SKELETAL AGE ESTIMATION IN MODERN EUROPEAN-AMERICAN ADULTS: THE EFFECTS OF ACTIVITY, OBESITY, AND OSTEOARTHRITIS ON AGE-RELATED CHANGES IN THE ACETABULUM

By

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SKELETAL AGE ESTIMATION IN MODERN EUROPEAN-AMERICAN ADULTS: THE EFFECTS OF ACTIVITY, OBESITY, AND OSTEOARTHRITIS ON AGE-RELATED CHANGES IN THE ACETABULUM

By

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This research investigated the nature of progressive changes in the acetabulum, with the aim of determining whether they are metamorphic or degenerative and ascertaining whether they are useful for the estimation of age at death. If degenerative, these changes could be linked with osteoarthritis, potentially affected by factors like physical activity and obesity, and deemed less useful for skeletal age estimation. In order to investigate these problems, the researcher analyzed a sample of 409 female and male European-American skeletal individuals from the *W.M. Bass Donated Skeletal Collection* (University of Tennessee, Knoxville). Acetabular changes and osteoarthritis were observed and scored, and these data were compared with documented demographic data (age, sex, body mass index, and habitual/occupational activities) for the 409 individuals. Statistical tests compared acetabular changes with age, osteoarthritis, activity, and obesity data.

Acetabular changes were found to correlate strongly positively with osteoarthritis and age. This indicates that the changes occurring in the joint are degenerative rather than metamorphic, but they are still useful for age estimation. Acetabular changes were

relatively resistant to the effects of obesity and physical activity, also arguing for their relevance to studies of age. In other joints, osteoarthritis also showed strong positive correlations with age, further undermining the metamorphic vs. degenerative dichotomy of age-relevant skeletal change. Osteoarthritis exhibited limited positive correlations with obesity, but no relationship with activity. The disease likely has both biomechanical and systemic components.

In summary, the degenerative changes of the acetabulum are valid skeletal indicators of age. The etiology of osteoarthritis is multifactorial, but age is a major contributing factor. The impact of physical activity is less straightforward than once thought, and it may in some cases improve joint health. Thus, weight loss and exercise should be considered palliative for both osteoarthritis and obesity. These findings have implications not only for the study and identification of the dead, but also for the improvement of health outcomes and interventions for the living.

CHAPTER 1
INTRODUCTION

In forensic anthropological and bioarchaeological analyses, accurate interpretations of individual or group characteristics depend upon the accuracy of the scientific methods used to determine the *biological profile* (age, sex, ancestry, and stature) of skeletal remains. Accurate and precise methods of age-at-death estimation are vital to this process. In the context of medicolegal identification, broad age intervals contribute little to the elimination of potential matches from the universe of missing persons, while narrow intervals may exclude the correct missing person from consideration. In the bioarchaeological sphere, inaccurate age estimates can lead to skewed mortality profiles that incorrectly characterize past populations.

Challenges of Adult Age Estimation

In spite of its key role in the biological profile, adult age estimation still challenges the biological anthropologist (see detailed discussion in Chapter 2). Unlike in juveniles, where age-related changes follow predictable developmental patterns, age changes in adults involve limited post-developmental milestones and a more complex interaction of genes and environment (Meindl and Russell, 1998).

Biological anthropologists frequently examine joint surfaces to inform their adult age estimates. All joints change with age; but some exhibit *metamorphic* change, while others exhibit *degenerative* change (Stewart, 1979). It is generally believed that degeneration has a weaker correlation with age than does metamorphosis: skeletal degeneration is difficult to quantify and subject to many influences other than age. These include intra-individual variation and population-level differences in heredity, nutrition, mechanical loading, and activity. Further, these complicated degenerative

processes are more pronounced and variable in elderly individuals, meaning that aging accuracy decreases with advancing age (Aykroyd et al., 1999; Latham and Finnegan, 2010; Nawrocki, 2010; Winburn and Brown, 2010; 2011). Adult age estimation proves particularly challenging for forensic anthropologists, tasked with generating age estimates for an increasingly elderly U.S. population. According to missing persons data curated by the FBI's National Crime Information Center (NCIC, 2014), of the 43,646 active adult missing-persons cases, approximately 50% of missing adult females and 65% of missing adult males are 40 years of age or older. While the NCIC sample is limited by the accuracy of the data reported and entered into the database (and the fact that not all missing persons are reported), the dataset approximates the universe of unidentified decedents analyzed by forensic scientists.

Biological anthropologists must identify skeletal indicators of adult aging processes and validate methods based on these indicators, so that they can estimate age with accuracy and precision—even in older adult individuals. Much research on adult age estimation has centered on the pelvis, due to the late-onset age-related changes that occur in its *amphiarthrodial* (relatively immobile) joints: the pubic symphysis and the sacro-iliac joint. The age-related changes of the pubic symphysis—the fibrocartilagenous joint where the halves of the *os pubis* meet—have been studied for over a century (Berg, 2008; Brooks and Suchey, 1990; Cleland, 1889; Djuric et al., 2007; Gilbert and McKern, 1973; Hanihara and Suzuki, 1978; McKern and Stewart, 1957; Schmitt, 2004; Todd, 1920). In particular, the metamorphic changes of the ventral rampart (a late-forming portion of the pubic symphysis) allow accurate and precise age estimates in young adults (Brooks and Suchey, 1990; Katz and Suchey,

1989). However, the lack of precise age estimates in older individuals and the often-poor preservation of the pubic symphysis in adverse depositional environments undermine its utility as an age indicator. Likewise, the auricular surface of the ilium—the pelvic half of the sacroiliac joint—has received widespread attention in the past several decades (Buckberry and Chamberlain, 2002; Falys et al., 2006; Lovejoy et al., 1985; Mulhern and Jones, 2005; Murray and Murray, 1991; Osborne et al., 2004; Sashin, 1930; Schmitt, 2004). However, as a purely degenerative age indicator, the iliac auricular surface lacks an informative region of metamorphic change, leading to methods that either fail to characterize the variability of change in the joint (Lovejoy et al., 1985) or classify it into broad phases resulting in imprecise age estimates (Osborne et al., 2004). The auricular surface of the sacrum has also been considered for age estimation (Brown, 2015; Passalacqua, 2009; 2010), though age-related changes are even less pronounced in this region than in the problematic iliac auricular surface (Passalacqua, 2009; 2010).

Recently, biological anthropologists have also begun to explore the age-estimation potential of the acetabulum—the pelvic component of the hip (Calce, 2012; Calce and Rogers, 2011; Rissech et al., 2006; 2007; Rougé-Maillart et al., 2004; 2007; 2009). This indicator has the potential to improve adult age estimation: proponents of acetabular aging methods report high accuracy and narrow age ranges leading to precise age estimates, even for elderly individuals (Rissech et al., 2006). The acetabulum is a robust joint with a relatively protected anatomical location, often surviving extreme depositional environments (e.g., the burial environments of archaeological contexts, the fragmentation scenarios common in forensic cases of

homicide and mass disaster) when fragile joints like the pubic symphysis are not preserved.

Another strong argument for use of the acetabulum in age estimation would be evidence that the changes observed in this joint are metamorphic—akin to the tightly age-correlated changes of the pubic symphysis ventral rampart—rather than merely degenerative. Metamorphic skeletal changes occur after the attainment of skeletal maturity but independent of skeletal degeneration, and they are believed to be more relevant to age estimation than degenerative changes. In the pubic symphysis, for example, metamorphic changes of the ventral rampart allow accurate and precise age estimates in adults younger than 40 years. After more than a decade of research on acetabular aging, however (Calce, 2012; Calce and Rogers, 2011; Miranker, 2016; Powanda, 2008; Rissech et al., 2006; 2007; Rougé-Maillart et al., 2004; 2007; 2009), the nature of acetabular changes (i.e., metamorphic vs. degenerative) remains obscure. Further, the metamorphic vs. degenerative dichotomy itself remains relatively unexplored. Thus, it also remains unclear to what degree, if any, metamorphic changes truly outperform degenerative changes as skeletal indicators of age.

Many questions remain about the validity and utility of the acetabulum as an age indicator. Acetabular age estimation is the focus of this dissertation, and it is discussed in detail in Chapter 2.

Understanding Age-Related Change in the Acetabulum

Several factors have the potential to complicate the straightforward age correlation of the progressive changes observed in the acetabulum. Like the pubic symphysis and auricular surface, the acetabulum participates in the transfer of body mass from the upper body to the lower limb. It is thus possible that obesity—an excess

of adipose tissue—may affect acetabular changes. Unlike the amphiarthrodial joints of the pelvis, however, the acetabulum is a *diarthrodial* joint; it is relatively mobile and used in daily physical activities. Thus, it is also possible that differing levels of physical activity can influence the way the acetabulum changes with age. Further, diarthrodial joints commonly develop osteoarthritis (OA)—the degeneration of skeletal articular surfaces. Biological anthropologists have long noted that OA increases with age (e.g., Stewart, 1979); certainly, elderly individuals suffer more frequently from OA than younger individuals (Hunter and Eckstein, 2009). However, the correlation between advancing age and increasing OA is generally viewed as weak, due to the many other factors influencing OA development. These factors include both vigorous physical activity (Felson and Zhang, 1998; Larsen, 1982; 1997) and obesity (Coggon et al., 2001; Couchman, 2009; Felson et al., 1988; Felson et al., 2000; Fransen, 2011; Mandl, 2007). If the changes observed in the acetabulum are akin to the processes of OA observed in the other diarthrodial joints of the body, then acetabular changes are likely degenerative rather than metamorphic. This could mean that they are more highly correlated with factors like activity and obesity than with age.

The effects of activity and obesity on the age-related changes of the acetabulum—and the relationship of these changes with OA—have not been investigated. Before biological anthropologists can responsibly use the acetabulum to estimate age, research must ascertain: Are acetabular changes affected by patterns of physical activity and obesity? Are the changes truly metamorphic, or are they degenerative? What does that mean in terms of their correlation with age? If they merely constitute degenerative change, are they still useful for adult age estimation?

Research Objectives

The goal of this research is to understand how age, activity, obesity, and OA affect changes in the acetabulum. Biological anthropologists cannot accurately estimate age using skeletal indicators unless they understand the biology underlying the changes observed in those indicators. Thus, this dissertation aims to provide a more complete understanding of progressive acetabular changes that will enable the validation and/or refinement of acetabular age estimation methods, improving their objectivity and reliability. The current research compares age-related changes in the acetabulum with OA in other joints of the body, in order to determine whether acetabular changes constitute degenerative or metamorphic change. This research also investigates the relative influences of age, activity, and obesity on the acetabulum in particular, and OA in general, in order to untangle the relationships between joint degeneration and these contributing factors.

The current research is particularly relevant to forensic anthropological age estimation in an increasingly elderly U.S. population, in light of reports that acetabular changes are informative for elderly adult age estimation (Rissech et al., 2006). This research responds to the standard set by the *Daubert* court decision for rigorously tested forensic science methods that are widely accepted within the scientific community and for which error rates and standards for application have been generated (Daubert v. Merrell Dow Pharmaceuticals, 1993). It also constitutes a response to the recent call by the National Academy of Sciences (NAS) for studies establishing the scientific bases demonstrating the validity, reliability, and accuracy of forensic methods (NRC, 2009). Method validation and refinement are consistent with both NAS

recommendations (NRC, 2009) and the *Daubert* standards (Daubert v. Merrell Dow Pharmaceuticals, 1993).

The implications of this research are not, however, purely medicolegal. Improved age-estimation methods also benefit bioarchaeologists, paleoanthropologists, and any other biological anthropologists studying the aging process in past populations. A more nuanced understanding of the etiology of OA informs bioarchaeological research, which has often attempted to link observed joint degeneration with posited physical activities and other lifestyle variables. Finally, and perhaps most importantly, this research contributes to the growing body of medical research on aging, much of which has focused on OA: its multifactorial etiology, the relative importance of various risk factors, and the palliative potential of interventions like exercise and weight loss. Thus, these dissertation findings may have repercussions not only for interpretations of the dead, but also for health outcomes in living populations.

Chapter Outline

This dissertation explores the relationships among age, activity, obesity, and OA and their relative contributions to progressive changes of the acetabulum. Chapter 2 ("Skeletal Age Estimation") provides an overview of the biological processes of human aging and the method and theory underlying skeletal age estimation, with particular emphasis on the acetabulum as an age indicator. Chapter 3 ("The Human Hip") reviews the growth, development, and basic biomechanics of the hip, in addition to providing an evolutionary context for bipedal pelvic adaptations and outlining pathological hip conditions. Chapter 4 ("Essentials of Osteoarthritis") discusses major contributing factors to OA (age, activity, and obesity) and changing interpretations of disease etiology through time.

Chapter 5 (“Materials and Methods”) describes the study protocols used to test for correlations among age, activity, obesity, OA, and acetabular changes in a sample of donated skeletal individuals with documented demographic information (*n*=409). A Bayesian statistical method of acetabular aging is used to estimate age in these individuals (Rissech et al., 2006), and the appropriateness of this method is assessed. Frequentist statistical methods (e.g., correlation tests, linear regression) are used to test whether OA has a relationship with acetabular changes and to ascertain which of several factors (age, activity, obesity) are associated with generalized OA throughout the body and specific progressive changes in the acetabulum.

Chapter 6 (“Results”) presents study outcomes: while acetabular changes are found to be degenerative (strongly correlated with OA), they prove to be strongly correlated with age and relatively resistant to the effects of activity and obesity; these findings undermine the metamorphic vs. degenerative dichotomy in skeletal aging and validate the acetabulum as an age indicator. In other joints of the body, OA is also strongly correlated with age and relatively resistant to the effects of activity, though limited evidence emerges for a positive association with obesity. Finally, Chapter 7 (“Discussion”) considers the implications of these results, and Chapter 8 (“Conclusions”) summarizes and contextualizes the research.

CHAPTER 2
SKELETAL AGE ESTIMATION

Estimating Adult Age

The estimation of age at death is a vital component of the biological profile—the biological snapshot of a deceased individual that allows their medicolegal identification or a bioarchaeological interpretation of their life and death. Age estimates in juvenile individuals frequently are both accurate (close to documented chronological age) and precise (with small ranges). Yet, accurate and precise adult age-at-death estimation still challenges the biological anthropologist.

Estimating age at death from the developing skeleton hinges on assessments of predictable patterns of dental and skeletal growth. In contrast, the aging processes visible in the adult skeleton are far more variable. After the completion of development, skeletal milestones indicating age progression are limited. With the exception of unique loci of metamorphic change in the adult skeleton (e.g., the pubic symphysis ventral rampart; see *Pelvic Age Estimation*, below), most of the age-related changes assessed in adult age estimation involve skeletal degeneration. Degeneration is a complicated biological process that is both genetically programmed and environmentally impacted (Meindl and Russell, 1998). Inter-individual differences in obesity, bone density, mechanical loading, and hormonal levels affect skeletal degeneration in ways that are poorly understood (see Chapter 4). Skeletal age estimation methods that attempt to condense these complex biological processes into simplified age-progressive phases often fail (e.g., Lovejoy et al., 1985; see *Pelvic Age Estimation*, below). Inter- and intra-observer error further contribute to the challenges of estimating adult age.

The degenerative processes that complicate adult age estimation only become more pronounced and variable as an individual ages, and this trajectory effect (Nawrocki, 2010) translates to further-decreased accuracy with advancing age (Aykroyd et al., 1999; Winburn and Brown, 2010; 2011; cf. Milner and Boldsen, 2012). Thus, age estimates for adults of advanced age are more difficult to achieve, less accurate, and less precise than age estimates for young adults, and anthropological aging methods tend to underestimate age in the oldest individuals (Buikstra and Ubelaker, 1994; Komar and Buikstra, 2008; Latham and Finnegan, 2010). Yet, due to advances in health care and standards of living, modern individuals are more likely than prehistoric peoples to reach extreme old age (Vaupel, 2004). This presents a particular problem for forensic anthropologists, who face the challenge of providing the accurate age estimates that enable identifications for a world population with a rising mean age at death.

Statistical Theory of Age Estimation

Developing an age estimation method from a skeletal sample is a three-step process. First, age-related morphological features are identified in a reference sample; second, these features are linked with chronological age in the reference sample; finally, the method is used to estimate age in individuals from a target sample (Hoppa and Vaupel, 2002). Often, observations are grouped into age-progressive descriptive categories called phases or stages, each associated with descriptive statistics generated from the reference sample (e.g., mean, standard deviation, age range). Once the method is developed, the actual process of age estimation involves observation of an unknown set of remains, comparison with the stages/phases and descriptions generated from the reference sample, and assignment of an estimated age range—moving “from stage to age” (Hoppa and Vaupel, 2002, p. 1).

This conversion of observed skeletal features into chronological ages relies on statistical inference. The major theories of statistical inference applied to skeletal age estimation are the frequency-based (or frequentist) and Bayesian approaches. Frequentist approaches include the most commonly used analytical procedures (e.g., Student's *t*-test, linear regression), many of which come with assumptions that are not always supported by biological data like those generated from aging studies (e.g., normality, repeatable and random sampling during which parameters remain constant). Bayesian approaches are not governed by the same assumptions, and thus have been gaining attention for their relevance to the statistical needs of biological anthropology. Both approaches are used in the current research.

At the heart of both frequency-based and Bayesian statistics is the goal of using probabilities to describe uncertainties; the differences between the approaches lie in the type of uncertainties that each approach describes and measures. Frequentist statisticians only use probabilities to describe the types of uncertainties inherent in the occurrence of random events (e.g., tossing a coin), using the frequency definition of probability: the long-run frequency with which an event occurs if it is repeated indefinitely. In contrast, Bayesian statisticians use probabilities to describe the types of uncertainties that are due not to randomness, but to lack of knowledge. These types of uncertainties differ not only between propositions, but also between people, whose knowledge about the propositions necessarily differs (O'Hagan, 2004). All researchers undertake statistical analysis in order to learn about parameters and reduce the second type of uncertainty. But since they do not make statements about the probabilities of those parameters, frequency-based inferences (e.g., significance tests, confidence

intervals) engage with the parameters only indirectly, and only in terms of repeated sampling. Bayesian inferences, on the other hand, describe how "the acquisition of data modifies (and usually reduces) the uncertainty about a parameter, from 'prior' uncertainty to 'posterior' uncertainty" (O'Hagan, 2004, p. 133).

A Bayesian approach incorporates prior knowledge (informed or uninformed priors) in order to strengthen probability statements. In this scheme, posterior probabilities are those probabilities of a variable that are dependent on other variables; prior probabilities are those probabilities of a variable that are independent of other variables. Bayes' theorem describes how the knowledge of prior probabilities is used to find probabilities of unknown events (Byers and Roberts, 2003); it relates posterior to prior probabilities in such a way that observations about one variable (*A*) are modified by the observation of another variable (*B*): $P(A|B)=[P(B|A)f(A)]/P(B)$.

In terms of skeletal age estimation, Bayes' theorem might be interpreted as follows: the posterior probability that a skeleton represents an individual of age *A* (given observed characteristics *B* of the remains) is equal to the posterior probability that those characteristics (*B*) are observed in a reference sample given age *A*, times the prior probability distribution of ages-at-death in the target population of interest (*fA*), divided by the prior probability of those characteristics (*B*) in the target population (Hoppa and Vaupel, 2002; Love and Müller, 2002). The fact that in order to assess $P(A|B)$ one must first estimate the probability distribution of ages-at-death has spawned an interest in the quality and quantity of reference populations, and in the methods necessary to analyze them and estimate their mortality distributions. This work—as well as multiple calls for physical anthropologists and osteologists to embrace Bayesian approaches to age-at-

death estimation—forms the basis of what is known as the “Rostock Manifesto” for paleodemography (Hoppa and Vaupel, 2002, p. 2).

Unlike frequency-based practitioners, Bayesian paleodemographers following the recommendations of the “Rostock Manifesto” can make probability statements directly about the parameters of a distribution (here, the probability that a set of skeletal remains are from an individual of a given age, given the suite of characteristics exhibited). The initial (prior) probability represents the chance of an individual belonging to an age group before any information about the individual is known (Lucy et al., 1996). The likelihood represents the conditional probability of any given suite of skeletal characteristics occurring in an individual of a given age (Hoppa and Vaupel, 2002). The posterior probability is the conditional probability of an individual being in a given age group, given observations from reference samples and the individual’s stage ranking (Lucy et al., 1996). Prior distributions can come from estimates of the age-at-death distribution for the target population (Boldsen et al., 2002) or from information independent of the target: priors can be uniform (i.e., uninformed priors), they can represent an arbitrary lifespan distribution, or they can comprise a distribution known to be similar to the population under study (Hoppa and Vaupel, 2002; Lucy et al., 2002). For example, in his study of acetabular degeneration in London’s Spitalfields skeletal sample (18th-19th-century A.D.), Mays (2012) conducted a nonparametric Bayesian analysis using five sets of prior probabilities of age: uniform priors; priors from model life tables; priors from the Spitalfields age distribution; and priors from documentary sources on mortality in Medieval England and in the Roman Empire.

In the years since the publication of the “Rostock Manifesto,” using a Bayesian approach to ascertain posterior probability of age given stage has become the norm among paleodemographers estimating age in adult remains (Mays, 2012). Forensic anthropological age estimation methods have also begun to incorporate Bayesian approaches (e.g., Brennaman et al., 2016; Lucy et al., 1996; Rissech et al., 2006). In particular, multifactorial Bayesian age estimation methods incorporating data from multiple skeletal age indicators are currently experiencing a surge in popularity (Boldsen et al., 2002; Bullock et al., 2013; Cappella et al., 2015; Langley-Shirley and Jantz, 2010; Rougé-Maillart et al., 2009). However, unlike paleodemographic studies, which undertake to generate population trends in mortality and life expectancy, forensic identification necessitates accurate and precise age estimates for single individuals (Passalacqua, 2009). Compared with a population age structure, a single age estimate for any one individual is inherently imprecise, since physiological age indicators vary widely among the individuals in any one age group (Aykroyd et al., 1997).

This raises the question of how to identify the target population from which an unknown, unidentified individual originates. While the violation of assumptions of frequentist age analyses (e.g., that age variables are independent, that they vary linearly with age, that errors are normally distributed) affects Bayesian analyses to a lesser degree (Byers and Roberts, 2003; Lucy et al., 1996), Bayesian approaches do make a major assumption of their own: that the mortality distribution of the prior sample resembles that of the target sample. Prior age-at-death distribution data from homicide victims can be used to estimate age in forensic cases (Boldsen et al., 2002). Steadman and colleagues (2006, p. 17) have emphasized the necessity for the prior distribution in

forensic cases to capture the “population at large,” noting that its definition will change depending on context. For example, for a military anthropologist, the “population at large” might include all individuals killed in action in a particular conflict. Clearly, however, this population is not representative of the overall U.S. “population at large.” The importance of a representative prior population cannot be overestimated: choosing an inappropriate source biases the prior probabilities calculated from these sources (Byers and Roberts, 2003); these biases have a greater impact in forensic than in paleodemographic age estimation, since they can potentially lead to elimination of the correct individual from a list of possible unidentified decedents. In other words, when inappropriate priors are chosen, the advantages of Bayesian reasoning become liabilities.

Bayesian approaches allow forensic anthropologists to quantify the probability that their identifications are correct (Sironi and Taroni, 2015)—akin to the probability of a unique DNA identification (Steadman et al., 2006). These types of probabilistic statements have become increasingly important in the years since the Daubert v. Merrell Dow Pharmaceuticals (1993) call for increasingly quantified and objective forensic methods. Compared with the broad age ranges generated by frequency-based approaches (e.g., the auricular surface aging method of Osborne et al., 2004, in which one phase has a mean of 58.9 years and a two-standard-deviation range of 28.4-89.4 years), the accurate and precise age estimates reported by the authors of Bayesian methods seem attractive (e.g., Rissech et al., 2006; Rougé-Maillart et al., 2009). However, the narrow age intervals originally proposed for methods like that of Rissech and colleagues (2006) may be overly optimistic (see *Acetabular Age Estimation*, below);

as described above, degeneration is variable and difficult to quantify, particularly for the elderly. Age estimates based on degenerative changes of the pelvic joints (see *Pelvic Age Estimation*, below) may not be precise enough to be useful in the Bayesian probabilistic approach to identification touted by Steadman et al. (2006). The broad age ranges of the more traditional frequentist approaches to aging may merely reflect the biological reality of degenerative change.

In summary, both frequentist and Bayesian approaches continue to be used in biological anthropological age estimation. In most frequentist (i.e., regression-based) aging techniques, age is treated as the dependent variable being predicted by the independent variable of the observed skeletal traits. Bayesian aging techniques take a slightly more biologically realistic approach, in which prior knowledge is used to predict the transition of an individual from a given developmental phase to the next phase in an ordered sequence (Konigsberg et al., 2008). The former is better suited to continuous, normally distributed data; the latter is appropriate for analyzing discrete, age-progressive phases when scores from relevant reference samples are available. Bayesian approaches have gotten more attention in paleodemographic studies, though forensic anthropologists are also developing ways to incorporate this type of inference into their age estimation studies.

Both approaches are relevant to the current research, with Bayesian inference employed to generate age estimates based on the acetabulum and frequentist statistics forming the basis for testing the significance of associations among age, activity, obesity, OA, and acetabular changes. The applications of these approaches are detailed in Chapter 5.

Pelvic Age Estimation

Much research in adult skeletal age estimation has focused on the way that articular surfaces change with age. Intuitively, it seems likely that degenerative processes operate more slowly and regularly in the body's relatively immobile amphiarthrodial joints, in comparison with the more mobile diarthrodial joints that are implicated in locomotion and other habitual physical activities. Two amphiarthrodial joints are found in the pelvis, making this body region a focus of adult skeletal age estimation. In the pubic symphysis and sacro-iliac joint, certain age-related changes occur slowly, beginning early and continuing through the later decades of life (Meindl and Russell, 1998). The pubic symphysis is distinguished by the fact that some of its age changes represent metamorphic rather than solely degenerative joint changes—resulting in accurate and precise age estimates in adults up to approximately age 40. The strength of the iliac auricular surface allegedly lies in its robusticity; it is often described as more resistant than the pubic symphysis to post-depositional processes like weathering and breakage (Buckberry and Chamberlain, 2002; Igarashi et al., 2005; Lovejoy et al., 1985; Meindl and Lovejoy, 1989). The auricular surface of the sacrum has also been considered for age estimation, though age-related changes in this region are not pronounced (Brown, 2015; Passalacqua, 2009; 2010).

In contrast with the traditional focus on amphiarthrodial joints, recent research has indicated that the acetabulum—the pelvic component of the diarthrodial hip joint—may also be valuable for estimating adult age (Calce, 2012; Calce and Rogers, 2011; Rissech et al., 2006; 2007; Rougé-Maillart et al., 2004; 2007; 2009). Researchers have touted both its resistance to postmortem damage (Rougé-Maillart et al., 2007) and its utility in estimating age in elderly adults (Rissech et al., 2006); age change in this

diarthrodial joint may even represent metamorphic change. Methods of age estimation using the pubic symphysis and auricular surface are briefly reviewed in the following subsections. Age estimation using the sacrum is not discussed further in this review. As the focus of this dissertation, age estimation using the acetabulum is discussed in detail in its own section (see *Acetabular Age Estimation*, below).

Pubic Symphysis Age Estimation

Pubic symphysis aging has a reputation for reliability, and the joint has purportedly received more scholarly attention than any other skeletal age indicator (Meindl and Russell, 1998; Aykroyd et al., 1999; Krogman, 1962). In life, the left and right symphyses are united by fibrocartilage at the anterior aspect of the pelvic girdle. Research has focused on the relationship of the metamorphic and degenerative changes of the pubic symphysis with age for over 120 years (Berg, 2008; Brooks and Suchey, 1990; Cleland, 1889; Djuric et al., 2007; Gilbert and McKern, 1973; Hanihara and Suzuki, 1978; Hartnett, 2010; Katz and Suchey, 1989; McKern and Stewart, 1957; Sinha and Gupta, 1995; Schmitt, 2004).

The relevance of the pubic symphysis ventral rampart for adult age estimation was not recognized, however, until the work of Todd (1920). A strip of bone beveling that forms on the ventral face of the symphyseal surface, the ventral rampart appears and fuses by approximately age 35. These “delayed epiphyseal events” distinguish the pubic symphysis from other, primarily degenerative, skeletal indicators of adult age (Meindl and Russell, 1998, p. 385). Essentially, while the rest of the adult skeleton has already begun the process of degenerative change, the pubic symphysis is still undergoing metamorphic change. This metamorphic change translates to accurate and precise age estimates until approximately age 40. In addition to the formation of the

ventral rampart, age-related bone changes in the pubic symphysis include the loss of the dense, ridged and furrowed symphyseal surface texture characteristic of youth and its replacement with a pitted, porous, and irregularly textured symphyseal surface, delineated by the ventral rampart on one side and dorsal osteophytic lipping on the other.

Most pubic symphysis age estimation methods rely on comparing joint surfaces with photographic, diagrammatic, or cast exemplars representing these different phases of joint change, with separate standards and statistics for females and males to reflect the sexes' hormonal and functional differences (e.g., Brooks and Suchey, 1990; Todd, 1920). In a recent survey, practicing forensic anthropologists ranked the pubic symphysis as their most preferred skeletal region for adult age estimation; the vast majority (95%) used the Brooks and Suchey (1990) method (Garvin and Passalacqua, 2012). This popular method provides sex-specific standards for six phases of symphyseal change, depicted in resin reference casts.

The ventral rampart—the great benefit of the pubic symphysis for adult age estimation up to age 40—is also its downfall once skeletal metamorphsis is complete. After the formation of this metamorphic landmark, the pubic symphysis itself begins to degenerate, a process that proves as variable as degeneration in any other joint of the adult skeleton (Meindl and Russell, 1998). Consequently, methods of aging based on the pubic symphysis tend to yield accurate age estimates only in individuals less than 40 years of age (Brooks and Suchey, 1990; Djuric et al., 2007; Gilbert and McKern, 1973; Hanihara and Suzuki, 1978; Krogman, 1962; Meindl and Russell, 1998; Sinha and Gupta, 1995; cf. Berg, 2008 and Hartnett, 2010). The loss of precision experienced

by the Brooks and Suchey (1990) method after the completion of metamorphic change exemplifies the impact of biological variability on phase-based skeletal aging. In the early Suchey-Brooks Phases I and II, 95% confidence intervals comprise tight age ranges of 8-21 years. However, in later Phases III-VI, 95% confidence intervals can stretch to 45 or even 58 years (Brooks and Suchey, 1990). Although broad age ranges and limited old-age discrimination may be satisfactory for bioarchaeological research, the later phases of the method may be too broad and variable to be useful forensically (Brooks and Suchey, 1990). The joint's utility is also undermined by its fragility and relatively unprotected anterior anatomical position: post-depositional processes frequently damage the delicate pubic symphysis, particularly in burial settings (Buckberry and Chamberlain, 2002; Lovejoy et al., 1985; Miller, 1993).

Auricular Surface Age Estimation

The iliac auricular surface was recognized as a potential age indicator nearly 100 years ago (Todd, 1920; Sashin, 1930), and it has received widespread attention for the past several decades (Buckberry and Chamberlain, 2002; Falys et al., 2006; Igarashi et al., 2005; Lovejoy et al., 1985; Moraitis et al., 2014; Mulhern and Jones, 2005; Murray and Murray, 1991; Osborne et al., 2004; Rissech et al., 2012; Rougé-Maillart, 2009; Schmitt, 2004). The joint surface is alleged to be more robust and resistant to post-depositional processes than the fragile pubic symphysis, recommending it for both forensic and bioarchaeological analyses (Buckberry and Chamberlain, 2002; Igarashi et al., 2005; Lovejoy et al., 1985; Meindl and Lovejoy, 1989).

Comprised in youth of fibrous cartilage with a columnar alignment running dorso-ventrally, the soft tissue covering the auricular surface becomes more rough and frayed beginning in the 30's (Meindl and Lovejoy, 1989), with corresponding age-related

changes to the underlying bone (Lovejoy et al., 1985). Unlike in the pubic symphysis, these changes appear to occur independently of sex, but they are solely degenerative; no informative region of metamorphic change has been identified in the auricular surface (Lovejoy et al., 1985; Murray and Murray, 1991; Osborne et al., 2004). Auricular surface changes include the loss of finely grained, horizontally billowed bone texture and its replacement first with coarsely granular bone, and later with irregular, densified bone marked with porosity and marginal osteophytic activity.

Lovejoy and colleagues (1985) codified the first auricular surface aging method, linking eight phases of auricular surface change with narrow five-year age ranges. However, even the authors themselves reported that the method was difficult to apply (Lovejoy et al., 1985), and subsequent research highlighted further problems, including: high inter-observer error; low replicability; questionable statistical validity of the five-year age intervals; a range of error too large for applicability in single cases; and the overestimation of age in young adults and underestimation of age in older adults (Buckberry and Chamberlain, 2002; Falys et al., 2006; Mulhern and Jones, 2005; Murray and Murray, 1991; Osborne et al., 2004; Rissech et al., 2012; Schmitt, 2004). Many of these flaws may be due to the complexity and variability of age-related degenerative change occurring in this region. Indeed, later methodological and statistical modifications to the auricular surface method have acknowledged this complexity by utilizing broader age intervals that allow easier application and higher accuracy (Buckberry and Chamberlain, 2002; Falys et al., 2006; Mulhern and Jones, 2005; Osborne et al., 2004). In spite of these improvements, however, the method of Lovejoy and colleagues (1985) remains the most frequently used auricular surface

technique among forensic anthropologists (Garvin and Passalacqua, 2012). The auricular surface (independent of method) ranks third among the preferred skeletal regions for adult age estimation (Garvin and Passalacqua, 2012).

Problems with auricular surface age estimation include the lack of metamorphic change in the region, the corresponding variability of the degenerative processes occurring in the joint, and the broad, forensically meaningless age intervals (and imprecise age estimates) that necessarily result (Winburn and Brown, 2010; 2011).

Acetabular Age Estimation

Like the other joints of the pelvis, the acetabulum exhibits age-related changes. The acetabulum is a robust joint with a relatively protected anatomical location (Calce and Rogers, 2011; Rougé-Maillart et al., 2007), often surviving extreme depositional environments (e.g., burial, fragmentation) and postmortem carnivore scavenging (Haglund et al., 1989) in which the pubic symphyses and even the auricular surfaces are damaged (Powanda, 2008). Recently developed acetabular methods have been shown to yield accurate age estimates with narrow age ranges (Rissech et al., 2006)—rivaling the performance of even the frequently used pubic symphysis. The acetabulum has the potential to discriminate age in the elderly, while the pubic symphysis reaches its discriminatory peak by 40 years (Rissech et al., 2006). Perhaps most importantly, the changes observed in the acetabulum may be metamorphic—akin to the tightly age-correlated changes of the pubic symphysis ventral rampart.

These age changes, first noted in the work of Rougé-Maillart and colleagues (2004), occur in four regions of the acetabulum: the acetabular rim; acetabular fossa; lunate surface; and apex. In their seminal study of the male acetabulum, Rougé-Maillart and colleagues (2004) used terminology derived from an iliac auricular surface aging

method (Lovejoy et al., 1985) to describe the morphological characteristics of these four acetabular regions. The appearance of each of the four regions was categorized in one of three-to-five stages and tested using non-parametric statistical methods to determine the correlation between age and stage. Rougé-Maillart and colleagues (2004) found that variables of three of the regions (characteristics of the acetabular rim, acetabular fossa, and apex) correlated significantly with age, as well as with the original criteria for age estimation in the auricular surface (Lovejoy et al., 1985). Lunate surface activity, while significantly correlated with age, proved too variable to be a meaningful indicator of age (Rougé-Maillart et al., 2004).

In a subsequent study of the male acetabulum, Rissech and colleagues (2006) refined and expanded the Rougé-Maillart methodology by eliminating the lunate surface and further quantifying observations of the remaining informative areas of the acetabulum: the acetabular rim; the acetabular fossa; and the fossa's outer edge (Figure 2-1). They presented seven variables to be scored in the three regions, with each one assigned to any of a series of states describing the morphological characteristics observed and the degree of their expression (Table 2-1 and Figures 2-2 through 2-8). Scores obtained by this method (Rissech et al., 2006) are analyzed via a Bayesian statistical program (IDADE2) that estimates a relative likelihood distribution for the target individuals and produces age-at-death estimates and 95% confidence intervals.

Rather than confining estimates to comparison with one reference collection, the IDADE2 software has the capacity to estimate age based on any and all skeletal populations. The only criteria are that the chosen reference sample is of known-age,

and that its mortality distribution resembles that of the target sample. That all users of IDADE2 can choose different reference scores against which to estimate age in their desired sample should, in theory, lend the method flexibility and greater applicability to diverse populations. However, acetabular variable scores from multiple previously scored skeletal samples are not currently available to the user of IDADE2.

Rissech and colleagues' use of Bayesian estimation reflects a trend by some aging researchers away from traditional linear regression models of estimation and toward a prediction model that incorporates both prior and posterior probabilities (Lucy et al., 1996; Hoppa and Vaupel, 2002; Schmitt et al., 2002). Age-related skeletal changes are variable and non-linear, and the variables chosen to describe them are frequently categorical (Lucy et al., 1996). Bayesian inference has been demonstrated to describe these data more appropriately than linear regression (Lucy et al., 1996; Schmitt et al., 2002). Proponents allege that age estimates made using Bayesian inference are more accurate and less biased than those made using traditional methods (Aykroyd et al., 1999; Lucy et al., 2002; Miranker, 2016; Powanda, 2008; Rissech et al., 2006).

Indeed, Rissech and colleagues (2006) initially reported high accuracy (the difference between known and estimated age was within 10 years in over 89% of the sample) and narrow age ranges—even for the elderly. Low intra- and inter-observer error (possibly due to the standardized numerical scoring technique) and significant correlation between observed variables and age were also reported (Rissech et al., 2006). Subsequent research exploring population variation in acetabular age changes further supported the acetabulum as a valid age indicator and indicated that the method

is applicable to multiple European and European-American populations (Miranker, 2016; Powanda, 2008; Rissech et al., 2007). While the method was developed using an all-male skeletal sample, patterns of aging in the acetabulum are similar between females and males (San-Millán et al., 2016), suggesting that the method could also be applied to females.

Acetabular method revisions

The original research on the acetabulum has inspired subsequent studies as well as methodological changes (Calce, 2012; Calce and Rogers, 2011; Mays, 2012; 2014; San-Millán et al., 2016). Some research has supported the original conclusions of Rissech and colleagues (2006), finding that the seven acetabular variables are relevant for age estimation in individuals of European ancestry—importantly, for both female and male samples (Miranker, 2016; San-Millán et al., 2016). Other research has revealed problems with the original method. The IDADE2 program is difficult to use (Calce, 2012), and it does not currently contain a database of previously scored individuals from multiple populations. Overaging of the very young has been reported (Rissech et al., 2007). More problematically, the narrow age ranges originally reported (Rissech et al., 2006) may have been overly optimistic (Calce, 2012). In Mays' (2012) Bayesian analysis of acetabular variables, posterior probabilities of age (given stage) were so dispersed as to be uninformative. Further, some research has shown that only three-to-four of the seven age-related variables originally proposed (Rissech et al., 2006) correlate with age (Calce, 2012; Mays, 2012). Authors have also reported difficulty in assessing the acetabular variables as originally described (Calce, 2012; Stull and James, 2010). Thus, method revisions have been proposed that either maintain the commitment of the original method to Bayesian age estimation based on seven

variables (San-Millán et al., 2016) or completely overhaul the system to create broad age categories (young adult, middle adult, and old adult) to which acetabula are assigned using traditional stage-to-age methodology (Calce, 2012). Age-related changes of the acetabulum simply may not be strong enough age indicators to enable the kind of precise age estimates originally reported by Rissech and colleagues (2006), as even subsequent modifications that incorporate broad age categories (Calce, 2012) have been found to yield inaccurate age estimates (Mays, 2014). It is also possible, however, that acetabular changes are valid age indicators and that appropriate method refinements will improve precision and accuracy sufficiently to enable the use of acetabular aging methods in forensic anthropological and bioarchaeological research (San-Millán et al., 2016).

Unresolved questions in acetabular aging

Unlike the other joints of the pelvis (the amphiarthrodial pubic symphysis and auricular surface), the acetabulum is a mobile, diarthrodial joint. It is also directly involved in the process of bipedal locomotion. This raises the concern that age-related changes in the acetabulum may merely constitute OA, a disease linked not with skeletal metamorphosis, but with skeletal degeneration. Indeed, some of the changes noted in the acetabulum closely resemble OA changes. These include acetabular traits involving osteophyte formation (e.g., roughening/bone growth of the acetabular rim, osteophytic activity of the apex of the posterior cornua, crest formation on the edge of the acetabular fossa) and possibly the development of macroporosity (e.g., on the acetabular rim and fossa). However, pitting and porosity, and their relationship with OA, are poorly understood (Rothschild, 1997). Further, some age changes noted in the

acetabulum appear unrelated to OA (e.g., formation of a groove around the acetabular rim, changes in the level of the acetabular fossa).

It is possible that some of these changes have not only a degenerative but also a metamorphic component. This possibility is investigated in the current research via within-individual analyses of the correlation between age-related hip changes and generalized OA (in other, particularly non-weight-bearing, joints). This analysis aims to illuminate the nature of the observed acetabular variables (i.e., osteoarthritic or metamorphic). If age-related changes in the acetabulum do not correlate with generalized OA in the same individual, it suggests that these changes may be metamorphic in nature. If this is the case, the descriptions of metamorphic joint change in the literature on pubic symphyseal aging (e.g., Brooks and Suchey, 1990) can provide a template for identifying metamorphic age-related changes. Descriptions of the non-osteoarthritic femoral head may also prove informative. In Solomon's (1976) study of 327 surgically removed femoral heads with OA, he observed that normal, aged femoral heads did not exhibit any of the changes typically associated with OA. In his antemortem antero-posterior radiographs, the femoral heads of aged individuals without OA appeared spherical, with 2/3 of the head circumscribed by the acetabulum and visible joint space between them maintained by preserved cartilage superiorly and medially (Solomon 1976). Upon gross morphological examination, the normal aged femoral head exhibited a smooth articular surface, particularly in the weight-bearing portion; however, some mild degenerative changes to the rim (i.e., minor marginal osteophytes) were visible—likely related not to disease, but to normal age progression (Solomon 1976). The diagnosis of these osteophytes as normal, healthy, and age

related was supported by internal and radiographic analyses of the femur: when the normal aged femoral head was sectioned, the articular cartilage appeared thick and homogeneous; postmortem radiographs showed intact subchondral bone and unbroken trabeculae (Solomon 1976). Many of these purported traits of healthy, aging hip joints could be discernable on skeletal remains and confirmed through radiography. The descriptions of Solomon (1976) could thus inform an analysis of age-related changes in the acetabulum, should these changes prove to be metamorphic and unrelated to OA.

If this research demonstrates a correlation between acetabular changes and generalized OA, however, it indicates a degenerative origin for the age-related changes of the acetabulum. This means that anthropologists must untangle the complexities of osteoarthritic change in the acetabulum before they can identify which indicators reflect age, and which reflect the multitude of other OA risk factors. However difficult, this task may prove fruitful. There is evidence that certain osteoarthritic changes are more positively correlated with age than with factors like activity and obesity. In radiographic clinical diagnosis of OA patients, the presence of osteophytes is frequently used as a criterion; and osteophytes are alleged to correlate well with OA symptoms (Felson and Zhang, 1998). Weiss and Jurmain (2007) have posited that these marginal osteophytes are more closely linked with age progression than with activity.

In particular, hip OA may be more closely linked with advancing age than OA in other diarthrodial joints (Jurmain, 1980; Jurmain and Kilgore, 1995). In general, and across multiple geographically and temporally distinct populations, frequencies of hip OA are relatively low (Bridges, 1991; Felson and Zhang, 1998; Fransen et al., 2011; Jurmain, 1980; 1990; Larsen, 1982; Watkins, 2010). Studies of hip OA have also

shown less conclusive correlations with risk factors like obesity, suggesting that studies of the acetabulum may be more productive for age estimation than studies of more frequently affected joints like the knee (Felson and Zhang, 1998; Hochberg, 2004). This may be because the hip is subjected to lower contact and shearing forces than the knee (Wearing et al., 2006), and the convex surface of the femoral head distributes these stresses more evenly, rendering the hip joint less subject to functional factors (i.e., activity, obesity) and more sensitive to systemic (i.e., age) changes (Jurmain, 1980).

The lower frequencies of hip than knee OA, combined with the less conclusive correlations between body mass, activity, and the degeneration of the hip, suggest that studies of the acetabulum may be more productive for age estimation than studies of joints like the knee. In essence, even if age-related changes of the acetabulum are shown to be degenerative in origin, the acetabulum may still have potential as an age indicator. Finally, there is a possibility that degenerative changes in all joints of the body will prove highly correlated with age, in which case the metamorphic vs. degenerative dichotomy for skeletal aging may require revision.

Summary

This chapter has summarized the biological, theoretical, and methodological challenges of adult age estimation. The advantages of frequentist and Bayesian statistics for skeletal age estimation were discussed and the relevance of both approaches to different facets of this research established. A review of skeletal age estimation compared and contrasted the various pelvic age indicators, focusing on the acetabulum. The next chapter continues the exploration of this joint, providing an overview of the development, biomechanics, evolutionary context, and diseases of the human hip.

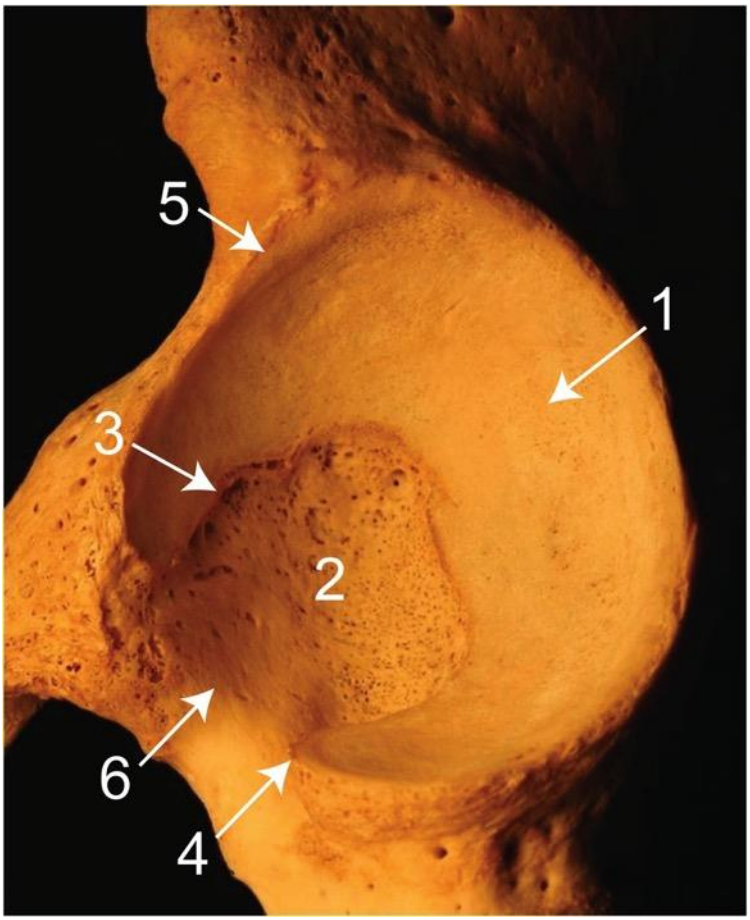


Figure 2-1. Terminology for the Rissech et al. (2006) method: 1. Lunate surface; 2. Acetabular fossa; 3. Outer edge of fossa; 4. Apex; 5. Acetabular rim; 6. Acetabular notch. Photograph by A.P. Winburn.

Table 2-1. Morphological descriptions of the seven acetabular variables and their states (after Rissech et al., 2006, p. 215-217).

Variable	Description of variable	Characteristics of the states
Acetabular groove	Groove surrounding the internal acetabular rim	In youth: no anatomical interruption between lunate surface and rim. With age: pronounced groove. States 0-3.
Acetabular rim shape	The outer rim of the acetabulum	In youth: dense, round and smooth. With age: rough, with osteophytic growth. States 0-6.
Acetabular rim porosity	The texture/activity of the outer rim	In youth: smooth, little porosity. With age: macroporosity and destruction. States 0-5.
Apex activity	Osteophytic activity on the apex of the posterior cornua	In youth: round, smooth apex. With age: osteophytic growth of one (sometimes both) horns of the lunate surface. States 0-4.
Fossa edge activity	Crest formation between fossa and lunate surface	In youth: smooth, no delineation between surfaces. With age: visible crest. States 0-5.
Fossa activity	Level and activity of the acetabular fossa	In youth: dense, level acetabular fossa. With age: irregular with much activity. States 0-5.
Fossa porosity	Texture and integrity of the acetabular fossa	In youth: dense, smooth fossa. With age: macroporosity and destruction. States 0-6.

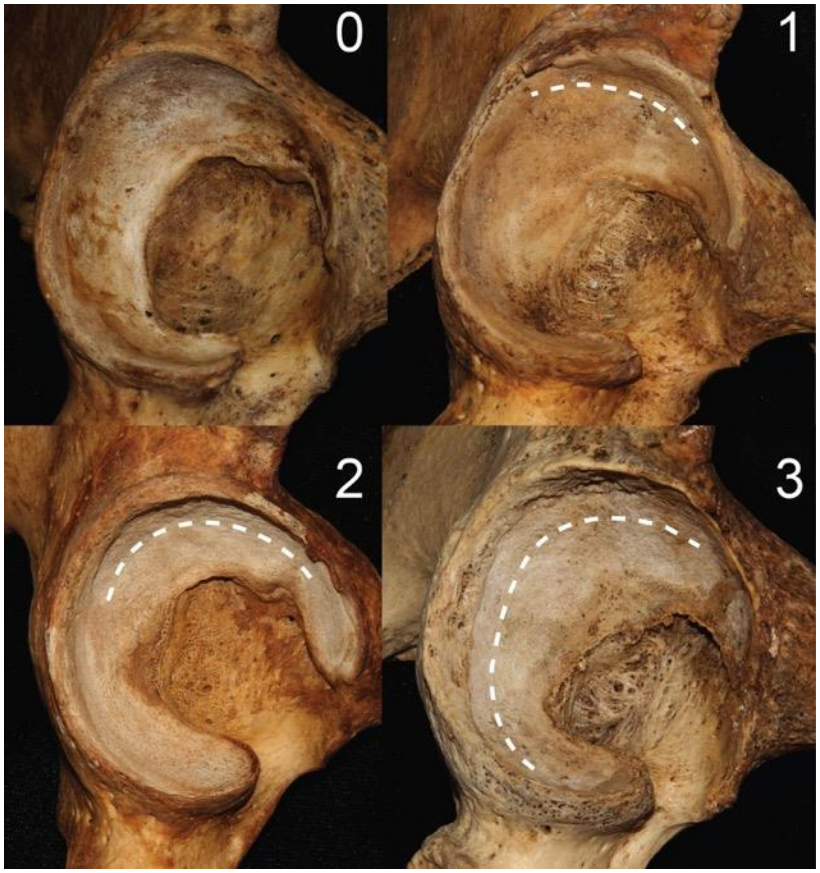


Figure 2-2. Variable 1: acetabular groove. State 0 (no groove) shows no groove below the acetabular rim; state 1 (groove) shows a short, shallow anatomical interruption between the lunate surface and acetabular rim; state 2 (pronounced groove) shows a deeper groove surrounding a larger part of the acetabular rim; state 3 (very pronounced groove) shows a groove surrounding nearly all of the acetabular rim. White dashed lines show extent of grooves in states 1, 2, and 3. Photographs by A.P. Winburn.

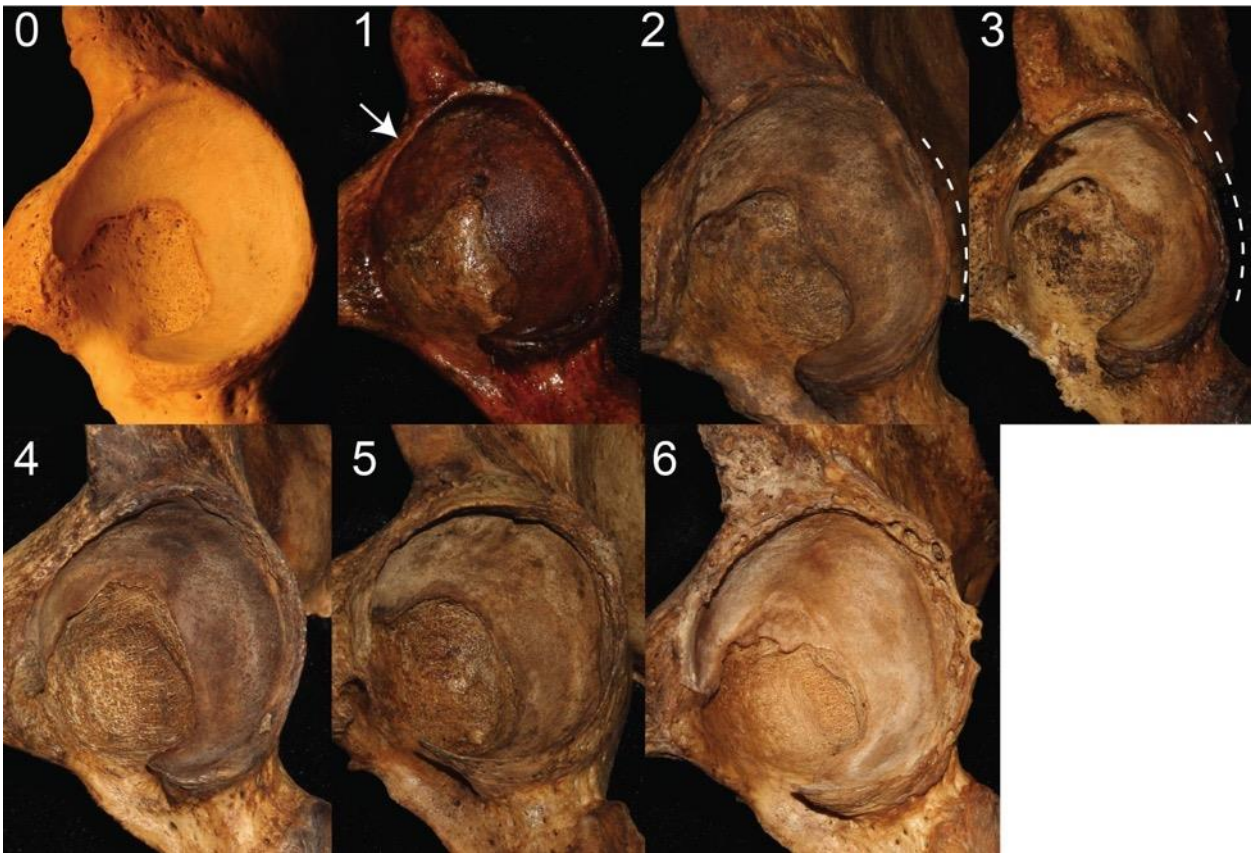


Figure 2-3. Variable 2: acetabular rim shape. State 0 (rounded acetabular rim) shows a dense, round, and smooth rim; state 1 (partially narrow acetabular rim) shows a partially rounded but partially narrow (arrow) rim; state 2 (narrow or rough acetabular rim) shows a region of roughness on the rim (white dashed line); state 3 (partially crested rim) shows a small chain of osteophytes along a small part of the rim (white dashed line); state 4 (crested rim) shows a 1-mm-high chain of osteophytes along the entire acetabular rim; state 5 (very high crested rim) shows a >4-mm-high chain of osteophytes along the entire acetabular rim; state 6 (destructured rim) shows an extremely high (>8-mm), fragile crest along the rim. Photographs by A.P. Winburn.

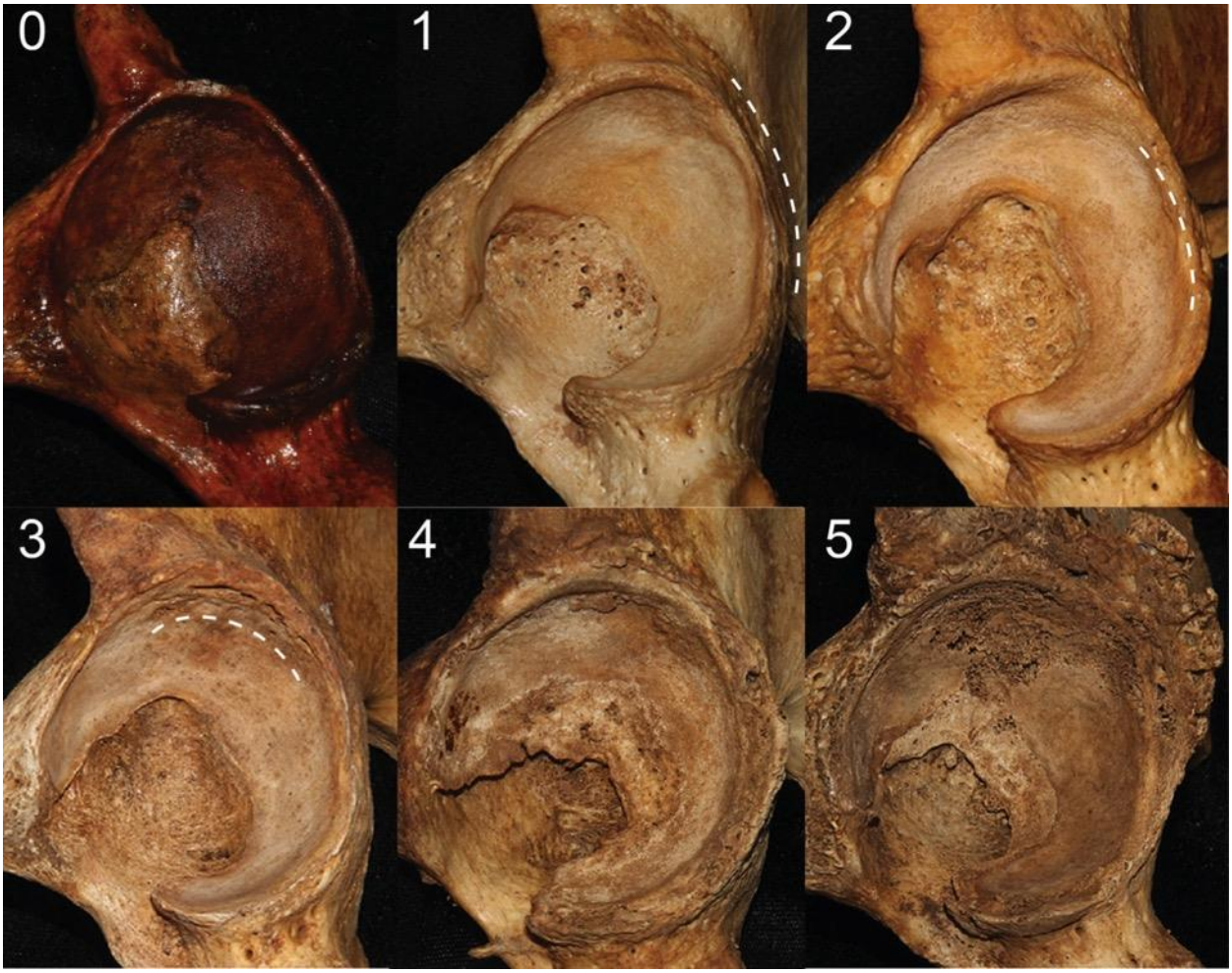


Figure 2-4. Variable 3: acetabular rim porosity. State 0 (normal porosity) shows a smooth rim; state 1 (external porosity) shows porosity posterior to the rim (white dashed line); state 2 (rim porosities) shows microporosities on a portion of the rim (white dashed line); state 3 (rough rim) shows a region of roughness and macroporosity on the rim (white dashed line); state 4 (destructured rim) shows macroporosities and destruction along the entire rim; state 5 (extremely destructured rim) shows macro- and microporosities and destruction of entire rim, with invasion of porosity below the anterior inferior iliac spine. Photographs by A.P. Winburn.

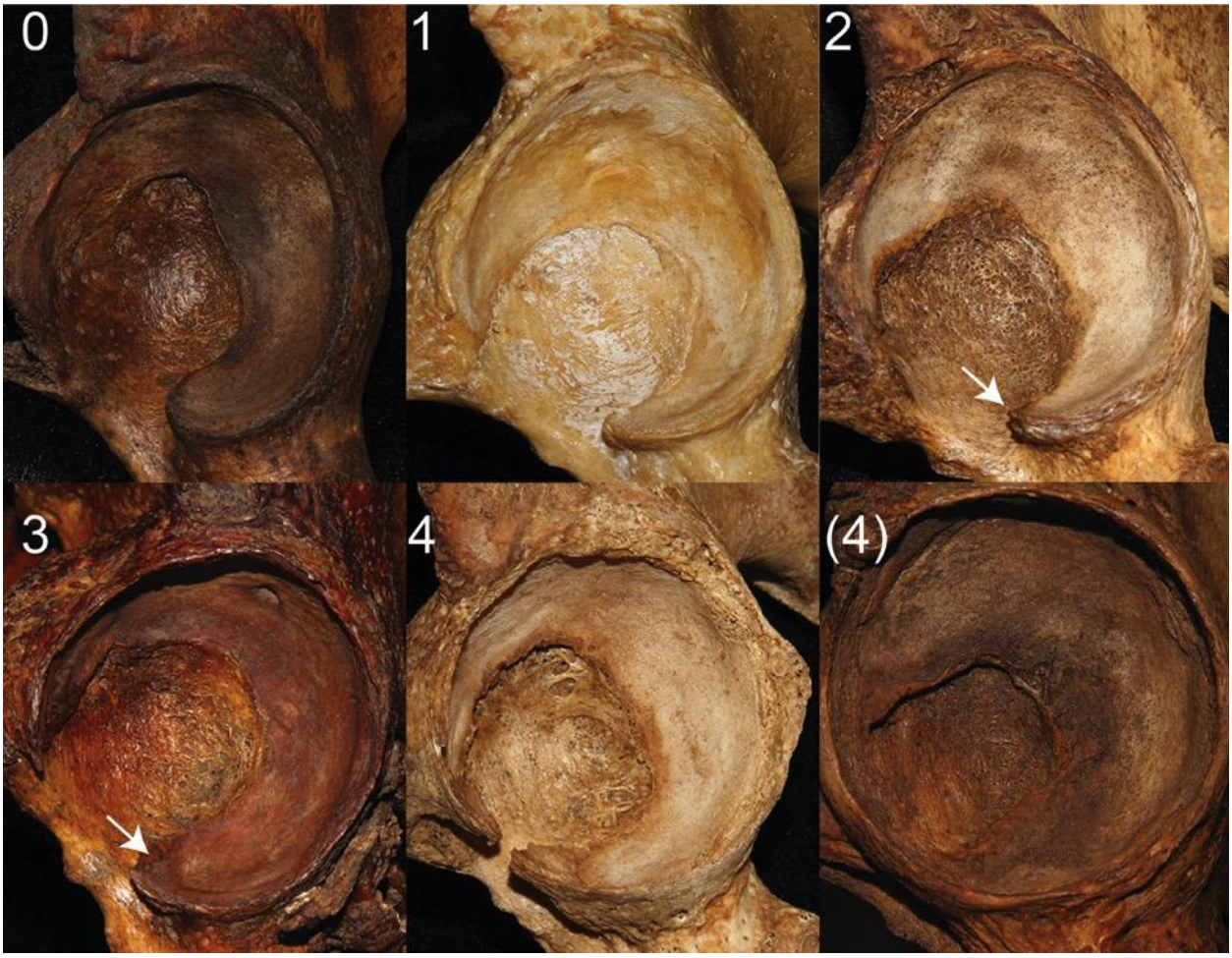


Figure 2-5. Variable 4: apex activity. State 0 (no activity) shows a round, smooth apex with no osteophyte formation; state 1 (apex activity) shows an apex that has become slightly elongated and sharp to the touch; state 2 (osteophyte activity) shows a developed and conspicuous osteophyte larger than 1 mm (arrow); state 3 (much osteophyte activity) shows an osteophyte larger than 3 mm, covering the entire horn of the apex (arrow); state 4 (very much osteophytic activity) shows an osteophyte so large (>5 mm) that it crosses the acetabular notch, also involving osteophyte formation of the anterior horn of the lunate surface; variant (4) shows complete osteophytic bridging of the anterior and posterior horns. Photographs by A.P. Winburn.

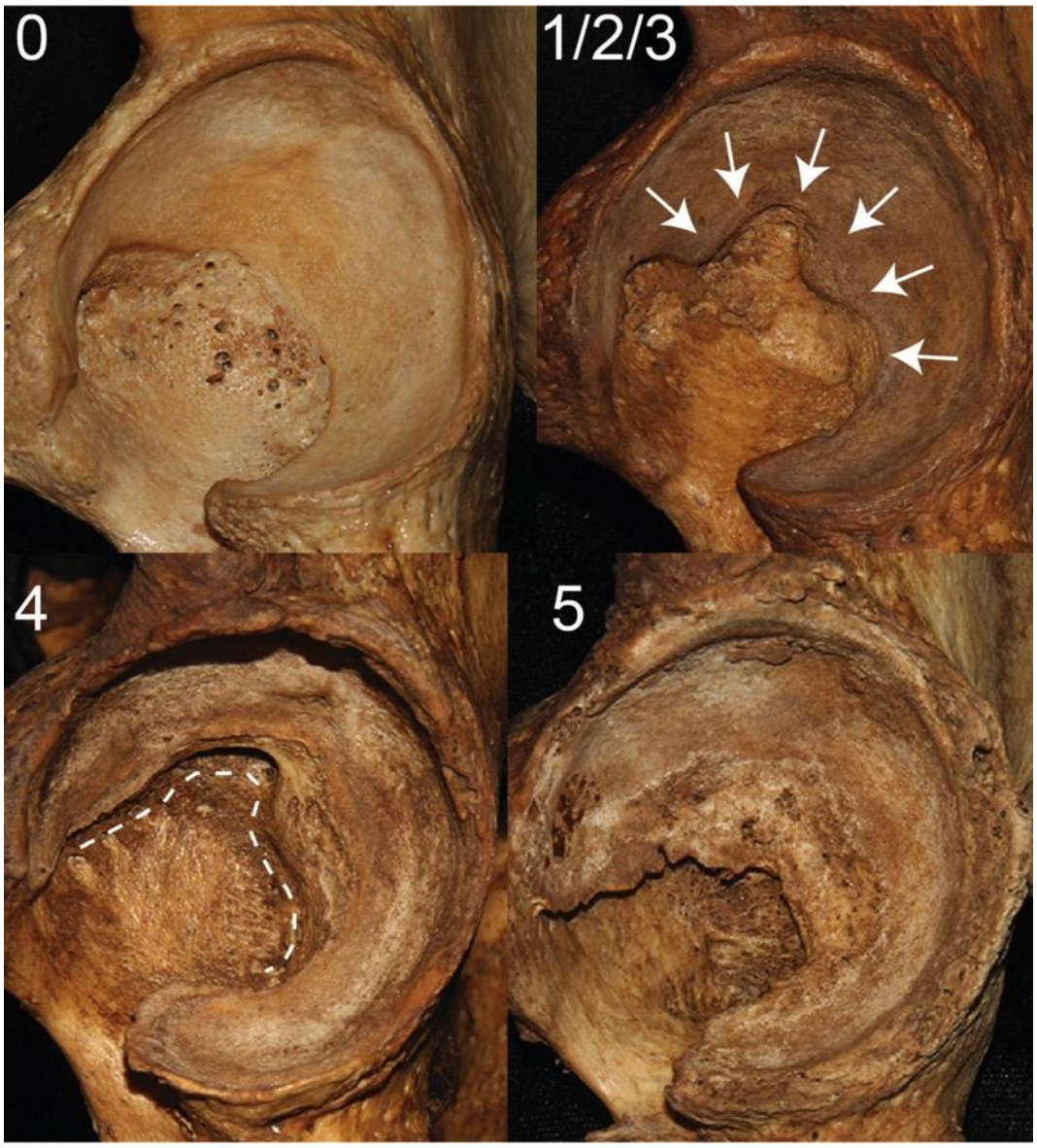


Figure 2-6. Variable 5: acetabular fossa edge activity; crest formation. State 0 (no activity on the outer edge) shows a smooth-edged acetabular fossa; state 1 (slight activity, <1/4 of the outer edge), state 2 (medium activity, <1/2 of the outer edge), and state 3 (much activity, 3/4 of the outer edge) involve a fossa with a crest that is palpable but not visible (arrows indicate how to move a finger along the outer edge of the acetabular fossa in order to palpate this state of crest); state 4 (extreme activity, >3/4 of the outer edge) shows a crest that can be both palpated and seen along most of the fossa edge (white dashed line); state 5 (destructured outer edge) shows so much visible growth on the outer fossa edge that it partially covers the fossa parallel to the outer edge. Photographs by A.P. Winburn.

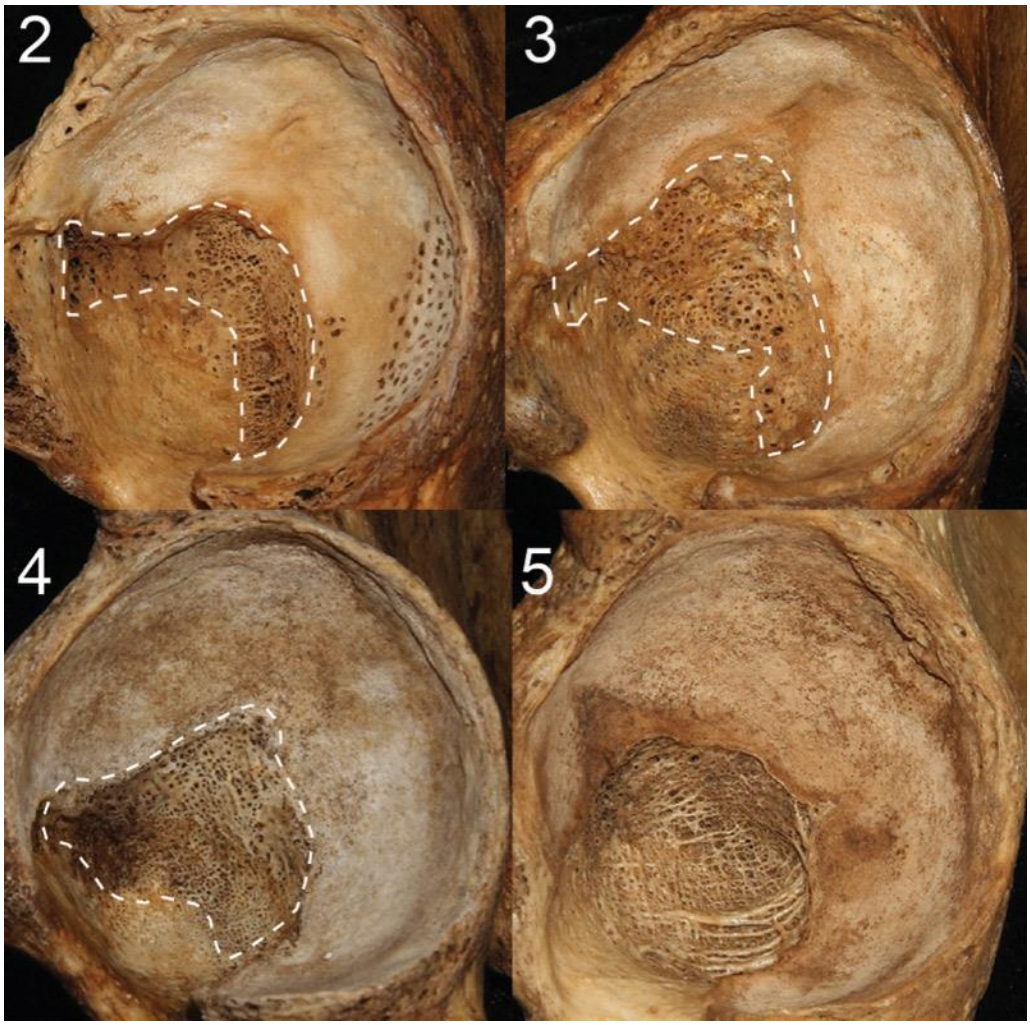


Figure 2-7. Variable 6: acetabular fossa activity. States 0 (no activity) and 1 (slight activity) are not depicted, as they were not observed in this sample. State 2 (peripheral activity) shows activity (relief, porosities, and bone production) on between 1/4 and 1/2 of the fossa along the peripheries (white dashed line); state 3 (central activity) shows fossa activity on approximately 1/2 of the fossa, extending to the middle (white dashed line); state 4 (major activity) shows activity on more then 3/4 of the fossa, though the fossa retains its density (white dashed line); state 5 (generalized activity) shows a fossa entirely covered by extensive formation, with loss of bone density and consistency. Photographs by A.P. Winburn.

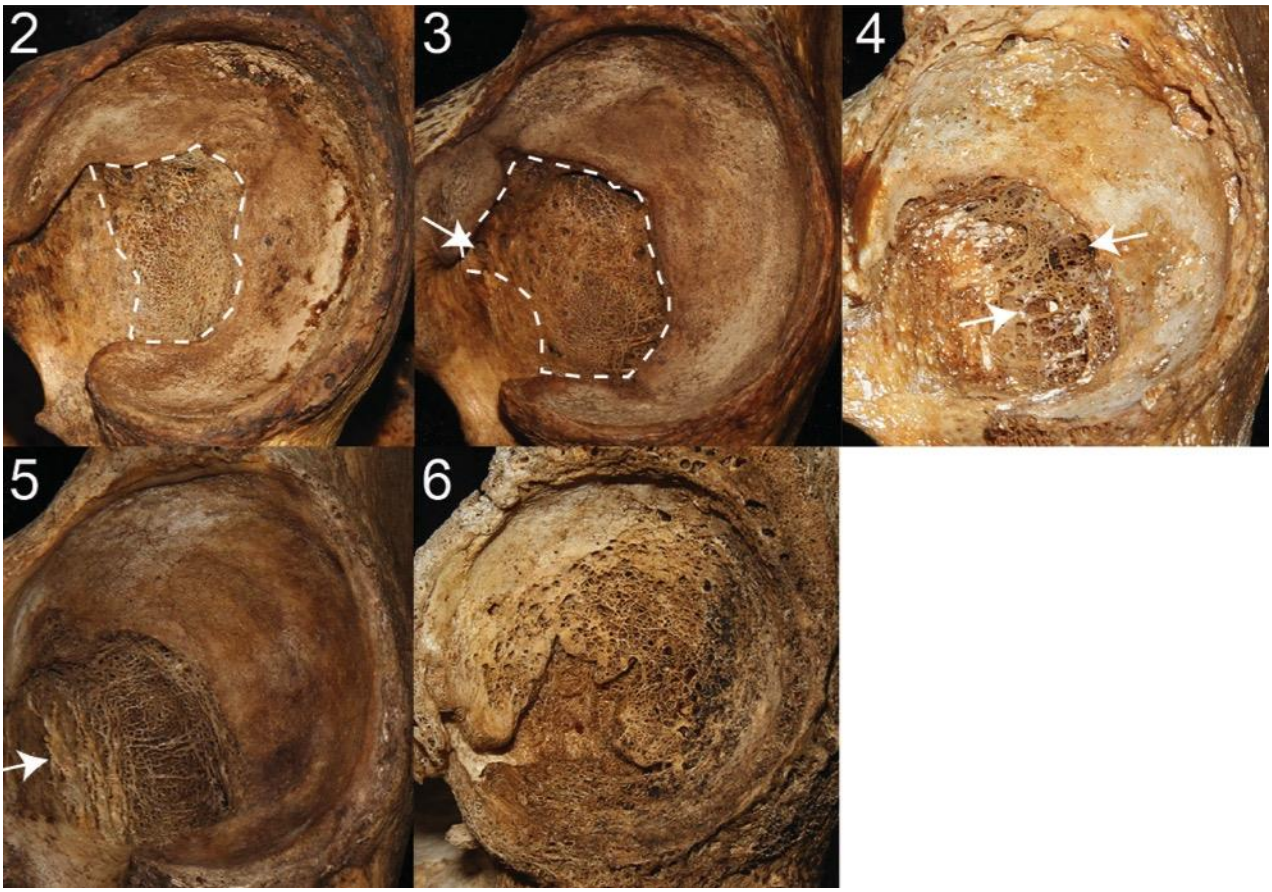


Figure 2-8. Variable 7: acetabular fossa porosity. States 0 (dense acetabular fossa) and 1 (acetabular fossa with microporosities) are not depicted, as they were not observed in this sample. State 2 (macroporosities or peripheral trabecular bone) shows porosities covering approximately 1/2 of the fossa; state 3 (macroporosities on the three lobes) shows microporosities and macroporosities (arrow) covering approximately 3/4 of the fossa; state 4 (macroporosities with destruction) shows large macroporosities with areas of sharp-edged destruction (arrows) on a base of microporosities and smaller macroporosities; state 5 (bone destruction on most of the fossa) shows a fossa covered with bone that is swollen and trabecular in appearance, with loss of consistency (arrow); state 6 (bone proliferation) shows a fossa that has been obliterated by bone proliferation. Photographs by A.P. Winburn.

CHAPTER 3
THE HUMAN HIP

The human hip joint, situated at the lateral aspect of the *os coxa* or pelvic bone, comprises the articulation of the dome-shaped acetabulum and the globular head of the femur, which represents approximately 2/3 of a sphere. Like all mobile, diarthrodial joints, the hip is comprised of skeletal, cartilaginous, and ligamentous elements, closed within a soft tissue capsule and lubricated by synovial fluid (Figure 3-1). The hip is often described as a ball-and-socket joint, and its multi-directional mobility results from the articulation of the convex surface of the femoral head (ball) within the concave surface of the acetabulum (socket). The *ligamentum teres* extending from the fovea capitis of the femoral head to the acetabular fossa provides stability, as do the ligaments surrounding the hip and the cartilaginous *labrum* that extends from the acetabular rim, deepening the socket of the cup-shaped acetabulum (Sariali et al., 2008). Twenty-seven muscles cross the hip, acting to produce movement as well as to stabilize the joint (Sariali et al., 2008) and balance a center of gravity that is located above the hips (Radin, 1980). Within the joint capsule, the subchondral articular surfaces of the acetabulum and femoral head are lined with thin (1-3mm) layers of hyaline cartilage (Hodge et al., 1986), which tends to be thicker on the femoral head than on the acetabulum (Greenwald and O'Connor, 1971). Viscous synovial fluid lubricates and nourishes the cartilage surfaces (Afoke et al., 1987; Greenwald and O'Connor, 1971).

Hip Growth and Development

In the developing human embryo, the femoral head and acetabulum differentiate out of the same block of primitive mesenchymal cells (Committee on Quality Improvement, 2000). At approximately six weeks' gestation, the acetabulum forms as a

shallow depression proximal to the femoral head, differentiating from the precursor cells that will become the three components of the os coxa: the ilium, ischium, and pubis (Lee and Eberson, 2006). The cartilaginous models of the acetabulum and femoral head are developed by approximately seven weeks' gestation, and by 11 weeks, all muscular and soft-tissue portions of the fetal hip are also formed and visible (Lee and Eberson, 2006). Primary ossification centers form for the femoral head at approximately eight weeks' gestation and for the iliac, ischial, and pubic components of the acetabulum by approximately 16 weeks (Lee and Eberson, 2006), though at birth, both the acetabulum and femoral head remain largely cartilaginous (Walker, 1981).

Situated at the junction of the three components of the os coxa, the postnatal acetabulum is formed by their articulation. In the developing child, the Y-shaped *triradiate* cartilage separates the iliac, ischial, and pubic components of the acetabulum along its medial wall, while the ring-shaped acetabular cartilage forms the acetabulum's lateral rim (Lee and Eberson, 2006). Changing in depth throughout development, the acetabulum is at its most shallow at the time of birth, likely contributing to the high rates of hip dislocation that occur at this time (see *Pathological Conditions of the Human Hip: Developmental Dysplasia*, below). After birth, this trend reverses, and the depth of the acetabulum increases with the increasingly globular femoral head, creating a more stable joint (Scheuer and Black, 2004). Indeed, the cup-shaped morphology of the acetabulum develops in response to the presence of the spherical femoral head, deepening with age as bone is deposited in the central, triradiate region as well as at the edges of the joint (Ponseti, 1978). Likewise, contact with the acetabulum helps to maintain regular, semi-spherical growth of the femoral head (Lee and Eberson, 2006).

At all stages of development, the acetabulum and femoral head act as two components of a functional whole: each needs the other in order to develop properly. Disruptions in blood supply or articulation to either component can cause disease or deformity to the other (e.g., Legg-Calvé-Perthes disease, in which anomalies of vascular development in the femoral head lead to corresponding acetabular deformities; Lee and Eberson, 2006).

At puberty, secondary acetabular ossification centers develop, ultimately forming the anterior and posterior walls of the acetabulum (Ponseti, 1978). By the age of approximately 15 years in females and 17 years in males, the triradiate cartilage fuses, joining the iliac, ischial, and pubic components of the acetabulum (Scheuer and Black, 2004). The secondary ossification center of the femoral head is fully ossified by puberty and typically fuses to the femoral neck by approximately 16 years in females and 19 years in males (Scheuer and Black, 2004). At this point, the joint is skeletally mature.

Evolution of the Bipedal Hip

The human hip is characterized by adaptations to bipedalism, a hallmark of the human condition. Because of its role in locomotion, the human hip underwent unique selective pressures during its evolution. The following sections review fossil evidence of changes in pelvis and hip design over the course of the evolution of humans and *hominins*—the bipedal primates on our evolutionary lineage. Implications of this evidence for interpretations of the nature of hominin bipedalism, obstetrics, and gestational development are also discussed.

Fossil Evidence of Hip Evolution

The pelvis is a rich source of information on locomotion. However, few fossilized hominin pelvic remains have been recovered. Hominin pelvic remains from the

Miocene are still unknown. Pliocene hominin pelvic remains include one partial adult *Ardipithecus ramidus* specimen (ARA-VP-6/500; Lovejoy et al., 2009), one partial adult *Australopithecus africanus* specimen (STS-14; Abitbol, 1995), and one partial adult *Australopithecus afarensis* specimen (AL 288-1; Johanson et al., 1982). Hominin pelvic specimens dating from the early Pleistocene include the partial adult Gona pelvis (BSN49/P27; Ruff, 2010; Simpson et al., 2008), partial adult and partial subadult *Paranthropus robustus* specimens (SK 50 and SK 3155; Brain et al., 1974; Broom and Robinson, 1950), partial adult and partial subadult *Australopithecus sediba* specimens (MH1, MH2; Kibii et al., 2011), one partial adult early *Homo* specimen (KNM-ER 3228; Rose, 1984), one partial juvenile *Homo erectus* specimen (KNM-WT 15000; Brown et al., 1985), one partial adult *H. erectus* specimen (OH 28; Day, 1971), and several more fragmentary specimens. Pelvic remains originating from later Pleistocene contexts are more numerous, and include multiple specimens of *Homo neanderthalensis* and archaic and anatomically modern *Homo sapiens*.

In cases where hominin pelvic remains have not been recovered or preserved, pelvic characteristics such as acetabular size and shape can be estimated using metric and morphological traits of the femoral head. Proximal femoral remains have been recovered for Miocene hominin *Orrorin tugenensis* (BAR 1002'00; Senut et al., 2001), Pliocene *Au. afarensis* (e.g., MAK-VP-1/1; Lovejoy et al., 2002) and *Au. africanus* (e.g., MLD 46; Reed et al., 1993), and multiple Pleistocene hominins (e.g., SK 82, SK 97; Ruff et al., 1999).

Reconstructions have been attempted for several of the above remains, including pelves STS-14 (Abitbol, 1995), KNM-WT 15000 (Walker and Ruff, 1993), BSN49/P27

(Simpson et al., 2008), MH1 and MH2 (Kibii et al., 2011), and ARA-VP-6/500 (Lovejoy et al., 2009), along with multiple *Australopithecus* and *Homo* femora (e.g., Walker, 1973). Such reconstructions have been hindered by the incomplete preservation of the specimens and the destructive taphonomic processes that have acted upon them (Gibbons, 2009; Ruff, 2010). Still, these pelvic and femoral remains have enabled paleoanthropologists to assess both biomechanical hip function and obstetric constraints for hominins.

Temporal Changes in Hominin Pelvis Design

Changes in the pelvis are among the most dramatic differences between the skeletal remains of quadrupedal and bipedal primates. These changes involve the shift from pronograde to upright posture, and include: the vertical shortening and horizontal broadening of the pelvis that lowers the bipedal primate's center of gravity, acts as a base for the torso, and provides a mechanically advantageous attachment for derived gluteal musculature; the different pattern of muscle-markings reflecting bipedal musculature, such as the increasingly robust anterior inferior iliac spine; and the increasingly large, increasingly stable hip joint.

Miocene hominins: *O. tugenensis* and *Sahelanthropus tchadensis*

Indirect evidence of bipedalism is found in the earliest fossils postdating the chimpanzee-human last common ancestor. The *O. tugenensis* femur (dated to approximately 6 million years ago [MYA]) exhibits features indicative of bipedalism, including the presence of a groove for the *obturator externus* tendon and cortical thickening in the inferior aspect of the femoral neck (Pickford et al., 2002). The inferior placement of the foramen magnum in the *S. tchadensis* fossilized cranium (dated to 7-6 MYA) also suggests bipedalism in the earliest hominins (Brunet et al., 2002).

Early Pliocene hominins: *Ar. ramidus*

Direct evidence for bipedalism can be observed in the fossilized pelvic remains of hominins postdating these early members of the hominin lineage. Changes from the long, narrow chimpanzee pelvic form are observable in the *Ar. ramidus* fossils dated to approximately 4.4 MYA: in specimen ARA-VP-6/500, the greater sciatic notch and anterior inferior iliac spine resemble those of later hominins, and the iliac blades are shorter and broader than those of chimpanzees (White et al., 2009). However, the *Ar. ramidus* pelvis is not as vertically short and horizontally broad as the pelves of later hominins, and the ischium still retains apelike traits (Lovejoy et al., 2009).

Later Pliocene hominins: genus *Australopithecus*

The apelike traits seen in the *Ar. ramidus* pelvis are modified in the direction of humanlike expression in members of the genus *Australopithecus*. The pelves of *Au. afarensis* and *Au. africanus* are characterized by wide horizontal breadths and flared ilia. The position of their anterior gluteal muscles has been alleged to be mechanically advantageous for bipedality (Lovejoy, 1979). The hip joints of these species are larger than the hip joints of arboreal quadrupeds (Jungers, 1988), and their femoral head and acetabular morphologies are within the range of normal variation seen in modern humans (Asfaw, 1985). However, *Australopithecus* pelves are not entirely humanlike in morphology and dimensions. *Australopithecus* acetabula may be larger than those of the African apes, but they are markedly smaller than those of anatomically modern humans; there remains a large “relative hip joint disparity” between modern humans and members of the genus *Australopithecus* (Corruccini and McHenry, 1978, p. 148). Some researchers have posited that the morphology of the *Au. afarensis* pelvis represents a compromise between terrestrial bipedalism and arboreal climbing (McHenry, 1986;

Stern and Susman, 1983; Susman et al., 1984). These authors have predicted that the earliest specimens of the species would look like a "generalized ape" with a distinctly un-humanlike mode of locomotion (Stern and Susman, 1983, p. 279). While affirming the un-humanlike nature of the *Au. afarensis* gait, Rak (1991) has described the horizontally broad AL 288-1 pelvis not as a compromise between chimpanzee-like and humanlike locomotion, but as a unique solution to the problem of bipedal locomotion with short hindlimbs.

There is evidence that the pelvis of *Au. africanus*—a possible daughter species of the earlier *Au. afarensis*—has more humanlike morphology (Haeusler, 2002). However, McHenry (1986) has highlighted similarities between the pelves of some *Au. africanus* and *Au. afarensis* pelvic specimens (e.g., STS 14 and AL 288), and other researchers have cited similarities between the postcrania of both *Australopithecus* and *Paranthropus* (Walker, 1973). According to Asfaw and colleagues (1999), *Australopithecus garhi* (~2.5 MYA) may be the first of its genus with humanlike postcrania. One *Au. garhi* specimen (BOU-VP 12/1) exhibits humanlike humerus-to-femur proportions—making it derived relative to *Au. afarensis* (Asfaw et al., 1999). However, based on the specimen's apelike forelimb-to-hindlimb proportions, it is likely that modern limb proportions did not evolve until after *Au. garhi*—probably with *H. erectus* (Asfaw et al., 1999).

Early Pleistocene hominins: *Au. sediba* and early members of the genus *Homo*

Dating to approximately 1.98 MYA, South African hominin *Au. sediba* famously displays a mosaic of primitive and derived features (Berger, 2013). In the hindlimb, for example, while tibial and femoral measurements are similar to *Au. africanus*, and the foot

skeleton is relatively primitive, several derived pelvic features are present (Berger et al., 2010). The pelves of MH1 and MH2, like specimens from the genus *Homo*, exhibit vertically oriented iliac blades with S-shaped curvature and medially displaced anterior superior iliac spines, along with robust regions of weight transfer in the iliac bodies (Kibii et al., 2011). Like members of the genus *Australopithecus*, however, they also exhibit elongated pubic rami, a wide bi-acetabular diameter, and small sacral and coxal joint surfaces (Kibii et al., 2011).

From the genus *Australopithecus* to early members of the genus *Homo*, the pelvis increases in size and the sacrum rotates anteriorly (McHenry and Coffing, 2000; Rak, 1991). The absolute and relative size of the hip joint increases dramatically (McHenry and Coffing, 2000). While no pelvic remains attributed to *H. habilis* (*sensu stricto*) have been recovered, a pelvic specimen attributed to *H. rudolfensis* (KNM-ER 3228; ~1.9 MYA) already exhibits a human-sized acetabulum (McHenry and Coffing, 2000), suggesting that the functional complexes of humanlike bipedalism were in place before the emergence of *H. erectus* (Rose, 1984). This implies that a significant locomotor change occurred early in the hominin lineage.

The pelvis of subadult *H. erectus* specimen KNM-WT 15000 (~1.6 MYA) is similar in morphology to KNM-ER 3228, exhibiting a considerable degree of *Australopithecus*-like iliac flare, but with human-sized hip joints (Brown et al., 1985). A large, humanlike acetabulum is also seen in *H. erectus* specimen OH 28 (~0.5 MYA; Leakey, 1971); this trait, along with the presence of a pronounced vertical iliac pillar and medial rotation of the ischium, suggest humanlike bipedal musculature (Day, 1971).

However, not all change within specimens attributed to the genus *Homo* is in the direction of humanlike morphology. The Early Pleistocene pelvis BSN 49/P27 (1.4-0.9

MYA), attributed by Simpson et al. (2008) to *H. erectus*, exhibits a mediolaterally broad birth canal similar to AL 288-1 and small acetabula and femoral heads within the size range for *Australopithecus* rather than *Homo* (Ruff, 2010). Due to this primitive pelvic anatomy, Ruff (2010) has suggested that BSN 49/P27 be attributed not to *H. erectus*, but to *Paranthropus boisei.*

Later Pleistocene hominins: *Homo neanderthalensis* and *Homo sapiens*

Fossilized Neandertal remains have been dated to as early as ~200,000 years ago (KYA) in Europe and as early as ~70 KYA in Western Asia (Klein, 2009). While Neandertals were undoubtedly obligate bipeds, the Neandertal pelvic—and particularly pubic—morphology is unique among hominins. The pubic rami of most Neandertal specimens are elongated and slender (Trinkaus, 1976). Some (e.g., Krapina 208) also exhibit superior-inferior flattening, and others (e.g., Shanidar 1) exhibit a distinct rim on the ventral aspect of the superior pubic ramus (Trinkaus, 1976). This suite of Neandertal pubic morphological traits does not seem to reflect sexual dimorphism, as many known Neandertal specimens are male. The “disproportionately long” Neandertal pubic ramus may have served to facilitate slight postural and locomotor differences (Rak and Arensburg, 1987, p. 228), or may merely reflect the uniquely broad upper bodies of the Neandertals (Greene and Sibley, 1986; Klein, 2009). Alternately, the elongated Neandertal pubic shape may have allowed the birth of large-headed, large-bodied babies—predicted characteristics for the offspring of short, muscular, heavily built Neandertal mothers (Rosenberg, 1988). Regardless, it now seems that human pelvic morphology was still experiencing significant changes long after the evolution of bipedalism (Trinkaus, 1984).

By approximately 200 KYA, an ancestral population of anatomically modern humans had evolved in Africa; this population likely had spread widely by around 50 KYA (Klein, 2009). At this point, the human neural, muscular, and skeletal systems had achieved modern proportions, enabling modern cultural and technological developments. Anatomically modern human pelves indisputably facilitate obligate bipedalism and are characterized by: broad, transversely ovoid inlets; short, broad, vertically oriented ilia; and marked gluteal muscle attachments. In these and other pelvic and femoral traits, modern humans can be considered even more derived than the Neandertals (Trinkaus, 2006).

Reversals in hominin pelvic/locomotor anatomy

The evolution of hominin pelvic and hindlimb anatomy does not represent unidirectional progress toward obligate bipedalism. In contrast with earlier studies asserting that *Au. africanus* and *Au. afarensis* have functionally similar locomotor anatomies (e.g., McHenry, 1986), some recent studies have alleged that *Au. africanus* has more apelike limb proportions than *Au. afarensis* (Green et al., 2007; Haeusler and McHenry, 2007). Assuming the former is a daughter species of the latter, this implies an evolutionary reversal. It may suggest that *Au. africanus* was more arboreal than *Au. afarensis*, and was thus subject to different selective pressures. Further complicating issues of phylogeny and selection, while most forelimb proportions for both *Au. afarensis* and *H. habilis* are humanlike, one specimen of *H. habilis* (KNM-ER 3735) has been shown to exhibit more apelike limb proportions than *Au. afarensis* specimen AL 288 (Haeusler and McHenry, 2007). This may imply that *H. habilis* is a descendant of the more postcranially apelike *Au. africanus*, rather than *Au. afarensis*. Regardless, these examples of reversals serve to underscore the nonlinear nature of evolutionary

change, and highlight problem of using limb proportions—traits under heavy selection pressures for locomotor adaptation—for inferring phylogeny (Green et al., 2007; Jungers and Cole, 1992).

Interpreting Hominin Pelvic Adaptations

Many animals (including chimpanzees, the closest primate relatives of the hominin lineage) can use bipedal locomotion opportunistically. Anatomically modern humans, however, are obligate bipeds, walking on two legs habitually. Between these two extremes, different members of the hominin lineage may have occupied different places on the spectrum of bipedalism. However, experts disagree on the extent to which the earliest hominins relied on terrestrial bipedalism vs. arboreal quadrupedalism or brachiation, whether early bipedal locomotion resembled modern human locomotion, and at what point in the hominin lineage obligate terrestrial bipedalism became the norm rather than the exception.

Facultative vs. obligate bipedalism

One school of thought holds that early members of the genus *Australopithecus* undertook a partly arboreal, partly terrestrial lifestyle, employing a transitional locomotor mode distinct from modern human bipedality (e.g., Stern and Susman, 1991). To scholars espousing this view, morphological forms reflect their biomechanical function: primitive-looking traits observed in fossil hominins must still indicate the fossil's way of life; otherwise, the environment would have selected against them (Susman and Stern, 1991). On the opposite side of the debate, another school of thought holds that the locomotion of early bipeds like *Au. afarensis* closely resembled human bipedality, and that the habitats of these habitual bipeds were purely terrestrial (e.g., Latimer and Lovejoy, 1990). Scholars espousing this view emphasize that not every morphological

trait has a functional, adaptive significance: while form is infinite, function is not (*sensu* Lovejoy, 1975). In the opinions of these anthropologists, seemingly arboreal morphologies may actually represent evolutionary holdovers, retained from arboreal ancestors via neutral selection. As Cartmill and Smith (2009, p. 176) have summarized, these differences in fossil interpretation center around the issue of phylogenetic inertia: "How much of the anatomy of an organism reflects its own way of life, and how much reflects the adaptations of its ancestors?"

Recent *Ar. ramidus* publications exemplify this debate. While some researchers have interpreted the shortened, broadened *Ar. ramidus* pelvis with its pronounced anterior inferior iliac spine as clear evidence of the primate's bipedalism (Lovejoy et al., 2009), other researchers have pointed to the fact that these traits occur in other early hominoids—including those that practice quadrupedal locomotion and those that are not members of the hominin lineage (Sarmiento, 2010). Much of the difficulty in identifying obligate bipedalism in the fossil record lies in the mosaic nature of hominin remains. The *Ar. ramidus* remains (ARA-VP-6/500), for example, combine both humanlike and apelike pelvic traits (Lovejoy et al., 2009). It is possible that the hominin's locomotion also incorporated elements of both bipedalism and arboreal quadrupedalism, or that it utilized a more primitive form of bipedalism than members of the genus *Australopithecus* (White et al., 2009).

Even in the *Australopithecus* fossils that postdate ARA-VP-6/500, the evidence for obligate bipedalism is unclear. Some research suggests that the evolution of any form of terrestrial bipedality by australopithecines must have precluded them from undertaking arboreal locomotion—that hominins became obligate bipeds as soon as

they left the trees (Latimer and Lovejoy, 1990; Lovejoy, 1988). Lovejoy (2005) has argued that the kinetics of the human hip were established early in our evolutionary history—perhaps as early as 3.5 million years ago, with the evolution of *Au. afarensis*.

Others have cited the thickened inferior cortical bone of *Au. afarensis* and modern human femoral necks as evidence of humanlike bipedality in both species (Ohman et al., 1997)—in spite of the fact that inferior thickening of the femoral neck has also been observed in multiple arboreal species (Demes et al., 2000; Stern and Susman, 1991). Indeed, the mosaic pelvic traits of *Au. africanus* and especially *Au. afarensis* seem to indicate a combination of terrestrial bipedalism and arboreal quadrupedalism—a locomotion that was distinctly un-humanlike, yet unlike any locomotion seen in extant hominoids (McHenry, 1986). In particular, the small hip joints of *Australopithecus* specimens seem un-humanlike, and these differences in hip morphology may have translated to differences in gait (Corruccini and McHenry, 1978). Additionally, the posterior orientation of the iliac blade of *Au. afarensis* specimen AL 288-1 indicates that pelvic balance was maintained through partially flexed rather than fully extended thighs; and the anterior horn of the AL 288-1 acetabular surface is small, an apelike trait (Stern and Susman, 1983). Based on the unique anatomical features of *Au. afarensis* specimens, Stern and Susman (1991) concluded that their locomotion was similarly unique; and Duncan and colleagues (1994) have agreed that *Au. afarensis* locomotion might have been intermediate between modern humans and African apes. Indeed, when Susman and Demes (1994) simulated the relative foot length of the AL 288-1 individual in a kinematic experiment, they found that angular excursions at the hip, knee, and ankle increased with increasing foot size, rendering

"Lucy's" gait distinctly un-humanlike. Stern and Susman (1983) have interpreted the *Au. afarensis* pelvic evidence as lacking some of the key traits necessary for modern human locomotion (e.g., hyperextension of the hip during terminal stance phase). This posited primitive bipedalism, termed "bent-hip, bent-knee" or "BHBK" (Carey and Crompton, 2005, p. 25), might have been a stable and relatively long-lived adaptation (McHenry, 1986). Indeed, if the small hip joints in many specimens of the genus *Australopithecus* are indicators of primitive gait, this "Lucy-like locomotor adaptation" may have persisted until the time of *H. erectus* (Jungers, 1988, p. 263). Alternately, humanlike locomotion may have evolved in later species of *Australopithecus.* Haeusler (2002) has cited the apelike pelvic muscle attachments in *Au. afarensis* specimen AL 288-1 as evidence for a bent-legged gait in this species, with more humanlike muscle attachments in later *Au. africanus* specimen STW 431 suggesting a more humanlike gait.

However, thermoregulatory data from studies on human subjects indicate that BHBK gait is extremely metabolically costly and thus unlikely to have been selected as a locomotor adaptation in early hominins (Carey and Crompton, 2005; Crompton et al., 1998). Based on predictive dynamic modeling using the AL 288-1 remains, some researchers have asserted that even early members of the genus *Australopithecus* likely walked with legs extended (Crompton et al., 1998). Analysis of an *Au. afarensis* fourth metatarsal reveals humanlike morphology that suggests obligate, terrestrial bipedalism (Ward et al., 2011). Some researchers have interpreted the adducted toe, apparent arches, and in-line striding pattern of the Laetoli footprints as evidence for a humanlike gait in early hominins (Day and Wickens, 1980; Leakey and Hay, 1979).

Tuttle (1985, p. 130) has even gone so far as to describe the foot structure and inferred gait for two of the Laetoli individuals as “indistinguishable” from that of *H. sapiens*. Stern and Susman (1983) however, have disagreed, citing features of the footprints that resemble chimpanzee gait, not that of modern humans. More recent kinematic data suggest that the pattern of the Laetoli footprints could have been formed by a biped with either a humanlike stride or a BHBK gait (Raichlen et al., 2008). The interpretation of the Laetoli footprints is further obscured by the fact that the substrates on which organisms walk (e.g., dry gravel, wet sand, mud) have the potential to obscure anatomical and biomechanical indicators to varying degrees (Morse et al., 2013).

Even if members of the genus *Australopithecus* walked with a BHBK gait, the dorsally projecting hamstring attachment site on the ischium of *Au. afarensis* pelvic specimens suggests that their posture was less crouched than chimpanzees’ (Pontzer et al., 2009). Further, while extended-leg locomotion is undoubtedly more efficient than BHBK, studies on the metabolic costs of bipedal locomotion in chimpanzees suggest that even the earliest, most primitive form of hominin bipedalism may have been more energetically economical than quadrupedalism (Pontzer et al., 2009). Finally, the pelvic and lower limb morphology of *Au. sediba* suggests that it was a habitual biped by 1.98 MYA (Berger et al., 2010), likely walking with a fully extended leg and inverted foot during swing phase (DeSilva et al., 2013). This raises the possibility that members of the genus *Australopithecus* were practicing a diversity of bipedal locomotor adaptations during the late Pliocene and early Pleistocene. Regardless, scholars of the evolution of bipedality do not dispute that Australopiths as early as *Au. afarensis* walked bipedally

with a valgus knee (Stern and Susman, 1991); they merely differ in their interpretation of the nature of that bipedality, and the substrate on which it was habitually undertaken.

Certainly, by the ascendancy of the genus *Homo*, humanlike bipedal gait was the hominin locomotor norm. Dated to approximately 1.9 MYA, the posited *H. rudolfensis* pelvic specimen (KNM-ER 3228; McHenry and Coffing, 2000) may represent one of the earliest habitually bipedal hominins (Rose, 1984). Studies on human subjects indicate that the longer the hindlimbs, the lower the locomotor cost (Steudel et al., 2007). Pontzer and colleagues (2010) have posited functionally modern locomotion for the long, humanlike Dmanisi hominin femur (D4167) dated to approximately 1.8 MYA. Certainly, the humanlike pelvic traits of OH 28 indicate that *H. erectus* was a habitual biped by around 0.5 MYA (Day, 1971). The obligate bipedal locomotion of members of the genus *Homo* would have resulted in lower metabolic costs than the primitive bipedalism practiced by *Australopithecus* individuals, regardless of whether the latter walked with bent or extended legs (Pontzer et al., 2009; 2010).

What triggered the evolution of bipedalism in early members of the hominin lineage and its subsequent refinement in the lineages of *Australopithecus* and *Homo*? Susman and colleagues (1984) used microfaunal, macrofaunal, and palynological data to reconstruct the environment in which Australopiths practiced their early, imperfect form of bipedality: the Pliocene Hadar environment was a mosaic of forest, woodland, and open habitats; Laetoli during the Pliocene, while likely drier than Hadar, still shows evidence of forest-dwelling species—most conspicuously, tree-dwelling monkeys. These authors portrayed increasingly bipedal hominins as ranging across a varied and changing landscape—relying on the resources and protection of trees, but increasingly

employing terrestrial bipedality as forested regions fluctuated in size—and supported it with behavioral data from extant primates (Susman et al., 1984). In this model, concurrent with increased hominin bipedality came an increase in the sizes of their bodies, brains, and capacity for creating material and social culture; it was only when they were "freed…from reliance on the trees" that they became human (Susman et al., 1984, p. 152).

Other researchers have suggested that the increased need to carry loads (e.g., altricial infants) prompted early hominins to use their forelimbs for transportation rather than locomotion; others have indicated that the efficiency of bipedal locomotion in and of itself conveyed a selective advantage to its practitioners (Watson et al., 2008). Lovejoy (1988) has posited that bipedality evolved in hominins concurrently with a modern human behavioral suite, including the practices of male-female monogamy, nuclear family groupings, and sex-specific food-gathering behaviors (i.e., early hominin males used their forelimbs to carry food items long distances to their monogamous female partners). Some researchers have proposed a link between cursorial walking/running and more advanced bipedalism in members of the genus *Homo*, potentially correlated with new foraging demands (e.g., meat acquisition) and the increased ranges that accompanied them (Pontzer et al., 2010). The large hip joints and long hindlimbs that characterize members of the genus *Homo* were likely one of the fundamental changes of later hominin evolution—possibly selected for traveling longer distances or repetitive hindlimb loading (Jungers, 1988).

The lack of consensus on the origin, degree, and nature of bipedalism in early hominins calls into question the importance of bipedalism to a fossil primate's place on

the hominin family tree. Is obligate bipedalism the most important indicator of hominin status? Is it a fundamentally human trait, or an arbitrary benchmark? What degree of bipedalism meets the mark? Does the primitive (likely facultative) bipedalism posited by White and colleagues (2009) for *Ar. ramidus* qualify the early primate for a spot on the hominin lineage? Does whether *Au. afarensis* specimen AL 288-1 walked with bent or extended knees change whether she is considered a human ancestor? The more fully researchers understand the mosaic nature of hominin evolution, the more likely it seems that a full package of humanlike traits was not evolved at once (*sensu* Susman et al., 1984), but rather that bipedalism was incorporated long before the encephalization and extensive use of culture that are considered hallmarks of humanity.

Obstetric constraints

Bipedal locomotion is not the only evolutionary constraint that has acted on the hominin pelvis. The bipedal pelvis also plays a role in posture, support of the viscera, and, perhaps most importantly, birth (Tague and Lovejoy, 1986). Thus, the human pelvis represents an evolutionary compromise among these factors. For example, human female pelvic shape, rather than representing bipedal optimization, "accommodates childbearing within the bipedal-walking adaptation" (Greene and Sibley, 1986, p. 518). The ability to birth relatively large-headed young is a defining hominin trait, particularly among later members of the lineage. However, the biomechanics of bipedalism have tended to constrain the breadth of the hominin pelvis, resulting in different obstetric and gestational adaptations over the course of hominin evolution. A brief discussion of hominin obstetrics is relevant to an understanding of human pelvic anatomy.

In the great apes and other non-hominin primates, the pelvis is supero-inferiorly elongated and broadest in the anterio-posterior dimension; the fetal head remains anterio-posteriorly oriented throughout the birth process (Rosenberg and Trevathan, 2002; Tague and Lovejoy, 1986). Over the course of hominin evolution, the stability required for bipedal locomotion led to the development of a shortened, mediolaterally broadened pelvic form. This mediolaterally broad pelvis characterizes many early members of the hominin lineage, including members of the genus *Australopithecus* (Claxton et al., 2016; Tague and Lovejoy, 1986), and this adaptation may have persisted even in early members of the genus *Homo* (Ruff, 1995). The mediolateral expansion of the bipedal pelvic shape led to a transverse rather than antero-posterior orientation for the fetal head during the birth process in Australopiths and early members of the genus *Homo* (Ruff, 1995; Tague and Lovejoy, 1986). Since mediolateral pelvic breadth is constrained by biomechanical factors, it is possible that this constrained cranial capacity in these earlier hominins until the evolution of rotational birth later in the lineage (Ruff, 1995).

The marked adult encephalization ultimately seen in later members of the genus *Homo* was enabled by two subsequent adaptations: enlarged birth canals; and the birth of neonates with heads that, while increasingly encephalized, yet comprised a smaller percentage of their eventual adult brain size (Rosenberg and Trevathan, 2002). This latter adaptation led to the *secondary arltriciality* seen in modern humans: the birth of relatively more helpless young compared with our closest primate relatives—even those with similar gestational lengths (Rosenberg and Trevathan, 2002). Secondary altriciality may have evolved as early as the time of *H. erectus* (Rosenberg, 1988) or as late as the

Mid-Pleistocene (Trinkaus, 1984), depending on differing interpretations of the fossil evidence from the genus *Homo*. In particular, the Neandertal pelvis, with its elongated pubis, has been interpreted either as evidence of the birth of large-headed, secondarily altricial neonates by correspondingly large-bodied Neandertal mothers (Rosenberg, 1988) or as evidence of longer gestation time in Neandertals (i.e., 11-12 months) and a lack of secondary altriciality (Trinkaus, 1984).

Ultimately, the increasing fetal encephalization of later members of the genus *Homo* exerted a strong selective pressure for a more spacious birth canal. Shoulder size may have also played a role (Trevathan and Rosenberg, 2000). Since the hominin pelvis was already broad in the mediolateral dimension (and constrained by bipedal locomotion from broadening further), this space increase evolved in the antero-posterior dimension (Tague and Lovejoy, 1986). The heads of fetal hominins had entered the birth canal in a transverse orientation for millions of years, at least since the evolution of the mediolaterally broad Australopith pelvis (Rosenberg, 1992). This later, genus-*Homo* era increase in antero-posterior pelvic inlet space led to modern human birth mechanics—including rotational birth (wherein the fetal head enters the birth canal in one orientation and exits in another, facing posteriorly) and the behavioral adaptation of assisted birth (Rosenberg and Trevathan, 2002). The current human condition involves a transversely broad (but relatively rounded) pelvic inlet through which the encephalized human fetus passes with head oriented transversely early in the birth process and antero-posteriorly later in the birth process (Rosenberg and Trevathan, 2002).

Thus, the evolution of modern human pelvic dimensions was not contemporaneous with the evolution of bipedalism, but rather was a later (Plio-

Pleistocene) phenomenon. Some characteristically "*Homo*-like" pelvic traits likely evolved due to locomotor rather than obstetric constraints, as indicated by recent evidence of the small endocranial volumes and pelvic inlet diameters of *Au. sediba,* co-occurring with *Homo*-like aspects of pelvic morphology (Kibii et al., 2011). However, it is likely that fully modern human pelvic dimensions evolved concurrently with the later evolution of encephalization (Tague and Lovejoy, 1986).

Putting it All Together: A Summary of Hominin Pelvic Evolution

Bipedalism in the hominin lineage dates back to approximately 6 MYA, though the exact form it took and the nature of the selective pressures behind its evolution remain disputed. Though early hominins likely incorporated varying degrees of arboreality into their locomotor strategies, they had already lost the long, blade-like pelvic design characteristic of quadrupeds, evolving shorter, broader pelves that enabled stability and balance during bipedal locomotion. Subsequent species became increasingly committed to obligate bipedalism, and further changes to pelvic design evolved concurrently with changes in birth mechanics and altriciality. Australopiths and their contemporaries evolved mediolaterally broad pelves through which a transversely oriented fetal head could pass; later, members of the genus *Homo* evolved a rounder, more spacious pelvic inlet through which a fetus entered transversely and exited antero-posteriorly. It was only relatively late in hominin evolutionary history that marked encephalization and secondary altriciality evolved.

Most relevant to the current research, the general evolutionary trend has been for hominin hip joints to become larger and more stable as species committed more fully to obligate bipedalism and evolved larger body sizes with *allometrically* scaling (disproportionately large) joints. As early as 1.9 MYA, *H. rudolfensis* and other early

members of the genus *Homo* displayed large, humanlike hip joints. Rather than suffering high levels of degeneration, these large hominin hip joints adapted during the transition from quadrupedal to bipedal locomotion to withstand the unique stresses of walking on two legs (the idea that modern human hip morphology minimizes OA is explored further below; see *Potential Factors Impacting Hip OA: Bipedalism*). Our commitment to bipedalism—and the large lower limb joints that have evolved along with it—truly represent hallmarks of the human condition.

Modern Human Hip Biomechanics

The bipedal hip represents a compromise between mobility and stability. It is "the pivot upon which the human body is balanced in gait," and its stability depends upon the integrity of the configuration between the acetabulum and the femoral head (Radin, 1980, p. 28). Human gait during walking consists of a repeated cycle of heel-strike through toe-off, alternating between the left and right feet. The phase of the gait cycle between heel-strike and toe-off for one foot describes the period of time during which the foot is in contact with the ground and is referred to as the stance phase for that foot. The phase between toe-off and the subsequent heel-strike consists of the period of time during which the foot is in the air and is referred to as the swing phase. During the period of transition from the initial contact of one foot to the swing phase of the opposite foot, there is also a period of double support. Stance phase comprises approximately 60% of the gait cycle, and swing comprises approximately 40%. Peak hip joint forces are incurred during stance phase, when a single lower limb bears the brunt of the body mass.

While referred to as a ball-and-socket, the femoral head and acetabulum do not fit with the congruence expected of a sphere nested within a cup (Afoke et al., 1987).

Rather, their articular surfaces are incongruous in an unloaded state (Greenwald and O'Connor, 1971), leading to irregularities in force patterns during loading (Rydell, 1966).

Areas of Loading in the Human Hip

In a study of 51 cadaveric hips loaded in different positions under varying loads, Greenwald and O'Connor (1971) identified portions of acetabular and femoral head articular cartilage that do not come into habitual contact with each other, as well as portions that make contact only under heavy loading. Surprisingly, they found that the dome of the acetabulum—frequently referred to as the weight-bearing portion (e.g., Chuckpaiwong et al., 2009)—comes out of contact with the femoral head during light loading scenarios like the swing phase of the gait cycle (Greenwald and O'Connor, 1971), during which hip joint forces have been measured at only 10-40% of body weight (Bergmann et al., 1993).

Experimental data from instrumental femoral head prostheses indicate that local pressures in the hip joint are high and non-uniform and that the articular cartilage of the acetabulum and femoral head does not distribute this pressure uniformly (Hodge et al., 1986). Likewise, in an experimental setting in which five healthy hips were loaded in different positions with different loads, pressure-sensitive film applied to the femoral head yielded tortuous, irregular pressure print signatures (Afoke et al., 1987). Afoke and colleagues (1987) identified a region of high pressure on the anterosuperior surface of the femoral head cartilage in the five cadaveric hips examined in their loading experiment. They drew a connection between the high pressure sustained by this cartilage and the evidence of OA-related cartilage loss that is frequently seen in this region (Afoke et al., 1987). Bergmann and colleagues (1993) also posited that cartilage destruction and femoral head remodeling may be related to forces acting consistently

on a small area of pressure transfer, noting that there is little variation in force direction (in the frontal plane) during peak hip loading. The area of anterosuperior contact cited by these researchers corresponds with one of the regions identified by Greenwald and O'Connor (1971) as an area of habitual contact (i.e., where the femoral head and acetabular articular cartilage make contact even under the smallest loads), but these researchers have instead posited that the majority of cartilage degeneration occurs on the periphery of these areas, where contact is load-dependent, or infrequent. In their two-dimensional finite element analysis, Vasu and colleagues (1982) also identified the superior acetabulum as a region of high compressive stress, with tensile and bending stresses distributed in peripheral regions of the subchondral bone, and throughout the medial portions of the os coxa. Carter (1987) viewed these compressive stresses on the acetabular roof and corresponding superior surface of the femoral head as beneficial for cartilage health, in contrast with the tensile strains sustained by the peripheral areas of the acetabulum and femoral head, regions where cartilage degeneration and osteoarthritic changes have been observed.

The pelvis receives body weight transmitted from the sacrum at the auricular articulations and via the lumbosacral ligaments (Pal, 1989). The primary role of the pelvis in its the interaction with the femoral head (at the acetabulum) is to support this body weight and transfer the load to the lower extremities; as such, it must withstand forces several times body weight (Dalstra and Huiskes, 1995). To do so, the pelvis has evolved a 'sandwich' structure in which a core of trabecular bone is surrounded by a thin layer of cortical shell (Dalstra and Huiskes, 1995; Jacob et al., 1976; Vasu et al., 1982). In essence, the human pelvis represents a high-strength, low-weight solution to

a difficult problem of engineering (Dalstra and Huiskes, 1995). In both the acetabulum and the femur, the trabecular bone underlying the cortical shell plays a supporting role, allowing deformation without structural damage (Radin, 1980).

An early loading experiment on a pelvic model with an epoxy resin 'cortical shell' filled with epoxy foam 'trabecular bone' (materials with Young's moduli ratios mirroring the ratio of actual cortical and trabecular bone) revealed that the cortical materials were highly stressed in both tension and compression, as stresses traveled from the subchondral bone of the acetabulum to the acetabular rim, then across the cortical shell (Jacob et al., 1976). Later, Dalstra and Huiskes (1995) utilized a three-dimensional finite element model of the human pelvis (incorporating a bilateral pelvic mesh, values and directions of hip joint forces and 21 muscular forces during eight phases of the gait cycle) in order to investigate its behavior under normal physiological loading conditions. They also found that the majority of the load passing through the pelvis is transferred through the cortical shell, in which stresses are 50 times higher than the stresses sustained in the underlying trabecular bone, which is, alternately, subjected to higher levels of strain (Dalstra and Huiskes, 1995). According to their model, the highest stresses transmitted from the femoral head to the acetabulum are sustained by the superior acetabular wall, from which they are transferred either to the sacro-iliac or pubic symphyseal joints (Dalstra and Huiskes, 1995).

Previous research had suggested that not only hip joint force but also muscular forces play a large contributing role in hip stress (Krebs et al., 1988). While the Dalstra and Huiskes (1995) model illuminated the stabilizing role played by the pelvic musculature, it also helped them to identify that hip joint force, not muscular force, is the

most important aspect of load transfer across the pelvis—playing a particularly important role in the transfer of load from the femoral head to the anterosuperior acetabulum. Their conclusions that the acetabulum is loaded in a non-uniform fashion, and that its anterosuperior edge is the major locus of load transfer from the femoral head, were supported by observations of dense trabeculae and thick cortical shell in this region (Dalstra and Huiskes, 1995), as well as by the experimental pressure print data of Afoke and colleagues (1987). Chuckpaiwong and colleagues (2009) also cited this portion of the acetabulum as essential to the stability of the joint. Other researchers have identified the posterosuperior region of the acetabulum as the region of highest acetabular contact pressure (Kim et al., 2006; Krebs et al., 1988). It is possible that this conflicting evidence is related to the areas of habitual/loading-dependent contact identified by Greenwald and O'Connor (1971): according to their research, the anterior- and posterior-most portions of the acetabular lunate surface are habitually in contact with the femoral head, but these areas of habitual contact merge into one continuous area of contact during high loading scenarios. Depending on the degree of loading of their experimental hips, different researchers may have identified different portions of the habitual/loading-dependent contact zones as the areas of highest acetabular contact pressure.

Human Hip Joint Forces

Until the advent of advanced instrumented hip prostheses, *in vivo* measurements of hip joint loads were rare, and most studies inferred or indirectly approximated loads by modeling body segments as series of rigid links and solving the inverse dynamics problem using force-plate data (Crowninshield et al., 1978). These hip joint force calculations were frequently higher than *in vivo* measurements (Bergmann et al., 1993).

For example, one study calculated that hip joint loads during stair climbing would exceed seven times body weight (Crowninshield et al., 1978)—a magnitude that subsequent *in vivo* measurements have not borne out (Bergmann et al., 1993; 2001; Rydell, 1966).

In a seminal *in vivo* study, Rydell (1966) measured peak hip joint contact forces at 182% of body weight (during walking) in one patient, and at 433% of body weight (during running) in a second. The discrepancy between these peak force data may be due to the poor fit of the former patient's prosthetic femoral head (Bergmann et al., 1993). In a subsequent study of hip joint forces measured in two patients via telemetering femoral head implants, Bergmann and colleagues (1993) reported that hip joint forces increased with speed from approximately 280% of body weight during slow walking to a maximum during fast walking/jogging of 467% in one patient, 584% in the other, corroborating the peak force data for the second patient in Rydell's (1966) study. The highest hip joint forces reported for the patients in the study of Bergmann and colleagues (1993) were incurred during stumbling: 720% and 870% of body weight, respectively—a maximum that was not approximated during any other physical activity undertaken by the study patients (including running). In that study (Bergmann et al., 1993), the hip joint force magnitudes in a patient with disturbed muscle function proved exceptionally high, likely due to her gait anomalies. Indeed, a subsequent study of four patients with telemetered femoral prostheses revealed lower average peak forces during normal walking (on average, 238% of body weight); in this study, even stair climbing resulted in the comparatively low peak contact forces of (on average) 251% of body weight ascending, and 260% descending (although torque is on average 23%

higher during stair climbing than during normal walking; Bergmann et al., 2001). Further, when compared with walking, jogging, and stair climbing, all other tested routine activities (e.g., knee-bending, sitting down, and standing up) resulted in small hip joint loading forces (Bergmann et al., 2001). Still, comparative studies of humans and quadrupedal animals indicate that the hip joint force magnitudes experienced by humans during normal walking are approximately three times higher than those experienced by sheep (Bergmann et al., 1999). The above data confirm estimates quoted by Huang and colleagues (2000) that human hip joint forces approximate two-to-four times body weight. This is lower than knee joint forces, which are estimated at three-to-six times body weight (Huang et al., 2000).

