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Methodological Considerations:
Osteoarthritis and the Significance of Porosity in the
William M. Bass Donated Skeletal Collection

A Thesis
Presented for the
Masters of Arts Degree
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Kimberly Tenese Wren
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DEDICATION

This Masters Thesis is dedicated first and foremost to my Lord and Savior Jesus Christ for giving me the strength to go on and to my mother, Therolan Wren, for the little things that mean so much.

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ABSTRACT

Osteoarthritis is one of many diseases that accompany the ascension into old age. It has been documented in animals and humans alike. The current research examines porosity's relationship to osteoarthritis. European American females and African American and European American males from the contemporary William M. Bass Donated Skeletal Collection are analyzed.

The objective of this study is to determine whether or not a change in the relationship of osteoarthritis severity to porosity severity will occur as the criteria used to identify osteoarthritis changes from osteophytes alone (method I), to eburnation alone (method II), and then both osteophytes and eburnation (method III). The significance of the relationship of osteoarthritis severity to porosity severity is also examined for each method to determine whether or not porosity is related to osteoarthritis. The data reveal that in most instances there is not a change in the relationship of porosity severity to osteoarthritis severity when utilizing method I versus method III to identify osteoarthritis. However, the relationship of osteoarthritis severity to porosity severity changes when utilizing method II (eburnation) as the sole identifying criterion of osteoarthritis. There also appears to be a significant relationship between porosity severity and osteoarthritis severity in the left hips of African American and European American males. Furthermore, in the knee, porosity's significance to OA, using any of the three methods, tended to be positively correlated with sample size. This suggests that porosity might be related to osteoarthritis and consequently should not be excluded as a criterion for osteoarthritis presence.

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Chapter I

Introduction

Aim of the Pathologist

“The pattern of disease or injury that affects any group of people is never a matter of chance. It is invariably the expression of stresses and strains to which they were exposed, a response to everything in their environment and behaviour. It reflects their genetic inheritance (which is their internal environment), the climate in which they lived, the soil that gave them sustenance and the animals or plants that shared their homeland. It is influenced by their daily occupations, their habits of diet, their choice of dwelling and clothes, their social structure, even their folklore and mythology.” (Wells 1964:17)

If the goal of anthropology is to explore and understand all aspects of human variation through time and space, pathology is one way of doing just that. Pathology is a physical expression of how people respond to the misfortunes of their environment. Through the study of ancient diseases, in conjunction with present diseases, one can make inferences concerning various population lifestyles (i.e., activities, diets, migration patterns, etc.) through time and space. The aim of the pathologist, in this context the paleopathologist, is to reconstruct as accurately as possible what might have led to a disease, the significance of the disease and the disease process, and in so doing provide a means by which one might avoid such disease (Johnson 1965). Both the pathologist and the paleopathologist seek to know whether certain changes in the disease process and disease patterning are more common in one population versus another, what their distribution is throughout the body, in addition to the history and geography of disease (Jarcho 1965). They also seek to determine, disease subsets, and the role age, sex, ancestry, heredity, and obesity play with regard to disease (Moskowitz 1984a).

Research Agenda

Osteoarthritis is one such disease in which paleopathologists can make inferences regarding changes in population lifestyles. Osteoarthritis is one of the most widely distributed diseases across both time and space, yet, it is also one of the most poorly understood amongst clinicians, rheumatologists, and anthropologists alike (Moodie 1923, Wells 1964, Moskowitz et al. 1984, Hall 1995). The current research involves the examination of the hips and knees of 231 individuals from the William M. Bass Donated Skeletal Collection. This research seeks to determine if a change in the relationship between porosity severity scores and osteoarthritis severity scores, when defining osteoarthritis by osteophytes alone (method I), secondly by eburnation alone (method II), and lastly by osteophytes and eburnation (method III), will occur. This research also seeks to determine if there is a consistently significant relationship between porosity severity and osteoarthritis severity (when defining osteoarthritis by methods, I, II, and III) for the joint surfaces examined herein as a whole. The primary purpose of this research is to determine porosity's relationship to osteoarthritis, and in doing so, provide data that will accept or refute Rothschild's (1997) assertion that porosity is not related to osteoarthritis and should thus be excluded as an identifier of osteoarthritis. First, however, a brief history on the establishment of osteoarthritis as a disease of its own and of some current issues that concern osteoarthritis identification in various disciplines follows.

Osteoarthritis itself was not identified as a separate form, from that of other forms, of arthritis until 1898 when Bannatyne (Janssens 1970) identified it as such due to

its characteristic formation of new bone. It was not until the early 1900's, however, that a clear distinction was made between hypertrophic (osteoarthritis) and atrophic (rheumatoid) arthritis, and osteoarthritis was defined as a clinical entity (Moskowitz 1984a:1). Experts have since defined osteoarthritis as a disease of the synovial joints characterized by degeneration of joint tissue and remodeling of underlying bone (Sokoloff 1969, Hall 1995, Ortner 2003a). They have also acknowledged that it is highly correlated with aging, though the significance of aging to osteoarthritis is still debatable (Sokoloff 1969, Peyron 1988, Hughes and Dunlop 1995, Verbrugge 1995).

Furthermore osteoarthritis experts have yet come to an agreement on a proper name for osteoarthritis. "Osteoarthrosis", "Osteoarthritis" and "Degenerative Joint Disease" are among the various names employed to describe this disorder of the synovial joints (Sokoloff 1969, Janssens 1970, Wood 1976, Radin 1976, Steinbock 1976, Wood 1978, Jurmain 1990). A number of authors (Sokoloff 1969, Janssens 1970, Wood 1976, and Jurmain 1990) argue that the term osteoarthritis is misleading because it implies inflammation as the primary inciting cause. The alternate term "Degenerative Joint Disease" implies degeneration as the main factor, in essence specifying etiology, which creates semantic difficulties when the changes can be shown to result from other causes such as metabolic abnormalities (Sokoloff 1969, Wood 1976, Jurmain 1990).

"Osteoarthrosis," on the other hand, takes into account the destructive components of the disease as well as the production of new bone and cartilage, and places inflammation in secondary position (Sokoloff 1969, Jurmain 1990). "Osteoarthrosis is, therefore, the most appropriate term for this ailment of the synovial joints. Osteoarthritis is also

sometimes referred to as hypertrophic arthritis because of its characteristic formation of sclerotic subchondral bone and marginal spurring (osteophytes) (Steinbock 1976). For this investigation, the term osteoarthritis, denoted as OA, will be utilized because of its widespread use in anthropological, rheumatic, clinical, and pathologic literature in the United States.

Although radiological, histological, and chemical analyses of OA have contributed greatly to knowledge concerning the process of OA, they have contributed very little regarding the etiology of OA. Admittedly, knowledge of the etiology of a disease is very important in providing treatment that hinders or slows down the disease process. However, paleopathologists, anthropologists, and other individuals encountering dry, defleshed, and in many cases poorly preserved bone are not, given the circumstances, able to analyze osteoarthritic lesions outside of the osteological realm. For this reason Johnson (1965:71-72), a paleopathologist, argues that pathology is the study of the *structure of disease* and not its etiology, and that “the fundamental function of the pathologist is to unravel the morphologic characteristics of something that is already defined.” Thus, a paleopathologist or anthropologist examining skeletal remains must focus most of their time on identifying and describing the changes that occur in bone afflicted by various ailments.

Paleopathologists, pathologists or anthropologists dealing with bones must first recognize that bone responds by resorbing previously established bone or adding new bone during the disease process. The task is to *describe* and *define* the reactive patterns of abnormal bone through both qualitative and quantitative analysis. This includes

listing, measuring, mapping, and recording the pattern of variation in addition to the stages of the abnormalities present. Lastly, researchers must examine these data in light of biological data on age, sex, and ancestry as well as behavioral data of the individual(s) being examined (Johnson 1965). Examining disease in light of biological and behavioral data enables paleopathologists to make inferences concerning population lifestyles (i.e., activities, diets, migration patterns, etc.) and to reconstruct, as accurately as possible, what might have led to a disease, the significance of the disease and the disease process, and in so doing provide a means by which one might avoid such disease (Johnson 1965).

If describing and defining the reactive patterns of abnormal bone, according to Johnson (1965), is the role of the anthropologist when examining bone in the pathological realm, then why, according to Bridges (1993), are there so many inconsistencies among anthropologists in regards to the prevalence and distribution of OA among and within populations, age groups, sexes and joints? Moskowitz (1984a) suggests that such inconsistencies occur in studies outside of anthropology because autopsy studies, for example, tend to define OA earlier than clinical studies which depend on symptomatic responses from the patient that are often triggered by *significant* pathologic changes. Moskowitz also notes that the way in which a disease is defined, the techniques utilized to analyze the disease, as well as the number and location of joints analyzed all have an affect on the results obtained in any given study. Ortner (2003b) also suggests that, given the differences in the data available to the clinician and the paleopathologist, there will be

incongruities in what the clinician observes and what the paleopathologist sees in the skeleton.

Anthropologists, for the most part, are not confronted with the same short comings as in autopsy analysis, clinical analysis, and/or roentgenographic analysis. Whether they are analyzing paleo- or modern populations, they are, for the most part, still dealing with bare bones and in many cases the entire skeleton. Given that age, sex and sometimes ancestry must be determined by the anthropologist, one can understand if there are subtle inconsistencies among these demographic data due to observer error and biases (Ortner 2003c). Such inconsistencies, however, should not affect the overall identification of OA. Inconsistencies in the identification of OA stem from unaddressed observer biases. Anthropologists use whatever criteria they see fit to identify OA. Lieverse et al. (2007) used osteophytes, eburnation, and porosity; Rothschild (1997) used only osteophytes; Waldron (1992) emphasized the importance of eburnation being present. The discrepancy among anthropologists in what morphological criteria denote OA not only hinders comparisons between studies but might also have an affect on interpretations regarding porosity's relationship to osteoarthritis in any given skeletal sample.

The significance of porosity, defined by the presence of discontinuity (pitted, disorganized lesions) of subchondral bone in the absence of erosive processes (Rothschild, 1997), to OA further complicates observer biases. Woods (1995) examination of 486 OA affected individuals in the Hamman-Todd Collection demonstrated a lack of correlation between porosity and eburnation in the knee joint.

This contradicts Ortner's (1968) assumption that eburnation rarely occurs without porosity, although, Ortner (2003a) later suggested that porosity *can* occur in the presence eburnation. Additionally, Rothschild's (1997) re-examination of the knees of 400 individuals in the Hamman-Todd Collection further concluded that no significant relationship existed between porosity and OA as defined by the presence of osteophytes. For this reason Rothschild (1997) suggested that porosity be excluded as an identifying characteristic of OA.

The identifying characteristics utilized, the various scoring techniques, as well as the relationship of the identifying characteristics to one another affect OA prevalence, distribution and severity data. The current research seeks to determine whether or not porosity is related to OA and whether or not the relationship of porosity to OA changes depending on the method used to identify OA. The following sections address the theoretical and methodological issues that must be considered when identifying or hypothesizing diseases and disease lesions in the skeletal realm.

Theoretical Considerations

The data an epidemiologist collects on any given population regarding a particular disease ought to represent the distribution of that disease within that population. For the most part it does. Clinicians have at their disposal the living individual, flesh and all. Thus they are able to account for disease in soft tissue and to some extent in bone, through the use of radiographs.

In contrast, paleopathologists can examine an abnormal skeleton in its entirety, obtaining detailed information on the type and distribution of skeletal lesions which the clinician would not have been able to obtain (Ortner 1991, Ortner 2003b). Yet, because paleopathologists deal most often with disease as it is manifested in bone, they operate under different theoretical guidelines and assumptions. They are also faced with different theoretical issues. One guideline that was also addressed in the introduction is that the paleopathologist ought to be concerned with the structure of disease and the morphological aspect of disease, not its etiology (Johnson 1965, Ortner 2003c). The paleopathologist describes and defines the reactive patterns of diseases that have already been well established scientifically (Johnson 1965).

Furthermore paleopathologists are faced with the issue of relating pathological bone changes to actual diseases. Paleopathology depends on the assumption that data regarding pathological conditions have proven themselves consistent with a given disease and thus accurately represent that disease (Ortner 2003b, 2003c). However, despite careful descriptions and precautions taken to ensure a sound diagnosis, one must keep in mind that there “are no such things as infallible diagnostic criteria” (Johnson 1965:69). Bone, when confronted with a pathological agent, only reacts by creating more bone, resorbing already existing bone or a combination of the two. Consequently several diseases may trigger a similar response in bone (Mann and Murphy 1990).

Yet another issue the paleopathologist encounters is the realization that these data is *not* an accurate r, or even the sample he has in front of him. Data obtained by the paleopathologist are an *under* representation of the distribution of disease in both the

living population and the sample population analyzed. For the paleopathologist has at their disposal bare bone with little if any soft tissue. As a result, they can only address how a disease is manifested skeletally which, at least in the case of OA, would be an under-representation because in most cases of OA soft tissues are involved first; it might take a considerable amount of time before skeletal tissues are involved (Ortner 2003a). Consequently criteria used by paleopathologists to identify OA, such as eburnation, osteophyte development and sclerosis, are predominantly, especially in regard to eburnation, secondary to soft tissue changes like joint space narrowing and cartilage destruction (Meachim and Brooke 1984, Ortner 2003a).

Furthermore, paleopathologists seek to know what their data mean in light of human evolution, human biology, and the interaction between humans and their environments (Ortner 1991, Ortner 2003c). Genetics acts to either increase or decrease one's resistance to disease, while culture acts to minimize or increase the incidence of disease, both of which play a role in human history and evolution (Ortner 2003c). The evolutionary relationship between humans and disease agents raises questions concerning conclusions made based on paleopathological data (Ortner 2003c). The hope is that such conclusions accurately address the relationship between disease and human populations across time and space (Ortner 1991).

In summary, in order to work in the paleopathological realm it is imperative that individuals have a clear understanding of the theoretical context in which they toil. Paleopathologists ought not be concerned with the etiology of disease but with the structure of disease (i.e. those morphological characteristics of disease that manifest

themselves in the skeleton). They must also realize that there are no perfect criteria for identifying disease and that the data they obtain, be they accurate, are always an under-representation of both the living population and the sample that came from that population. Even so, paleopathology has the potential to contribute to our understanding of the role disease has played in human biology and evolution amongst varying populations, the multifaceted relationship between disease and human cultural evolution, as well as our understanding of skeletal responses to disease (Ortner 1991).

Methodological Considerations

Given that no criteria or criterion is with 100% certainty indicative of a particular disease when dealing with pathological conditions on bone, paleopathologists can only aim at achieving the best identification possible based on *well* established identifying criteria. *Well* established criteria are those that have been found to consistently occur with a given disease. The first step in achieving the most accurate identification possible is to develop a standard descriptive and classificatory system for analyzing pathological conditions in bone (Ortner 1991). All of the abnormal bone lesions encountered in the human skeleton must be described in great detail (Mann and Murphy 1990, Ortner 2003d). A careful and all-inclusive descriptive terminology of pathological bone lesions that integrates pathological information from various disciplines must also be established (Mann and Murphy 1990, Ortner 2003d). A standard descriptive and classificatory system not only serves as a guideline for paleopathological research but also gives researchers the option of arriving at a different diagnosis (Ortner

2003c, Mann and Murphy 1990, Ortner and Aufderheide 1991, Ortner 1991). Buikstra and Ubelaker (1994) provide a great example of a standardized descriptive and classificatory system for analyzing human skeletal remains. Unfortunately, this standard has not been embraced by the entire anthropological community in regards to pathological data collection.

Data basic to a standard pathological descriptive and classificatory system includes demographic and context information such as age, sex, ancestry or population, geographic location and time period (Ortner 2003e). Paleopathological data includes the location and distribution of bone lesions in the joint, the extent of involvement of bone or the entire skeleton, as well as bone destruction and hypertrophy (Ortner 2003e). Bone lesions should be described in light of the cells that produce them so that they might be comprehensive across disciplines (Ortner 1991, Ortner and Aufderheide 1991). In other words, one should describe lesions in terms of osteoblastic (bone formation) or osteoclastic (bone resorption) activity (Ortner 1991). Pathological lesions should also be analyzed using one or more of the following techniques: gross analysis, radiographic analysis, light and scanning electron microscope examination, and chemical analysis (Ortner 2003e). All pathological features apparent in a skeleton must be obtained before a diagnosis or diagnostic options are given (Jurmain 1977, Ortner 2003c).

In addition to the need for the acceptance of a standard descriptive and classificatory system, paleopathology is also faced with the problem of a lack of comparability in pathological reports (Ortner 1991). If two or more reports concerning the same disease but utilizing different populations living during different time periods

and in distinct geographical regions cannot be compared due to methodological issues, research regarding the distribution of disease between groups, across time and space is hindered. This proves detrimental to paleopathology, as paleopathology seeks to reconstruct the relationship between human biology, human culture history, human evolution and disease (Ortner 1991). Ortner (1991) gives three reasons for the lack of comparability among research reports. To begin with, researchers view skeletal disease according to *their own* knowledge of skeletal disease, which varies greatly among researchers. Second, there is a lack of consistency in the type of data collected and published among researchers. The third reason for the lack of comparability which was also mentioned by Ortner and Aufderheide (1991) is that in published sources many authors simply give a diagnostic *opinion* regarding paleopathological specimens in addition to vague and imprecise descriptions. This not only prohibits independent assessment of specimens but also “limits any statements regarding variation in disease prevalence in antiquity to highly speculative observations” (Ortner 1991:8).

According to Ortner (2003e) the main objective of a descriptive analysis is to identify the disease process, which ultimately may assist other specialists in identifying its etiology. If a disease cannot be directly correlated with the pathological lesions observed, a detailed description of such lesions still provides important information on the frequency and prevalence of descriptive categories (Ortner 2003e). Later research might call for the establishment of a new disease category or a new subfield under an existing category. Ultimately, a standard descriptive and classificatory system like that of Buikstra and Ubelaker (1994) allows paleopathology to advance the overall substance of

its research so that questions concerning the theoretical implications of pathological data can be addressed (Ortner and Aufderheide 1991).

The current research relates to Ortner's (1991) first and second reason for the lack of comparability between reports. The first being that researchers view skeletal disease according to *their own* knowledge of skeletal disease and the second being the lack of consistency in the type of data collected and published among researchers. Rothschild (1997) viewed osteophytes as the only means by which to identify OA, consequently porosity was only addressed in light of its relationship to osteophytes. Data on porosity's relationship to eburnation was not collected because Rothschild (1997:530) viewed eburnation as "a sign of severity of arthritis, not of the form of arthritis that caused cartilage loss." Other researchers (Waldron 1992, Woods 1995, and Lieveise et al. 2007) would have identified OA based on other criteria such as eburnation, porosity, and osteophytes, or a combination of these criteria. Perhaps, in identifying OA differently, Rothschild (1997) would have obtained different results in regards to porosities relationship to OA and not of concluded that porosity should be excluded as an identifying characteristic of OA. The theoretical and methodological issues posed here can not be adequately addressed in regards to OA if researchers have yet to ascertain *well* established identifying criteria for OA. Accordingly, the current research aims to assist researchers in the establishment of sound criteria for the identification of OA by testing the significance of one questionable criterion, porosity, to OA (as defined by varying methods).

The following chapter reviews literature that has addressed porosity's relationship to OA lesions as well as porosity's relationship to OA directly. Chapter III will discuss normal and abnormal bone formation processes, the disease process and types of OA as well as the prevalence of OA in distinct populations and related diseases. Theories related to the etiology of OA and porosity are discussed in chapter IV. Chapter V discusses how the biomechanics of the hip and knee joints play a role in the occurrence and location of OA lesions on distinct joint surfaces. Chapter VI discusses how anthropologists, clinicians, and rheumatologists view porosity in regards to OA and why such views have taken root in the varying professions. Accordingly, chapter VII discusses in detail the materials and methods used in the current research. Chapter VIII lays out the results of the current research and chapter IX considers these results in light of the implications they make in regards to porosity's relationship to OA as it is defined by various methods. The conclusion, chapter X, follows and acts to summarize the importance the current study in regards to identifying criteria of OA, in particular, porosity's significance as an identifying criterion of OA.

