OSTEOARTHRITIS OF THE HUMAN SKELETON: AN EVALUATION OF AGE, ACTIVITY, AND BODY SIZE IN LOAD-BEARING JOINT REGIONS

by

Stephanie Elizabeth Calce
B.Sc., M.Sc., University of Toronto, 2010

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Supervisory Committee

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Dr. Helen Kurki, Department of Anthropology
Supervisor

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Departmental Member

Dr. Darlene Weston, Department of Anthropology, University of British Columbia
Outside Member

Abstract

Supervisory Committee

Dr. Helen Kurki, Department of Anthropology, University of Victoria
Supervisor

Dr. Lisa Gould, Department of Anthropology, University of Victoria
Departmental Member

Dr. Darlene Weston, Department of Anthropology, University of British Columbia
Outside Member

Osteoarthritis (OA) is the most common joint disease in human populations with onset and severity influenced by mechanical loading, aging effects, genetics, anatomy, and body mass. Despite major advancements in knowledge, the aetiopathogenesis of OA is complex and still poorly understood. Lack of standardization in methods to quantify skeletal OA make it difficult to study the effects of interacting explanatory variables on arthritic response, and prevents comparison of results between bioarchaeological studies. Joint changes of OA as a function of both the natural aging process and of mechanical stress can make an individual appear older than their chronological age, potentially impacting current methods to derive accurate skeletal age at death estimates, particularly in load-bearing regions.

This project addressed these issues through three studies, using a large skeletal sample of modern Europeans for which sex, age, and occupation were available. The first study used principal component analysis (PCA) as a standardized procedure to compute aggregate scores for joint complexes and a systemic measure of OA in each region of the lumbar spine, pelvis, and knee. The second study analyzed the composite scores with a multiple regression model to determine the relative contribution of three predictors: age, activity, and body size, and their effect on skeletal expression of OA in each region. Body size (stature and mass) was calculated from postcranial skeletal measurements; torsional strength (*J*) of the femoral midshaft was calculated from three-dimensional surface models, size standardized and used as a proxy for measure of activity. The third study considered the effect of OA severity on the validity and reliability of three methods to estimate age at death from load-bearing joints of the os coxa: the pubic symphysis, auricular

surface, and acetabulum. The study was designed to determine whether OA in adults acts as a potential limitation or benefit in deriving accurate skeletal age at death estimates from pelvic joint morphology that will contribute to standardized methods in establishing physiological degeneration of the skeleton due to aging.

Body size and activity factors did not contribute significantly to OA pathology outside of the age-related expression in either of the lumbar vertebrae or knee regions, and only demonstrated a weak association at pelvic joints. Differences in adult patterns of age are reflected in joint arthritic changes of the os coxa and OA severity has an effect on the accuracy of age estimates from the pelvis; those with OA consistently aging faster in all three joint areas. This influence is most significant for young individuals at the auricular surface and pubic symphysis, over-aging at both. Oldest persons with little arthritic patterning at the acetabulum were under-aged, but accuracy of the age estimate improved as OA severity increased. Systemic measures of OA determined through PCA as an indicator of age, appear useful to identify the very old, but may also help to distinguish between systemic age-related stresses and localized biomechanical effects. Interpreting OA as evidence for old age, measures of habitual activity, and larger body mass should be exercised with caution in skeletal populations.

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For Eloise

❤

Because the dreams that you dare to dream really can come true.
Reach beyond your rainbow bum-bums.

Chapter 1.
Introduction

1.1 Aims and objectives

Interpreting skeletal expression of disease has significant theoretical applications in bioarchaeology when aiming to construct the biology, behaviour, ecology, and social structure of past populations. Osteoarthritis (OA) is the most commonly found pathological condition in archaeological collections and the most frequent musculoskeletal disorder in contemporary populations with onset and severity highly correlated with advancing age and activity (Felson et al., 2000). Early paleopathological research of OA focused heavily on description and classification (Rogers, 1966; Ortner, 1968; Rogers et al., 1987), noting the distribution of progressive degenerative qualities and attributing increased prevalence to specific behavioural interpretations (Jurmain, 1977; Merbs, 1983; Bridges, 1985). Clinical data from the last decade show that OA can affect varied joint tissues, is regenerative, reparative and highly linked with mechanical use (Felson and Nevitt, 2004; Dieppe, 2011); and osteologists have since recognized the diverse aetiology of OA by exercising caution in interpreting specific activities from bone pathologies (Jurmain et al., 2012).

Despite its long history of study in biological anthropology, methods for defining joint arthritic expression and for analysis of OA data vary widely in the literature (Rogers, 1966; Jurmain and Kilgore, 1995; Rothschild, 1997; Klaus et al., 2009; Molnar et al., 2011; Nikita et al., 2013). Lack of standardization in methods to quantify skeletal OA makes it difficult to compare results between studies, and to significantly study the effects of interacting explanatory variables (e.g., sex, age, activity, body size) on osteoarthritic response. Bioarchaeological studies must examine the simultaneous impact of multiple underlying factors on the expression of OA in a meaningful way to improve behavioural reconstruction and the interpretation of disease determinants in the past, particularly when the causative factors that produce OA are unknown (see section 1.3.2 for the difference between two subcategories of 'idiopathic' and 'secondary' OA).

Also, cumulative or abnormal mechanical loading, changes in anatomical alignment, and movement are capable of producing greater severity and distribution of joint osteoarthritic lesions (i.e., osteophytes, porosity), which are also known to co-vary with age (Lovejoy et al., 1985; Spector et al., 1996; Loeser, 2010) and can make an individual's skeleton appear older than their chronological age. These variables may have a significant impact on our current methods to derive

accurate skeletal age at death estimates based on morphology of joint structures, especially in load-bearing joint regions affected by mechanical stress. Assessing the role of arthritic features in estimating adult age is necessary because we do not yet know how to separate age from mechanical effects in analyses of physiological degeneration due to OA.

The purpose of this study is to determine the effects of idiopathic OA on skeletal age markers through an examination of age, body size, and bone robusticity in three modern European skeletal samples of adults of known sex, age, and occupation (N=289). This research addresses the following three questions: 1) How should we quantify OA severity that will accurately explain the cumulative effects of age and mechanical stress on bone quality? 2) How much of the variation associated with OA can be explained by age, stature, body mass, or structural adaptation related to habitual use? And 3) Does the presence of OA affect our ability to accurately estimate age at death from the adult skeleton?

The remainder of this chapter provides a background on OA in biological anthropology and the contribution of the current project to this field. It begins by reviewing normal joint anatomy and the pathogenesis of OA, including a discussion that contrasts clinical and paleopathological diagnoses of the condition. It then reviews in some detail, what we know of its causal mechanisms and how OA has been used in bioarchaeological analyses to interpret activity, variation in anatomy, aging, and sex-based roles in the past. A basic introduction of skeletal biology related to aging is presented, followed by a relevant discussion of how age-progressive arthritic traits are used in our current methods to estimate age at death from the human skeleton. The necessity to formulate a meaningful representation of arthritic severity from skeletal analysis is presented. The chapter concludes with a brief outline of the dissertation and the three papers that comprise it. Details of the skeletal populations analyzed for this study are outlined in Chapter 2, *Skeletal material*.

1.2 Normal joint anatomy and movement

Bones of the skeleton are inflexible, and movement can only occur at joints. Each joint reflects a compromise between the need for strength and the need for mobility. Also known as articulations, joints of the human body are specialized in anatomical shape and structure to control for range of motion between connecting surfaces (Carter and Beaupré, 2001). There are three functional classes of joints differentiated by histological features and allowable range of motion: fibrous, cartilaginous (primary, secondary), and synovial joints.

Fibrous joints (synarthroses) are relatively immoveable joints held together by fibrous connective tissues, but lack cartilage and a cavity between bones. These synarthrotic joints are extremely strong, with examples including interlocking cranial sutures and the distal articulation of the tibiofibular joint. Cartilaginous joints (amphiarthroses) permit slight joint movement where bones are separated by a plate of cartilage. There are two types: (1) primary cartilaginous joints bridged by hyaline cartilage and only capable of limited movement, e.g. between the rib ends and the sternum, and (2) secondary cartilaginous joints separated by a wedge or pad of fibrocartilage and permitting more movement, e.g. public symphysis or intervertebral disks of the spine, where each individual vertebral joint provides only slight movement and collectively the vertebral column demonstrates a high degree of mobility (Gosling et al., 1995).

Synovial joints (diarthroses) are freely moveable complex structures held together by fibrous connective tissues and cartilage. Bone ends are covered by a protective layer of dense hyaline cartilage and encompassed by both a fibrous capsule and synovial membrane that is supported by ligaments to form the joint cavity. The joint cavity contains a clear, slightly yellow viscous synovial fluid that is secreted by the membrane and has three primary functions: (1) to provide joint lubrication, (2) to distribute nutrients for the articular cartilage, and (3) for shock absorption (Waldron, 2009; Dieppe, 2011). Accessory structures, such as ligaments, reinforce synovial joints for additional stability and may either pass outside (extra-capsular) or inside (intra-capsular) the joint capsule. Synovial joints comprise the majority of joints in the body and permit the highest range of motion. A joint cannot be both highly mobile and very strong. As a result, the greater the range of motion at a joint, the weaker it becomes. Synovial joints are often classified according to the shape of the joint surface (e.g., plane, saddle, ball and socket), or by the type of movement they permit (e.g., sliding, hinge, pivot) (Gosling et al., 1995; Palastanga, 2002). Examples of these include the elbow, hip, knee, shoulder, wrist, and small articulations of the hands.

Joint cartilage has several functions. It distributes load over a wide area thus reducing contact stress; provides protective lubrication that minimizes friction and mechanical wear at the joint; protects the joint periphery; improves joint fit by limiting slip between articulating bones; and absorbs shock (Carter and Beaupré, 2001). Articular joint cartilage is transparent in radiographs and the apparent displacement between the ends of the bones is referred to as the joint space. The subchondral bone plate, made of thin cortical lamellar bone, sits immediately beneath the articular cartilage and is supported by trabeculae. Subchondral trabecular bone exerts important shock absorbing and supportive functions in normal joints and may also be important for

cartilage nutrient supply and metabolism (Li et al., 2013). Articular connective tissues such as tendons (connect muscles to bones) and ligaments (connect bones to bones) promote joint stability, i.e., the ability of the joint to resist abnormal displacement of articulating bones. In normal non-pathological joints, articulating surfaces move with remarkably little friction, the physiology of the joint is dynamic and it is capable of considerable repair (Jurmain, 1999; Waldron, 2009; Dieppe, 2011).

1.3 Osteoarthritis

Joint disease was initially differentiated into two main groups based on observable pathological features of (1) bone erosion and (2) bone hypertrophy. Erosive rheumatic diseases (e.g., septic and rheumatoid arthritis) were found to occur predominantly in young persons and were classified based on their extreme inflammatory response. Hypertrophic tissue manifestations associated with older age and deteriorating joint cartilage, became known as "degenerative joint disease," or "osteoarthritis" (Brandt et al., 2009). Great progress has been made in distinguishing the group of erosive joint diseases but the same developments have not progressed for OA (Weiss and Jurmain, 2007).

Osteoarthritis is a complex proliferative bone condition that has been documented in human populations as one of the most common skeletal pathologies (Karsenty, 2003; Ortner, 2003; Felson and Nevitt, 2004). Despite extensive study, relatively little is known about its causal mechanisms and origins, except that OA slowly evolves from some combination of systemic and local biomechanical risk factors to alter the anatomy and matrix composition of articular cartilage and the bone underneath it (Brandt et al., 2009). The poorly understood aetiopathogenesis of OA suggests that it is not a single disease, but a heterogeneous cluster of conditions that lead to similar clinical and pathological alterations (Altman et al., 1986; Dieppe, 1990; Felson et al., 2000; Sowers, 2001). This fact makes OA a particularly stimulating study subject as we try to define it pathologically and to understand the multifactorial reasons for its widespread prevalence.

1.3.1 The pathogenesis of osteoarthritis

The pathogenesis of OA includes chronic, inflammatory and degenerative changes in joints with cartilage components as a result of confounding effects such as aging, mechanical stress, genetic susceptibility, normal anatomical variation, joint injury, and body size (Radin et al., 1972; Merbs, 1983; Jurmain, 1999; Ortner, 2003; Spector and McGregor, 2004; Weiss and Jurmain, 2007). Regarded as whole-joint failure, OA may originate in any and all tissues surrounding a joint including cartilage, subchondral bone, ligaments, periarticular muscles, and/or the synovium (Dieppe, 2011). Breakdown of contiguous joint tissue causes bone surfaces to rub together, resulting in pain, stiffness, restricted range of motion, and eventual loss of joint use (Jurmain and Kilgore, 1995; Tepperman, 1981).

Generally, the earliest changes of OA occur in articular cartilage. Cartilage degeneration is characterized by two phases of cell metabolism. In the biosynthetic phase, chondrocytes (cartilage cells) attempt repair of the extracellular matrix that has become damaged by metalloproteinases (Sandell and Aigner, 2001; Visse and Nagase, 2003). Enzymes produced by the chondrocytes attack the matrix and erosion of cartilage is accelerated in the degradative phase. Cartilaginous tissue degeneration continues when biosynthetic anabolic activity can no longer keep pace with the degradative catabolic activity (Sandell and Aigner, 2001).

In subchondral bone, microtrabecular fractures and their subsequent healing increase stiffness, transmitting increased load to overlying cartilage that contributes to its damage and reduces its function as a shock absorber (Felson and Neogi, 2004). Reactive bone formation in trabeculae underlies porous degenerative changes visible on the bone surface and is possibly associated with cyst formation (Hough, 2001; Ortner, 2003). Increased osteoclast activity just below articular cartilage may lead to perforations of the subchondral bone plate (porosity) that becomes more permeable as a result, causing a higher than normal fluid exudation and leading to a net loss of fluid from the cartilage, which subsequently becomes damaged (Botter et al., 2011). Eventually, progenitor cells are activated as a result of tissue injury, which leads to secondary cartilage formation that ossifies into bone overgrowth at both the joint margin and on the articular joint surface (i.e., osteophytes), as well as other hypertrophic bony responses, such as sclerosis of the subchondral plate (eburnation), which becomes thickened (Sandell and Aigner 2001; Felson and Neogi, 2004; Klaus et al., 2009).

Osteophytes grow in both horizontal and vertical directions to produce a change in the shape of the joint contour (widening or flattening) (Rogers et al., 1987; Jurmain, 1990). Multiple proliferative osteophytic spurs originating at the joint rim may grow towards each other and eventually fuse (Larsen, 1997; Ortner, 2003). Following the total loss of joint cartilage, eburnation results from the rubbing (or contact) of two bone surfaces and leads to polishing and mechanical scoring (grooves) parallel to the line of motion observable on the subchondral compact bone surface (Klaus et al., 2009).

The pathogenesis of OA involves complex interactions between joint anatomy, physiology, biochemistry, and biomechanical function, arising from the attempt to repair damage driven by abnormal joint loading. Osteoarthritis has been conceptualized as a disease of both bone and cartilage, though it is not clear which of these is the primary triggering organ. Regardless, OA is not a discrete disease with a common pathophysiologic pathway. For example, both clinical and longitudinal experimental studies have shown that subchondral microcysts precede cartilage damage and may be the first sign of OA (Botter et al., 2011; Binks et al., 2013; Sulzbacher, 2013). Porosity may occur in isolation or in association with eburnation (Hough, 2001). There is some research to suggest that ossification at insertion sites of ligaments, tendons and joint capsules (enthesophytes) may also define OA (Rogers et al., 2004). Finally, discordant evidence from animal experiments where OA was induced (Radin et al., 1973; Dedrick et al., 1993) suggests that cartilage loss and bone sclerosis are two independent consequences of increased mechanical stress (Felson and Neogi, 2004).

1.3.2 Idiopathic vs. secondary osteoarthritis

Traditional subcategories of OA, (1) idiopathic and (2) secondary, are used to identify localized and generalized causative factors. Idiopathic OA involves patients with no underlying predisposing factors, whereas secondary OA is associated with a known event or underlying condition, which may include trauma (e.g., bone fracture, ligament injury), inflammatory disease (e.g., rheumatoid arthritis), metabolic conditions (e.g., diabetes), or congenital deformations (e.g., hip dislocation, extreme valgus/varus misalignment). As all OA may be secondary to phenomena not yet discovered, the term ‘idiopathic’ OA has been recommended to replace ‘primary’ in classifications of OA subsets (Altman et al., 1986; Jurmain, 1999; Brandt et al., 2009).

1.3.3 Osteoarthritis in load-bearing skeletal regions

Osteoarthritis occurs most often in the spine, knee, hip, and hands as a result of the concentration of force across the joint and the rate of mechanical loading. The effects of differential loading will produce variable appearances of OA in bone, which is significant because joints that do not move do not develop OA (Jurmain, 1991; Carter and Beaupré, 2001; Waldron, 2009). Both too little and too much mechanical stress seems to promote development of OA (Brandt et al., 2009; Dieppe, 2011), and severe bone expressions increase with age, but are less common in adults younger than 40 years (Ortner, 2003). This study focuses on arthritic traits directly observable on bone surfaces of the lumbar vertebrae, pelvis, and knee that serve as stabilizing and load-bearing joint regions responsible for mobility and movement. Each area is examined separately in the following analyses because OA will manifest differently in varied joint locations of the body based on function and load-type. For example, eburnated surfaces of the ball and socket joints of the hip and shoulder appear smoothly polished similar to porcelain, or the shine of a bowling ball (Fig. 1.1). Whereas areas in hinge-like joints, such as the elbow or knee, the polished surface is accompanied by deep parallel grooves (Fig. 1.2); and in joints of the spine with less overall rotational movement, eburnation may be minimal or absent (Ortner, 2003).

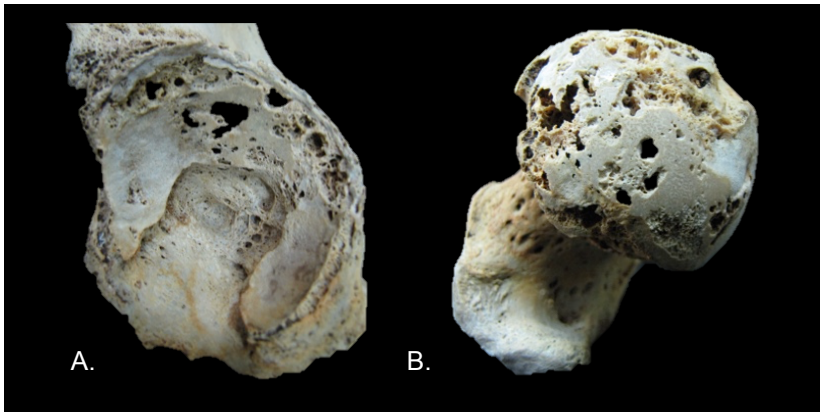


Figure 1.1. Polishing of the acetabulum (A) and femoral head (B). Photos A and B: Athens 56, Male 39 years.

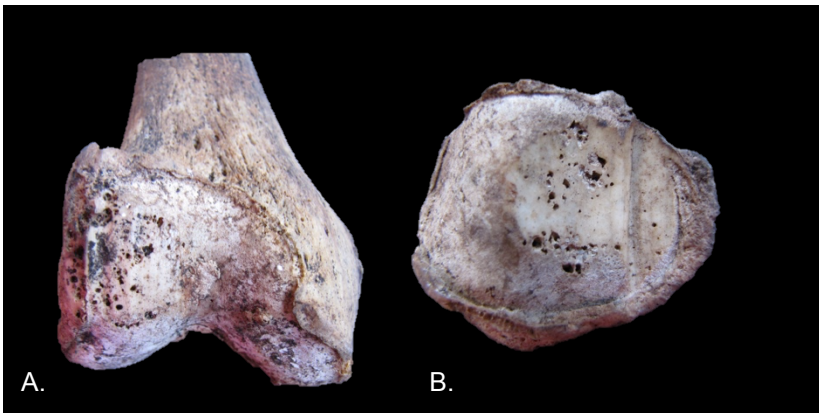


Figure 1.2. Parallel grooves in the distal femur (A) and patella (B). Photos A and B: Lisbon 351, Female 90 years.

Arthritic changes at fibrocartilaginous joints of the spinal column typically exhibit a greater degree of marginal osteophytosis in comparison to joints of the hip and/or knee regions, which move more freely (Jurmain and Kilgore, 1995; Gold et al., 2007). Some studies classify modifications of vertebral body margins as 'vertebral osteophytosis', or 'spondylosis' reserving the term 'osteoarthritis' for strict synovial joint involvement (Bridges, 1994; Jurmain and Kilgore, 1995; Sofaer-Derevenski, 2000), but following the lead of other paleopathology researchers (Larsen et al., 1995; Lieverse et al., 2007; Klaus et al., 2009; Waldron, 2009) degeneration of the intervertebral disks are subsumed under the term OA in this study. In fact, facet joints of the spine are considered true synovial articulations that slide upon one another in a cranio-caudal direction to stabilize flexion and extension movements (Lewin, 1964). Intervertebral synovial joints are quite flexible to enable bending and twisting of the body, and undergo degenerative changes identical to those of OA seen in other synovial joint areas (Lewin, 1964). Also important is the differential diagnosis of spondyloarthropathies that may appear in concert with OA, such as diffuse idiopathic skeletal hyperostosis. Several studies have demonstrated that osseous change related to OA is the most frequent and severe in the lumbosacral region (Bridges, 1994; Sofaer-Derevenski, 2000). Peak involvement of the lumbosacral segment occurs around the points of maximum curvature of the spine, while minimal wear is associated with cervical and thoracic vertebrae lying along (or behind) the plane of the center of gravity. Articular facets of the lumbar vertebrae and sacrum are prone to OA due to the dorsiflexed nature of the lower spine, their position in front of the line of center of gravity, and significant load-bearing capacity as a result of bipedality (Lewin, 1964). In particular, arthritic patterning is commonly observed in the lumbosacral joint (L5-S1) (Fig. 1.3), which allows for considerable rotation so that the pelvis and hips may swing when walking and running.

With persistent loading of the hip and knee during locomotor tasks, OA is the most common reason for total hip and knee replacement (Felson et al., 2000). With the development of OA at the hip, the convex joint surface of the femoral head is enlarged and deformed; its curvature resembling a mushroomed effect formed from marginal exostoses that overhang the femoral neck (Fig. 1.4a). Chondral defects such as eburnation and erosion are most marked on the superior aspect of the femoral head, while osteophytes develop in the intracapsular portion of the neck where lytic lesions are also common. In the acetabulum, marginal lipping, osteophyte development of the posterior horn of the lunate surface, and deepening of the socket are observed, as is sclerosis of the acetabular roof, which may contain cystic cavities (Fig. 1.4b).

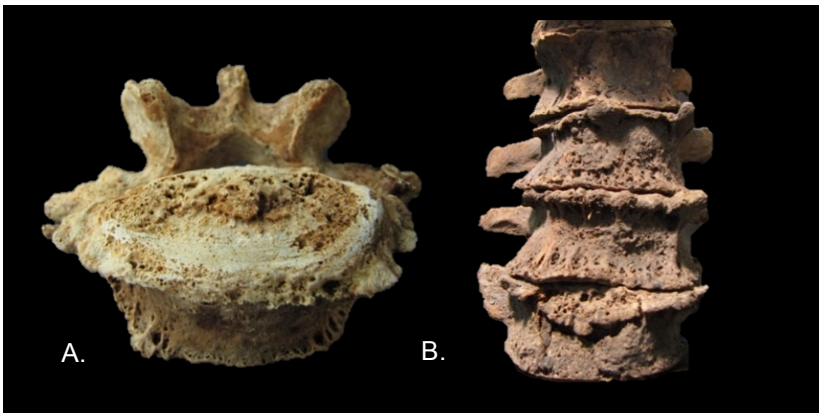


Figure 1.3. Marginal osteophytes of the lumbar spine. Photo A: Athens 150, Female 82 years. Photo B: Sassari 27, Male 82 years.

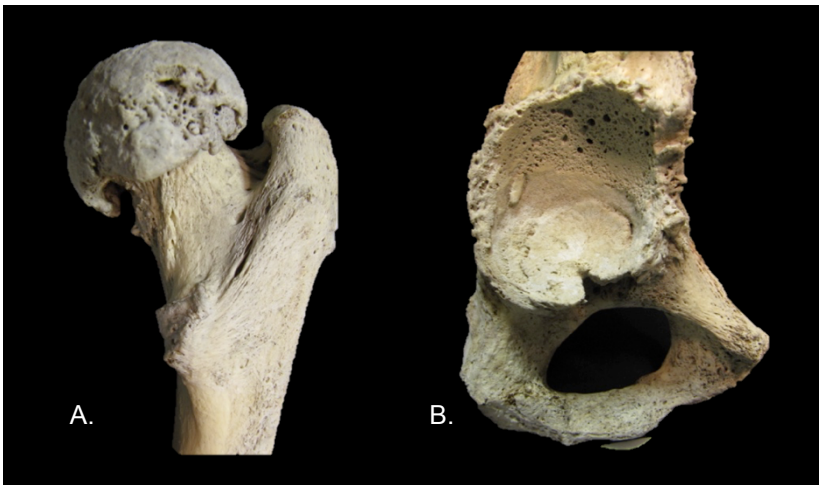


Figure 1.4. Mushroom effect of the femoral head (A) and degeneration of the acetabular surface. Photos A and B: Sassari 308, Male 69 years.

Synovial-lined articulations of the sacroiliac are highly specialized joints that permit stable (yet flexible) support to the upper body (Vleeming et al., 2012). Functionally, the pubic symphysis resists tensile, shearing and compressive forces and is also capable of a small amount of movement, which may become accentuated during pregnancy from hormonal influences that result in softening and relaxation of surrounding ligaments (Resnick et al., 1977; Gamble et al., 1986; Becker et al., 2010). Biomechanically, movements at both the sacroiliac and pubic symphyseal joints are induced by motions occurring at other locations in the body; however, both joint areas are subject to a variety of internal and external forces that lead to marginal lipping, cystic lesions (presenting as either micro- and macro-porosity of the bone surface), degeneration/erosion and ankylosis (Resnick et al., 1977; Lovejoy et al., 1985; Campanacho et al., 2012) (Fig. 1.5). In particular, pelvic stress injury (e.g., osteitis pubis) and sacroiliac abnormalities can lead to coarsely porous new bone remodelling, and eburnation on any of the six articular faces of the pelvis (Major and Helms, 1997; Judd, 2010; Pfeiffer, 2011). Repetitive, strenuous, and prolonged physically demanding activity may contribute to pelvic joint degeneration, though differential diagnoses to rule out ankylosing spondylitis, Reiter's syndrome, rheumatoid arthritis, tuberculosis and trauma as contributing to the rate of degeneration are advised (Ortner, 2003).

Frequent and severe knee OA forms as a result of joint stress during flexion, hyperextension, and adduction (Felson et al., 2000). The knee joint is exposed to high contact and shear forces with compressive loads in excess of three-to-six times body weight during walking, running, and stair climbing. Severe sclerosis can occur in any of the three major compartments of the knee: medial, lateral, and patellofemoral. Arthritic patterning, and especially articular surface osteophytes are more common in the medial tibiofemoral and patellofemoral compartments, but less so in the lateral tibiofemoral area (Fig. 1.6).

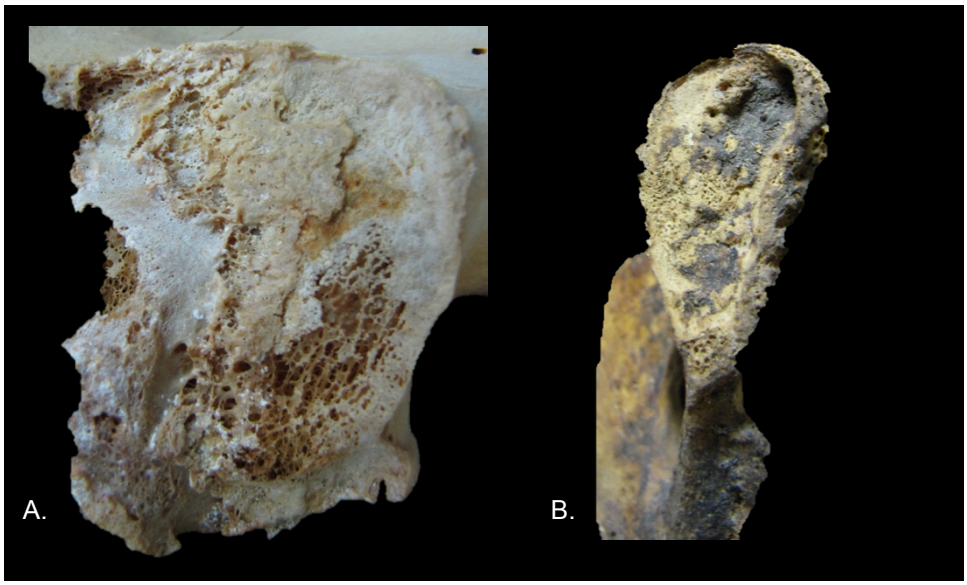


Figure 1.5. Marginal lipping, erosion, and surface osteophytes of the auricular surface (A) and pubic symphyseal face (B). Photo A: Athens 149, Male 66 years. Photo B: Sassari 25, Male 73 years.

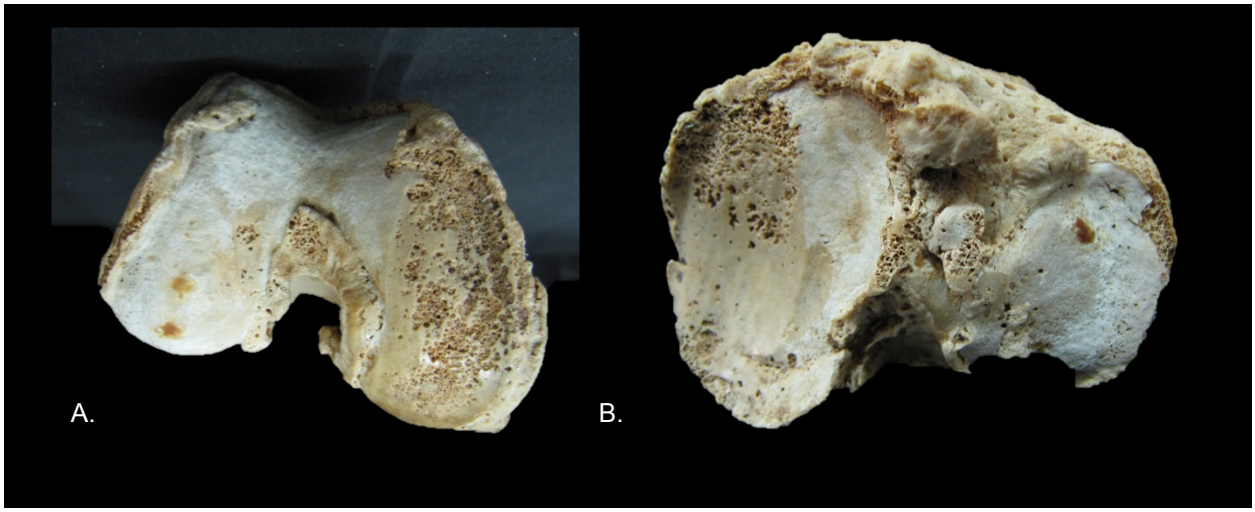


Figure 1.6. Eburnation and surface osteophytes of the medial compartment in the distal femur (A) and proximal tibia (B). Photos A and B: Athens 012, Female 84 years.

1.3.4 Clinical vs. paleopathological diagnosis

Both clinicians and paleopathologists are at a disadvantage when studying OA, since neither has the whole picture of what constitutes joint pathology. Joint pain and radiological diagnostic criteria (e.g., joint space narrowing or marginal osteophytes) serve as clinical indicators of arthritic severity (Kellgren and Lawrence, 1957), but the two are not correlated. Patients with radiological features of acute OA may be asymptomatic, whereas individuals without radiological evidence may experience debilitating pain (Altman et al., 1986; Dieppe, 2011). Without a clear understanding of arthritic progression and its affects on both articular cartilage and the underlying bone surface, efforts to standardize and clarify the clinical definition of OA remain a challenge.

In the absence of soft tissues, diagnostic dry-bone OA criteria are informed from clinical sources, but adapted to suit the circumstances of paleopathology where OA prevalence is likely under-represented (Ortner, 2003; Mays, 2012). Because of bone's limited response to disease (i.e., forming or resorbing), specific diagnoses on the basis of skeletal lesions alone are difficult, and likely to reflect the later stages of the condition or its more severe manifestations (Ortner, 2003). In this context, 'severity' describes the progression of skeletal lesions, rather than disease duration, impact on quality of life, or endured pain that are typically measured in clinical investigations of joint pathology. For example, in archaeological skeletal analyses eburnation has been regarded as the most reliable indicator of severe OA because it follows end-stage cartilage degeneration and bone-on-bone contact (Weiss and Jurmain, 2007). In the absence of eburnation, Waldron (1991) suggested that the presence of any two of the following factors can be used to classify a joint as osteoarthritic: (a) new bone around the joint margin, (b) new bone on the joint surface, (c) pitting on the joint surface, or (d) deformation of the normal joint. Rogers and Waldron (1995) and Weiss and Jurmain (2007) have recommended that osteophyte formation alone should not be taken as evidence of OA, because this process is also related to the natural course of biological aging. Clinically, the presence of polyarticular osteophytes (visible in x-ray) distinguishes idiopathic joint OA from other arthritides more than any other pathological feature (Altman et al., 1986; Sadnell and Aigner, 2001).

Differences in diagnoses highlight the inherent multidisciplinary nature of OA, which must be investigated using varied data sets. Despite the limitations of gross anatomy and patient history, paleopathologists do have a significant advantage in their ability to examine the whole extent of

joint abnormalities. Such detailed information on the type and distribution of arthritic joint lesions are impossible to obtain in a living patient (Ortner, 2003; Rogers et al., 2004). The medicohistorical approach of paleopathological analyses is helpful to understand the natural progression of OA from a populational perspective. The purpose of identifying and evaluating stress in a skeletal sample helps to illuminate the general burden of disease in a population as well as physiological adaptations. This differs significantly from typical clinical examinations of case-by-case joint abnormalities, where the aim is to provide disease prognosis and to instigate effective treatment.

1.4 Paleopathology and aetiology of osteoarthritis

A full understanding of the OA disease process has not been reached. Nor has agreement on the theoretical and methodological context for interpreting the meaning of arthritic data in terms of health and activity of past human populations. The following section will present a short summary of different causal mechanisms known to affect arthritic expression, followed by examples of how bioarchaeologists have investigated varied aetiological factors of OA to understand the lives of past populations. This section also includes a discussion of varied techniques to calculate aggregate measures of skeletal OA and concerns for the limitations of such in analyses of OA pathology.

1.4.1 Multifactorial nature of osteoarthritis

Onset and severity of OA are most closely correlated with advancing age and activity and OA is generally accepted as a multifactorial disease with multiple causes (Jurmain, 1999; Weiss and Jurmain, 2007; Waldron, 2009). Complexities of the condition arise from the interplay between systemic influences (e.g., age, sex, hormones, nutrition, genetics) and local biomechanical risk factors (e.g., muscle weakness, obesity, physical activity). The most significant intrinsic factor is age, leading to biochemical and histological changes to both articular cartilage and to the underlying bone that affect joint morphology (Jurmain, 1991; Loeser, 2010). Aging cartilage is less capable of resisting the forces of repetitive mechanical loading and is therefore more susceptible to microtrauma resulting in early stages of arthritic development (Resnick and Niwayama, 1995; Ferrucci et al., 2002). While the greatest risk factor for OA is older age, OA is not an inevitable

consequence of growing old. Early onset of OA-related traits has been observed in young individuals, not all older adults develop OA, and not all joints of the body are affected to the same degree. This evidence supports the idea that joint arthritic patterning is primarily a mechanical response to loading, either from natural wear-and-tear (a cumulative response), or from short, intense periods of abnormal loading, i.e., obesity, trauma, or muscle weakness (Jurmain, 1999; Dieppe, 2011). Large body size correlates with generalized bone hypertrophy (Spector et al., 1996) and the positive relationship between body mass and severe OA in load-bearing joint regions (for example, the hip, knee and ankle) has been demonstrated extensively in living populations (Srikanth et al., 2005; Sandford et al., 2014). Anatomical variation may be important, but conflicting evidence in the evaluation of hinge joints in archaeological samples (Plomp et al., 2013) has failed to confirm the relationship between joint shape and OA that has been observed in clinical studies (Shepstone et al., 1999; 2001). There is also research to suggest that there is a genetic or heritable component (Spector et al., 2004; Gestsdóttir et al., 2006).

1.4.2 Bioarchaeological analyses of osteoarthritis

The history of paleopathological investigations of OA dates back to the mid-20th century when J. Lawrence Angel and his then graduate student, Donald J. Ortner described a curious condition of the distal humerus, "atlatl elbow" related to spear-throwing, in three different hunter-gatherer populations from Alaska, Peru, and California (Angel, 1966; Ortner, 1968). Ortner attributed remodelling and degenerative changes at the elbow to a combination of factors that included age, anatomy, and type and intensity of use. Using patterns and distribution of OA in other joints of the body to describe activities between populations became very popular in the decades that followed. Landmark studies by Jurmain (1977) and Merbs (1983) tying prevalence of arthritis to specific activities, such as harpoon throwing and kayak paddling (in men) and domestic cleaning and hide-preparation for clothing (in females), spurred a number of investigations to determine indicators of activity levels in archaeological skeletal samples from OA, including sex-based behavioural differences. Bridges' (1991; 1992; 1994) analyses of more than 25 skeletal samples of Amerindians found a varied patterning of OA among major appendicular joints, suggestive of more broadly-based behavioural interpretations. Bridges proposed that previously linking OA with specific activities might be too, "simplistic" (1992:85), and that high-intensity, infrequent forces

may be more important to the development and progression of arthritic characteristics, rather than low-level habitual activities. As a deeper understanding of the complex aetiology of OA ensued, interpretations based entirely on activity as a causative factor have waned, though some more recent studies continue to interpret very specific behaviour patterns from arthritic prevalence (Lieverse et al., 2007; Klaus et al., 2009).

Research on the effects of mechanical stress and physical activity as major contributing factors to the expression of OA severity in archaeological samples is abundant (Jurmain, 1999; Sofaer-Derevenski, 2000; Lieverse et al., 2007; Klaus et al., 2009; Shrader, 2012; Lieverse et al., 2015). While specific habitual activities are not likely informed through an examination of musculoskeletal disorders (Jurmain et al., 2012), it is possible to use OA as a general marker of occupational stress to approximate how humans interact with their environment. Support for the "stress hypothesis" has been demonstrated widely (cf. Weiss and Jurmain, 2007), proposing that OA develops more-so in one population over another due to higher levels of biomechanical stresses on the joint, and particularly if these stresses begin early in childhood (for example, individuals working as children in physical activities related to farming and agriculture). However, because of the complex multiple aetiologies associated with OA, different joints, or even different populations may not respond in the same way to similar stresses (cf. Weiss and Jurmain, 2007; Gestsdóttir, 2014). This interpretive framework combines skeletal studies and principles of biomechanics (e.g., Wolff's Law) with ethnographic and archaeological evidence to suggest that differences in OA prevalence are informative about activity in the past.

Much of the focus on OA in osteoarchaeology has been to prove or disprove the influence of *one* aetiological factor. For example, Weiss (2006) considered the effects of body mass on OA in a Californian hunter-gatherer population (500-1500 AD), an important study given that obesity is a known risk factor for OA (Sanford et al., 2014) and because body size, especially body mass will affect how bone responds to external forces (Merritt, 2015; Wescott and Drew, 2015). Unfortunately, the pattern demonstrated by Weiss' study mainly reflects sex-specific size variation, rather than a true estimation of body mass differences in OA severity and expression. Plomp et al. (2013) examined the relationship between joint shape and OA in the elbows and knees of 147 individuals from 4th-19th century England, but contrary to results from longitudinal studies (Miyazaki et al., 2002), they found that (1) joint shape was unlikely to influence the development of OA, (2) shape changes produced by OA were not systematic or quantifiable, and (3) no particular joint shape was predisposing to the condition. This is significant and contrary to results by Miyazaki

et al. (2002), where the medial compartment of the knee has been shown to change structurally based on varus deformity (malalignment responsible for increases in knee adduction moment). Klaus et al. (2009) associated all arthritic patterning and changes in OA prevalence among native Peruvians (AD 900-1532) to activity alone, providing a very detailed context of regional economic intensification in mining, agriculture, and pastoralism as the only explanation for the complex distributions of OA observed in their sample. While each of these studies brings us closer to understanding normal variation in anatomy and the role of biomechanical factors in tissue degeneration, we still do not know whether all types of altered joint loading share a common pathway of trait onset and progression. If the key to understanding OA is abnormal mechanical stress (i.e., overuse, immobilization, joint instability from muscle weakness) (Bridges, 1992; Dieppe, 2011), then what are the circumstances in which its contribution can be determined? With new powerful research tools and sophisticated statistical techniques, bioarchaeologists can also contribute to diagnostic considerations of OA to determine the relative impact and interaction of *multiple* underlying factors that shape OA pathogenesis.

1.4.3 Calculating and interpreting arthritic severity from skeletal remains

Unlike other diseases, OA is not clearly associated with mortality risk; that is, people do not die from OA. Rather, it is the presence of co-morbid conditions that increase the risk of death in individuals with OA. For example, obesity may lead to walking disability (or immobility) that increases the risk of death from cardiovascular causes (Nüesch et al., 2011). As a result, reconstructing the pathological process of OA in bioarchaeology is difficult because skeletal pathologies are only presented at a single point in the disease process, arrested at the time of death, and not necessarily related to the cause of death. In this way, health and differential risk of illness (and of death) are poorly understood, both of which can impact the makeup of a skeletal assemblage which may not adequately reflect a randomized subset of the parent population (Wood et al., 1992).

Osteoarthritis is typically reported as a measure of both prevalence (number of OA cases/number of individuals in the sample) and risk, calculated as odds ratios, a summary statistic that expresses the overall difference in prevalence between two populations as an age-related proportion (or some other grouping); its significance assessed by a chi square test. As Baker and

Pearson (2006) point out, these tests may produce misleading or inflated statistical significance in bioarchaeological studies where it is necessary to standardize population units but often unclear whether a small skeletal series adequately represents the larger population of interest. Other problems such as preservation bias, may introduce sampling error and individuals for whom joint surface morphology is preserved may not be representative of their age group or pathological state. Waldron and Rogers (1991) demonstrated that inconsistent descriptive terminology leads to large inter-observer error in estimates of OA status (mild, moderate, severe interpretations), a reflection of an imperfect relationship between bony indicators and true physiological ages or OA pathological states.

Biocultural interpretations of OA have been hindered by a lack of appropriate techniques for reducing and interpreting large volumes of OA data. Twenty years ago, researchers identified the need for a more scientific approach to paleopathological analysis with increased emphasis on standardized methods of recording OA to ensure comparability of data from different studies, as well as an increase in statistical testing of results to glean whether these can be seen as meaningful (Bridges, 1993; Jurmain and Kilgore, 1995). In response, several coding systems were developed (Jurmain, 1990; Waldron and Rogers, 1991; Buikstra and Ubelaker, 1994; Jurmain and Kilgore, 1995) to record varied information related to changes in bone quality and differences in OA severity, but none of which have been adopted by all researchers. Buikstra and Ubelaker's (1994) seminal publication on documenting osteological data (referred to simply as, "Standards") included a uniform OA coding scheme to document separate arthritic traits (lipping, porosity, eburnation, surface osteophytes), and remains to this day the most detailed evaluative method encouraging two separate measurements of (1) OA severity (progression of traits) and (2) distribution (the amount of surface area affected). Though no 'gold standard' of recording OA has been adopted, Buikstra and Ubelaker's (1994) method is cited often in recent bioarchaeological investigations of OA (Klaus et al., 2009; Shrader, 2012; Lieverse et al., 2015) and is also employed in this study.

Still, the large database of raw observations produced by Buikstra and Ubelaker's coding system is nearly impossible to interpret without some manipulation of the data into representative joint scores, and unfortunately there is no agreement among researchers on how to effectively summarize or determine the number of joint surfaces that may comprise a joint complex. This can have a significant impact on interpreting results given that the more joint surfaces included, the smaller the average score will be (Bridges, 1993). Since OA may be a systemic disorder of bone (Rogers et al., 2004), we need a systemic measure of OA that accounts for heterogeneous wear

patterns across multiple joint complexes. Understanding the mechanobiology of moveable joints in past populations is critical to test the assumption that OA pathology is a reflection of behaviour; but to do so we need (1) a reliable method to quantify OA expression and (2) a tool to measure both individual variation (by skeletal region) and population differences in biomechanics and biology.

1.5 Skeletal biology, aging, and osteoarthritis

While the greatest risk factor for OA is older age, OA is not an inevitable consequence of growing old. Aging is not a causal mechanism for OA, but age-related changes that affect both joint function and surrounding joint tissues increase susceptibility to developing OA in older adults, especially when other factors (e.g., joint injury, obesity, genetics) are also present (Sowers, 2001; Anderson and Loeser, 2010). Since changes both outside the joint (e.g., sarcopenia) and within the joint (such as cell and matrix alterations) contribute to the development of observable OA characteristics (osteophytes, eburnation, porosity), osteologists interpreting OA in past populations need to know the basic biology of musculoskeletal aging, informed through clinical immunology and rheumatology research, to truly appreciate the variation in arthritic severity.

The aging process of the musculoskeletal system contributes to OA pathogenesis in several ways, and includes chondrocyte senescence, oxidative damage, and gradual loss of the cartilage matrix. This includes age-related loss in the ability of cells and tissues to maintain homeostasis (Anderson and Loeser, 2010), particularly when placed under abnormal mechanical stress that aging joint tissues could not compensate for as easily as younger tissues (Ferrucci et al., 2002). As a person ages, major components of the cartilage extracellular matrix (ECM) undergo changes that alter its biomechanical properties by decreasing in size and structural organization (Goldring and Goldring, 2006). Also important in the increased susceptibility to OA is a reduced anabolic response to growth factors, which declines with age (Sharma et al., 2013). Chondrocytes, the only cells to inhabit the cartilage ECM, become limited in their regenerative capacity to remodel, repair, and respond to growth factors, igniting a disequilibrium between catabolic and anabolic activities that lead to accelerated cartilage tissue degeneration and eventually, the formation of osteophytes, as described in section 1.3.1 (Goldring and Goldring, 2006; Anderson and Loeser, 2010; Sharma et al., 2013).

In addition to understanding the cellular processes that regulate the functional activities of chondrocytes in cartilage integrity, age changes that affect bone as part of the pathophysiology of

OA are particularly important. A recent study by Burr and Gallant (2012) illustrates that the rate of bone remodelling differs across the course of the disease. An imbalance in bone turnover, favouring the rate of bone formation, leads to an altered bone structure with an increased volume of cancellous bone and the formation of osteophytes that can be used to identify the late stages of OA (Burr and Gallant, 2012). With age, the subchondral bone volume and trabecular thickness significantly increase as cartilage degenerates, while the number of trabecular strata and degree of trabecular bone separation decrease (Bobinac et al., 2003). Subchondral bone, highly vascularized with many nerves and blood vessels that nourish joint tissues, hardens in a process of sclerosis (see 1.3.1) (Cox et al., 2013). Age changes expressed at the cellular level are also expressed at the surface level, which comprise the changes in osteophytes, porosity, and eburnation that are evaluated by paleopathologists to assess the progression and severity of OA. Osteoarthritis results from a more complex biological process of matrix degradation than is described here, but is generally accepted as a classic age-related disorder in both clinical and paleoepidemiological interpretations.

1.5.1 Adult skeletal age estimation from osteoarthritic traits

Adult age estimation methods are created from rates of skeletal remodelling and periods of degeneration after skeletal maturity has been reached. Reconstructing the biological attributes of adult age is a critical, but difficult tool because no universal system of biological aging exists. Skeletal morphology is influenced by a combination of variables such as sex, ancestry, diet, mechanical forces, and environmental and genetic constraints that produce unilateral changes at a variable pace. As well, disease can make a person appear older than their chronological age. That said, age at death can be detected from skeletal elements, as is evidenced by the various skeletal regions that have shown to change predictably with age, such as the pelvic joints, ribs, and cranial sutures (İşcan et al., 1984; Meindle and Lovejoy, 1985; Lovejoy et al., 1985;). Degenerative arthritic changes may be particularly important data for establishing and narrowing age estimates for older individuals. Elderly populations are expanding globally, increasing as a result of declining fertility, improved health, and increased life expectancy (Kinsella and Velkoff, 2001; Ice, 2003). The ability to accurately model age in the elderly will be significant in forensic and bioarchaeological studies.

Predictable age-progressive morphological changes that include features of OA (marginal lipping, osteophytic outgrowths, marginal erosion, eburnation and pitting/porosity) have been

identified in various skeletal joints and incorporated into analyses of skeletal age at death. Table 1.1 lists skeletal methods known to incorporate age-related degenerative changes in the assessment of chronological age. As well, Sharman (2015) has demonstrated that evaluating arthritic traits as skeletal age indicators alongside published methods, improves the accuracy of the overall age estimate, particularly for adults 60+ years in her sample for whom osteophytes and OA in multiple joints became more common as age increased.