Evidence suggests that wearing shoes has little effect on hip joint loading (Bergmann et al., 1993; 1995). In an individual with bilateral telemetered femoral head implants, the lowest hip joint loads were incurred walking barefoot, but wearing shoes caused only a slight increase in peak forces (Bergmann et al., 1995). Neither did the characteristics of the shoes worn (e.g., stiffness) cause any detrimental effects or confer any significant advantage (Bergmann et al., 1995). While Bergmann and colleagues (1993) posited that impact forces from the floor must be dampened by the time they reach the hip joint, their later research indicated that jogging with soft heel strikes caused an appreciable (~10%) decrease in maximum hip joint forces compared with normal jogging (Bergmann et al., 1995).

Major Factors Impacting Lower Limb Mechanics: OA, Aging, and Obesity

In spite of the biomechanical constraints placed upon them by their postural and locomotor roles, the acetabulum and femoral head exhibit marked inter-individual variations in orientation and morphology, leading to wide ranges and patterns of

variation in hip joint motion (Dujardin et al., 1997). For example, in a study of 55 female and male individuals with normal body weights and no known locomotion disorders, hip rotation around a vertical axis varied between 3° and over 30° (Dujardin et al., 1997). The inter-cartilage space between the articular surfaces of the femoral head and acetabular lunate surface varies in size and location and changes shape with different joint positions (Afoke et al., 1987). Moreover, the ball and socket components of the hip joint themselves change shape within an individual over the course of life, with the femoral head maintaining its slightly aspherical shape, but the acetabulum becoming more regular and spherical with age (Greenwald and O'Connor, 1971). When developmental abnormalities are considered, variations in hip morphology are even more extreme, with hereditary dysplasias (Iglič et al., 1993), metabolic disorders (Bálint and Szebenyi, 2000), and subtler and less-frequently diagnosed femoro-acetabular impingement scenarios contributing to changes in hip mechanics, and ultimately cartilage and bone damage (Ganz et al., 2008; Kim et al., 2006). However, three major factors impacting human gait are of particular relevance to the current research: OA, aging, and obesity.

Knee and hip OA. Age-related gait-limiting factors like OA of the hip—and particularly of the knee—have major repercussions for lower limb mechanics. In their cross-sectional study of the biomechanical changes associated with asymptomatic (*n*=60), moderate (*n*=60), and severe knee OA (*n*=61), Astephen and colleagues (2007) identified multiple kinetic and kinematic differences (at the hip, knee, and ankle) between asymptomatic individuals and moderate knee OA patients and between moderate and severe knee OA patients. Stance phase knee flexion angles, early

stance knee extension moments, peak stance phase hip internal rotation moments, and peak ankle dorsiflexion moments decreased with the progression of the disease, along with incremental decreases between the severity groups in speed, stance percentage, stride time, and stance time (Astephen et al., 2007). In addition, both moderate and severe OA patients experienced increased mid-stance knee adduction moments, decreased peak knee flexion moments, decreased peak hip adduction moments, and decreased peak hip extension moments compared with asymptomatic individuals (Astephen et al., 2007). As summarized by Arokoski and colleagues (2006), studies have shown that static balance and proprioreception are worse in patients with knee OA than in healthy individuals. Knee OA-related gait adaptations may be more severe in females than in males: in a study of 42 healthy males and females and 39 male and female OA patients, females with moderate knee OA exhibited biomechanical changes not seen in males with moderate knee OA, including less torque at the knee and ankle, and less range of motion at the knee (McKean et al., 2007).

Unlike patients with knee OA, patients with hip OA may not experience a reduction in postural control—at least, not if they are male. In a study comparing 27 males with hip OA to a control group of 30 healthy, age-matched males, Arokoski and colleagues (2006) found no difference between the groups in sensory organization test results (assessing ability to use visual, vestibular, and somatosensory inputs for balance maintenance) and no difference in center point of force velocity during one- and two-footed standing. In contrast, even normal walking can lead to higher hip joint stress in elderly females—contributing to a dangerous loading situation for women predisposed

to hip OA and providing support for the idea that increased female joint contact stress correlates with high frequencies of hip OA in females (Boyer et al., 2008).

In summary, lower-limb OA adversely affects bipedal gait (particularly in females), though adverse effects of knee OA are more significant than hip. Hip OA is discussed further in the following section of this chapter (*Pathological Conditions of the Human Hip: OA*), and an in-depth discussion of OA in general (including its etiology and interpretations) is provided in the ensuing Chapter 4.

Aging. Age-related changes in strength, range-of-motion, and gait mechanics are also common, even in individuals not afflicted with OA (Boyer et al., 2008; Hernández et al., 2009; Lindle et al., 1997; Nonaka et al., 2002). Aging detrimentally affects the human musculoskeletal system. Muscle mass, muscle strength, and bone density decrease with age. Older muscles take a longer time to produce a smaller amount of force than younger muscles do, and with age, joints become increasingly stiff (Lindle et al., 1997). As DeVita and Hortobagyi (2000) summarize, researchers have long noted the decreased joint torques and lower extremity powers of the elderly—and these changes amount to more than a mere age-related speed decrease. The elderly tend to adapt their gait patterns to reduce mediolateral center of mass accelerations (Hernández et al., 2009). They may experience progressive decreases in range-of-motion in the individual joints of the lower limb, as well as decreases in their interactive ranges-of-motion, due to musculoskeletal degenerative changes; in particular, lumbar spinal degeneration can affect hip mobility (Nonaka et al., 2002).

Moreover, with age comes a shift in motor performance and a complete redistribution of joint torques and powers (Boyer et al., 2008) that creates an altered

motor pattern favoring greater power in different joints than those used by younger adults (DeVita and Hortobagyi, 2000). In a study on lower limb joint powers and kinematics during gait, healthy elderly adults (*n*=12) exhibited significantly different joint angular kinematics, joint torques, and joint powers compared to healthy young adults (*n*=14)—specifically, the elderly produced more work with their hip extensors and less with their knee extensors and plantar flexors than the young adults (DeVita and Hortobagyi, 2000). This finding was strengthened by the work of Savelberg and colleagues (2007), who found that both inactive (*n*=10) and active, physically fit elderly males (*n*=10) exhibited this age-related redistribution of joint torques from ankle flexors to hip extensors, compared with inactive (*n*=10) and active (*n*=10) young males, whose plantar flexors accomplished six times the work of their hip extensors. Riley and colleagues (2001) also found that elderly individuals (*n*=14) exhibited significantly reduced ankle joint power compared with young adults (*n*=14), as well as kinematic changes (e.g., reduced maximum hip extension) that limited their gait speed. The finding that reduced plantar flexor support is a key factor limiting gait speed in the elderly was confirmed by Goldberg and Neptune (2007), whose forward dynamics simulation model enabled them to alter musculoskeletal parameters and observe their effects on model movement, isolating specific compensatory mechanisms in the gait of the elderly.

Sex-correlated differences have been identified in the hip joint moments of healthy older males (*n*=21) vs. females (*n*=21)—with older female gait characterized by greater external hip adduction and internal rotation moments and significantly higher cadence, regardless of imposed or self-selected speed (Boyer et al., 2008). The finding

of greater hip adduction angles (and therefore narrower step width relative to pelvic width) in elderly female gait mirrors similar findings in young adult females, and, coupled with the higher cadence of elderly female gait, contributes to greater hip joint moments per unit weight and height for females (Boyer et al., 2008). Thus, while the gait adaptations of elderly females resemble aspects of younger female gait, age exacerbates an already-dangerous loading situation in the hip of females, for whom even normal walking produces elevated hip joint contact stress (Boyer et al., 2008).

The underlying mechanisms behind gait adaptations in older adults are not fully understood. Because of muscle redundancy, "various neuromotor strategies may exist to compensate for decreased muscle strength and joint stiffness" (Goldberg and Neptune, 2007, p. 361). Further, because of dynamic coupling, identifying which muscles are taking compensatory action can be both difficult and counterintuitive. Regardless, it is now clear that even healthy aging individuals (and particularly, aging females) may experience adverse gait modifications.

Obesity. In the research summarized above (see *Knee and hip OA*), Astephen and colleagues (2007) identified significant biomechanical gait changes associated with knee OA. However, these researchers have warned that some aspects of the observed gait alterations may in fact be attributable to obesity (Astephen et al., 2007). In their study of healthy young adults (10 obese; 10 of normal body mass), Browning and Kram (2007) tackled the conundrum surrounding obesity, OA, and exercise: obesity is the main modifiable risk factor for knee OA, but the exercise that can help people to lose weight may also play a role in the biomechanical loading that causes OA. These researchers found that obese young adults had greater knee-joint loads than young

adults of normal body mass: the obese individuals experienced significantly greater absolute ground reaction forces, along with larger step width, and greater sagittal-plane net muscle moments (and joint loads) at the hip, knee, and ankle (Browning and Kram, 2007). This study emphasized absolute rather than normalized GRF, to emphasize actual loads on joints (which have been shown not to scale with body mass on an individual level; Lieberman et al., 2001; Ruff et al., 1991). In both obese individuals and those of normal body mass, however, ground reaction forces decreased significantly at slower walking speeds, leading Browning and Kram (2007) to suggest slow-speed walking as a mechanically safe exercise alternative for the obese.

The forces acting across the hip joint during walking and jogging are several times higher than an individual's body weight, and the loads vary with weight, and speed of walking (Bergmann et al., 1993). Joints compromised by OA can have reduced capacity to dissipate these loads, increasing joint stress. It is unsurprising, then, that obesity impacts the biomechanics of the hip and exacerbates the effects of both age and OA. As discussed in the following section, aging, obesity, and normal and pathological patterns of bipedal gait interact with myriad other factors to produce hip OA, the joint pathology most relevant to anthropological studies of age-related change in the acetabulum.

Pathological Conditions of the Human Hip: OA

Probably the most common pathological condition of the hip, OA is a major cause of disability in modern populations. In 1998, symptomatic hip OA was estimated to occur in approximately 4% of adults in the United States (Lawrence et al., 1998). In the United States, knee and hip OA account for more lower limb disabilities in elderly individuals than any other disease (Felson and Zhang, 1998). However, hip OA is not

only a disease of the elderly. Multiple factors contribute to a modern human individual's propensity to develop hip OA, including (but not limited to) age, obesity, degree of mechanical loading/joint stress, activity patterns, anatomy, heredity, and any of a variety of congenital, developmental, and post-traumatic abnormalities. Researchers have recognized a genetic component to OA in all joints of the body (Couchman, 2009), and the occurrence of OA in individuals younger than 50 years of age may indicate a genetic predisposition to the disease (Bálint and Szebenyi, 2000).

Researchers are only beginning to understand the intricacies of degenerative changes in the hip. Over the course of its evolutionary history, has the bipedal hip become more or less susceptible to hip OA? How do factors like aging and obesity affect degenerative change in the human hip? The evolutionary contexts within which bipedalism, aging, and obesity affect the development of hip OA are considered in the following sections (etiology, pathogenesis, and interpretations of OA are discussed in detail in Chapter 4).

Potential Factors Impacting Hip OA: Bipedalism

Intuitively, one might posit that the change from quadrupedalism to bipedalism in the hominin lineage led to higher rates of hindlimb OA. Since "large body size and locomotor patterns are implicated in the distribution and frequency of degenerative joint disease in mammals", and hominins combine large body size with unique bipedal locomotion, patterning of joint degeneration in the hominin lineage may correlate with locomotor adaptations (Cook et al., 1983, p. 96). Perhaps the bipedal loading of the human hindlimb predisposes our hindlimb joints to develop OA. However, rather than indicating a simple locomotion-to-OA correlation (i.e., with bipeds exhibiting high levels of OA in the hindlimb), there is evidence that the large hindlimb joints of large-bodied

hominins are adapted to reduce the amount of biomechanical loading. Research indicates that acetabula increase in size progressively from quadrupeds to early hominins (e.g., *Au. afarensis*) to anatomically modern humans (Jungers, 1988; see *Evolution of the Bipedal Hip*, above). The large hip joints of modern humans represent a derived condition; in essence, our large hindlimbs (and, particularly, our large hindlimb joints) make us unique (Jungers, 1988). According to Jungers (1988), they also act as a preventative for joint degeneration. Large joints, in large-bodied species, may exist to decrease the relationship between load and area such that damage to joint cartilage and subchondral bone is minimized (Radin, 1982). Indeed, data from human samples with developmental hip dysplasia indicate that the distribution of repeated, high-magnitude stresses over the small surface area of dysplastic joints contributes to the development of OA (Iglič et al., 1993).

Limited fossil hominin data support the idea that bipedal hindlimb joints were selected to minimize the joint stresses that cause OA. While joint degeneration has been observed in several hominin species, many instances occur in the vertebrae rather than the hind limb. *Australopithecus afarensis* specimen AL 288-1 exhibits localized vertebral degenerative pathologies; interestingly, while their pattern does not match typical OA patterning in extant primate species, localized vertebral lesions similar to those seen in AL 288-1 have been linked to heavy lifting/loading in modern human populations (Cook et al., 1983). Due to the similarities between the AL 288-1 pathologies and those of modern humans with extreme upper body strength, Cook and colleagues (1983) suggest that climbing and acrobatics may have played a major role in *Australopithecus* locomotion. However, caution must be used when interpreting direct

correlations between locomotor activity and OA in the fossil record. As discussed in Chapter 4, overt activity-based etiologies for OA have been largely rejected in recent years. In fact, physical activity is often viewed as beneficial for joint health. Studies on primates and other animals suggest that captive animals exhibit greater incidence of joint disease than wild animals—possibly linked to the *in*activity of the captive individuals (DeRousseau, 1985; Reed et al., 1993; Rothschild and Woods, 1992).

Neandertal remains also exhibit vertebral degeneration—in addition to acetabular and other appendicular joint pathology. The La Chapelle-aux-Saints 1 partial skeleton exhibits pathologies of the vertebrae (cervical and thoracic degeneration, osteophytes, and depressions consistent with those caused by Schmorl's nodules), forelimb (articular porosity and pitting), and hindlimb (OA of the left acetabulum and one interphalangeal joint; Trinkaus, 1985). Due to the extreme degeneration of the left acetabulum (e.g., exostosis formation, roughening, subchondral degeneration, eburnation, porosity, and possible abscess), Trinkaus (1985) has speculated that this pathological condition has a traumatic origin. It is interesting to note that Trinkaus has attributed the only remarkable hindlimb OA seen in La Chapelle-aux-Saints 1 to trauma—not to physical activity, loading, or the stresses of bipedal locomotion. Unilateral joint degeneration in the Shanidar Neandertals may also be associated with trauma, "particularly in the lower limb where normal biomechanical stress, and hence rates of degeneration, should be relatively symmetrical" (Trinkaus and Zimmerman, 1982). While mild vertebral degeneration and upper limb degeneration in two of the Shanidar individuals appear normal, the advanced, asymmetrical degeneration of several joints in Shanidar

individuals 1 and 3 appears to be the result of traumatic injuries (Trinkaus and Zimmerman, 1982).

Meanwhile, although wild populations of non-human apes (e.g., *Gorilla gorilla*, *Gorilla beringei*, *Pan paniscus*, *Pan troglodytes troglodytes*, and *Pan troglodytes schweinfurthii*) consistently exhibit higher frequencies of erosive arthritis and spondyloarthropathy than modern human populations (Lovell, 1990; Nunn et al., 2006; Rothschild and Rühli, 2005a,b; Rothschild and Woods, 1991), they exhibit comparatively low frequencies of OA (Jurmain, 1989; Rothschild and Rühli, 2005a,b). For example, in a study of several large samples of great apes (including chimpanzees, gorillas, and bonobos; *n*=167), although patterns and frequencies of appendicular OA vary, frequencies of vertebral degeneration are always statistically significantly lower than in archaeological and modern human samples (*n*=523), where spinal OA is both uniform and prevalent (Jurmain, 2000). Indeed, humans consistently evince more spinal OA than other primates (Brown et al., 2008); and human spinal degeneration tends to occur between the weight-bearing vertebral centra, while spinal OA in great apes tends to occur between the zygapophyseal articulations (Cook et al., 1983). In an experimental biomechanical study of the dissected spines of 22 human individuals with OA, Brown and colleagues (2008) found that apophyseal joint load-bearing correlated positively and significantly with scores for bone and cartilage degeneration, particularly when load-bearing exceeded 50% of the spine's total compressive force. They concluded that the degenerative changes of the human spine are directly related to the high levels of compressive load bearing caused by bipedal posture (Brown et al., 2008). Jurmain and Kilgore (1995) observed that while locations and frequencies of peripheral

joint OA differ widely among human populations, frequencies of spinal OA are consistent (and consistently higher than frequencies in non-human primates), leading them to posit a common functional etiology. This suggests that differences in biomechanical loading in quadrupedal primates and bipedal humans may entail different patterning and severity of vertebral OA. Bipedal locomotion increases torsional loading of the spine and exerts more compressive stress on the bipedal vertebral column than is sustained by the vertebrae of quadrupeds (Jurmain, 2000). It is possible that as hominins evolved from quadrupeds to bipeds, "biomechanic stresses, especially of the lower thoracic and lumbar regions, were significantly altered" and "as a consequence, the pattern and severity of vertebral lesions also…changed" (Jurmain, 1989, p. 235). Perhaps in bipeds, the spine takes the brunt of the biomechanical load. The important role of the spine in supporting the bipedal torso, associated with increased vertebral OA in bipedal primates, may explain why comparative mammal studies do not find evidence for a link between joint degeneration and biomechanical function in mammalian limbs; as Fox (1939, p. 118) stated, "it does not appear that these functions [i.e., locomotion]" have "any relationship to arthritis of the legs".

Over the course of its evolution, the hominin pelvis has changed from the chimpanzee's vertically long, horizontally narrow prototype to a vertically short, horizontally broad structure (see *Evolution of the Bipedal Hip*, above). As hominins became first facultative and later obligate bipeds, the horizontal orientation of the pelvis has become vertical, buttressed with gluteal musculature for stability. The small acetabula and femoral heads of chimpanzees and the earliest hominins have become the large, derived hip joints of the striding bipeds. The structures of the pelvis play an

integral part in bipedal locomotion as practiced by modern humans. Some of them may have evolved to minimize degeneration of the bipedal hip, with other skeletal structures (i.e., the spine) bearing the brunt of bipedalism's mechanical load.

Potential Factors Impacting Hip OA: Aging

The evolutionary pressure of bipedal locomotion is only one of many factors influencing bone changes in the human hindlimb. Age is a major systemic risk factor for the development of hip OA. In humans and other primates, degenerative changes of the hip are associated with increasing age (DeRousseau, 1985; Jurmain, 1977). Evidence of OA in larger-bodied primates may be the result of their lifespans—which tend to be longer for larger-bodied animals (Kappeler, 1996). In their analyses of trauma on Neandertal remains, Trinkaus and Zimmerman (1982; Trinkaus, 1984) have noted that barring trauma, hindlimb joint degeneration in bipedal hominins is regular, symmetrical, and age-progressive.

This correlation between aging and OA may hold particularly true in the hip. Recent forensic studies confirm that joint degeneration and osteoarthritic activity in the diarthrodial acetabulum progress in a fashion similar to age-related changes in the more stable amphiarthrodial pubic symphysis and iliac auricular surface (see Chapter 2). Additionally, bioarchaeological research suggests that the hip is less affected by biomechanical stress than more distal joints like the knee and ankle; "the hip, least affected by functional factors, is correspondingly more under the influence of systemic ones"—such as age (Jurmain, 1977, p. 364). These preliminary results bode well for the joint's potential as an age indicator, though factors such as trauma and developmental abnormalities must be considered. As exemplified by Trinkaus' (1985) Neandertal study, primary hip OA follows "regular patterns of bone formation," while

secondary OA caused by injury, trauma, or congenital pathologies can mimic the appearance of age-related changes, causing a joint to appear older than it truly is (Reed et al., 1993, p. 11). Age-related, sex-specific, and anatomical inter-individual variations in gait and hip joint motion also contribute to differential levels of joint degeneration (Dujardin et al., 1997; McKean et al., 2007).

Finally, and particularly in the context of the modern obesity epidemic, obesity must be considered as a major factor contributing to hip OA. Thus, though advancing age is one of the most important contributors to acetabular degeneration (Jurmain, 1980), obesity may be the main modifiable risk factor (Browning and Kram, 2007).

Potential Factors Impacting Hip OA: Obesity

Experimental evidence indicates that among mammals, joint forces are proportional to body mass$^{2/3}$ (Alexander, 1980). Joint stresses are defined as joint force/unit area (Ruff, 1988); larger articular surfaces more effectively dissipate the load from the forces acting on a joint, resulting in lower joint stresses. As summarized by Ruff (1988), research indicates that joint stresses in humans are of the same order of magnitude in all of the joints of the body—from the weight-bearing joints of the lower limb to the articulations of the metacarpals and phalanges. Moreover, in many organisms, joint stresses are reduced (or maintained at similar magnitudes) by the positive allometric scaling of certain articular surfaces: particularly, the ones involved in locomotion (e.g., the disproportionately large humeral heads of the gibbon, the disproportionately large femoral heads of humans; Jungers, 1988; 1991; Ruff, 1988). This suggests a functional correlation between joint size and locomotor adaptation. In a study of two species of brachiating primates (*Hylobates lar* and *Ateles geoffroyi*), Swartz (1989) also found that articular surfaces scale with positive allometry; she posited that

this reflects the necessity for larger-bodied species to evolve relatively larger weight-bearing joints (although Godfrey et al. [1991] subsequently attributed this positive allometry to functional, rather than size, similarities among animals adopting similar locomotor modes).

There is evidence that articular surface areas evolve in response to biomechanical pressures at the species level and are insensitive to intra-species inter-individual variation (e.g., in body mass). The research of Lieberman and colleagues (2001) has indicated that although articular surface areas are determined by mechanical (i.e., locomotor) constraints at the species level, individual variation in loading does not cause significant articular surface remodeling during life. Likewise, in a radiographic study of 80 black and white U.S. females and males with documented current and previous (ca. 18 years of age) body weight, Ruff and colleagues (1991) found that while femoral head dimensions correlated with body weight at 18 years, femoral shaft dimensions correlated with current body weight. Where the compact cortical bone of diaphyses remodels with changes in body mass, reflecting individual loading conditions during life, the predominantly trabecular bone of the articular surfaces remodels less readily, rendering the more conservative articular surfaces an appropriate proxy for body size at the species level (Ruff et al., 1991). Similar results were reported by Harrington and Wescott (2015), with a modern U.S. skeletal sample of white U.S. females and males exhibiting no significant differences in femoral head diameter between obese and normal-weight individuals, despite the fact that both femoral and tibial shaft diameters were statistically significantly larger in obese individuals.

Early studies of OA posited a correlation between large-bodied animals and OA. For example, Fox (1939) noted that in his study of 1,749 wild and captive mammals, small mammals such as rodents rarely exhibited OA. However, more recent research indicates that the size and shape of a species' joints scale allometrically with that species' body size (e.g., Jungers, 1988; Radin, 1982). The evolution of joint sizes and shapes is influenced by a species' habitual posture and locomotor adaptation (Jungers, 1988). Large joints, in large-bodied species, likely evolved to decrease the relationship between load and area such that damage to joint cartilage and subchondral bone is minimized (Radin, 1982). This assertion is supported by the extremely low frequencies of OA observed in the large-bodied *Gorilla gorilla* (8%; *n*=99) and *Gorilla beringei* (3%; *n*=38; Rothschild and Rühli, 2005a), as well as in *Pan paniscus* (3%; *n*=34), *Pan troglodytes troglodytes* (8%; *n*=79), and *Pan troglodytes schweinfurthii* (4%; *n*=26; Rothschild and Rühli, 2005b). Further, Jurmain (2000) has noted that larger-bodied gorillas exhibit only slightly more OA than smaller-bodied chimpanzees, and that individuals of the smallest-bodied African ape (*Pan paniscus*) exhibit the most OA. However, frequencies of all three species are extremely low, even in individuals of advanced age (Jurmain, 2000).

In fact, some of the only evidence for high levels of primate OA comes from DeRousseau (1985), who observed high levels of hip and vertebral degeneration in 55 captive rhesus macaques. DeRousseau's proposition that the conditions of captivity—not body size—lead to increased OA was supported by Rothschild and Woods (1992), whose study of 153 prosimians and 1,250 non-prosimian Old World primates revealed differences in OA frequencies between captive and wild prosimians (4.8% vs. 0.8%, or

six times more common) and captive and wild non-prosimian Old World primates (3.7% vs. 0.9%, or four times more common). Hip and elbow OA were only observed in captive primates, while knee OA was more common in wild; since both OA frequencies and distribution differed between captive and free-ranging primates, the authors rejected the idea that the differences were due to the longer lifespans of captive primates (Rothschild and Woods, 1992). Rather, it may be due to the fact that captive animals are frequently more obese and less physically active than free-ranging animals (Jurmain, 2000; Smith and Jungers, 1997). Smith and colleagues (2006) found that lifelong diet restriction in Labrador Retrievers delays the onset and reduces the severity of hip OA, when compared to littermates from age- and weight-matched control samples.

Likewise, in humans, overall body *size* (i.e., tall stature) has little effect on OA (Weiss, 2006). In fact, shorter individuals have been demonstrated to exhibit more OA (Weiss and Jurmain, 2007). Yet, body *mass* (i.e., obesity) consistently correlates with OA in medical studies (Coggon et al., 2001; Couchman, 2009; Felson et al., 1988; Felson and Zhang, 1998; Fransen et al., 2011). The above-summarized research suggests that *intra*specific skeletal dimensions scale *isometrically*, not allometrically: although an animal's joint surfaces are appropriate for species-level body mass and locomotion behaviors, they cannot accommodate radical inter-individual variations like extremes of body mass. The modern human tendency toward hip and other forms of OA may be due to the high levels of obesity in many of today's human populations.

Some of this correlation may be explained by skeletal biomechanics (though some may be metabolic; see discussion in Chapter 4). As summarized above, human

hip joint forces during walking and jogging are several times an individual's body weight, and the loads vary with weight and speed (Bergmann et al., 1993). Joint forces and stresses change with changing body mass, and an excess of adipose tissue leads to higher forces across weight-bearing joints. However, given that articular surface dimensions are conservative (*sensu* Lieberman et al., 2001; Ruff et al., 1991), the joints do not change correspondingly. This disproportionate loading contributes to the cartilage breakdown that is a part of the OA disease process (Felson and Zhang, 1998; Gill et al., 2011; Iannone and Lapadula, 2010; Wearing et al., 2006). Thus, obesity can affect the hip biomechanics and contribute to hip OA.

Pathological Conditions of the Human Hip: Developmental Dysplasia

Though it is a focus of this dissertation, OA is not the only pathological condition affecting the hip. In fact, in the hip more than other joints, OA is frequently secondary to developmental defects, abnormalities, and malformations (Felson, 1988; Ganz et al., 2008; Solomon, 1976). These include: acetabular retroversion, in which the joint space between the femoral head and acetabulum is abnormally narrow (Kim et al., 2006); hereditary cartilage disorders (e.g., multiple epiphyseal dysplasia, in which the abnormal ossification of the hip leads to irregular joint surfaces and early-onset OA; Bálint and Szebenyi, 2000); metabolic disorders (e.g., ochronosis, in which a genetic mutation results in a lack of enzyme activity leading to tendon calcification and destruction of the hips and knees; Bálint and Szebenyi, 2000); Legg-Calvé-Perthes disease, in which anomalies of vascular development lead to femoral head and corresponding acetabular deformities (Lee and Eberson, 2006); and various femoro-acetabular impingement scenarios contributing to changes in hip mechanics and ultimately to cartilage and bone damage (Ganz et al., 2008).

Probably the most common abnormality leading to early-onset hip OA, however, is developmental dysplasia of the hip (DDH). Formerly known as congenital hip dysplasia, or congenital hip dislocation, the disorder was recently renamed DDH in order to downplay the criterion of complete dislocation for diagnosis with the disorder and to reflect the role of development in its etiology (Aronsson et al., 1994). Researchers now recognize that DDH is frequently not present at birth; developmental dysplasias can occur during the prenatal or postnatal periods, and they can take many forms (Harcke, 1999). DDH can be defined as "the loss of the normal relationship between the femoral head and the acetabulum"—an appropriately general definition for a term that has come to encompass many variations on the spectrum of hip abnormality (Aufderheide and Rodríguez-Martín, 1998, p. 69; Harcke, 1999). While the most severe cases of DDH involve *luxation* (complete dislocation of the femoral head from the acetabulum), many involve *subluxation* (partial dislocation) and hip instability (in which the femoral head can move in and out of the acetabulum), or merely constitute minor acetabular or femoral malformations (Aronsson et al., 1994; Bialik et al., 1999; Committee on Quality Improvement, 2000). Additionally, most infants with DDH improve with time, even in absence of treatment (Bialik et al., 1999; Harcke, 1999). For that reason, estimating prevalence of DDH is difficult and dependent on the point in an infant's development at which DDH is diagnosed (Bialik et al., 1999).

DDH, Development, and Etiology

Several periods of embryonic, fetal, and early childhood development have been identified as sensitive periods for the development of DDH. 1. The period of time immediately following the development of the hip is a period of increased risk; if the femoral head dislocates at this stage of embryonic development, the entire development

of the hip proceeds abnormally, and the fetus is born with a shallow acetabulum, a distended hip joint capsule, and a small, malformed femoral head (Aronsson et al., 1994). 2. When the hip muscles develop *in utero* at around 18 weeks' gestation, abnormal muscular development can lead to hip dislocation (Aronsson et al., 1994). 3. During the last few weeks *in utero*, a fetus subjected to abnormal mechanical forces (e.g., breech positioning) can suffer dislocation (Shefelbine and Carter, 2004). 4. Finally, an infant experiences a risk of hip dislocation soon after birth, due to the laxity of the hip capsule, the continuing development of the acetabular labrum, and the cartilaginous state of the femoral head and acetabulum (Committee on Quality Improvement, 2000). Because of the laxity of the neonatal hip ligaments, femoral heads dislocated during this post-natal period frequently reduce without treatment, and development proceeds normally (Aronsson et al., 1994)—only approximately 10% of unstable hips require treatment (Shefelbine and Carter, 2004).

While DDH is the most common orthopedic problem in infants (Shefelbine and Carter, 2004), accounting for approximately 75% of congenital defects, incidences of DDH have been over-reported in the literature (Bialik et al., 1999). In fact, in a study of 18,060 infant hip ultrasound and clinical examinations, Bialik et al. (1999) found that the newborn DDH incidence of 55.1/1000 reduced without intervention within 2-6 weeks after birth to a true DDH incidence of only 5/1000. In reality, incidence of true DDH is low, and most cases resolve themselves, either with or without treatment.

However, when DDH is undiagnosed, untreated, or otherwise unresolved, it has a major impact on bone and soft tissue development, biomechanics, and the development of hip OA. If partial or complete dislocation persists, the femoral head

migrates supero-laterally (Aufderheide and Rodríguez-Martín, 1998), leading to flattening on its postero-medial side (Aronsson et al., 1994). Asymmetries on the growth front of the developing femur lead to increased femoral anteversion (Shefelbine and Carter, 2004), limb shortening, and pelvic tilt (in unilateral cases; Aufderheide and Rodríguez-Martín, 1998). With the femoral head repositioned and remodeled outside of its normal concentric position within the acetabulum, the acetabulum becomes shallow and flat (Aronsson et al., 1994), and the labrum can flatten and evert (Committee on Quality Improvement, 2000). In extreme cases, the supero-lateral migration of the femur can result in the formation of a false acetabulum (located superior to the shallow, malformed true acetabulum), in which the flattened femoral head rests (Aufderheide and Rodríguez-Martín, 1998). In such cases, the increased space between the femoral head and true acetabulum fills with fibro-fatty tissue, pushing the femoral head further supero-laterally and pulling the inferior joint capsule over the inferior aspect of the acetabulum (Aronsson et al., 1994).

Risks for DDH can be mechanical: for example, swaddling (Aronsson et al., 1994) or abnormal fetal contact with the maternal abdominal wall (Shefelbine and Carter, 2004). Breech positioning poses an increased risk (Shefelbine and Carter, 2004); breech-delivered infants are at an approximately six times higher risk of DDH than normally positioned fetuses (Aufderheide and Rodríguez-Martín, 1998). Researchers have also recognized a genetic component of DDH (Aronsson et al., 1994; Aufderheide and Rodríguez-Martín, 1998): children of healthy parents with previous children with DDH experience a 6% risk; children of affected parents experience a 12% risk; and children of affected parents with previous affected children experience a 36%

risk (Committee on Quality Improvement, 2000). Females more frequently suffer from DDH than males (an approximately five-to-eight times greater risk; Aufderheide and Rodríguez-Martín, 1998), possibly because females are more sensitive to the maternal hormone relaxin, which can lead to the *in-utero* ligamentous laxity that allows hip instability (Committee on Quality Improvement, 2000). The condition is rare in American blacks, Africans, and Chinese individuals (Aronsson et al., 1994; Aufderheide and Rodríguez-Martín, 1998) and more common in American whites (Aronsson et al., 1994) and Native Americans (Turkel, 1989).

DDH and OA

A discussion of DDH is relevant to the current research, as presence of the pathological condition could undermine the use of the acetabulum for age estimation. Yet, truly congenital, and particularly very early prenatal, scenarios of DDH—the ones that are the most devastating for hip joint stability, and the ones that typically do not reduce without surgical intervention—may be the only pathological DDH scenarios. All other, milder forms of hip abnormality tend to resolve themselves in the developing child, and can be classified as normal developmental variants. If diagnosed and treated early, most forms of DDH do not preclude the normal development of the hip joint (Shefelbine and Carter, 2004). However, if left untreated, DDH can result in femoral head osteonecrosis, pain, limited mobility, and early-onset OA (Committee on Quality Improvement, 2000; Shefelbine and Carter, 2004). The involvement of DDH in hip OA means that the abnormality also leads to increased risk for progression to total hip arthroplasty (Hochberg, 2004). It may be the reduced size of the dysplastic acetabulum that contributes to early hip degeneration: prolonged, high-magnitude stress on a disproportionately small weight-bearing area may lead to OA (Iglič et al., 1993). One

theory even states that all forms of hip OA are secondary to underlying hip defects like DDH, however subtle (Ganz et al., 2008; Murray, 1965; Solomon, 1976).

Certainly, the more severe pathological forms of DDH can be expected to correlate positively with hip OA. Researchers investigating age-related acetabular changes have warned that forensic anthropologists must recognize severe, pathological instances of DDH in skeletal remains and avoid using acetabular indicators in these individuals (Rissech et al., 2006). Fortunately for forensic anthropologists, however, these instances of severe DDH are highly diagnostic in skeletal remains, making the recommendation to eliminate such acetabula from age estimation studies easy to follow. Additionally, some research suggests that the age-related variables of the acetabulum may remain unaffected by DDH (Rougé-Maillart et al., 2007).

Thus, due to natural and surgical resolutions of DDH, incidence of true, pathological hip dysplasia is far lower than originally reported. Research suggests that sensitive study of the acetabulum can enable the identification of even the subtle forms of this abnormality, which may not interfere with the forensic anthropological estimation of age from the acetabulum.

Summary

This chapter provided an evolutionary framework for understanding the human hip, in addition to contextualizing forensic anthropological studies of the acetabulum with information on the modern human hip joint's growth, development, and biomechanics. It was argued that the evolutionary history of the hominin hip reflects a trend toward larger body sizes and allometrically scaling hip joints that have, over time, become sufficiently large and stable to withstand the stresses of bipedal locomotion. Finally, two pathological hip conditions were discussed: OA, a common condition linked

with aging and obesity; and DDH, a rarer condition less likely to impact anthropological analyses of the acetabulum. Hip OA was emphasized, and an evolutionary context was provided for the potential effects of age, activity, and obesity on its development. The next chapter provides a detailed analysis of this condition, discussing the etiology, pathogenesis, risk factors, and traditions of interpreting OA.

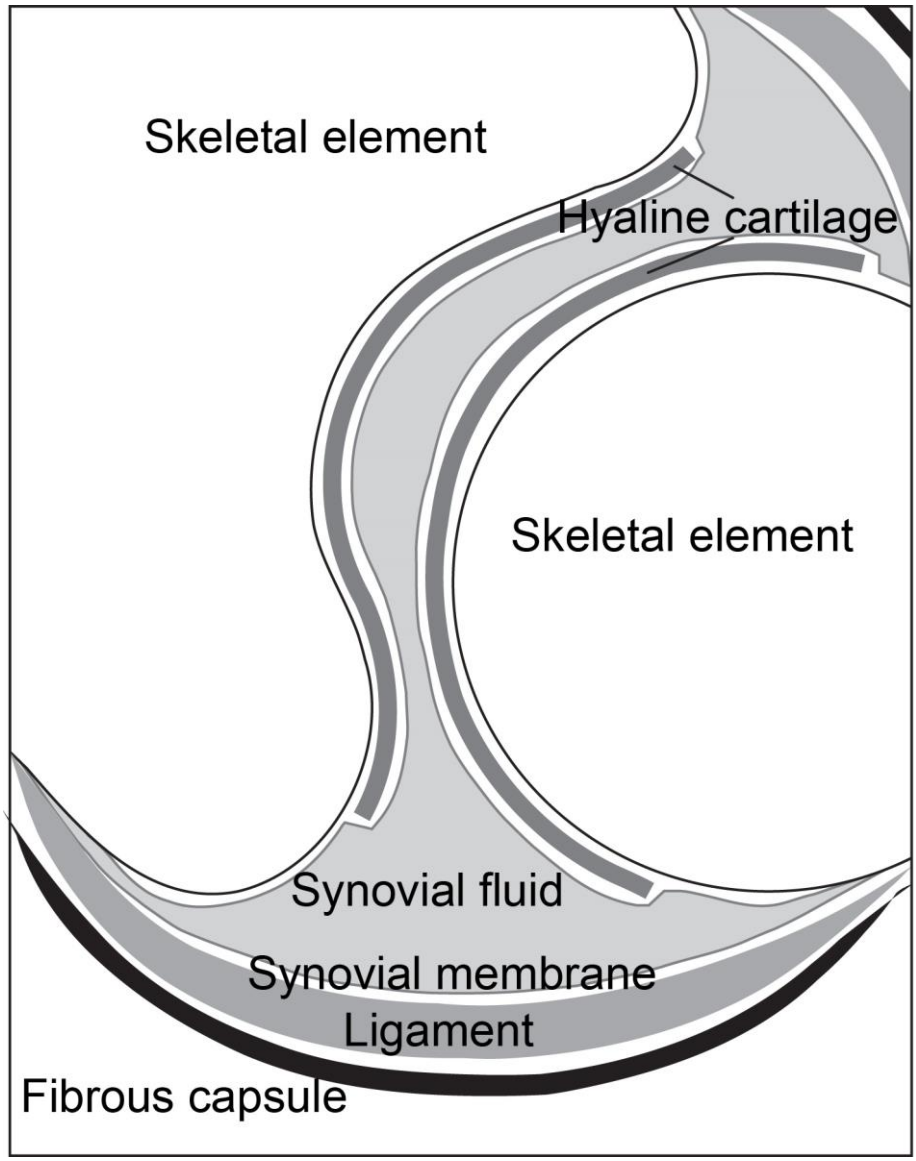


Figure 3-1. Illustration depicting the hard and soft tissues of the idealized diarthrodial joint. In addition to these basic components, the hip joint also displays a cartilaginous labrum that deepens the acetabular socket and a ligament connecting the acetabulum and the femoral head. Image by A.P. Winburn.

CHAPTER 4
ESSENTIALS OF OSTEOARTHRITIS

Often defined as the degeneration of articular surfaces in the skeleton, OA is a leading cause of disability in the elderly (Hunter and Eckstein, 2009; Mandl, 2007). In individuals of advanced age, knee and hip OA account for more lower limb disabilities than any other disease in the U.S. (Felson and Zhang, 1998). In 1998, symptomatic knee OA was estimated to occur in 6.1% of U.S. adults 30 years of age and older (Felson and Zhang, 1998); symptomatic hip OA approximated 4% (Lawrence et al., 1998). Approximately 15% (40 million) of Americans suffered from some form of arthritis ca. 1995; it is estimated that by 2020, 18.2% (59.4 million) will be affected (Lawrence et al., 1998).

Alternately called osteoarthrosis or degenerative joint disease, OA has also been observed in most non-human mammals, including bovids, canids, cervids, suids, extant primates, and the fossils of bipedal hominins (Cook et al., 1983; Fox, 1939; Jungers, 1988; Jurmain, 2000; Nichols and Zihlman, 2002; Smith et al., 2006; Trinkaus, 1985)—in addition to the skeletal remains of past peoples studied by bioarchaeologists (Jurmain and Kilgore, 1995; Weiss and Jurmain, 2007). Researchers in the fields of anthropology, primatology, and clinical and veterinary medicine acknowledge that OA is multifactorial, with factors such as age, body mass, joint size, bone mineral density, degree of mechanical loading/joint stress, activity patterns, and heredity contributing to an individual's propensity to develop OA. However, understanding the intricacies of this common condition has proved challenging, and conceptions of its complex etiology have changed over time.

Biological anthropologists have long recognized that factors like sex, ancestry, and body composition influence disease prevalence in past human populations (Damon, 1964). However, in the subfield of forensic anthropology, researchers have tended to emphasize the positive correlation of OA with advancing age (e.g., Stewart, 1979). In contrast, much research in the subfield of bioarchaeology has focused on activity as the main contributing factor to OA (e.g., Larsen, 1997). In the past, medical research also focused on the contribution of activity to OA (e.g., Felson and Zhang, 1998). In recent years, however, the medical fields have increasingly focused on obesity as the main modifiable risk factor for the disease and age as its major systemic contributor (Weiss and Jurmain, 2007).

This chapter describes current conceptions of the etiology of OA. It then presents research in the fields of biological anthropology and medicine summarizing the contributions of the major risk factors contributing to OA. As the focus of the current dissertation, age, activity, and obesity receive the most attention. Because of this dissertation's focus on age-related changes in the lower limb, this review also emphasizes the knee and hip, with particular emphasis placed upon the latter joint.

The Etiology of OA

Osteoarthritis is the most common form of joint pathology seen in human populations, both in the past (Jurmain and Kilgore, 1995; Weiss and Jurmain, 2007) and in the present (Mandl, 2007). Its etiology differs from that of rheumatoid arthritis (RA), an autoimmune disorder in which the immune system attacks joints throughout the body, causing inflammation and erosion. The progression of OA is degenerative rather than inflammatory, leading to its oft-repeated classification as "wear-and-tear" arthritis (Peterson et al., 2010, p. 1124). Its etiology, however, is more complicated and less

well understood than this simple description implies. Researchers emphasize the contribution to OA of both systemic factors (e.g., age, sex, ancestry, genetics, bone density, hormones) and local factors (e.g., injury, obesity, joint deformity, muscle weakness). Systemic factors act to increase an individual's risk of OA susceptibility, while local, often biomechanical, factors are the influences that determine the site or severity of the disease (Sharma, 2001). Aging may render tissues vulnerable to injury, for example, while obesity may add biomechanical stress (Felson and Zhang, 1998; Hunter and Eckstein, 2009).

The Pathogenesis of OA: Cartilage and Bone Changes

The preservation of a healthy joint organ is dependent in large part upon the maintenance of its major structural components: cartilage and bone. Alterations to these tissues lead to the pain, loss of motion, and joint instability characteristic of OA (Martel-Pelletier et al., 2007). The development and progression of OA involves complex interactions between cartilage and subchondral bone, leading to distinctive morphological changes within the joint organ. Subchondral bone metabolism increases, leading to the rapid deposition of hypomineralized bone, while the overlying articular hyaline cartilage thins and fibrillates (Figure 4-1). As the disease progresses, the joint space narrows and subchondral bone *sclerosis* (thickening/densification) becomes visible radiographically (Figure 4-2). Osteophytes form around joint margins (Figure 4-3), and pitting and porosity of the articular surface may occur (Figure 4-4). Ultimately, joint space narrowing and cartilage thinning leads to eburnation, the "bone-on-bone" polishing that results when articulating skeletal elements grind against each other without the protective cushion of articular cartilage (Figure 4-5). In extreme cases, the joint may experience ankylosis, or fusion (Figure 4-6). Finally, cysts comprised of

fibrovascular tissue or metaplastic cartilage may develop, surrounded by newly formed bone (Dequeker et al., 1997). However, the order in which these changes occur, as well as the nature of the relationship between cartilaginous and bony changes, have long been debated (Radin and Paul, 1970; Radin et al., 1972).

Human joints have evolved to function well under the unique conditions of bipedal locomotion (see Chapter 3), but they inevitably change as an individual ages; the more deformed a joint becomes, the less well it functions under load (Radin and Paul, 1970). Most researchers have assumed that it is the resilient joint cartilage and lubricating synovial fluid that provide the majority of joint compliance and joint force attenuation (Radin and Paul, 1970). Thus, OA—the breakdown of joint function—has traditionally been seen as primarily triggered by cartilage destruction (Martel-Pelletier et al., 2007). However, despite decades of research, the mechanisms involved in that cartilage destruction have remained incompletely understood, both at the macroscopic and the molecular levels. By the time most OA patients present with clinical symptoms, cartilage degeneration is often advanced, obscuring the early stages of the disease (Martel-Pelletier et al., 2007). Unlike in the well-understood molecular pathogenesis of inflammatory RA, no unequivocally implicated cytokine has been identified in OA's cartilage degeneration (Couchman, 2009). Further, cartilage changes are notoriously difficult to dissociate from changes to the underlying subchondral bone, even in the earliest stages of OA (Sokoloff, 1969).

In contrast with the traditional view of joint compliance as cartilage-driven, subchondral bone may play a more important role in protecting joints from impact loading than previously recognized. Subchondral bone actually attenuates joint forces

through its cortical and trabecular structures more effectively than does the thinner articular cartilage (Radin and Paul, 1970)—proving 30 times more effective in dissipating force through joints (Radin et al., 1970). Perhaps, then, OA is not a primary disorder of cartilage that leads to secondary bony changes. Subchondral bone defects (e.g., metabolically or systemically caused) may in actuality be the primary lesions—potentially reducing shock-absorption in the bone, transferring loading stress to cartilage, and causing secondary cartilage changes (Dequeker et al., 1997). When insulted by injury or inflammation, adult *chondrocytes* (cartilage cells) attempt to regress to their early role in cartilage formation; failure of these adult cells to replicate developmental conditions leads to degradation of the cartilage matrix (Goldring, 2007). Perhaps in OA, the initial cartilage insult results from detrimental changes to underlying bone structure. Research has long investigated links between bone density and OA, under the hypothesis that larger individuals with denser bone exhibit more OA (Burr et al., 1983; Carter, 1987; Dequeker et al., 1997). While bone density research is far from conclusive (see *Other Factors Impacting OA*, below), subchondral bone may play a more important role in initiating OA development than previously recognized (Martel-Pelletier et al., 2007). Indeed, it now appears that the pathogenic event actually occurs at the level of the bony tissue, not within the cartilage (Felson and Neogi, 2004; Radin et al., 1972).

The pathogenesis of OA has been linked to an increased stiffness and density of the subchondral bone—the sclerosis visible in radiographs of affected joints (Radin and Rose, 1986). With less-effective shock-absorbing capacities, this stiffened subchondral bone may be subject to trabecular microfractures that, when healed and remodeled, can

lead to further trabecular stiffening (Radin, 1982). This can in turn lead to loss of joint congruence, cartilage fibrillation, and ultimately OA. Alternately, the increased subchondral bone stiffness associated with the disease's progression may in fact be part of a general pattern of bone alteration seen in individuals predisposed to OA, independent of microfracture events (Dequeker et al., 1997). It is also possible that the variability in stiffness and density that characterizes osteoarthritic subchondral bone may be more detrimental to joint health and cartilage integrity than either demineralization or excess density in isolation (Fazzalari and Parkinson, 1998; Crane et al., 1990).

Thus, while OA is still understood to involve cartilage degeneration, it is now viewed as a generalized bone disease of the entire joint organ rather than an isolated disease of articular cartilage (Felson, 2004; Presle et al., 2007). It is seen as the outcome of injurious factors/activities acting on a vulnerable joint and leading to structural and functional joint failure, including: loss/degeneration of cartilage, alteration to subchondral bone, meniscal degeneration, inflammation, and osteophytic overgrowth (Felson, 2004; Hunter and Eckstein, 2000). Within these complicated interactions, bone integrity, not cartilage integrity, may be the key to prolonging joint health (Radin and Paul, 1970). The cartilage degeneration that has long been the focus of OA investigations may in reality be caused by changes in the mechanical properties of subchondral bone (i.e., microfractures, increased stiffness, decreased energy-absorption capacity).

The Pathogenesis of OA: Abnormal Stresses and Abnormal Tissues

Mechanical attrition likely plays a role in the pathogenesis of OA—but not always in the intuitive sense of "wear and tear." While some research indicates that joint

degeneration occurs in regions of cartilage that receive the heaviest loading (Afoke et al., 1987), OA seems to be more than the simple degenerative result of tissue-wounding (Couchman, 2009; Wollheim and Lohmander, 2007). Subchondral bone metabolism actually increases during some stages of OA, likely triggering the structural changes that ultimately degrade the overlying cartilage (Martel-Pelletier et al., 2007). Far from being merely *catabolic* (promoting tissue break-down), some cartilage injuries actually trigger *anabolic* (tissue-building) mechanisms (Couchman, 2009). In fact, "the pathophysiology of OA is a dynamic process that involves some regeneration and increased turnover of cartilage matrix components, new bone formation and joint remodeling, and degeneration of articular tissues" (Nuki and Salter, 2007, p. 33). Not strictly "wear and tear," then, OA occurs when there is an imbalance in the dynamic equilibrium between joint tissue repair and degeneration (Hunter and Eckstein, 2009).

Carter (1987) used the acetabulum to illustrate a proposed mechanism for the role of mechanical stress in skeletal maintenance and degeneration. He observed that the regions of the acetabulum that receive the highest magnitude of compressive subchondral bone stress (the acetabular roof and corresponding superior surface of the femoral head) are the regions where articular cartilage is thickest (Carter, 1987). Since bone is stronger in compression than in tension, these regions of high compressive stress rarely evince degenerative change. In contrast, degenerative change (e.g., cartilage fibrillation and osteophyte formation) is common in the medial-inferior and peripheral areas of the acetabular roof and femoral head—regions subjected to high tensile stresses. He concluded that while compressive stress is beneficial to cartilage and subchondral bone health, tensile strain might be the component contributing more

directly to OA, possibly because of the vascular invasion allowed when compressive and shear stresses are reduced (Carter, 1987).

Since bone forms, strengthens, and regenerates in response to activity, mechanical stress plays a large role in maintaining, as well as degenerating, joint tissue (Carter, 1987). Mechanical loading, at least during the process of maturation, has been shown to increase the formation of cartilage (Wearing et al., 2006). Habitual under-loading, in contrast, can lead to disuse atrophy, in which cartilage degrades and fibrillates (Wollheim and Lohmander, 2007). Moderate loading is thus more beneficial to joints than either extremely low or extremely high loading (Videman et al., 1990). But while normal joint loading is necessary for maintaining healthy joint cartilage, "acute injurious mechanical loading" (abnormal loading of normal cartilage or normal loading of abnormally formed cartilage) leads to chondrocyte death and cartilage degeneration, thus contributing to OA (Couchman, 2009, p. 47).

Within this proposed mechanism for OA, normal loading is beneficial for bone and cartilage health, but abnormal loading is detrimental; and systemic factors like age and heredity can predispose individuals to OA, but local biomechanical factors as obesity, injury, or extremes of physical activity can contribute to its progression, location, and severity (Figure 4-7). Abnormal cellular activity also plays a role in the pathogenesis of OA (Figure 4-7). In essence, OA can result either from abnormal mechanical stresses damaging normal tissues or from the failure of abnormal tissues under normal mechanical stresses (Nuki and Salter, 2007).

Interpreting OA

The disease is traditionally classified into primary (idiopathic) OA and OA secondary to another condition (e.g., hip dysplasia, traumatic injury). According to this

dichotomy, the former is seen as an intrinsic, age-progressive process of cartilage and bone degeneration, and the latter is believed to occur prematurely, subsequent to major trauma or disease. However, this primary-secondary dichotomy has a long history of criticism (Murray, 1965; Solomon, 1976). The list of conditions that can predispose an individual to secondary OA is extensive, and includes anything from metabolic disorders (e.g., gout) to endocrine disorders (e.g., acromegaly; Mandl, 2007). Even post-traumatic OA is strongly influenced by factors like obesity, sex, or hereditary predisposition (Couchman, 2009). It is becoming increasingly clear that even so-called primary forms of OA in fact result from multiple etiological factors (Nuki and Salter, 2007). Further, all OA (regardless of classification) tends to cluster in certain sites more than others, and these sites (e.g., the hip and knee) are the joints where other injuries, defects, and abnormalities most frequently occur (Solomon, 1976). In particular, dichotomizing hip OA into traditional primary-secondary categories implies etiological distinctness that may not exist (Solomon, 1976). An estimated 20-50% of adults with hip OA develop the hip degeneration secondary to DDH subluxation or full dysplastic luxation (Shefelbine and Carter, 2004). One theory even holds that all hip OA is in fact secondary to joint injury or developmental abnormality—however subtle (Ganz et al., 2008; Kim et al., 2006; Solomon, 1976). Indeed, studies report that approximately 90% of cases of hip OA are associated with underlying joint abnormalities (i.e., they were not traditional cases of primary OA), ranging from extreme traumatic events to subtle developmental defects (Ganz et al., 2008; Solomon, 1976). In essence, this research argues, all OA is secondary OA.

Osteoarthritis can be localized to a single joint or generalized throughout the body. In the latter case, generalized OA is often identified by the presence of the disease in both small joints (i.e., hands) and large joints (i.e., limbs). When three or more of these joints exhibit symptoms, a diagnosis of generalized OA is accepted (Mandl, 2007).

Yet, recognizing OA in these joints is far from straightforward. In living patients, diagnoses differ depending on whether clinical symptoms or radiographic findings are considered (Felson and Zhang, 1998). In the longitudinal Framingham Osteoarthritis Study, for example, 33% of individuals between ages 63 and 93 exhibited radiographic knee OA, but only 9.5% were symptomatic (Felson et al., 1987). The opposite can also be true, as patients with debilitating pain can exhibit minimal or no radiographic signs of OA (Mandl, 2007). This may be due to the fact that radiographic assessments rely on hard-tissue findings (e.g., osteophyte formation), while many of the symptoms of OA are linked with soft-tissue changes (e.g., cartilage fibrillation, inflammation).

The most common system used to evaluate and define OA radiographically is that developed by Kellgren and Lawrence in 1957. Using the Kellgren-Lawrence (1957) system, radiographs of a patient's joints are compared with a standard radiographic atlas, and the patient is assigned a grade of 0 (normal joint), 1 (possible osteophytes present), 2 (definite osteophytes present, with possible joint-space narrowing), 3 (moderate osteophytes present, with definite joint-space narrowing, some sclerosis, and possible attrition or deformity of bone contour), or 4 (large osteophytes present, with marked joint-space narrowing, severe sclerosis, and definite attrition or deformity of bone contour). A patient's radiographs could be considered positive for OA if they

receive a grade of 2 or higher on the Kellgren-Lawrence (1957) scale; however, most clinicians would also consider the patient's experience of pain before making a determination (Mandl, 2007). Thus, common epidemiological definitions combine radiographic evidence of OA with reported pain on most days of a month within the preceding year (Mandl, 2007). Such functional definitions have been approved by the American College of Rheumatology for the hand (Altman et al., 1990), hip (Altman et al., 1991), and knee (Altman et al., 1986).

Recognizing OA is equally complicated in the skeletal remains of deceased individuals. Without the presence of the cartilage, muscles, ligaments, tendons, and other soft-tissue structures that hold joints in anatomical alignment during *in vivo* clinical assessments, radiographic systems like that of Kellgren and Lawrence (1957) are inapplicable. Instead, biological anthropologists rely on OA scoring systems that describe changes visible on disarticulated skeletal material. Ordinal scoring systems such as those proposed by Buikstra and Ubelaker (1994) and Jurmain (1990) rely on the identification of traits like marginal lipping and osteophyte formation, porosity, eburnation, and ankylosis. Joint-space narrowing cannot be assessed on disarticulated skeletal remains, nor can the integrity of cartilage be assessed when soft tissue is decomposed.

As with the radiographic systems used to identify OA in the clinical setting, the relationships between skeletal indicators of OA and *in vivo* experiences of pain and disability are unknown. Some researchers allege that osteophytes (identifiable both on radiographs and skeletal remains) correlate well with OA symptoms (Felson and Zhang, 1998), while others believe that these marginal changes are of questionable clinical

significance (Loeser, 2007). Still others allege that marginal osteophytic changes are correlated with age, while changes observed on the articular surfaces are influenced by activity (Weiss and Jurmain, 2007). Meanwhile, porosity has been shown to have no significant relationship with OA (in study of the knees of 400 modern U.S. skeletal individuals; Rothschild, 1997). Moreover, while it is frequently utilized in skeletal studies, porosity is not acknowledged as a component of OA in the medical literature, and appears to have no clinical correlation (Rothschild, 1997). Ankylosis also goes unmentioned in clinical assessment systems like that of Kellgren and Lawrence (1957). Unfortunately, these areas of disconnect between clinical and skeletal OA assessment systems mean that medical and anthropological findings are necessarily incompatible.

Major Factors Impacting OA: Age, Activity, and Obesity

The differing theoretical perspectives adopted by OA researchers further compound the methodological difficulties of scoring OA in a meaningful manner based on radiographs or skeletal remains. Over the decades, medical and anthropological research has variously emphasized the impacts of age, activity, and obesity on the development and progression of OA. The influences of these three major OA risk factors are discussed below, along with changing attitudes in various fields toward their relative importance. Other risk factors for OA (not considered further in the current dissertation) are also briefly addressed.

Age

Osteoarthritis predominantly affects older adults. In fact, advanced age is the single most important systemic risk factor for the development of OA—for all joints and in both human and non-human species (Loeser, 2007; Mandl, 2007; Weiss and Jurmain, 2007). Multiple explanations have been proposed for this positive age

correlation: with age, chondrocytes become less responsive to growth/repair, rates of anabolic activity decrease, higher proportions of cartilage calcify, ligaments become lax, muscles weaken, and joints are more susceptible to injury (Felson and Zhang, 1998; Loeser, 2007). All of the above factors contribute to OA. However, since OA has little effect on an individual's fitness during reproductive years, there has been little selective pressure against the genes that may predispose older adults to the disease (Loeser, 2007).

The disease positively correlates with age in pre-contact Southwestern and Alaskan natives (*n*=208; *n*=146) and 20th century black and white American populations (*n*=444)—particularly significantly in the shoulder and hip (Jurmain, 1980). The notion that "hip disease is strongly correlated with advancing age" is one that has emerged repeatedly in the work of Jurmain (1980; Jurmain and Kilgore, 1995, p. 447), and it is the foundation for recent forensic anthropological studies studying age-related changes of the acetabulum (Calce, 2012; Calce and Rogers, 2011; Mays, 2012; Rissech et al., 2006; 2007; Rougé-Maillart et al., 2004; 2007). Forensic anthropologists also use the general occurrence of OA to inform estimates of age at death in unknown skeletons (e.g., the presence of OA indicates an age at death of 40 years or older; Stewart, 1979). Still, most forensic anthropologists would agree with Aykroyd and colleagues (1999, p. 59) that "the rate at which it occurs is dependent on so many variables…that using both osteoarthritis and osteoporosis as methods of adult aging must be considered ancillary (if not a last resort) to the other morphological methods."

However, it is important to note that despite its correlation with age, OA is not a normal part of the aging process (Wollheim and Lohmander, 2007). Bone may lose

density and cartilage may thin and roughen as joints age normally, with no subsequent OA changes. The joint-organ changes that come along with normal aging (e.g., ligamentous laxity) do not themselves result directly in OA; rather, these systemic age changes render the joints of older adults more susceptible to the local factors that directly lead to the disease's development (Loeser, 2007). These local factors include joint injuries and instabilities, abnormal mechanical loading, obesity, and heredity—all of which typically predate advanced age and thus drive the location and severity of the eventual development of OA (Loeser, 2007). Age is a risk factor for OA because age-related joint changes make it easier for another factor to influence the development of the disease; but whereas many older adults have healthy, aged joints, OA is a pathological condition of the joint organ (Solomon, 1976).

Activity

Intuitively, the assumption that repetitive, stressful physical activities lead to excessive joint loading—and ultimately, to joint damage and increased frequencies of OA—seems sound. Indeed, in the fields of bioarchaeology and biological anthropology, much research has been based on the assumption that "the primary contributing factor to osteoarthritis is mechanical stress and physical activity" (Larsen, 1997, p. 163). Bioarchaeological research has traditionally focused on the effects of activity on the development of OA (Bridges, 1991; Lallo, 1973; Larsen, 1982; Kennedy, 1989; Ortner, 1968), seeking to correlate lifestyle (e.g., subsistence, occupation) with the presence, frequency, and severity of joint degeneration in past populations. This type of research has attempted to draw conclusions about the cultural behaviors of past peoples via the patterning of OA: in an archaeological population with high levels of elbow OA, for

example, bioarchaeologists might posit repetitive arm movements such as spear-throwing as the cause (*sensu* Angel, 1966).

However, despite the intuitive connection between OA and activity, and the long history of bioarchaeological interest in the topic, no strong correlation between activity and OA has emerged. Different archaeological populations seem to have responded to similar physical stresses in different ways—and to different stresses in similar ways (Jurmain, 1977; 1980; 1990). Further, different authors have inferred activity in different ways and hypothesized differing pathways for its contribution to OA (e.g., habitual, repetitive activity [Williams, 2005] vs. infrequent, traumatic activity [Bridges, 1991]). Moreover, inconsistencies in the scoring and/or presentation of OA data abound, and it is often difficult to discern connections among the conflicting patterns that emerge from the bioarchaeological analyses of activity and OA. Likewise, in the medical literature, a definitive correlation between OA and activity has remained obscure.

Occupational physical activities

Early medical investigations into the etiology of OA focused largely on activity as the main contributing factor for OA. However, many early studies are plagued by methodological inconsistencies that preclude a valid determination of causal relationships (Maetzel et al., 1997). Weiss and Jurmain (2007) have estimated that the majority of clinical studies showing a strong positive correlation between activity and OA predate 1970, with later studies acknowledging the myriad other factors influencing the disease.

Still, a correlation between OA and physically demanding, repetitive (i.e., occupational) activities has emerged from investigations of the knee (Allen et al., 2010; Croft et al., 1992), and research has indicated that mechanical stress contributes to

knee OA, regardless of age, sex, or adiposity (Dahaghin et al., 2009). Knee bending (Cooper et al., 1994; Maetzel et al., 1997) and squatting (Dahaghin et al., 2009; Fransen et al., 2011) have been identified as particular risk factors for knee OA. Cooper and colleagues (1994) found that, after controlling for factors like obesity and Heberden's nodes (often used as a proxy for generalized, hereditary OA), knee OA was significantly higher in British men and women (*n*=218) whose occupations involved more than 30 minutes per day of squatting, kneeling, or stair climbing than in a control population (*n*=218). In their large (*n*=2,729) cross-sectional study of modern U.S. African American and Caucasian males and females, Allen and colleagues (2010) found no statistically significant associations between knee/hip OA and specific occupations; but they identified several specific activities that increased the odds of both knee and hip OA (heavy lifting, crawling, and doing heavy work while standing) and concluded that both knee and hip OA are associated with physically demanding tasks.

In general, however, the relationship between occupational activity and hip OA is less clear, and it has been less extensively investigated than in the knee. In a systematic literature review, Maetzel and colleagues (1997) reported a weak, but consistently positive link between occupational activity and hip OA (in contrast to the strong positive relationship between occupational knee bending and knee OA). Subsequent reviews of more recent literature reported a moderate positive association between physical workload and hip OA (Lievense et al., 2001). However, in a large-scale study of 85,191 Swedish males, hip disorders were the least prevalent form of musculoskeletal disorder among construction workers—in contrast with locations like the neck, upper limb, and lower back, which evinced high levels of degenerative

disease that the researchers attributed to occupational exposure to demanding physical work (Holmström and Engholm, 2003). In their study of hip OA and occupational activity in British males (hip OA patient *n*=245; control *n*=294), Croft and colleagues (1992) found no clear association between hip OA and any sampled occupation. However, when they restricted their analysis to those with severe disease, severe hip OA was observed more commonly in males whose long-term occupations involved farming, heavy lifting, or prolonged standing (Croft et al., 1992).

This correlation between farming and hip OA has been observed in multiple studies (Lievense et al., 2001; Maetzel et al., 1997; Thelin et al., 2004; Thelin and Holmberg, 2007); but the connection may be more than merely mechanical (Croft et al. [1992] have suggested an infectious origin for occupation-related OA in farmers). Research on the U.S. agricultural sector has also revealed a high risk of lower back and upper limb musculoskeletal disorders in farmers (Davis and Kotowski, 2007), although these researchers also emphasized that this is likely due not only to mechanical stresses, but also to farming-related injuries and other job hazards.

Thus, it appears that only certain high-intensity occupations predispose individuals for lower-limb OA, specifically those involving bending and lifting. Further, the positive correlation between strenuous occupations and lower-limb OA is stronger in the knee than in the hip.

Recreational physical activities

There is still less evidence for positive correlations between recreational physical activities and lower-limb OA. The initial findings of the Framingham Study suggested that physical activity led to higher levels of knee OA (Felson and Zhang, 1998). However, a recent analysis of the Framingham data showed that recreational activity

did not correlate with increased knee OA—even in obese individuals (Felson et al., 2007). These findings by the Framingham researchers have helped to bring about a sea change among OA researchers regarding the effect of physical activity on risk of OA.

A review of the literature on exercise and OA reveals that individuals with normal joints who participate in vigorous, low-impact, recreational exercise do not have an increased risk of OA (Hunter and Eckstein, 2009). In fact, some clinical researchers have even asserted that physical activity can improve joint tissue health—in particular, of the articular cartilage that lines the joint surfaces (Urquhart et al., 2011). Increasing muscular strength can also decrease pain and increase function in osteoarthritic joints (e.g., strengthening the quadriceps muscles can have a beneficial effect on knee OA; Hunter and Eckstein, 2009). Indeed, in its 2000 update on recommendations for the medical management of lower limb OA, the American College of Rheumatology Subcommittee on Osteoarthritis Guidelines recommended both strength training and exercise as beneficial non-pharmacological therapies. Exercise programs specifically targeting older adults with hip OA have also yielded positive results: Tak and colleagues (2005) reported that hip OA patients (*n*=55) experienced reduced levels of self-reported pain and disability and improved hip function after an eight-week exercise program, compared to controls (*n*=54).

Research also indicates that recreational sporting activities are not significantly correlated with hip OA (Croft et al., 1992). Five-, eight-, and nine-year longitudinal studies on the effects of aging and exercise on the hip and knee found no increased risk of OA in runners compared with age-matched non-runners (Lane et al., 1993; 1998;

Panush et al., 1995). One study of long-distance runners showed no increase in hip OA even after decades of running marathons (Puranen et al., 1975).

However, the intensity of the recreational activity may play some role in its contribution to OA. Elite-level competition in professional sports, for example, may be more injurious to lower-limb joints than recreational sporting activities (e.g., elite runners; Spector et al., 1996). However, the joint injuries often sustained by elite athletes may be the confounding factor in their subsequent OA, not the athletic activities themselves. Professional soccer players with anterior cruciate ligament and meniscal injuries, for example, are far more likely to develop knee OA than non-injured athletes (Neyret et al., 1993). Unfortunately, the fine line between beneficial and injurious loading remains poorly defined and poorly understood (Couchman, 2009). The difference may be between the repetitive microtrauma incurred by the high mechanical stresses of heavy physical tasks (e.g., repeated squatting)—tantamount to joint injury—as compared with the beneficial mechanical loading of low-impact recreational exercise (Hunter and Eckstein, 2009).

The now-acknowledged complexity of factors contributing to OA makes it impossible to attribute the disease to mechanical loading alone. As in medical research, the anthropological tide is turning against such simplistic interpretations. Researchers who previously touted OA as an indicator of culturally patterned mechanical stresses (e.g., Jurmain, 1977) now urge caution, calling activity-OA correlations “extremely tenuous” (Jurmain and Kilgore, 1995, p. 446). Even the recent research of C.S. Larsen, one of the strongest bioarchaeological proponents of an

activity-based etiology for OA, has suggested that OA is not merely attributable to excessive joint use (Peterson et al., 2010).

Rather, it seems that excessive mechanical stress can only effect degenerative change when combined with systemic susceptibility and/or local mechanical risk factors like injuries or mal-alignments (Figure 4-7). In the lower limb, for example, anatomical (i.e., varus/valgus) joint mal-alignments can alter adduction moment: in such cases, excessive compressive loading of the medial (*genu varum*) or lateral (*genu valgum*) compartments of the knee can dramatically increase risk of OA (Hunter and Eckstein, 2009).

Obesity

The sea change in the medical community away from an activity-driven etiology for OA has led many researchers to a new etiological focus. Increasingly, the medical literature emphasizes the importance of obesity to the development and progression of OA. As summarized in Chapter 3, the joints most frequently used in a species' locomotor adaptation are optimized to reduce the amount of biomechanical loading. Human knee and hip joints are extremely large relative to other primates—even compared with our large-bodied great ape relatives (Jungers, 1988). The large size of human leg joints was likely evolutionarily selected by locomotor pressures to reduce stress and limit OA during bipedal locomotion.

However, joint surfaces are conservative—representing not individual variations in mechanical loading but species-level adaptations for body mass support during locomotion (Lieberman et al., 2001; Ruff et al., 1991). The issue affecting joint degeneration, then, is not an individual's body *size*, but their body *mass*—specifically, it is likely that when an individual is carrying excess weight, they will exhibit earlier and

more severe OA (Smith et al., 2006). Indeed, heavier individuals have been shown to exhibit higher rates of lower limb OA (Felson et al., 2000). Studies have identified a clear connection between knee OA and body mass in multiple human populations (Coggon et al., 2001; Couchman, 2009; Felson et al., 1988; 2000; Felson and Zhang, 1998; Fransen et al., 2011). Obese individuals are three-to-four times more likely to develop knee OA than individuals with lower body mass indices (Couchman, 2009; Muthuri et al., 2011), and this increased risk of knee OA applies regardless of the individuals' level of physical activity (Felson et al., 1988). Indeed, researchers from the longitudinal Framingham Study have cited obesity as the most important modifiable risk factor for knee OA in females (Felson et al., 2000), stating unequivocally that obesity—or factors related to obesity—*causes* knee OA (Felson et al., 1988). Moreover, research indicates that when combined with other risk factors (e.g., injury, genetic predisposition to generalized OA), obesity interacts more than additively with each of these risk factors: for example, in an obese individual with previous knee injury, risk of knee OA rises from 8.2 to 21.6 (Coggon et al., 2001). There is less evidence for a relationship between increased body mass and increased risk of OA in the hip than in the knee (Felson and Zhang, 1998; Hochberg, 2004). This may be due in part to the fact that hip joint forces are lower than knee joint forces (Huang et al., 2000). Specific effects of obesity on the biomechanics of the hip joint are discussed above, in Chapter 3.

Since obesity is a modifiable risk factor for OA, research has focused on weight loss as a treatment for OA symptoms (Coggon et al., 2001; Felson et al., 1992; Huang et al., 2000; Richette et al., 2011). Each additional kg of body mass increases the

compressive load of the knee by ~4kg (Hunter and Eckstein, 2009). For example, obese young adults have greater knee-joint loads than young adults of normal body mass, experiencing significantly greater absolute ground reaction forces, along with larger step width, and greater sagittal-plane net muscle moments (and joint loads) at the hip, knee, and ankle (Browning and Kram, 2007). Thus, even slight weight loss can cause dramatic reduction in compressive stress. Huang and colleagues (2000) found that obese Chinese individuals with bilateral knee OA (*n*=126) who received weight loss treatment (with and without electrotherapy) experienced greater pain reduction, increased walking speed, and reduced disability index scores compared with individuals who received electrotherapy in absence of weight loss treatment. Research from the Framingham Study indicated that loss of ~5.1kg over 10 years reduced the risk of incident knee OA by 54% in American females (Felson et al., 1992). Richette and colleagues (2011) found that OA pain and inflammation decreased with massive weight loss in 44 French knee OA patients who received gastric bypass surgery. Coggon and colleagues (2001) have gone so far as to estimate that if all overweight and obese individuals reduced their body mass index to within the recommended normal range, the number of surgical cases of knee OA could be reduced by half; losing just 5kg of body mass could eliminate 24% of cases. The possibility of reducing knee OA risk by 50% in U.S. populations by preventing obesity was confirmed by Muthuri and colleagues (2011). The Johns Hopkins Arthritis Center also emphasizes the role of body weight in OA and recommends weight loss as a way to reduce joint stress and alleviate arthritis pain—particularly in the knee (Bartlett, 2012). A recent review of the obesity, OA, and weight-loss literature confirms that in general, weight loss improves OA symptoms in

both the knee and the hip (Gill et al., 2011)—though the distinction must be made between losing adipose tissue (which can have a palliative effect on OA) and losing absolute mass (often including muscle mass, which can reduce joint strength and thus worsen the progression of the disease; Mandl, 2007). Losing weight is thus a "logical" way to reduce pain in joints and slow the progression of degeneration (Huang et al., 2000, p. 404), as it decreases the forces across weight-bearing joints like the knee and hip.

However, the mechanisms by which obesity contributes to OA are not merely biomechanical. Obese individuals suffer from circulatory problems that can lead to cartilage breakdown, triggering OA (Huang et al., 2000). Obesity is a complex syndrome, a low-grade inflammatory disease triggering abnormal neuroendocrine and pro-inflammatory pathways that can lead to metabolic changes (Iannone and Lapadula, 2010; Richette et al., 2011). Obese individuals experience high levels of OA not only in weight-bearing joints, but also in joints like the hands, indicating a systemic effect of obesity (Cicuttini et al., 1996; Felson and Zhang, 1998; Kalichman et al., 2005; Richette et al., 2011). Obese individuals tend to have higher bone mineral density, which has been linked to OA (Huang et al., 2000). Further, obesity can cause the overproduction of the pro-inflammatory molecules that promote synovial inflammation and contribute to OA symptoms (Gill et al., 2011; Iannone and Lapadula, 2010). Some types of adipose tissue can even act as an endocrine organ, releasing into the blood stream harmful cytokines—cell-signaling proteins that trigger cartilage degeneration and bone remodeling (Iannone and Lapadula, 2010; Richette et al., 2011). Specifically, *leptin*, a protein produced by *adipocytes* (fat cells) and sometimes secreted directly into joint

capsules, has been found in high levels in the tissues of OA patients (Lajeunesse et al., 2004). Leptin induces the expression of growth factors that can promote articular damage (Presle et al., 2007). A product of the obese (*ob*) gene, leptin may be the metabolic link between obesity and OA, influencing the cartilage degradation and abnormal subchondral osteoblast activities characteristic of the disease (Bai et al., 1996; Presle et al., 2007). Recent research on systemic biochemical markers of cartilage synthesis and degeneration indicates that in addition to improving OA pain, massive weight loss can also improve metabolic function, reduce inflammation and promote cartilage production (Richette et al., 2011). This means that OA may not only be a biomechanical disease, but also a metabolic one (Eaton, 2004; Lajeunesse et al., 2005; Presle et al., 2007).

Effects of activity and age notwithstanding, the recent medical research increasingly points to obesity as a major factor in the development and progression of OA. Still, the correlation between obesity and OA is stronger in the knee than in the hip.

Other Factors Impacting OA

Heredity

In recent years, researchers have engaged with the contribution of heredity to OA. Felson and Zhang (1998) have reported that an individual is more likely to develop OA if his parents also had the disease—particularly if it affected more than one articular surface. In a comprehensive Icelandic study that compared ~30 years of recorded total hip arthroplasties with an extensive genetic database, 2,713 individuals with hip OA were significantly more related to each other than they were to age- and sex-matched controls (Ingvarsson et al., 2000). Heberden's nodes (as a proxy for generalized, inherited OA) are significantly correlated with knee OA (Coggon et al., 2001) and hip OA

(Marks et al., 1979), indicating a generalized, heritable, systemic pattern of OA. In fact, individuals with hand OA (as a proxy for heritable OA) may be three times more likely to develop knee OA after meniscal injury than individuals without generalized OA (Couchman, 2009). Bálint and Szebenyi (2000) have suggested that occurrence of OA in individuals younger than 50 years of age indicates a genetic predisposition to the disease. Additionally, many of the developmental disorders contributing to OA (e.g., hip dysplasia) are hereditary (Ganz et al., 2008). Other hereditary disorders (including osteochondrodysplasias like achondroplasia and Stickler's syndrome) can cause genetically determined articular cartilage defects, leading to biomechanical abnormalities and early-onset OA (Bálint and Szebenyi, 2000). Hereditary metabolic disorders can also cause secondary OA (Bálint and Szebenyi, 2000). However, untangling the contribution of heredity to OA has proven challenging, as genetic polymorphisms impact OA in complex ways (Couchman, 2009).

Previous injury

Major joint trauma leads to biomechanical changes that increase joint stresses (Felson and Zhang, 1998). Several studies have found a significant positive correlation between previous knee injury and knee OA (Coggon et al., 2001; Couchman, 2009; Neyret et al., 1993). The results of the Framingham Study have confirmed that males with major knee injuries are at a five-to-six times higher risk of developing knee OA than those without joint injuries; injured females have a three times higher risk (Zhang et al., 1996). In fact, knee injuries have been cited as the most important modifiable risk factor for knee OA in U.S. males and the second most important factor (after obesity) in females (Felson et al., 2000). In the hip, acetabular fractures have been cited as leading to post-traumatic arthritis (Chuckpaiwong et al., 2009), as have labral tears and

other labral lesions (McCarthy et al., 2001). Previous joint injury also emerged as the leading cause of hip OA in a recent study of Asian individuals (Fransen et al., 2011). Finally, in Solomon's (1976) study of 327 hips, 91.7% of the osteoarthritic hips also exhibited underlying joint abnormalities, ranging from extreme traumatic events to subtle developmental defects.

Female sex

Coggon and colleagues (2001) have reported higher incidence of OA in females than in males; and the Framingham Study has found that overweight females are at greater risk of knee OA than overweight males (Felson et al., 1988). McKean and colleagues (2007) found that adult females with moderate knee OA underwent biomechanical changes in gait that were not observed in males, suggesting that the biomechanics associated with knee OA are sex-dependent. It remains difficult, however, to identify whether sex-specific gait differences contribute to higher knee OA in females, or whether these differences are a measure of the different effect knee OA has on females (McKean et al., 2007). Although young men may suffer from higher levels of OA than young women, the sex imbalance shifts after age 50, and the higher prevalence in women only increases with age (Felson and Zhang, 1998). A multitude of factors may contribute to this imbalance, including differences in male and female anatomy, hormonal influences, and, in the case of obese individuals, the disproportionate loading of small female joint surfaces (Weiss and Jurmain, 2007). Additionally, females suffer more frequent acetabular dysplasia than males, itself a factor in the development and progression of hip OA (Ganz et al., 2008; Solomon, 1976).

Bone density

Some research supports an inverse relationship between osteoporosis and OA: essentially, larger individuals tend to have higher bone mineral content and more OA; smaller, frailer individual tend to have lower bone mineral content and less OA (Carter, 1987; Dequeker et al., 1997). However, other research has shown the correlation to be less clear. Burr and colleagues (1983) found that in a native Alaskan sample (*n*=123), male and female individuals exhibited different patterning of bone mineral content and OA: the most severe female OA was noted in osteoporotic individuals; while the most severe male OA was observed in individuals with high bone mineral content (Burr et al., 1983). They concluded that while high bone mineral content may contribute to OA, low bone mineral content does not protect against it (Burr et al., 1983). Some of this conflicting evidence may be due to the difficulty of assessing bone quality via bone mineral content, which often represents a volumetric measurement rather than a direct measurement of bone mineralization.

The literature provides similarly conflicting views on the correlation between propensity for excessive bone growth and propensity for OA. In a sample of 337 skeletal individuals, Rogers and colleagues (1997) found a significant positive correlation between osteophytes and enthesophytes. However, in his study of acetabular degeneration in the Spitalfields skeletal collection, Mays (2012) found evidence to the contrary: degenerative changes were not correlated with diffuse idiopathic skeletal hypertrophy (as proxy for the tendency for excessive bone formation). The influence of bone density on OA thus remains contested in the literature.

Nutrition

In investigating differing rates of age-related degenerative change in individuals of different racial affiliations, forensic anthropological researchers have posited poor nutrition as a risk factor for joint degeneration (Katz and Suchey, 1989); bioarchaeological researchers investigating the transition from foraging to agriculture have done the same (Larsen et al., 2001). A recent animal study provides evidence to support this correlation: in a fifty-year study of a protected moose population (*n*=1,099), Peterson and colleagues (2010) found that poor moose nutrition early in life is linked with higher OA and reduced life expectancy. The researchers used size as a proxy for early nutrition, and found that the smallest moose (<33rd size percentile) were 32% more likely to die with OA than the largest moose (>66th percentile; Peterson et al., 2010). They drew a connection between the link between moose nutrition and OA, and the higher incidences of OA observed in native American populations after the introduction of agriculture, and the presumed concurrent nutritional depletion (Peterson et al., 2010). While this is a promising line of future research, studies of the effect of nutrition on the development and progression of OA are still in their infancy.

Summary

This chapter highlighted the multifactorial etiology of OA, which, while often characterized as degenerative “wear and tear,” in actuality includes both anabolic and catabolic components and involves both subchondral bone and the overlying hyaline cartilage. Theoretical and methodological challenges of interpreting OA were reviewed. Research investigating potential associations between OA and age, activity, obesity, and other factors was synthesized, with an emphasis on the effects of these factors on OA in the lower limb. While age is a definite risk factor and obesity seems to impact OA

(particularly in the knee), the effects of physical activity seem less clear. The next chapter introduces the materials and methods used in the current study to investigate the effects of age, activity, obesity, and OA on progressive changes in the acetabulum.

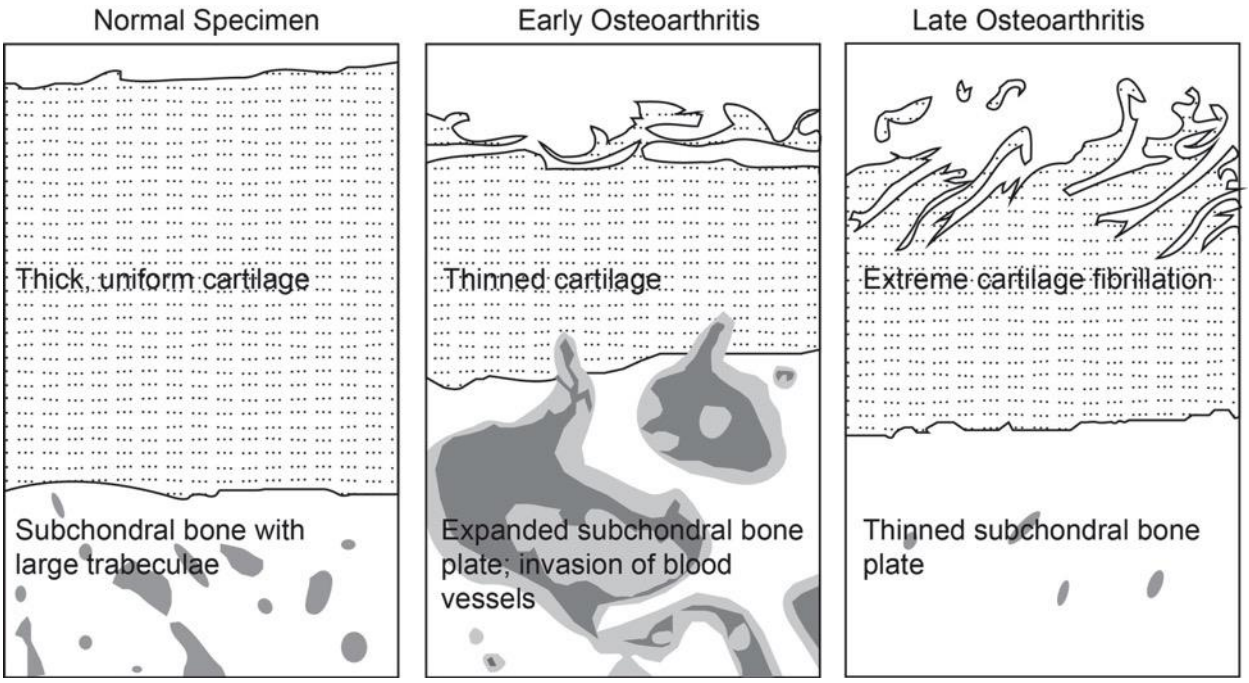


Figure 4-1. Illustration depicting histological sections of subchondral bone and cartilage from normal, early OA, and late OA patients. Based on Hematoxylin and Eosin stain at x60 magnification depicted by Martel-Pelletier and colleagues (2007:Plate 1). Image by A.P. Winburn.

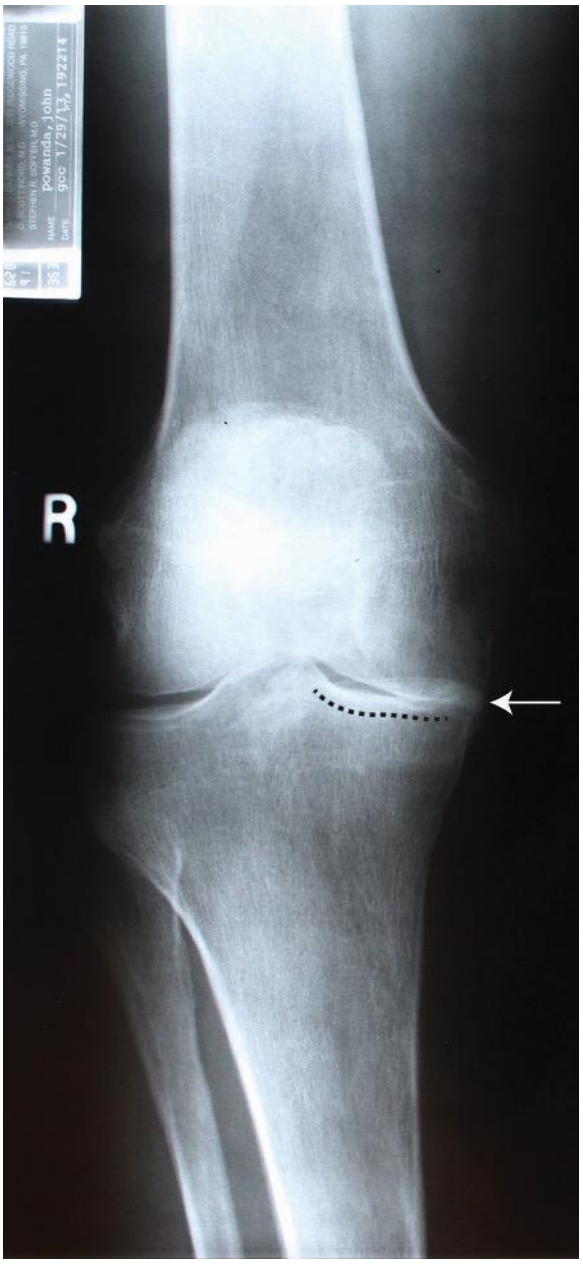


Figure 4-2. Radiographic knee OA, with antero-posterior radiograph of the right knee showing joint-space narrowing (white arrow) and subchondral bone sclerosis (black dashed line). Radiograph courtesy of John R. Powanda.

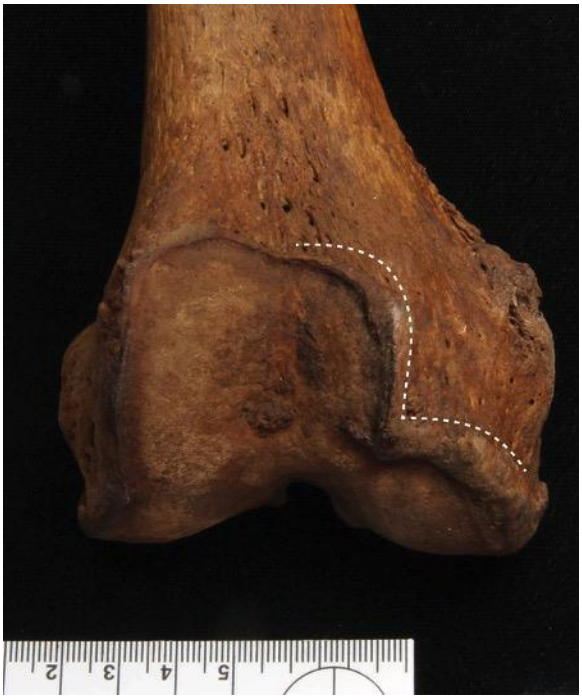


Figure 4-3. Anterior photograph of distal right femur exhibiting osteophyte formation around the margins of the articular surface (white dashed line). Scale is in cm. Photograph by A.P. Winburn.

Figure 4-4. Posterior photograph of left proximal femur exhibiting porosity of the articular surface. Eburnation and osteophytes are also present. Photograph by A.P. Winburn.

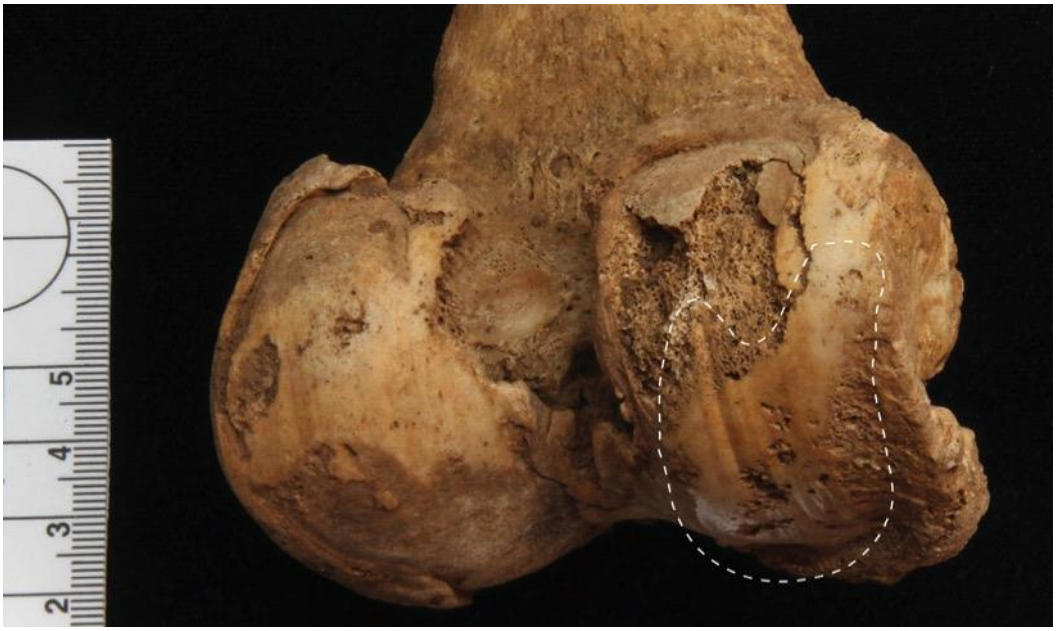


Figure 4-5. Posterior photograph of distal left femur exhibiting eburnation (white dashed line). Scale is in cm. Note shiny, grooved texture where the articulating tibia has polished the femoral condyle. Osteophytes are also present. Photograph by A.P. Winburn.

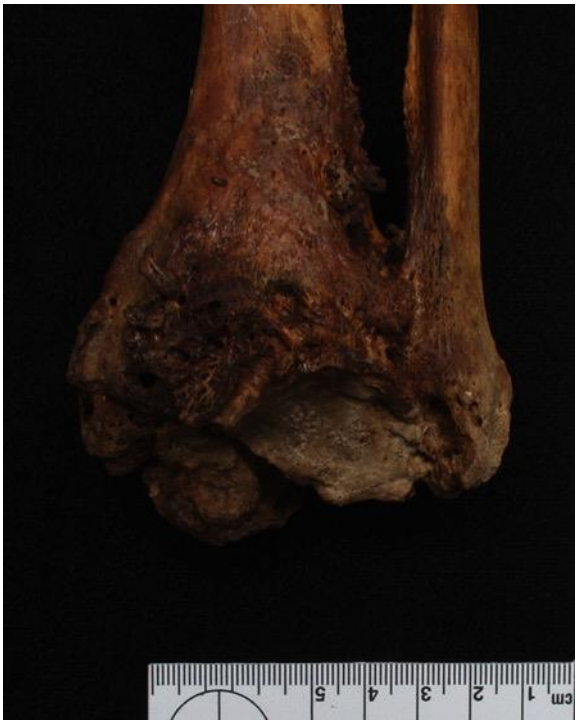


Figure 4-6. Posterior photograph of distal right tibia, fibula, and talus exhibiting pathological (post-surgical) ankylosis (complete joint fusion). Photograph by A.P. Winburn.

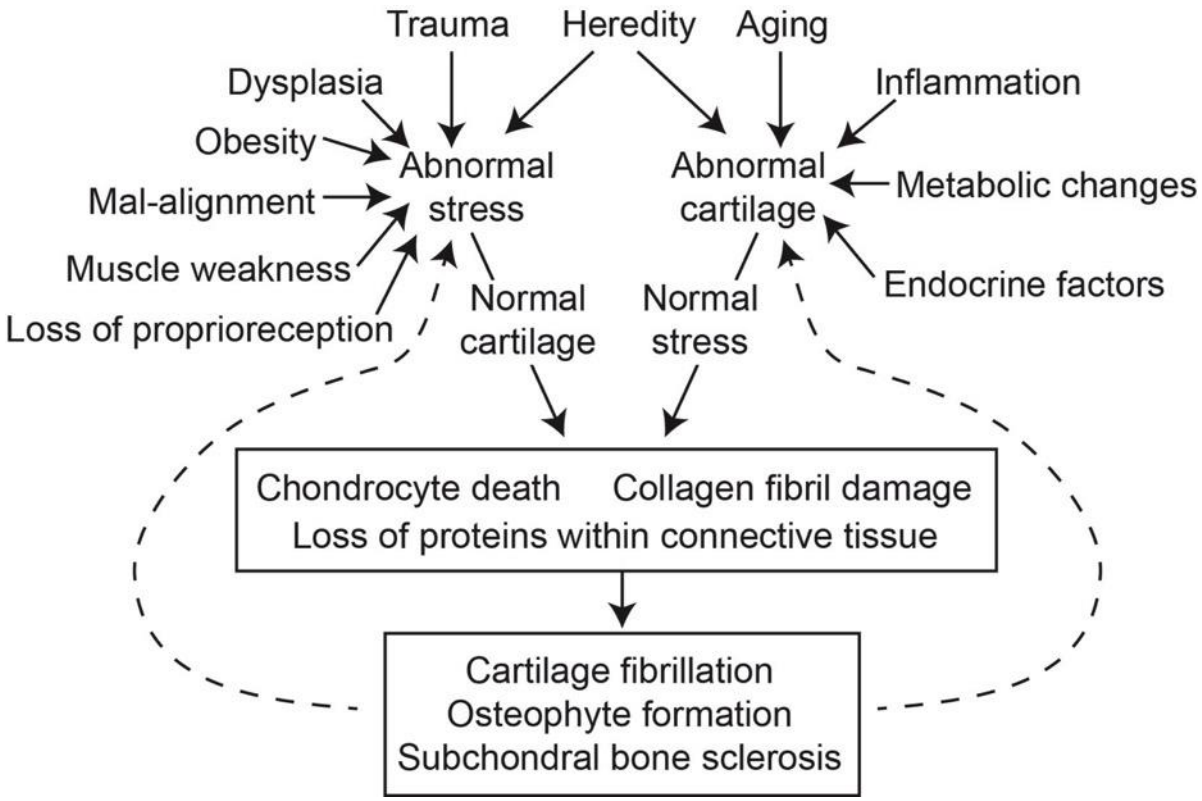


Figure 4-7. The effect of mechanical stress (normal and abnormal) on the development of OA, after Nuki (2005) and Nuki and Salter (2007, p. 34). Image by A.P. Winburn.

CHAPTER 5
MATERIALS AND METHODS

This research investigated the relationship of macroscopic acetabular age changes with overall OA in a sample of 409 European-American skeletal individuals. Documented demographic data for these individuals enabled an analysis of the effects of factors like age, sex, obesity, and occupational/habitual physical activities.

Skeletal Sample: The W.M. Bass Donated Skeletal Collection

The *W.M. Bass Donated Skeletal Collection* is a large donated collection of modern U.S. skeletal individuals (*n*>1700) housed at the University of Tennessee, Knoxville. Approximately 700 individuals within the collection are associated with exceptionally complete personal data, including: age, sex, and ancestry; height and weight; illnesses and injuries; and occupational/habitual activities. This documented sample provides an opportunity to investigate the effects of age, activity, and obesity on the development of OA and the progressive changes of the acetabulum, in individuals for whom other important demographic data are also known.

A stratified random sample was assembled[1] using eight age groups approximating 10-year intervals: 19-29 years, 30-39 years, 40-49 years, 50-59 years, 60-69 years, 70-79 years, 80-89 years, and 90-101 years. A goal of 30 individuals per age cohort was set for both females and males. However, because of the demographic makeup of the *Bass Collection*, (fewer females than males; fewer extremely young and

[1] Using this sampling strategy, individuals previously determined to meet the study's documentary requirements (i.e., data on age, ancestry, sex, height, body mass, habitual/occupational activities) were chosen at random from pre-defined age and sex groups. These documentary requirements formed the basis for most individuals' inclusion in the study sample. However, some of the youngest and oldest individuals in the *Bass Collection* lacked certain of these demographic data. These individuals were still included in order to create balanced, sufficiently large sample sizes in the least well-represented age and sex groups. This means that the sample sizes for the various analyses detailed below varied based on the availability of demographic data for the included individuals (see Chapter 6).

extremely old individuals) some of these 10-year age categories could not be fully populated (also see *Study Limitations*, below, for a discussion of the collection's ancestral limitations).

The resulting 409 European-American females (*n*=198) and males (*n*=211) comprise the overall study sample (Table 5-1). In addition to the 10-year age groups, documented ages for all individuals were also used in statistical analyses. For the sample as a whole, the mean age is 61.5 years, median age is 61 years, and minimum and maximum ages are 19 and 101 years, respectively. The age distribution for the overall sample appears in Figure 5-1, and the sample distribution by sex appears in Figure 5-2.

Hypotheses

This research tested a series of hypotheses examining the relationship between acetabular changes and OA (Hypothesis 1), the interactions of age, activity, and obesity with acetabular changes (Hypotheses 2a, 3a, and 4a) and overall OA (Hypotheses 2b, 3b, and 4b), and the relative contributions of these factors to acetabular changes and OA, respectively (Hypotheses 5a and 5b). Most hypotheses were paired, with one acetabulum-specific component (a) and one general-OA component (b).

Summaries of the hypotheses and their testing protocols are listed below. Further details about statistical testing are provided below (see *Testing*) and in Chapter 6.

Hypothesis 1: Acetabular Changes Correlate Positively with OA

Individuals with higher composite acetabulum scores (CAS; see Mays, 2012 and *Data Analysis*, below) and higher individual acetabulum aging scores for the seven variables (Rissech et al., 2006) were predicted to exhibit more OA in the other joints of

the body. Within-body comparisons (e.g., acetabulum vs. knee, elbow) were conducted. Acetabular CAS and each of the seven individual acetabular variables were tested for positive associations with OA scores (Jurmain, 1990) in individual joints and combined joint regions (overall OA, upper limb OA, and lower limb OA).

Associations between ordinal datasets were visualized via box-and-whisker plots and assessed via Spearman's rank-order correlation tests (*rho*) and Spearman's tests using an asymptotic approximation of the exact distribution (*p*). Associations between continuous datasets were visualized via scatterplots and assessed via Spearman's rank-order correlation tests (*rho*) and linear regression (*p*, adjusted r^2).

Hypothesis 2: Age

Hypothesis 2a: Acetabular changes correlate positively with age

Older individuals were expected to exhibit "older-looking" acetabula. Acetabular age estimates (Rissech et al., 2006) for the sample individuals were compared with the individuals' documented ages. Scores for the seven individual acetabular variables and overall CAS were tested for positive associations with age.

Measures of aging error included: percentage of individuals correctly classified into an age group; percentage of prediction errors; inaccuracy; bias; maximum over- and under-estimation; and *rho* values for correlations between estimated and documented ages. Associations between ordinal datasets were visualized via box-and-whisker plots and assessed via Spearman's rank-order correlation tests (*rho*) and Spearman's tests using an asymptotic approximation of the exact distribution (*p*). Associations between continuous datasets were visualized via scatterplots and assessed via Spearman's rank-order correlation tests (*rho*) and linear regression (*p*, adjusted r^2).

Hypothesis 2b: OA correlates positively with age

Older individuals were expected to show more OA in all body regions. In particular, a primarily systemic cause for OA (age) was expected to lead to OA not only in the lower limbs, but also in joints not implicated in locomotion (e.g., shoulder, elbow). Scores for OA in individual joints and overall body regions were tested for positive associations with the individuals' documented ages.

Associations between ordinal datasets were visualized via box-and-whisker plots and assessed via Spearman's rank-order correlation tests (*rho*) and Spearman's tests using an asymptotic approximation of the exact distribution (*p*). Associations between continuous datasets were visualized via scatterplots and assessed via Spearman's rank-order correlation tests (*rho*) and linear regression (*p*, adjusted r^2).

Hypothesis 3: Activity

Hypothesis 3a: Acetabular changes correlate positively with activity

Individuals who had undertaken more rigorous occupational and habitual physical activities were expected to exhibit "older-looking" acetabula than age-matched individuals undertaking less rigorous physical activities. Scores for the seven individual acetabular variables and overall CAS were tested for positive associations with the MET level of documented physical activities (see *Data Analysis*, below). In particular, tests assessed whether high acetabular scores were associated with rigorous documented habitual physical activities utilizing the lower limb (e.g., running, wrestling). To control for age, individuals were compared within age-matched 10-year subsamples (e.g., 30-39 years, 40-49 years).

Associations between ordinal datasets were visualized via box-and-whisker plots and assessed via Spearman's rank-order correlation tests (*rho*) and Spearman's tests

using an asymptotic approximation of the exact distribution (*p*). Associations between continuous datasets were visualized via scatterplots and assessed via Spearman's rank-order correlation tests (*rho*) and linear regression (*p*, adjusted r^2). Wilcoxon rank-sum tests assessed differences between individuals engaging in strenuous physical activities utilizing the lower limb and individuals who did not engage in these rigorous activities.

Hypothesis 3b: OA correlates positively with activity

Individuals who had undertaken more rigorous occupational and habitual physical activities were expected to exhibit more OA than age-matched individuals undertaking less rigorous physical activities. Scores for OA in individual joints and overall body regions were tested for positive associations with the MET level of documented physical activities. In particular, tests assessed whether high hip, knee, and ankle OA scores were associated with rigorous documented habitual physical activities utilizing the lower limb (e.g., running, wrestling). To control for age, individuals were compared within age-matched 10-year subsamples.

Associations between ordinal datasets were visualized via box-and-whisker plots and assessed via Spearman's rank-order correlation tests (*rho*) and Spearman's tests using an asymptotic approximation of the exact distribution (*p*). Associations between continuous datasets were visualized via scatterplots and assessed via Spearman's rank-order correlation tests (*rho*) and linear regression (*p*, adjusted r^2). Wilcoxon rank-sum tests assessed differences between individuals engaging in strenuous physical activities utilizing the lower limb and individuals who did not engage in these rigorous activities.

Hypothesis 4: Obesity

Hypothesis 4a: Acetabular changes correlate positively with obesity

Obese individuals were expected to exhibit "older-looking" acetabula than age-matched normal-weight or underweight individuals. This hypothesis employed the body mass index (BMI) as a proxy for obesity. Scores for the seven individual acetabular variables and overall CAS were tested for positive associations with BMI values (see *Data Analysis*, below). To control for age, individuals were compared within age-matched 10-year subsamples.

Associations between ordinal datasets were visualized via box-and-whisker plots and assessed via Spearman's rank-order correlation tests (*rho*) and Spearman's tests using an asymptotic approximation of the exact distribution (*p*). Associations between continuous datasets were visualized via scatterplots and assessed via Spearman's rank-order correlation tests (*rho*) and linear regression (*p*, adjusted r^2).

Hypothesis 4b: OA correlates positively with obesity

Obese individuals were expected to exhibit more OA than age-matched normal-weight or underweight individuals. Scores for OA in individual joints and overall body regions were tested for a positive association with BMI values. To control for age, individuals were compared within age-matched 10-year subsamples.

Associations between ordinal datasets were visualized via box-and-whisker plots and assessed via Spearman's rank-order correlation tests (*rho*) and Spearman's tests using an asymptotic approximation of the exact distribution (*p*). Associations between continuous datasets were visualized via scatterplots and assessed via Spearman's rank-order correlation tests (*rho*) and linear regression (*p*, adjusted r^2).

Hypothesis 5: Relative Contributions of Age, Activity, and Obesity

Hypothesis 5a: Of the above factors, age has the most influence on acetabular changes

Once the above-proposed associations between acetabular changes and age, activity, and obesity had been supported or rejected, this research endeavored to determine the relative importance of these contributing factors. Multiple regression tests isolated the variables exerting the greatest influence on acetabular changes.

Hypothesis 5b: Of the above factors, age has the most influence on OA

Once the above-proposed associations between OA and age, activity, and obesity had been supported or rejected, this research endeavored to determine the relative importance of these contributing factors. Multiple regression tests isolated the variables exerting the greatest influence on OA.

Ancillary Research Goals

In addition to the primary research hypotheses stated above, the current research also tested for associations between: previous hip trauma and acetabular aging scores; previous trauma and OA throughout the joints of the body; biological sex and acetabular aging scores; and biological sex and OA throughout the joints of the body. Further, in a subsample of the 409 individuals under study (n=292), the preservational states of the three pelvic joints (acetabulum, iliac auricular surface, pubic symphysis) were assessed, to test whether previous claims about the relative robusticity of the acetabulum were supported in the *Bass Collection* sample.

Wilcoxon rank-sum tests were used to assess differences in these datasets. To assess associations between previous traumatic injuries/surgical interventions and OA/acetabular changes, repeated resampling and comparison with median values for

affected joints evaluated whether unaffected joints would yield comparable levels of OA/acetabular changes.

Data Collection

All skeletal individuals included in the sample (*n*=409) were analyzed by the researcher on-location at the *W.M. Bass Donated Skeletal Collection*. All skeletal analyses were completed "in the blind" (i.e., after sample selection, the researcher did not consult data on age, sex, obesity, or occupational/habitual activities until the completion of data collection).

For each individual in the sample, left and right acetabula were scored for the seven acetabular variables relevant to age estimation, following the protocol of Rissech and colleagues (2006; Table 2-1; Figures 2-1 through 2-8; Chapter 2). Both female and male acetabula were scored, despite use of a method developed exclusively on males (Rissech et al., 2006). Recent research has shown that patterns of acetabular aging are similar for females and males (San-Millán et al., 2016; 2017), indicating the potential applicability of the original acetabular aging method (Rissech et al., 2006) to individuals of both sexes. Acetabular variables were utilized in other forms of testing than age estimation (e.g., testing individual variables' associations with age, calculating overall CAS for each acetabulum), further arguing for the collection of both female and male data. Acetabula showing macroscopic signs of trauma, surgery, or pathological conditions were included in the sample, in order to address questions of the effect of previous injury/intervention on acetabular changes. However, these pathological acetabula were not used in subsequent age estimation using the method of Rissech and colleagues (2006). All acetabula were photographed.

All major appendicular joints were macroscopically examined (Table 5-2). Both left and right skeletal elements were scored. Skeletal elements showing macroscopic signs of trauma, surgery, or pathological conditions were included in the sample, in order to address questions of the effect of previous injury/intervention on skeletal degeneration. The major joints of the body (including the hip) were scored for the presence and severity of OA following the ordinal scoring system of Jurmain (1990) and the combined-joint protocol of Weiss (2006). Using the Jurmain (1990) ordinal scoring system (Table 5-3 and Figure 5-3), each element implicated in a joint was assigned an individual score (e.g., acetabulum, proximal femur), and then the individual scores were added and divided by the number of available elements in order to achieve a combined joint score (e.g., “left hip” = [left femoral head score + left acetabulum score]/2). Left and right combined joint scores were initially kept separate. However, subsequent Wilcoxon rank-sum testing indicated no statistically significant differences between left and right joint scores for females or males: for females, the largest difference in means was 0.04 (all medians were the same); for males, the largest difference in means was 0.08 (medians were slightly higher for the right elbow and wrist). In 14 Wilcoxon rank-sum tests (significance level, α=0.004 with Bonferroni correction), female *p*-values ranged from 0.42 to 0.74 (Spearman's *rho* values ranged from 0.641 to 0.748), and male *p*-values ranged from 0.09 to 0.90 (*rho* values ranged from 0.571 to 0.774). Thus, left and right joint scores were combined and averaged in accordance with the protocol of Weiss (2006). In the case of missing data (i.e., damaged or missing articular surfaces), fewer surfaces were used in the calculation of the combined joint variable. A sample of joints showcasing the various expressions of OA was photographed.

Traumatic skeletal injuries, pathological conditions, or surgical interventions were noted as present during macroscopic analysis. During the course of data collection, it became clear that the different joints of the pelvis (pubic symphysis, iliac auricular surface, acetabulum) exhibited differential levels of postmortem damage. A simple ordinal scale was designed by the researcher to capture preservational variation in these joints relevant to age estimation (Table 5-4). For a subset of individuals (*n*=292), this scale was used to score the preservation of the left and right acetabula, iliac auricular surfaces, and pubic symphyses (Table 5-4) so that subsequent testing could determine if differences in their preservational condition were statistically significant.

Data Management and Organization

During data collection and analysis, data were stored in separate digital (*Microsoft Excel*) spreadsheets, containing: 1. OA data on appendicular joints (ordinal scores per Jurmain, 1990; brief qualitative descriptions); 2. Acetabulum aging data (ordinal scores per Rissech et al., 2006); 3. Pelvic joint preservation data (ordinal scores described above); 4. Associated demographic information. Spreadsheets were designed prior to the beginning of data collection. No one other than the researcher collected data for this study, and all skeletal elements were examined in person.

Individuals were tracked by their assigned, anonymized *Bass Collection* numbers throughout data collection, management, and analysis. Photographs were loaded from digital cards to a laptop daily during data collection and regularly saved to an external hard drive. Spreadsheets were backed up daily to an external hard drive during data collection and analysis.

Data Analysis

Coding

Subsequent to data collection, demographic data were reintroduced. Documented ages and biological sexes were re-associated with each individual in the sample. Documented height and body mass data were used to calculate BMI, using the following formula: BMI=mass [kg]/height [m]^2. Resulting BMI values were then categorized according to National Institutes of Health standards current as of 2017: obese ≥30; overweight 25-29.9; normal-weight 18.5-24.9; underweight <18.5 (Table 5-5).

Using the Bayesian IDADE2 software (Rissech et al., 2006), acetabular age estimates were calculated and added to the acetabular aging spreadsheet. Pathological, post-traumatic, and post-surgical acetabula were scored for use in analyses of trauma and tests of the five research hypotheses, but these individuals were omitted from the age estimation component of Hypothesis 2a. Only non-pathological left acetabula were utilized for age estimation, with right acetabula substituted in cases of pathological conditions, previous trauma or surgery, or extreme postmortem damage to the left side (per Rissech et al., 2006). Female as well as male ages were estimated despite the original development of the Rissech and colleagues (2006) method exclusively on males, in light of recent research suggesting that patterns of acetabular aging are similar for both sexes (San-Millán et al., 2016; 2017). However, female and male ages were estimated separately, against sex-specific reference distributions, in accordance with the recommendations of San-Millán and colleagues (2017). At this time, reference distributions of acetabular scores from multiple previously analyzed populations are not available to the user of IDADE2 (see Chapter

2). Thus, in order to obtain relevant prior distributions of acetabular variable scores, the female and male samples were arranged by known ages and randomly divided in half, with approximately equal numbers of individuals from all age categories represented in each half of the sample. The acetabular scores from one half of the sample were then used to estimate age in the remaining half of the sample. While the application of a prior distribution drawn from the same population as the test sample is tautological and far from ideal, this was a way to simulate the use of data from a relevant independent population. For each individual in the test sample, the IDADE2 program generated a point age estimate and a confidence interval bounded by the youngest and oldest ages in the age groups comprising the central 95% of the estimating distribution.

In order to enable parametric, multivariate statistical testing of normally distributed acetabular variable scores, the ordinal scores were transformed into a CAS for each individual (Mays, 2012). Acetabular variable scores (which can range from 0-3 to 0-7 in the system of Rissech et al., 2006) were transformed into a common scale of 0-10, so that each variable would be weighted equally. For example, if a variable received a score of 1 on a scale of 0-3, this was transformed to 3.33 (see Mays, 2012). Transformed scores were summed and averaged to achieve a CAS for each individual, and these were added to the acetabular aging spreadsheet.

All occupational and habitual physical activities documented for the individuals in the sample were assigned a metabolic equivalent (MET) intensity level using the activity codes and MET values listed in the Compendium of Physical Activities (Ainsworth et al., 2011). A MET value consists of the measured or estimated metabolic rate of a given activity divided by the resting metabolic rate for quiet sitting, classified as 1 MET

(Ainsworth et al., 2000; Tudor-Locke et al., 2009). In this classification system, a 3-MET activity involves three times the energy expenditure of a 1-MET activity. For example, outdoor construction work is estimated as a 4-MET activity, and conducting seated office work (e.g., computer typing) is estimated as a 1.5-MET activity (Ainsworth et al., 2011). The current version of the Compendium of Physical Activities contains MET values for 821 activities, many of which were obtained from published measurements (Ainsworth et al., 2011). Within the Compendium, activities are assigned a specific descriptive code and MET value, but they can also be more broadly classified as light-intensity (<3 METs), moderate-intensity (3-6 METs), and vigorous-intensity (>6 METs).

The Compendium is typically used to assign standardized codes to the physical activities self-reported by survey participants, in order to facilitate comparison among studies. The activity and occupation data recorded for a subsample of the individuals in the *Bass Collection* were sometimes quite specific (e.g., describing a specific sport or musical instrument played). In these cases, individual activities and occupations were assigned to specific Compendium categories. Even when reported activities were less specific (e.g., reporting “exercise” rather than a specific sport), they were sufficient to allow coding using the modified protocol of Tudor-Locke and colleagues (2009). Using this system, all associated example activities sharing similar 6-digit Compendium codes were averaged to create an estimated MET value for the overall category (Tudor-Locke et al., 2009). For example, if a sample individual listed “walking” as a habitual physical activity, rather than forcing a categorization into one of the more than 50 walking-related activities listed in the Compendium (e.g., household walking, 2 MET; walking the dog, 3 MET), all walking-related MET values were averaged, and the activity was assigned the

resulting value (Tudor-Locke et al., 2009). If an individual listed more than one activity, the aggregate MET values for each listed activity were averaged to create an overall estimated MET level for that individual. This coding system was applied to both reported occupations and habitual activities (Tables 5-6 and 5-7). When listed activities/occupations could not be coded using the Compendium (e.g., unemployment, time served in prison), they were not scored or used in further testing. In order to enable an assessment of the impact of strenuous activities utilizing the lower limb, habitual physical activities involving the lower limb (e.g., running, wrestling) were noted during scoring so that they could be tested separately.

Finally, before the commencement of statistical testing, all qualitative data captured in the spreadsheets were transformed to ordinal or categorical scores. For example, noted instances of skeletal trauma, pathological conditions, or surgical interventions were scored as "1" for "present." Ultimately, all data were moved into a single master spreadsheet for testing.

Testing

Individual acetabular scores, acetabular age estimates and confidence intervals (Rissech et al., 2006), transformed CAS (Mays, 2012), and OA scores (Jurmain, 1990) were used in statistical testing, in addition to trauma and preservation scores and the demographic data reincorporated into analyses after the completion of "blind" data collection (i.e., sex, age, activity MET level, BMI). The seven individual variables assessed for each acetabulum (per Rissech et al., 2006) were used for multiple purposes. The raw acetabular variable scores were used to generate an age estimate for each individual (Rissech et al., 2006), tested for correspondence with documented age (Hypothesis 2a). The individual variables themselves were tested for associations

with OA, age, activity, and BMI (Hypotheses 1, 2a, 3a, and 4a). Finally, the individual variable scores were transformed, summed, and averaged to create CAS that approximated continuous data and were normally distributed, allowing regression analysis in Hypotheses 1, 2a, 3a, 4a, and 5a (see below). Likewise, the OA scores were used in the analyses of individual joints (as ordinal data for Hypotheses 1, 2b, 3b, and 4b) and averaged to achieve overall, upper limb, and lower limb OA scores (which approximated continuous data and were normally distributed, useful for regression analysis in Hypotheses 1, 2b, 3b, 4b, and 5b).

These various types of scores and codes comprised ordinal, categorical, and continuous data. Ordinal data included: raw acetabulum scores; raw OA scores; and preservation scores. Categorical presence/absence data included trauma and pathology scores. Continuous, numeric data included: age, CAS, BMI, activity MET level, and overall, upper limb, and lower limb OA scores. It should be noted that while "at-last-birthday" ages represent discrete categories, they were treated as continuous because the underlying concept of biological age is indeed continuous. Similarly, CAS and overall, upper limb, and lower limb OA scores approximated continuity when the ordinal scores on which they were based were standardized and averaged.

All statistical analyses were conducted using *R* (*R* Core Team, 2015). In general, simpler statistical tests were preferred to more complex ones. Where ordinal scores were assessed, non-parametric statistical tests were used in order to accommodate the non-normality of the data (e.g., investigating whether acetabular changes correlate with OA; Hypothesis 1). Where continuous and normally distributed data were assessed, parametric statistical tests were used (e.g., isolating the variable

exerting the greatest influence on CAS; Hypothesis 5a). Testing mostly relied on frequentist statistics (e.g., correlation tests, significance tests); however, Bayesian inference was utilized in estimating ages based on acetabular changes. Statistical testing included both univariate analysis (e.g., correlation tests) and multivariate analysis (e.g., multiple regression). In tests of statistical significance, results were considered significant when they fell below the p=0.05 threshold. However, when multiple iterations of significance or correlation tests were run (e.g., Wilcoxon rank-sum tests), the Bonferroni correction was applied. In these cases, the α-level of 0.05 was divided by the number of test iterations in order to render the significance level appropriate to the scale of the testing. It should also be noted that the *R* function used to run the Wilcoxon rank-sum tests computed an exact *p*-value when samples contained less than 50 finite values and there were no ties; when ties in data ranking were present, a normal approximation was used. For each analysis, combined-sex and sex-specific tests were conducted, in order to enable comparisons between females and males.

In general, the below testing procedures was followed. For Hypotheses 1 through 4, the relationships between the variables were first examined visually, using box-and-whisker plots (for ordinal variables) and scatterplots (for continuous variables). These relationships were further investigated using Spearman's rank-order correlation tests, in which correlation values (*rho*) can take any value between -1 and +1 (where a *rho* value of +1 would indicate a perfect positive correlation and a value of -1 would indicate a perfect negative correlation). Finally, the statistical significance of any correlation was investigated. For ordinal variables, Spearman's tests calculated *p*-

values using the conditional null distribution of the test statistic (asymptotic approximation of the exact distribution), with ties in rank resolved using mid-ranks. For continuous variables, linear regression assessed the statistical significance of the relationships (*p*-values and adjusted r^2 values).

For the component of Hypothesis 1 testing the accuracy of acetabular age estimates, the Bayesian IDADE2 software was used to predict age based on the acetabular variables (Rissech et al., 2006). For the IDADE2 results, measures of aging error were generated based on comparisons between known ages, estimated point estimates of age, and 95% confidence intervals generated from the estimating distribution. These measures of aging error included: percentage of individuals correctly classified by the 95% confidence intervals; percentage of prediction errors (based on known and estimated ages); inaccuracy (the sum of the absolute differences between known and estimated age divided by sample size); bias (the sum of estimated age minus actual age divided by sample size); maximum over- and under-estimation of known ages based on point estimates of age; and *rho* values for correlations between known ages and estimated point estimates of age (tested for statistical significance using asymptotic Spearman tests). The IDADE2 program also generated a variable called "fit"—a measure of age estimation accuracy calculated as the sum over age classes of the minimum number of years that must be added to or subtracted from the known age to get an age that fell into that age class, times the probability of that age class (Rissech et al., 2006). In order to test the stability of the age-estimation model, ten additional test samples of females (*n*=60) and ten additional test samples of males (*n*=62) were randomly selected in which all age classes were evenly represented.

Then, IDADE2 was used to estimate age in these subsamples using the remainder of the females (*n*=135) and males (*n*=147) as reference samples. Comparisons of mean "fit" values from each of the 20 IDADE2 iterations with 95% confidence intervals generated from bootstrapped mean estimates from each run (with each run resampled 10,000 times) indicated whether the model yielded similar errors across all runs.

For Hypothesis 5, multiple regression was used to isolate the most important factors contributing to acetabular changes and OA. First, combined linear models were constructed including all contributing factors, along with their interaction effects. These full models were then simplified via automated backwards-stepwise selection informed by Akaike's Information Criterion (AIC) in order to acieve the minimal adequate models.

In the ancillary analysis of the effects of trauma/surgery on acetabular changes and OA, repeated resampling was used to test whether observed median OA/acetabular variable scores for affected joints consistently exceeded median scores for 10,000 random, equivalent-sized samples of unaffected joint scores. Finally, Wilcoxon rank-sum tests were used to test for statistically significant differences between the means for pelvic joint preservation scores and between females and males for each variable. Where statistically significant sex differences were indicated, sex-specific results were reported. Where no statistically significant differences were indicated, combined-sex results were reported.