Chapter II

Literature Review

The purpose of this chapter is to provide a basis for the current research endeavor by elaborating on what previous researchers have done concerning porosity's relationship to OA lesions and OA directly. Studies by three researchers (Ortner 1968, Merbs 1983, and Woods 1995) are addressed to demonstrate how their research on porosity's relationship to eburnation and non-contact areas (i.e., those areas that are out of contact with forces throughout normal locomotion) culminated in Rothschild's (1997) investigation of porosity. Consequently, Rothschild's (1997) findings, that no significant relationship was found between porosity and OA among sexes, ancestral groups, or with increasing age, led him to conclude that porosity is not related to OA and should thus be excluded as an identifying criterion of OA. In light of Rothschild's (1997) conclusion concerning porosity, the current research was undertaken to test the validity of his assertion by not only using different methods to identify OA but also by including the hip in the analysis.

Most anthropologists when attributing OA to the skeleton look for the occurrence of osteophytes, eburnation, and porosity (Ortner 1968, Jurmain 1977, Merbs 1983, Bridges 1993, Ortner 2003a). Radiographically, rheumatologists and clinicians look for joint space narrowing, subchondral sclerosis, eburnation, osteophytes, and cysts formation (Hoaglund 1976, Moll 1976, Altman et al. 1986, Altman et al. 1991). Porosity/pitting is mentioned quite often by anthropologists without explanation or any attempt to explore its significance to OA (Jurmain 1977, Ortner 2003a, Lieveise et al.

2007). Ortner (1968), in an attempt to better establish classification criteria for OA recorded the frequency, in the form of presence or absence, of porosity, eburnation, and osteophytes among the different joint surfaces of the elbow in 165 Alaskan Eskimos and 485 Peruvian Indians. The elbow, like the knee, is a hinge joint, but unlike the knee is a non-weight bearing joint. He examined the relationship between localized areas of stress on the joint surface and the production of OA lesions. He found that porosity was frequent on areas under the most amount of stress; these areas include the trochlea and capitulum. He also found that eburnation rarely occurred without porosity, though porosity frequently occurred without eburnation, suggesting that porosity precedes eburnation as a lesion during the OA process. He did not, however, explore directly the relationship of porosity to OA.

Merbs (1983) explored the relationship between porosity and eburnation to investigate Ortner's (1968) suggestion that eburnation rarely occurred without porosity. He examined the articulating surfaces of the hips and knees along with several other bone joint surfaces of a Canadian Inuit population consisting of 97 Saddlermiut individuals from the site of Native Point on Southampton Island. Eburnation, osteophyte development and porosity were used to identify OA; they were scored as absent, trace, mild, moderate or severe. Merbs (1983) found that eburnation was not prevalent throughout the entire population, however when eburnation occurred in the individual it occurred in several different joints. Porosity also appeared to precede almost all cases of eburnation with the exception of a few instances in which eburnation appeared to precede porosity. Merbs (1983) also found that porosity *did* occur in the absence of eburnation.

His findings, in accordance with Ortner (1968), seem to suggest that porosity might precede eburnation in the arthritic process. Merbs (1983), however, did not look at the significance of porosity to OA directly.

Woods (1995), however, demonstrated that porosity did not correlate with eburnation in the knee joint. In his observation of 486 OA affected individuals in the Hamman-Todd Collection, he also found differences in the frequency of porosity and eburnation with regards to subgroups and joint surfaces (contact vs. non-contact). Black females were much more prone to porosity than any other group, accounting for 17.7% of porosity presence, while both Black and White females were prone to eburnation of the knee joint. White males seemed to be resistant to both, whereas Black males were moderately prone to both.

In addition, porosity tended to concentrate on the posterior most portions of the condyles. These areas are out of contact with forces throughout normal locomotion thus they are non-contact areas. On other non-contact areas, porosity tends to concentrate on the medial and lateral aspects of the patellofemoral grooves of the knees, the non-contact areas of the medial and lateral femoral condyles, as well as the medial and lateral facets of the retropatellar surfaces of the knees. On the habitual contact areas of the medial and lateral condyles, the medial patellofemoral facet, and on the medial and lateral tibial plateaus, porosity was sparse.

Porosity was also present in only 163(34%) of the 486 individuals with OA, 15 of which had porosity exclusively in the absence of osteophytes and or eburnation. Furthermore, 314(65%) of the 486 had osteophytes exclusively and none had eburnation

exclusively. Porosity in conjunction with osteophytes but in the absence of eburnation occurred in 124(26%) individuals. All three lesions occurred in 24(5%) of the 486 and in no individual did porosity occur together with eburnation without osteophytes. What's more is that in no instance did eburnation manifest itself in the absence of osteophytes.

Eburnation, in contrast, was most often located in habitual contact areas. Such areas include the medial and lateral facets of the patellofemoral surface of the knees, contact areas of the medial and lateral femoral condyles, medial and lateral tibial plateaus plus the lateral facet of the retropatellar area of the left knee.

Woods' (1995) findings suggest that porosity and eburnation are not only unrelated to each other but also the result of different etiologies since they do not occur on the same type of joint surfaces, contact vs. non-contact. Consequently Woods (1995) stressed the importance of research being done that would explore the occurrence of porosity in light of other forms of arthritis to clarify its role as an identifying characteristic of OA.

On the other hand Woods (1995) scored porosity as slight, moderate or severe and gave a detailed description with photos of how each score was represented skeletally. But, both eburnation and osteophyte development were scored as either present or absent. Due to these inconsistencies in scoring method, Woods' (1995) analysis lacks data regarding relationships between porosity severity and eburnation severity, porosity severity and osteophyte severity, as well as severity between all three criteria combined.

Rothschild (1997) investigated porosity further when he did an intense study of the significance of porosity to OA in the knee of 400 individuals, again using the

Hamman-Todd Collection. Rothschild's (1997) analysis involved African American males and females as well as European American males and females. He pointed out that neither porosity nor eburnation fulfill the American College of Rheumatology established criteria for recognition of osteoarthritis, even though both of them continue to be used as diagnostic criteria for the recognition of OA in the skeleton (Jurmain 1980 & 1990, Altman et al., 1986 & 1991, Ortner, 2003a). Rothschild (1997) set out to test the assumed relationship between porosity and OA. Porosity was recognized as discontinuity (pitted, disorganized lesions) of subchondral bone in the absence of an erosive process. OA was recognized by the presence of osteophyte formation and or evidence of subchondral bone sclerosis, both of which fulfill the American College of Rheumatology criteria and clinical criteria for recognizing OA (Altman et al., 1986 & 1991). OA severity was not addressed. Knee porosity was found in 70 of 400 skeletons (17.5%), OA was present in 118 (29%), while porosity and OA coexisted in 21 individuals (5%). Thus 49 (70%) individuals with porosity had no OA and 97 (82%) of individuals with OA lacked porosity. *Rothschild (1997) utilized Student t-test to determine differences in the average age of individuals with porosity and osteoarthritis (as defined by osteophytes).* Chi-square was also used to determine whether there was a relationship between age groups, ancestral groups, or sex and either porosity or osteoarthritis; chi-square was also employed to test the possible effect of age on porosity. No significant relationship was found between sexes, ancestral groups, or with increasing age. Rothschild (1997) concluded that no significant relationship existed between porosity and OA and that porosity should thus be excluded as an identifying characteristic of OA.

Consequently, the current study aims at testing Rothschild's (1997) assertion that porosity is not significantly related to OA by testing whether there is a relationship between porosity severity and OA severity as OA is defined first by osteophytes (method I), then by eburnation (method II), and last by both osteophytes and eburnation (method III). The current research also addresses porosity in light of both the hip and knee to determine whether the type of joint, ball-and-socket versus hinge (respectively), plays a role in the relationship of porosity to OA. However, before launching directly into materials, methods, results, discussion, and conclusion, chapters containing sections on bone formation, disease processes regarding OA, types of OA, the prevalence of OA in similar and distinct populations, related diseases, theories and etiological orientations regarding OA and porosity, the biomechanics of OA, and identifying criteria of OA are addressed. Such chapters aim at providing the reader with a thorough understanding of not only several interrelated components of OA but also the current issues concerning OA.

Chapter III

Bone Formation and Osteoarthritis

This chapter gives an overview of what characterizes normal and abnormal bone formation. An understanding of bone formation sets the stage for what morphological traits can and cannot represent OA in the skeleton. Also included in this chapter is a section on the disease process that characterizes OA. This section contains information regarding the gradual increase in joint degradation (as evidenced by loss of cartilage, and the destruction and abnormal formation of both bone and cartilage) after the onset of OA. Knowledge of the disease process also sets the stage for what morphological traits characterize OA. Sections regarding the different types of OA, the prevalence of OA in distinct populations, as well as the diseases related to OA follow. These sections provide information on how OA and other forms of arthritis manifest themselves throughout the skeleton, among sexes, ancestral groups, and age groups.

Bone Formation

“A thorough knowledge of normal bone growth and remodeling and normal bone structure is necessary before one can appreciate the response of bone to disease and the consequent changes in its gross appearance” (Steinbock 1976:3). Bone is a connective tissue made up of living cells that is 92% mineral and 8% water; it supports the body, protects organs, and provides attachment for muscles and soft tissues (Ross 1985, Carter 2003a). Long bones have at their articulating ends epiphyses that bear articular cartilage and fuse to the metaphyses during maturation (Ross 1985, Carter 2003a) (Figure 1).

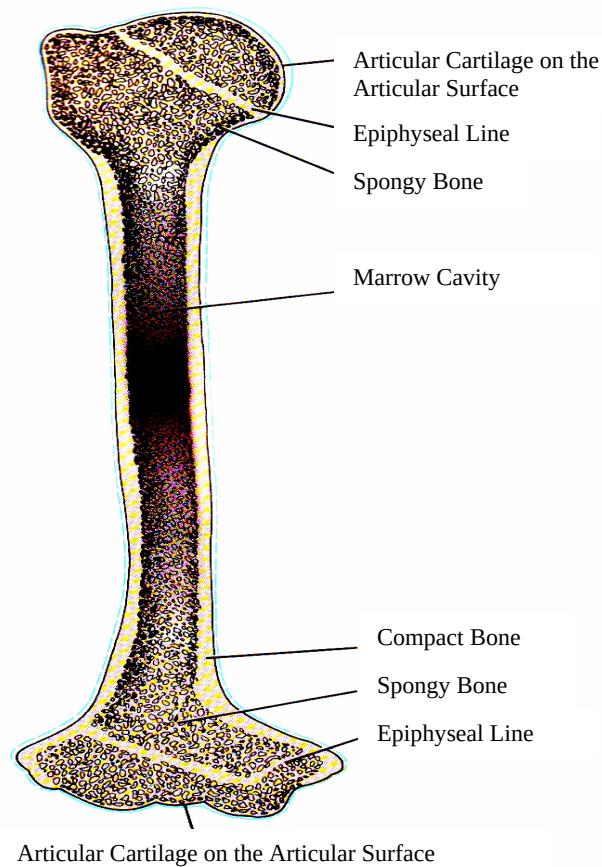


Figure 1: The Bone Structure of a Typical Long Bone (Ross 1985).

Metaphyses are immediately beneath the epiphyses at each articulating end in growing bone; they extend into the cortex of the diaphysis; the diaphysis is the shaft or central portion of the long bone (Ross 1985, Carter 2003a).

There are two types of bone, woven and lamellar. Woven bone consists of collagen fibrils forming an irregular matrix, it is primarily in the embryonic skeleton and is gradually replaced by lamellar after the first year of life (Ross 1985, Carter 2003a). Lamellar bone includes trabecular/cancellous bone and compact/cortical bone (Ross 1985, Carter 2003a). Trabecular bone is porous and associated with the interior of the

epiphyses and metaphyses and bone marrow (Ross 1985, Carter 2003a). Compact bone is dense and makes up the thick bone cortex of the shafts/diaphyses of long bones as well as the thin outer layer of bone around long bone epiphyses and irregular bones (Ross 1985, Carter 2003a).

Intramembranous ossification forms the frontal and parietal bones and other flat bones. It is also responsible for increasing the diameter and thickness of long bones by subperiosteal bone apposition (Ross 1985). Endochondral ossification is the other process by which normal bone forms. During endochondral ossification the cartilaginous model, which was laid down by chondroblasts, is converted into bone through osteoblastic ossification which starts in the midshaft and spreads to both ends of the bone simultaneously (Ross 1985). Osteoblasts form the bone matrix through the deposition of bone, osteocytes maintain the bone matrix, and osteoclasts remove or dissolve the bone matrix (Ross 1985, Carter 2003a). Endochondral ossification is associated with growth and maturation of the skeleton (Ross 1985, Carter 2003a).

Both intramembranous and endochondral ossification are referred to as bone *modeling* and are associated with the early processes of bone formation (Boskey 1999, Dempster 1999). Remodeling involves the process of osteoclastic bone resorption and osteoblastic bone deposition (Boskey 1999, Dempster 1999). *Remodeling* results in major changes in the size and shape of bone during growth and persists until death (Boskey 1999).

Both normal bone resorption (osteoclastic activity) and normal bone formation (osteoblastic activity) are needed to establish and maintain the bone matrix (Ross 1985,

Carter 2003a). Bone reacts to pathological or abnormal conditions by forming new bone, resorbing existing bone, or a combination of the two processes at different locations in the bone (Steinbock 1976, Lian and Stein 1999). Resorption can be caused by contact pressure erosion from tumors, infection, or cancer (Lian and Stein 1999). Deposition of bone is most often the result of periosteal inflammation, which results when infections or muscle injuries pull the periosteum away from bone, but can also be associated with tumors and hematological disorders (Ross 1985, Lian and Stein 1999).

Disease Process

OA is a degenerative disease of the synovial joints as well as those joints between the articular processes of vertebrae. Only fixed, immovable, synarthroses joints, such as the sutures of the skull and pubic symphysis are spared (Steinbock 1976, Wells 1964, Meachim & Brooke 1984). Synovial joints are freely movable and covered with articular cartilage at their articulating bone surfaces (Hall, 1995). The onset of OA in synovial joints leads to cartilage degradation, the destruction of bone, the formation of hypertrophic bone and cartilage as well as the formation of osteophytes (Wells 1964, Howell 1984, Moskowitz 1984a, Ortner 2003a). The degradation of articular cartilage also leads to a sclerotic osteoblastic response in the subchondral bone, mechanical attrition, eburnation of the exposed bony articular surface and porosity of the underlying subchondral bone (Ortner 2003a). Osteoarthritis generally attacks cancellous/trabecular areas of bone first and then other areas of bone (Steinbock 1976).

OA manifests itself in the skeleton three different ways according to Ortner (2003a). One way in which OA manifests itself in the skeleton, following the destruction of articular cartilage, is through abnormal abrasion of the underlying subchondral bone as a result of bone on bone contact. Sclerosis in the form of subchondral cysts or subchondral bone sclerosis (eburnation) is also indicative of OA in the skeleton. Bone also responds to OA by initiating new bone and cartilage growth along joint margins and joint surfaces (Ortner 2003a).

Types of Osteoarthritis

OA manifests itself by means of primary (idiopathic) and secondary processes. OA is considered primary when there appears to be no underlying cause to its onset. Primary OA usually occurs later in life, is milder and affects more than one joint in the skeleton (Sokoloff 1969, Moll, 1976; Meachim and Brooke, 1984, Ortner 2003a). Secondary OA, in contrast, occurs when an underlying local or systemic factor is found to be the cause of onset, such as wear and tear, trauma, or congenital abnormalities (Sokoloff 1969, Mann and Murphy, 1990, Ortner 2003a). Secondary OA usually manifests itself at an early age and is isolated among the joints it affects (Sokoloff 1969, Moll, 1976, Mann and Murphy, 1990, Ortner 2003a). Examples of secondary OA include, but are not limited to: inflammatory arthritis, congenital dislocation of the hip and subcapital femoral epiphysiolysis (Norman 1984). Cartilage and bone changes are similar for both primary and secondary OA (Meachim & Brooke, 1984; Mann and Murphy, 1990).

Prevalence

OA is not only the most common form of arthritis, but is one of the most widespread and ancient diseases (Wells 1964; Roney 1965; Sokoloff 1969; Steinbock 1976; Moskowitz 1984a; Norman 1984; Ortner 2003). Moodie (1923), Wells (1964) and Putschar (1965) reported it in dinosaurs and fossil animals in the form of OA of the synovial joints and vertebral osteophytosis. Vertebral osteophytosis was also documented in a Lower Miocene Cuban crocodile, as well as a cave bear and hyena from the Pleistocene (Wells 1964). The mandibular condyles of Neanderthals from Krapina, La Ferrassie, La Quina and La Chapelle-aux-Saints showed signs of osteoarthritis as well (Wells 1964).

OA has made quite an impact on human populations globally and temporally as well. Miles (1965) after examining individuals from the Mesa Verde site in Colorado found that not only were several young individuals afflicted by degenerative arthritis but that all but one individual over the age of 35 had significant degenerative arthritis (Miles 1965). The joints most frequently involved in the present study, as well as more recent studies, include the spine, knee, shoulder and hip (Miles 1965; Kettelkamp & Colyer 1984). Roney (1965) also found OA to be associated with adulthood in a California population in which only individuals over the age of 20 had OA. In addition to this, Roney (1965) also found that of the 17 out of 27 adults that showed signs of arthritis 14 were males. The most involved vertebrae and joints included the thoracic and lumbar spines, the sacroiliac, and the elbow and knee joints. Roney (1965) notes *however* that some of these instances of arthritis were associated with trauma, recognized by the

presence of healed fractures. Ortner (1968) suggests that variation in the distribution and severity of OA is likely between and within populations due to differences in environmental stress and role playing. Thus the males, in Roney's (1965) population were perhaps more likely to hunt or get injured in battle. None the less, arthritis was not only shown to be correlated with advancing years or adulthood but also with sex in this and other studies (Miles 1965; Roney 1965; Norman 1984).

Weight-bearing joints such as the hip, knee and spine are most commonly affected by OA (Steinbock 1976; Moll 1976; Hoaglund 1976; Wood 1976, Ortner 2003b). Hip OA, also known as coxarthrosis, is the most common form of OA (Sokoloff 1969). A slightly higher incidence of OA is also found in males (Roney 1965, Steinbock 1976, Agha and Webb 2006). Females, however, appear to be afflicted more often with knee and hip OA than males (Radin 1976; Hoaglund 1976, Davis et al. 1991).

Up until 45 years of age, OA is globally rare and slightly more prevalent in males (Peyron 1984). The localization of OA in the knees of women seems to appear after age 45; the hip is statistically the joint to be involved latest. Between 45 and 55 years of age it appears to be equally frequent in both males and females, but after 55 years of age, it occurs more frequently in females (Peyron 1984). By 60 years of age the knee is usually afflicted in both sexes (Sokoloff 1969). OA not only tends to be more widespread and more severe in women age 50 and older, but the involvement of multiple joints is also more prevalent in women (Peyron 1984). Peyron (1984) also noted that 33-66% of individuals over the age of 35 in any given population will show signs of OA.

In the knee, the OA involvement of the medial tibiofemoral joint together with the patellofemoral joint is more frequent than OA involvement of all three joints of the knee (medial and lateral tibiofemoral joint and the patellofemoral joint) (Norman 1984). In addition, OA of the patellofemoral joint is just as common as OA of the medial tibiofemoral joint (Norman 1984). OA in the hip tends to occur in the perfoveal and the peripheral inferior region of the femoral head, with OA being most severe in the perfoveal region (Meachim et al., 1973, Jeffrey 1975).

Lastly, OA of the knee was found to be much more common among Jamaicans than White Europeans from Wensleydale. In contrast, hip OA was rare among Africans from the rubber plantation of Cavalla and the Igbo-Ora and Isheri villages of Liberia and Nigeria and Jamaicans but quite common among White Europeans from Wensleydale and Leigh (Lawrence & Sebo 1981).

Related Diseases

Other forms of arthritis include rheumatoid arthritis, vertebral osteophytosis, Gout, and Diffuse Idiopathic Skeletal Hyperostosis (DISH). Rheumatoid arthritis is an erosive disorder associated with rheumatism. It affects multiple joints, especially the hands and feet, and typically affects females (Bridges 1992, Ortnier 2003a). It is generally characterized by erosion and resorption of the articular surface or subchondral bone; eburnation is rare (Leisen, et. al 1991).

Vertebral osteophytosis also known as spondylosis deformans and hypertrophic spondylitis is characterized by degeneration of the spine. It involves both the true

synovial joints or apophyseal joints and the intervertebral joints. Intervertebral joints are secondary cartilaginous joints which lack a synovial membrane and thus are not true synovial joints (Steinbock 1976). Vertebral osteophytosis may affect any portion of the spine and is characterized most often by osteophyte development on the vertebral bodies (Steinbock 1976, Bridges 1992, Ortner 2003a).