Table 1.1. Published age estimation methods that use arthritic traits in age assessments

Method	Sample	System	OA Traits
Brennaman, 2015	N=206 European/African M/F Late 20th C.	6-8 traits scored at four surfaces of the gleno-humeral joint: acromial facet , clavicular lateral facet, glenoid fossa, humeral head	Marginal & surface osteophytes Porosity Eburnation
Falys and Prangle, 2015	N=564 M/F European/African 18th-late 20th C.	3 features of the sternal end of the clavicle	Surface exostoses Porosity Osteophyte formation
Kunos et al., 1999	N=74 European/African M/F 19th-20th C.	Several traits for each of the: costal face, rib head, tubercle facet of the first rib	Surface exostoses Marginal & surface osteophytes Porosity
İşcan et al., 1984	N=204 European M/F 19th-20th C.	4 features of the sternal border of the fourth rib divided into 8 age phases	Marginal osteophytes Porosity
Listi and Manheim, 2012	N=104 European/African M/F 20th C.	3 variables of each superior and inferior borders of cervical, thoracic, and lumbar vertebra	Marginal osteophytes
Lovejoy et al., 1985	N=764 European/African M/F 19th-20th C.	8 features of the auricular surface divided into 8 age phases	Marginal & surface osteophytes Porosity
Buckberry and Chamberlain, 2002	N=180 European M/F 18th-19th C.	5 features of the auricular surface divided into 7 age phases	Marginal & surface osteophytes Lipping Porosity
Todd, 1920; 1921	N= 353 European/African M/F 19th-20th C.	10 age phase scoring system of the pubic symphysis	Marginal & surface osteophytes Porosity
Brooks and Suchey, 1990	N=1012 European/African/ Asian/Hispanic M/F	6 age phase scoring system of the pubic symphysis	Marginal & surface osteophytes Porosity
Rougé-Maillart et al., 2009	N=462 European M/F 19th-20th C.	3 features of the acetabulum divided into 8 age phases	Marginal & surface osteophytes Porosity
Calce, 2012	N=100 European/African M/F	3 features of the acetabulum divided into 3 age phases	Marginal & surface osteophytes Porosity
Passalacqua, 2009	N=633 European/African M/F 19th-20th C.	7 features of the sacrum divided into 6 age phases	Marginal osteophytes Porosity

1.6 Present research

As noted earlier, the three studies presented here aim to address key issues related to paleopathological analyses of OA by taking advantage of a large and well-preserved cemetery sample with documented human subjects. Using Buikstra and Ubelaker's (1994) arthritis recording system, the first study (Chapter 3) employs principal component analysis (PCA) as a standardized procedure to compute aggregate scores for separate joint complexes and a systemic measure of OA in each region of the lumbar spine, pelvis, and knee. Multivariate methods, like PCA have not been used to quantify OA previously. Accordingly, PCA was employed as an alternative to traditional methods of averaging point estimate scores that are biased by the total number of included surfaces, and fail to account for arthritic trait distribution and variance in joint-by-joint comparisons.

The second study (Chapter 4) analyzes the composite scores with a multiple regression model to determine the relative contribution of three predictors: age, activity, and body size, and their effect on skeletal expression of OA in weight-bearing regions. Body size (stature and mass) was calculated from postcranial skeletal measurements and torsional strength (*J*) of the femoral midshaft was calculated from three-dimensional surface models, size standardized and used as a proxy for measures of activity. Differences in adult patterns of age, activity, and body size can plausibly influence joint arthritic changes, and because many of our current age estimation methods utilize the progression of arthritic traits to determine chronological age, the third study examines the potential consequences of these.

The third study (Chapter 5) seeks to quantify the influence of error from OA on three methods to estimate age at death from load-bearing joints of the os coxa: the pubic symphysis, auricular surface, and acetabulum. The study was designed to determine whether OA in adults acts as a potential limitation or benefit in deriving accurate age estimates from pelvic joint morphology in order to contribute to standardized methods in establishing physiological degeneration of the skeleton due to aging. This research compliments other studies that consider the effects of body mass on the rate of skeletal aging (Merritt 2015; Wescott and Drew, 2015), but is also unique because evaluating progressive arthritic development from systemic measures of pelvic OA may help to distinguish between two very different processes: (1) systemic age-related stresses and (2) localized biomechanical effects.

Collectively, these studies strive to answer two questions: (1) how can we derive an accurate measure of OA severity from skeletal populations? and (2) how well do arthritic characteristics perform as age indicators in skeletal age estimation methods? These are important goals because skeletal joints wear differently based on function, anatomical structure, and habitual loading to produce varied osseous pathological bone responses (Ortner, 2003; Moskowitz et al., 2004; Arden and Nevitt, 2006); and a wide range of inferences about the behaviour and age structure of past populations are based on distributions of demographic prevalence, patterning, and severity of OA (Jurmain, 1999). If these are not accurate, interpretations of adaptive subsistence practices, sex-related divisions of labour, and rates of aging must be reconsidered. These results may have a considerable impact on our ability to accurately model age from the skeleton, and is especially relevant given that OA prevalence is increasing rapidly in young adults as a result of lifestyle characteristics, e.g., diet, disease, physical activity, and body mass (Barbour et al., 2013; Cross et al., 2014). In this regard, the results of the three studies described here have significant implications for paleodemography, bioarchaeology, and forensic anthropology.

Chapter 2.
Skeletal material

2.1 The study sample

This chapter details the skeletal sample used for this study and the three collections from which they are drawn. Details of the study sample are first described as a whole to demonstrate shared characteristics between individuals, and appropriateness of aggregating the three samples for analyses. Demographic details for each skeletal collection are then presented separately. For context, the process of amassing each collection is briefly discussed.

The study sample is comprised of 289 individuals from three modern, documented skeletal collections in Europe. These unclaimed bodies were exhumed from municipal cemeteries around Lisbon, Sassari, and Athens (Fig. 2.1) as part of traditional funerary customs that make room in the cemetery for new graves. Educational institutions purposefully appropriated the remains to form research and teaching collections. Biographic details accompanied the remains and were collected from coffin plates, cemetery records, and in many cases, the individual death certificate. The study sample includes complete and well-preserved adult skeletons of known sex and age at death interred between 1880-1996, although most of the individuals died between 1920-1989 (Fig. 2.2). Other individualizing information, such as home address and occupation, are included for most. Male occupations vary between skilled and non-skilled labourers, most of which are related to agriculture and farming professions. A significant portion of females worked in a domestic capacity, though is it not clear the difference between working in their own homes (i.e., housewife), or in others' (i.e., housekeeper). The balance of female professions included skilled work as a seamstress or teacher. Inferred from the reported occupations, this sample most likely represents the low to middle working class. Age at death is normally distributed in the sample, as assessed by a Shapiro-Wilk's test ($P > 0.05$) (Appendix Table A1). A one-way ANOVA revealed no significant differences ($P > 0.05$) in age between Lisbon (n=94, 58.1 ± 19.6), Sassari (n=101, 53.6 ± 18.9), or Athens (n=94, 54.4 ± 19.8) individuals (Appendix Table A2) and as such, the samples were pooled for all analyses. These collections are appropriate to aggregate for this study because both sexes and all adult ages are well represented (Table 2.1), but also because these populations are derived from a limited

temporal and geographic range from Southern Europe with similar activities and lifestyles, representing the low to middle socioeconomic strata of rural-to-urban populations (Belcastro et al., 2008; Cardoso, 2006; Eliopoulos et al., 2007).

Figure 2.1. Map of southern Europe indicating (left to right) Lisbon, Sassari, and Athens.

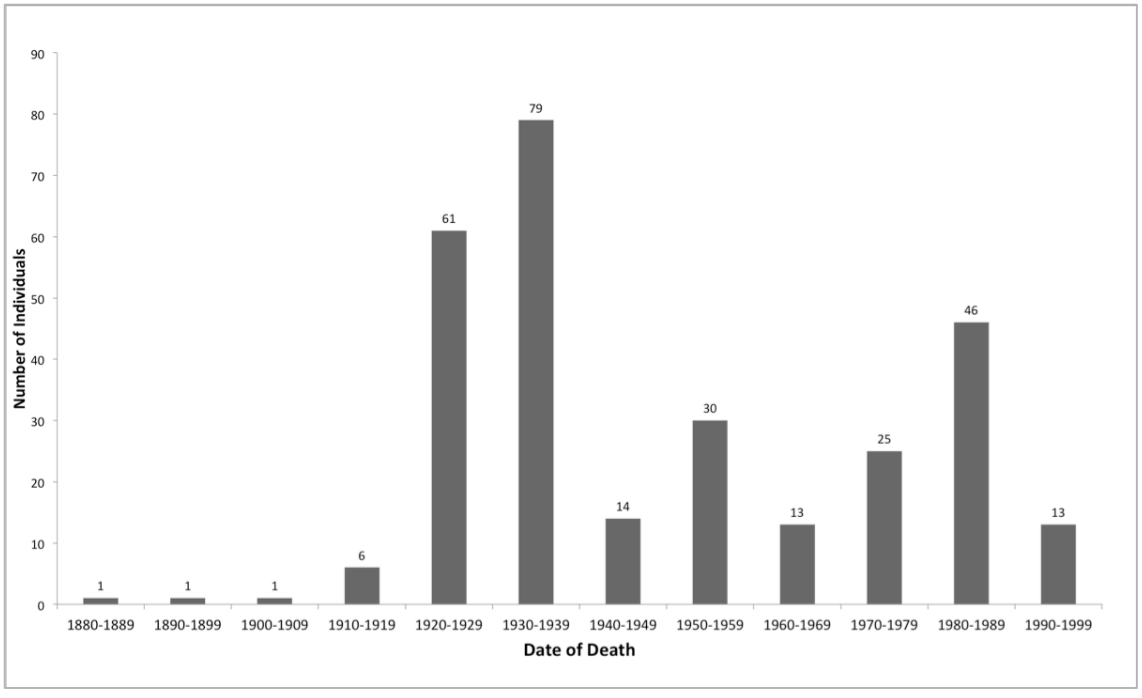


Figure 2.2. Distribution of the sample based on year of death (1880-1996).

Table 2.1. Sample size and distribution

	Athens				Lisbon				Sassari				Combined Sample	
	Male		Female		Male		Female		Male		Female			
Age Class	N	Mean age (years)	N	Mean age (years)	N	Mean age (years)	N	Mean age (years)	N	Mean age (years)	N	Mean age (years)	N	Mean age (years)
17-29	6	27.3	5	24.0	5	24.2	8	23.5	8	23.8	6	22.7	38	24.2
30-39	5	34.8	4	35.8	5	33.2	4	35.3	4	34.8	7	34.9	29	34.7
40-49	4	48.3	9	46.4	12	45.8	6	45.5	6	42.0	8	45.4	45	45.5
50-59	8	54.6	7	53.7	8	53.9	11	55.1	12	54.2	9	53.9	55	54.3
60-69	9	64.2	9	64.9	9	66.0	4	65.8	12	63.7	4	63.3	46	64.6
70-79	5	74.4	7	75.7	3	72.7	6	75.7	7	75.3	10	73.4	38	74.6
80-89	5	84.4	8	83.25	7	81.0	4	84.0	3	75.3	3	82.7	30	82.7
90+	2	94	1	99.0	0	NA	3	92.3	0	NA	2	94.0	8	94.0
Total	44	57.5	50	58.7	48	53.8	46	55.2	52	53.2	49	54.1	**289**	**55.4**

2.2 The Sassari Collection

The Sassari Collection is an assemblage of human remains exhumed from cemeteries in the region of Sassari on the island of Sardinia, the second largest island in the Mediterranean Sea and an autonomous region of Italy. Traditional funerary methods require that individual burials be exhumed after a post-depositional period of 10 years to create space in the cemetery for new interments. Unclaimed remains are kept for a period of time in individual boxes, and later deposited into a communal burial pit. Before reaching their final ossuary destination, Fabio Frassetto, director of the Institute of Anthropology, University of Bologna (1908-1947) acquired 606 individuals for which biographic details including sex, age, cause of death, and occupations were known from coffin plates, cemetery record books and hospital archives (Rastelli, 2005; Belcastro and Mariotti, 2012). Frassetto actively acquired human remains between 1930-1950 from Sardinian and Bolognese cemeteries. The Sassari collection is part of a larger skeletal assemblage housed at the

University of Bologna known as the Frassetto Collections. Containing more than 1000 individuals, the collections have been used to test osteological methods of age and sex determination (Belcastro et al., 2008; Benazzi et al., 2009; Hens and Belcastro, 2012; Godde and Hens, 2015), as well as to develop standards for recording and interpreting occupational stress markers (Mariotti et al., 2007; Milella et al., 2012).

The Sassari Collection spans a period of just over 100 years, with individuals living between 1828-1932. Known age at death is available for most (93%, n=565) and ranges from 9-98 years (Rastelli, 2005). Occupation is known for about half of the sample (n=337). Most of the variation in male professions includes a workforce divided between skilled and unskilled manual labourers (e.g., farmer, carpenter, machinist, miner, tailor, fisherman, military employee), the largest percentage (47%) associated with farming-related professions, suggestive of middle-to-lower socioeconomic status (Rastelli, 2005; Belcastro et al., 2008). Females did not typically work outside the family home (Belcastro et al., 2008). Ninety-four percent of females are recorded as housewife or housekeeper, with seamstress or teacher professions comprising the balance. Each individual is housed in a wooden box organized by call number. The author visited this collection in September 2012, with data collection lasting four weeks. During this time 101 adults (52 males, 49 females) ranging in age from 17-98 years (mean age 53.6 years) were analyzed (Table 2.1). In addition, 62 femora were imaged (34 males, 28 females) using the Next Engine™ laser scanner. Postcranial elements are relatively well-preserved, though postmortem damage at articular surfaces is common due to handling.

2.3 The Athens Collection

Greek funerary customs permit the exhumation of human burials after a period of 3-5 years, after which they are placed into individual metal boxes and moved to an indoor ossuary within the cemetery (Eliopoulos et al., 2007). The boxes of unclaimed skeletons are then emptied into a secondary underground ossuary pit at the back of the cemetery property. Before such practice, 225 individual skeletons were acquired from Athenian cemeteries over two periods: 1996-1997 and 2001-2003. Information for 214 individuals was collected from the death certificate and includes: sex, age, cause of death, occupation, and place of birth. The collection includes 11 additional undocumented subadult skeletons that are used primarily for teaching purposes. The first part of the collection (n=72), collected between 1996-1997, was compiled at the Wiener Laboratory of the

American School of Classical Studies and is known as "the Wiener Lab Collection". This assemblage was donated in 1998 to the Department of Animal and Human Physiology, University of Athens, where it is housed today. Together with the second part (n=153), collected between 2001-2003, the skeletal assemblage is collectively known as the University of Athens Human Skeletal Reference Collection, or the Athens Collection, for short. Much of the research conducted on this collection has been age and sex validation studies (Eliopoulos, 2006). Additional research includes the development of new techniques specific to variation found in the Greek population (Fox et al., 2003) and distribution of trauma and disease (Abel et al., 2003; Lagia et al., 2007).

The Athens Collection individuals lived during the late 19th–20th centuries, 1884-1996. Both sexes are represented (males n=114, females n=100),with age at death ranging from birth to 99 years (Eliopoulos et al., 2007). Male occupations vary and include service workers such as electrician, clothes manufacturer, automotive engineer, painter, merchant, and military employee. Female occupations are largely documented as "household", the meaning of which is unknown and may refer to either a housewife or a housekeeper (M. Chovalopoulos, pers. com.). The skeletal sample represents the middle-to-low social class (Eliopoulos et al., 2007). The collection is exceptionally well-preserved and housed in a climate-controlled laboratory. Each individual is kept neatly in acid-free boxes lined with protective padding to minimize postmortem damage at articular surfaces. The author visited the Athens collection in October 2012, with data collection lasting four weeks. Ninety-four individuals were evaluated for this study (44 males, 50 females), with age at death ranging from 20-99 years, with a mean age of 58.1 years (Table 2.1). Unfortunately, the Next Engine™ laser scanner ceased functioning during data collection, precluding 3D scanning of the femora. As a result, Athens individuals were not included in analyses of activity outlined in Chapter 4.

2.4 The Lisbon Collection

Funerary custom in Portugal follows the exhumation practices observed in other Southern European countries. Bodies are exhumed from temporary graves after the legally stipulated period of 5 years and kept in an above-ground ossuary (*ossários*) for a fee paid by relatives. If the remains are unclaimed, or if the family neglects payment for the *ossários*, the bones are either incinerated or reburied into a communal grave (Cardoso, 2006). Following a major fire that destroyed much of an existing skeletal collection, Luís Lopes, assistant professor of anthropology at the Faculty of

Sciences of the University of Lisbon (1977-1989), began to amass skeletal remains from three large cemeteries in the city of Lisbon. Today, the sample known as the Luís Lopes Collection (aka, the Lisbon Collection, or the ‘‘new Lisbon Collection’’) is comprised of nearly 1700 individuals of known age, sex, cause of death, and occupation, and is housed at the Bocage Museum in Lisbon, Portugal (Cardoso, 2006). Approximately half of the collection is available for study (H. Cardoso, pers. comm.). Since 2000, numerous research studies have been carried out on the collection, particularly on the large subadult portion of the sample (Cardoso, 2005; Rogers, 2009a), but also to document activity-related changes in the postcranial skeleton (Cardoso and Henderson, 2010; 2013).

While the collection spans the better part of two centuries (1805-1975), most of the individuals were born between 1861-1940 and died between 1921-1960. Males and females are both well represented, and ages at death range from birth to 98 years (Cardoso, 2006). As in the Sassari and Athens collections, male occupations vary between service and sales workers (30%), skilled workers (i.e., craftsmen) (23%), and general labourers (47%), representing the low-to-middle socioeconomic strata of an urban population. Female occupation is reported as housewife (85%), with remaining professions including maid, teacher, or student (Cardoso, 2006). The collection is in a good state of preservation with skeletons kept separate in tidy single drawers. The author visited this collection in April 2013, with data collection lasting four weeks. Ninety-four individuals were studied (48 males, 46 females), with age at death ranging between 18-94 years, with a mean age of 54.4 years (Table 2.1). Sixty-two complete femora were 3D laser scanned (29 males, 33 females).

Chapter 3.
Principal component analysis in the evaluation of joint osteoarthritis

3.1 Introduction

The pathophysiology of osteoarthritis (OA) includes chronic, inflammatory and degenerative changes in joints with cartilage components as a result of confounding effects such as aging, mechanical stress, genetic susceptibility, normal anatomical variation, joint injury, and body size (Radin et al., 1972; Merbs, 1983; Jurmain, 1999; Ortner, 2003; Spector and McGregor, 2004; Weiss and Jurmain, 2007). Osteoarthritis, a proliferative bone condition that generally presents as hypertrophic joint lesions, has been documented in human populations as one of the most common skeletal pathologies (Rogers et al., 1984; Karsenty, 2003; Ortner, 2003; Felson and Nevitt, 2004). In life, OA can originate in any and all tissues surrounding a joint including cartilage, subchondral bone, ligaments, periarticular muscles, and/or the synovium (Dieppe, 2011). A breakdown of contiguous joint tissue, particularly in cartilage, causes bone surfaces to rub together resulting in pain, stiffness, and eventual loss of joint use (Jurmain and Kilgore, 1995; Tepperman, 1981). While pain and narrowing of joint space are clinical indicators of arthritic severity and distribution, archaeological investigations of OA can only evaluate changes observed directly on the bone surface, which are likely to reflect the later stages of the disease, or its more severe manifestations (Ortner, 2003).

Occurring most often in the spine, knee, hip, and hands, cartilage tissue degeneration is accelerated when biosynthetic anabolic activity can no longer keep pace with the degradative catabolic activity (Sandell and Aigner, 2001; Visse and Nagase, 2003; Sharma et al., 2013). As a result of tissue injury, secondary cartilage forms along with bone overgrowth at the joint, also known as osteophyte (OPH) formation (Sandell and Aigner, 2001; Klaus et al., 2009). Osteophytes grow in both horizontal and vertical directions to produce a change in the shape of the joint contour (also described as lipping), and are most commonly observed in vertebral margins (Merbs, 1983; Walker and Hollimon, 1989; Jurmain, 1990; Bridges, 1991; Lieverse et al., 2007; Klaus et al., 2009). In addition, multiple proliferative osteophytic spurs originating at the joint rim may grow towards each other and eventually fuse (Larsen, 1997; Ortner, 2003). Under some circumstances,

osteophytes form more centrally in the joint and are associated with articular surface deformation (Pritzker et al., 2006). Clinically, the presence of polyarticular OPHs (visible on radiographs) distinguishes idiopathic joint OA from other arthritides more than any other pathological feature (Altman et al., 1986; Sadnell and Aigner, 2001), whereas in skeletal analyses eburnation is regarded as the clear indicator of severe OA (Weiss and Jurmain, 2007). Following the total loss of joint cartilage, eburnation results from the rubbing (or contact) of two bone surfaces that leads to polishing and grooves parallel to the line of motion (Klaus et al., 2009). Other changes in subchondral bone include resorption of the surface, recognized as pitting or porosity (Hough, 2001; Ortner, 2003).

Methods for defining joint OA expression and the analysis of OA data vary widely in the literature (Rogers, 1966; Jurmain and Kilgore, 1995; Rothschild, 1997; Klaus et al., 2009; Molnar et al., 2011; Nikita et al., 2013), leading to difficulties comparing studies when a mixed number of OA criteria are employed (Weiss and Jurmain, 2007). Quantifying OA continues to be a major challenge in the absence of standards for data collection and manipulation, including using statistical analyses to move beyond simply documenting OA occurrence (Bridges, 1993; Waldron, 2012). Several systems for coding osteoarthritic lesions have been proposed (Jurmain, 1990; Waldron and Rogers, 1991; Buikstra and Ubelaker, 1994; Jurmain and Kilgore, 1995; Larsen et al., 1995), but inconsistencies in the criteria used to evaluate arthritis, and varied approaches to manipulating data into accurate representative (or composite) scores for collapsed joint categories make it difficult to (1) compare results between studies, and (2) study the effects of interacting explanatory variables (e.g., sex, age, activity, body size) on osteoarthritic response.

Quantifying skeletal arthritic expression in a meaningful way has proven difficult for a few reasons. First, OA is a systemic disorder of bone (Rogers et al., 2004), which affects multiple joints simultaneously at varied rates (Dieppe, 2011). As a result, it is impractical to establish a "skeleton" or overall body score that encompasses this heterogeneity. Second, pathological bone responses vary dependent on joint type. As skeletal joints wear differently based on function, structure, and habitual loading, it is unlikely that an analogous progression of pathological osseous change across multiple joint complexes will be observed (Ortner, 2003; Moskowitz et al., 2004; Rogers et al., 2004; Arden and Nevitt, 2006). Third, the traditional method of averaging OA scores to represent categories of specific joint systems must be reviewed carefully for each individual study. Finally, there is no agreement among researchers on how to summarize the raw data, and determining how many joint surfaces will comprise a joint complex is somewhat arbitrary (Bridges, 1993). This can

have a significant impact on interpreting results given that the more joint surfaces included, the smaller the average score will be.

The large volume of data resulting from OA analysis is highly complex and multidimensional. Biocultural interpretations of OA have been hindered by a lack of appropriate techniques for reducing and interpreting large volumes of OA data (Astephen and Deluzio, 2004). Recently, Nikita and colleagues (2013) have demonstrated the potential of applying regression models to examine the simultaneous impact of multiple underlying factors on the expression of OA. Though they did not assess the actual impact of each factor, their results reveal the ability of multivariate statistics as tools for use in exploratory data analysis and predictive models. Likewise, simple non-parametric methods like principal component analysis (PCA) may be valuable in studies of OA to examine the internal structure of such complicated datasets and to explore the interrelations among variables (i.e., indicators of OA). Principal component analysis summarizes variation by clustering similar observations together based on multi-attributes (Krzanowski, 2000) and may be a way to reduce variable redundancy and to explore the relative impact and the interaction of underlying factors (such as age, activity, or body size) on OA expression. The purpose of this study is to demonstrate the advantages of PCA as a standardized procedure in the evaluation of idiopathic OA to: (1) compute aggregate scores for joint complexes that accurately capture pathological expression, (2) reveal which variables describe the most variation in OA within our sample, and (3) enable inter- and intra-sample comparison of results.

3.2 Materials and Methods

The study sample is comprised of skeletons from three modern European identified skeletal collections: (1) the Luís Lopes Skeletal Collection (Portugal), (2) the Sassari Collection (Italy), and (3) the University of Athens Human Skeletal Reference Collection (Greece). The sample size includes 289 specimens (144 males and 145 females). Age at death ranged from 17 to 99, with a mean age of 55.4, S.D.=19.4 (Table 3.1). These collections are appropriate to aggregate for this study because they are well preserved and derived from a limited temporal and geographic range from Southern Europe with similar activities and lifestyle, representing the low to middle socioeconomic strata of rural-to-urban populations (Belcastro et al., 2008; Cardoso, 2006; Eliopoulos et al., 2007).

The skeletons were exhumed from municipal cemeteries in the last 50 years, and represent individuals of known age, sex, and occupation who died between 1880 and 1996 (Belcastro et al., 2008; Hens et al., 2008; Eliopoulos et al., 2007; Cardoso, 2006). Specimens affected by OA related to fractures were excluded to avoid confusion with secondary OA that forms as a result of pre-existing abnormalities in joint tissues. Other criteria for selection were (1) bone preservation of individual elements showing no more than 25% of post mortem damage at articular surfaces and margins, and (2) sample representation of adults across all age-classes (young, middle, old).

Osteoarthritis was scored macroscopically using the ordinal scale proposed by Buikstra and Ubelaker (1994) for both severity and distribution of lipping, porosity, eburnation, and surface osteophytes (Table 3.2, Figs. 3.1-3.4) in stabilizing and weight bearing joints of the lumbar vertebrae, os coxa, and knee (Table 3.3), that are responsible for mobility and movement of the skeleton. Data on periarticular resorptive foci was not collected because it is only present in rheumatoid arthritis, and is not the focus of this study. Levels of OA severity are measured on a relative scale to qualify progression of the disease marked by gross morphological changes; which in paleopathological skeletal analyses, is defined by the combination of continued growth (lipping, osteophytes) and/or deterioration (porosity, eburnation) observable on the bone surface. In this context, 'severity' is meant to describe pathological expression of OA, not related to disease duration, impact on quality of life, or endured pain, as is typically expressed in clinical investigations of joint pathology (Dieppe, 2011). Distribution is defined here specifically to represent the amount of joint area affected, and not meant to describe frequencies of OA in relation to other joints of the skeleton, as has been done in other studies (Lieverse et al., 2007; Klaus et al., 2009; Schrader, 2012). Individuals with diffuse idiopathic skeletal hyperostosis (DISH) were not excluded from the study since OA may develop in concert; however, cases of DISH (particularly where lumbar vertebrae were ankylosed) were differentiated from those with OA on the basis of the following DISH characteristics: (1) maintenance of normal intervertebral disk space in fused vertebrae (including the thoracic region), (2) absence of pathology (lipping, porosity, eburnation) in vertebral facet joints, and (3) extraspinal manifestations of new bone growth in ligament and tendon insertion sites (e.g., linea aspera, deltoid tubercle, and iliac crest) (Rogers et al., 1987). If these characteristics were present, then the pathological diagnosis was DISH, not OA.

Table 3.1. Summary age and sex data for the test sample

Sex	Lisbon				Athens				Sassari				**Combined sample**			
	n	Mean Age (years)	SD	Range (years)	n	Mean Age (years)	SD	Range (years)	n	Mean Age (years)	SD	Range (years)	n	Mean Age (years)	SD	Range (years)
Males	48	53.8	18.0	20-88	44	57.5	19.8	24-94	52	53.2	18.0	20-82	**144**	**54.7**	**18.5**	**20-94**
Females	46	55.2	21.6	18-94	50	58.7	19.6	20-99	49	54.1	20.1	17-98	**145**	**56.0**	**20.4**	**17-99**
Total	94	54.4	19.8	18-94	94	58.1	19.6	20-99	101	53.6	19.0	17-98	**289**	**55.4**	**19.4**	**17-99**

Table 3.2. Scoring system used to record OA severity and distribution, after guidelines proposed by Buikstra and Ubelaker (1994), page 115 attachment 66.

Feature	**Description**	**Degree of Expression (Severity)**	**Extent of Area Affected (Distribution)**
Lipping (Fig. 3.1)	Marginal proliferation of new bone in either a horizontal or vertical direction that produces a change in the shape of the joint contour.	0 – Not present 1 – Barely discernible 2 – Sharp ridge or curled spicule(s) 3 – Extensive spicule formation 4 – Ankylosis	0 – Not present 1 – < 25% 2 – 25-75% 3 – > 75%
Porosity (Fig. 3.2)	Pitting and/or erosion of the joint surface.	0 – Not present 1 – Pinpoint 2 – Coalesced 3 – Both pinpoint & coalesced	0 – Not present 1 – < 25% 2 – 25-75% 3 – > 75%
Eburnation (Fig. 3.3)	Polished subchondral bone with or without ridges (mechanical scoring).	0 – Not present 1 – Barely discernible 2 – Polish only 3 – Polish with grooves	0 – Not present 1 – < 25% 2 – 25-75% 3 – > 75%
Surface Osteophytes (Fig. 3.4)	Multiple proliferative changes of bone growth (bony spurs) that originate either at the joint rim, or on the articular surface.	0 – Not present 1 – Barely discernible 2 – Clearly present	NA

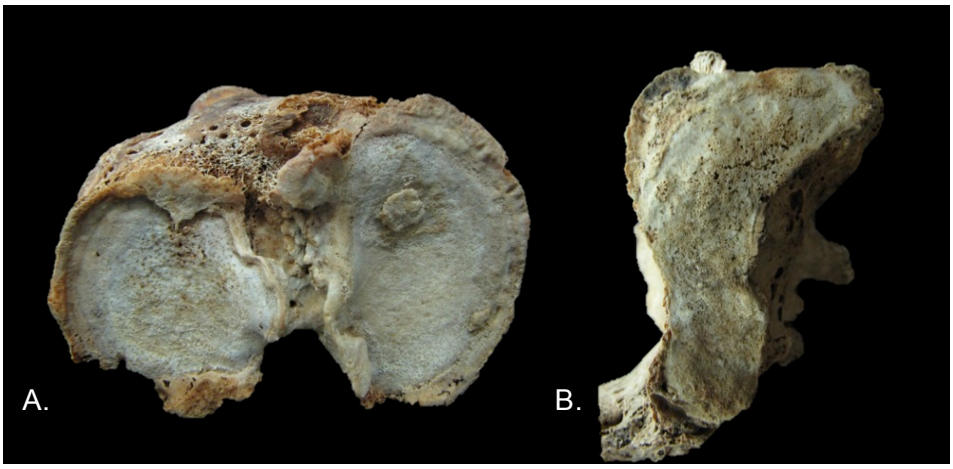


Figure 3.1. Lipping and associated joint surface contour changes of the proximal tibia (A) and sacro-iliac surface of the sacrum (B). Photo A: Athens 136, Female 62 years. Photo B: Athens 181, Male 94 years.

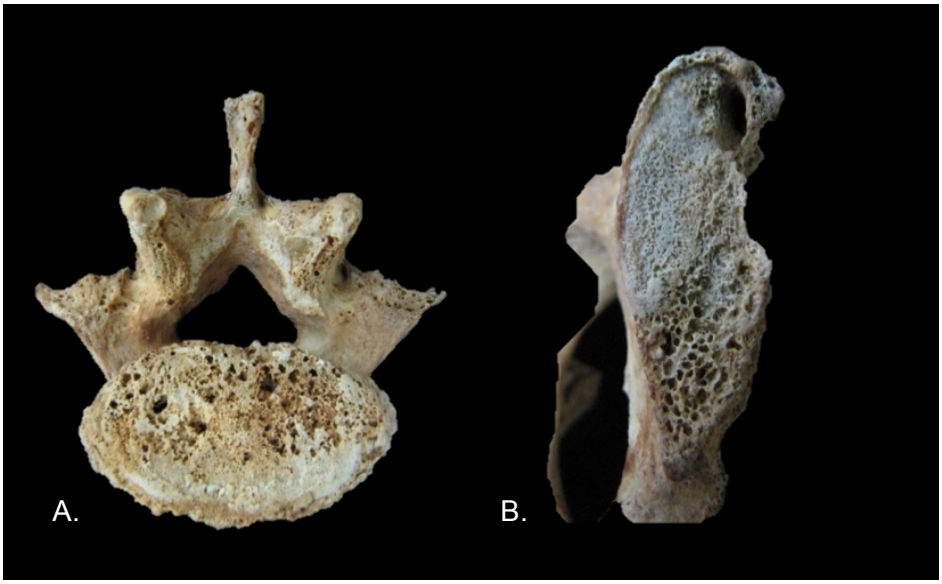


Figure 3.2. Subchondral surface porosity of the lumbar vertebra (A) and pubic symphyseal face (B). Photo A: Athens 150, Female 82 years. Photo B: Athens 178, Male 73 years.

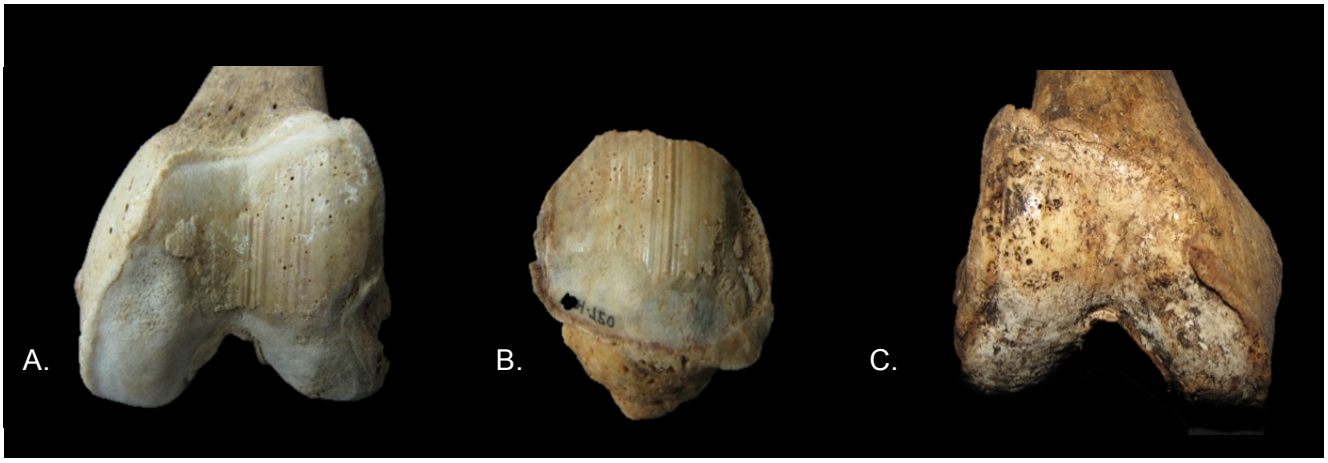


Figure 3.3. Eburnation on the distal femoral condyles (A) and associated posterior patellar surface (B) where parallel mechanical scoring and pitting are observed. Polish and porosity of the lateral femoral condyle (C). Photos A and B: Athens 150, Female 82 years. Photo C: Lisbon 351, Female 90 years.

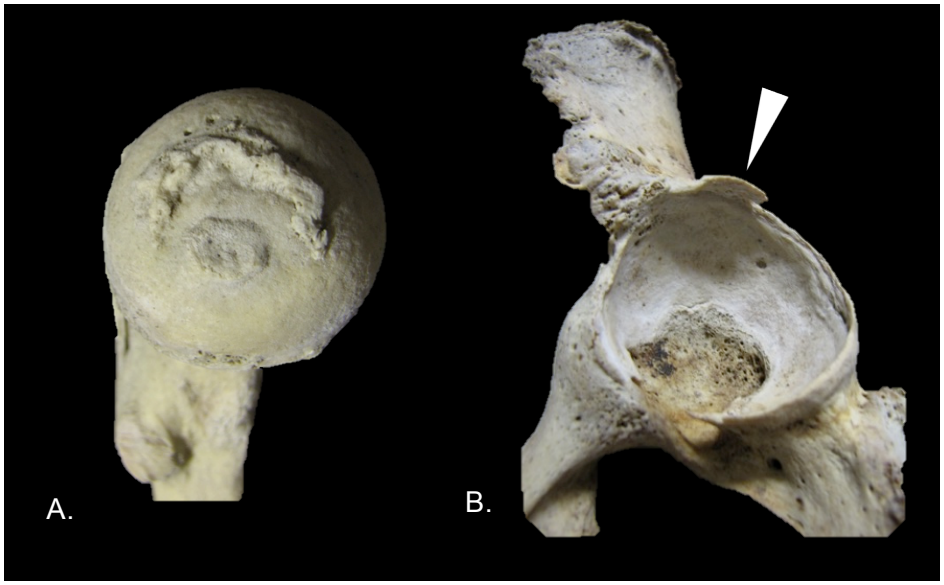


Figure 3.4. Osteophytic bone growth on the articular surface of the femoral head (A), and at the acetabular margin (B). Photo A: Sassari 308, Male 69 years. Photo B: Sassari 329, Male 78 years.

Table 3.3. Articular surfaces examined and scored separately for OA in the lumbar vertebrae, pelvic, and knee regions

Lumbar Vertebral Region (11—13 surfaces)[a]	Pelvic Region (5 surfaces)[a]	Knee Region (3 surfaces)[a]
1. Lumbar[b]: Superior intervertebral body, superior articular facets (evaluated together as one surface)	1. Sacrum: Sacroiliac joint	1. Femur: Medial and lateral condyles
2. Lumbar[b]: Inferior intervertebral body, inferior articular facets (evaluated together as one surface)	2. Os Coxa: Auricular surface	2. Patella: Condylar surfaces
3. Sacrum: Superior intervertebral body of first sacral vertebra, superior articular facets (evaluated together as one surface)	3. Os Coxa: Pubic symphysis	3. Tibia: Medial and lateral condyles
	4. Os Coxa: Acetabulum	
	5. Femur: Head	

[a] Each element was scored separately, but an average score was calculated in each joint region (lumbar, os coxa, knee) for each variable by adding scores together and dividing by the number of available elements. For example, the composite score for *lipping* of the knee region = (femoral condyles *lipping score* + patella *lipping score* + tibial plateau *lipping score*)/3.

[b] L1-L6 surfaces scored separately; individuals who had a sixth lumbar vertebra, n=2.

Elements from the left side were evaluated unless missing from the collection, in which case the right side was substituted. Skeletal elements were scored separately for all available surfaces, and then collapsed into categories of specific joint systems (Table 3.3). A mean score was calculated for each variable by joint region, where individual variable scores were summed and divided by the number of observed surfaces to produce an average. For example, pelvis OPH score = (sacroiliac joint OPH score + auricular surface OPH score + pubic symphysis OPH score + acetabulum OPH score + femoral head OPH score)/5. Summation of scores across all variables for each element (or surface) could not be computed to accurately reflect OA expression because the ordinal scoring system differed between variables, that is, each variable (lipping, porosity, eburnation, surface osteophytes) was not measured by the same ranked scale. Involvement of OA at multiple joints is common; therefore, joint regions of the lumbar spine, pelvis, and knee were analyzed as separate conditions here to differentiate between distributions of idiopathic OA (Arden and Nevitt, 2006; Cushnaghan and Dieppe, 1991).

3.2.1 Statistical procedures

Friedman's two-way analysis of variance by ranks (a non-parametric measurement for related samples of non-normally distributed data) and post-hoc tests were performed to identify

differences among category means for each of Buikstra and Ubelaker's (1994) seven variables by skeletal location (lumbar, pelvis, and knee). Regional differences suggest varied wear patterns of OA expression. Females and males are examined together in the analyses given no significant differences ($P > 0.05$) between median OA scores as determined by Mann-Whitney U tests in each skeletal region. Statistical analyses were performed using SPSS 20 (IBM, 2011) and PAST 3.04 (Hammer et al., 2001).

Principal component analysis is an exploratory tool favoured to reduce redundancy in multivariate data and, as in the current study, is also useful to reveal measurements that best reflect the dynamics of a particular phenomenon; in this case which traits contribute most of the variation in the development and expression of idiopathic joint OA (Jolliffe, 2002; Krzanowski, 2000). To examine the configuration and variability of OA data in a multivariate space, PCA was conducted on the scores of the seven variables defined by Buikstra and Ubelaker (1994): lipping severity (LIP-S), lipping distribution (LIP-D), porosity severity (POR-S), porosity distribution (POR-D), eburnation severity (EBR-S), eburnation distribution (EBR-D), and severity of osteophytes (OPH) (Table 3.4) by skeletal region (lumbar, pelvis, knee). Principal components (PC) were extracted as a linear combination of the variables and estimated from the eigenvectors of the correlation matrix. The correlation matrix was used because variables in the dataset are measured on different scales, and is therefore equivalent to standardizing the variables to zero mean and unit standard deviation (Krzanowski, 2000). Principal component loadings (eigenvectors) measure the importance of each variable in accounting for the variability of OA in each PC. Loadings from the first and second PCs were used to form the weighted component scores ($Y1, Y2$) as a linear combination of the original seven variables for each individual. Eigenequations given by $Y1, Y2$ are combined to represent a composite OA expression score ($Y3 = |Y1\ \lambda1| + |Y2\ \lambda2|$) where λi is the variance explained by each PC (eigenvalue) that accounts for most of the variation in OA at each skeletal region.

Buikstra and Ubelaker's (1994) method is the only published method that separates severity measures from distribution measures (i.e., amount of surface area affected) (Table 3.2). A second PCA of their four severity variables (LIP-S, POR-S, EBR-S, and OPH) was conducted to determine how well severity traits could distinguish OA characteristics within the dataset. Following the PCA procedure described above, a secondary composite OA score was generated from eigenequations of the first and second PCs and compared with each of Buikstra and Ubelaker's original distribution traits to look for preserved linear relationships, reduced amount of variation in each category, outliers, and overlap within the dataset.

Table 3.4. Abbreviations and descriptions of OA variables used in PCA

Abbreviation	Variable Name	Description
LIP-S	Lipping Severity	Severity of lipping at the margin or contour of the joint surface
LIP-D	Lipping Distribution	Distribution of lipping at the margin or contour of the joint surface
POR-S	Porosity Severity	Severity of pitting and/or erosion of the joint surface
POR-D	Porosity Distribution	Distribution of pitting and/or erosion of the joint surface
EBR-S	Eburnation Severity	Severity of polished subchondral bone on the joint surface
EBR-D	Eburnation Distribution	Distribution of polished subchondral bone on the joint surface
OPH	Surface Osteophytes	Severity of bone growth (spur) originating either on the articular surface, or at the joint rim

3.3 Results

Descriptive statistics for OA variables are provided in Table 3.5. Damage to individual specimens resulted in a slightly reduced sample size for the lumbar vertebrae. The Friedman test results (Table 3.6) indicate significant differences ($P < 0.05$) in means among skeletal regions for all seven variables tested. Pairwise comparisons post-hoc tests were performed with a Bonferroni correction for multiple comparisons. Post-hoc analysis revealed statistically significant differences between skeletal regions for all variables, except for LIP-S between the pelvic and knee regions ($P = 0.82$) (Table 3.6). Both POR-S and POR-D occurred in very low frequencies in the knee region, 167 individuals (58%) were scored as '0', or 'not present' for these variables. Likewise, extremely low frequencies for both EBR-S and EBR-D were observed in all skeletal regions; only 29% (lumbar), 8% (os coxa), and 14% (knee) of individuals within the sample received a score of '1', or more. The distribution of original scores for each variable by skeletal region (lumbar, pelvis, knee) can be found in the Appendix, Figures A1-A7.

3.3.1 Multivariate analysis

Principal component analysis. Missing values are ignored while optimizing the PCA model, so individuals with missing data points were excluded from the analysis. For seven variables defined by Buikstra and Ubelaker (1994) (Table 3.4), PCA was conducted separately on the lumbar spine, os coxa, and knee. In all regions PCA revealed two components that had eigenvalues greater than one and explained 87%, 83%, and 79% of the total variance in the lumbar spine, pelvis, and knee areas respectively (Table 3.7), therefore two PCs were retained in each model for further analysis. In each of the regional analyses, lipping, porosity, and osteophyte variables (LIP-S, LIP-D, POR-S, POR-D, OPH) loaded heavily on the first PC, while eburnation variables (EBR-S and EBR-D) weighed heavily onto the second PC (Table 3.7). Both porosity (POR-S, POR-D) and OPH are more variable on PC2 with less effect of OPH at pelvic and knee areas, and more influence for porosity in the knee. The first PC may be interpreted as representing earlier stages of cartilage degeneration, where lipping, porosity, and osteophyte development, occurring as a suite of traits, determine most of the variation present in the sample. Since PCA clusters similar observations together based on multi-attributes, it is likely that EBR-S and EBR-D variables are separated from the others (in each skeletal region) because they represent a more advanced state of contiguous cartilage wear and are less frequent in the sample overall. The second PC contrasts eburnation (positive eigenvector coefficients) with lipping, porosity, and osteophyte growth (negative coefficients) (Table 3.7) and suggests that individuals with high PC2 scores tend to have developed eburnation expression, but a low collective pattern of all other variables (lipping, porosity, and osteophytes). Plots of individual component scores for PC1 versus PC2 are provided in the Appendix, Figure A8.

Table 3.5. Summary statistics for original scores of the study variables by skeletal region

Variable	Lumbar (n=286)		Pelvis (n=289)		Knee (n=289)	
	Mean	S.D	Mean	S.D	Mean	S.D
1–LIP-S	2.10	0.70	1.51	0.58	1.46	0.86
2–LIP-D	1.84	0.67	1.30	0.55	1.54	0.90
3–POR-S	1.36	0.76	1.20	0.71	0.49	0.60
4–POR-D	1.12	0.68	1.01	0.55	0.44	0.50
5–EBR-S	0.31	0.48	0.04	0.16	0.22	0.53
6–EBR-D	0.18	0.28	0.04	0.14	0.16	0.39
7–OPH	0.98	0.64	0.65	0.57	0.42	0.57

Table 3.6. Results of Friedman's two-way analysis of variance (ANOVA) and post-hoc[a] tests for study variables by skeletal region

					Post-hoc P-value (adjusted)		
Variable	**N[b]**	***t***	**df**	***p*[c]**	**Lumbar—Pelvis**	**Lumbar—Knee**	**Pelvis—Knee**
LIP-S	1608	926.04	2	*0.00*	*0.00*	*0.00*	0.82
LIP-D	1608	558.80	2	*0.00*	*0.00*	*0.00*	*0.00*
POR-S	1608	1320.62	2	*0.00*	*0.01*	*0.00*	*0.00*
POR-D	1608	1265.59	2	*0.00*	*0.00*	*0.00*	*0.00*
EBR-S	1608	519.71	2	*0.00*	*0.00*	*0.00*	*0.00*
EBR-D	1608	470.21	2	*0.00*	*0.00*	*0.00*	*0.00*
OPH	1608	2525.33	2	*0.00*	*0.00*	*0.00*	*0.00*

[a] Pairwise comparisons with Bonferroni correction; significance level $P < 0.05$ for all tests and indicated by italics.
[b] Number of surfaces evaluated.
[c] Asymptotic Significance (2-tailed test).

Table 3.7. Eigenvector coefficients for principal components of seven variables from the lumbar, pelvis, and knee regions

	Lumbar		Pelvis		Knee	
	Component		Component		Component	
	1	2	1	2	1	2
LIP-S	.423	-.162	.425	-.213	.402	-.193
LIP-D	.426	-.159	.439	-.162	.379	-.272
POR-S	.401	-.177	.433	-.127	.398	-.235
POR-D	.410	-.233	.431	-.133	.366	-.311
EBR-S	.291	.643	.235	.663	.357	.592
EBR-D	.293	.640	.231	.667	.347	.613
OPH	.415	-.197	.377	.093	.391	-.091
Eigenvalue	4.685	1.388	4.118	1.700	4.280	1.211
% Total Variance	66.93%	19.83%	58.83%	24.29%	61.15%	17.30%

Sample sizes and descriptive statistics for weighted PCs (*Y1, Y2*) and composite OA scores (*Y3*) are presented in Table 3.8. In each region, PCA produced the same general pattern with eburnation scoring driving significant changes in composite OA scores. When eburnation is not present/barely discernible (scores 0, 1) and all other variables (lipping, porosity, osteophytes) are also not present/barely discernible (scores 0, 1), an overall low OA score (~3) is observed. When polish is observed (scores 2, 3) and all other variables are also clearly present (scores 2, 3), an overall high OA score (~20) is observed. Little to no eburnation (scores 0, 1) combined with developed expression of all other variables (scores 2, 3) produce a moderate overall OA score (~11); but most interesting is the higher OA score (>15) when advanced stage eburnation (polishing and grooves) is observed in isolation. Bone growth variables (LIP-S, OPH) weigh heavily on PC1 in all three skeletal areas and when extensive spicules/fusion develops (scores 3, 4) the highest OA scores are observed (~25+) (see illustration in Figure 3.5). Larger composite OA scores reflect the most advanced stages of arthritic osseous change in terms of joint mobility within the sample. The distribution of arthritic traits determined by PCA produces an OA score that quantifies the expression of joint changes in varied biological joint structures from most moveable to least mobile, the final stage being joint fusion (Fig. 3.5). Composite OA scores are most highly variable in

the lumbar region for both males and females, as compared to the pelvis and knee (Fig. 3.6). Throughout the skeleton, OA scores are not normally distributed for nearly equal sample sizes of both males and females as assessed by Shapiro-Wilk's test ($P < 0.05$) (Appendix Table A3). Males and females are pooled in the analysis given no significant differences ($P > 0.05$) between median OA scores as determined by Mann-Whitney U tests in each region (Appendix Table A4).