Study Limitations

Ancestral Diversity in the *Bass Collection*

Any donated skeletal collection necessarily has gaps in documentation. The *Bass Collection* only includes information on height, body mass, and occupational/habitual activities for approximately 700 individuals. Additionally, although

the collection includes individuals of various ancestries (including African-American, Asian-American, and Native-American), most of the completely documented individuals are of European-American ancestry. For example, of the over 1700 individuals curated in the *Bass Collection*, fewer than 100 are of African-American descent. Even fewer of these individuals are associated with complete documentary data. Fewer still identified as members of other ancestral or ethnic groups (e.g., Asian-American, Native-American). Because this disparity in representation and documentation precluded the assemblage of sufficient sample sizes for non-European individuals, all individuals in the current skeletal sample are of European-American descent. Unfortunately, this precludes an analysis of the effects of ancestry—a factor linked with heredity that undoubtedly contributes both to OA and to age-related skeletal changes. However, acetabular age estimation research has in its preliminary studies focused almost entirely on individuals of European ancestry (e.g., Calce, 2011; Mays, 2012; Rissech et al., 2006; 2007; San-Millán et al., 2016), and therefore the ancestral makeup of this skeletal sample is appropriate to the capabilities of the age estimation methods, as they are currently understood.

Obesity and BMI

The use of BMI to approximate obesity has been criticized (Smalley et al., 1990; Wellens et al., 1996). The measure merely describes the relationship of height to body mass; it does not measure an individual's percent body fat, differentiate adipose tissue from muscle, bone, or organs, or describe an individual's abdominal girth. A muscular athlete may have a high BMI but a low percent body fat, for example; conversely, an older individual may have a higher percent body fat than a younger individual with the same BMI. Body fat proportions may also differ among individuals of ancestries (Rush

et al., 2009; Wang et al., 1994) and biological sexes (Smalley et al., 1990). Thus, BMI may not be sensitive enough to identify the dangerous levels of adiposity that have been correlated with morbidity and mortality in the obese (Wellens et al., 1996)—at least in non-European populations, for whom relationships between BMI and adiposity have not been sufficiently established (Wang et al., 1994).

However, methods that do directly measure or approximate body fat composition (e.g., underwater body density studies) are often time-consuming and expensive (Wellens et al., 1996). Calculating BMI remains a conveniently simple approximation for adiposity (Keys et al., 1972), and while imperfect, its lower and upper limits likely do capture extremes of adiposity (i.e., underweight, BMI <18.5; obese, BMI ≥30). More accurate methods of measuring adiposity are impractical when dealing with deceased individuals, and impossible when analyzing with skeletal remains. Finally, the lack of ancestral diversity in the development and testing of BMI equations is not a factor in the current study. Percent body fat is highly correlated with BMI in European-American populations (Flegal et al., 2010), and this sample is comprised exclusively of European-American individuals. Thus, BMI is an indirect indicator of adiposity valid for use this study.

Error

A brief discussion of the possibility for random and systematic error in this research is warranted. The researcher could have introduced random human error during data collection (e.g., by transposing numbers during ordinal scoring). However, the potential for this error was mitigated by standardized data collection procedures, standardized spreadsheets with clearly marked individual identifiers and joint labels, and the regular checking of data input. Potential problems with the documentation of

the skeletal collection (e.g., labeling errors, incorrectly attributed demographic information) represent another form of human error that cannot be mitigated in this study. However, it is hoped that the relatively large sample size results in sufficient data points to minimize this type of error.

Any systematic error in this research is largely a byproduct of the variability of the human processes of aging (see Chapter 2) and OA development (see Chapter 4). Simply stated, condensing variable biological processes into simplified ordinal scores necessarily glosses over the complexity of these processes. Thus, the resolution of the current analyses may not be fine-grained enough to identify some of the subtler biological connections between the processes of aging, degeneration, and acetabular change. Developing methods that employ continuous variables to capture degenerative and other age-related changes may account for some of this variability. While this is indeed an avenue the researcher hopes to pursue in the future (see Chapter 7), it is outside the scope of the current study.

Summary

This chapter described the current skeletal sample (*n*=409 documented skeletal individuals of European-American ancestry), culled from the University of Tennessee's *William M. Bass Donated Skeletal Collection*. Five research hypotheses were presented: Hypothesis 1, that acetabular changes correlate positively with OA; Hypotheses 2a and 2b, that acetabular changes and OA correlate positively with age; Hypotheses 3a and 3b, that acetabular changes and OA correlate positively with activity; Hypotheses 4a and 4b, that acetabular changes and OA correlate positively with obesity; Hypotheses 5a and 5b, that age is the most important contributing factor to acetabular changes and OA. Methods of data collection and management were

outlined, including the application of ordinal scoring systems relevant to acetabular age estimation and the analysis of OA in multiple joints throughout the body. Analytical methods were discussed, including a Bayesian method for estimating age in the acetabulum (Rissech et al., 2006) and frequentist methods for assessing the statistical significance of associations between acetabular changes, OA, and their various potential contributing factors (e.g., correlation tests, linear regression). Finally, limitations of the current research were considered. The next chapter presents the analytical results of the current study.

Table 5-1. Total sample of European-American adults analyzed in the current study, arranged by age group, with mean and median ages.

Sex	Age group	*n*	Mean age	Median age
Female	20-29	3	27	27
	30-39	14	35.7	36.5
	40-49	33	45.3	46
	50-59	33	54.7	55
	60-69	33	64.8	66
	70-79	33	74.7	75
	80-89	33	83.9	84
	90+	16	92.4	92
Female total		198	64.3	65.5
Male	19-29	10	24.3	25.5
	30-39	32	34.9	35
	40-49	34	45.4	45.5
	50-59	31	54.5	55
	60-69	32	64.2	65
	70-79	33	73.9	74
	80-89	33	83.5	83
	90+	6	94.7	95
Male total		211	58.8	59
TOTAL		409	61.5	61

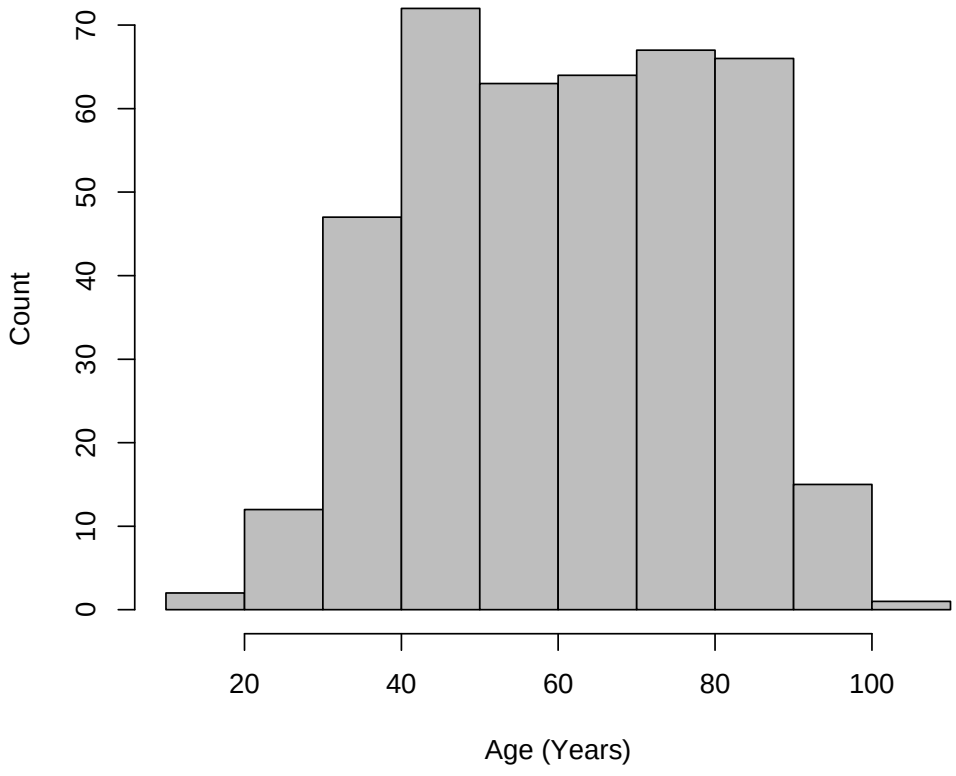
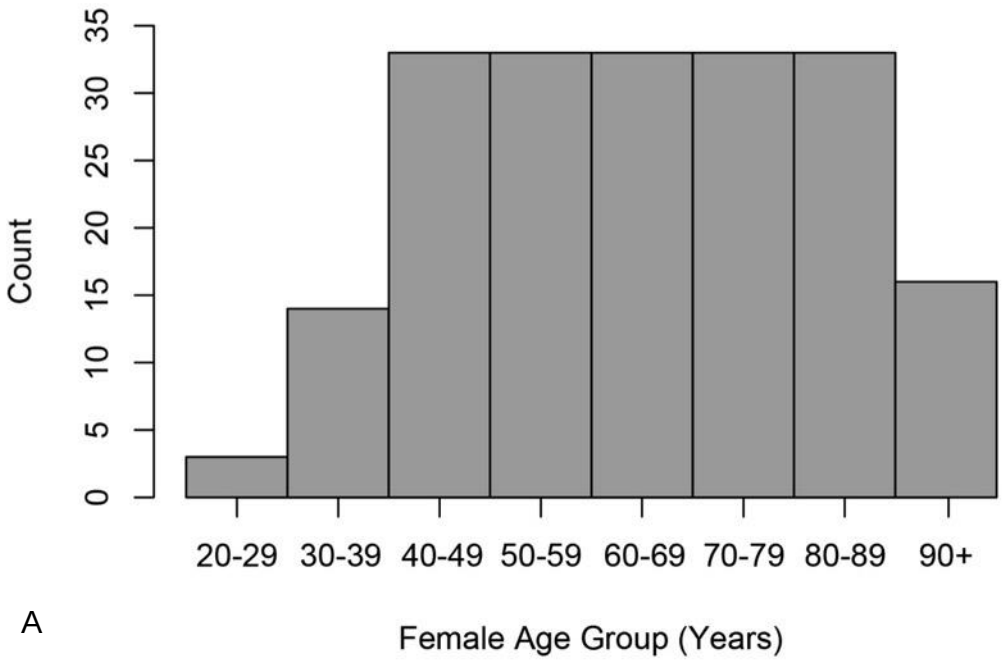
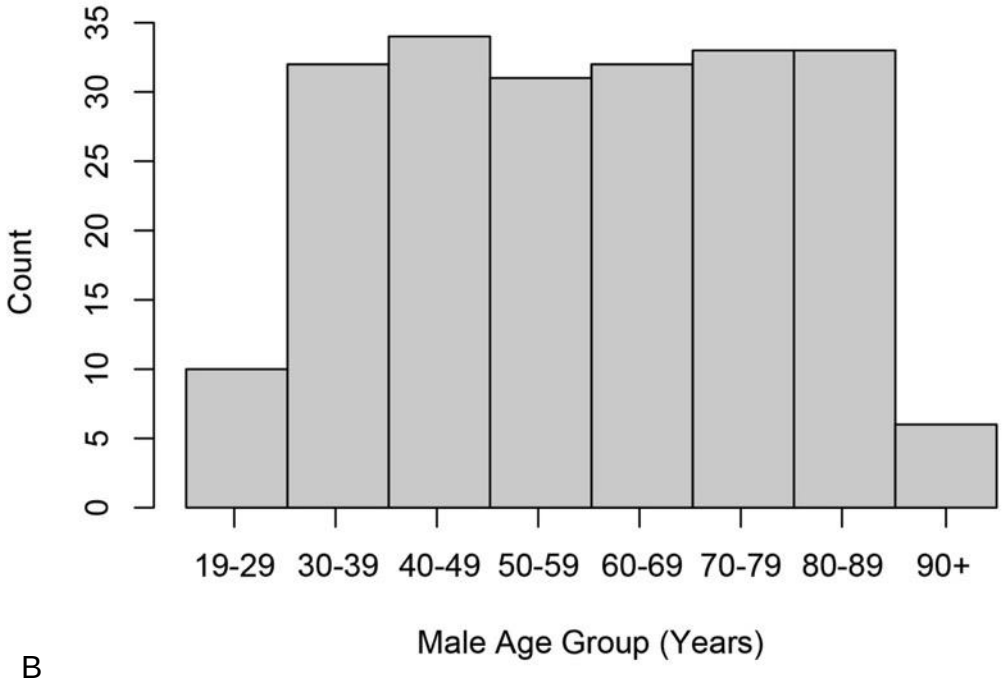


Figure 5-1. Age distribution of sample (*n*=409).



A



B

Figure 5-2. Sample distribution by age group and sex. A) Females, B) Males.

Table 5-2. List of joints and skeletal elements examined for the current analyses of acetabular changes (per Rissech et al., 2006) and OA (per Jurmain, 1990).

Joint	Element	Type of analysis
Temporomandibular joint (TMJ)	Mandibular fossa	OA
	Mandibular condyle	OA
Shoulder	Scapular glenoid	OA
	Proximal humerus	OA
Elbow	Distal humerus	OA
	Proximal radius	OA
	Proximal ulna	OA
Wrist	Distal radius	OA
	Distal ulna	OA
	Scaphoid	OA
	Lunate	OA
Hand	Based on presence/absence of OA in any hand element, per Jurmain (1990)	OA (not used in testing due to different scoring criteria)
Hip	Acetabulum	Acetabular aging, OA
	Proximal femur	OA
Knee	Distal femur	OA
	Patella	OA
	Proximal tibia	OA
Ankle	Distal tibia	OA
	Distal fibula	OA
	Talus	OA
Foot	Based on presence/absence of OA in any foot element, per Jurmain (1990)	OA (not used in testing due to different scoring criteria)

Table 5-3. Osteoarthritis scoring criteria adapted from Jurmain (1990, p. 85).

Appendicular OA
0 none/slight
1 moderate (small osteophyte; and/or pitting over <10% of articular surface)
2 severe (very large osteophyte remodeled and concave with original surface; and/or pitting over >10% of articular surface; or any evidence of eburnation)
3 ankylosis (joint fusion)

Figure 5-3. Images of the acetabulum (left) and distal femur (right), corresponding with OA scores 0, 1, and 2 in the Jurmain (1990) OA scoring system. Photograph by A.P. Winburn.

Table 5-4. Scoring criteria designed by the researcher to capture the preservational condition of the pelvic joints frequently used for age estimation (pubic symphysis, iliac auricular surface, acetabulum).

Degree of postmortem damage
0 none
1 slight postmortem damage (most/all variables observable)
2 extreme postmortem damage (most variables obscured)
3 joint surface destroyed or completely obscured by postmortem damage (no scoring possible)

Table 5-5. Sample sizes (*n*) for the various BMI categories.

Sex	Underweight (<18.5)	Normal weight (18.5-24.9)	Overweight (25-29.9)	Obese (≥30)
Female	20	68	38	61
Male	10	68	62	48
TOTAL	30	136	100	109

Table 5-6. Sample sizes (*n*) for the various MET categories for occupational activities.

Sex	Light intensity (<3 METs)	Moderate intensity (3-6 METs)	Vigorous intensity (>6 METs)
Female	95	83	0
Male	67	122	3
TOTAL	162	205	3

Table 5-7. Sample sizes (*n*) for the various MET categories for habitual activities.

Sex	Light intensity (<3 METs)	Moderate intensity (3-6 METs)	Vigorous intensity (>6 METs)
Female	35	27	9
Male	20	41	8
TOTAL	55	68	17

CHAPTER 6
RESULTS

Sample sizes for each of the variables tested in the below analyses differed based on availability of demographic data and preservation of the observed skeletal elements. Sample sizes for each variable are listed in Table 6-1.

Preliminary statistical analyses (Wilcoxon rank-sum tests) indicated no statistically significant differences for left and right CAS (p>0.25) and acetabular variables (p>0.02; α=0.007; Bonferroni correction: α=0.05/7) for either sex. Spearman's rank-order correlation tests also indicated left and right scores to be highly correlated (CAS: female *rho*=0.849, male *rho*=0.864; acetabular variables: *rho* values ranged from 0.608 to 0.762 for females and from 0.592 to 0.793 for males). Thus, left and right CAS were averaged to create an overall CAS for each individual. This averaging was deemed appropriate for a summary measure of acetabular changes, akin to the combined-joint OA scores averaged according to the protocol of Weiss (2006; see Chapter 5). This also created an approximation of continuous, normally distributed data for these variables. Rather than averaging the acetabular variables, only left-side variables were used in the below analyses (with right-side variables substituted in cases of damaged or pathological acetabula), following the protocol of Rissech and colleagues (2006). This preserved the original, ordinal nature of these individual acetabular variable data.

Wilcoxon rank-sum tests revealed statistically significant differences between the means for female and male CAS (p=0.01) and female and male MET values for occupations (p=0.001). Female and male OA score means also differed significantly in several joints (seven Wilcoxon rank-sum tests; α=0.007 with Bonferroni correction):

females exhibited more OA in the TMJ (p=0.004) and knee (p=0.003); males exhibited more OA in the ankle (p<0.001). Sex differences in hip OA also approached statistical significance, with females exhibiting more hip OA (p=0.007). Because of these significant differences, tests of CAS, MET values for occupations, and TMJ, knee, ankle, and hip OA data utilized separate female and male analyses. Sex-specific results are presented separately, below. Wilcoxon rank-sum tests revealed no statistically significant differences in the female and male means of: acetabular variables (α=0.007; Bonferroni correction: α=0.05/7); overall, upper limb, and lower limb OA; MET values for habitual activities; or BMI values, so combined-sex analyses were undertaken. For these data, combined-sex results are presented below, except where comparisons involve distributions that show sex differences (e.g., OA scores).

Results of Hypothesis Testing

Hypothesis 1: Acetabular Changes Correlate Positively with OA

CAS. Box-and-whisker plots and scatterplots were used to visualize the relationships between CAS and OA in the various joints in females and males (Figures 6-1 through 6-10). In boxplots, the boxes display the median and first and third quartiles for CAS per OA score in each of the major joints. For both females and males, this visual assessment of the data suggested a general trend of increasing CAS with increasing OA in the shoulder, elbow, wrist, hip, and knee (Figures 6-1, 6-2, 6-4, and 6-5 [females] and Figures 6-6, 6-7, 6-9, and 6-10 [males]). Hip OA in particular appeared to show a positive association with CAS (Figures 6-4 and 6-9). However, for all plots, there was considerable overlap in the dispersion of values per OA score, and TMJ and ankle OA appeared to have a more ambiguous relationship with CAS. For both females and males, the box-and-whisker plots for CAS and wrist OA appeared relatively

complex due to the number of possible scores available (*n*=31 for females; *n*=36 for males). As the wrist OA scores approximated continuous data, these relationships were also graphed as scatterplots fitted with linear regression lines, showing statistically significant positive associations with CAS (*p*=4.35e-15 and r^2=0.267 in females; *p*<2e-16 and r^2=0.315 in males; Figures 6-3 and 6-8).

These visual assessments were further investigated using Spearman's rank-order correlation. Correlation values (*rho*) were computed for each joint and CAS in females and males (Table 6-2). *Rho* values for all joints were greater than zero, and asymptotic Spearman tests indicated that positive correlations with female and male CAS were statistically significant in all joints (*p*<6.184e-07; α=0.007; Bonferroni correction: α=0.05/7; Table 6-2).

Scatterplots were used to visualize relationships between female and male CAS and the female and male scores for overall OA, upper limb OA, and lower limb OA (Figures 6-11 and 6-12). Spearman's rank-order correlation values (*rho*) were greater than zero for all associations between CAS and overall OA, upper limb OA, and lower limb OA (Table 6-3). Linear regression indicated that positive associations with female and male CAS were statistically significant in all regions (*p*<0.001; *α*=0.02; Bonferroni correction: *α*=0.05/3; Table 6-4). The data seemed to conform moderately well to linear models—particularly in males (Figure 6-12). For both sexes, r^2 values were highest in the CAS-overall OA models (female r^2=0.45; male r^2=0.62; Figures 6-11 and 6-12).

Acetabular variables. Sex-specific acetabular variable associations were assessed for those joints with sex differences in OA: the TMJ, knee, ankle, and hip (while the latter only approached significant sex differences, the sexes were still

analyzed separately). Combined-sex analyses were undertaken for all other joints. Box-and-whisker plots were used to visualize relationships between the acetabular variables and OA in the individual joints of the body (Figures 6-13 through 6-33). The relationships between the acetabular variables and overall, upper limb, and lower limb OA were also visually assessed using box-and-whisker plots (Figures 6-34 through 6-40). In all plots, boxes display the median and first and third quartiles for OA score per acetabular variable score. The individual-joint comparison plots appeared to show weak positive associations between all seven acetabular variables and OA, with much overlap in the dispersion of values (Figures 6-13 through 6-33). For some joints (e.g., the hip), the positive relationship appeared to be stronger (Figures 6-14, 6-17, 6-20, 6-23, 6-26, 6-29, and 6-32). The plots depicting the relationships between the acetabular variables and overall, upper limb, and lower limb OA also appeared to show positive associations between all variables and OA, albeit with much overlap (Figures 6-34 through 6-40). Associations seemed to be slightly weaker in Variables 6 and 7, with more overlap in the dispersion of values (Figures 6-39 and 6-40).

This visual assessment was further investigated using Spearman's rank-order correlation. *Rho* values were computed for each acetabular variable with OA in each of the joints (Tables 6-5 through 6-11) and with overall, upper limb, and lower limb OA (Table 6-12). All *rho* values for the acetabular variables were greater than zero (Tables 6-5 through 6-12). In the individual-joint comparisons, asymptotic Spearman tests indicated that all acetabular variables had statistically significant positive correlations with OA in the female and male hip, the male knee, and the combined shoulder, elbow, and wrist (α=0.0007; Bonferroni correction: α=0.05/70; Tables 6-5 through 6-11). All

except Variables 3 and 7 had statistically significant positive correlations with female knee OA and male ankle OA (Tables 6-7 and 6-11). Only Variables 1 and 3 had statistically significant positive correlations with female and male TMJ, and only Variable 1 had a statistically significant positive correlation with female ankle OA (Tables 6-5 and 6-7). In the summary OA comparisons, all acetabular variables had statistically significant positive correlations with overall, upper limb, and lower limb OA (p<2.85e-11; α=0.002; Bonferroni correction: α=0.05/21; Table 6-12).

Hypothesis 2: Age

Hypothesis 2a: Acetabular changes correlate positively with age

Age estimates. Ages were estimated using IDADE2 (Rissech et al., 2006) for randomly selected female (n=97) and male (n=104) subgroups of the overall skeletal samples, using the remainder of the female and male samples as the reference distributions (Table 6-13). Because error rates have been shown to differ between younger and older adult age estimates (Aykroyd et al., 1999; Nawrocki, 2010; Winburn and Brown, 2010; 2011), the female and male subsamples were divided at the approximate median age (60 years), and error rates were generated for individuals <60 years and 60+ years, in addition to individuals of all ages (Table 6-13). These measures of error included: percent correctly classified; percent prediction error; inaccuracy; bias; maximum over- and under-estimation; and *rho* values for the associations between estimated and documented ages (Table 6-13).

In females, age estimates for the <60-year group showed no statistically significant correlation with known ages, while age estimates for the combined-age and 60+ females were statistically significantly correlated with known ages (p<0.011; α=0.02; Bonferroni correction: α=0.05/3; Table 6-13). Likewise, the lowest percentage

of correctly classified females was observed in the <60-year age group (44%, compared with 60% in the combined-age group and 72% in the 60+ group). Similarly, the highest female percentage prediction error was seen in <60-year females (36%, compared with 24% in the combined-age group and 16% in the 60+ group). Bias values indicated a tendency to overestimate age in younger females (by 13.4 years) and underestimate age in older females (by 6.8 years); the positive and negative biases of these groups virtually cancelled each other out in the measure of bias for combined-age females (1.3 years). Finally, inaccuracy values were similar for the three groups (<60 females: 16.1 years; 60+ females: 12.0 years; combined-age females: 13.6 years), and full ranges of error were always large (<60 females: -17.4 to 38.5 years; 60+ females: -34 to 26 years; combined-age females: -34 to 38.5 years).

In males, age estimates for all three groups showed statistically significant correlations with known ages (p<0.006; α=0.02; Bonferroni correction: α=0.05/3; Table 6-13). Percentages of correctly classified individuals were similar in the three age groups, ranging from 60% in males <60 years to 66% in males 60+ years. As in females, however, percentage prediction error was highest in the <60-year males (30%, compared with 23% in combined-age males and 16% in 60+ males). Also as in females, bias values indicated a tendency to overestimate age in younger males (by 7.8 years) and underestimate age in older males (by 9.7 years); the positive and negative biases of these groups virtually cancelled each other out in the measure of bias for combined-age males (-1.1 years). Finally, inaccuracy values were nearly identical for the three groups (ranging from 12.3 to 12.6 years), and full ranges of error were always

large (<60 males: -17 to 37.6 years; 60+ males: -42 to 10.7 years; combined-age males: -42 to 37.6 years).

The "fit" variables produced by IDADE2 indicated that age estimation inaccuracy was generally lower in males than in females—sometimes statistically significantly so, even when α-levels were adjusted for 100 repetitions of Wilcoxon rank-sum tests (α=0.0005 with Bonferroni correction). In the ten randomly selected female and male test samples, female "fit" means ranged from 14.06 to 16.90, and male "fit" means ranged from 11.87 to 14.24. In terms of model stability, mean "fit" values from each of the ten female and male test samples typically fell within the 95% confidence intervals generated from bootstrapped mean estimates from all other iterations. Exceptions included: four cases in which female means were higher than the 95% confidence intervals generated for other runs; three cases in which female means were lower; four cases in which male means were higher than the 95% confidence intervals generated for other runs; and two cases in which male means were lower.

CAS. Scatterplots were used to visualize relationships between female and male CAS and the documented ages of the sampled individuals (Figures 6-41 and 6-42). *Rho* values were computed for the associations between age and each acetabular variable (Table 6-14). Spearman's rank-order correlation values (*rho*) were greater than zero in both females and males (Table 6-14), and linear regression indicated significant positive associations between age and CAS for both sexes (p<0.001; Table 6-15). The data seemed to conform moderately well to linear models—particularly in males (female r^2=0.34; male r^2=0.52; Figures 6-41 and 6-42).

Acetabular variables. Box-and-whisker plots were used to visualize relationships between each of the left acetabular variables and the documented ages of the sampled individuals (Figures 6-43 and 6-44). In these plots, boxes display the median age and first and third quartiles for age per acetabular variable score. All plots appeared to reveal a general trend of increasing age with increasing acetabular variable score (Figures 6-43 and 6-44). However, there was some overlap in the dispersion of values per acetabular variable score, and the positive relationship between age and score seemed to be less clear for Variable 6 (Figure 6-44).

This visual assessment was further investigated using Spearman's rank-order correlation. *Rho* values were computed for the associations between age and each acetabular variable (Table 6-16). *Rho* values for age and all seven acetabular variables were greater than zero, and asymptotic Spearman tests indicated statistically significant positive age correlations for all seven acetabular variables (p<5.58e-12; α=0.007; Bonferroni correction: α=0.05/7; Table 6-16).

Hypothesis 2b: OA correlates positively with age

Sex-specific age associations were assessed for those joints with sex differences in OA: the TMJ, knee, ankle, and hip (while the latter only approached significant sex differences, the sexes were still analyzed separately). Combined-sex analyses were undertaken for all other joints. Box-and-whisker plots and scatterplots were used to visualize relationships between OA scores in each of the individual joints and the documented ages of the sampled individuals (Figures 6-45 through 6-49). In the boxplots, boxes display the median age and first and third quartiles for age per OA score. Weak trends toward increasing age with increasing OA score were discernable in the plots for female and male TMJ, female and male knee OA, and combined elbow

and wrist OA (Figures 6-45 through 6-48). Stronger positive associations appeared to be present between age and OA in the combined shoulder and female and male hip (Figures 6-45 and 6-48). Positive associations between age and OA in the female and male ankle appeared to be absent or extremely weak (Figure 6-49). Overlap in the dispersion of values per OA score was present in all plots (Figures 6-45 through 6-49). The box-and-whisker plot for age and wrist OA (Figure 6-46) appeared relatively complex due to the number of possible scores available (n=39). As the combined wrist OA scores approximated continuous data, their relationship with age was also graphed as a scatterplot fitted with a linear regression line, showing a statistically significant positive association (p<2e-16; r^2=0.264; Figure 6-47).

This visual assessment was further investigated using Spearman's rank-order correlation. *Rho* values were computed for the associations between age and OA in each joint (Table 6-17). All *rho* values for age and OA were greater than zero, and asymptotic Spearman tests indicated statistically significant positive correlations between age and OA in all joints except the female ankle (p<6.396e-04; α=0.007; Bonferroni correction: α=0.05/7; Table 6-17).

Finally, scatterplots were used to visualize relationships between overall, upper limb, and lower limb OA and the documented ages of the individuals in the combined-sex sample (Figure 6-50). *Rho* values were computed for the associations between age and OA in each summary region (Table 6-18). All *rho* values for age and summary OA were greater than zero (Table 6-18), and linear regression indicated statistically significant positive associations between age and OA in all three regions (overall, upper limb, and lower limb OA; p<0.001; α=0.02; Bonferroni correction: α=0.05/3; Table 6-19).

A linear model seemed to describe the relationship between age and summary OA moderately well (overall OA r^2=0.40; upper limb OA r^2=0.38; lower limb OA r^2=0.21; Figure 6-50).

Hypothesis 3: Activity

In order to control for age, testing for Hypothesis 3 proceeded within the study's 10-year age groups. However, because of insufficient sample sizes in the youngest and oldest age groups, these were combined with adjacent age groups for testing. In the analyses of MET values for occupation (females and males analyzed separated), this resulted in five age groups tested for females (*n*=30 to 47) and six age groups tested for males (*n*=26 to 38). In the combined-sex sample used in analyses of MET values for habitual physical activities, this resulted in five age groups tested (*n*=22 to 32). In all of the below activity analyses, lower MET values indicate lower-intensity physical activities, and higher MET values indicate higher-intensity physical activities.

Hypothesis 3a: Acetabular changes correlate positively with activity

CAS and occupational activities. Scatterplots were used to visualize relationships between CAS and MET values for occupational activities in females and males of different ages (Figures 6-51 through 6-54). No positive associations were discernable in the scatterplots for females or males of any age group (Figures 6-51 through 6-54). Spearman's *rho* values for most female and male age groups were greater than zero (Table 6-20). However, linear regression indicated that no associations between CAS and occupation were statistically significant for any female or male age group (female p>0.07; male p>0.40; α=0.01 for females [Bonferroni correction: α=0.05/5] and α=0.008 for males [Bonferroni correction: α=0.05/6]; Table 6-21). Further, linear models poorly described all relationships between CAS and

occupation: most female models and all male models were non-significant; in the one female group with a significant linear model, the r^2 value was 0.070 (Figures 6-51 through 6-54).

Acetabular variables and occupational activities. Box-and-whisker plots were used to visualize the relationships between acetabular variable scores and MET values for occupation in females and males of different ages (Figures 6-55 through 6-76). Boxes display the median and first and third quartiles for occupational MET value per acetabular variable score. For both females and males, no clear positive or negative associations were discernable in any age group, and extensive overlap in the dispersion of MET values per acetabular variable score was present in most plots (Figures 6-55 through 6-76).

This visual assessment was further investigated using Spearman's rank-order correlation. *Rho* values were computed for associations between acetabular variable scores and MET values for occupation in females and males of different ages (Table 6-22). For most variables, the majority of *rho* values were less than zero, though some *rho* values (e.g., in Variable 7) were greater than zero (Table 6-22). Asymptotic Spearman tests indicated that no associations between acetabular scores and occupation values were statistically significant for any of the seven variables in females or males of any age group ($p>0.02$; $\alpha=0.001$; female Bonferroni correction: $\alpha=0.05/35$; male Bonferroni correction: $\alpha=0.05/42$; Table 6-22).

CAS and habitual physical activities. Despite statistically significant sex differences present in female and male CAS distributions, habitual physical activities

tests were run within a combined-sex sample, due to the limited sample size with documented data for habitual activities (Table 6-1).

Scatterplots were used to visualize the relationships between CAS and MET values for habitual physical activities in different age groups within the combined-sex sample (Figures 6-77 and 6-78). A possible positive association and Spearman's *rho* value greater than zero were discernable in only one age group (50-59 years; Figure 6-77; Table 6-23). All other age groups exhibited what appeared to be negative associations between CAS and MET values for habitual physical activities, and they had *rho* values less than zero (Figures 6-77 and 6-78; Table 6-23). Linear regression indicated that none of these associations were statistically significant ($p>0.03$; $\alpha=0.01$; Bonferroni correction: $\alpha=0.05/5$; Table 6-24). Further, linear models poorly described the relationships depicted by most scatterplots: some models were non-significant; in the others, r^2 values ranged from 0.005 to 0.144 (Figures 6-77 and 6-78).

Finally, Wilcoxon rank-sum tests discerned no statistically significant differences in CAS means between individuals engaging in strenuous physical activities utilizing the lower limb compared with individuals who did not engage in these rigorous activities ($p>0.188$ for all groups; $\alpha=0.01$; Bonferroni correction: $\alpha=0.05/5$).

Acetabular variables and habitual physical activities. Box-and-whisker plots were used to visualize the relationships between acetabular variable scores and MET values for habitual physical activities in different age groups within the combined-sex sample (Figures 6-79 through 6-88). Boxes display the median and first and third quartiles for habitual activity MET per acetabular variable score. No clear positive or negative associations were discernable in any age group, and extensive overlap in the

dispersion of MET values per acetabular variable score was present in most plots (Figures 6-79 through 6-88). However, possible negative associations with habitual activities were noted in some plots (e.g., Variables 1, 2, and 4 in the <50-year-old age group; Figure 6-79), and possible positive associations were noted in plots of Variable 5 (e.g., in the 50-59-year-old age group; Figure 6-82).

This visual assessment was further investigated using Spearman's rank-order correlation. *Rho* values were computed for associations between acetabular variable scores and MET values for habitual activities in the various age groups (Table 6-25). In most variables, the majority of *rho* values were less than zero, though in some (e.g., Variable 5), *rho* values were greater than zero (Table 6-25). However, asymptotic Spearman tests indicated that no associations between acetabular variable scores and MET values for habitual activities were statistically significant for any of the seven variables in any age group ($p>0.003$; $\alpha=0.001$; Bonferroni correction: $\alpha=0.05/35$; Table 6-25).

Finally, Wilcoxon rank-sum tests discerned no statistically significant differences in acetabular variable means between individuals engaging in strenuous physical activities utilizing the lower limb compared with individuals who did not engage in these rigorous activities ($p>0.01$ for all variables and all groups; $\alpha=0.001$; Bonferroni correction: $\alpha=0.05/35$).

Hypothesis 3b: OA correlates positively with activity

Occupational activities. Box-and-whisker plots were used to visualize relationships between OA scores in each of the individual joints and the MET values for occupational activities in various age groups in the female and male samples (Figures 6-89 through 6-110). Boxes display the median and first and third quartiles for

occupational MET values per OA score. No clear positive or negative associations were discernable in any age group, and extensive overlap in the dispersion of MET values per OA score was present in most plots (Figures 6-89 through 6-110). However, possible positive associations with occupational activities were noted in the plots for male TMJ OA in the 50-59-year age group and male shoulder and elbow OA in the 80+ age group (Figures 6-103 and 6-109); and a possible negative association with occupation was noted in the plot for male TMJ OA in the 70-79-year age group (Figure 6-107).

This visual assessment was further investigated using Spearman's rank-order correlation. *Rho* values were computed for associations between OA scores and MET values for occupational activities in the various age groups of the female and male samples (Table 6-26). In most joints, the majority of age groups exhibited *rho* values greater than zero, though in some (e.g., female and male TMJ), the majority of *rho* values were less than zero (Table 6-26). Asymptotic Spearman tests indicated that no associations between OA and occupation were statistically significant for any of the joints in females or males of any age group ($p>0.004$; $\alpha=0.001$; Bonferroni correction for females: $\alpha=0.05/35$; for males: $\alpha=0.05/42$; Table 6-26).

Scatterplots were used to visualize relationships between overall, upper limb, and lower limb OA and MET values for occupational activities in the various age groups of the female and male samples (Figures 6-111 through 6-121). In the majority of cases, Spearman's *rho* values were greater than zero (Table 6-27), but linear regression indicated than no associations were statistically significant in females or males of any age group ($p>0.01$; $\alpha=0.003$; Bonferroni correction: $\alpha=0.05/15$; Tables 6-

28 and 6-29). In general, linear models did not seem appropriate for describing relationships between occupation MET values and summary OA: some models were non-significant; in others, r^2 values ranged from 6.818e-06 to 0.201 (Figures 6-111 through 6-121).

Habitual physical activities. Despite statistically significant sex differences present in female and male score distributions for TMJ, knee, and ankle OA, habitual physical activities tests were run within a combined-sex sample, due to the limited sample size with documented data for habitual activities (Table 6-1).

Box-and-whisker plots were used to visualize relationships between OA scores in each of the individual joints and MET values for habitual physical activities in combined-sex individuals of the various age groups (Figures 6-122 through 6-131). Boxes display the median and first and third quartiles for habitual activity MET values per OA score. Both negative and positive associations were observed throughout the joints, though some plots displayed no discernable relationship, and marked overlap in the dispersion of MET values per OA score was present in many plots (Figures 6-122 through 6-131). In particular, negative associations were observed for nearly all joints in the <50-year age group (Figures 6-122 and 6-123), and positive associations were observed for nearly all joints in the 50-59-year age group (Figures 6-124 and 6-125).

This visual assessment was further investigated using Spearman's rank-order correlation. *Rho* values were computed for associations between OA scores and MET values for habitual physical activities in the various age groups (Table 6-30). In most joints, the majority of *rho* values were less than zero, but asymptotic Spearman tests indicated that no associations between OA scores and MET values for habitual activities

were statistically significant for any of the joints in any age group (p>0.01; α=0.001; Bonferroni correction: α=0.05/35; Table 6-30).

Scatterplots were used to visualize relationships between overall, upper limb, and lower limb OA and MET values for habitual physical activities in the various age groups of the combined-sex samples (Figures 6-132 through 6-136). Most associations appeared to be negative, with Spearman's *rho* values less than zero (Table 6-31). However, linear regression indicated that no associations between summary OA and habitual physical activities were statistically significant in combined-sex individuals of any age group (p>0.005; α=0.003; Bonferroni correction: α=0.05/15; Table 6-32). Further, all associations were poorly described by linear models: most models were non-significant; in others, r^2 values ranged from 0.009 to 0.011 (Figures 6-134 through 6-136).

Finally, Wilcoxon rank-sum tests discerned no statistically significant differences in hip, knee, or ankle OA score means between individuals engaging in strenuous physical activities utilizing the lower limb compared with individuals who did not engage in these rigorous activities (p>0.06 for all lower-limb joint scores and all groups; α=0.003; Bonferroni correction: α=0.05/15).

Hypothesis 4: Obesity

For most individuals in the sample (n=375), BMI was calculated from reported height and weight data; for a minority of individuals, BMI could also be calculated from measured cadaver weights (n=25). For only 12 of these 25 individuals, BMI category changed when the measured body weight figures were added to the calculation. Wilcoxon rank-sum tests indicated no statistically significant difference between the BMI means (p=0.682), and Spearman's correlation tests indicated that the two distributions

were highly correlated (*rho*=0.963). Thus, the below obesity analyses used reported (rather than measured) BMI, for consistency among the sampled individuals. As in the above activity analyses, obesity tests were run within approximately 10-year age groups, in order to control for age effects. This resulted in five age groups tested for females (*n*=32 to 47), six age groups tested for males (*n*=24 to 36), and six age groups tested for the combined-sex sample (*n*=37 to 83).

Hypothesis 4a: Acetabular changes correlate positively with obesity

CAS. Scatterplots were used to visualize relationships between CAS and BMI values in females and males of different ages (Figures 6-137 through 6-140). Most associations appeared weakly positive (Figures 6-137 through 6-140), but Spearman's *rho* values both less than and greater than zero were observed (Table 6-33). Linear regression indicated that no associations between CAS and BMI were statistically significant in females or males of any age group ($p>0.014$; $\alpha=0.01$ for females [Bonferroni correction: $\alpha=0.05/5$] and $\alpha=0.008$ for males [Bonferroni correction: $\alpha=0.05/6$]; Table 6-34). Further, most of these relationships were poorly described by linear models: some models were non-significant; in others, r^2 values ranged from 0.024 to 0.159 (Figures 6-137 through 6-140).

Acetabular variables. Box-and-whisker plots were used to visualize relationships between acetabular variable scores and BMI values in combined-sex individuals of the various age groups (Figures 6-141 through 6-152). Boxes display the median and first and third quartiles for BMI value per acetabular variable score. Few BMI associations were discernable from the plots, except possible positive associations with Variables 4 and 5 in individuals <40 years (Figures 6-141 and 6-142), a possible positive association with Variable 2 in individuals 40-49 years (Figure 6-143), and a

possible negative association with Variable 7 in individuals 80+ years (Figure 6-152). Marked overlap in the dispersion of BMI values per acetabular variable score was present in many plots (Figures 6-141 through 6-152).

This visual assessment was further investigated using Spearman's rank-order correlation. *Rho* values were computed for associations between acetabular variable scores and BMI values in the various age groups (Table 6-35). The majority of *rho* values were greater than zero (Table 6-35). However, asymptotic Spearman tests indicated that none of the associations between acetabular variables and BMI values were statistically significant in any age group ($p>0.002$; $\alpha=0.001$; Bonferroni correction: $\alpha=0.05/42$; Table 6-35)

Hypothesis 4b: OA correlates positively with obesity

Box-and-whisker plots were used to visualize relationships between OA scores in each of the individual joints and BMI values in females, males, and combined-sex individuals of the various age groups (Figures 6-153 through 6-169). Boxes display the median and first and third quartiles for BMI value per OA score. In females and males, some positive associations were noted with knee and ankle OA (e.g., in females 50-59 years; Figure 6-160), fewer positive associations were noted with hip OA (e.g., in males 50-59 years; Figure 6-159), and some weak negative associations were noted with TMJ OA (e.g., in males<40 years; Figure 6-153). In the combined sample, positive associations were noted with shoulder, elbow, and wrist OA (e.g., shoulder OA in individuals 40-49 years; Figure 6-155). Other plots displayed no discernable relationships, and marked overlap in the dispersion of BMI values per OA score was present in many plots (Figures 6-153 through 6-169).

This visual assessment was further investigated using Spearman's rank-order correlation. *Rho* values were computed for associations between OA scores and BMI values in the various age groups; most were greater than zero (Table 6-36). Asymptotic Spearman tests indicated a statistically significant positive correlation with BMI in the ankle of females <50 years (p=0.001; female/combined-sex α=0.003 [Bonferroni correction: α=0.05/20 and α=0.05/18, respectively]; Table 6-36). A positive correlation in the 40-49-year-old combined shoulder sample also approached statistical significance (p=0.003; Table 6-36). However, it is interesting to note that, had a less-conservative α-level been employed, several additional correlations between BMI and knee and ankle OA would have been statistically significant—particularly in females, for whom p-values were generally lower than male p-values for the same joints (e.g., 50-59-year-old female knee and ankle OA p=0.005 and 0.004, respectively; 50-59-year-old male knee and ankle OA p=0.012 and 0.071, respectively).

Scatterplots were used to visualize relationships between overall, upper limb, and lower limb OA and BMI values in the various age groups of the combined-sex sample (Figures 6-170 through 6-175). All associations appeared to be positive (Figures 6-170 through 6-175), with Spearman's *rho* values greater than zero (Table 6-36). Linear regression indicated that several of these associations were statistically significant (α=0.003; Bonferroni correction: α=0.05/18; Table 6-38). Positive associations between BMI and overall OA were statistically significant in the 50-59-year age group (p=1.88e-04; Table 6-38). Positive associations between BMI and lower limb OA were statistically significant in the <40, 50-59-year, and 70-79-year age groups (p-values ranged from 8.46e-06 to 0.001; Table 6-38). Linear models seemed more

adequate to describe the relationships between lower limb OA and BMI (r^2 values ranged from 0.008 to 0.264) than between overall and particularly upper limb OA and BMI (some models were non-significant; in others, r^2 values ranged from 0.032 to 0.190 and 0.033 to 0.070, respectively; Figures 6-170 through 6-175).

Hypothesis 5: Relative Contributions of Age, Activity, and Obesity

Combined linear models were constructed for the below hypotheses, including all relevant contributing factors and their interaction effects. The full models were then simplified via automated backwards-stepwise selection informed by Akaike's Information Criterion (AIC) in order to acieve the minimal adequate models. The original model and the simplified model are reported for each subsample tested in the below hypotheses.

Hypothesis 5a: Of the above factors, age has the most influence on acetabular changes

Multiple regression was used to assess the influence of age, habitual activities, occupational activities, and BMI on acetabular changes (CAS) in females and males. In the female sample, a linear model was created with age, habitual activities, and BMI as the explanatory variables and CAS as the response variable (r^2=0.18; Table 6-39). When backwards-stepwise, automated model simplification was used to simplifiy the model based on AIC, age was the only remaining term, and it was statistically significant (p=1.99e-05; r^2=0.23; Table 6-40). A similar model was created with the larger sample size available for testing the effects of age, occupational activities, and BMI on female CAS (r^2=0.32; Table 6-41). Again, when backwards-stepwise, automated model simplification was used to simplifiy the model based on AIC, age was the only remaining term, and it was statistically significant (p<2e-16; r^2=0.33; Table 6-42). In the male

sample, a linear model was created with age, habitual activities, and BMI as the explanatory variables and CAS as the response variable (r^2=0.58; Table 6-43). When backwards-stepwise, automated model simplification was used to simplifiy the model based on AIC, age and BMI remained in the model, and both were statistically significant (p<0.01; r^2=0.60; Table 6-44). A similar model was created with the larger sample size available for testing the effects of age, occupational activities, and BMI on male CAS (r^2=0.42; Table 6-45). When backwards-stepwise, automated model simplification was used to simplifiy the model based on AIC, age, BMI, occupation, and the BMI:occupation interaction term remained in the model, but only age was statistically significant (p<2e-16; r^2=0.42; Table 6-46).

Hypothesis 5b: Of the above factors, age has the most influence on OA

Multiple regression was used to assess the relative contributions of age, habitual activities, and BMI to overall, upper limb, and lower limb OA in the combined-sex sample and the contributions of age, occupational activities, and BMI to overall, upper limb, and lower limb OA in females and males.

For the combined sample, a linear model was created with age, habitual activities, and BMI as the explanatory variables and overall OA as the response variable (r^2=0.38; Table 6-47). When backwards-stepwise, automated model simplification was used to simplifiy the model based on AIC, age, BMI, habitual activities, and the BMI:activity interaction term remained in the model, but only age and habitual activities were statistically significant (p<0.05; r^2=0.38; Table 6-48). Next, a combined-sex linear model was created with age, habitual activities, and BMI as the explanatory variables and upper limb OA as the response variable (r^2=0.34; Table 6-49). When backwards-stepwise, automated model simplification was used to simplifiy the model based on AIC,

age, BMI, habitual activities, and the age:activity and BMI:activity interaction terms remained in the model, but only age, activity, and the BMI:activity interaction term were statistically significant (p<0.05; r^2=0.34; Table 6-50). A final combined-sex linear model was created with age, habitual activities, and BMI as the explanatory variables and lower limb OA as the response variable (r^2=0.28; Table 6-51). When backwards-stepwise, automated model simplification was used to simplifiy the model based on AIC, age and BMI remained in the model, and both were statistically significant (p<2.22e-07; r^2=0.28; Table 6-52).

For females, a linear model was created with age, occupational activities, and BMI as the explanatory variables and overall OA as the response variable (r^2=0.41; Table 6-53). When backwards-stepwise, automated model simplification was used to simplifiy the model based on AIC, age, BMI, and occupational activities remained in the model, but only age and BMI were statistically significant (p<0.001; r^2=0.42; Table 6-54). Next, a linear model was created for the female sample with age, occupational activities, and BMI as the explanatory variables and upper limb OA as the response variable (r^2=0.40; Table 6-55). When backwards-stepwise, automated model simplification was used to simplifiy the model based on AIC, age, BMI, and occupational activities remained, and all three were statistically significant (p<0.035; r^2=0.41; Table 6-56). Finally, a linear model was created for the female sample with age, occupational activities, and BMI as the explanatory variables and lower limb OA as the response variable (r^2=0.26; Table 6-57). When backwards-stepwise, automated model simplification was used to simplifiy the model based on AIC, age and BMI remained in the model, and both were statistically significant (p<1.87e-07; r^2=0.27; Table 6-58).

For males, a linear model was created with age, occupational activities, and BMI as the explanatory variables and overall OA as the response variable (r^2=0.39; Table 6-59). When backwards-stepwise, automated model simplification was used to simplifiy the model based on AIC, age, BMI, and occupational activities remained in the model, and all three were statistically significant (p<0.007; r^2=0.40; Table 6-60). Next, a linear model was created for the male sample with age, occupational activities, and BMI as the explanatory variables and upper limb OA as the response variable (r^2=0.38; Table 6-61). When backwards-stepwise, automated model simplification was used to simplifiy the model based on AIC, age, BMI, and occupational activities again remained in the model, and again, all three were statistically significant (p<2.24e-04; r^2=0.39; Table 6-62). Finally, a linear model was created for the male sample with age, occupational activities, and BMI as the explanatory variables and lower limb OA as the response variable (r^2=0.28; Table 6-63). Again, when backwards-stepwise, automated model simplification was used to simplifiy the model based on AIC, age, BMI, and occupational activities remained in the model, and all three were statistically significant (p<0.05; r^2=0.28; Table 6-64).

Ancillary Research Goals

Trauma

In this sample, frequencies of traumatic injury and/or surgical intervention were quite low (Table 6-65). Thus, repeated resampling was used to test whether observed median OA/acetabular variable scores for affected joints consistently exceeded median scores for unaffected joint scores. For each left and right joint in the female and male samples, 10,000 random samples of unaffected joint scores were selected, each of an equivalent size to the sample of affected individuals in that joint. The number of times

that the median scores for these random samples of unaffected individuals exceeded the median scores for affected individuals was calculated and divided by the number of repetitions (10,000). This generated *p*-values indicating the probability that acetabular changes and OA were unaffected by trauma/surgical intervention (Tables 6-70 and 6-71). The possibility that age complicated these analyses (i.e., that increased age led to both increased OA and increased instances of trauma/surgical intervention) could not be investigated in this sample, as observed frequencies of traumatic injuries and surgical interventions were too low to enable comparisons of scores within age-matched sub-groups (Table 6-65). In females, instances of trauma/surgery were highest in the hip, followed by the wrist and knee; in males, instances of trauma/surgery were highest in the knee, followed by the hip and ankle (Table 6-65)

Few analyses of hip trauma/surgery and acetabular variable scores showed statistically significant effects (Table 6-66). For females, Variable 3 (right side) and Variables 6 and 7 (left and right sides) showed statistically significant trauma effects (p=0.000; α=0.003 with Bonferroni correction of 0.05/14; Table 6-66). For males, only Variables 5 (right side) and 7 (left side) showed statistically significant trauma effects (p<0.002; α=0.003 with Bonferroni correction of 0.05/14; Table 6-66).

In many OA analyses, previous instances of trauma/surgical intervention affected OA in the various joints statistically significantly (Table 6-67). In females, previous trauma/surgery had a statistically significant effect on OA scores in approximately half of the tests (p<0.001 for the left shoulder, left elbow, left and right wrist, right hip, and right ankle; α=0.003 with Bonferroni correction of 0.05/14; Table 6-67). In males, previous trauma/surgery had a statistically significant effect on OA scores in over half of the tests

(p<0.002 for the right TMJ, left and right elbow and wrist, left hip, and left and right ankle; α=0.003 with Bonferroni correction of 0.05/14; Table 6-67).

Sex

See the beginning of Chapter 6 for a discussion of sex-based differences in the tested variables. Overall frequencies of OA in females and males appear in Table 6-68. In both females and males, mean OA scores were highest in the hip. In females, frequencies of OA (from highest to lowest) were: hip, knee, shoulder, elbow, TMJ, wrist, and ankle (Table 6-68). In males, frequencies of OA (from highest to lowest) were: hip, shoulder, knee, elbow, wrist, ankle, and TMJ (Table 6-68).

Preservation

Preservation scores for the three pelvic joints were first tested for side, sex, and age differences. Wilcoxon rank-sum tests revealed no statistically significant differences between left and right preservation scores for females (p-values ranged from 0.098 to 0.360) or males (p-values ranged from 0.275 to 0.818), so left and right scores for the pubic symphyses, auricular surfaces, and acetabula were averaged for each sex. Further Wilcoxon rank-sum testing revealed no statistically significant differences between female and male preservation scores for each joint, though female means were always slightly higher than male means (p-values ranged from 0.076 to 0.972). Thus, female and male preservation scores were averaged for each joint. The combined-sex preservation-score sample was then divided into six age groups (<40 years, 40-49 years, 50-59 years, 60-69 years, 70-79 years, and 80+ years). In general, preservation score means increased with age for each joint. Wilcoxon rank-sum tests revealed statistically significant differences among pubic symphysis preservation scores between the 80+ age group and all other age groups (p-values ranged from 6.975e-08

to 0.001), so this age group was kept separate for further testing (tests among all other age groups resulted in *p*-values ranging from 0.05 to 0.990; α=0.003 with Bonferroni correction of 0.05/15). Wilcoxon rank-sum tests revealed statistically significant differences among auricular surface preservation scores between the 70-79 and 80+ age group and all other age groups (*p*-values for the 70-79-year age group ranged from 0.001 to 0.306; *p*-values for the 80+ age group ranged from 1.299e-13 to 7.897e-05), so these age groups were kept separate for further testing (tests among all other age groups resulted in *p*-values ranging from 0.055 to 0.800; α=0.003 with Bonferroni correction of 0.05/15). Age differences were less marked in the acetabulum, with only the <40-year and 40-49-year age groups showing statistically significant differences in preservation from the 80+ age group (*p*-values=7.751e-05 and 1.139e-04) and approaching significant differences from the 70-79-year age group (*p*-values=0.005 and 0.006); *p*-values for other inter-group tests ranged from 0.054 to 0.986. Still, the 70-79-year and 80+ age groups were kept separate for further testing, for consistency with the other pelvic joints.

Testing proceeded within these groups (combined-sex pubic symphysis scores for individuals <80 years [*n*=215] and 80+ years [*n*=69]; combined-sex auricular surface scores for individuals <70 years [*n*=179], 70-79 years [*n*=35], and 80+ years [*n*=69]; and combined-sex acetabular scores for individuals <70 years [*n*=183], 70-79 years [*n*=37], and 80+ years [*n*=71]). Mean and median pelvic joint scores appear in Table 6-69, and *p*-values for the pelvic joint scores appear in Table 6-70. Acetabular damage scores were the lowest in all age-matched comparisons (Table 6-69). They were also the most resistant to age-related changes in preservation, only showing statistically significant

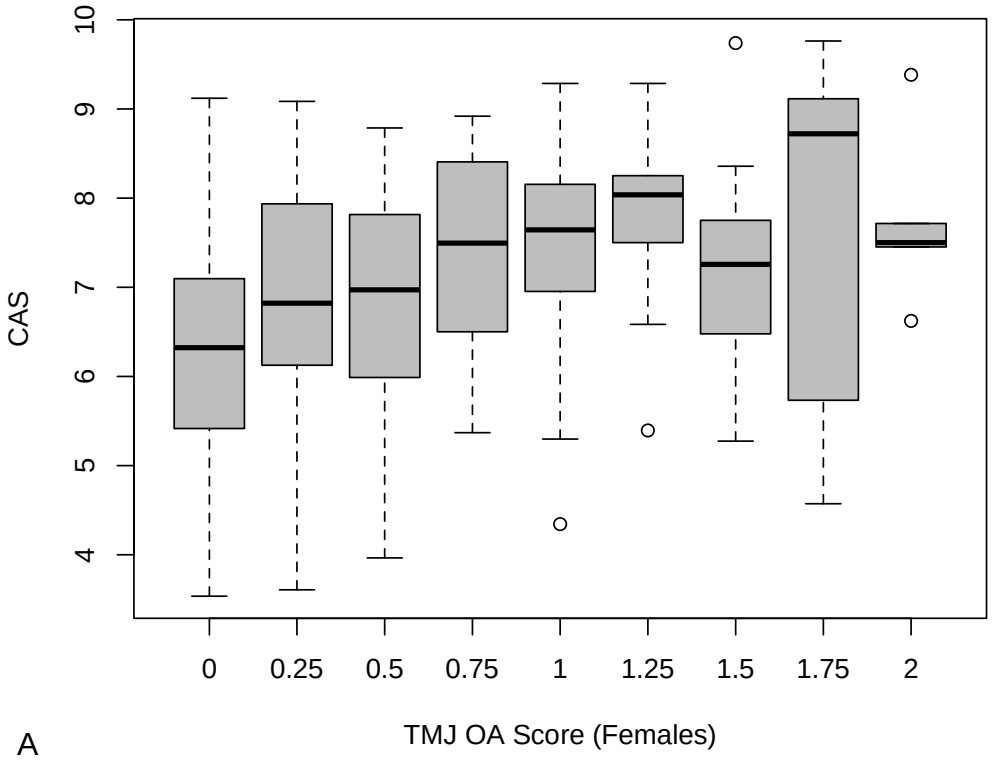
differences between the oldest and youngest age groups (Table 6-70). Acetabulum means were statistically significantly lower than pubic symphysis means in the 80+ age group, and acetabulum means for the <70-year-age group were also statistically significantly lower than pubic symphysis means in the <80-year age group. Auricular surface means in the <70-year age group were also statistically significantly lower than pubic symphysis means in the <80-year age group; but in the 80+ age group, auricular surface means were statistically significantly higher than pubic symphysis means (Table 6-70). Auricular surface and acetabulum means were similar for the <70-year and 70-79-year age groups, but acetabular means were statistically significantly lower than auricular surface means in the 80+ age group (Table 6-70). Thus, acetabula appeared to be statistically significantly better preserved than pubic symphyses and auricular surfaces in most age-matched comparisons—particularly those involving the oldest individuals.

Summary

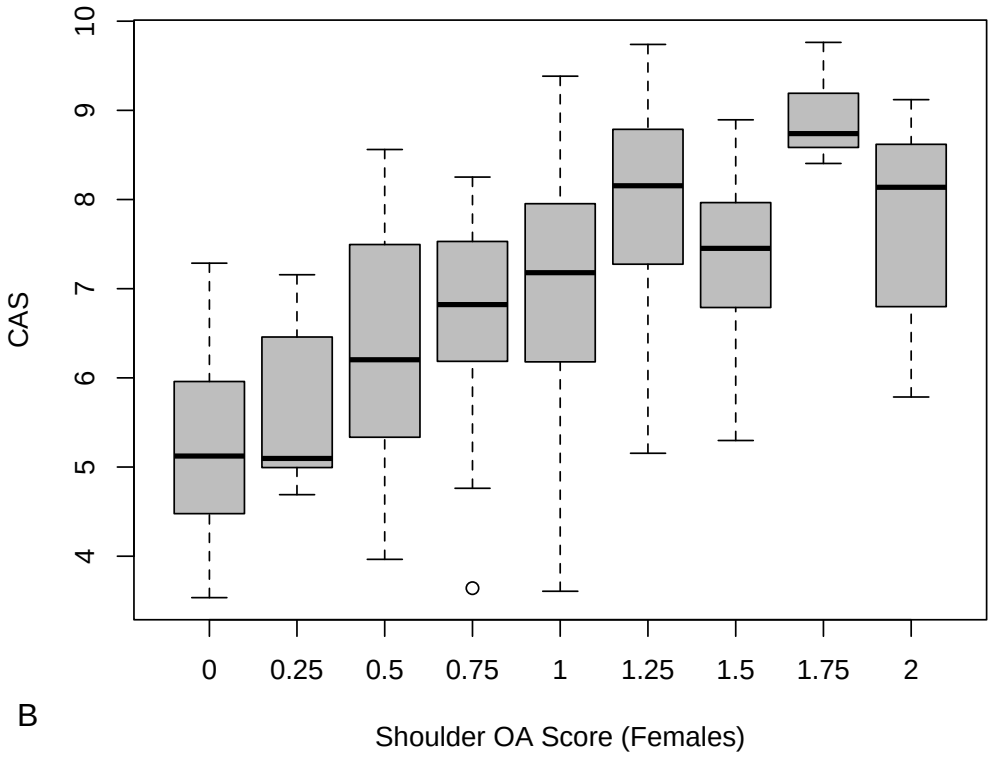
This chapter presented the results of the current analyses. These findings have provided support for Hypotheses 1 (acetabular changes correlate positively with OA), 2a (acetabular changes correlate positively with age), 2b (OA correlates positively with age), and 5a (age is the major contributing factor to acetabular changes. These results have provided limited support for Hypotheses 4b (OA correlates positively with obesity) and 5b (age is the major contributing factor to OA). On the basis of these results, Hypotheses 3a (acetabular changes correlate positively with activity), 3b (OA correlates positively with activity) and 4a (acetabular changes correlate positively with obesity) can be rejected. The next chapter discusses and contextualizes these results.

Table 6-1. Sample sizes for each of the variables used in statistical analyses.

Variable	*n* (female)	*n* (male)	*n* (combined-sex)
Acetabular age estimates	195	209	404
CAS	198	210	408
Acetabular variables 1, 2, 3, 4, 6, and 7	195	210	405
Acetabular variable 5	195	209	404
TMJ OA	198	210	408
Shoulder OA	198	210	408
Elbow OA	198	210	408
Wrist OA	197	210	407
Hip OA	198	210	408
Knee OA	195	203	398
Ankle OA	194	208	402
Overall OA	198	210	408
Upper limb OA	198	210	408
Lower limb OA	198	210	408
BMI	187	188	375
MET level (habitual activities)	71	69	140
MET level (occupational activities)	178	192	370
Preservation (pubic symphysis)	148	137	285
Preservation (auricular surface)	148	136	284
Preservation (acetabulum)	149	143	292

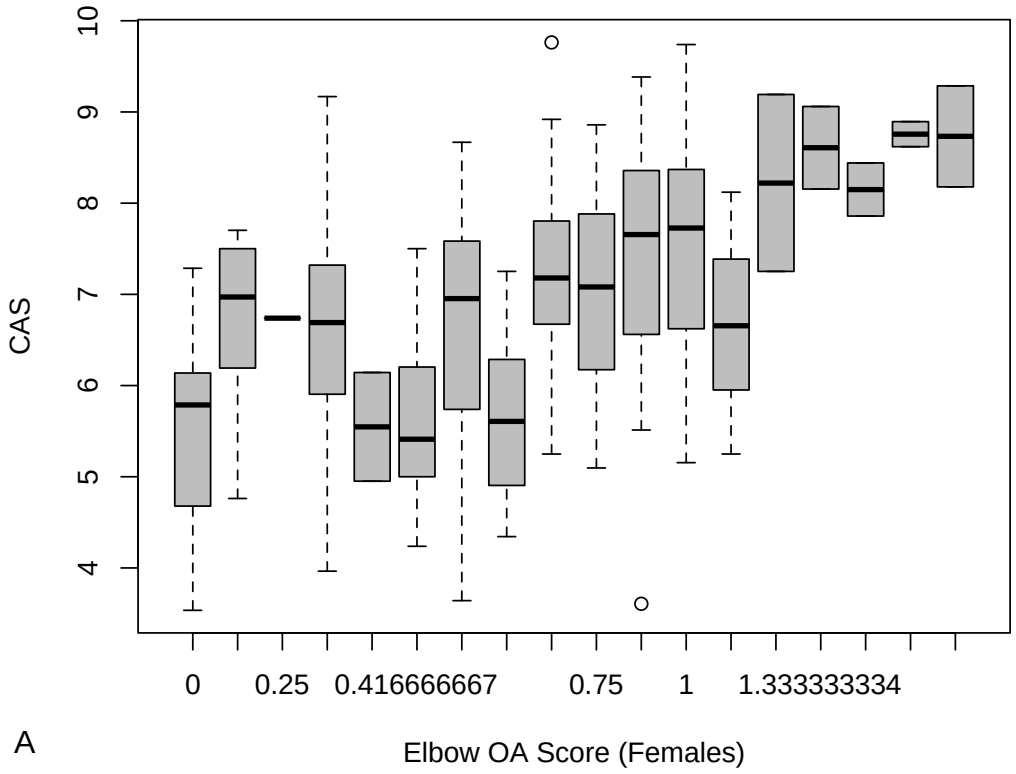


A

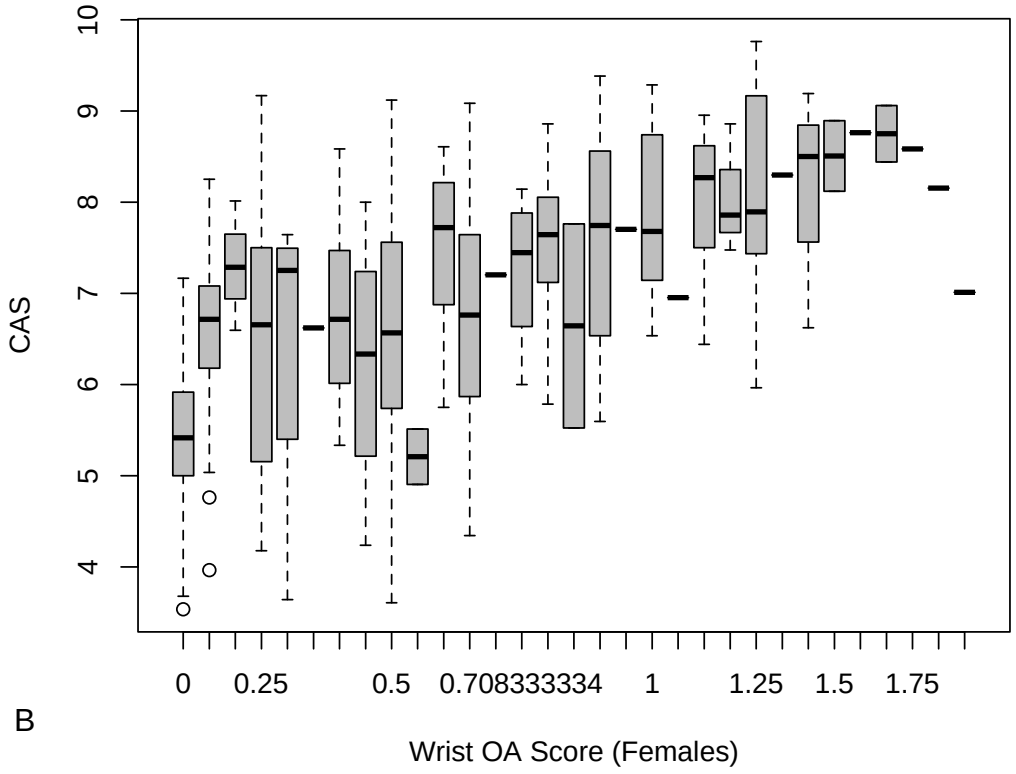


B

Figure 6-1. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots of CAS per OA score in females. A) TMJ, B) Shoulder.



A



B

Figure 6-2. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots of CAS per OA score in females. A) Elbow, B) Wrist.

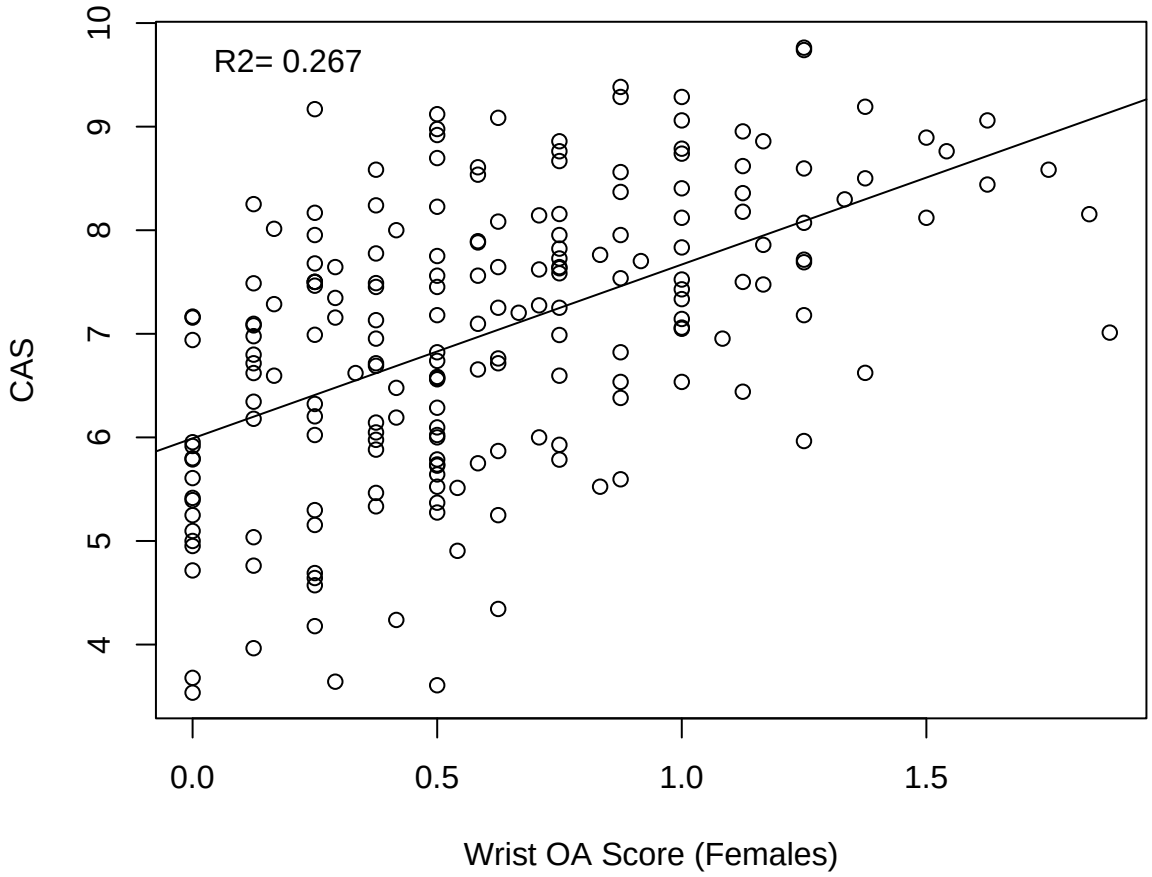
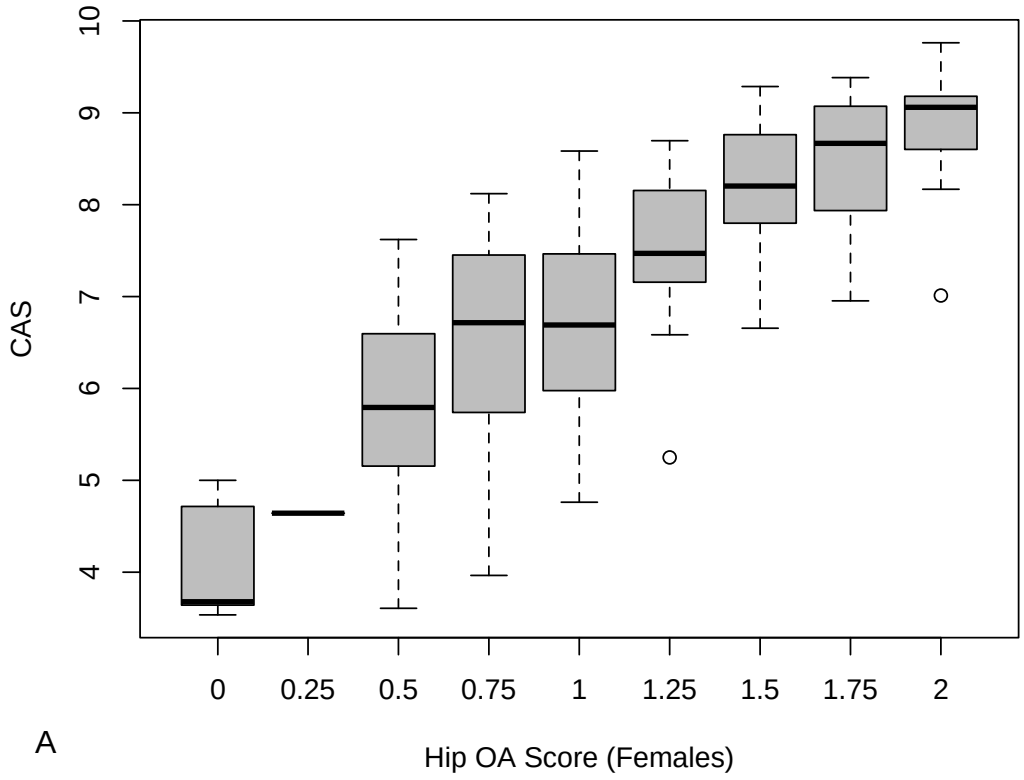


Figure 6-3. Hypothesis 1: acetabular changes correlate positively with OA; scatterplot for CAS and wrist OA score in females. Black line indicates regression model.



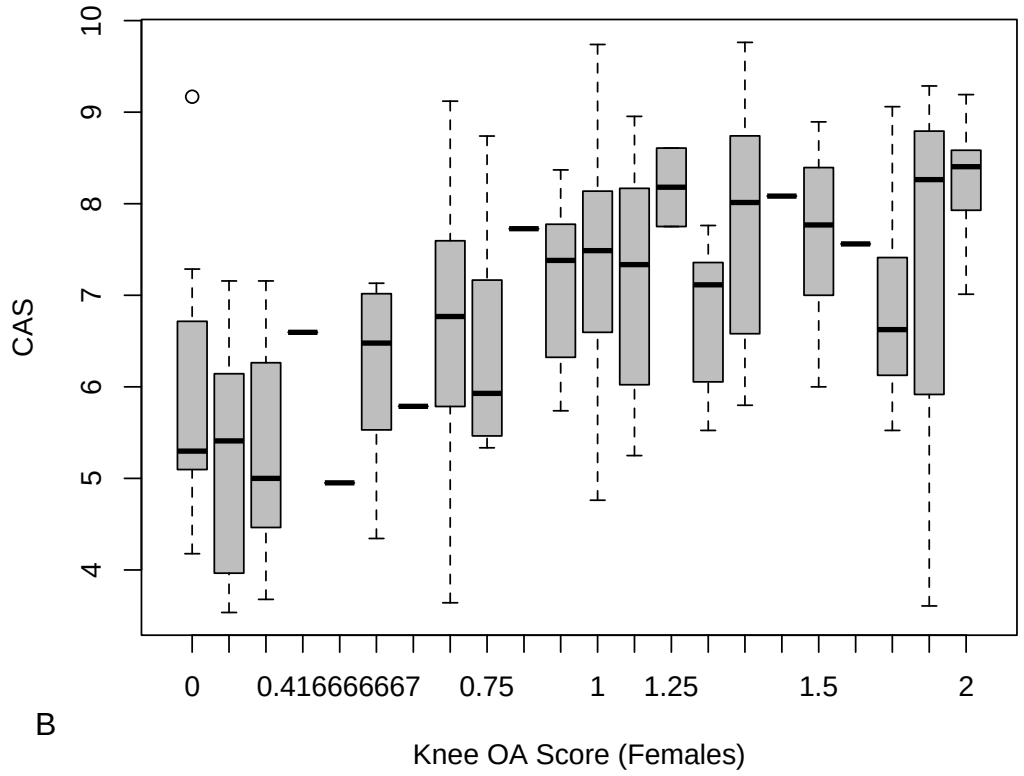


Figure 6-4. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots of CAS per OA score in females. A) Hip, B) Knee.

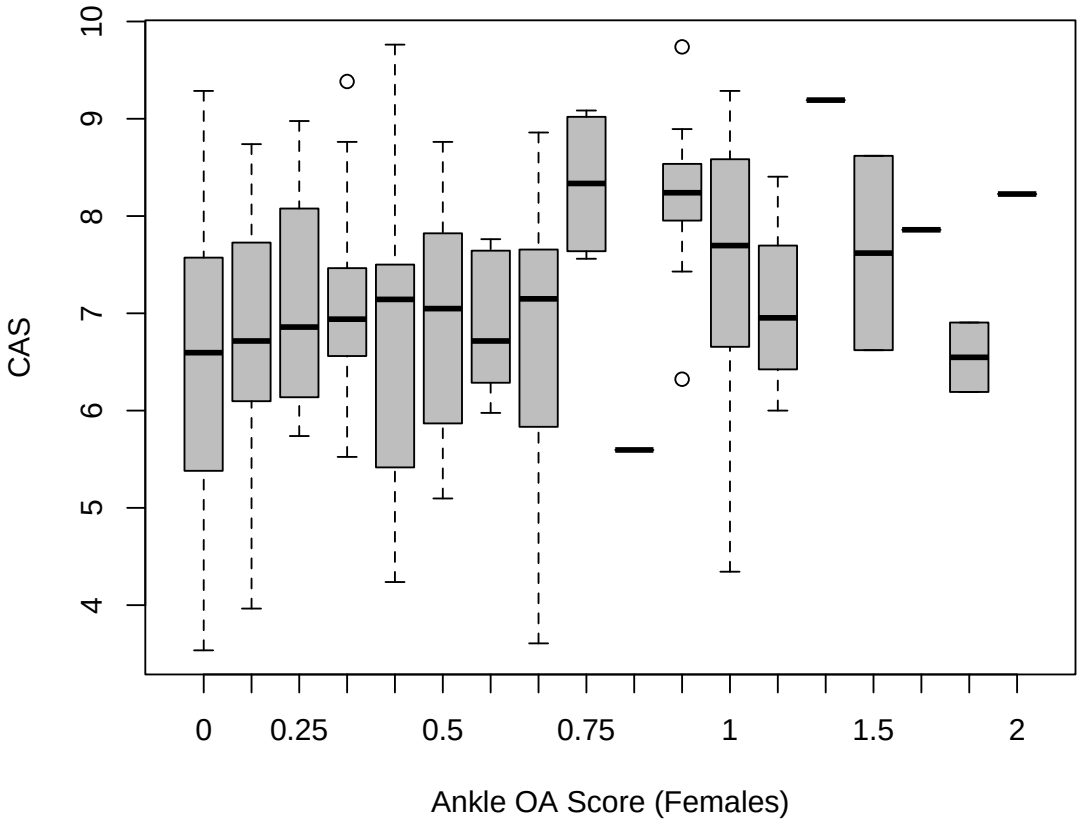


Figure 6-5. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plot of CAS per OA score in the female ankle joint.

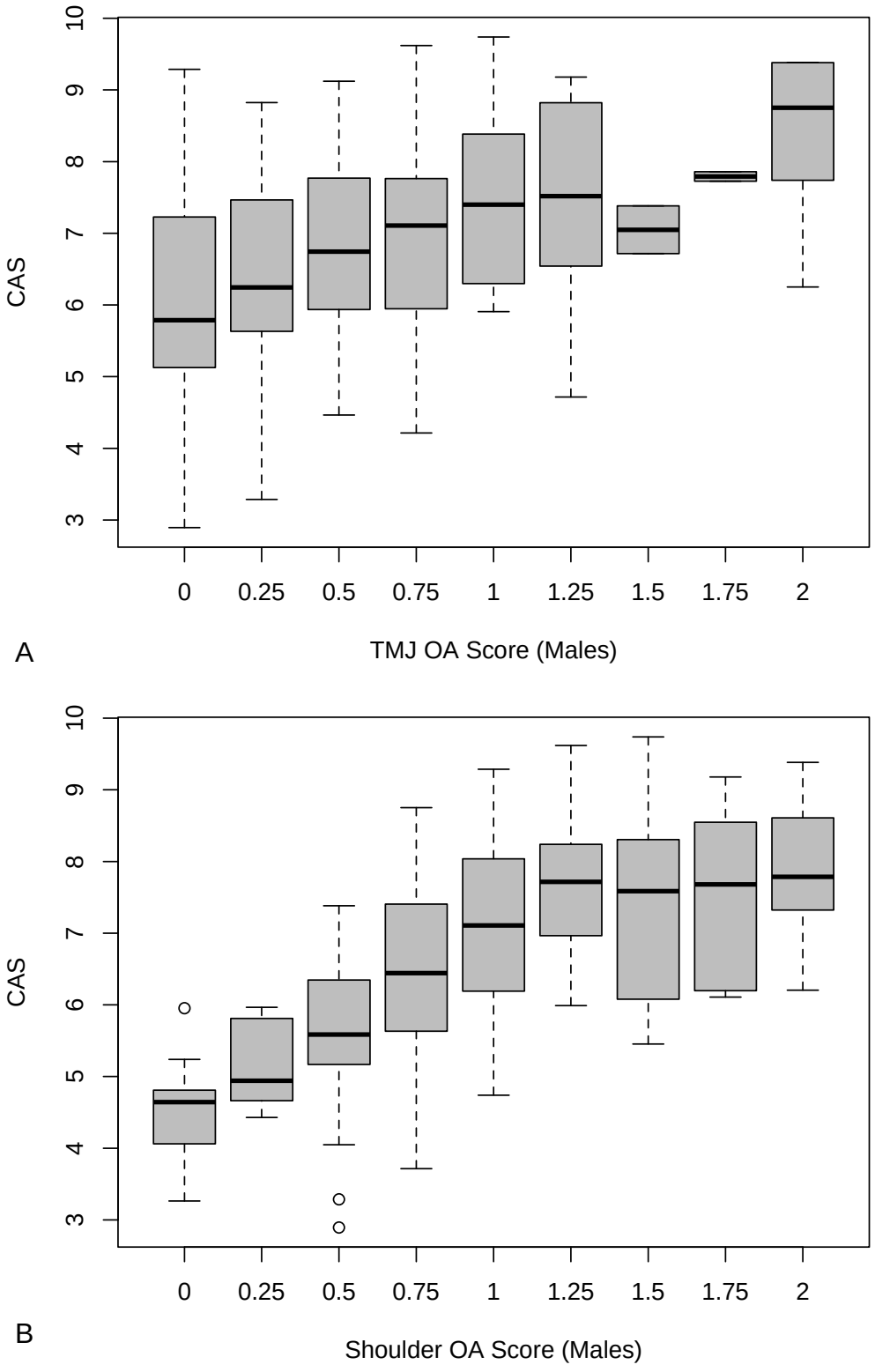


Figure 6-6. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots of CAS per OA score in males. A) TMJ, B) Shoulder.

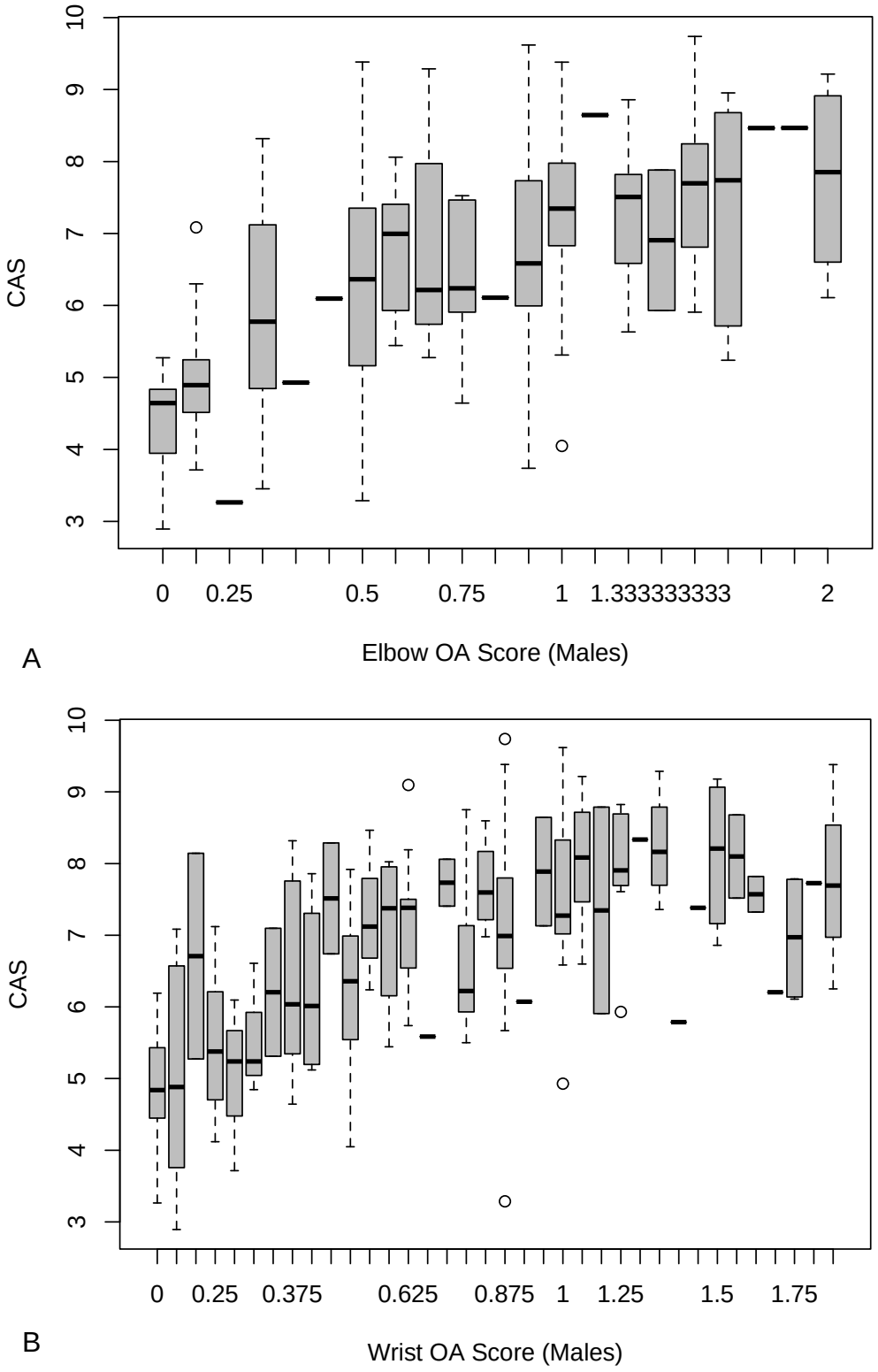


Figure 6-7. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots of CAS per OA score in males. A) Elbow, B) Wrist.

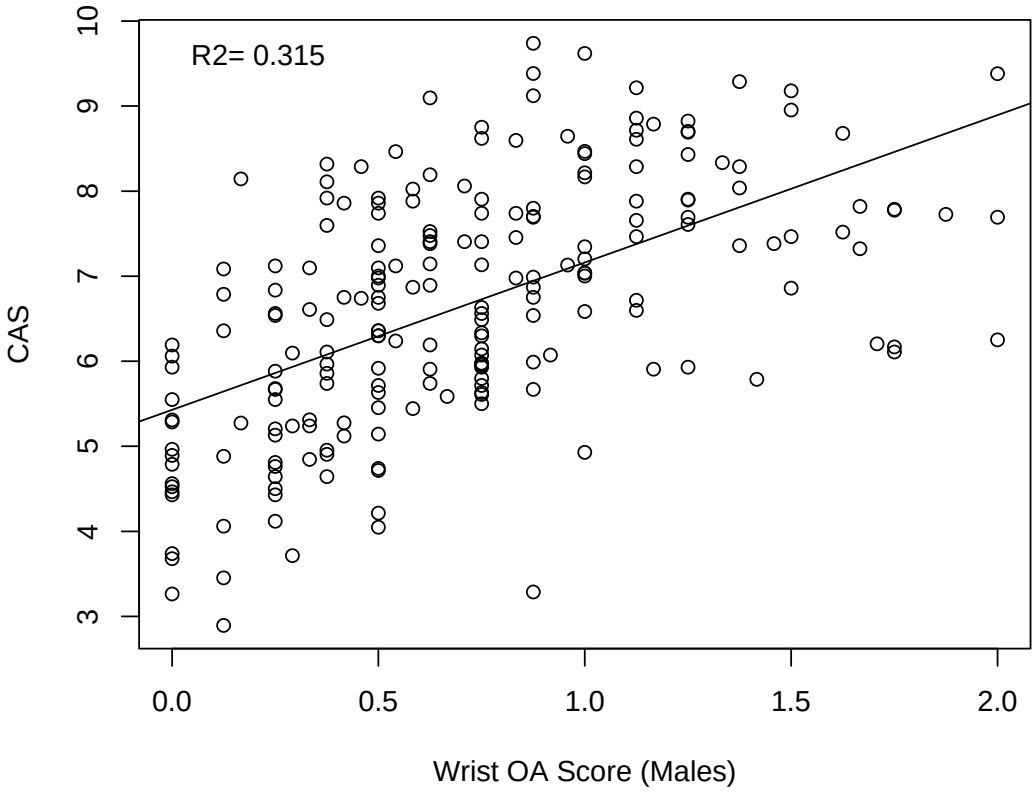
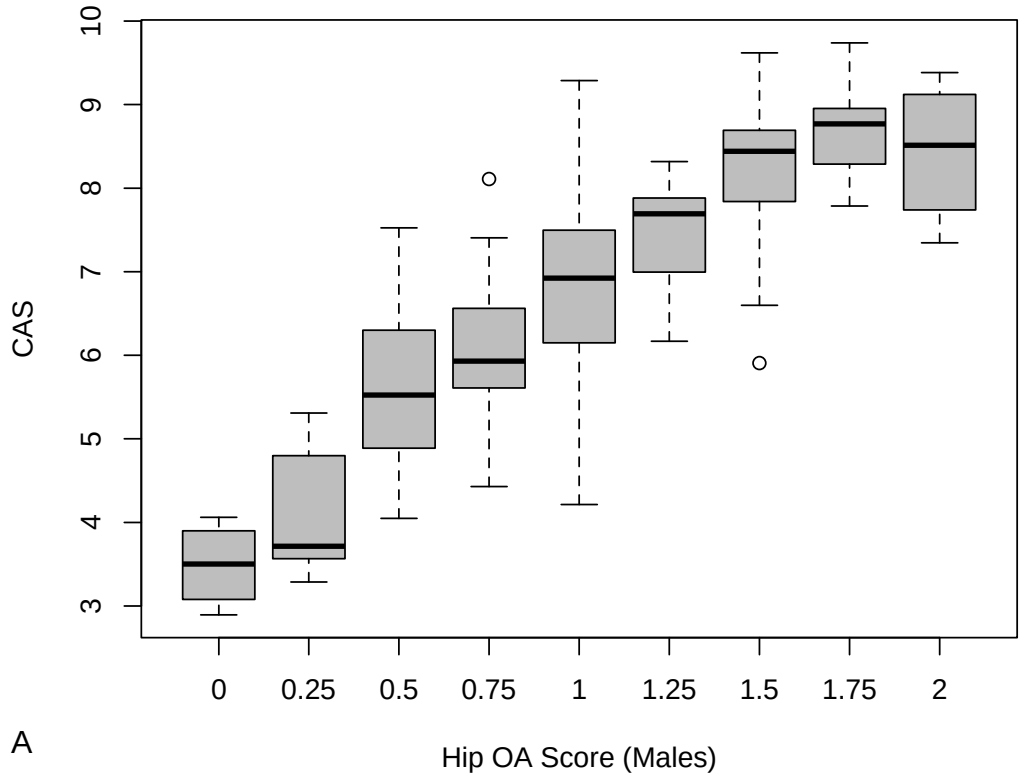
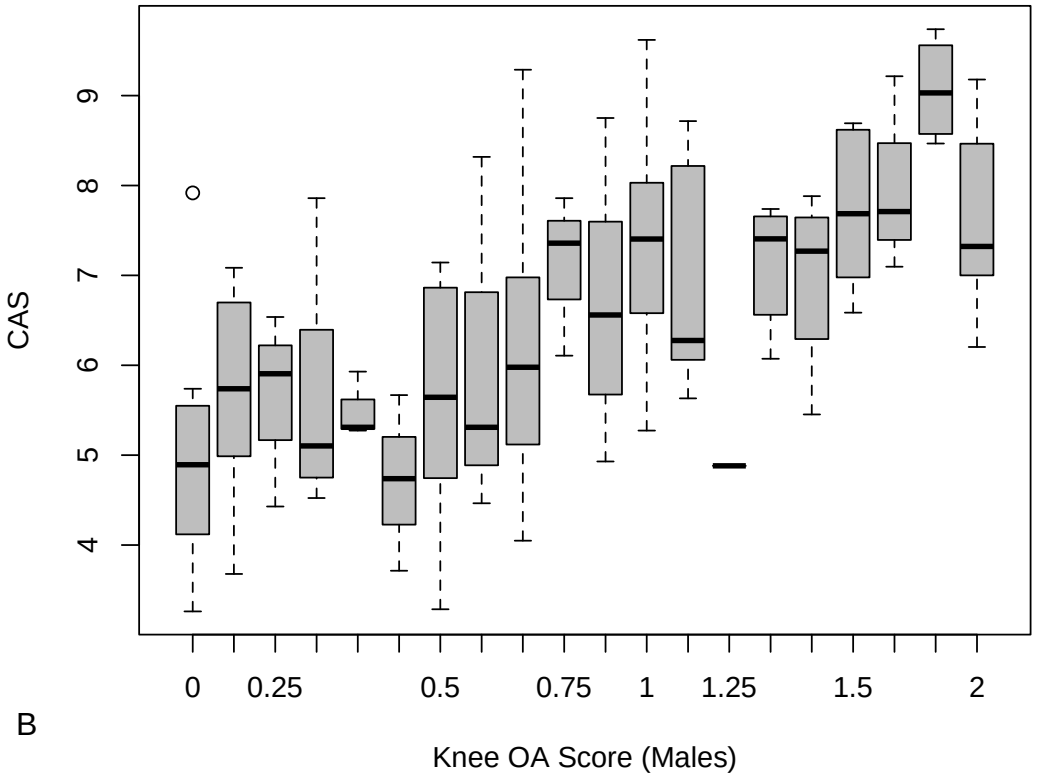


Figure 6-8. Hypothesis 1: acetabular changes correlate positively with OA; scatterplot for CAS and wrist OA score in males. Black line indicates linear regression model.



A



B

Figure 6-9. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots of CAS per OA score in males. A) Hip, B) Knee.

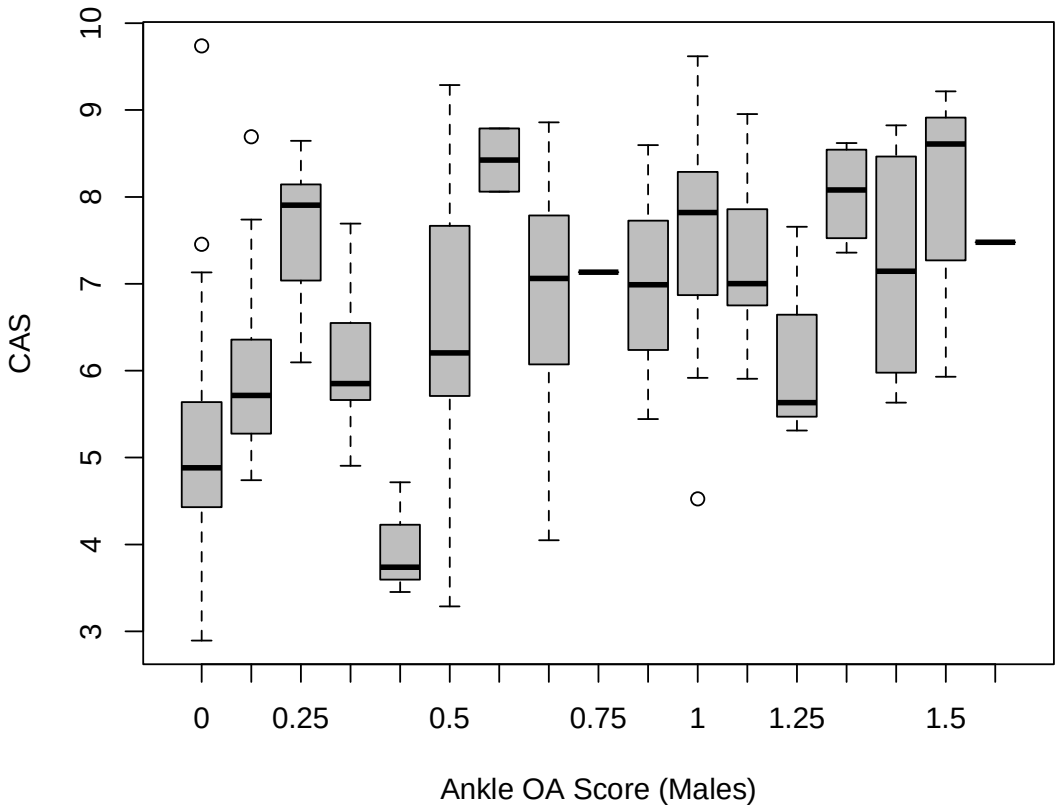


Figure 6-10. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plot of CAS per OA score in the male ankle joint.

Table 6-2. Spearman's rank-order correlations for CAS and OA in the various joints in females and males.

Joint	*rho* (female)	*rho* (male)
TMJ	0.331*	0.345*
Shoulder	0.456*	0.654*
Elbow	0.428*	0.520*
Wrist	0.525*	0.603*
Hip	0.745*	0.772*
Knee	0.435*	0.564*
Ankle	0.282*	0.498*

*Statistically significant at α=0.007 (Bonferroni correction: α=0.05/7).

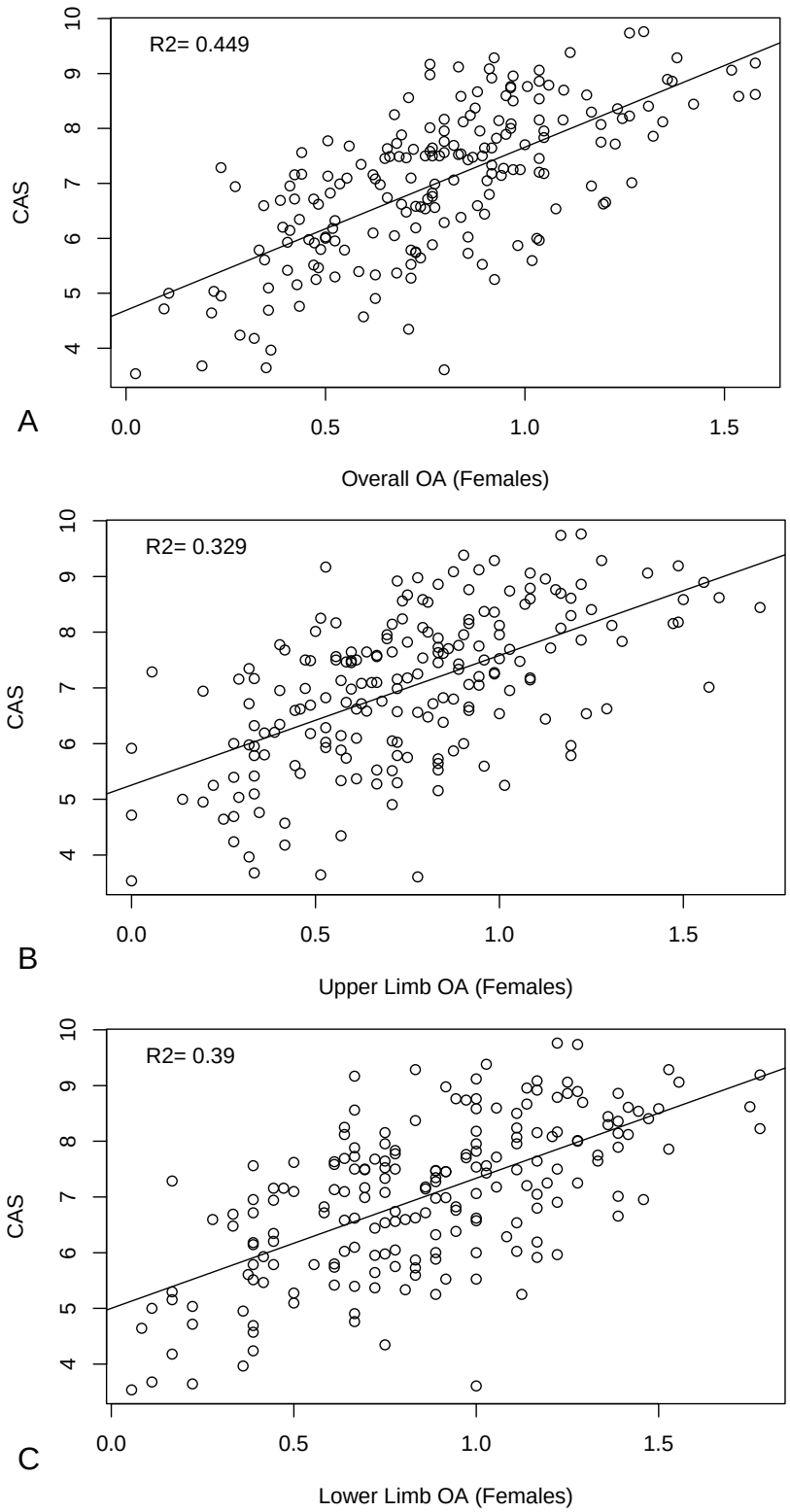


Figure 6-11. Hypothesis 1: acetabular changes correlate positively with OA; scatterplots for CAS and OA in females. A) Overall, B) Upper limb, C) Lower limb. Black line indicates linear regression model.

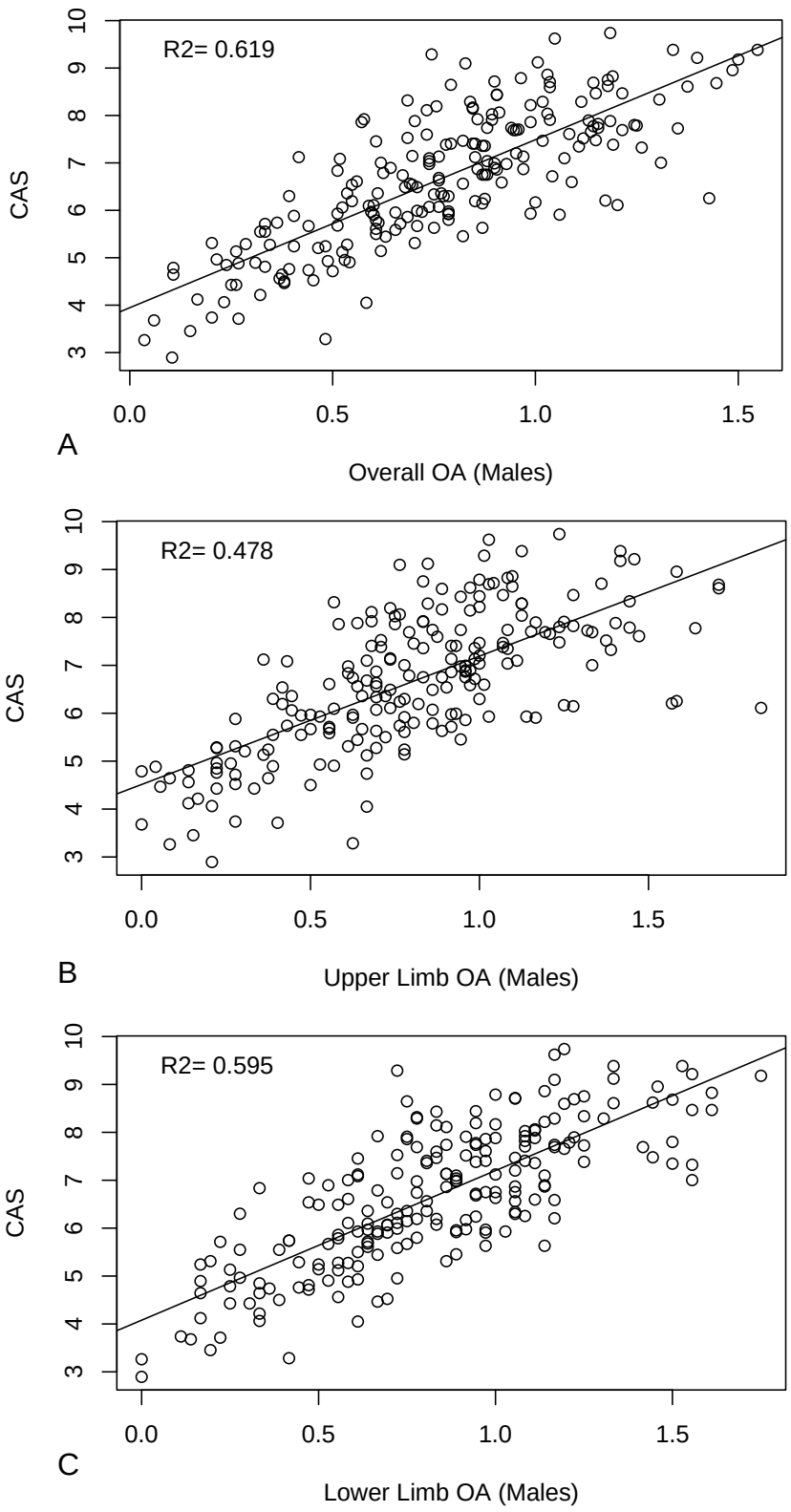


Figure 6-12. Hypothesis 1: acetabular changes correlate positively with OA; scatterplots for CAS and OA in males. A) Overall, B) Upper limb, C) Lower limb. Black line indicates linear regression model.

Table 6-3. Spearman's rank-order correlations for CAS and overall, upper limb, and lower limb OA in females and males.

Summary OA	*rho* (females)	*rho* (males)
Overall	0.664*	0.779*
Upper limb	0.572*	0.703*
Lower limb	0.606*	0.765*

*Statistically significant at α=0.02 (Bonferroni correction: α=0.05/3).

Table 6-4. Regression results for CAS and overall, upper limb, and lower limb OA in females and males.

Variable	Estimate	Std. Error	t value	Probability
Intercept	4.690	0.197	23.84	<2e-16*
Female overall OA	2.970	0.234	12.68	<2e-16*
Intercept	5.257	0.1949	26.971	<2e-16*
Female upper limb OA	2.326	0.236	9.857	<2e-16*
Intercept	5.004	0.193	25.88	<2e-16*
Female lower limb OA	2.334	0.207	11.27	<2e-16*
Intercept	3.947	0.159	24.78	<2e-16*
Male overall OA	3.543	0.192	18.44	<2e-16*
Intercept	4.514	0.170	26.51	<2e-16*
Male upper limb OA	2.678	0.193	13.88	<2e-16*
Intercept	4.077	0.160	25.46	<2e-16*
Male lower limb OA	3.124	0.178	17.55	<2e-16*

*Statistically significant at α=0.02 (Bonferroni correction: α=0.05/3).

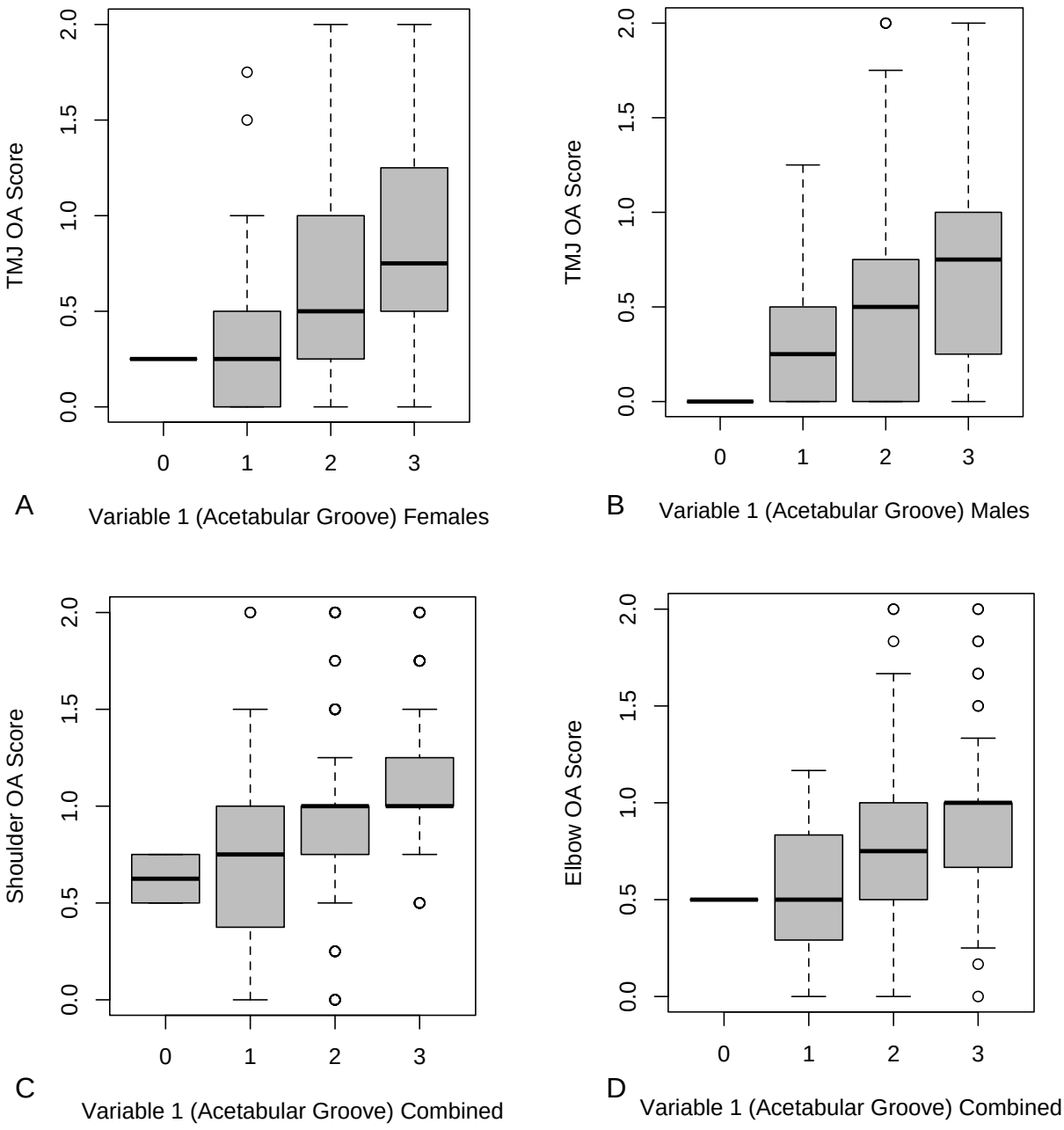


Figure 6-13. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for OA score per acetabular score (Variable 1). A) Female TMJ OA, B) Male TMJ OA, C) Combined shoulder OA, D) Combined elbow OA.

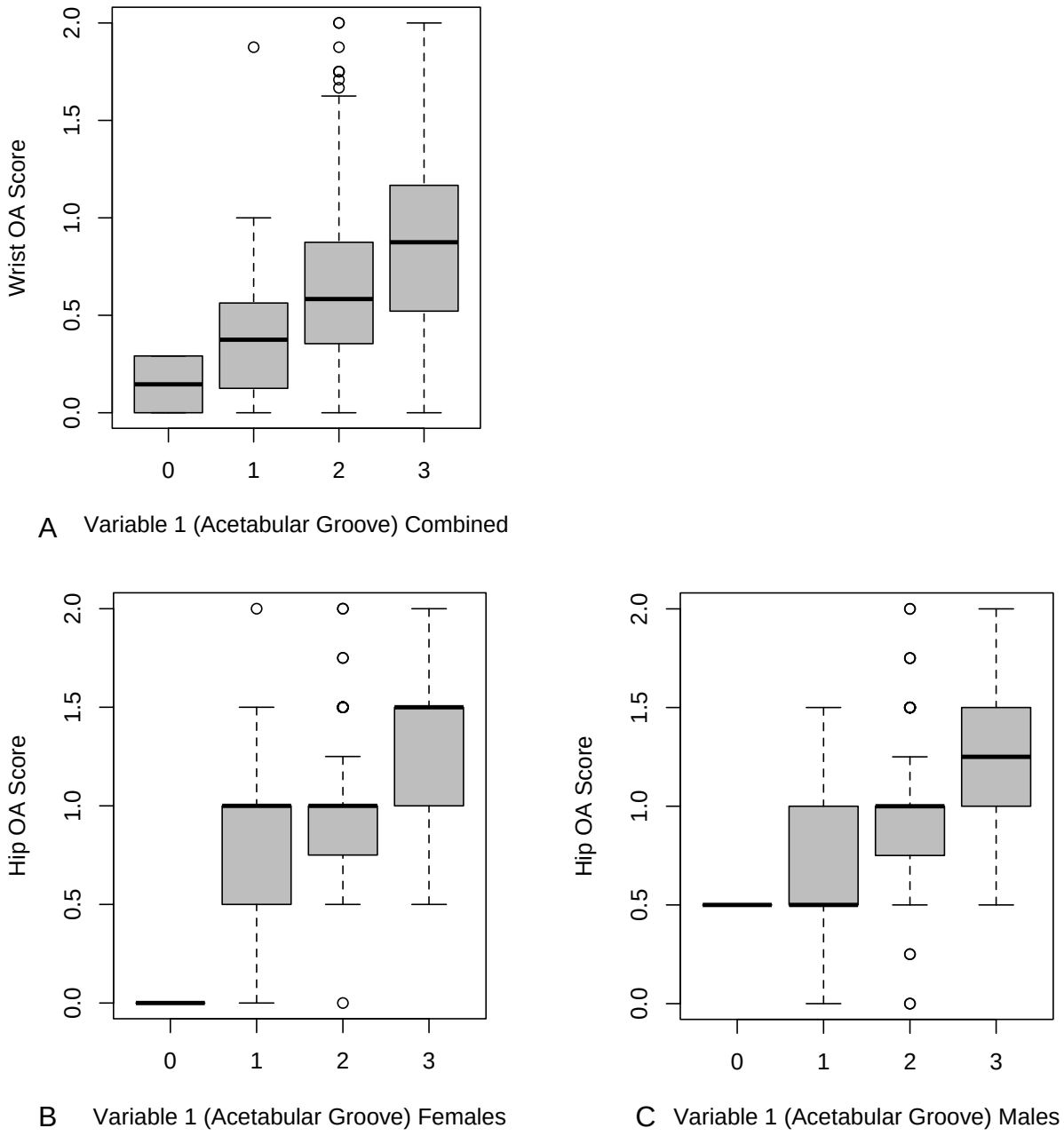


Figure 6-14. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for OA score per acetabular score (Variable 1). A) Combined wrist OA, B) Female hip OA, C) Male hip OA.

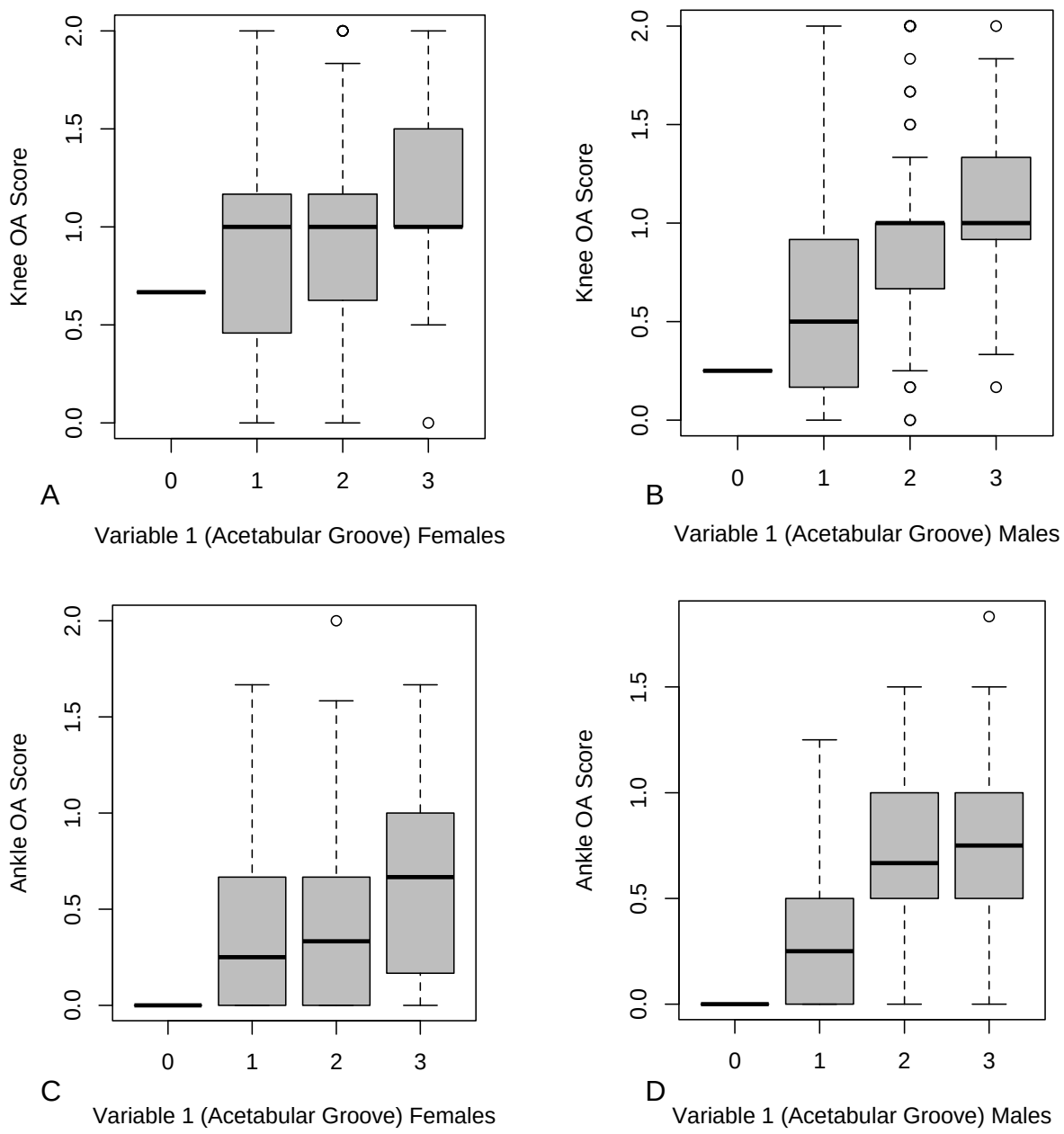


Figure 6-15. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for OA score per acetabular score (Variable 1). A) Female knee OA, B) Male knee OA, C) Female ankle OA, D) Male ankle OA.

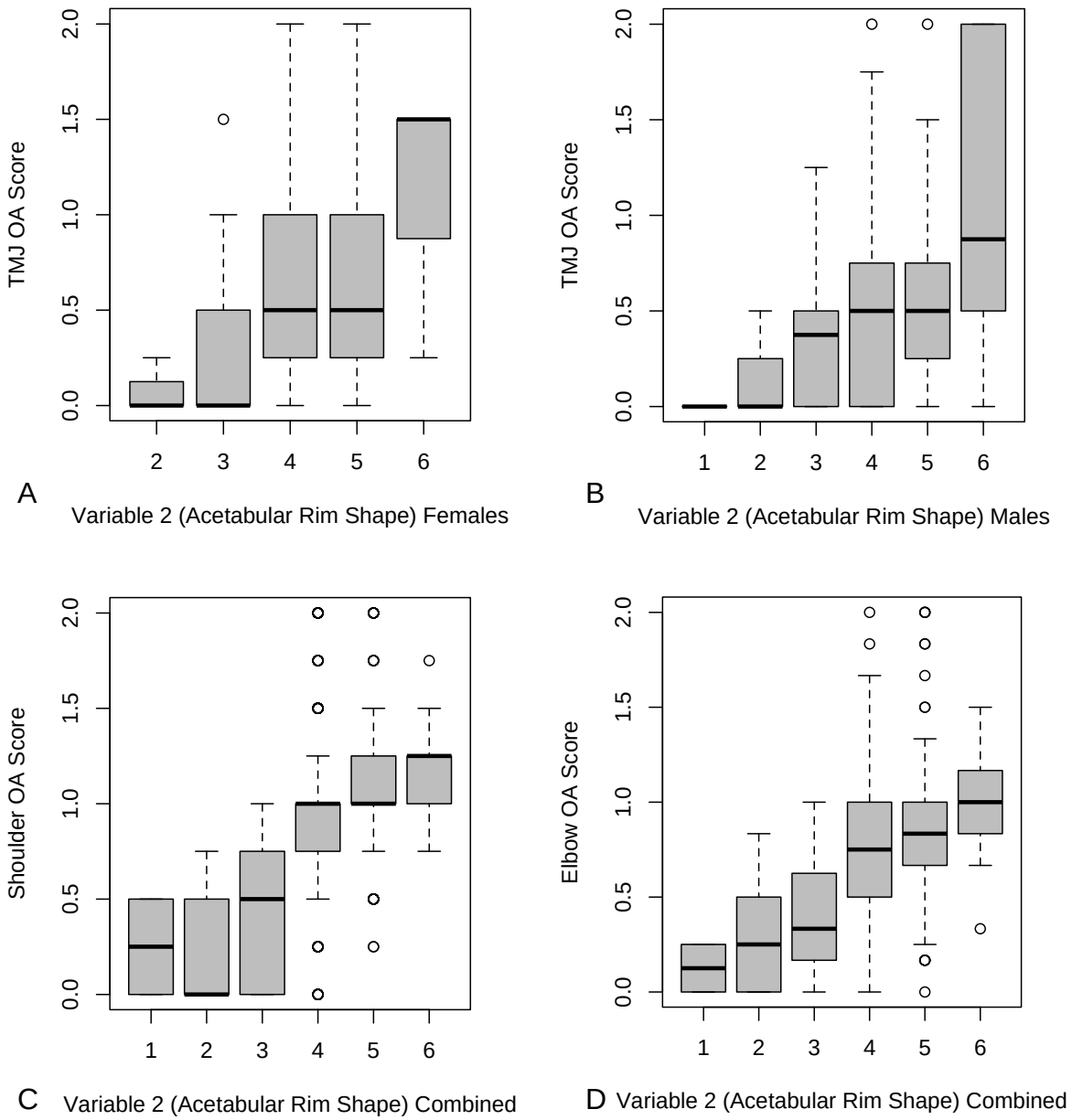


Figure 6-16. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for OA score per acetabular score (Variable 2). A) Female TMJ OA, B) Male TMJ OA, C) Combined shoulder OA, D) Combined elbow OA.