Gout is an erosive arthropathy associated with excessive amounts of sodium urate (Ortner 2003a). Gout generally affects the hands and feet, most notably the big toe; it is characterized by pressure erosion (Steinbock 1976, Ortner 2003a). Diffuse Idiopathic Skeletal Hyperostosis (DISH) is yet another form of arthritis that tends to “produce excessive amounts of bone at joint margins and the entheses” (Ortner 2003a:559). It is most notable in the spine but does affect other joints in the skeleton (Ortner 2003a).

Chapter IV

Theoretical and Etiological Orientations of Osteoarthritis and Porosity

Osteoarthritis is, in spite of its wide distribution through time and space, still one of the most poorly understood diseases amongst clinicians, rheumatologists, and anthropologists alike (Moodie 1923, Wells 1964, Moskowitz et al. 1984, Hall 1995). Thus, this chapter is devoted to the theoretical and etiological postulations concerning the onset of OA and porosity. Biochemical and biomechanical theories, in addition to other theories regarding OA onset are addressed in brief. Following these sections are sections on the etiological orientations of clinicians, rheumatologists, and anthropologists concerning OA in general, and then concerning OA more specifically in the hip, knee, and in light of age. Theoretical and etiological orientations of clinicians, rheumatologists, and anthropologists are also addressed in regards to porosity's onset in osteoarthritic joints. The purpose of this chapter is to supply the reader with a general understanding of the theoretical and etiological debates among experts concerning OA and porosity.

Biochemical Theory

One major biochemical theory attributes primary OA to aging or senescence according to Jaffe (1972). The body, as it ages, gradually loses its ability to maintain joint cartilage. Joint cartilage becomes thin and discolored. It undergoes fibrillation and begins to crack. Articular cartilage begins to erode due to continued friction in the joint that acts to flake away the already fibrillated cartilage. The lack of joint integrity due to

increasing age enables stress to injure the joint; inflammation occurs in the synovium which in turn responds by releasing enzymes and inflammatory chemicals that further attack the cartilage. Other factors that influence the onset of primary OA are occupation, daily activities, stance and balance (Jaffe 1972).

Biomechanical Theory

The biomechanical theory of OA emphasizes loading stresses as the main factor leading to cartilage degeneration (Radin 1984). Cartilage after enduring such attacks becomes soft, cracks and begins to flake (Kushner, et. al. 1984). Inflammation, as a result of this damage, chemically attacks the remaining cartilage ensuring a cycle of accelerated damage to the joint (Kushner, et. al. 1984).

In conjunction with these processes, changes in the underlying bone may arise. The destruction of cartilage exposes the underlying subchondral bone. This exposed bone, as a result of continued bone on bone friction (Steinbock, 1976) and extensive remodeling produces thick polished or eburnated bone resembling ivory (Meachim and Brooke 1984). Other areas of bone display osteoclastic or resorptive activity as demonstrated by the development of subchondral microcysts (pseudocysts), areas of rarefaction surrounded by rims of sclerotic new bone (Steinbock 1976), and small fractures (Steinbock 1976). Ossified outgrowths known as osteophytes develop on the joint margins as well as the joint surfaces (Kushner, et. al. 1984). It has been postulated that osteophytes, both peripheral and surface, develop as an attempt to favorably redistribute load across the joint after damage or complete destruction of articular

cartilage (Sokoloff 1969, Norman, 1984). They can however, according to Sokoloff (1969), hinder the joint mechanically and further impede the normal function of the joint.

The majority of the new bone formation however, takes place in the marrow beneath the erosions and is associated with sclerosis and extensive reorganization of the internal architecture of the epiphyses (Sokoloff, 1969); this is most readily detected by clinicians rather than anthropologists because clinicians have greater access to radiological technology. The bone changes mentioned above are utilized as identifying characteristics of OA by clinical investigators, rheumatologists and to some extent by anthropologists. In summary they include, narrowing of joint space (cartilage loss), soft tissue swelling (inflammation), osteophyte development, altered bone contour, subchondral bony sclerosis, eburnation, and cysts (microcysts/pseudocysts) (Hoaglund, 1976; Moll, 1976; Steinbock, 1976 Norman 1984). The same bone changes that occur in primary OA, according to this theory, are also believed to hold true for secondary OA.

Additional Theories

Howell (1984) attributes OA to three major theories concerning the biomechanical and inflammatory response of the body to a noxious agent. The first theory stresses the importance of physical forces and biomaterial failure of articular cartilage. The second emphasizes the failure of articular chondrocytes (cartilage cells) to respond in light of both degradation and repair. The third theory “considers bony remodeling, synovial responses, microfractures, vascular changes, and other

extracartilaginous factors as primary problems, with cartilage change being secondary” (Howell 1984:130-131).

Etiology of Osteoarthritis: General

The etiology of OA is unclear, poorly understood and diverse (Wells 1964; Howell 1984; Mann and Murphy 1990; Ortner 2003a). Systemic-predisposing factors such as age, sex, metabolism, nutrition, the inability of cartilage to repair itself, hormones (endocrine) and heredity, as well as mechanical-functional factors such as chronic or acute trauma, stress, wear and tear, and obesity, in addition to cold temperatures, dampness, and sunlight are amongst the suggested causes of OA (Wells 1964; Sokoloff 1969; Janssens 1970; Radin 1976; Steinbock 1976; Moll 1976; Kettelkamp & Colyer 1984; Mann and Murphy 1990; Jurmain, 1997; Ortner 2003a). Wells (1964) deems stress from forces exerted on joints during daily activities as the single most important factor in determining which bones will most likely be affected by OA. Jurmain (1977, 1980) on the other hand, suggests that OA must be explained by a multifactoral model including hereditary factors, endocrine agents, age and sex, in addition to functional stress. Both Wells (1964) and Jurmain (1990) also propose that OA is concomitant of the normal aging processes and the cumulative affect of stress on the joints over many years. Such stress is intensified by culturally patterned wear-&-tear factors (Jurmain 1990). Hence the arthritic mandibular condyles of some prehistoric Native American populations are indicative of the stress of chewing tough and grit-laden foods (Wells 1964).

Etiology of Hip Osteoarthritis

Hip OA is attributed to these two mechanical factors: (1) normal forces acting on an abnormal hip joint, e.g., a hip with congenital hip dysplasia, congenital dislocation of the hip, acetabular dysplasia, coxa valga, and/or coxa vara, or/and (2) increased forces acting on a normal hip joint (Stulberg 1984). Amstutz & Kim (1984) suggest that hip OA is usually secondary in nature. Hip OA in women is most often due to dysplasia of the hip joint (Stulberg 1984). In short, hip OA is due to abnormally matched forces resulting in an abnormal distribution of stress on the joint and ending in the manifestation of OA (Amstutz & Kim 1984).

Etiology of Knee Osteoarthritis

Knee OA has been attributed most often to the abnormal distribution of loads or force in an abnormally configured knee joint. Thus individuals having genu varum (bowleg deformity) or genu valgum (knock-knee deformity) are more likely to exhibit signs of OA due to the abnormal distribution of weight-bearing loads in such individuals (Kettelkamp & Colyer 1984). Flexion deformity in addition to fractures can also be the cause of knee OA (Kettelkamp and Jacobs 1972, Kettelkamp & Colyer 1984). Of course OA in the hip or knee due to any of the above abnormalities or ailments is secondary in nature, i.e., the cause is known. However Moskowitz (1984) and Bluestone (1984) suggest that most cases of hip and knee OA are primary in nature, in which case there is no known underlying cause.

Etiology: Osteoarthritis and Aging

OA is repeatedly cited as being concomitant of age, a disease of advancing years, and correlated with age (Sokoloff 1969, Moskowitz 1984, Ortner 2003a). Whether OA is just correlated with age or is actually apart of the normal aging process is debatable (Sokoloff 1969, Mann and Murphy 1990). It is, however, important to mention the unique relationship shared between OA and aging. Aging is either viewed as the cumulative acquisition of injuries of extrinsic origin, or as a property governed by genetics that causes progressive deterioration of the body in the absence of any known cause (Sokoloff 1969). The concept of OA as an ailment of physiological aging or senescence suggests that during the process of aging there is “(1) loss of some genetically governed, self-replication capacity of the chondrocytes; and (2) physiochemical changes in the intercellular matrix of the [joint] tissue” (Sokoloff 1969: 25). According to Sokoloff (1969), OA ought to be viewed as the result of interaction between many variables both genetic and accidental, with age as a factor in that interaction.

Moskowitz (1984a) further suggests that OA is not totally dependent upon age but instead is the result of years of exposure to pathophysiological processes that took place earlier in life. OA is the result of definable alterations in biomechanical and biochemical mechanisms (Moskowitz 1984b). It ensues when degenerative changes exceed the rate at which cartilage can repair itself (Moskowitz 1984a). Various forms of OA likely represent subsets of the disorder, with major differences in the roles played by age, sex, biomechanical alteration, hormones, inflammatory mediators, and the immune process (Moskowitz 1984b).

Theoretical and Etiological Orientation of Porosity

Rheumatologists, clinicians and anthropologists alike have theorized as to the cause of porosity, yet there still exists no definite explanation as to the etiology of this destructive bone lesion. Ortner (1968) suggested that porosity was a vascular response of the subchondral bone to cartilage degeneration and bone changes. Sokoloff (1969) suggested that porosity is related to subchondral cysts (spheroid or pyriform defects of para-articular bone) which exist in a great deal of severely osteoarthritic joints, suggesting a relationship between porosity and OA severity. These cysts are not true cysts but rather polymorphous connective tissues bordered by thick rims of sclerotic new bone which replace trabeculae and marrow. Quite often the edges of such cysts abut upon the eroded articular surface. Smaller lesions of this sort, according to Sokoloff (1969), are to blame for the amount of porosity on some eburnated joint surfaces. Subchondral cysts are common in both the hip and knee (Sokoloff, 1969; Moll, 1976), implying the common occurrence of porosity in these joints as well.

Merbs (1983) also suggests that porosity reflects the occurrence of cysts that tend to open directly at the bony surface of the joint, making the bone appear porous. On the other hand, Merbs (1983) proposes that porosity may be attributable to abrasion, the result of bone on bone friction, and the exposure of the underlying trabecular bone. This seems to reinforce Ortner's (1968) assertion that eburnation rarely occurs without porosity, seeing that eburnation is the result of bone on bone friction.

Due to findings showing the occurrence of porosity in habitually non-contact areas (areas that only come into contact with forces during extreme motions), Woods

(1995) proposes the following theory concerning porosity. The constant application and transmission of magnitude joint forces of approximate body-weight are necessary for synovial fluid to be thoroughly pumped throughout the collagen medium. When adequate replenishing of synovial fluid does not occur, localized areas of malnourished cartilage develop. This malnourished cartilage is prone to failure under loads as a result of deficiencies in the tensile strength of its individual collagen fibers. It soon generates a reaction from the trabecular beds beneath the subchondral bone, which initiates vascular invasion through the subchondral bone into deep layers of calcified cartilage. Small blood vessels penetrate the cartilage/subchondral bone boundary producing channels that provide ports through which synovial fluid is pumped into the trabecular beds beneath the subchondral bone. Remodeling of the trabecular and subchondral bone matrix occurs in localized areas, initiating the phenomenon known as porosity.

It is also important to note that occasionally high magnitude joint loads, in combination with cartilage malnutrition and diminished structural properties are necessary to cause porosity within habitually non-contact areas (Woods 1995).

Chapter V

Biomechanics of the Hip, Knee, and Osteoarthritis

The current chapter outlines how the biomechanics of articular joints, more specifically the biomechanics of the hip and knee, play a role in the onset and distribution of OA and OA lesions. The first section gives a brief overview of the biomechanics of articular joints. Sections on the biomechanics of the hip and hip OA and on the biomechanics of the knee and knee OA follow.

Articular Joints

All articular joints including the hip and the knee are covered with articular cartilage. Articular cartilage is a dense, white fibrous tissue that provides protective lubrication to the joint (Hall 1995). It acts to transmit load and provide a maximum surface contact area for the joint (Radin 1984). Cartilage also acts to reduce friction and wear at the joint during movement (Nordin and Frankel 1989).

Articular cartilage is not in direct contact with the underlying subchondral bone; instead it has a calcified base that rest directly on the subchondral bone. Synovial fluid envelops articular cartilage and serves as a source of nutrition to the cartilage (Radin 1984). A fibrous articular capsule enfolds the entire joint space (Hall 1995). The synovium is the innermost lining of the joint fibrous capsule. It acts as a filter for transmission of materials into the synovial fluid (Radin 1984).

Menisci, articular fibrocartilage that intervenes between articulating bones, also act to distribute load and absorb shock, particularly in the knee joint (Radin 1984, Hall

1995). Ligaments, tendons, and muscles provide joint stability (Radin 1984, Hall 1995). When articular cartilage is worn away OA ensues (Radin 1984, Hall 1995, Ortnier 2003a).

Biomechanics of the Hip and Hip Osteoarthritis

The hip is a proximal appendicular ball-and-socket joint that is “primarily concerned with stance and in serving as the fulcrum for the body weight during those moments of single-leg stance that occur during walking” (Radin 1984:94). The head of the femur is the ball and it forms two thirds of a sphere, all of which is contained by the acetabulum (Norman 1984, Hall 1995). The acetabulum makes up the concave socket in which the head of the femur resides (Hall 1995). The articular surfaces of the femur and acetabulum are congruent in the normal hip (Norman 1984).

In the acetabulum cartilage is thickest at the peripheries, this is not so for the femoral head (Hall 1995). On the femoral head cartilage is thickest at the weight-bearing segment lateral to the fovea centralis (capitis) (Norman 1984). Cartilage in the femur measures 4mm at the weight-bearing segments and decreases gradually to 1-2mm at the periphery (Norman 1984).

The stability of the hip is maintained by several strong ligaments which act to rotate the femur head about the acetabulum. Muscles of the hip are responsible for flexion, extension, abduction, and adduction. Each hip supports one-half the body weight above the hip or about one-third of the total body weight when standing in an upright position where upper body weight is equally dispersed across each leg. “However, the total load on each hip in this situation is greater than the weight supported because

tension in the large, strong hip muscles further adds to compression at the joint” (Hall 1995:228)

When stresses endured in the joint exceed its strength, mechanical failure ensues. Such applied stress can be the result of a single excessive peak dynamic stress or recurring lower stresses that exceed the endurance limit (Amstutz and Kim 1984). On the other hand, a lack of stress applied to the joint could also result in mechanical failure. Ailments due to the lack of stress in the joint are disuse atrophy and stress shielding (Amstutz and Kim 1984). In essence there must be equilibrium between the stress received by a joint and its strength in order to maintain normal joint functioning.

Yoshida et al., (2006) found that peak pressure in the hip during daily activities involving fast, normal and slow walking, standing up and sitting down, knee bending and going up and down stairs were situated on the lateral roof and posterior horn of the acetabulum. Accordingly, these areas should be areas where eburnation and marginal osteophytes (as opposed to surface osteophytes) are most prevalent and where porosity is rare, seeing that these areas would be defined as contact areas according to Woods (1995). Also, the perifoveal region of the femoral head, a non-contact area, tends to be an area of frequent osteophytic development (Jeffrey 1975).

Biomechanics of the Knee and Knee Osteoarthritis

The knee is a middle appendicular linkage or hinge joint which is also structured for weight bearing and locomotion (Radin 1984, Hall 1995). It is composed of the medial tibiofemoral joint, lateral tibiofemoral joint, and patellofemoral joint. The medial

condyles of the tibia and femur articulate to form the medial tibiofemoral joint, the lateral condyles of the tibia and femur articulate to form the lateral tibiofemoral joint, and articulation between the patella and the femur form the patellofemoral joint (Hall 1995). The medial and lateral tibiofemoral joints enable the knees to rotate axially (Radin 1984).

All of the articulating surfaces of the knee are covered with articular cartilage. Cartilage is thickest in the patellofemoral joint (Sokoloff 1969). The menisci, fibrocartilaginous discs located between the tibial and femoral condyles help to absorb shock and distribute load across the knee (Hall 1995). Menisci also provide stability for the joint (Radin 1984). Hinge joints would have small articular cartilage contact areas if it were not for menisci (Radin 1984). Injury or loss of the menisci increases the likelihood of one developing OA (Norman 1984). Numerous ligaments and muscles also aid in maintaining stability in the knee (Hall 1995).

The patella acts to increase the contact area between the patellar tendon and the femur, which in turn decreases patellofemoral joint contact stress. The patella also increases the leverage of the quadriceps muscle and affords some protection for the anterior aspect of the knee (Radin 1984, Hall 1995).

The maximum joint force at the knee ranges between two and four times the body weight (Radin 1984). Joint forces at the medial and lateral tibiofemoral joints range from three times the body weight when standing to about four times the body weight when climbing up stairs. When standing the bulk of the load is borne by the medial tibial plateaus, while the smaller loads inflicted throughout the swing phase are borne by the tibial plateaus (Morris 1970). Joint force at the patellofemoral joint ranges from one-half

of the body weight when walking to more than three times the body weight during stair climbing (Reilly and Martens 1972).

OA in the knee appears quite often on the articular surfaces of the patella because the patella undergoes exceedingly high load amounts when the knee is flexed in the squatting position (Steinbock 1976). Also in the knee, cysts lie under the pressure zone while osteophytes tend to occur marginally to weight bearing areas and appear more prominent when joint space narrowing is greatest (Norman 1984).

The knee is also a commonly reported site of porosity (Ortner 1968, Woods 1986, Woods 1995, Rothschild 1997). Porosity is most prevalent on non-contact areas of the knee such as the posterior most portions of the condyles, the medial and lateral aspects of the patellofemoral grooves, and the non-contact areas of the medial and lateral femoral condyles (Woods 1995). Osteophytes also tend to concentrate on non-contact areas such as the periphery of the joint surfaces. Eburnation, on the other hand, tends to occur most often on load bearing areas such as the medial and lateral facets of the patellofemoral surface of the knees, contact areas of the medial and lateral femoral condyles, and the medial and lateral tibial plateaus (Woods 1995).

Chapter VI

Identifying Criteria of Osteoarthritis

Criteria that clinicians, rheumatologists, and anthropologists use to identify OA vary. Ortner (2003b) suggests that this is due to differences in the data available to the clinician and rheumatologists as opposed to the anthropologist. Anthropologists are generally not exposed to clinical and rheumatologic criteria such as muscular atrophy, cartilage degradation, and loose bodies in the joint when identifying OA because in most cases they have only bones with perhaps a minimal amount of soft tissue. The following sections highlight the reasons for porosity's inclusion or lack of inclusion as an identifier of OA and give a detailed description of the criteria used to identify OA by anthropologists, clinicians, and rheumatologists.

Considerations of Porosity

Of the identifying characteristics significant to OA, porosity's significance has yet to be explored in great detail. Porosity is accepted anthropologically as an identifying characteristic of OA perhaps because of its common association with two clinically (as well as anthropologically) accepted OA criteria, osteophyte development and eburnation (Woods 1995, Rothschild 1997). Porosity itself is not, for the most part, accepted as an identifying characteristic of OA by clinicians and rheumatologist (Woods, 1995; Altman et al. 1986, 1991), although Sokoloff (1969) and Steinbock (1976), both clinicians, mentioned it.

The rejection of porosity by clinicians is perhaps due to the fact that osteophyte development and eburnation are more visible radiographically, whereas porosity is not recognized radiographically until it produces bone density changes greater than 30-50% as required for radiological detection, and because most porous changes are less than 30-50% detection is unlikely clinically (Ortner 1991, Woods 1995).

Clinical, Rheumatologic & Anthropological Criteria

Criteria used by clinicians and rheumatologists to identify OA can be seen in Table 1. Most anthropologists identify OA by the presence of one or more of the following characteristics: (1) eburnation, (2) marginal/peripheral osteophytes, (3) surface osteophytes (4) hypertrophic bone buildup, (5) surface porosity, and (6) subchondral cyst (Wells 1964, Roney Jr. 1965, Mann and Murphy 1990, Ortner 2003a).