Table 3.8. Summary statistics for principal components and OA composite scores by skeletal region

		PC1 (Y_1)		PC2 (Y_2)		OA Score (Y_3)					
	N	**Mean**	**S.D.**	**Mean**	**S.D.**	**Mean**	**S.D**	**Minimum**	**Median**	**Maximum**	**IQR**
Lumbar	286	3.75	1.351	1.28	0.602	19.33	6.821	0.4	20.46	30.16	9.4
Pelvis	289	2.44	1.073	0.84	0.346	11.47	4.912	0	11.76	24.85	6.8
Knee	289	1.83	1.314	0.81	0.521	8.81	5.959	0	7.77	27.11	8.1

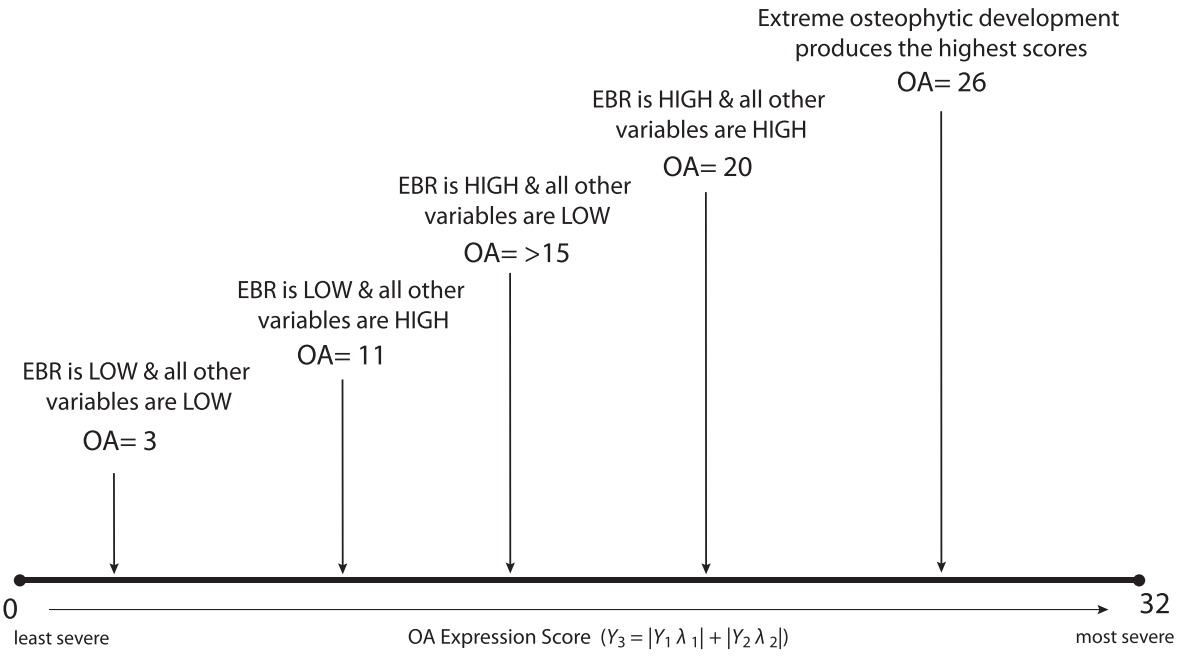


Figure 3.5. Illustrative example of how trait expression affects overall OA score.

Severity versus distribution trait analysis using PCA. In all regions, there is a high correlation between severity and distribution variables (Table 3.9), all significant at the $P < 0.01$ level. This result indicates that as severity in OA increases at any particular joint location, so does the distribution of OA within the joint surface, that is, more joint surface area is affected by osteoarthritic response.

Principal component analysis of four severity variables (LIP-S, POR-S, EBR-S, OPH) resulted in a similar loading pattern of two PCs, as seen in the preceding analysis where LIP-S, POR-S, and OPH (representing early contiguous tissue degeneration) weighed heavily onto PC1, and EBR-S (late stage tissue deterioration) weighed heavily onto PC2 (Table 3.10). Together, two PCs (*Y1,Y2*) explained more than 80% of the total variance in each region of the lumbar, os coxa, and knee (Table 3.10). Plots of individual component scores for PC1 versus PC2 are provided in the Appendix Figure A9. In each region, composite OA scores ($Y3=|Y1\ \lambda1| + |Y2\ \lambda2|$) were positively correlated with each of Buikstra and Ubelaker's (1994) original distribution traits (Table 3.11), which suggest that severity measurements alone are capable of capturing all of the sample variation in OA. Since larger composite scores reflect the linear changes in individual trait scoring, inclusion of data from distribution variables (LIP-D, POR-D, EBR-D) are redundant in formulating an aggregate measure of OA.

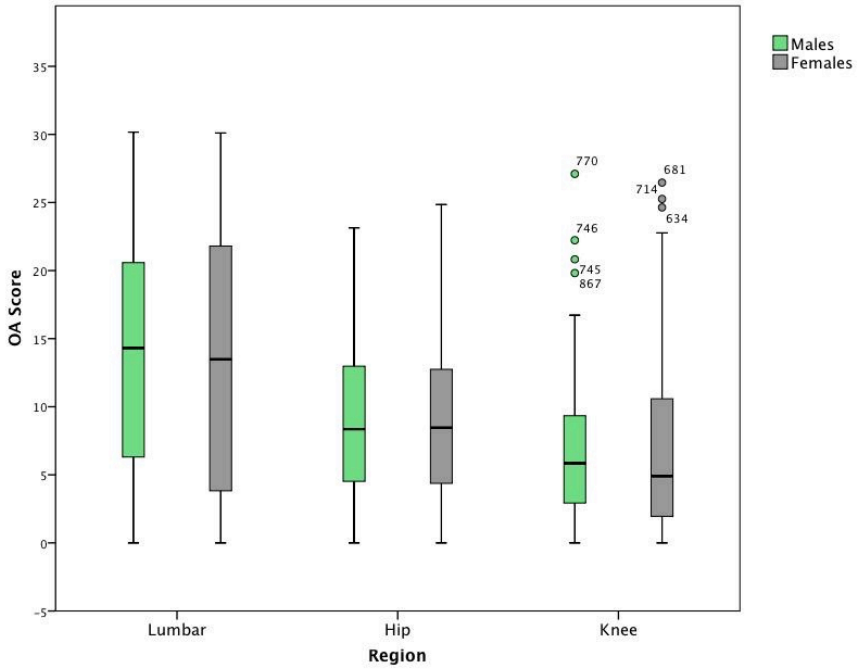


Figure 3.6. Distribution of OA scores by region calculated from loadings on the first and second PCs and separated by sex.

Table 3.9. Spearman's rank-order correlation coefficients[a] ($P < 0.01$) between severity and distribution variables

	Lumbar	Pelvis	Knee
	r_s	r_s	r_s
LIP-S —LIP-D	0.774	0.856	0.877
POR-S—POR-D	0.827	0.861	0.963
EBR-S—EBR-D	0.987	0.956	0.998

[a] Spearman's rho (r_s): .90—1.00 = very high positive correlation; 0.70—0.90 = high positive correlation; 0.50—0.70 moderate positive correlation; 0.30—0.50 = low positive correlation; 0.00—0.30 = negligible correlation (Hinkle et al., 2003).

Table 3.10. Eigenvector coefficients for principal components of four severity variables from the lumbar, pelvis, and knee regions

	Lumbar		**Pelvis**		**Knee**	
	Component		Component		Component	
	1	2	1	2	1	2
LIP-S	.561	-.192	.557	-.257	.513	-.252
POR-S	.501	-.163	.566	-.094	.499	-.272
EBR-S	.354	.934	.279	.953	.450	.892
OPH	.557	-.253	.539	.128	.532	-.256
Eigenvalue	2.849	0.737	2.412	0.889	2.631	0.585
% Total Variance	71.22%	18.44%	60.31%	22.24%	65.75%	14.63%

Table 3.11. Spearman's rank-order correlation coefficients[a] ($P < 0.01$) between OA scores[b] from PCA on four severity variables and Buikstra and Ubelaker's (1994) original distribution traits

	Lumbar OA Score (Y_3)	Pelvis OA Score (Y_3)	Knee OA Score (Y_3)
	r_s	r_s	r_s
LIP-D	0.811	0.819	0.830
POR-D	0.710	0.759	0.693
EBR-D	0.407	0.308	0.502

[a] See Table 3.9 for description of Spearman's rho (r_s) correlation sizes.
[b] OA score ($Y_3 = |Y_1 \lambda_1| + |Y_2 \lambda_2|$) determined via PCA using a reduced set of severity variables: LIP-S, POR-S, EBR-S, OPH

3.4 Discussion

There are several benefits to employing PCA in bioarchaeological studies of OA pathology. PCA as an analytic technique is useful for understanding the multivariate structure of large OA datasets that contain groups of correlated variables (Jackson, 2003). In regression, *multicollinearity* refers to predictors that are correlated with other predictors, and results in factor redundancy. Given that adult bone remodelling cycles of resorption and formation are closely linked, all of the pathological changes we see in skeletal tissue are inherently interrelated, which make it difficult to fully understand how trait indicators of OA should be combined (or weighted) in a measurement of OA severity; an important fact ignored by the traditional method of averaging point estimate scoring. Multivariate techniques, like PCA are necessary to remove the correlation between variables so we can determine how much of the variation in each trait is contributing to differences in arthritic expression, and to use the variable coefficients (PC loadings) to examine their relative importance in arthritic patterning throughout the skeleton. The distribution of traits underlies the overall skeletal expression of OA that is represented by an aggregate score, which must be an accurate reflection of the mechanobiology of the whole joint. Composite OA scores generated from multidimensional scaling of PCA provide a more comprehensive and meaningful representation of mechanical stress than calculations of sample means (averages) for joint complexes that are biased by the total number of scored surfaces, and fail to account for trait distribution and variance.

More detailed and complete descriptions of arthritic lesions are favourable to understand the complex aetiopathogenesis of OA, which are captured by an ordered recording system like the one used in this study (after Buikstra and Ubelaker [1994]). However, choosing an appropriate scoring methodology depends largely on the question asked by the researcher. For example, paleoepidemiological investigations of OA typically score arthritic criteria dichotomously (i.e., present or absent) (Larsen et al., 1995), which is useful to explain prevalence and patterning of OA as it relates to age of onset, frequency of involvement of varied joints, as well as to compare incidence of age- and sex-specific variations associated with repetitive in-vivo behaviours in a specific population (Sofaer-Derevenski. 2000; Lieverse et al., 2007; Klaus et al., 2009; Schrader, 2012). A binary evaluative method provides a simplified platform for determining the appearance of OA, but by lumping observations into a single “arthritis present” score we lose the opportunity to record information about the individual joint surface examined, including changes in bone quality and differences in severity. Other, more complex evaluative methods that separate coding by joint

surface and by indicators of OA (e.g., lipping, porosity, eburnation, or surface osteophytes) into mild, moderate, and severe categories (Jurmain, 1990; Waldron and Rogers, 1991; Buikstra and Ubelaker, 1994; Jurmain and Kilgore, 1995) have the potential to produce a more detailed description of wear expression, but also result in a large database of raw observations that are nearly impossible to interpret without some manipulation of the data into representative joint scores. Principal component analysis is an effective tool with which to simplify and accurately describe complicated OA datasets that include scoring of multiple surfaces from varied joint locations using several discreet variables. In this study alone, up to 133 individual surface scores were generated for each specimen case, which over the entire sample equates to more than 38,000 records of OA in the skeleton. Considering the overwhelming amount of data produced here, results of PCA show that multidimensional scaling is useful to summarize variation in OA by grouping variables that are statistically associated, and to reveal wear patterns in the data that would otherwise be unnoticeable. Recognizing wear patterns is possible with PCA because OA data are represented by fewer dimensions making it easier to store, analyze, and handle the data; but at the same time, PCA preserves most of the information that the OA data represents, since a reduction in the number of dimensions will also result in a loss of some information.

Given the complex relationship between natural and activity-induced stresses related to skeletal expression of arthritic defects (Meyer et al., 2011), PCA allows for a systematic examination of the associations among marginal lipping, pitting, osteophyte formation and eburnation at the individual level as they differ in expression between (1) joint locations and (2) population groups. As expected, expressions of OA varied significantly in this study between the lumbar, pelvis, and knee regions (Fig. 3.6), the result of differential mechanical loads placed on these weight-bearing joints related to structure and mobility function (Rogers et al., 1987; Jurmain and Kilgore, 1995; Arden and Nevitt, 2006; Brown et al., 2008). For example, arthritic changes at fibrocartilaginous joints of the lumbar vertebrae typically exhibit a greater degree of marginal osteophytes in comparison to synovial joints of the os coxa and/or knee regions (Jurmain and Kilgore, 1995; Gold et al., 2007), which move more freely. Significant differences ($P < 0.001$) in LIP-S were observed between the lumbar and both the pelvic and knee regions (Table 3.6) indicating that osteophyte development at vertebral margins was quite different than arthritic lipping morphology occurring elsewhere in the skeleton. On this point, some studies classify modifications of vertebral body margins as ‘vertebral osteophytosis’, or ‘spondlyosis’, reserving the term ‘osteoarthritis’ for synovial joint involvement (Jurmain and Kilgore, 1995), but following the lead of other

paleopathology researchers (Larsen et al., 1995; Lieverse et al., 2007; Klaus et al., 2009) and to demonstrate how multivariate techniques can help to explain varied wear patterns, that distinction is not made here. Since we do not see a homogenous patterning of osseous change throughout moveable skeletal joints, weighting subjects by variable coefficients generated from joint-specific PCA is an effective way to capture regional OA variation, rather than relying on the distribution of a singular trait (i.e., lipping), with the possibility to then compare these results between groups and between individual skeletal sites to consider how abnormal joint loading leads to its failure.

Regional variation in idiopathic joint OA can have a significant effect on our interpretation of the past. Lieverse et al. (2015) demonstrate behavioural variability in middle Holocene foragers from Cis-Baikal through a comparative examination of OA severity in upper versus lower limb joints from three cemetery populations (8000-4000 cal. BP). Their results of OA analyses in the elbow show a different activity wear pattern than was found in other hunter-gatherer populations studied by Ortner (1968). The contrast suggests that degenerative changes at the distal humerus results from varied mechanical stress, prompting a critical review of adaptive response to loading at this area of the upper limb as a consequence of in-vivo behaviours and activity. Other studies, such as that by Klaus et al. (2009) show complex distributions of OA related to economic intensification as a result of mining, agriculture, and pastoralism in native Peruvians (AD 900-1532). Whereas Shrader (2012) was able to ascribe the patterning of OA in New Kingdom Nubians to low levels of physical activity that the middle-to-upper socioeconomic classes living in the Egyptian Empire (16th-11th century BC) would have enjoyed.

Eburnation, however rare in a paleopathological context (Shrader, 2012) is the most reliable indicator of OA (Weiss and Jurmain, 2007) and significant to interpreting later stages of joint tissue degeneration (Shepstone et al., 1999; 2001). Its separation on PC2 is supported by this fact, as well as by the effect of EBR-S/D scoring on the overall composite evaluation (*Y3*), which is driven up to reflect a more severe expression of tissue damage (Fig. 3.5). Bone eburnation is a common anatomical indicator of chronic arthropathy (Lagier, 2006), so it makes sense that later stage expressions of tissue damage would be separated in PCA. We might also expect to see lower frequencies of EBR-S/D compared to all other variables (lipping, porosity, osteophytes) due to varied mechanical stress operating on different joint systems (Fig. A5). Regardless of their irregularity, features of eburnation should not be left out of analyses.

An important finding from clinical research has demonstrated that OA is in fact an active repair process, and not always progressively degenerative (Dieppe, 2011). Herein lies a key

difference between clinical and paleopathological interpretations of OA, where skeletal analyses focus on describing OA as a deteriorating cartilage disease, culminating in bone-on-bone contact. On this point it is no wonder why eburnation has been considered the most severe expression of OA in paleoepidemiological studies. Based on the fact that (1) joint failure can result from problems in any of the surrounding joint tissues, and that (2) periods of joint stabilization do occur (i.e., OA can become *inactive*) (Dieppe et al., 1993), it is plausible that eburnation may occur without any evidence of porosity or osteophytic development (as seen in this study and reflected by moderate OA scores ~15, see Fig. 3.5). Likewise, patterns of osseous change mediated by habitual loading and abnormal mechanical stress may also result in complete ankylosis of the joint, bypassing bone-to-bone contact (i.e., no evidence of eburnation) and rendering the joint immobile (as demonstrated by the highest OA scores, see Fig. 3.5). Surely, joint immobility—leading to disability could be considered the "most severe" in the context of evaluating gross anatomy, particularly since pain (symptomatic of OA in life) is not visible in the archaeological record. Understanding the mechanobiology of moveable joints in past populations is critical to test the assumption that OA pathology is a reflection of behaviour; but to do so we need (1) a reliable method to quantify OA expression and (2) a tool to measure both individual variation (by skeletal region) and population differences in biomechanics and biology.

In the sample population, variables were summarized into two components that can be plotted based on groupings by age, sex (Figs. A8–A9), or any other variable (e.g., body size) to look for patterns among individuals within the dataset. Yet, PCA is also useful to generate population-specific distributions of OA for standardized comparison between skeletal samples. As other studies have shown, dissimilarities in the number of scored variables/joints/surfaces can result in widely different estimates of OA severity leading to difficulties in relative comparison between analyses (Bridges, 1993; Jurmain and Kilgore, 1995). But OA itself is an observed variable that *cannot be measured directly*; therefore, we must capture (infer) its expression indirectly using other variables that can be observed (such as those used in this study: lipping, porosity, osteophytes, and eburnation). Components are empirically determined aggregates of the variables (e.g., LIP-S, POR-S etc.) without presumed theory of their interrelatedness (Krzanowski, 2000), so it is possible to identify the underlying structure of those variables (how they combine) to describe their contribution to variation in how OA is presented skeletally. For example, LIP-S/D, POR-S/D, and OPH had loadings >0.40 on the first PC, while EBR-S/D was weighted lower at 0.29 (Table 3.7). Together lipping, porosity, and osteophytes determined most of the variance explained by PC1,

which in this case is the suite of traits that has a strong affect on early stage expressions of joint tissue damage. Principal components have the benefit of being uncorrelated since they are orthogonal to each other, and are beneficial in analyses of OA to reduce biological noise within the sample. In addition to comparisons of the aggregate OA scores (*Y3*) to interpret severity in bioarchaeological populations, investigators should look for statistically significant differences between eigenvector loadings, eigenvalues (i.e., population variance), and individual PC scores that are weighted linear combinations of the variables derived from PCA. Regression loadings are optimal because no other series of weights could produce a set of components that are more successful in explaining the variation in the observed variables (Vinayakam and Sekar, 2013). Comparing PCs between populations and within samples allows us to consider the mechanobiology of arthropathies, such as OA, as well as the variation in joint pathology that results from a suite of inclusive traits.

3.4.1 Severity and distribution of OA in the skeletal record

Interpretations of the severity and distribution of OA joint pathology have been used to broadly explain mechanically strenuous lifestyles and patterns of activity in a bioarchaeological context (Sofaer-Derevenski, 2000; Lieverse et al., 2007; Klaus et al., 2009; Schrader, 2012). While specific habitual activities are not likely informed through an examination of musculoskeletal disorders (Jurmain et al., 2012), it is possible to use OA as a general marker of occupational stress to approximate how humans interact with their environment. More recently, OA characteristics have been successfully employed in the assessment of skeletal age-at-death for much older adults (i.e., 65+ years) (Calce, 2012; Falys and Prangle, 2015). Progressive changes related to osteophytes and porosity show a significant correlation with increasing age in European ancestral populations, particularly for individuals living into, and well past their seventh decade (Calce, 2012; Falys and Prangle, 2015). The cumulative effects of age and mechanical stress on bone quality clearly warrant further inspection, particularly to interpret OA expression as it relates to past behaviour and biological aging of skeletal structures.

Though often incorporated into both archaeological and osteological analyses, there is no agreement among bioarchaeologists on the number of criteria to establish the presence and severity of joint OA (Weiss and Jurmain, 2007). Individual researchers seem to select skeletal

evaluations of marginal changes (lipping, and/or osteophytic bone spur development) and those observed on articular surfaces (porosity, eburnation, and exostoses) somewhat arbitrarily, but the discrepancies are likely owing to disparities between clinical epidemiological and paleopathological investigations of OA. For example, in her study on hand OA, Weiss (2013) did not include measures of eburnation, which has been consistently argued as the best and most reliable indicator of OA and has also been presented by others as late stage arthritic development (Shepstone et al., 1999; Arden and Nevitt, 2006; Weiss and Jurmain, 2007; Plomp et al., 2013; Weiss, 2014). Waldron and Rogers (1991) advocate for the examination of exostoses (raised bony nodules on articular joint surfaces) in evaluating OA severity because these are typically observed radiographically (as in Habermeyer et al., 2006); and only Buikstra and Ubelaker (1994) encourage a separate measurement of the amount of surface area affected by various OA indicators (lipping, porosity, eburnation). In this study both POR-S and POR-D variables demonstrated a strong association with osteophytic bone formation in the lumbar and pelvic regions, yet the validity of these features as good indicators of OA have been questioned (Rogers et al., 1997; Rothschild, 1997; Sowers, 2001; Schmitt et al., 2007). While we do not quite understand the pathogenic process of pitting in articular surfaces that may form simply as a result of natural occurrence on bone (Rothschild, 1997; Jurmain, 1999; Weiss, 2014), or from subchondral microcysts (Binks et al., 2013), both clinical and longitudinal experimental studies have shown that porotic changes in subchondral bone were found to precede cartilage damage (Botter et al., 2011; Sulzbacher, 2013), which supports a primary alteration characteristic of early phase OA development. It may be premature to discard either OPHs or porosity as reliable indicators of OA where more research is needed to determine whether these develop mainly as a result of biological aging and/or metabolic risk factors (Rogers et al., 1997; Sowers, 2001; Schmitt et al., 2007).

Buikstra and Ubelaker's (1994) OA scoring method is the most detailed among those published using an ordinal system (e.g., Jurmain, 1990; Waldron and Rogers, 1991), categorizing ranked degrees of OA expression beyond simple measures of 'mild', 'moderate', and 'severe' (Table 3.2) and including separate ordered measures for percentage of surface area affected. It is relevant to consider the nature of the relationship between severity and distribution variables to determine whether both are necessary to capture variation in pathological response to activity, or if severity measures alone can accurately qualify progression in arthritic traits. Through PCA, this study has shown that to include them together as separate indicators of OA may be introducing redundancy in scoring (i.e., measuring the same marker of severity twice), which highlights another advantage

of PCA to extract and simplify relevant OA variation. Naturally, this does not exclude the possibility that a different pattern and relationship between OA severity and distribution may be observed in other population datasets, for which PCA should be employed to (1) describe discrete OA samples, and (2) as a standardized procedure in computing population-specific representative measurements of idiopathic joint OA.

3.4.2 Other statistical methods to evaluate OA

Observations that degenerative lesions occur, or even that they can be occasionally severe are by themselves of no particular interest (Jurmain, 1990). Rather, we must also investigate reasons for pathological expression in skeletal biology to explain OA pathogenesis and to discover the aetiology of arthritic patterning in joint systems. Early research of OA by biological anthropologists focused heavily on description and classification of the disease in past populations (Rogers et al., 1987; Rogers, 1966), but with new powerful research tools bioarchaeologists can also contribute to diagnostic considerations of OA (Nikita et al., 2013; Weiss, 2006). Nikita and colleagues (2013) demonstrate the potential of generalized linear models in analyses of OA from dichotomous data (i.e., presence/absence). Since the aetiology of OA is multifactorial, the study by Nikita et al. (2013) is relevant to the investigation of OA in a bioarchaeological context in that it supports the possibility to explore the simultaneous effects of several predictive variables on the expression of skeletal OA. Other conventional methods of statistical analyses such as odds ratios of binomial distributions have been useful to examine the effects of behaviour on arthritic development (Klaus et al., 2009), but are limited in their power by considering a single independent variable, or the relationship between only two variables on OA response. In order to determine the relative impact and interaction of *multiple* underlying factors that shape the pathogenesis of skeletal OA, a more advanced and sophisticated statistical approach, like the use of multiple regression models is required. Bioarchaeologists are responsible for improving their skills to diagnose and interpret skeletal abnormalities, and to develop and implement technical standards. The results of this study add to Nikita and colleagues' research by proposing a standardized procedure for computing composite OA scores (from a ranked multipoint ordinal scale) using PCA that can later be applied in regression models to investigate the relationship between OA and other (multiple) independent variables.

3.5 Conclusions

This paper examines the potential of PCA for dealing with OA data of multiple variables that have been recorded using a ranked ordinal scoring system, and shows that PCA is an appropriate way to create a representative arthritis score for major joints or combined joint regions. Researchers should avoid estimating a general skeleton score to account for joint-specific OA variation affected by differential mechanical loadings that will produce varied osseous patterns in lipping, porosity, eburnation, and surface osteophytes. Joint-by-joint comparisons can be performed separately by PCA and OA scores comprised of a different number of traits, or by different weights for each trait. As a data reduction technique, PCA is a simple, non-parametric method of extracting relevant information from complex datasets and summarizes the variation based on correlated multi-attributes to reveal a simplified structure of OA expression (Krzanowski, 2000; Astephen and Deluzio, 2004). Bioarchaeologists should employ multivariate techniques like PCA to describe discrete OA samples, and as a standardized procedure in computing population-specific representative measurements for idiopathic joint OA. Composite scores can later be analyzed through the robust technique of multiple regression models to investigate the combined effects of systemic and local mechanical risk factors such as age, sex, activity, and body size on skeletal responses in joints. Finally, osteologists must continue to critically review both their methods and study samples to provide the most objective analysis of human adaptation and disease in the past. This research demonstrates that PCA is an effective tool with which to investigate the relationship between severity and distribution of OA in a skeletal population.

Chapter 4.
Effects of age, activity, and body size on skeletal expression of osteoarthritis in a modern European sample

4.1 Introduction

Osteoarthritis (OA) is the most common joint pathology in human populations, studied extensively for more than 60 years by both clinicians and osteologists to produce an amazingly deep and rich literature base (Rogers et al., 1987; Jurmain, 1999; Karsenty, 2003; Arden and Nevitt, 2006; Waldron, 2012). Clinical research has focused largely on cellular destruction of articular cartilage, but new diagnostic tools (e.g., MRI, bone scans), and biomarker discoveries have revealed four important results: (1) OA is not entirely a cartilage problem and can affect varied joint tissues, (2) OA is not a purely degenerative disorder, but also a reparative one, (3) OA is not necessarily progressive with potential to stabilize from changes in joint anatomy, and (4) OA is a focal pathology occurring in habitually loaded joint areas (Felson and Nevitt, 2004; Dieppe, 2011; Waldron, 2012). If OA is joint failure driven by cumulative or abnormal joint loading, rather than disease, then paleoepidemiological analyses of OA have much to contribute to a discussion of how environmental factors influence human biology via questions regarding mobility, habitual behaviours, and aging in archaeological populations.

Despite major advancements in knowledge, the aetiopathogenesis of OA is complex and still poorly understood (Felson et al., 2000). The interplay between systemic influences (e.g., age, sex, hormones, nutrition, genetics) and local biomechanical risk factors (e.g., muscle weakness, obesity, and physical activity) requires further study to determine whether OA represents a single state or a heterogeneous cluster of conditions that share a common final pathway (Felson et al., 2000; Sowers, 2001). Research on OA prevalence has consistently demonstrated that it increases with age (Jurmain, 1991; Loeser, 2010) and that sex-specific differences are evident (Oliveria et al., 1995), but complexities of bone biology, limited samples, and fragmentary preservation have restricted behaviourally-oriented research in the archaeological record, and osteologists are rightfully cautious in interpreting both causal mechanisms and specific activities from bone pathologies (Weiss, 2005; Weiss, 2006; Jurmain et al., 2012). Studies that consider joint changes of OA as a

function of both the natural aging process and of mechanical stress (wear-and-tear) are necessary to recognize diverse aetiologies of OA, particularly in relation to clinical diagnoses, risks, and treatment, but also to uniquely document bony lesions associated with OA biology that can only be studied through direct observation of articular bone surfaces.

The relationship between joint OA and excessive mechanical stress can be assessed through variation in long bone diaphyseal shape. The distribution of cortical bone reflects its loading history; therefore, skeletal remodelling of the long bone diaphysis is a useful measure of behavioural inference (Ruff and Hayes, 1983). The seminal research by Ruff and colleagues (1982; 1983; 1984; 2000; 2006), Stock and Shaw (2007), and Sparacello and Pearson (2010) have shown how measures of bone robusticity address long standing anthropological inquiries regarding adaptive subsistence practices, variability in skeletal growth and development, and relative limb strength and locomotion. As such, long bone cross sectional geometric (CSG) properties (e.g., strength) may also be useful to investigate reasons for the onset and development of musculoskeletal disorders, such as OA, that are driven by biomechanical forces as much as by genetic influence and biological aging. Cumulative or abnormal loading of the skeleton demands that weight-bearing joints respond and adapt to mechanical stresses and strains, which may eventually be compensated by arthritic changes to bone (Stürmer et al., 2000; Berenbaum and Sellam, 2008). It is important to consider whether arthritic joint lesions (e.g., osteophyte formation) are simply a functional adaptation to loads imposed on the plastic skeleton to promote joint stability (Dieppe et al., 1993), generated by different lifestyles.

Body size estimates are important to reconstruct various attributes of past populations that include demographic characteristics, to assess health, and to recognize patterns of skeletal morphology affected by activity loading patterns (Steckel and Rose, 2002; Ruff et al., 2006; Cohen and Crane-Kramer, 2007). Large body size correlates with generalized bone hypertrophy and with OA characteristics, of which osteophytes are the most consistent variable (Spector et al., 1996). Body weight is distributed primarily in the lower limb (Ruff, 2000), and moveable joints such as the hip and knee, are highly affected by OA (Larsen, 1997; Felson et al., 2000; Wearing et al., 2006; Jiang et al., 2011). Forces transmitted across the knee joint during normal gait range between two-and-three times body weight, a load effect that explains the increased risk for OA among overweight persons (D'Lima et al., 2012). Sandford and colleagues (2014) recently found that joint loads in the hip and knee increase approximately linearly with body mass, which strengthens the findings that body mass could be a factor linking obesity to OA (Oliveria et al., 1999; Powell et al., 2005; D'Lima

et al., 2012). An understanding of structural factors/limitations that contribute to both hip and knee OA is advancing rapidly (Lane et al., 2000).

Effects of stature on arthritic development are less well understood, although new research using biological markers (i.e., single nucleotide polymorphisms, SNPs) to identify genetic variants associated with height has identified a link between short stature and susceptibility to OA (Sanna et al., 2008). Gene polymorphisms may be linked to increased susceptibility to OA, because mutations that reduce the functional levels of cartilage matrix proteins or alter key interactions in their assembly and function could be expected to compromise the biomechanics of joint cartilage (Kannu et al., 2009). Genome-wide association studies represent a promising way to study complex, common, chronic disorders like OA that incorporate body size variables highly influenced by both genes and the environment. For paleopathologists, the relationship between overall body size and the mechanical stress threshold of weight-bearing joints may be specifically important factors to control for in evaluating joint failure and arthritic patterning in once-living populations.

The purpose of this study is to test the hypothesis that differences in adult patterns of age, activity, and body size are reflected in joint arthritic changes through an examination of OA in weight-bearing regions of the lumbar spine, pelvis, and knee for a modern 19th-20th century European skeletal population. Explaining population variation in OA by age, activity, or body size is relevant because OA aetiology is multifactorial. This bioarchaeological study examines the simultaneous impact of multiple underlying factors on the expression of idiopathic OA in weight bearing joints of the lower limb from a skeletal series; results of which aim to improve behavioural reconstruction and interpretation of disease determinants in the past.

4.2 Materials and methods

4.2.1 Sample

The study sample (n=124) was derived from two large modern European identified skeletal populations from Portugal (Luís Lopes, University of Lisbon) and Italy (Sassari, University of Bologna), grouped together based on a shared population history of Southern Europe with similar activities and lifestyle at the turn of the 20th century (Cardoso, 2006; Belcastro et al., 2008). Both populations represent a low socioeconomic class, evidenced by documented occupations in rural farming/manual trades (males) and managing the family household (females). Exhumed from

municipal cemeteries in the last 50 years, complete skeletons for individuals who died between 1912-1970 were evaluated and known sex and age-at-death were recorded for each individual from documents accompanying the collections. Summary data for age and sex of the test sample are provided in Table 4.1.

Sample individuals were selected randomly, but only individuals presenting with the requisite morphology were evaluated. Specimens affected by OA related to fractures were excluded, as were individuals with gross pathological evidence of infectious disease to avoid confusion with secondary OA that forms as a result of pre-existing abnormalities in joint tissues, and which may also influence diaphyseal morphology. Other criteria for selection were bone preservation and representation of adults in all age-classes. Elements from the left side were evaluated, unless missing from the collection in which case the right side was substituted.

Table 4.1. Summary age and sex data for the test sample

	Luís Lopes, University of Lisbon, Portugal				**Sassari, University of Bologna, Italy**				**Combined sample**			
Sex	n	Mean Age (years)	SD	Range (years)	n	Mean Age (years)	SD	Range (years)	n	Mean Age (years)	SD	Range (years)
Males	29	56.2	20.5	20-88	34	54.1	18.9	21-82	**63**	**55.1**	**19.5**	**20-88**
Females	33	61.2	21.0	18-94	28	61.0	19.4	24-98	**61**	**61.0**	**20.1**	**18-98**
Total	62	58.7	20.8	18-94	62	57.6	19.2	21-98	**124**	**58.1**	**20.0**	**18-98**

4.2.2 Variables

Osteoarthritis. Articular surfaces of the lumbar vertebrae, pelvis, and knee joints were scored macroscopically after the ordinal scale proposed by Buikstra and Ubelaker (1994) for severity of four arthritic traits: lipping, porosity, eburnation, and surface osteophytes (Table 4.2). Individuals presenting with diffuse idiopathic skeletal hyperostosis (DISH) were not excluded from the study since OA may develop in concert; however, cases of DISH (particularly where lumbar vertebrae were ankylosed) were differentiated from those with OA on the basis of the following DISH characteristics: (1) normal intervertebral disk space in fused vertebrae (including the thoracic

region), (2) absence of pathology (lipping, porosity, eburnation) in vertebral facet joints, and (3) extraspinal manifestations of new bone growth in ligament and tendon insertion sites (e.g., linea aspera, deltoid tubercle, and iliac crest) (Rogers et al., 1987). If the features described in 1-3 were present then the pathological diagnosis was DISH, and not OA. Each element was scored separately (Table 4.3) and a maximum of twenty-one articular surfaces were evaluated for each individual (Fig. 4.1). A principal component analysis (PCA) of the mean trait scores by region (lumbar vertebrae, pelvis, knee) was conducted to derive component-based OA scores reflective of severity in joint arthritic expression from the combination of loadings from the first and second principal components (see Chapter 3 for a complete discussion of the method to formulate an OA score using PCA). Components are empirically determined aggregates of the OA variables (i.e., lipping, porosity, eburnation, osteophytes) that are inherently related. A multivariate technique, like PCA, removes the correlation between these variables to determine how much of the variation in each trait is contributing to differences in arthritic expression, and uses the variable coefficients (PC loadings) to examine their relative importance in arthritic patterning throughout the skeleton. As a result, composite OA scores account for trait distribution and variance within the sample, reflecting a comprehensive and meaningful representation of mechanical stress. Involvement of OA at multiple joints is common and a heterogeneous pattern of osseous change is observed in joints affected by differential mechanical loadings (Arden and Nevitt, 2006; Brown et al., 2008). The lumbar vertebrae, pelvis, and knee regions were analyzed separately to differentiate between distributions of idiopathic OA in the skeleton (Arden and Nevitt, 2006; Cushnaghan and Dieppe, 1991).

Body size. Variables selected for this study were chosen on the basis of their performance in previous analyses to estimate body mass (kg) and stature (cm) from postcranial elements (Ruff, 2002; Auerbach and Ruff, 2004; Kurki et al., 2010; Ruff et al., 2012). Four skeletal measurements were collected (Fig. 4.2): superior-inferior femoral head breadth (FHB), maximum femur length (FXL), maximum tibia length (TIB), and bi-iliac breadth (BIB). Stature (ST) was estimated using sex-specific regression formulae based on long bone lengths of the femur and tibia of Holocene Southern Europeans, which is closest in geographical and temporal range to the target sample (Table 4.4) (Ruff et al., 2012). Body mass (kg) was calculated by averaging estimates from the 'mechanical' and 'morphometric' equations provided by Ruff et al. (2005; 2012) (Table 4.4). All variables were measured to the nearest millimeter with an osteometric board or digital sliding

calipers; then re-collected for 18 randomly selected individuals, with a one-week time lapse to test for intraobserver reliability (mean percent errors < 2%) (Appendix Table A5).

Activity. Three-dimensional surface models of complete femora were generated using the NextEngine™ laser scanner on the high-definition setting. Cross-sectional geometric properties were calculated using automated AsciiSection software (http://www.pave.bioanth.cam.ac.uk/software.html) to calculate polar second moments of area (*J*) at the 50% diaphyseal midshaft. The accuracy of calculating CSG from the periosteal contour has been demonstrated by other studies (Stock and Shaw 2007; Davies et al., 2012; Macintosh et al., 2013). These parameters were size standardized to the product of body mass and maximum bone length (Ruff, 2000) and used as a proxy for measures of activity (Bridges, 1989; Stock, 2006; Lieverse et al., 2011; Stock and Macintosh, 2015).

Table 4.2. Scoring system used to record OA severity after guidelines proposed by Buikstra and Ubelaker (1994), page 115 attachment 66

Feature	Description	Degree of Expression (Severity)
Lipping	Marginal proliferation of new bone in either a horizontal or vertical direction that produces a change in the shape of the joint contour.	0 – Not present 1 – Barely discernible 2 – Sharp ridge or curled spicule(s) 3 – Extensive spicule formation 4 – Ankylosis
Porosity	Pitting and/or erosion of the joint surface.	0 – Not present 1 – Pinpoint 2 – Coalesced 3 – Both pinpoint & coalesced
Eburnation	Polished subchondral bone with or without ridges (mechanical scoring).	0 – Not present 1 – Barely discernible 2 – Polish only 3 – Polish with grooves
Surface Osteophytes	Multiple proliferative changes of bone growth (bony spurs) that originate either at the joint rim, or on the articular surface.	0 – Not present 1 – Barely discernible 2 – Clearly present

Table 4.3. Articular surfaces scored separately for OA in the lumbar vertebrae, pelvis, and knee regions

Lumbar Vertebral Region (11—13 surfaces)[a]	Pelvic Region (5 surfaces)	Knee Region (3 surfaces)
1. Lumbar : Superior intervertebral body, superior articular facets (evaluated together as one surface) 2. Lumbar : Inferior intervertebral body, inferior articular facets (evaluated together as one surface) 3. Sacrum: Superior intervertebral body of first sacral vertebra, superior articular facets (evaluated together as one surface)	1. Sacrum: Sacroiliac joint 2. Os Coxa: Auricular surface 3. Os Coxa: Pubic symphysis 4. Os Coxa: Acetabulum 5. Femur: Head	1. Femur: Medial and lateral condyles 2. Patella: Condylar surfaces 3. Tibia: Medial and lateral condyles

[a] L1-L6 surfaces scored separately; individuals who had a sixth lumbar vertebra, n=2.

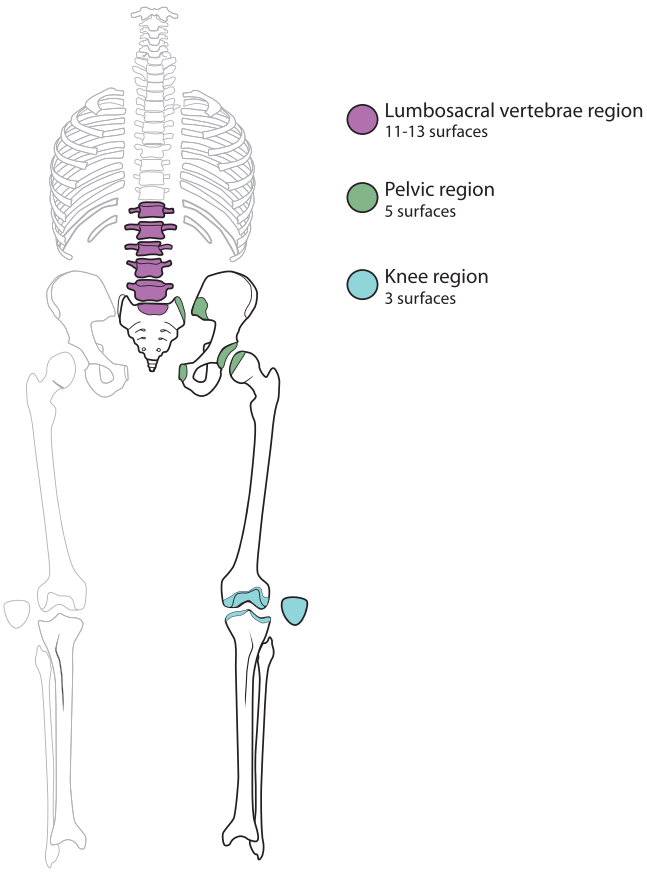


Figure 4.1. Joint surfaces for which OA scores were recorded, by anatomical region. (See also Table 4.3.)

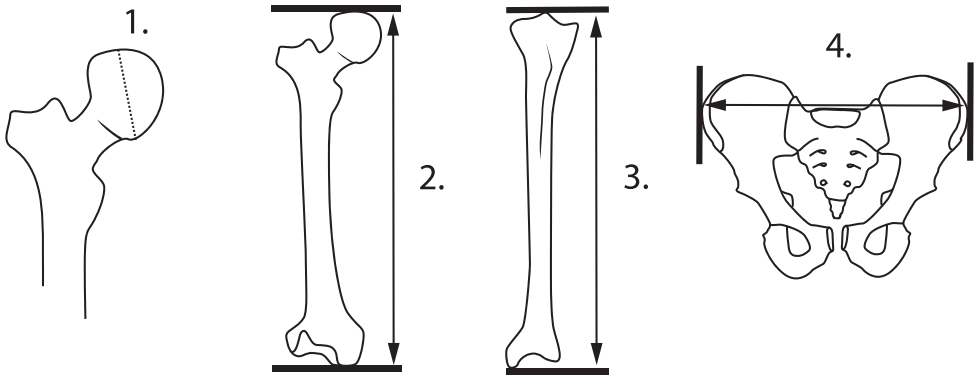


Figure 4.2. Post-cranial elements showing linear measurements used to calculate body size. 1. Superior-inferior femoral head breadth (FHB); 2. Maximum femur length (FXL); 3. Maximum tibia length (TIB); 4. Bi-iliac breadth (BIB).

Table 4.4. Published regression equations for estimating body mass and stature

Abbreviation	Method	Male	Female	Reference
ST	Stature (cm)	1.40*(FXL+TIB)+49.68	1.47*(FXL+TIB)+42.96	Ruff et al., 2012
FHB	Body mass (kg) "mechanical"	2.80*FHB–66.70	2.18*FHB–35.81	Ruff et al., 2012
ST/BIB[a]	Body mass (kg) "morphometric"	0.422*ST+3.126*BIB–92.9	0.504*ST+1.804*BIB-72.6	Ruff et al., 2005

[a] Corrected for "Living BIB" (1.17*BIB–3) (Ruff et al, 1997).

4.2.3 Analyses

A standard multiple regression was conducted separately by skeletal region to evaluate how well age, activity, and body size (independent variables) explain the variation in OA (dependent variable) at the lumbar spine, pelvis, and knee. A multiple regression analysis is appropriate to determine the overall fit of the model, as well as the relative contribution of each of the predictors

(age, activity, body size) that may have an effect on skeletal expression of OA in varied joint areas (McDonald, 2009).

The sample was pooled by sex given no significant differences ($P > 0.05$) in regional OA scores as determined by Mann Whitney U-tests. Data were normalized using base-10 log (*Y+1*) transformation of the OA knee variable, and a square root (*Y+1*) transformation for OA variables of the lumbar spine and pelvis regions. Except for *J* (a measure of torsional strength or overall loading on a bone), which was naturally log-transformed, all other independent variables (age, body mass, stature) were normally distributed. Assumptions of linearity, independence of errors, homoscedasticity, outliers, and normality of residuals were met in each of the regional analyses, presented together herein. All statistical analyses were performed in SPSS Version 22.0.

4.2.4 Expectations

The statistical approach of this study has two main goals: first, to test for significance of the overall regression model that will help to determine how much of the variation in OA can be explained by the combined effects of age, activity, and body size; and second, to test for significance of each independent variable in the model that will help to understand the relative contribution of each predictor on arthritic expression, as well as functional relationships between them. Based on these, the null hypotheses (H_0) are: (1) that there is no relationship between age, activity, and body size variables with severity of joint arthritic expression; and (2) that the addition of each independent variable (age, *J*, stature, body mass) to the multiple regression does not improve the fit of the model or contribute significantly to the prediction of OA beyond what could be expected by chance.

In our sample we expect to observe a strong positive linear relationship between the expression of arthritic defects and increasing age that may simply be the result of accumulated use over time, and then to determine what other factors contribute to OA pathology outside of the age-related expression. We expect to observe a positive correlation between OA severity and J that reflects habitual loading from repetitive activities and mechanical stress over the life course. Since both body mass and stature had to be estimated for the samples in this study, BMI could not be calculated. Instead, stature and body mass are evaluated as separate variables in the regression analysis to identify their independent relationships with OA. Although stature and mass are

positively correlated (taller people tend to be heavier), of the body size variables, we expect that body mass will have a greater effect on OA severity than stature based simply on increased biomechanical loading associated with carrying heavier weight.

4.3 Results

Summary data of OA measurements, body mass (kg), stature (cm) and *J* measurements are provided in Table 4.5.

4.3.1 Multiple regressions

In all joint regions (lumbar, pelvis, knee), the linear combination of age at death, body mass (kg), stature (cm), and torsional rigidity *(J)* of the femoral midshaft was significantly related to differences in OA expression as determined by an F-test of the model (Table 4.6, $P < 0.001$). A moderate-to-strong association was found between the independent (explanatory) variables and OA (Table 4.6), with the strongest association in the lumbar spine ($r = 0.806$, $P < 0.001$). Standard errors of the estimate (SEE) are low (0.232–0.469) across all joint regions, indicating that the regression model is a good fit of the data. Results of the model show that approximately 65%, 49%, and 30% of the variance in OA severity is accounted for by age, activity, and body size in joint areas of the lumbar spine, pelvis, and knee respectively (Table 4.6). Based on these results, we reject H_0; at least one of the independent variables is useful in explaining/predicting OA expression in these skeletal joint regions.

Although body mass and stature are correlated (R^2=0.599, P=0.001), the assumption of multicollinearity is not violated since tolerance is high (0.37) (Appendix Tables A6-A8) and greater than the recommended minimum of 0.25 (Huber and Stephens, 1993). As a result, both variables are retained in the analyses of multiple regressions.

Age was the strongest contributor to the prediction of arthritic expression across all skeletal joints examined in the analyses ($P < 0.001$, Table 4.7, Fig. 4.3), uniquely explaining between 25-56% of the variance in the total OA severity score (see semi-partial correlations listed in Table 4.7). Again, we reject H_0; the age variable does contribute and should remain in the model. In addition, body mass and increased torsional rigidity *(J)* at the femoral midshaft made significant

contributions to predicting OA expression in the pelvis (reject H_0 for these variables, $P < 0.05$, Table 4.7), but accounts for very little unique variance (6% combined) in the outcome of pelvic OA. Of these, *J* demonstrates a negative correlation with pelvic OA when controlled for both age and body size; that is, as femoral robusticity increases, the OA score decreases ($B = -0.914$, Table 4.7). The effect of increased activity on OA expression is not more severe as was expected; rather, higher activity levels may offer some kind of protective benefit from joint failure at the pelvis.

Neither body size, nor activity variables demonstrated a statistical relationship with OA expression in either of the lumbar vertebrae, or in the knee (accept H_0), which suggests that body mass, stature, and torsional rigidity are not relatively important in determining arthritic patterning in the study sample, and have little-to-no effect on differences in OA severity at the lumbar and knee joint complexes for this group.