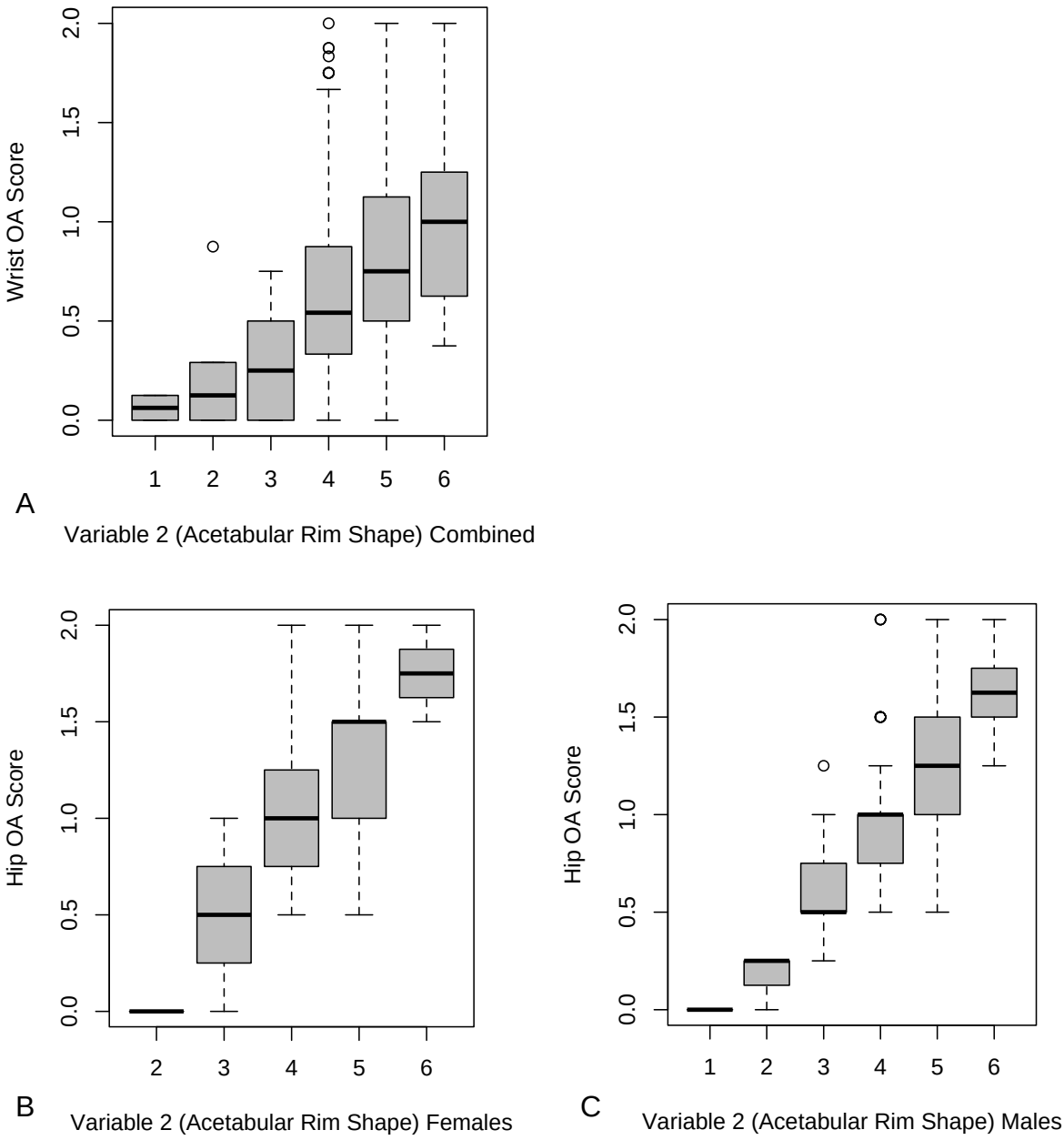


Figure 6-17. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for OA score per acetabular score (Variable 2). A) Combined wrist OA, B) Female hip OA, C) Male hip OA.

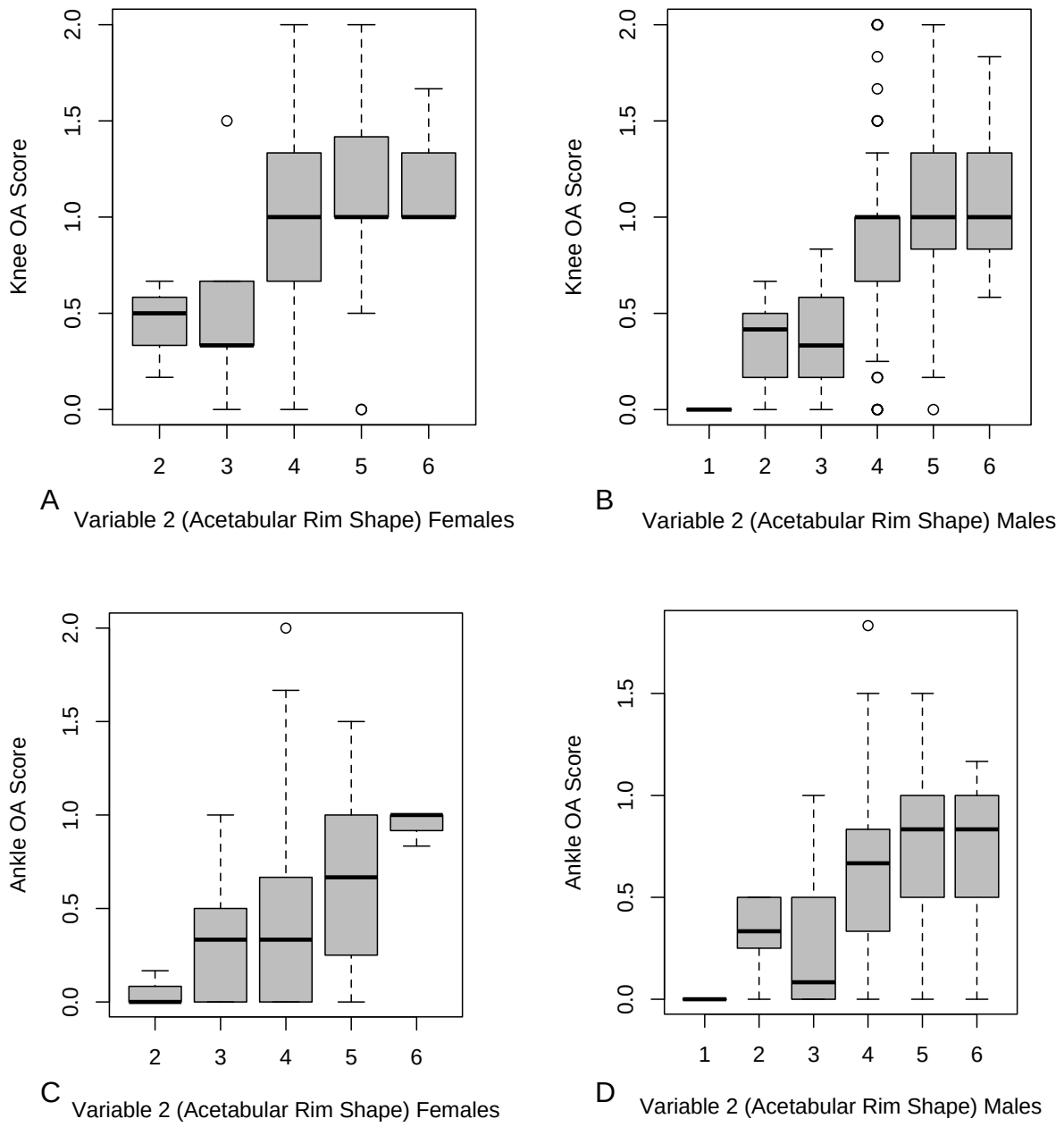


Figure 6-18. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for OA score per acetabular score (Variable 2). A) Female knee OA, B) Male knee OA, C) Female ankle OA, D) Male ankle OA.

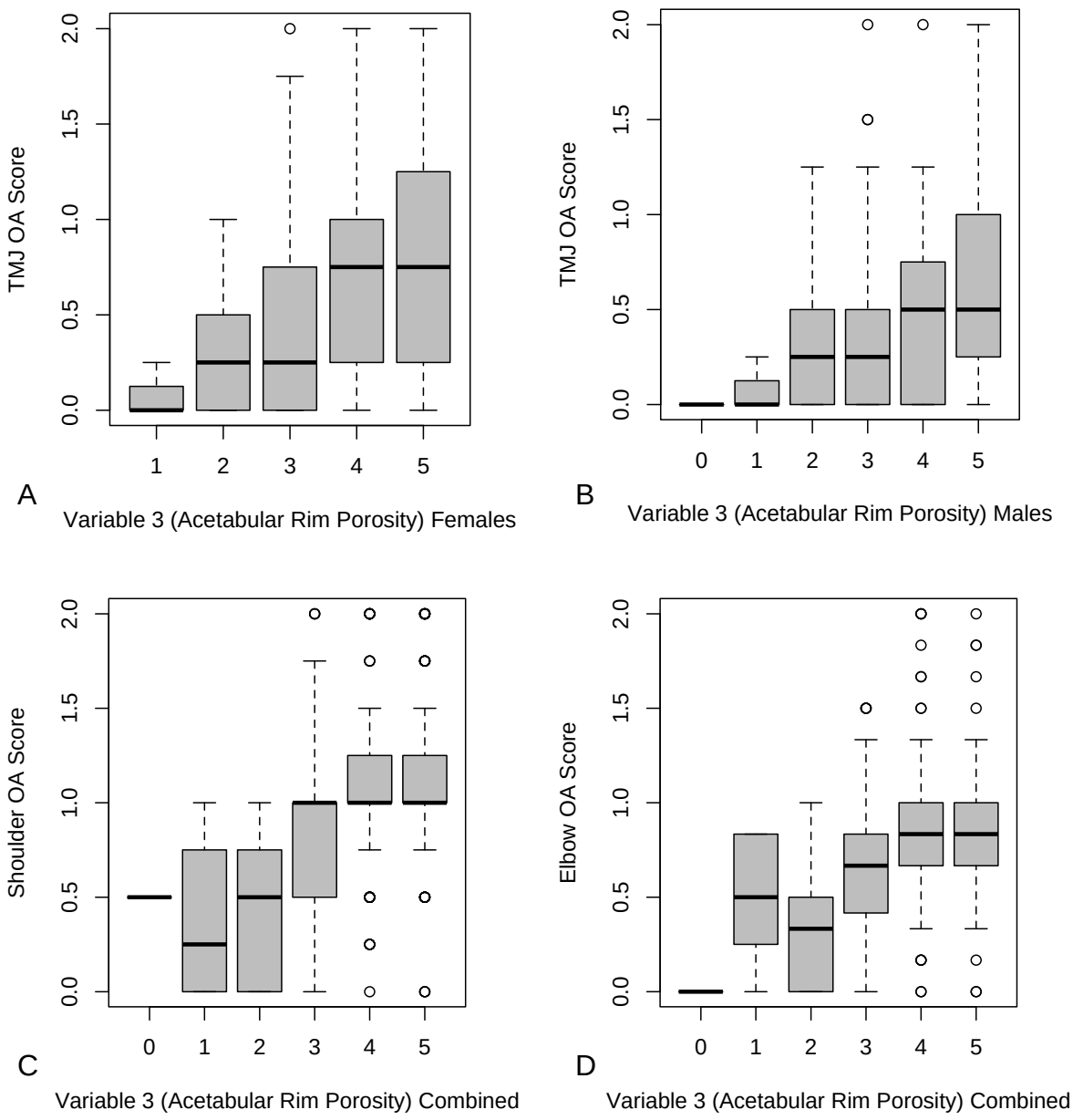


Figure 6-19. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for OA score per acetabular score (Variable 3). A) Female TMJ OA, B) Male TMJ OA, C) Combined shoulder OA, D) Combined elbow OA.

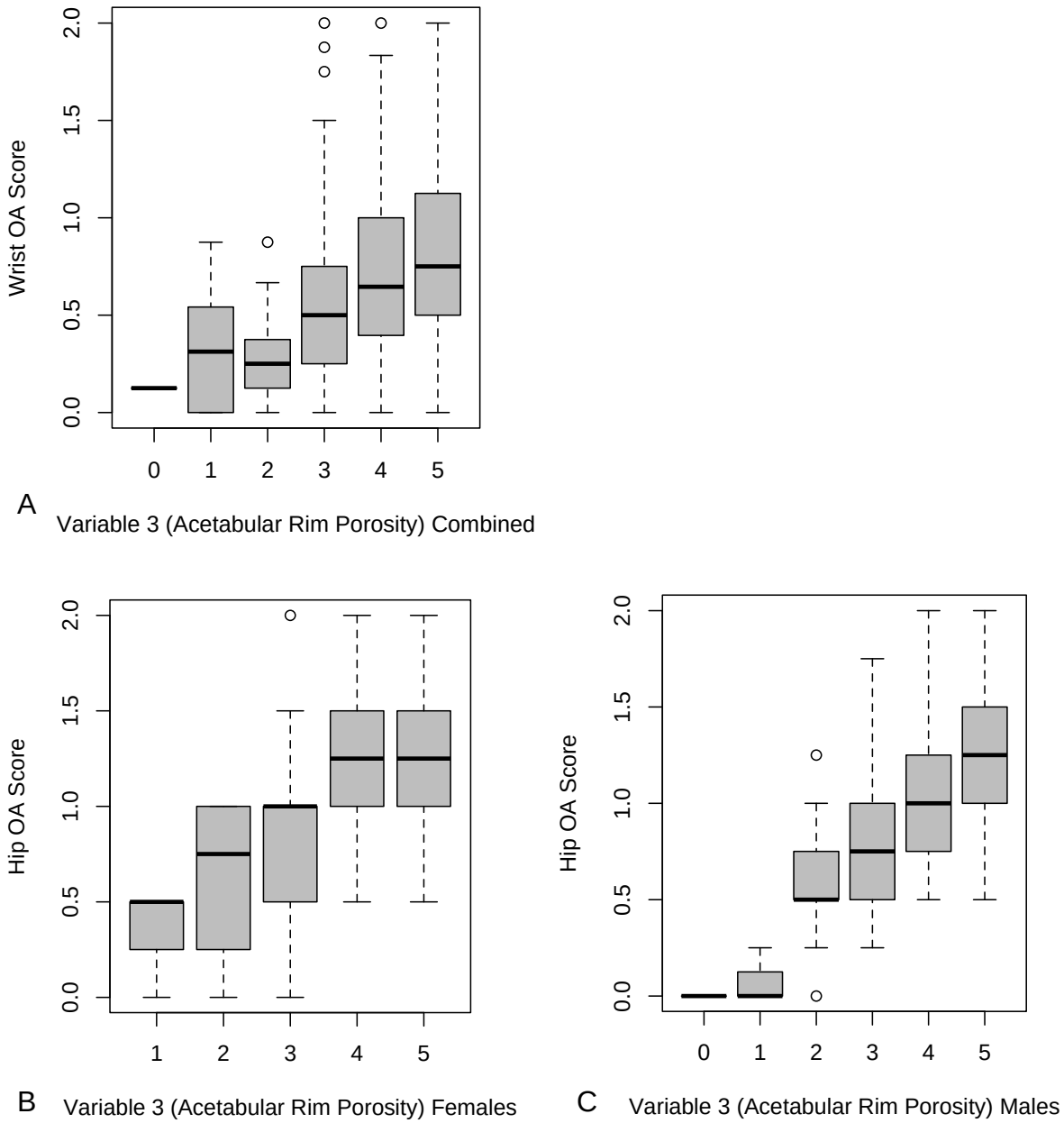


Figure 6-20. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for OA score per acetabular score (Variable 3). A) Combined wrist OA, B) Female hip OA, C) Male hip OA.

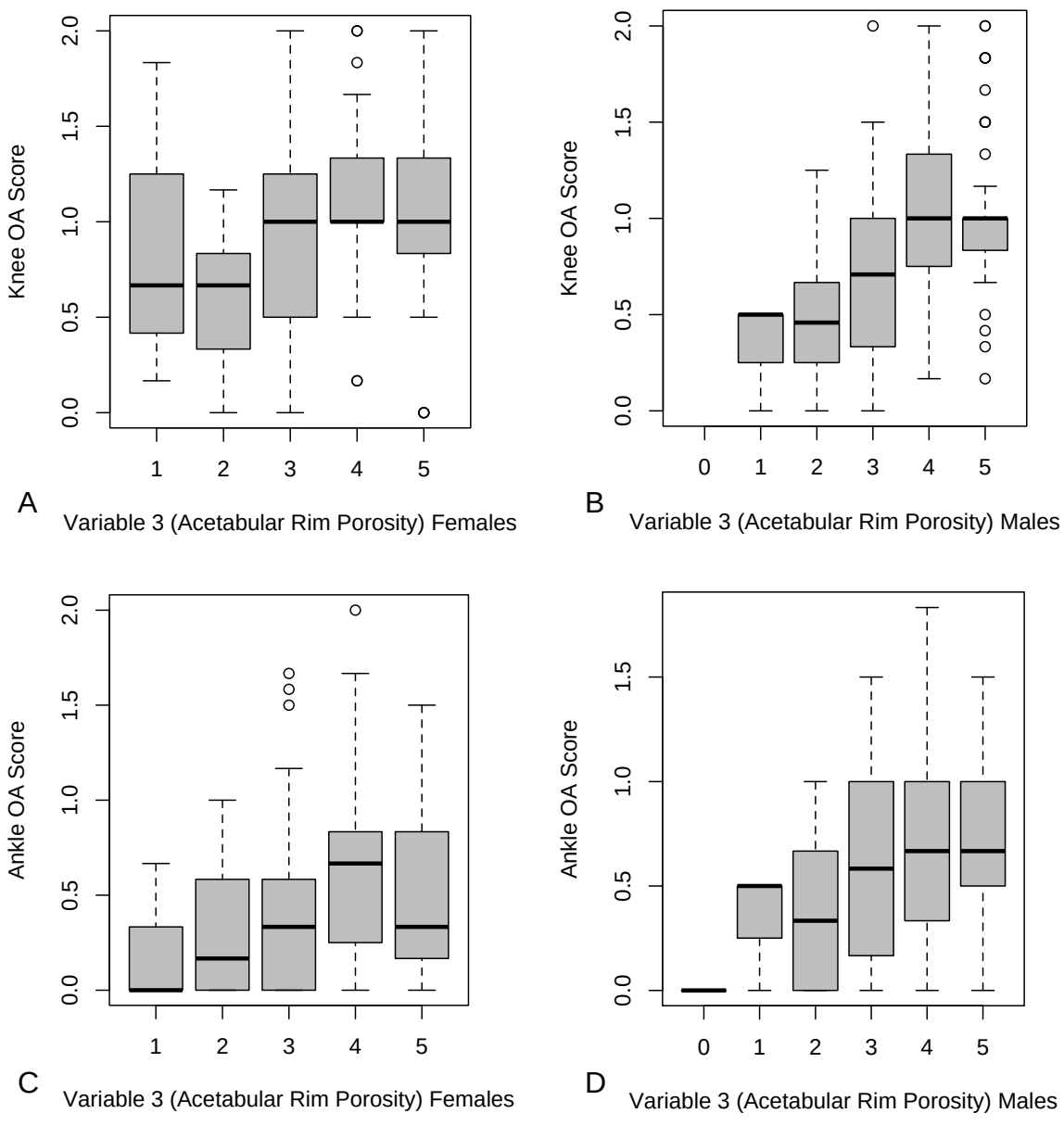


Figure 6-21. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for OA score per acetabular score (Variable 3). A) Female knee OA, B) Male knee OA, C) Female ankle OA, D) Male ankle OA.

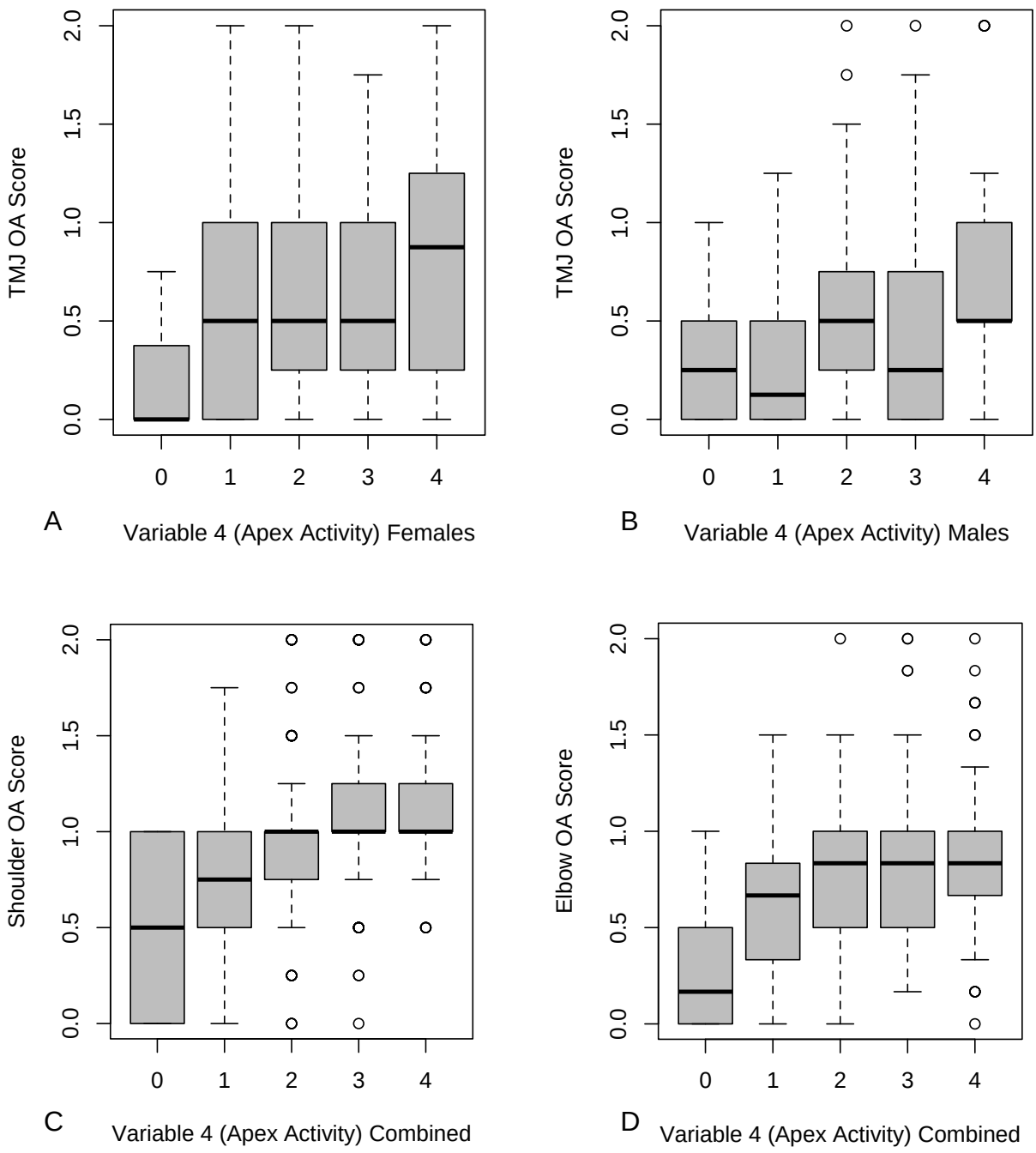


Figure 6-22. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for OA score per acetabular score (Variable 4). A) Female TMJ OA, B) Male TMJ OA, C) Combined shoulder OA, D) Combined elbow OA.

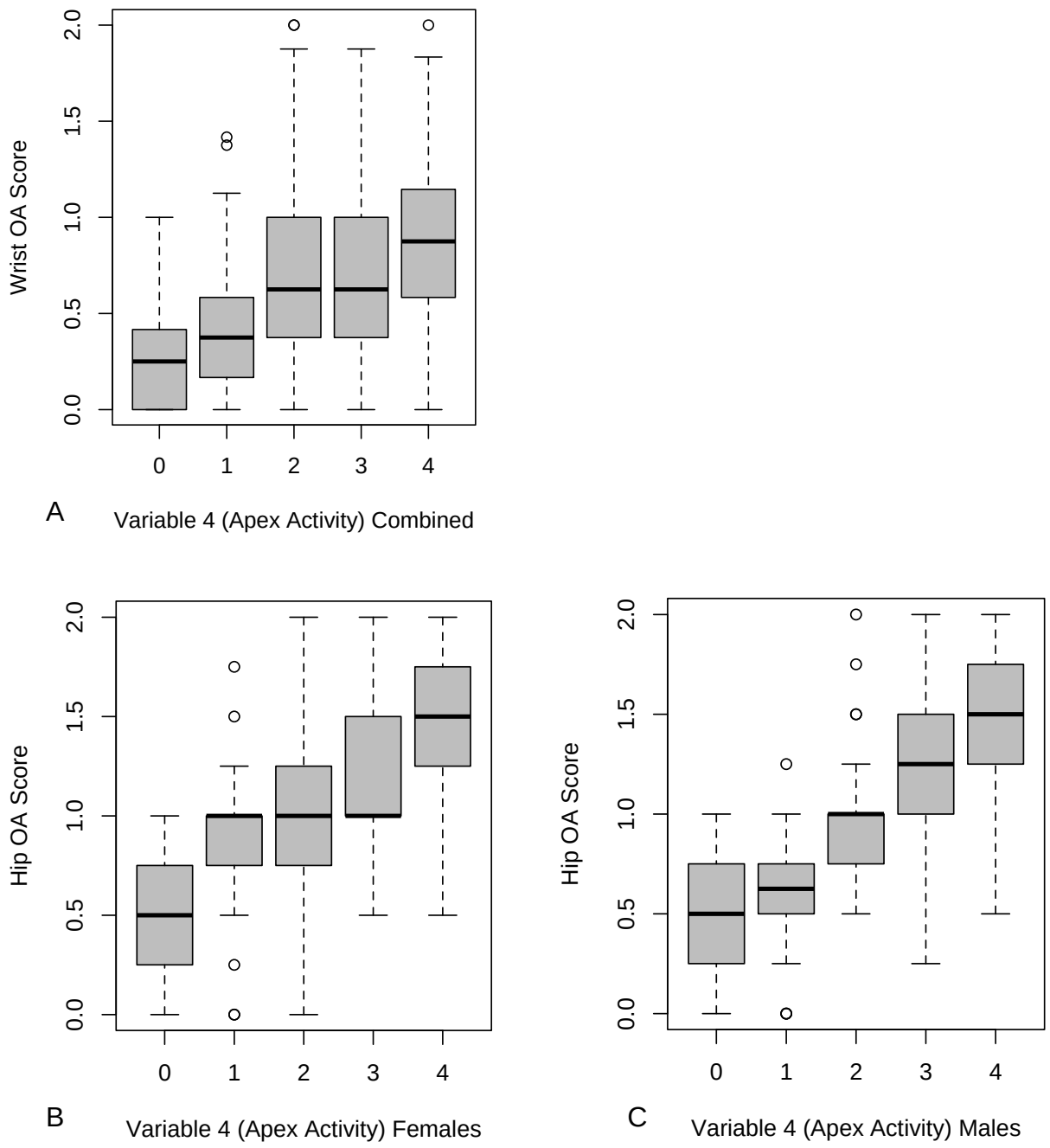


Figure 6-23. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for OA score per acetabular score (Variable 4). A) Combined wrist OA, B) Female hip OA, C) Male hip OA.

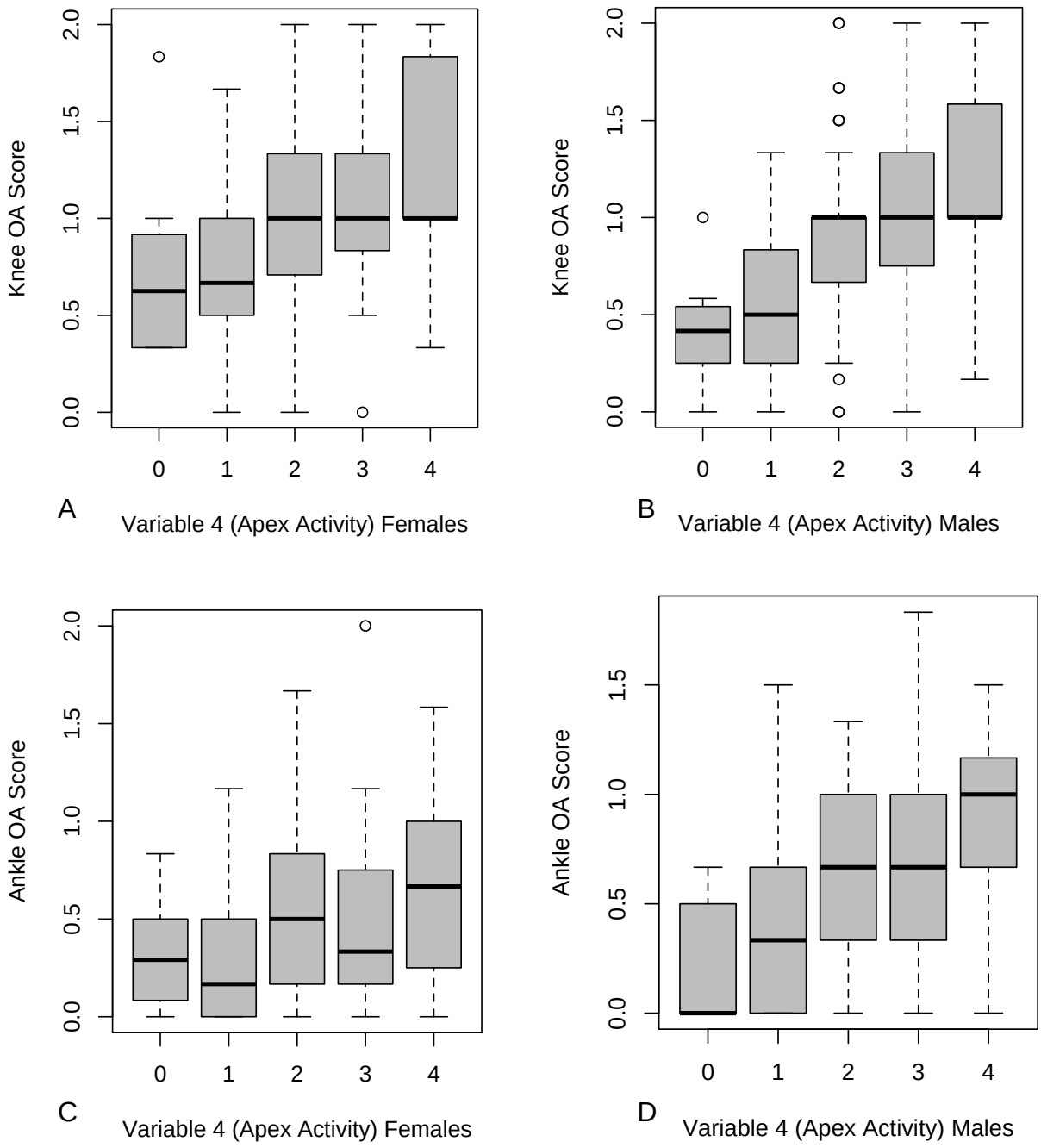


Figure 6-24. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for OA score per acetabular score (Variable 4). A) Female knee OA, B) Male knee OA, C) Female ankle OA, D) Male ankle OA.

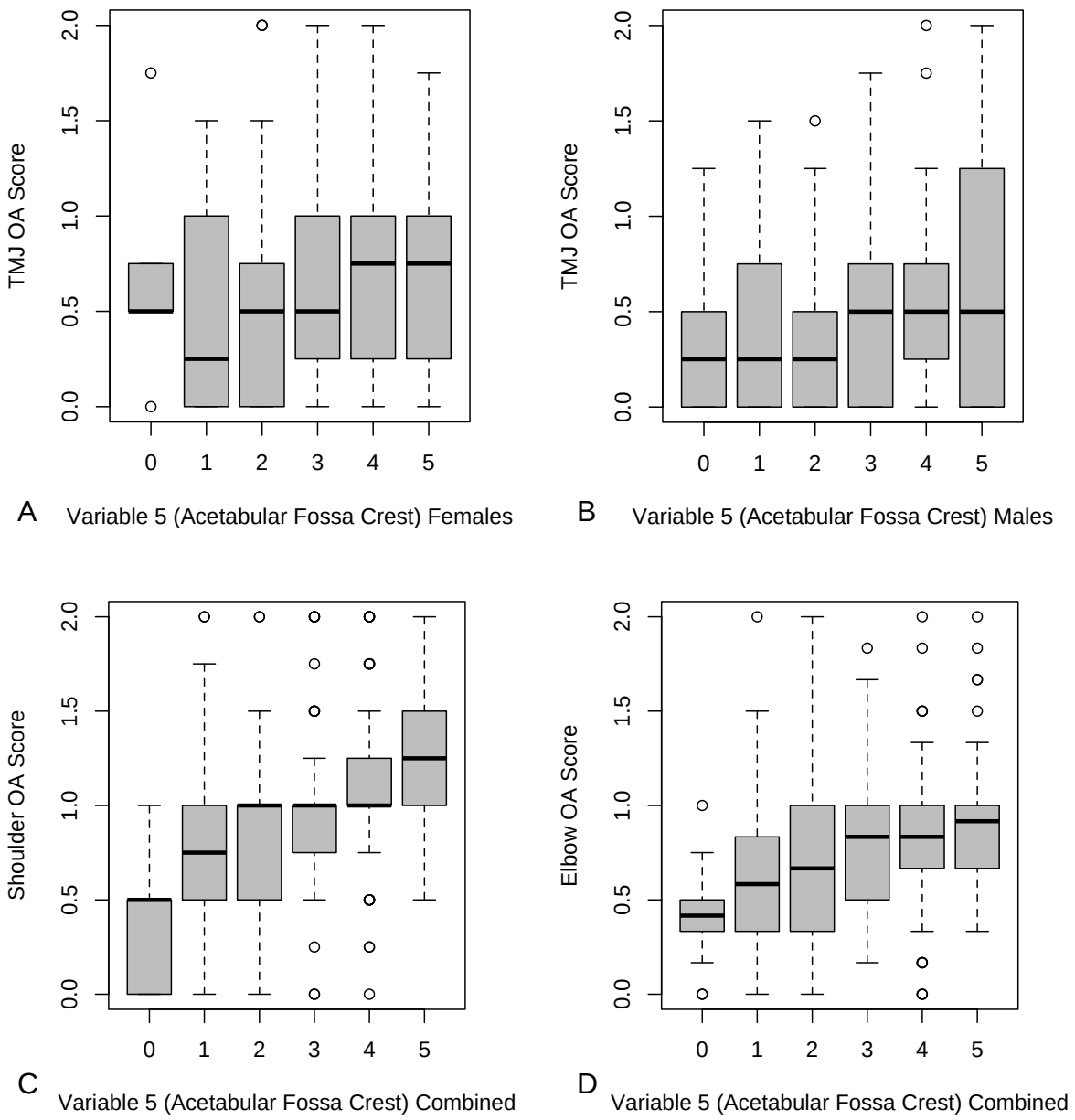


Figure 6-25. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for OA score per acetabular score (Variable 5). A) Female TMJ OA, B) Male TMJ OA, C) Combined shoulder OA, D) Combined elbow OA.

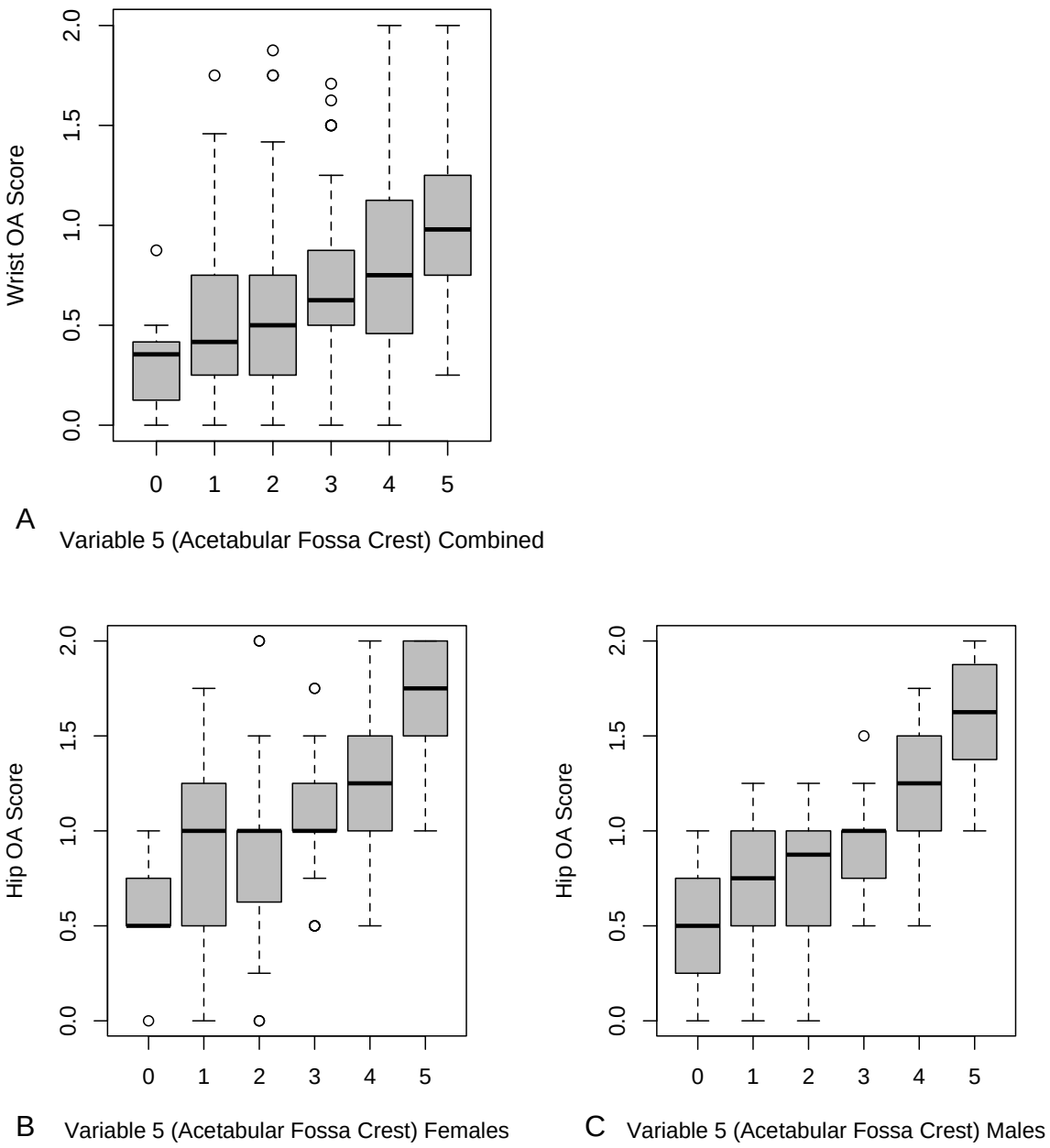


Figure 6-26. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for OA score per acetabular score (Variable 5). A) Combined wrist OA, B) Female hip OA, C) Male hip OA.

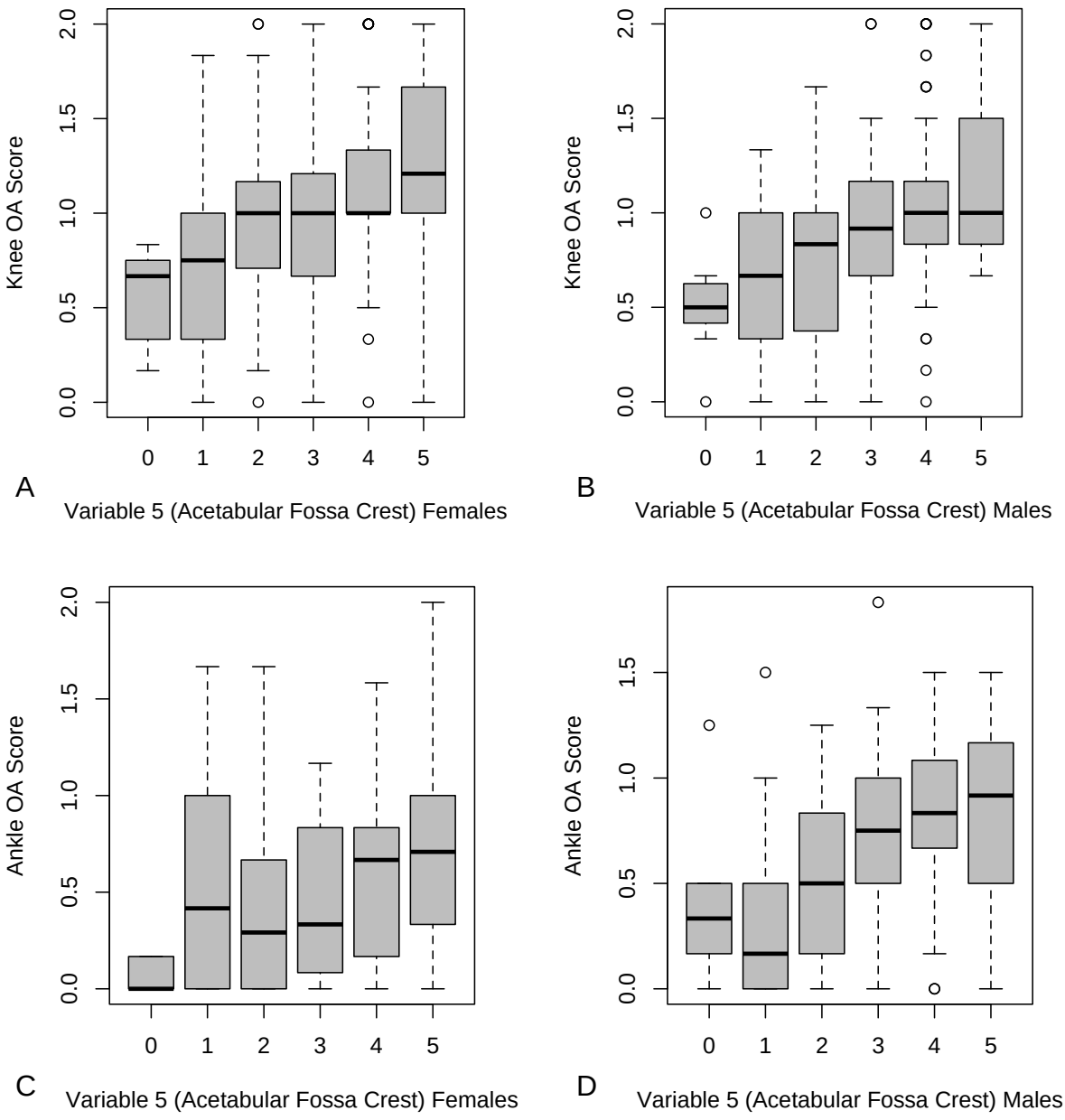


Figure 6-27. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for OA score per acetabular score (Variable 5). A) Female knee OA, B) Male knee OA, C) Female ankle OA, D) Male ankle OA.

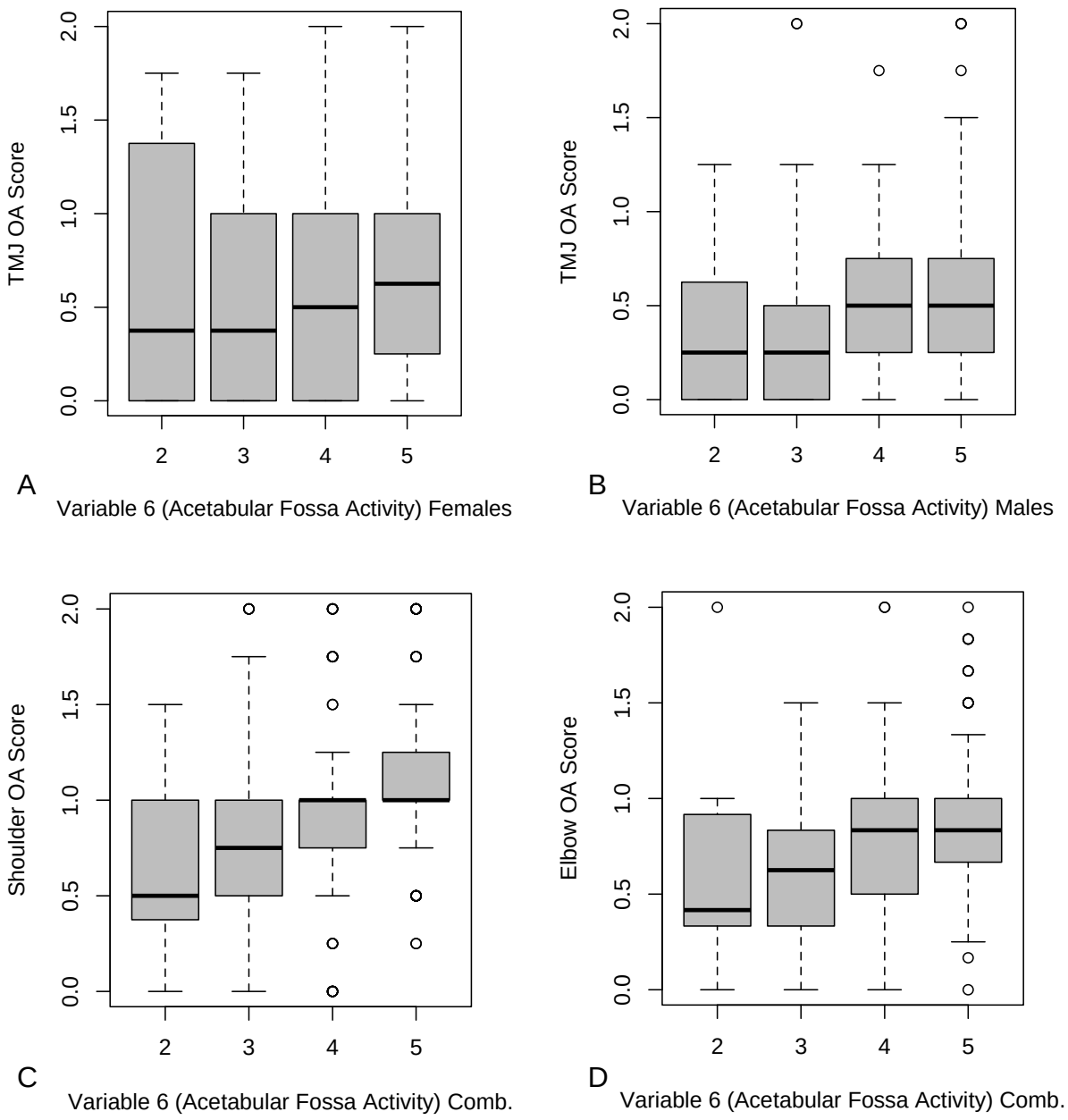


Figure 6-28. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for OA score per acetabular score (Variable 6). A) Female TMJ OA, B) Male TMJ OA, C) Combined shoulder OA, D) Combined elbow OA.

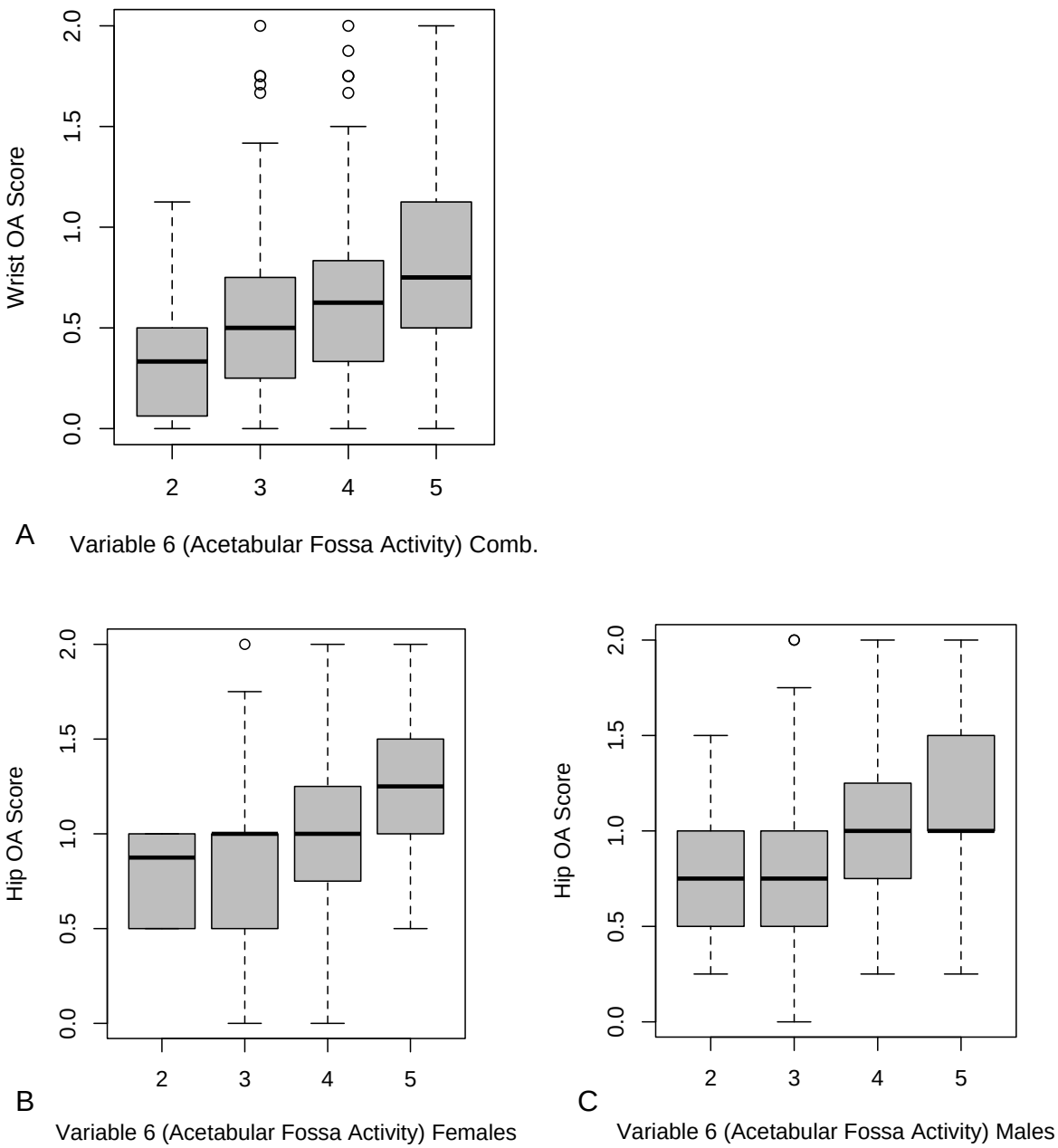


Figure 6-29. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for OA score per acetabular score (Variable 6). A) Combined wrist OA, B) Female hip OA, C) Male hip OA.

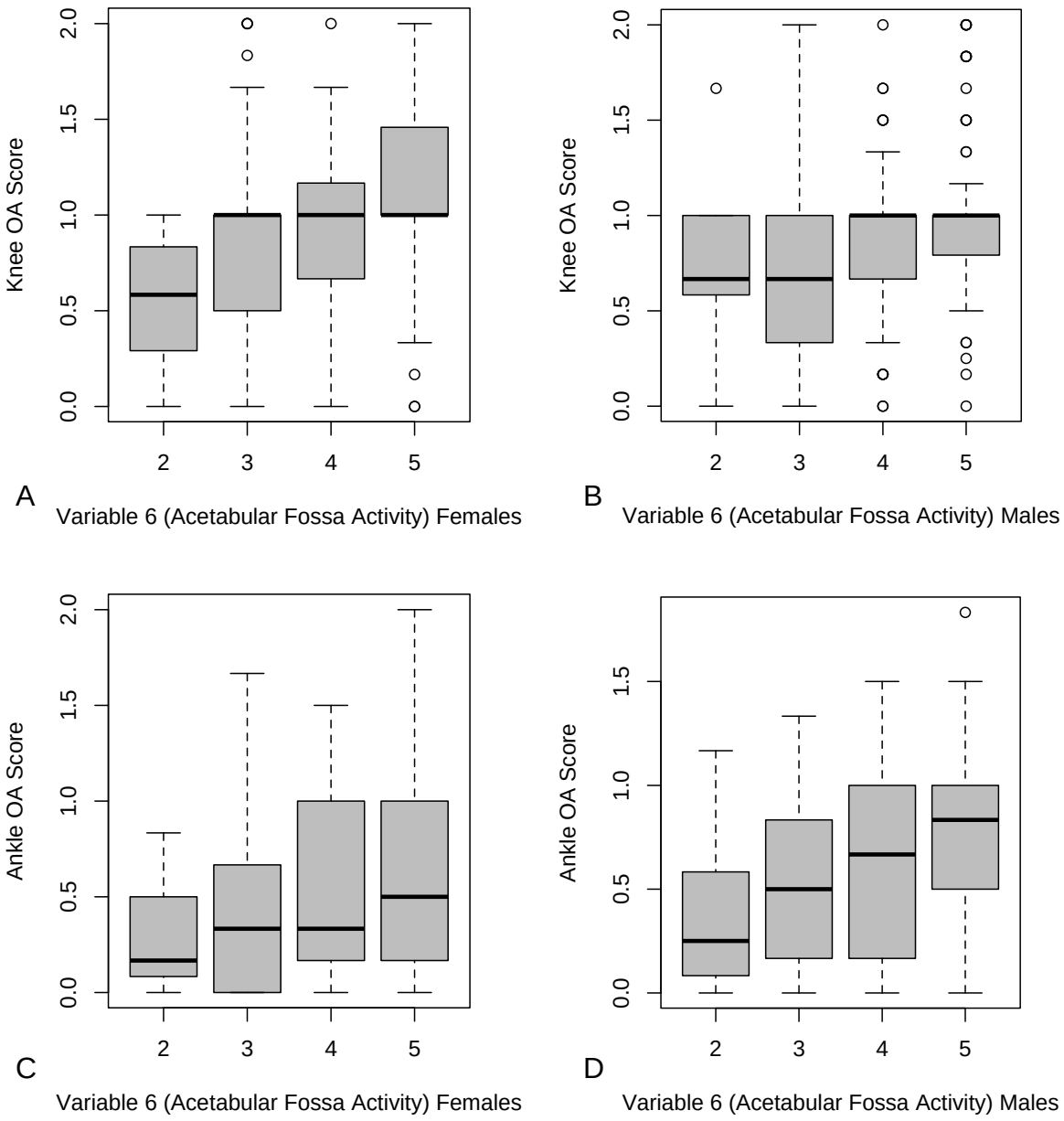


Figure 6-30. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for OA score per acetabular score (Variable 6). A) Female knee OA, B) Male knee OA, C) Female ankle OA, D) Male ankle OA.

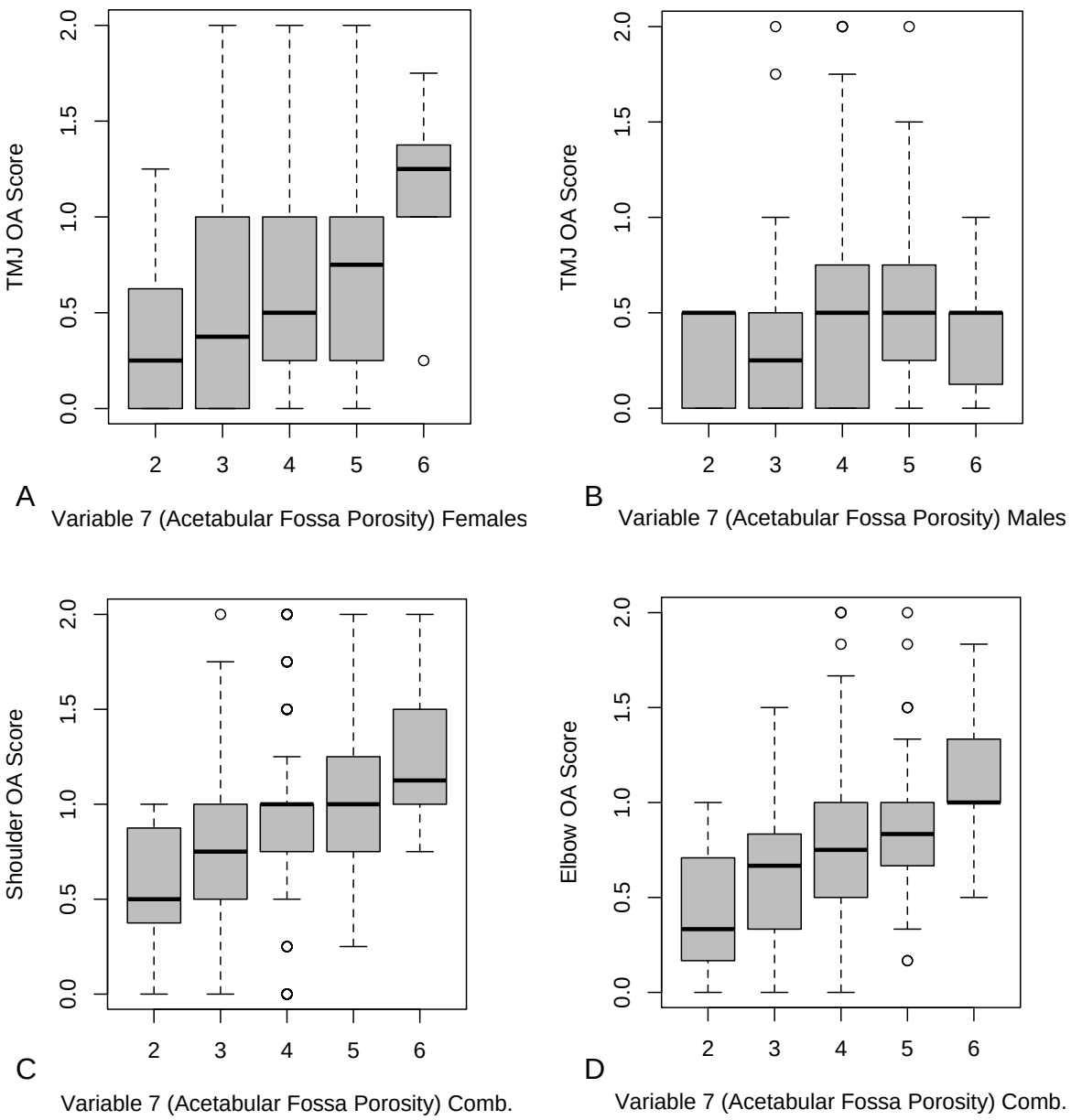
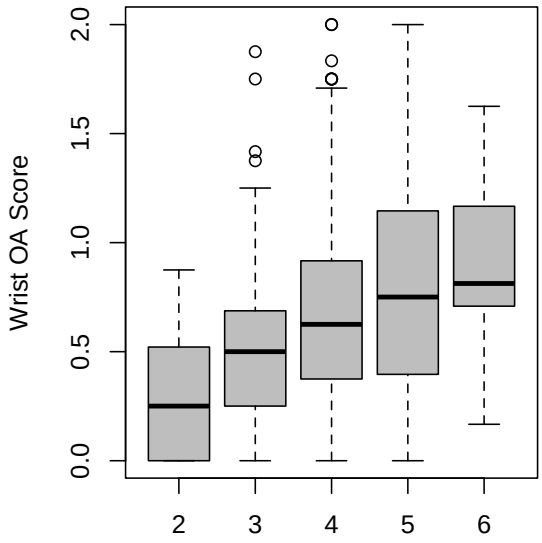
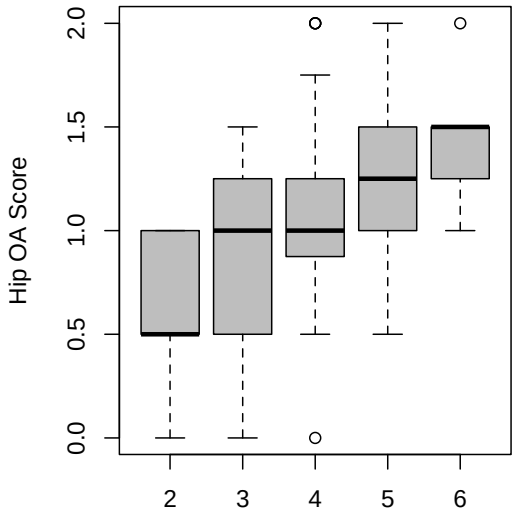


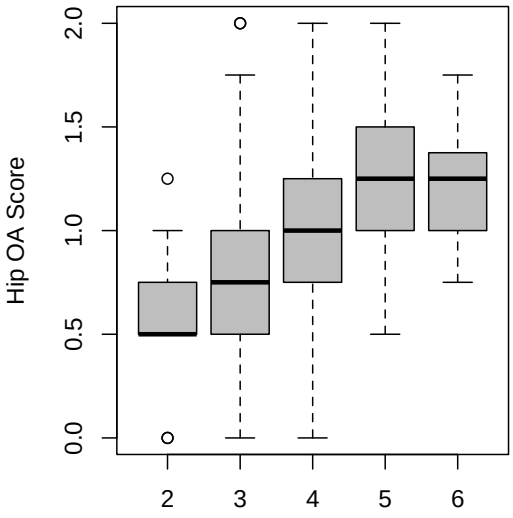
Figure 6-31. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for OA score per acetabular score (Variable 7). A) Female TMJ OA, B) Male TMJ OA, C) Combined shoulder OA, D) Combined elbow OA.



A Variable 7 (Acetabular Fossa Porosity) Comb.



B Variable 7 (Acetabular Fossa Porosity) Females



C Variable 7 (Acetabular Fossa Porosity) Males

Figure 6-32. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for OA score per acetabular score (Variable 7). A) Combined wrist OA, B) Female hip OA, C) Male hip OA.

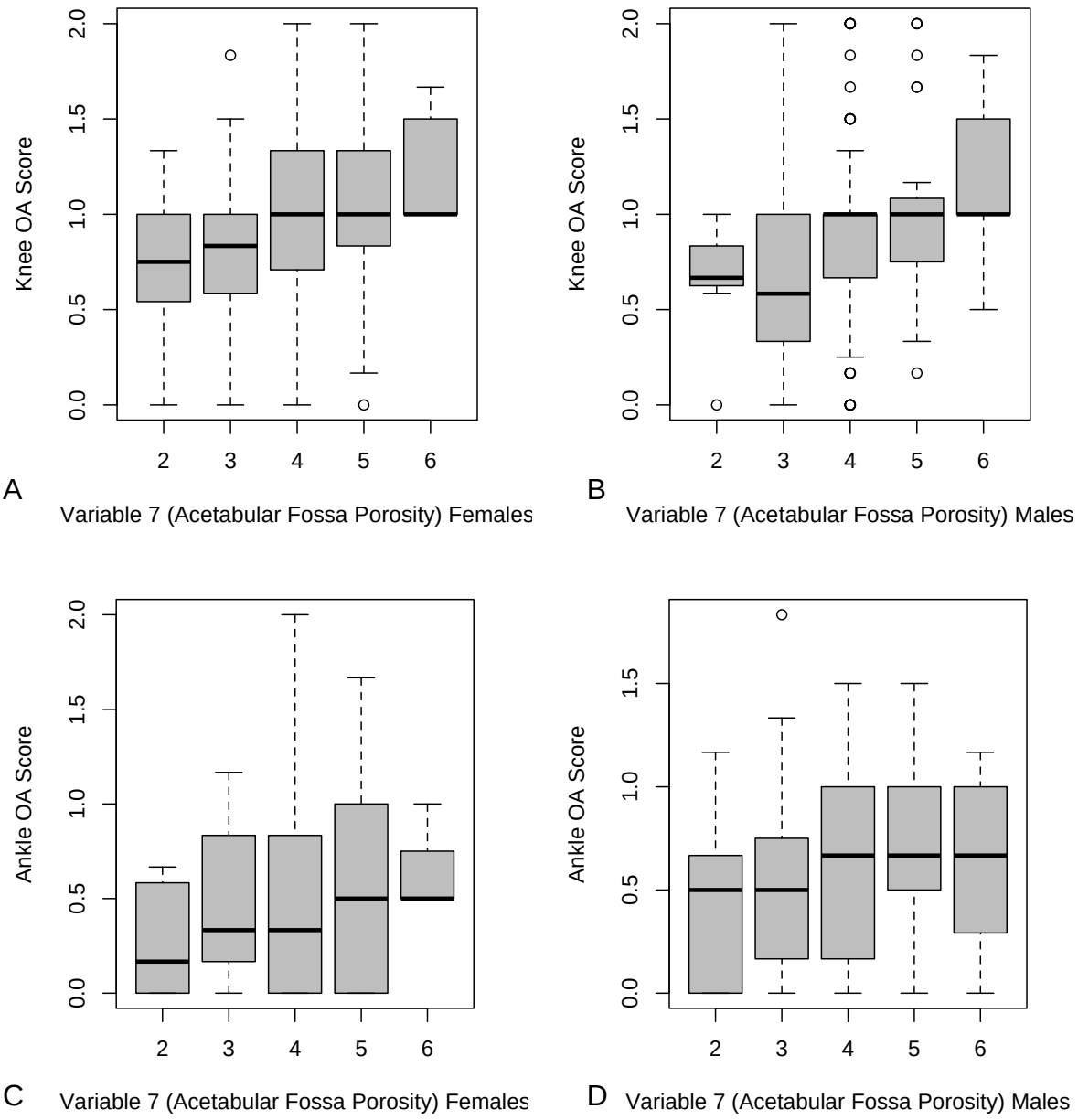


Figure 6-33. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for OA score per acetabular score (Variable 7). A) Female knee OA, B) Male knee OA, C) Female ankle OA, D) Male ankle OA.

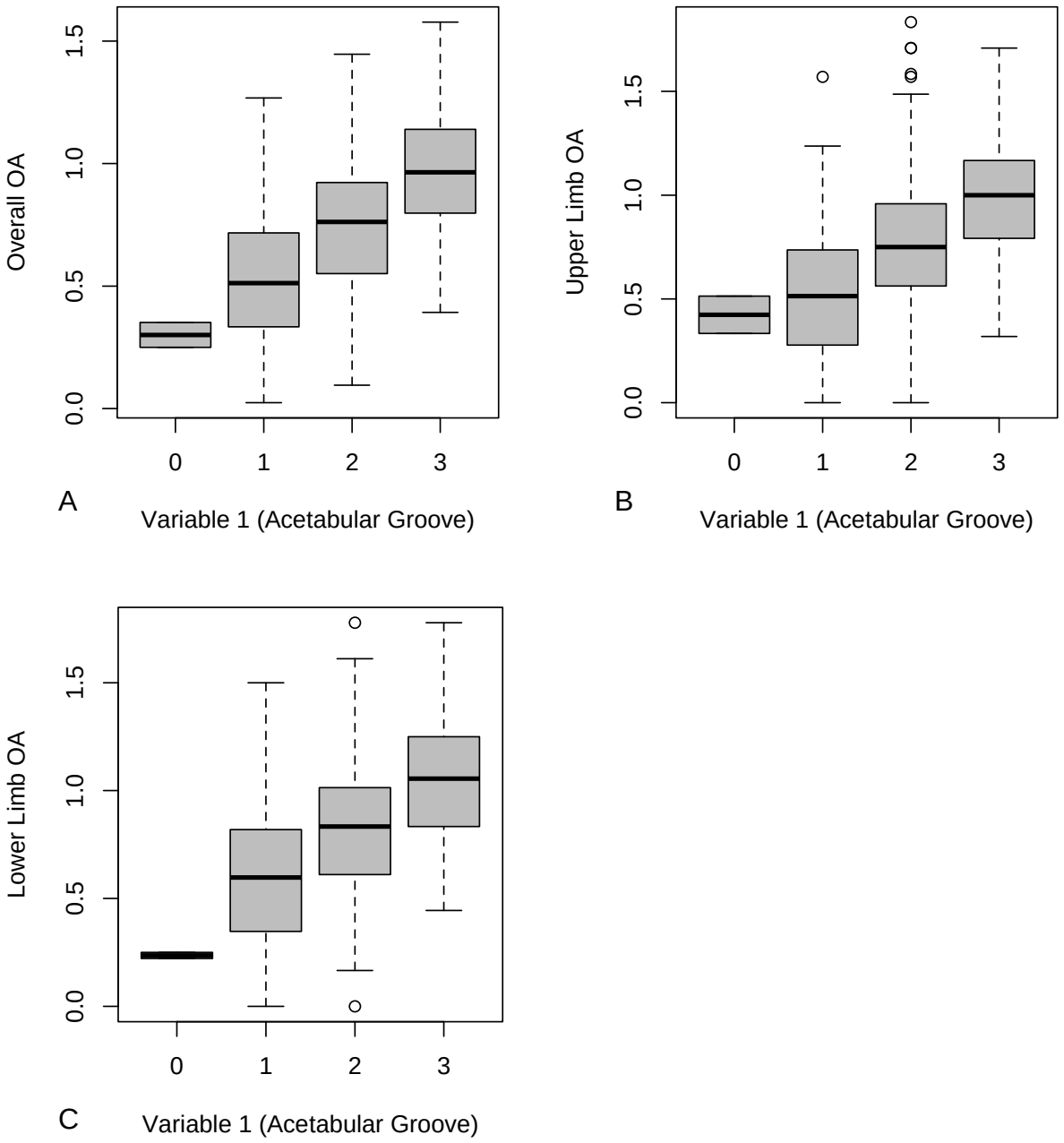


Figure 6-34. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for summary OA score per acetabular score in the combined sample (Variable 1). A) Overall OA, B) Upper limb OA, C) Lower limb OA.

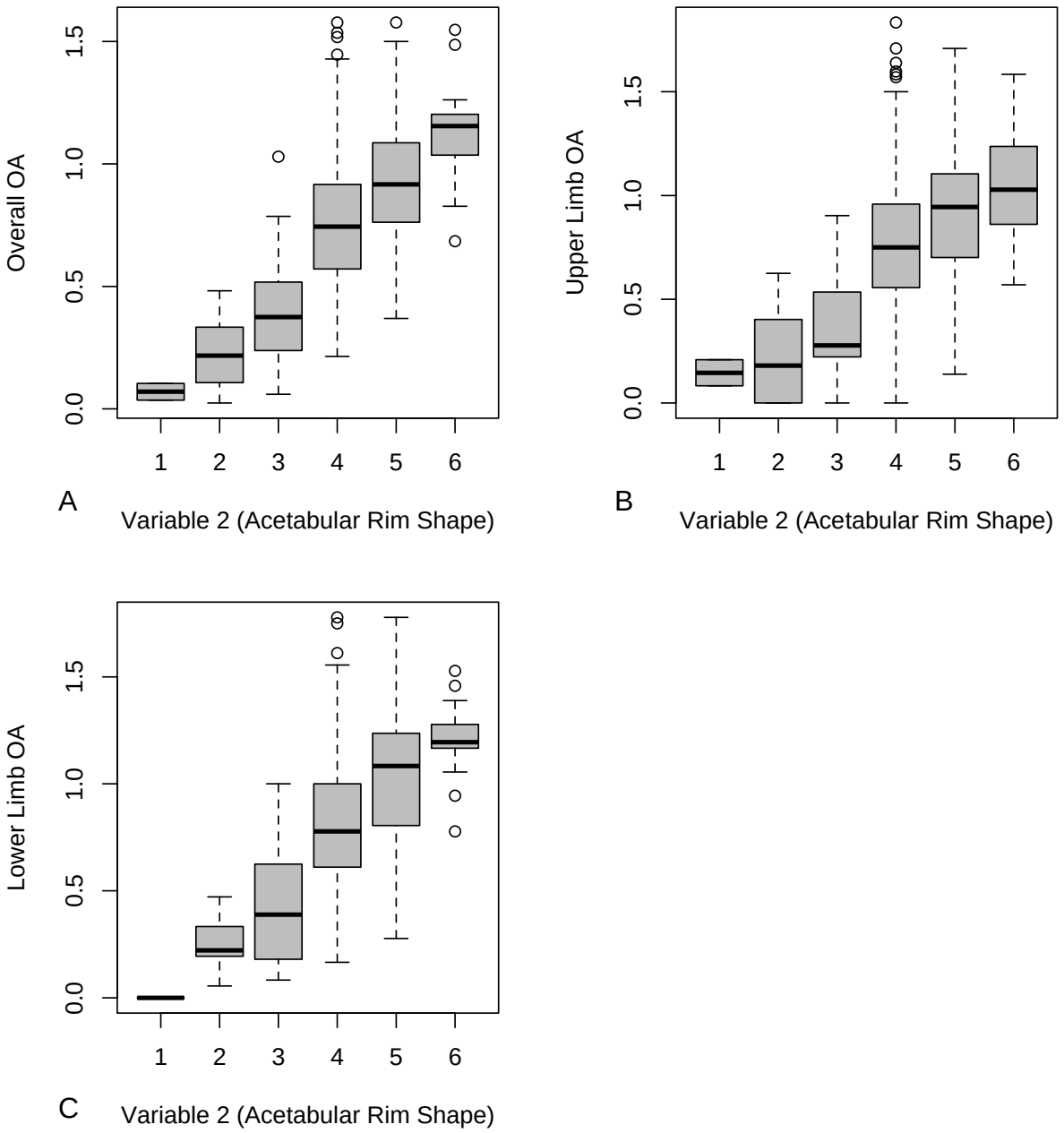


Figure 6-35. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for summary OA score per acetabular score in the combined sample (Variable 2). A) Overall OA, B) Upper limb OA, C) Lower limb OA.

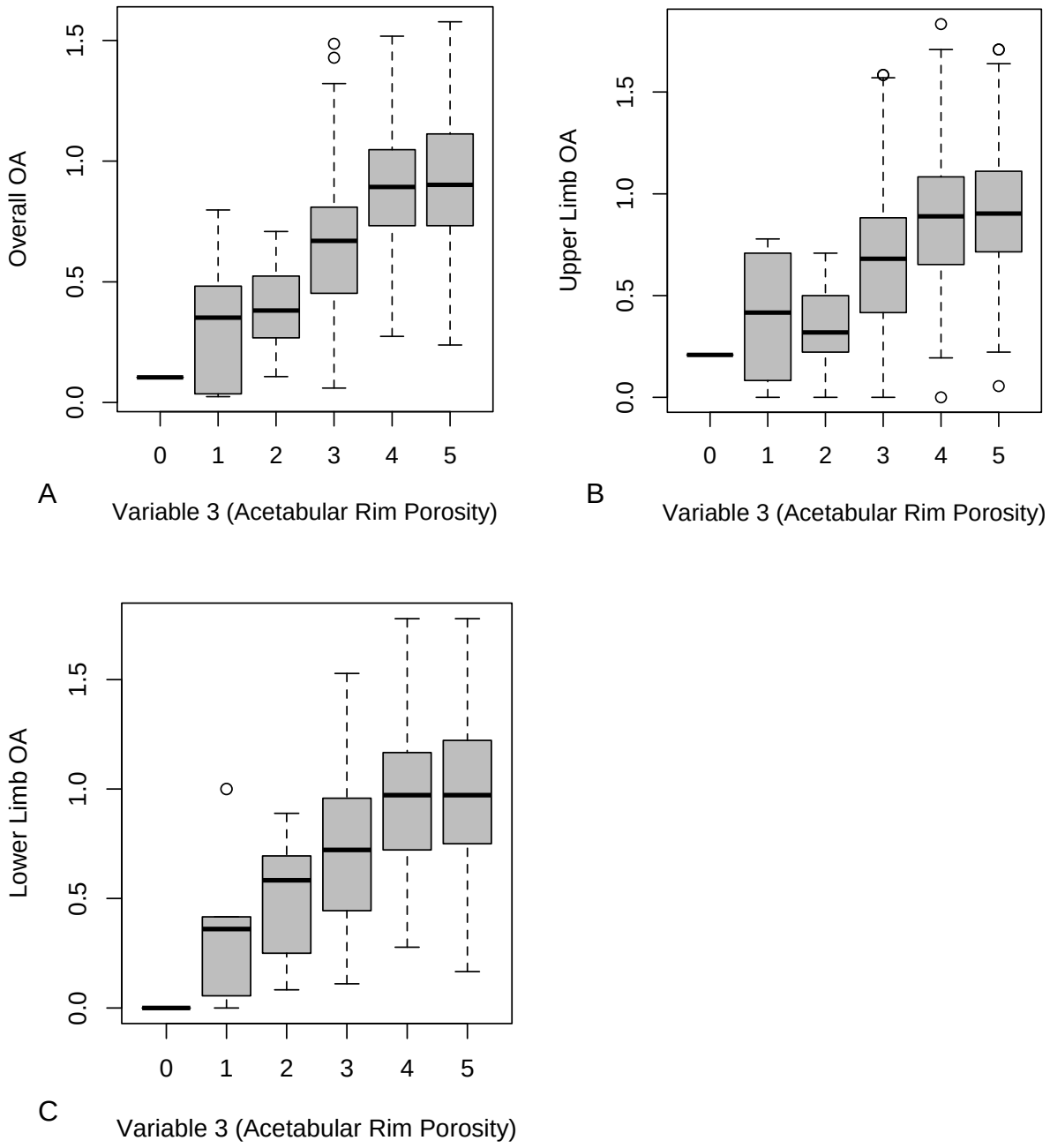


Figure 6-36. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for summary OA score per acetabular score in the combined sample (Variable 3). A) Overall OA, B) Upper limb OA, C) Lower limb OA.

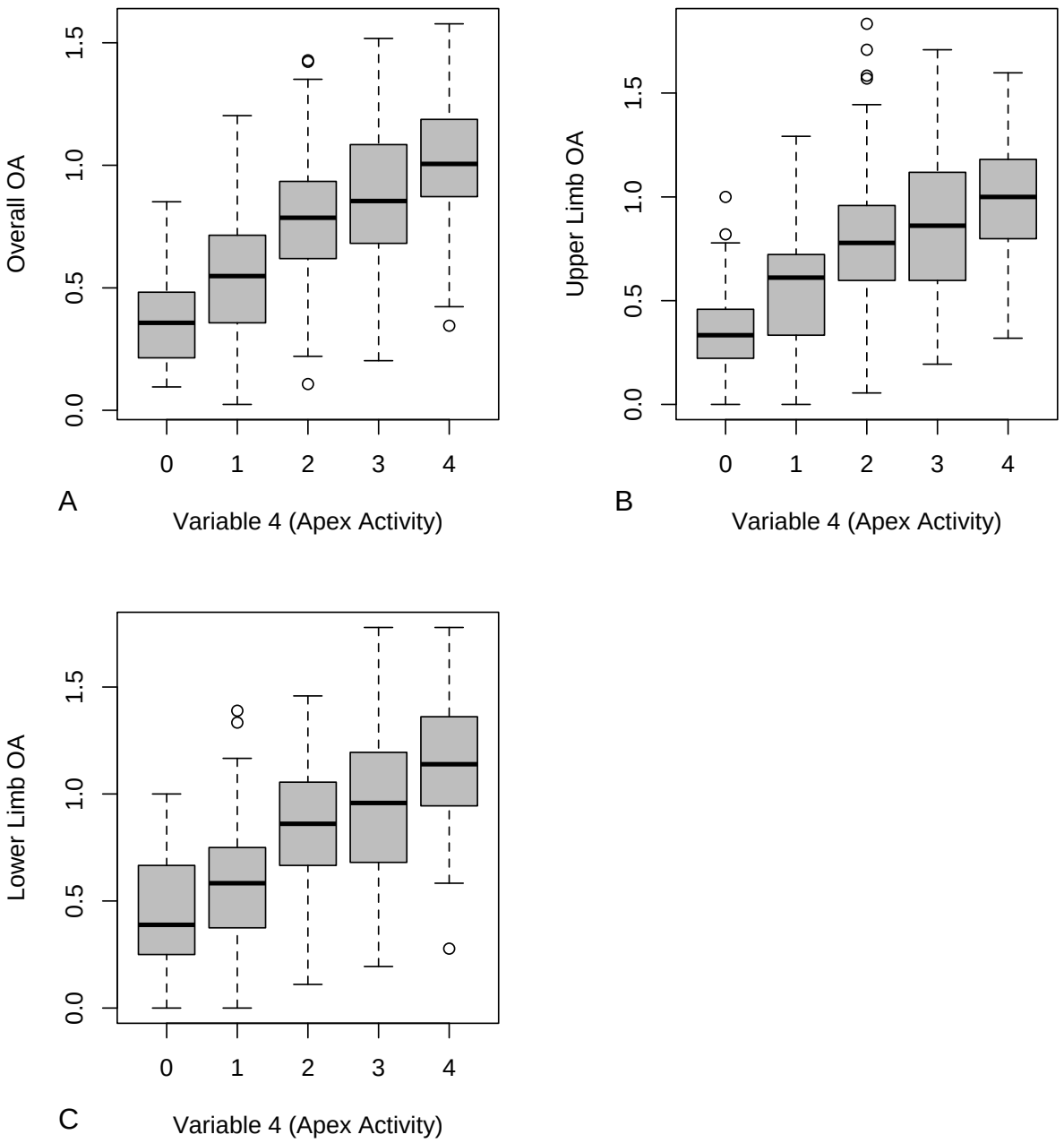


Figure 6-37. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for summary OA score per acetabular score in the combined sample (Variable 4). A) Overall OA, B) Upper limb OA, C) Lower limb OA.

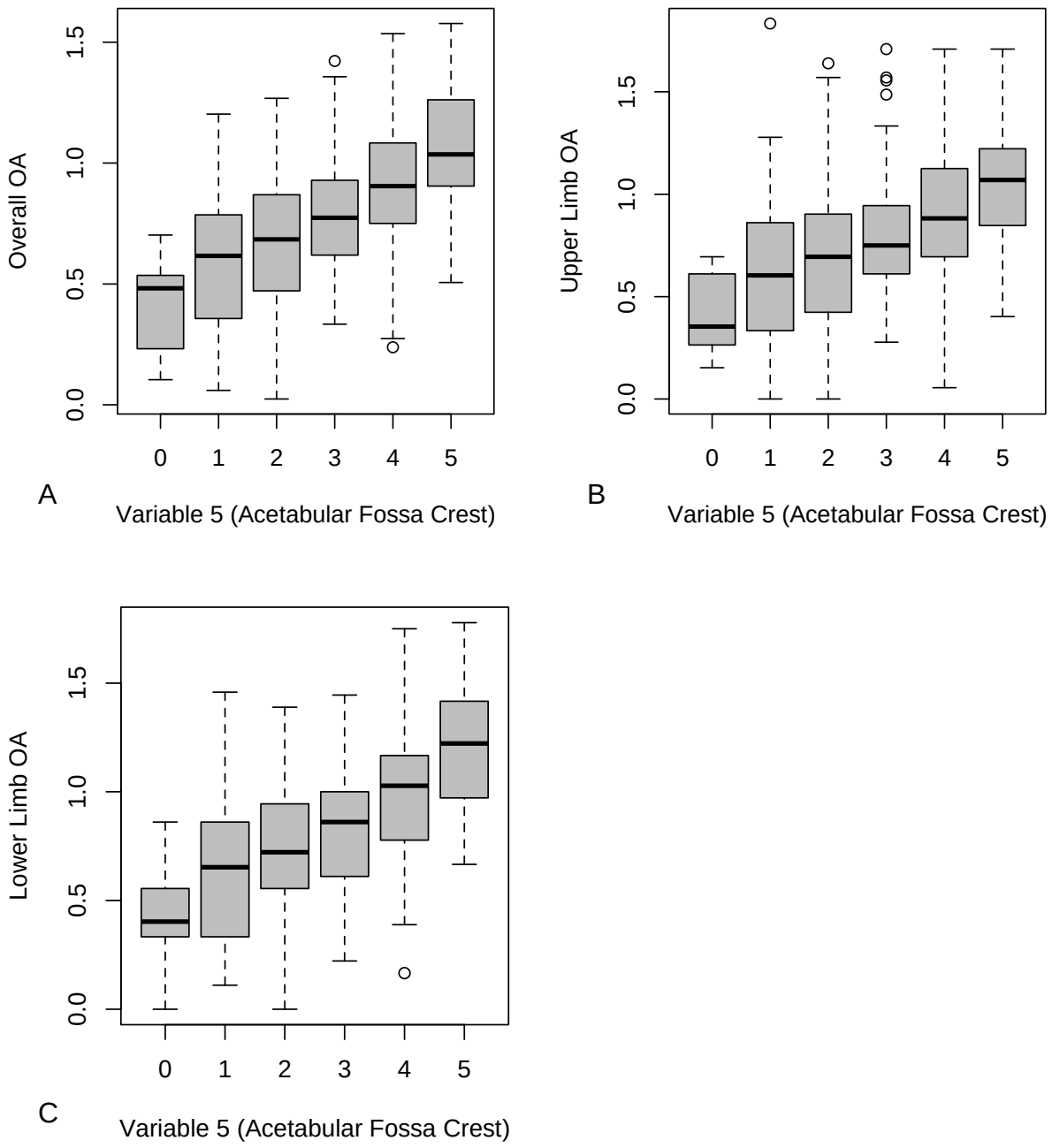


Figure 6-38. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for summary OA score per acetabular score in the combined sample (Variable 5). A) Overall OA, B) Upper limb OA, C) Lower limb OA.

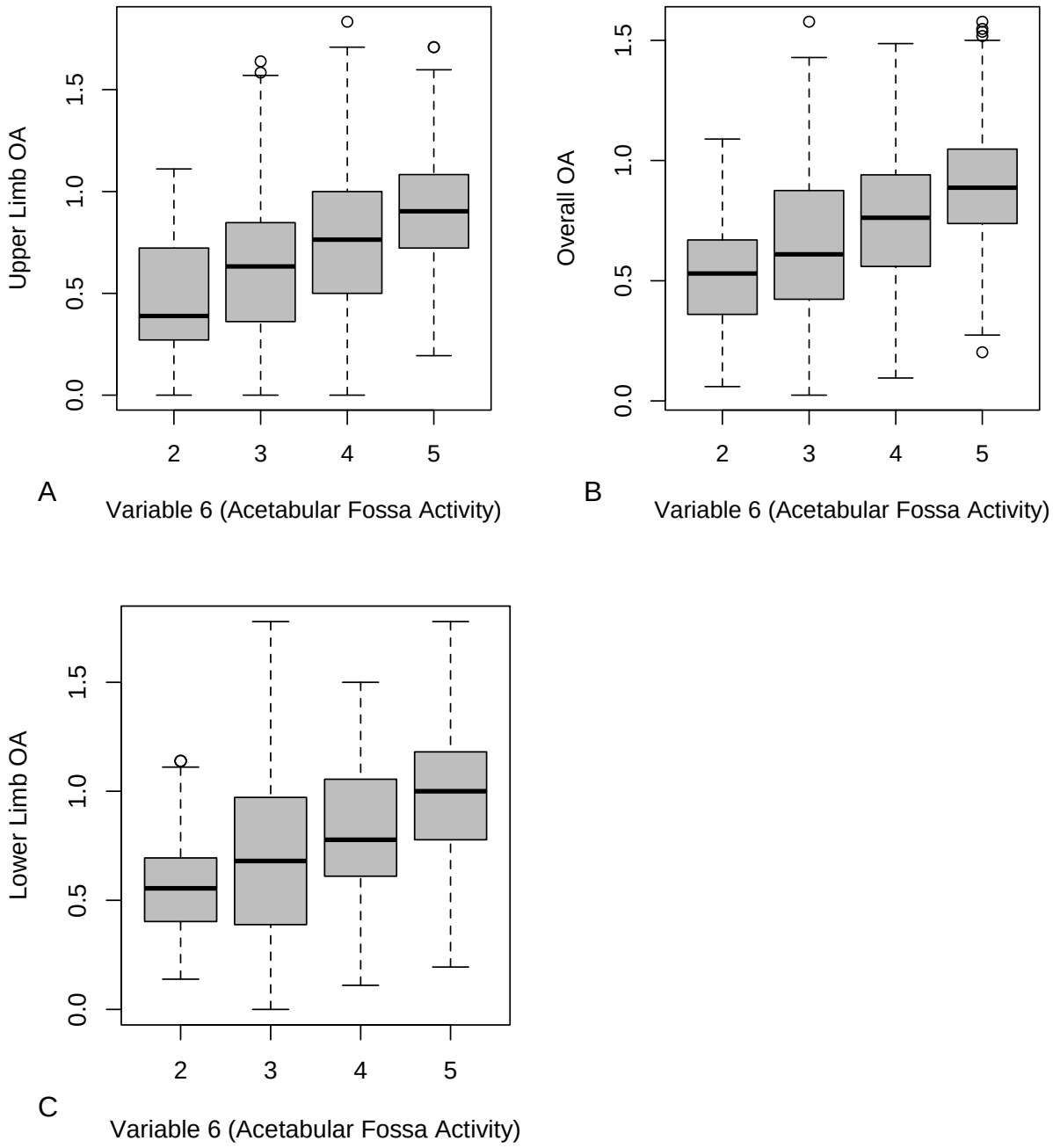


Figure 6-39. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for summary OA score per acetabular score in the combined sample (Variable 6). A) Overall OA, B) Upper limb OA, C) Lower limb OA.

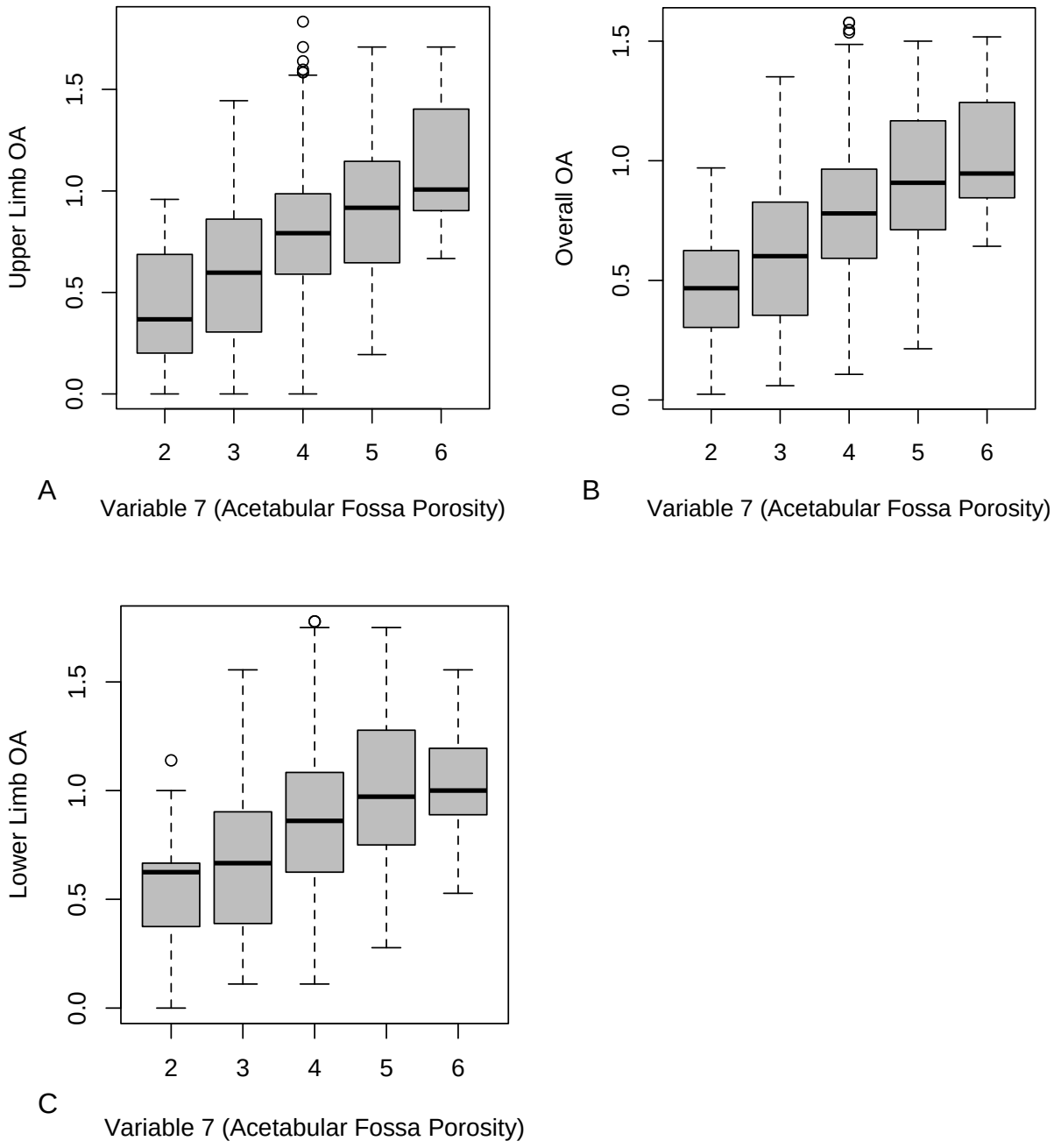


Figure 6-40. Hypothesis 1: acetabular changes correlate positively with OA; box-and-whisker plots for summary OA score per acetabular score in the combined sample (Variable 7). A) Overall OA, B) Upper limb OA, C) Lower limb OA.

Table 6-5. Spearman's rank-order correlations for acetabular Variable 1 and OA in the various joints in females, males, and the combined sample.

Joint	*rho* (female)	*rho* (male)	*rho* (combined)
TMJ	0.309*	0.281*	NA
Shoulder	NA	NA	0.426*
Elbow	NA	NA	0.371*
Wrist	NA	NA	0.424*
Hip	0.515*	0.537*	NA
Knee	0.265*	0.380*	NA
Ankle	0.254*	0.357*	NA

*Statistically significant at α=0.0007 (Bonferroni correction: α=0.05/70).
NA indicates that tests were not conducted (the combined-sex sample was only used in testing when no sex differences were detected in OA distributions).

Table 6-6. Spearman's rank-order correlations for acetabular Variable 2 and OA in the various joints in females, males, and the combined sample.

Joint	*rho* (female)	*rho* (male)	*rho* (combined)
TMJ	0.089	0.215	NA
Shoulder	NA	NA	0.372*
Elbow	NA	NA	0.329*
Wrist	NA	NA	0.353*
Hip	0.553*	0.629*	NA
Knee	0.302*	0.445*	NA
Ankle	0.245	0.319*	NA

*Statistically significant at α=0.0007 (Bonferroni correction: α=0.05/70).
NA indicates that tests were not conducted (the combined-sex sample was only used in testing when no sex differences were detected in OA distributions).

Table 6-7. Spearman's rank-order correlations for acetabular Variable 3 and OA in the various joints in females, males, and the combined sample.

Joint	*rho* (female)	*rho* (male)	*rho* (combined)
TMJ	0.331*	0.252*	NA
Shoulder	NA	NA	0.422*
Elbow	NA	NA	0.346*
Wrist	NA	NA	0.371*
Hip	0.510*	0.576*	NA
Knee	0.227	0.432*	NA
Ankle	0.170	0.221	NA

*Statistically significant at α=0.0007 (Bonferroni correction: α=0.05/70).
NA indicates that tests were not conducted (the combined-sex sample was only used in testing when no sex differences were detected in OA distributions).

Table 6-8. Spearman's rank-order correlations for acetabular Variable 4 and OA in the various joints in females, males, and the combined sample.

Joint	*rho* (female)	*rho* (male)	*rho* (combined)
TMJ	0.206	0.234	NA
Shoulder	NA	NA	0.424*
Elbow	NA	NA	0.286*
Wrist	NA	NA	0.381*
Hip	0.472*	0.645*	NA
Knee	0.326*	0.485*	NA
Ankle	0.192	0.394*	NA

*Statistically significant at α=0.0007 (Bonferroni correction: α=0.05/70).
NA indicates that tests were not conducted (the combined-sex sample was only used in testing when no sex differences were detected in OA distributions).

Table 6-9. Spearman's rank-order correlations for acetabular Variable 5 and OA in the various joints in females, males, and the combined sample.

Joint	*rho* (female)	*rho* (male)	*rho* (combined)
TMJ	0.188	0.198	NA
Shoulder	NA	NA	0.368*
Elbow	NA	NA	0.306*
Wrist	NA	NA	0.397*
Hip	0.499*	0.673*	NA
Knee	0.300*	0.414*	NA
Ankle	0.209	0.478*	NA

*Statistically significant at α=0.0007 (Bonferroni correction: α=0.05/70).
NA indicates that tests were not conducted (the combined-sex sample was only used in testing when no sex differences were detected in OA distributions).

Table 6-10. Spearman's rank-order correlations for acetabular Variable 6 and OA in the various joints in females, males, and the combined sample (used in testing when no sex differences were detected in OA distributions).

Joint	*rho* (female)	*rho* (male)	*rho* (combined)
TMJ	0.169	0.194	NA
Shoulder	NA	NA	0.323*
Elbow	NA	NA	0.300*
Wrist	NA	NA	0.318*
Hip	0.417*	0.330*	NA
Knee	0.310*	0.249*	NA
Ankle	0.173	0.328*	NA

*Statistically significant at α=0.0007 (Bonferroni correction: α=0.05/70).
NA indicates that tests were not conducted (the combined-sex sample was only used in testing when no sex differences were detected in OA distributions).

Table 6-11. Spearman's rank-order correlations for acetabular Variable 7 and OA in the various joints in females, males, and the combined sample (used in testing when no sex differences were detected in OA distributions).

Joint	*rho* (female)	*rho* (male)	*rho* (combined)
TMJ	0.229	0.158	NA
Shoulder	NA	NA	0.329*
Elbow	NA	NA	0.268*
Wrist	NA	NA	0.265*
Hip	0.371*	0.363*	NA
Knee	0.241	0.300*	NA
Ankle	0.112	0.176	NA

*Statistically significant at α=0.0007 (Bonferroni correction: α=0.05/70).
NA indicates that tests were not conducted (the combined-sex sample was only used in testing when no sex differences were detected in OA distributions).

Table 6-12. Spearman's rank-order correlations for acetabular Variable 1 through Variable 7 and summary OA in the combined sample.

Summary OA	*rho* (1)	*rho* (2)	*rho* (3)	*rho* (4)	*rho* (5)	*rho* (6)	*rho* (7)
Overall	0.532*	0.483*	0.495*	0.533*	0.521*	0.401*	0.367*
Upper limb	0.481*	0.417*	0.445*	0.442*	0.430*	0.377*	0.344*
Lower limb	0.469*	0.520*	0.438*	0.531*	0.538*	0.384*	0.331*

*Statistically significant at α=0.002 (Bonferroni correction: α=0.05/21).

Table 6-13. Error rates for females and males using the IDADE2 acetabular aging program (Rissech et al., 2006): percentage correctly classified, percentage prediction error, inaccuracy, bias, maximum underestimation ("Min."), maximum overestimation ("Max."), and *rho* values between known and estimated ages.

Group	*n*	Correctly Classified	Prediction Error	Inacc. (yrs.)	Bias (yrs.)	Min. (yrs.)	Max. (yrs.)	*Rho*
Females <60	41	44%	36%	16.1	13.4	-17.4	38.5	0.243
Females 60+	56	72%	16%	12.0	-6.8	-34	26	0.353*
All females	97	60%	24%	13.6	1.3	-34	38.5	0.414*
Males <60	53	60%	30%	12.6	7.8	-17	37.6	0.420*
Males 60+	51	66%	16%	12.3	-9.7	-42	10.7	0.394*
All males	104	63%	23%	12.4	-1.1	-42	37.6	0.562*

*Statistically significant at α=0.02 (Bonferroni correction: α=0.05/3).

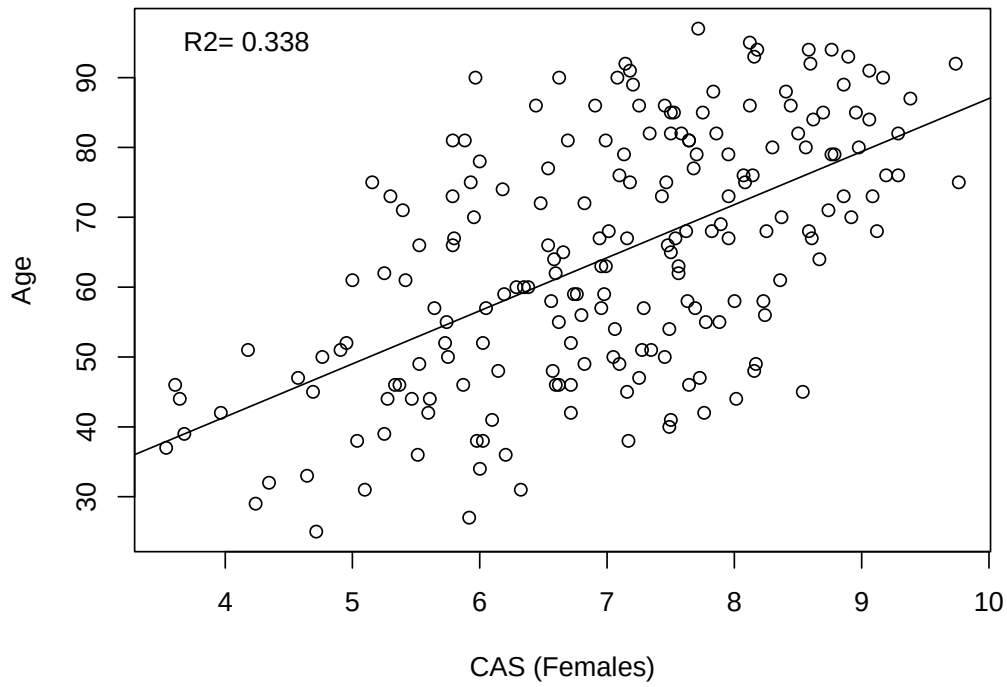


Figure 6-41. Hypothesis 2a: acetabular changes correlate positively with age; scatterplot for age and CAS in females. Black line indicates linear regression model.

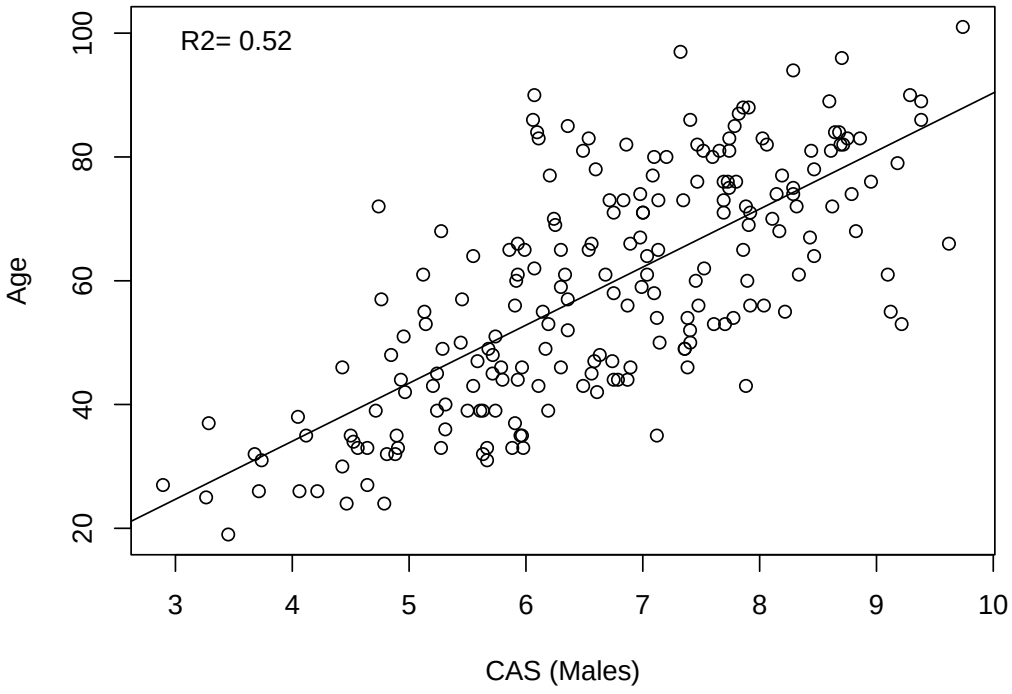


Figure 6-42. Hypothesis 2a: acetabular changes correlate positively with age; scatterplot for age and CAS in males. Black line indicates linear regression model.

Table 6-14. Spearman's rank-order correlations for age and CAS in the female and male samples.

Variable	*rho*
Female CAS	0.565*
Male CAS	0.713*

*Statistically significant at the α=0.05 level.

Table 6-15. Regression results for age and CAS in the female and male samples.

Variable	Estimate	Std. Error	t value	Probability
Intercept	11.034	5.383	2.05	0.042*
Female CAS	7.600	0.754	10.07	<2e-16*
Intercept	-3.450	4.233	-0.815	0.416
Male CAS	9.379	0.621	15.093	<2e-16*

*Statistically significant at the α=0.05 level.

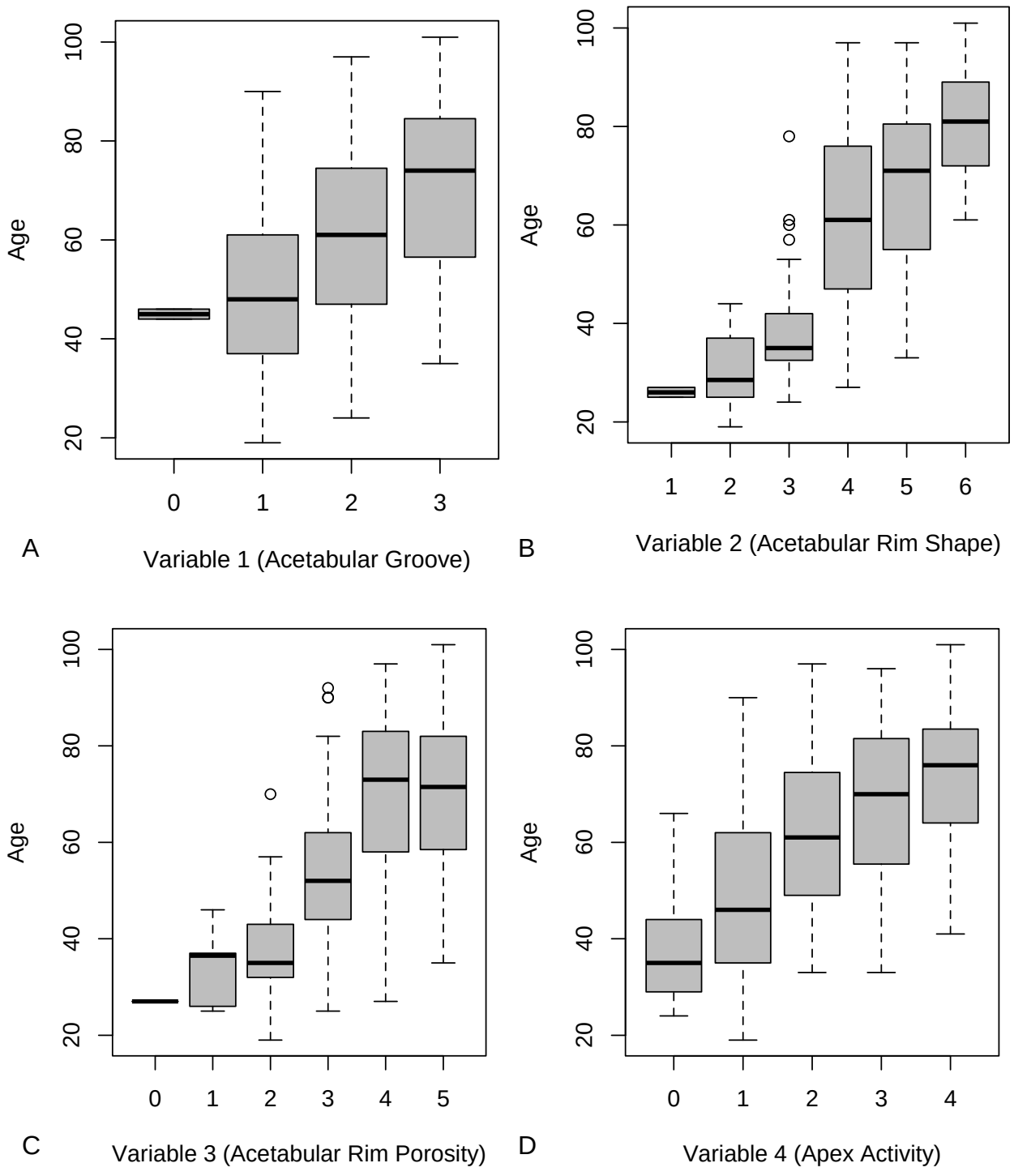


Figure 6-43. Hypothesis 2a: acetabular changes correlate positively with age; box-and-whisker plots of age per acetabular variable in the combined-sex sample (left side). A) Variable 1, B) Variable 2, C) Variable 3, D) Variable 4.

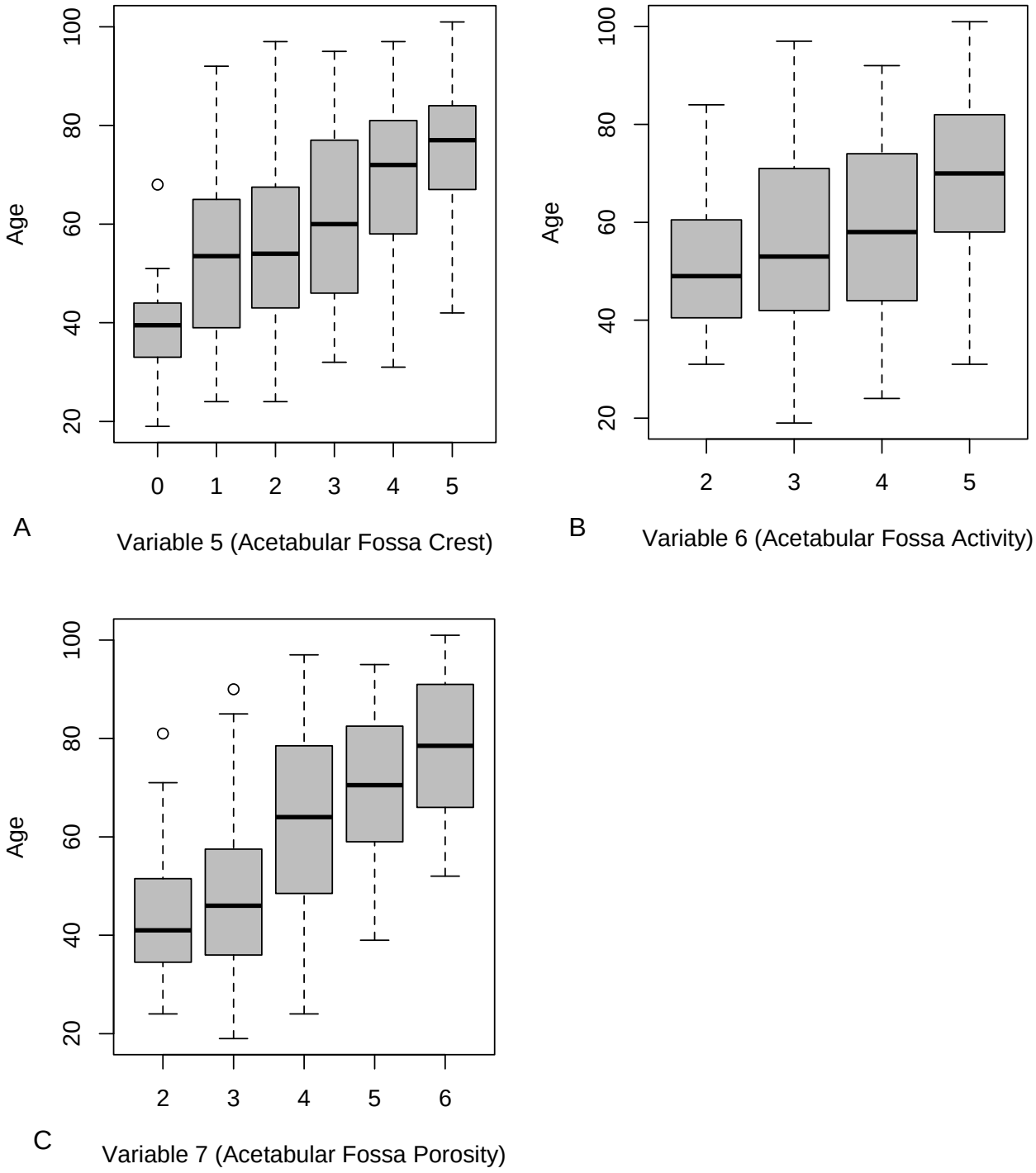


Figure 6-44. Hypothesis 2a: acetabular changes correlate positively with age; box-and-whisker plots of age per acetabular variable in the combined-sex sample (left side). A) Variable 5, B) Variable 6, C) Variable 7.

Table 6-16. Spearman's rank-order correlations for age and acetabular variables in the combined-sex sample (left variables only).

Acetabular variable	*rho*
1. Acetabular groove	0.405*
2. Acetabular rim shape	0.414*
3. Acetabular rim porosity	0.507*
4. Apex activity	0.484*
5. Acetabular fossa crest	0.434*
6. Acetabular fossa activity	0.343*
7. Acetabular fossa porosity	0.435*

*Statistically significant at α=0.007 (Bonferroni correction: α=0.05/7).

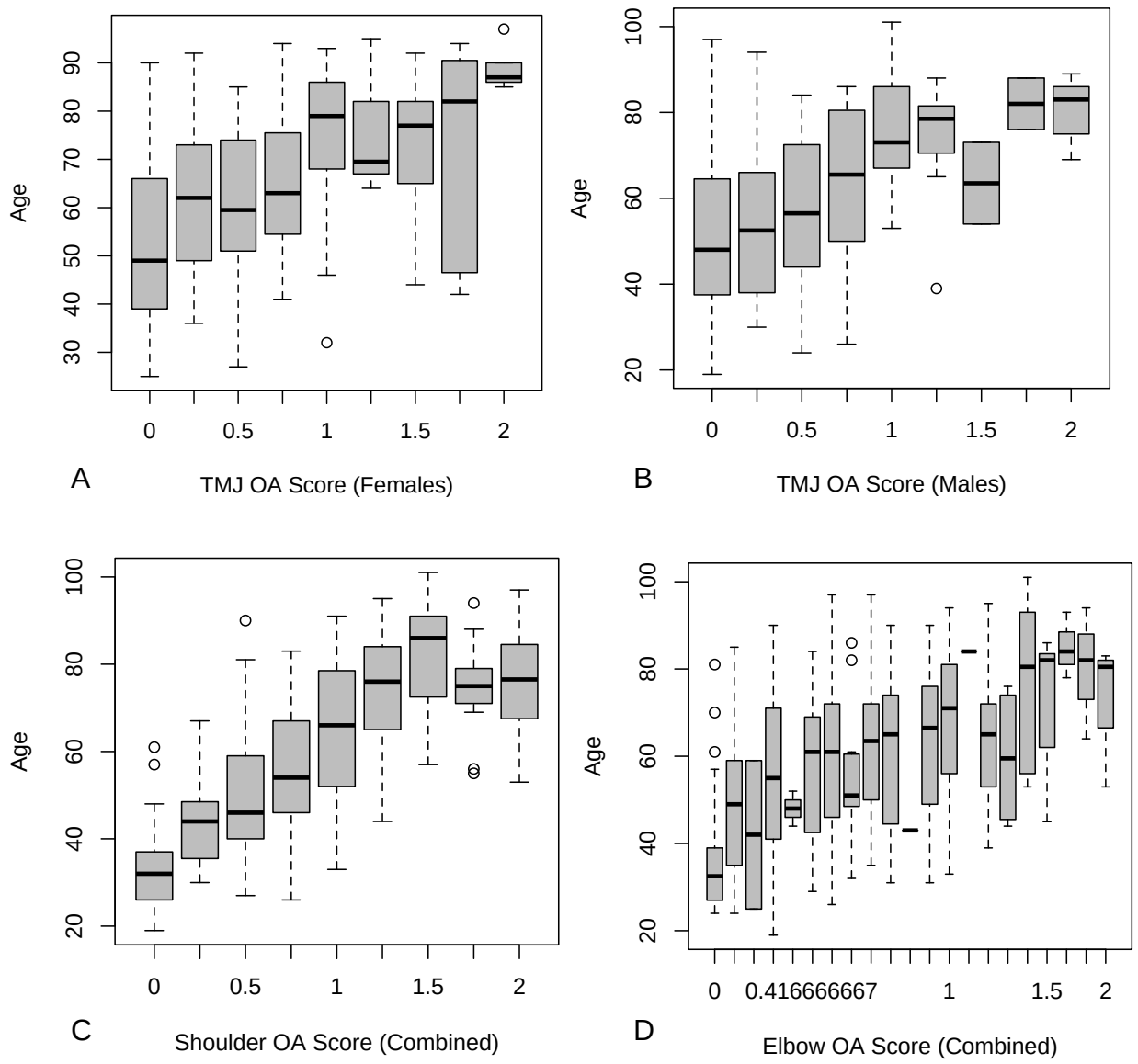


Figure 6-45. Hypothesis 2b: OA correlates positively with age; box-and-whisker plots for age per OA score. A) Female TMJ OA, B) Male TMJ OA, C) Combined shoulder OA, D) Combined elbow OA.

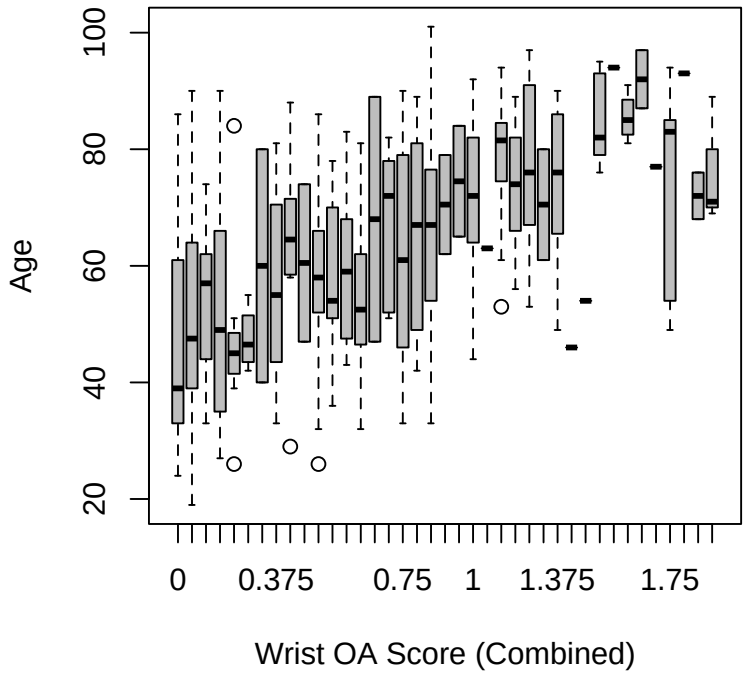


Figure 6-46. Hypothesis 2b: OA correlates positively with age; box-and-whisker plot for age per combined wrist OA score.

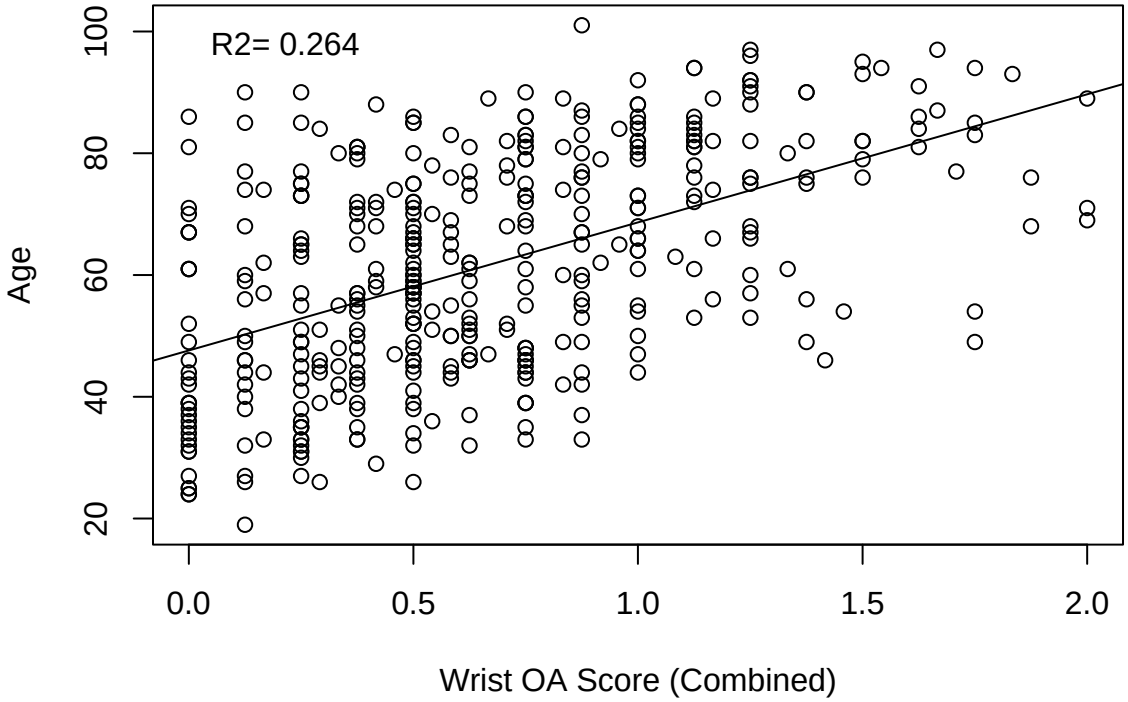


Figure 6-47. Hypothesis 2b: OA correlates positively with age; scatterplot for age and combined wrist OA. Black line indicates linear regression model.