Changes that typically occur in both the OA afflicted hip and knee follow. Eburnation, porosity and marginal osteophytes frequent innominates. Marginal osteophytes are also one of the first indicators of hip OA (Carter 2003b). Innominates may also exhibit deepening of the socket, acetabular roof sclerosis and in some cases cystic cavities (Ortner 2003a). The femur head typically exhibits periarticular bone, flattened areas of bone where degeneration and resorption have occurred, subchondral cysts, surface osteophytes, marginal osteophytes, and surface porosity (Meachim and Brooke 1984, Norman 1984, Chew 1989). In the knee, the distal femur exhibits both marginal and surface osteophytes along with surface porosity (Meachim and Brooke 1984, Norman 1984, Chew 1989). The proximal tibia exhibits tibial spine spiking,

surface osteophytes, eburnation, and marginal osteophytes (Meachim and Brooke 1984, Norman 1984). Peripheral osteophytes, surface osteophytes, and surface porosity occur most often on the OA afflicted patella (Meachim and Brooke 1984, Norman 1984).

The femur is rarely afflicted with eburnation, with the exception of elderly individuals (Jaffe 1972, Meachim and Brooke 1984, Norman 1984). Flattened areas of bone on the femur head, subchondral cysts, along with surface and marginal osteophytes also frequent the femur head in elderly individuals (Jaffe 1972, Meachim and Brooke 1984, Norman 1984). The most severe hip OA changes occur on the femoral head. These changes include eburnation, erosion, osteophytes, and marginal exostoses (mushroom deformity), which may hang over the femoral neck (Ortner 2003a).

Table 1: Clinical and Rheumatological Criteria for Identifying Osteoarthritis				
General Criteria		Hip Criteria		Knee Criteria
1	Cartilage degeneration		Cartilage degeneration	Cartilage degeneration
2	Subchondral bone sclerosis		Subchondral bone sclerosis	Subchondral bone sclerosis
3	Subchondral cysts		Subchondral cysts	Subchondral cysts
4	Osteophytes		Osteophytes	Osteophytes
5	Osteoclastic resorption		Osteoclastic resorption	Osteoclastic resorption
6	Eburnation		Eburnation	Eburnation
7	Loose bodies in the joint			Loose bodies in the joint
8	Tiny irregular pits			Tibial spines
9	Bony enlargements			Joint effusions
10	Gross deformity with subluxation			Thickened synovium
11	Joint surface remodeling			Joint capsule tenderness
12	Osteochondrophytes			
13	Cartilage proliferation			
14	Subchondral bone collapse			
15	Muscle atrophy			
16	*Porosity			

*Note: Porosity was mentioned by Sokoloff (1969) and Steinbock (1976) both of whom are clinicians. (Steinbock 1976, Sokoloff 1969, Janssens 1970, Adams 1976, Hoaglund 1976, Hoaglund 1976, Moll 1976, Radin 1976, Moskowitz 1984a, Moskowitz 1984c, Norman 1984, Amstutz and Kim 1984).

Now that criteria accepted by clinicians, rheumatologists and anthropologists for OA identification has been addressed the current research can proceed in its examination of one debated criterion, porosity, and its relationship to OA. Rothschild's (1997) assertion that porosity is not related to OA and should thus be excluded as an identifying characteristic of OA prompted this research. For it is imperative that paleopathologists have a good understanding of what criteria characterize OA in order to advance in better understanding the OA disease process, its prevalence among distinct populations and its relationship to varying lifestyles. Though a number of researchers have hinted at or explored the relationship of porosity to eburnation (Ortner 1968, Merbs 1983, Woods 1995) only Rothschild (1997) explored, directly, the relationship of porosity to OA. Rothschild (1997), however, like other anthropologists, is also subject to biases on what characterizes OA, seeing that in his research OA was identified solely by the presence of osteophytes. In addition, Rothschild (1997) only investigated the knee joint. Thus, the current research seeks to address the relationship of porosity to OA as it is defined by osteophytes alone (method I), eburnation alone (method II), and then by both osteophytes and eburnation (method III). In addition, this research not only examines the knee but also the hip in order to determine whether or not porosity's relationship to OA will be expressed similarly in different joint types (i.e. hinge (knee) and ball-and-socket (hip)).

Chapter IV

Materials and Methods

Materials

In order to carry out the present research, severity data concerning osteophytes, eburnation, and porosity were recorded from the hips and knees of 231 individuals 35 to 101 years of age in the William M. Bass Donated Skeletal Collection at the University of Tennessee, Knoxville. The Bass collection was initiated in 1981 by Dr. William M. Bass. It is a contemporary collection made up of primarily donated individuals with the exception of unclaimed individuals from Medical Examiner Offices. The primary objective of this collection is to establish a skeletal collection of modern individuals with known demographic information. The number of individuals examined for this study is displayed in Table 2. This sample included African American males, European American females, and African American males. There were only 5 African American females in this collection at the time of the examination; the sample was thus too small to be representative of the population. The sample under examination includes the most complete and best preserved individuals in order to avoid over estimating the presence of OA.

Individuals were excluded from the sample if taphonomic changes were extensive, in which case the bulk or all of the articulating ends of the hip and knee were eroded or endured extensive postmortem damage. Individuals were also excluded in cases where soft tissue covered most or all of the knee or hip joint. As mentioned earlier, this investigation is not concerned with whether or not an individual had secondary or

primary OA. However, individuals exhibiting obvious cases of secondary OA were excluded in order to prevent inflation with regards to the prevalence and distribution of OA. Individuals suffering from osteoporosis were not excluded from the sample, although some have proposed an inverse relationship between OA and osteoporosis (Cooper et al., 1991, Hart et al., 1994). Lastly, some of the earliest individuals, who were shellacked for preservation purposes, were also excluded from the investigated sample because shellacking made one or more of the identifying criteria indiscernible.

Table 2: Demography of Individuals Analyzed from the William M. Bass Donated Skeletal Collection

Age	*EA Females	*AA Males	*EA Males
35-44	5	6	17
45-54	14	7	23
55-64	22	5	28
65-74	21	5	17
75-84	16	3	16
85-94	9		14
95-101			3
Total	87	26	118

*Note: AA denotes African American.

*Note: EA denotes European American.

Individuals 35 years and older were chosen because studies have shown that in any population over age 35 OA should be present in 1-2 out of every 3 individuals (Miles 1965, Peyron 1984). The hip and knee joints are utilized not only because they are the most commonly reported sites of OA (Hoaglund 1976, Bridges 1992) but also because OA generally affects weight bearing joints (Steinbock 1976, Mann and Murphy 1990). The knee is the most involved large joint, and OA lesions in this joint appear at a much younger age than in any other joint (Jurmain 1990, Sokoloff 1969). It is also a commonly reported site of porosity (Ortner 1968, Woods 1986, Woods 1995, Rothschild 1997). The inclusion of the hip, due to its prominence as a skeletal region affected by OA, in this study adds diversity to the previous studies by allowing the question of porosity's significance to be extended to yet another joint with similarities (weight-bearing) and differences (ball-and-socket vs. hinge). In short the hip and the knee were chosen because of their prominence as skeletal regions affected by OA.

The hip and knee are also both bilaterally involved joints (Jurmain 1980, 1997). Bilaterally involved joints are best suited for this analysis because they suggest that the forces applied to one side of the body are also applied to the other side of the body. Hence, if there is a lot of bilateral involvement amongst individuals in the Bass collection then these joints are perhaps less likely to be attributed to secondary OA.

Differences in the distribution and severity of OA lesions in the hip and knee are expected because the knee and hip are subject to different weight bearing loads (Engin 1974, Bergman 2001, Yoshida et al., 2006). Such differences, however, would not likely be attributed to weight vs. non-weight bearing, but instead to the type of joint, ball and

socket vs. hinge. Age effects are also more pronounced in the hip than in the knee (Jurmain 1977, Jurmain 1980).

Methods

The hips and knees of all the individuals herein were examined thoroughly over a period of eight months. Three surfaces of the hip for both the right and left sides were examined. These surfaces include the lunate surface of the acetabulum, femoral head, and perifoveal region of the femoral head (Figure 2). The perifoveal region of the femoral head was examined separately because this region is known to only come into contact with the acetabulum during extremes of daily walking (Nordin and Frankel 1980a). Such areas have tended to be areas of frequent osteophytic development (Jeffrey 1975, Woods 1995). Inclusion of this region with the overall femoral head region would over estimate the prevalence and severity of OA on the femoral head, hence the separation of the two regions.

Eight articular surfaces of both the left and right knees were examined for this study. These surfaces include the lateral patellar articular facet and medial patellar articular facet, lateral patellofemoral articular surface, medial patellofemoral articular surface, lateral femoral condyle, medial femoral condyle, lateral tibial plateau, and medial tibial of both the left and right knee joints (Figure 3). A total of sixteen joint surfaces were examined for the knees with eight on each side.

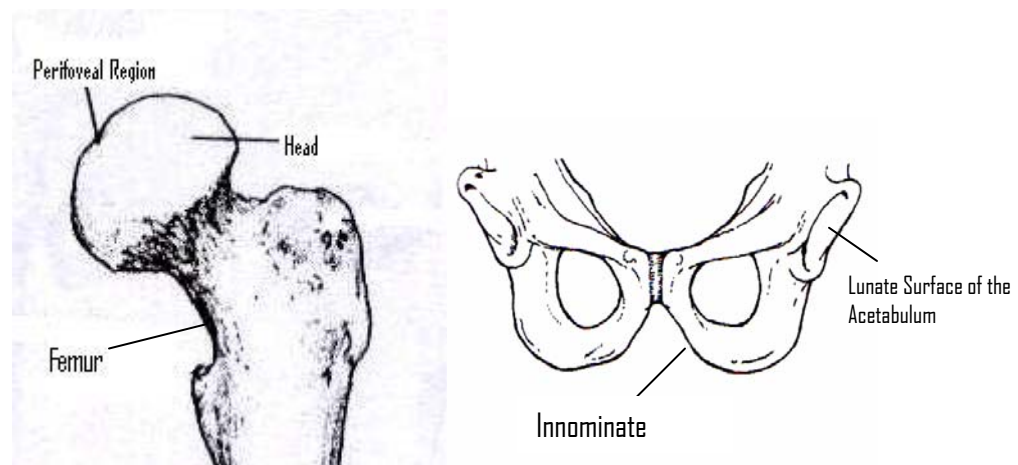


Figure 2: Hip Joint Surfaces Examined (from Bass 1995, Hall 1995).

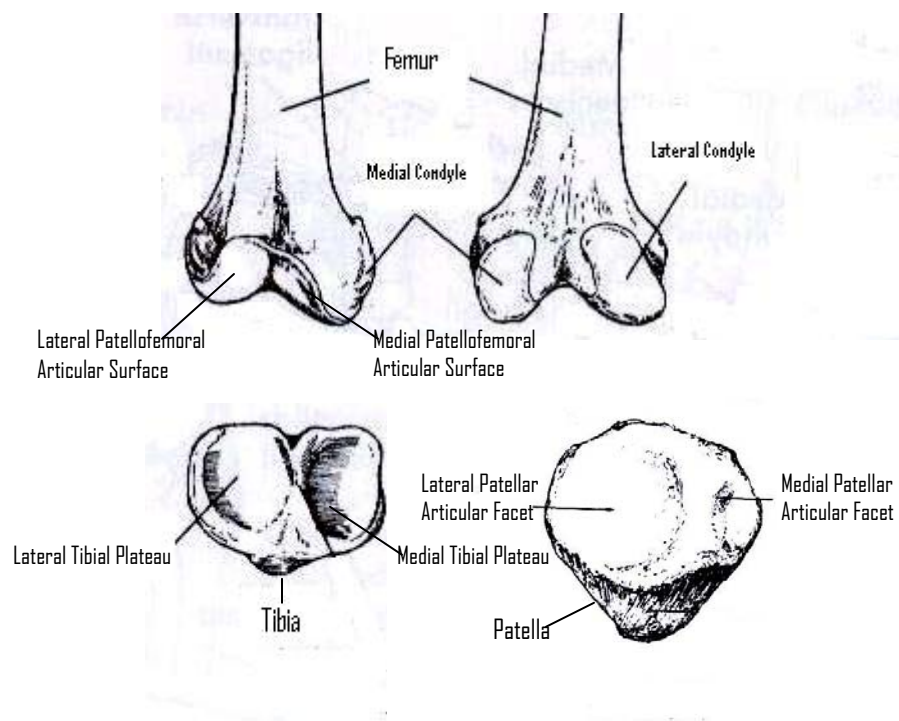


Figure 3: Knee Joint Surfaces Examined (from Bass 1995, Hall 1995).

Every surface was examined for the presence of osteophytic lesions, eburnation and porosity. Hypertrophic bone formation along with tibial spines was included under osteophytic development. All three pathological lesions were rated on a scale of severity that included absent (0), trace (1), slight (2), moderate (3), and severe (4). Ortner (2003a) also used this same coding system when identifying OA severity. Absent for all three lesions meant that there was no trace of any osteoarthritic activity on a joint surface (Figures 4 & 5; Appendix A). Trace for all three lesions meant that a trait was barely discernable regardless of its surface coverage (Figures 4, 5, 6, 7 & 8; Appendix A). Slight was expressed by the presence, but not prominence, of a particular trait on less than 1/3 of the surface and/or periphery of the joint (Figures 9, 10, 11 & 12; Appendix A). Moderate was expressed by the expression of a prominent trait on more than 1/3 but less than 2/3 of the joint surface and/or periphery (Figures 8, 12, 13 & 14; Appendix A). Severe lesions occurred when the joint surface and/or periphery were more than 2/3 covered by a discernable trait and/or a surface exhibited extensive development of the trait (Figures 6, 7, 8, 14 & 15; Appendix A).

Osteophytes were recognized by the presence of new bone growth along the margin or surface of the joints which includes what Ortner (2003a) calls hypertrophic bone buildup (Moll 1976, Merbs 1983, Mann and Murphy 1990, Ortner 2003a). Eburnation was recognized by the presence of polished, ivory like bone (Merbs 1983, Mann and Murphy 1990, Ortner 2003a), which results from continued friction between sclerotic subchondral bones after complete cartilage destruction (Steinbock 1976).

Porosity was recognized by the presence of discontinuity (pitted, disorganized lesion) of subchondral bone in the absence of erosive processes (Rothschild 1997).

OA severity levels are analyzed in regards to age, sex, ancestral group, and joint type. Porosity severity levels are analyzed in light of OA severity levels for sex, ancestral, and joint groups. OA is examined using three distinct methods to determine how the use of different methods might affect the outcome of results and hinder comparability. The first method identifies OA on the basis of osteophytes alone; the second method identifies OA by the presence of eburnation alone; and the third method identifies OA on the basis of both osteophytes and eburnation. Porosity is not used as a criterion for identifying OA because it is the character being analyzed. Also it must be noted that the severity score, when using both osteophytes and eburnation to identify OA, was determined by selecting the highest severity score of the two lesions. For instance, if osteophytes on a particular joint surface were given a severity score of 3 for moderate and eburnation on that same joint surface was given a score of 2 for slight that particular joint surface would receive a severity score of 3 for moderate.

All of the statistics in this research were done using SAS 9.1.3 (2002-2003). Fisher's Exact tests were utilized to determine if there were significant differences in porosity severity scores across each level of severity for method I, method II, and method III. Fisher's Exact tests are especially appropriate for cells with small cell frequencies or when sample sizes are small, both of which are the case in this analysis. A p-value of less than or equal to 0.05 was indicative of significant differences in porosity severity scores across OA severity scores for whichever method was used. This indicated that a

significant relationship existed between porosity severity and OA severity for whichever method was used. This research is not only interested in documenting changes in the relationship of porosity severity and OA severity for each method, but this research also seeks to determine whether the relationship between porosity severity and OA severity appears to be consistently significant for either of the three methods. The latter information would act to support or refute Rothschild's assumption that porosity is not significantly correlated with OA and thus should be deleted as an identifying characteristic of OA.

Chapter V

Results

As stated earlier Fisher's Exact tests were utilized to determine if there were significant differences in porosity severity scores across each level of severity for method I, method II, and method III. In the event that a method is invariant for severity no test will be listed for Fisher's Exact test. The Fisher's Exact Test tables for all but the left perfoveal region of the hip (Table 3) and the left medial patellar articular facet of the knee (Table 9) are in appendix B. Tables containing data on the prevalence of OA lesions and cross tabulations are in Appendix C. Further results are presented by hip surfaces then knee surfaces. Also, from this point on the use of osteophytes alone to identify OA will be referred to as method I; the use of eburnation alone to identify OA will be referred to as method II; and the use of both osteophytes and eburnation to identify OA will be referred to as method III.

Hip

Left Perifoveal Region

Fisher's Exact test of the relationship between porosity severity and osteophyte severity is only present for method I in European American females and African American males because eburnation (method II) is invariant (Table 3). In European American males the relationship between porosity severity and osteophyte severity is the same for all three methods (Table 3). Porosity severity is significantly related to OA

severity for method I in African American males and for all three methods in European American males (Table 3).

Right Perifoveal Region

The relationship between porosity severity and osteophyte severity is the same for all three methods in European American females and African American males (Table 4). In European American males only the Fisher's Exact test of the relationship between porosity severity and osteophyte severity for method I is present because eburnation is invariant (Table 4). Porosity severity is not significantly related to OA severity for any of the methods in European American females, African American males, and European American males (Table 4).

Table 3: Fisher's Exact Test for the Left Perifoveal Region Showing the Relationship Between Porosity Severity Scores and each Method

Group	Method	Table Probability	P-Value	n=
*EA Females:	Osteophytes	4.39E-05	0.0894	77
*AA Males:	Osteophytes	1.22E-04	0.0292	24
*EA Males:	Osteophytes	5.66E-11	0.0016	108
	Eburnation	0.0467	0.0467	107
	Osteophytes and Eburnation	3.78E-12	2.02E-04	108

*Note: AA denotes African American.

*Note: EA denotes European American.

Left Femoral Head

The relationship between porosity severity and osteophyte severity is the same for all three methods for European American females and European American males (Table 5). In African American males the relationship between porosity severity and osteophyte severity is the same for methods I and III but different for method II (Table 5). Porosity severity is significantly related to OA severity for method II in African American males and for all three methods in European American males (Table 5).

Right Femoral Head

The relationship between porosity severity and osteophyte severity is the same for all three methods in European American females (Table 6). In African American males and European American males only the Fisher's Exact test of the relationship between porosity severity and osteophyte severity for method I is present because eburnation (method II) is invariant (Table 6). Porosity severity is not significantly related to OA severity for any of the methods for European American females, African American males, and European American males (Table 6).

Left Lunate Acetabular Surface

The relationship between porosity severity and osteophyte severity is the same for all three methods in European American females (Table 7). In African American males and European American males only the Fisher's Exact test of the relationship between porosity severity and osteophyte severity for method I is present because eburnation

(method II) is invariant (Table 7). Porosity severity is significantly related to OA severity for method I in European American males (Table 7).

Right Lunate Acetabular Surface

The relationship between porosity severity and osteophyte severity is the same for all three methods in European American females and African American males (Table 8). In European American males only the Fisher's Exact test of the relationship between porosity severity and osteophyte severity for method I is present because eburnation (method II) is invariant (Table 8). Porosity severity is not significantly related to OA severity for any of the methods in European American females, African American males, and European American males (Table 8).

Knee

Left Medial Patellar Articular Facet

The relationship between porosity severity and osteophyte severity is the same for method I and method III in European American females; this relationship changed when using severity data from method II to identify OA on this surface (Table 9). In African American males only the Fisher's Exact test of the relationship between porosity severity and osteophyte severity for method I is present because eburnation (method II) is invariant (Table 9). In European American males the relationship between porosity severity and osteophyte severity is the same for method I and method II but different for method III (Table 9). Porosity severity is significantly related to OA severity for

Table 9: Fisher's Exact Test for the Left Medial Patellar Articular Facet Showing the Relationship Between Porosity Severity Scores and each Method

Group	Method	Table Probability	P-Value	n=
*EA Females:	Osteophytes	5.99E-07	0.0586	71
	Eburnation	0.1449	0.7465	71
	Osteophytes and Eburnation	5.99E-07	0.0586	71
*AA Males:	Osteophytes	1.84E-04	0.651	24
*EA Males:	Osteophytes	4.06E-08	0.1503	99
	Eburnation	0.0071	0.0788	100
	Osteophytes and Eburnation	4.16E-09	0.0504	99

*Note: AA denotes African American.

*Note: EA denotes European American.

method I and III in European American females and for method III in European American males (Table 9).

Left Lateral Patellar Articular Facet

The relationship between porosity severity and osteophyte severity is the same for methods I and III but different for method II in European American females (Table 10). In African American males the relationship between porosity severity and osteophyte severity is the same for all three methods (Table 10). In European American males the relationship between porosity severity and osteophyte severity was the same for method II and method III but different for method I (Table 10). Porosity severity is significantly

related to OA severity for methods I and III in European American females and for methods II and III in European American males (Table 10).