Table 4.5. Summary data for each variable

	Males (n=63)				Females (n=61)				Combined sample (n=124)			
Variable	**n**	**Mean**	**SD**	**Range**	**n**	**Mean**	**SD**	**Range**	**n**	**Mean**	**SD**	**Range**
OA Lumbar[a]	62	5.79	3.63	0-12.28	61	6.37	3.87	0-13.93	123	6.08	3.75	0-13.93
OA Pelvis	63	3.99	2.52	0-10.82	61	4.18	2.66	0-11.01	124	4.07	2.59	0-11.01
OA Knee	63	2.63	2.02	0-9.43	61	3.19	2.98	0-12.39	124	2.91	2.54	0-12.39
Body Mass (kg)[b]	63	60.0	7.29	43.7-75.2	61	53.6	6.00	40.6-69.2	124	56.8	7.40	40.6-75.2
Stature (cm)[c]	63	162	6.51	145-175	61	154	5.53	142-167	124	158	7.15	142-175
Polar second moment of area (*J*)	63	433.3	87.5	232.5-729.9	61	393.7	82.4	232.1-673.8	124	413.8	87.0	232.1-728.9

[a] Composite OA scores for each region calculated via PCA (see Chapter 3)
[b] Body mass, calculated as the average of FHB and ST/BIB equations by Ruff et al. (2005; 2012)
[c] Stature, calculated from long bone lengths of the femur and tibia (Ruff et al., 2012)

Table 4.6. Summary of the multiple linear regression model by region

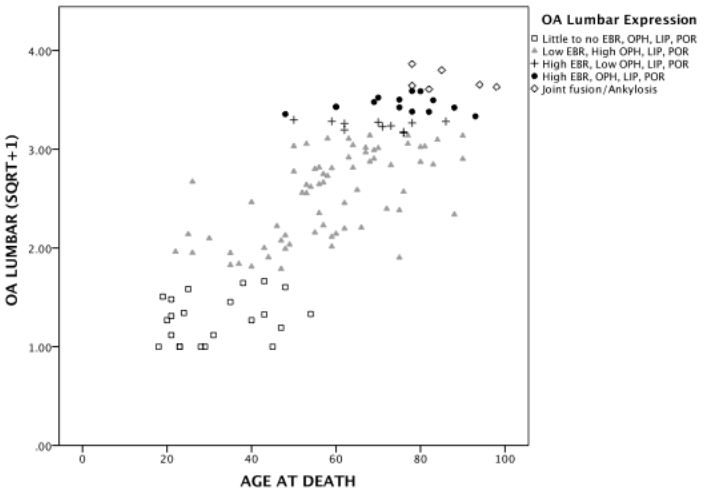
Dependent Variable	*R*	R^2	SEE	*P*-value
OA Lumbar [a]	0.806	0.650	0.469	**0.000**
OA Pelvis	0.700	0.490	0.425	**0.000**
OA Knee	0.552	0.304	0.232	**0.000**

R, multiple correlation coefficient; R^2, proportion of variance explained; SEE, standard error of the estimate; Significance level $P < 0.05$ for all tests
[a] Predictors: (constant), age at death, *J*, body mass (kg), stature (cm)

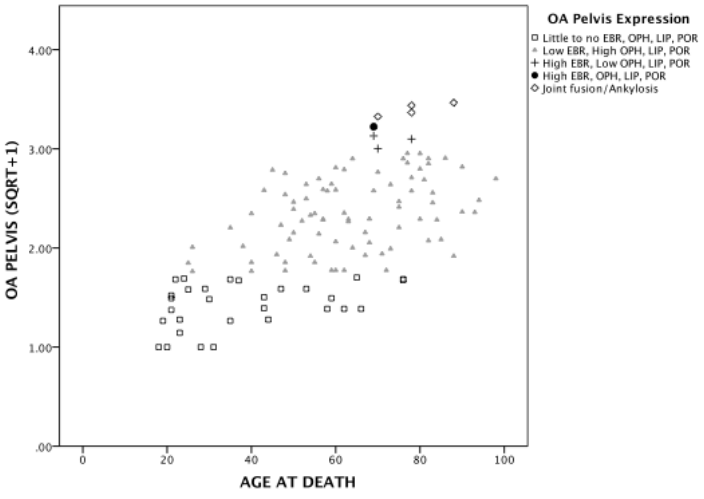
Table 4.7. Multiple linear regression coefficients by region

Model 1	OA Lumbar					OA Pelvis					OA Knee				
Predictor	*B*	*SE B*	*β*	*P*-value	Part	*B*	*SE B*	*β*	*P*-value	Part	*B*	*SE B*	*β*	*P*-value	Part
(Constant)	-0.579	1.739				4.008	1.567				-1.527	0.858			
Age	0.031	0.002	0.787	**0.000**	0.747	0.019	0.002	0.633	**0.000**	0.601	0.007	0.001	0.534	**0.000**	0.507
J	0.385	0.476	0.045	0.421	0.044	-0.914	0.429	-0.141	**0.035**	-0.139	0.392	0.235	0.129	0.098	0.127
Body mass (kg)	0.010	0.009	0.094	0.294	0.057	0.026	0.008	0.328	**0.003**	0.202	-0.004	0.005	-0.103	0.408	-0.063
Stature (cm)	-0.001	0.010	-0.013	0.883	-0.008	-0.013	0.009	-0.155	0.152	-0.094	0.005	0.005	0.133	0.292	0.081

B, unstandardized regression coefficient; SE B, standard error; β, standardized regression coefficient; Part, semi-partial correlations. Significance level $P < 0.05$ bolded.



A.



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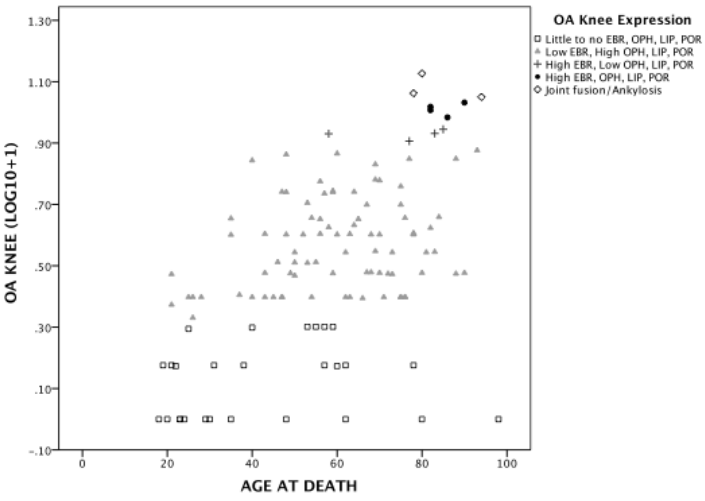

C.

Figure 4.3. Relationship of age to OA severity in the (a) lumbar spine, (b) pelvis, and (c) knee showing a positive linear relationship.

4.4 Discussion

Bioarchaeological investigations of OA commonly cite a significant, positive effect of age on OA severity (Weiss, 2006; Plomp et al., 2013), even after adjusting for differences in population age-structure, important in studies of skeletal pathologies that are known to co-vary with age (Baker and Pearson, 2006; Klaus et al., 2009). Results of this study agree with others that age-related factors are driving most of the changes associated with OA severity.

Large population-based clinical studies on joint-specific prevalence and incidence of OA in the elderly have consistently demonstrated an increase with age, with OA prevalence doubling in age cohorts 70+ years (Felson et al., 1995; Jordan et al., 2009). While the greatest risk factor for OA is older age, uniquely explaining up to 56% of the variation in OA severity of the lumbar spine in the study sample, OA is not an inevitable consequence of growing old. Aging *itself* is not a causal mechanism for OA, but age-related changes that affect both joint function and surrounding joint tissues increase susceptibility for OA to develop in older adults, especially when other factors (e.g., joint injury, obesity, genetics) are also present (Sowers, 2001; Anderson and Loeser, 2010).

To understand why aging is so highly correlated with the development of OA, the physiological aging process and associated pathological changes in arthritic joints must be established. Paleopathologists are indeed limited to diagnoses from the gross appearance of skeletal lesions, arrested at the time of death, and where all other soft tissues (muscles, ligaments, tendons) have decayed. Since changes both outside the joint (e.g., sarcopenia) and within the joint (e.g., cell and matrix alterations) contribute to the development of observable OA characteristics (osteophytes, eburnation, porosity), osteologists interpreting OA in past populations need to know the basic biology of musculoskeletal aging to truly appreciate the variation in arthritic severity presented skeletally, informed through clinical immunology and rheumatology research.

Not all older adults develop OA and not all joints of the body are affected to the same degree; study of individuals in the oldest senior age categories (70+ years) who have *not* developed OA will be important to discover what factors may be protective. In addition, consideration of other explanatory variables (e.g., nutritional, hormonal and genetic effects, along with joint shape analysis) is important to explain the remaining proportion of variance not described by the regression model of this study (e.g., approximately 70% unexplained at the knee).

4.4.1 Body Size and Activity with OA

Despite males being taller, heavier and more femorally robust than females, no differences in the severity of lumbar spine, pelvis, or knee OA were found between sexes, which is contrary to results from both paleopathological and clinical literature (Oliveria et al., 1995; Sofaer-Derevenski, 2000). Mechanical loading during life can have an effect on bone morphology (Ruff et al., 2006), suggesting two explanations for this result. First, similarities of OA severity between males and females imply variations in applied loading during life—a diverse activity pattern wherein no evidence of specific (habitual) behaviours is observed. Klaus et al. (2009) and Lieverse et al. (2015) have linked OA to sex-related divisions of labour, but such populations are also known for their distinct and unique periods of cultural transition detailing post-contact adaptive shifts in economy, and in behavioural variability of Cis-Baikal hunter-gatherers (8000-4000 cal. BP). In contrast, both the Lisbon and Sassari samples are relatively modern, representing the low to middle socioeconomic strata of urban populations, that include a workforce divided between service/sales, skilled occupations such as craftsmen, housekeeper, teacher, or student with both sexes equally contributing to the household income.

Second, these results indicate that sex-specific body size variability has no affect on the severity of joint arthritic response. When controlling for age and sex, body mass did not correlate with OA, except at the pelvic region where it was significant, but weak (R^2=0.202, P=0.003). This result is in line with previous research by Weiss (2006) who also found a slight correlation of body size to pelvic OA in a hunter-gatherer population (500-1500AD), even though the clinical literature points to a clear association between body mass and severe knee OA with higher prevalence in women (Hart and Spector, 1993; Manninen et al., 1996; Felson et al., 1995; Srikanth et al., 2005). Perhaps the relationship between increased body mass and severe OA is undetectable in certain archaeological populations, given that most individuals are of relatively normal body mass (~40–75kg, as seen in the current study) and almost none are considered classically obese (>80kg). Obesity is a risk factor for lumbar, hip and knee OA resulting from joint overloading during weight-bearing activities, as well as from systemic/metabolic mechanisms that adversely affect cartilage and other joint structures (Arden and Nevitt, 2006; Gellhorn, 2013; Sandford et al., 2014). In living populations, this relationship has been studied extensively by comparing the presence of joint space narrowing and osteophytes from radiographs with individuals of high body mass index (BMI, kg/m^2), as well as through in-vivo gait analysis to measure compressive loads (Hochberg et al.,

1995; Sanford et al., 2014). This relationship has been difficult to study in the skeletal record because only a few anatomical collections contain documented height and weight at death; additionally, we have no knowledge of how BMI may have fluctuated in life. Further study of how body size affects joint arthritic severity is possible using more recent identified skeletal collections that contain a large proportion of individuals with a living body mass of greater than 80kg (Merritt, 2015).

Stature had no effect on OA severity in any joint region of this study, and as expected, did not contribute more variability to differences in OA than body mass. Compared to other European samples (Ruff et al., 2012), the sample is average in height, even though 30% of individuals were estimated at less than 5 feet tall. Stature is a complex polygenic trait influenced by both genes and environmental factors; more than 80% of the variation in height may be genetically determined (Sanna et al., 2008). Though none here are suffering from any obvious chondrodysplasias (e.g., dwarfism) known to be associated with an arthritic phenotype, population studies of molecular mechanisms may be important to explain the link between short stature and elevated risk of OA (Kannu et al., 2009).

Except for the pelvic area, neither body size, nor activity measures made a significant impact on the severity of OA expression in the lumbar spine or knee, for which there are three possible explanations. First, that pelvic OA demonstrates a mechanical stress (wear-and-tear) response to behavioural strains; second, that joints may differ in what strains they respond to adaptively (e.g., pelvis versus lumbar and knee); and third, that activity levels and variation in activity might simply be too low to detect in this relatively sedentary sample (as opposed to other terrestrial or marine mobility foraging populations).

Unexpectedly, increased femoral robusticity was significantly correlated with *lower* pelvic OA scores, suggesting that more torsional strength as a result of habitual loading over the life course is in some way protective from joint arthritic response, or that OA is not necessarily progressive from long-term repetition of force (Dieppe, 2011). One explanation for this result could be the fact that adults in this population were incredibly active as children, working in activities related to farming and agriculture that would have produced more robust bones measureable in adulthood (Cardoso and Henderson, 2010). Bone's considerable plasticity during the subadult growth period is crucial to formation and remodelling of cortical bone in response to mechanical loading throughout an individual's life, and is perhaps the only period wherein patterns of activity can be truly isolated (Pearson and Lieberman, 2004). Other unknown factors such as the age at

which individuals began their occupations, changes in profession, and additional activities (e.g., sport) also influence skeletal robusticity and the development of OA (Cardoso and Henderson, 2010; Shaw and Stock, 2009a). Or, as Bridges (1989; 1991) noted, OA and biomechanical measures of bone strength may be responses to different kinds of forces or types of activities, where OA forms as a result of a shorter, more intense period of abnormal loading.

Variability in pelvic OA that is affected by both body mass and activity has potentially significant consequences on our current methods to estimate age. Well-accepted methods (e.g., pubic symphysis, auricular surface) that rely heavily on texture degeneration and osteophytic bone growth to determine patterns of skeletal aging are actually evaluating the stages of OA progression that clearly arise from more than a cumulative biological response to loading in the pelvis. Though the combination of body size and activity accounted for very little unique variance (6% combined), future biological studies of age should control for both body mass and activity to explain portions of the unattributed variance in skeletal age estimations from the os coxa.

4.5 Conclusions

Most of the variation in lumbar, pelvic, and knee OA was explained by age in the sample. Age, body mass, and torsional strength of the femoral midshaft have explanatory and predictive power of OA severity at the pelvis, but this conclusion is model-specific. For this sample, osteoarthritis in pelvic joints demonstrates a different kind of mechanical stress (wear-and-tear) response to behavioural strains than diaphyseal strength, though it is possible that a new set of independent variables could yield a different relationship to pelvic OA expression. In this sample, the combination of body mass, stature, and torsional rigidity are not relatively important in determining arthritic patterning at the lumbar spine, or in the knee; but joints may differ in what strains they respond to adaptively. The in-vivo strain environment that drives both degenerative joint changes and bone functional adaptation is both complex and variable. Studies of activity-related skeletal changes, such as OA, would benefit from more nuanced approaches to understanding skeletal aging (e.g., changes in bone microarchitecture), particularly in females affected by hormonal changes related to the onset of menopause, as well as much older individuals in the senior age categories (70+ years) who show no progression of the disease. The point at which an activity is initiated is central to studying behaviourally based osseous changes (Shaw and Stock,

2009b); and so, the period of growth and development may be especially important to determine how bone robusticity affects arthritic response in weight bearing joints of the adult skeleton.

Multivariate research design provides greater insight into the potentially interactive effects of age, activity, body size and their affect on osteoarthritic joint severity. Such distinctions will allow us to further consider whether idiopathic joint OA acts as a potential limitation or benefit in deriving accurate skeletal age at death estimates that will contribute to standardized methods of OA measurement in biological anthropology and physiological degeneration of the skeleton due to aging.

Chapter 5.
The effects of osteoarthritis on age at death estimates from the human pelvis

5.1 Introduction

Determining age at death from skeletal elements has critical applications in both forensic science and bioarchaeology to aid in victim identification and to construct the biology, behaviour, ecology, and social structure of past populations. Growth and developmental periods of skeletal maturity cease by the onset of the third decade, after which rates of skeletal remodelling and degeneration, from which most methods of adult age estimation are derived, occur at a variable pace. Since the accuracy of current adult age at death estimation methods decrease as chronological age increases, degenerative changes within the skeleton could potentially yield useful data for establishing and narrowing age estimates for older individuals (Listi and Manheim, 2012; Falys and Prangle, 2015).

Osteoarthritis (OA), the most common joint disease in human populations, has long been used to describe age related changes in the skeleton. Eighty-five years ago, Sashin (1930) an orthopaedic surgeon, first interpreted the changes seen in articular cartilage of the sacroiliac joint as "osteoarthritic", and found that these changes were "progressive, and increase in extent and intensity with the age of the individual" (Sashin, 1930 p.909). He particularly noted the simultaneous formation of osteophytes as an age-related degenerative change. Subsequent to these evaluations, Lovejoy and colleagues (1985) developed the popular skeletal age estimation method of the auricular surface, which has been used widely in studies of biological anthropology and adult skeletal aging for more than a quarter century.

The usefulness of joint OA as an adult age at death indicator has been demonstrated in numerous other studies as well. Stewart (1958) studied vertebral OA and its significance in skeletal age estimation and found that osteophytic vertebral markers increase with age. Further research confirmed this fact, but that the highly variable pattern of OA in the vertebral column is only beneficial to derive general age estimates (Snodgrass, 2004; Watanabe and Terazawa, 2006; Kim et al., 2006; Listi and Manheim, 2012). Mapping of arthritic degeneration in the upper limb has shown

that progressive changes in surface topography, porosity, and osteophyte formation can refine age estimates in adults over 50 years at both the glenohumeral joint and the sternal end of the clavicle (Brennaman et al., 2015; Falys and Prangle, 2015). Finally, predictable age-progressive morphological changes that include features of OA have been identified in load-bearing pelvic joint regions of the pubic symphysis (Todd 1920; 1921; Brooks and Suchey, 1990), the sacroiliac (Lovejoy et al., 1985; Buckberry and Chamberlain, 2002), and more recently, the acetabulum (Rougé-Maillart et al., 2004; 2007; Calce 2012). Joint arthritic changes of the pubic symphysis evaluated by Brooks and Suchey's (1990) method include: lipping of the dorsal border, erosion of the rim, and pitting/porosity of the pubic symphyseal face. Buckberry and Chamberlain's (2002) auricular surface method captures age-related OA variation in micro/macroporosity of subchondral bone, osteophytic outgrowths, and lipping of the apical surface contour. Calce's (2012) acetabular method scores osteophyte development both of the marginal rim and of the anterior horn of the lunate surface, including porosity of the lunate surface. Well-developed methods using the pubic symphysis and auricular surface are the most commonly used approaches to determine patterns of skeletal aging (Garvin and Passalaqua, 2012). New research has shown that using additional arthritic traits in combination with these methods improves the accuracy and precision of estimating age at death for both young and older (70 years+) individuals (Sharman, 2014).

The os coxa is a popular choice for age estimation because it provides up to three independent sites of evaluation. However, its biomechanical role in transferring loads between the spine and lower limbs is known to affect joint surface morphology, changes that are also highly influenced by diet, disease, physical activity, and body size (Merritt, 2015; Wescott and Drew, 2015). As a result, cumulative or abnormal mechanical loading, changes in anatomical alignment, and movement are capable of producing greater severity and distribution of joint osteoarthritic lesions (i.e., osteophytes, porosity). These are also known to co-vary with age (Lovejoy et al., 1985; Spector et al., 1996; Loeser, 2010) and can make an individual appear older than their chronological age. These variables may have a significant impact on our current methods to derive accurate skeletal age at death estimates based on morphology of joint structures, especially in load-bearing joint regions affected by mechanical stress.

Osteoarthritis has a serious impact on quality of life and is a major cause of morbidity, disability, and health care utilization, affecting 1 in 10 North American adults (Brault et al., 2009; Hootman and Helmick, 2006; Bombardier et al., 2011). Osteoarthritis is a long term health condition that has been perceived as a disease of the elderly. However, nearly 3 in 5 people affected (58%) are younger than 65 years of age (Public Health Agency of Canada, 2011), 15% of which are

less than 44 years old (Barbour et al., 2013). By 2030, the number of North Americans with OA is predicted to increase by 124%, equating to approximately 18 million more people (Hootman and Helmick, 2006; Public Health Agency of Canada, 2011). Globally, musculoskeletal conditions affecting the hip represent a major health crisis in aging due largely to an increase in obesity, longer life expectancy, and an aging baby boomer population (Cross et al., 2014). It is clear that more individuals than ever before suffer from OA (particularly young adults), and prevalence is expected to increase significantly in the very short term. Reasonably, the modern skeletal record will reflect an extremely high prevalence of arthritic defects in joint areas currently used to estimate skeletal age at death, prompting the need for a critical re-evaluation of these techniques due to their impacts on efforts to identify individuals from forensic contexts.

The purpose of this study is to determine the effect of OA severity on the validity and reliability of three methods to estimate age at death from load-bearing joints of the os coxa: (1) the pubic symphysis (Brooks and Suchey, 1990), the auricular surface (Buckberry and Chamberlain, 2002), and the acetabulum (Calce, 2012). Results will reveal whether OA in adults acts as a potential limitation or benefit in deriving accurate skeletal age at death estimates from pelvic joint morphology and will contribute to standardized methods in establishing physiological degeneration of the skeleton due to aging. It is not the purpose of this study to revise or improve skeletal age estimation methods, but rather to assess the role of osteoarthritic features in the estimation of adult age, so that we may properly interpret the expression of this pathological condition in the skeleton.

5.2 Materials and Methods

5.2.1 Sample

The study sample of 252 adults (128 males and 124 females) was derived from three large modern European identified skeletal collections: (1) the Luís Lopes Skeletal Collection (Portugal), (2) the Sassari Collection (Italy), and (3) the University of Athens Human Skeletal Reference Collection (Greece). These collections are appropriate to aggregate for this study because they are well preserved and derived from a limited temporal and geographic range from Southern Europe with similar activities and lifestyle, representing the low to middle socioeconomic strata of rural-to-urban populations at the turn of the 20th century (Belcastro et al., 2008; Cardoso, 2006; Manolis et

al., 2007). Exhumed from municipal cemeteries in the last 50 years, complete skeletons for individuals who died between 1880-1996 were evaluated and known sex and age at death were recorded for each individual from documents accompanying the collections.

Age at death ranged from 17 to 79 years, with a mean age of 50.9, S.D.=16.6; summary data for age and sex of the test sample are provided in Table 5.1. Individuals over 80 years were not used because the median age for Buckberry and Chamberlain's (2002) highest stage (VII) is 73 years, and the mean ages for Suchey-Brooks' last phase (VI) and Calce's oldest phase (III) are 61.2 years and 76.7 years respectively. Therefore, age in individuals 80-99 years would be most certainly underestimated; tests to evaluate error in age estimates would produce erroneous results regardless of OA severity or other factors.

Individuals were sampled randomly from each collection, and only those for which all three joint surfaces could be evaluated (auricular surface, pubic symphysis, acetabulum) were included in the study. To avoid confusion with secondary OA that forms as a result of pre-existing abnormalities in joint tissues, specimens affected by OA related to fractures and individuals with gross pathological evidence of infectious disease were excluded. Other criteria for selection were (1) less than 25% area damage at articular surfaces and margins and (2) sample representation of adults across all age classes (young, middle, old).

Table 5.1. Sample size and distribution

	Athens						**Lisbon**						**Sassari**						**Combined Sample**		
	Male			**Female**			**Male**			**Female**			**Male**			**Female**					
Age Class	N	Mean age (years)	S.D.	N	Mean age (years)	S.D.	N	Mean age (years)	S.D.	N	Mean age (years)	S.D.	N	Mean age (years)	S.D.	N	Mean age (years)	S.D.	N	Mean age (years)	S.D.
17-29	6	27.3	2.07	5	24.0	3.08	5	24.2	4.08	8	23.5	4.24	8	23.8	3.20	6	22.7	3.88	38	24.2	3.59
30-39	5	34.8	2.77	4	35.8	2.22	5	33.2	3.96	4	35.3	2.87	4	34.8	2.63	7	34.9	2.73	29	34.7	2.78
40-49	4	48.3	0.50	9	46.4	1.94	12	45.8	3.24	6	45.5	2.16	6	42.0	3.16	8	45.4	3.02	45	45.5	3.00
50-59	8	54.6	3.11	7	53.7	3.09	8	53.9	2.64	11	55.1	3.30	12	54.2	2.98	9	53.9	3.52	55	54.3	3.02
60-69	9	64.2	2.90	9	64.9	3.14	9	66.0	2.55	4	65.8	2.63	12	63.7	3.60	4	63.3	3.40	47	64.6	3.09
70-79	5	74.4	1.82	7	75.7	3.25	3	72.7	2.08	6	75.7	1.86	7	75.3	2.93	10	73.4	3.75	38	74.6	2.96
Total	37	51.8	16.1	41	53.0	16.5	42	49.5	15.0	39	49.4	17.8	49	51.5	17.1	44	50.3	17.4	252	50.9	16.6

5.2.2 Osteoarthritis

Five articular surfaces comprising joints of the pelvis were evaluated for OA expression: the sacroiliac joint of the sacrum, auricular surface, pubic symphysis, acetabulum, and the femoral head. Each surface was scored macroscopically using the ordinal scale proposed by Buikstra and Ubelaker (1994) for severity of four arthritic traits: lipping, porosity, eburnation, and surface osteophytes (Table 5.2). Individuals presenting with diffuse idiopathic skeletal hyperostosis (DISH) were not excluded from the study since OA may develop in concert with this condition. However, cases of DISH (particularly where lumbar vertebrae were ankylosed) were differentiated from those with OA on the basis of the following DISH characteristics: (1) maintenance of intervertebral disk space in fused vertebrae (including the thoracic region), (2) absence of lipping, porosity, and eburnation in vertebral facet joints, and (3) presence of extraspinal manifestations of new bone growth in ligament and tendon insertion sites (e.g., linea aspera, deltoid tubercle, and iliac crest) (Rogers et al., 1987). If these characteristics were present, then the pathological diagnosis was DISH, not OA. Elements from the left side were evaluated. If absent, elements from the right side were substituted.

A principal component analysis (PCA) of the mean trait scores was conducted to derive component-based OA scores reflective of severity in joint arthritic expression from the combination of loadings from the first and second principal components (see Chapter 3 for a complete discussion of the method to formulate an OA score using PCA). Components are empirically determined aggregates of the OA variables (i.e., lipping, porosity, eburnation, surface osteophytes) that are inherently related. A multivariate technique, like PCA, removes the correlation between these variables to determine how much of the variation in each trait is contributing to differences in arthritic expression, and uses the variable coefficients (PC loadings) to examine their relative importance in arthritic patterning throughout the skeleton. As a result, composite OA scores account for trait distribution and variance within the sample, reflecting a comprehensive and meaningful representation of systemic stress in the pelvis.

5.2.3 Age at death indicators

Three separate methods to document age-related changes in the os coxa were applied to each skeleton in the sample. Blind age assessments from the pubic symphysis (Brooks and Suchey, 1990), auricular surface (Buckberry and Chamberlain, 2002) and the acetabulum (Calce, 2012) were performed separately. All methods were applied as described by the authors and known ages at death recorded afterward. Sex-specific criteria for the Suchey-Brooks methods were employed, and in all cases the left side was evaluated. If missing, the right side was substituted. Each individual was assigned to an age phase (specified by the method), for each of the three methods employed.

Table 5.2. Scoring system used to record OA severity after guidelines proposed by Buikstra and Ubelaker (1994), page 115 attachment 66

Feature	Description	Degree of Expression (Severity)
Lipping	Marginal proliferation of new bone in either a horizontal or vertical direction that produces a change in the shape of the joint contour.	0 – Not present 1 – Barely discernible 2 – Sharp ridge or curled spicule(s) 3 – Extensive spicule formation 4 – Ankylosis
Porosity	Pitting and/or erosion of the joint surface.	0 – Not present 1 – Pinpoint 2 – Coalesced 3 – Both pinpoint & coalesced
Eburnation	Polished subchondral bone with or without ridges (mechanical scoring).	0 – Not present 1 – Barely discernible 2 – Polish only 3 – Polish with grooves
Surface Osteophytes	Multiple proliferative changes of bone growth (bony spurs) that originate either at the joint rim, or on the articular surface.	0 – Not present 1 – Barely discernible 2 – Clearly present

5.2.4 Analyses

Age and OA variables are normally distributed as assessed by Shapiro-Wilk's test ($P > 0.05$) (Appendix Tables A1 and A9). As independent t-tests of both age and OA variables revealed no significant differences ($P > 0.05$) between males and females, the sexes were pooled for all analyses (Appendix Table A10). The relationship between OA and age at death in the sample (n=252) was examined using Pearson's correlation.

Bias (directional error) and inaccuracy (absolute error) were calculated for each age method to determine: (1) whether the method under- or over-estimated age in individuals of differing age classes (bias = $\sum$ (estimated age–known age)/n) and (2) the average absolute error of age estimation (inaccuracy= $\sum$(|estimated age–known age|)/n). To evaluate the effect of OA on the validity and reliability of the Buckberry-Chamberlain, Suchey-Brooks, and Calce methods, a third variable, *estimate error* (a measure for accuracy of the age estimate) was calculated for each method for each individual. Estimate error was calculated by estimated age minus the known age, where the estimated age was the median of the age interval for the auricular surface, and the mean ages for the pubic symphysis and acetabulum (derived from statistics provided in the original studies) (Brooks and Suchey, 1990; Buckberry and Chamberlain, 2002; Calce, 2012). Individuals were collapsed into three age categories for comparison, (1) young adults 17-39 years, (2) middle adults 40-59 years, and (3) old adults 60-79 years. In addition, the sample was sorted by OA severity calculated by +/- 1 standard deviation from the mean OA score to produce three comparative groups: low, midrange, and high OA scores.

One-way ANOVAs and post-hoc tests were performed separately for each age method to identify differences in bias, inaccuracy, and estimate error between adult age groups and between OA severity groups. Tukey's honest significance difference (HSD) post hoc multiple comparison test was used, which controls for type 1 errors and is also appropriate for analysis of uneven sample sizes. Where homogeneity of variance was violated (Levene's test for equality, $P < 0.05$) Welch's ANOVA and Games-Howell post-hoc tests, which incorporate the samples sizes and variances of each group, were used.

An ordinary least squares regression examined the relationship between OA severity (independent variable) and estimate error (dependent variable) among age groups (young, middle, old) for each joint location. A neutral slope (zero error) would indicate that OA severity does not affect the accuracy of the age estimate.

A cumulative probit model was used to calculate the mean, standard deviation, and standard error of the ages-of-transition for each age estimation phase in the pubic symphysis, auricular surface, and acetabulum. Transition analysis is a parametric estimation procedure for modeling the passage of individuals from one developmental stage to the next higher stage in an ordered sequence, calculating the probability of the timing (i.e., age) of the transition from one phase to another (Boldsen et al., 2002; Kimmerle et al., 2008; Konigsberg et al., 2008). For this study, the estimated age phase served as the dependent variable; independent variables were log-age and OA. The natural log-scale assures that the transition distribution is log-normal. The asymmetry of the log-normal distribution is important to exclude ages-at-transition that are extremely young or negative ages-at-transition that would be invalid (Konigsberg et al., 2008). Likelihood ratio tests assessed significant differences between the log-age models and the log-age/OA models. Statistical analyses were performed in R version 3.1.1 (https://www.r-project.org/) and in SPSS version 23.0. Comparison of age at transition between OA severity groups was generated using the Nphases2 program (http://konig.la.utk.edu/nphases2.htm).

5.2.5 Expectations

Since age uniquely explains more than 1/3 of the variance in pelvic arthritic expression for the Lisbon and Sassari sub-samples (n=124) (see Chapter 4), we expect to observe a strong positive linear relationship between OA severity and increasing chronological age in the total sample combined (n=252). If this is true, and if osteoarthritic changes make an individual's skeleton appear older than their true age, then three hypotheses emerge. First, age at death would be overestimated more frequently for young individuals (≤39 years) with high OA severity scores. Second, as a result of over-aging young persons with OA, variation in absolute error of the age estimate will be greater for individuals with high composite OA scores compared to those with lower scores. Third, persons with evidence of severe joint arthritic expression will have lower ages at transition between phases for each age method, i.e., transition from one phase to the next at an earlier age.

5.3 Results

Summary data of OA measurements and sample distribution of age phases by method are provided in Tables 5.3 and 5.4. There was a moderate positive correlation between OA severity and age at death in the sample (R =0.641, $P < 0.01$), with OA severity explaining 41.1% of the variation in age (Fig. 5.1). Bias and inaccuracy results are presented in Table 5.5. There was a moderate to strong correlation between the estimated and chronological age at death for each method: Buckberry and Chamberlain ($R = 0.653$, $P < 0.001$), Suchey-Brooks ($R = 0.695$, $P < 0.001$), and Calce ($R = 0.765$, $P < 0.001$).

Table 5.6 shows the descriptive statistics for the ages at transition for each age estimation method generated from the cumulative probit model without considering the factor of OA. As expected, age ranges for skeletons from old individuals are broader than those for young individuals, evidenced by lower standard deviations for early phases (I-II) across all methods. Inter-individual variation in the timing of age-related senescent changes are much greater as chronological age increases; this trend is consistent among all three joint locations of the os coxa. Generally, standard error was minimal for the predicted probability ages at transition between phases, except for the transition stage VI-VII of Buckberry and Chamberlain's method (standard error=14.15), indicating a high amount of variation in late stage degenerative changes at the auricular surface. Comparatively, the slowest rate of age progression was observed at the acetabulum (i.e., highest age at transition between phases I-II), and the earliest age-related changes were found in the auricular surface (i.e., demonstrated by the lowest ages at transition between phases I-II).

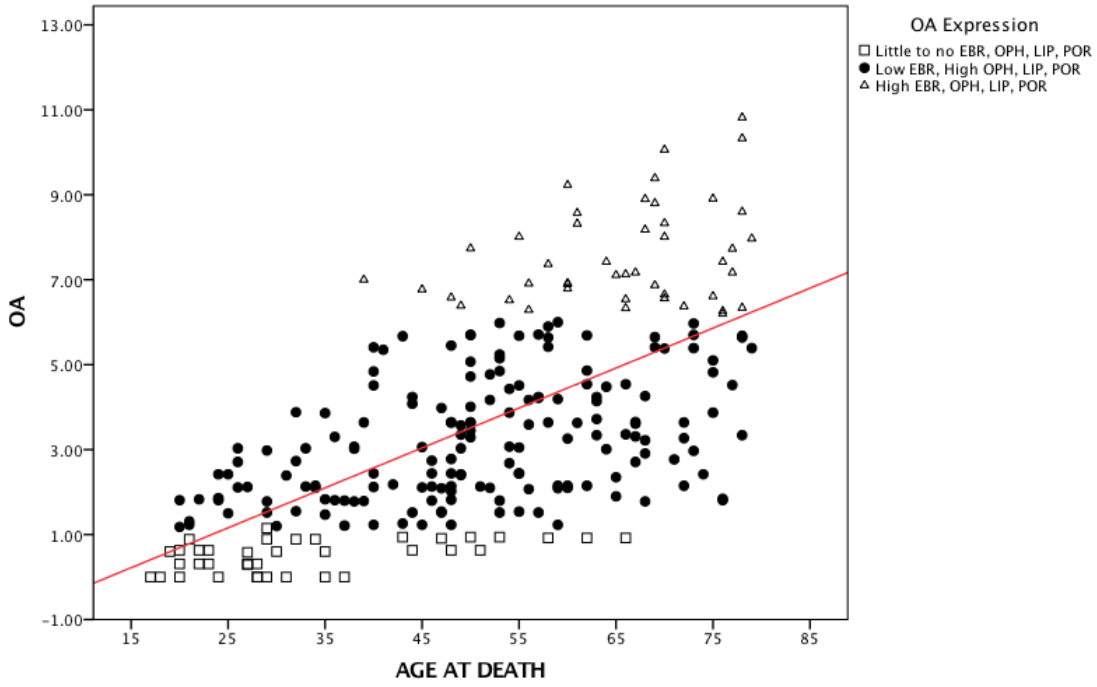

Figure 5.1. Relationship of age to OA severity (n=252) showing a moderate positive linear relationship (R=0.641, P<0.01; R^2=0.411)

Table 5.3. Summary data for OA composite scores[a]

Collection	Sex	N	Mean	SD	Minimum	Median	Maximum
Athens	Male	37	4.00	2.12	0.58	3.64	8.57
	Female	41	3.61	1.92	0.29	3.80	7.97
Lisbon	Male	42	2.03	1.40	0	2.05	5.70
	Female	39	1.87	1.76	0	1.82	7.17
Sassari	Male	44	5.20	2.44	1.23	5.30	10.82
	Female	49	4.48	2.68	0	4.54	10.06
Combined Sample	Male	128	3.82	2.44	0	3.64	10.82
	Female	124	3.37	2.42	0	3.59	10.06
	Total	252	3.60	2.43	0	3.63	10.82

[a] composite OA scores calculated via PCA (see Chapter 3)

Table 5.4. Sample distribution across age-related phases for each method

Method	Phase	N	Mean Age	SD	Median	Range
Pubic Symphysis Brooks and Suchey (1990)	I	10	20.5	2.12	20.5	17-24
	II	15	26.1	4.92	25	20-35
	III	26	37.2	12.01	34.5	23-73
	IV	77	49.3	12.85	48	24-78
	V	59	57.1	11.23	57	32-79
	VI	63	64.1	11.01	66	34-79
Auricular Surface Buckberry and Chamberlain (2002)	I	5	20.2	3.96	19	17-27
	II	31	32.1	10.03	29	20-57
	III	43	41.4	13.51	43	20-72
	IV	62	54.3	13.78	54	25-78
	V	58	55.7	14.12	57	24-79
	VI	36	62.9	10.43	65.5	40-79
	VII	17	64.3	10.73	66	39-78
Acetabulum Calce (2012)	I	75	31.5	9.94	29	17-72
	II	137	56.6	10.04	55	34-79
	III	40	67.9	9.95	70	39-78

Table 5.5. Bias and inaccuracy of age estimation by method

	Auricular Surface		Pubic Symphysis		Acetabulum	
Age	Bias[a]	Inaccuracy[b]	Bias	Inaccuracy	Bias	Inaccuracy
17-29	1.44	1.53	0.23	0.44	1.00	1.00
30-39	1.04	1.25	0.32	0.80	0.08	0.57
40-49	0.65	1.77	-0.90	1.30	0.59	1.51
50-59	0.33	1.53	-1.60	2.07	0.00	0.74
60-69	-0.60	1.01	-2.18	2.19	-0.85	1.93
70-79	-1.86	1.88	-2.90	2.90	-1.19	1.49
17-39[c]	2.47	2.78	0.54	1.24	1.08	1.57
40-59	0.98	3.30	-2.50	3.37	0.59	2.25
60-79	-2.46	2.89	-5.08	5.09	-2.09	3.42
Overall	0.97	8.98	-7.03	9.71	-0.42	7.24

[a] Directional error, over- and under-estimation of age in years.
[b] Absolute error, average absolute error of age estimation in years
[c] Collapsed into three age categories: young (17-39), middle (50-59), old (60-79)

Table 5.6. Descriptive statistics for the sample of the ages at transition by method

Method	Phase	Mean Age at Transition (years)	S.D.	S.E
Auricular Surface	I–II	18.1	4.12	1.93
	II–III	25.6	15.54	2.53
	III–IV	39.2	17.01	1.81
	IV–V	55.8	25.51	2.21
	V–VI	73.9	22.69	3.40
	VI–VII	101.2	29.45	14.15
Pubic Symphysis	I–II	20.6	3.02	0.85
	II–III	25.3	5.94	1.09
	III–IV	32.7	13.19	1.82
	IV–V	52.4	17.55	1.60
	V–VI	68.3	18.84	2.35
Acetabulum	I–II	41.2	8.23	1.14
	II–III	73.8	15.32	2.52

5.3.1 Auricular surface and osteoarthritis

When evaluating the auricular surface, bias and estimate error were significantly different between young, middle, and old adult age classes, as well as between OA severity groups ($P <$ 0.001), whereas inaccuracy (the amount of absolute error) did not differ between groups (Table 5.7). Estimation error was most variable for individuals with the highest OA scores (Table 5.8); though individuals with both low OA scores ($\bar{x}$ =3.73 ± 10.49 S.D.) and high OA scores ($\bar{x}$-=3.37 ± 12.31 S.D.) were aged incorrectly compared to those with midrange OA severity scores ($\bar{x}$ =1.76 ± 13.15 S.D.) using Buckberry and Chamberlain's (2002) method. When error levels were examined between age classes (young, middle, old), significant differences were found between all age group pairs (Table 5.9). When OA score was regressed on age estimate error within each of the three age groups (Table 5.10, Fig. 5.2), significant positive relationships were detected. For young and middle adults (≤59 years), 13% and 16% of the variation in age estimate error can be explained by severity

in OA score respectively, overestimating age as severity in arthritic expression increased. For the oldest age category (60–79 years), the relationship is not significant. Regression analyses of estimate error on OA suggest that severity of arthritic expression does not influence the accuracy of age at death estimates from the auricular surface for individuals ≥60 years in the sample.

The likelihood ratio test between the log-age model and the OA/log-age model shows significant differences between the transition ages for each phase ($\chi^2_{(2)} = 60.554$, $P < 0.001$). As severity in OA scoring increases, the transition age for each phase also increases. Table 5.11 shows the statistics from the log-age/OA cumulative probit regression applied to the sample for the Buckberry and Chamberlain method. Age at transition distributions are illustrated in Figure 5.5a–f. The three lines in each figure represent the predicted transition distributions for individuals in each OA group with mean OA values of 1.80, 3.60, and 5.41 calculated from the 1st, 2nd, and 3rd quartiles. Individuals with the highest OA scores transition between phases at an earlier age compared to those with less severe expressions of arthritic patterning, meaning that they exhibit an accelerated rate of skeletal aging at the auricular surface, appearing older than their chronological age. In contrast, individuals with low OA scores appear younger longer.

5.3.2 Pubic symphysis and osteoarthritis

Damage to individual specimens resulted in a slightly reduced sample size for the pubic symphysis (n=250). Bias, inaccuracy, and estimate error were significantly different ($P < 0.001$) between all groups considered using the Suchey-Brooks method (Table 5.7). Most of the variation in error of the age estimate was found between individuals with the lowest OA scores (Table 5.8). Age estimate error increased between low ($\bar{x}$ =-0.99 ± 7.23 S.D.), to midrange ($\bar{x}$ =-8.31 ± 12.04 S.D.), to high OA scores ($\bar{x}$ =-13.82 ± 10.91 S.D.), suggesting that persons with more severe arthritic expression were aged incorrectly using Brooks and Suchey's (1990) method. When estimate error was examined between age classes (young, middle, old), a significant difference was also found among them (Table 5.7), with all age groups displaying significant differences from each other (Table 5.9). However, the regression of estimate error on OA was only significant in young adults (17-39 years) (Table 5.10, Fig. 5.3) where age was overestimated as severity in arthritic expression increased. Approximately 16% of the variation in age estimate error is explained by OA for young adults (Fig. 5.3). For middle and old adults (40-79 years), the relationship is not significant.

Regression analyses of estimate error on OA suggest that severity of arthritic expression does not influence the accuracy of age at death estimates from the pubic symphysis for individuals ≥40 years in the sample.

The likelihood ratio test between the log-age model and the OA/log-age model shows significant differences between the transition ages for each phase ($\chi^2_{(2)} = 19.986$, $P < 0.001$); as severity in OA scoring increases, the transition age for each phase also increases. See Table 5.11 for the statistics from the log-age/OA cumulative probit regression applied to the sample for the Suchey-Brooks method. The age at transition distributions are illustrated in Figure 5.6a–e. As in the auricular surface, individuals with the highest OA scores have a lower age at transition between phases, demonstrating an accelerated rate of skeletal aging at the pubic symphysis.

Table 5.7. Results of one-way analysis of variance (ANOVA) for bias, inaccuracy, and age estimate error by method

		Auricular Surface			Pubic Symphysis			Acetabulum		
		F[f]	df	*P*-value	*F*	df	*P*-value	*F*	df	*P*-value
Between OA groups [a]	Bias [c]	3.93	2	**0.021**	18.98	2	**0.000**	8.32	2	**0.001**
	Inaccuracy [d]	2.34	2	0.098	22.52	2	**0.000**	2.83	2	0.065
	Estimate Error [e]	3.86	2	**0.022**	19.07	2	**0.000**	8.32	2	**0.001**
Between Age groups [b]	Bias	71.53	2	**0.000**	84.82	2	**0.000**	26.97	2	**0.000**
	Inaccuracy	1.88	2	0.157	36.15	2	**0.000**	13.76	2	**0.000**
	Estimate Error	70.75	2	**0.000**	86.39	2	**0.000**	26.97	2	**0.000**

Significance level $P < 0.05$ bolded.
[a] Three groups defined by +/-2.43 (1SD) of the mean (3.60): Low OA score (≤1.17, n=38), Midrange OA Score (1.18—6.02, n=169), High OA score (≥6.03, n=45). Except for the auricular surface, Welch's ANOVA was used.
[b] Three adult age groups defined by Young (17-39) n=67, Middle (40-59) n=100, Old (60-79) n=85; Welch's ANOVA used for all methods.
[c] Directional error
[d] Absolute error
[e] Estimated age – actual age
[f] Asymptotically F distributed

5.3.3 Acetabulum and osteoarthritis

When evaluating the acetabulum, bias, inaccuracy, and estimate error were significantly different ($P < 0.01$) between young, middle and old adult age classes, but between OA severity groups, inaccuracy (the amount of absolute error) did not differ (Table 5.7). Most of the variation in error of the age estimate was found between low to midrange OA scores (Table 5.8). Age estimate error was highest for individuals with low OA scores ($\bar{x}$ =3.27 ± 7.16 S.D.) compared to midrange ($\bar{x}$ =-1.97 ± 10.58 S.D.) and high scores ($\bar{x}$ =1.59 ± 13.29 S.D.), suggesting that persons with less severe arthritic expression were aged incorrectly using Calce's (2012) method. When error was examined between age classes (young, middle, old), a significant difference was also found among them (Table 5.7). However, it was the oldest age group that displayed significant differences in error from both the young and middle-aged cohorts (Table 5.9). From the results presented in Figure 5.4, the Calce method appears to underage old adults (60-79 years) with low OA scores, but the accuracy of the method to estimate age in old adults improves as OA severity increases (i.e., estimate error is reduced for old adults with severe OA). When OA score was regressed on age estimate error, there was a significant positive relationship in old adults (60-79 years), where 17.7% of the variation in estimate error is explained by OA (Table 5.10, Fig. 5.4). The relationship is not significant for younger individuals (17-59 years). Regression analyses of estimate error on OA suggest that severity of arthritic expression does not influence the accuracy of age at death estimates from the acetabular method for individuals less than 59 years in the sample.

The likelihood ratio test between the log-age model and the OA/log-age model shows significant differences between the transition ages for each phase ($\chi^2_{(2)} = 35.5363$, $P < 0.001$). As severity in OA scoring increases, the transition age for each phase also increases. Statistics from the log-age/OA cumulative probit regression applied to the sample for the Calce method are found in Table 5.11. The age at transition distributions are illustrated in Figure 5.7a–b. Consistent with results from other joint regions where an accelerated rate of skeletal aging was observed, individuals with the highest OA scores have a lower age at transition between phases at the acetabulum.

Table 5.8. Results of post-hoc tests for differences between OA groups by method

	Auricular Surface			Pubic Symphysis			Acetabulum		
	Tukey's HSD Post-hoc *P*-value [d]			Games-Howell Post-hoc *P*-value			Games-Howell Post-hoc *P*-value		
Dependent Variable	Low - Midrange	Midrange - High	Low - High	Low - Midrange	Midrange - High	Low - High	Low - Midrange	Midrange - High	Low - High
Bias [a]	0.689	**0.038**	**0.031**	**0.000**	0.071	**0.000**	**0.000**	0.228	0.622
Inaccuracy [b]	0.080	0.837	0.381	**0.000**	0.147	**0.000**	0.210	0.429	0.78
Estimate Error [c]	0.659	**0.043**	**0.031**	**0.000**	0.058	**0.000**	**0.000**	0.228	0.622

Significance level $P < 0.05$ bolded.
[a] Directional error
[b] Absolute error
[c] Estimated age – actual age
[d] Asymptotically F distributed

Table 5.9. Results of Games-Howell post-hoc tests for differences between age groups by method

	Auricular Surface			Pubic Symphysis			Acetabulum		
	Post-hoc *P*-value [d]			Post-hoc *P*-value			Post-hoc *P*-value		
Dependent Variable	Young-Middle	Middle-Old	Young-Old	Young-Middle	Middle-Old	Young-Old	Young-Middle	Middle-Old	Young-Old
Bias [a]	**0.000**	**0.000**	**0.000**	**0.000**	**0.000**	**0.000**	0.058	**0.000**	**0.000**
Inaccuracy [b]	0.133	0.284	0.896	**0.000**	**0.000**	**0.000**	0.959	**0.000**	**0.000**
Estimate Error [c]	**0.000**	**0.000**	**0.000**	**0.000**	**0.000**	**0.000**	0.058	**0.000**	**0.000**

Significance level $P < 0.05$ bolded.
[a] Directional error
[b] Absolute error
[c] Estimated age – actual age
[d] Asymptotically F distributed

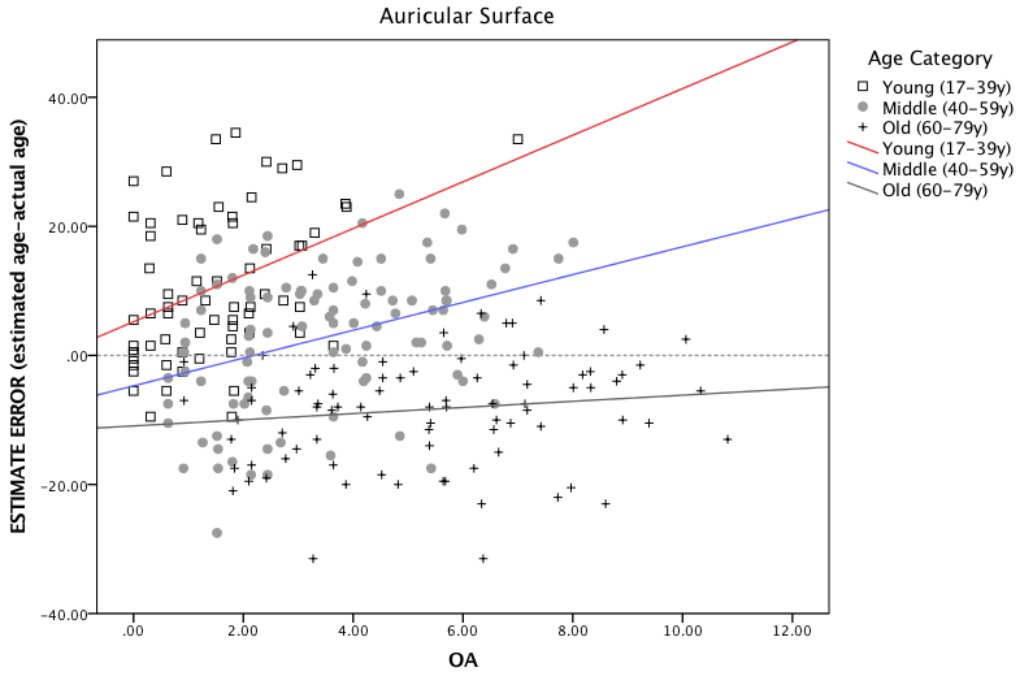

Figure 5.2. Regression plot showing the relationship between estimate error and OA for the auricular surface by age group (young, middle, old).