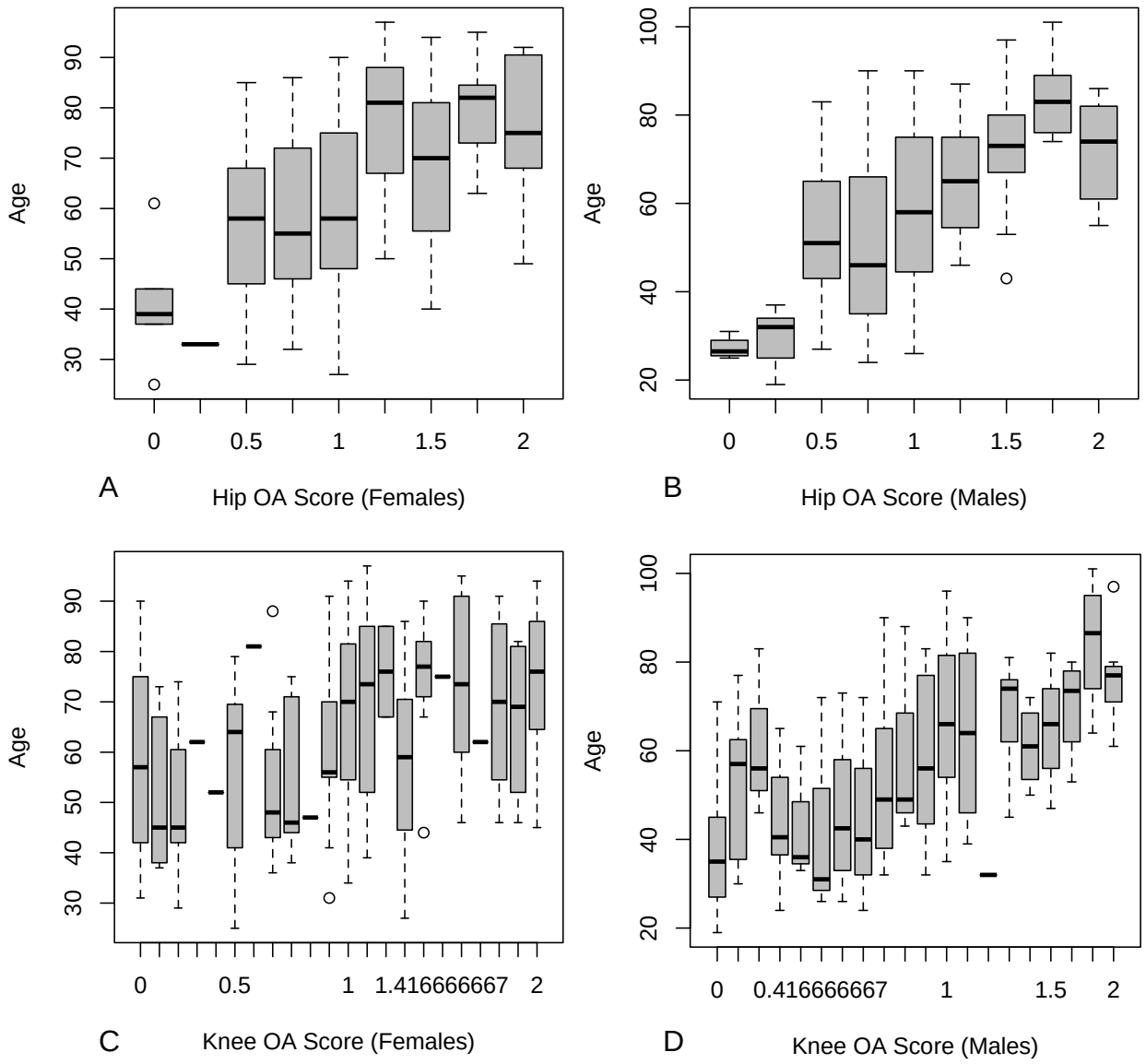


Figure 6-48. Hypothesis 2b: OA correlates positively with age; box-and-whisker plots for age per OA score. A) Female hip OA, B) Male hip OA, C) Female knee OA, D) Male knee OA.

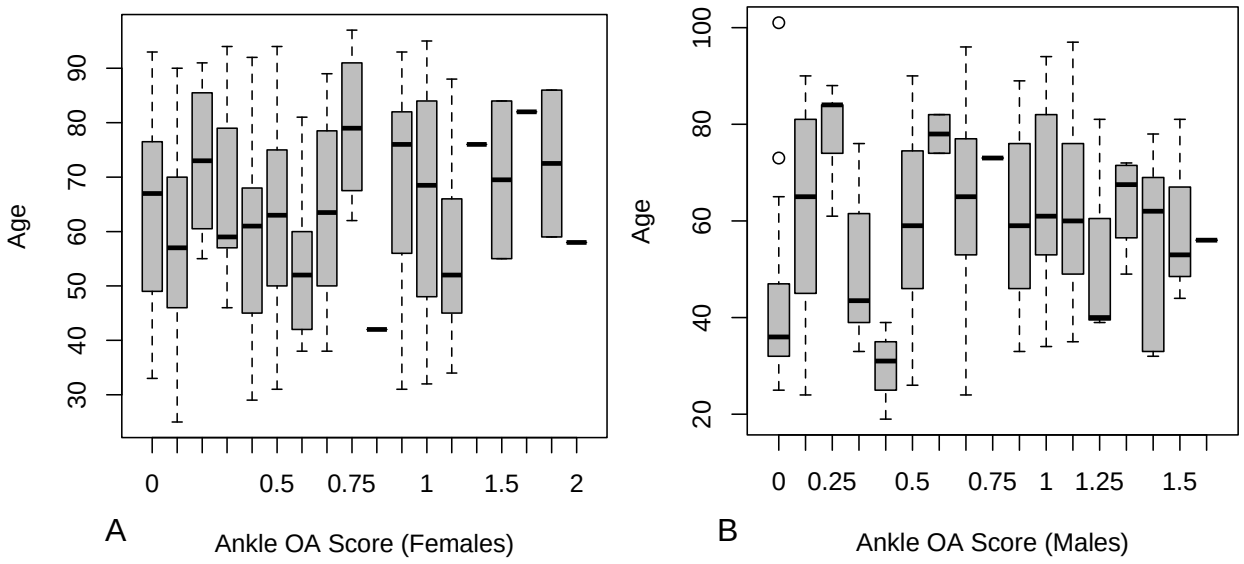
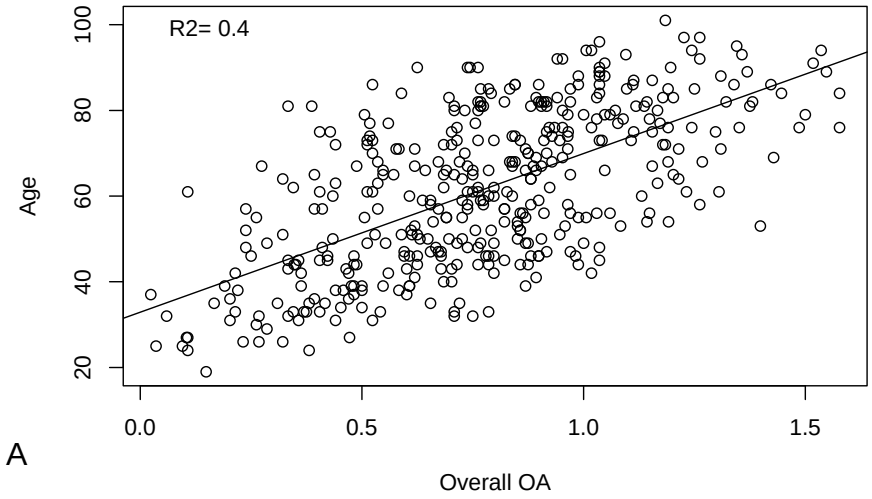


Figure 6-49. Hypothesis 2b: OA correlates positively with age; box-and-whisker plots for age per OA score. A) Female ankle OA, B) Male ankle OA.

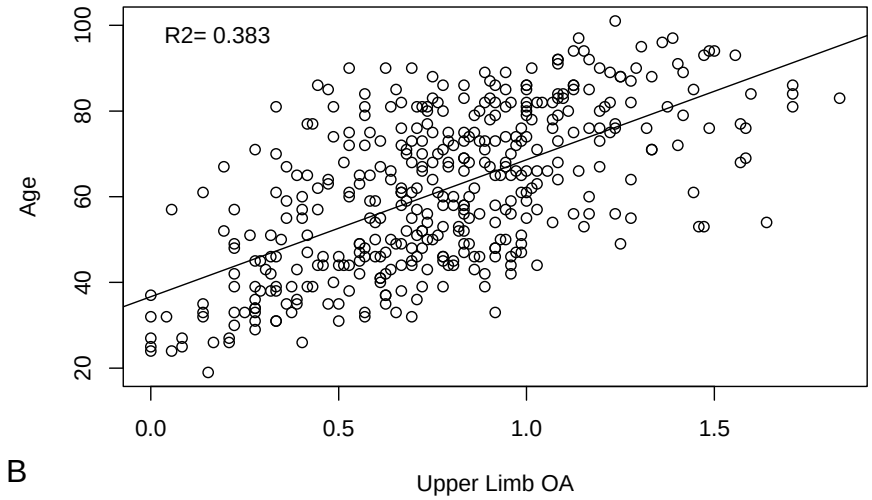
Table 6-17. Spearman's rank-order correlations for age and OA in the various joints in females, males, and the combined sample.

Joint	*rho* (female)	*rho* (male)	*rho* (combined)
TMJ	0.474*	0.409*	NA
Shoulder	NA	NA	0.613*
Elbow	NA	NA	0.386*
Wrist	NA	NA	0.508*
Hip	0.438*	0.525*	NA
Knee	0.353*	0.496*	NA
Ankle	0.063	0.237*	NA

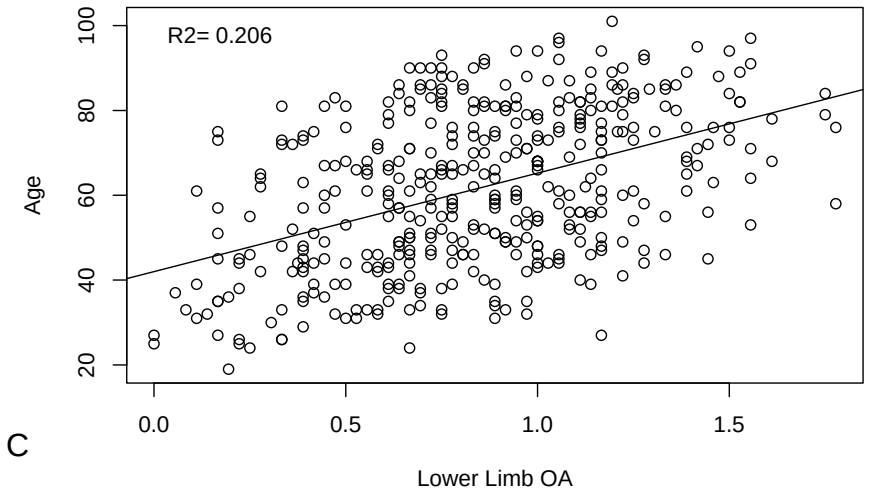
*Statistically significant at α=0.007 (Bonferroni correction: α=0.05/7).
NA indicates that tests were not conducted (the combined-sex sample was only used in testing when no sex differences were detected in OA distributions).



A



B



C

Figure 6-50. Hypothesis 2b: OA correlates positively with age; scatterplot for age and summary OA in the combined sample. A) Overall OA, B) Upper limb OA, C) Lower limb OA. Black line indicates linear regression model.

Table 6-18. Spearman's rank-order correlations for age and overall, upper limb, and lower limb OA in the combined-sex sample.

Variable	*rho*
Overall OA	0.622*
Upper limb OA	0.609*
Lower limb OA	0.438*

*Statistically significant at α=0.02 (Bonferroni correction: α=0.05/3).

Table 6-19. Regression results for age and overall, upper limb, and lower limb OA in the combined-sex sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	32.913	1.875	17.56	<2e-16*
Overall OA	37.014	2.245	16.49	<2e-16*
Intercept	36.670	1.718	21.35	<2e-16*
Upper limb OA	31.956	2.006	15.93	<2e-16*
Intercept	41.998	2.063	20.36	<2e-16*
Lower limb OA	23.218	2.252	10.31	<2e-16*

*Statistically significant at α=0.02 (Bonferroni correction: α=0.05/3).

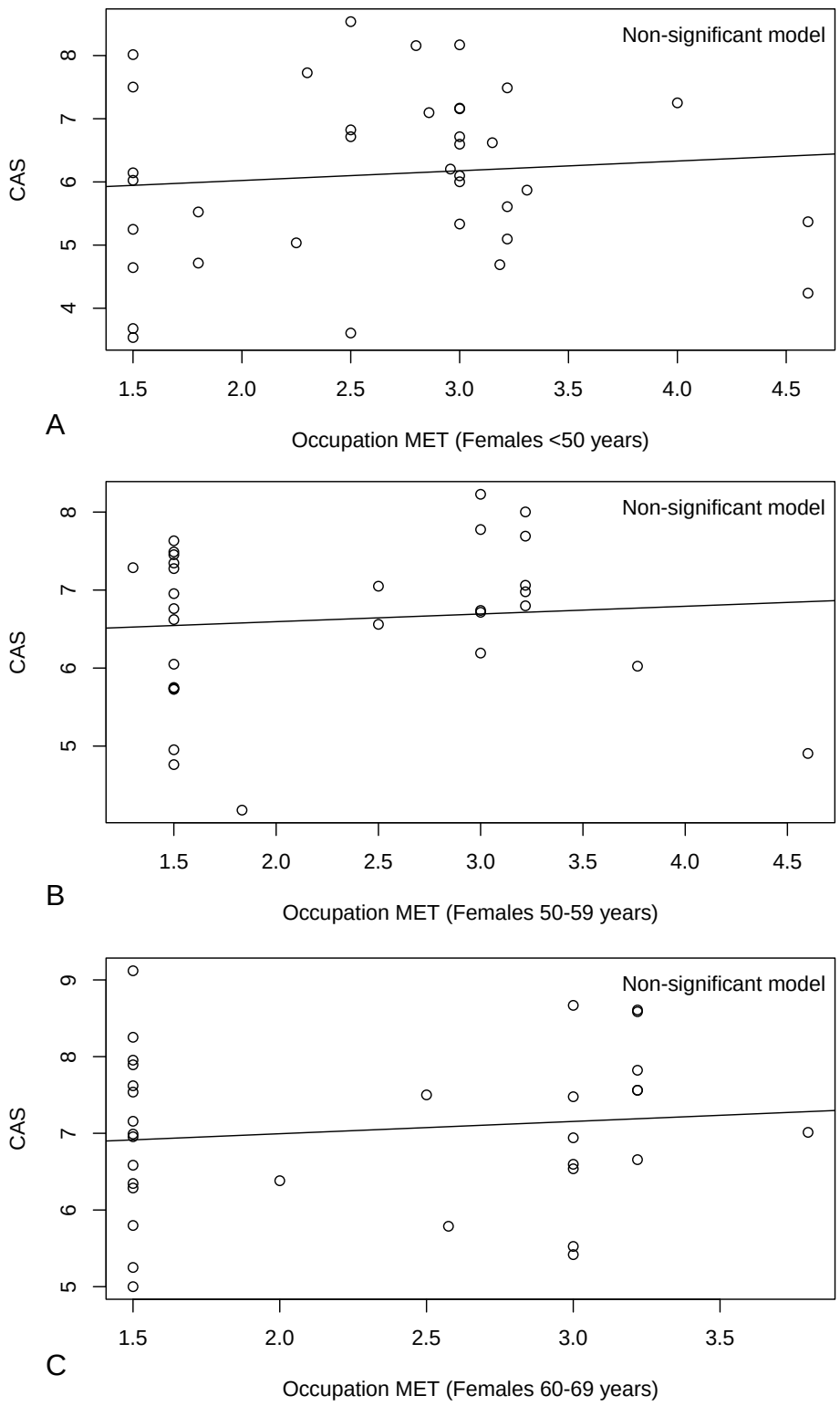


Figure 6-51. Hypothesis 3a: acetabular changes correlate positively with activity; scatterplots for CAS and MET occupation values in females. A) Female <50 years, B) Females 50-59 years, C) Females 60-69 years. Black line indicates linear regression model.

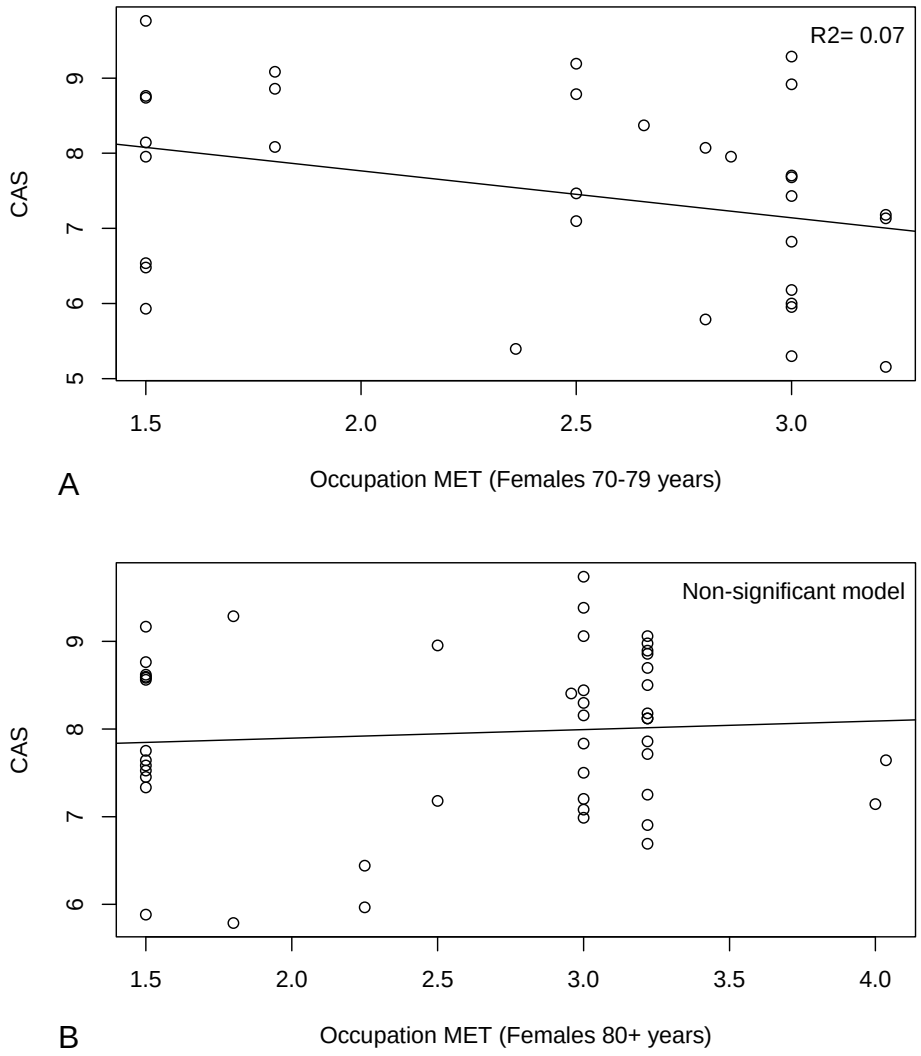


Figure 6-52. Hypothesis 3a: acetabular changes correlate positively with activity; scatterplots for CAS and MET occupation values in females. A) Females 70-79 years, B) Females 80+ years. Black line indicates linear regression model.

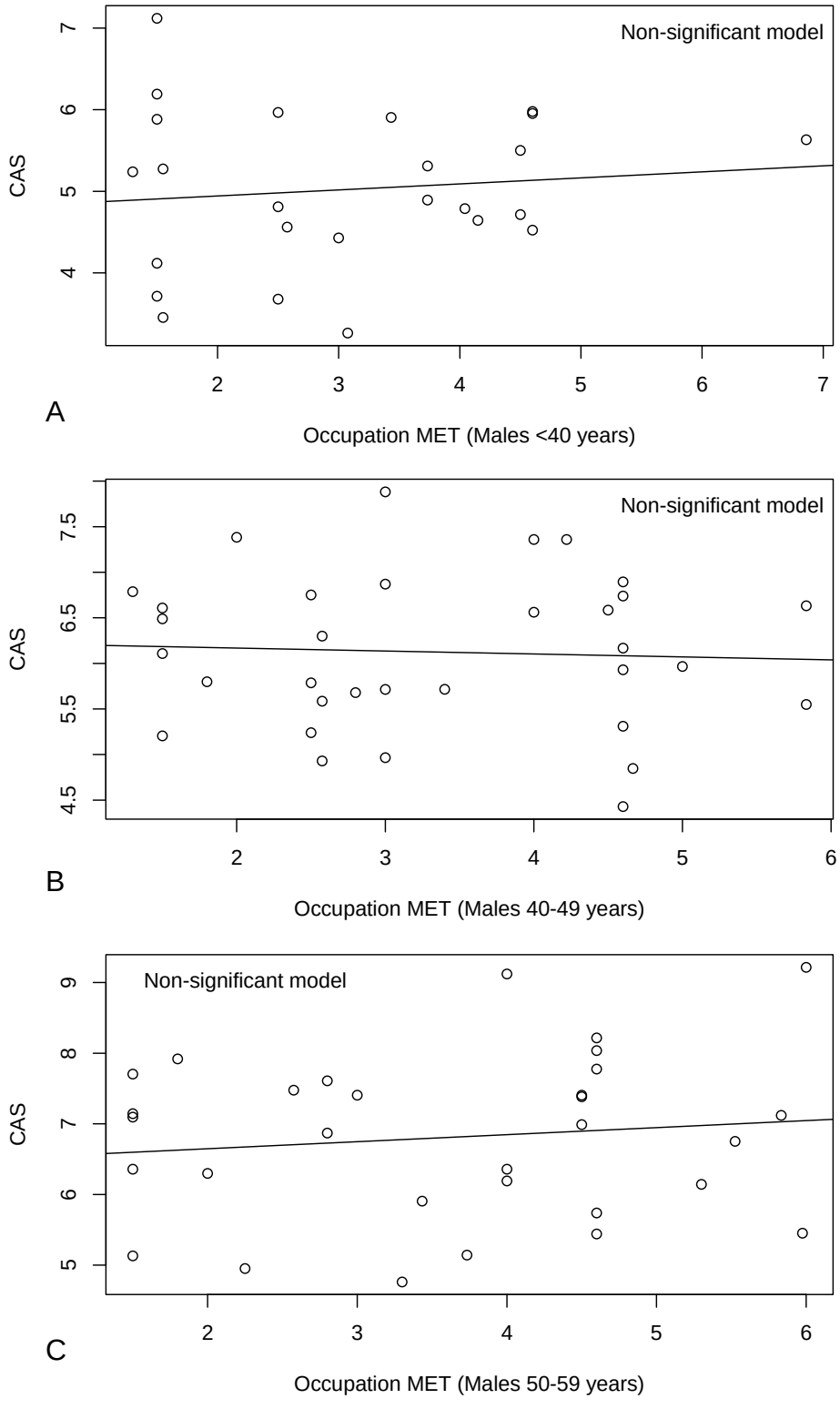


Figure 6-53. Hypothesis 3a: acetabular changes correlate positively with activity; scatterplots for CAS and MET occupation values in males. A) Males <40 years, B) Males 40-49 years, C) Males 50-59. Black line indicates linear regression model.

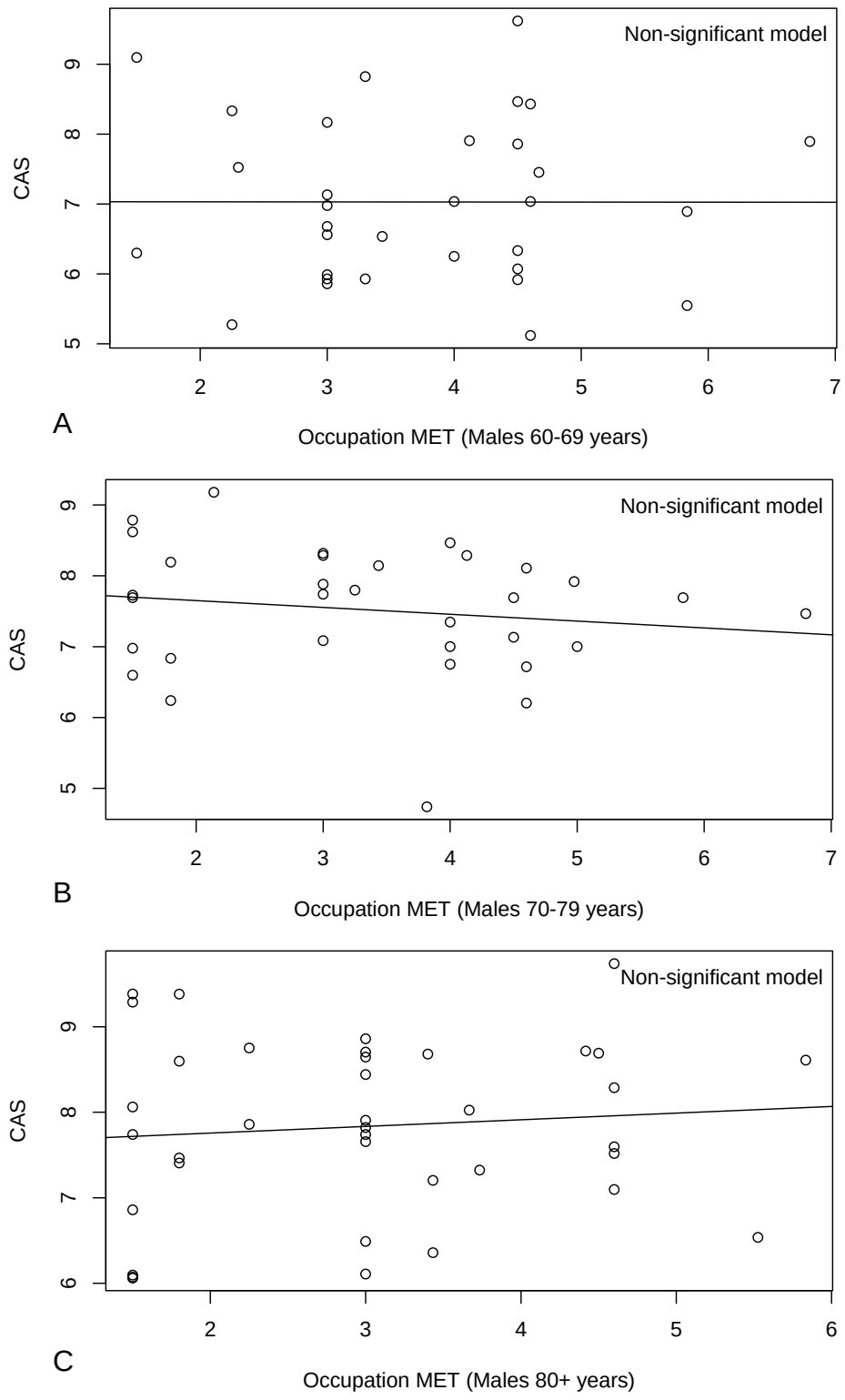


Figure 6-54. Hypothesis 3a: acetabular changes correlate positively with activity; scatterplots for CAS and MET occupation values in males. A) Males 60-69 years, B) Males 70-79 years, C) Males 80+ years. Black line indicates linear regression model.

Table 6-20. Spearman's rank-order correlations for CAS and MET values for occupation in females and males.

Group	*rho*
Females <50 years	0.067
Females 50-59 years	0.094
Females 60-69 years	0.170
Females 70-79 years	-0.313
Females 80+ years	0.034
Males <40 years	0.082
Males 40-49 years	-0.077
Males 50-59 years	0.088
Males 60-69 years	0.015
Males 70-79 years	-0.179
Males 80+ years	0.069

*Statistically significant at α=0.01 for females and α=0.008 for males (Bonferroni correction for females: α=0.05/5; for males: α=0.05/6).

Table 6-21. Regression results for CAS and MET values for occupation in females and males.

Variable	Estimate	Std. Error	t value	Probability
Intercept	5.715	0.758	7.542	9.3e-09*
Females <50 years	0.154	0.273	0.564	0.576
Intercept	6.399	0.513	12.493	5.73e-13*
Females 50-59 years	0.098	0.208	0.472	0.641
Intercept	6.674	0.574	11.62	1.23e-12*
Females 60-69 years	0.160	0.236	0.68	0.502
Intercept	9.014	0.853	10.566	8.46e-12*
Females 70-79 years	-0.624	0.338	-1.846	0.074
Intercept	7.701	0.487	15.821	<2e-16*
Females 80+ years	0.098	0.180	0.542	0.59
Intercept	4.795	0.463	10.351	3.97e-10*
Males <40 years	0.074	0.137	0.539	0.595
Intercept	6.232	0.404	15.410	4.43e-16*
Males 40-49 years	-0.032	0.113	-0.284	0.778
Intercept	6.450	0.573	11.251	4.26e-12*
Males 50-59 years	0.100	0.147	0.676	0.504
Intercept	7.036	0.684	10.283	2.38e-11*
Males 60-69 years	-0.002	0.174	-0.009	0.993
Intercept	7.845	0.413	19.010	<2e-16*
Males 70-79 years	-0.097	0.113	-0.855	0.4
Intercept	7.602	0.441	17.228	<2e-16*
Males 80+ years	0.078	0.135	0.573	0.57

*Statistically significant at α=0.01 for females and α=0.008 for males (Bonferroni correction for females: α=0.05/5; for males: α=0.05/6).

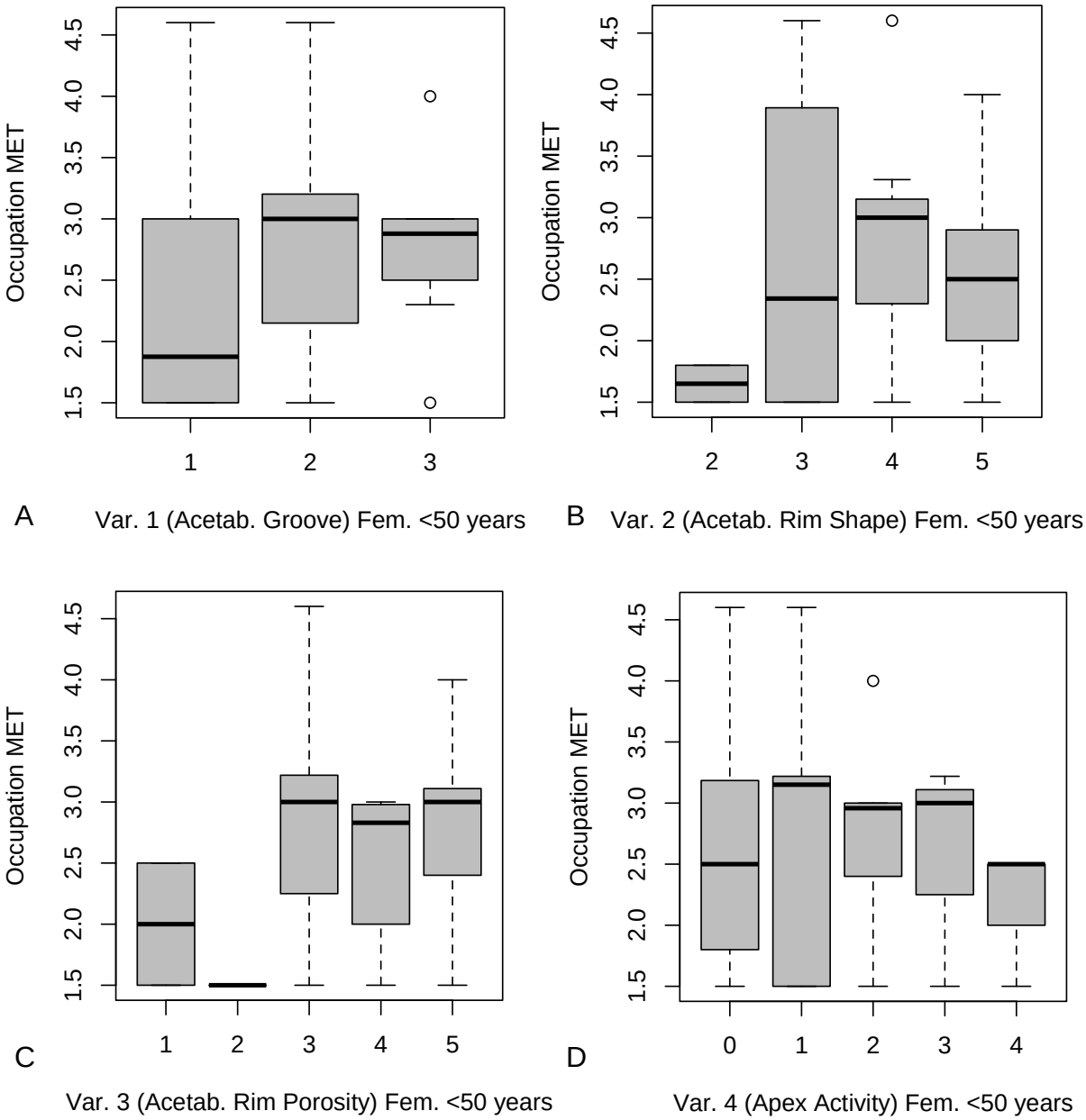


Figure 6-55. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for occupation MET value per acetabular variable score in females under 50 years. A) Variable 1, B) Variable 2, C) Variable 3, D) Variable 4.

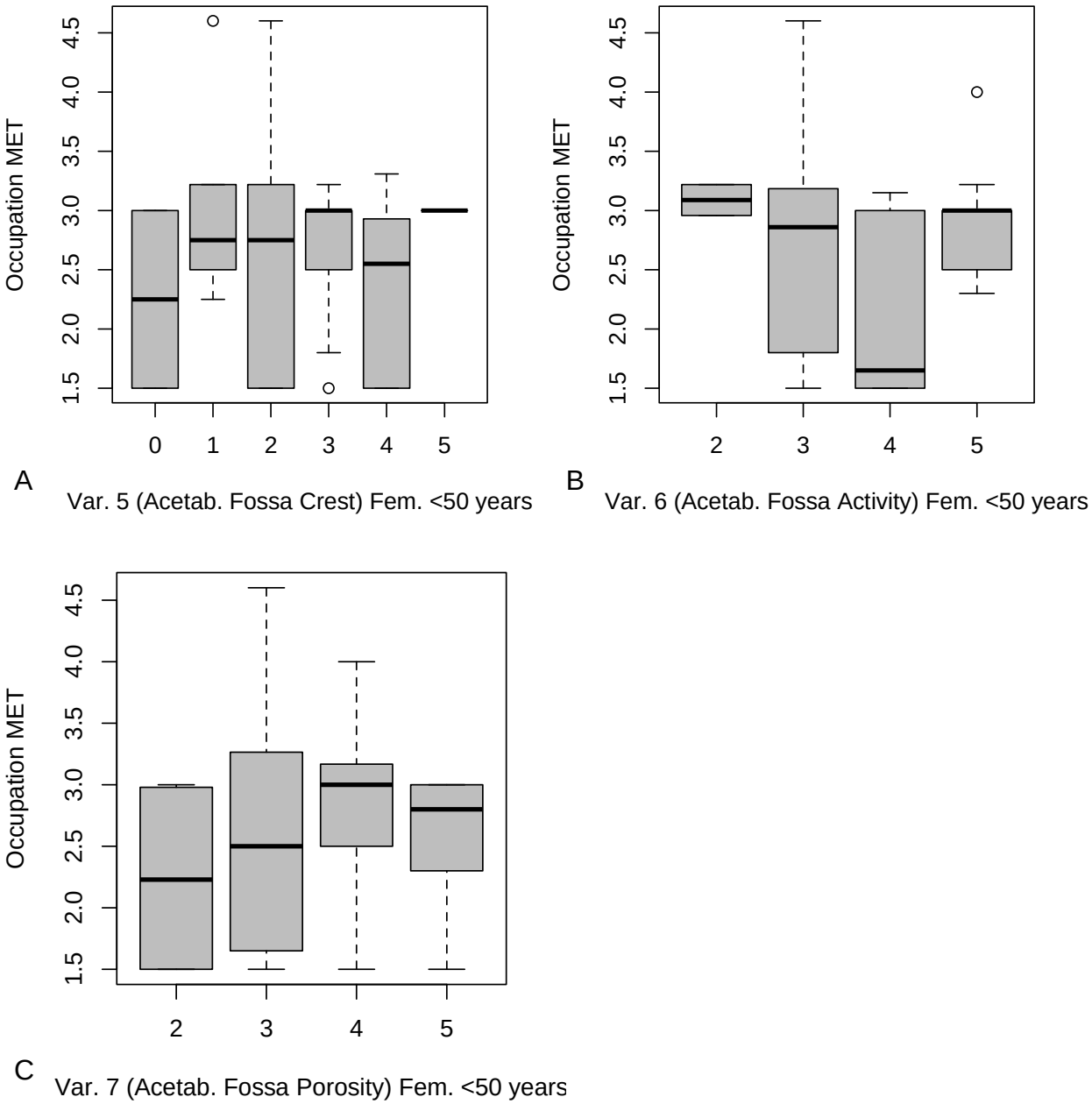


Figure 6-56. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for occupation MET value per acetabular variable score in females under 50 years. A) Variable 5, B) Variable 6, C) Variable 7.

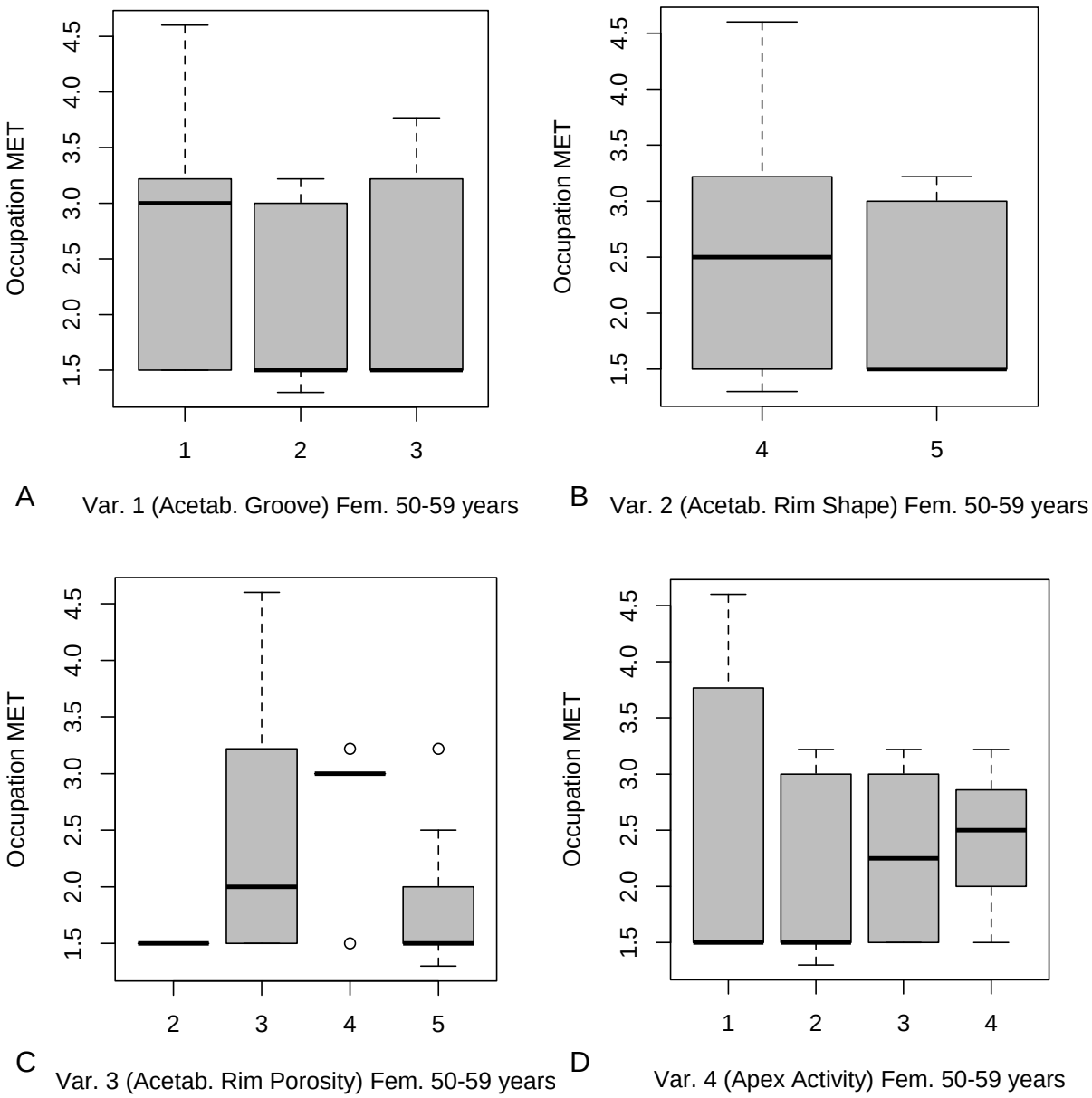
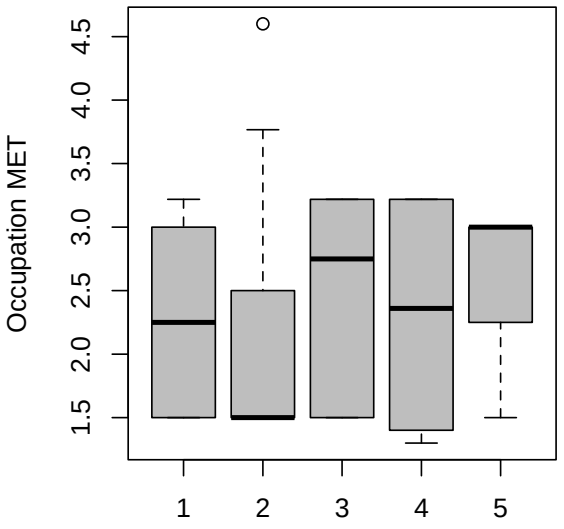
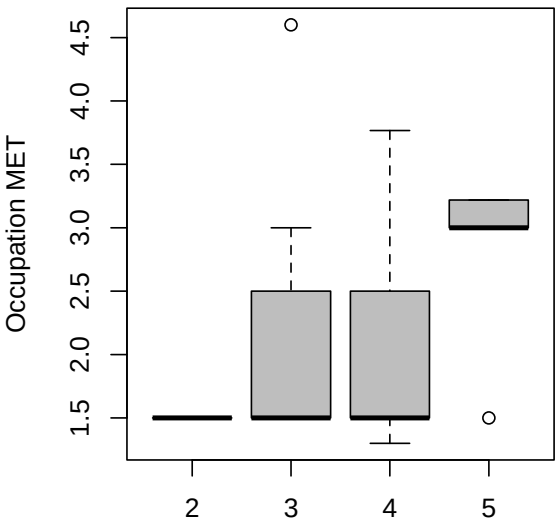


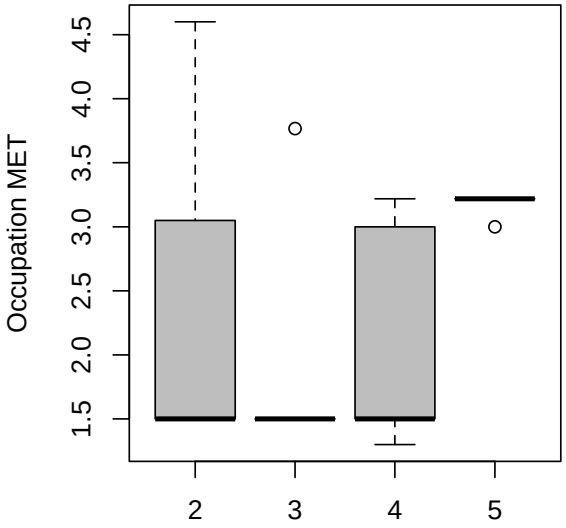
Figure 6-57. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for occupation MET value per acetabular variable score in females 50-59 years. A) Variable 1, B) Variable 2, C) Variable 3, D) Variable 4.



A Var. 5 (Acetab. Fossa Crest) Fem. 50-59 years



B Var. 6 (Acetab. Fossa Activity) Fem. 50-59 yrs.



C Var. 7 (Acetab. Fossa Porosity) Fem. 50-59 yrs.

Figure 6-58. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for occupation MET value per acetabular variable score in females 50-59 years. A) Variable 5, B) Variable 6, C) Variable 7.

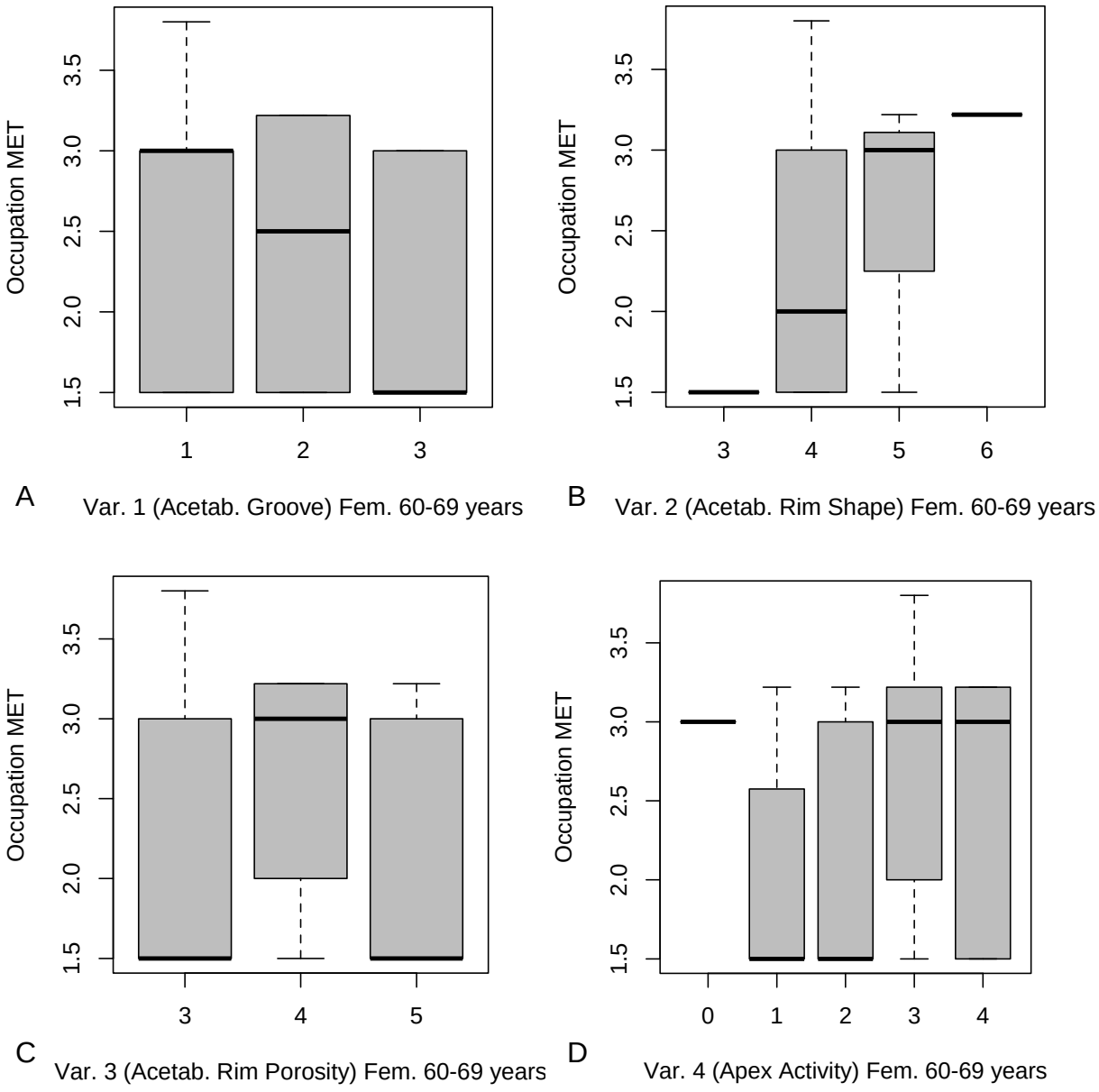


Figure 6-59. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for occupation MET value per acetabular variable score in females 60-69 years. A) Variable 1, B) Variable 2, C) Variable 3, D) Variable 4.

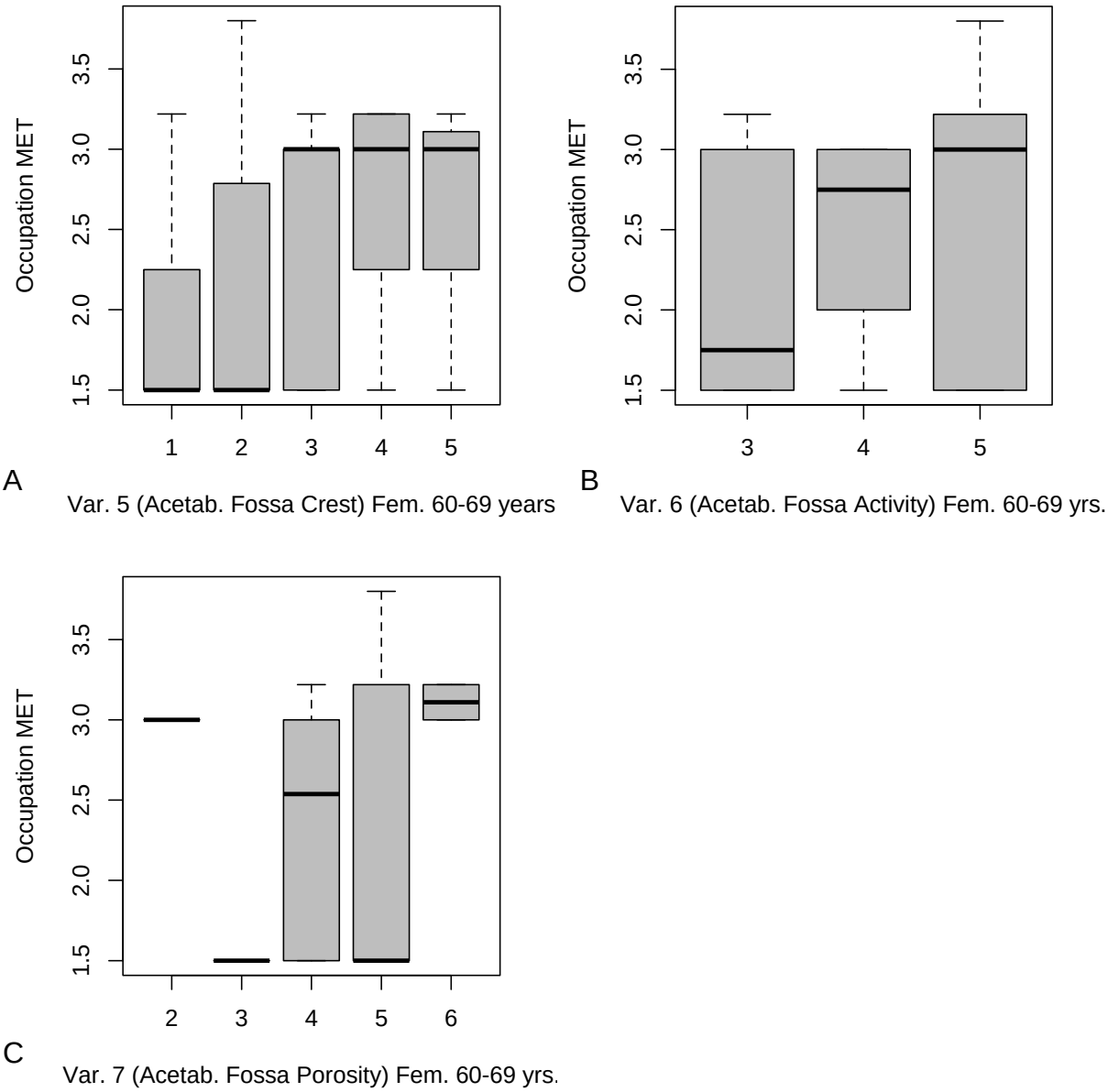


Figure 6-60. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for occupation MET value per acetabular variable score in females 60-69 years. A) Variable 5, B) Variable 6, C) Variable 7.

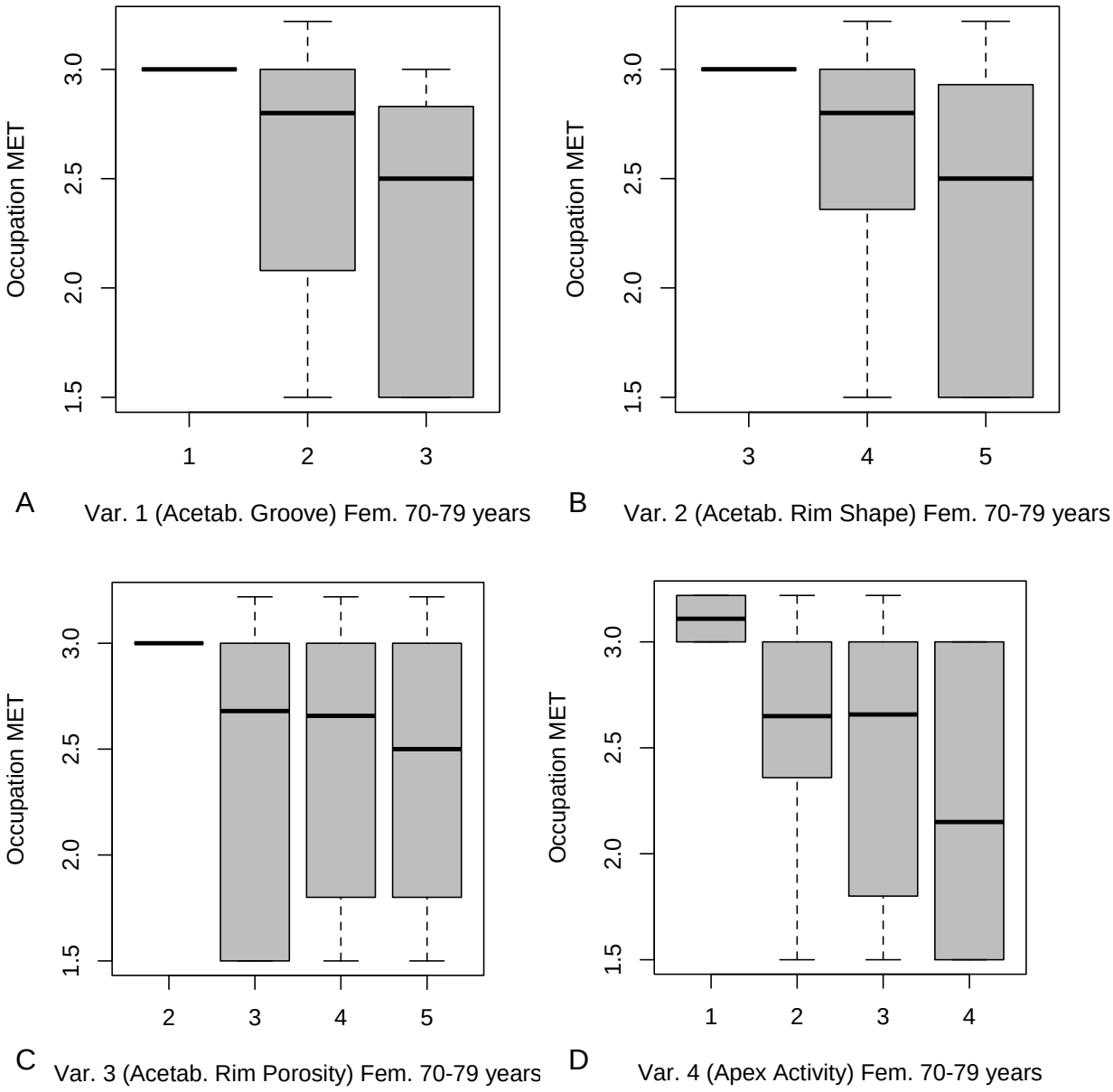
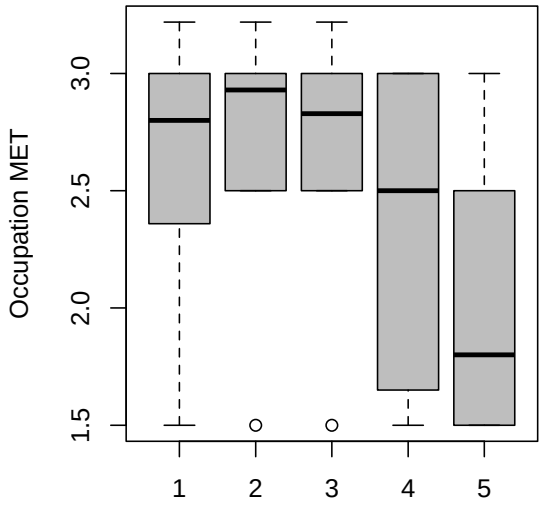
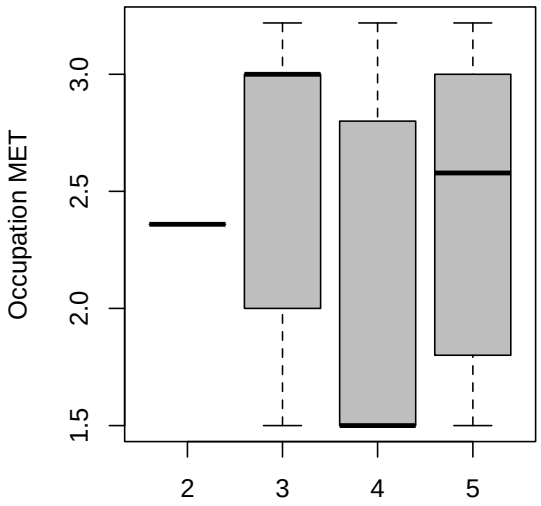


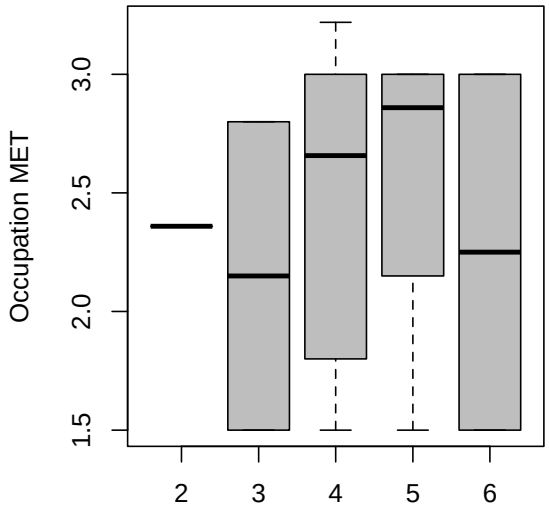
Figure 6-61. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for occupation MET value per acetabular variable score in females 70-79 years. A) Variable 1, B) Variable 2, C) Variable 3, D) Variable 4.



A Var. 5 (Acetab. Fossa Crest) Fem. 70-79 years



B Var. 6 (Acetab. Fossa Activity) Fem. 70-79 yrs.



C Var. 7 (Acetab. Fossa Porosity) Fem. 70-79 yrs.

Figure 6-62. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for occupation MET value per acetabular variable score in females 70-79 years. A) Variable 5, B) Variable 6, C) Variable 7.

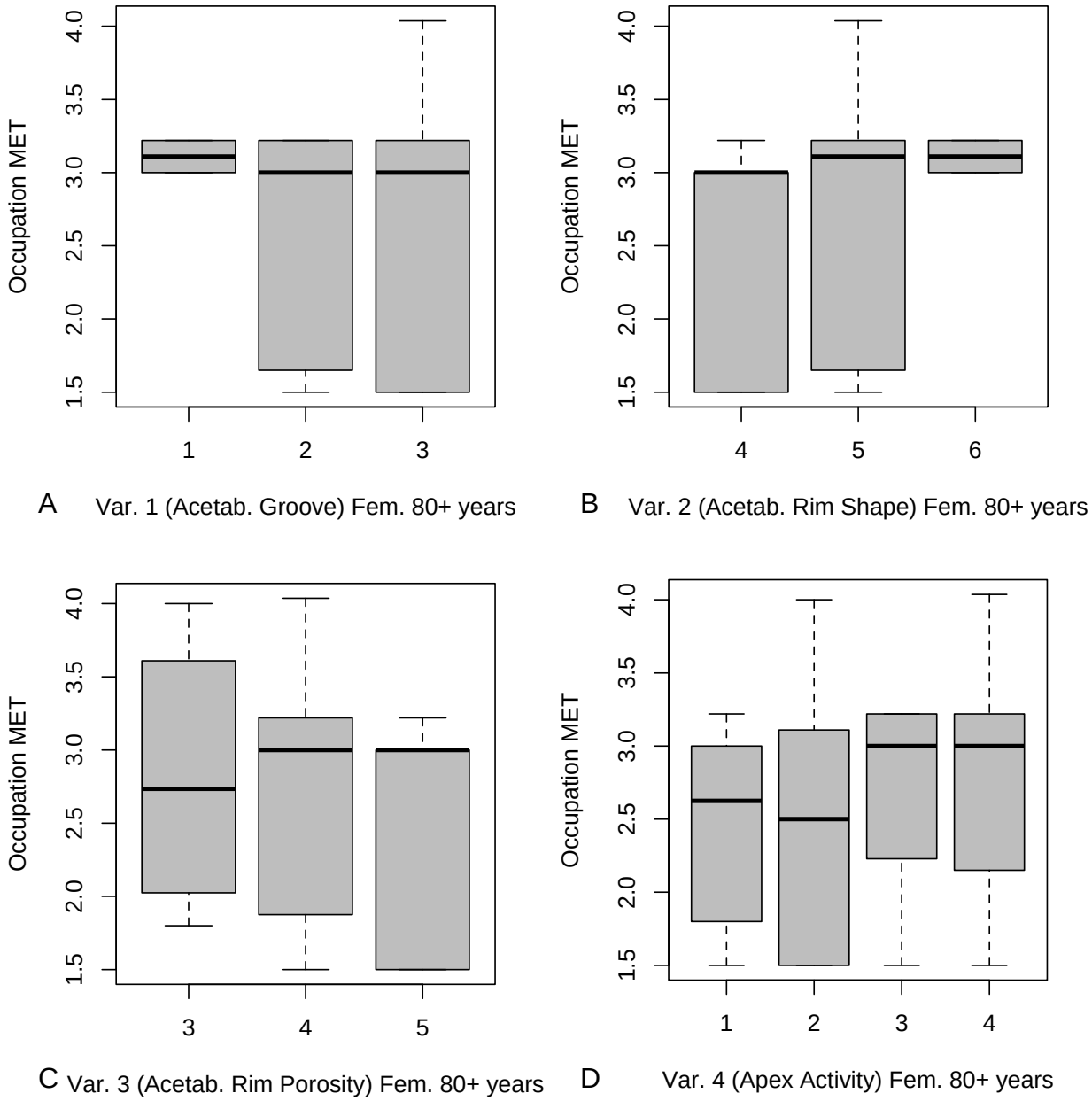
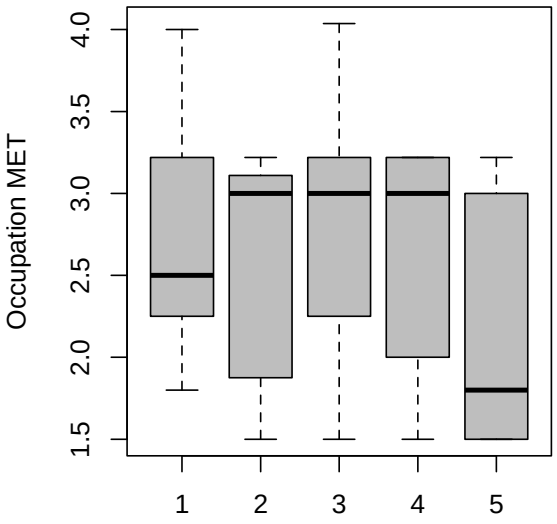
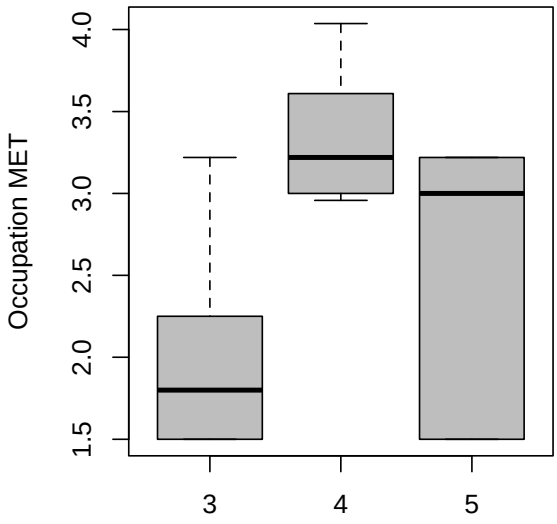


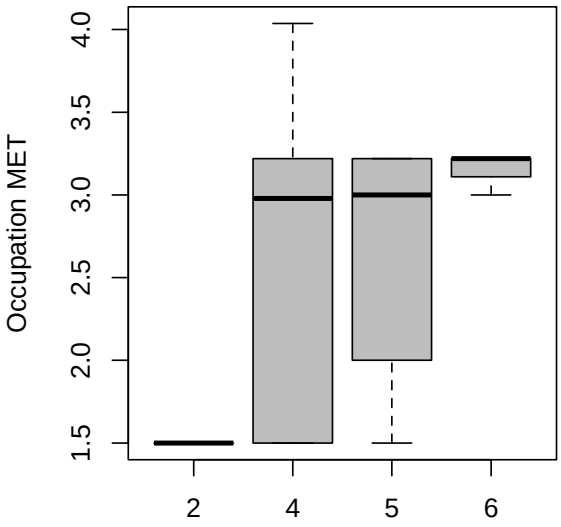
Figure 6-63. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for occupation MET value per acetabular variable score in females 80+ years. A) Variable 1, B) Variable 2, C) Variable 3, D) Variable 4.



A Var. 5 (Acetab. Fossa Crest) Fem. 80+ years



B Var. 6 (Acetab. Fossa Activity) Fem. 80+ years



C Var. 7 (Acetab. Fossa Porosity) Fem. 80+ yrs.

Figure 6-64. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for occupation MET value per acetabular variable score in females 80+ years. A) Variable 5, B) Variable 6, C) Variable 7.

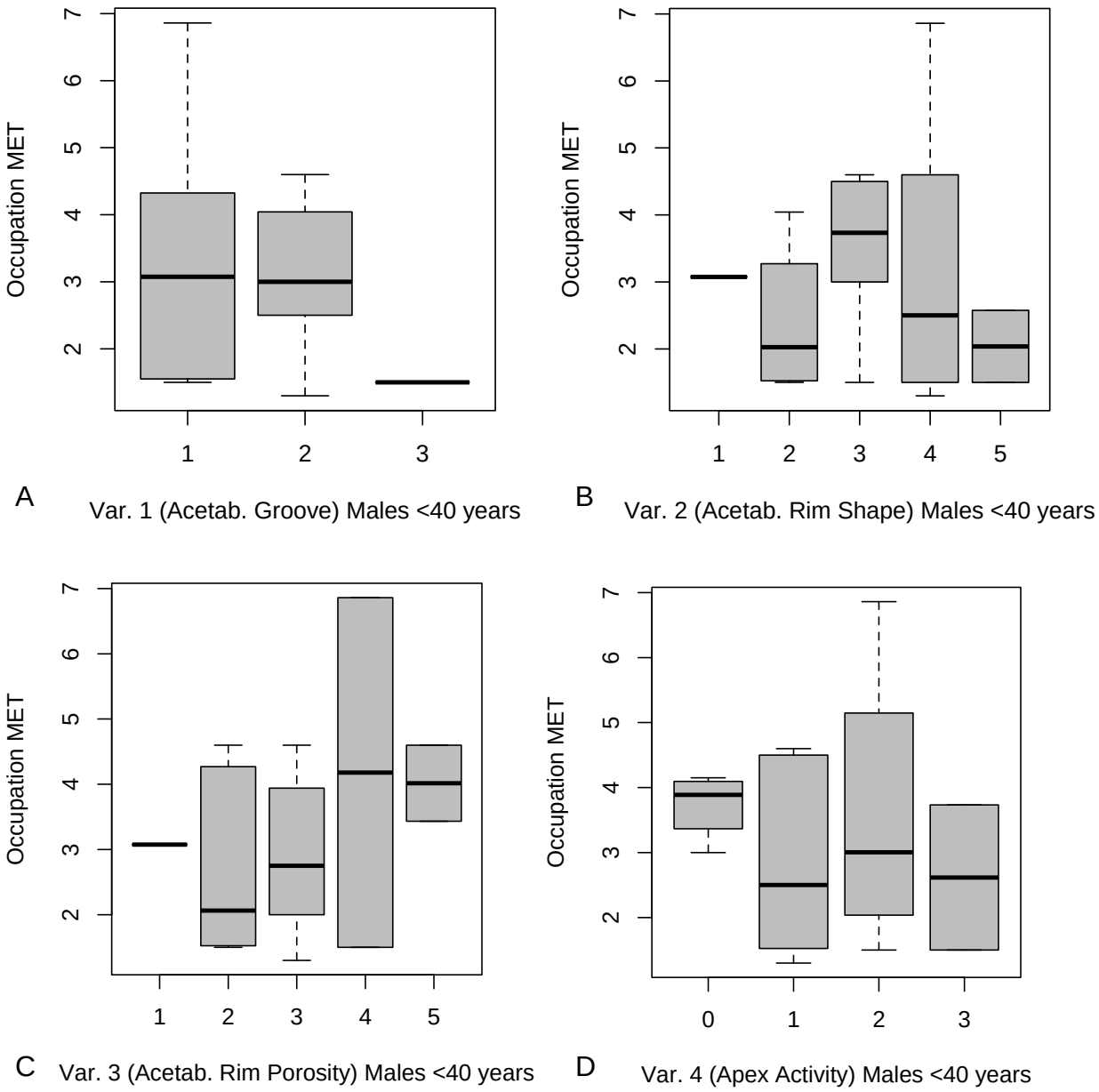
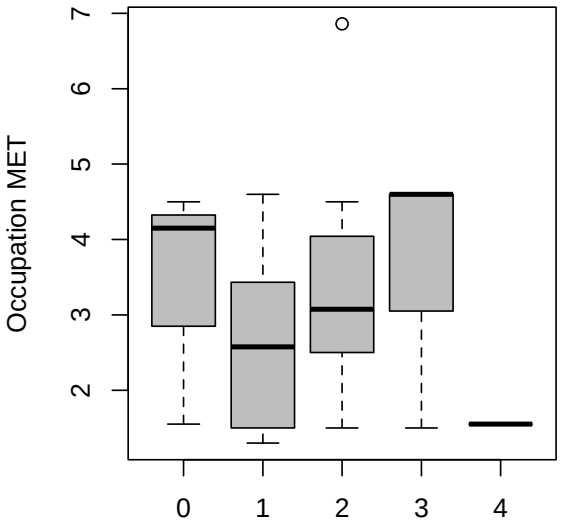
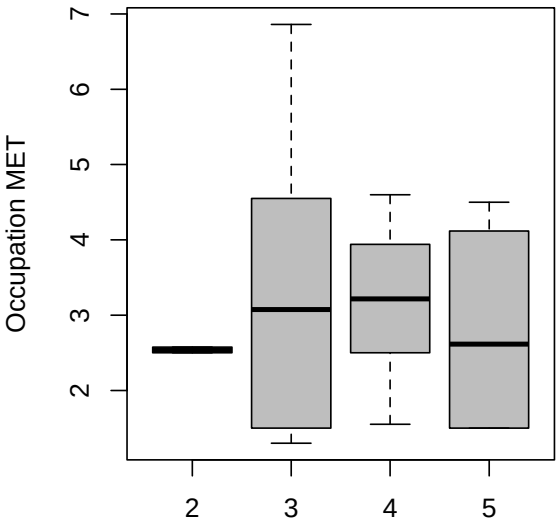


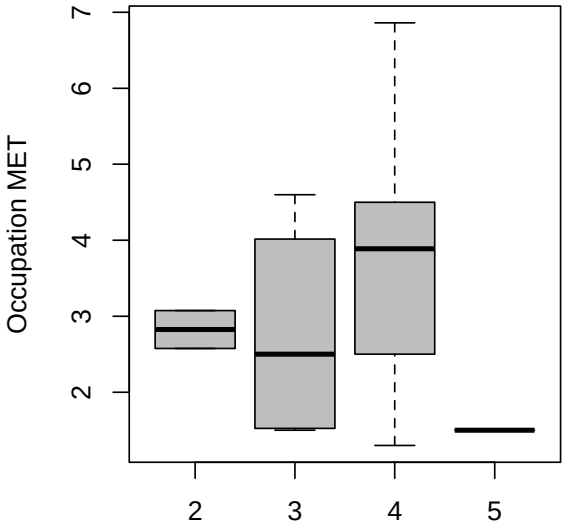
Figure 6-65. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for occupation MET value per acetabular variable score in males under 40 years. A) Variable 1, B) Variable 2, C) Variable 3, D) Variable 4.



A Var. 5 (Acetab. Fossa Crest) Males <40 years



B Var. 6 (Acetab. Fossa Activity) Males <40 years



C Var. 7 (Acetab. Fossa Porosity) Males <40 yrs.

Figure 6-66. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for occupation MET value per acetabular variable score in males under 40 years. A) Variable 5, B) Variable 6, C) Variable 7.

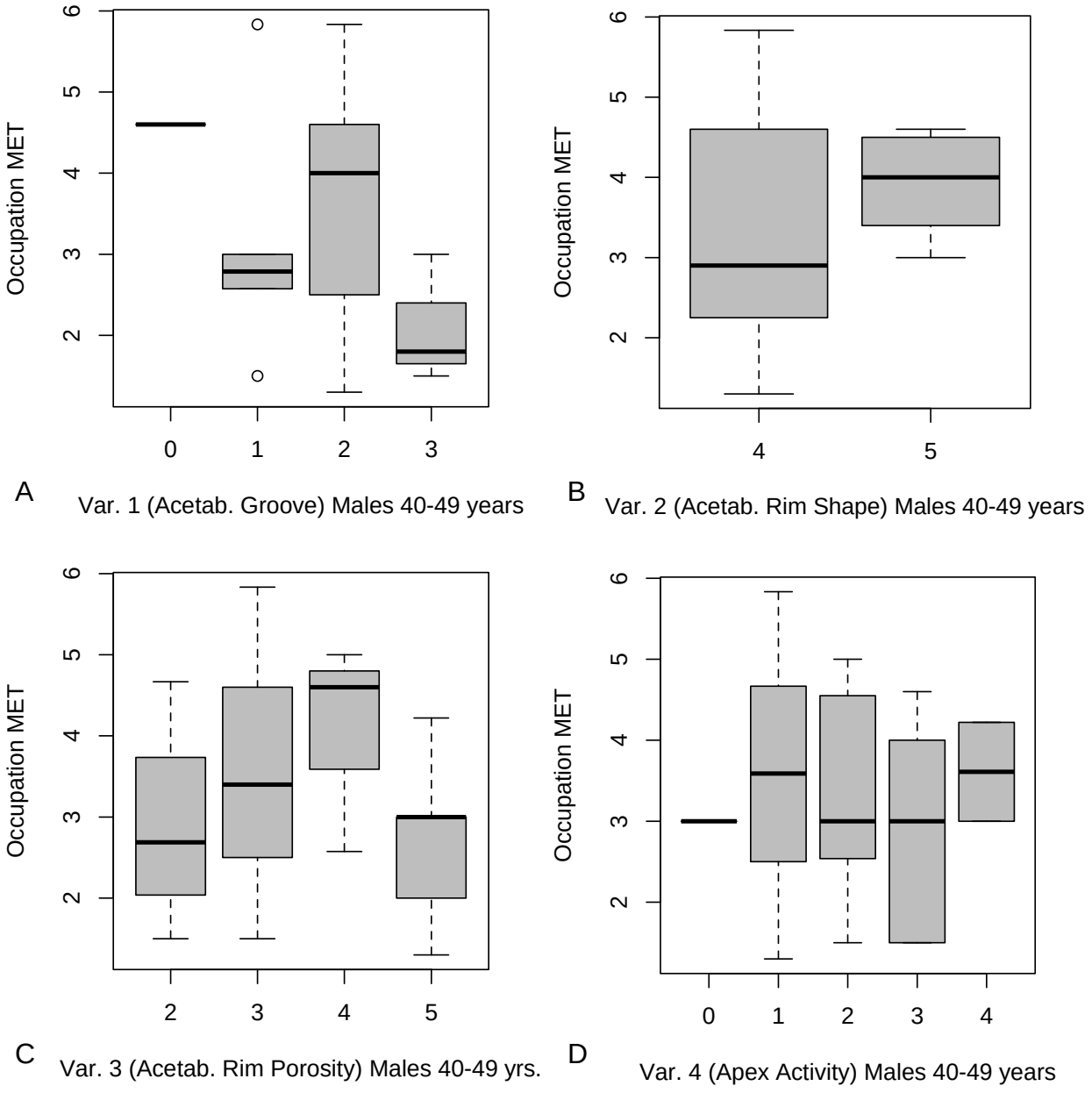


Figure 6-67. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for occupation MET value per acetabular variable score in males 40-49 years. A) Variable 1, B) Variable 2, C) Variable 3, D) Variable 4.

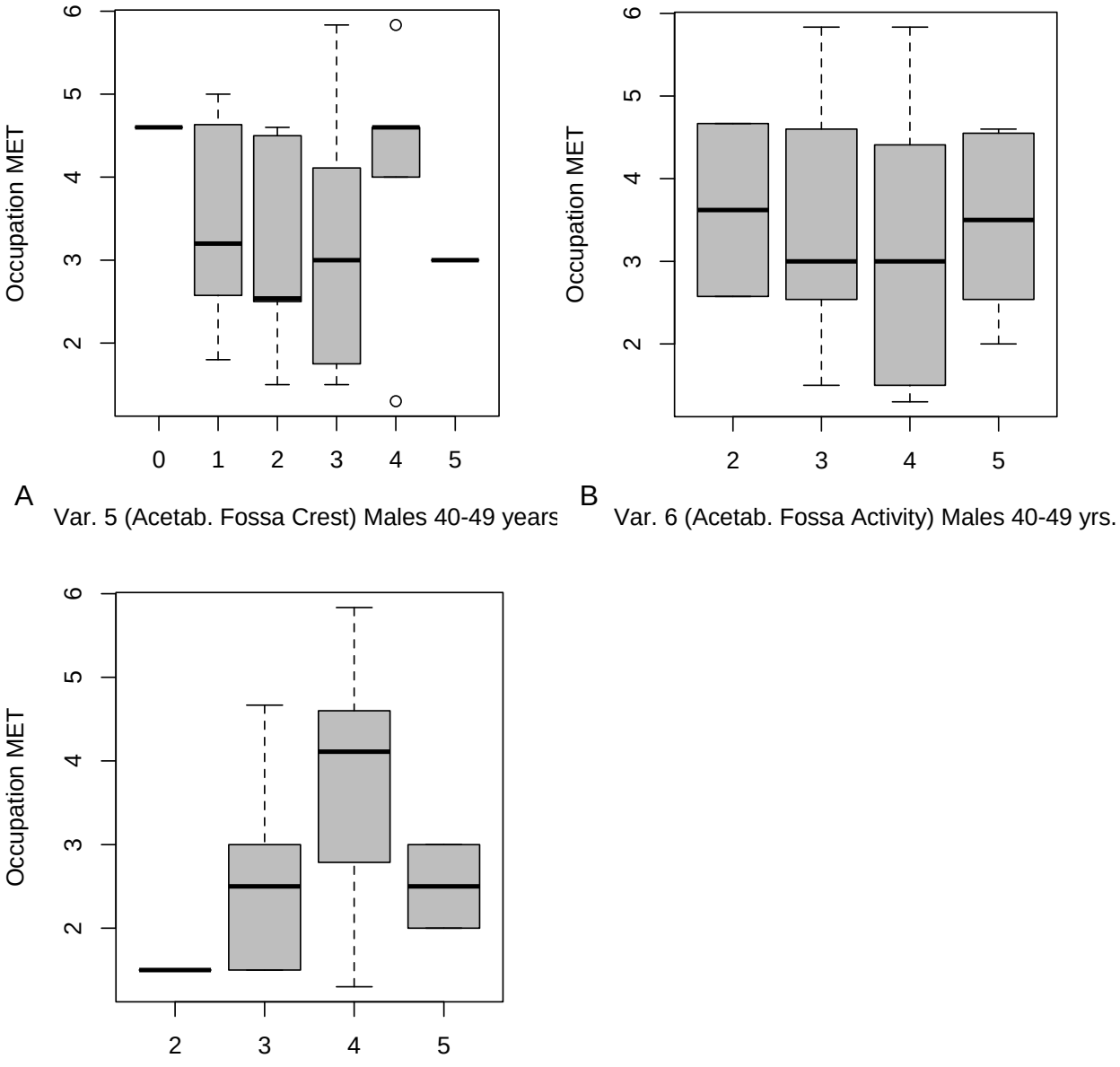


Figure 6-68. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for occupation MET value per acetabular variable score in males 40-49 years. A) Variable 5, B) Variable 6, C) Variable 7.

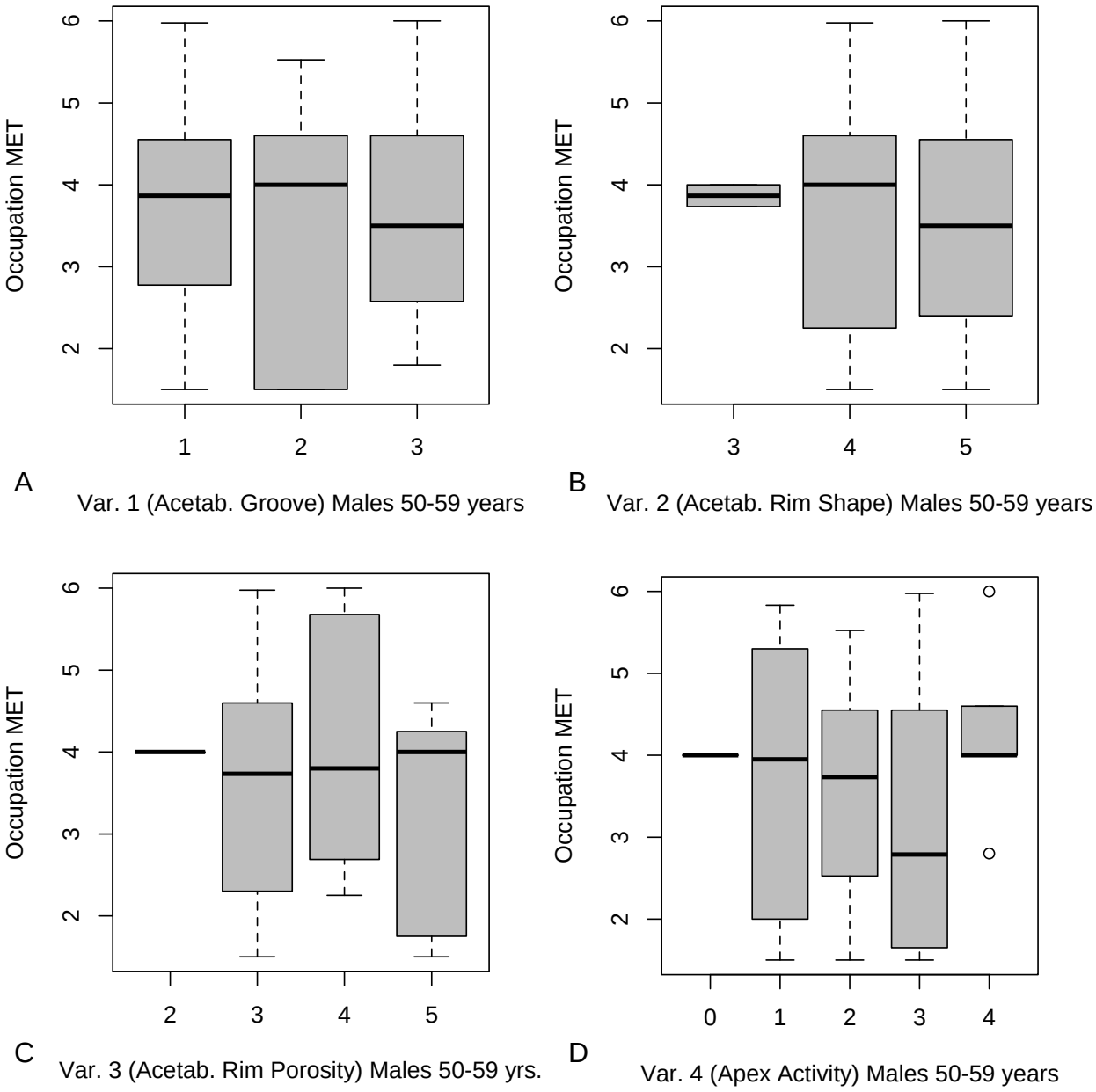


Figure 6-69. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for occupation MET value per acetabular variable score in males 50-59 years. A) Variable 1, B) Variable 2, C) Variable 3, D) Variable 4.

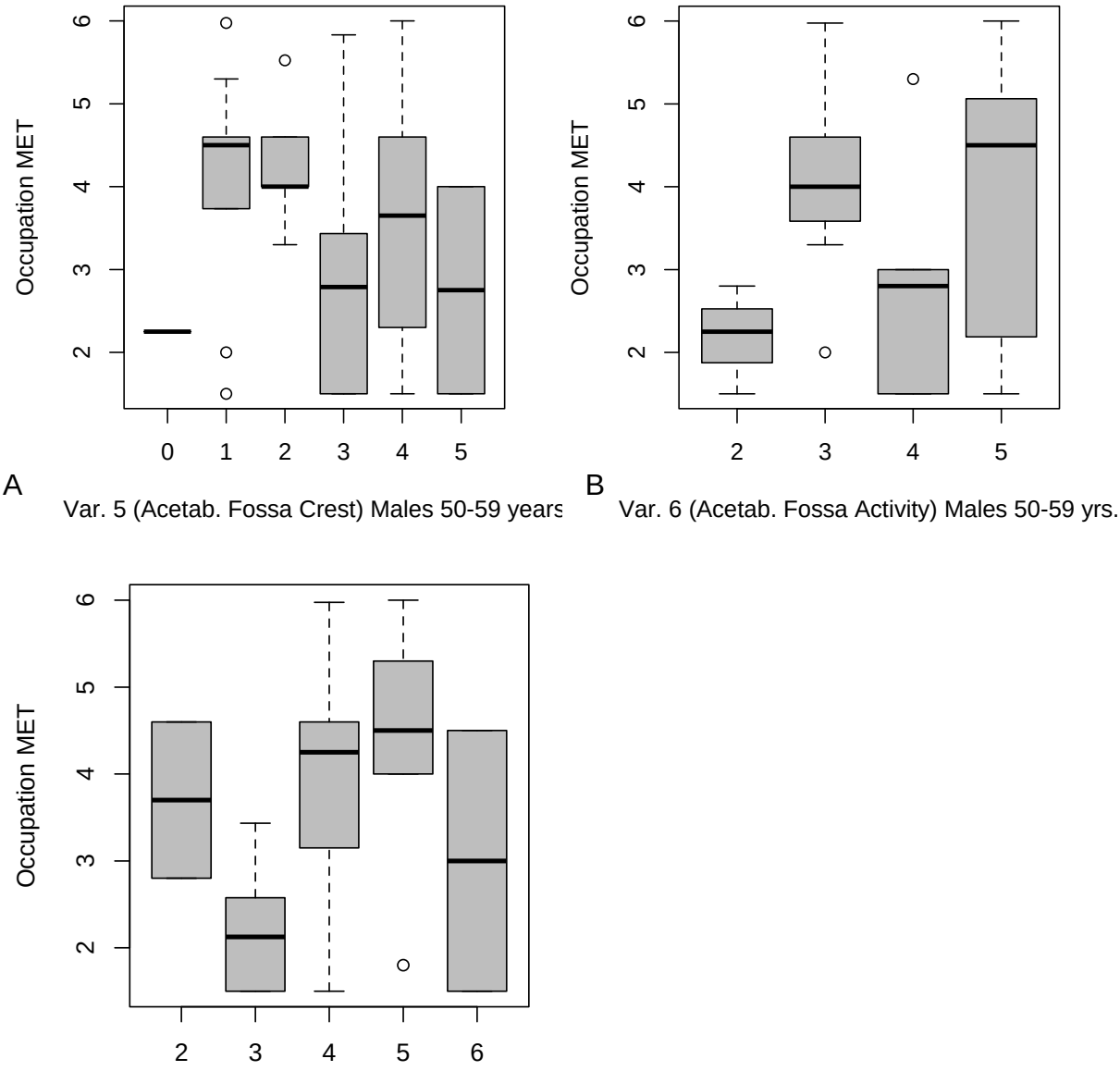


Figure 6-70. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for occupation MET value per acetabular variable score in males 50-59 years. A) Variable 5, B) Variable 6, C) Variable 7.

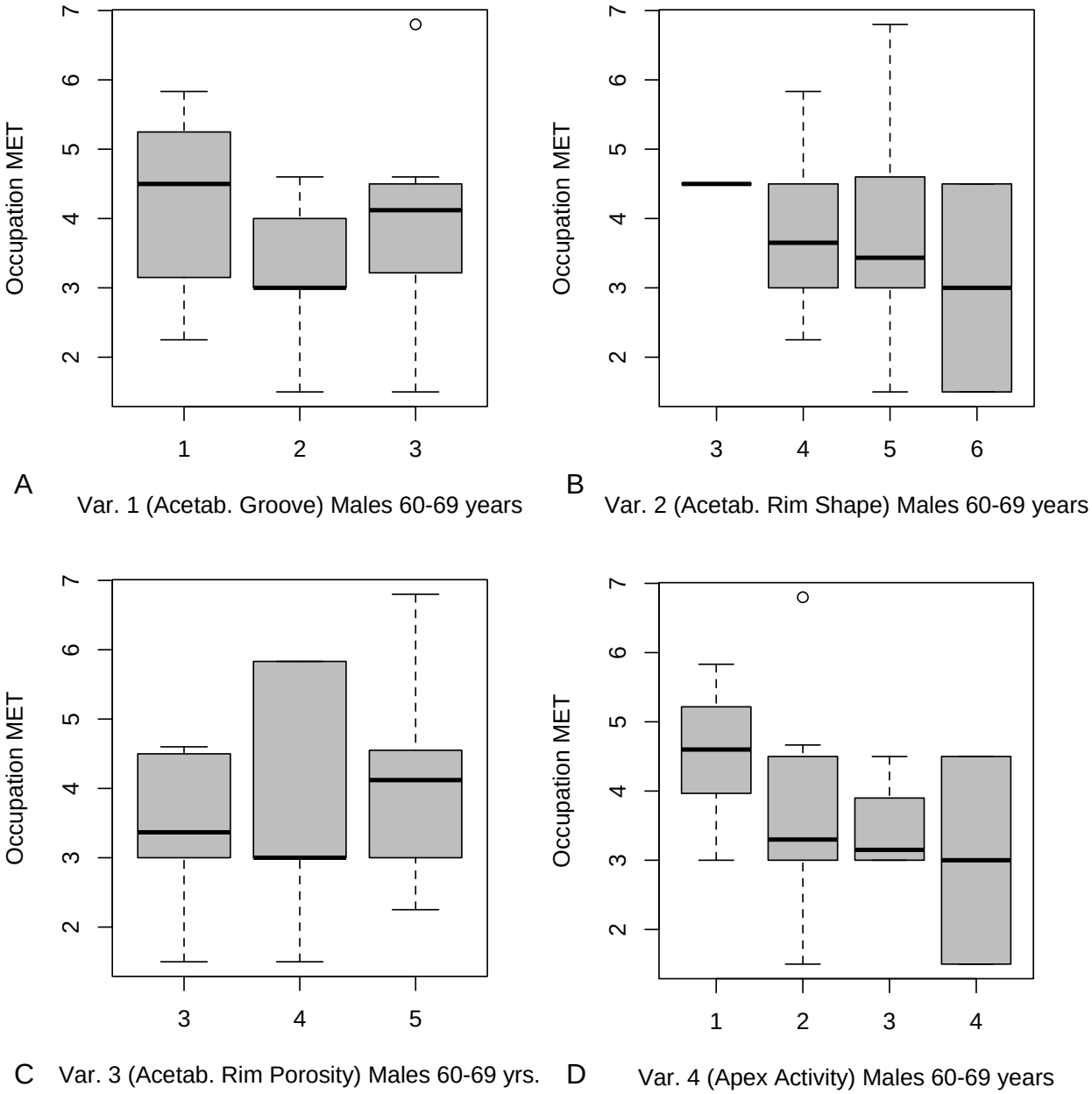


Figure 6-71. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for occupation MET value per acetabular variable score in males 60-69 years. A) Variable 1, B) Variable 2, C) Variable 3, D) Variable 4.

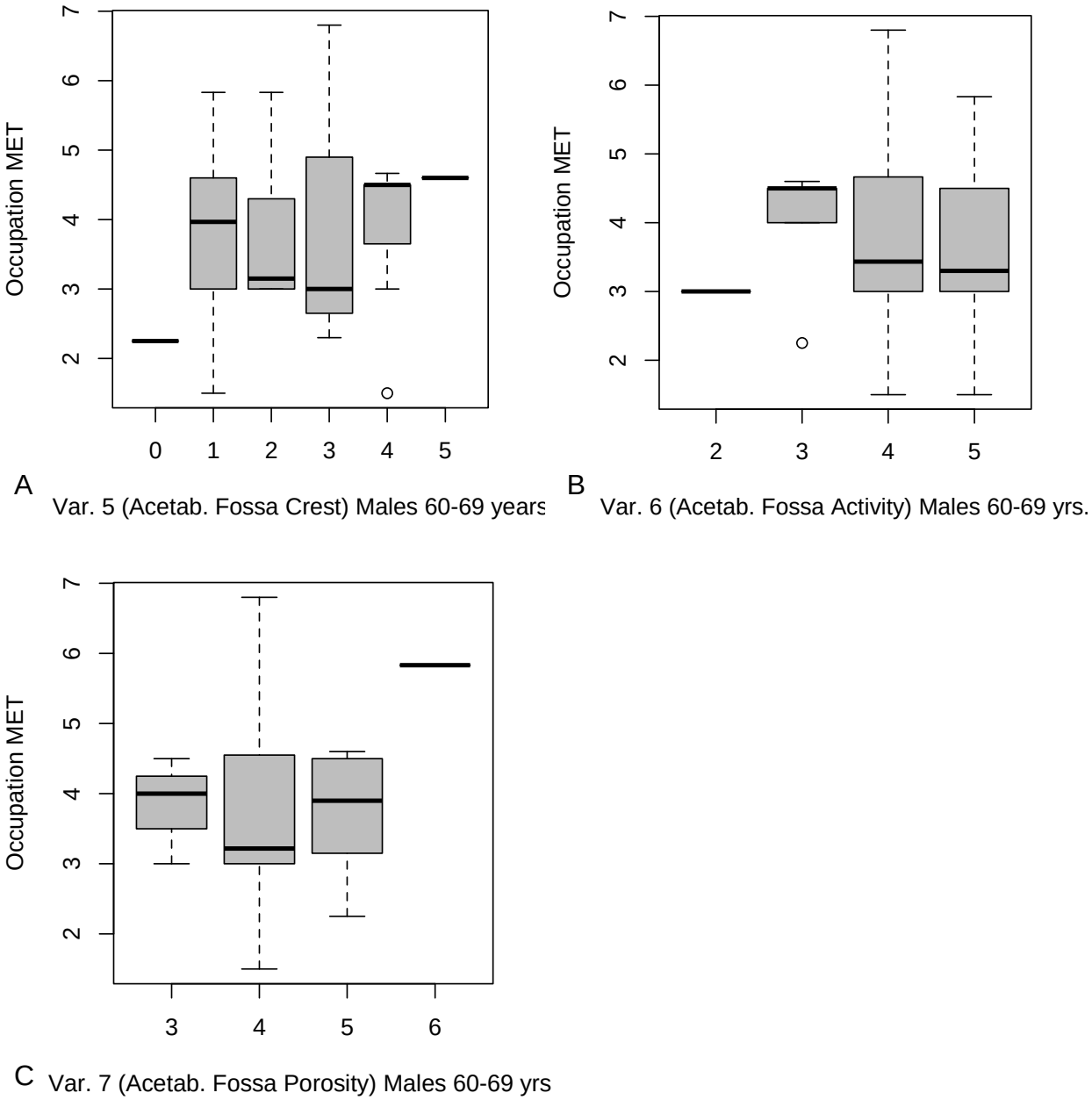


Figure 6-72. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for occupation MET value per acetabular variable score in males 60-69 years. A) Variable 5, B) Variable 6, C) Variable 7.

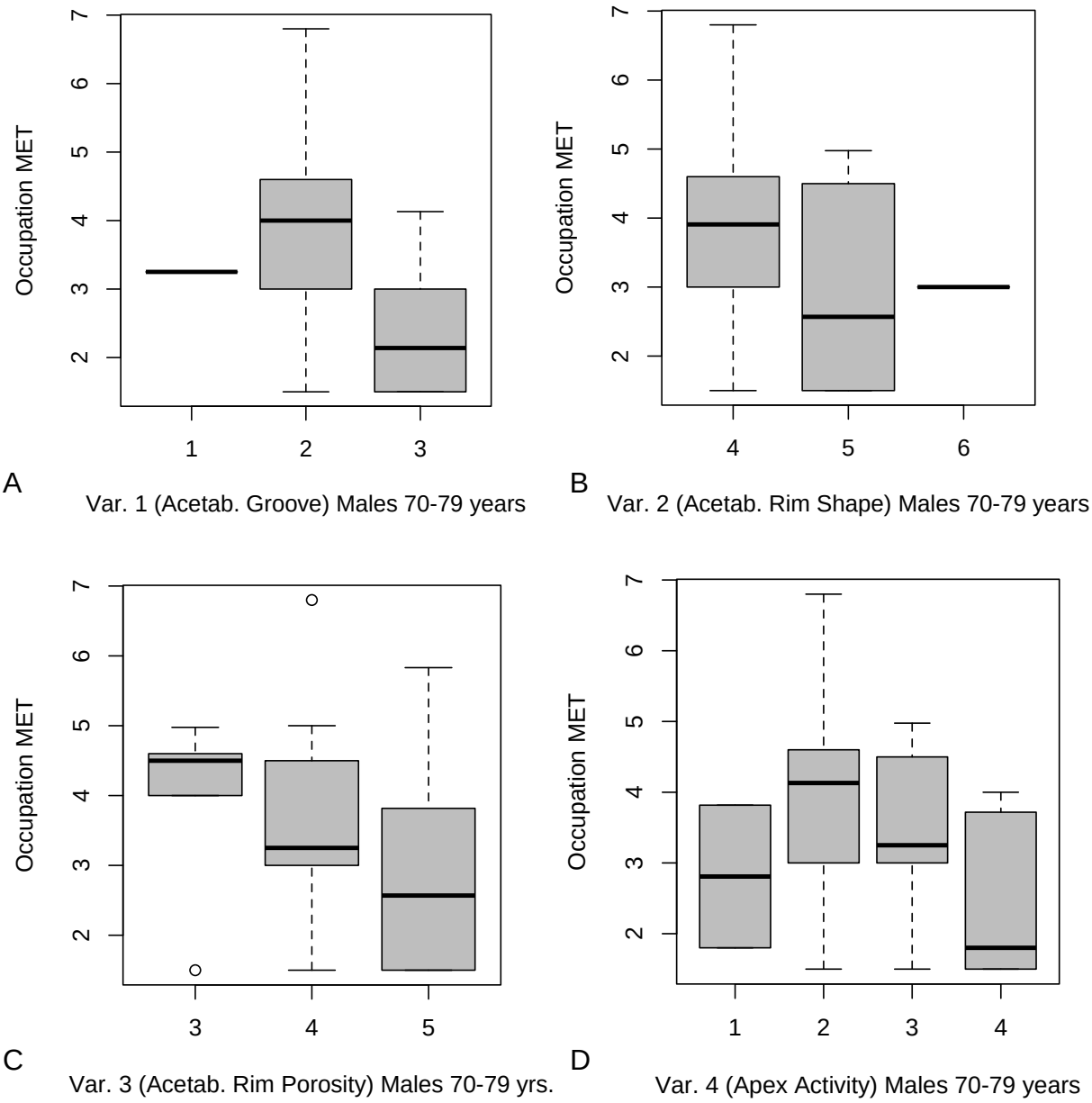


Figure 6-73. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for occupation MET value per acetabular variable score in males 70-79 years. A) Variable 1, B) Variable 2, C) Variable 3, D) Variable 4.

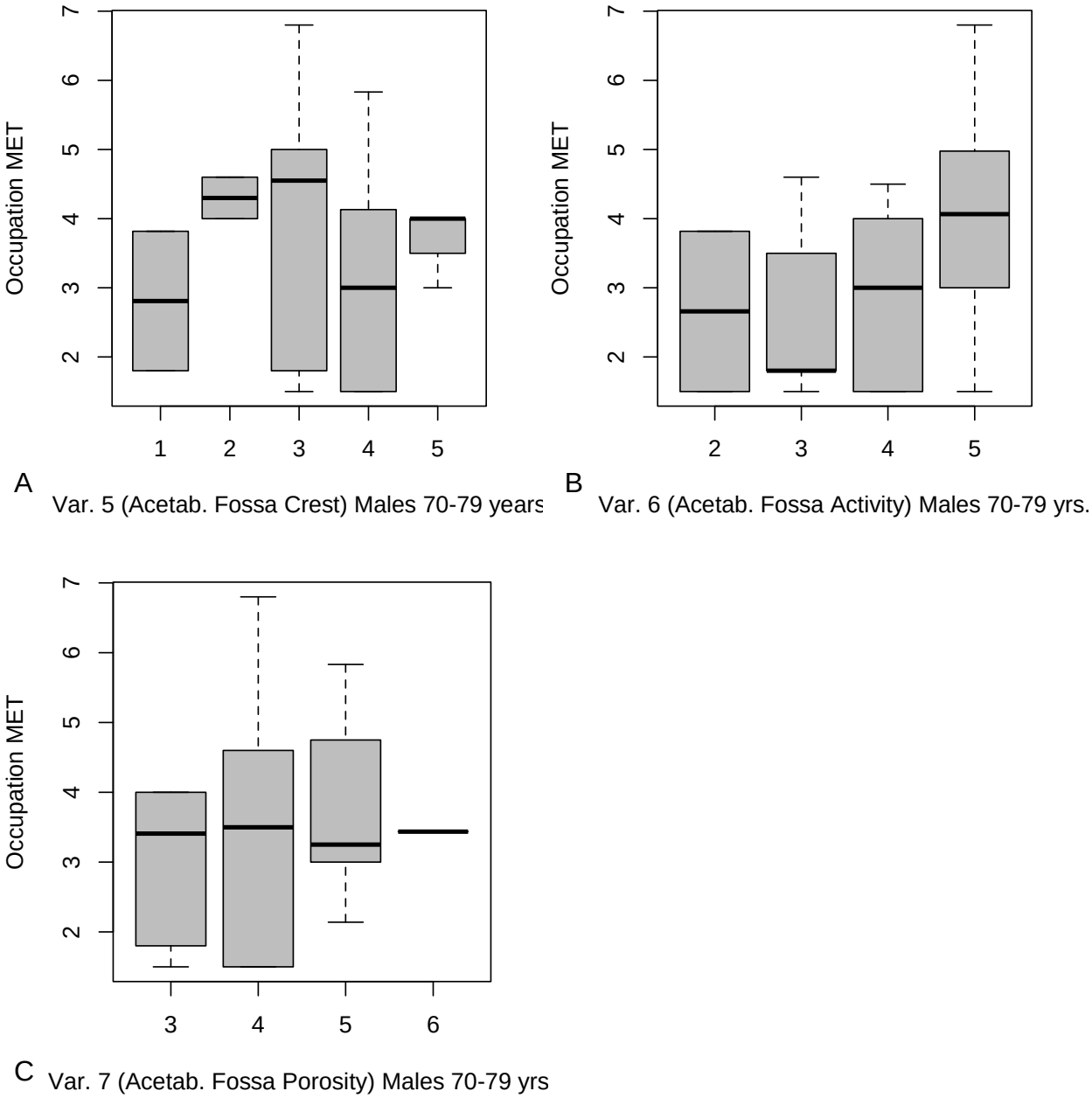


Figure 6-74. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for occupation MET value per acetabular variable score in males 70-79 years. A) Variable 5, B) Variable 6, C) Variable 7.

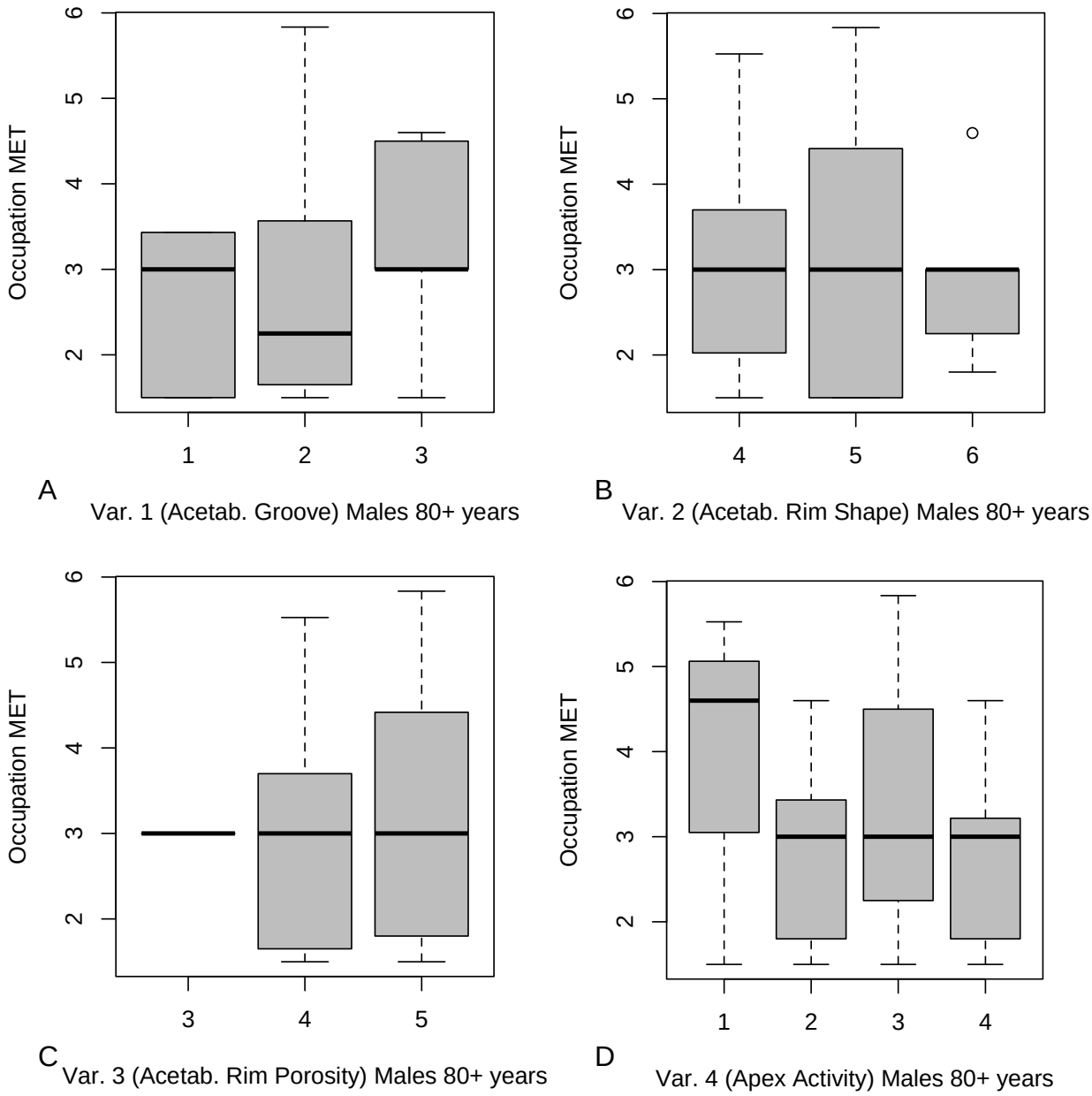


Figure 6-75. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for occupation MET value per acetabular variable score in males 80+ years. A) Variable 1, B) Variable 2, C) Variable 3, D) Variable 4.

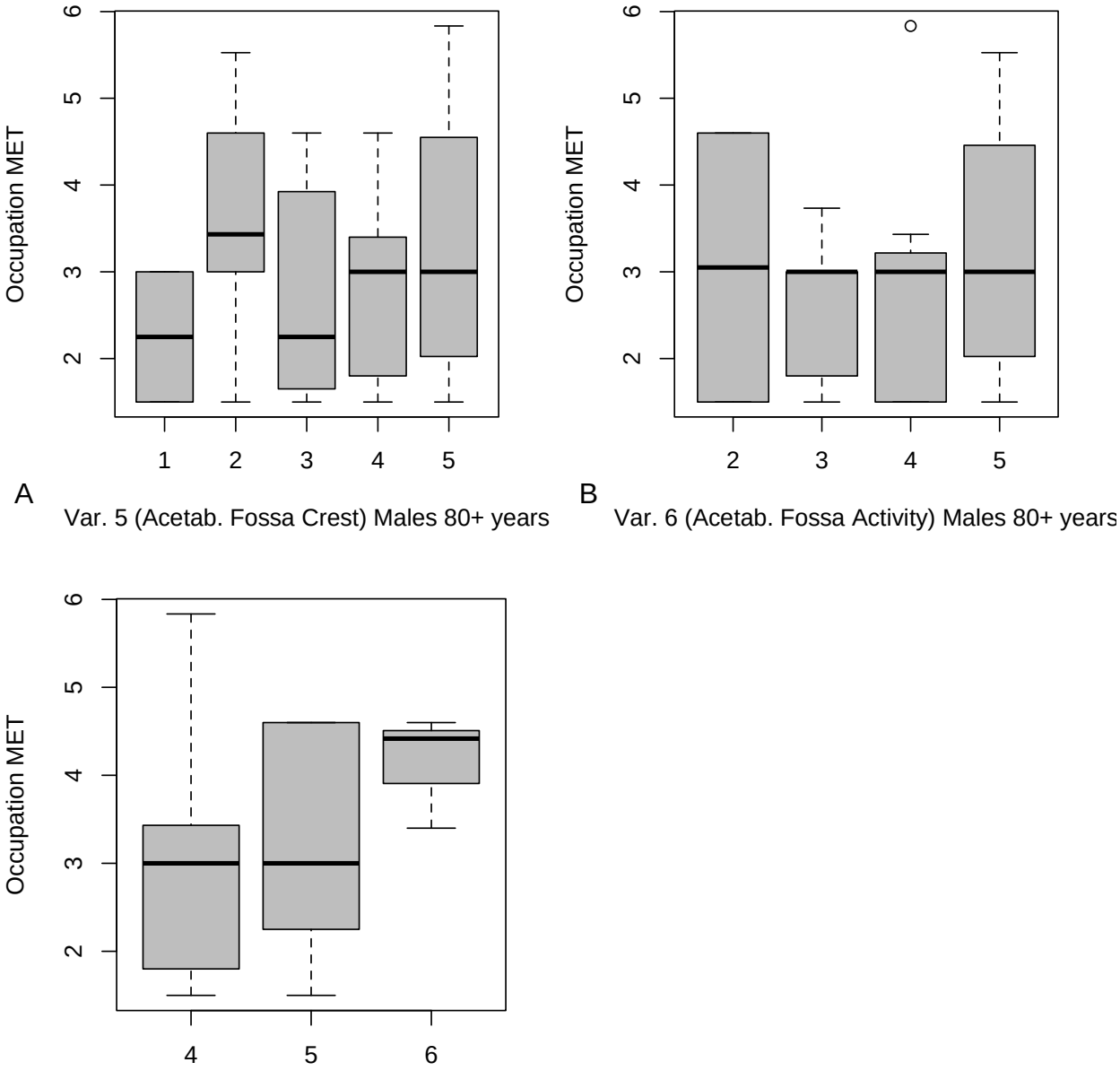


Figure 6-76. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for occupation MET value per acetabular variable score in males 80+ years. A) Variable 5, B) Variable 6, C) Variable 7.

Table 6-22. Spearman's rank-order correlations for MET occupation values and scores for acetabular Variable 1 through Variable 7 in females and males.

Group	*rho* (1)	*rho* (2)	*rho* (3)	*rho* (4)	*rho* (5)	*rho* (6)	*rho* (7)
Females <50 years	0.174	.0178	0.125	-0.108	-0.104	-0.035	0.103
Females 50-59 years	-0.076	-0.162	-0.129	0.003	0.084	0.461	0.365
Females 60-69 years	-0.134	0.307	-0.130	0.243	0.363	0.168	0.228
Females 70-79 years	-0.380	-0.267	-0.053	-0.272	-0.279	-0.003	0.083
Females 80+ years	-0.041	0.240	-0.216	0.176	-0.157	0.044	0.171
Males <40 years	-0.138	0.158	-0.092	-0.148	0.056	0.010	0.070
Males 40-49 years	-0.152	0.183	-0.009	-0.083	-0.026	-0.071	0.345
Males 50-59 years	0.038	-0.047	-0.060	0.017	-0.178	0.172	0.276
Males 60-69 years	-0.070	-0.036	0.169	-0.363	0.192	-0.125	0.139
Males 70-79 years	-0.408	-0.250	-0.289	-0.306	-0.133	0.438	0.193
Males 80+ years	0.202	-0.085	0.007	-0.090	0.048	0.161	0.262

*Statistically significant at α=0.001 (Bonferroni correction for females: α=0.05/35; for males: α=0.05/42).

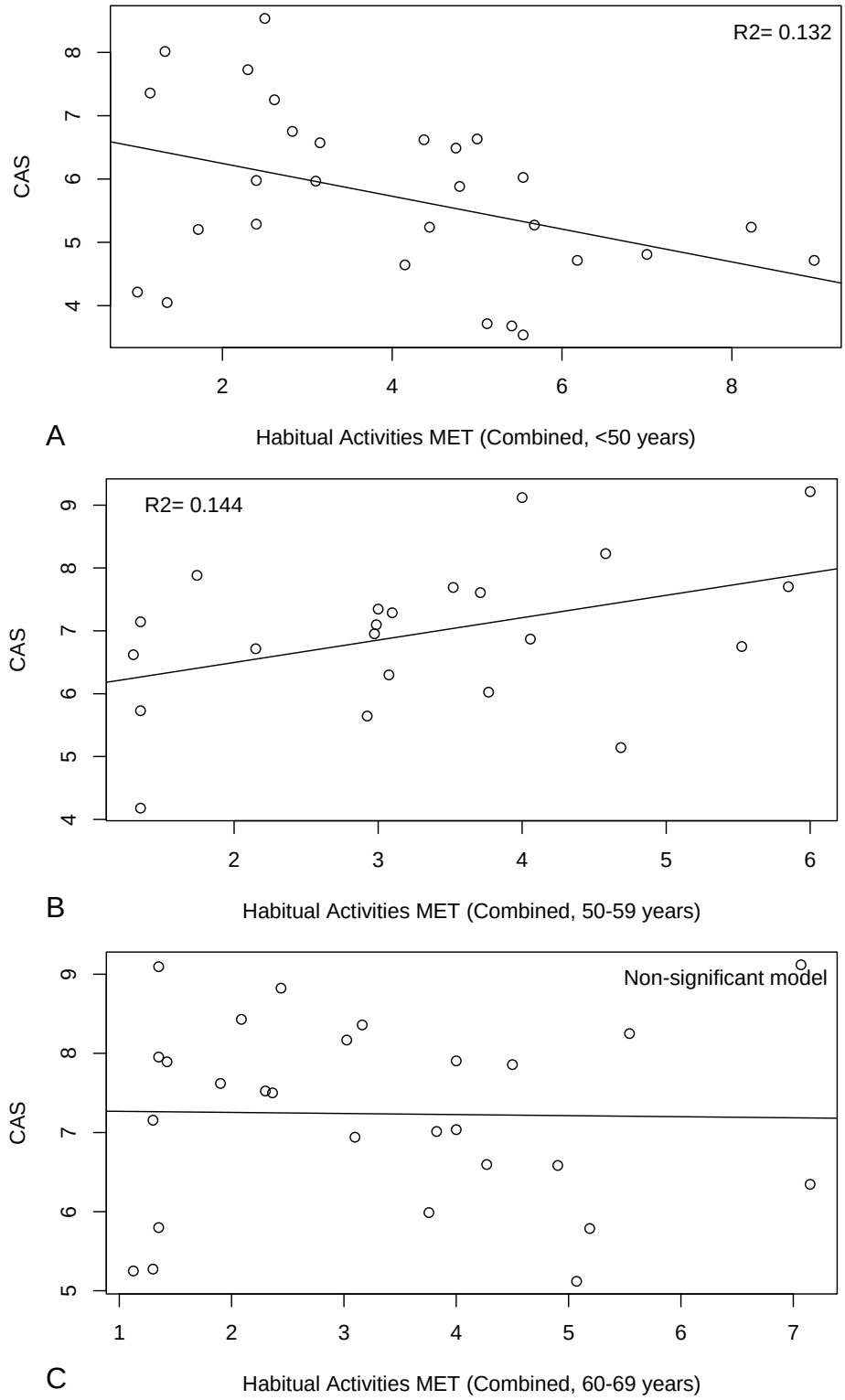


Figure 6-77. Hypothesis 3a: acetabular changes correlate positively with activity; scatterplots for CAS and MET habitual activities values in the combined-sex sample. A) <50 years, B) 50-59 years, C) 60-69 years. Black line indicates linear regression model.

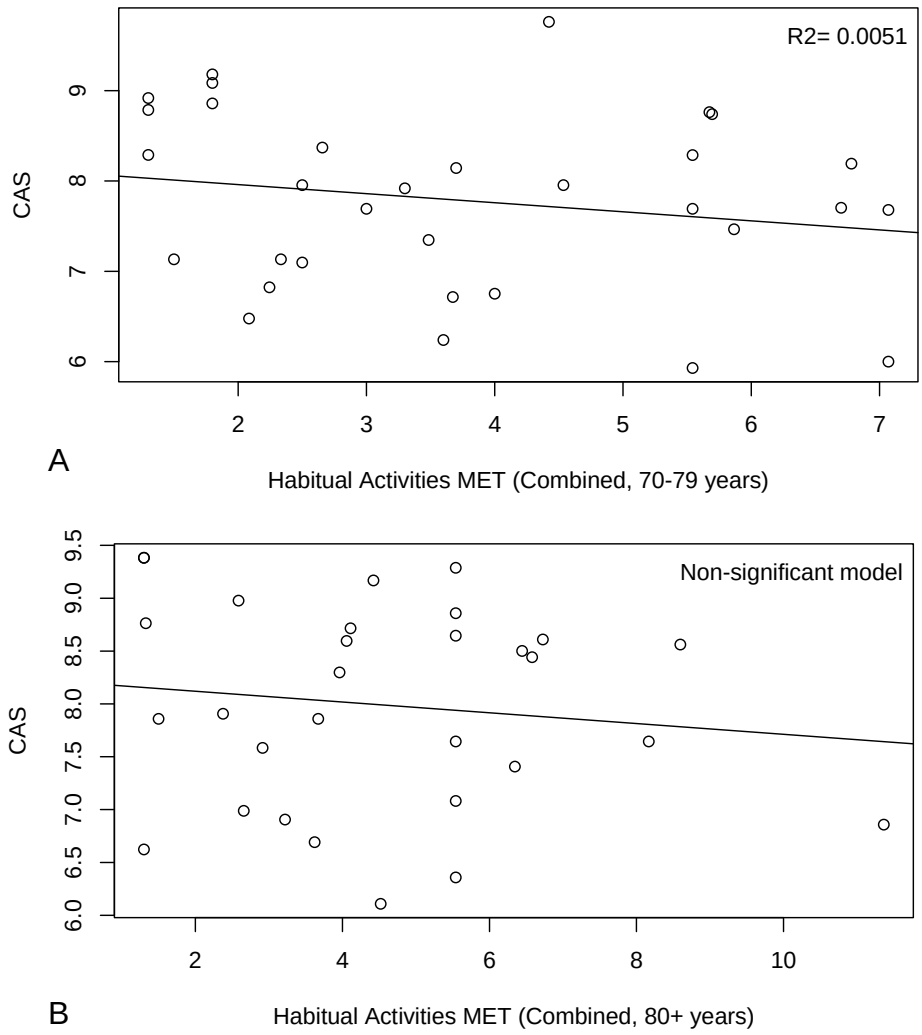


Figure 6-78. Hypothesis 3a: acetabular changes correlate positively with activity; scatterplots for CAS and MET habitual activities values in the combined-sex sample. A) 70-79 years, B) 80+ years. Black line indicates linear regression model.