Right Medial Patellar Articular Facet

The relationship between porosity severity and osteophyte severity is the same for all three methods in European American females, African American males, and European American males (Table 11). In European American males porosity severity and osteophyte severity are significantly related for all three methods (Table 11).

Right Lateral Patellar Articular Facet

The relationship between porosity severity and osteophyte severity is the same for all three methods in European American females and European American males (Table 12). In African American males only the Fisher's Exact test of the relationship between porosity severity and osteophyte severity for method I is present because eburnation (method II) is invariant (Table 12). Porosity severity is significantly related to OA severity for all three methods in European American males (Table 12).

Left Medial Patellofemoral Surface

The relationship between porosity severity and osteophyte severity is the same for methods I and III but different for method II in European American females (Table 13). In African American males only the Fisher's Exact test of the relationship between porosity severity and osteophyte severity for method I is present because eburnation

(method II) is invariant (Table 13). In European American males the relationship between porosity severity and osteophyte severity is the same for all three methods (Table 13). Porosity severity is significantly related to OA severity for methods I and III in European American females and all three methods in European American males (Table 13).

Left Lateral Patellofemoral Surface

The relationship between porosity severity and osteophyte severity is the same for methods I and III but different for method II in European American females and African American males (Table 14). In European American males the relationship between porosity severity and osteophyte severity is the same for all three methods (Table 14). Porosity severity is significantly related to OA severity for methods I and III in European American females and in African American males (Table 14).

Right Medial Patellofemoral Surface

The relationship between porosity severity and osteophyte severity is the same for methods I and III in European American females and European American males; this relationship changed when using method II to identify OA severity (Table 15). In African American males only the Fisher's Exact test of the relationship between porosity severity and osteophyte severity for method I is present because eburnation (method II) is invariant (Table 15). Porosity severity is significantly related to OA severity for methods I and III in European American females and in European American males (Table 15).

Right Lateral Patellofemoral Surface

The relationship between porosity severity and osteophyte severity is the same for methods I and III in European American females and European American males; this relationship changed when using method II to identify OA severity (Table 16). In African American males only the Fisher's Exact test of the relationship between porosity severity and osteophyte severity for method I is present because eburnation (method II) is invariant (Table 16). Porosity severity is significantly related to OA severity for methods I and III in European American females and European American males (Table 16).

Left Medial Femoral Condyle

The relationship between porosity severity and osteophyte severity is the same for all three methods in European American females and African American males (Table 17). In European American males the relationship between porosity severity and osteophyte severity is the same for method I and method III but different for method II (Table 17). Porosity severity is significantly related to OA severity for methods I and III in European American males (Table 17).

Left Lateral Femoral Condyle

The relationship between porosity severity and osteophyte severity is the same for methods I and III in European American females; this relationship changed when using method II to identify OA severity (Table 18). In African American males only the Fisher's Exact test of the relationship between porosity severity and osteophyte severity

for method I is present because eburnation (method II) is invariant (Table 18). In European American males the relationship between porosity severity and osteophyte severity is the same for all three methods (Table 18). Porosity severity is significantly related to OA severity for method II in European American females and for all three methods in European American males (Table 18).

Right Medial Femoral Condyle

The relationship between porosity severity and osteophyte severity is the same for all three methods in European American females and African American males (Table 19). In European American males the relationship between porosity severity and osteophyte severity was the same for method I and method III but different for method II (Table 19). Porosity severity is significantly related to OA severity for methods II in European American males (Table 19).

Right Lateral Femoral Condyle

The relationship between porosity severity and osteophyte severity is the same for all three methods in European American females and African American males (Table 20). In European American males the relationship between porosity severity and osteophyte severity is the same for method II and method III but different for method I (Table 20). Porosity severity is significantly related to OA severity for methods II and III in European American males (Table 20).

Left Medial Tibial Plateau

The relationship between porosity severity and osteophyte severity is the same for all three methods in European American females and European American males (Table 21). In African American males the relationship between porosity severity and osteophyte severity is the same for method II and method III but different for method I (Table 21). Porosity severity is significantly related to OA severity for method I in African American males and all three methods in European American males (Table 21).

Left Lateral Tibial Plateau

The relationship between porosity severity and osteophyte severity is the same for all three methods in European American females (Table 22). In African American males only the Fisher's Exact test of the relationship between porosity severity and osteophyte severity for method I is present because eburnation (method II) is invariant (Table 22). In European American males the relationship between porosity severity and osteophyte severity is the same for method I and method III; this relationship is different when using method II to identify OA severity (Table 22). Porosity severity is significantly related to OA severity for methods I and III in European American males (Table 22).

Right Medial Tibial Plateau

The relationship between porosity severity and osteophyte severity is the same for all three methods in European American females, African American males, and European

American males (Table 23). Porosity severity is significantly related to OA severity for all three methods in European American males (Table 23).

Right Lateral Tibial Plateau

The relationship between porosity severity and osteophyte severity is the same for methods I and III in European American females; this relationship is different when using method II to identify OA severity (Table 24). In African American males only the Fisher's Exact test of the relationship between porosity severity and osteophyte severity for method I is present because eburnation (method II) is invariant (Table 24). In European American males the relationship between porosity severity and osteophyte severity is the same for all three methods (Table 24). Porosity severity is significantly related to OA severity for method II in European American females and for all three methods in European American males (Table 24).

Chapter VI

Discussion

The purpose of the present research was to determine porosity's relationship to OA as OA was defined using three distinct methods. To reiterate, method I involved the use of osteophytes alone to identify OA, method II involved the use of eburnation alone to identify OA, and method III involved the use of both osteophytes and eburnation to identify OA. Using data from the William M. Bass Donated Skeletal Collection, the present research found that porosity appeared to be significantly related to OA, regardless of the method used to identify OA, in the left hip joints of African and European males. There were no significant correlations between porosity severity and OA severity for European American females in the hip. The only significant relationships between OA severity and porosity severity were amongst African American males for the left perifoveal region and left femoral head and amongst European American males for the left perifoveal region, left femoral head, and the left lunate acetabular surface (Tables 1, 5 & 9). There appears to be a sex and side bias in males. Perhaps the significance of porosity severity to OA severity in the left sides of males can be explained by biomechanics.

In addition, as previously mentioned, the significance of porosity severity to OA does not appear to favor one particular method. In European American males all three methods demonstrated a significant relationship to porosity severity for the left perifoveal region and left femoral head; only methods I and III exhibited significant relationships for the left lunate acetabular surface. In African American males methods I and III were

significant for the left perfoveal region but only method II was significant for the left femoral head. Thus, porosity appears to be related to OA regardless of the method used to identify OA at least in among the left hip joints of African and European males.

Furthermore, in the knee, porosity's significance to OA, using any of the three methods, tended to be positively correlated with sample size. The greatest number of significant relationships between porosity severity and OA severity were found in European American males, then European American females, and lastly African American males. Simply put, as sample size increased, the chance of porosity's significance to OA increased for all three methods. This is completely different from the apparent male sex and side bias of porosity in the hip. The hip and knee are different types of weight-bearing joints (the knee is a hinge joint and the hip is a ball and socket joint) and the forces acting on each is different. In this research, the type of joint examined appears to play a role in the results one obtains regarding porosity's significance to OA. It should be stressed, however, that these results are on severity data and not absence-presence data. It is likely that one might obtain different results using absence-presence data or other scoring techniques.

The results of this research also corroborate Bridges (1993) assessment that the methods one uses will undoubtedly affect the outcome of their results. In light of this study there was generally no change in the relationship of porosity severity to OA severity as defined by methods I and III in both the hip and knee. Method II, on the other hand, differed most often from either of the other methods in its relationship to age and porosity severity. Then again, the occurrence of eburnation, (which is representative of

method II), is so rare that for several joint surfaces it is invariant, which in this research signified that it did not occur at all on a particular joint surface, consequently the results of the Fisher's Exact test for method I and method III would be exactly the same (Tables 3-24). This was expected seeing that eburnation rarely occurred during the course of this research.

The general lack of change in the outcome of results when using methods I and III could also be attributed to the fact that eburnation never occurred without osteophytes (Tables 25-45; Appendix C) and in most cases was less severe than osteophytic development, as evidenced by the cross tabulations in Tables 31-45 (Appendix C). Consequently, the severity score for osteophytic development, as explained in the methods chapter, would have taken precedent over the severity score for eburnation in most instances. Thus in many cases the severity scores for methods I and III are almost identical. This in itself is a methodological issue. If one were to calculate severity differently when analyzing osteophytes and eburnation they might obtain different results than those presented here. Nonetheless, method II almost always exhibited a different relationship than that of methods I and III, suggesting that methods do affect results and perhaps measures should be taken to make studies using different methods more comparable.

Changes in the relationship of porosity to OA depend on the methods used to identify OA, joint type, and sample size. Porosity severity appears to have a significant relationship with OA severity in the left hip of African American males and European American males. Significance appears to be dependent on sex and side in the hip and

sample size in the knee. This difference in the relationship of porosity to OA in the hip and knee suggests that the prevalence of porosity depends of the type of joint as well as the forces acting on the joint. Also porosity significance does not appear to favor one particular method over another in the hip or knee. This evidence refutes Rothschild's (1997) suggestion that porosity should be eliminated as a criterion of OA identification. If anything, porosity needs to be investigated further amongst other populations and different joints with a greater sample size.

Chapter VII

Conclusion

The William M. Bass Donated Skeletal Collection represents a sample of contemporary African, European and Latino American males and females. The current research only examined 231 European American females and African and European American males ages 35 to 101. Though this sample is small and lacking in diversity the results it presents in regards to porosity's significance to OA should not be overlooked.

First, porosity does appear to be significantly related to hip OA, particularly the left hip, in African and European American males. This suggests that porosity should be investigated further in regards to specific joint types in order to see if porosity is more prone to one joint type as opposed to another. If this is the case, questions concerning whether or not porosity's significance is related to OA or biomechanical factors should be raised and addressed.

Secondly, porosity appeared to be significantly related to knee OA in European American males. However, porosity's apparent lack of significance to OA in the knees of European American females and African American males does not prove noteworthy, seeing that as sample size increased (from African American males to European American females and then to European American males) the occurrence of significant relationships increased. Future research should include larger and more evenly distributed sample sizes to determine if porosity appears significant across distinct populations.

In addition, porosity's significance to OA in both the hip and knee did not favor any particular identifying method of OA. Thus, porosity appears to occur whether or not osteophytes or eburnation occur. This suggests that the process that causes porosity is distinct from the processes that cause eburnation and osteophytes. More research is needed to further explore this presumption.

In summation, this research does not support Rothschild's (1997) assertion that porosity is not related to OA and should thus be excluded as an identifying characteristic of OA. The current research suggests that porosity is related to hip OA in African and European American males and knee OA in European American males. More research must be undertaken with larger and more diverse samples before any concrete conclusions can be made in regards to porosity's significance to OA.

BIBLIOGRAPHY

- Adams ID. 1976. Osteoarthrosis and Sport. *Clinics in Rheumatic Diseases* 2(3):523-541.
- Agha RA, Webb, B. 2006. A cadaveric investigation into the links between macroscopic and microscopic osteoarthritic changes at the hip. *Clinical Anatomy* 19:115-124.
- Altman R, et al.,. 1986. Development of criteria for the classification and reporting of osteoarthritis: Classification of osteoarthritis of the knee. *Arthritis Rheum* 29(8):1039-1049.
- Altman R, et al.,. 1991. The American college of rheumatology criteria for classification and reporting of osteoarthritis of the hip. *Arthritis Rheum* 34(5):505-514.
- Amstutz HC, Kim, W.C. 1984. Osteoarthritis of the Hip. In: Moskowitz RW, Howell, D.S., Goldberg, V.M., Mankin, H.J., editor. *Osteoarthritis: Diagnosis and management*. Philadelphia: W.B. Saunders Company. p 423-442.
- Angel JL. 1981. History and development of paleopathology. *Am J Phys Anthropol* 56:509-515.
- Akesson K. 1999. Osteoarthritis and degenerative spine pathologies. In: Seibel MJ, Robins SP, Bilezikian JP., editor. *Dynamics of bone and cartilage metabolism*. New York: Academic Press. p. 637-648.
- Bass WM. 1995. *Human osteology: A laboratory and field manual*, 4th edition. Columbia, MO: Missouri Archaeological Society.
- Bergmann G, et al., . 2001. Hip contact forces and gait patterns from routine activities. *J Biomechanics* 34:859-871.
- Bluestone R. 1984. General aspects of differential diagnosis. In: Moskowitz RW, Howell, D.S., Goldberg, V.M., Mankin, H.J., editor. *Osteoarthritis: Diagnosis and management*. Philadelphia: W.B. Saunders Company. p 275-284.
- Boskey AL. 1999. Mineralization, structure, and function of bone. In: Seibel MJ, Robins SP, Bilezikian JP., editor. *Dynamics of bone and cartilage metabolism*. New York: Academic Press. p. 153-164.
- Bridges PS. 1992. Prehistoric arthritis in the Americas. *Ann Rev Anthropol* 21:67-91.
- Bridges PS. 1993. The effect of variation in methodology on the outcome of osteoarthritic studies. *Int J Osteoarchaeol* 3:289-295.
- Buikstra JE, Ubelaker, D.H., eds. 1994. *Standards for data collection for human skeletal remains: Proceedings of a seminar at the Field Museum of Natural History*,

Arkansas Archeological Survey Research Series No. 44. Fayetteville: Arkansas Archeological Survey.

Carter MA. 2003. Anatomy and physiology of bones and joints. In: Price SA, Wilson LM, editor. Pathophysiology: Clinical concepts of disease processes. St. Louis, Missouri: Mosby, Inc. p. 1019-1024.

Carter MA. 2003b. Osteoarthritis. In: Price SA, Wilson LM, editor. Pathophysiology: Clinical concepts of disease processes. St. Louis, Missouri: Mosby, Inc. p. 1037-1040.

Chew FS. 1989. Skeletal radiology: The bare bones. Rockville, Maryland: Aspen Publishers, Inc.

Cooper C, et al.,. 1991. Osteoarthritis of the hip and osteoporosis of the proximal femur. *Annals of the Rheumatic Diseases* 50:540-542.

Croft P. 2005. An introduction to the atlas of standard radiographs of arthritis. *Rheumatology* 44(Suppl.4):iv42.

Danielsson L. 1966. Incidence of osteoarthritis of the hip (coxarthrosis)*. *Clin Orthop and Related Research* 45:67-72.

Danielsson L, Hernborg, J. 1970. Morbidity and mortality of osteoarthritis of the knee (gonarthrosis) in Malmo, Sweden. *Clin Orthop and Related Research* 69:224-226.

Davis MA, et al.,. 1991. Knee osteoarthritis and physical functioning: Evidence from the NHANES I epidemiologic followup study. *Journal of Rheumatology* 18:591-598.

Dempster DW. 1999. New concepts in bone remodeling. In: Seibel MJ, Robins SP, Bilezikian JP., editor. Dynamics of bone and cartilage metabolism. New York: Academic Press. p. 261-273.

Engin AE, Milind, S.K. 1974. Biomechanics of normal and abnormal knee joint. *J Biomechanics* 7:325-334.

Felson DT, et al.,. 1995. The incidence and natural history of knee osteoarthritis in the elderly: The Framingham osteoarthritis study. *Arthritis Rheum* 38(10):1500-1505.

Freeman MAR, Pinskerova, V. 2005. The movement of the normal tibio-femoral joint. *J Biomechanics* 38:197-208.

Hall SJ. 1995. Basic Biomechanics. St. Louis: Mosby-Year Book, Inc.

- Hart DJ, et al., 1994. The relationship between osteoarthritis and osteoporosis in the general population: the Chingford Study. *Annals of the Rheumatic Diseases* 53:158-162.
- Hernborg JS, Nilsson, B.E. 1977. The natural course of untreated osteoarthritis of the knee. *Clin Orthop and Related Research* 123:130-137.
- Hoaglund FT. 1976. Clinical manifestations of osteoarthrosis. *Clinics in Rheumatic Diseases* 2(3):543-556.
- Holderbaum D, Haqqi, T.M., Moskowitz, R.W. 1999. Genetics and osteoarthritis: Exposing the iceberg. *Arthritis Rheum* 42(3):397-405.
- Howell DS. 1984. Etiopathogenesis of osteoarthritis. In: Moskowitz RW, Howell, D.S., Goldberg, V.M., Mankin, H.J., editor. *Osteoarthritis: Diagnosis and management*. Philadelphia: W.B. Saunders Company. p 129-146.
- Hughes SL, Dunlop, D. 1995. The prevalence and impact of arthritis in older persons. *Arthritis Care and Research* 8(4):257-264.
- Jaffe HL. 1972. *Metabolic, degenerative, and inflammatory diseases of bones and joints*. Philadelphia, Lea & Febiger.
- Janssens PA. 1970. *Palaeopathology: Diseases and injuries of prehistoric man*. London: John Baker LTD.
- Jarcho S. 1965. The development and present condition of human palaeopathology in the United States. In: Jarcho S, editor. *Human palaeopathology*. New Haven and London: Yale University Press. p 3-30.
- Jeffrey AK. 1975. Osteophytes and the osteoarthritic femoral head. *Journal of Joint and Bone Surgery* 57B:314-324.
- Johnson LC. 1965. Discussion. In: Jarcho S, editor. *Human Palaeopathology*. New Haven and London: Yale University Press. p 68-81.
- Jurmain RD. 1977. Stress and the etiology of osteoarthritis. *Am J Phys Anthropol* 46:353-366.
- Jurmain RD. 1980. The pattern of involvement of appendicular degenerative joint disease. *Am J Phys Anthropol* 53:143-150.
- Jurmain RD. 1990. Paleoepidemiology of a Central California prehistoric population from CA-ALA-329. II. Degenerative disease. *Am J Phys Anthropol* 83:83-94.

- Kaufman KR, et al., . 2001. Gait characteristics of patients with knee osteoarthritis. *J Biomechanics* 34:907-915.
- Kettelkamp DB, Colyer, R.A. 1984. Osteoarthritis of the knee. In: Moskowitz RW, Howell, D.S., Goldberg, V.M., Mankin, H.J. , editor. *Osteoarthritis: Diagnosis and management*. Philadelphia: W.B. Saunders Company. p 403-421.
- Kettelkamp DB, Jacobs, A.W. 1972. Tibiofemoral contact area-determination and implications. *J Bone Joint Surg* 54A:349-356.
- Kushner I, Forer, A, McGuire, A. 1984. *Understanding Arthritis: Arthritis foundation*. New York, Charles Scribner's Sons.
- Lawrence JS, Sebo, M. 1981. The geography of osteoarthritis. In: Nuki G., editor. *The Aetiopathogenesis of osteoarthrosis*. Tunbridge Wells, Pitman Medical Publishing Company. p 155-183.
- Leisen JCC, Duncan, H., Riddle, J.M. 1991. Rheumatoid erosive arthropathy as seen in macerated (dry) bone specimens. In: Ortner DJ, Aufderheide, A.C., editor. *Human paleopathology: Current syntheses and future options*. Washington: Smithsonian Institution Press. p 211-215.
- Lieverse AR, et al., . 2007. Osteoarthritis in Siberia's Cis-Baikal: Skeletal Indicators of Hunter-Gatherer Adaptation and Cultural Change. *Am J Phys Anthropol* 132:1-16.
- Lian JB, Stein, GS. 1999. The cells of bone. In: Seibel MJ, Robins SP, Bilezikian JP., editor. *Dynamics of bone and cartilage metabolism*. New York: Academic Press. p. 165-185.
- Macys JR, et al., . 1980. Coxarthrosis: A study of the natural history based on a correlation of clinical, radiographic, and pathologic findings. *Sem Arth Rheum* 10(1):66-80.
- Mann RW, Murphy, S.P. 1990. *Regional atlas of bone disease: A guide to pathologic and normal variation in the human skeleton*. Springfield: Charles C Thomas Publisher.
- Meachim G, Brooke, G. . 1984. The pathology of osteoarthritis. In: Moskowitz RW, Howell, D.S., Goldberg, V.M., Mankin, H.J., editor. *Osteoarthritis: Diagnosis and management*. Philadelphia: W.B. Saunders Company. p 29-42.
- Merbs CF. 1983. Patterns of activity induced pathology in Canadian population. *Arch Serv, Canada Paper* 119. Hull, Canada: Museum of Civilization.