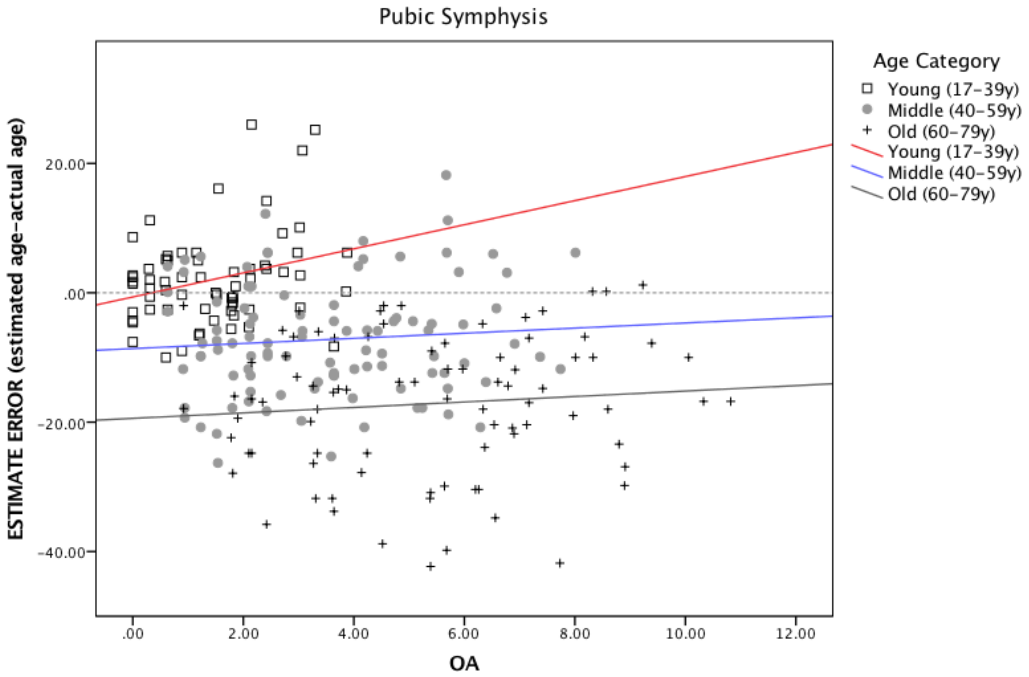

Figure 5.3. Regression plot showing the relationship between estimate error and OA for the pubic symphysis by age group (young, middle, old).

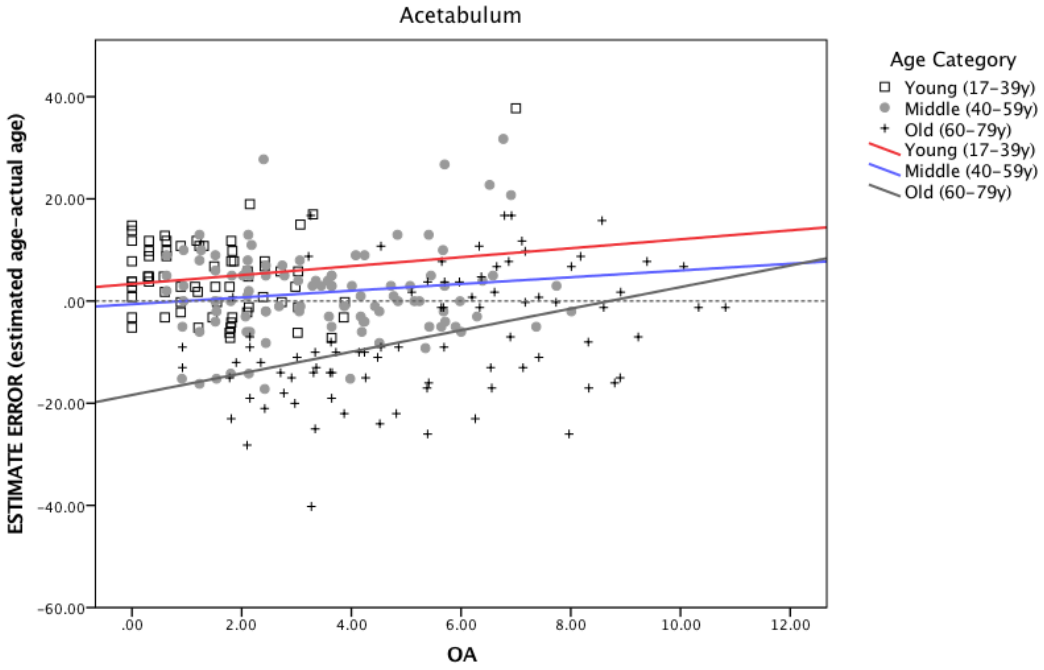

Figure 5.4. Regression plot showing the relationship between estimate error and OA for the acetabulum by age group (young, middle, old).

Table 5.10. Results of linear regression of OA on age estimate error by method

Model: Regression Estimate Error [a] and OA [b]

		Young (17-39)	Middle (40-59)	Old (60-79)	Overall
Auricular Surface	N	67	100	85	252
	R	0.406	0.365	0.13	-0.179
	R^2	0.164	0.133	0.017	0.032
	SEE	10.49	10.28	8.76	12.61
	P-value	**0.001**	**0.000**	0.234	**0.004**
Pubic Symphysis	N	66	99	85	250
	R	0.398	0.083	0.094	0.302
	R^2	0.159	0.007	0.009	0.091
	SEE	7.01	8.89	10.77	11.4
	P-value	**0.001**	0.412	0.39	**0.000**
Acetabulum	N	67	100	85	252
	R	0.146	0.14	0.421	-0.056
	R^2	0.021	0.019	0.177	0.003
	SEE	7.63	8.76	11.01	10.88
	P-value	0.238	0.166	**0.000**	0.374

R, Pearson's correlation coefficient; R^2, proportion of variance explained; SEE, standard error of the estimate; Significance level $P < 0.05$ bolded.

[a] Dependent variable: Estimate Error (estimated age – actual age)

[b] Predictors: (constant),

Table 5.11. Log-age/OA statistics for each method using the cumulative probit model

Method	Model	Phase	Intercept
Auricular Surface	Model: Log age and OA Log age coefficient = –2.285 OA coefficient = 0.347	I–II	6.394
		II–III	8.154
		III–IV	9.166
		IV–V	10.131
		V–VI	11.043
		VI–VII	11.935
Pubic Symphysis	Model: Log age and OA Log age coefficient = –3.790 OA coefficient = 0.353	I–II	11.610
		II–III	12.733
		III–IV	13.831
		IV–V	15.353
		V–VI	16.248
Acetabulum	Model: Log age and OA Log age coefficient = –5.201 OA coefficient = 0.267	I–II	19.170
		II–III	22.503

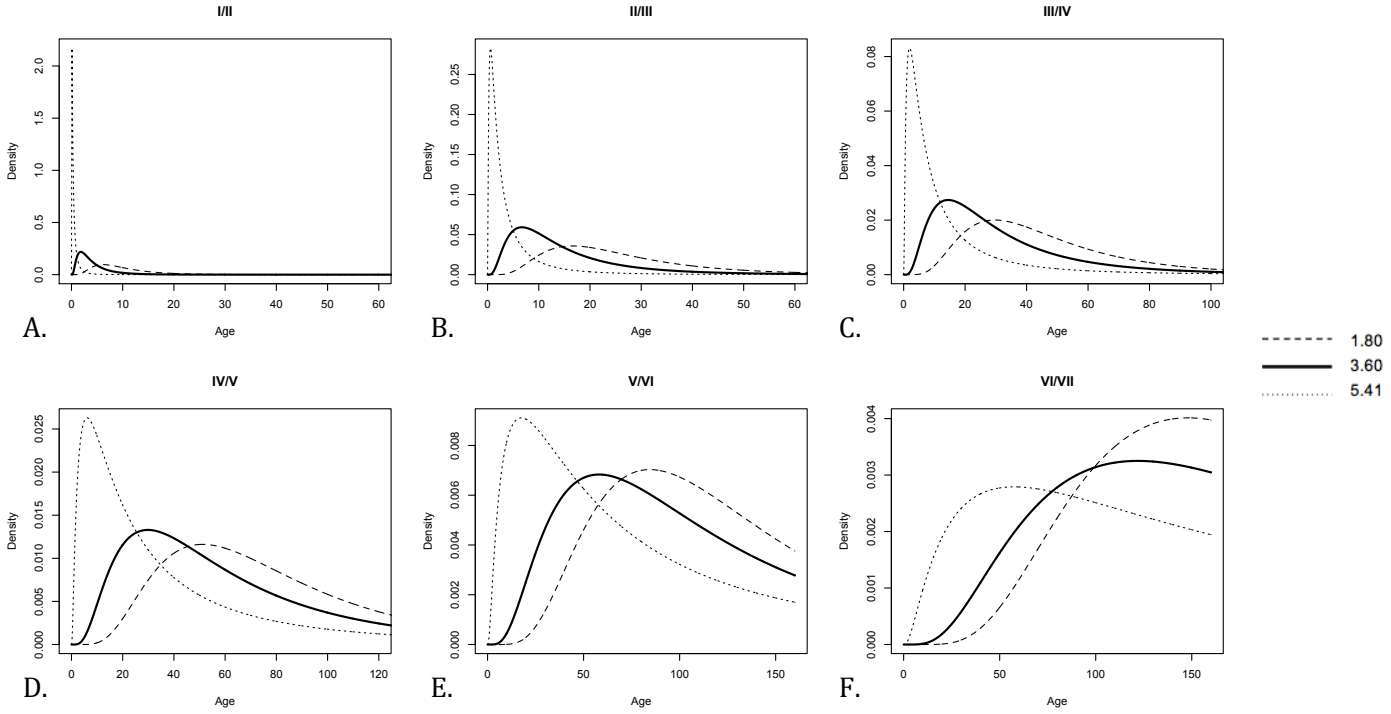

Figure 5.5. Ages-at-transition for the OA/log-age model of the auricular surface using Buckberry & Chamberlain (2002) method.

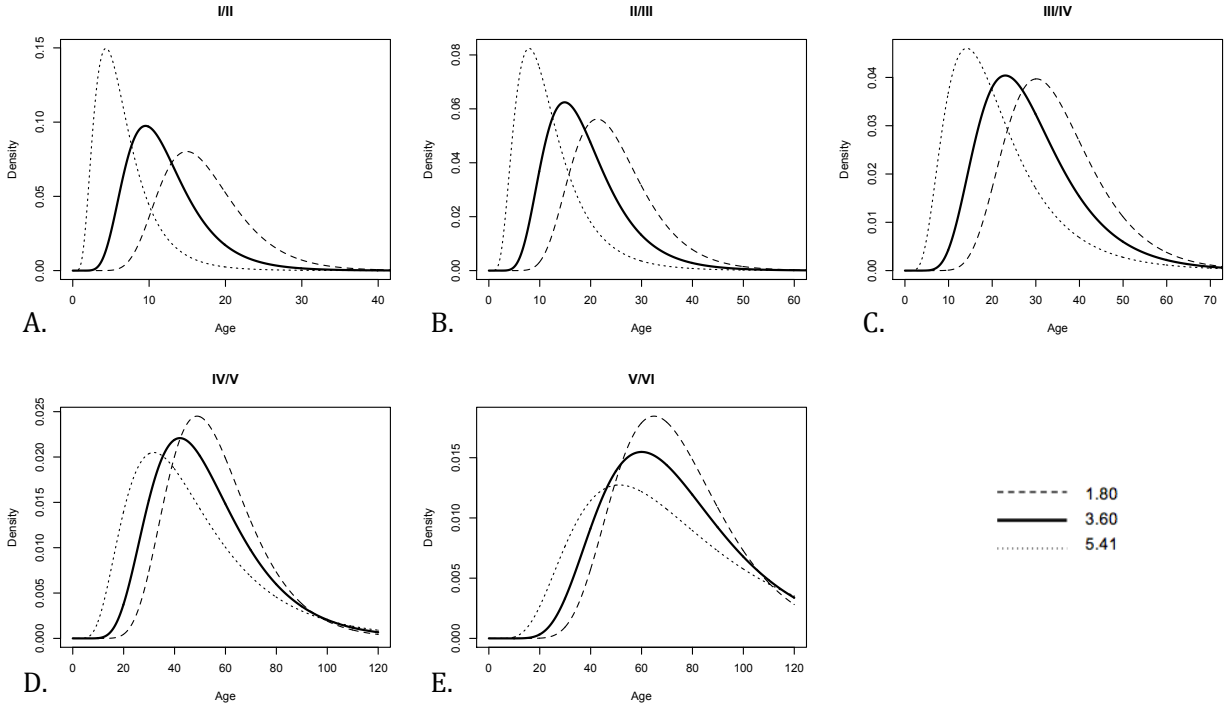

Figure 5.6. Ages-at-transition for the OA/log-age model of the pubic symphysis using Brooks and Suchey (1990) method.

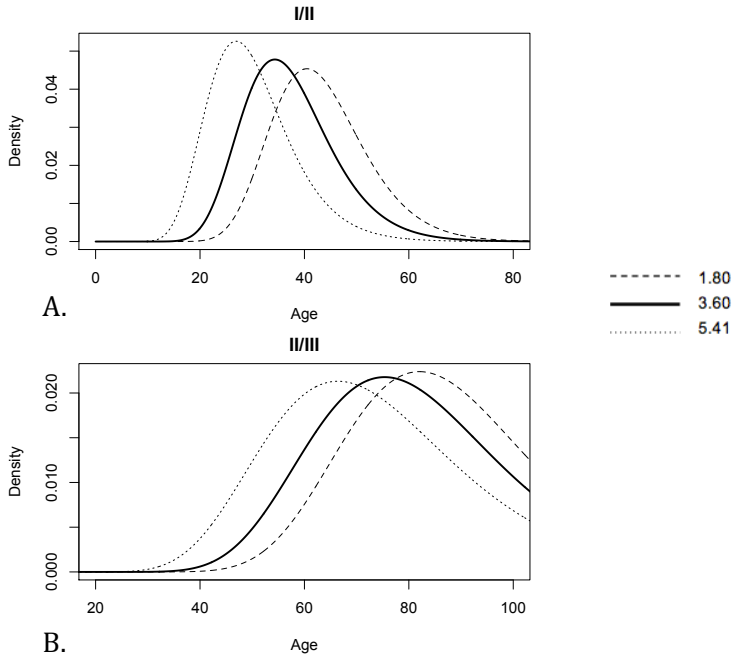

Figure 5.7. Ages-at-transition for the OA/log-age model of the acetabulum using Calce (2012) method.

5.4 Discussion

Three significant results emerge from these data. First, OA severity has an effect on the accuracy of age estimates from joints of the os coxa. Second, this influence is most significant for different age cohorts in each of the joint regions, demonstrating that varied rates of arthritic trait progression occur on the auricular surface, pubic symphyses, and acetabulum. Third, those with OA appear to be aging faster, a consistent trend in all three joint regions.

Based on the results of this study, methods to estimate age from degenerative arthritic traits may be suitable to employ on the joints of the os coxa for persons in senior age categories. This is consistent with other findings that degenerative qualities persist well past the sixth decade in these regions (Mulhern and Jones, 2005; Falys et al., 2006; Hartnett, 2010). At the acetabulum, testing of age-related traits that rely heavily on texture degeneration and osteophytic development to distinguish individuals with a high probability of being over 60 years old have been conducted with promising results (Rougé-Maillart et al., 2004; 2009; Rissech et al., 2006; Calce, 2012), and this study has shown that old individuals with little evidence of arthritic degenerative patterning are under-aged at this region. In this study, accuracy of the age estimate improves for individuals 60-79 years as OA severity increases using Calce's method, and therefore may play an important role in identifying the elderly.

Differential rates of age progression of skeletal traits were observed between methods, the earliest of traits developed at the auricular surface (lowest mean age at transition from phase I-II), and the pubic symphysis aging the fastest overall (earliest age transitions between phases II-VI) (Table 5.6). When OA was considered, individuals appeared to progress through the stages of each method more rapidly (Figs. 5.5-5.7). Those with OA appeared older than their chronological age at each joint location. The potential to overage individuals using Buckberry and Chamberlain's method is consistent with results by Wescott and Drew (2015), who also showed an accelerated rate of aging at the auricular surface when controlled for body mass. They did not find a significant difference in the reliability of the Suchey-Brooks method between normal and obese groups, whereas in this sample, OA did affect the accuracy of age estimates from the pubic symphysis in young persons (≤39 years), over-aging them as well.

Body mass has shown to be a factor in age estimation for multiple methods (Merritt, 2015; Wescott and Drew, 2015) and risk of developing OA is associated with obesity (D'Lima et al., 2012). Though none of the individuals in our sample met the conditions for obesity classification (>80kg),

results of our previous research (see Chapter 4) demonstrated a significant (but weak) partial correlation of body mass (kg) to OA severity in the pelvis (PR^2=0.202, P=0.003). As such, partial correlations controlling for body mass for each of the three os coxa methods were carried out for this sample to remove the effect of body size from joint arthritic severity and its potential influence on age estimate error. These results are presented in the Appendix Tables A11-A13 and indicate that body mass had no influence in the relationship between estimate error and OA in any of the pelvic joint locations. In addition to these analyses, a cumulative probit model was run to approximate the relationship between body mass and age, without considering the factor of OA (see Appendix Figures A10-A12). For the acetabular method, body mass affected the timing of transition between age phases II-III. The heaviest individuals transitioned earlier, aging faster than the lighter individuals. Large body mass is a confounding factor of OA and may be contributing to the increased estimate error found in the old individuals (60-79 years) of this study when using Calce's method. This study demonstrated that body mass had no effect on the timing of transitions between age phases of the pubic symphyseal or auricular surface methods, which differs from Merritt (2015) and Wescott and Drew's (2015) results. However, in both of these studies the independent variable, body mass index (BMI, kg/m^2), was derived from samples with recorded weight at death, whereas in this study body mass was estimated from postcranial skeletal measurements (Appendix Table A14), calculations which are known to be associated with significant error (Elliot et al., 2015).

As other studies (Weiss, 2006; Plomp et al., 2013) have also reported a correlation between OA and chronological age, we might expect degenerative qualities to progress linearly, with the oldest persons with advanced OA expression being aged accurately using any one of the os coxa methods applied at a single joint surface. But joint changes of OA are a function of both the natural aging process and of the mechanical stress response to loading. How can we tease apart these two very different processes to determine whether arthritic characteristics can be used as appropriate age indicators? The difference between how OA is measured by this study versus the information provided by arthritic patterning of a single specific joint is that age-related (and potentially genetic-related) variation is captured by a systemic measurement of pelvic OA and is significant for two reasons. First, the systemic pelvic OA measurement takes into account that severe expressions of the condition occur more consistently across multiple joints for individuals in the sample. Severe OA found only at one specific joint, and not in others, may be a biomechanical effect. Second, in determining a final skeletal age estimate, we use a combination of multiple aging methods from multiple joint locations. For this reason it is important to consider the distribution of pelvic arthritic

lesions to provide more accurate determinations of age, rather than focusing on one specific joint system with a potential to bias results by over-or underestimating age at death.

The os coxa joints are distinctly biomechanically stressed areas of the skeleton with potential to invoke a large amount of variation in the development or progression of degenerative changes. The femoroacetabular joint is unique in the fact that it is never fully unloaded during daily activities. Although the duration of maximum loads experienced by the articular surfaces may be short, a residual compressive force acts across the joint at all times, with an average magnitude approximately equal to body weight (Bowman et al., 2010). Biomechanically, movement at both the sacroiliac and pubic symphyseal joints are induced by motions occurring at other locations in the body; however, both joint areas are subject to a variety of internal and external forces that can lead to OA. The fact that the auricular surface bears more weight than the pubic symphysis (Vleeming et al., 2012) may explain the earliest changes of sacroiliac joint morphology. Variation in arthritic joint lesions of the os coxa may simply be functional adaptations to loads imposed on the plastic skeleton throughout life. The specific role that OA plays in the aging of musculoskeletal structures is difficult to examine because its aetiology is multifactorial; while risk of developing OA is largely dependent on environmental factors that may accelerate and accentuate these processes, there is also evidence to suggest that OA may be hereditary and therefore genetically influenced as well (Spector and MacGregor, 2004; Gestsdóttir, 2014).

The pelvis itself is a complex anatomic structure composed of osseous, ligamentous, and muscular assemblages responsible for transferring the weight of the body from the axial skeleton into the lower extremities; which must be accomplished while allowing for dynamic loading during activities such as gait and balance (Pauwels, 1976). Many factors contribute to the forces encountered in the os coxa, including daily and athletic activities, the contribution of weight and obesity, and the limitations of motion. Studies that consider the effects of weight on joint degeneration (Merritt, 2015; Wescott and Drew, 2015) make progress towards understanding how body size influences the rate of skeletal aging in the pelvis, and possibly also the relationship between body size and OA in joints of the lower limb. This may be especially important for the area of the femoroacetabular joint where an association has been found between being overweight and increased peak hip moments that may independently increase the risk of injury, dysfunction, and intra-articular pathology (McMillan et al., 2009). It may not be possible to separate the singular effect of age from other mechanical factors that also produce changes in pelvic bone morphology.

Perhaps OA is not a disease, but rather the endpoint of normal degeneration (Forbes, 1997; Kirkwood, 1997). After all, to consider using features such as osteophytic bone spurs, marginal lipping, and porosity of subchondral bone in methods of skeletal age estimation presupposes this fact, that is, we have accepted arthritic degeneration as a universal related to the aging process, rather than a true disease that affects only a subset of the population. Osteoarthritis is not an inevitable consequence of growing old (Felson et al. 2000; Anderson and Loeser, 2010), though age is arguably the greatest risk factor. Sticking to the formal pelvic aging methods as described here, this study has shown that systemic OA severity has an effect on the accuracy of age estimates from joints of the os coxa, particularly in young adults. Yet, evaluating the quality of bone and distribution of joint OA in various other skeletal locations (e.g., the upper limb) has proven to be successful in generating more accurate age estimates when used in addition to pelvic methods (Garvin and Passalacqua, 2012; Milner and Boldsen, 2012; Sharman, 2014). The exact mechanisms of OA and aging are not clearly understood, which underscores the importance of research to determine how morphological features (e.g., porosity, osteophytes, eburnation) are combined in formal methods to accurately estimate age at death from the os coxa, since (1) joint degeneration affects multiple skeletal sites at varied rates, and (2) pathological bone responses vary dependent on joint function, anatomical structure and loading (Rogers et al., 2004; Dieppe, 2011).

Research that uses OA to allow for accurate age assessment beyond the fifth decade (Brennaman et al., 2015; Falys and Prangle, 2015) incorporates age estimates for a large portion of the adult age cohort not typically represented by other methods. Additional research that specifically targets degenerative pelvic joint changes in the very old (70+ years) will help to further determine the usefulness of OA in age estimation using current techniques. In this study, the oldest cohort of individuals (80-99 years, n=39) was excluded to avoid large artificial margins of error, certain to bias results in the direction of the younger age distributions from which the aging methods were created. Standards to describe aging criteria that distinguish individuals 70+, 80+, and 90+ years is necessary because globally, many more people are entering the mortality sample at these ages as a result of declining fertility, improved health, and increased life expectancy (Kinsella and Velkoff, 2001; Ice, 2003). The ability to accurately model age in the elderly will be significant to narrow the scope of forensic investigations, as well as to inform wider bioarchaeological studies of demography that aim to understand cultural attitudes towards social age categories.

5.5 Conclusions

In this sample, OA severity affected the accuracy of age estimates from each of the methods by Buckberry and Chamberlain (2002), Brooks and Suchey (1990), and Calce (2012), with individuals appearing older than their true chronological age as expected. This result will have a significant impact on our ability to accurately model age from the skeleton, and is especially relevant given that OA prevalence is increasing rapidly in young adults as a result of lifestyle characteristics, e.g., diet, disease, physical activity, and body mass. Systemic measures of OA, as an indicator of age, may be very useful to identify those in the most senior age category using joint areas of the pubic symphysis, sacroiliac, and femoroacetabulum, and in particular, to distinguish between joint failure from abnormal mechanical loading. The acetabulum demonstrated the slowest progression of age-related traits with higher ages-at-transition between phases. The oldest persons with little arthritic patterning at the femoroacetabular joint were underaged using Calce's (2012) method, but accuracy of the age estimate improved for persons 60 years and older as OA severity increased.

Methods to estimate age at death using the pelvis are widely popular. The continued use of such markers supports the necessity of evaluating joint surfaces in relation to OA (unaffected by trauma), to understand bone health and how OA directly affects the rate of skeletal aging. In this study, transition analysis demonstrated differential rates between methods of age progression of skeletal traits accelerated by OA, highlighting the importance of determining a final age estimate from varied skeletal locations on the body. Other areas not biomechanically loaded in the same way as the pelvis (e.g., the upper limb), have shown promise in effectively estimating age at death from skeletal arthritic degeneration (Falys and Prangle, 2015), which suggests that the biological processes that cause OA share a common final pathway with other age-associated diseases, rather than OA occurring as a time-dependent disorder distinct from normal aging with separate causative mechanisms at work. Until this becomes clear, osteologists must recognize the limitations of our methods to accurately describe aging from discreet skeletal arthritic traits.

Chapter 6.
General discussion and conclusions

The study of OA in a paleopathological context presents several limitations that must be considered before the described results are accepted. The first relates to the fact that OA is ill-defined. Basic to the quantification of OA is deciding how much detail to collect (e.g., number of elements, number of articular surfaces), documenting the nature of skeletal expression (i.e., number of variables) and the distribution of lesions (i.e., location, amount of surface area affected). Researchers use a mixed number of these criteria to establish the presence and severity of OA from an ordered progression of degeneration, arguing from effect-to-cause, rather than the other way around. A hierarchy of weighting severity is problematic because OA is neither purely degenerative, nor linearly progressive (Felson and Nevitt, 2004; Dieppe, 2011; Waldron, 2012). Yet, all of these decisions are made by the paleopathologist a-priori without an accepted operational definition of OA. And as we have seen, dissimilarities in the number of scored variables/joints/surfaces have resulted in widely different estimates of OA prevalence and severity leading to difficulties in relative comparison between analyses (Bridges, 1993; Jurmain and Kilgore, 1995; Waldron, 2012).

One of the major stumbling blocks in paleopathological research of OA has been in the definition of OA *severity*. Defining OA severity from skeletal lesions (in the absence of soft tissue) is complicated, particularly when causative factors are unknown. In the living population, we are able to qualitatively assess severity by symptomatic markers such as pain and limitations in mobility. But clinical research has consistently proven that pain and florid joint changes are unrelated (Waldron, 2012). A small bone spur can lead to debilitating pain and immobility, while the most degenerative joint morphology may not be painful at all. So how can we remedy this in the bioarchaeological record where pain and mobility are not preserved? We cannot go back in time and ask people how they felt when they moved around and we certainly cannot infer specific behaviours from pathological lesions that share a common pathway with other stress loading effects. Can we really determine OA severity in past populations?

In spite of these data collection and analyses roadblocks, it is not being suggested that we abandon the study of OA. Rather, the studies presented in this thesis have systematically argued for the use of principal component analysis (PCA) as a method to accurately synthesize very large and complicated inter-related OA datasets to reveal discrete patterns of pathological expression from

which a representative and systemic measurement of OA severity is composed. Since components are empirically determined aggregates of the variables (e.g., osteophytosis, porosity, eburnation), a 'severity' measurement from PCA is valuable to describe composite OA pathology, and is reflective of the variation in joint-specific wear patterns.

Several systems for coding OA pathology have been proposed (Jurmain, 1990; Waldron and Rogers, 1991; Buikstra and Ubelaker, 1994; Jurmain and Kilgore, 1995; Larsen et al., 1995) and no universal method has been accepted. However, it was not the purpose of this study to determine which method would be favourable, only to demonstrate that the OA scoring proposed by Buikstra and Ubelaker (1994) could be effectively summarized through PCA. In fact, PCA could also be employed for either of the methods proposed by Jurmain (1990) or Waldron and Rogers (1991).

The challenges in OA research listed here are not insurmountable and PCA as a standardized procedure in computing population-specific distributions of OA severity overcomes them. PCA is an exploratory and descriptive multivariable tool useful for summarizing variation in OA by grouping variables that are statistically associated (Jolliffe, 2002), and for revealing both joint-specific and population-specific wear patterns in the dataset that would otherwise be unnoticeable using the traditional summary technique (e.g., averaging raw scores for joint complexes that are biased by the total number of surfaces). The standardization of data recording is obviously critical and as a discipline, paleopathology must operationalize a coherent definition of OA. But standardization in analyzing data is also fundamentally important and serves larger scale analyses. Being explicit in the use of published coding protocols and data manipulation will alleviate problems of non-comparability, making meta-analyses possible.

6.1 Body size and activity with osteoarthritis

Despite the clear association between joint loading and OA (Dieppe, 2011), neither body size nor activity variables affected the expression of OA in this sample. However, a few limitations of the study variables must be discussed before we can accept that body size and activity are not relatively important in determining arthritic patterning. First, individuals in this sample are estimated to be of relatively normal body mass (~40–75kg), whereas body size correlates with OA are usually linked to an obese phenotype (Hochberg et al., 1995; Sanford et al., 2014). While males were taller and heavier than females, within-group body size variability may be too low to detect differences in this sample. Further study of how body size affects joint arthritic severity may be

possible using other documented skeletal collections that contain a large proportion of individuals with a living body mass of greater than 80kg (Merritt, 2015; Wescott and Drew, 2015).

Second, in the absence of joint injury there is no strong evidence to suggest that vigorous low-impact exercise is associated with an accelerated rate of OA development (Hunter and Eckstein, 2009). This fact may serve to explain the negative correlation between torsional strength of the femur with OA in our sample; that long-term, repetitive physical work capacity is in some way protective against the development of OA, as reported by longitudinal studies (Foley et al., 2007). While exercise is known to have advantageous trophic effects on periarticular bone, muscle, and tendon (Magnusson et al., 2007), in the absence of soft tissue, paleopathologists are limited in their interpretation of how low-level cumulative mechanical loading really affects the whole joint organ. However, other studies, such as those by Shaw and Stock (2009a; 2009b) that consider how elite sports participation and intensity of loading affect diaphyseal strength and shape, will be very important for understanding occupational risk of OA development, particularly since exercise has, and will continue to play, an important role in both the pathogenesis and management of OA in the living population (Roddy et al., 2005). Future controlled studies such as these will inform behavioural interpretation of the past and are necessary to understand the role of physical activity and features of osseous change.

There are additional reasons to explain the low association between activity and OA in this sample. For example, modern urban populations are unlikely to exhibit marked differences in post cranial skeletal robusticity as compared to terrestrial or marine mobility hunter-gatherer groups with more specific behavioural characteristics, and/or repetitive loading histories. Next, documented skeletal collections as a source of behavioural information present their own limitations and/or biases. Unknown factors such as age at which individuals began their occupations, changes in profession, and additional activities (e.g., sport) influence skeletal robusticity and the development of OA (Cardoso and Henderson, 2010; Shaw and Stock, 2009a). The point at which an activity is initiated is especially important to studying behaviourally-based osseous changes (Shaw and Stock, 2009b). Bone's considerable plasticity during the subadult growth period is crucial to formation and remodelling of cortical bone in response to mechanical loading throughout an individual's life, and is perhaps the only period wherein patterns of activity can be truly isolated (Pearson and Lieberman, 2004). Finally, the in-vivo strain environment that drives both degenerative joint changes and bone functional adaptation is complex and variable, and bioarchaeologists are only privy to observations suspended at the time of death. Research to

address the complex nature of activity from skeletal morphology in past populations should incorporate subadult tissue. Data from the upper limb may also reflect a broader spectrum of habitual behaviours.

6.2 The effects of osteoarthritis on age estimation

The systemic measure of OA in each of the lumbar spine, pelvic, and knee regions takes into account that expressions of the condition occur more consistently across multiple joints. This fact has been especially useful to examine differences in age-related OA variation for this sample, and is relevant because we evaluate multiple skeletal surfaces in determining a final age estimate. As expected, error in age estimates from pelvic joints was more significant for young individuals (≤39 years) with OA, who appeared older than their chronological ages. As well, individuals with more advanced arthritic expression progressed more rapidly through the phases of the aging methods, demonstrating an accelerated rate of skeletal aging compared to those with low OA scores. These results have a potentially significant consequence on how we understand the rate of bone remodelling as it is related to disease, aging and the evaluation of age indicators from macroscopic age estimation methods. The relationship between factors that affect skeletal aging, such as bone remodelling rates and bone mineral density in persons with OA are discussed.

6.2.1 Bone remodelling, bone mineral density, and osteoarthritis

Bone remodelling is the process of bone renewal to maintain strength and mineral homeostasis that begins before birth and continues until death (Clarke, 2008). It is based on the concerted action of resorptive and formative cell populations that replace old bone with new, a relationship that changes with age to produce variations in bone morphology (Eriksen, 2010). As age increases, the rate of bone removal usually outpaces the rate of bone deposition as a consequence of hormonal factors and a decrease in osteoblastic activity (Szulc and Seeman, 2009; cf. Merritt, 2015). These changes in bone remodelling rates also affect bone mineral density (BMD), which peaks near age 30, and decreases predictably thereafter (Boskey and Coleman, 2010). The effects of bone remodelling and the decline in BMD at the microscopic level translate to observable macroscopic age changes on the bone surface, from which most methods of adult age estimation are

derived (Rogers, 2009b). By the onset of the third decade, changes in surface texture are evident: striae become dense and granular, billowing smoothes and microporosity forms. With increasing age into the fifth, sixth and seventh decades, surface morphology generally becomes more degenerative: dense granularity becomes course, smooth surfaces become roughened and concave, macroporosity begins to replace microporosity, osteophytic activity occurs, and bone becomes more fragile. But how do we explain reasons for this advanced aging morphology when it is observed in young individuals? And how is BMD affected when OA is present?

As demonstrated by this study, changes in joint surface morphology are also accelerated by disease—individuals with more advanced OA displayed increased rates of bone remodelling over those with lower scores (i.e., transitioning between age phases at earlier ages). While this trend was observed across all age classes of the sample, it was most significant in young individuals who were consistently over-aged when OA was present. Yet, how this affects BMD is still not clear. Clinical research has shown that persons with high systemic BMD are at an increased risk of developing OA (Nevitt et al., 2010), and OA has been presented largely as a proliferative hypertrophic bone disease in this context (Burr and Gallant, 2012). Only a few studies have attempted to identify this concept of *bone formers* in a skeletal series. Studies by Rogers et al. (1997; 2004) derived simple indices of bone formation from the strong association between osteophytes and enthesophytes in 9th–16th century English populations, noting that skeletal response to stress is dependent on common cellular mechanisms under genetic control that affect joints differentially. Their results are generally echoed by Crubézy et al. (2002) who also noted that genetic factors conferring susceptibility to OA were already present in Central European populations of the early Neolithic period (5700 BC). Schmitt and colleagues (2007) examined bone turnover rates in the hands of a modern Portuguese sample to find a strong correlation between age and osteophytic development; but failed to confirm an association between OA and high bone mass density that is typically cited in clinical research trials (Hochberg et al., 2004). In a similar study, Weiss (2013) found that the cortical index of the second metacarpal was a poor indicator of bone loss in a sample of prehistoric American Indians, but a study by Mays (2015) indicates a positive relationship between bone mass and ligamentous ossification in 18-19th century Londoners.

Studies by Mays (2015), Weiss (2013) and Schmitt et al. (2007), have all begun to make progress towards understanding complicated metabolic bone processes in skeletal samples, but more research is needed. Together, these results prompt a critical evaluation of identifying 'bone formers' and 'bone losers' in a skeletal population to determine whether persons who form bone at

a uniquely rapid rate have differential risks of developing the range of bone-forming arthritic disorders, such as osteophytosis, OA, or advanced spondyloarthropathies (e.g., DISH). Population-based assessments to identify a high bone density phenotype, and whether increased BMD is a cause or consequence of OA forms the next likely arm of this research, helping us to better interpret the usefulness of arthritic characteristics as age indicators, and to estimate age at death more accurately. The relationship between the rate of bone remodelling, BMD, age, and age indicators is particularly important for the young adult population, where OA prevalence is increasing rapidly as a result of lifestyle characteristics, e.g., diet, disease, physical activity, and body mass. Future research that quantifies variable bone formation to tease apart genuine age effects, a systemic predisposition of skeletal remodelling, and the impact of local biomechanical factors will allow us to recognize diverse aetiologies of OA in both the context of normal bone metabolism and pathological osseous change, particularly in relation to concepts of disability and aging within archaeological populations with significant implications for paleodemography, paleoanthropology, bioarchaeology, and forensic anthropology.

6.3 Conclusions

This research had three primary goals: (1) to accurately and meaningfully quantify OA expression in load-bearing skeletal joint analyses; (2) to determine how factors of age, activity, and body size contribute to variation in skeletal OA pathology; and (3) to calculate the amount of error in age at death estimates when severe OA is present in three pelvic joint areas.

Since arthritic joint changes occur as a function of both the natural aging process and the mechanical stress response to loading, it was expected that increasing chronological age, higher general activity levels, and larger body size would have a significant effect on OA severity in three load-bearing joint regions of the lumbar spine, pelvis, and knee. Body size and activity factors did not contribute significantly to OA pathology outside of the age-related expression in either of the lumbar vertebrae or knee regions, and only demonstrated a weak association at the os coxa joints. Increased activity levels demonstrated a negative correlation with OA severity, supporting the notion that continued exercise and habitual loading is in some way protective from the development of OA. The in-vivo strain environment that drives both degenerative joint changes and bone functional adaptation is both complex and variable. Joints may differ in what strains they respond to adaptively (e.g., hip versus lumbar and knee), and activity levels and variation in activity

might simply be too low to detect in this relatively sedentary sample, as opposed to other terrestrial or marine mobility foraging populations.

Exploratory data analysis of PCA is crucial to understanding the behaviour of a dataset, as a means of recognizing new patterns and relationships among variables that serve as a basis for diagnosing skeletal abnormalities such as OA, and must be distinct from significance testing (Ortner, 2012; Stodder, 2012). This thesis has shown that it is possible to determine age-related variation from a systemic measure of OA that combines trait distribution (e.g., osteophytes, porosity, eburnation) from multiple joint locations using PCA.

OA was highly correlated with age in the sample, and as expected, error in age estimates was most significant for young individuals when pelvic OA was present, making them appear older than their chronological ages. Overall, persons with evidence of severe joint arthritic expression progressed through the age phases of each pelvic method more rapidly, appearing to age faster. As appropriate to the research questions, accelerated rates of skeletal aging as a result of OA must be considered in determining a final age estimate from skeletal joint morphology, particularly in young adults for whom OA prevalence is increasing rapidly from rises in obesity and diabetes (Schett et al., 2013). Further analyses involving large samples of known individuals are needed to resolve the issues identified in these studies. Future work on the relationship between bone mass, age indicators and age may lie in the attempt to identify individuals or populations lying at different points on a bone-former/bone-loser spectrum. In the meantime, osteologists must recognize the limitations of our methods to accurately describe aging from discreet skeletal arthritic traits at pelvic joints; though refinements of age at death may be possible from OA occurring elsewhere in the skeleton (Milner and Boldsen, 2012; Sharman, 2014). Finally, interpreting OA as evidence for old age, measures of habitual activity, and larger body mass should be exercised with caution in skeletal populations.

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Appendix 1

Supplementary Figures

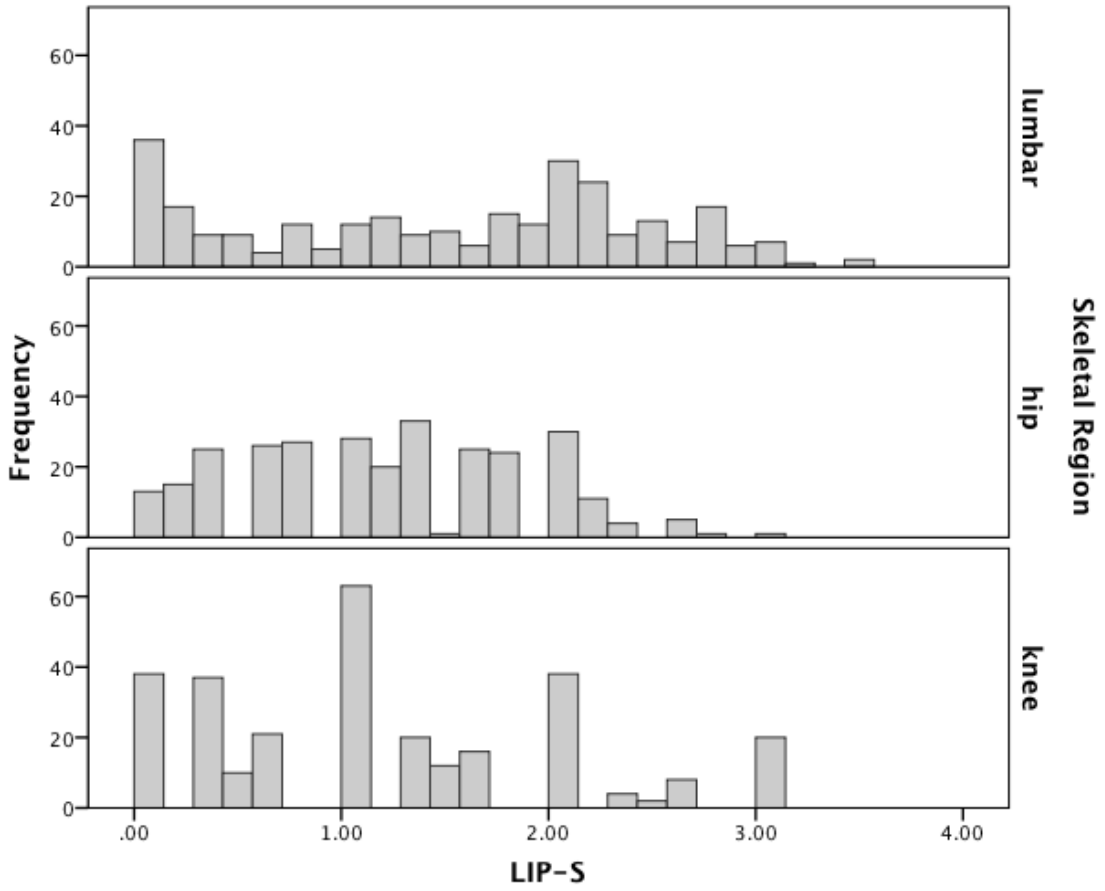

Figure A1. Distribution of ranked scores for lipping severity (LIP-S) compared between lumbar, pelvis, and knee regions.

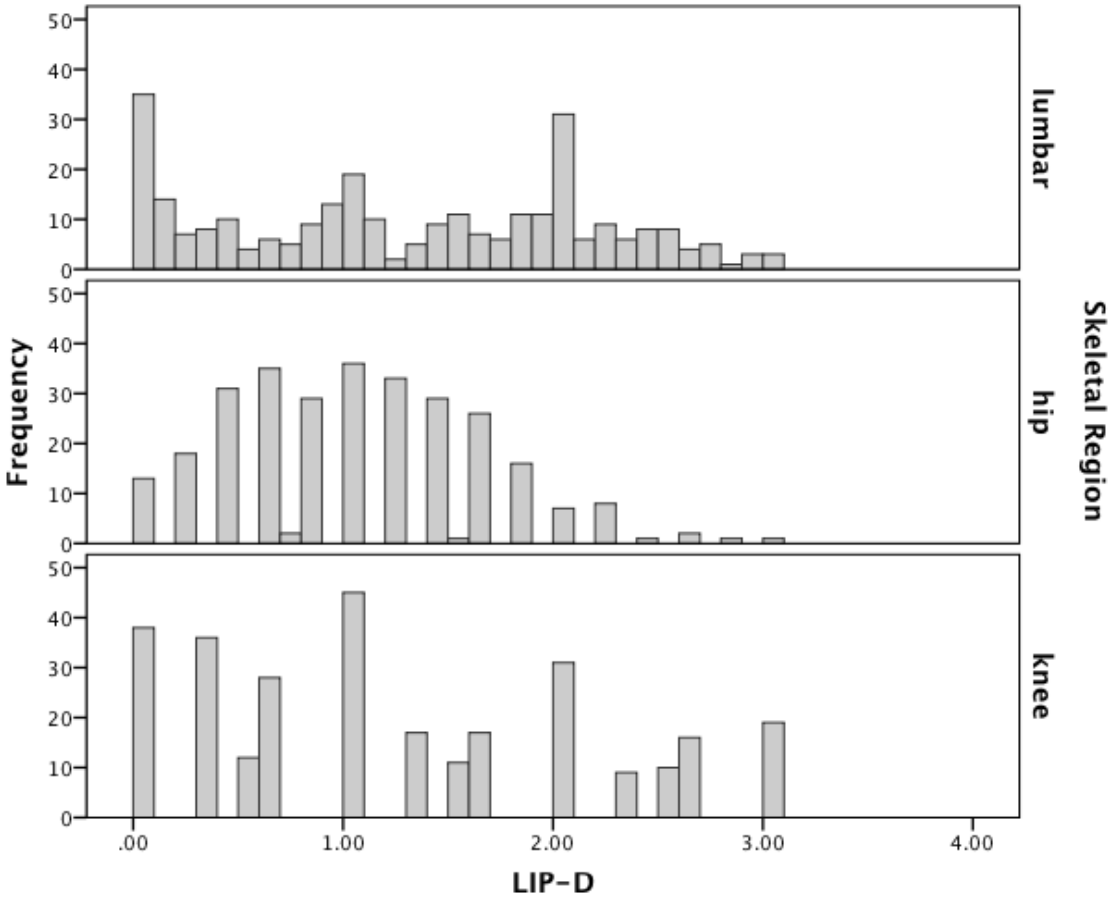

Figure A2. Distribution of ranked scores for the amount of area affected by lipping (LIP-D) compared between lumbar, pelvis, and knee regions.