Table 6-23. Spearman's rank-order correlations for CAS and MET values for habitual activities in the combined-sex sample.

Group	*rho*
<50 years	-0.395
50-59 years	0.409
60-69 years	-0.020
70-79 years	-0.247
80+ years	-0.124

*Statistically significant at α=0.01 (Bonferroni correction: α=0.05/5).

Table 6-24. Regression results for CAS and MET values for habitual activities in the combined-sex sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	6.765	0.521	12.98	7.18e-13*
<50 years	-0.259	0.115	-2.26	0.033
Intercept	5.785	0.603	9.593	6.33e-09*
50-59 years	0.356	0.167	2.126	0.046
Intercept	7.282	0.496	14.688	8.42e-14*
60-69 years	-0.014	0.133	-0.103	0.919
Intercept	8.161	0.390	20.902	<2e-16*
70-79 years	-0.100	0.093	-1.077	0.29
Intercept	8.222	0.383	21.473	<2e-16*
80+ years	-0.051	0.075	-0.682	0.501

*Statistically significant at α=0.01 (Bonferroni correction: α=0.05/5).

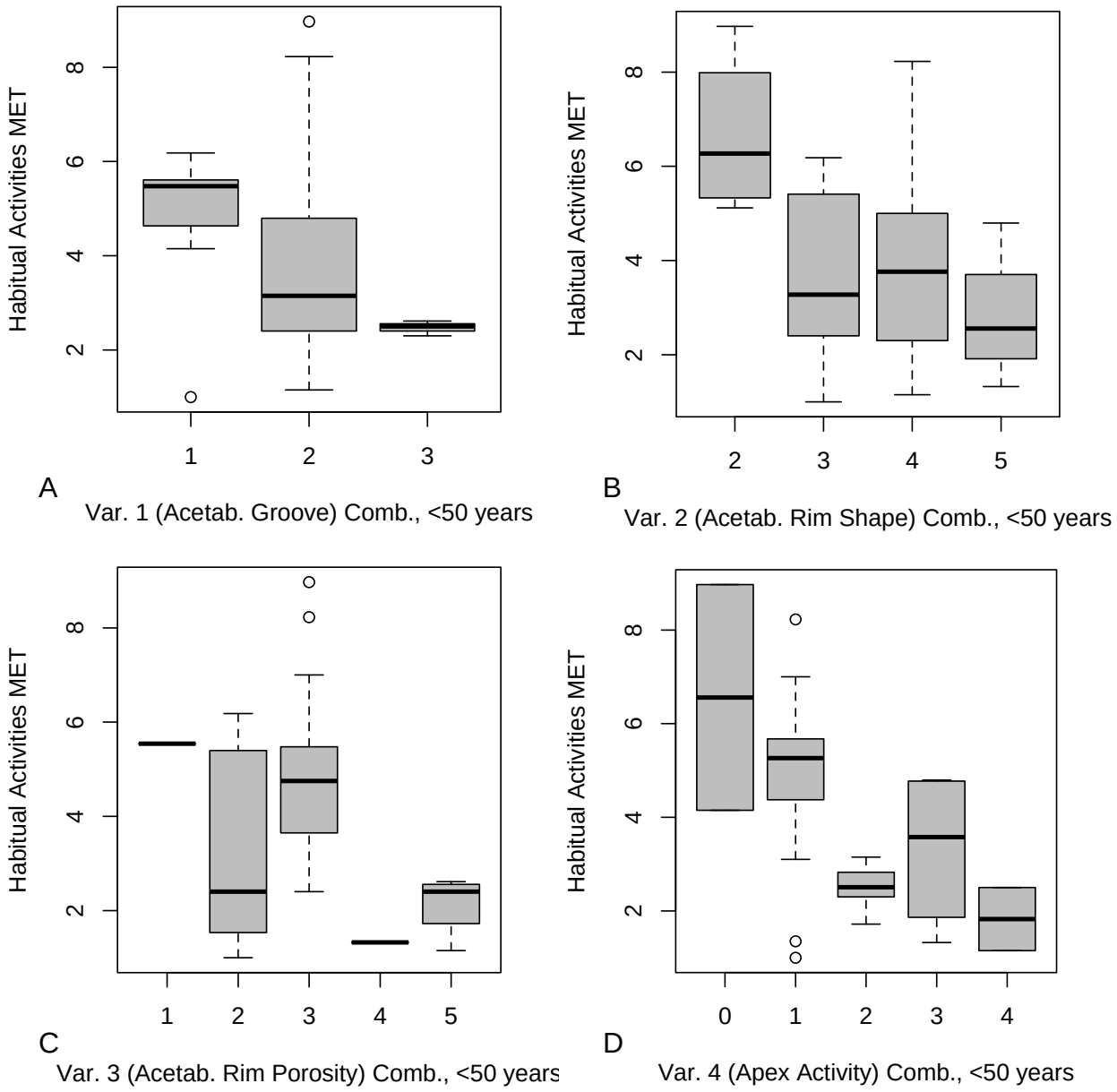


Figure 6-79. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for habitual activity MET value per acetabular variable score in the combined sample (<50 years). A) Variable 1, B) Variable 2, C) Variable 3, D) Variable 4.

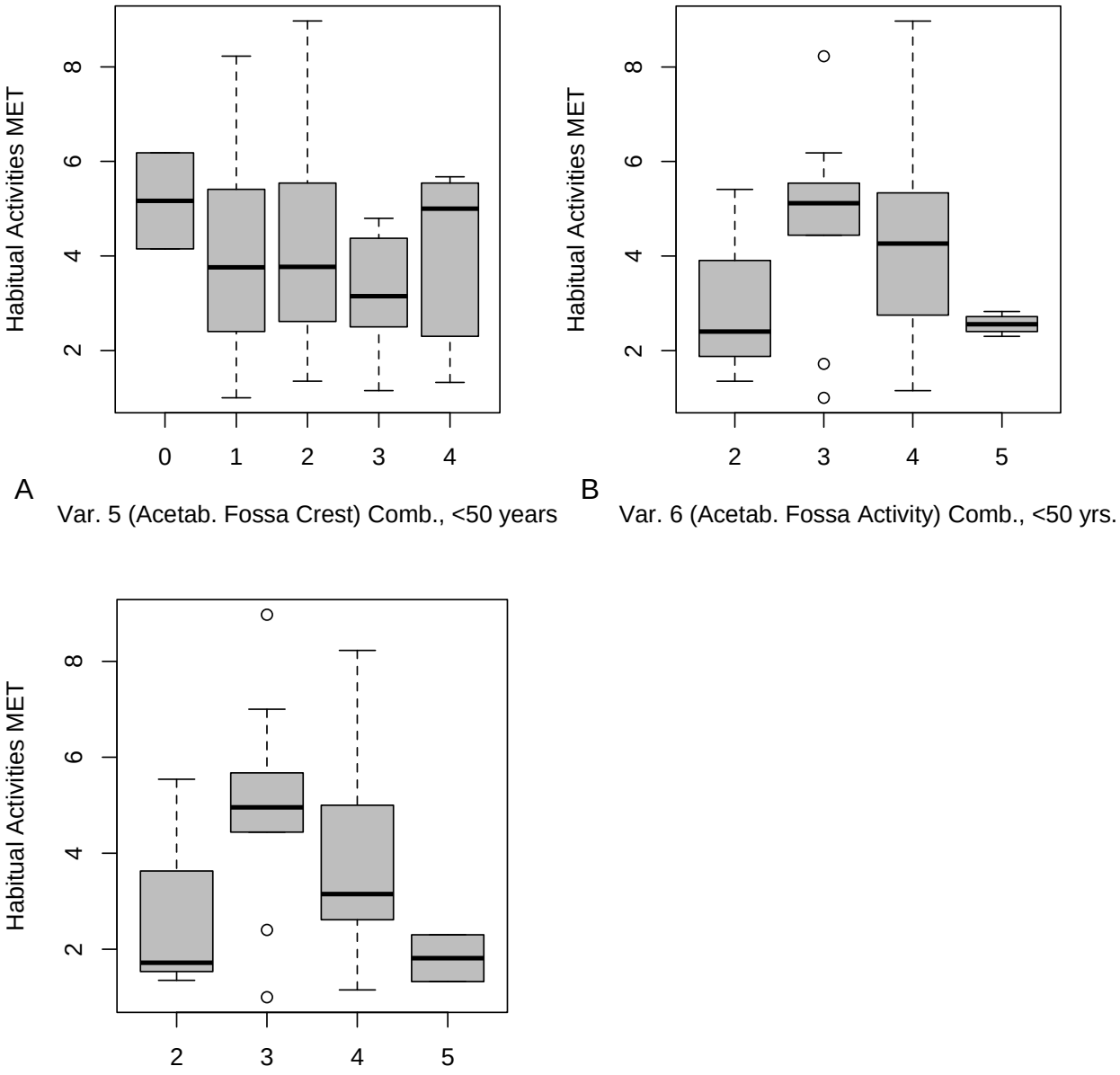


Figure 6-80. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for habitual activity MET value per acetabular variable score in the combined sample (<50 years). A) Variable 5, B) Variable 6, C) Variable 7.

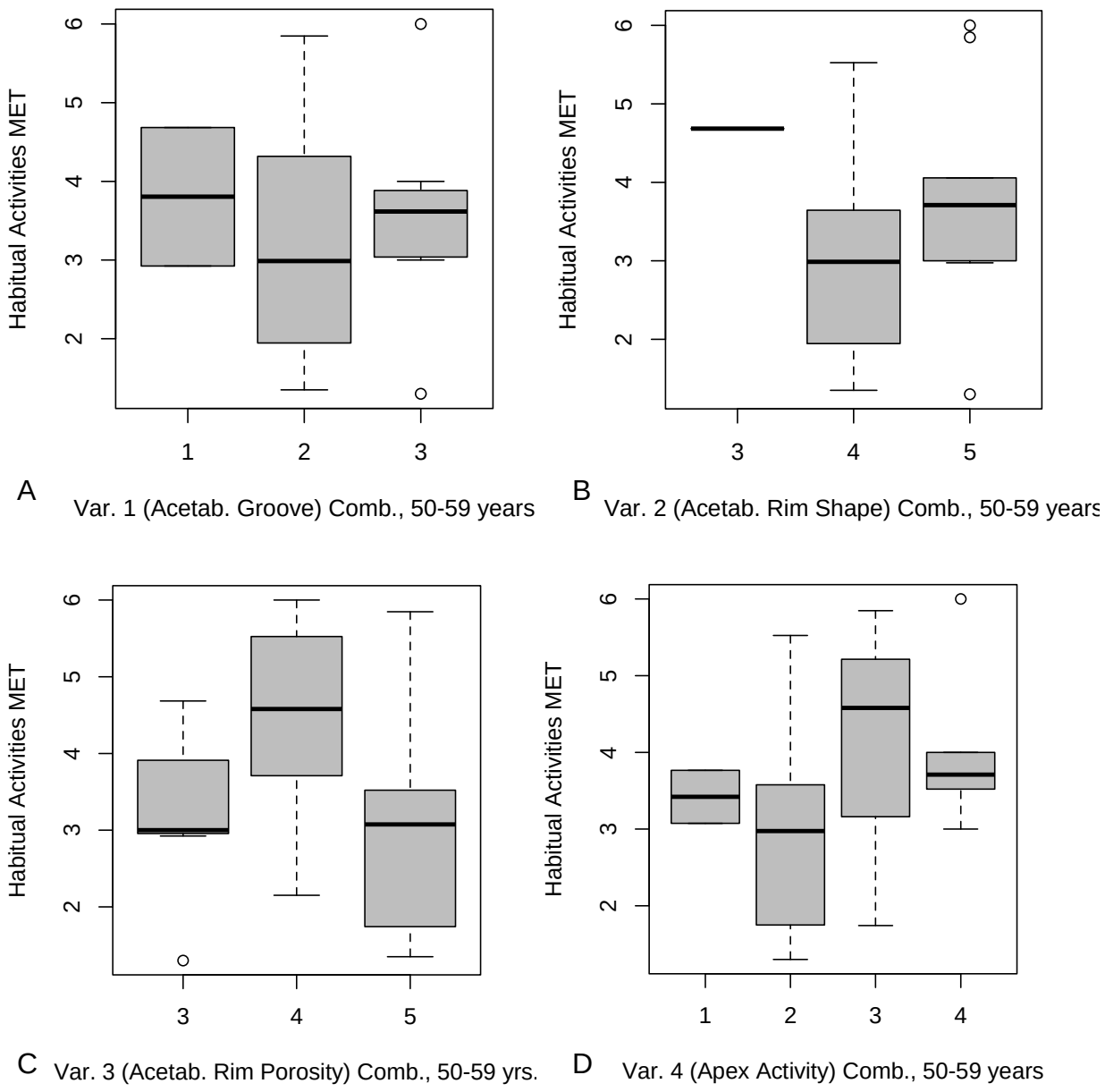
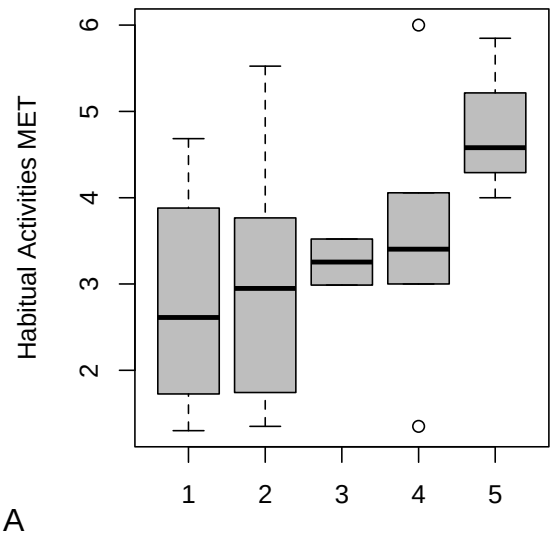
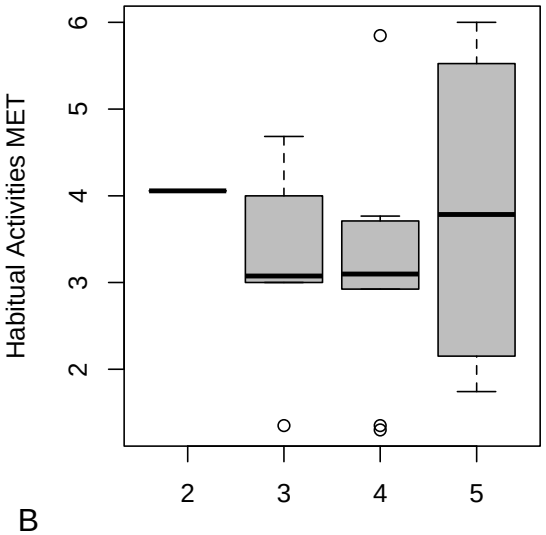


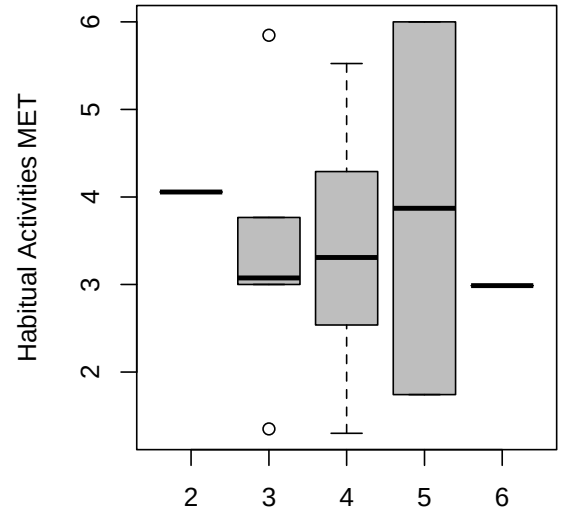
Figure 6-81. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for habitual activity MET value per acetabular variable score in the combined sample (50-59 years). A) Variable 1, B) Variable 2, C) Variable 3, D) Variable 4.



A Var. 5 (Acetab. Fossa Crest) Comb., 50-59 yrs.



B Var. 6 (Acetab. Fossa Activity) Comb., 50-59 yrs



C Var. 7 (Acet. Fossa Porosity) Comb., 50-59 yrs.

Figure 6-82. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for habitual activity MET value per acetabular variable score in the combined sample (50-59 years). A) Variable 5, B) Variable 6, C) Variable 7.

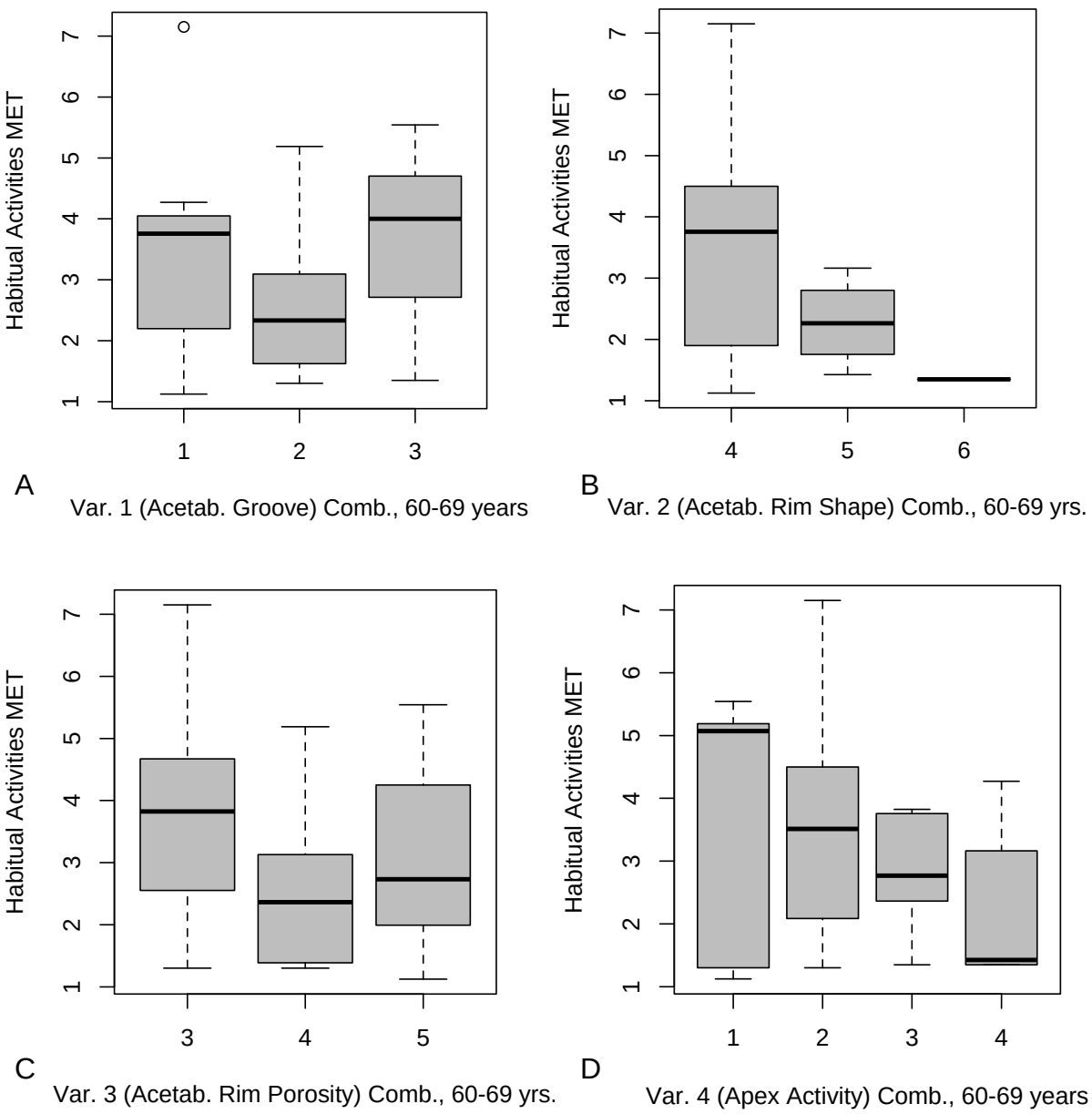
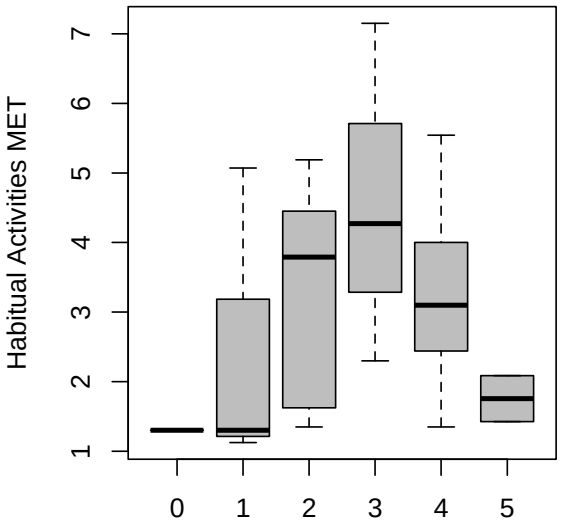
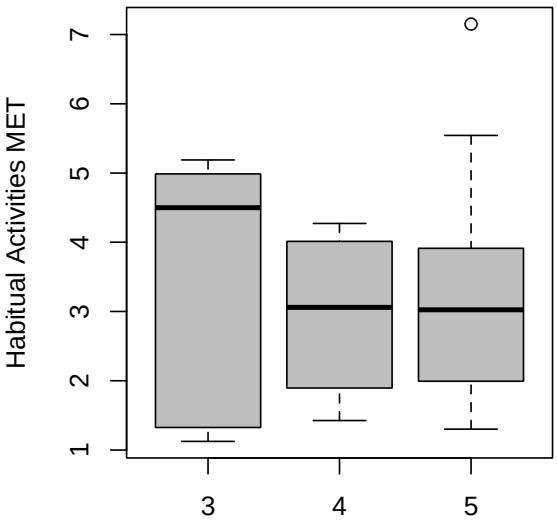


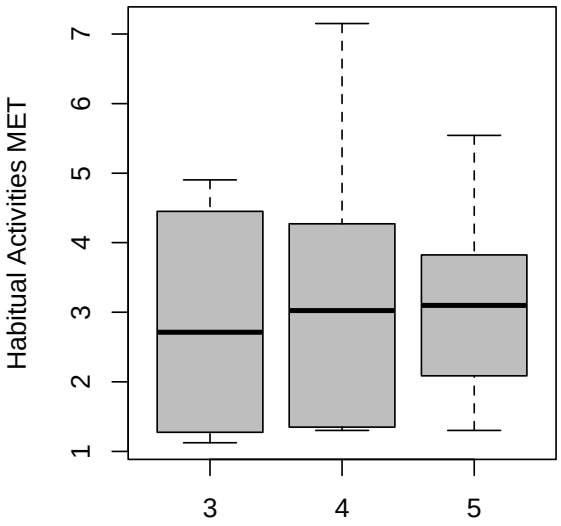
Figure 6-83. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for habitual activity MET value per acetabular variable score in the combined sample (60-69 years). A) Variable 1, B) Variable 2, C) Variable 3, D) Variable 4.



A Var. 5 (Acetab. Fossa Crest) Comb., 60-69 yrs.



B Var. 6 (Acetab. Fossa Activity) Comb., 60-69 yrs



C Var. 7 (Acet. Fossa Porosity) Comb., 60-69 yrs.

Figure 6-84. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for habitual activity MET value per acetabular variable score in the combined sample (60-69 years). A) Variable 5, B) Variable 6, C) Variable 7.

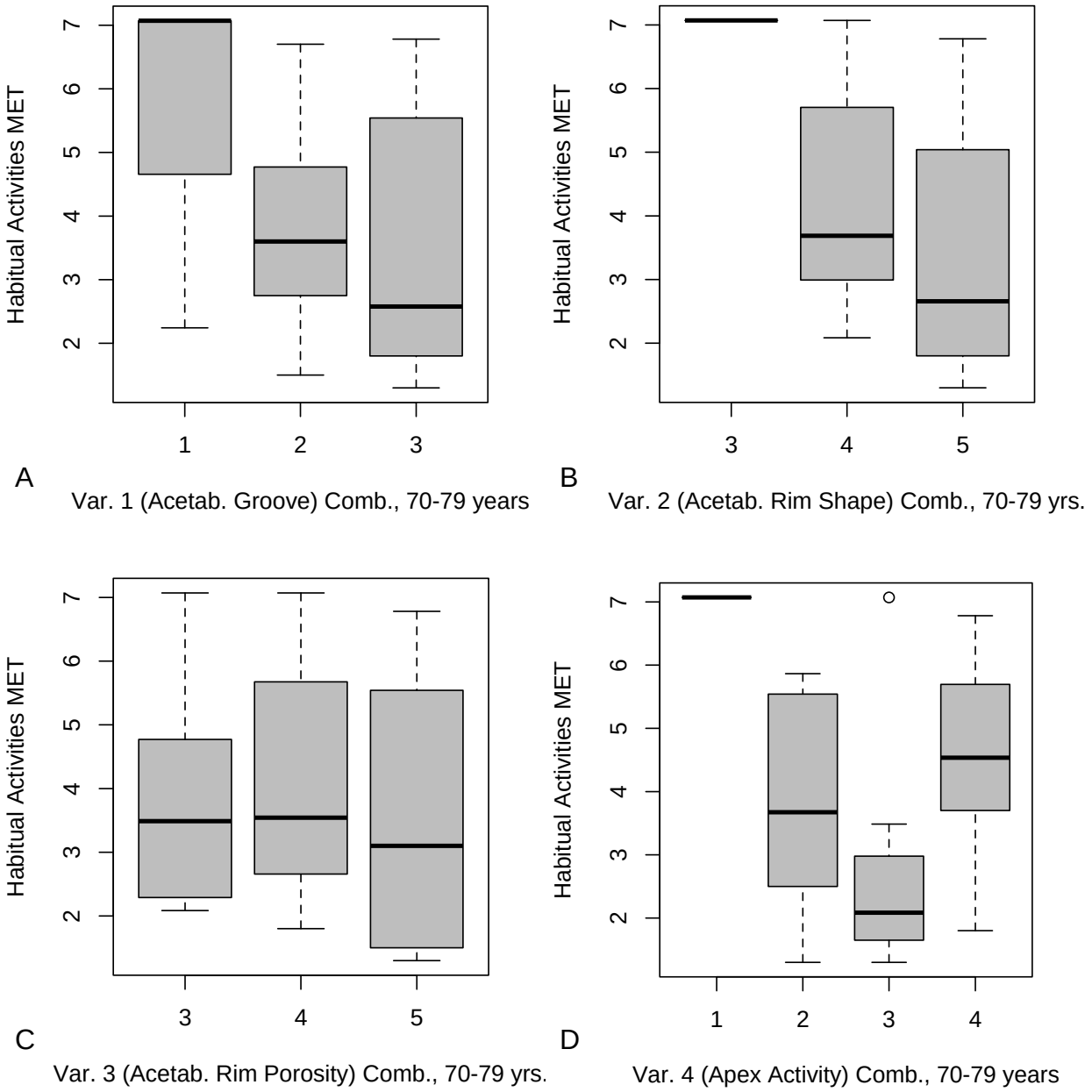


Figure 6-85. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for habitual activity MET value per acetabular variable score in the combined sample (70-79 years). A) Variable 1, B) Variable 2, C) Variable 3, D) Variable 4.

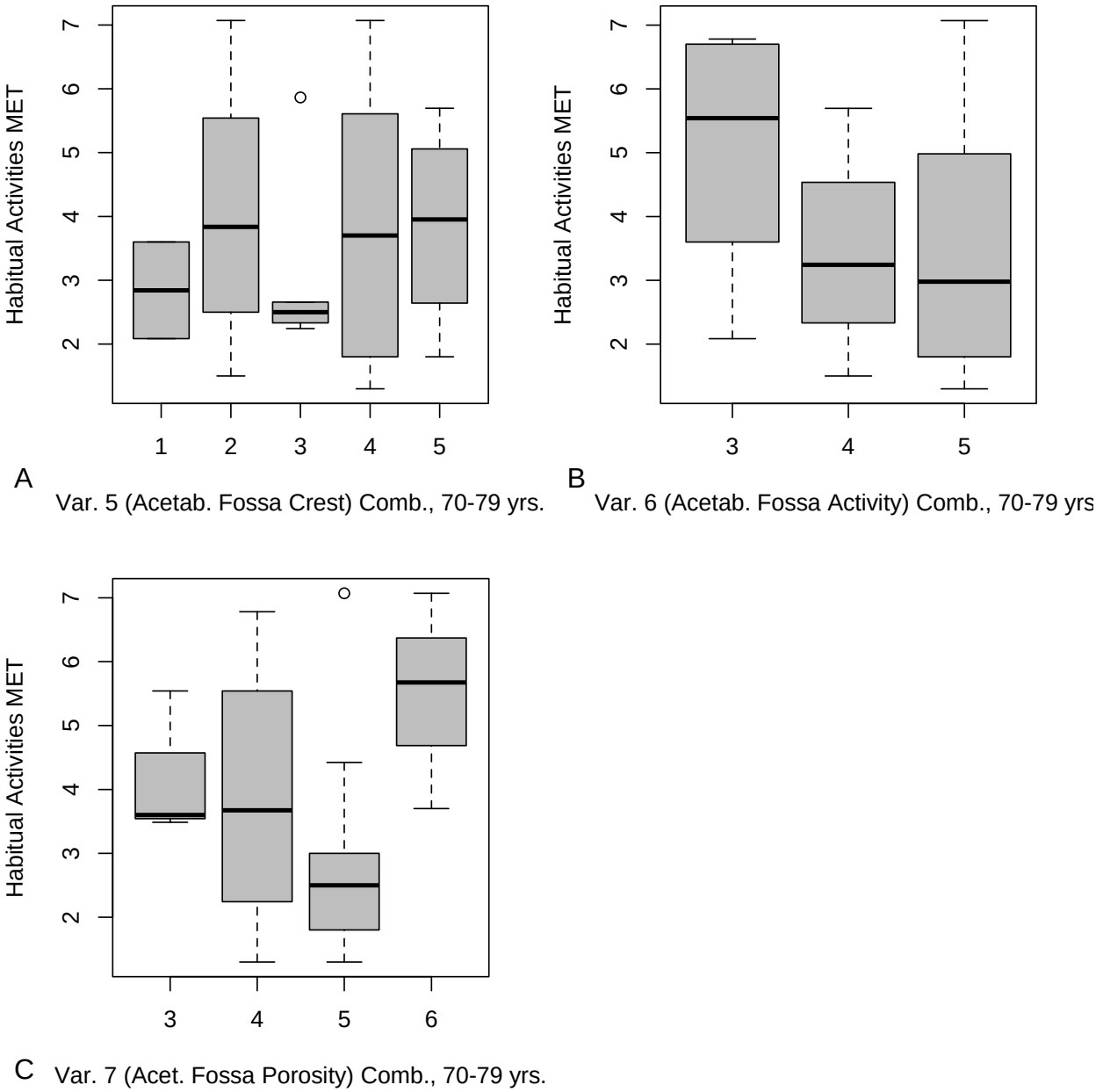


Figure 6-86. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for habitual activity MET value per acetabular variable score in the combined sample (70-79 years). A) Variable 5, B) Variable 6, C) Variable 7.

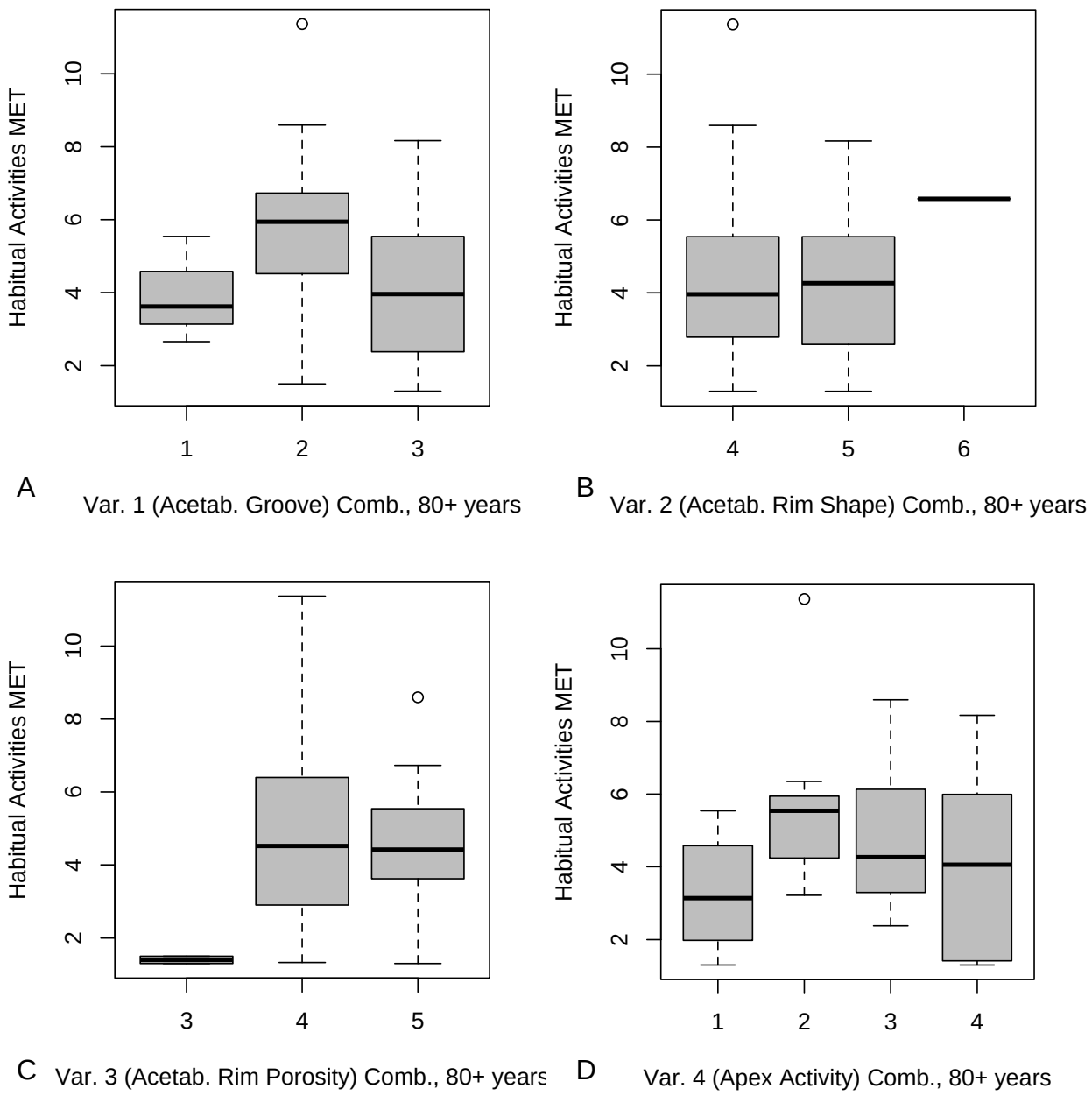


Figure 6-87. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for habitual activity MET value per acetabular variable score in the combined sample (80+ years). A) Variable 1, B) Variable 2, C) Variable 3, D) Variable 4.

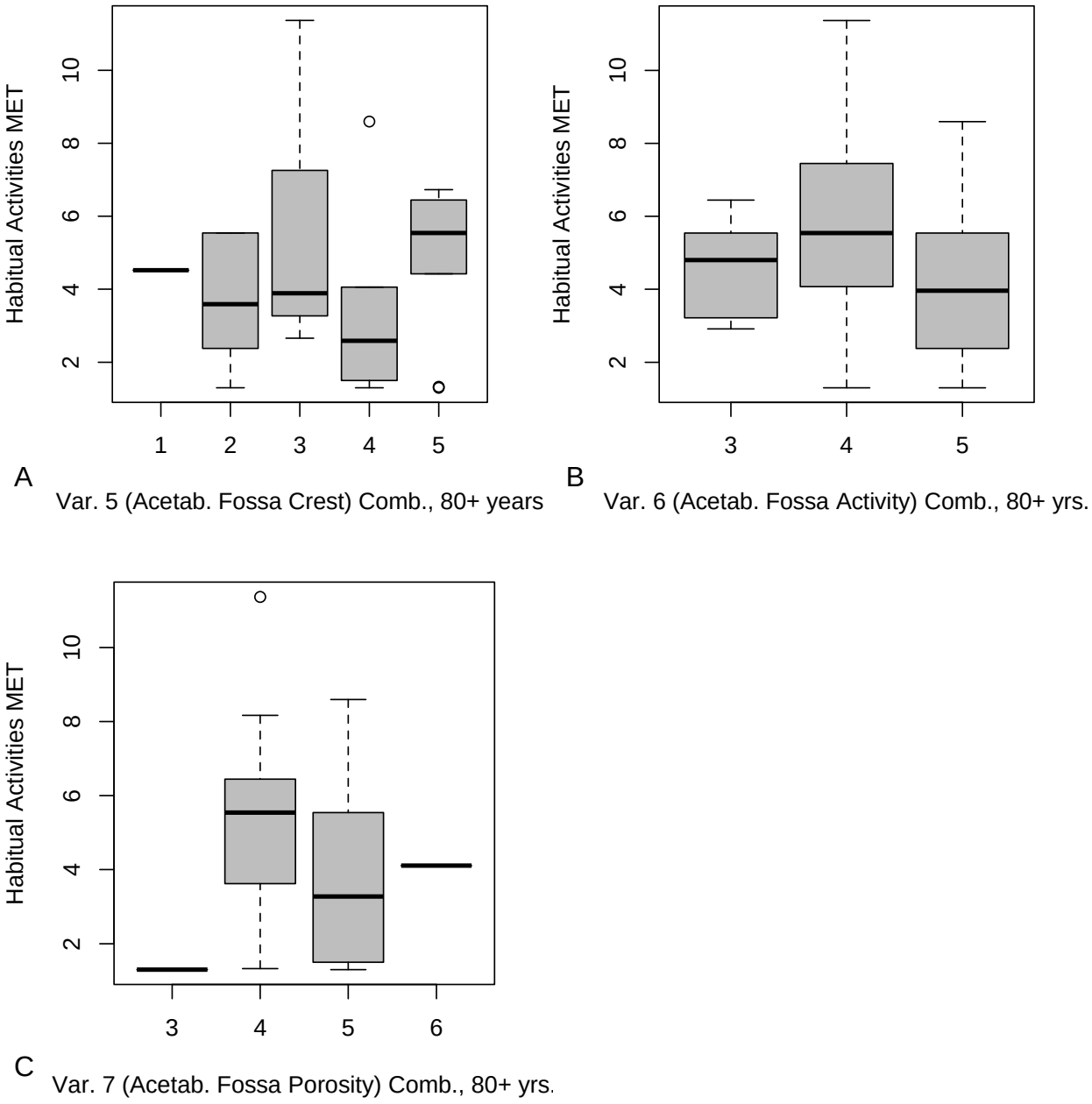


Figure 6-88. Hypothesis 3a: acetabular changes correlate positively with activity; box-and-whisker plots for habitual activity MET value per acetabular variable score in the combined sample (80+ years). A) Variable 5, B) Variable 6, C) Variable 7.

Table 6-25. Spearman's rank-order correlations for MET values for habitual activities and scores for acetabular Variable 1 through Variable 7 in the combined-sex sample.

Group	*rho* (1)	*rho* (2)	*rho* (3)	*rho* (4)	*rho* (5)	*rho* (6)	*rho* (7)
<50 years	-0.385	-0.372	-0.221	-0.565	-0.106	-0.164	-0.193
50-59 years	0.078	0.130	-0.104	0.295	0.443	0.019	-0.096
60-69 years	0.143	-0.265	-0.070	-0.227	0.147	-0.046	0.052
70-79 years	-0.274	-0.359	-0.133	0.054	0.058	-0.234	-0.049
80+ years	-0.305	0.077	0.144	-0.041	0.174	-0.218	-0.185

*Statistically significant at α=0.001 (Bonferroni correction: α=0.05/35).

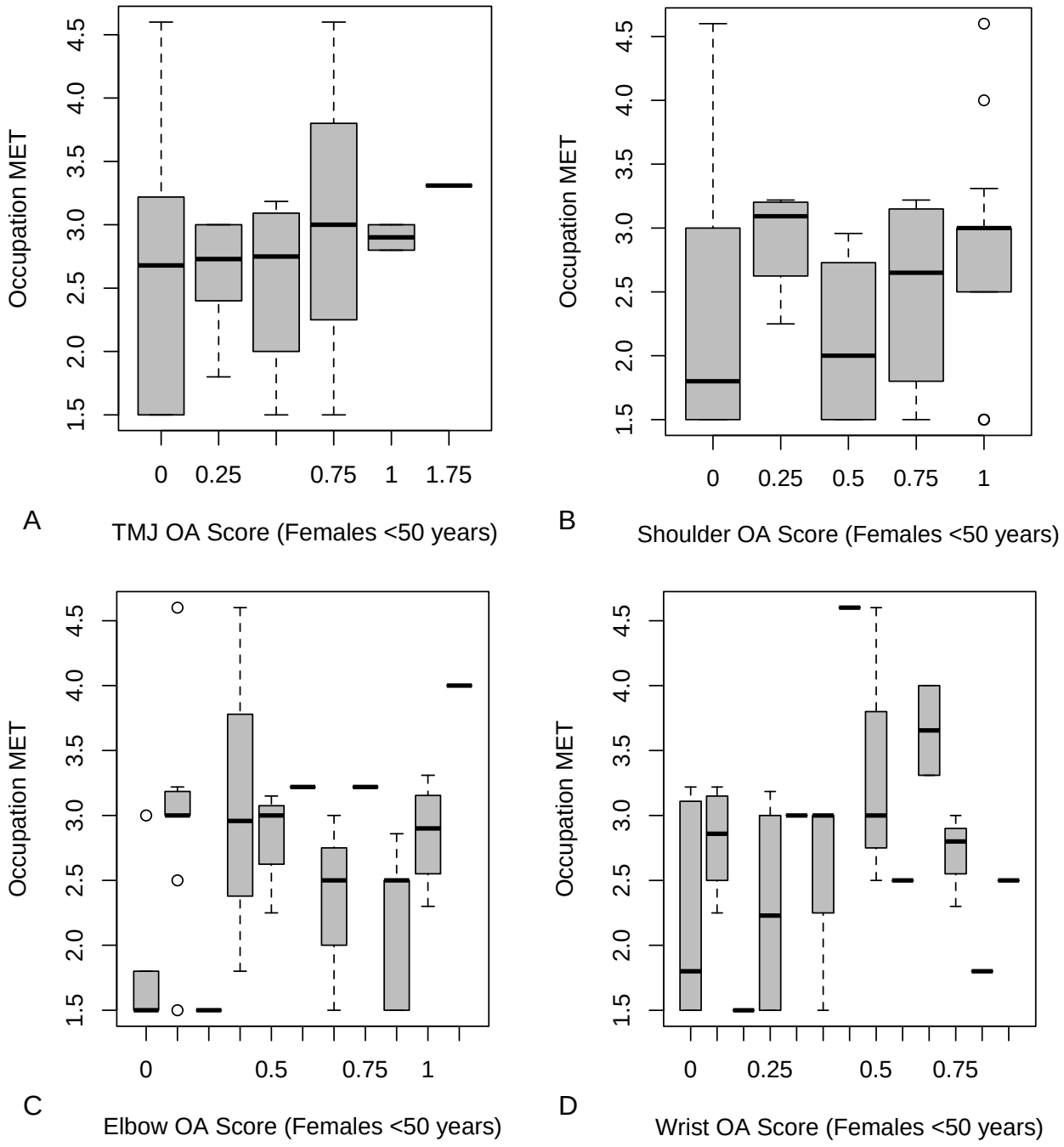


Figure 6-89. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for occupational activity MET value per OA score in females (<50 years). A) TMJ OA, B) Shoulder OA, C) Elbow OA, D) Wrist OA.

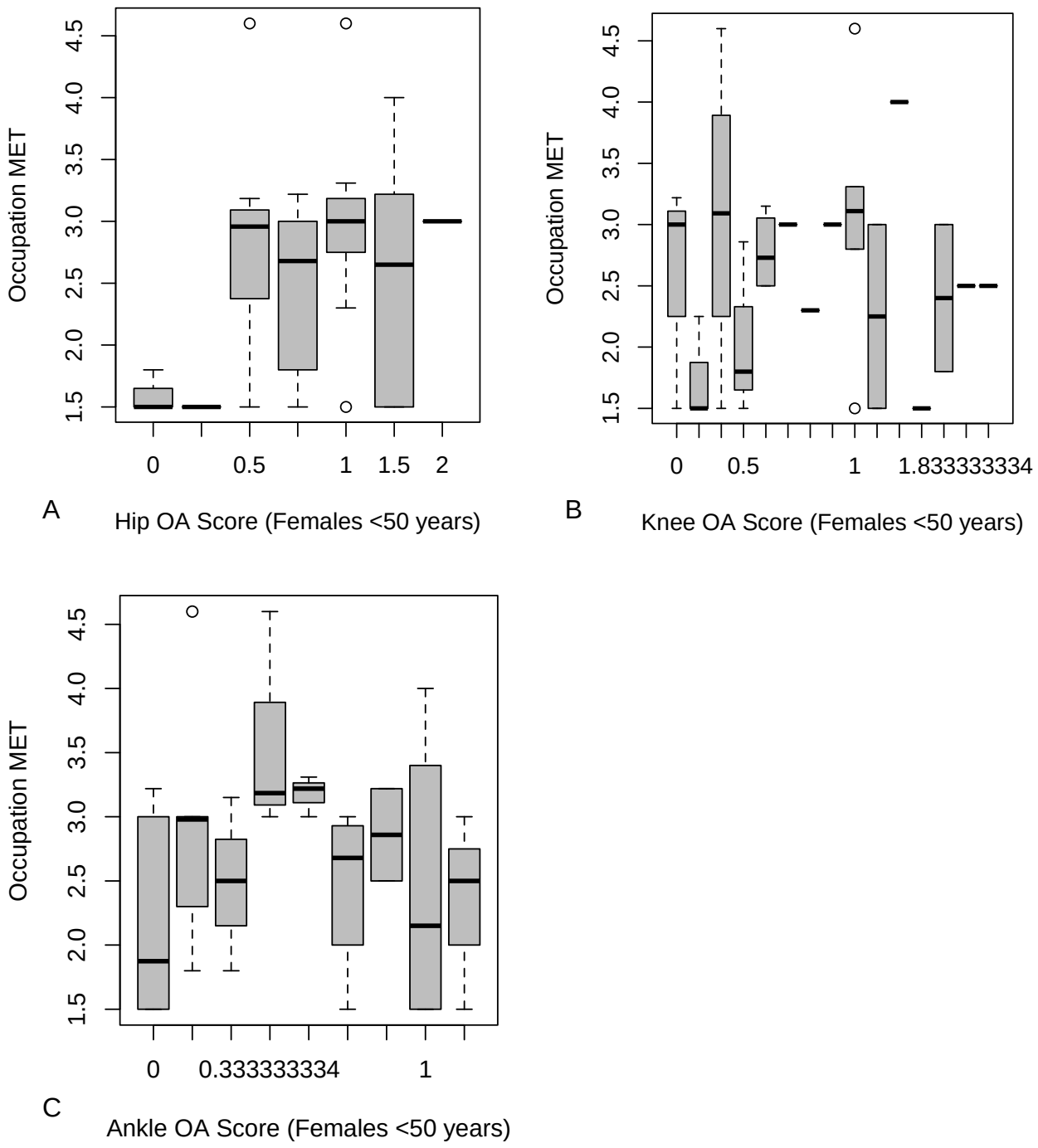


Figure 6-90. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for occupational activity MET value per OA score in females (<50 years). A) Hip OA, B) Knee OA, C) Ankle OA.

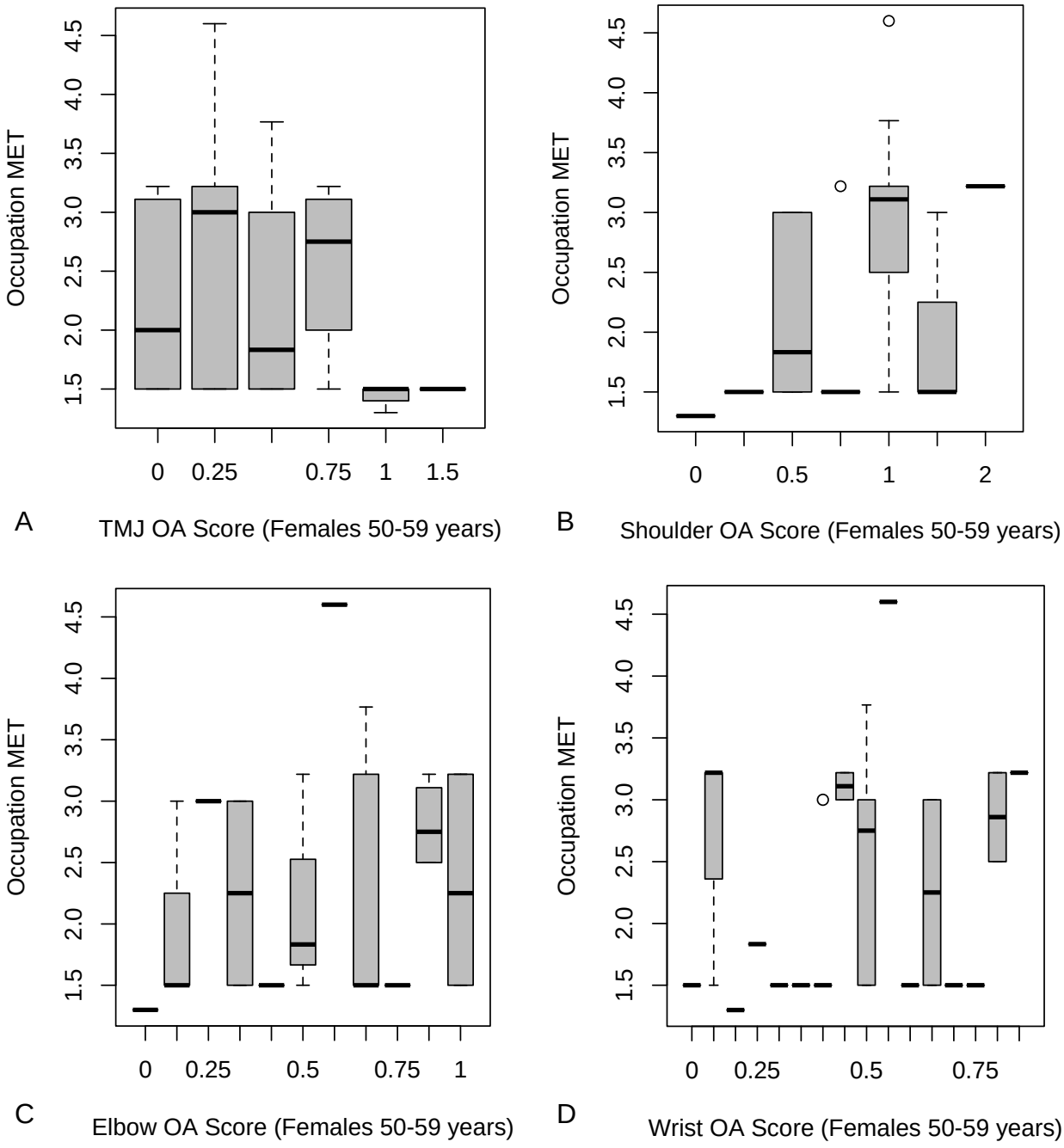


Figure 6-91. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for occupational activity MET value per OA score in females (50-59 years). A) TMJ OA, B) Shoulder OA, C) Elbow OA, D) Wrist OA.

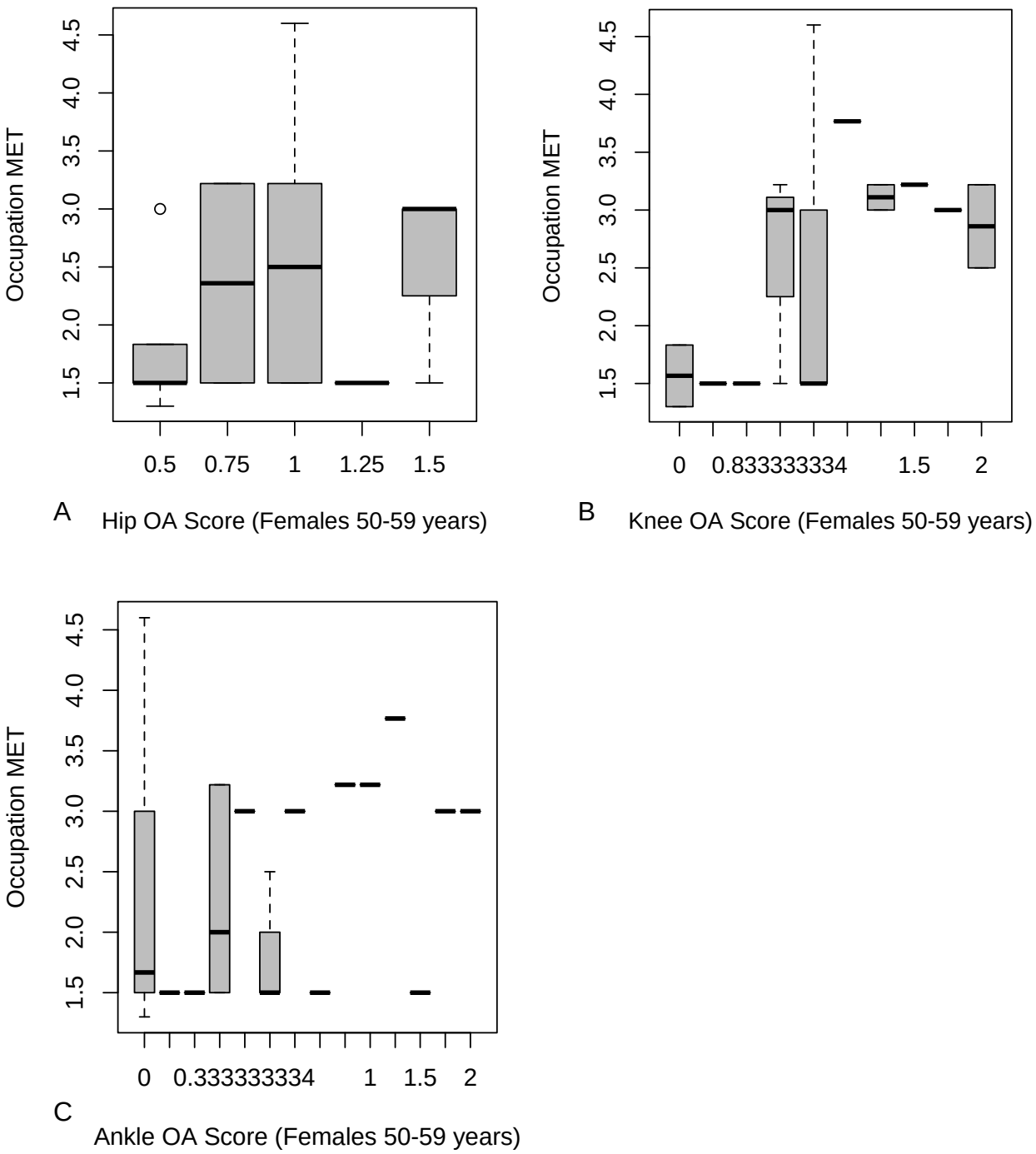


Figure 6-92. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for occupational activity MET value per OA score in females (50-59 years). A) Hip OA, B) Knee OA, C) Ankle OA.

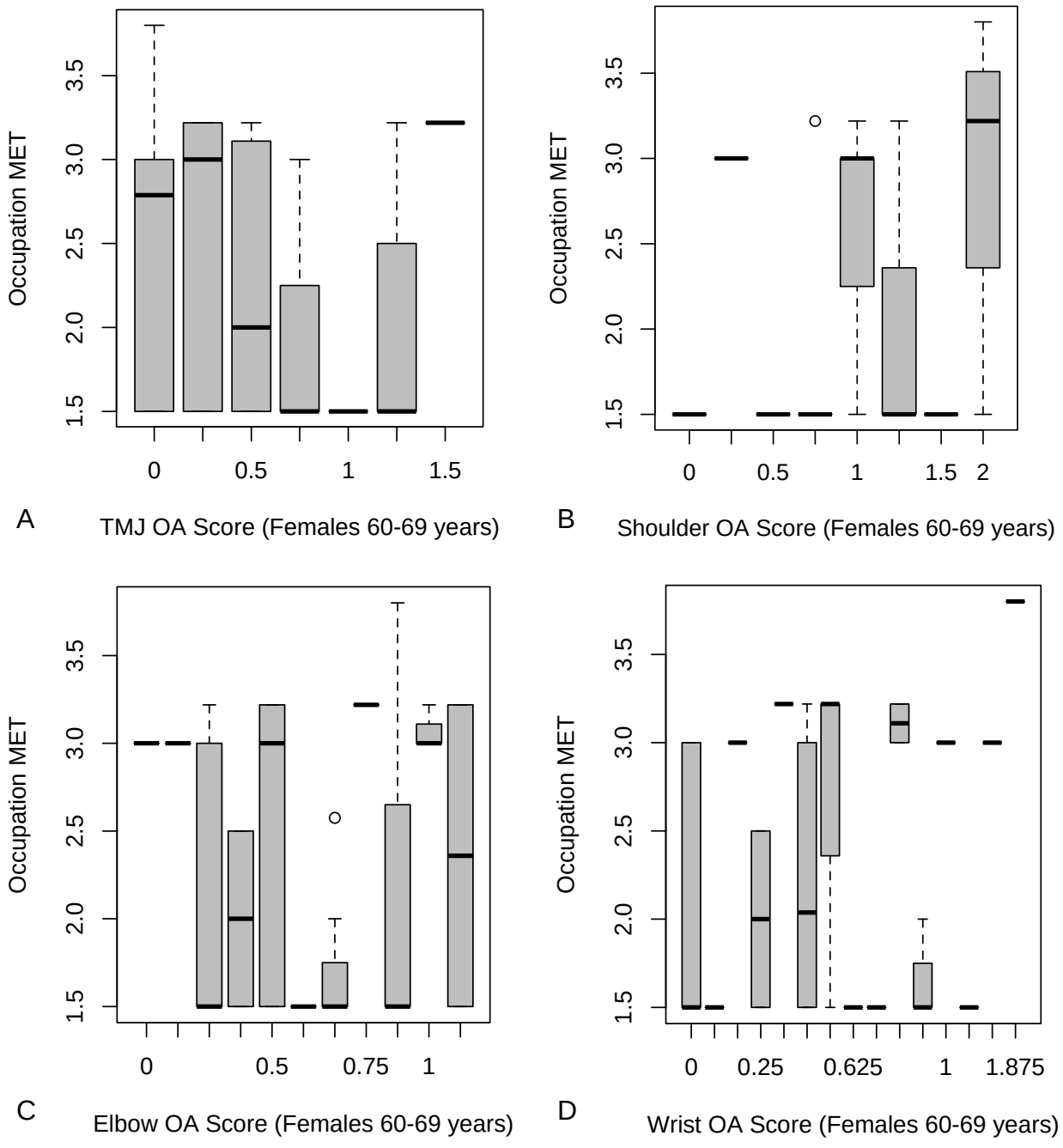


Figure 6-93. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for occupational activity MET value per OA score in females (60-69 years). A) TMJ OA, B) Shoulder OA, C) Elbow OA, D) Wrist OA.

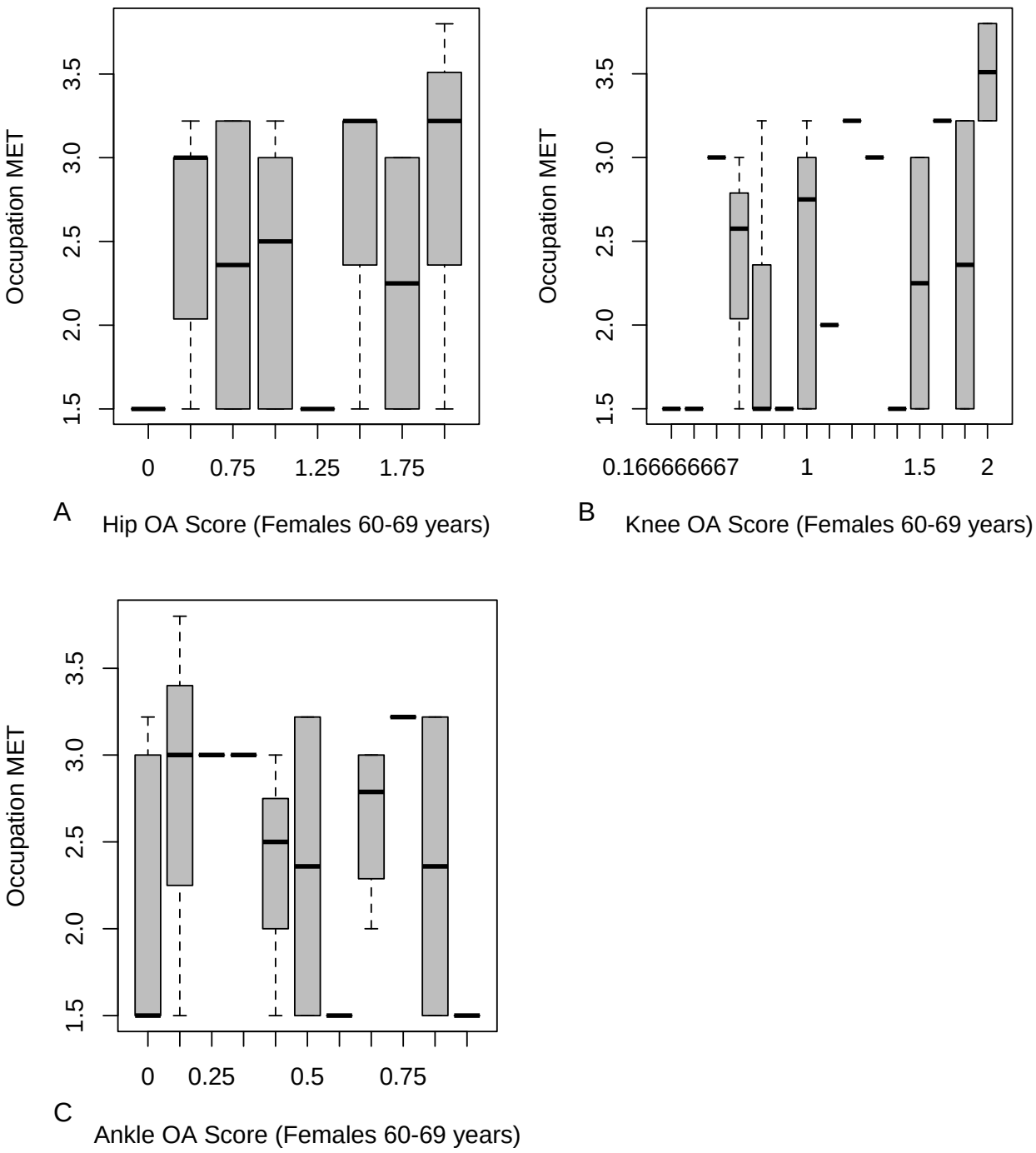


Figure 6-94. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for occupational activity MET value per OA score in females (60-69 years). A) Hip OA, B) Knee OA, C) Ankle OA.

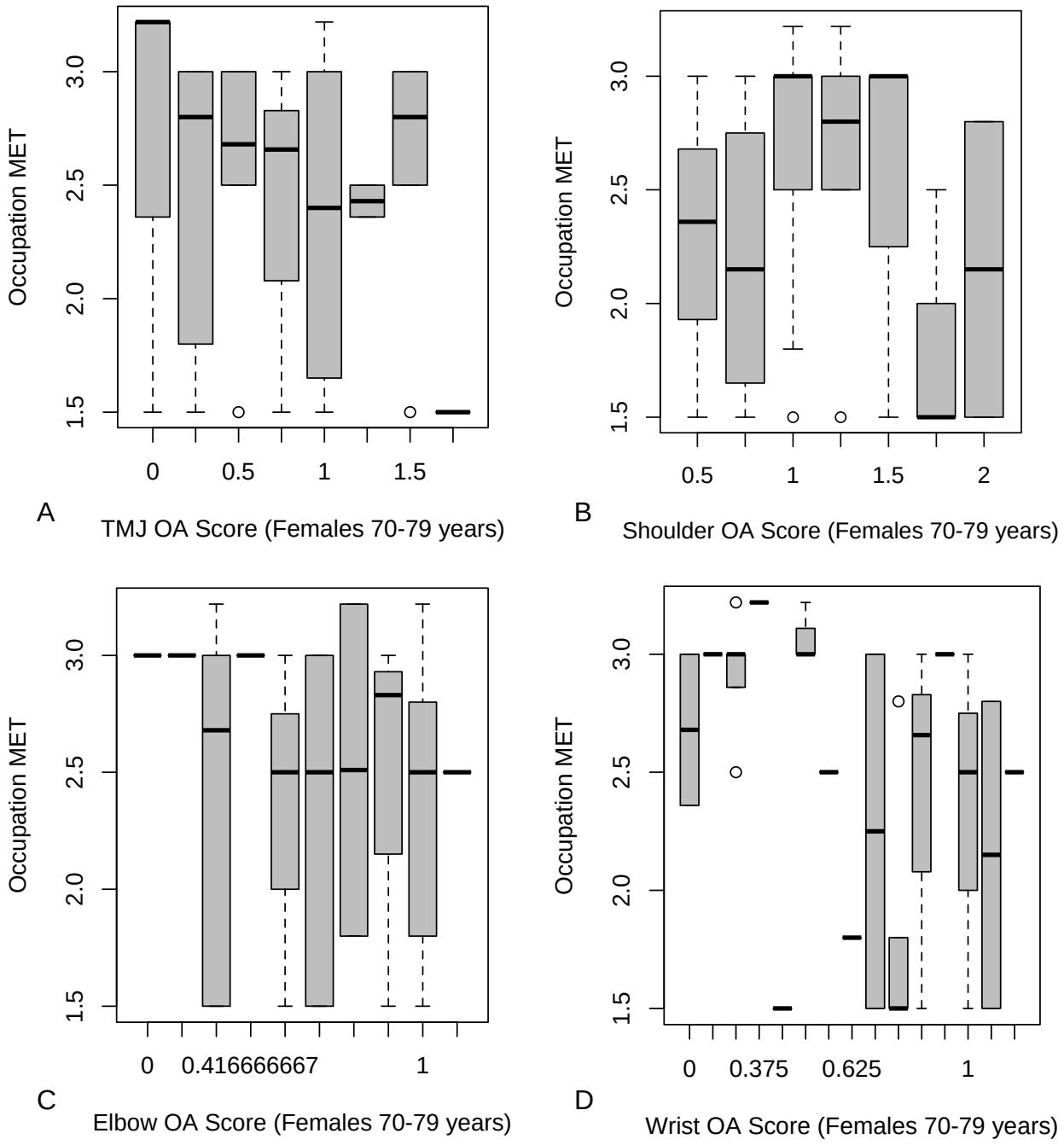


Figure 6-95. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for occupational activity MET value per OA score in females (70-79 years). A) TMJ OA, B) Shoulder OA, C) Elbow OA, D) Wrist OA.

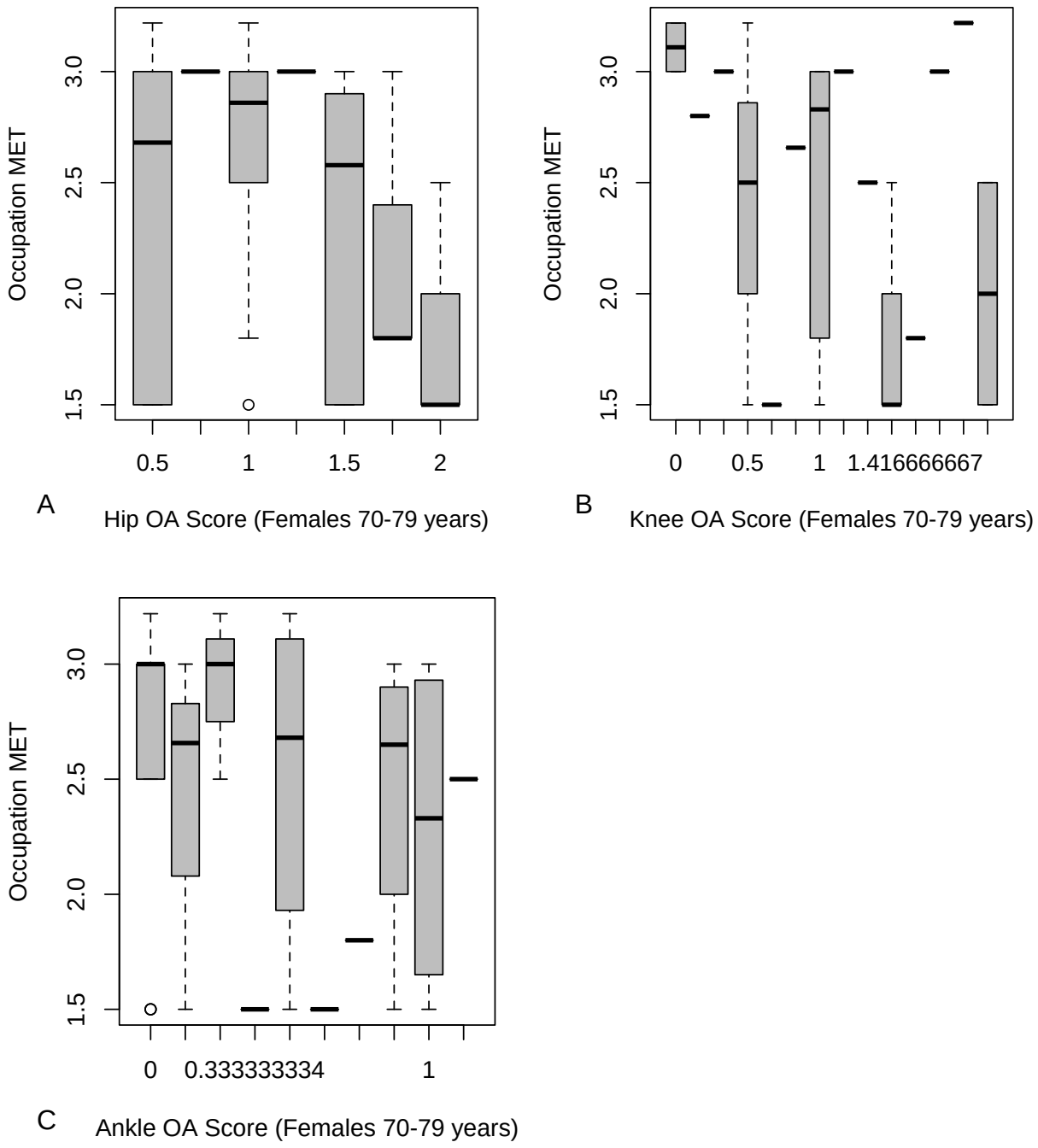


Figure 6-96. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for occupational activity MET value per OA score in females (70-79 years). A) Hip OA, B) Knee OA, C) Ankle OA.

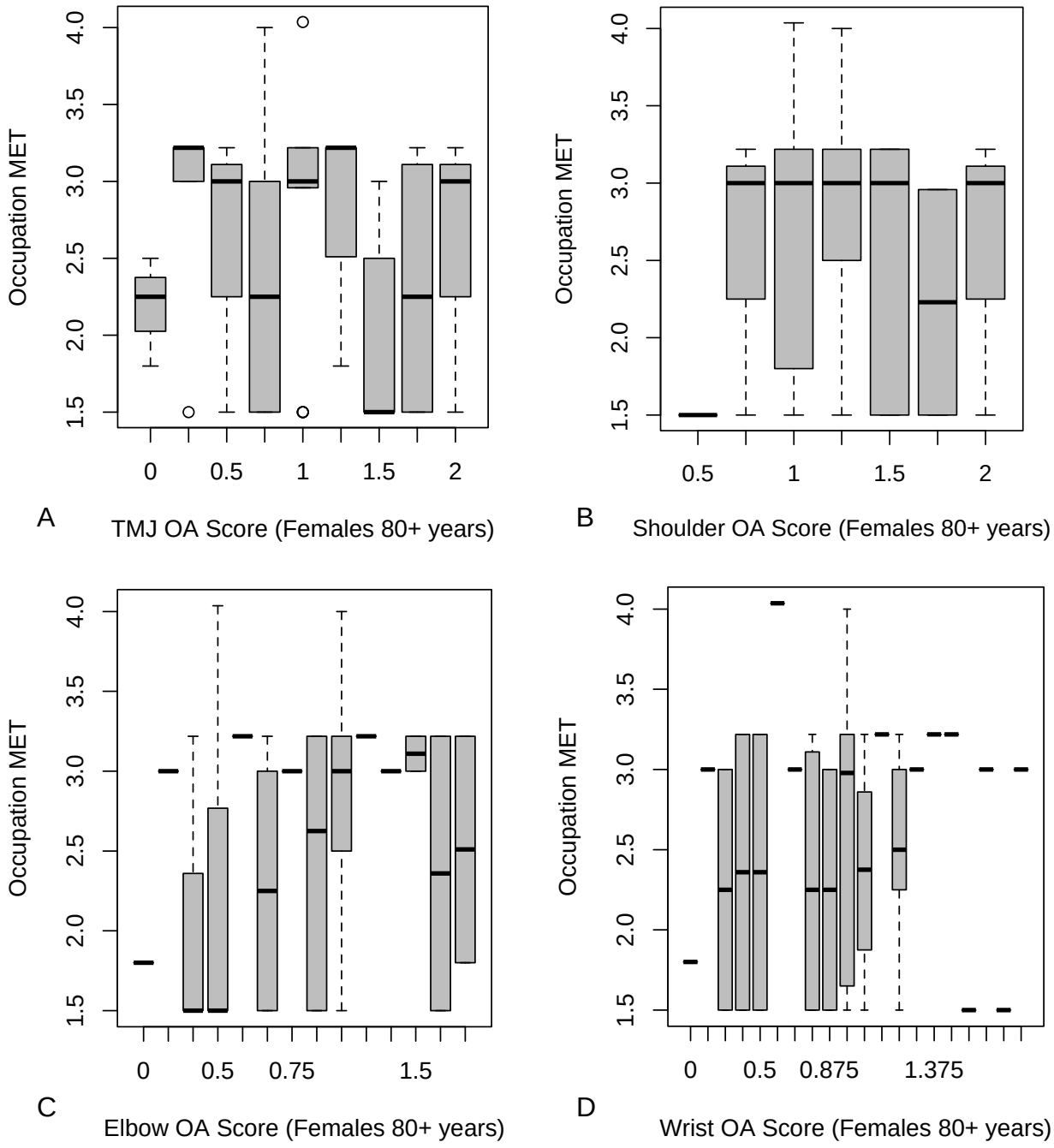


Figure 6-97. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for occupational activity MET value per OA score in females (80+ years). A) TMJ OA, B) Shoulder OA, C) Elbow OA, D) Wrist OA.

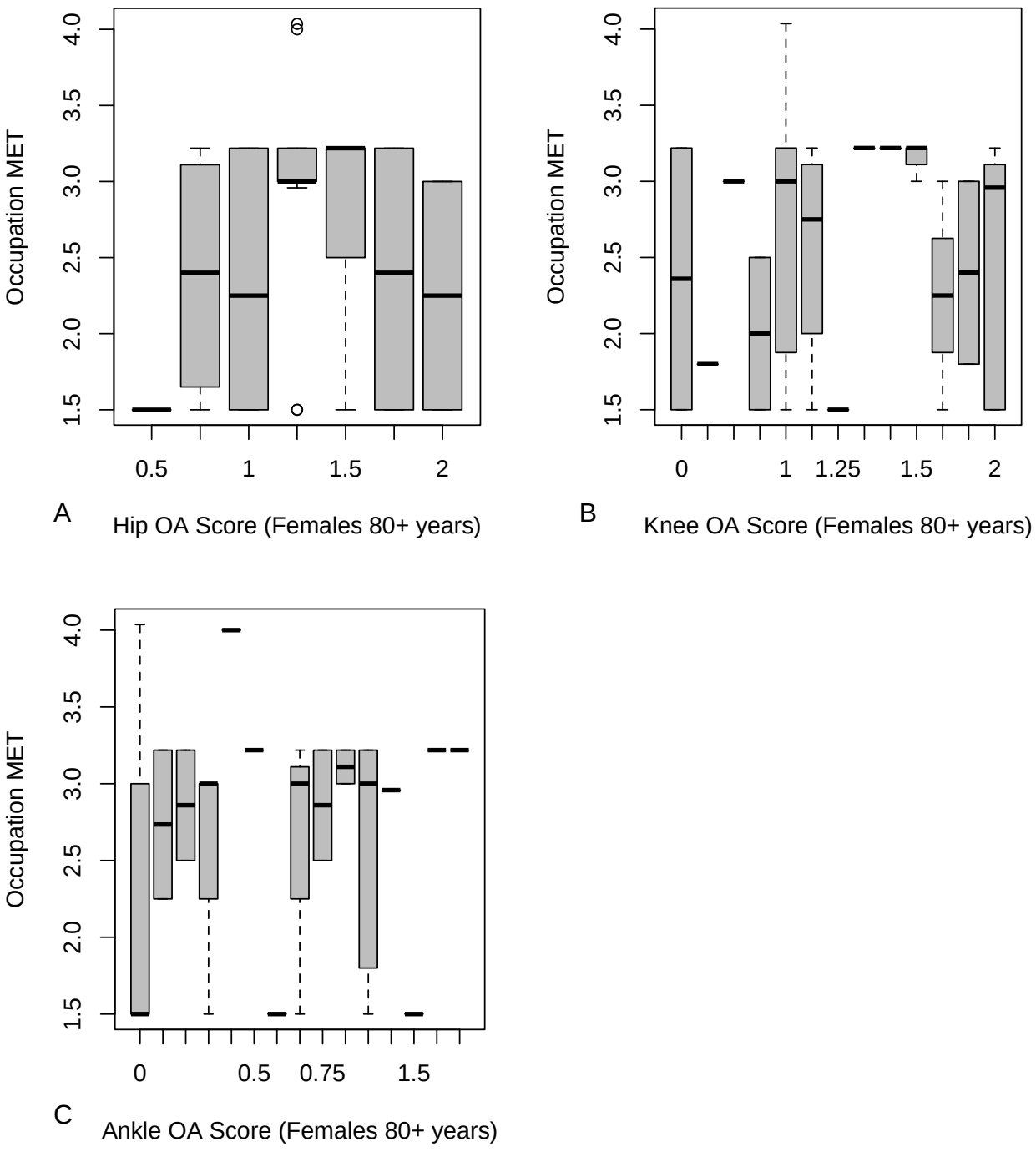


Figure 6-98. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for occupational activity MET value per OA score in females (80+ years). A) Hip OA, B) Knee OA, C) Ankle OA.

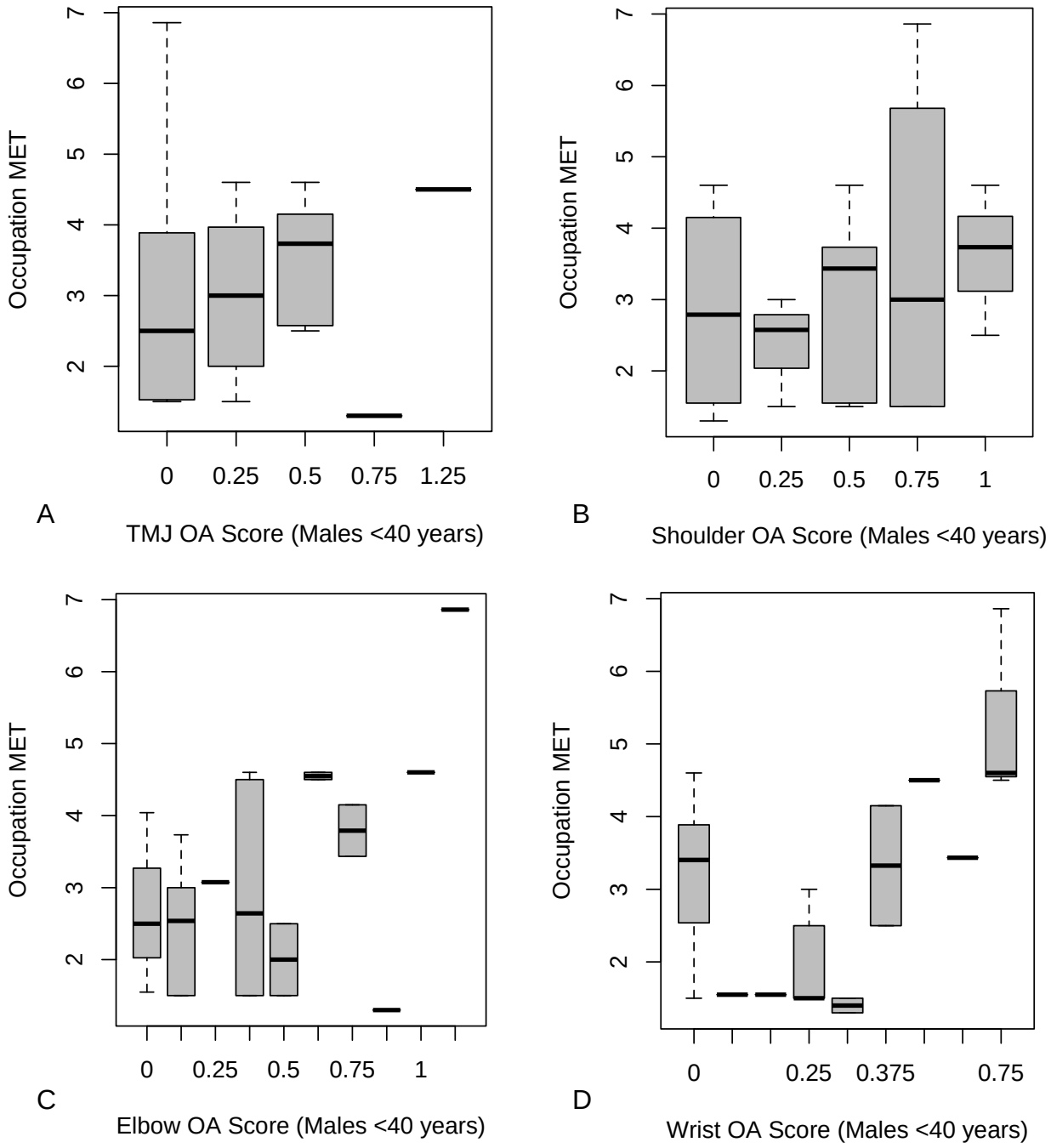


Figure 6-99. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for occupational activity MET value per OA score in males (<40 years). A) TMJ OA, B) Shoulder OA, C) Elbow OA, D) Wrist OA.

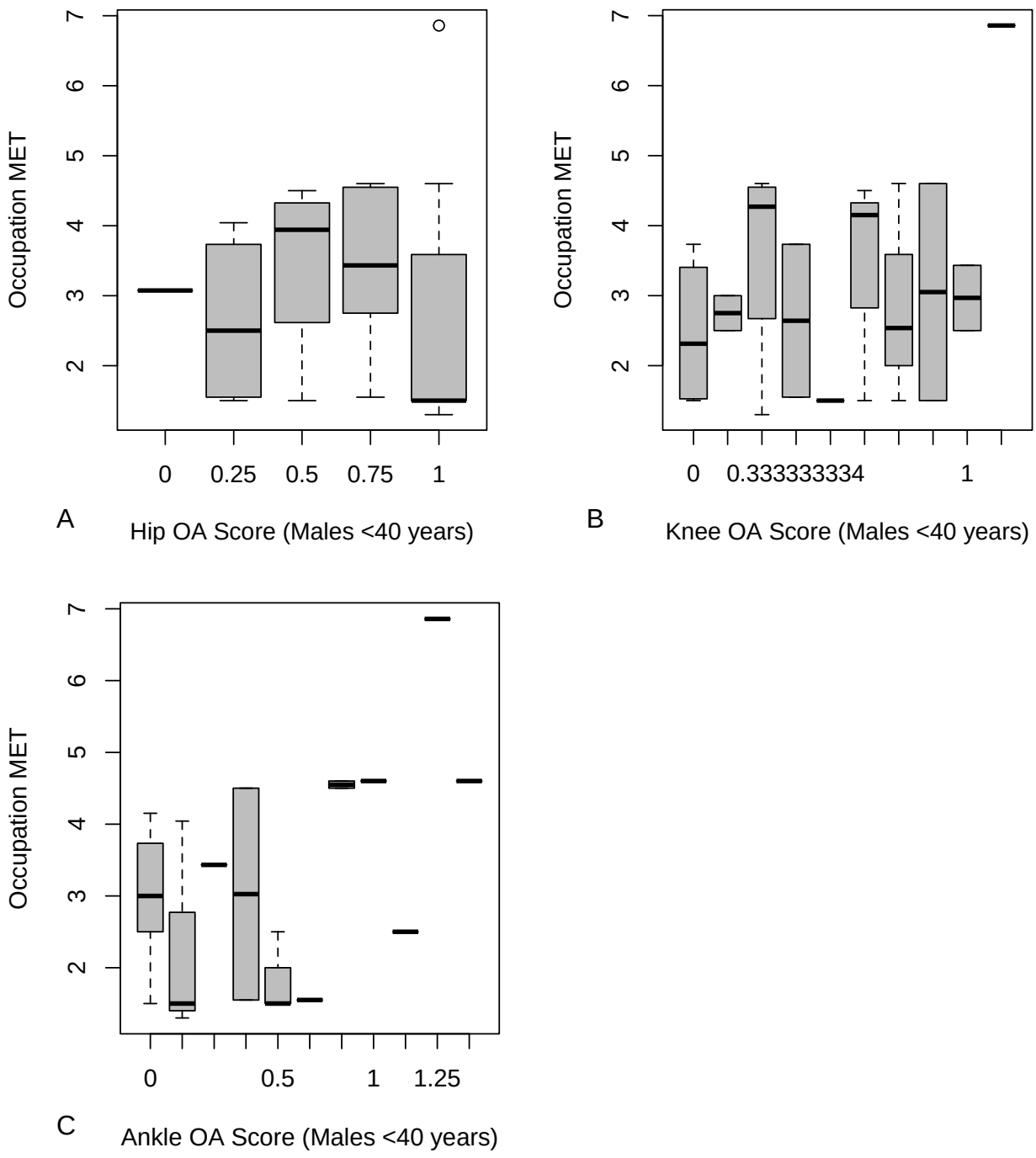


Figure 6-100. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for occupational activity MET value per OA score in males (<40 years). A) Hip OA, B) Knee OA, C) Ankle OA.

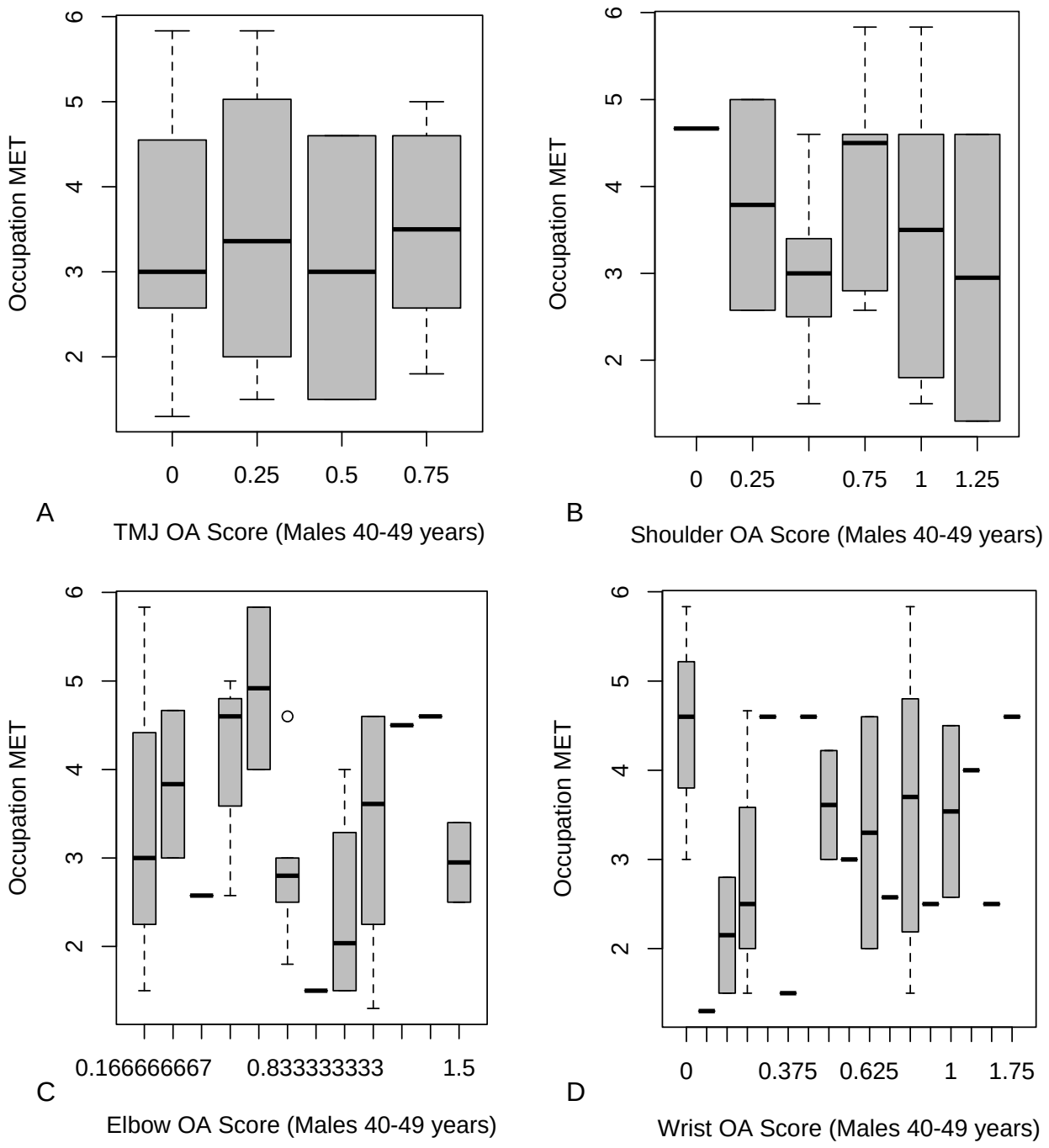


Figure 6-101. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for occupational activity MET value per OA score in males (40-49 years). A) TMJ OA, B) Shoulder OA, C) Elbow OA, D) Wrist OA.

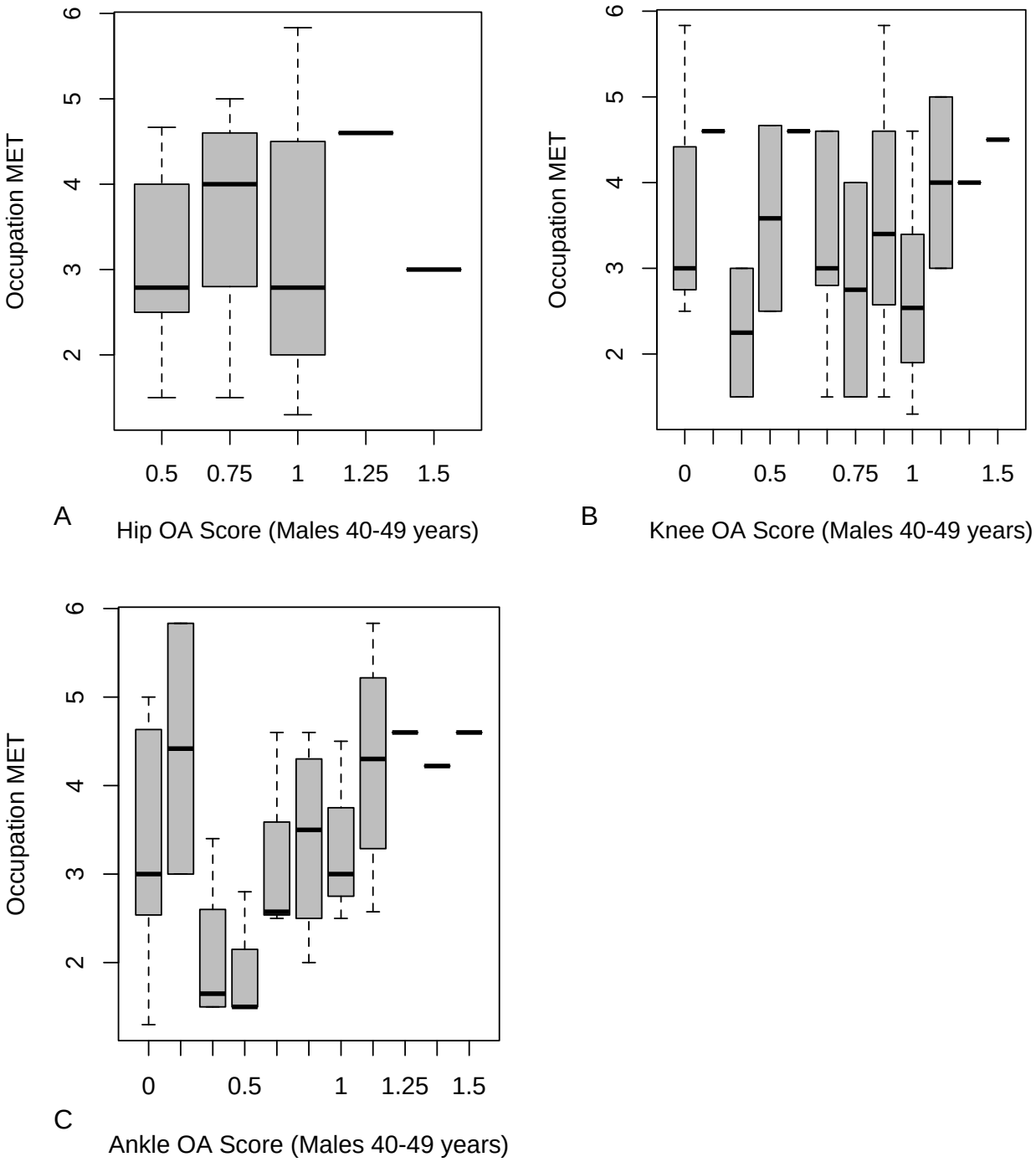


Figure 6-102. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for occupational activity MET value per OA score in males (40-49 years). A) Hip OA, B) Knee OA, C) Ankle OA.

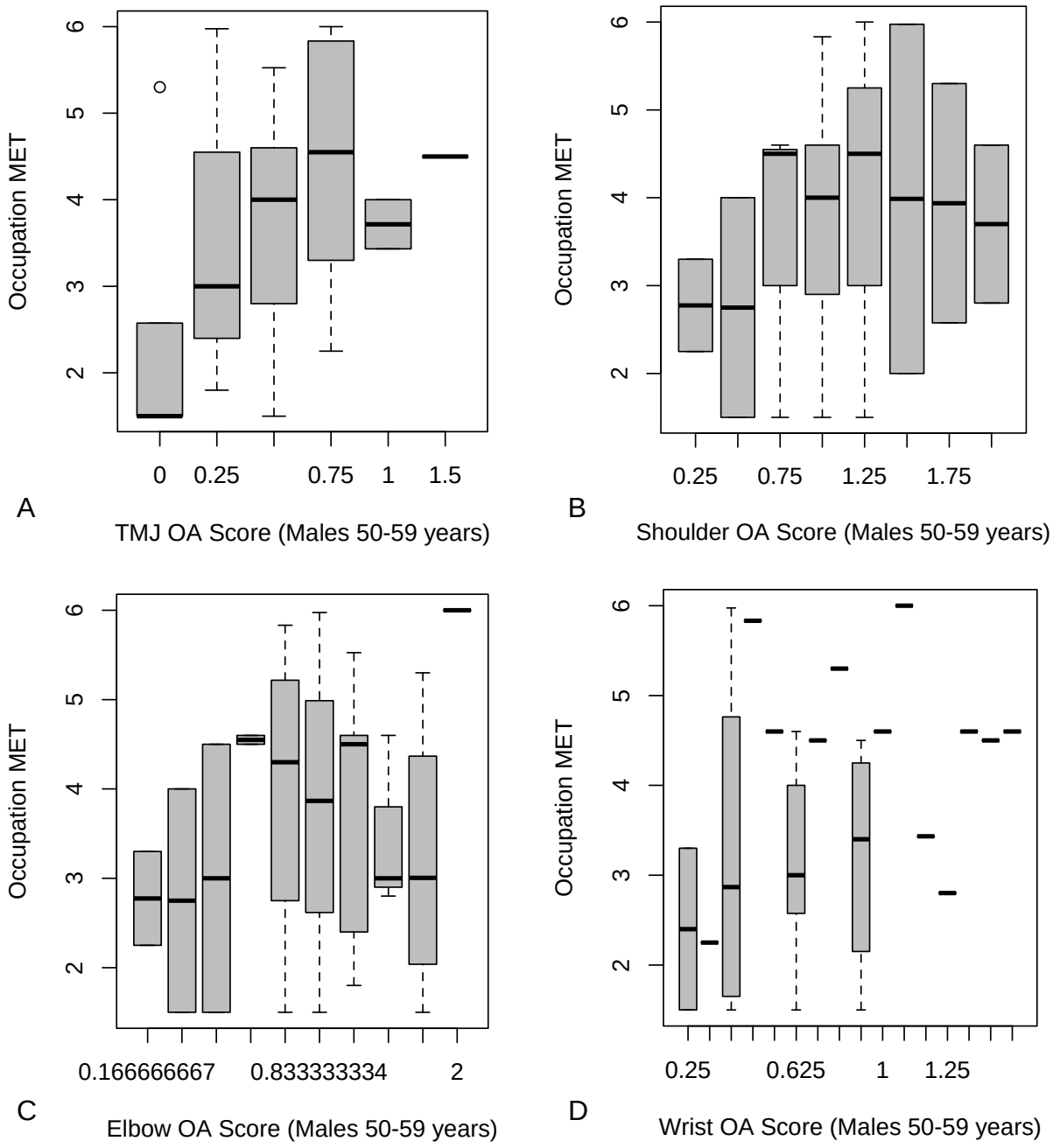


Figure 6-103. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for occupational activity MET value per OA score in males (50-59 years). A) TMJ OA, B) Shoulder OA, C) Elbow OA, D) Wrist OA.

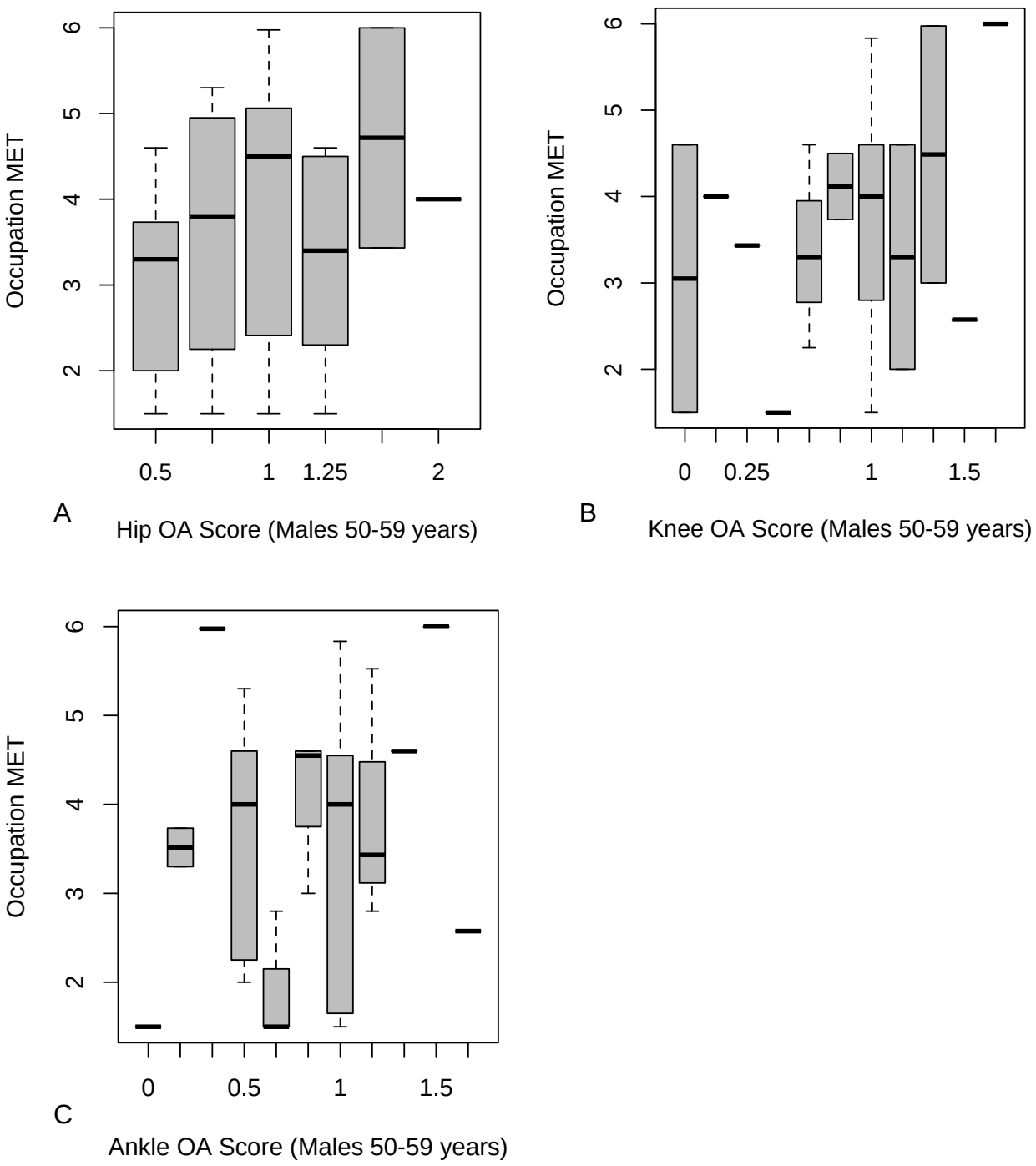


Figure 6-104. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for occupational activity MET value per OA score in males (50-59 years). A) Hip OA, B) Knee OA, C) Ankle OA.

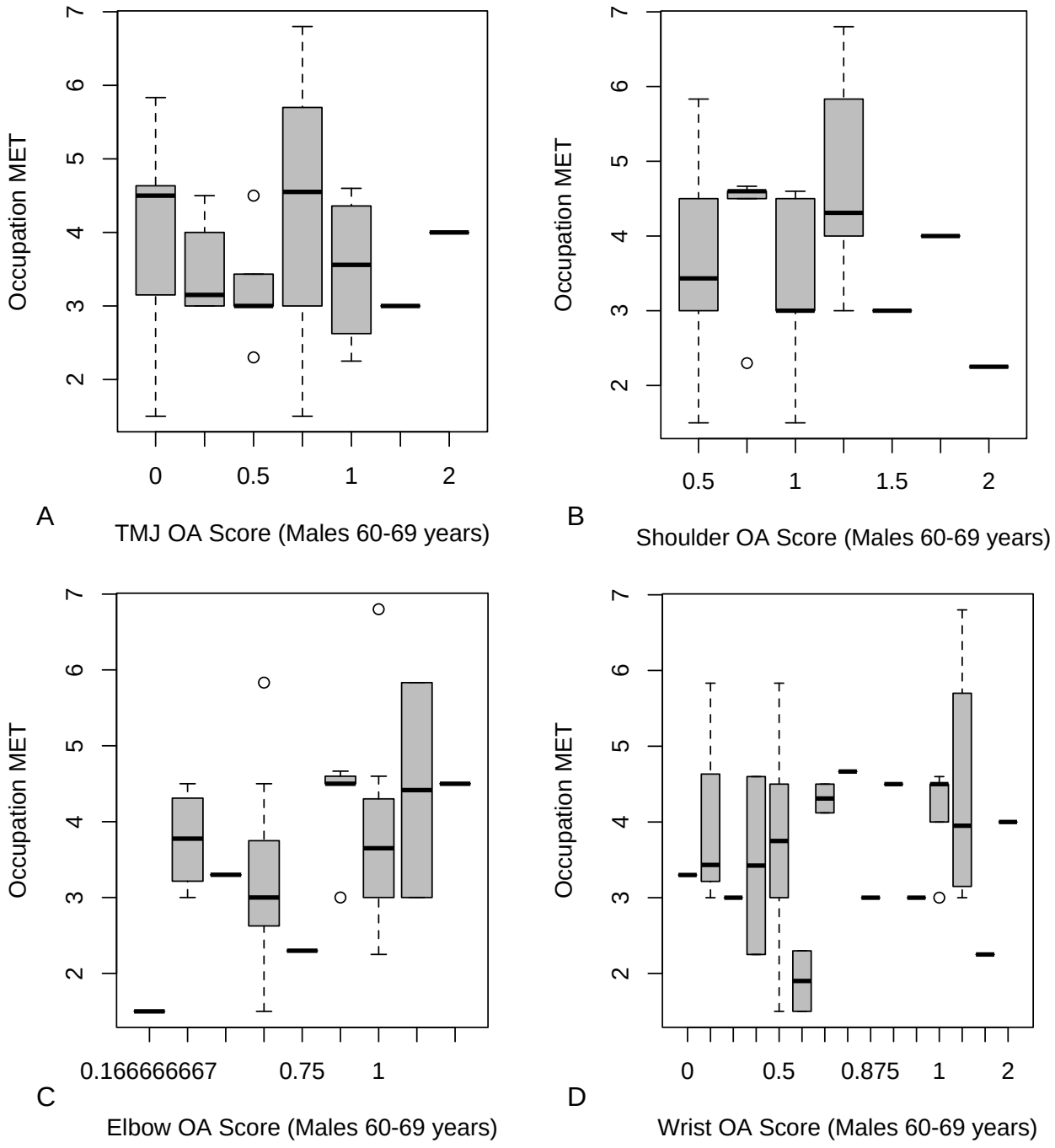


Figure 6-105. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for occupational activity MET value per OA score in males (60-69 years). A) TMJ OA, B) Shoulder OA, C) Elbow OA, D) Wrist OA.

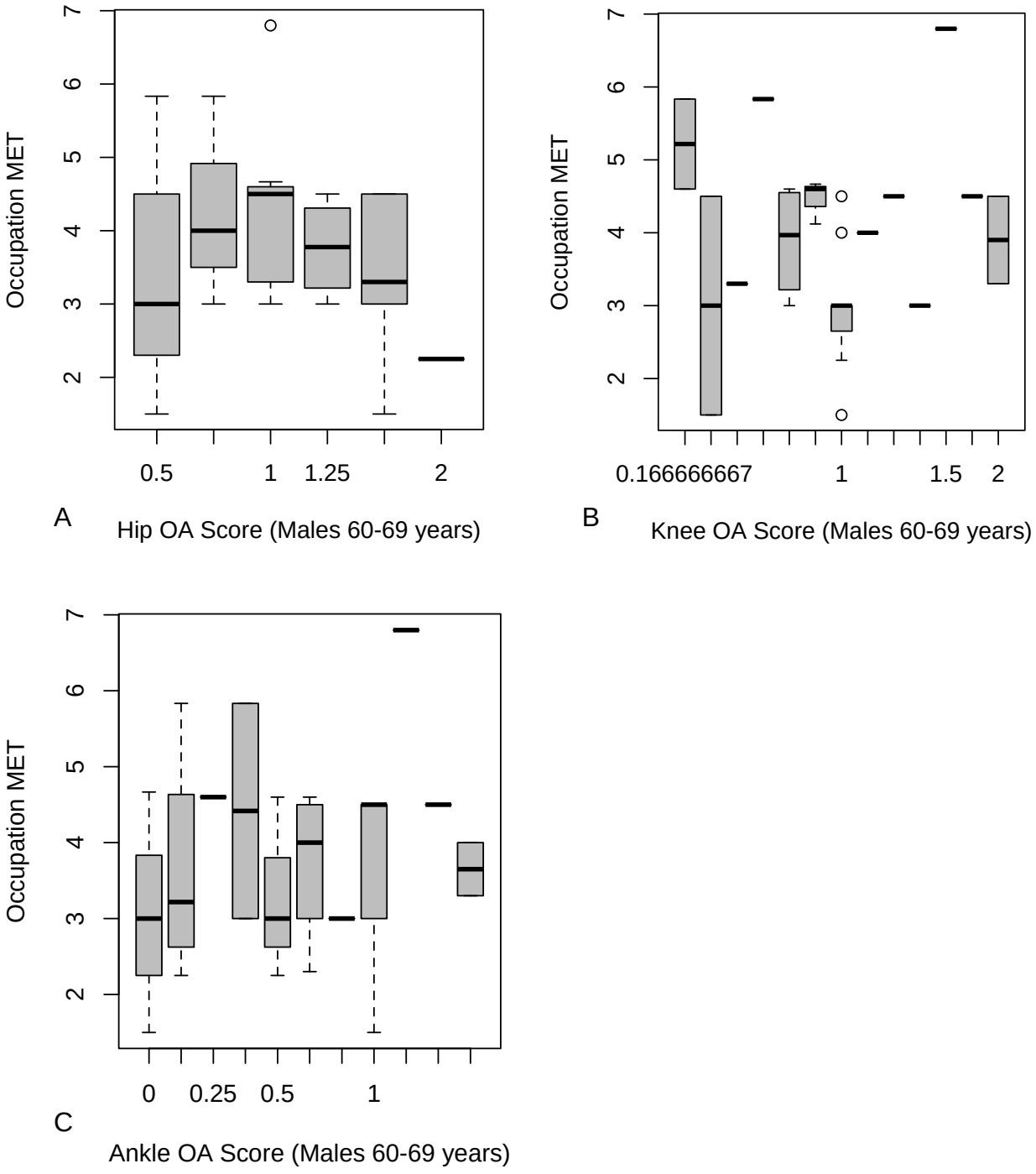


Figure 6-106. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for occupational activity MET value per OA score in males (60-69 years). A) Hip OA, B) Knee OA, C) Ankle OA.

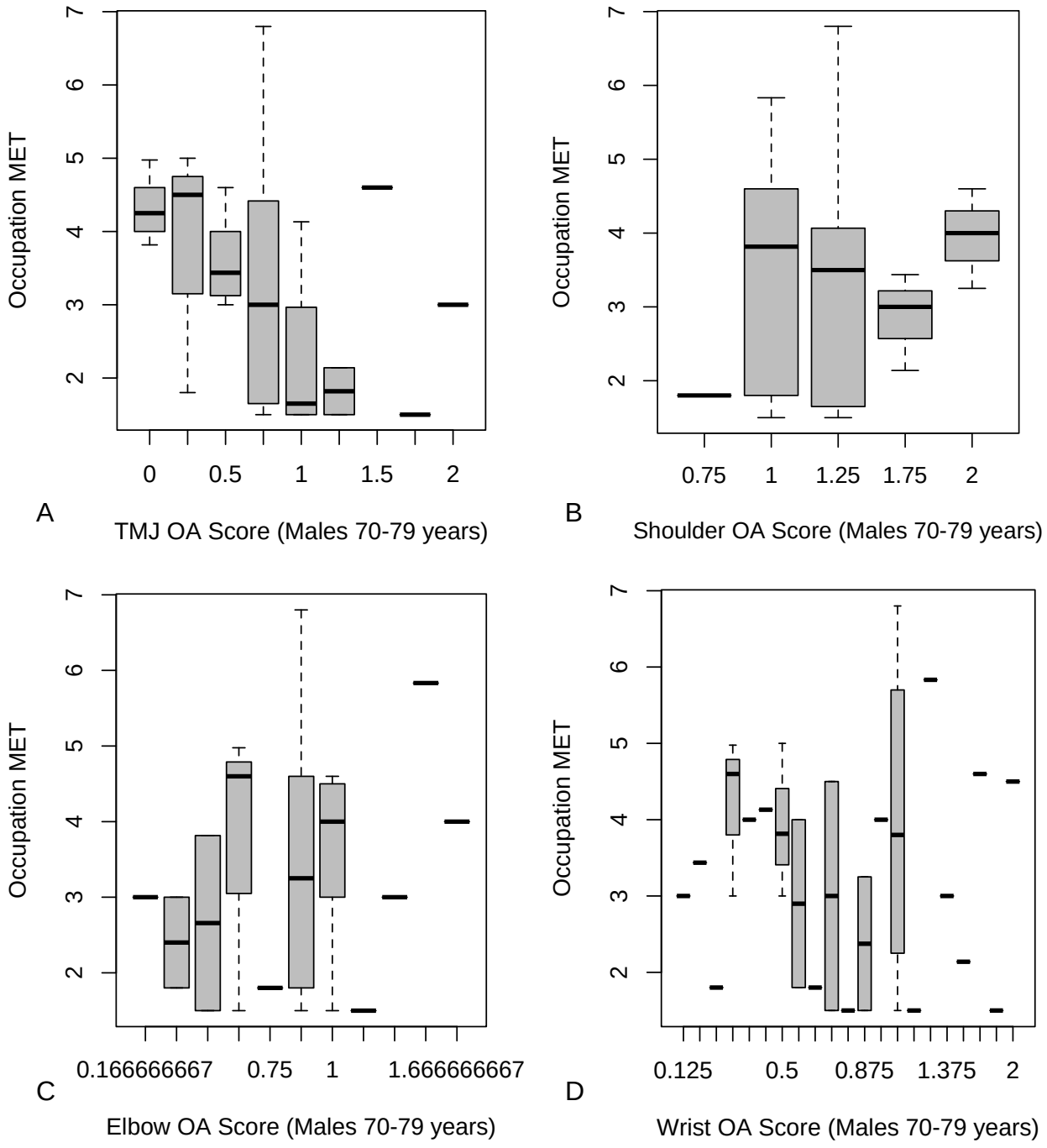


Figure 6-107. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for occupational activity MET value per OA score in males (70-79 years). A) TMJ OA, B) Shoulder OA, C) Elbow OA, D) Wrist OA.

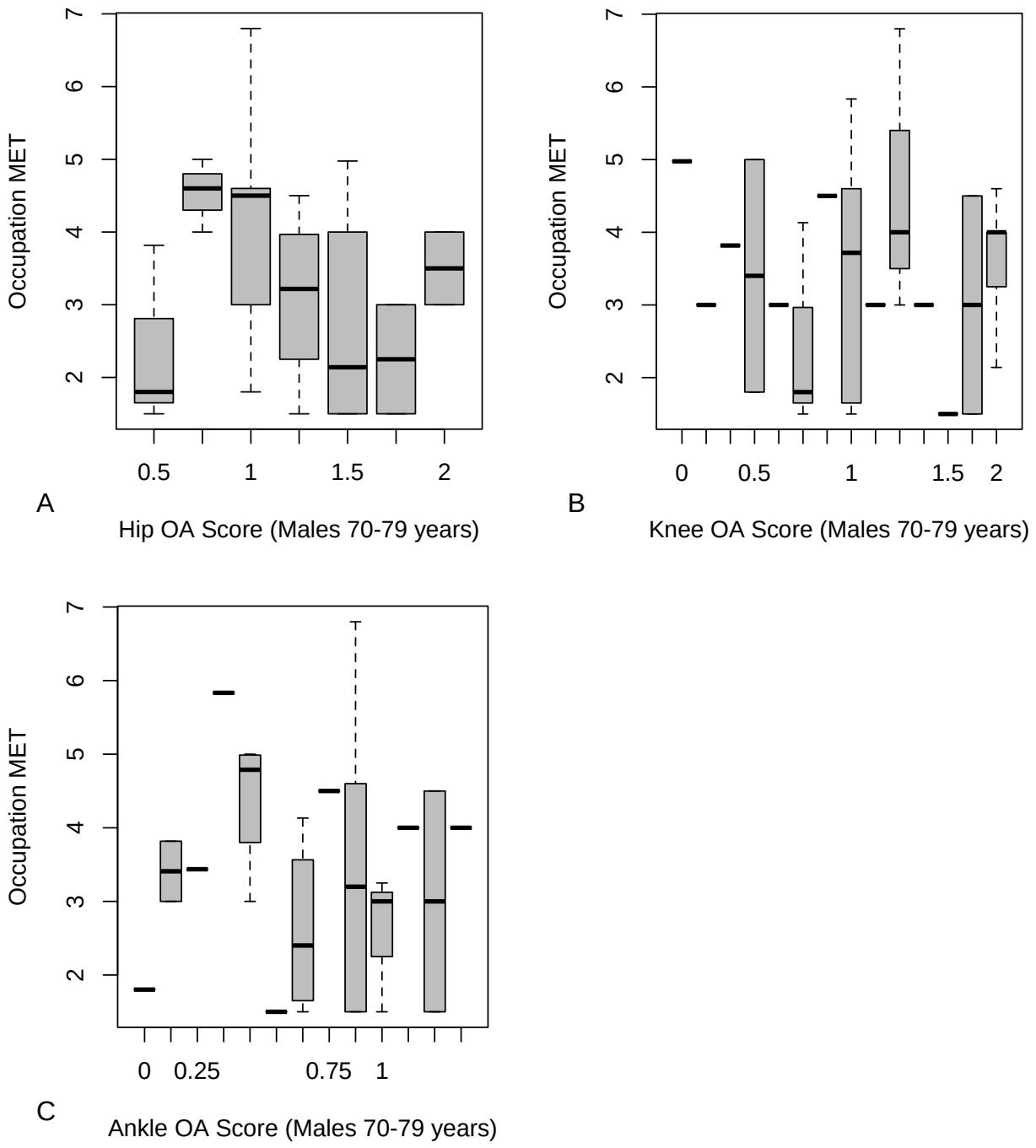


Figure 6-108. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for occupational activity MET value per OA score in males (70-79 years). A) Hip OA, B) Knee OA, C) Ankle OA.

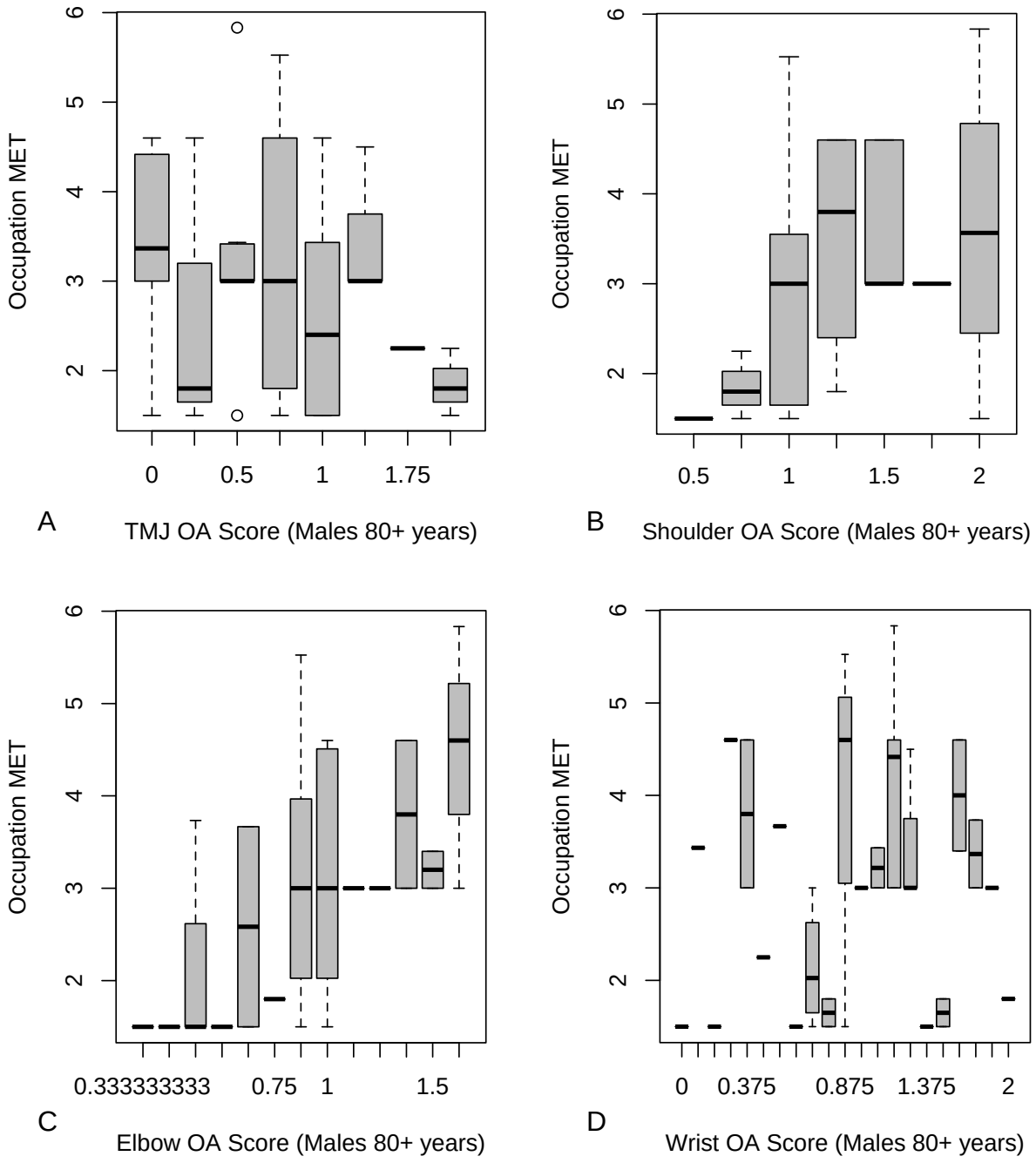


Figure 6-109. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for occupational activity MET value per OA score in males (80+ years). A) TMJ OA, B) Shoulder OA, C) Elbow OA, D) Wrist OA.