- Miles JS. 1965. Diseases encountered in Mesa Verde, Colorado. II. Evidences of disease. In: Jarcho S, editor. Human Palaeopathology. New Haven and London: Yale University Press. p 91-97.
- Moll JMH. 1976. Investigation of osteoarthrosis. Clinics in Rheumatic Diseases 2(3):587-613.
- Moodie RL. 1923. Paleopathology: An introduction to the study of ancient evidences of disease. Urbana, Illinois: University of Illinois Press.
- Morris on JB. 1970. The mechanics of the knee joint in relation to normal walking. J of Biomech 3:51-.
- Moskowitz RW. 1984a. Introduction. In: Moskowitz RW, Howell, D.S., Goldberg, V.M., Mankin, H.J., editor. Osteoarthritis: Diagnosis and management. Philadelphia: W.B. Saunders Company. p 1-6.
- Moskowitz RW. 1984b. Experimental models in osteoarthritis. In: Moskowitz RW, Howell, D.S., Goldberg, V.M., Mankin, H.J., editor. Osteoarthritis: Diagnosis and management. Philadelphia: W.B. Saunders Company. p 109-128.
- Moskowitz RW. 1984c. Osteoarthritis-symptoms and signs. In: Moskowitz RW, Howell, D.S., Goldberg, V.M., Mankin, H.J., editor. Osteoarthritis: Diagnosis and management. Philadelphia: W.B. Saunders Company. p 149-154.
- Murray RO. 1965. The aetiology of primary osteoarthritis of the hip. Br J Radiol 38:810-824.
- Naish JH, et al., . 2006. MR measurement of articular cartilage thickness distribution in the hip. OsteoArthritis and Cartilage 14:967-973.
- Neilson M, et al., . 2004. Changes in bone architecture in the femoral head and neck in osteoarthritis. Clinical Anatomy 17:378-391.
- Nordin M, Frankel, V.H. 1980a. Biomechanics of the hip. In: Frankel VH, Nordin, M., editor. Basic biomechanics of the skeletal system. Philidelphia: Lea and Febiger. p 149-177.
- Nordin M, Frankel, V.H. 1980b. Biomechanics of the knee. In: Frankel VH, Nordin, M., editor. Basic biomechanics of the skeletal system. Philidelphia: Lea and Febiger. p 113-148.

- Nordin M, Frankel, V.H. 1989. Basic biomechanics of the skeletal system, 2nd ed. Philadelphia: Lea and Febiger.
- Norman A. 1984. Roentgenologic diagnosis. In: Moskowitz RW, Howell, D.S., Goldberg, V.M., Mankin, H.J., editor. Osteoarthritis: Diagnosis and management. Philadelphia: W.B. Saunders Company. p 155-184.
- Ortner DJ. 1968. Description and classification of degenerative bone changes in the distal joint surfaces of the humerus. *Am J Phys Anthropol* 28:139-156.
- Ortner DJ, Utermohle, C.J. 1981. Polyarticular Inflammatory Arthritis in a Pre-Columbian skeleton from Kodiak Island, Alaska, U.S.A. *Am J Phys Anthropol* 56:23-31.
- Ortner DJ. 1991. Theoretical and methodological issues in paleopathology. In: Ortner DJ, Aufderheide, A.C., editor. *Human paleopathology: Current syntheses and future options*. Washington: Smithsonian Institution Press. p 5-11.
- Ortner DJ. 2003a. Osteoarthritis and diffuse idiopathic skeletal hyperostosis. In: Ortner DJ, editor. *Identification of pathological conditions in human skeletal remains*. 2nd ed. New York: Academic Press. p 545-560.
- Ortner DJ. 2003b. Background data in paleopathology. In: Ortner DJ, editor. *Identification of pathological conditions in human skeletal remains*. 2nd ed. New York: Academic Press. p 37-44.
- Ortner DJ. 2003c. Theoretical issues in paleopathology. In: Ortner DJ, editor. *Identification of pathological conditions in human skeletal remains*. 2nd ed. New York: Academic Press. p 109-118.
- Ortner DJ. 2003d. Introduction. In: Ortner DJ, editor. *Identification of pathological conditions in human skeletal remains*. 2nd ed. New York: Academic Press. p 1-10.
- Ortner DJ. 2003e. Methods used in the analysis of skeletal lesions. In: Ortner DJ, editor. *Identification of pathological conditions in human skeletal remains*. 2nd ed. New York: Academic Press. p 45-64.
- Ortner DJ, Aufderheide, A.C. 1991. Introduction. In: Ortner DJ, Aufderheide, A.C., editor. *Human paleopathology: Current syntheses and future options*. Washington: Smithsonian Institution Press. p 1-4.

- Ortner DJ, Turner-Walker, G. 2003. The biology of skeletal tissues. In: Ortner DJ, editor. Identification of pathological conditions in human skeletal remains. 2nd ed. New York: Academic Press. p 11-35.
- Peyron JG. 1984. The epidemiology of osteoarthritis. In: Moskowitz RW, Howell, D.S., Goldberg, V.M., Mankin, H.J., editor. Osteoarthritis: Diagnosis and management. Philadelphia: W.B. Saunders Company. p 9-27.
- Putschar WGJ. 1965. Problems in pathology and palaeopathology of bone. In: Jarcho S, editor. Human Palaeopathology. New Haven and London: Yale University Press. p 57-65.
- Radin EL. 1976. Aetiology of osteoarthrosis. Clinics in Rheumatic Diseases 2(3):509-522.
- Radin EL. 1984. Biomechanical considerations. In: Moskowitz RW, Howell, D.S., Goldberg, V.M., Mankin, H.J., editor. Osteoarthritis: Diagnosis and management. Philadelphia: W.B. Saunders Company. p 93-107.
- Reilly DT, Martens, M. 1972. Experimental analysis of the quadriceps muscle force and patello-femoral joint reaction force for various activities. Acta Orthop Scand 43:126-.
- Rogers J, Shepstone, L., Dieppe, P. 2004. Is osteoarthritis a systemic disorder of bone? Arth & Rheum 50(2):452-457.
- Roney Jr. JG. 1965. Palaeoepidemiology: An example from California. In: Jarcho S, editor. Human palaeopathology. New Haven and London: Yale University Press. p 99-107.
- Ross MH, Reith, EJ. 1985. Histology: A text and atlas. New York: Harper & Row, Publishers, J.B. Lippincott Company.
- Rothschild BM. 1997. Porosity: A curiosity without diagnostic significance. Am J Phys Anthropol 104:529-533.
- SAS Institute Inc., SAS 9.1.3 Help and Documentation, Cary, NC: SAS Institute Inc., 2000-2004.
- Schumacher HR. 1984. Secondary osteoarthritis. In: Moskowitz RW, Howell, D.S., Goldberg, V.M., Mankin, H.J., editor. Osteoarthritis: Diagnosis and management. Philadelphia: W.B. Saunders Company. p 235-264.
- Sokoloff L. 1969. The biology of degenerative joint disease. Chicago: The University of Chicago Press.

- Sokoloff L, Hough, A. 1985. Pathology of osteoarthritis. In: McCarthy DJ, editor. Arthritis and allied conditions: A Textbook of Rheumatology, 10th edition. Lea & Febiger. p 1377-1399.
- Solomon L, Beighton, P., Lawrence, J.S. 1975. Rheumatic disorders in the South African Negro: II Osteoarthrosis. S Afr Med 49:1737-1740.
- Steinbock TR. 1976. Paleopathological diagnosis and interpretation: Bone disease in ancient human populations. Springfield: Charles C. Thomas Publisher.
- Stulberg SD. 1984. The etiology and natural course of osteoarthritis of the hip (coxarthrosis). In: Moskowitz RW, Howell, D.S., Goldberg, V.M., Mankin, H.J., editor. Osteoarthritis: Diagnosis and management. Philadelphia: W.B. Saunders Company. p 265-273.
- Verbrugge LM. 1995. Women, men, and osteoarthritis. Arthritis Care and Research 8(4):212-220.
- Waldron T. 1992. Osteoarthritis in a black death cemetery in London. Int J Osteoarchaeol 2: 235-240.
- Waldron T. 1995. Changes in the distribution of osteoarthritis over historical time. Int J Osteoarchaeol 5:385-389.
- Wells C. 1964. Bones, bodies, and disease: Evidence of disease and abnormality in early man. New York: Praeger.
- Wood PHN. 1976. Osteoarthrosis in the community. Clinics in Rheumatic Diseases 2(3):495-507.
- Woods RJ. 1986. Biomechanics and degenerative joint disease in humans, gorillas, and chimpanzees. Kent: Kent State University.
- Woods RJ. 1995. Biomechanics and osteoarthritis of the knee. Columbus: Ohio State University.
- Yoshida H, et al.,. 2006. Three-dimensional dynamic hip contact area and pressure distribution during activities of daily living. J Biomechanics 39(11):1996-2004.
- *2002. Letter to the editor. ISB recommendation on definitions of joint coordinate system of various joints for the reporting of human joint motion-part I: ankle, hip, and spine. J Biomechanics 35: 543-548.
- *2005. Atlas of standard radiographs of arthritis. Rheumatology 44:iv43-iv72.

APPENDICES

Appendix A
Photographic Examples of Osteoarthritic Severity

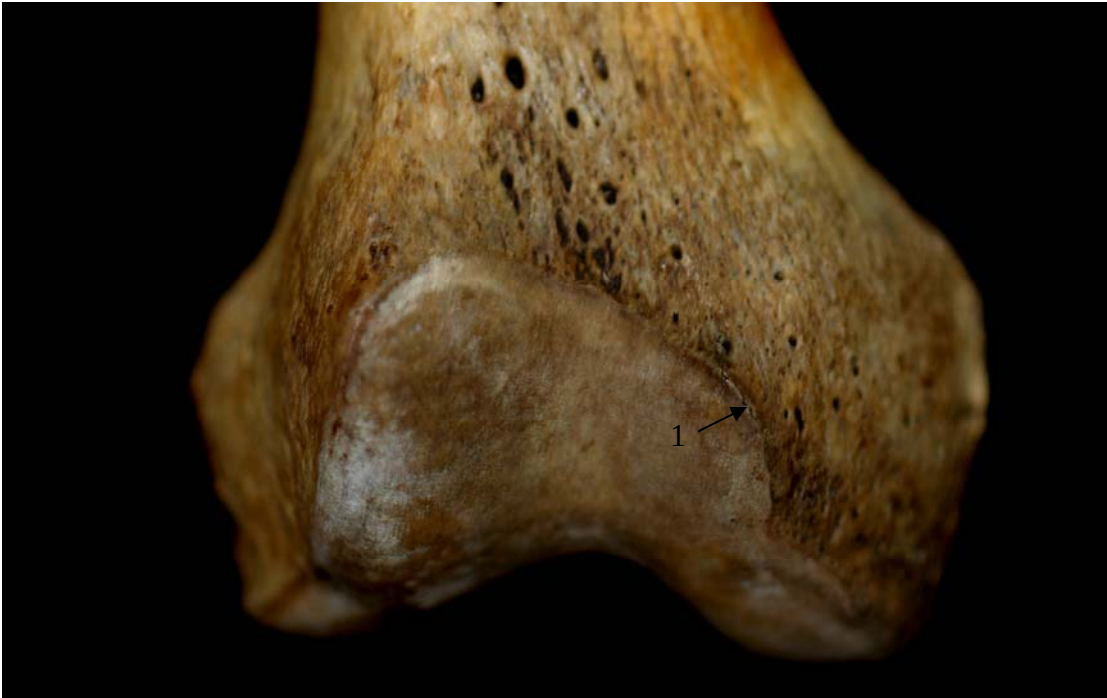


Figure 4: Example of Osteoarthritis Scored as Absent (0) on the Lateral Patellofemoral Surface and Trace (1) Marginal Osteophytic development on the Medial Patellofemoral Surface.



Figure 5: Example of Trace (1) Osteophytic Development on the Lateral Condyle and Osteoarthritis Scored as Absent (0) of on the Medial Condyle.



Figure 6: Example of Trace (1) Eburnation on the Lateral Intercondylar Tubercle and Severe (4) Eburnation on the Medial Tibial Plateau.



Figure 7: Example of Severe (4) Eburnation and Trace (1) Porosity on the Lateral Patellofemoral Surface.



Figure 8: Example of Severe (4) Eburnation, Trace (1) Porosity, and Moderate (3) Osteophytic Development on the Lateral Tibial Plateau.



Figure 9: Example of Slight (2) Osteophytic Development on the Perifoveal Region.



Figure 10: Example of Slight (2) Marginal Osteophytic Development on the Patellofemoral Surfaces.

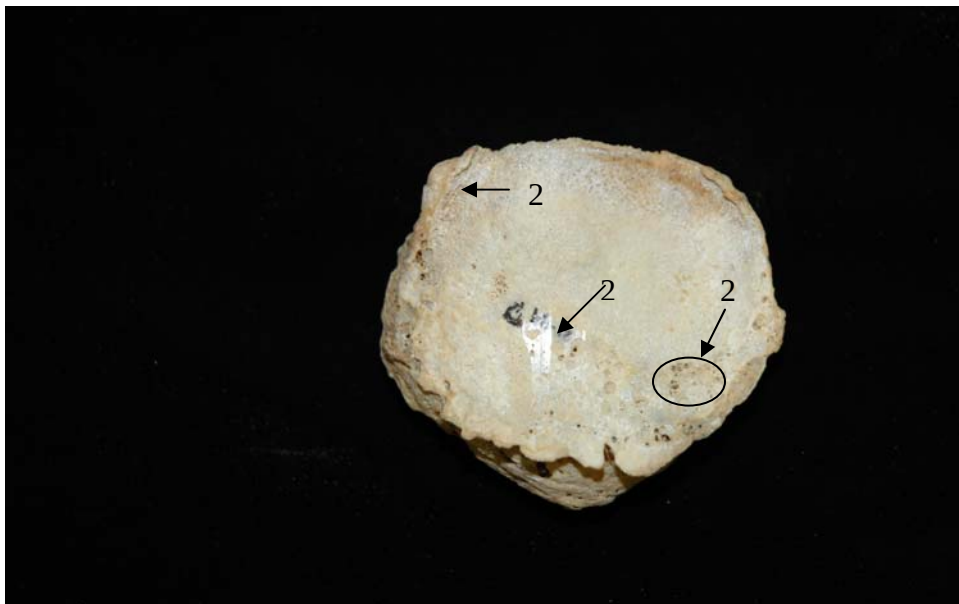


Figure 11: Example of Slight (2) Eburnation, Slight (2) Osteophytic Development and Slight (2) Porosity on Both Articular Surfaces of the Right Patella.



Figure 12: Example of Slight (2) Eburnation, Slight (2) Porosity, and Moderate (3) Osteophytic Development on the Lateral Tibial Plateau.

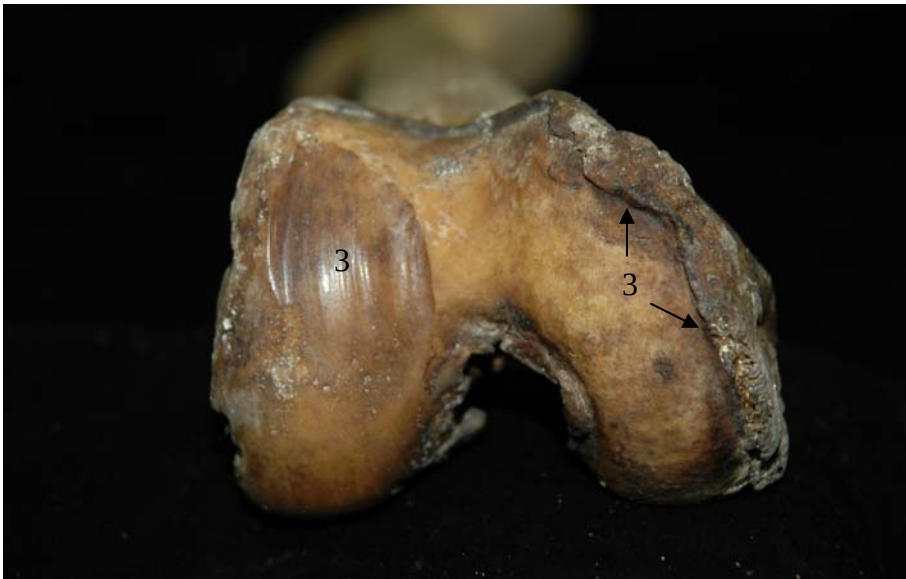


Figure 13: Example of Moderate (3) Eburnation on the Lateral Femoral Condyle and Moderate (3) Osteophytic Development on the Medial Femoral Condyle.

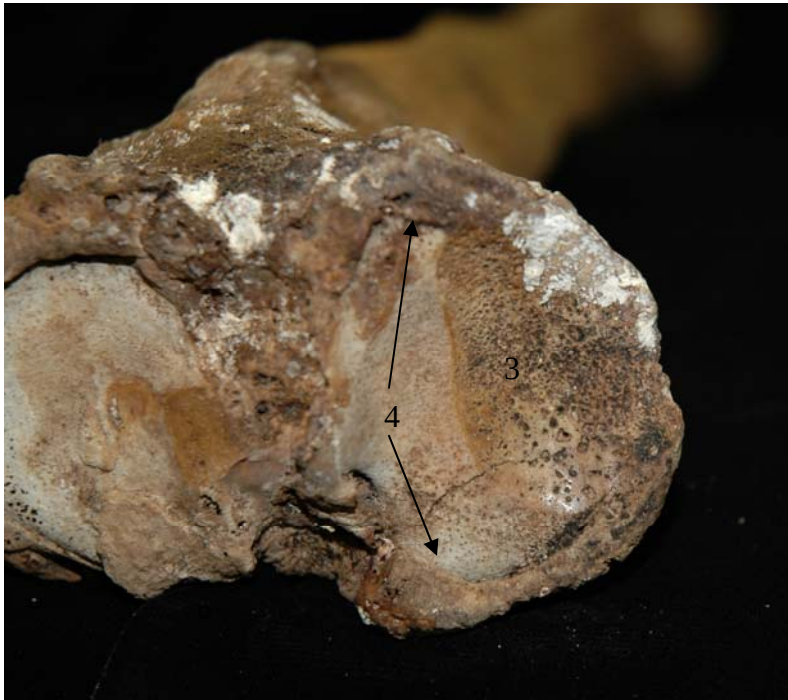


Figure 14: Example of Severe (4) Osteophytic Development, Moderate (3) Eburnation, and Moderate (3) Porosity on the Medial Tibial Plateau. Eburnation and Porosity are Enclosed Together.



Figure 15: Severe (4) Porosity on the Lunate Surface.

Appendix B

Fisher's Exact Test Tables

Table 4: Fisher's Exact Test for the Right Perifoveal Region Showing the Relationship Between Porosity Severity Scores and each Method

Group	Method	Table Probability	P-Value	n=
*EA Females:	Osteophytes	3.25E-04	0.3328	78
	Eburnation	0.8101	1	79
	Osteophytes and Eburnation	3.25E-04	0.3328	78
*AA Males:	Osteophytes	0.0014	0.1434	24
	Eburnation	0.75	1	24
	Osteophytes and Eburnation	0.0014	0.1434	24
*EA Males:	Osteophytes	6.64E-06	0.6187	113

*Note: AA denotes African American.

*Note: EA denotes European American.

Table 5: Fisher's Exact Test for the Left Femoral Head Showing the Relationship Between Porosity Severity Scores and each Method

Group	Method	Table Probability	P-Value	n=
*EA Females:	Osteophytes	6.85E-05	0.437	79
	Eburnation	0.4557	1	79
	Osteophytes and Eburnation	6.85E-05	0.437	79
*AA Males:	Osteophytes	0.0028	0.9015	25
	Eburnation	0.04	0.04	25
	Osteophytes and Eburnation	0.0028	0.9015	25
*EA Males:	Osteophytes	9.51E-09	0.003	111
	Eburnation	0.027	0.036	111
	Osteophytes and Eburnation	9.51E-09	0.003	111

*Note: AA denotes African American.

*Note: EA denotes European American.

Table 6: Fisher's Exact Test for the Right Femoral Head Showing the Relationship Between Porosity Severity Scores and each Method

Group	Method	Table Probability	P-Value	n=
*EA Females:	Osteophytes	3.65E-04	0.8768	79
	Eburnation	0.2532	1	79
	Osteophytes and Eburnation	3.65E-04	0.8768	79
*AA Males:	Osteophytes	0.0148	1	25
*EA Males:	Osteophytes	5.78E-07	0.1751	113

*Note: AA denotes African American.

*Note: EA denotes European American.