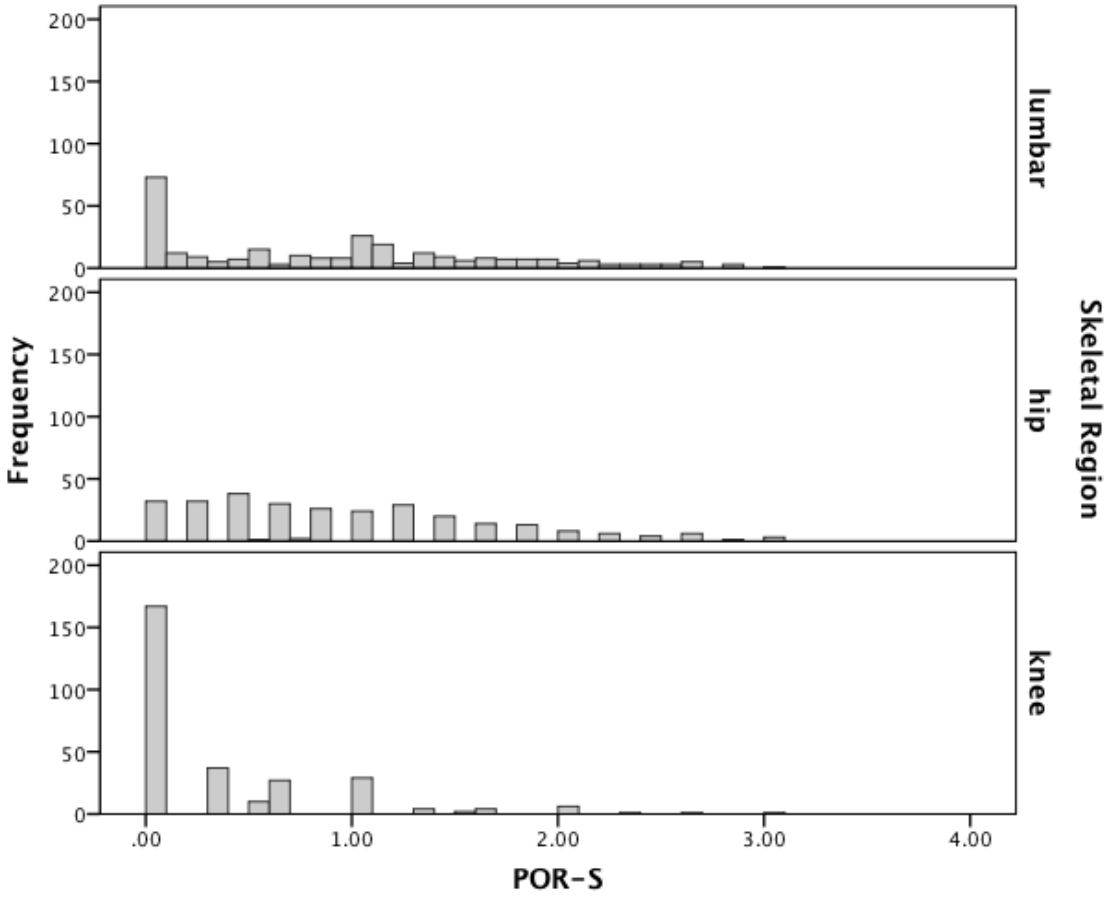

Figure A3. Distribution of ranked scores for porosity severity (POR-S) compared between lumbar, pelvis, and knee regions.

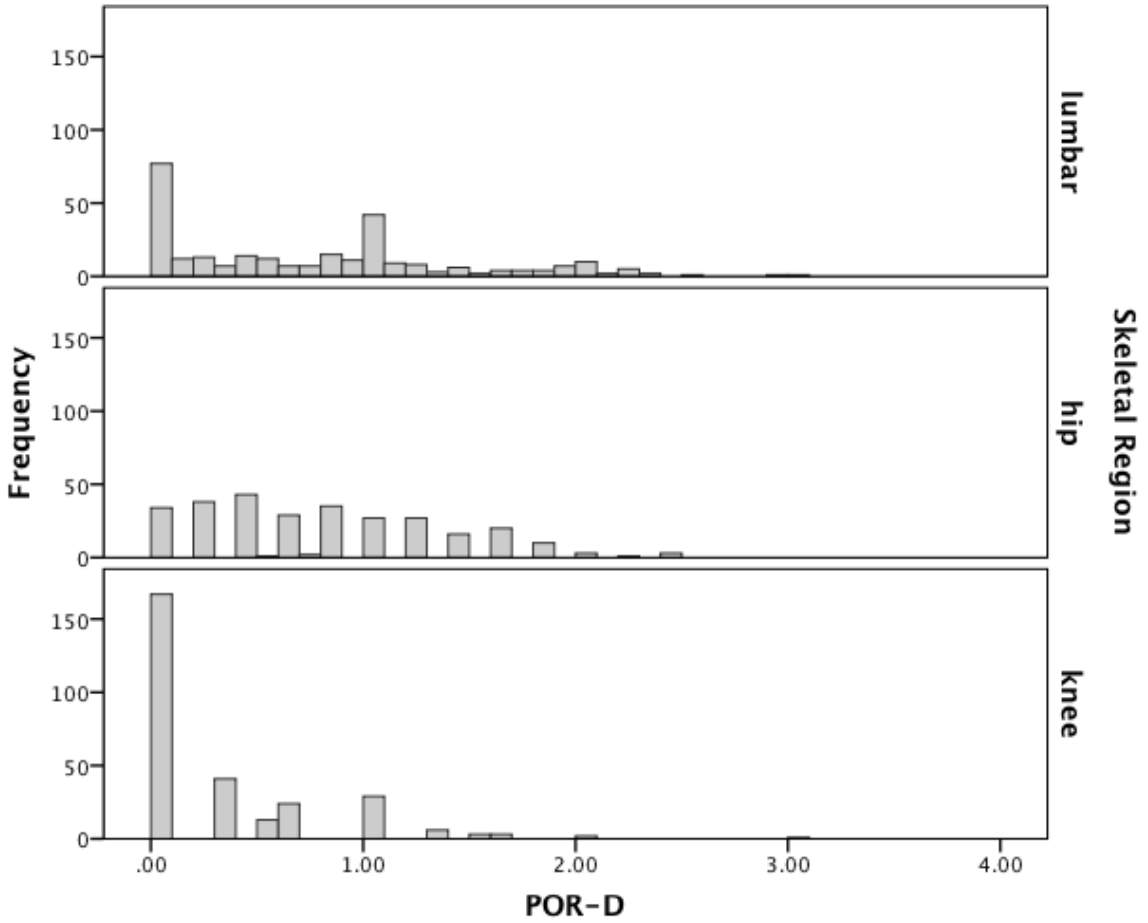

Figure A4. Distribution of ranked scores for amount of area affected by porosity (POR-D) compared between lumbar, pelvis, and knee regions.

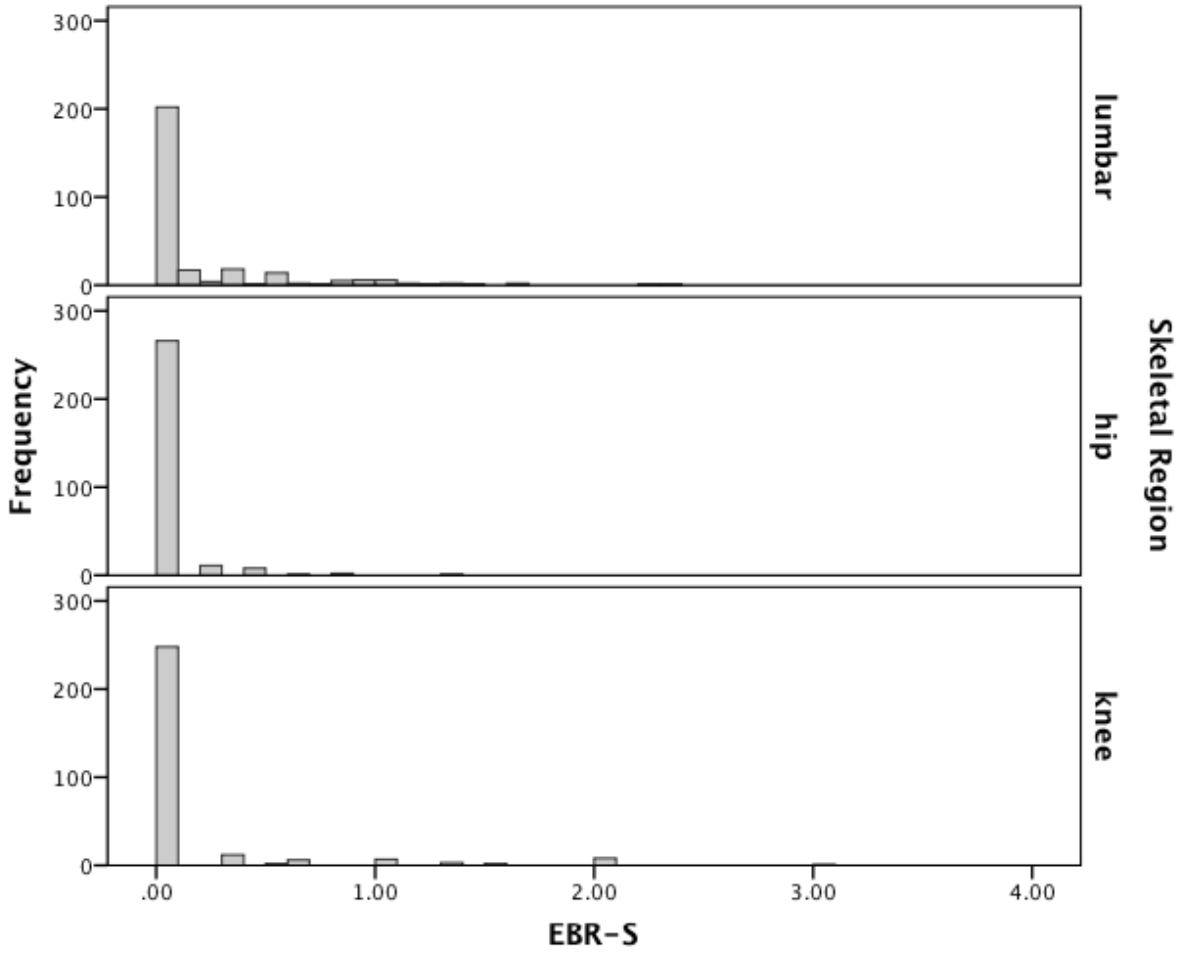

Figure A5. Distribution of ranked scores for eburnation severity (EBR-S) compared between lumbar, pelvis, and knee regions.

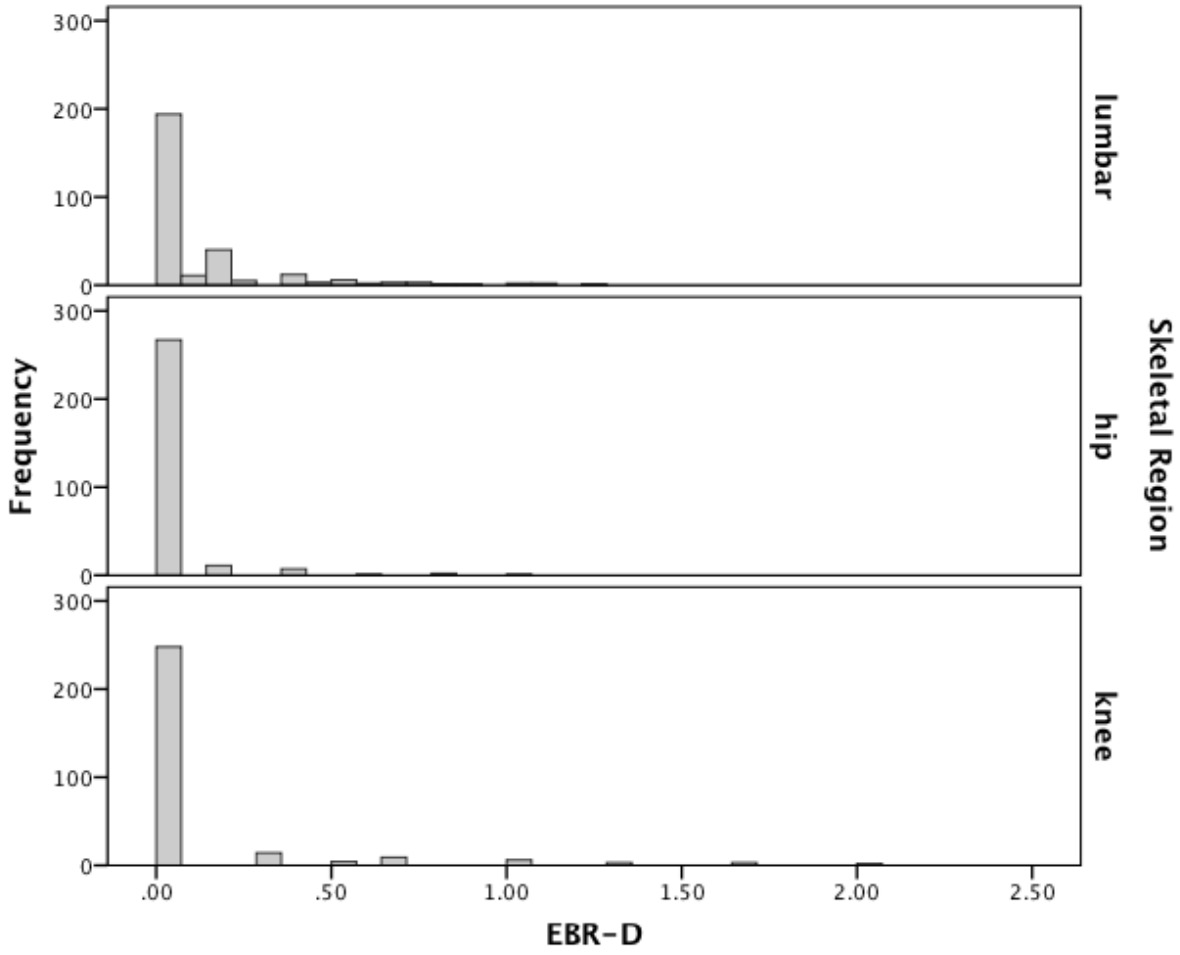

Figure A6. Distribution of ranked scores for amount of area affected by eburnation (EBR-D) compared between lumbar, pelvis, and knee regions.

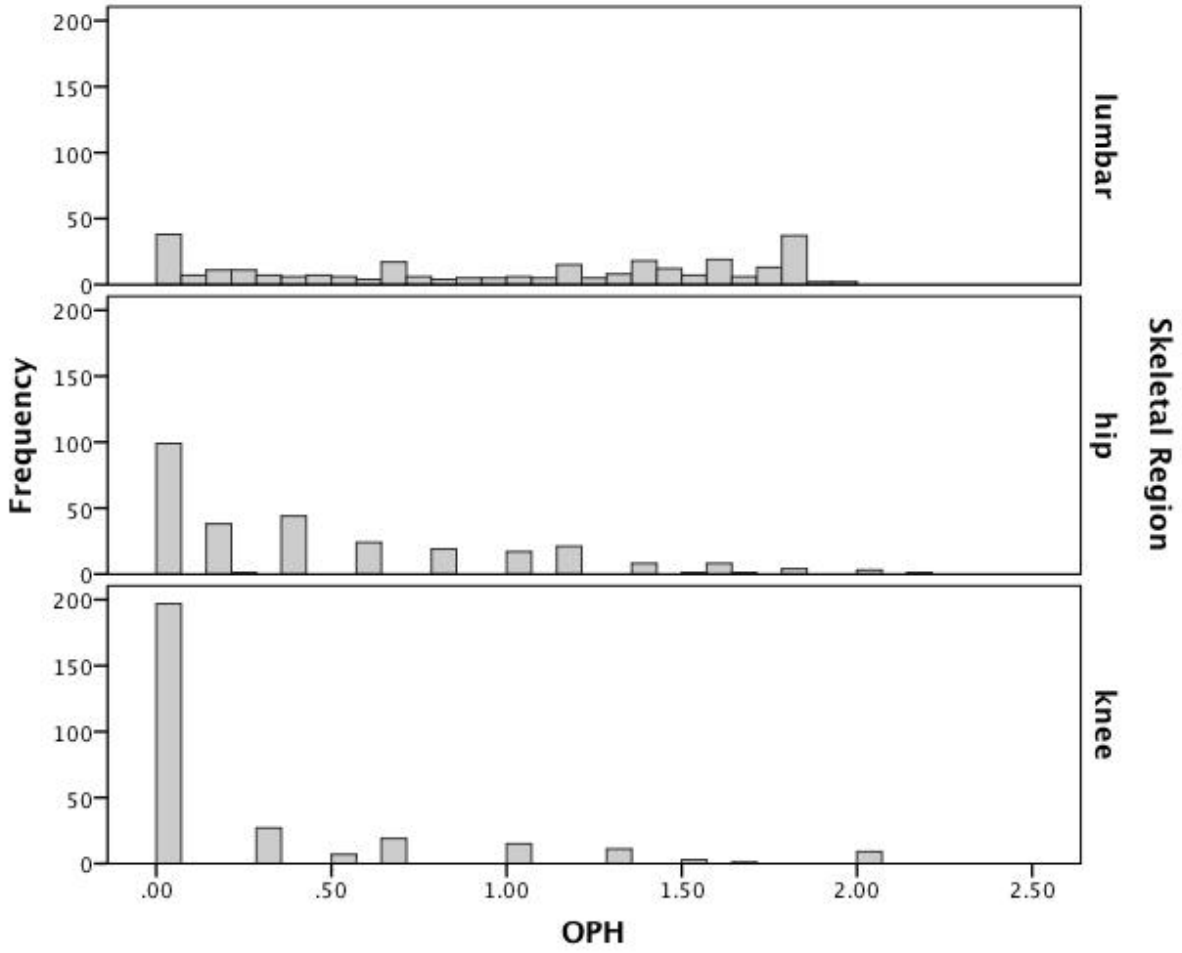

Figure A7. Distribution of ranked scores for osteophytes (OPH) compared between lumbar, pelvis, and knee regions.

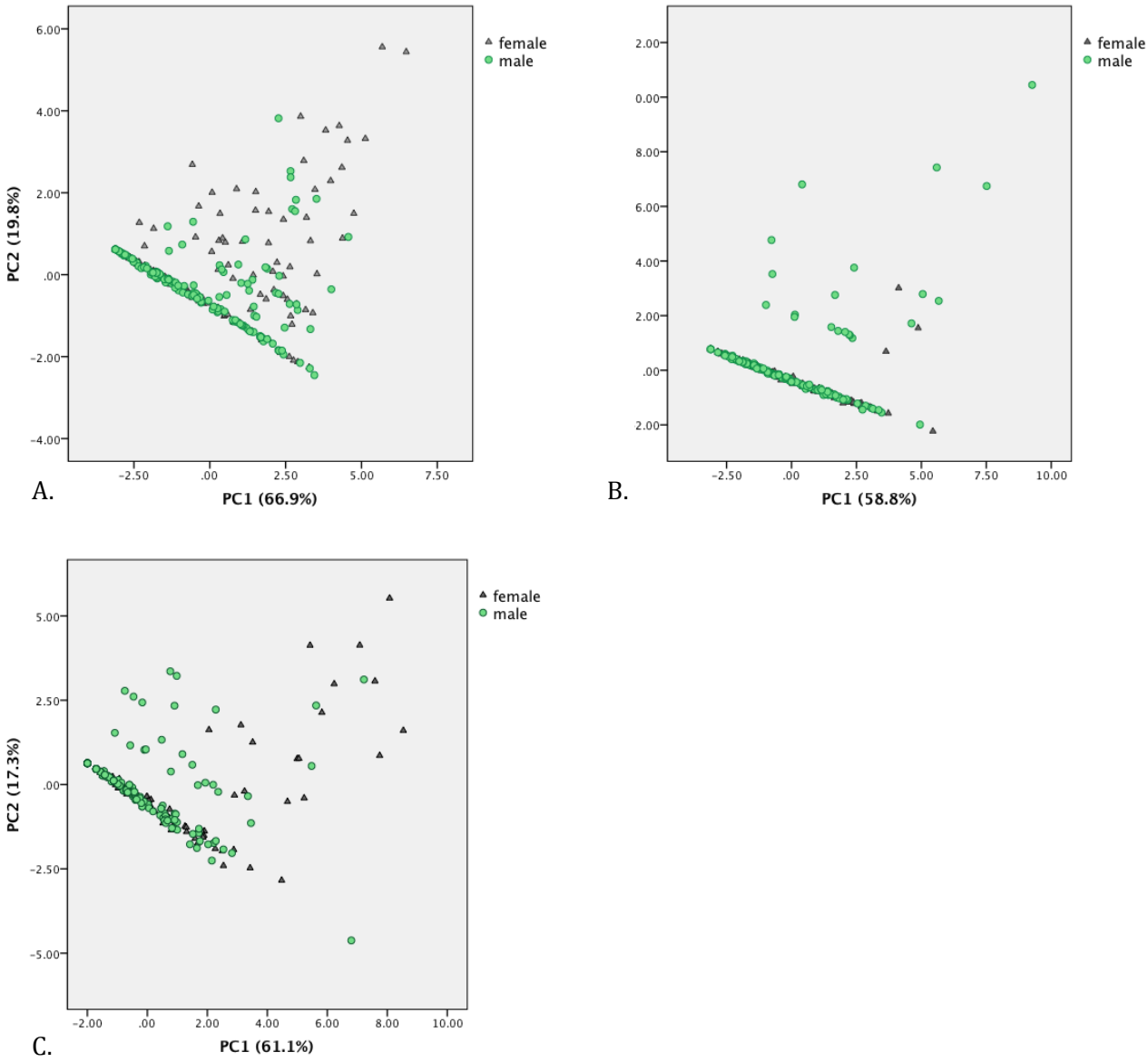

Figure A8. Individual component scores for principal components; PC1 versus PC2 of seven OA variables from the lumbar (A), pelvis (B), and knee (C) regions, plotted by sex.

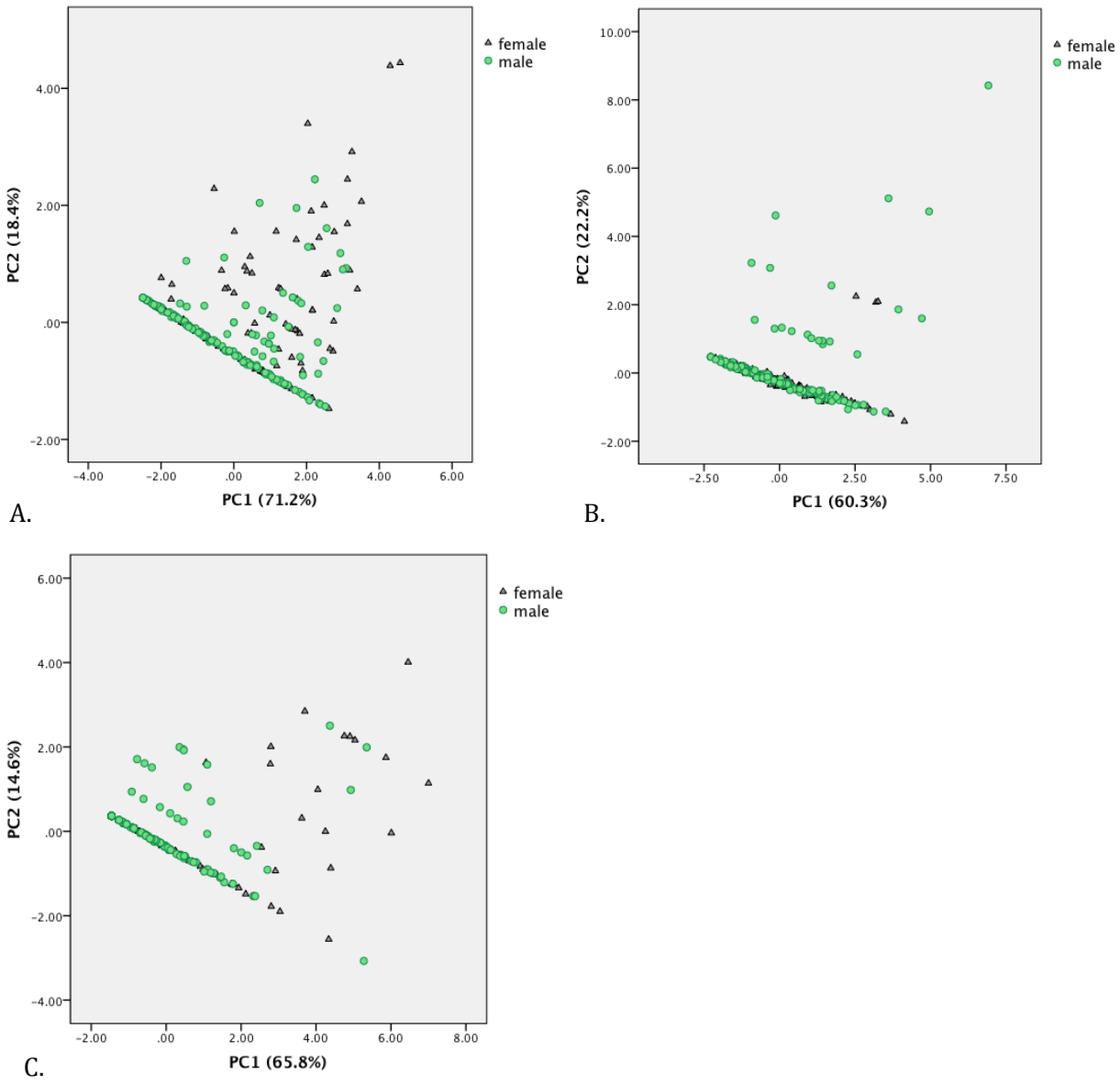

Figure A9. Individual component scores for PC1 versus PC2 of the severity variables set (LIP-S, POR-S, EBR-S, and OPH) from the lumbar (A), pelvis (B), and knee (C) regions, plotted by sex.

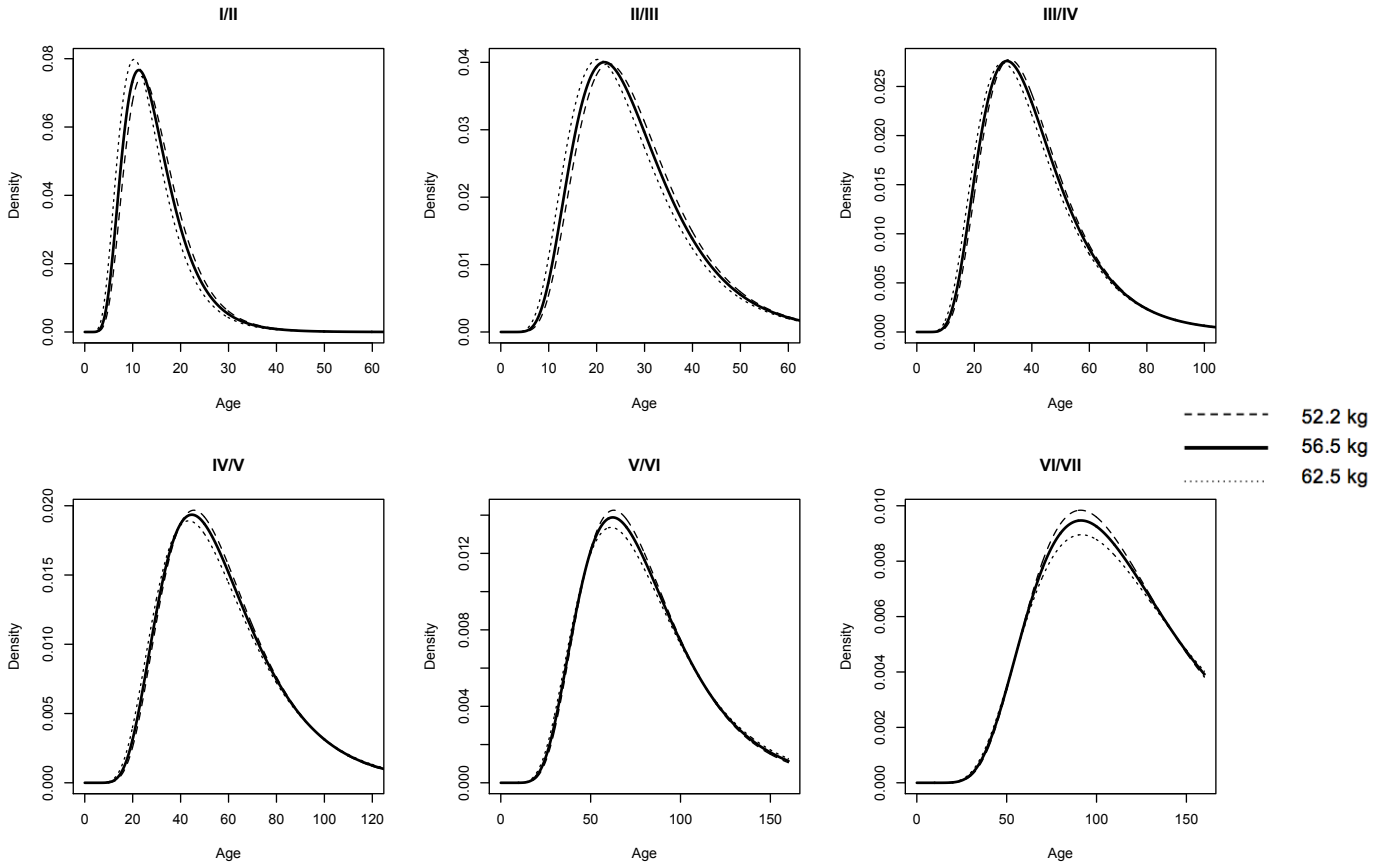

Figure A10. Ages-at-transition of the body mass/log age model using Buckberry and Chamberlain's (2002) method of the auricular surface. The three lines in each figure represent the predicted transition distributions for individuals in each BM group with mean OA values of 52.2kg, 56.5kg, and 62.5kg calculated from the 1st, 2nd, and 3rd quartiles.

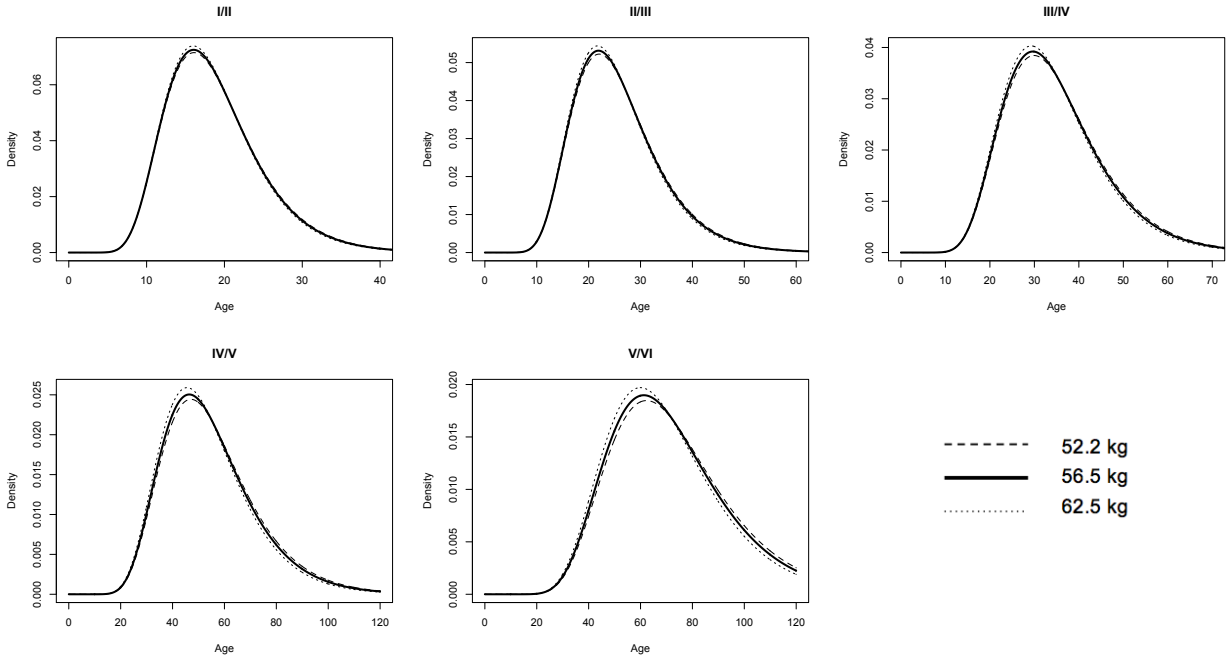

Figure A11. Ages-at-transition of the body mass/log age model using Brooks and Suchey's (1990) method of the pubic symphysis.

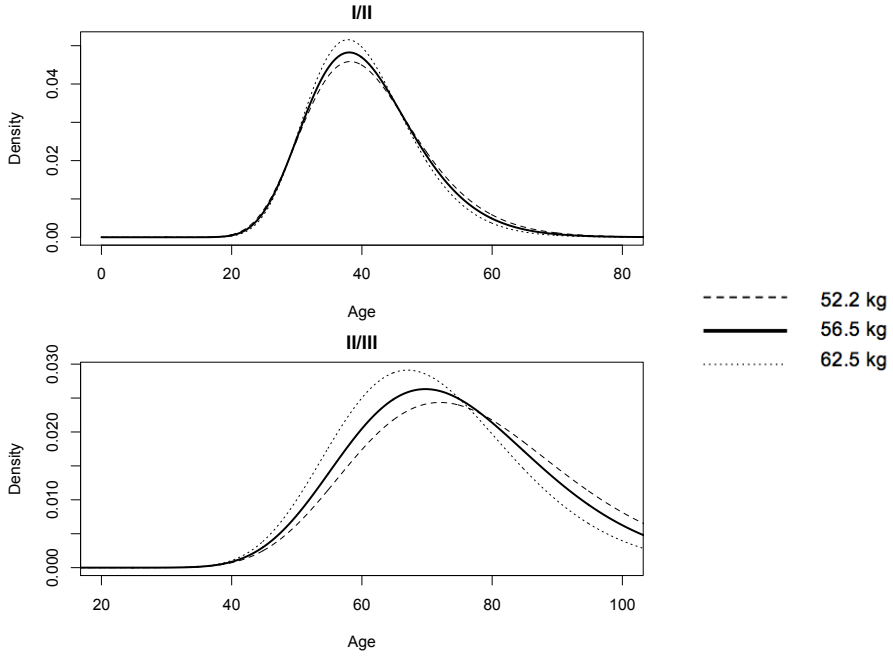

Figure A12. Ages-at-transition of the body mass/log age model using Calce's (2012) method of the acetabulum.

Appendix 2

Supplementary Tables

Table A1. Shapiro-Wilk's test of normality (*P* > 0.05) for age at death in the sample

		Shapiro-Wilk		
	Collection	Statistic	df	Sig.
Age at Death	Sassari	.978	101	.082
	Athens	.975	94	.072
	Lisbon	.976	94	.081

Table A2. Results of one-way analysis of variance (ANOVA) for age at death between Lisbon, Sassari, and Athens samples of individuals

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	1106.357	2	553.179	1.468	.232
Within Groups	107753.643	286	376.761		
Total	108860.000	288			

Table A3. Shapiro-Wilk's test of normality (*P* > 0.05) for OA scores, by region and by sex

		Shapiro-Wilk		
	Documented Sex	Statistic	df	Sig.
OA Score (Lumbar)	male	.948	142	.000
	female	.931	144	.000
OA Score (Hip)	male	.959	142	.000
	female	.964	144	.001
OA Score (Knee)	male	.880	142	.000
	female	.832	144	.000

Table A4. Results of Mann-Whitney U tests for OA scores between males and females by skeletal region

Test Statistics[a]	OA Score (Lumbar)	OA Score (Pelvis)	OA Score (Knee)
Mann-Whitney U	10102.500	9938.000	10063.000
Wilcoxon W	20542.500	20523.000	20648.000
Z	-.174	-.707	-.532
Asymp. Sig. (2-tailed)	.862	.480	.595

a. Grouping Variable: Documented Sex

Table A5. Mean percent errors from skeletal measurements

				Mean Percent Error			
Collection	Collection ID	Sex	Side	FH	FXL	TIB	BIB
Sassari	34	M	R*	0.15	0.00	0.30	0.41
Sassari	36	M	L	0.04	0.24	0.30	0.76
Sassari	75	F	L	0.71	0.26	0.00	1.05
Sassari	87	F	L	0.15	0.00	0.31	1.96
Sassari	117	M	L	0.63	0.24	0.29	1.59
Sassari	134	M	L	1.19	0.44	0.00	0.70
Sassari	183	F	L	0.21	0.28	0.33	1.21
Sassari	208	F	L	0.09	0.00	0.31	0.40
Sassari	214	F	L	0.28	0.23	0.29	0.00
Sassari	308	M	R*	1.90	0.47	1.28	1.97
Lisbon	475	F	L	1.04	0.00	0.00	0.35
Lisbon	334	F	L	0.43	0.00	0.00	0.38
Lisbon	203	F	L	0.38	0.27	0.00	0.87
Lisbon	61	F	R*	0.76	0.46	0.00	2.00
Lisbon	439	M	L	0.23	0.00	0.00	0.41
Lisbon	302	M	L	0.24	0.97	0.27	0.42
Lisbon	198	M	L	0.56	0.23	0.29	2.01
Lisbon	176	M	L	0.40	0.23	0.28	0.40

Table A6. Multiple linear regression coefficients for the lumbar region

Coefficients[a]	Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95.0% Confidence Interval for B		Correlations			Collinearity Statistics	
Model	B	Std. Error	Beta			Lower Bound	Upper Bound	Zero-order	Partial	Part	Tolerance	VIF
1 (Constant)	-.579	1.739		-.333	.740	-4.022	2.864					
AGE AT DEATH	.031	.002	.787	13.716	.000	.026	.035	.801	.784	.747	.902	1.109
J_LOG10	.385	.476	.045	.808	.421	-.558	1.328	.171	.074	.044	.974	1.027
BM (KG)	.010	.009	.094	1.055	.294	-.009	.028	.123	.097	.057	.377	2.655
STATURE (CM)	-.001	.010	-.013	-.147	.883	-.021	.018	-.049	-.014	-.008	.371	2.698

a. Dependent Variable: OA LUMBAR (SQRT+1)

Table A7. Multiple linear regression coefficients for the pelvic region

Coefficients[a]	Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95.0% Confidence Interval for B		Correlations			Collinearity Statistics	
Model	B	Std. Error	Beta			Lower Bound	Upper Bound	Zero-order	Partial	Part	Tolerance	VIF
1 (Constant)	4.008	1.567		2.557	.012	.905	7.111					
AGE AT DEATH	.019	.002	.633	9.176	.000	.015	.023	.647	.644	.601	.902	1.109
J_LOG10	-.914	.429	-.141	-2.128	.035	-1.764	-.064	-.033	-.191	-.139	.974	1.027
BM (KG)	.026	.008	.328	3.079	.003	.009	.043	.240	.272	.202	.377	2.655
STATURE (CM)	-.013	.009	-.155	-1.440	.152	-.030	.005	.019	-.131	-.094	.371	2.698

a. Dependent Variable: OA PELVIS (SQRT+1)

Table A8. Multiple linear regression coefficients for the knee region

Coefficients[a]		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95.0% Confidence Interval for B		Correlations			Collinearity Statistics	
Model		B	Std. Error	Beta			Lower Bound	Upper Bound	Zero-order	Partial	Part	Tolerance	VIF
1	(Constant)	-1.527	.858		-1.780	.078	-3.225	.172					
	AGE AT DEATH	.007	.001	.534	6.627	.000	.005	.010	.531	.519	.507	.902	1.109
	J_LOG10	.392	.235	.129	1.666	.098	-.074	.857	.209	.151	.127	.974	1.027
	BM (KG)	-.004	.005	-.103	-.830	.408	-.013	.005	.027	-.076	-.063	.377	2.655
	STATURE (CM)	.005	.005	.133	1.059	.292	-.004	.015	-.024	.097	.081	.371	2.698

a. Dependent Variable: OA KNEE (LOG10+1)

Table A9. Shapiro-Wilk's test of normality (*P* > 0.05) for OA scores from the pelvic region

	Shapiro-Wilk		
	Statistic	df	Sig.
OA_PELVIS_SQRT+1	.984	289	.229

Table A10. Results of independent samples T-test for age and OA variables between males and females

		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
Age at Death	Equal variances assumed	.448	.504	.020	250	.984	.042	2.096	-4.085	4.170
	Equal variances not assumed			.020	247.516	.984	.042	2.098	-4.090	4.174
OA	Equal variances assumed	.161	.688	1.455	250	.147	.44551	.30619	-.15754	1.04856
	Equal variances not assumed			1.455	249.861	.147	.44551	.30615	-.15746	1.04849

Table A11. Partial correlation analysis for Buckberry and Chamberlain's method controlling for body size at the auricular surface

Control Variables			Estimate Error (estimated age-actual age)	OA	Body Mass (kg)
-none-[a]	Estimate Error (estimated age-actual age)	Correlation	1.000	-.179	.024
		Significance (2-tailed)		.004	.706
		df	0	250	250
Body Mass (kg)	Estimate Error (estimated age-actual age)	Correlation	1.000	-.182	
		Significance (2-tailed)		.004	
		df	0	249	

a. Cells contain zero-order (Pearson) correlations.

Table A12. Partial correlation analysis for Suchey-Brooks' method controlling for body size at the pubic symphysis

Control Variables			Estimate Error (estimated age-actual age)	OA	Body Mass (kg)
-none-[a]	Estimate Error (estimated age-actual age)	Correlation	1.000	-.302	.086
		Significance (2-tailed)		.000	.175
		df	0	248	248
Body Mass (kg)	Estimate Error (estimated age-actual age)	Correlation	1.000	-.303	
		Significance (2-tailed)		.000	
		df	0	247	

a. Cells contain zero-order (Pearson) correlations.

Table A13. Partial correlation analysis for Calce's method controlling for body size at the acetabulum

Control Variables			Estimate Error (estimated age-actual age)	OA	Body Mass (kg)
-none-[a]	Estimate Error (estimated age-actual age)	Correlation	1.000	-.056	-.026
		Significance (2-tailed)		.374	.683
		df	0	250	250
Body Mass (kg)	Estimate Error (estimated age-actual age)	Correlation	1.000	-.054	
		Significance (2-tailed)		.391	
		df	0	249	

a. Cells contain zero-order (Pearson) correlations.

Table A14. Published regression equations for estimating body mass and stature

Abbreviation	Method	Male	Female	Reference
ST	Stature (cm)	1.40*(FXL+TIB)+49.68	1.47*(FXL+TIB)+42.96	Ruff et al., 2012
FHB	Body mass (kg) "mechanical"	2.80*FHB–66.70	2.18*FHB–35.81	Ruff et al., 2012
ST/BIB[a]	Body mass (kg) "morphometric"	0.422*ST+3.126*BIB– 92.9	0.504*ST+1.804*BIB-72.6	Ruff et al., 2005

ST, stature; FHB, femoral head breadth (mm); BIB, bi-iliac breadth (cm); FXL, maximum femoral length (cm); TIB, maximum tibia length (cm)

[a] Corrected for "Living BIB" (1.17*BIB–3) (Ruff et al, 1997).

Appendix 3

Raw Data Tables

A. Age estimates and skeletal measurements

Collection	Calce _ID	Sex	Age	Calce _2012	Buckberry_ Chamberlain _2002	Suchey_ Brooks_ 1990	Max_Femoral_ head_diameter _mm	Max_Femoral _length_mm	Max_Tibia_ length _mm	BIB_ mm
Sassari	1	male	60	>65	53-92	27-66	46.11	470	410	272
Sassari	2	male	62	>65	39-91	27-66	45.02	411	335	262
Sassari	3	male	60	40-64	29-81	27-66	43.5	420	338	265
Sassari	4	male	25	17-39	29-81	21-46	46.57	429	354	269
Sassari	5	male	75	40-64	29-81	≥34	48.56	448	369	261
Sassari	6	male	73	>65	53-92	≥34	48.54	413	336	276
Sassari	7	male	82	>65	53-92	≥34	48.69	419	361	284
Sassari	8	male	57	40-64	29-88	27-66	40.42	412	347	236
Sassari	9	male	26	17-39	21-38	21-46	39.89	398	330	245
Sassari	10	male	50	40-64	29-88	27-66	45.43	443	366	270
Sassari	11	male	26	17-39	21-38	19-34	49.86	474	397	287
Sassari	12	male	50	40-64	29-88	27-66	40.77	422	343	251
Sassari	13	male	21	17-39	21-38	≤23	45.16	465	370	251
Sassari	14	male	35	17-39	29-88	23-57	43.72	446	371	241
Sassari	15	male	58	40-64	39-91	27-66	45.04	414	337	269
Sassari	16	male	58	40-64	29-81	≥34	44.7	407	340	248
Sassari	17	male	53	40-64	29-81	23-57	48.22	426	344	242
Sassari	18	male	37	17-39	29-88	23-57	48.71	462	362	274
Sassari	19	male	40	17-39	29-81	21-46	44.84	448	384	280
Sassari	20	male	64	40-64	53-92	≥34	42.43	412	347	255
Sassari	21	male	69	40-64	39-91	27-66	49.56	452	372	264
Sassari	22	male	70	40-64	29-88	23-57	38.3	419	340	259
Sassari	23	male	78	>65	39-91	≥34	48.09	479	381	297
Sassari	24	male	22	17-39	21-38	≤23	43.19	411	357	249
Sassari	25	male	55	40-64	39-91	27-66	43.39	413	350	265
Sassari	26	male	77	>65	29-81	27-66	43.67	410	347	253
Sassari	27	male	43	40-64	39-91	≥34	46.67	450	372	285
Sassari	28	male	20	17-39	16-65	≤23	42.61	438	358	263
Sassari	29	male	41	17-39	29-88	23-57	43.61	426	375	249
Sassari	30	male	60	40-64	29-88	≥34	48.68	451	387	275
Sassari	31	male	80	>65	53-92	≥34	42.22	411	344	244
Sassari	32	male	40	40-64	29-81	23-57	41.96	423	341	247
Sassari	33	male	40	40-64	39-91	27-66	47.27	434	365	260

Sassari	34	male	48	40-64	16-65	27-66	43.7	465	358	254
Sassari	35	male	53	40-64	29-81	23-57	46.06	415	364	255
Sassari	36	male	66	40-64	29-88	27-66	41.27	432	348	244
Sassari	37	male	66	40-64	29-88	27-66	43.46	450	375	265
Sassari	38	male	56	40-64	29-88	23-57	45.55	426	348	269
Sassari	39	male	67	40-64	39-91	23-57	41.73	411	341	244
Sassari	40	male	76	>65	39-91	≥34	45.21	455	391	291
Sassari	41	male	61	40-64	29-88	≥34	45.86	412	350	253
Sassari	42	male	29	17-39	29-88	23-57	45.27	461	381	269
Sassari	43	male	36	40-64	29-81	≥34	45.85	423	349	252
Sassari	44	male	31	17-39	16-65	23-57	39.69	405	337	262
Sassari	45	male	69	>65	29-88	≥34	43.04	428	352	237
Sassari	46	male	80	40-64	29-81	27-66	48.15	410	350	242
Sassari	47	male	78	>65	53-92	≥34	43.44	424	362	245
Sassari	48	male	60	40-64	29-81	23-57	42.65	394	326	248
Sassari	49	male	55	40-64	29-88	≥34	43.73	422	354	265
Sassari	50	male	50	>65	29-88	≥34	45.24	422	341	246
Sassari	51	male	55	40-64	53-92	≥34	47.67	442	360	252
Sassari	52	male	21	17-39	16-65	19-34	47.53	462	378	257
Sassari	53	female	78	≥65	29-81	≥42	40.8	381	316	273
Sassari	54	female	57	40-64	29-88	26-70	41.31	412	342	283
Sassari	55	female	98	≥65	29-88	26-70	40.34	379	323	277
Sassari	56	female	80	40-64	39-91	25-83	41.88	417	342	277
Sassari	57	female	54	40-64	29-88	25-83	43.04	408	327	258
Sassari	58	female	70	≥65	53-92	≥42	43.59	434	356	307
Sassari	59	female	68	40-64	39-91	26-70	42.04	422	341	285
Sassari	60	female	59	40-64	29-81	25-83	43.65	445	369	290
Sassari	61	female	75	40-64	29-81	≥42	44.93	420	345	273
Sassari	62	female	60	40-64	39-91	26-70	38.36	399	334	261
Sassari	63	female	63	40-64	53-92	26-70	34.56	373	303	242
Sassari	64	female	22	17-39	16-65	≤24	41.21	408	320	272
Sassari	65	female	17	17-39	16-19	≤24	36.85	362	302	193
Sassari	66	female	78	≥65	29-88	26-70	43.52	414	331	271
Sassari	67	female	20	17-39	21-38	19-40	42.08	383	313	264
Sassari	68	female	70	40-64	29-88	26-70	41.54	377	313	268
Sassari	69	female	88	≥65	53-92	≥42	42.84	388	329	284
Sassari	70	female	58	40-64	29-88	25-83	38.3	400	321	251
Sassari	71	female	54	≥65	39-91	≥42	39.96	368	318	253
Sassari	72	female	80	≥65	53-92	≥42	42.35	413	340	282
Sassari	73	female	45	≥65	29-88	25-83	45.72	429	352	0
Sassari	74	female	48	40-64	21-38	26-70	40.36	410	333	257