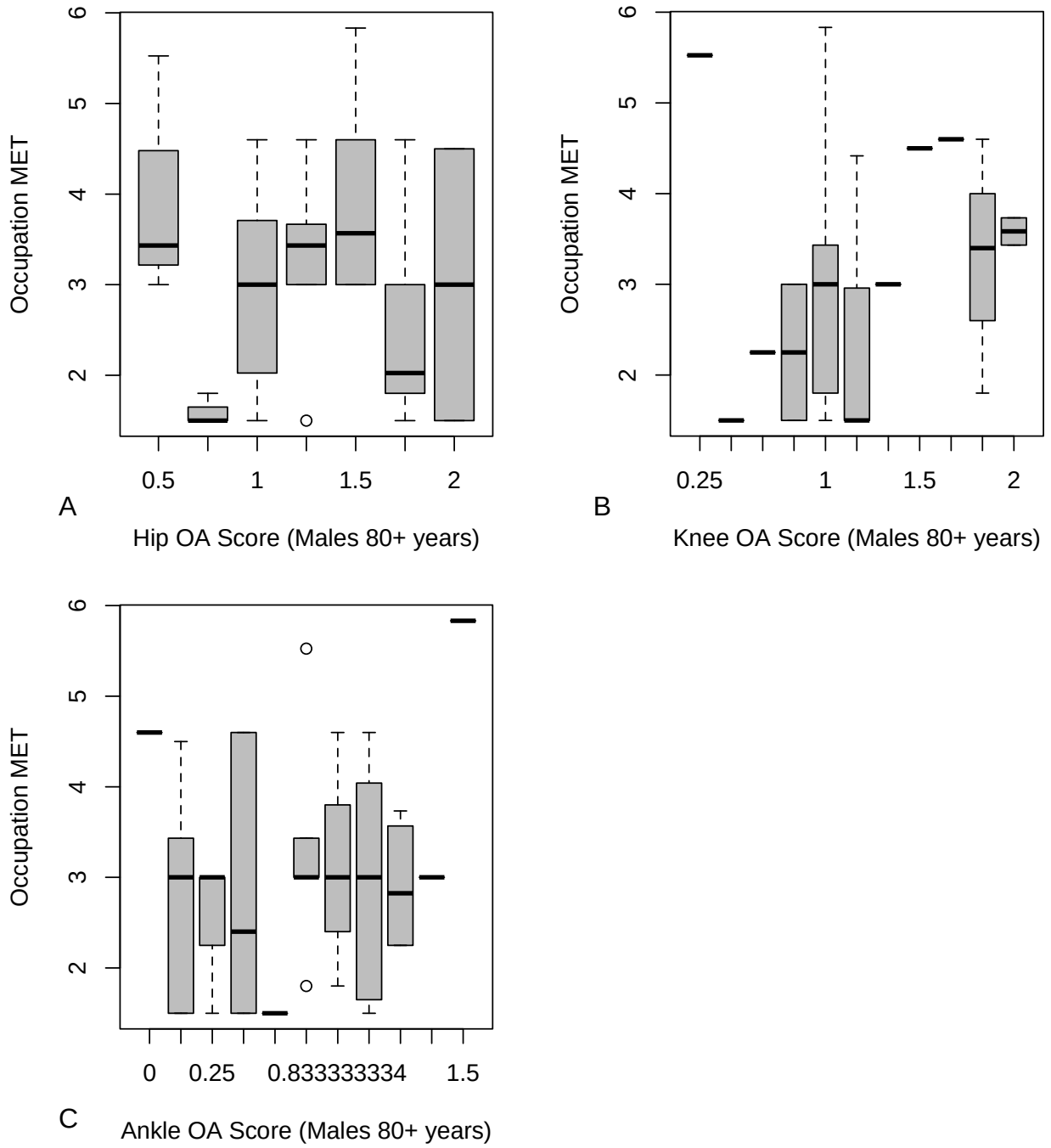


Figure 6-110. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for occupational activity MET value per OA score in males (80+ years). A) Hip OA, B) Knee OA, C) Ankle OA.

Table 6-26. Spearman's rank-order correlations for MET occupation values and scores for OA in the various joints in females and males.

Group	*rho* (TMJ)	*rho* (Shoulder)	*rho* (Elbow)	*rho* (Wrist)	*rho* (Hip)	*rho* (Knee)	*rho* (Ankle)
Females <50 years	0.140	0.139	0.140	0.178	0.283	0.085	0.058
Females 50-59 years	-0.203	0.460	0.192	0.236	0.147	0.524	0.337
Females 60-69 years	-0.153	0.219	0.120	0.210	0.042	0.422	0.099
Females 70-79 years	-0.167	-0.096	-0.174	-0.376	-0.301	-0.163	-0.244
Females 80+ years	-0.108	0.038	0.261	0.091	0.015	-0.031	0.196
Males <40 years	0.120	0.131	0.327	0.301	-0.046	0.170	0.338
Males 40-49 years	-0.021	-0.064	-0.114	0.052	0.134	-0.062	0.223
Males 50-59 years	0.335	0.215	0.145	0.330	0.102	0.223	0.149
Males 60-69 years	-0.156	-0.072	0.267	0.099	-0.021	-0.187	0.105
Males 70-79 years	-0.489	0.040	0.184	-0.051	-0.294	-0.069	-0.074
Males 80+ years	-0.204	0.403	0.478	0.122	0.085	0.332	0.118

*Statistically significant at α=0.001 (Bonferroni correction for females: α=0.05/35; for males: α=0.05/42).

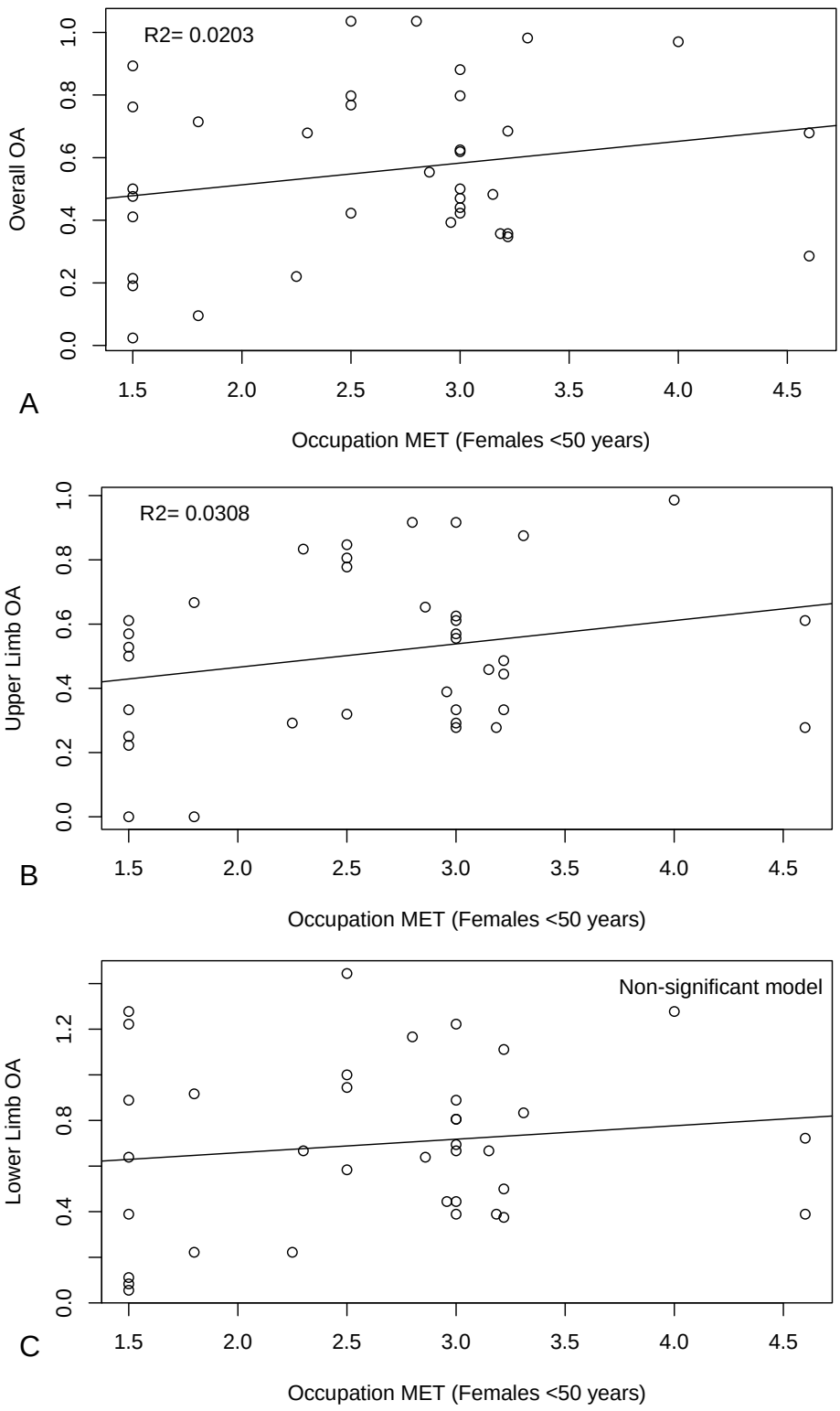


Figure 6-111. Hypothesis 3b: OA correlates positively with activity; scatterplots for summary OA and MET occupation values in females <50 years. A) Overall OA, B) Upper limb OA, C) Lower limb OA. Black line indicates linear regression model.

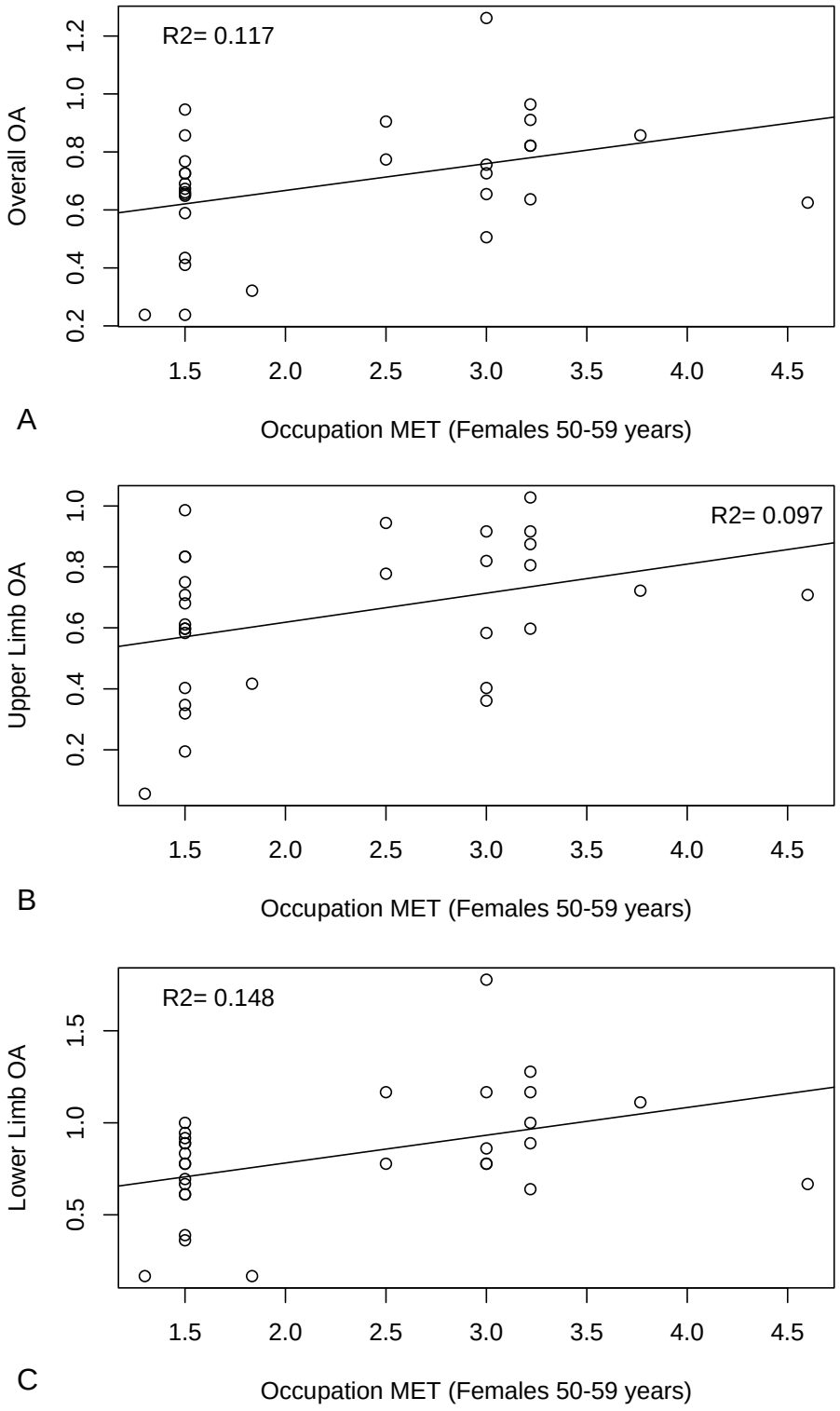


Figure 6-112. Hypothesis 3b: OA correlates positively with activity; scatterplots for summary OA and MET occupation values in females 50-59 years. A) Overall OA, B) Upper limb OA, C) Lower limb OA. Black line indicates linear regression model.

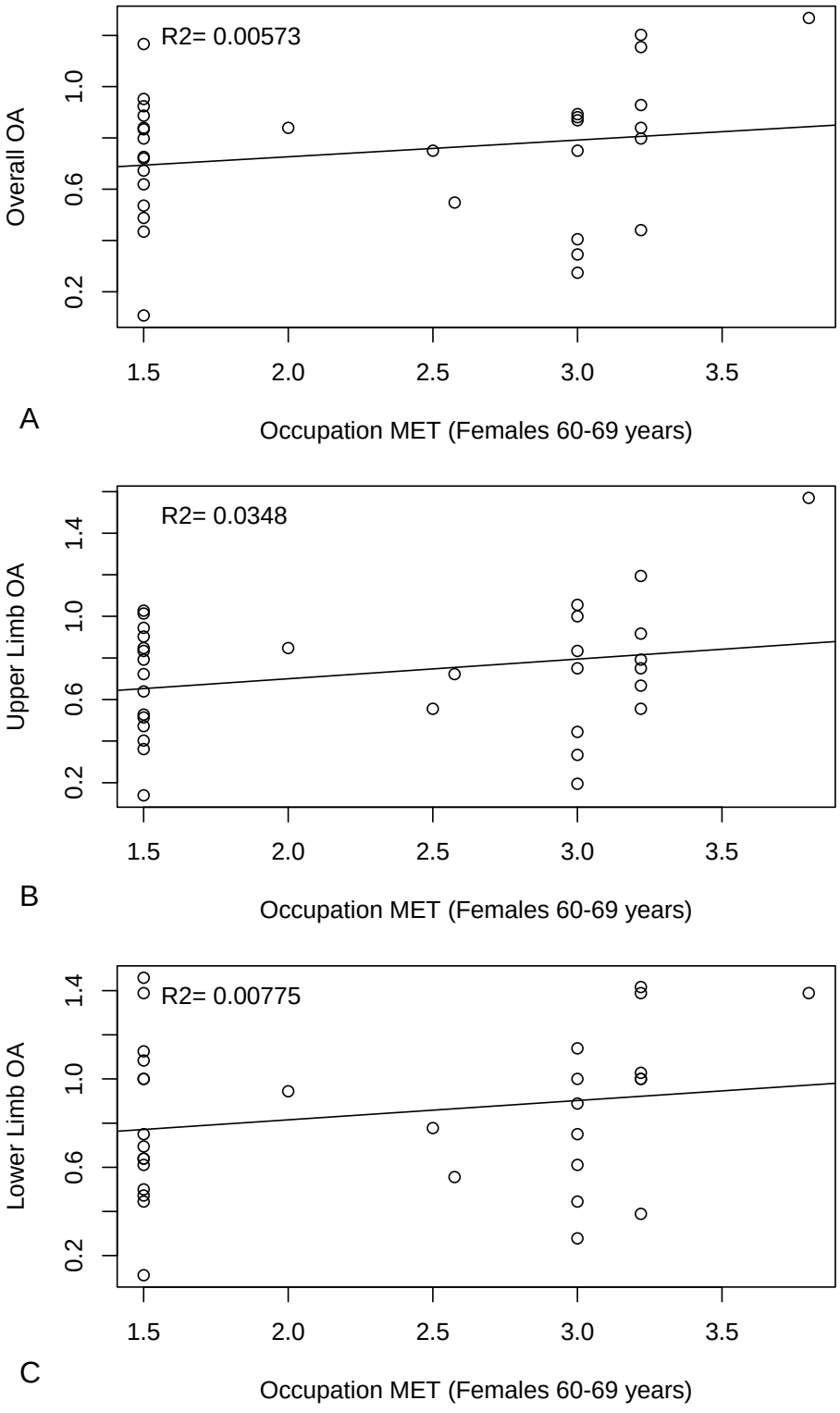


Figure 6-113. Hypothesis 3b: OA correlates positively with activity; scatterplots for summary OA and MET occupation values in females 60-69 years. A) Overall OA, B) Upper limb OA, C) Lower limb OA. Black line indicates linear regression model.

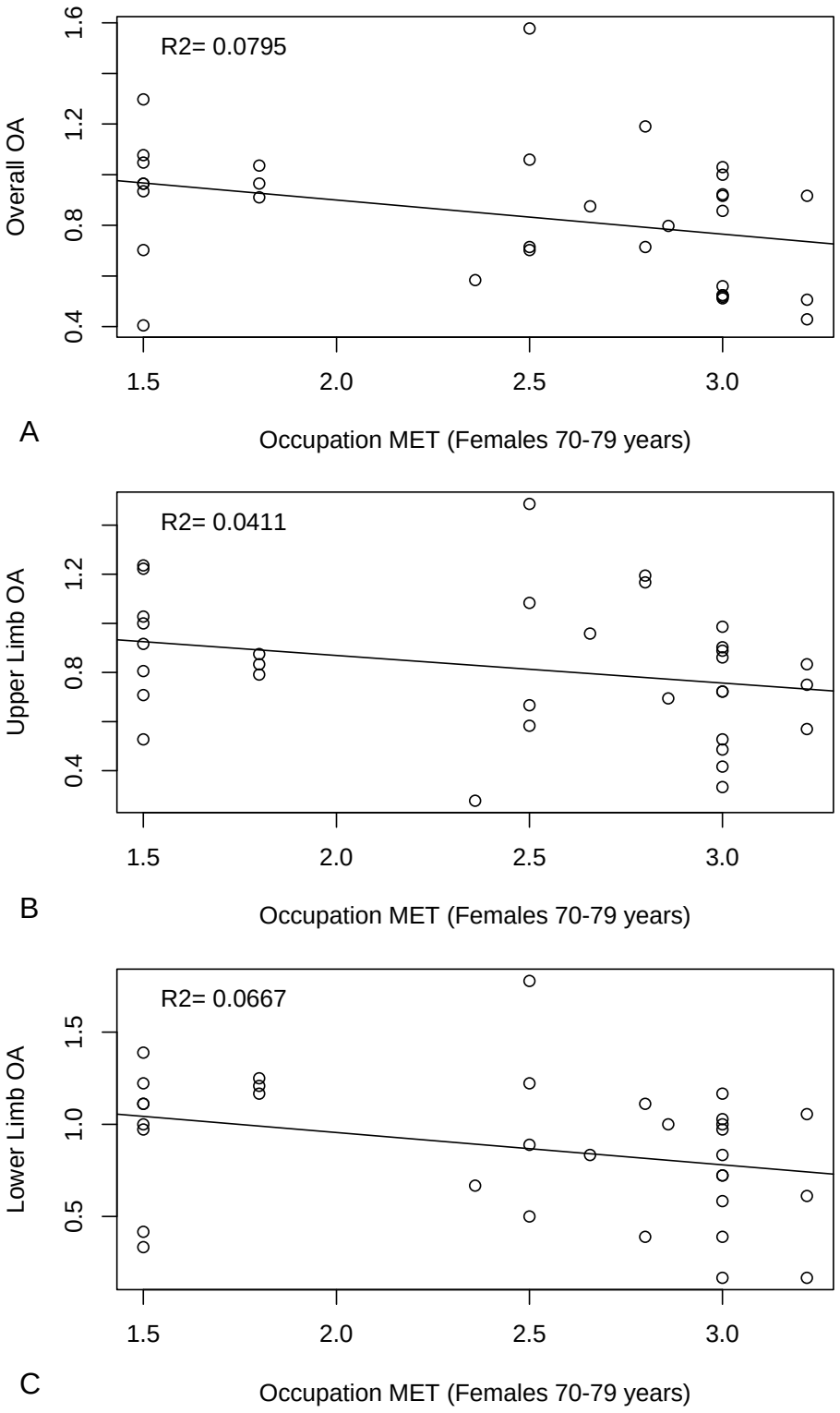


Figure 6-114. Hypothesis 3b: OA correlates positively with activity; scatterplots for summary OA and MET occupation values in females 70-79 years. A) Overall OA, B) Upper limb OA, C) Lower limb OA. Black line indicates linear regression model.

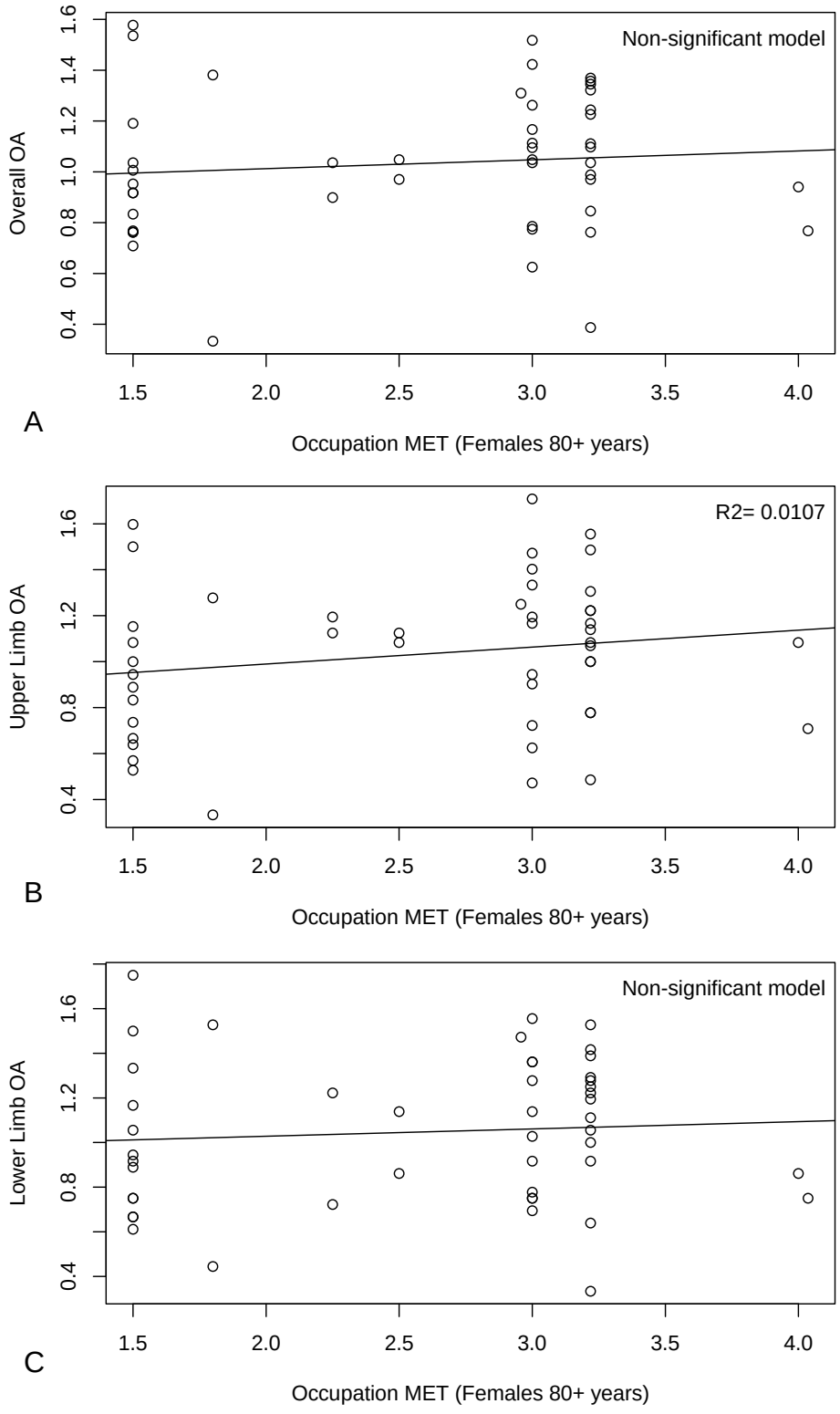


Figure 6-115. Hypothesis 3b: OA correlates positively with activity; scatterplots for summary OA and MET occupation values in females 80+. A) Overall OA, B) Upper limb OA, C) Lower limb OA. Black line indicates linear regression model.

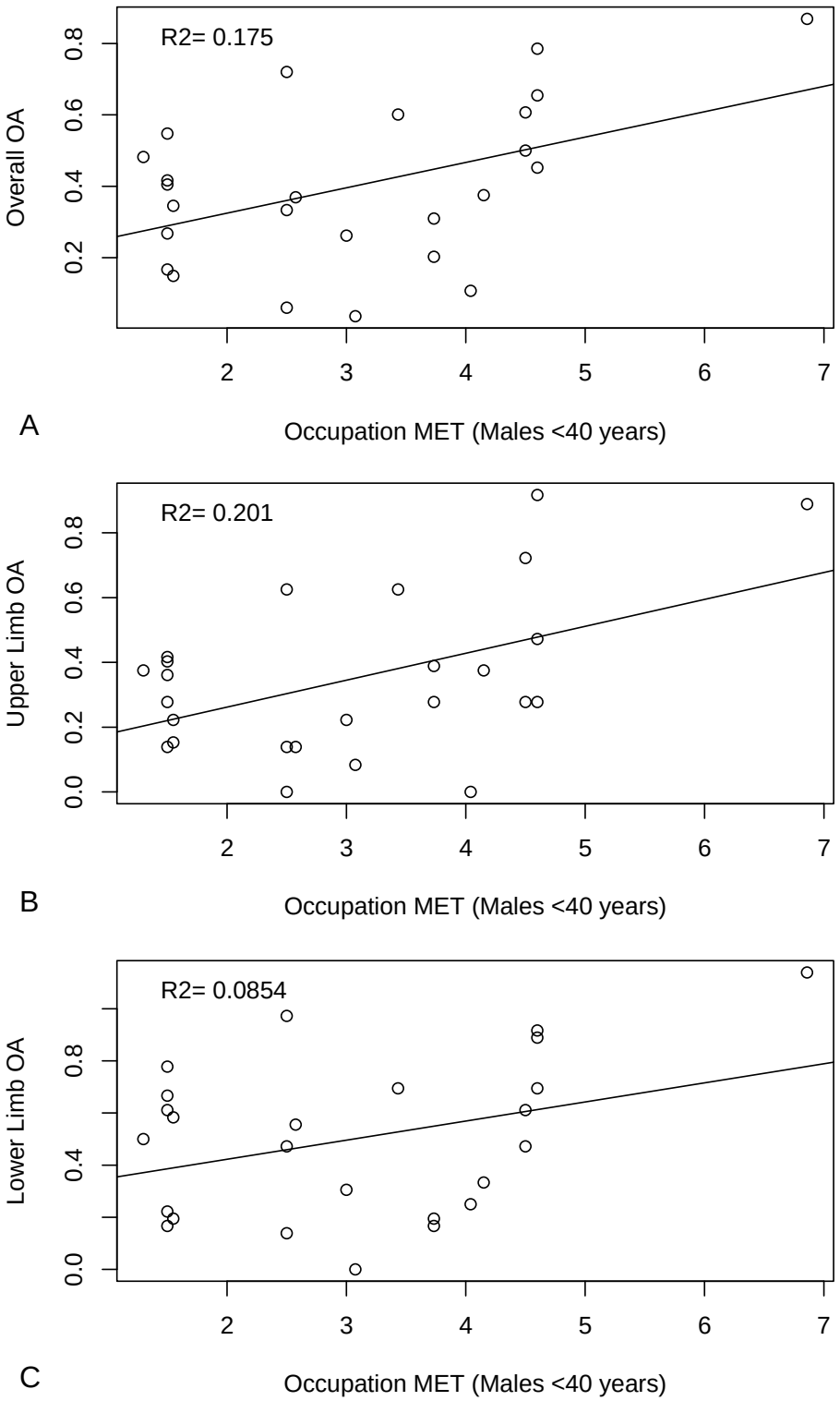


Figure 6-116. Hypothesis 3b: OA correlates positively with activity; scatterplots for summary OA and MET occupation values in males <40 years. A) Overall OA, B) Upper limb OA, C) Lower limb OA. Black line indicates linear regression model.

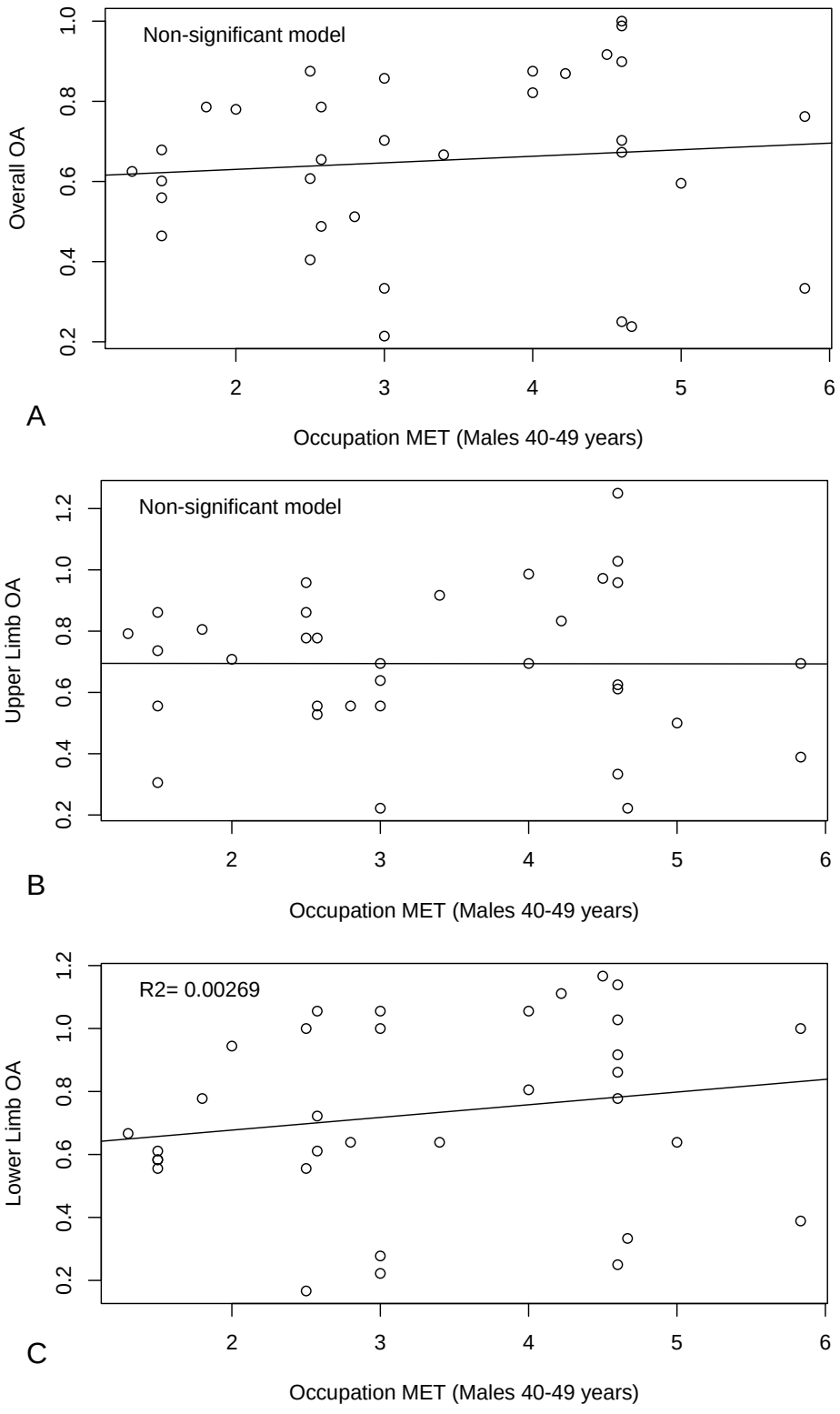


Figure 6-117. Hypothesis 3b: OA correlates positively with activity; scatterplots for summary OA and MET occupation values in males 40-49 years. A) Overall OA, B) Upper limb OA, C) Lower limb OA. Black line indicates linear regression model.

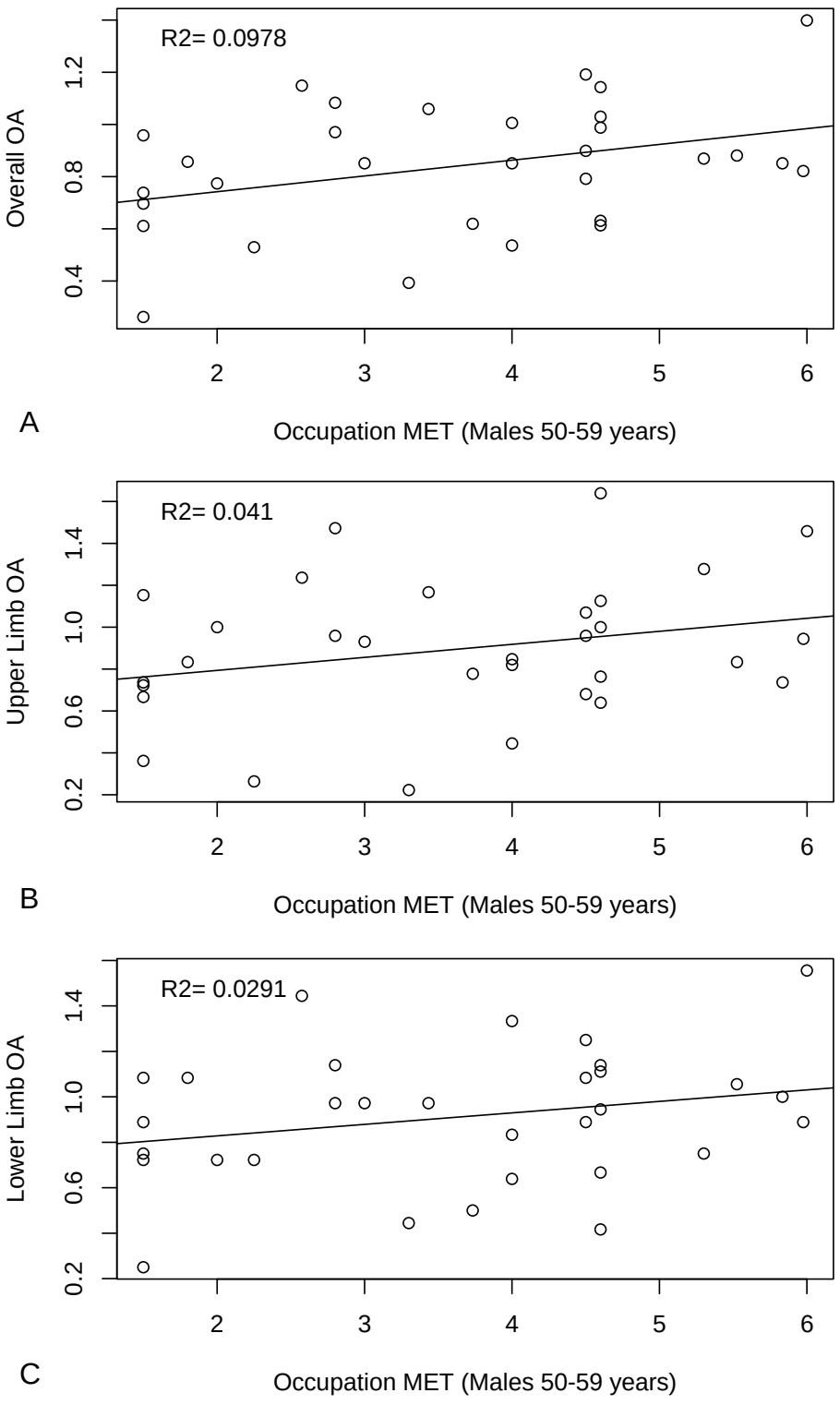


Figure 6-118. Hypothesis 3b: OA correlates positively with activity; scatterplots for summary OA and MET occupation values in males 50-59 years. A) Overall OA, B) Upper limb OA, C) Lower limb OA. Black line indicates linear regression model.

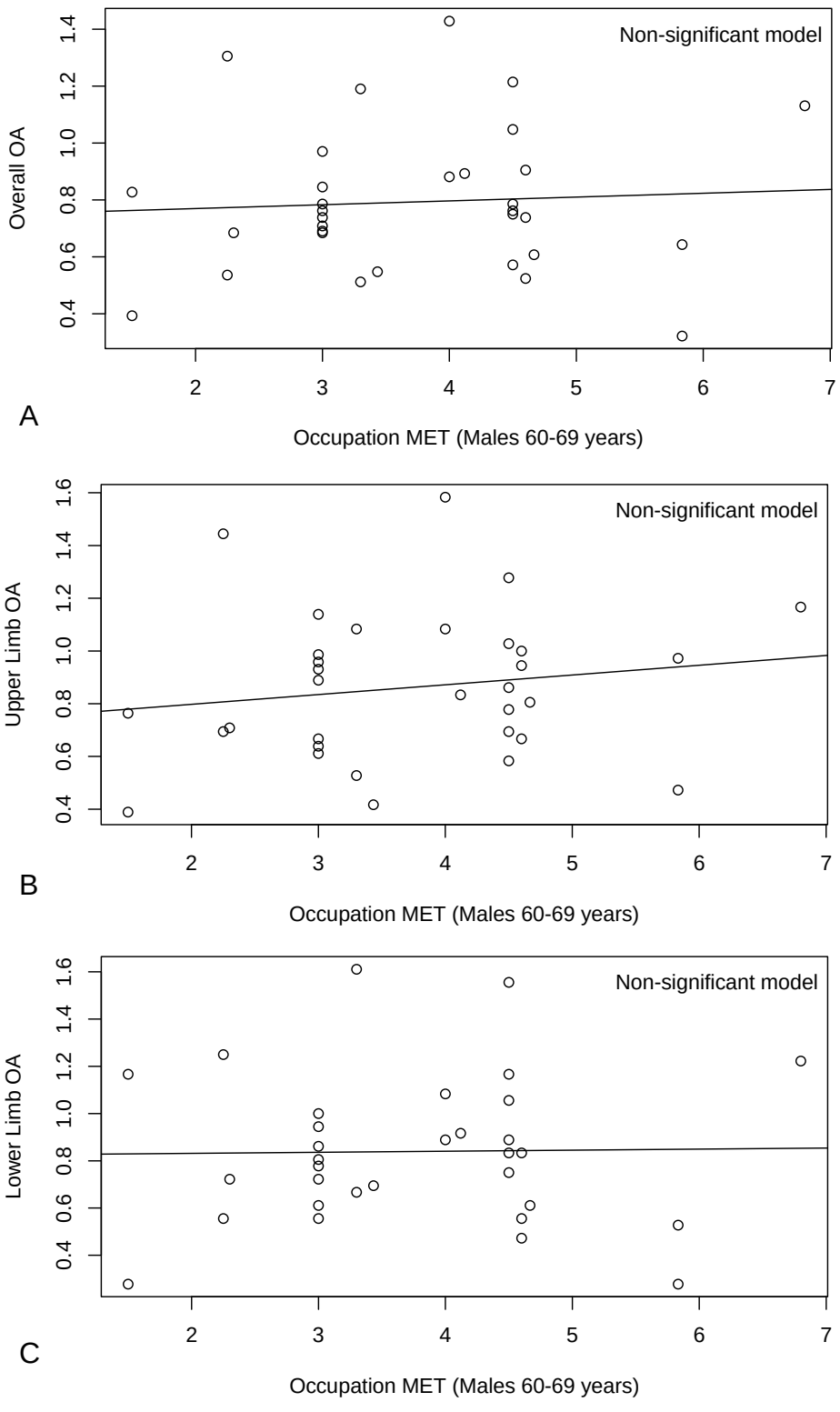


Figure 6-119. Hypothesis 3b: OA correlates positively with activity; scatterplots for summary OA and MET occupation values in males 60-69 years. A) Overall OA, B) Upper limb OA, C) Lower limb OA. Black line indicates linear regression model.

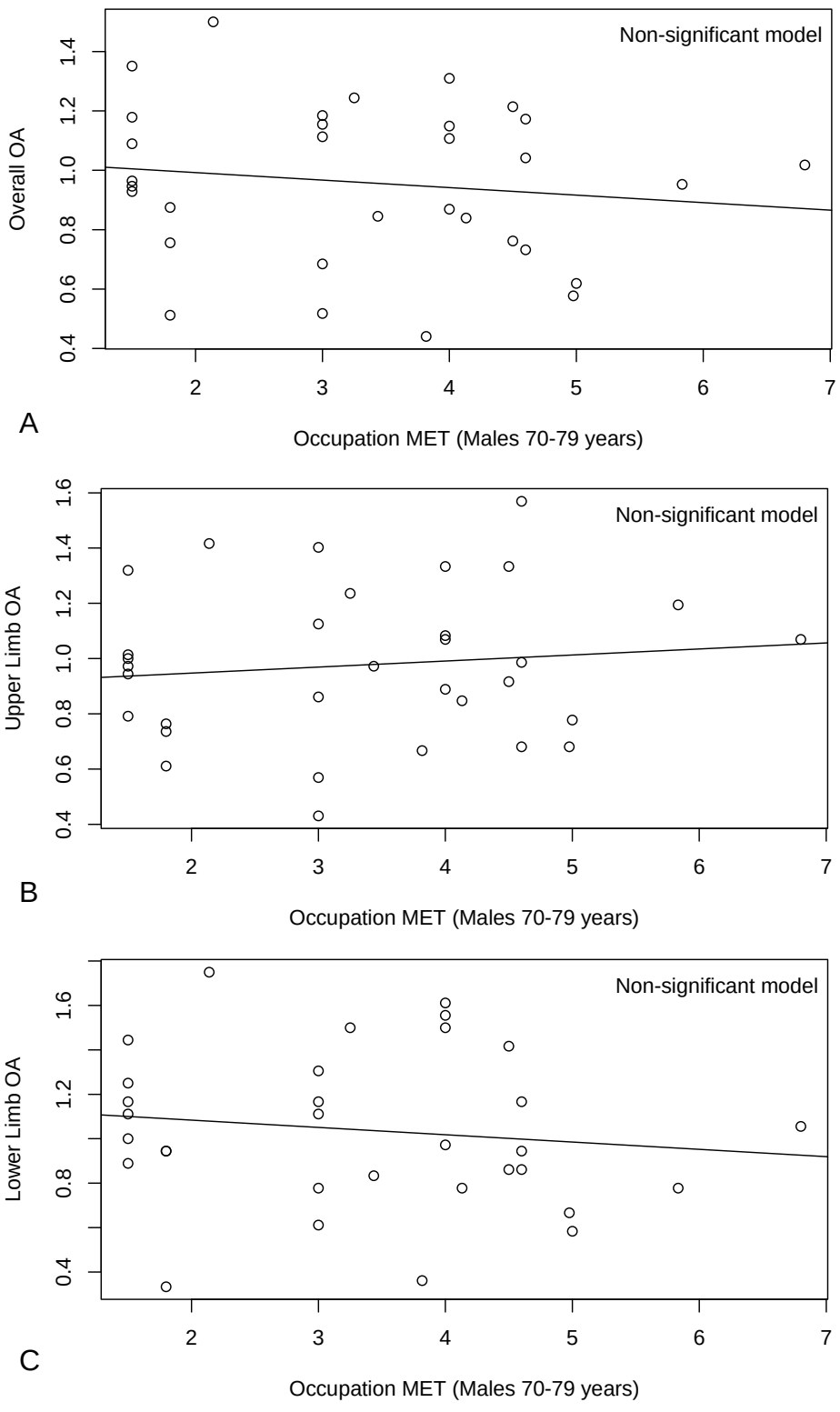


Figure 6-120. Hypothesis 3b: OA correlates positively with activity; scatterplots for summary OA and MET occupation values in males 70-79 years. A) Overall OA, B) Upper limb OA, C) Lower limb OA. Black line indicates linear regression model.

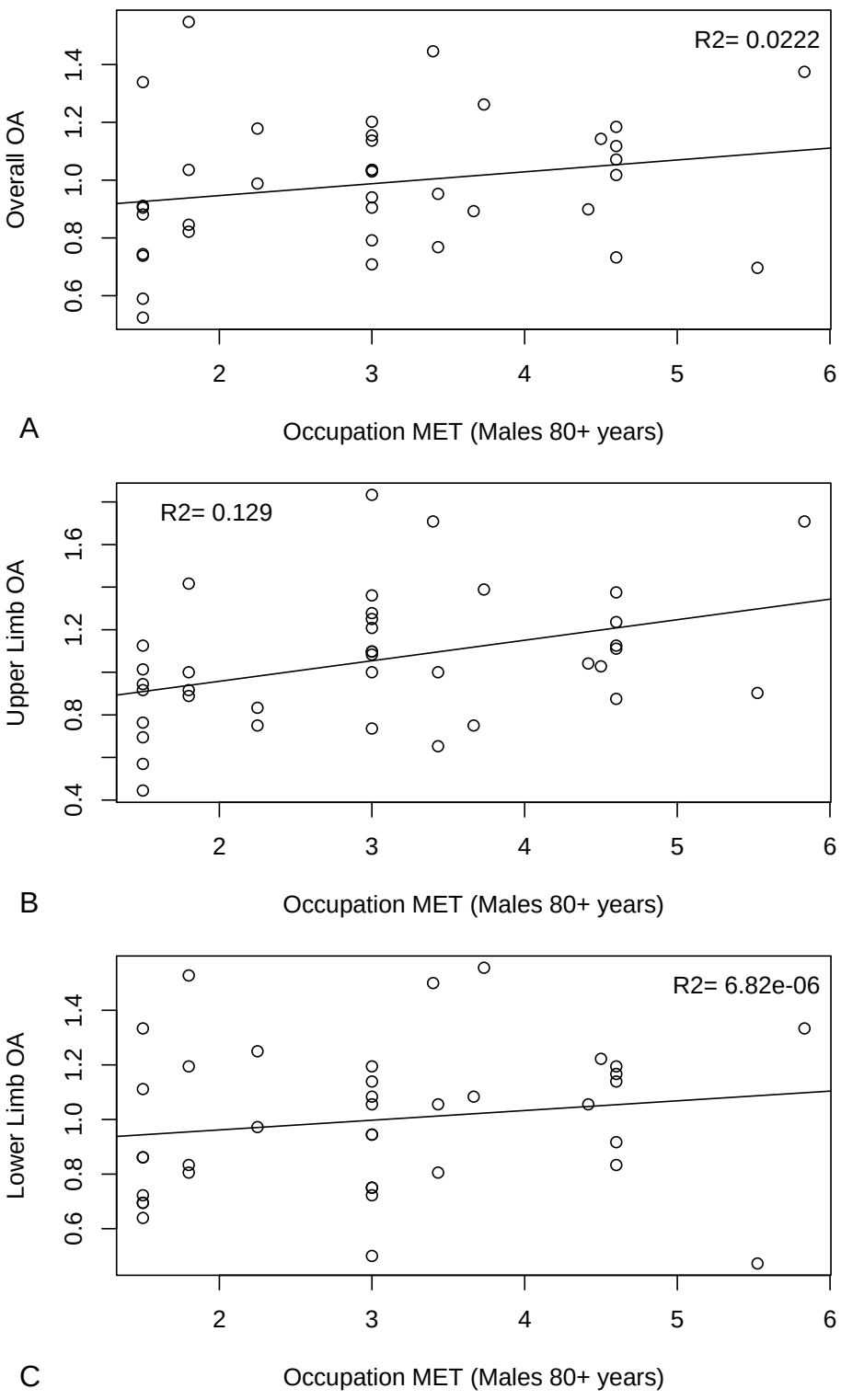


Figure 6-121. Hypothesis 3b: OA correlates positively with activity; scatterplots for summary OA and MET occupation values in males 80+. A) Overall OA, B) Upper limb OA, C) Lower limb OA. Black line indicates linear regression model.

Table 6-27. Spearman's rank-order correlations for summary OA and MET values for occupation in females and males.

Group	*rho*
Females <50 years overall OA	0.152
Females <50 years upper limb OA	0.164
Females <50 years lower limb OA	0.113
Females 50-59 years overall OA	0.388
Females 50-59 years upper limb OA	0.395
Females 50-59 years lower limb OA	0.424
Females 60-69 years overall OA	0.244
Females 60-69 years upper limb OA	0.198
Females 60-69 years lower limb OA	0.218
Females 70-79 years overall OA	-0.419
Females 70-79 years upper limb OA	-0.322
Females 70-79 years lower limb OA	-0.365
Females 80+ years overall OA	0.137
Females 80+ years upper limb OA	0.153
Females 80+ years lower limb OA	0.114
Males <40 years overall OA	0.346
Males <40 years upper limb OA	0.303
Males <40 years lower limb OA	0.261
Males 40-49 years overall OA	0.132
Males 40-49 years upper limb OA	-0.092
Males 40-49 years lower limb OA	0.215
Males 50-59 years overall OA	0.321
Males 50-59 years upper limb OA	0.261
Males 50-59 years lower limb OA	0.212
Males 60-69 years overall OA	0.001
Males 60-69 years upper limb OA	0.142
Males 60-69 years lower limb OA	-0.066
Males 70-79 years overall OA	-0.158
Males 70-79 years upper limb OA	0.086
Males 70-79 years lower limb OA	-0.215
Males 80+ years overall OA	0.256
Males 80+ years upper limb OA	0.385
Males 80+ years lower limb OA	0.253

*Statistically significant at α=0.003 (Bonferroni correction: α=0.05/15).

Table 6-28. Regression results for summary OA and MET values for occupation in females.

Variable	Estimate	Std. Error	t value	Probability
Intercept	0.374	0.147	2.546	0.016
Females <50 years overall OA	0.070	0.053	1.314	0.120
Intercept	0.320	0.139	2.309	0.027
Females <50 years upper limb OA	0.073	0.050	1.454	0.155
Intercept	0.541	0.206	2.629	0.013
Females <50 years lower limb OA	0.059	0.074	0.794	0.433
Intercept	0.482	0.104	4.656	7.1e-05*
Females 50-59 years overall OA	0.093	0.042	2.199	0.036
Intercept	0.427	0.116	3.694	9.493-04*
Females 50-59 years upper limb OA	0.096	0.047	2.029	0.052
Intercept	0.480	0.151	3.186	0.004
Females 50-59 years lower limb OA	0.151	0.061	2.460	0.020
Intercept	0.596	0.147	4.066	3.18e-04*
Females 60-69 years overall OA	0.065	0.060	1.086	0.286
Intercept	0.511	0.158	3.229	0.003
Females 60-69 years upper limb OA	0.095	0.065	1.455	0.156
Intercept	0.640	0.191	3.347	0.002*
Females 60-69 years lower limb OA	0.087	0.078	1.115	0.274
Intercept	1.169	0.175	6.675	1.83e-07*
Females 70-79 years overall OA	-0.135	0.069	-1.940	0.062
Intercept	1.093	0.184	5.955	1.4e-06*
Females 70-79 years upper limb OA	-0.112	0.073	-1.540	0.134
Intercept	1.306	0.244	5.356	7.74e-06*
Females 70-79 years lower limb OA	-0.175	0.097	-1.813	0.080
Intercept	0.943	0.141	6.672	3.12e-08*
Females 80+ years overall OA	0.035	0.052	0.667	0.508
Intercept	0.842	0.163	5.162	5.36e-06*
Females 80+ years upper limb OA	0.074	0.060	1.224	0.227
Intercept	0.963	0.166	5.797	6.24e-07*
Females 80+ years lower limb OA	0.033	0.062	0.532	0.597

*Statistically significant at α=0.003 (Bonferroni correction: α=0.05/15).

Table 6-29. Regression results for summary OA and MET values for occupation in males.

Variable	Estimate	Std. Error	t value	Probability
Intercept	0.183	0.098	1.873	0.074
Males <40 years overall OA	0.071	0.029	2.467	0.022
Intercept	0.096	0.106	0.907	0.374
Males <40 years upper limb OA	0.083	0.031	2.653	0.014
Intercept	0.276	0.138	2.007	0.057
Males <40 years lower limb OA	0.073	0.041	1.801	0.085
Intercept	0.598	0.106	5.623	3.6e-06*
Males 40-49 years overall OA	0.016	0.030	0.550	0.586
Intercept	0.695	0.117	5.920	1.54e-06*
Males 40-49 years upper limb OA	-2.88e-04	0.033	-0.009	0.993
Intercept	0.597	0.139	4.311	1.53e-04*
Males 40-49 years lower limb OA	0.040	0.039	1.042	0.305
Intercept	0.621	0.114	5.442	7.44e-06*
Males 50-59 years overall OA	0.061	0.029	2.062	0.048*
Intercept	0.670	0.160	4.179	2.45e-04*
Males 50-59 years upper limb OA	0.062	0.041	1.511	0.142
Intercept	0.727	0.143	5.089	1.98e-05*
Males 50-59 years lower limb OA	0.051	0.037	1.378	0.179
Intercept	0.743	0.151	4.909	3.01e-05*
Males 60-69 years overall OA	0.014	0.039	0.350	0.729
Intercept	0.724	0.163	4.442	1.12e-04*
Males 60-69 years upper limb OA	0.037	0.041	0.893	0.379
Intercept	0.823	0.186	4.436	1.14e-04*
Males 60-69 years lower limb OA	0.004	0.047	0.093	0.926
Intercept	1.043	0.123	8.509	1.7e-09*
Males 70-79 years overall OA	-0.025	0.034	-0.752	0.458
Intercept	0.904	0.128	7.078	7.19e-08*
Males 70-79 years upper limb OA	0.022	0.035	0.624	0.537
Intercept	1.150	0.163	7.052	7.7e-08*
Males 70-79 years lower limb OA	-0.033	0.045	-0.735	0.468
Intercept	0.864	0.099	8.744	1.97e-10*
Males 80+ years overall OA	0.041	0.030	1.357	0.183
Intercept	0.765	0.124	6.198	3.79e-07*
Males 80+ years upper limb OA	0.096	0.038	2.541	0.012
Intercept	0.891	0.116	7.702	4.03e-09*
Males 80+ years lower limb OA	0.036	0.036	1.000	0.324

*Statistically significant at α=0.003 (Bonferroni correction: α=0.05/18).

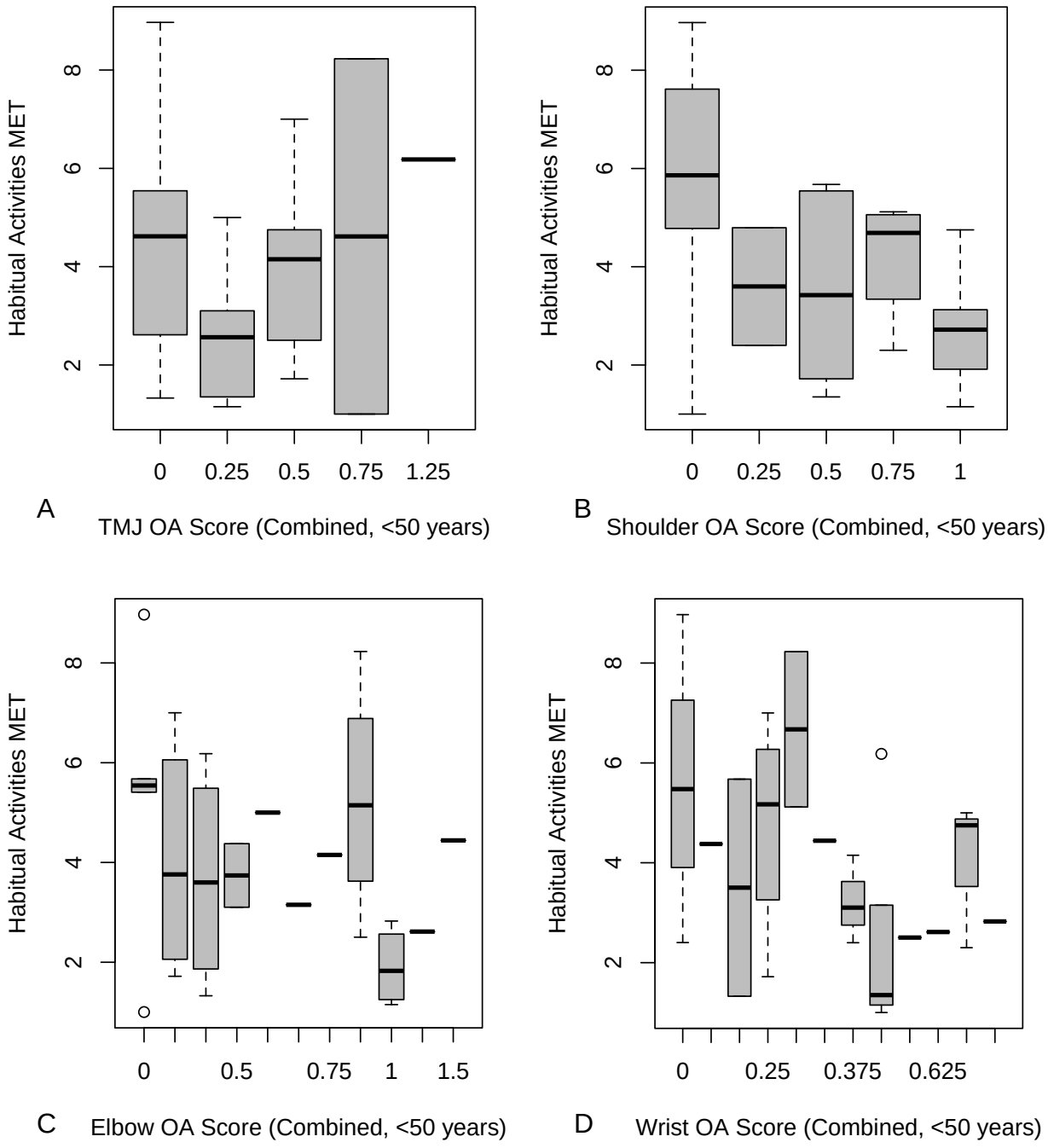


Figure 6-122. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for habitual activity MET value per OA score in the combined sample (<50 years). A) TMJ OA, B) Shoulder OA, C) Elbow OA, D) Wrist OA.

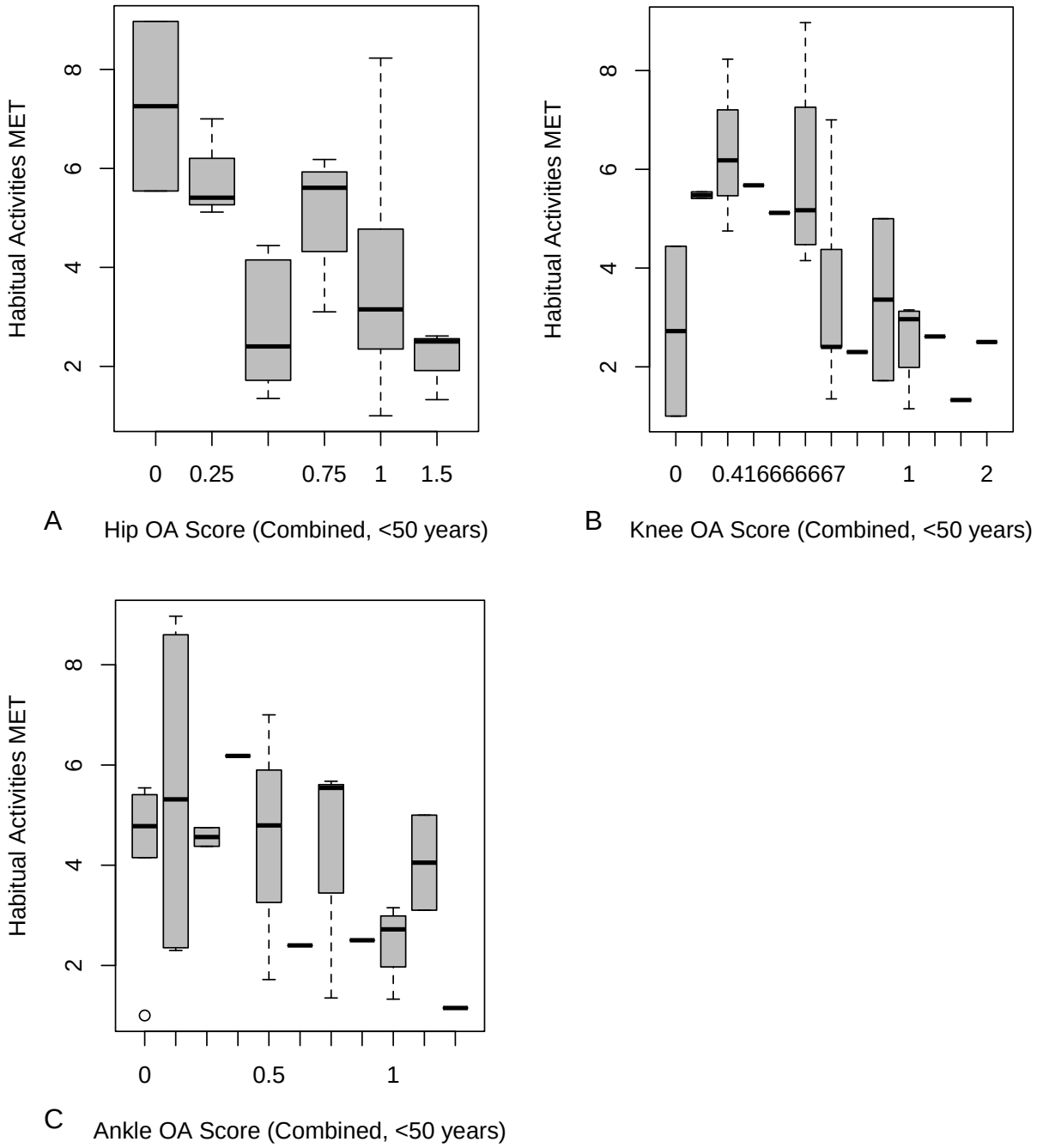


Figure 6-123. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for habitual activity MET value per OA score in the combined sample (<50 years). A) Hip OA, B) Knee OA, C) Ankle OA.

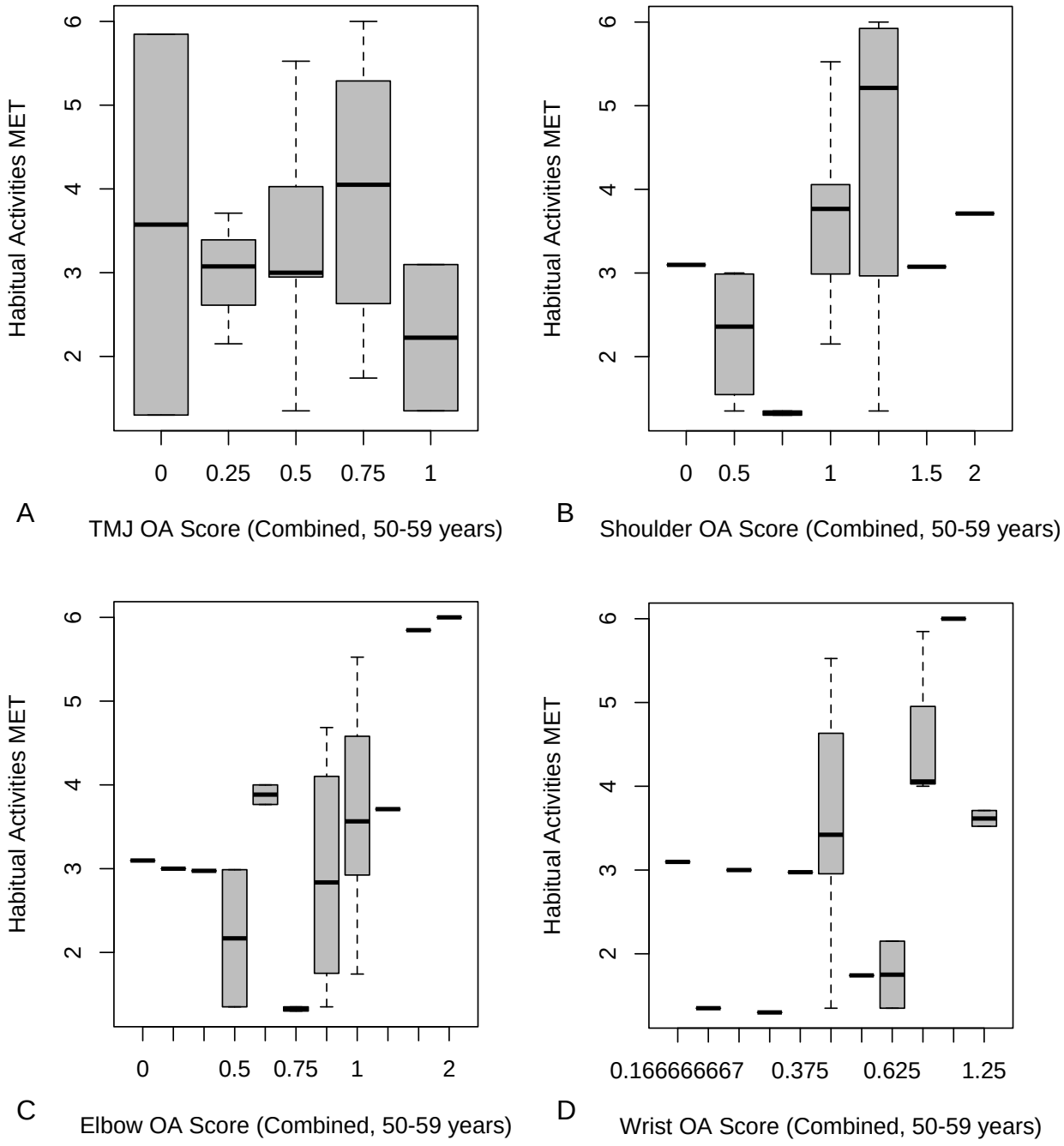


Figure 6-124. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for habitual activity MET value per OA score in the combined sample (50-59 years). A) TMJ OA, B) Shoulder OA, C) Elbow OA, D) Wrist OA.

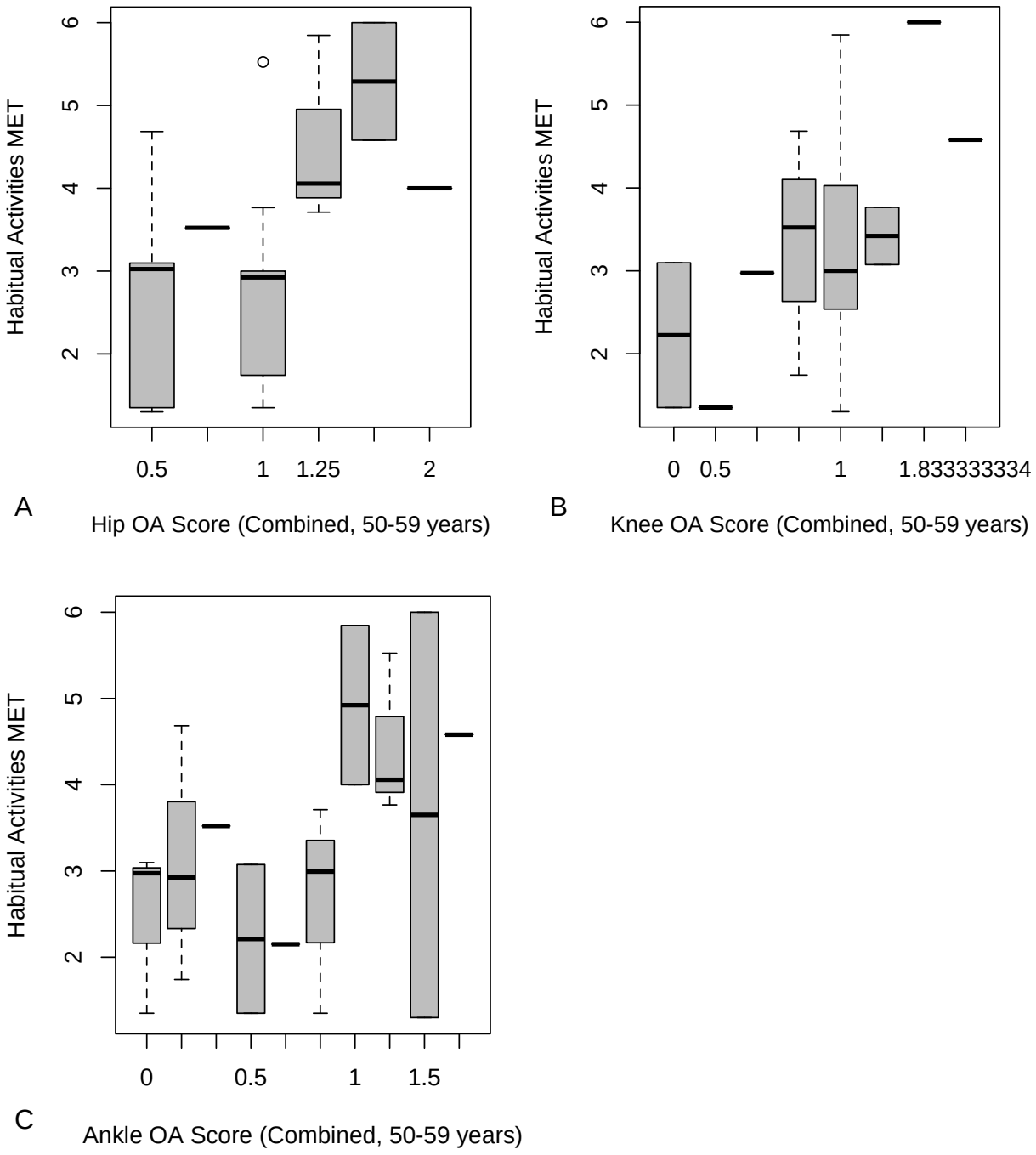


Figure 6-125. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for habitual activity MET value per OA score in the combined sample (50-59 years). A) Hip OA, B) Knee OA, C) Ankle OA.

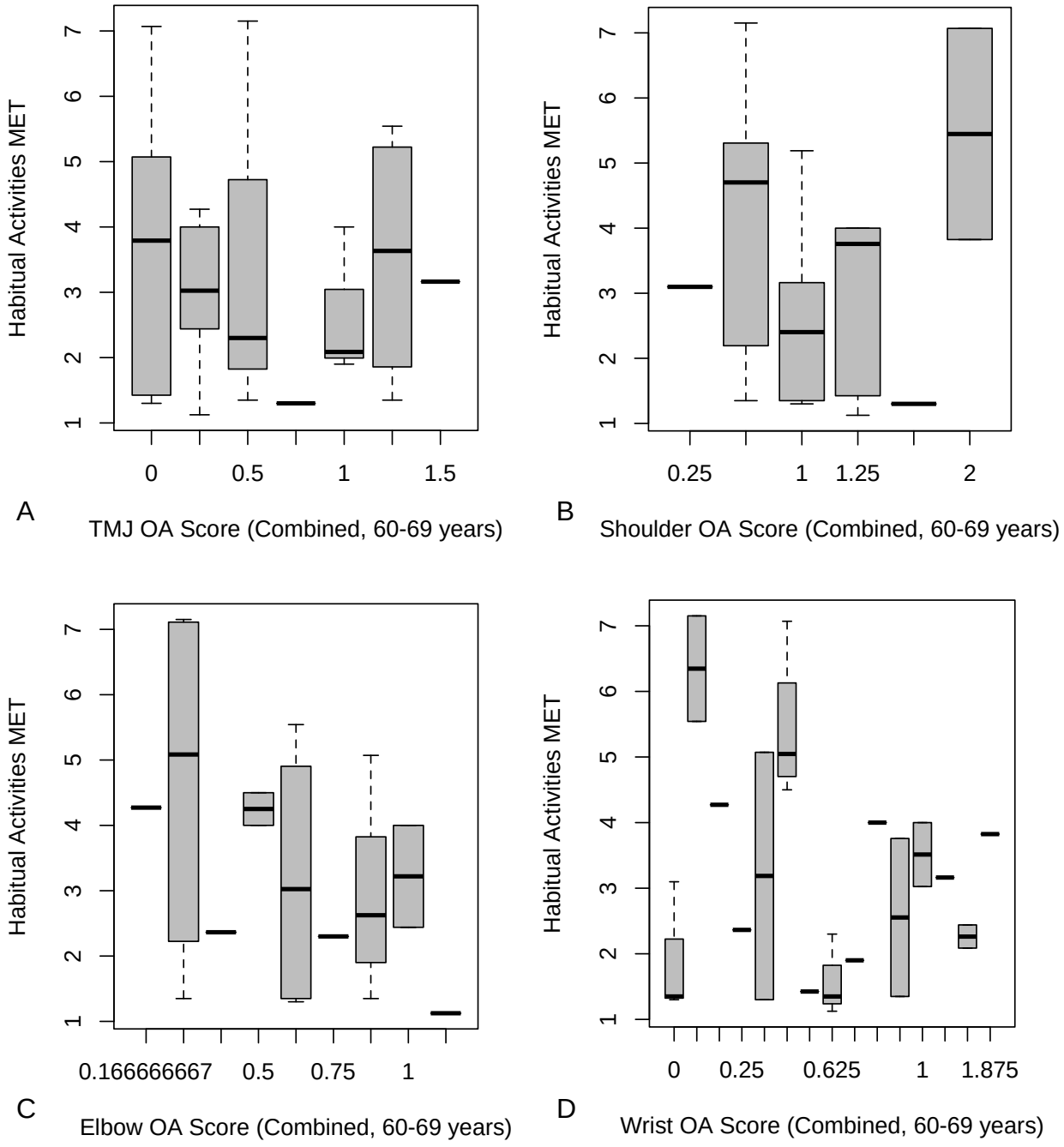


Figure 6-126. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for habitual activity MET value per OA score in the combined sample (60-69 years). A) TMJ OA, B) Shoulder OA, C) Elbow OA, D) Wrist OA.

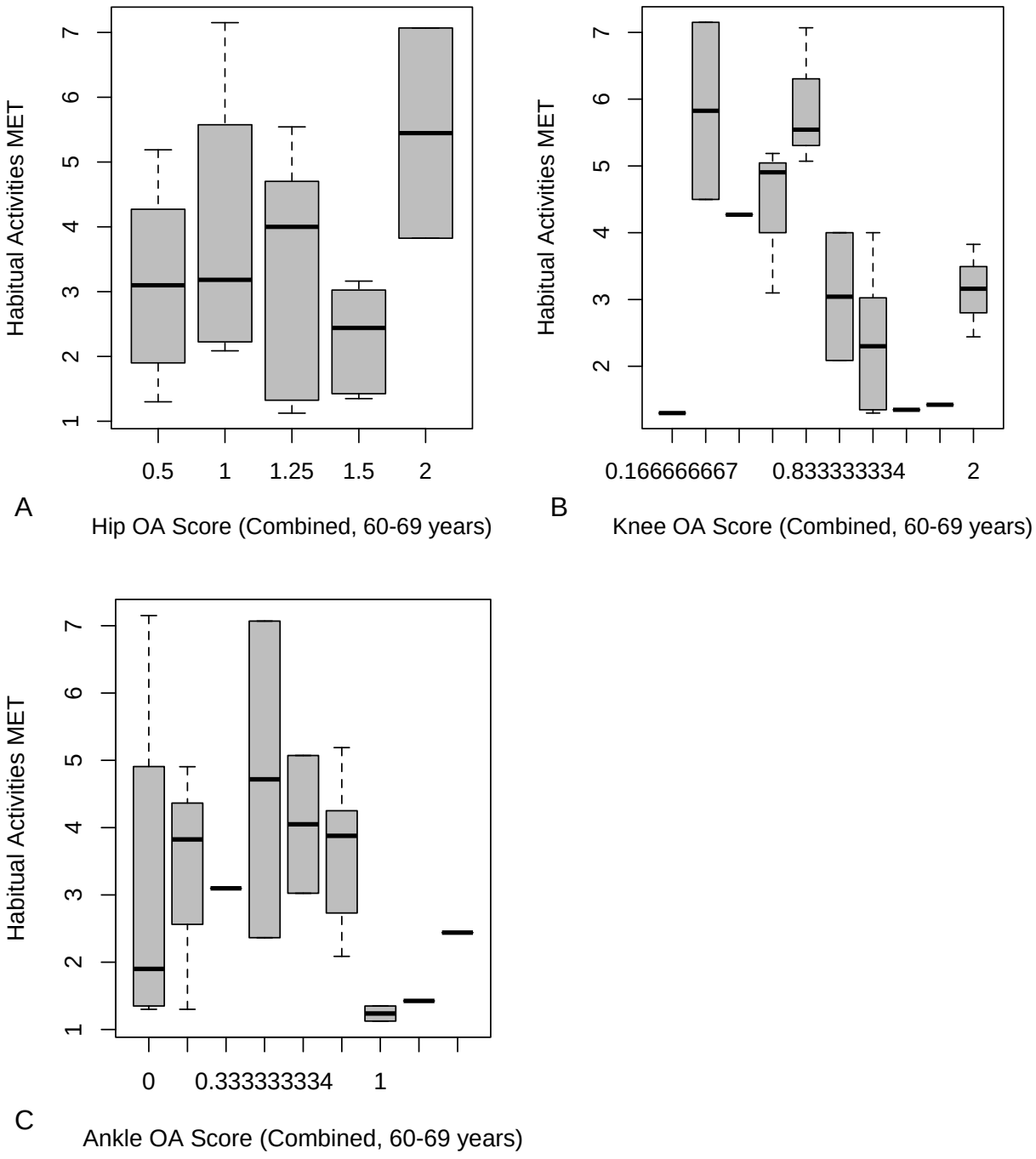


Figure 6-127. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for habitual activity MET value per OA score in the combined sample (60-69 years). A) Hip OA, B) Knee OA, C) Ankle OA.

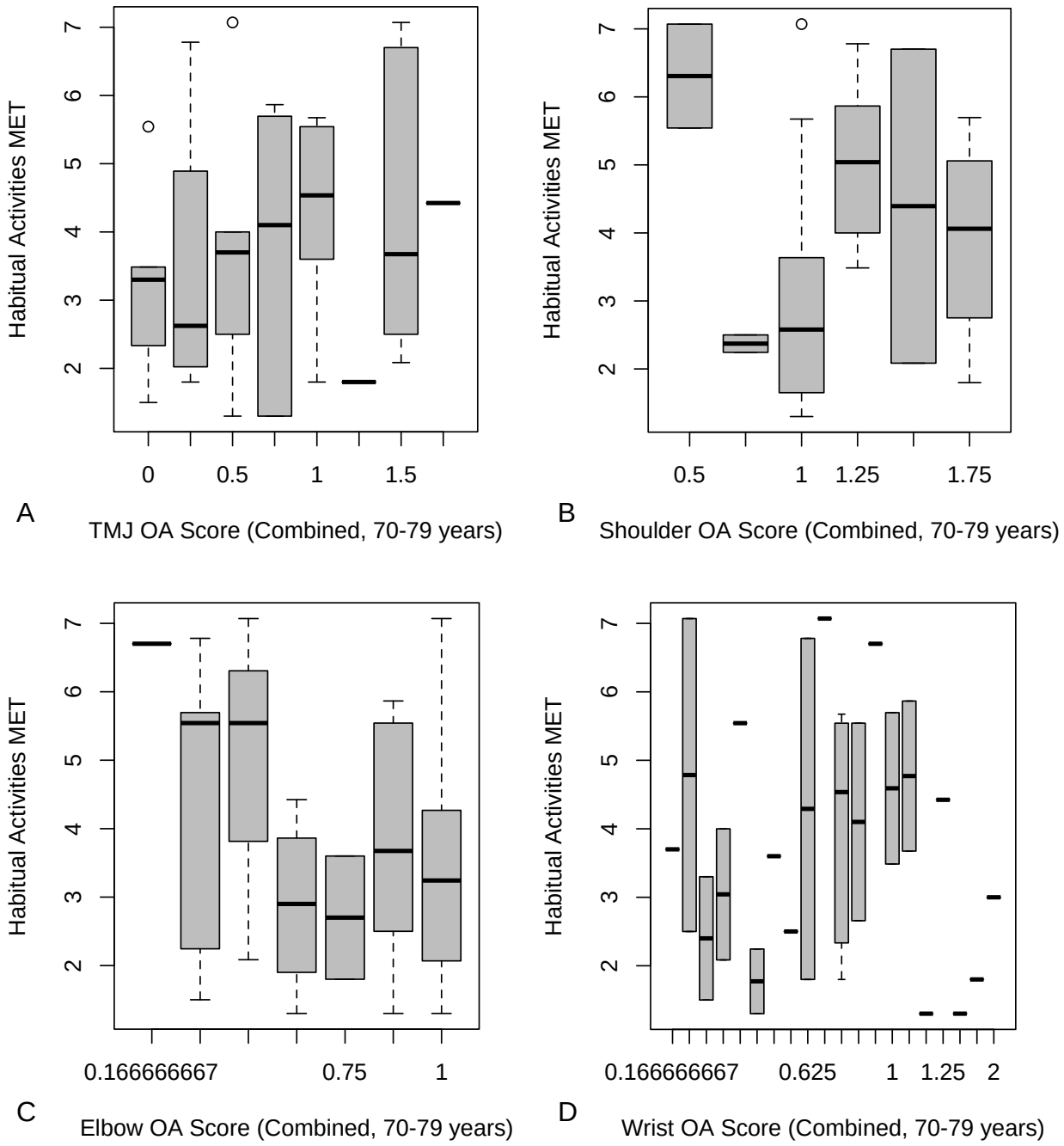


Figure 6-128. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for habitual activity MET value per OA score in the combined sample (70-79 years). A) TMJ OA, B) Shoulder OA, C) Elbow OA, D) Wrist OA.

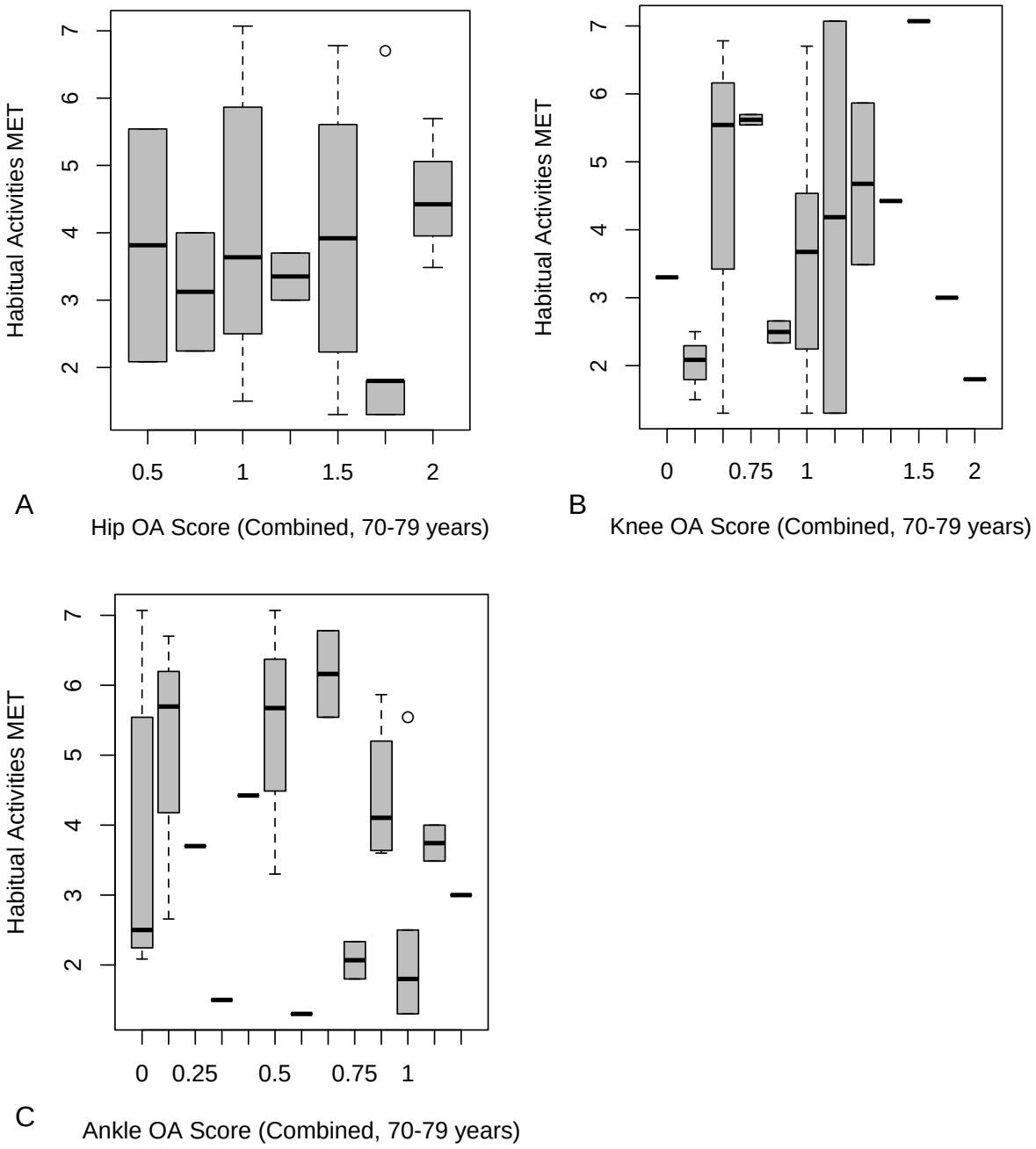


Figure 6-129. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for habitual activity MET value per OA score in the combined sample (70-79 years). A) Hip OA, B) Knee OA, C) Ankle OA.

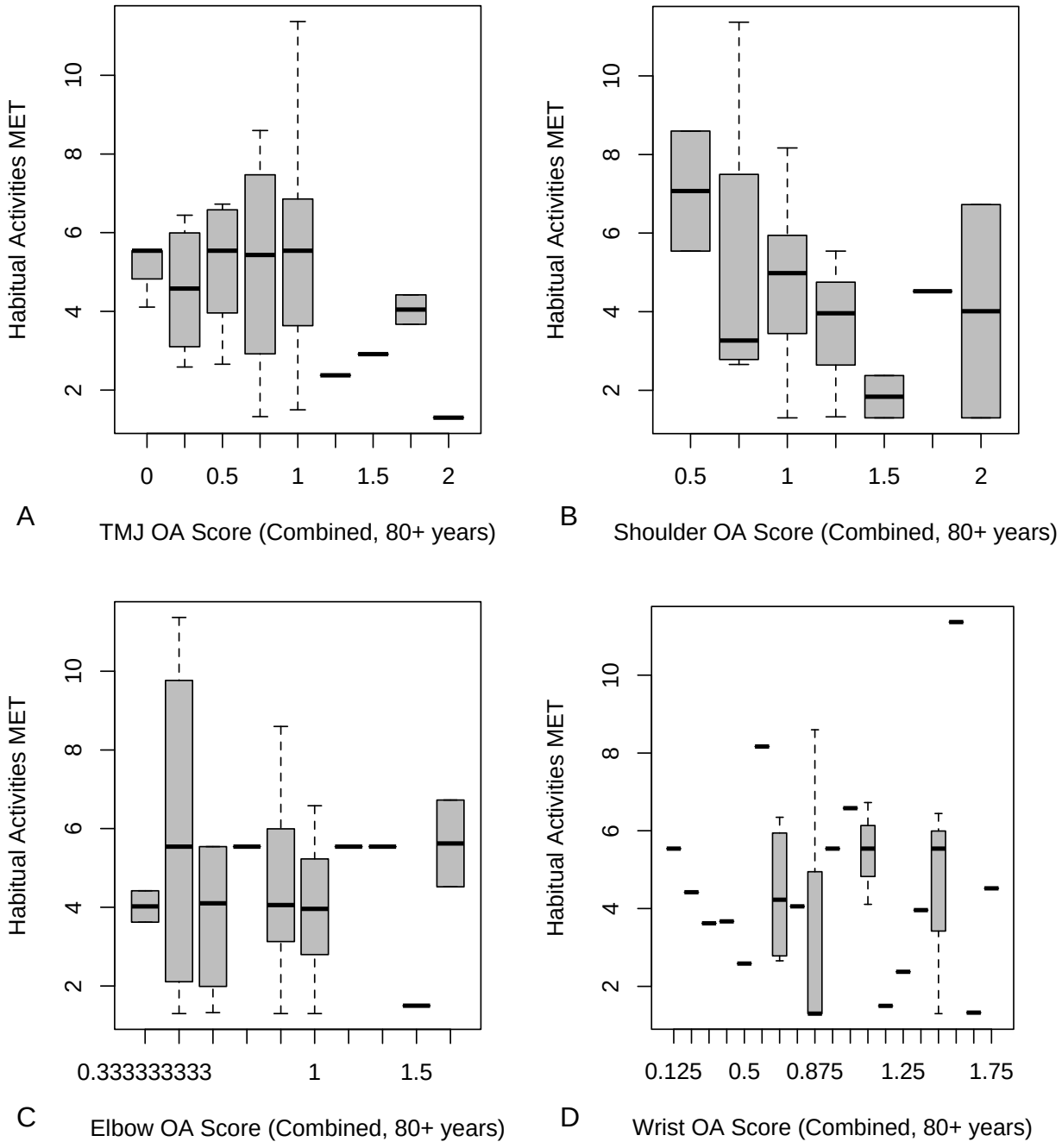


Figure 6-130. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for habitual activity MET value per OA score in the combined sample (80+ years). A) TMJ OA, B) Shoulder OA, C) Elbow OA, D) Wrist OA.

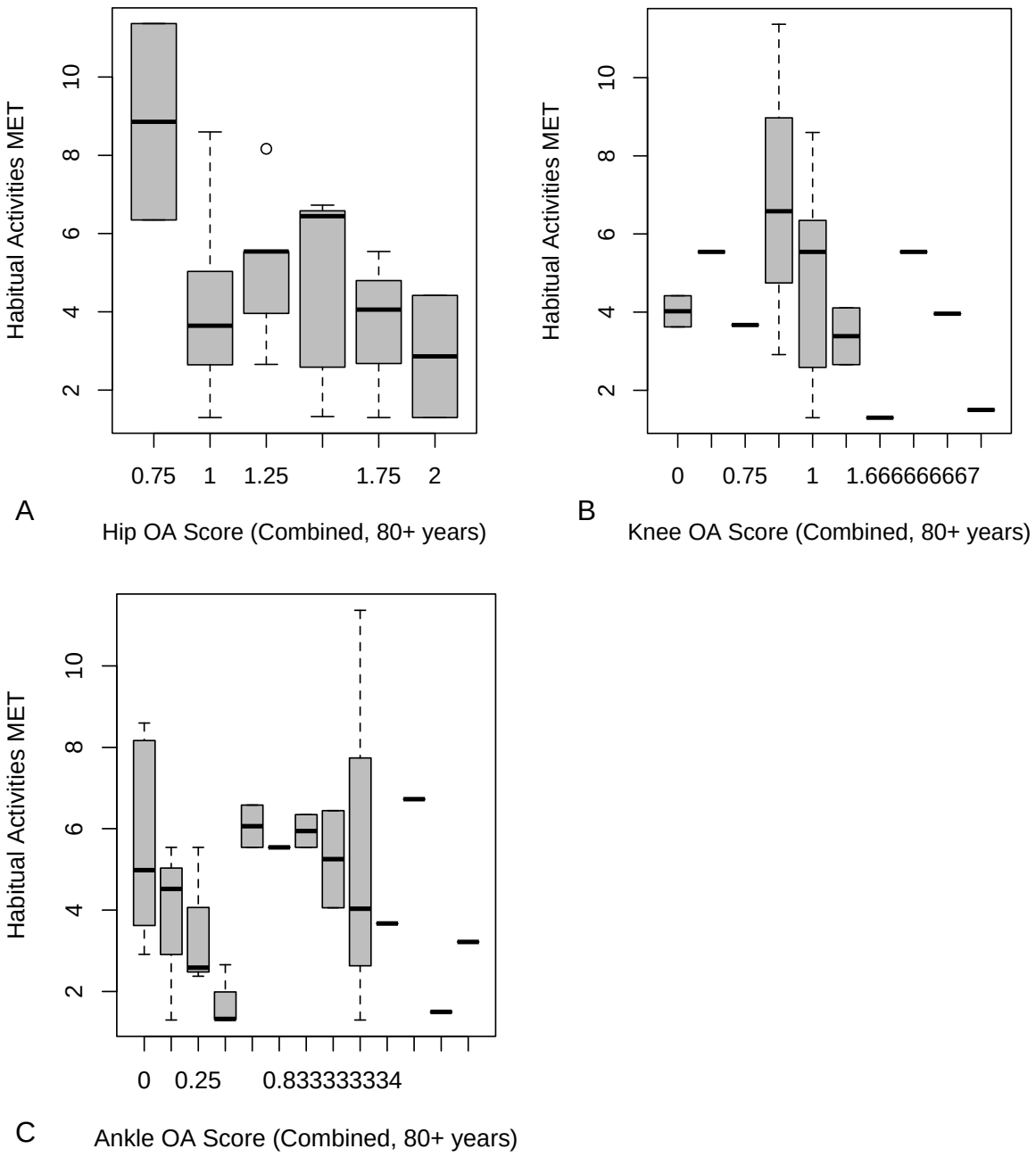


Figure 6-131. Hypothesis 3b: OA correlates positively with activity; box-and-whisker plots for habitual activity MET value per OA score in the combined sample (80+ years). A) Hip OA, B) Knee OA, C) Ankle OA.

Table 6-30. Spearman's rank-order correlations for MET values for habitual physical activities and scores for OA in the various joints in the combined-sex sample.

Group	*rho* (TMJ)	*rho* (Shoulder)	*rho* (Elbow)	*rho* (Wrist)	*rho* (Hip)	*rho* (Knee)	*rho* (Ankle)
Combined <50 years	-0.093	-0.474	-0.302	-0.370	-0.460	-0.508	-0.291
Combined 50-59 years	0.049	0.479	0.458	0.410	0.491	0.427	0.442
Combined 60-69 years	-0.090	-0.178	-0.311	-0.115	0.015	-0.463	-0.114
Combined 70-79 years	0.178	0.198	-0.181	-0.022	-0.093	0.152	-0.199
Combined 80+ years	-0.399	-0.278	0.045	-0.024	-0.137	-0.244	-0.042

*Statistically significant at α=0.001 (Bonferroni correction: α=0.05/35).

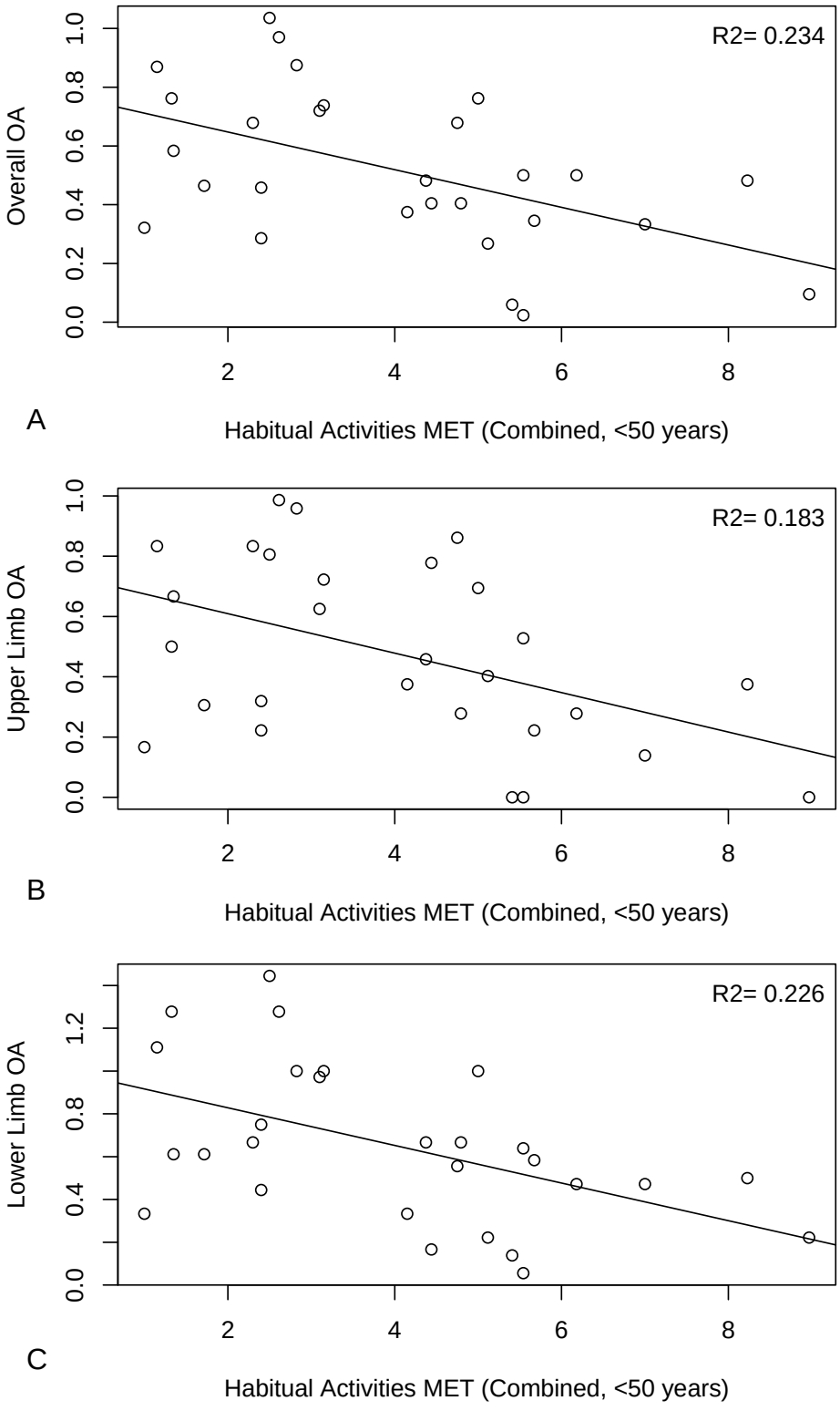


Figure 6-132. Hypothesis 3b: OA correlates positively with activity; scatterplots for summary OA and MET habitual activity values in combined-sex individuals <50 years. A) Overall OA, B) Upper limb OA, C) Lower limb OA. Black line indicates linear regression model.

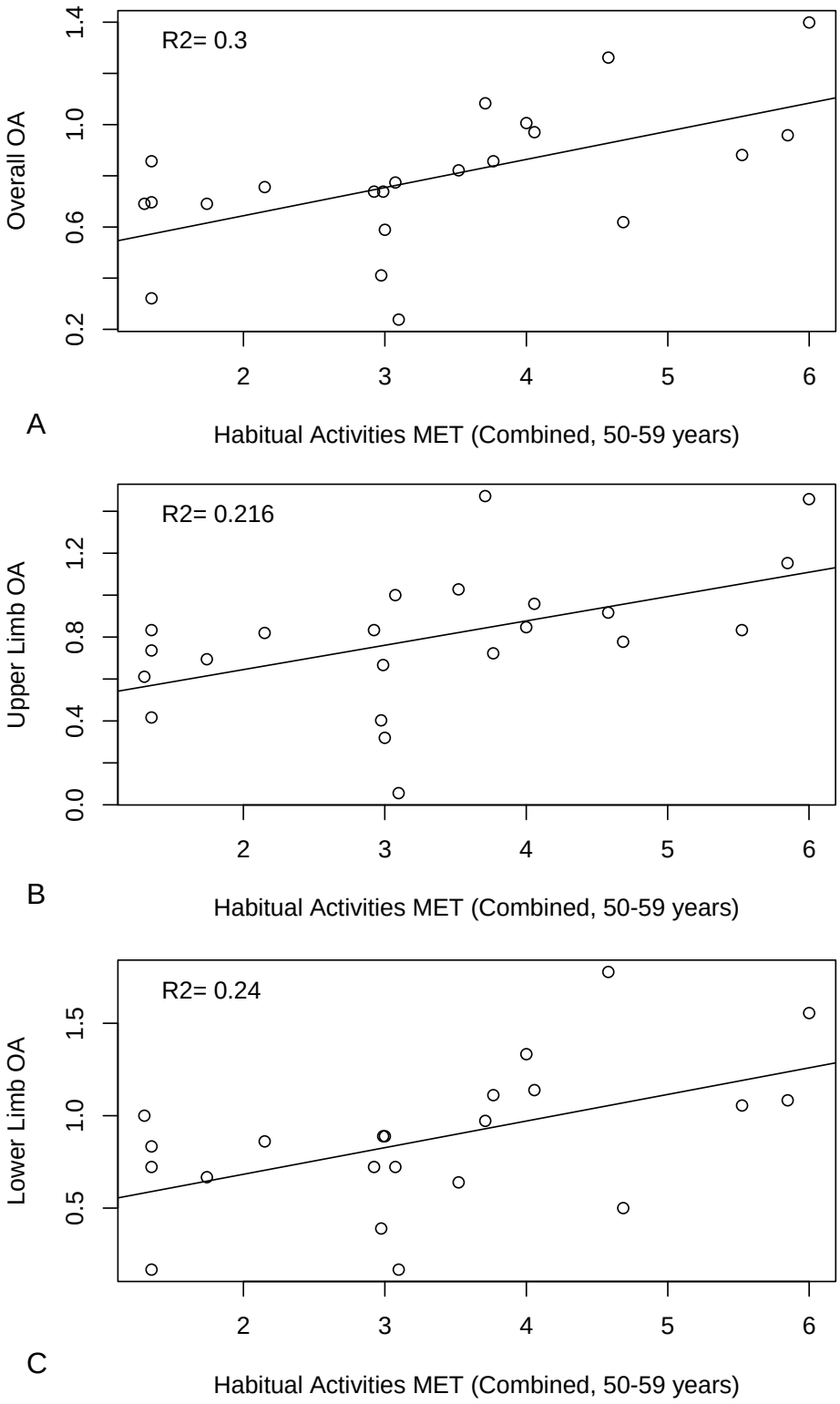


Figure 6-133. Hypothesis 3b: OA correlates positively with activity; scatterplots for summary OA and MET habitual activity values in combined-sex individuals 50-59 years. A) Overall OA, B) Upper limb OA, C) Lower limb OA. Black line indicates linear regression model.

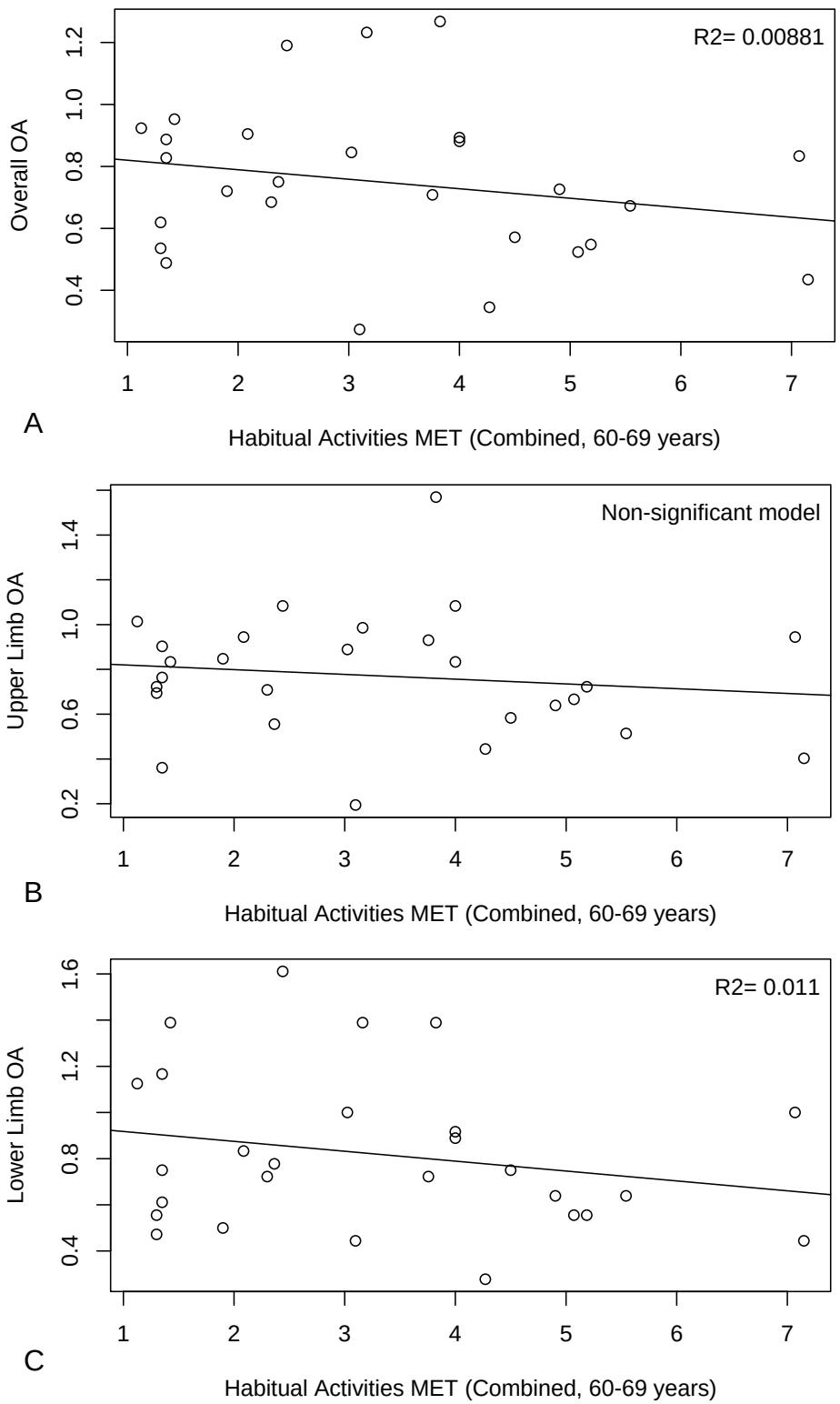


Figure 6-134. Hypothesis 3b: OA correlates positively with activity; scatterplots for summary OA and MET habitual activity values in combined-sex individuals 60-69 years. A) Overall OA, B) Upper limb OA, C) Lower limb OA. Black line indicates linear regression model.

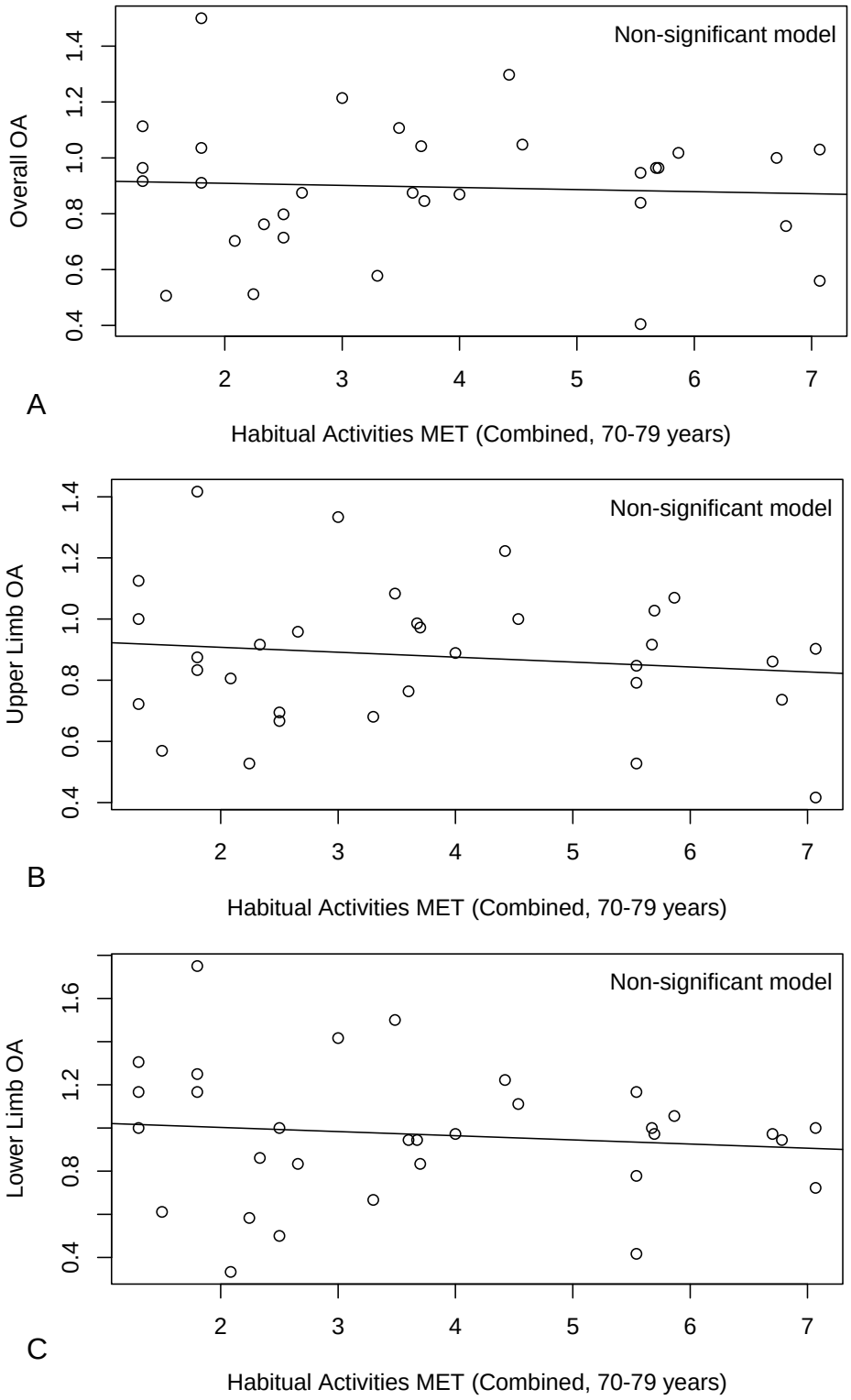


Figure 6-135. Hypothesis 3b: OA correlates positively with activity; scatterplots for summary OA and MET habitual activity values in combined-sex individuals 70-79 years. A) Overall OA, B) Upper limb OA, C) Lower limb OA. Black line indicates linear regression model.

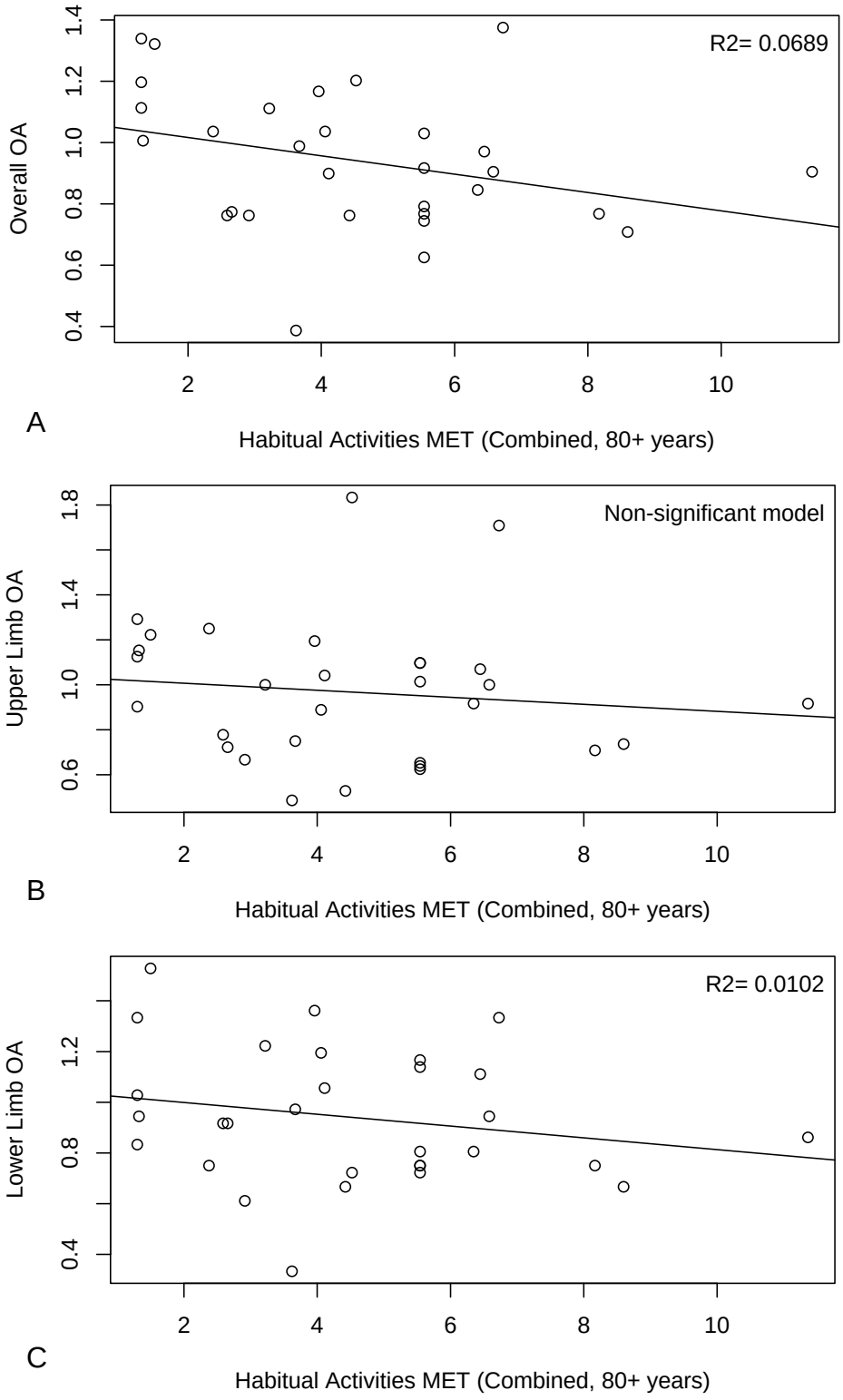


Figure 6-136. Hypothesis 3b: OA correlates positively with activity; scatterplots for summary OA and MET habitual activity values in combined-sex individuals 80+. A) Overall OA, B) Upper limb OA, C) Lower limb OA. Black line indicates linear regression model.

Table 6-31. Spearman's rank-order correlations for summary OA and MET values for habitual physical activities in the combined-sex sample.

Group	*rho*
<50 years overall OA	-0.427
<50 years upper limb OA	-0.421
<50 years lower limb OA	-0.482
50-59 years overall OA	0.613
50-59 years upper limb OA	0.563
50-59 years lower limb OA	0.531
60-69 years overall OA	-0.236
60-69 years upper limb OA	-0.199
60-69 years lower limb OA	-0.174
70-79 years overall OA	-0.013
70-79 years upper limb OA	-0.026
70-79 years lower limb OA	-0.141
80+ years overall OA	-0.347
80+ years upper limb OA	-0.193
80+ years lower limb OA	-0.188

*Statistically significant at α=0.003 (Bonferroni correction: α=0.05/15).

Table 6-32. Regression results for summary OA and MET values for habitual physical activities in the combined sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	0.776	0.096	8.114	1.36e-08*
<50 years overall OA	-0.064	0.021	-3.044	0.005
Intercept	0.741	0.112	6.604	5.29e-07*
<50 years upper limb OA	-0.066	0.025	-2.652	0.014
Intercept	1.005	0.134	7.509	5.68e-08*
<50 years lower limb OA	-0.088	0.030	-2.983	0.006
Intercept	0.424	0.125	3.382	0.003
50-59 years overall OA	0.110	0.035	3.163	0.005
Intercept	0.413	0.161	2.571	0.018
50-59 years upper limb OA	0.116	0.045	2.607	0.017
Intercept	0.395	0.188	2.099	0.049
50-59 years lower limb OA	0.144	0.052	2.762	0.012
Intercept	0.851	0.103	8.266	1.29e-08*
60-69 years overall OA	-0.031	0.028	-1.110	0.278
Intercept	0.842	0.116	7.265	1.3e-07*
60-69 years upper limb OA	-0.021	0.031	-0.683	0.501
Intercept	0.961	0.141	6.838	3.62e-07*
60-69 years lower limb OA	-0.043	0.038	-1.135	0.267
Intercept	0.924	0.096	9.637	1.07e-10*
70-79 years overall OA	-0.008	0.023	-0.328	0.745
Intercept	0.940	0.091	10.304	2.27e-11*
70-79 years upper limb OA	-0.016	0.022	-0.737	0.467
Intercept	1.041	0.125	8.355	2.52e-09*
70-79 years lower limb OA	-0.019	0.030	-0.647	0.523
Intercept	1.076	0.086	12.454	6.18e-13*
80+ years overall OA	-0.030	0.017	-1.773	0.087
Intercept	1.038	0.125	8.318	4.74e-09*
80+ years upper limb OA	-0.016	0.024	-0.643	0.525
Intercept	1.045	0.104	10.022	9.18e-11*
80+ years lower limb OA	-0.023	0.020	-1.139	0.264

*Statistically significant at α=0.003 (Bonferroni correction: α=0.05/15).

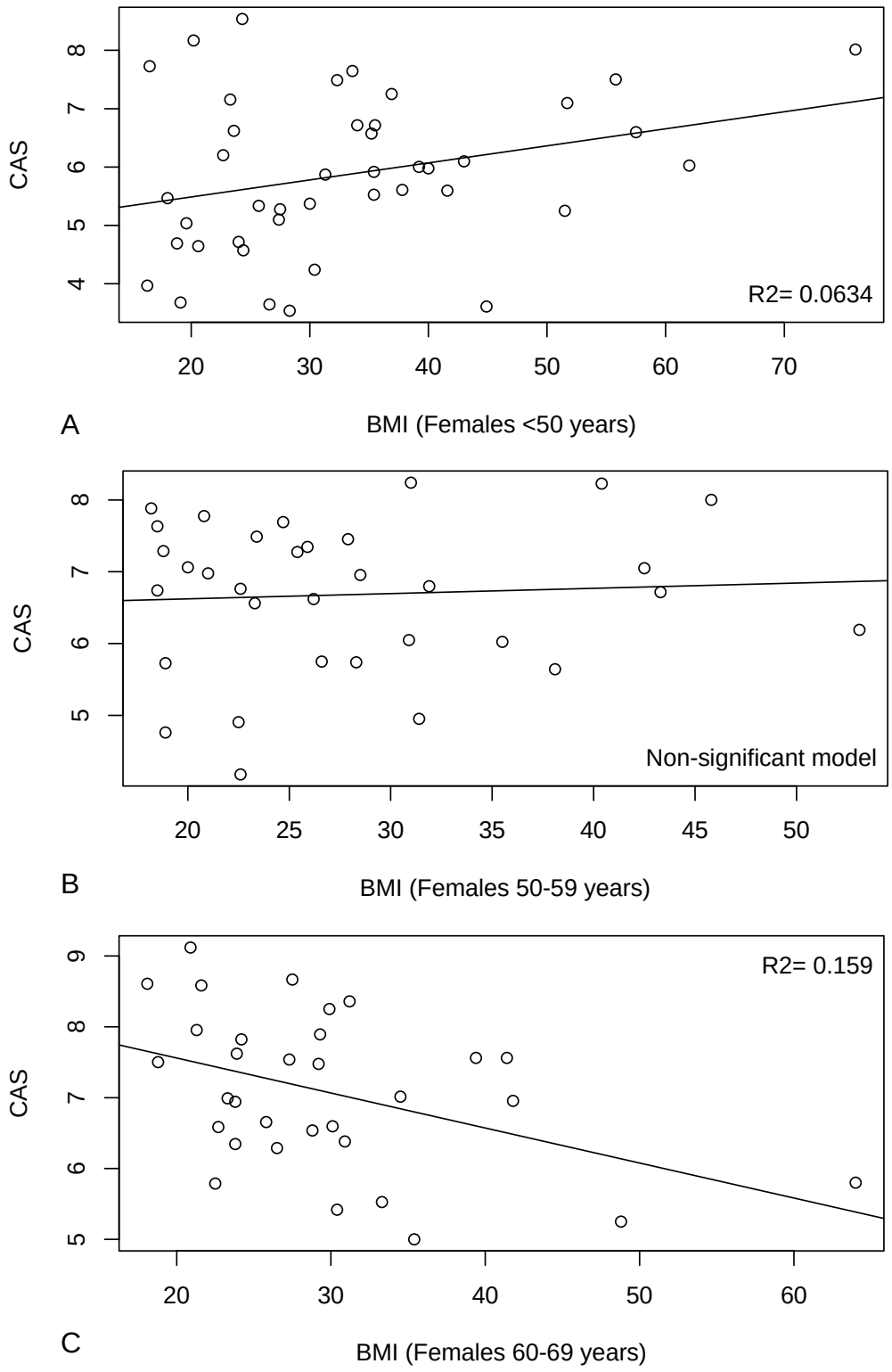


Figure 6-137. Hypothesis 4a: acetabular changes correlate positively with obesity; scatterplots for CAS and BMI values in females. A) <50 years, B) 50-59 years, C) 60-69 years. Black line indicates linear regression model.