Table 7: Fisher's Exact Test for the Left Lunate Acetabular Surface Showing the Relationship Between Porosity Severity Scores and each Method

Group	Method	Table Probability	P-Value	n=
*EA Females:	Osteophytes	5.24E-04	0.6909	84
	Eburnation	0.7024	1	84
	Osteophytes and Eburnation	5.24E-04	0.6909	84
*AA Males:	Osteophytes	0.0108	0.4553	25
*EA Males:	Osteophytes	5.92E-09	0.018	117

*Note: AA denotes African American.

*Note: EA denotes European American.

Table 8: Fisher's Exact Test for the Right Lunate Acetabular Surface Showing the Relationship Between Porosity Severity Scores and each Method

Group	Method	Table Probability	P-Value	n=
*EA Females:	Osteophytes	6.81E-04	0.957	85
	Eburnation	0.1256	0.5622	85
	Osteophytes and Eburnation	6.81E-04	0.957	85
*AA Males:	Osteophytes	0.0333	0.07	25
	Eburnation	0.92	1	25
	Osteophytes and Eburnation	0.0333	0.07	25
*EA Males:	Osteophytes	6.66E-06	0.1646	116

*Note: AA denotes African American.

*Note: EA denotes European American.

Table 10: Fisher's Exact Test for the Left Lateral Patellar Articular Facet Showing the Relationship Between Porosity Severity Scores and each Method

Group	Method	Table Probability	P-Value	n=
*EA Females:	Osteophytes	2.56E-06	0.0505	72
	Eburnation	0.1667	0.1806	72
	Osteophytes and Eburnation	2.56E-06	0.0505	72
*AA Males:	Osteophytes	1.06E-04	0.3647	24
	Eburnation	0.25	0.5833	24
	Osteophytes and Eburnation	1.06E-04	0.3647	24
*EA Males:	Osteophytes	2.22E-08	0.0628	98
	Eburnation	2.25E-04	0.0226	98
	Osteophytes and Eburnation	5.56E-09	0.0418	98

*Note: AA denotes African American.

*Note: EA denotes European American.

Table 11: Fisher's Exact Test for the Right Medial Patellar Articular Facet Showing the Relationship Between Porosity Severity Scores and each Method

Group	Method	Table Probability	P-Value	n=
*EA Females:	Osteophytes	1.27E-06	0.1578	75
	Eburnation	0.12	0.76	75
	Osteophytes and Eburnation	1.27E-06	0.1578	75
*AA Males:	Osteophytes	0.003	0.9397	22
	Eburnation	0.2727	1	22
	Osteophytes and Eburnation	0.003	0.9397	22
*EA Males:	Osteophytes	1.55E-10	3.96E-04	98
	Eburnation	0.0227	0.024	98
	Osteophytes and Eburnation	1.55E-10	3.96E-04	98

*Note: AA denotes African American.

*Note: EA denotes European American.

Table 12: Fisher's Exact Test for the Right Lateral Patellar Articular Facet Showing the Relationship Between Porosity Severity Scores and each Method

Group	Method	Table Probability	P-Value	n=
*EA Females:	Osteophytes	1.04E-05	0.1148	76
	Eburnation	0.1974	0.1974	76
	Osteophytes and Eburnation	1.04E-05	0.1148	76
*AA Males:	Osteophytes	0.0021	0.5909	22
*EA Males:	Osteophytes	6.87E-08	0.0126	96
	Eburnation	0.0058	0.044	97
	Osteophytes and Eburnation	6.87E-08	0.0126	96

*Note: AA denotes African American.

*Note: EA denotes European American.

Table 13: Fisher's Exact Test for the Left Medial Patellofemoral Surface Showing the Relationship Between Porosity Severity Scores and each Method

Group	Method	Table Probability	P-Value	n=
*EA Females:	Osteophytes	4.75E-07	0.0197	78
	Eburnation	0.2179	0.4231	78
	Osteophytes and Eburnation	4.75E-07	0.0197	78
*AA Males:	Osteophytes	1.61E-04	0.5401	23
*EA Males:	Osteophytes	2.43E-10	0.0032	105
	Eburnation	0.0108	0.0595	106
	Osteophytes and Eburnation	2.43E-10	0.0032	105

*Note: AA denotes African American.

*Note: EA denotes European American.

Table 14: Fisher's Exact Test for the Left Lateral Patellofemoral Surface Showing the Relationship Between Porosity Severity Scores and each Method

Group	Method	Table Probability	P-Value	n=
*EA Females:	Osteophytes	1.88E-08	0.0031	79
	Eburnation	0.1772	0.1899	79
	Osteophytes and Eburnation	1.88E-08	0.0031	79
*AA Males:	Osteophytes	7.09E-06	0.0122	22
	Eburnation	0.4091	1	22
	Osteophytes and Eburnation	7.09E-06	0.0122	22
*EA Males:	Osteophytes	2.08E-08	0.0637	107
	Eburnation	0.0088	0.8226	107
	Osteophytes and Eburnation	1.04E-08	0.0661	107

*Note: AA denotes African American.

*Note: EA denotes European American.

Table 15: Fisher's Exact Test for the Right Medial Patellofemoral Surface Showing the Relationship Between Porosity Severity Scores and each Method

Group	Method	Table Probability	P-Value	n=
*EA Females:	Osteophytes	4.09E-07	0.0109	82
	Eburnation	0.0861	0.2936	82
	Osteophytes and Eburnation	4.09E-07	0.0109	82
*AA Males:	Osteophytes	7.06E-05	0.3195	24
*EA Males:	Osteophytes	2.15E-10	0.008	107
	Eburnation	0.0875	0.3536	107
	Osteophytes and Eburnation	2.15E-10	0.008	107

*Note: AA denotes African American.

*Note: EA denotes European American.

Table 16: Fisher's Exact Test for the Right Lateral Patellofemoral Surface Showing the Relationship Between Porosity Severity Scores and each Method

Group	Method	Table Probability	P-Value	n=
*EA Females:	Osteophytes	5.15E-10	2.71E-05	84
	Eburnation	0.0292	0.2792	84
	Osteophytes and Eburnation	6.86E-10	4.17E-05	84
*AA Males:	Osteophytes	1.80E-04	0.0786	23
*EA Males:	Osteophytes	5.26E-11	0.0035	111
	Eburnation	0.0123	0.3575	111
	Osteophytes and Eburnation	4.34E-11	0.0037	111

*Note: AA denotes African American.

*Note: EA denotes European American.

Table 17: Fisher's Exact Test for the Left Medial Patellofemoral Surface Showing the Relationship Between Porosity Severity Scores and each Method

Group	Method	Table Probability	P-Value	n=
*EA Females:	Osteophytes	1.70E-05	0.8425	78
	Eburnation	0.0313	0.6679	78
	Osteophytes and Eburnation	1.70E-05	0.8425	78
*AA Males:	Osteophytes	0.0033	0.1613	22
	Eburnation	0.4091	1	22
	Osteophytes and Eburnation	0.0016	0.2676	22
*EA Males:	Osteophytes	3.78E-09	0.0027	104
	Eburnation	0.0053	0.2477	104
	Osteophytes and Eburnation	3.78E-09	0.0027	104

*Note: AA denotes African American.

*Note: EA denotes European American.

Table 18: Fisher's Exact Test for the Left Lateral Patellofemoral Surface Showing the Relationship Between Porosity Severity Scores and each Method

Group	Method	Table Probability	P-Value	n=
*EA Females:	Osteophytes	7.96E-08	0.1961	74
	Eburnation	4.71E-05	4.46E-04	74
	Osteophytes and Eburnation	1.99E-08	0.0896	74
*AA Males:	Osteophytes	9.48E-04	0.1581	22
*EA Males:	Osteophytes	2.12E-09	0.0086	107
	Eburnation	0.001	0.001	108
	Osteophytes and Eburnation	1.27E-09	0.0067	107

*Note: AA denotes African American.

*Note: EA denotes European American.

Table 19: Fisher's Exact Test for the Right Medial Patellofemoral Surface Showing the Relationship Between Porosity Severity Scores and each Method

Group	Method	Table Probability	P-Value	n=
*EA Females:	Osteophytes	2.82E-06	0.1314	82
	Eburnation	0.0177	0.5701	83
	Osteophytes and Eburnation	9.22E-07	0.1018	82
*AA Males:	Osteophytes	6.18E-05	0.2329	24
	Eburnation	0.0181	0.1377	24
	Osteophytes and Eburnation	1.54E-05	0.0989	24
*EA Males:	Osteophytes	2.66E-08	0.1228	107
	Eburnation	1.80E-04	0.0205	108
	Osteophytes and Eburnation	6.66E-09	0.0674	107

*Note: AA denotes African American.

*Note: EA denotes European American.

Table 20: Fisher's Exact Test for the Right Lateral Patellofemoral Surface Showing the Relationship Between Porosity Severity Scores and each Method

Group	Method	Table Probability	P-Value	n=
*EA Females:	Osteophytes	1.03E-05	0.5001	81
	Eburnation	0.112	0.5722	81
	Osteophytes and Eburnation	5.61E-06	0.4882	81
*AA Males:	Osteophytes	0.0013	0.5859	23
	Eburnation	0.0435	0.087	23
	Osteophytes and Eburnation	0.0013	0.5859	23
*EA Males:	Osteophytes	2.02E-08	0.1015	108
	Eburnation	6.07E-04	0.0031	108
	Osteophytes and Eburnation	8.06E-09	0.0561	108

*Note: AA denotes African American.

*Note: EA denotes European American.

Table 21: Fisher's Exact Test for the Left Medial Tibial Condyle Showing the Relationship Between Porosity Severity Scores and each Method

Group	Method	Table Probability	P-Value	n=
*EA Females:	Osteophytes	1.23E-05	0.1327	76
	Eburnation	0.1062	0.7326	77
	Osteophytes and Eburnation	1.23E-05	0.1327	76
*AA Males:	Osteophytes	3.69E-04	0.0415	22
	Eburnation	0.5	1	22
	Osteophytes and Eburnation	1.84E-04	0.0608	22
*EA Males:	Osteophytes	2.05E-09	0.0091	104
	Eburnation	0.0011	0.043	104
	Osteophytes and Eburnation	2.05E-09	0.0091	104

*Note: AA denotes African American.

*Note: EA denotes European American.

Table 22: Fisher's Exact Test for the Left Lateral Tibial Condyle Showing the Relationship Between Porosity Severity Scores and each Method

Group	Method	Table Probability	P-Value	n=
*EA Females:	Osteophytes	5.99E-06	0.0989	71
	Eburnation	0.0102	0.1174	72
	Osteophytes and Eburnation	2.40E-06	0.1273	71
*AA Males:	Osteophytes	6.50E-03	0.3778	23
*EA Males:	Osteophytes	9.51E-10	0.0011	103
	Eburnation	0.011	0.0718	105
	Osteophytes and Eburnation	8.81E-10	0.0014	103

*Note: AA denotes African American.

*Note: EA denotes European American.

Table 23: Fisher's Exact Test for the Right Medial Tibial Condyle Showing the Relationship Between Porosity Severity Scores and each Method

Group	Method	Table Probability	P-Value	n=
*EA Females:	Osteophytes	2.20E-05	0.2299	80
	Eburnation	0.0762	1	80
	Osteophytes and Eburnation	1.21E-05	0.1707	80
*AA Males:	Osteophytes	0.0057	0.7859	24
	Eburnation	0.0217	0.1667	24
	Osteophytes and Eburnation	4.35E-04	0.2661	24
*EA Males:	Osteophytes	7.93E-10	0.0045	101
	Eburnation	7.78E-06	2.42E-04	104
	Osteophytes and Eburnation	3.96E-10	0.0024	101

*Note: AA denotes African American.

*Note: EA denotes European American.

Table 24: Fisher's Exact Test for the Right Lateral Tibial Condyle Showing the Relationship Between Porosity Severity Scores and each Method

Group	Method	Table Probability	P-Value	n=
*EA Females:	Osteophytes	4.38E-06	0.1934	75
	Eburnation	0.0031	0.0255	75
	Osteophytes and Eburnation	2.36E-07	0.0659	75
*AA Males:	Osteophytes	8.04E-04	0.1949	22
*EA Males:	Osteophytes	2.31E-09	0.0062	100
	Eburnation	5.49E-05	0.001	101
	Osteophytes and Eburnation	6.93E-10	0.0025	100

*Note: AA denotes African American.

*Note: EA denotes European American.

Appendix C

Prevalence of OA Lesions and Cross Tabulations

Table 25: Prevalence of Osteoarthritic Lesions in European American Females by Joint Surface

Method	Absent	*OP	*EB	*OP & *EB
Left Medial Patellar Articular Facet	12	56	2	56
Left Lateral Patellar Articular Facet	14	48	1	48
Right Medial Patellar Articular Facet	5	66	2	66
Right Lateral Patellar Articular Facet	16	48	1	48
Left Medial Patellofemoral Surface	17	57	1	57
Left Lateral Patellofemoral Surface	27	40	1	40
Right Medial Patellofemoral Surface	16	62	2	62
Right Lateral Patellofemoral Surface	26	53	3	53
Left Medial Femoral Condyle	3	62	5	62
Left Lateral Femoral Condyle	8	44	4	44
Right Medial Femoral Condyle	7	69	5	69
Right Lateral Femoral Condyle	9	54	2	54
Left Medial Tibial Plateau	9	57	3	57
Left Lateral Tibial Plateau	12	51	4	51
Right Medial Tibial Plateau	6	67	4	67
Right Lateral Tibial Plateau	5	64	3	64
Left Perifoveal Region	0	76	0	76
Right Perifoveal Region	0	77	1	77
Left Femoral Head	6	58	1	58
Right Femoral Head	5	60	2	60
Left Lunate Acetabular Surface	1	79	1	79
Right Lunate Acetabular Surface	1	82	3	82

*Note: OP denotes osteoporphyses; EB denotes eburation.

Table 26: Prevalence of Osteoarthritic Lesions in African American Males by Joint Surface

Method	Absent	*OP	*EB	*OP & *EB
Left Medial Patellar Articular Facet	1	21	0	21
Left Lateral Patellar Articular Facet	4	17	1	17
Right Medial Patellar Articular Facet	0	22	1	22
Right Lateral Patellar Articular Facet	5	15	0	15
Left Medial Patellofemoral Surface	3	19	0	19
Left Lateral Patellofemoral Surface	5	16	1	16
Right Medial Patellofemoral Surface	4	18	0	18
Right Lateral Patellofemoral Surface	5	14	0	14
Left Medial Femoral Condyle	0	22	1	22
Left Lateral Femoral Condyle	3	17	0	17
Right Medial Femoral Condyle	1	21	2	21
Right Lateral Femoral Condyle	5	13	1	13
Left Medial Tibial Plateau	4	17	1	17
Left Lateral Tibial Plateau	2	20	0	20
Right Medial Tibial Plateau	2	21	2	21
Right Lateral Tibial Plateau	2	18	0	18
Left Perifoveal Region	1	23	0	23
Right Perifoveal Region	1	23	1	23
Left Femoral Head	1	20	1	20
Right Femoral Head	0	21	0	21
Left Lunate Acetabular Surface	0	25	0	25
Right Lunate Acetabular Surface	0	25	1	25

*Note: OP denotes osteopyhtes; EB denotes eburnation.

Table 27: Prevalence of Osteoarthritic Lesions in European American Males by Joint Surface

Method	Absent	*OP	*EB	*OP & *EB
Left Medial Patellar Articular Facet	8	83	2	83
Left Lateral Patellar Articular Facet	11	71	5	71
Right Medial Patellar Articular Facet	12	78	2	78
Right Lateral Patellar Articular Facet	15	67	2	67
Left Medial Patellofemoral Surface	13	82	2	82
Left Lateral Patellofemoral Surface	22	66	5	66
Right Medial Patellofemoral Surface	13	85	2	85
Right Lateral Patellofemoral Surface	23	61	4	61
Left Medial Femoral Condyle	0	91	6	91
Left Lateral Femoral Condyle	6	76	2	76
Right Medial Femoral Condyle	3	89	5	89
Right Lateral Femoral Condyle	9	76	3	76
Left Medial Tibial Plateau	3	81	6	81
Left Lateral Tibial Plateau	5	86	3	86
Right Medial Tibial Plateau	7	82	5	82
Right Lateral Tibial Plateau	7	83	4	83
Left Perifoveal Region	0	106	1	106
Right Perifoveal Region	0	110	0	110
Left Femoral Head	23	52	1	52
Right Femoral Head	13	55	0	55
Left Lunate Acetabular Surface	2	110	0	110
Right Lunate Acetabular Surface	0	113	0	113

*Note: OP denotes osteopyhtes; EB denotes eburnation.

Table 28: Occurrence of Porosity with Other Osteoarthritic Lesions in European American Females

	*PR Only	*OP & *PR Only	*EB & *PR Only	*OP *EB *PR Only	*PR Total
Left Medial Patellar Articular Facet	3	31	0	1	35
Left Lateral Patellar Articular Facet	10	26	0	1	37
Right Medial Patellar Articular Facet	4	33	0	1	38
Right Lateral Patellar Articular Facet	12	22	0	1	35
Left Medial Patellofemoral Surface	4	28	0	1	33
Left Lateral Patellofemoral Surface	12	25	0	1	38
Right Medial Patellofemoral Surface	4	33	0	1	38
Right Lateral Patellofemoral Surface	5	33	0	2	40
Left Medial Femoral Condyle	13	49	0	5	67
Left Lateral Femoral Condyle	22	36	0	3	61
Right Medial Femoral Condyle	6	53	0	4	63
Right Lateral Femoral Condyle	18	35	0	2	55
Left Medial Tibial Plateau	10	37	0	3	50
Left Lateral Tibial Plateau	8	24	0	3	35
Right Medial Tibial Plateau	7	49	0	3	59
Right Lateral Tibial Plateau	6	37	0	2	45
Left Perifoveal Region	1	76	0	0	77
Right Perifoveal Region	1	76	0	1	78
Left Femoral Head	15	39	0	1	55
Right Femoral Head	14	44	0	2	60
Left Lunate Acetabular Surface	4	70	0	1	75
Right Lunate Acetabular Surface	2	70	0	2	74

*Note: OP denotes osteopyhtes; EB denotes eburnation; PR denotes porosity.

Table 29: Occurrence of Porosity with Other Osteoarthritic Lesions in African American Males

	*PR Only	*OP & *PR Only	*EB & *PR Only	*OP *EB *PR Only	*PR Total
Left Medial Patellar Articular Facet	2	13	0	0	15
Left Lateral Patellar Articular Facet	3	10	0	1	14
Right Medial Patellar Articular Facet	0	15	0	1	16
Right Lateral Patellar Articular Facet	2	9	0	0	11
Left Medial Patellofemoral Surface	1	11	0	0	12
Left Lateral Patellofemoral Surface	1	13	0	1	15
Right Medial Patellofemoral Surface	1	10	0	0	11
Right Lateral Patellofemoral Surface	4	5	0	0	9
Left Medial Femoral Condyle	0	12	0	1	13
Left Lateral Femoral Condyle	2	15	0	0	17
Right Medial Femoral Condyle	2	13	0	2	17
Right Lateral Femoral Condyle	5	10	0	1	16
Left Medial Tibial Plateau	1	15	0	1	17
Left Lateral Tibial Plateau	1	14	0	0	15
Right Medial Tibial Plateau	1	13	0	2	16
Right Lateral Tibial Plateau	2	12	0	0	14
Left Perifoveal Region	0	23	0	0	23
Right Perifoveal Region	0	22	0	1	23
Left Femoral Head	4	17	0	1	22
Right Femoral Head	4	18	0	0	22
Left Lunate Acetabular Surface	0	24	0	0	24
Right Lunate Acetabular Surface	0	24	0	1	25

*Note: OP denotes osteopyhtes; EB denotes eburnation; PR denotes porosity.