Sassari	75	female	42	40-64	29-88	26-70	38.02	410	329	243
Sassari	76	female	48	40-64	29-81	26-70	37.4	361	294	239
Sassari	77	female	70	≥65	39-91	≥42	43.9	447	355	276
Sassari	78	female	78	≥65	29-81	≥42	39.91	387	312	257
Sassari	80	female	39	17-39	16-65	21-53	39.3	379	309	239
Sassari	81	female	50	40-64	29-81	25-83	40.07	418	340	267
Sassari	82	female	34	17-39	29-81	19-40	48.15	441	368	251
Sassari	83	female	48	40-64	29-88	26-70	38.61	417	332	253
Sassari	84	female	50	40-64	29-81	26-70	38.59	393	333	255
Sassari	85	female	28	17-39	29-81	19-40	41.83	437	363	274
Sassari	86	female	35	17-39	21-38	26-70	35.51	362	306	246
Sassari	87	female	50	40-64	39-91	26-70	38.17	382	324	258
Sassari	88	female	70	40-64	39-91	≥42	38.2	395	323	250
Sassari	89	female	75	≥65	39-91	25-83	41.1	390	316	262
Sassari	90	female	53	40-64	16-65	26-70	38.57	416	340	267
Sassari	91	female	32	17-39	29-81	26-70	37.19	357	291	245
Sassari	92	female	34	40-64	29-88	≥42	40.67	426	344	274
Sassari	93	female	38	40-64	29-81	≥42	39.5	421	360	251
Sassari	94	female	47	17-39	29-88	21-53	37.55	409	336	263
Sassari	95	female	45	40-64	29-81	26-70	40.08	415	345	260
Sassari	96	female	40	17-39	29-88	21-53	39.5	386	325	266
Sassari	97	female	32	17-39	29-81	25-83	36.94	408	342	244
Sassari	98	female	25	17-39	29-88	19-34	36.94	387	310	266
Sassari	99	female	70	≥65	29-81	≥42	39.75	424	353	302
Sassari	100	female	24	17-39	29-88	19-40	39.81	423	355	277
Sassari	101	female	62	40-64	29-88	≥42	38.96	391	317	278
Sassari	102	female	90	40-64	39-91	≥42	37.83	392	334	261
Athens	103	female	79	40-64	29-88	≥42	40.28	394	309	270
Athens	104	female	84	40-64	29-88	26-70	41.82	419	335	274
Athens	105	female	67	>65	29-88	≥42	38.52	403	342	261
Athens	106	female	49	17-39	16-65	21-53	41.1	404	328	277
Athens	107	female	66	40-64	29-88	≥42	42.47	434	376	280
Athens	108	female	44	40-64	29-88	25-83	41.83	406	335	284
Athens	109	female	46	17-39	29-81	21-53	40.11	388	313	273
Athens	110	female	44	40-64	16-65	26-70	42.38	433	347	274
Athens	111	female	27	17-39	16-65	21-53	42.22	448	346	267
Athens	112	female	81	>65	29-88	≥42	44.36	456	370	299
Athens	113	female	72	40-64	29-81	26-70	41.27	413	343	271
Athens	114	female	54	40-64	29-88	25-83	43.67	448	379	289
Athens	115	female	48	17-39	29-81	26-70	42.19	409	331	274
Athens	116	female	54	40-64	29-81	25-83	42	403	329	269

Athens	117	female	51	40-64	29-81	26-70	39.5	425	352	270
Athens	118	female	72	>65	16-65	25-83	44.57	418	348	298
Athens	119	female	37	17-39	16-65	21-53	37.26	398	331	249
Athens	120	female	47	17-39	21-38	26-70	44.47	412	340	272
Athens	121	female	82	>65	53-92	≥42	38.29	398	338	265
Athens	122	female	33	17-39	16-65	21-53	40.7	390	322	254
Athens	123	female	65	40-64	39-91	25-83	40.37	400	325	280
Athens	124	female	85	40-64	39-91	25-83	41.18	400	320	293
Athens	125	female	45	40-64	29-81	26-70	42.38	404	336	278
Athens	126	female	78	40-64	39-91	≥42	39.84	389	318	283
Athens	127	female	27	17-39	16-65	21-53	40.8	421	353	247
Athens	128	female	77	40-64	29-88	26-70	41.95	435	358	272
Athens	129	female	46	40-64	29-81	26-70	41.2	417	333	273
Athens	130	female	69	>65	29-88	25-83	40.64	405	339	279
Athens	131	female	56	>65	53-92	25-83	40.41	406	305	260
Athens	132	female	62	40-64	29-88	≥42	34.04	419	352	259
Athens	133	female	52	40-64	29-88	25-83	38.8	413	335	278
Athens	134	female	60	>65	29-88	25-83	39.36	399	333	261
Athens	135	female	63	40-64	29-81	26-70	46.9	461	395	310
Athens	136	female	82	>65	39-91	≥42	39.87	387	316	265
Athens	137	female	87	>65	39-91	≥42	39.41	388	324	274
Athens	138	female	59	40-64	29-81	26-70	39.88	383	329	269
Athens	139	female	73	>65	39-91	21-53	38.33	402	327	264
Athens	140	female	84	>65	53-92	≥42	41.19	389	326	253
Athens	141	female	81	40-64	29-81	25-83	40.99	401	322	272
Athens	142	female	79	40-64	39-91	25-83	43.46	425	359	299
Athens	143	female	69	40-64	29-88	≥42	41.28	409	331	278
Athens	144	female	99	>65	39-91	≥42	40.15	395	335	278
Athens	145	female	49	40-64	29-81	26-70	40.57	428	357	272
Athens	146	female	38	17-39	29-81	25-83	36.58	387	321	264
Athens	147	female	20	17-39	16-65	19-40	40.1	408	342	260
Athens	148	female	35	17-39	16-65	21-53	39.74	407	343	271
Athens	149	female	50	40-64	39-91	NA*	40.08	398	314	269
Athens	150	female	24	17-39	16-65	26-70	42.83	427	361	255
Athens	151	female	22	17-39	21-38	≤24	43.01	428	376	258
Athens	152	female	63	40-64	29-81	25-83	42.05	410	342	278
Athens	153	male	29	17-39	21-38	19-34	43.78	458	385	269
Athens	154	male	33	17-39	16-65	23-57	48.16	462	393	253
Athens	155	male	32	17-39	16-65	23-57	49.24	458	382	262
Athens	156	male	26	17-39	29-81	23-57	47.4	458	390	268
Athens	157	male	55	40-64	39-91	≥34	46.08	420	350	263

Athens	158	male	94	≥65	29-88	≥34	43.38	420	350	281
Athens	159	male	73	≥65	39-91	≥34	46.35	434	358	295
Athens	160	male	66	≥65	39-91	≥34	40.75	413	369	282
Athens	161	male	58	40-64	29-88	27-66	44.08	429	352	282
Athens	162	male	88	≥65	39-91	≥34	53.54	501	412	309
Athens	163	male	85	≥65	39-91	≥34	45.92	464	395	280
Athens	164	male	61	40-64	29-81	27-66	47.47	470	393	306
Athens	165	male	81	≥65	39-91	≥34	48.95	474	381	298
Athens	166	male	66	≥65	53-92	≥34	49.12	487	418	295
Athens	167	male	55	40-64	16-65	≥34	45.5	396	358	272
Athens	168	male	84	≥65	39-91	≥34	46.48	460	390	304
Athens	169	male	84	≥65	39-91	≥34	42.71	454	369	284
Athens	170	male	50	40-64	16-65	27-66	47.14	439	361	290
Athens	171	male	61	≥65	39-91	≥34	45.96	448	360	265
Athens	172	male	72	17-39	16-65	27-66	44.14	407	350	250
Athens	173	male	29	17-39	21-38	21-46	47.86	498	410	294
Athens	174	male	68	≥65	39-91	≥34	47.22	451	363	291
Athens	175	male	24	17-39	21-38	19-34	43.87	458	397	270
Athens	176	male	50	40-64	29-88	23-57	42.79	450	384	262
Athens	177	male	49	40-64	29-81	23-57	46.27	446	367	288
Athens	178	male	56	40-64	29-81	≥34	47.91	472	409	280
Athens	179	male	27	17-39	21-38	21-46	44.31	431	356	276
Athens	180	male	48	40-64	29-81	23-57	47.1	459	378	290
Athens	181	male	29	17-39	16-65	23-57	50.99	482	392	289
Athens	182	male	48	40-64	16-65	23-57	46.06	428	363	282
Athens	183	male	75	≥65	39-91	≥34	44.23	379	464	273
Athens	184	male	34	17-39	16-65	21-46	46.98	489	414	294
Athens	185	male	65	≥65	39-91	≥34	46.09	428	351	251
Athens	186	male	48	40-64	29-88	23-57	44.19	432	359	257
Athens	187	male	94	40-64	29-81	23-57	45.87	451	376	290
Athens	188	male	67	40-64	29-88	23-57	44.95	419	352	273
Athens	189	male	76	≥65	29-88	27-66	43.45	441	370	278
Athens	190	male	36	17-39	16-65	23-57	49.74	431	369	277
Athens	191	male	64	40-64	29-88	≥34	45.32	444	371	258
Athens	192	male	55	40-64	39-91	23-57	44.95	422	352	280
Athens	193	male	39	≥65	53-92	≥34	42.82	419	364	226
Athens	194	male	58	40-64	16-65	27-66	49.73	446	369	294
Athens	195	male	76	40-64	53-92	27-66	47.37	432	350	280
Athens	196	male	60	17-39	16-65	23-57	42.96	410	331	243
Lisbon	197	female	78	≥65	29-88	25-83	41.55	428	353	268
Lisbon	198	female	18	17-39	16-19	≤24	39.43	409	322	237

Lisbon	199	female	54	40-64	16-65	26-70	45.56	450	380	281
Lisbon	200	female	62	40-64	29-81	≥42	41.79	374	307	255
Lisbon	201	female	19	17-39	16-19	≤24	41.79	428	351	251
Lisbon	202	female	56	40-64	16-65	21-53	40.26	402	333	275
Lisbon	203	female	66	40-64	39-91	25-83	42.24	433	355	253
Lisbon	204	female	48	40-64	21-38	26-70	38.68	411	342	258
Lisbon	205	female	94	≥65	39-91	≥42	41.05	419	350	289
Lisbon	206	female	67	40-64	39-91	≥42	37.5	376	338	240
Lisbon	207	female	59	40-64	29-81	26-70	42.45	412	350	268
Lisbon	208	female	76	40-64	29-81	25-83	38.63	395	341	259
Lisbon	209	female	28	17-39	21-38	21-53	37.74	396	337	259
Lisbon	210	female	35	17-39	21-38	21-53	38.8	374	310	264
Lisbon	211	female	76	≥65	29-88	≥42	38.8	391	321	267
Lisbon	212	female	23	17-39	21-38	19-40	39.78	410	340	244
Lisbon	213	female	44	40-64	29-81	26-70	40.11	412	347	279
Lisbon	214	female	85	40-64	16-65	21-53	41.27	411	356	273
Lisbon	215	female	59	40-64	29-81	≥42	36.54	372	328	232
Lisbon	216	female	20	17-39	16-65	≤24	38.74	426	339	258
Lisbon	217	female	29	17-39	21-38	21-53	42.24	409	335	282
Lisbon	218	female	50	40-64	29-81	21-53	39.42	424	357	246
Lisbon	219	female	27	17-39	16-19	26-70	39.15	410	415	266
Lisbon	220	female	93	≥65	39-91	25-83	38.87	413	339	295
Lisbon	221	female	39	17-39	21-38	26-70	40.66	432	355	262
Lisbon	222	female	51	40-64	16-65	25-83	40.52	386	317	260
Lisbon	223	female	43	40-64	16-65	25-83	39.92	424	360	257
Lisbon	224	female	68	≥65	39-91	25-83	45.12	392	329	294
Lisbon	225	female	48	40-64	16-65	25-83	42.05	421	330	260
Lisbon	226	female	44	40-64	16-65	25-83	38.89	400	334	267
Lisbon	227	female	46	17-39	21-38	26-70	41.93	428	351	248
Lisbon	228	female	32	17-39	21-38	26-70	38.03	405	331	255
Lisbon	229	female	35	17-39	21-38	19-40	40.41	433	362	275
Lisbon	230	female	74	40-64	29-81	26-70	41.59	424	357	272
Lisbon	231	female	24	17-39	21-38	≤24	42.04	449	359	286
Lisbon	232	female	73	40-64	29-88	≥42	38.02	396	333	242
Lisbon	233	female	53	40-64	53-92	25-83	40.56	456	364	244
Lisbon	234	female	56	40-64	29-81	≥42	40.33	404	323	265
Lisbon	235	female	57	40-64	39-91	25-83	42.95	395	326	286
Lisbon	236	female	52	40-64	53-92	≥42	39.9	406	328	275
Lisbon	237	female	59	40-64	29-81	≥42	38.26	397	335	286
Lisbon	238	female	83	≥65	39-91	≥42	39.43	393	316	244
Lisbon	239	female	77	≥65	53-92	≥42	38.86	384	311	290

Lisbon	240	female	86	≥65	39-91	≥42	41.26	413	336	273
Lisbon	241	female	82	≥65	39-91	≥42	43.85	433	357	320
Lisbon	242	female	90	≥65	53-92	≥42	37.65	383	324	246
Lisbon	243	male	53	40-64	39-91	23-57	39.88	425	352	243
Lisbon	244	male	47	40-64	16-65	23-57	47.18	464	392	273
Lisbon	245	male	65	40-64	29-81	27-66	38.73	380	303	250
Lisbon	246	male	81	≥65	53-92	≥34	47.17	434	362	302
Lisbon	247	male	67	40-64	29-81	≥34	45.27	452	372	266
Lisbon	248	male	48	40-64	16-65	27-66	46.34	448	370	277
Lisbon	249	male	37	17-39	29-88	27-66	43.23	449	363	246
Lisbon	250	male	47	40-64	39-91	27-66	45.18	459	410	287
Lisbon	251	male	30	17-39	29-88	23-57	46.41	440	376	285
Lisbon	252	male	46	40-64	16-65	27-66	43.73	427	348	253
Lisbon	253	male	64	40-64	29-88	≥34	42.11	431	359	272
Lisbon	254	male	49	≥65	39-91	≥34	45.66	450	389	273
Lisbon	255	male	68	40-64	53-92	≥34	41.87	431	377	257
Lisbon	256	male	53	40-64	29-81	23-57	41.36	425	355	260
Lisbon	257	male	58	40-64	29-88	≥34	42.7	405	345	251
Lisbon	258	male	63	40-64	29-81	23-57	46.32	439	385	276
Lisbon	259	male	68	40-64	29-81	27-66	41.75	424	349	258
Lisbon	260	male	52	40-64	29-81	23-57	42.36	439	374	293
Lisbon	261	male	40	40-64	16-65	23-57	46.29	439	368	242
Lisbon	262	male	50	40-64	29-88	23-57	39.97	430	361	238
Lisbon	263	male	82	≥65	29-88	27-66	46.3	441	378	283
Lisbon	264	male	75	≥65	53-92	≥34	43.95	442	349	265
Lisbon	265	male	20	17-39	16-19	NA	44.66	440	368	261
Lisbon	266	male	40	40-64	29-81	27-66	41.37	415	350	235
Lisbon	267	male	43	40-64	21-38	23-57	38.5	418	350	251
Lisbon	268	male	30	17-39	21-38	≤23	45.4	438	352	271
Lisbon	269	male	69	≥65	53-92	≥34	47.01	452	371	279
Lisbon	270	male	57	40-64	21-38	23-57	47.8	492	424	273
Lisbon	271	male	29	17-39	16-65	21-46	48.58	446	372	266
Lisbon	272	male	83	≥65	29-88	≥34	47.52	473	396	280
Lisbon	273	male	38	17-39	16-65	23-57	44.71	441	339	233
Lisbon	274	male	72	40-64	29-81	≥34	41.39	404	332	244
Lisbon	275	male	31	17-39	21-38	≤23	46.99	440	356	270
Lisbon	276	male	47	17-39	21-38	23-57	41.12	440	369	254
Lisbon	277	male	84	≥65	29-88	≥34	45.24	467	377	267
Lisbon	278	male	21	17-39	21-38	≤23	44.44	446	368	245
Lisbon	279	male	71	40-64	29-81	≥34	46.92	461	380	281
Lisbon	280	male	23	17-39	21-38	21-46	39.94	382	302	233

Lisbon	281	male	49	40-64	29-88	27-66	45.08	437	353	253
Lisbon	282	male	68	40-64	29-88	≥34	40.11	424	364	260
Lisbon	283	male	55	40-64	16-65	21-46	42.93	413	359	248
Lisbon	284	male	28	17-39	21-38	21-46	47.38	465	385	270
Lisbon	285	male	49	40-64	29-88	23-57	48.04	461	377	290
Lisbon	286	male	53	40-64	16-65	27-66	43.84	424	340	262
Lisbon	287	male	45	40-64	29-81	23-57	42.81	443	373	252
Lisbon	288	male	62	40-64	29-81	27-66	44.65	410	347	231
Lisbon	289	male	88	≥65	53-92	≥34	41.3	413	347	253
Lisbon	290	male	82	≥65	39-91	≥34	39.55	422	370	240

B. Lumbar spine OA scores

Collection	Calce_ID	Sex	Age	LIP_S_ LUMBAR	LIP_D_ LUMBAR	POR_S_ LUMBAR	POR_D_ LUMBAR	EBR_S_ LUMBAR	EBR_D_ LUMBAR	OPH_S_ LUMBAR
Sassari	1	male	60	2.67	2.22	2.11	1.78	0.11	0.11	1.60
Sassari	2	male	62	2.82	2.18	1.18	1.45	0.00	0.00	1.64
Sassari	3	male	60	#NULL!	#NULL!	#NULL!	#NULL!	#NULL!	#NULL!	#NULL!
Sassari	4	male	25	0.91	0.91	0.55	0.55	0.00	0.00	0.65
Sassari	5	male	75	2.64	2.45	2.09	1.27	0.00	0.00	1.64
Sassari	6	male	73	2.36	2.73	1.91	2.09	0.00	0.00	1.36
Sassari	7	male	82	2.73	2.73	2.45	2.36	0.91	0.55	1.80
Sassari	8	male	57	1.45	1.27	0.18	0.18	0.00	0.00	0.65
Sassari	9	male	26	1.00	2.00	1.00	1.00	1.00	1.00	1.20
Sassari	10	male	50	2.18	2.55	2.18	2.27	0.00	0.00	1.53
Sassari	11	male	26	0.27	0.27	1.36	2.55	0.09	0.09	0.11
Sassari	12	male	50	2.09	2.36	1.18	2.91	0.36	0.18	1.47
Sassari	13	male	21	0.18	0.18	0.00	0.00	0.00	0.00	0.49
Sassari	14	male	35	0.27	0.36	1.00	1.00	0.09	0.18	0.16
Sassari	15	male	58	1.82	1.55	1.91	1.73	0.09	0.09	1.42
Sassari	16	male	58	2.09	2.09	1.64	1.27	0.00	0.00	1.42
Sassari	17	male	53	1.27	0.91	1.00	2.00	0.09	0.09	0.98
Sassari	18	male	37	0.09	0.09	1.00	1.00	0.00	0.00	0.38
Sassari	19	male	40	0.27	0.27	0.73	0.73	0.00	0.00	0.38
Sassari	20	male	64	2.00	1.82	1.55	1.64	0.18	0.09	1.31
Sassari	21	male	69	2.00	2.00	1.18	1.91	0.09	0.09	1.47
Sassari	22	male	70	2.64	2.45	2.27	2.36	0.18	0.18	1.91
Sassari	23	male	78	3.09	2.27	2.09	1.55	0.27	0.18	1.80
Sassari	24	male	22	0.00	0.00	1.09	0.73	0.00	0.00	0.65
Sassari	25	male	55	1.55	1.55	1.18	0.64	0.00	0.00	1.31
Sassari	26	male	77	2.18	2.09	1.27	1.00	0.00	0.00	1.75
Sassari	27	male	43	1.09	1.00	0.27	0.27	0.00	0.00	0.38
Sassari	28	male	20	0.22	0.11	0.11	0.11	0.00	0.00	0.67
Sassari	29	male	41	0.82	0.55	1.00	1.09	0.00	0.00	0.55
Sassari	30	male	60	2.73	2.64	2.64	2.00	0.00	0.00	1.80
Sassari	31	male	80	2.36	2.82	2.82	2.27	0.64	0.27	1.80
Sassari	32	male	40	2.00	2.36	0.27	0.27	0.00	0.00	1.75
Sassari	33	male	40	1.09	0.82	0.00	0.00	0.00	0.00	1.36
Sassari	34	male	48	2.18	2.09	2.18	1.00	0.00	0.00	1.75
Sassari	35	male	53	2.09	1.91	1.09	0.91	0.00	0.00	1.69
Sassari	36	male	66	2.09	1.91	1.55	0.82	0.00	0.00	1.75
Sassari	37	male	66	2.27	2.09	2.45	1.82	0.00	0.00	1.75

Sassari	38	male	56	2.00	2.00	0.00	0.00	0.00	0.00	0.60
Sassari	39	male	67	2.18	1.91	1.73	1.09	0.00	0.00	1.75
Sassari	40	male	76	2.09	1.91	1.73	1.18	0.55	0.18	1.47
Sassari	41	male	61	2.27	2.27	1.09	1.91	0.00	0.00	1.80
Sassari	42	male	29	0.18	0.18	0.45	0.27	0.00	0.00	0.05
Sassari	43	male	36	0.73	0.73	1.00	1.00	0.00	0.00	0.76
Sassari	44	male	31	1.45	0.82	1.18	1.00	0.00	0.00	0.98
Sassari	45	male	69	2.27	2.00	2.55	1.91	0.36	0.18	1.75
Sassari	46	male	80	1.56	1.11	1.33	1.00	0.00	0.00	1.40
Sassari	47	male	78	3.27	2.45	2.20	3.00	0.00	0.00	1.80
Sassari	48	male	60	1.00	0.45	0.45	0.00	0.00	0.00	0.65
Sassari	49	male	55	0.82	0.64	1.00	1.00	0.00	0.00	0.38
Sassari	50	male	50	2.55	1.55	1.36	1.00	0.00	0.00	1.64
Sassari	51	male	55	1.82	1.82	0.18	0.09	0.00	0.00	1.31
Sassari	52	male	21	0.00	0.00	0.09	0.00	0.00	0.00	0.33
Sassari	53	female	78	2.55	2.27	1.91	2.00	0.18	0.18	1.69
Sassari	54	female	57	1.64	0.91	1.09	0.82	0.00	0.00	1.15
Sassari	55	female	98	2.82	2.91	2.36	1.45	1.36	1.00	1.80
Sassari	56	female	80	1.73	1.45	1.64	1.18	0.00	0.00	1.47
Sassari	57	female	54	0.09	0.09	0.27	0.27	0.00	0.00	0.11
Sassari	58	female	70	2.09	2.00	2.18	0.82	0.91	0.36	1.36
Sassari	59	female	68	0.36	0.27	0.45	0.18	0.09	0.09	0.22
Sassari	60	female	59	2.09	2.09	1.91	1.27	0.00	0.00	1.80
Sassari	61	female	75	0.55	0.36	0.45	0.36	0.00	0.00	0.55
Sassari	62	female	60	2.60	2.60	1.80	1.40	0.80	0.40	1.80
Sassari	63	female	63	2.00	2.00	0.55	0.55	0.00	0.00	1.80
Sassari	64	female	22	0.18	0.18	0.00	0.00	0.00	0.00	0.11
Sassari	65	female	17	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Sassari	66	female	78	2.14	2.43	1.14	0.86	1.14	0.71	1.80
Sassari	67	female	20	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Sassari	68	female	70	2.09	2.09	1.64	1.73	0.00	0.00	1.69
Sassari	69	female	88	2.18	2.36	2.18	1.73	1.09	1.09	1.80
Sassari	70	female	58	1.27	1.18	1.09	0.64	0.18	0.18	1.47
Sassari	71	female	54	0.00	0.00	0.00	0.00	0.00	0.00	0.11
Sassari	72	female	80	2.55	2.27	2.64	1.82	0.55	0.36	1.80
Sassari	73	female	45	#NULL!	#NULL!	#NULL!	#NULL!	#NULL!	#NULL!	#NULL!
Sassari	74	female	48	0.55	0.36	0.09	0.09	0.00	0.00	0.27
Sassari	75	female	42	0.82	0.82	1.00	1.00	0.00	0.00	0.00
Sassari	76	female	48	0.91	0.64	0.27	0.27	0.00	0.00	0.55
Sassari	77	female	70	1.64	1.45	1.82	1.18	0.00	0.00	1.36
Sassari	78	female	78	2.78	2.56	2.56	2.11	2.33	1.22	1.85

Sassari	80	female	39	1.18	0.91	0.00	0.00	0.00	0.00	0.65
Sassari	81	female	50	1.78	1.67	1.11	1.00	0.00	0.00	1.07
Sassari	82	female	34	0.27	0.18	0.00	0.00	0.00	0.00	0.33
Sassari	83	female	48	1.18	0.91	0.18	0.18	0.00	0.00	0.76
Sassari	84	female	50	1.91	1.36	1.18	0.64	0.36	0.36	1.47
Sassari	85	female	28	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Sassari	86	female	35	1.00	1.00	0.00	0.00	0.00	0.00	0.60
Sassari	87	female	50	1.73	1.18	1.18	1.00	0.00	0.00	1.69
Sassari	88	female	70	2.09	2.18	2.55	1.82	0.00	0.00	1.80
Sassari	89	female	75	2.30	2.40	2.64	1.91	0.00	0.00	1.80
Sassari	90	female	53	1.91	1.45	1.18	1.27	0.00	0.00	1.64
Sassari	91	female	32	0.36	0.36	0.27	0.27	0.00	0.00	0.22
Sassari	92	female	34	0.45	0.45	0.00	0.00	0.00	0.00	0.27
Sassari	93	female	38	0.82	0.64	0.00	0.00	0.00	0.00	0.16
Sassari	94	female	47	1.18	0.91	0.00	0.00	0.00	0.00	0.71
Sassari	95	female	45	0.73	0.73	0.00	0.00	0.00	0.00	0.49
Sassari	96	female	40	1.18	1.18	1.00	1.00	0.36	0.18	0.76
Sassari	97	female	32	0.36	0.27	0.27	0.27	0.00	0.00	0.33
Sassari	98	female	25	0.27	0.18	0.55	0.55	0.00	0.00	0.11
Sassari	99	female	70	2.82	2.00	1.55	0.91	1.64	0.73	1.80
Sassari	100	female	24	0.18	0.18	0.00	0.00	0.00	0.00	0.27
Sassari	101	female	62	2.18	2.00	1.36	1.64	0.36	0.18	1.80
Sassari	102	female	90	2.18	2.00	1.18	1.00	0.09	0.09	1.80
Athens	103	female	79	2.82	2.64	3.00	1.64	0.82	0.55	1.80
Athens	104	female	84	2.45	2.45	2.36	1.45	1.18	0.82	1.80
Athens	105	female	67	2.78	2.56	1.00	2.00	1.22	0.67	1.80
Athens	106	female	49	1.91	1.18	1.00	0.45	0.00	0.00	1.58
Athens	107	female	66	2.82	2.36	1.45	1.00	0.55	0.18	1.80
Athens	108	female	44	1.27	1.09	0.64	0.64	0.00	0.00	0.49
Athens	109	female	46	1.27	0.91	0.91	0.91	0.00	0.00	1.20
Athens	110	female	44	1.27	0.91	0.00	0.00	0.00	0.00	1.15
Athens	111	female	27	0.36	0.36	0.00	0.00	0.00	0.00	0.22
Athens	112	female	81	2.86	2.71	2.14	2.14	0.00	0.00	1.80
Athens	113	female	72	2.82	3.00	1.18	1.00	0.00	0.00	1.80
Athens	114	female	54	2.00	2.00	0.00	0.00	0.00	0.00	1.20
Athens	115	female	48	1.18	1.09	0.00	0.00	0.00	0.00	0.87
Athens	116	female	54	1.67	1.00	0.00	0.00	0.00	0.00	1.27
Athens	117	female	51	2.00	2.00	0.45	0.45	0.00	0.00	1.42
Athens	118	female	72	3.00	2.73	1.45	0.82	1.45	0.91	1.80
Athens	119	female	37	0.64	0.45	0.00	0.00	0.00	0.00	0.38
Athens	120	female	47	1.09	1.00	0.73	0.73	0.00	0.00	0.65

Athens	121	female	82	2.64	2.55	1.64	1.00	0.55	0.27	1.80
Athens	122	female	33	1.36	0.91	0.91	0.91	0.00	0.00	0.71
Athens	123	female	65	1.73	1.36	0.00	0.00	0.36	0.18	1.04
Athens	124	female	85	2.56	2.00	1.78	1.11	0.00	0.00	1.80
Athens	125	female	45	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Athens	126	female	78	2.27	2.09	0.55	0.27	0.00	0.00	1.42
Athens	127	female	27	0.36	0.18	0.00	0.00	0.00	0.00	0.22
Athens	128	female	77	2.91	1.64	1.64	1.55	0.82	0.55	1.80
Athens	129	female	46	0.89	0.89	1.44	1.00	0.00	0.00	0.67
Athens	130	female	69	2.45	2.27	1.18	1.91	0.36	0.18	1.80
Athens	131	female	56	2.45	1.55	1.00	1.00	0.91	0.36	1.47
Athens	132	female	62	2.73	1.73	1.45	1.00	0.00	0.00	1.80
Athens	133	female	52	1.91	1.45	1.00	1.00	0.00	0.00	1.20
Athens	134	female	60	2.36	2.09	1.73	2.09	0.36	0.18	1.64
Athens	135	female	63	2.38	1.50	1.36	1.00	0.36	0.18	1.53
Athens	136	female	82	3.00	2.91	2.82	1.91	0.00	0.00	1.80
Athens	137	female	87	2.82	2.64	1.18	2.09	0.36	0.18	1.80
Athens	138	female	59	1.82	1.82	0.91	0.91	0.00	0.00	1.47
Athens	139	female	73	3.00	2.45	2.45	1.91	0.36	0.18	1.80
Athens	140	female	84	2.64	2.00	1.64	1.27	0.55	0.18	1.31
Athens	141	female	81	2.82	2.18	2.64	2.27	0.00	0.00	1.53
Athens	142	female	79	2.82	2.00	2.64	2.00	0.36	0.18	1.69
Athens	143	female	69	1.91	1.36	0.73	0.73	0.55	0.18	1.04
Athens	144	female	99	2.86	2.57	1.71	1.86	0.14	0.14	1.71
Athens	145	female	49	1.36	0.91	1.36	1.00	0.55	0.18	0.71
Athens	146	female	38	1.09	1.09	1.00	1.00	0.00	0.00	0.60
Athens	147	female	20	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Athens	148	female	35	0.67	0.44	0.89	0.89	0.00	0.00	0.33
Athens	149	female	50	1.91	1.64	0.55	0.55	0.00	0.00	1.25
Athens	150	female	24	0.11	0.11	0.00	0.00	0.00	0.00	0.07
Athens	151	female	22	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Athens	152	female	63	2.36	2.18	1.18	0.82	0.00	0.00	1.47
Athens	153	male	29	0.27	0.18	0.91	0.91	0.00	0.00	0.11
Athens	154	male	33	0.36	0.18	1.00	0.82	0.36	0.18	0.00
Athens	155	male	32	0.64	0.45	1.00	1.00	0.00	0.00	0.16
Athens	156	male	26	0.00	0.00	0.18	0.18	0.00	0.00	0.00
Athens	157	male	55	2.18	1.73	0.91	0.55	0.00	0.00	1.20
Athens	158	male	94	2.73	2.55	2.00	1.64	0.00	0.00	1.75
Athens	159	male	73	2.27	2.27	1.73	1.36	0.55	0.18	1.53
Athens	160	male	66	1.55	1.91	1.36	1.00	0.00	0.00	0.82
Athens	161	male	58	2.45	1.64	1.36	1.00	0.00	0.00	1.58

Athens	162	male	88	2.91	2.91	2.82	2.09	0.00	0.00	1.75
Athens	163	male	85	2.90	1.60	2.27	1.27	0.89	0.67	1.74
Athens	164	male	61	1.73	1.73	1.91	1.18	0.00	0.00	1.04
Athens	165	male	81	3.09	1.55	1.89	1.11	0.00	0.00	1.75
Athens	166	male	66	2.78	2.44	1.44	0.89	0.22	0.22	1.80
Athens	167	male	55	1.91	1.91	1.00	0.64	0.36	0.18	1.36
Athens	168	male	84	#NULL!	#NULL!	#NULL!	#NULL!	#NULL!	#NULL!	#NULL!
Athens	169	male	84	2.18	2.55	1.55	0.82	0.91	0.55	1.64
Athens	170	male	50	1.36	1.45	0.55	0.55	0.00	0.00	0.98
Athens	171	male	61	2.11	2.78	1.67	1.44	0.00	0.00	1.33
Athens	172	male	72	2.18	1.82	1.36	1.36	1.09	0.64	1.20
Athens	173	male	29	0.00	0.00	0.73	0.73	0.00	0.00	0.00
Athens	174	male	68	3.44	2.00	1.20	1.00	0.00	0.00	1.98
Athens	175	male	24	0.09	0.09	1.00	1.00	0.00	0.00	0.05
Athens	176	male	50	1.89	1.89	1.33	1.00	0.00	0.00	1.40
Athens	177	male	49	1.89	1.78	0.00	0.00	0.00	0.00	1.33
Athens	178	male	56	1.36	1.36	0.91	0.91	0.00	0.00	0.93
Athens	179	male	27	0.27	0.27	0.91	0.91	0.00	0.00	0.16
Athens	180	male	48	1.73	1.18	1.00	1.18	0.00	0.00	1.09
Athens	181	male	29	0.00	0.00	0.82	0.82	0.00	0.00	0.00
Athens	182	male	48	0.82	0.73	0.55	0.55	0.00	0.00	0.49
Athens	183	male	75	2.45	2.00	1.18	2.00	0.36	0.18	1.69
Athens	184	male	34	0.09	0.09	0.45	0.45	0.00	0.00	0.05
Athens	185	male	65	2.22	2.11	1.89	1.00	0.00	0.00	1.60
Athens	186	male	48	1.00	0.91	1.18	0.82	0.00	0.00	0.71
Athens	187	male	94	2.15	2.08	1.54	1.38	0.00	0.00	1.43
Athens	188	male	67	2.00	1.69	1.77	1.23	0.23	0.23	1.15
Athens	189	male	76	1.33	1.11	0.89	0.78	0.00	0.00	0.80
Athens	190	male	36	0.45	0.45	0.55	0.45	0.00	0.00	0.33
Athens	191	male	64	2.00	1.73	1.27	0.91	0.18	0.18	1.36
Athens	192	male	55	2.09	1.91	0.73	0.45	0.00	0.00	1.36
Athens	193	male	39	0.33	0.33	0.89	0.56	0.00	0.00	0.20
Athens	194	male	58	2.18	2.36	0.55	0.36	0.00	0.00	1.53
Athens	195	male	76	2.55	1.55	1.45	1.09	0.55	0.18	1.64
Athens	196	male	60	2.27	1.55	1.18	1.00	0.00	0.00	1.47
Lisbon	197	female	78	2.45	2.09	1.55	1.09	0.36	0.36	1.64
Lisbon	198	female	18	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	199	female	54	1.82	1.09	0.18	0.09	0.00	0.00	1.36
Lisbon	200	female	62	1.27	1.00	0.73	0.45	0.73	0.36	0.71
Lisbon	201	female	19	0.00	0.00	0.82	0.55	0.00	0.00	0.00
Lisbon	202	female	56	1.36	1.27	1.09	0.64	0.64	0.55	0.98

Lisbon	203	female	66	0.91	0.82	0.36	0.18	0.91	0.36	0.38
Lisbon	204	female	48	0.91	0.64	0.64	0.45	0.00	0.00	0.55
Lisbon	205	female	94	3.00	2.55	2.36	2.27	0.91	0.73	1.80
Lisbon	206	female	67	1.64	0.82	1.91	1.09	0.55	0.55	1.20
Lisbon	207	female	59	2.18	1.55	0.00	0.00	0.00	0.00	1.75
Lisbon	208	female	76	1.45	0.91	0.55	0.36	0.00	0.00	1.25
Lisbon	209	female	28	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	210	female	35	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	211	female	76	2.09	2.00	1.27	1.09	1.64	0.73	1.09
Lisbon	212	female	23	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	213	female	44	0.45	0.45	0.00	0.00	0.00	0.00	0.16
Lisbon	214	female	85	3.11	3.00	1.86	0.86	2.29	1.14	1.88
Lisbon	215	female	59	1.09	1.09	0.00	0.00	0.00	0.00	0.65
Lisbon	216	female	20	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	217	female	29	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	218	female	50	0.73	0.64	0.18	0.20	0.00	0.00	0.49
Lisbon	219	female	27	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	220	female	93	2.73	3.00	1.45	0.91	1.00	0.64	1.58
Lisbon	221	female	39	0.09	0.09	0.36	0.18	0.18	0.18	0.05
Lisbon	222	female	51	1.55	1.00	0.18	0.18	0.45	0.36	0.82
Lisbon	223	female	43	0.82	0.82	0.00	0.00	0.18	0.18	0.16
Lisbon	224	female	68	2.00	2.18	1.82	1.09	0.18	0.18	1.42
Lisbon	225	female	48	0.82	0.64	0.00	0.00	0.00	0.00	0.44
Lisbon	226	female	44	0.82	0.82	0.09	0.09	0.00	0.00	0.60
Lisbon	227	female	46	0.45	0.45	0.00	0.00	0.00	0.00	0.27
Lisbon	228	female	32	0.45	0.45	0.00	0.00	0.00	0.00	0.27
Lisbon	229	female	35	0.36	0.36	0.18	0.09	0.00	0.00	0.11
Lisbon	230	female	74	0.27	0.27	0.00	0.00	0.00	0.00	0.00
Lisbon	231	female	24	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	232	female	73	2.18	1.91	0.18	0.36	0.27	0.18	1.64
Lisbon	233	female	53	2.45	1.82	0.75	0.50	0.00	0.00	1.64
Lisbon	234	female	56	2.00	2.00	0.55	0.45	0.00	0.00	1.47
Lisbon	235	female	57	1.91	1.91	0.36	0.27	0.55	0.18	1.15
Lisbon	236	female	52	1.64	1.45	0.55	0.36	0.36	0.45	0.98
Lisbon	237	female	59	1.27	1.00	0.00	0.00	0.00	0.00	0.71
Lisbon	238	female	83	1.91	1.64	1.09	1.00	0.18	0.18	1.15
Lisbon	239	female	77	1.91	2.00	1.82	1.18	0.55	0.36	1.15
Lisbon	240	female	86	2.27	1.82	1.91	1.27	0.18	0.18	1.58
Lisbon	241	female	82	2.36	2.36	2.00	2.27	0.00	0.00	1.53
Lisbon	242	female	90	2.27	1.82	0.56	0.56	0.89	0.44	1.31
Lisbon	243	male	53	1.36	0.82	0.00	0.00	0.00	0.00	0.55

Lisbon	244	male	47	0.00	0.00	0.27	0.27	0.00	0.00	0.00
Lisbon	245	male	65	2.00	1.91	0.00	0.00	0.00	0.00	1.25
Lisbon	246	male	81	1.82	1.45	1.64	1.45	0.18	0.18	1.36
Lisbon	247	male	67	2.00	2.27	1.36	0.82	0.00	0.00	1.25
Lisbon	248	male	48	0.09	0.09	0.00	0.00	0.00	0.00	0.00
Lisbon	249	male	37	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	250	male	47	0.82	0.73	0.00	0.00	0.00	0.00	0.44
Lisbon	251	male	30	0.11	0.11	0.00	0.00	0.00	0.00	0.07
Lisbon	252	male	46	1.45	1.36	0.00	0.00	0.18	0.18	0.76
Lisbon	253	male	64	2.36	2.00	0.00	0.00	0.00	0.00	1.58
Lisbon	254	male	49	1.09	1.00	0.00	0.00	0.00	0.00	0.65
Lisbon	255	male	68	1.73	1.18	0.64	0.36	0.00	0.00	1.09
Lisbon	256	male	53	1.50	1.00	0.00	0.00	0.00	0.00	0.90
Lisbon	257	male	58	2.00	1.78	0.56	0.44	0.33	0.11	1.13
Lisbon	258	male	63	2.55	2.27	0.82	0.45	0.18	0.18	1.64
Lisbon	259	male	68	1.82	1.18	1.45	0.64	0.18	0.18	1.09
Lisbon	260	male	52	0.36	0.45	0.36	0.36	0.00	0.00	0.27
Lisbon	261	male	40	0.18	0.18	0.00	0.00	0.00	0.00	0.16
Lisbon	262	male	50	1.27	0.91	0.09	0.09	0.00	0.00	0.55
Lisbon	263	male	82	1.82	1.91	0.82	0.55	0.00	0.00	1.20
Lisbon	264	male	75	1.64	1.18	0.00	0.00	0.00	0.00	1.04
Lisbon	265	male	20	0.18	0.09	0.00	0.00	0.00	0.00	0.16
Lisbon	266	male	40	0.50	0.50	0.45	0.27	0.00	0.00	0.30
Lisbon	267	male	43	0.27	0.27	0.00	0.00	0.00	0.00	0.16
Lisbon	268	male	30	1.11	0.56	0.11	0.11	0.00	0.00	0.73
Lisbon	269	male	69	2.00	1.55	0.91	0.82	0.09	0.09	1.42
Lisbon	270	male	57	2.00	1.55	0.36	0.18	0.00	0.00	1.31
Lisbon	271	male	29	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	272	male	83	3.55	1.82	0.80	0.40	1.00	0.40	1.98
Lisbon	273	male	38	0.45	0.36	0.00	0.00	0.00	0.00	0.27
Lisbon	274	male	72	1.55	1.00	0.27	0.27	0.00	0.00	0.93
Lisbon	275	male	31	0.09	0.09	0.00	0.00	0.00	0.00	0.05
Lisbon	276	male	47	0.27	0.18	0.00	0.00	0.00	0.00	0.16
Lisbon	277	male	84	2.64	1.45	0.73	0.45	0.55	0.27	1.53
Lisbon	278	male	21	0.09	0.09	0.00	0.00	0.00	0.00	0.05
Lisbon	279	male	71	2.36	1.82	1.33	1.11	1.33	0.44	1.42
Lisbon	280	male	23	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	281	male	49	1.36	1.00	0.09	0.09	0.00	0.00	0.82
Lisbon	282	male	68	2.27	1.45	0.73	0.45	0.18	0.18	1.20
Lisbon	283	male	55	2.13	1.88	1.45	0.91	0.00	0.00	1.58
Lisbon	284	male	28	0.00	0.00	0.00	0.00	0.00	0.00	0.00

Lisbon	285	male	49	1.00	0.73	0.09	0.09	0.00	0.00	0.71
Lisbon	286	male	53	1.73	1.00	0.73	0.73	0.00	0.00	1.04
Lisbon	287	male	45	0.63	0.50	0.55	0.45	0.00	0.00	0.30
Lisbon	288	male	62	1.18	1.00	0.27	0.18	0.00	0.00	0.76
Lisbon	289	male	88	1.36	1.00	0.18	0.18	0.55	0.18	0.93
Lisbon	290	male	82	2.91	2.00	1.18	1.00	1.09	0.36	1.80

C. Pelvis OA scores

Collection	Calce_ID	Sex	Age	LIP_S_ PELVIS	LIP_D_ PELVIS	POR_S_ PELVIS	POR_D_ PELVIS	EBR_S_ PELVIS	EBR_D_ PELVIS	OPH_ PELVIS
Sassari	1	male	60	1.00	0.80	0.60	0.60	0.20	0.20	0.60
Sassari	2	male	62	1.40	1.00	1.80	1.00	0.00	0.00	1.40
Sassari	3	male	60	2.00	2.20	1.00	0.80	0.20	0.20	0.80
Sassari	4	male	25	0.80	1.00	0.80	0.40	0.00	0.00	0.00
Sassari	5	male	75	1.60	1.60	1.00	1.00	0.00	0.00	0.60
Sassari	6	male	73	1.60	2.20	1.60	1.20	0.00	0.00	0.80
Sassari	7	male	82	2.20	2.80	1.20	1.60	0.20	0.40	1.60
Sassari	8	male	57	1.60	1.60	0.80	1.00	0.00	0.00	0.40
Sassari	9	male	26	1.40	1.20	0.40	0.40	0.20	0.20	0.20
Sassari	10	male	50	1.20	1.00	1.20	1.40	0.20	0.20	0.80
Sassari	11	male	26	0.40	0.40	0.40	0.40	0.60	0.60	0.20
Sassari	12	male	50	1.60	1.40	1.20	1.80	0.20	0.20	0.60
Sassari	13	male	21	0.40	0.40	0.20	0.20	0.40	0.40	0.00
Sassari	14	male	35	1.20	1.60	0.80	1.00	0.20	0.40	0.60
Sassari	15	male	58	1.60	1.20	1.40	1.60	0.20	0.20	0.80
Sassari	16	male	58	1.40	1.20	1.40	1.20	0.20	0.20	1.20
Sassari	17	male	53	1.00	0.60	1.60	1.60	0.40	0.40	1.00
Sassari	18	male	37	0.80	0.60	0.20	0.20	0.20	0.20	0.20
Sassari	19	male	40	1.40	0.80	1.40	1.00	0.00	0.00	0.20
Sassari	20	male	64	1.80	1.80	1.80	1.80	0.00	0.00	1.40
Sassari	21	male	69	2.00	2.40	1.80	1.20	0.40	0.40	2.20
Sassari	22	male	70	1.80	1.80	1.40	1.40	0.00	0.00	1.20
Sassari	23	male	78	2.67	2.67	3.00	1.67	1.33	1.00	1.67
Sassari	24	male	22	0.25	0.25	0.75	0.50	0.00	0.00	0.25
Sassari	25	male	55	1.60	1.60	0.40	0.40	0.00	0.00	1.00
Sassari	26	male	77	2.00	1.80	1.60	1.20	0.00	0.00	1.60
Sassari	27	male	43	1.60	1.40	1.20	0.80	0.00	0.00	1.00
Sassari	28	male	20	0.60	0.20	0.40	0.40	0.00	0.00	0.20
Sassari	29	male	41	1.60	1.40	1.00	1.00	0.20	0.20	1.00
Sassari	30	male	60	2.40	1.80	2.20	1.60	0.00	0.00	1.60
Sassari	31	male	80	2.00	3.00	2.60	2.00	0.00	0.00	2.00
Sassari	32	male	40	1.80	1.80	0.80	0.80	0.00	0.00	1.00
Sassari	33	male	40	1.80	1.20	0.80	0.80	0.00	0.00	0.60
Sassari	34	male	48	2.00	1.60	1.20	1.20	0.00	0.00	1.20
Sassari	35	male	53	2.00	1.40	0.60	0.60	0.00	0.00	0.80
Sassari	36	male	66	1.80	1.40	0.80	0.80	0.00	0.00	1.80
Sassari	37	male	66	1.80	1.60	1.40	0.80	0.00	0.00	1.60

Sassari	38	male	56	2.00	1.40	1.00	1.00	0.00	0.00	1.20
Sassari	39	male	67	1.20	1.00	0.20	0.20	0.00	0.00	0.80
Sassari	40	male	76	1.80	1.60	1.60	1.00	0.00	0.00	1.60
Sassari	41	male	61	2.00	1.80	2.20	1.60	0.00	0.00	1.40
Sassari	42	male	29	0.80	0.80	0.80	0.40	0.00	0.00	0.40
Sassari	43	male	36	1.00	1.00	0.80	0.60	0.00	0.00	0.40
Sassari	44	male	31	0.60	0.40	0.80	0.80	0.00	0.00	0.20
Sassari	45	male	69	2.40	2.20	2.60	2.40	0.80	0.80	1.40
Sassari	46	male	80	1.60	1.20	1.00	0.60	0.00	0.00	0.20
Sassari	47	male	78	2.60	2.00	2.40	1.60	0.40	0.40	2.00
Sassari	48	male	60	1.00	0.60	0.20	0.00	0.00	0.00	0.20
Sassari	49	male	55	1.00	0.80	0.40	0.40	0.00	0.00	0.20
Sassari	50	male	50	1.80	1.20	1.00	1.00	0.00	0.00	1.00
Sassari	51	male	55	2.00	2.20	1.60	1.40	0.00	0.00	1.80
Sassari	52	male	21	0.60	0.40	0.20	0.00	0.00	0.00	0.00
Sassari	53	female	78	2.00	1.80	2.20	1.40	0.00	0.00	1.60
Sassari	54	female	57	2.00	1.40	0.60	0.60	0.00	0.00	1.20
Sassari	55	female	98	2.00	1.60	1.00	0.60	0.00	0.00	1.20
Sassari	56	female	80	1.60	0.80	2.00	1.00	0.00	0.00	1.00
Sassari	57	female	54	0.80	0.80	1.60	0.80	0.00	0.00	0.60
Sassari	58	female	70	2.20	1.60	2.60	1.60	0.00	0.00	2.00
Sassari	59	female	68	2.00	1.40	2.60	1.60	0.00	0.00	1.40
Sassari	60	female	59	2.00	1.60	0.80	0.40	0.00	0.00	1.20
Sassari	61	female	75	1.00	1.00	0.80	0.80	0.00	0.00	0.80
Sassari	62	female	60	2.20	1.60	1.20	1.00	0.00	0.00	1.20
Sassari	63	female	63	1.60	1.60	0.80	1.20	0.00	0.00	0.40
Sassari	64	female	22	0.20	0.20	0.00	0.00	0.00	0.00	0.00
Sassari	65	female	17	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Sassari	66	female	78	1.80	1.60	0.60	1.20	0.00	0.00	1.40
Sassari	67	female	20	0.40	0.20	0.00	0.00	0.00	0.00	0.00
Sassari	68	female	70	1.60	1.20	1.00	1.20	0.00	0.00	1.00
Sassari	69	female	88	2.80	2.60	2.80	2.40	0.00	0.00	1.80
Sassari	70	female	58	1.40	1.80	2.20	1.60	0.00	0.00	1.40
Sassari	71	female	54	1.60	1.60	1.20	0.80	0.00	0.00	1.60
Sassari	72	female	80	1.80	2.00	2.20	1.60	0.00	0.00	1.20
Sassari	73	female	45	2.25	1.50	1.25	1.25	0.00	0.00	1.00
Sassari	74	female	48	1.00	0.80	0.60	0.60	0.00	0.00	0.00
Sassari	75	female	42	1.20	1.00	0.20	0.40	0.00	0.00	0.00
Sassari	76	female	48	2.00	1.20	1.20	0.80	0.00	0.00	0.40
Sassari	77	female	70	1.80	1.40	2.40	1.60	0.00	0.00	1.20
Sassari	78	female	78	2.20	2.20	1.40	1.20	0.00	0.00	0.60