Table 30: Occurrence of Porosity with Other Osteoarthritic Lesions in European American Males

	*PR Only	*OP & *PR Only	*EB & *PR Only	*OP *EB *PR Only	*PR Total
Left Medial Patellar Articular Facet	8	61	0	2	71
Left Lateral Patellar Articular Facet	16	44	0	4	64
Right Medial Patellar Articular Facet	8	55	0	2	65
Right Lateral Patellar Articular Facet	14	41	0	2	57
Left Medial Patellofemoral Surface	10	48	0	2	60
Left Lateral Patellofemoral Surface	19	44	0	5	68
Right Medial Patellofemoral Surface	9	49	0	2	60
Right Lateral Patellofemoral Surface	27	39	0	4	70
Left Medial Femoral Condyle	13	77	0	6	96
Left Lateral Femoral Condyle	25	64	0	2	91
Right Medial Femoral Condyle	15	72	0	5	92
Right Lateral Femoral Condyle	23	60	0	3	86
Left Medial Tibial Plateau	20	65	0	6	91
Left Lateral Tibial Plateau	12	68	0	3	83
Right Medial Tibial Plateau	12	60	0	5	77
Right Lateral Tibial Plateau	10	61	0	4	75
Left Perifoveal Region	1	99	0	1	101
Right Perifoveal Region	2	107	0	0	109
Left Femoral Head	36	39	0	1	76
Right Femoral Head	44	41	0	0	85
Left Lunate Acetabular Surface	5	103	0	0	108
Right Lunate Acetabular Surface	3	110	0	0	113

*Note: OP denotes osteopyhtes; EB denotes eburnation; PR denotes porosity.

Table 31: Cross Tabulations for the Hip of European American Females

Joint Surface	*OP Frequency	*EB Frequency			
<i>*Lt Perifoveal Region</i>	<i>Frequency</i>	Absent	Total		
	Absent	1	1		
	Trace	9	9		
	Slight	58	58		
	Moderate	9	9		
	Severe	1	1		
	Total	78	78		
<i>*Rt Perifoveal Region</i>	Absent	Absent	Slight	Total	
	Trace	1	0	1	
	Slight	8	0	8	
	Moderate	56	1	57	
	Severe	8	0	8	
	Total	4	0	4	
	Total	77	1	78	
<i>*Lt Femoral Head</i>	Absent	Absent	Slight	Total	
	Trace	22	0	22	
	Slight	47	0	47	
	Moderate	9	1	10	
	Total	1	0	1	
	Total	79	1	80	
<i>*Rt Femoral Head</i>	Absent	Absent	Trace	Slight	Total
	Trace	19	0	0	19
	Slight	43	0	0	43
	Severe	15	1	1	17
	Total	1	0	0	1
	Total	78	1	1	80
<i>*Lt Lunate Acetabular Surface</i>	Absent	Absent	Trace	Total	
	Trace	5	0	5	
	Slight	21	0	21	
	Moderate	53	1	54	
	Total	5	0	5	
	Total	84	1	85	
<i>*Rt Lunate Acetabular Surface</i>	Absent	Absent	Slight	Total	
	Trace	3	0	3	
	Slight	18	0	18	
	Moderate	58	3	61	
	Severe	2	0	2	
	Total	1	0	1	
	Total	82	3	85	

*Note: OP denotes osteophytes; EB denotes eburnation; Lt denotes Left; Rt denotes Right.

Table 32: Cross Tabulations for the Hip of African American Males

Joint Surface	*OP Frequency	*EB Frequency		
<i>*Lt Perifoveal Region</i>	<i>Frequency</i>	Absent	Total	
	Absent	1	1	
	Trace	2	2	
	Slight	10	10	
	Moderate	9	9	
	Severe	2	2	
	Total	24	24	
<i>*Rt Perifoveal Region</i>	Absent	Absent	Trace	Total
	Trace	1	0	1
	Slight	1	0	1
	Moderate	14	0	14
	Severe	6	0	6
	Severe	1	1	2
	Total	23	1	24
<i>*Lt Femoral Head</i>	Absent	Absent	Slight	Total
	Trace	5	0	5
	Slight	9	0	9
	Moderate	8	1	9
	Severe	2	0	2
	Total	24	1	25
<i>*Rt Femoral Head</i>	Absent	Absent	Total	
	Trace	4	4	
	Slight	9	9	
	Moderate	10	10	
	Severe	2	2	
	Total	25	25	
<i>*Lt Lunate Acetabular Surface</i>	Trace	Absent	Total	
	Slight	4	4	
	Moderate	19	19	
	Severe	1	1	
	Severe	1	1	
	Total	25	25	
<i>*Rt Lunate Acetabular Surface</i>	Trace	Absent	Slight	Total
	Slight	5	0	5
	Moderate	18	1	19
	Severe	1	0	1
	Severe	1	0	1
	Total	25	1	26

*Note: OP denotes osteophytes; EB denotes eburnation; Lt denotes Left; Rt denotes Right.

Table 33: Cross Tabulations for the Hip of European American Males

Joint Surface	*OP Frequency	*EB Frequency		
	Frequency	Absent	Severe	Total
<i>*Lt Perifoveal Region</i>	Absent	1	0	1
	Trace	22	0	22
	Slight	60	1	61
	Moderate	22	0	22
	Severe	3	0	3
	Total	108	1	109
<i>*Rt Perifoveal Region</i>	Absent	Absent	Total	
	Trace	2	2	
	Slight	11	11	
	Moderate	80	80	
	Severe	11	11	
	Total	115	115	
<i>*Lt Femoral Head</i>	Absent	Absent	Moderate	Total
	Trace	60	0	60
	Slight	33	0	33
	Severe	18	0	18
	Severe	0	1	1
	Total	111	1	112
<i>*Rt Femoral Head</i>	Absent	Absent	Total	
	Trace	59	59	
	Slight	34	34	
	Moderate	20	20	
	Severe	1	1	
	Total	114	114	
<i>*Lt Lunate Acetabular Surface</i>	Absent	Absent	Total	
	Trace	7	7	
	Slight	30	30	
	Moderate	70	70	
	Severe	9	9	
	Total	117	117	
<i>*Rt Lunate Acetabular Surface</i>	Absent	Absent	Total	
	Trace	3	3	
	Slight	32	32	
	Moderate	76	76	
	Severe	5	5	
	Total	117	117	

*Note: OP denotes osteophytes; EB denotes eburnation; Lt denotes Left; Rt denotes Right.

Table 34: Cross Tabulations for the Patellar Articular Facets of European American Females

Joint Surface	*OP Frequency	*EB Frequency			
<i>*Lt Medial Facet</i>	<i>Frequency</i>	Absent	Trace	Slight	Total
	Absent	16	0	0	16
	Trace	21	0	0	21
	Slight	30	1	0	31
	Moderate	4	0	1	5
	Total	71	1	1	73
<i>*Lt Lateral Facet</i>		Absent	Slight	Total	
	Absent	25	0	25	
	Trace	24	0	24	
	Slight	22	1	23	
	Moderate	2	0	2	
	Total	73	1	74	
<i>*Rt Medial Facet</i>		Absent	Trace	Slight	Total
	Absent	9	0	0	9
	Trace	26	0	0	26
	Slight	33	1	0	34
	Moderate	5	0	1	6
	Total	73	1	1	75
<i>*Rt Lateral Facet</i>		Absent	Slight	Total	
	Absent	28	0	28	
	Trace	24	0	24	
	Slight	19	1	20	
	Moderate	4	0	4	
	Total	75	1	76	

*Note: OP denotes osteophytes; EB denotes eburnation; Lt denotes Left; Rt denotes Right.

Table 35: Cross Tabulations for the Patellar Articular Facets of African American Males

Joint Surface	*OP Frequency	*EB Frequency		
<i>*Lt Medial Facet</i>	<i>Frequency</i>	Absent	Total	
	Absent	3	3	
	Trace	5	5	
	Slight	14	14	
	Moderate	2	2	
	Total	24	24	
<i>*Lt Lateral Facet</i>		Absent	Slight	Total
	Absent	7	0	7
	Trace	4	0	4
	Slight	11	1	12
	Moderate	1	0	1
	Total	23	1	24
<i>*Rt Medial Facet</i>		Absent	Trace	Total
	Trace	5	0	5
	Slight	14	0	14
	Moderate	2	1	3
	Total	21	1	22
<i>*Rt Lateral Facet</i>		Absent	Total	
	Absent	7	7	
	Trace	3	3	
	Slight	12	12	
	Total	22	22	

*Note: OP denotes osteophytes; EB denotes eburnation; Lt denotes Left; Rt denotes Right.

Table 36: Cross Tabulations for the Patellar Articular Facets of European American Males

Joint Surface						
*OP Frequency		*EB Frequency				
*Lt Medial Facet	Frequency	Absent	Moderate	Severe	Total	
	Absent	16	0	0	16	
	Trace	39	0	0	39	
	Slight	38	0	1	39	
	Moderate	3	1	0	4	
	Severe	1	0	0	1	
	Total	97	1	1	99	
*Lt Lateral Facet		Absent	Slight	Moderat	Severe	Total
	Absent	27	0	0	0	27
	Trace	45	0	0	0	45
	Slight	21	3	0	0	24
	Moderate	2	0	1	1	4
	Total	95	3	1	1	100
*Rt Medial Facet		Absent	Slight	Total		
	Absent	20	0	20		
	Trace	33	0	33		
	Slight	39	2	41		
	Moderate	3	0	3		
	Severe	1	0	1		
	Total	96	2	98		
*Rt Lateral Facet		Absent	Slight	Severe	Total	
	Absent	29	0	0	29	
	Trace	40	0	0	40	
	Slight	26	1	0	27	
	Severe	0	0	1	1	
	Total	95	1	1	97	

*Note: OP denotes osteophytes; EB denotes eburnation; Lt denotes Left; Rt denotes Right.

Table 37: Cross Tabulations for the Patellofemoral Surfaces of European American Females

Joint Surface	*OP Frequency	*EB Frequency				
<i>*Lt Medial Surface</i>	<i>Frequency</i>	Absent	Trace	Total		
	Absent	22	0	22		
	Trace	29	0	29		
	Slight	23	0	23		
	Moderate	3	1	4		
	Severe	1	0	1		
	Total	78	1	79		
<i>*Lt Lateral Surface</i>		Absent	Slight	Total		
	Absent	40	0	40		
	Trace	23	0	23		
	Slight	11	0	11		
	Moderate	4	1	5		
	Severe	1	0	1		
	Total	79	1	80		
<i>*Rt Medial Surface</i>		Absent	Trace	Slight	Total	
	Absent	20	0	0	20	
	Trace	31	0	0	31	
	Slight	26	0	1	27	
	Moderate	5	1	0	6	
	Total	82	1	1	84	
<i>*Rt Lateral Surface</i>		Absent	Trace	Slight	Moderate	Total
	Absent	32	0	0	0	32
	Trace	28	0	0	0	28
	Slight	15	1	1	1	18
	Moderate	7	0	0	0	7
	Total	82	1	1	1	85

*Note: OP denotes osteophytes; EB denotes eburnation; Lt denotes Left; Rt denotes Right.

Table 38: Cross Tabulations for the Patellofemoral Surfaces of African American Males

Joint Surface	*OP Frequency	*EB Frequency		
<i>*Lt Medial Surface</i>	<i>Frequency</i>	Absent	Total	
	Absent	5	5	
	Trace	6	6	
	Slight	10	10	
	Moderate	2	2	
	Severe	1	1	
	Total	24	24	
<i>*Lt Lateral Surface</i>		Absent	Trace	Total
	Absent	7	0	7
	Trace	6	0	6
	Slight	7	1	8
	Moderate	2	0	2
	Total	22	1	23
<i>*Rt Medial Surface</i>		Absent	Total	
	Absent	6	6	
	Trace	7	7	
	Slight	9	9	
	Moderate	2	2	
	Total	24	24	
<i>*Rt Lateral Surface</i>		Absent	Total	
	Absent	10	10	
	Trace	7	7	
	Slight	7	7	
	Total	24	24	

*Note: OP denotes osteophytes; EB denotes eburnation; Lt denotes Left; Rt denotes Right.

Table 39: Cross Tabulations for the Patellofemoral Surfaces of European American Males

Joint Surface		*OP Frequency		*EB Frequency			
*Lt Medial Surface	Frequency	Absent	Slight	Severe	Total		
	Absent	27	0	0	27		
	Trace	50	0	0	50		
	Slight	31	0	0	31		
	Moderate	3	1	0	4		
	Severe	0	0	1	1		
	Total	111	1	1	113		
*Lt Lateral Surface		Absent	Trace	Slight	Moderate	Severe	Total
	Absent	46	0	0	0	0	46
	Trace	42	0	0	0	0	42
	Slight	18	1	0	1	0	20
	Moderate	2	0	1	0	1	4
	Severe	0	1	0	0	0	1
	Total	108	2	1	1	1	113
*Rt Medial Surface		Absent	Trace	Slight	Total		
	Absent	25	0	0	25		
	Trace	48	0	0	48		
	Slight	37	0	0	37		
	Moderate	3	0	1	4		
	Severe	1	1	0	2		
	Total	114	1	1	116		
*Rt Lateral Surface		Absent	Slight	Moderate	Severe	Total	
	Absent	52	0	0	0	52	
	Trace	36	0	0	0	36	
	Slight	18	1	1	0	20	
	Moderate	5	0	0	0	5	
	Severe	0	0	1	1	2	
	Total	111	1	2	1	115	

*Note: OP denotes osteophytes; EB denotes eburnation; Lt denotes Left; Rt denotes Right.

Table 40: Cross Tabulations for the Femoral Condyles of European American Females

Joint Surface		*OP Frequency		*EB Frequency			
*Lt Medial Condyle	Frequency	Absent	Trace	Slight	Moderate	Total	
	Absent	17	0	0	0	17	
	Trace	27	1	0	0	28	
	Slight	25	0	0	0	25	
	Moderate	5	0	1	2	8	
	Severe	0	1	0	0	1	
	Total	74	2	1	2	79	
*Lt Lateral Condyle		Absent	Slight	Severe	Total		
	Absent	32	0	0	32		
	Trace	22	0	0	22		
	Slight	15	2	0	17		
	Moderate	6	0	1	7		
	Severe	1	0	1	2		
	Total	76	2	2	80		
*Rt Medial Condyle		Absent	Trace	Slight	Moderate	Severe	Total
	Absent	13	0	0	0	0	13
	Trace	32	0	0	0	0	32
	Slight	28	0	0	0	0	28
	Moderate	7	0	1	1	2	11
	Severe	0	1	0	0	0	1
	Total	80	1	1	1	2	85
*Rt Lateral Condyle		Absent	Moderate	Severe	Total		
	Absent	27	0	0	27		
	Trace	32	0	0	32		
	Slight	13	1	0	14		
	Moderate	9	1	1	11		
	Severe	1	0	0	1		
	Total	82	2	1	85		

*Note: OP denotes osteophytes; EB denotes eburnation; Lt denotes Left; Rt denotes Right.

Table 41: Cross Tabulations for the Femoral Condyles of African American Males

Joint Surface		*OP Frequency		*EB Frequency	
<i>*Lt Medial Condyle</i>	<i>Frequency</i>	Absent	Severe	Total	
	Trace	10	0	10	
	Slight	11	1	12	
	Moderate	1	0	1	
	Total	22	1	23	
<i>*Lt Lateral Condyle</i>	Absent	6	6		
	Trace	4	4		
	Slight	14	14		
	Total	24	24		
<i>*Rt Medial Condyle</i>	Absent	3	0	0	3
	Trace	7	0	0	7
	Slight	11	0	1	12
	Moderate	1	1	0	2
	Total	22	1	1	24
<i>*Rt Lateral Condyle</i>	Absent	10	0	10	
	Trace	5	0	5	
	Slight	8	1	9	
	Total	23	1	24	

*Note: OP denotes osteophytes; EB denotes eburnation; Lt denotes Left; Rt denotes Right.

Table 42: Cross Tabulations for the Femoral Condyles of European American Males

Joint Surface		*OP Frequency		*EB Frequency		
<i>*Lt Medial Condyle</i>	<i>Frequency</i>	Absent	Trace	Slight	Moderate	Total
	Absent	15	0	0	0	15
	Trace	45	0	0	0	45
	Slight	43	1	0	0	44
	Moderate	1	1	3	1	6
	Severe	0	0	1	0	1
	Total	104	2	4	1	111
<i>*Lt Lateral Condyle</i>		Absent	Slight	Moderate	Total	
	Absent	31	0	0	31	
	Trace	43	0	0	43	
	Slight	29	0	1	30	
	Moderate	5	0	0	5	
	Severe	1	1	0	2	
	Total	109	1	1	111	
<i>*Rt Medial Condyle</i>		Absent	Slight	Moderate	Severe	Total
	Absent	21	0	0	0	21
	Trace	45	0	0	0	45
	Slight	37	1	0	0	38
	Moderate	5	0	2	1	8
	Severe	0	1	0	0	1
	Total	108	2	2	1	113
<i>*Rt Lateral Condyle</i>		Absent	Trace	Slight	Severe	Total
	Absent	36	0	0	0	36
	Trace	41	0	0	0	41
	Slight	28	0	0	0	28
	Moderate	4	1	0	1	6
	Severe	2	0	1	0	3
	Total	111	1	1	1	114

*Note: OP denotes osteophytes; EB denotes eburnation; Lt denotes Left; Rt denotes Right.

Table 43: Cross Tabulations for the Tibial Plateaus of European American Females

Joint Surface		*OP Frequency		*EB Frequency		
<i>*Lt Medial Plateau</i>	<i>Frequency</i>	Absent	Slight	Moderate	Total	
	Absent	20	0	0	20	
	Trace	30	0	0	30	
	Slight	22	2	0	24	
	Moderate	3	0	1	4	
	Total	75	2	1	78	
<i>*Lt Lateral Plateau</i>		Absent	Trace	Slight	Severe	Total
	Absent	23	0	0	0	23
	Trace	29	0	0	0	29
	Slight	19	0	0	0	19
	Moderate	2	1	1	2	6
	Total	73	1	1	2	77
<i>*Rt Medial Plateau</i>		Absent	Slight	Moderate	Total	
	Absent	13	0	0	13	
	Trace	31	0	0	31	
	Slight	30	1	1	32	
	Moderate	3	1	1	5	
	Total	77	2	2	81	
<i>*Rt Lateral Plateau</i>		Absent	Moderate	Severe	Total	
	Absent	13	0	0	13	
	Trace	36	0	0	36	
	Slight	26	1	0	27	
	Moderate	3	0	2	5	
	Total	78	1	2	81	

*Note: OP denotes osteophytes; EB denotes eburnation; Lt denotes Left; Rt denotes Right.

Table 44: Cross Tabulations for the Tibial Plateaus of African American Males

Joint Surface		*OP Frequency		*EB Frequency	
<i>*Lt Medial Plateau</i>	<i>Frequency</i>	Absent	Severe	Total	
	Absent	6	0	6	
	Trace	4	0	4	
	Slight	13	1	14	
	Total	23	1	24	
<i>*Lt Lateral Plateau</i>		Absent	Total		
	Absent	4	4		
	Trace	4	4		
	Slight	16	16		
	Total	24	24		
<i>*Rt Medial Plateau</i>		Absent	Slight	Severe	Total
	Absent	4	0	0	4
	Trace	8	0	0	8
	Slight	11	1	1	13
	Total	23	1	1	25
<i>*Rt Lateral Plateau</i>		Absent	Total		
	Absent	5	5		
	Trace	6	6		
	Slight	13	13		
	Total	24	24		

*Note: OP denotes osteophytes; EB denotes eburnation; Lt denotes Left; Rt denotes Right.

Table 45: Cross Tabulations for the Tibial Plateaus of European American Males

Joint Surface		*OP Frequency		*EB Frequency		
<i>*Lt Medial Plateau</i>	<i>Frequency</i>	Absent	Trace	Slight	Moderate	Total
	Absent	15	0	0	0	15
	Trace	45	0	0	0	45
	Slight	43	1	0	0	44
	Moderate	1	1	3	1	6
	Severe	0	0	1	0	1
	Total	104	2	4	1	111
<i>*Lt Lateral Plateau</i>		Absent	Slight	Moderate	Total	
	Absent	31	0	0	31	
	Trace	43	0	0	43	
	Slight	29	0	1	30	
	Moderate	5	0	0	5	
	Severe	1	1	0	2	
	Total	109	1	1	111	
<i>*Rt Medial Plateau</i>		Absent	Slight	Moderate	Severe	Total
	Absent	21	0	0	0	21
	Trace	45	0	0	0	45
	Slight	37	1	0	0	38
	Moderate	5	0	2	1	8
	Severe	0	1	0	0	1
	Total	108	2	2	1	113
<i>*Rt Lateral Plateau</i>		Absent	Trace	Slight	Severe	Total
	Absent	36	0	0	0	36
	Trace	41	0	0	0	41
	Slight	28	0	0	0	28
	Moderate	4	1	0	1	6
	Severe	2	0	1	0	3
	Total	111	1	1	1	114

*Note: OP denotes osteophytes; EB denotes eburnation; Lt denotes Left; Rt denotes Right.

Vita

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