Sassari	80	female	39	1.40	1.00	0.80	0.80	0.00	0.00	0.20
Sassari	81	female	50	1.40	1.40	0.80	0.60	0.00	0.00	0.20
Sassari	82	female	34	0.20	0.20	0.40	0.40	0.00	0.00	0.00
Sassari	83	female	48	1.40	1.40	0.40	0.40	0.00	0.00	0.00
Sassari	84	female	50	2.00	1.40	0.40	0.60	0.00	0.00	0.20
Sassari	85	female	28	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Sassari	86	female	35	0.80	0.80	0.20	0.20	0.00	0.00	0.20
Sassari	87	female	50	2.00	1.40	1.80	1.40	0.00	0.00	1.40
Sassari	88	female	70	2.00	2.00	2.60	2.00	0.00	0.00	1.00
Sassari	89	female	75	2.20	1.80	2.00	1.60	0.00	0.00	1.80
Sassari	90	female	53	1.80	1.60	1.20	1.20	0.00	0.00	0.20
Sassari	91	female	32	1.00	0.60	1.20	1.00	0.00	0.00	0.40
Sassari	92	female	34	1.00	0.60	0.40	0.80	0.00	0.00	0.00
Sassari	93	female	38	1.40	1.00	0.60	0.60	0.00	0.00	0.00
Sassari	94	female	47	1.80	1.00	0.40	0.20	0.00	0.00	0.40
Sassari	95	female	45	1.40	1.00	0.20	0.20	0.00	0.00	0.40
Sassari	96	female	40	1.00	1.00	0.40	0.40	0.00	0.00	0.20
Sassari	97	female	32	0.80	0.60	0.20	0.40	0.00	0.00	0.00
Sassari	98	female	25	0.40	0.40	0.60	0.40	0.00	0.00	0.00
Sassari	99	female	70	2.40	1.80	1.40	1.40	0.00	0.00	0.60
Sassari	100	female	24	1.00	0.80	0.20	0.40	0.00	0.00	0.00
Sassari	101	female	62	1.60	1.20	1.40	1.20	0.00	0.00	0.00
Sassari	102	female	90	2.00	1.60	1.00	1.20	0.00	0.00	0.00
Athens	103	female	79	2.00	1.80	2.40	1.60	0.40	0.00	1.00
Athens	104	female	84	2.00	2.00	3.00	2.40	0.40	0.20	0.60
Athens	105	female	67	2.00	2.20	1.80	2.20	0.00	0.00	1.00
Athens	106	female	49	0.80	0.80	0.80	1.00	0.00	0.00	0.00
Athens	107	female	66	1.40	0.80	0.80	1.00	0.00	0.00	0.00
Athens	108	female	44	0.40	0.60	1.60	1.20	0.00	0.00	0.80
Athens	109	female	46	0.80	0.60	0.60	0.80	0.00	0.00	0.00
Athens	110	female	44	1.60	1.20	0.60	1.20	0.00	0.00	0.60
Athens	111	female	27	0.80	0.60	0.40	0.40	0.00	0.00	0.20
Athens	112	female	81	1.80	1.60	1.60	1.40	0.00	0.00	0.20
Athens	113	female	72	1.40	1.20	0.60	0.80	0.00	0.00	0.40
Athens	114	female	54	1.40	1.20	0.60	0.20	0.00	0.00	0.00
Athens	115	female	48	0.60	0.40	0.20	0.20	0.00	0.00	0.00
Athens	116	female	54	1.00	0.60	1.00	0.60	0.00	0.00	0.60
Athens	117	female	51	0.80	0.60	0.60	0.40	0.00	0.00	0.00
Athens	118	female	72	2.60	1.60	0.60	0.60	0.00	0.00	1.00
Athens	119	female	37	0.40	0.40	0.40	0.40	0.00	0.00	0.00
Athens	120	female	47	0.75	0.75	0.25	0.25	0.00	0.00	0.00

Athens	121	female	82	1.20	1.20	1.20	1.20	0.00	0.00	1.20
Athens	122	female	33	1.20	1.00	0.40	0.40	0.00	0.00	0.40
Athens	123	female	65	0.40	0.40	0.80	0.60	0.00	0.00	0.40
Athens	124	female	85	1.40	1.00	2.00	1.60	0.00	0.00	0.80
Athens	125	female	45	0.80	0.60	0.20	0.20	0.00	0.00	0.40
Athens	126	female	78	1.20	1.00	1.00	1.00	0.00	0.00	0.00
Athens	127	female	27	0.00	0.00	0.20	0.20	0.00	0.00	0.00
Athens	128	female	77	1.40	1.00	1.60	1.80	0.00	0.00	0.00
Athens	129	female	46	1.00	0.80	0.60	0.60	0.00	0.00	0.00
Athens	130	female	69	2.00	1.40	1.40	1.40	0.00	0.00	1.20
Athens	131	female	56	2.20	1.60	1.60	1.80	0.00	0.00	0.80
Athens	132	female	62	1.80	1.40	1.40	1.40	0.00	0.00	0.00
Athens	133	female	52	1.20	1.00	1.60	1.00	0.00	0.00	0.40
Athens	134	female	60	2.20	2.00	1.80	1.80	0.00	0.00	0.60
Athens	135	female	63	1.20	1.00	1.00	1.00	0.00	0.00	0.00
Athens	136	female	82	2.60	2.00	1.20	1.60	0.00	0.00	0.00
Athens	137	female	87	1.60	1.20	3.00	1.80	0.00	0.00	0.40
Athens	138	female	59	1.20	0.80	1.20	0.80	0.00	0.00	0.40
Athens	139	female	73	1.40	1.20	2.20	1.80	0.00	0.00	0.00
Athens	140	female	84	1.40	1.00	1.40	1.60	0.00	0.00	0.60
Athens	141	female	81	1.20	1.20	1.40	1.80	0.00	0.00	0.00
Athens	142	female	79	1.60	1.40	1.20	0.80	0.00	0.00	0.80
Athens	143	female	69	1.60	1.20	2.00	1.60	0.00	0.00	0.00
Athens	144	female	99	1.60	1.60	1.20	2.00	0.00	0.00	0.00
Athens	145	female	49	0.80	0.80	1.40	1.00	0.00	0.00	0.20
Athens	146	female	38	1.00	0.60	1.00	0.60	0.00	0.00	0.00
Athens	147	female	20	0.20	0.20	0.60	0.60	0.00	0.00	0.00
Athens	148	female	35	0.20	0.20	0.60	0.60	0.00	0.00	0.20
Athens	149	female	50	1.50	1.00	0.75	0.75	0.00	0.00	0.00
Athens	150	female	24	0.80	0.60	0.80	0.80	0.00	0.00	0.00
Athens	151	female	22	0.40	0.40	0.00	0.00	0.00	0.00	0.00
Athens	152	female	63	2.00	1.20	0.40	0.40	0.00	0.00	0.00
Athens	153	male	29	0.40	0.20	0.40	0.40	0.00	0.00	0.40
Athens	154	male	33	0.80	0.40	0.60	0.40	0.00	0.00	0.00
Athens	155	male	32	1.00	0.80	0.80	0.80	0.00	0.00	0.00
Athens	156	male	26	0.80	0.60	1.00	0.60	0.00	0.00	0.00
Athens	157	male	55	1.60	1.40	1.60	1.20	0.00	0.00	0.60
Athens	158	male	94	1.80	1.60	1.80	1.00	0.00	0.00	0.40
Athens	159	male	73	1.80	2.20	1.20	1.00	0.00	0.00	0.80
Athens	160	male	66	1.60	1.40	1.40	0.80	0.00	0.00	0.00
Athens	161	male	58	1.40	1.00	0.80	0.80	0.00	0.00	0.20

Athens	162	male	88	1.40	1.60	0.60	0.40	0.00	0.00	0.40
Athens	163	male	85	1.40	1.00	1.40	1.20	0.00	0.00	0.00
Athens	164	male	61	1.20	0.80	1.20	1.00	0.00	0.00	0.00
Athens	165	male	81	2.00	1.40	1.60	1.60	0.00	0.00	0.60
Athens	166	male	66	2.20	1.40	1.00	1.00	0.00	0.00	1.00
Athens	167	male	55	1.00	1.00	0.60	0.80	0.00	0.00	0.00
Athens	168	male	84	2.20	1.40	1.20	1.40	0.00	0.00	1.20
Athens	169	male	84	2.20	1.40	1.00	0.80	0.00	0.00	0.00
Athens	170	male	50	1.40	1.20	0.60	0.40	0.00	0.00	0.40
Athens	171	male	61	1.80	2.20	2.40	1.40	0.00	0.00	1.60
Athens	172	male	72	0.80	0.80	0.80	0.80	0.00	0.00	0.60
Athens	173	male	29	0.20	0.20	0.40	0.40	0.00	0.00	0.00
Athens	174	male	68	3.00	1.80	1.80	1.40	0.00	0.00	0.60
Athens	175	male	24	0.60	0.40	0.60	0.60	0.00	0.00	0.00
Athens	176	male	50	0.80	1.00	1.40	1.20	0.00	0.00	0.00
Athens	177	male	49	2.60	1.20	1.00	1.00	0.00	0.00	0.60
Athens	178	male	56	1.00	0.80	1.60	0.80	0.00	0.00	0.20
Athens	179	male	27	0.00	0.00	0.40	0.40	0.00	0.00	0.00
Athens	180	male	48	1.40	1.00	0.60	0.60	0.00	0.00	0.40
Athens	181	male	29	0.00	0.00	0.40	0.40	0.00	0.00	0.40
Athens	182	male	48	0.80	0.60	0.00	0.00	0.00	0.00	0.40
Athens	183	male	75	2.00	1.40	2.00	0.80	0.00	0.00	0.40
Athens	184	male	34	0.60	0.40	0.80	0.80	0.00	0.00	0.00
Athens	185	male	65	1.60	1.20	2.00	1.00	0.00	0.00	1.20
Athens	186	male	48	1.40	0.80	0.80	1.00	0.00	0.00	0.20
Athens	187	male	94	1.80	1.20	2.00	1.40	0.00	0.00	0.40
Athens	188	male	67	1.20	0.80	0.60	1.00	0.00	0.00	0.60
Athens	189	male	76	1.20	1.20	1.80	0.80	0.00	0.00	1.20
Athens	190	male	36	0.60	0.60	0.60	0.40	0.00	0.00	0.00
Athens	191	male	64	1.20	1.20	1.20	1.20	0.00	0.00	0.60
Athens	192	male	55	1.20	1.00	0.80	0.40	0.00	0.00	0.00
Athens	193	male	39	1.80	1.60	1.80	1.80	0.80	0.80	1.20
Athens	194	male	58	1.80	1.20	1.20	0.80	0.00	0.00	0.60
Athens	195	male	76	1.60	1.40	1.80	1.40	0.00	0.00	0.80
Athens	196	male	60	0.60	0.60	0.60	0.60	0.00	0.00	0.20
Lisbon	197	female	78	1.40	1.40	1.20	1.20	0.00	0.00	1.20
Lisbon	198	female	18	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	199	female	54	0.80	0.60	0.20	0.20	0.00	0.00	0.80
Lisbon	200	female	62	0.40	0.40	0.20	0.40	0.00	0.00	0.00
Lisbon	201	female	19	0.20	0.40	0.20	0.20	0.00	0.00	0.00
Lisbon	202	female	56	1.00	1.00	1.20	0.60	0.00	0.00	0.20

Lisbon	203	female	66	0.40	0.40	0.20	0.20	0.00	0.00	0.00
Lisbon	204	female	48	1.00	0.60	0.00	0.00	0.00	0.00	0.40
Lisbon	205	female	94	2.00	1.80	1.00	0.60	0.00	0.00	0.40
Lisbon	206	female	67	1.40	1.40	1.00	1.40	0.00	0.00	0.00
Lisbon	207	female	59	0.60	0.60	0.20	0.20	0.00	0.00	0.00
Lisbon	208	female	76	0.60	1.00	0.40	0.20	0.00	0.00	0.20
Lisbon	209	female	28	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	210	female	35	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	211	female	76	0.80	0.80	0.40	0.40	0.00	0.00	0.00
Lisbon	212	female	23	0.20	0.20	0.00	0.00	0.00	0.00	0.00
Lisbon	213	female	44	0.60	0.40	0.40	0.20	0.00	0.00	0.00
Lisbon	214	female	85	1.40	1.60	0.40	0.40	0.00	0.00	0.40
Lisbon	215	female	59	0.60	1.20	0.40	0.20	0.00	0.00	0.40
Lisbon	216	female	20	0.20	0.20	0.00	0.00	0.00	0.00	0.00
Lisbon	217	female	29	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	218	female	50	0.60	0.60	0.00	0.00	0.00	0.00	0.00
Lisbon	219	female	27	0.20	0.20	0.00	0.00	0.00	0.00	0.00
Lisbon	220	female	93	1.80	1.60	1.20	0.60	0.00	0.00	0.00
Lisbon	221	female	39	0.40	0.40	0.80	0.60	0.00	0.00	0.00
Lisbon	222	female	51	0.40	0.40	0.00	0.00	0.00	0.00	0.00
Lisbon	223	female	43	0.60	0.60	0.00	0.00	0.00	0.00	0.00
Lisbon	224	female	68	0.40	0.60	1.40	0.80	0.00	0.00	0.40
Lisbon	225	female	48	0.40	0.60	0.00	0.00	0.00	0.00	0.00
Lisbon	226	female	44	0.40	0.40	0.00	0.00	0.00	0.00	0.00
Lisbon	227	female	46	0.60	0.60	0.20	0.20	0.00	0.00	0.40
Lisbon	228	female	32	0.20	0.20	0.40	0.20	0.00	0.00	0.00
Lisbon	229	female	35	0.20	0.20	0.00	0.00	0.00	0.00	0.20
Lisbon	230	female	74	1.00	1.20	0.00	0.00	0.00	0.00	0.60
Lisbon	231	female	24	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	232	female	73	0.80	0.80	0.40	0.60	0.00	0.00	0.80
Lisbon	233	female	53	1.80	1.00	1.00	0.40	0.00	0.00	1.20
Lisbon	234	female	56	0.40	0.40	0.60	0.40	0.00	0.00	0.40
Lisbon	235	female	57	1.40	1.40	1.00	1.20	0.00	0.00	0.40
Lisbon	236	female	52	1.00	1.80	1.40	1.40	0.00	0.00	0.40
Lisbon	237	female	59	1.00	1.00	0.40	0.20	0.00	0.00	0.00
Lisbon	238	female	83	1.20	1.40	1.20	1.20	0.00	0.00	1.00
Lisbon	239	female	77	2.00	1.80	1.80	1.80	0.00	0.00	1.00
Lisbon	240	female	86	2.00	1.80	1.80	1.20	0.00	0.00	1.20
Lisbon	241	female	82	2.00	2.00	2.60	1.60	0.40	0.40	0.20
Lisbon	242	female	90	2.40	1.40	1.40	1.00	0.00	0.00	0.80
Lisbon	243	male	53	0.60	0.60	0.20	0.20	0.00	0.00	0.40

Lisbon	244	male	47	0.60	0.60	0.40	0.20	0.00	0.00	0.40
Lisbon	245	male	65	0.75	0.75	0.50	0.25	0.00	0.00	0.00
Lisbon	246	male	81	1.40	1.20	2.00	1.20	0.00	0.00	0.80
Lisbon	247	male	67	1.00	0.40	0.20	0.20	0.00	0.00	0.60
Lisbon	248	male	48	0.40	0.40	0.20	0.20	0.40	0.20	0.60
Lisbon	249	male	37	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	250	male	47	0.60	0.40	0.40	0.20	0.00	0.00	0.00
Lisbon	251	male	30	0.20	0.20	0.20	0.20	0.00	0.00	0.00
Lisbon	252	male	46	1.20	1.20	0.20	0.20	0.00	0.00	0.40
Lisbon	253	male	64	1.00	1.00	0.60	0.60	0.00	0.00	0.40
Lisbon	254	male	49	0.80	0.60	0.40	0.40	0.00	0.00	0.40
Lisbon	255	male	68	0.40	0.20	0.40	0.20	0.00	0.00	1.20
Lisbon	256	male	53	0.60	0.60	0.00	0.00	0.00	0.00	0.00
Lisbon	257	male	58	0.40	0.60	0.20	0.20	0.00	0.00	0.00
Lisbon	258	male	63	1.00	1.00	0.60	0.40	0.00	0.00	1.20
Lisbon	259	male	68	0.40	0.40	0.40	0.40	0.00	0.00	0.40
Lisbon	260	male	52	0.60	0.40	0.60	0.60	0.00	0.00	0.20
Lisbon	261	male	40	0.80	0.80	0.40	0.40	0.00	0.00	0.20
Lisbon	262	male	50	1.80	1.00	1.20	0.80	0.00	0.00	0.80
Lisbon	263	male	82	1.40	1.60	1.20	0.60	0.00	0.00	0.40
Lisbon	264	male	75	1.60	1.20	1.00	0.80	0.00	0.00	0.80
Lisbon	265	male	20	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	266	male	40	0.60	0.60	0.00	0.00	0.00	0.00	0.20
Lisbon	267	male	43	0.80	0.40	0.00	0.00	0.00	0.00	0.00
Lisbon	268	male	30	0.40	0.40	0.20	0.20	0.00	0.00	0.20
Lisbon	269	male	69	1.40	1.20	1.40	1.20	0.00	0.00	1.00
Lisbon	270	male	57	0.60	0.80	0.20	0.20	0.00	0.00	0.20
Lisbon	271	male	29	0.60	0.60	0.20	0.20	0.00	0.00	0.20
Lisbon	272	male	83	1.25	1.00	1.00	0.75	0.00	0.00	1.50
Lisbon	273	male	38	0.40	0.80	0.60	0.40	0.00	0.00	0.20
Lisbon	274	male	72	1.00	1.00	0.20	0.20	0.00	0.00	0.20
Lisbon	275	male	31	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	276	male	47	0.40	0.40	0.00	0.00	0.00	0.00	0.20
Lisbon	277	male	84	1.40	0.80	1.00	1.20	0.00	0.00	0.40
Lisbon	278	male	21	0.20	0.20	0.40	0.40	0.00	0.00	0.00
Lisbon	279	male	71	1.40	1.20	0.20	0.20	0.00	0.00	0.20
Lisbon	280	male	23	0.40	0.40	0.00	0.00	0.00	0.00	0.00
Lisbon	281	male	49	1.20	0.80	0.40	0.40	0.00	0.00	0.40
Lisbon	282	male	68	1.60	1.00	1.20	0.80	0.00	0.00	0.00
Lisbon	283	male	55	0.80	0.60	0.00	0.00	0.00	0.00	0.20
Lisbon	284	male	28	0.20	0.20	0.00	0.00	0.00	0.00	0.00

Lisbon	285	male	49	1.40	1.20	0.40	0.20	0.00	0.00	0.40
Lisbon	286	male	53	0.60	0.40	0.40	0.40	0.00	0.00	0.00
Lisbon	287	male	45	0.60	0.60	0.20	0.20	0.00	0.00	0.00
Lisbon	288	male	62	1.00	0.80	0.40	0.40	0.00	0.00	0.00
Lisbon	289	male	88	0.60	0.40	1.20	0.80	0.00	0.00	0.00
Lisbon	290	male	82	1.00	0.80	0.80	0.80	0.00	0.00	0.40

D. Knee OA scores

Collection	Calce_ID	Sex	Age	LIP_S_ KNEE	LIP_D_ KNEE	POR_S_ KNEE	POR_D_ KNEE	EBR_S_ KNEE	EBR_D_ KNEE	OPH_ KNEE
Sassari	1	male	60	0.33	0.33	0.00	0.00	0.00	0.00	0.33
Sassari	2	male	62	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Sassari	3	male	60	1.50	2.50	1.50	1.00	0.50	0.50	1.00
Sassari	4	male	25	0.33	0.33	0.00	0.00	0.33	0.33	0.00
Sassari	5	male	75	2.00	2.67	0.33	0.33	0.33	0.33	0.67
Sassari	6	male	73	0.33	0.33	1.00	1.00	0.00	0.00	0.00
Sassari	7	male	82	1.33	1.33	0.67	0.67	0.33	0.67	0.00
Sassari	8	male	57	1.67	1.33	0.67	0.67	0.67	0.33	0.33
Sassari	9	male	26	1.00	1.33	0.00	0.00	0.00	0.00	0.00
Sassari	10	male	50	1.00	1.33	0.00	0.00	0.33	0.33	0.33
Sassari	11	male	26	0.00	0.00	0.00	0.00	0.67	0.67	0.00
Sassari	12	male	50	0.67	0.67	0.00	0.00	0.67	0.67	0.00
Sassari	13	male	21	0.33	0.33	0.33	0.33	0.33	0.33	0.00
Sassari	14	male	35	1.50	1.00	0.00	0.00	1.00	1.00	0.00
Sassari	15	male	58	1.00	1.00	0.67	0.67	0.33	0.33	0.33
Sassari	16	male	58	1.33	0.67	0.00	0.00	1.00	1.00	0.00
Sassari	17	male	53	1.00	1.00	1.00	1.00	1.00	1.00	0.00
Sassari	18	male	37	0.33	0.33	0.00	0.00	0.67	0.67	0.00
Sassari	19	male	40	0.33	0.33	0.33	0.33	0.00	0.00	0.00
Sassari	20	male	64	1.00	1.00	0.33	0.33	1.00	0.67	0.00
Sassari	21	male	69	2.00	2.00	0.33	0.33	0.33	0.33	1.33
Sassari	22	male	70	2.00	2.00	1.67	1.33	0.33	0.33	1.00
Sassari	23	male	78	1.00	0.67	0.67	0.67	0.00	0.00	0.33
Sassari	24	male	22	0.00	0.00	0.33	0.33	0.00	0.00	0.00
Sassari	25	male	55	0.33	0.33	0.33	0.33	0.33	0.33	0.67
Sassari	26	male	77	2.00	1.67	0.67	1.33	0.00	0.00	1.33
Sassari	27	male	43	1.50	1.00	0.00	0.00	0.00	0.00	0.50
Sassari	28	male	20	0.67	0.67	0.67	0.67	0.00	0.00	0.00
Sassari	29	male	41	1.33	1.00	0.67	0.67	0.00	0.00	0.00
Sassari	30	male	60	2.33	2.67	0.33	0.33	0.33	0.33	1.33
Sassari	31	male	80	1.00	1.33	0.67	0.67	0.00	0.00	0.67
Sassari	32	male	40	1.33	1.67	0.00	0.00	0.00	0.00	0.33
Sassari	33	male	40	1.33	1.67	0.33	0.33	0.00	0.00	0.00
Sassari	34	male	48	2.00	2.00	0.33	0.33	0.33	0.33	1.67
Sassari	35	male	53	2.00	1.00	0.00	0.00	0.00	0.00	0.00
Sassari	36	male	66	0.33	0.33	0.00	0.00	0.00	0.00	0.00
Sassari	37	male	66	1.00	1.00	0.67	1.00	0.00	0.00	0.33

Sassari	38	male	56	2.00	2.00	0.33	0.67	0.00	0.00	0.00
Sassari	39	male	67	1.67	1.33	0.67	0.67	0.00	0.00	0.33
Sassari	40	male	76	1.00	0.67	0.00	0.00	0.00	0.00	0.00
Sassari	41	male	61	1.00	0.67	0.00	0.00	0.00	0.00	0.00
Sassari	42	male	29	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Sassari	43	male	36	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Sassari	44	male	31	1.00	0.50	0.00	0.00	0.00	0.00	0.00
Sassari	45	male	69	2.00	1.67	0.33	0.33	0.00	0.00	1.00
Sassari	46	male	80	1.33	1.00	0.00	0.00	0.00	0.00	0.00
Sassari	47	male	78	1.33	1.67	0.33	1.00	0.00	0.00	0.33
Sassari	48	male	60	0.00	0.00	0.33	0.33	0.00	0.00	0.00
Sassari	49	male	55	0.67	0.33	0.00	0.00	0.00	0.00	0.00
Sassari	50	male	50	2.00	2.67	0.33	0.33	0.00	0.00	0.33
Sassari	51	male	55	2.00	2.00	0.00	0.00	0.00	0.00	0.00
Sassari	52	male	21	0.33	0.33	0.00	0.00	0.00	0.00	0.00
Sassari	53	female	78	3.00	3.00	2.00	1.00	0.00	0.00	2.00
Sassari	54	female	57	0.33	0.33	0.00	0.00	0.00	0.00	0.00
Sassari	55	female	98	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Sassari	56	female	80	3.00	2.50	1.00	1.00	3.00	2.00	2.00
Sassari	57	female	54	1.00	1.00	0.00	0.00	0.00	0.00	0.00
Sassari	58	female	70	1.67	1.67	1.00	0.67	0.00	0.00	0.67
Sassari	59	female	68	2.00	1.00	0.67	0.33	0.00	0.00	0.00
Sassari	60	female	59	2.00	2.50	0.00	0.00	0.00	0.00	1.00
Sassari	61	female	75	1.00	1.00	0.00	0.00	0.00	0.00	0.00
Sassari	62	female	60	2.00	2.00	0.00	0.00	0.00	0.00	0.00
Sassari	63	female	63	1.33	0.67	0.33	0.33	0.00	0.00	0.33
Sassari	64	female	22	0.33	0.33	0.00	0.00	0.00	0.00	0.00
Sassari	65	female	17	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Sassari	66	female	78	0.50	0.50	0.00	0.00	0.00	0.00	0.00
Sassari	67	female	20	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Sassari	68	female	70	2.00	1.00	0.00	0.00	0.00	0.00	0.00
Sassari	69	female	88	2.00	2.50	0.50	0.50	0.00	0.00	1.50
Sassari	70	female	58	2.00	2.00	0.67	0.67	0.00	0.00	1.33
Sassari	71	female	54	0.33	0.33	0.00	0.00	0.00	0.00	0.00
Sassari	72	female	80	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Sassari	73	female	45	1.00	0.50	0.00	0.00	0.00	0.00	0.00
Sassari	74	female	48	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Sassari	75	female	42	0.00	0.00	0.67	0.67	0.00	0.00	0.00
Sassari	76	female	48	2.00	1.33	0.00	0.00	0.00	0.00	0.00
Sassari	77	female	70	1.33	1.33	0.00	0.00	0.00	0.00	0.00
Sassari	78	female	78	0.33	0.33	0.00	0.00	0.00	0.00	0.00

Sassari	80	female	39	0.33	0.33	0.00	0.00	0.00	0.00	0.00
Sassari	81	female	50	1.67	2.00	0.00	0.00	0.00	0.00	0.00
Sassari	82	female	34	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Sassari	83	female	48	1.00	0.67	0.00	0.00	0.00	0.00	0.00
Sassari	84	female	50	2.00	1.67	0.00	0.00	0.00	0.00	0.00
Sassari	85	female	28	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Sassari	86	female	35	1.67	2.33	0.33	0.33	0.00	0.00	0.00
Sassari	87	female	50	0.67	1.00	0.00	0.00	0.00	0.00	0.00
Sassari	88	female	70	2.00	2.33	0.67	0.67	0.00	0.00	0.00
Sassari	89	female	75	2.00	2.50	0.33	0.33	0.00	0.00	0.33
Sassari	90	female	53	1.00	1.00	0.00	0.00	0.00	0.00	0.67
Sassari	91	female	32	0.33	0.33	0.00	0.00	0.00	0.00	0.00
Sassari	92	female	34	1.00	1.00	0.00	0.00	0.00	0.00	0.00
Sassari	93	female	38	0.33	0.67	0.00	0.00	0.00	0.00	0.00
Sassari	94	female	47	1.00	0.67	0.00	0.00	0.00	0.00	0.00
Sassari	95	female	45	1.67	1.67	0.33	0.33	0.00	0.00	0.00
Sassari	96	female	40	3.00	2.00	1.00	2.00	0.00	0.00	0.00
Sassari	97	female	32	0.33	0.33	0.00	0.00	0.00	0.00	0.00
Sassari	98	female	25	1.00	0.67	0.00	0.00	0.00	0.00	0.00
Sassari	99	female	70	2.00	1.50	0.00	0.00	0.00	0.00	0.00
Sassari	100	female	24	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Sassari	101	female	62	1.67	1.00	0.00	0.00	0.00	0.00	0.00
Sassari	102	female	90	1.33	1.33	0.00	0.00	0.00	0.00	0.00
Athens	103	female	79	3.00	3.00	1.00	1.00	1.33	0.67	1.33
Athens	104	female	84	3.00	3.00	2.67	1.67	2.00	1.33	2.00
Athens	105	female	67	2.50	2.00	1.00	1.00	0.50	0.50	1.00
Athens	106	female	49	0.33	0.67	0.33	0.33	0.00	0.00	0.00
Athens	107	female	66	1.33	1.00	0.00	0.00	0.00	0.00	0.00
Athens	108	female	44	1.00	0.50	0.50	0.50	0.00	0.00	0.00
Athens	109	female	46	0.33	0.33	0.00	0.00	0.00	0.00	0.00
Athens	110	female	44	1.33	1.67	0.00	0.00	0.00	0.00	0.00
Athens	111	female	27	0.33	0.33	0.00	0.00	0.00	0.00	0.00
Athens	112	female	81	3.00	3.00	1.67	1.00	1.00	1.00	0.67
Athens	113	female	72	1.50	1.00	0.00	0.00	0.00	0.00	0.00
Athens	114	female	54	1.33	1.33	0.00	0.00	0.00	0.00	0.00
Athens	115	female	48	0.00	0.00	0.33	0.33	0.00	0.00	0.00
Athens	116	female	54	0.67	0.67	0.00	0.00	0.00	0.00	0.00
Athens	117	female	51	0.67	0.67	0.00	0.00	0.00	0.00	0.33
Athens	118	female	72	3.00	3.00	0.33	0.33	0.00	0.00	0.67
Athens	119	female	37	0.33	0.33	0.00	0.00	0.00	0.00	0.00
Athens	120	female	47	1.00	1.00	0.00	0.00	0.00	0.00	0.00

Athens	121	female	82	2.00	2.00	0.67	0.67	0.00	0.00	0.67
Athens	122	female	33	0.33	0.33	0.67	0.67	0.00	0.00	0.00
Athens	123	female	65	0.33	0.67	0.00	0.00	0.00	0.00	0.00
Athens	124	female	85	1.00	1.00	1.00	1.00	0.00	0.00	0.50
Athens	125	female	45	0.67	0.33	0.00	0.00	0.00	0.00	0.00
Athens	126	female	78	1.00	0.50	0.00	0.00	0.00	0.00	0.50
Athens	127	female	27	1.50	1.50	0.00	0.00	0.00	0.00	0.00
Athens	128	female	77	2.50	2.50	1.50	0.50	0.00	0.00	1.50
Athens	129	female	46	0.67	1.00	0.00	0.00	0.00	0.00	0.00
Athens	130	female	69	2.00	2.50	1.00	0.50	0.00	0.00	1.00
Athens	131	female	56	3.00	3.00	1.00	0.50	1.50	0.50	0.00
Athens	132	female	62	3.00	3.00	1.33	1.00	0.67	0.67	2.00
Athens	133	female	52	0.50	1.00	0.00	0.00	0.00	0.00	0.00
Athens	134	female	60	2.67	2.67	1.33	1.33	0.00	0.00	1.33
Athens	135	female	63	2.33	2.33	0.67	1.00	0.00	0.00	0.33
Athens	136	female	82	2.67	2.67	1.67	1.33	2.00	1.67	2.00
Athens	137	female	87	3.00	3.00	2.33	1.67	1.33	1.33	2.00
Athens	138	female	59	0.67	1.00	0.33	0.33	0.00	0.00	0.00
Athens	139	female	73	1.00	1.00	0.00	0.00	0.00	0.00	0.00
Athens	140	female	84	1.67	1.67	2.00	1.00	0.00	0.00	0.67
Athens	141	female	81	0.67	1.00	1.00	1.33	0.00	0.00	0.00
Athens	142	female	79	1.50	2.00	0.50	0.50	0.00	0.00	0.50
Athens	143	female	69	1.00	0.67	1.00	1.67	0.00	0.00	0.00
Athens	144	female	99	2.67	2.00	1.00	0.67	0.00	0.00	0.67
Athens	145	female	49	3.00	3.00	1.00	1.00	2.00	2.00	1.50
Athens	146	female	38	0.50	0.50	0.50	0.50	0.00	0.00	0.00
Athens	147	female	20	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Athens	148	female	35	1.00	1.00	0.00	0.00	0.00	0.00	0.00
Athens	149	female	50	1.67	2.00	0.67	0.67	0.00	0.00	0.00
Athens	150	female	24	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Athens	151	female	22	1.00	1.00	0.00	0.00	0.00	0.00	0.00
Athens	152	female	63	0.67	1.00	0.33	0.33	0.00	0.00	0.00
Athens	153	male	29	1.50	1.50	0.00	0.00	0.00	0.00	0.00
Athens	154	male	33	0.67	0.67	0.00	0.00	0.00	0.00	0.00
Athens	155	male	32	0.50	1.00	0.00	0.00	0.00	0.00	0.00
Athens	156	male	26	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Athens	157	male	55	0.33	0.33	0.00	0.00	0.00	0.00	0.00
Athens	158	male	94	1.33	1.00	1.00	1.00	0.00	0.00	0.00
Athens	159	male	73	1.00	1.50	2.00	1.00	0.00	0.00	0.50
Athens	160	male	66	1.67	1.67	1.00	1.00	0.00	0.00	0.00
Athens	161	male	58	2.00	3.00	0.50	0.50	0.00	0.00	1.00

Athens	162	male	88	1.50	2.50	1.00	1.50	0.00	0.00	0.00
Athens	163	male	85	1.67	1.67	1.00	1.00	0.00	0.00	0.67
Athens	164	male	61	1.33	1.33	0.67	0.67	0.00	0.00	0.00
Athens	165	male	81	1.00	1.33	0.33	0.33	0.00	0.00	0.00
Athens	166	male	66	2.00	3.00	0.00	0.00	0.00	0.00	1.00
Athens	167	male	55	2.00	2.00	0.00	0.00	0.00	0.00	0.00
Athens	168	male	84	3.00	3.00	1.00	1.00	1.50	0.50	2.00
Athens	169	male	84	3.00	2.67	1.67	1.33	2.00	1.67	1.33
Athens	170	male	50	1.00	1.50	0.00	0.00	0.00	0.00	0.00
Athens	171	male	61	1.00	2.00	1.00	1.00	0.00	0.00	0.00
Athens	172	male	72	1.00	1.00	0.00	0.00	0.00	0.00	0.00
Athens	173	male	29	0.50	0.50	0.00	0.00	0.00	0.00	0.00
Athens	174	male	68	1.33	2.00	0.67	0.67	0.00	0.00	0.67
Athens	175	male	24	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Athens	176	male	50	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Athens	177	male	49	0.50	0.50	0.50	0.50	0.00	0.00	0.00
Athens	178	male	56	1.50	2.50	0.00	0.00	0.00	0.00	1.00
Athens	179	male	27	0.50	0.50	0.00	0.00	0.00	0.00	0.00
Athens	180	male	48	1.00	1.33	0.00	0.00	0.00	0.00	0.00
Athens	181	male	29	0.33	0.33	0.00	0.00	0.00	0.00	0.00
Athens	182	male	48	1.33	1.67	0.00	0.00	0.00	0.00	0.00
Athens	183	male	75	1.00	1.50	2.00	2.00	0.00	0.00	0.00
Athens	184	male	34	1.00	2.00	0.00	0.00	0.00	0.00	0.00
Athens	185	male	65	1.00	2.50	1.00	1.50	0.00	0.00	0.00
Athens	186	male	48	1.50	2.00	0.00	0.00	0.00	0.00	0.00
Athens	187	male	94	3.00	2.00	1.00	1.00	0.00	0.00	1.00
Athens	188	male	67	1.00	1.50	1.00	1.00	0.00	0.00	0.00
Athens	189	male	76	1.50	1.50	1.00	1.50	0.00	0.00	1.00
Athens	190	male	36	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Athens	191	male	64	0.67	0.67	0.00	0.00	0.00	0.00	0.00
Athens	192	male	55	1.00	1.67	0.33	0.33	0.00	0.00	0.00
Athens	193	male	39	3.00	3.00	3.00	3.00	0.00	0.00	2.00
Athens	194	male	58	0.50	0.50	0.50	0.50	0.00	0.00	0.00
Athens	195	male	76	2.00	3.00	0.33	0.33	0.00	0.00	1.33
Athens	196	male	60	0.50	1.00	0.00	0.00	0.00	0.00	0.50
Lisbon	197	female	78	0.50	0.50	0.50	0.50	0.00	0.00	1.00
Lisbon	198	female	18	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	199	female	54	1.67	2.00	0.00	0.00	0.00	0.00	0.67
Lisbon	200	female	62	0.33	0.33	0.00	0.00	0.00	0.00	0.00
Lisbon	201	female	19	0.33	0.33	0.00	0.00	0.00	0.00	0.00
Lisbon	202	female	56	2.00	3.00	1.33	0.67	0.00	0.00	0.00

Lisbon	203	female	66	0.33	0.33	0.67	0.33	0.00	0.00	0.00
Lisbon	204	female	48	2.00	2.67	0.33	0.33	1.00	0.67	0.00
Lisbon	205	female	94	3.00	3.00	1.33	0.67	2.00	1.33	1.33
Lisbon	206	female	67	2.00	3.00	0.33	0.33	0.00	0.00	0.33
Lisbon	207	female	59	1.00	2.33	0.33	0.33	0.00	0.00	0.00
Lisbon	208	female	76	1.67	1.67	0.00	0.00	0.00	0.00	0.67
Lisbon	209	female	28	1.00	1.00	0.00	0.00	0.00	0.00	0.00
Lisbon	210	female	35	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	211	female	76	1.00	2.67	0.00	0.00	0.00	0.00	0.00
Lisbon	212	female	23	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	213	female	44	0.33	0.33	0.00	0.00	0.00	0.00	0.00
Lisbon	214	female	85	2.67	2.33	0.33	0.33	0.33	0.33	2.00
Lisbon	215	female	59	0.67	1.00	0.00	0.00	0.00	0.00	0.00
Lisbon	216	female	20	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	217	female	29	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	218	female	50	0.00	0.00	0.33	0.33	0.00	0.00	0.00
Lisbon	219	female	27	0.33	0.33	0.00	0.00	0.00	0.00	0.00
Lisbon	220	female	93	2.33	2.67	1.00	1.00	0.00	0.00	1.00
Lisbon	221	female	39	1.00	1.33	0.00	0.00	0.00	0.00	0.00
Lisbon	222	female	51	2.00	2.67	0.00	0.00	0.00	0.00	0.00
Lisbon	223	female	43	1.00	2.00	0.00	0.00	0.00	0.00	0.00
Lisbon	224	female	68	1.00	2.00	0.00	0.00	0.00	0.00	0.33
Lisbon	225	female	48	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	226	female	44	0.33	0.33	0.00	0.00	0.00	0.00	0.33
Lisbon	227	female	46	1.00	1.00	0.00	0.00	0.00	0.00	0.00
Lisbon	228	female	32	1.33	2.00	0.00	0.00	0.00	0.00	0.00
Lisbon	229	female	35	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	230	female	74	0.33	0.33	0.00	0.00	0.00	0.00	0.00
Lisbon	231	female	24	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	232	female	73	1.67	2.33	0.00	0.00	0.00	0.00	0.00
Lisbon	233	female	53	0.67	1.00	0.00	0.00	0.00	0.00	0.00
Lisbon	234	female	56	1.00	1.33	0.33	0.33	0.00	0.00	0.67
Lisbon	235	female	57	0.67	0.67	0.00	0.00	0.00	0.00	0.00
Lisbon	236	female	52	2.00	1.00	0.00	0.00	0.00	0.00	0.00
Lisbon	237	female	59	2.00	2.67	0.67	0.67	0.00	0.00	0.33
Lisbon	238	female	83	1.00	2.00	0.33	0.33	0.00	0.00	0.33
Lisbon	239	female	77	2.67	2.00	1.00	0.33	1.33	0.67	0.33
Lisbon	240	female	86	3.00	2.67	0.67	0.67	2.00	1.67	0.67
Lisbon	241	female	82	2.33	2.67	2.00	1.00	1.00	0.33	1.33
Lisbon	242	female	90	2.67	2.67	2.00	1.00	2.00	1.00	0.67
Lisbon	243	male	53	0.67	0.67	0.00	0.00	0.00	0.00	0.00

Lisbon	244	male	47	1.00	0.67	0.00	0.00	0.00	0.00	0.00
Lisbon	245	male	65	2.00	3.00	0.33	0.33	0.00	0.00	0.00
Lisbon	246	male	81	1.67	2.00	0.00	0.00	0.00	0.00	0.00
Lisbon	247	male	67	1.00	1.00	0.00	0.00	0.00	0.00	0.33
Lisbon	248	male	48	1.00	1.00	0.00	0.00	0.00	0.00	0.67
Lisbon	249	male	37	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	250	male	47	2.67	2.33	0.00	0.00	0.00	0.00	0.33
Lisbon	251	male	30	1.00	1.50	0.00	0.00	0.00	0.00	0.00
Lisbon	252	male	46	1.50	1.50	0.00	0.00	0.00	0.00	0.00
Lisbon	253	male	64	2.67	2.33	0.00	0.00	0.00	0.00	0.33
Lisbon	254	male	49	1.00	1.33	0.00	0.00	0.00	0.00	0.00
Lisbon	255	male	68	0.67	0.67	0.00	0.00	0.00	0.00	0.00
Lisbon	256	male	53	2.00	1.50	0.00	0.00	0.00	0.00	0.00
Lisbon	257	male	58	3.00	2.67	1.00	0.33	0.00	0.00	1.00
Lisbon	258	male	63	1.00	0.50	0.00	0.00	0.00	0.00	0.00
Lisbon	259	male	68	1.00	0.67	0.33	0.33	0.00	0.00	0.33
Lisbon	260	male	52	0.33	0.33	0.00	0.00	0.00	0.00	0.00
Lisbon	261	male	40	1.00	2.00	0.00	0.00	0.00	0.00	0.00
Lisbon	262	male	50	2.00	2.00	0.50	0.50	0.00	0.00	0.50
Lisbon	263	male	82	1.00	0.67	0.67	0.33	0.00	0.00	0.33
Lisbon	264	male	75	1.00	0.67	0.00	0.00	0.00	0.00	0.00
Lisbon	265	male	20	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	266	male	40	0.67	0.67	0.00	0.00	0.00	0.00	0.00
Lisbon	267	male	43	1.33	1.67	0.00	0.00	0.00	0.00	0.00
Lisbon	268	male	30	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	269	male	69	1.00	1.00	0.00	0.00	0.00	0.00	0.67
Lisbon	270	male	57	2.00	2.33	0.00	0.00	0.00	0.00	0.00
Lisbon	271	male	29	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	272	male	83	3.00	2.67	0.67	1.00	0.00	0.00	1.33
Lisbon	273	male	38	0.33	0.33	0.00	0.00	0.00	0.00	0.00
Lisbon	274	male	72	0.67	0.67	0.67	0.67	0.00	0.00	0.00
Lisbon	275	male	31	0.33	0.33	0.00	0.00	0.00	0.00	0.00
Lisbon	276	male	47	0.33	0.33	0.00	0.00	0.00	0.00	0.00
Lisbon	277	male	84	1.67	1.33	0.00	0.00	0.67	0.33	0.33
Lisbon	278	male	21	0.33	0.33	1.00	1.00	0.00	0.00	0.00
Lisbon	279	male	71	1.00	1.00	0.00	0.00	0.00	0.00	0.00
Lisbon	280	male	23	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	281	male	49	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Lisbon	282	male	68	2.00	2.00	0.00	0.00	0.00	0.00	0.00
Lisbon	283	male	55	1.33	1.67	0.00	0.00	0.00	0.00	0.33
Lisbon	284	male	28	0.00	0.00	0.00	0.00	0.00	0.00	0.00

Lisbon	285	male	49	1.00	2.00	0.33	0.33	0.00	0.00	0.00
Lisbon	286	male	53	1.00	1.00	0.50	0.50	0.00	0.00	0.00
Lisbon	287	male	45	1.00	1.00	0.00	0.00	0.00	0.00	0.00
Lisbon	288	male	62	1.00	1.00	0.00	0.00	0.00	0.00	0.00
Lisbon	289	male	88	0.67	0.67	0.67	0.33	0.00	0.00	0.00
Lisbon	290	male	82	3.00	3.00	1.00	1.00	2.00	1.00	1.00

E. Activity (n=124)

Collection	Calce_ID	J	occupation
Sassari	1	363.73	homeless
Sassari	2	548.03	laborer
Sassari	3	525.51	butcher
Sassari	4	427.20	farmer
Sassari	5	390.81	farmer
Sassari	6	439.03	farmer
Sassari	7	389.00	homeless
Sassari	8	336.92	
Sassari	9	441.76	tanner
Sassari	10	532.75	laborer
Sassari	11	306.57	farmer
Sassari	12	342.97	driver
Sassari	13	430.10	soldier
Sassari	14	367.27	trade worker
Sassari	15	513.57	farmer
Sassari	17	388.74	
Sassari	18	336.34	
Sassari	19	232.50	
Sassari	20	333.71	fisherman
Sassari	21	354.01	farmer
Sassari	23	344.28	mason
Sassari	24	400.61	
Sassari	25	439.46	farmer
Sassari	26	452.44	farmer
Sassari	27	434.08	laborer
Sassari	34	294.57	chef
Sassari	38	460.19	bellhop
Sassari	40	449.65	laborer
Sassari	45	388.89	
Sassari	46	431.57	
Sassari	47	656.10	
Sassari	48	379.44	
Sassari	49	536.23	
Sassari	52	439.36	laborer
Sassari	53	524.97	housewif
Sassari	54	366.60	housewif
Sassari	55	461.95	housewif
Sassari	56	389.48	housewif

Sassari	57	243.62	housewif
Sassari	58	355.13	housewif
Sassari	60	352.82	housewif
Sassari	61	352.89	housewif
Sassari	62	472.38	housewif
Sassari	63	299.82	housewif
Sassari	69	476.43	housewif
Sassari	72	232.09	housewif
Sassari	73	438.84	housewif
Sassari	74	295.09	housewif
Sassari	76	311.87	housewif
Sassari	77	239.43	housewif
Sassari	78	346.72	housewif
Sassari	81	347.13	housewif
Sassari	86	410.46	housewif
Sassari	89	456.98	housewif
Sassari	93	375.89	housewif
Sassari	94	304.94	housewif
Sassari	96	364.12	housewif
Sassari	98	282.09	housewif
Sassari	99	409.84	housewif
Sassari	100	341.08	housewif
Sassari	101	413.80	housewif
Sassari	102	317.05	housewif
Lisbon	197	395.00	housewif
Lisbon	198	475.99	housewif
Lisbon	199	336.57	housewif
Lisbon	200	374.03	housewif
Lisbon	201	298.44	housewif
Lisbon	202	385.55	housewif
Lisbon	203	432.41	housewif
Lisbon	204	472.40	housewif
Lisbon	205	372.91	housewif
Lisbon	206	504.52	housewif
Lisbon	207	472.17	housewif
Lisbon	208	573.72	housewif
Lisbon	209	385.98	housewif
Lisbon	211	393.81	housewif
Lisbon	212	435.43	housewif
Lisbon	214	673.85	housewif
Lisbon	215	405.43	housewif

Lisbon	220	337.77	housewif
Lisbon	223	298.45	housewif
Lisbon	224	597.80	housewif
Lisbon	226	421.68	housewif
Lisbon	229	352.32	housewif
Lisbon	232	400.36	housewif
Lisbon	233	369.86	housewif
Lisbon	234	395.06	housewif
Lisbon	235	427.10	housewif
Lisbon	236	430.08	housewif
Lisbon	237	352.12	housewif
Lisbon	238	453.06	housewif
Lisbon	239	407.02	housewif
Lisbon	240	441.83	housewif
Lisbon	241	356.72	housewif
Lisbon	242	404.51	housewif
Lisbon	244	459.25	commerce
Lisbon	245	551.63	retail
Lisbon	246	427.00	landlord
Lisbon	247	372.59	contractor
Lisbon	250	527.12	mailman
Lisbon	252	493.88	electrician
Lisbon	253	729.90	clerk
Lisbon	257	575.26	trader
Lisbon	258	605.58	laborer
Lisbon	261	504.44	railroad
Lisbon	264	440.00	chef
Lisbon	265	332.29	electrician
Lisbon	267	460.68	upholsteror
Lisbon	268	431.38	printer
Lisbon	269	486.79	retail
Lisbon	271	296.05	librarian
Lisbon	272	385.07	merchant
Lisbon	274	426.82	businessMan
Lisbon	275	489.73	electrician
Lisbon	277	432.12	forest rang
Lisbon	278	430.42	retail
Lisbon	279	489.40	tax man
Lisbon	280	401.10	commerce
Lisbon	282	434.14	trader
Lisbon	285	379.92	security

Lisbon	286	409.36	trader
Lisbon	288	369.37	police
Lisbon	289	382.39	painter
Lisbon	290	466.08	army captai