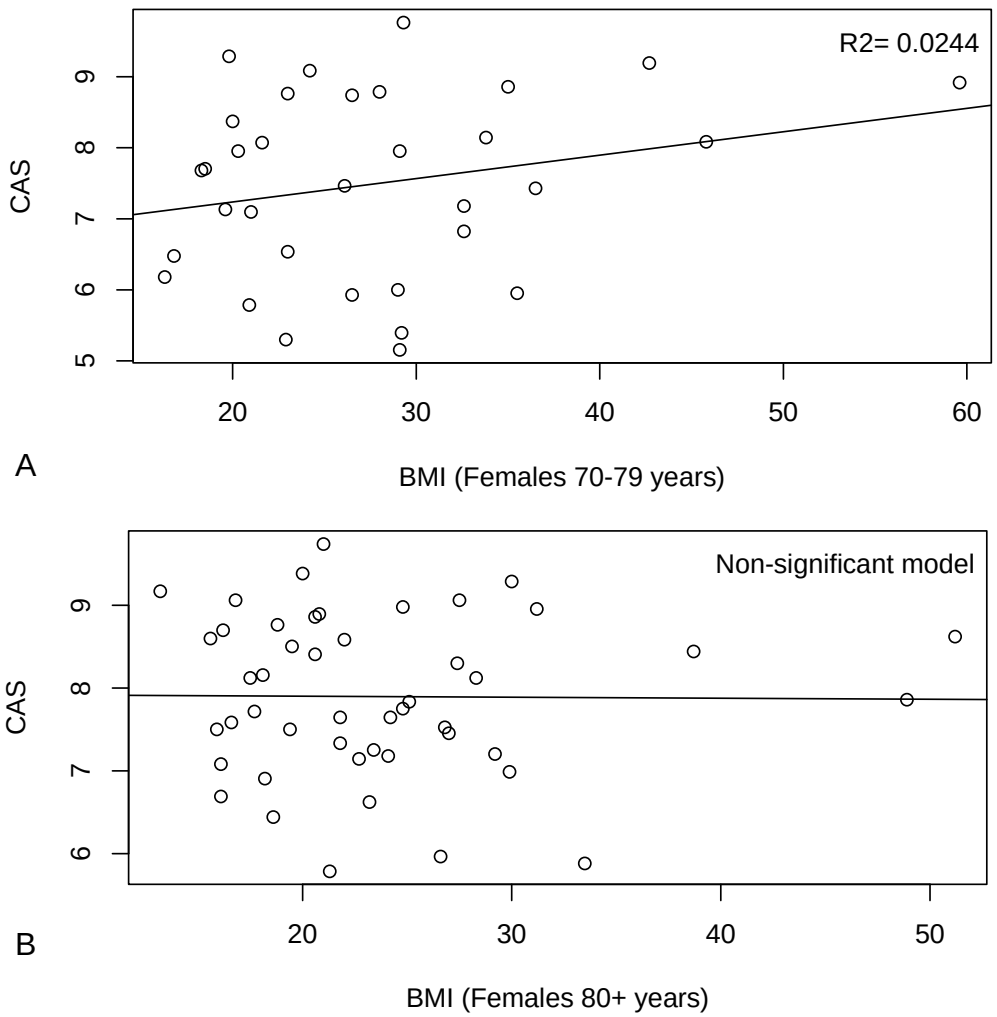


Figure 6-138. Hypothesis 4a: acetabular changes correlate positively with obesity; scatterplots for CAS and BMI values in females. A) 70-79 years, B) 80+ years. Black line indicates linear regression model.

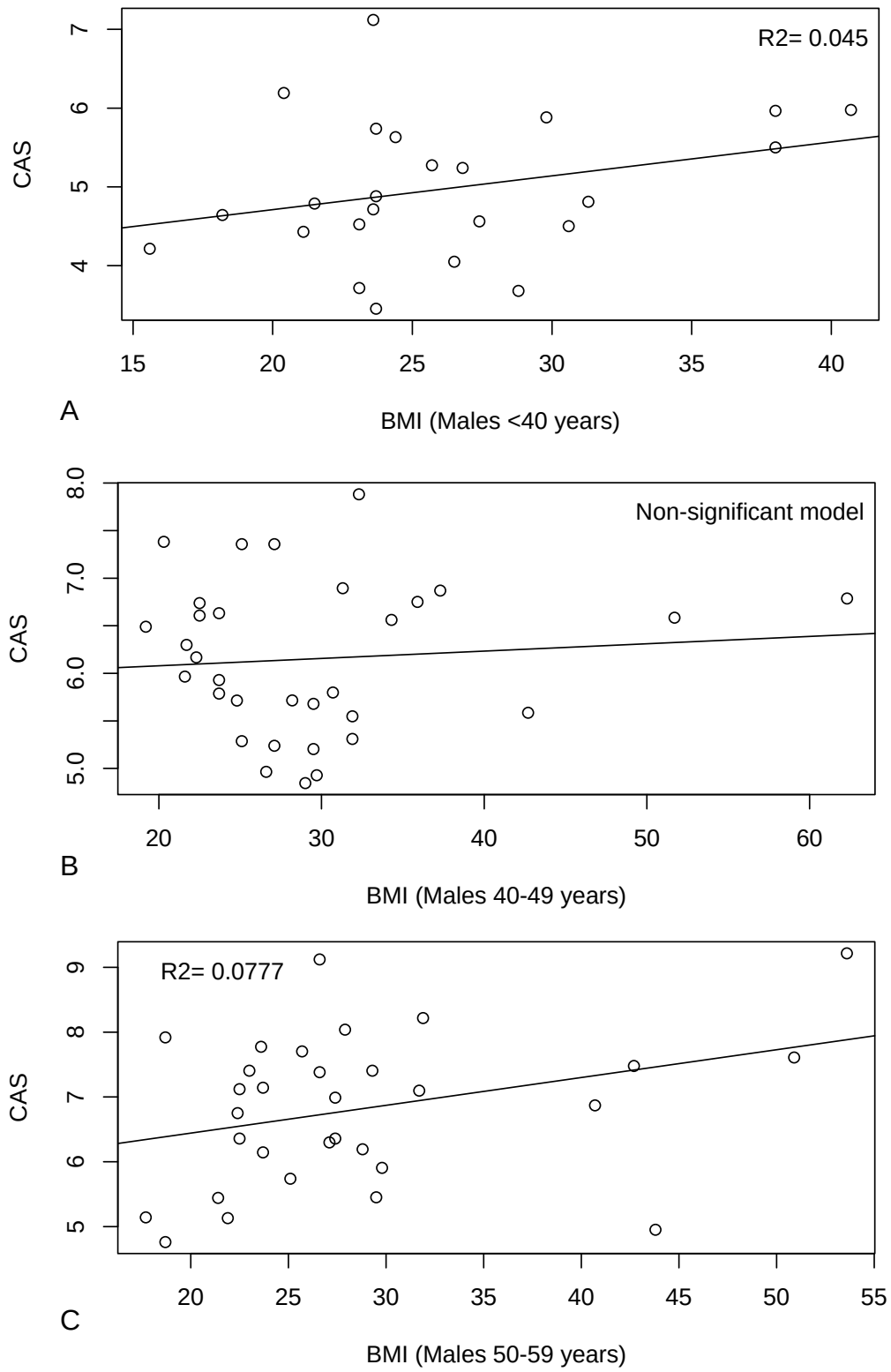


Figure 6-139. Hypothesis 4a: acetabular changes correlate positively with obesity; scatterplots for CAS and BMI values in males. A) <40 years, B) 40-49 years, C) 50-59 years. Black line indicates linear regression model.

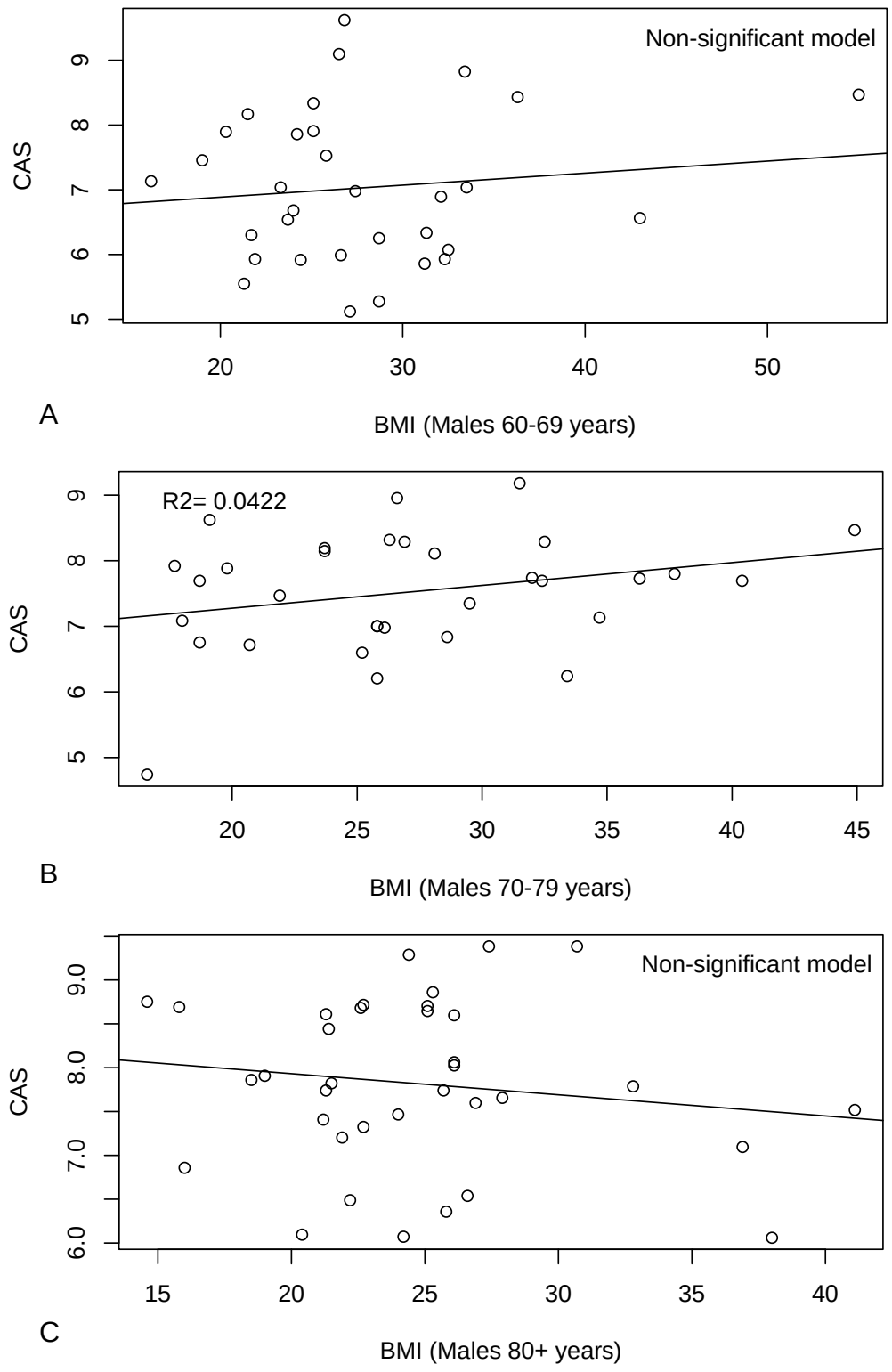


Figure 6-140. Hypothesis 4a: acetabular changes correlate positively with obesity; scatterplots for CAS and BMI values in males. A) 60-69 years, B) 70-79 years, C) 80+ years. Black line indicates linear regression model.

Table 6-33. Spearman's rank-order correlations for BMI values and CAS in the various joints in females and males.

Group	*rho*
Females <50 years	0.270
Females 50-59 years	-0.033
Females 60-69 years	-0.384
Females 70-79 years	0.191
Females 80+ years	-0.037
Males <40 years	0.256
Males 40-49 years	-0.023
Males 50-59 years	0.300
Males 60-69 years	0.011
Males 70-79 years	0.234
Males 80+ years	-0.061

*Statistically significant at α=0.01 for females and α=0.008 for males (Bonferroni correction for females: α=0.05/5; for males: α=0.05/6).

Table 6-34. Regression results for CAS and BMI values in females and males.

Variable	Estimate	Std. Error	t value	Probability
Intercept	4.903	0.538	9.106	2.68e-11*
Females <50 years	0.029	0.015	1.944	0.059
Intercept	6.475	0.611	10.595	7.92e-12*
Females 50-59 years	0.007	0.021	0.353	0.726
Intercept	8.548	0.588	14.546	3.95e-15*
Females 60-69 years	-0.049	0.019	-2.617	0.014
Intercept	6.579	0.715	9.201	2.25e-10*
Females 70-79 years	0.033	0.025	1.342	0.189
Intercept	7.927	0.465	17.043	<2e-16*
Females 80+ years	-0.001	0.019	-0.066	0.948
Intercept	3.852	0.800	4.815	8.26e-05*
Males <40 years	0.043	0.030	1.444	0.163
Intercept	5.924	0.497	11.91	6.75e-13*
Males 40-49 years	0.008	0.016	0.48	0.635
Intercept	5.584	0.683	8.179	5.1e-09*
Males 50-59 years	0.043	0.023	1.878	0.070
Intercept	6.514	0.816	7.980	6.6e-09*
Males 60-69 years	0.019	0.028	0.653	0.519
Intercept	6.583	0.632	10.415	1.76e-11*
Males 70-79 years	0.034	0.023	1.538	0.134
Intercept	8.413	0.711	11.84	1.3e-13*
Males 80+ years	-0.024	0.028	-0.86	0.396

*Statistically significant at α=0.01 for females and α=0.008 for males (Bonferroni correction for females: α=0.05/5; for males: α=0.05/6).

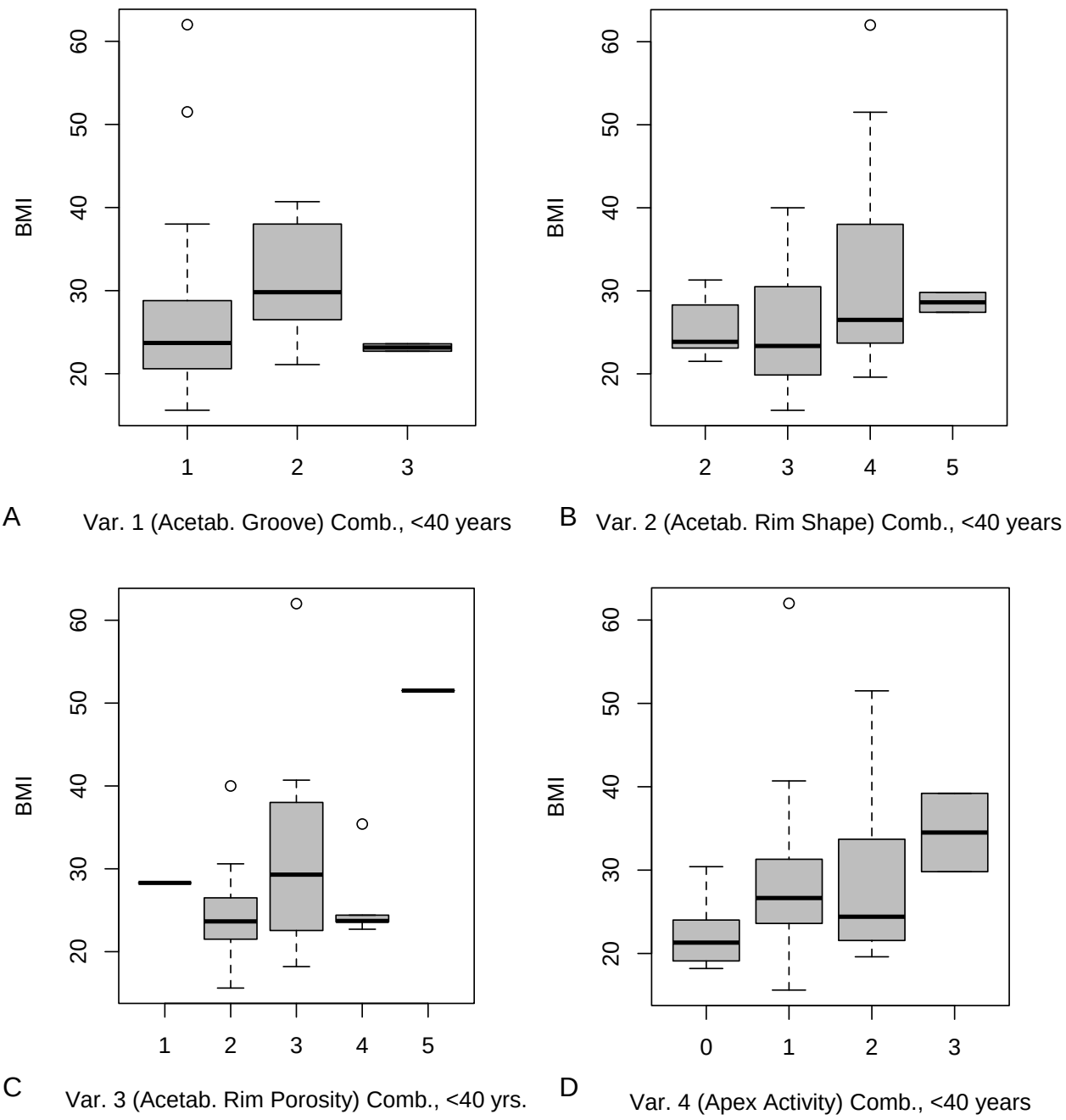


Figure 6-141. Hypothesis 4a: acetabular changes correlate positively with obesity; box-and-whisker plots for BMI value per acetabular variable score in the combined sample (<40 years). A) Variable 1, B) Variable 2, C) Variable 3, D) Variable 4.

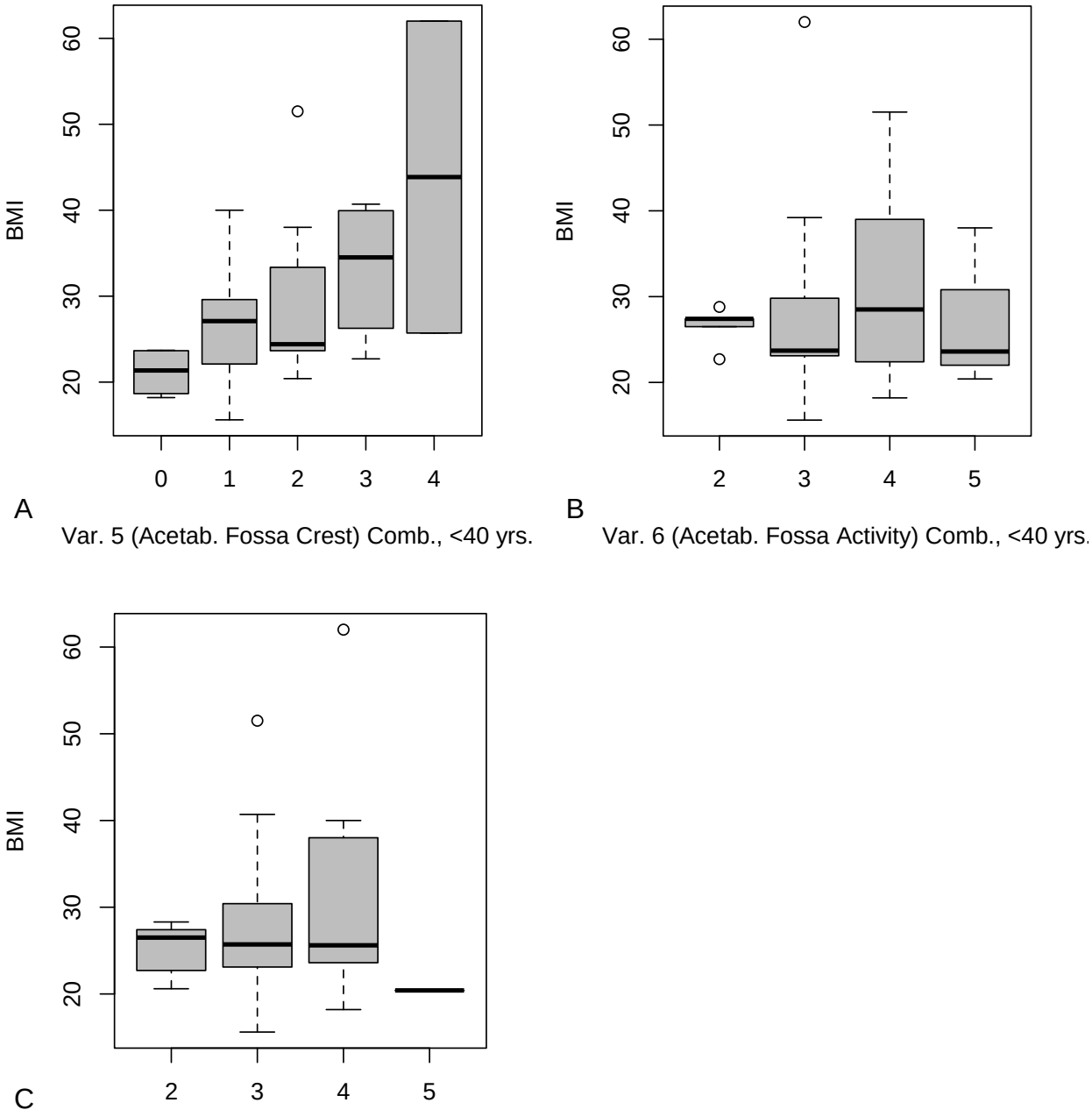


Figure 6-142. Hypothesis 4a: acetabular changes correlate positively with obesity; box-and-whisker plots for BMI value per acetabular variable score in the combined sample (<40 years). A) Variable 5, B) Variable 6, C) Variable 7.

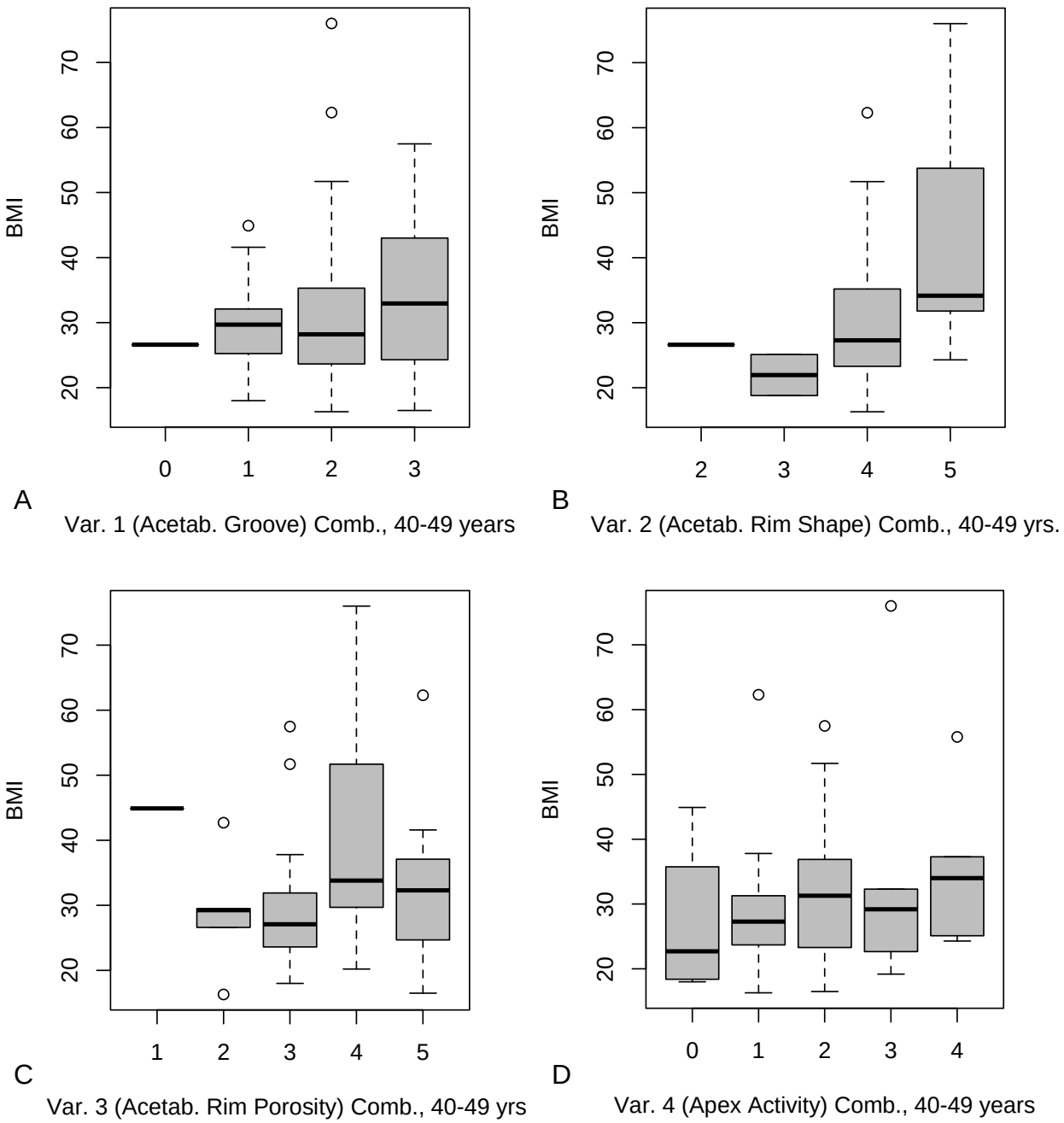


Figure 6-143. Hypothesis 4a: acetabular changes correlate positively with obesity; box-and-whisker plots for BMI value per acetabular variable score in the combined sample (40-49 years). A) Variable 1, B) Variable 2, C) Variable 3, D) Variable 4.

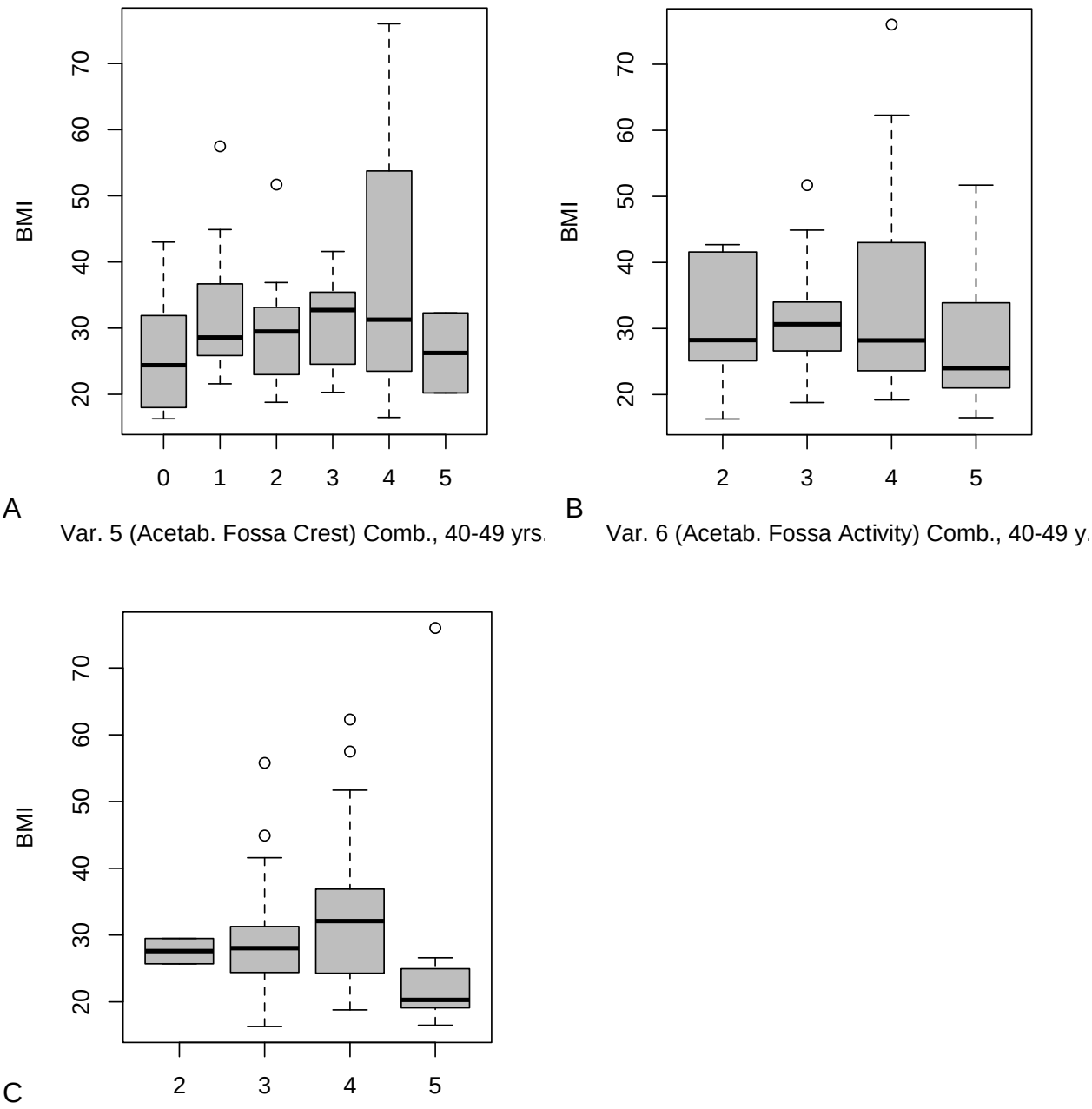


Figure 6-144. Hypothesis 4a: acetabular changes correlate positively with obesity; box-and-whisker plots for BMI value per acetabular variable score in the combined sample (40-49 years). A) Variable 5, B) Variable 6, C) Variable 7.

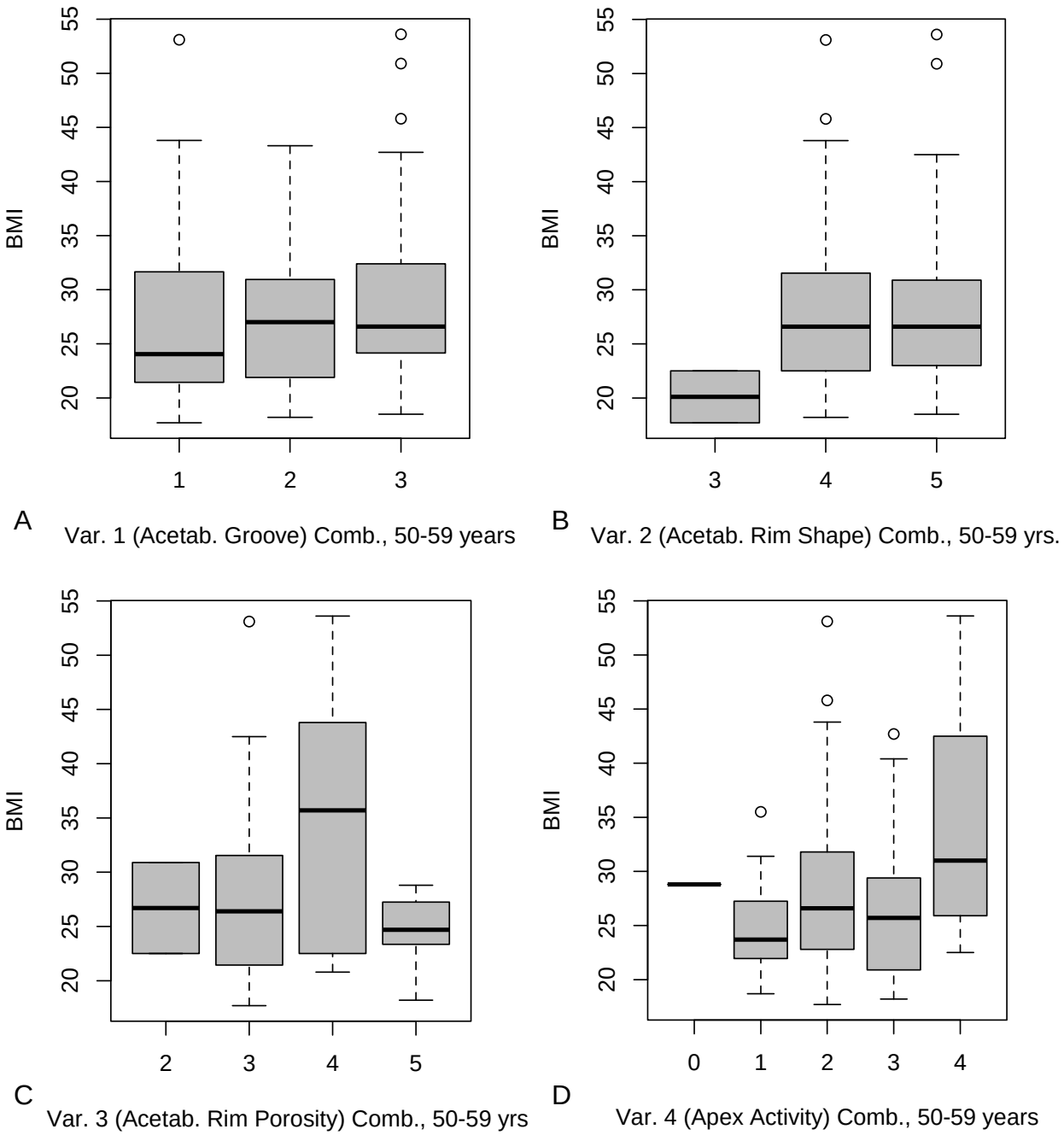


Figure 6-145. Hypothesis 4a: acetabular changes correlate positively with obesity; box-and-whisker plots for BMI value per acetabular variable score in the combined sample (50-59 years). A) Variable 1, B) Variable 2, C) Variable 3, D) Variable 4.

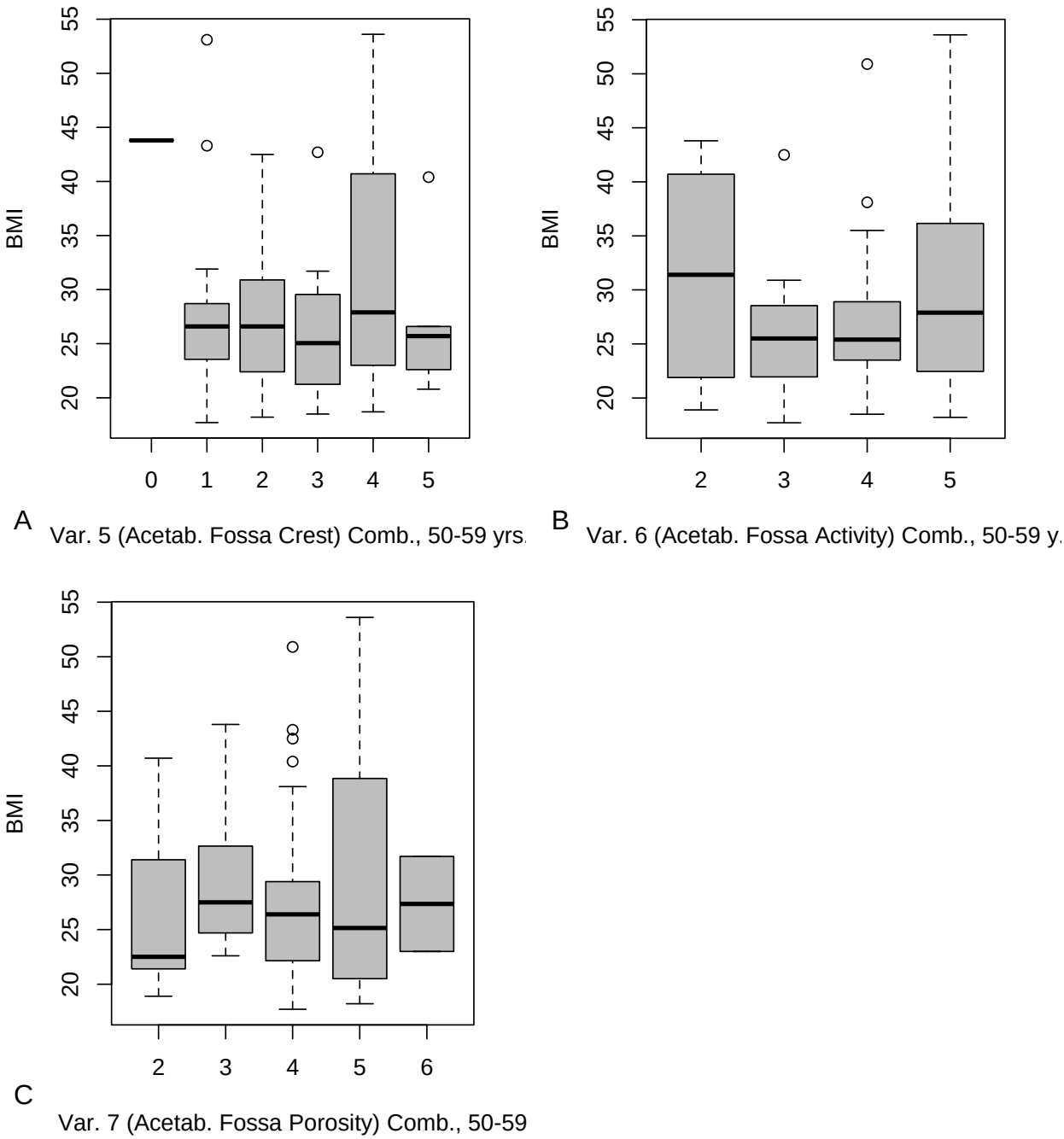


Figure 6-146. Hypothesis 4a: acetabular changes correlate positively with obesity; box-and-whisker plots for BMI value per acetabular variable score in the combined sample (50-59 years). A) Variable 5, B) Variable 6, C) Variable 7.

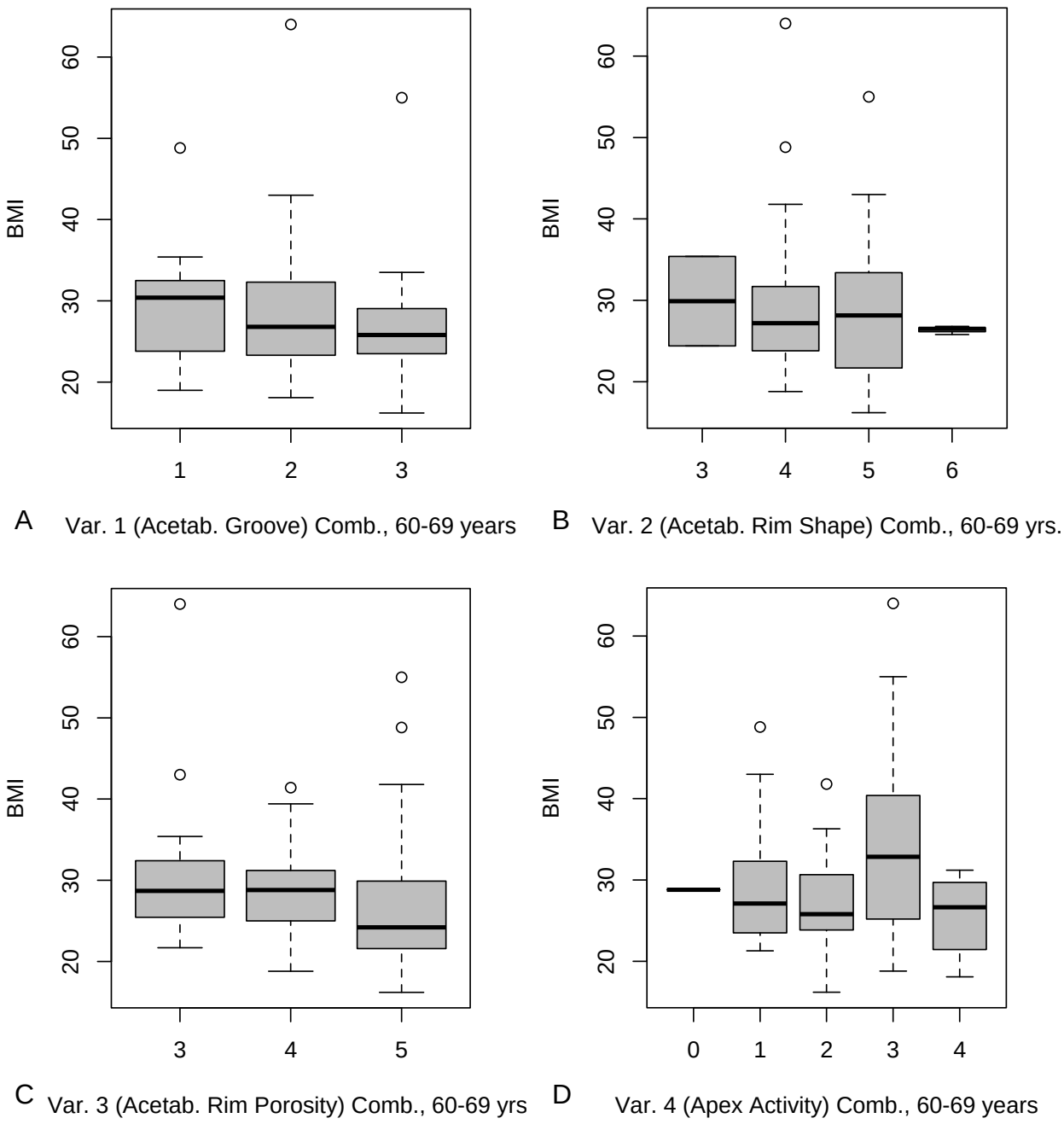


Figure 6-147. Hypothesis 4a: acetabular changes correlate positively with obesity; box-and-whisker plots for BMI value per acetabular variable score in the combined sample (60-69 years). A) Variable 1, B) Variable 2, C) Variable 3, D) Variable 4.

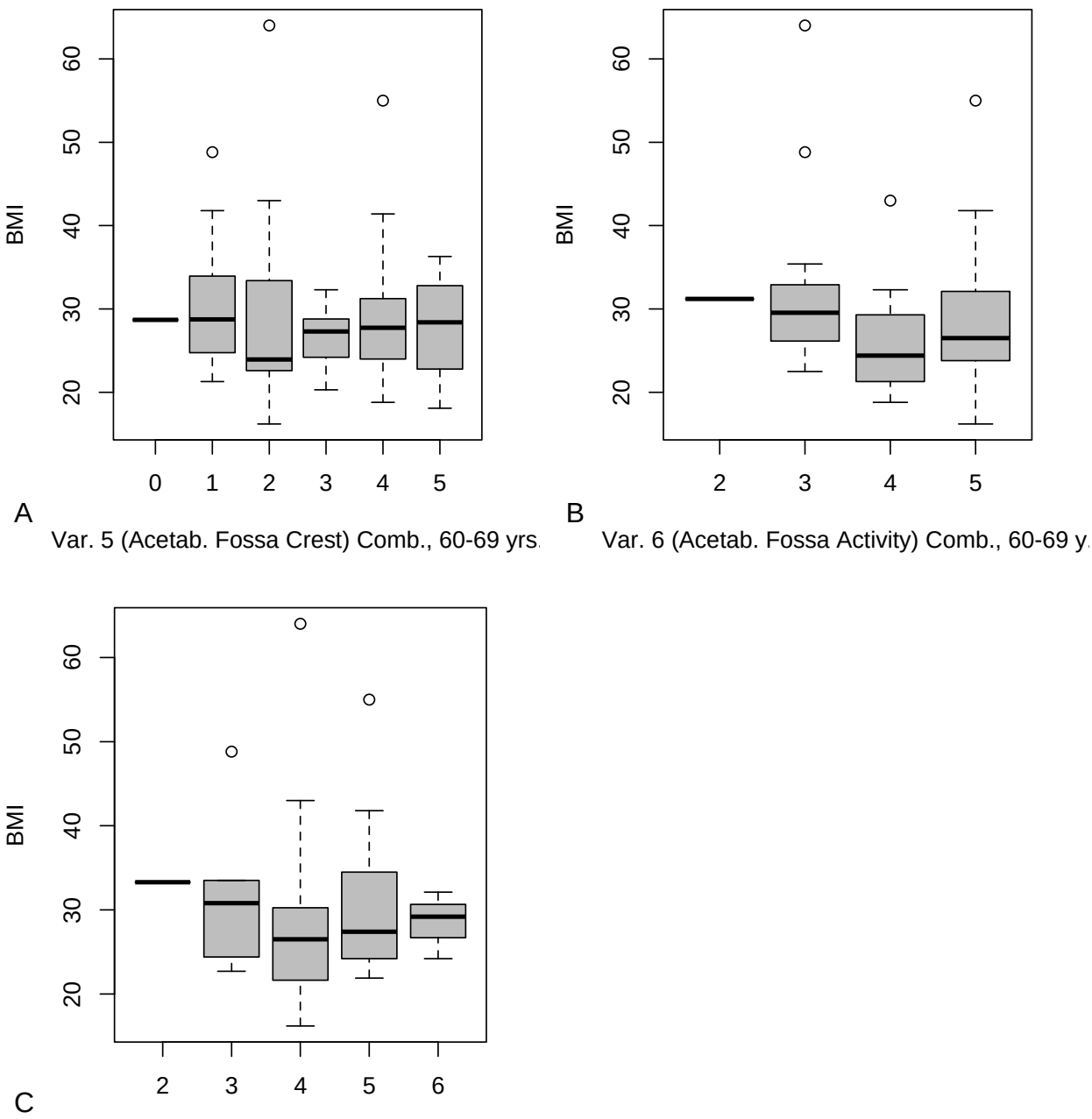


Figure 6-148. Hypothesis 4a: acetabular changes correlate positively with obesity; box-and-whisker plots for BMI value per acetabular variable score in the combined sample (60-69 years). A) Variable 5, B) Variable 6, C) Variable 7.

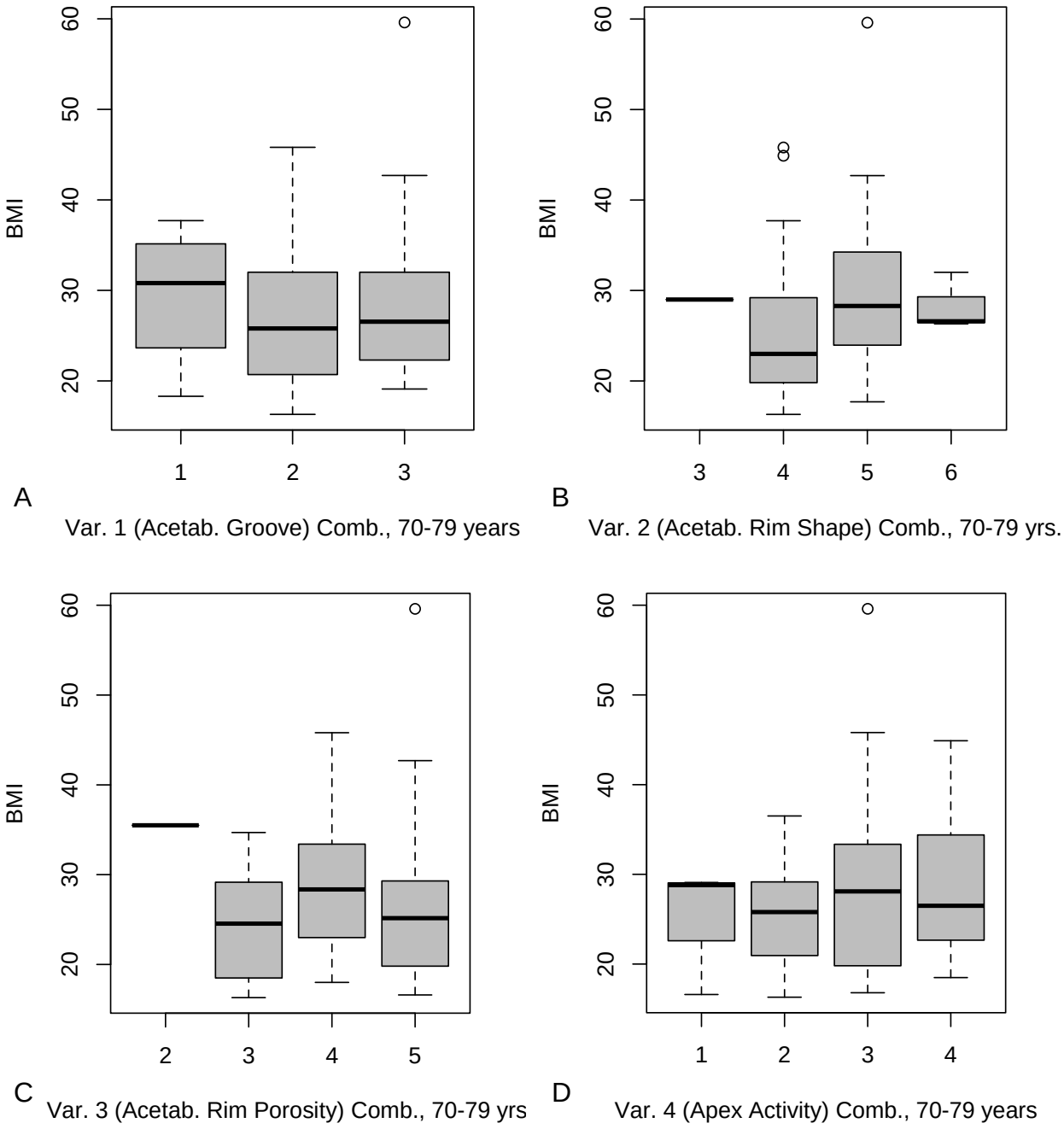


Figure 6-149. Hypothesis 4a: acetabular changes correlate positively with obesity; box-and-whisker plots for BMI value per acetabular variable score in the combined sample (70-79 years). A) Variable 1, B) Variable 2, C) Variable 3, D) Variable 4.

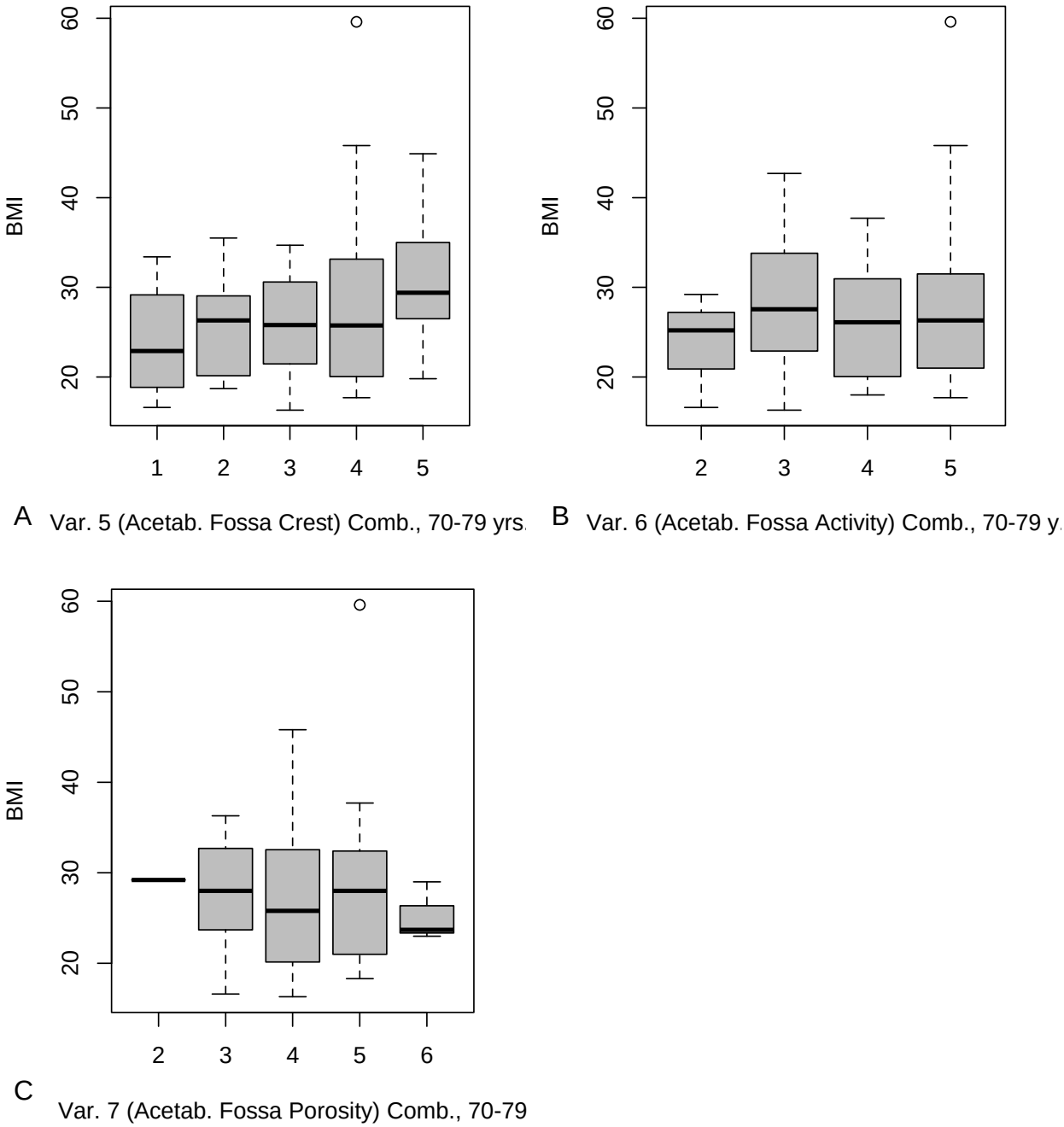


Figure 6-150. Hypothesis 4a: acetabular changes correlate positively with obesity; box-and-whisker plots for BMI value per acetabular variable score in the combined sample (70-79 years). A) Variable 5, B) Variable 6, C) Variable 7.

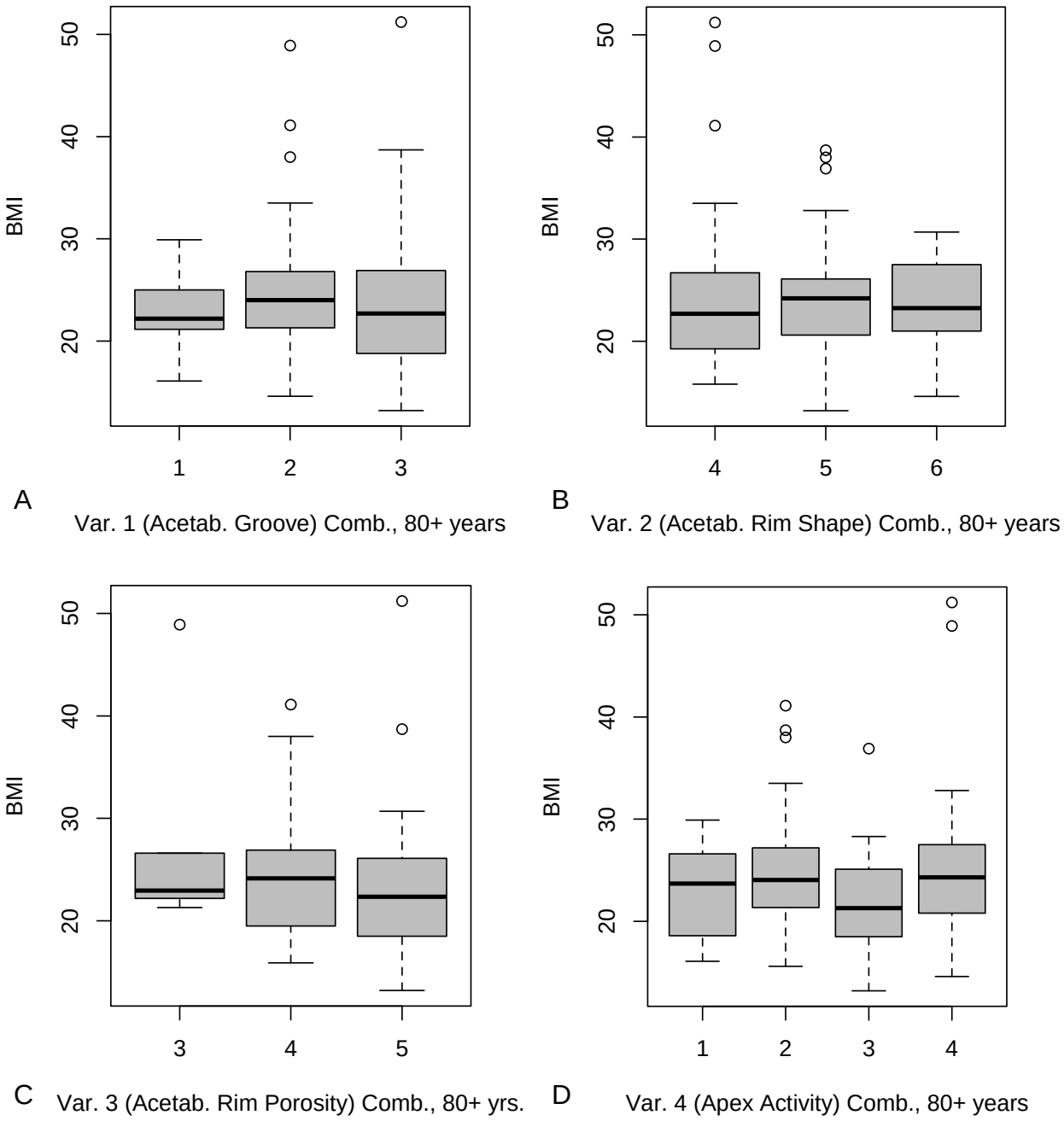
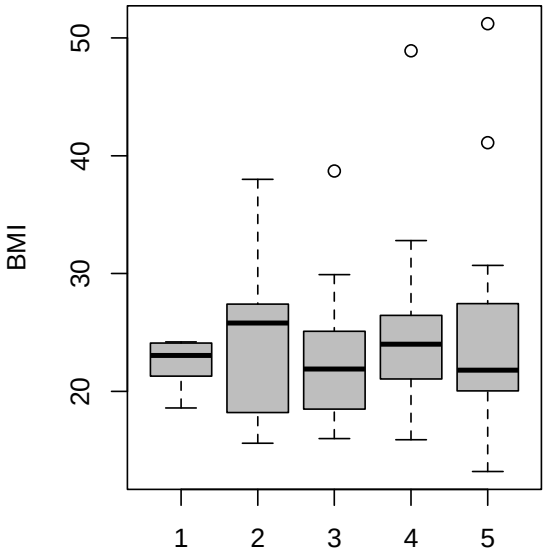
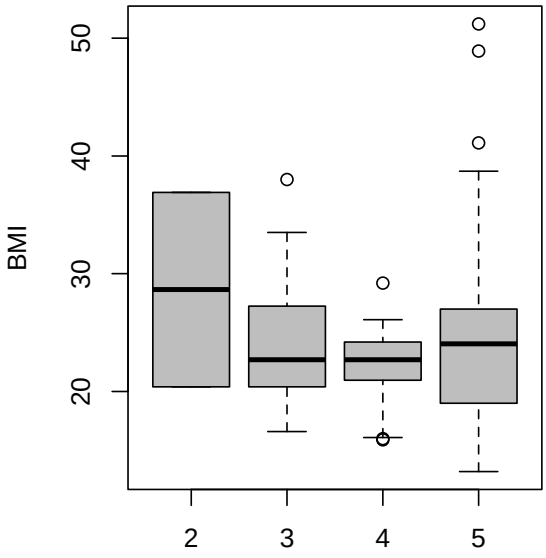


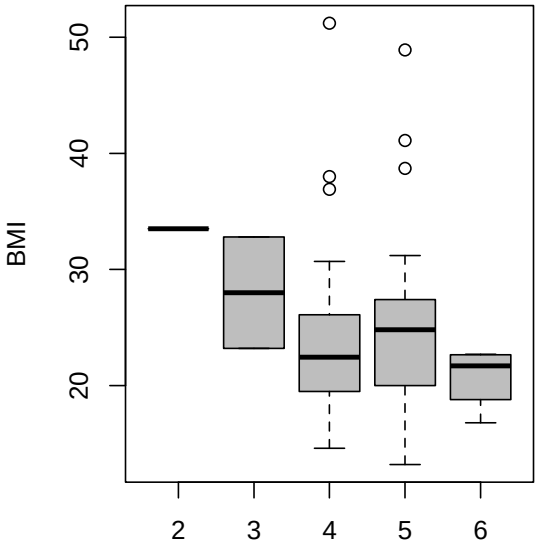
Figure 6-151. Hypothesis 4a: acetabular changes correlate positively with obesity; box-and-whisker plots for BMI value per acetabular variable score in the combined sample (80+ years). A) Variable 1, B) Variable 2, C) Variable 3, D) Variable 4.



A Var. 5 (Acetab. Fossa Crest) Comb., 80+ yrs.



B Var. 6 (Acetab. Fossa Activity) Comb., 80+ yrs.



C Var. 7 (Acetab. Fossa Porosity) Comb., 80+ yr.

Figure 6-152. Hypothesis 4a: acetabular changes correlate positively with obesity; box-and-whisker plots for BMI value per acetabular variable score in the combined sample (80+ years). A) Variable 5, B) Variable 6, C) Variable 7.

Table 6-35. Spearman's rank-order correlations for BMI values and scores for acetabular Variable 1 through Variable 7 in the combined-sex sample.

Group	*rho* (1)	*rho* (2)	*rho* (3)	*rho* (4)	*rho* (5)	*rho* (6)	*rho* (7)
<40 years	0.194	0.238	0.235	0.278	0.406	0.057	0.075
40-49 years	0.115	0.410	0.136	0.175	0.092	-0.172	-0.047
50-59 years	0.117	0.094	-0.012	0.143	-0.019	0.123	-0.037
60-69 years	-0.166	-0.061	-0.243	0.014	-0.031	-0.139	0.065
70-79 years	0.049	0.244	-0.000	0.141	0.233	0.002	-0.003
80+ years	-0.060	0.026	-0.126	0.022	0.034	-0.010	-0.062

*Statistically significant at α=0.001 (Bonferroni correction: α=0.05/42).

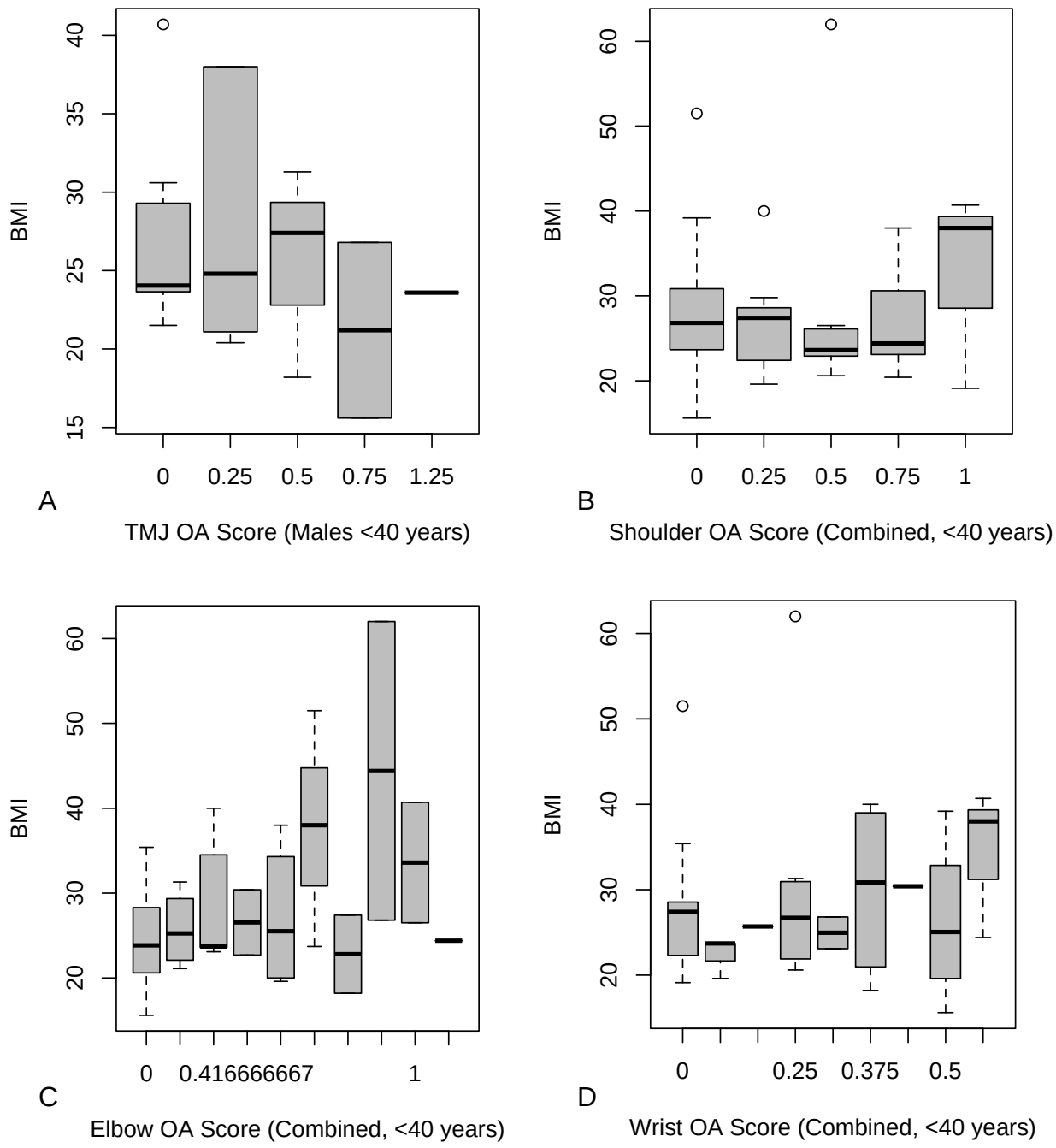


Figure 6-153. Hypothesis 4b: OA correlates positively with obesity; box-and-whisker plots for BMI value per OA score in males and combined sample <40 years. A) Male TMJ OA, B) Combined shoulder OA, C) Combined elbow OA, D) Combined wrist OA.

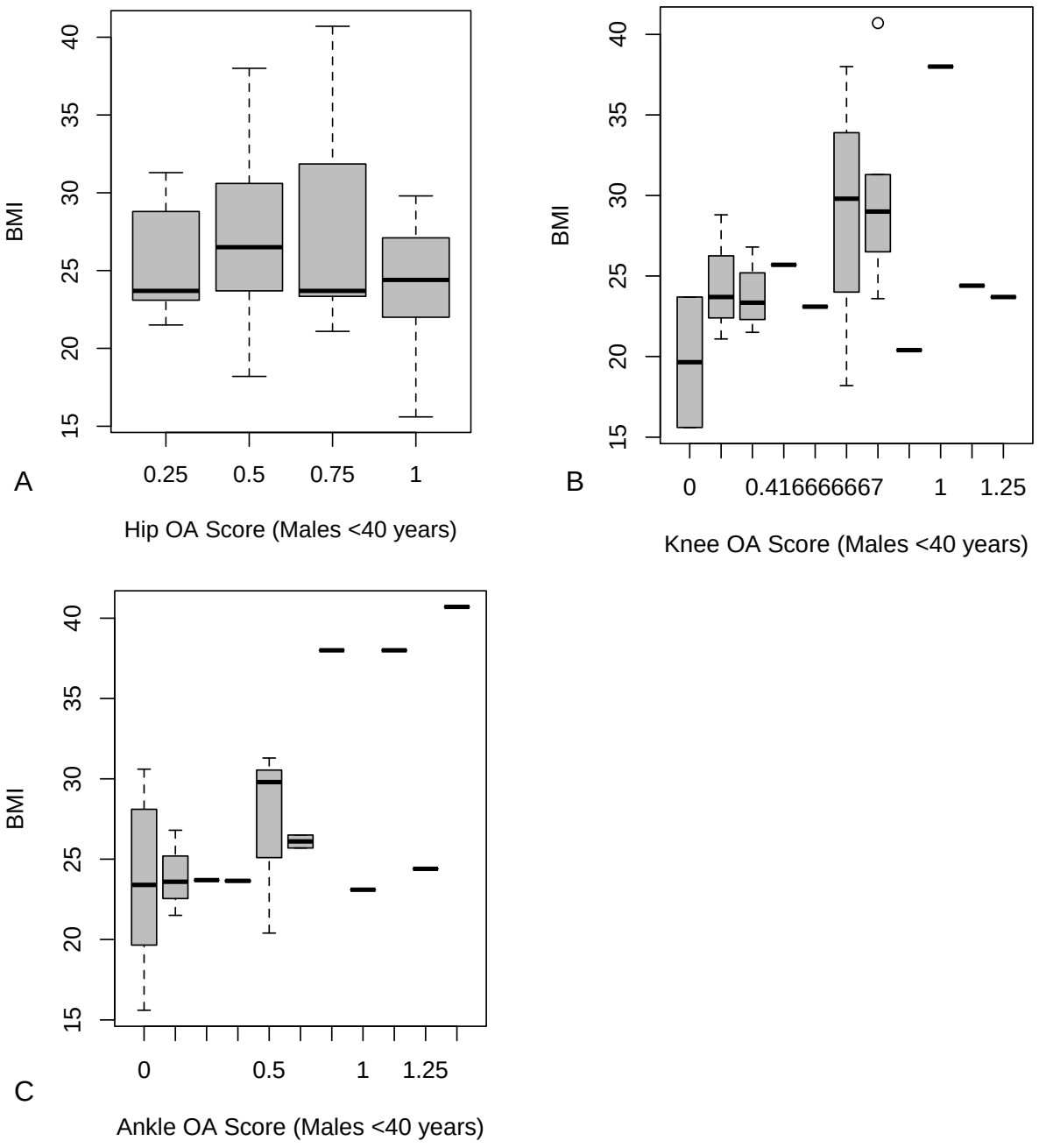


Figure 6-154. Hypothesis 4b: OA correlates positively with obesity; box-and-whisker plots for BMI value per OA score in males and combined sample <40 years. A) Male hip OA, B) Male knee OA, C) Male ankle OA.

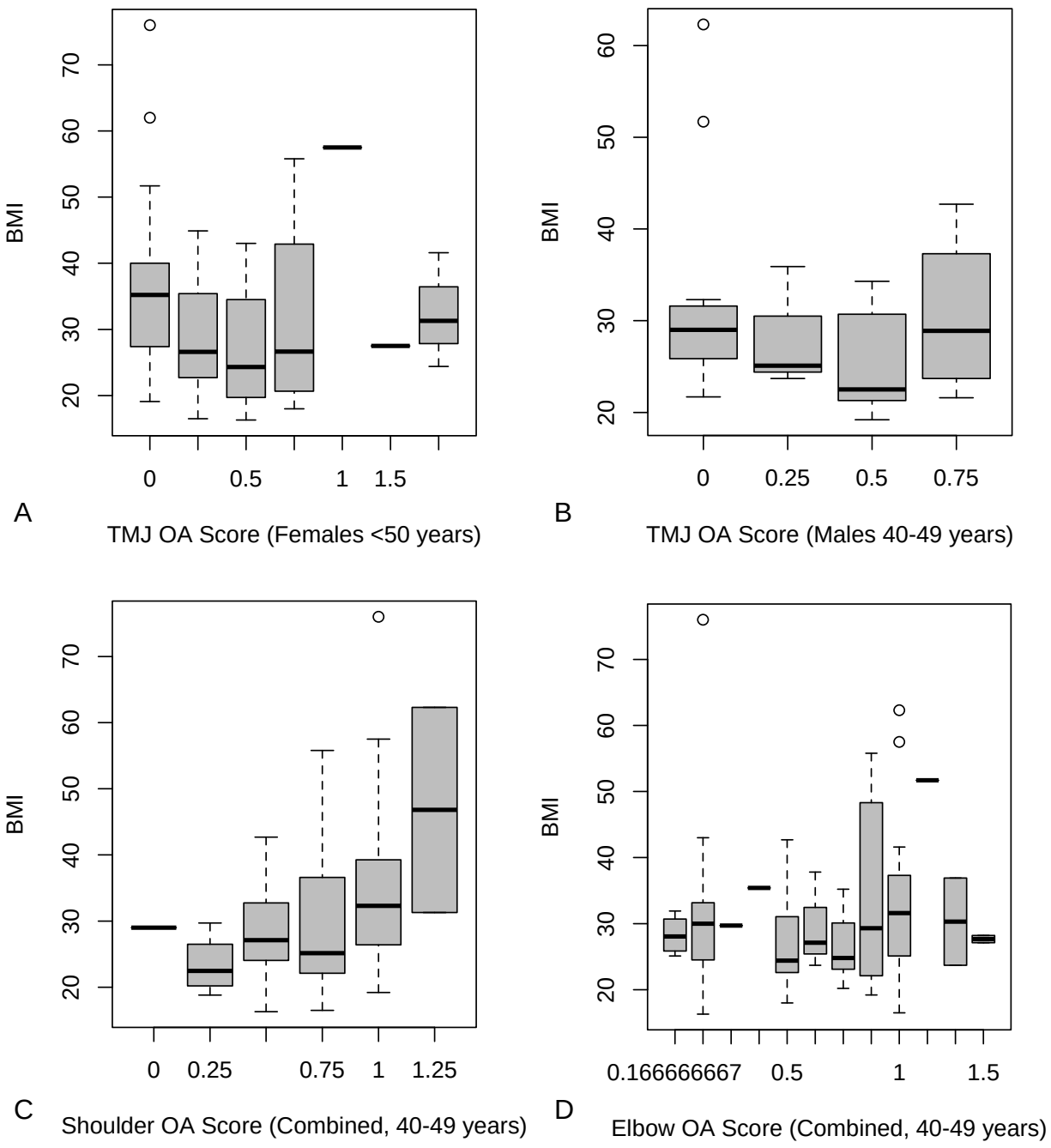


Figure 6-155. Hypothesis 4b: OA correlates positively with obesity; box-and-whisker plots for BMI value per OA score in females <50 years and males and combined sample 40-49 years. A) Female TMJ OA, B) Male TMJ OA, C) Combined shoulder OA, D) Combined elbow OA.

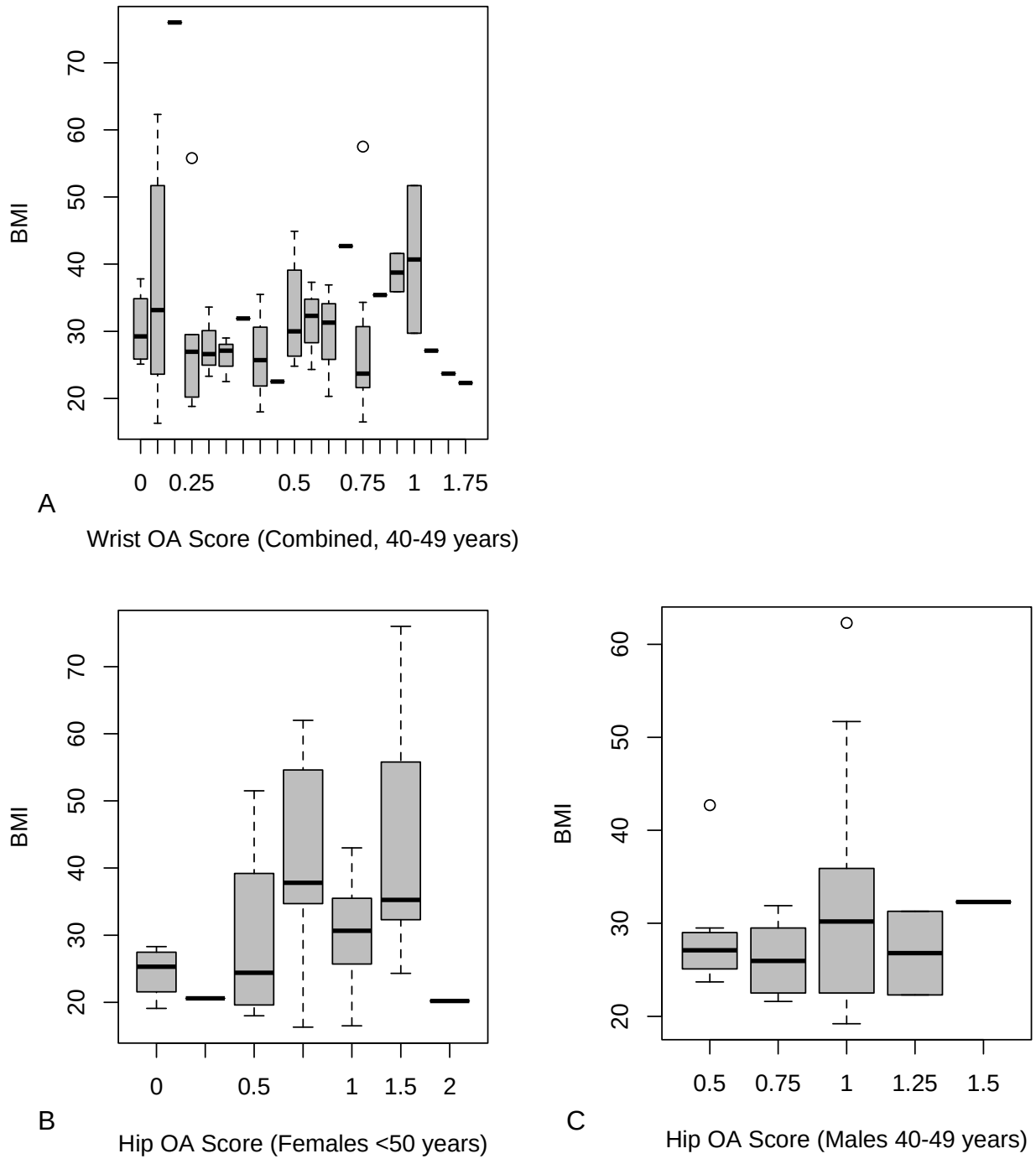


Figure 6-156. Hypothesis 4b: OA correlates positively with obesity; box-and-whisker plots for BMI value per OA score in females <50 years and males and combined sample 40-49 years. A) Combined wrist OA, B) Female hip OA, C) Male hip OA.

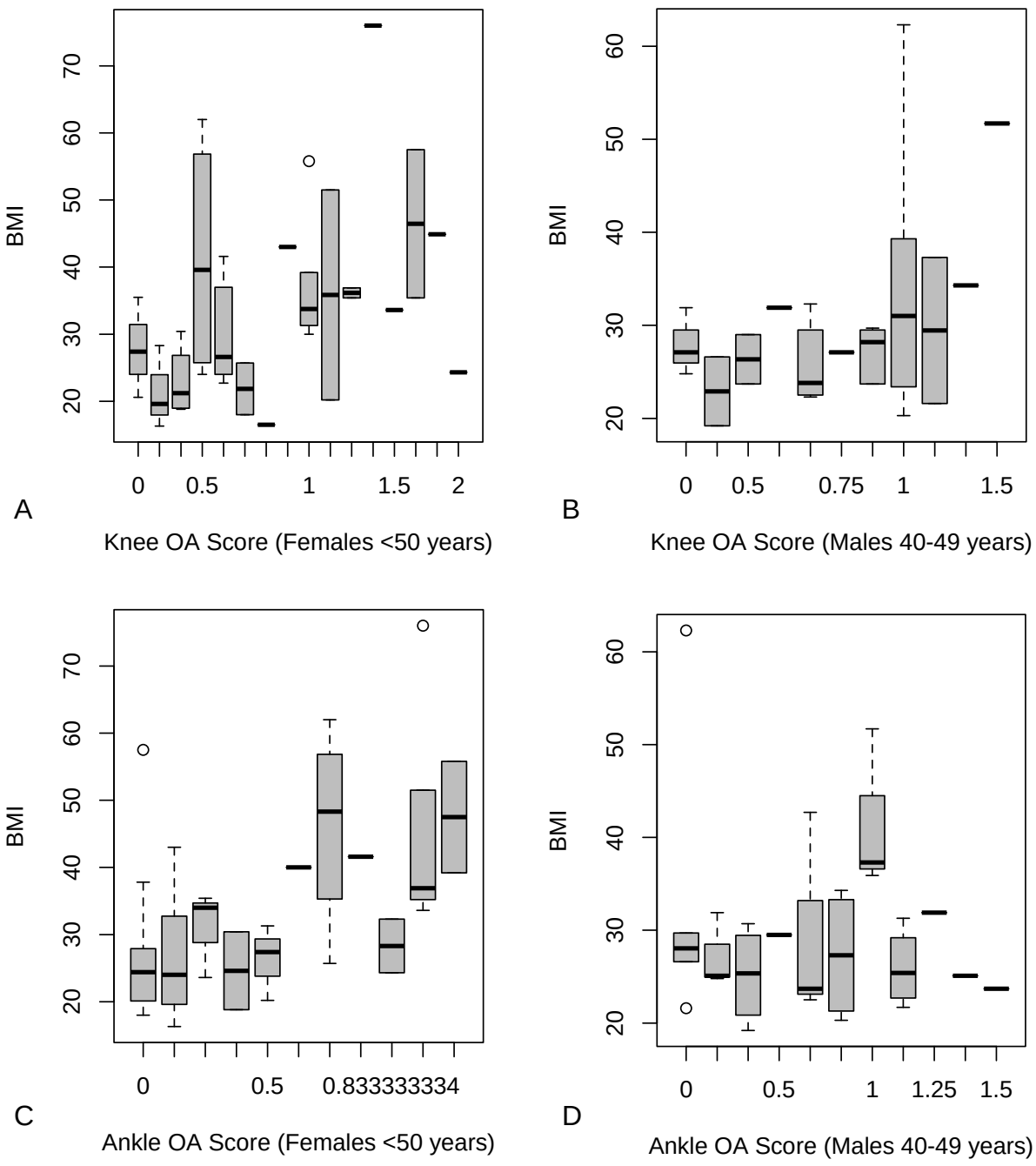


Figure 6-157. Hypothesis 4b: OA correlates positively with obesity; box-and-whisker plots for BMI value per OA score in females <50 and males 40-49 years. A) Female knee OA, B) Male knee OA, C) Female ankle OA, D) Male ankle OA.

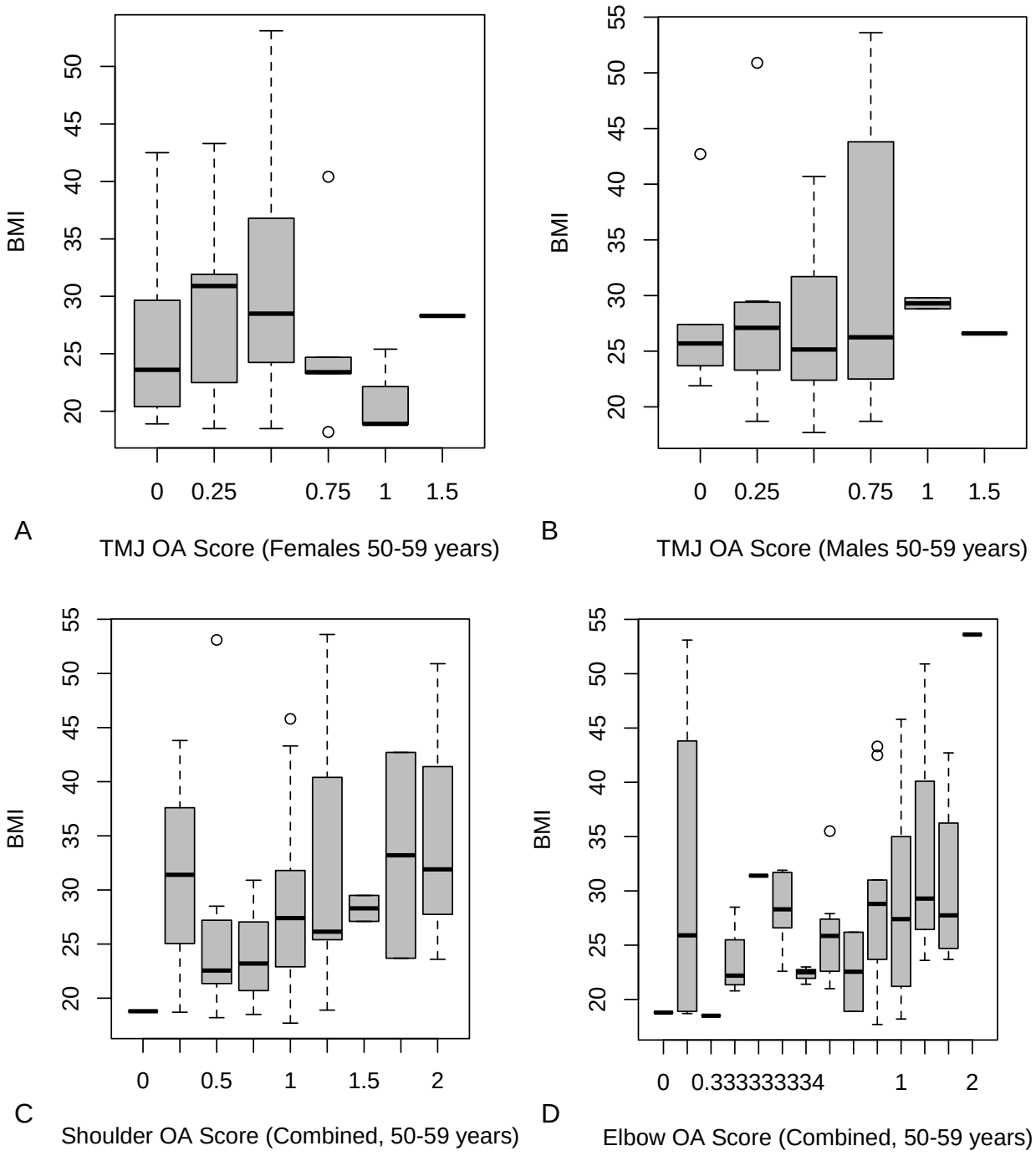


Figure 6-158. Hypothesis 4b: OA correlates positively with obesity; box-and-whisker plots for BMI value per OA score in females, males, and combined sample 50-59 years. A) Female TMJ OA, B) Male TMJ OA, C) Combined shoulder OA, D) Combined elbow OA.

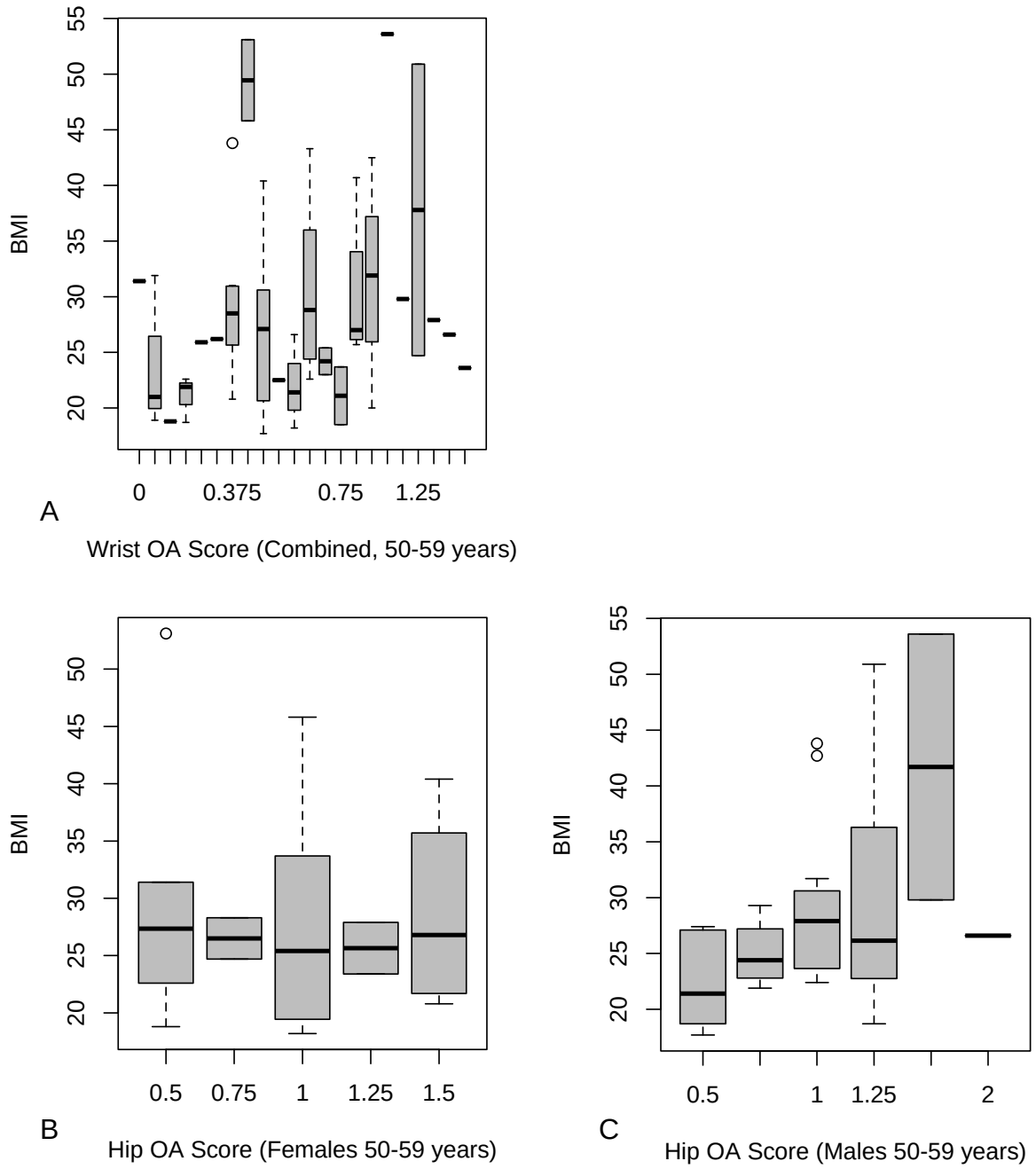


Figure 6-159. Hypothesis 4b: OA correlates positively with obesity; box-and-whisker plots for BMI value per OA score in females, males, and combined sample 50-59 years. A) Combined wrist OA, B) Female hip OA, C) Male hip OA.

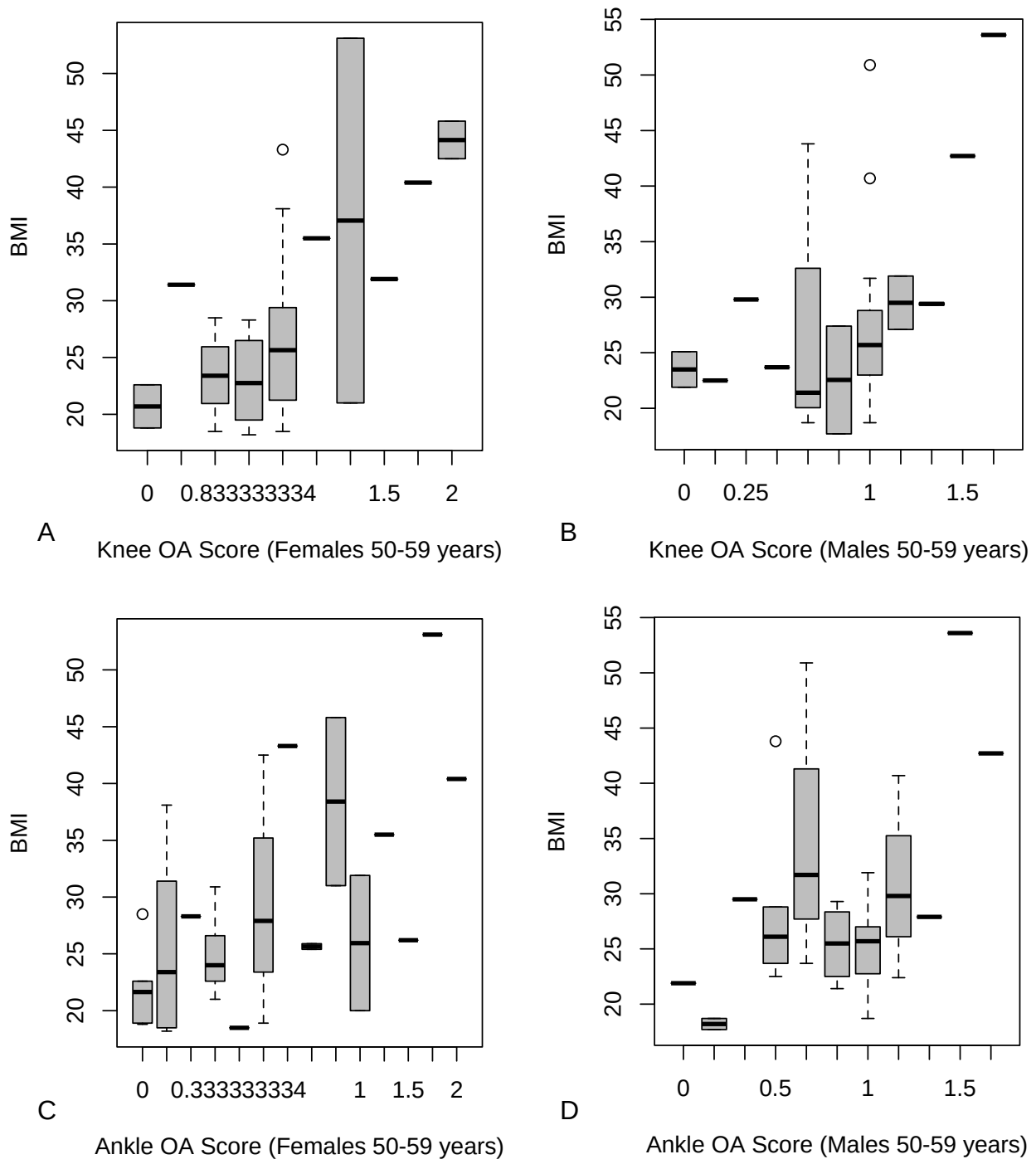


Figure 6-160. Hypothesis 4b: OA correlates positively with obesity; box-and-whisker plots for BMI value per OA score in females and males 50-59 years. A) Female knee OA, B) Male knee OA, C) Female ankle OA, D) Male ankle OA.

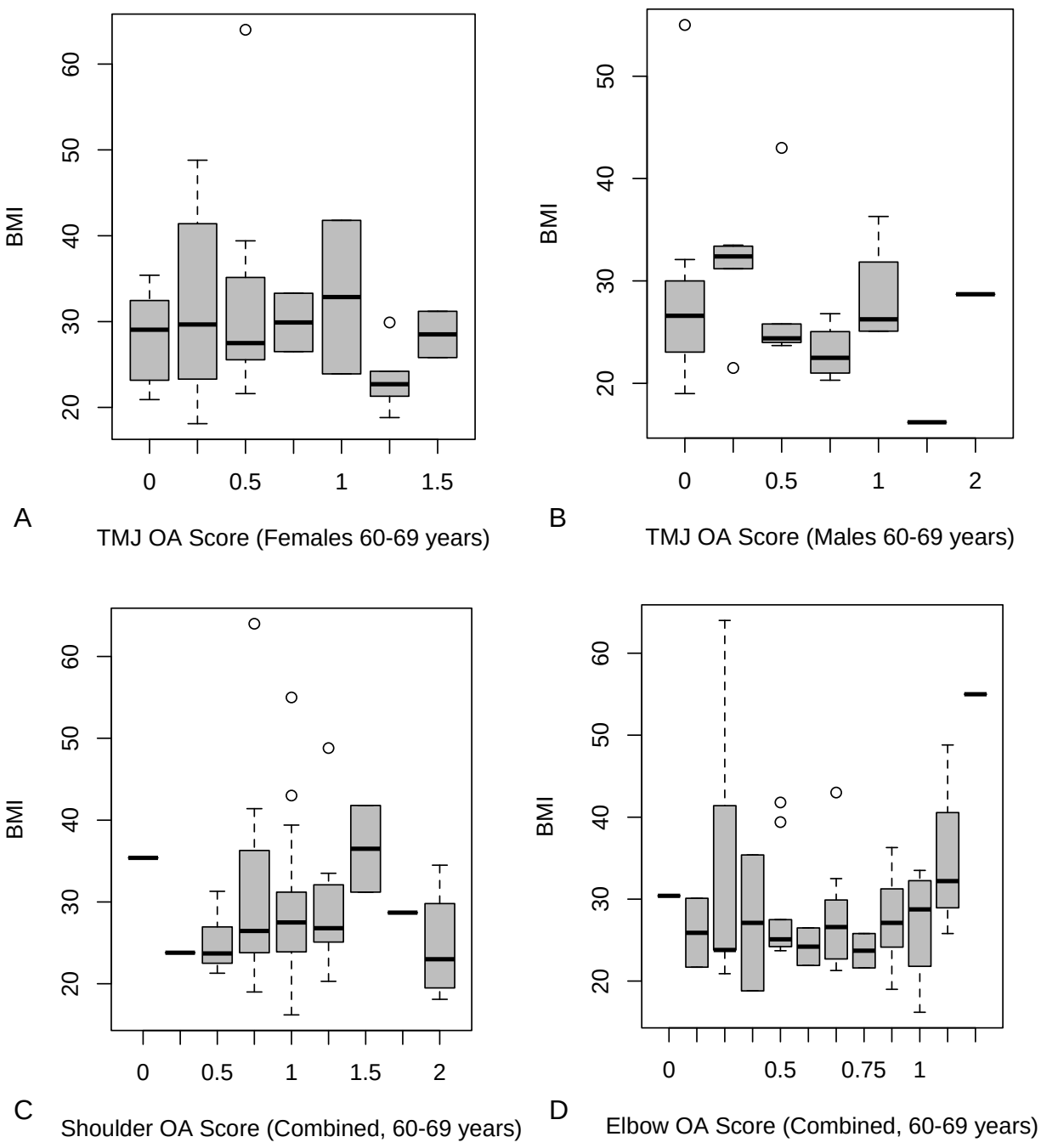


Figure 6-161. Hypothesis 4b: OA correlates positively with obesity; box-and-whisker plots for BMI value per OA score in females, males, and combined sample 60-69 years. A) Female TMJ OA, B) Male TMJ OA, C) Combined shoulder OA, D) Combined elbow OA.

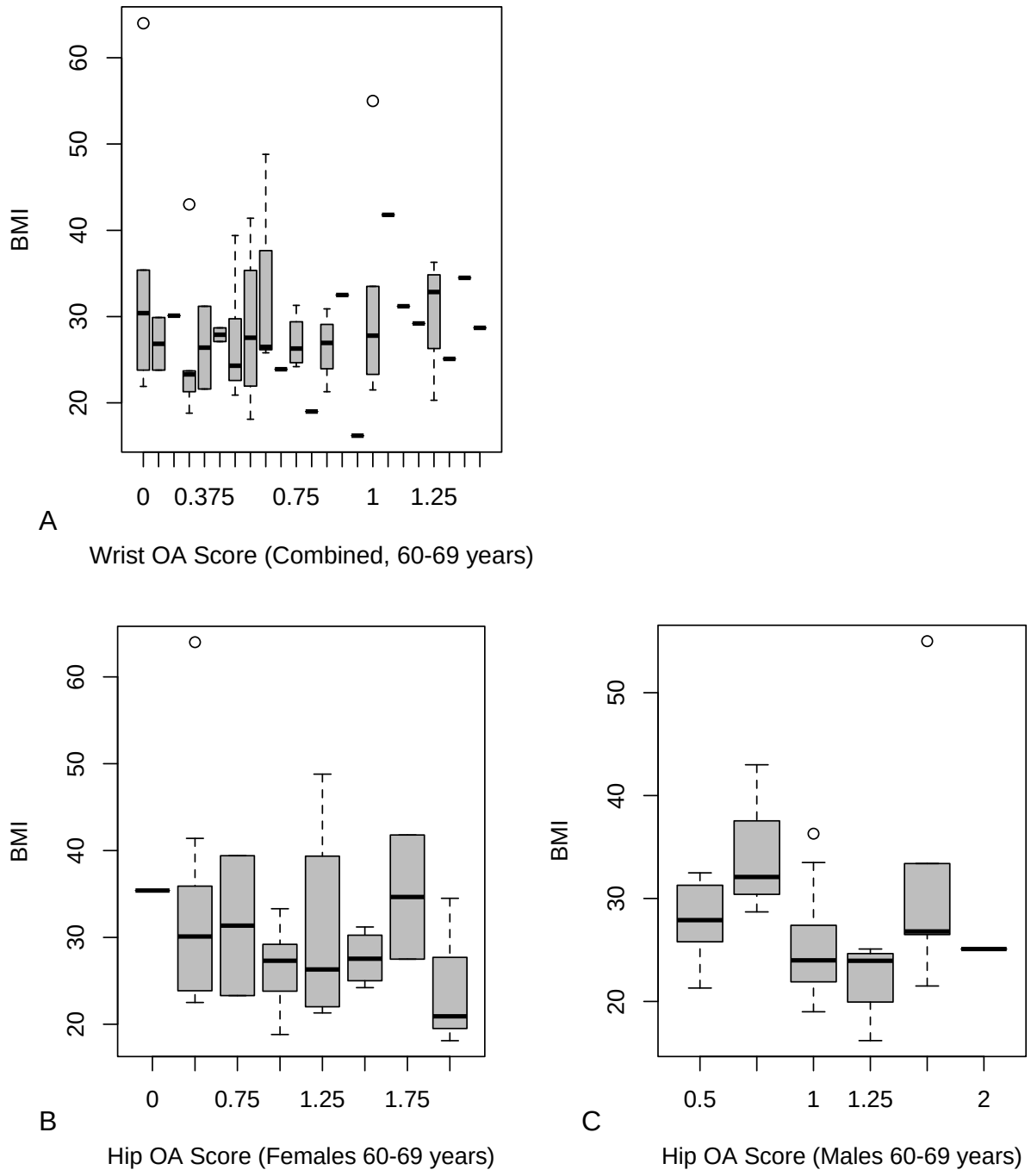


Figure 6-162. Hypothesis 4b: OA correlates positively with obesity; box-and-whisker plots for BMI value per OA score in females, males, and combined sample 60-69 years. A) Combined wrist OA, B) Female hip OA, C) Male hip OA.

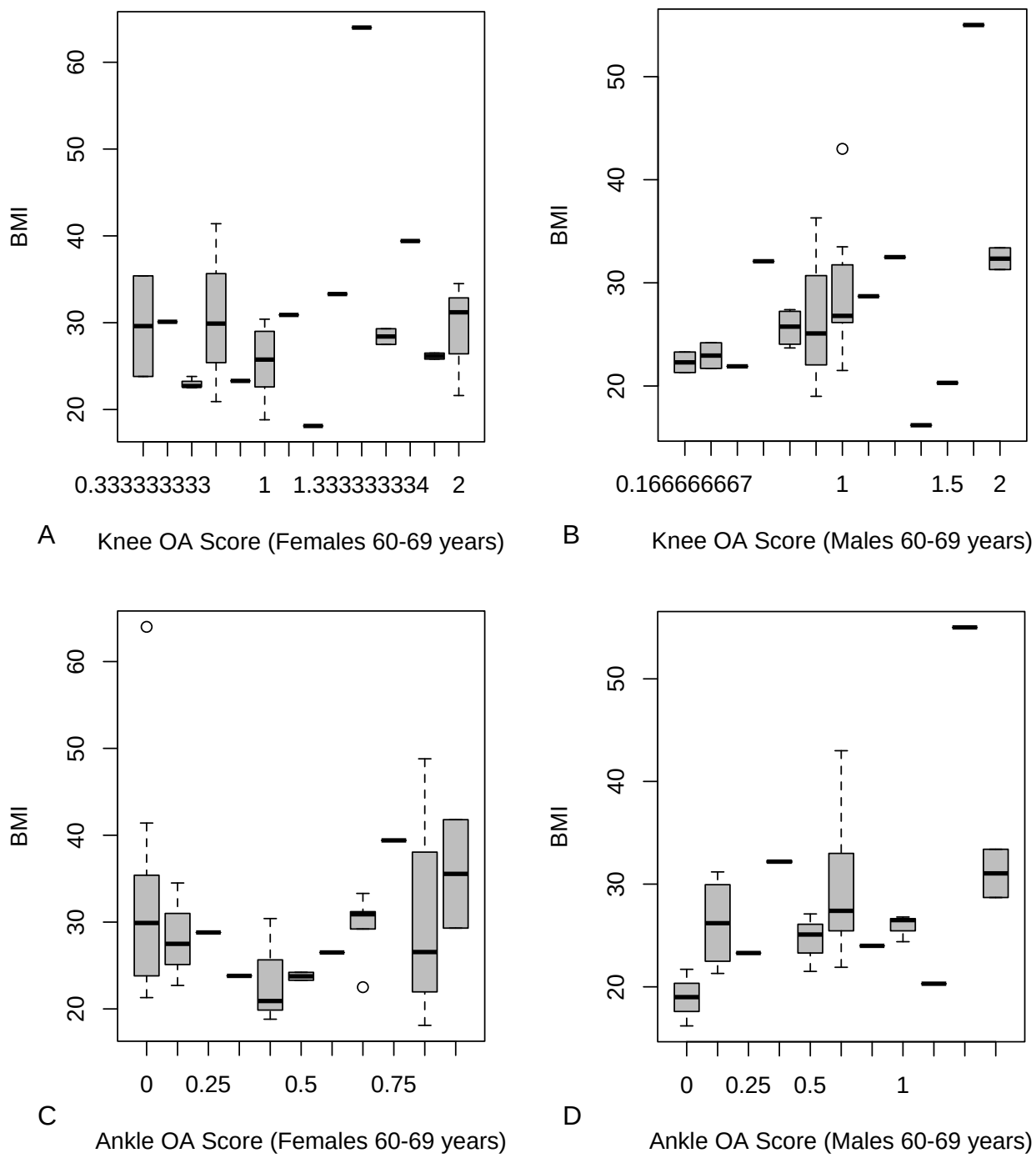


Figure 6-163. Hypothesis 4b: OA correlates positively with obesity; box-and-whisker plots for BMI value per OA score in females and males 60-69 years. A) Female knee OA, B) Male knee OA, C) Female ankle OA, D) Male ankle OA.

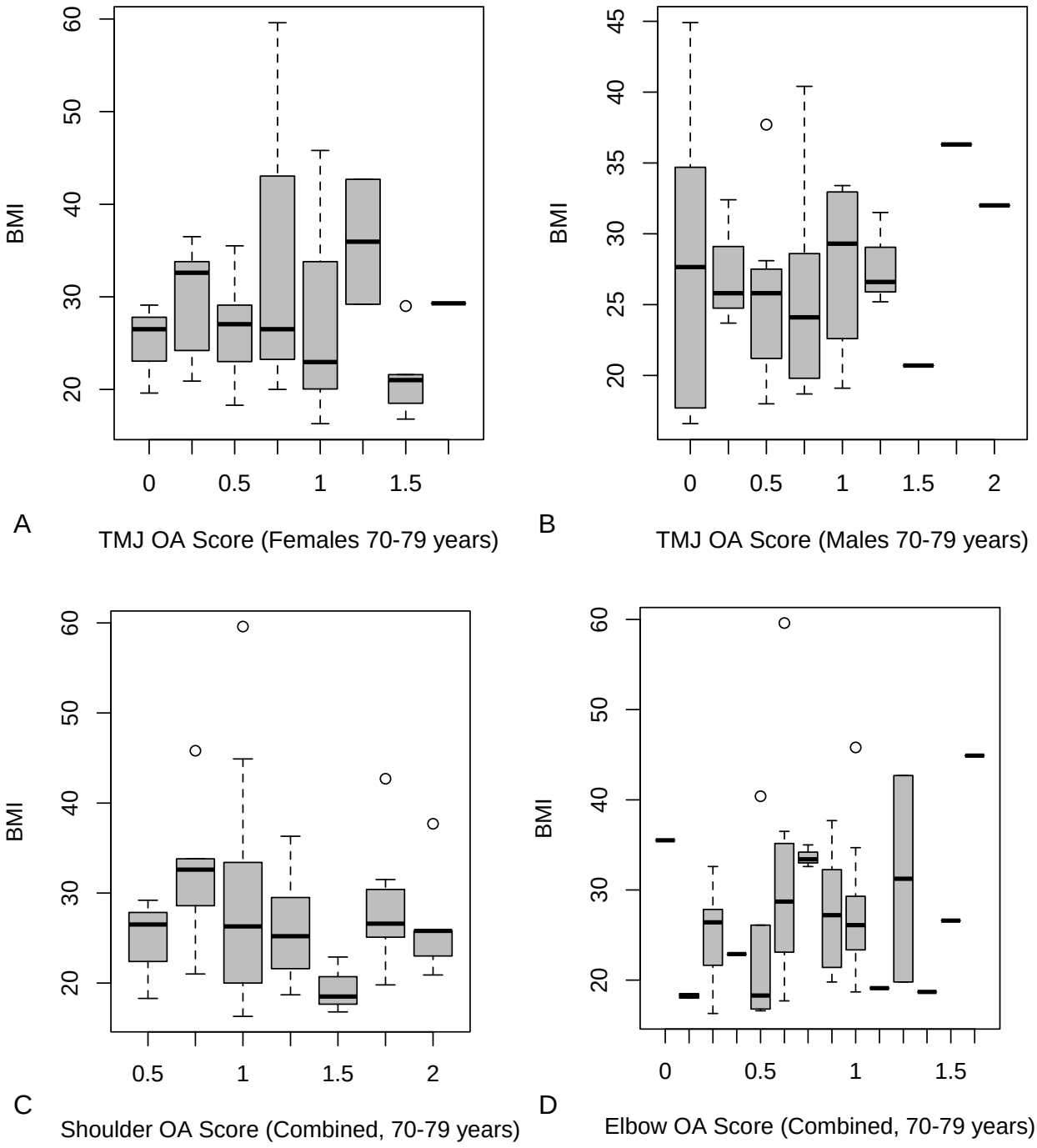


Figure 6-164. Hypothesis 4b: OA correlates positively with obesity; box-and-whisker plots for BMI value per OA score in females, males, and combined sample 70-79 years. A) Female TMJ OA, B) Male TMJ OA, C) Combined shoulder OA, D) Combined elbow OA.

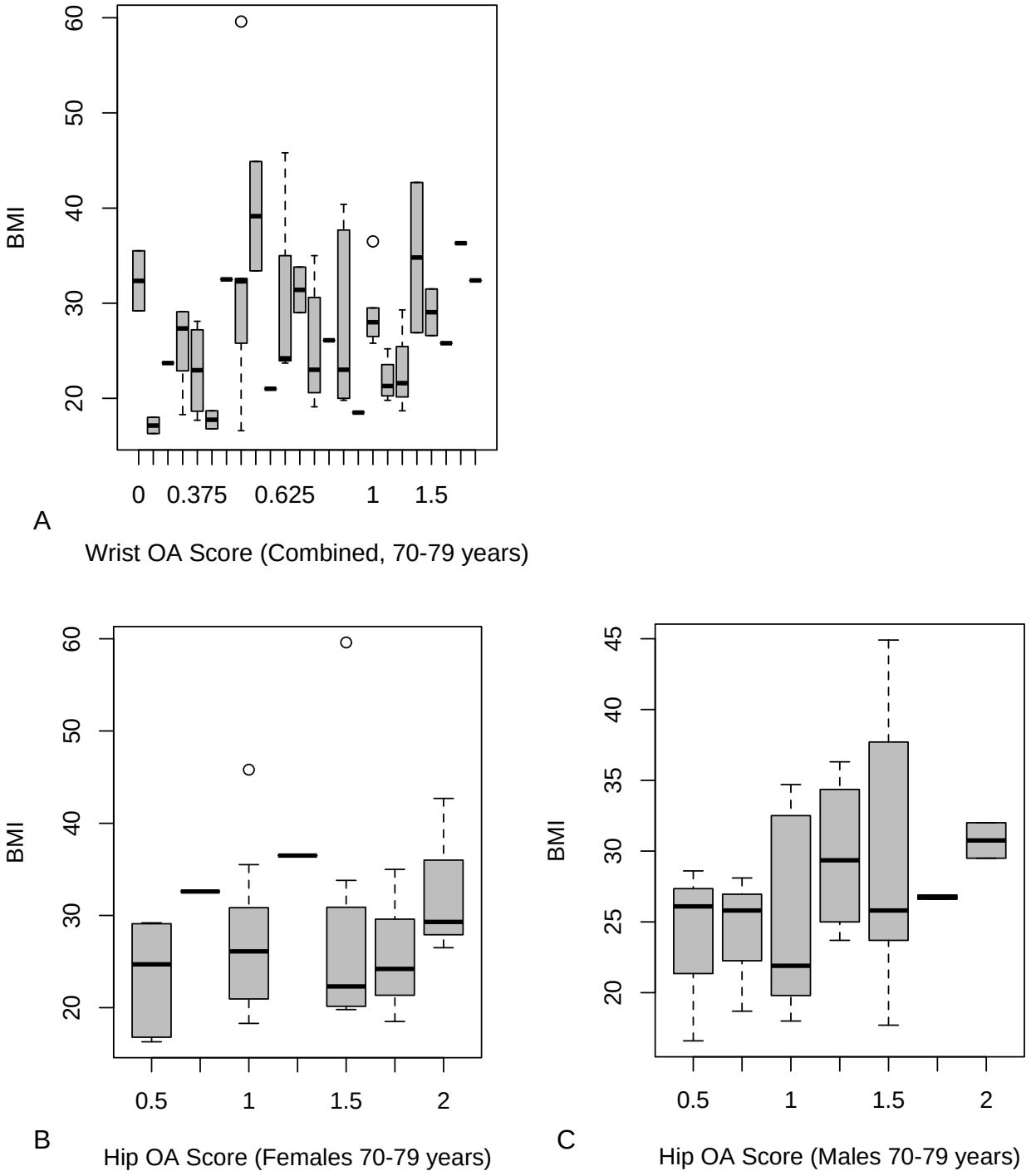


Figure 6-165. Hypothesis 4b: OA correlates positively with obesity; box-and-whisker plots for BMI value per OA score in females, males, and combined sample 70-79 years. A) Combined wrist OA, B) Female hip OA, C) Male hip OA.

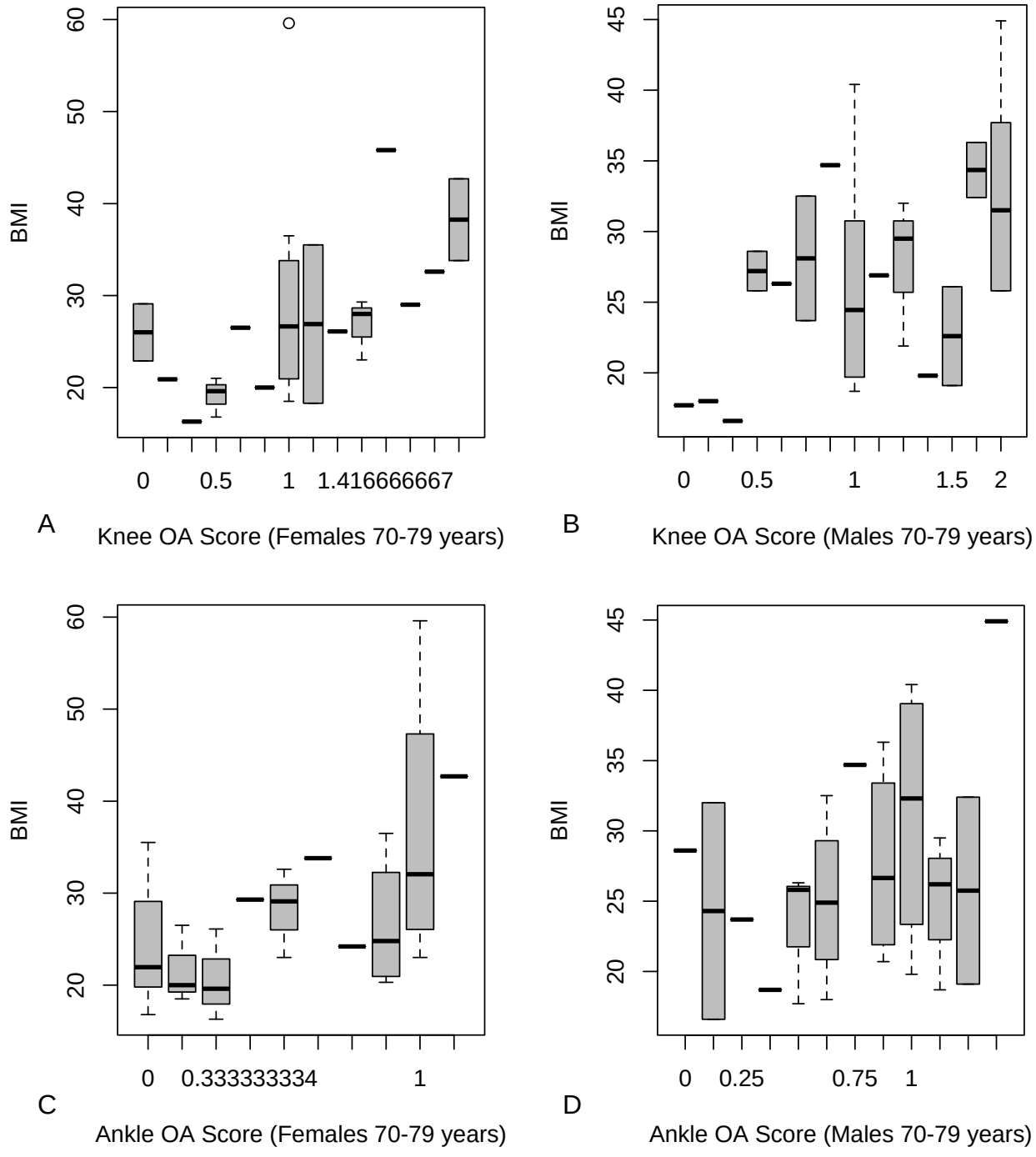


Figure 6-166. Hypothesis 4b: OA correlates positively with obesity; box-and-whisker plots for BMI value per OA score in females and males 70-79 years. A) Female knee OA, B) Male knee OA, C) Female ankle OA, D) Male ankle OA.

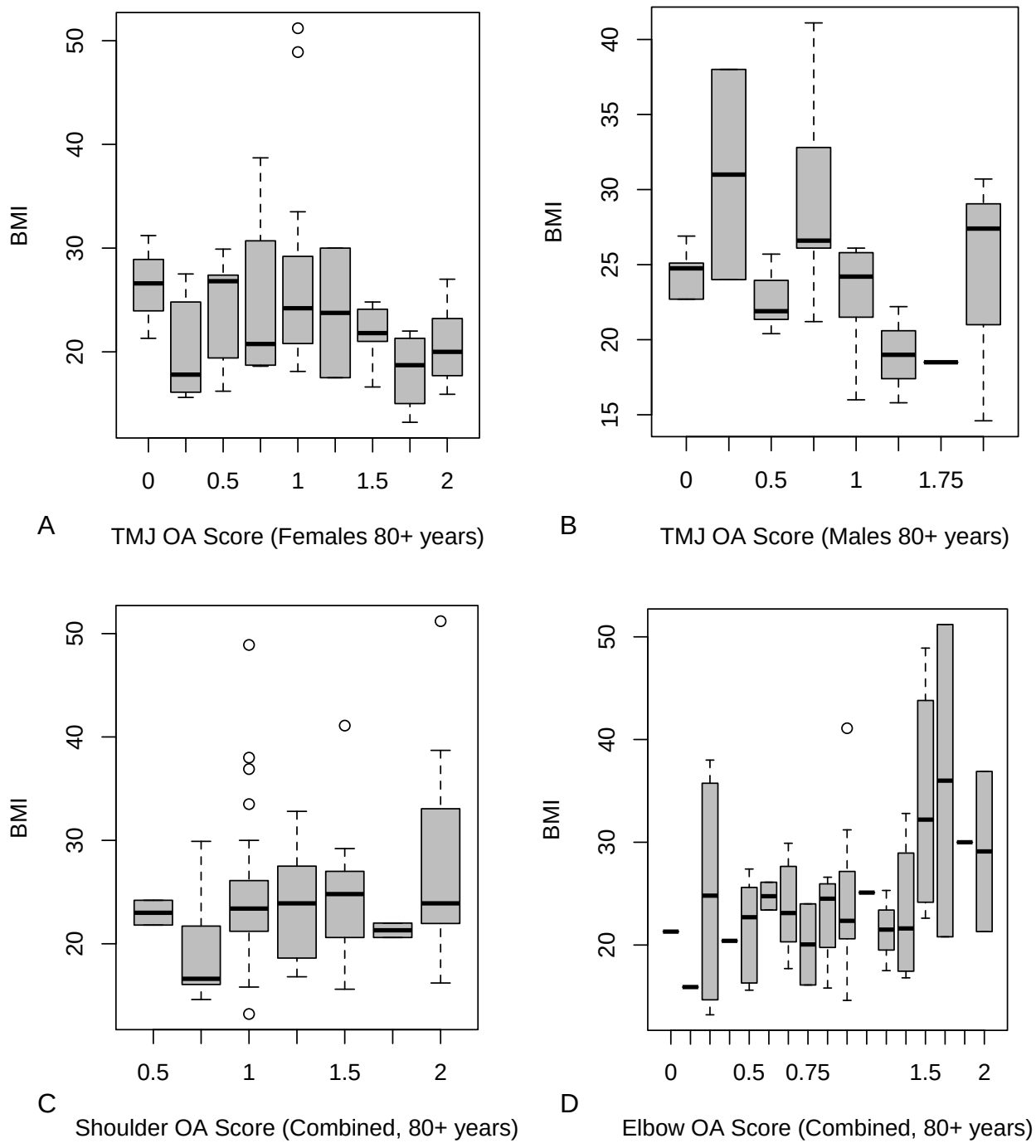


Figure 6-167. Hypothesis 4b: OA correlates positively with obesity; box-and-whisker plots for BMI value per OA score in females, males, and combined sample 80+ years. A) Female TMJ OA, B) Male TMJ OA, C) Combined shoulder OA, D) Combined elbow OA.

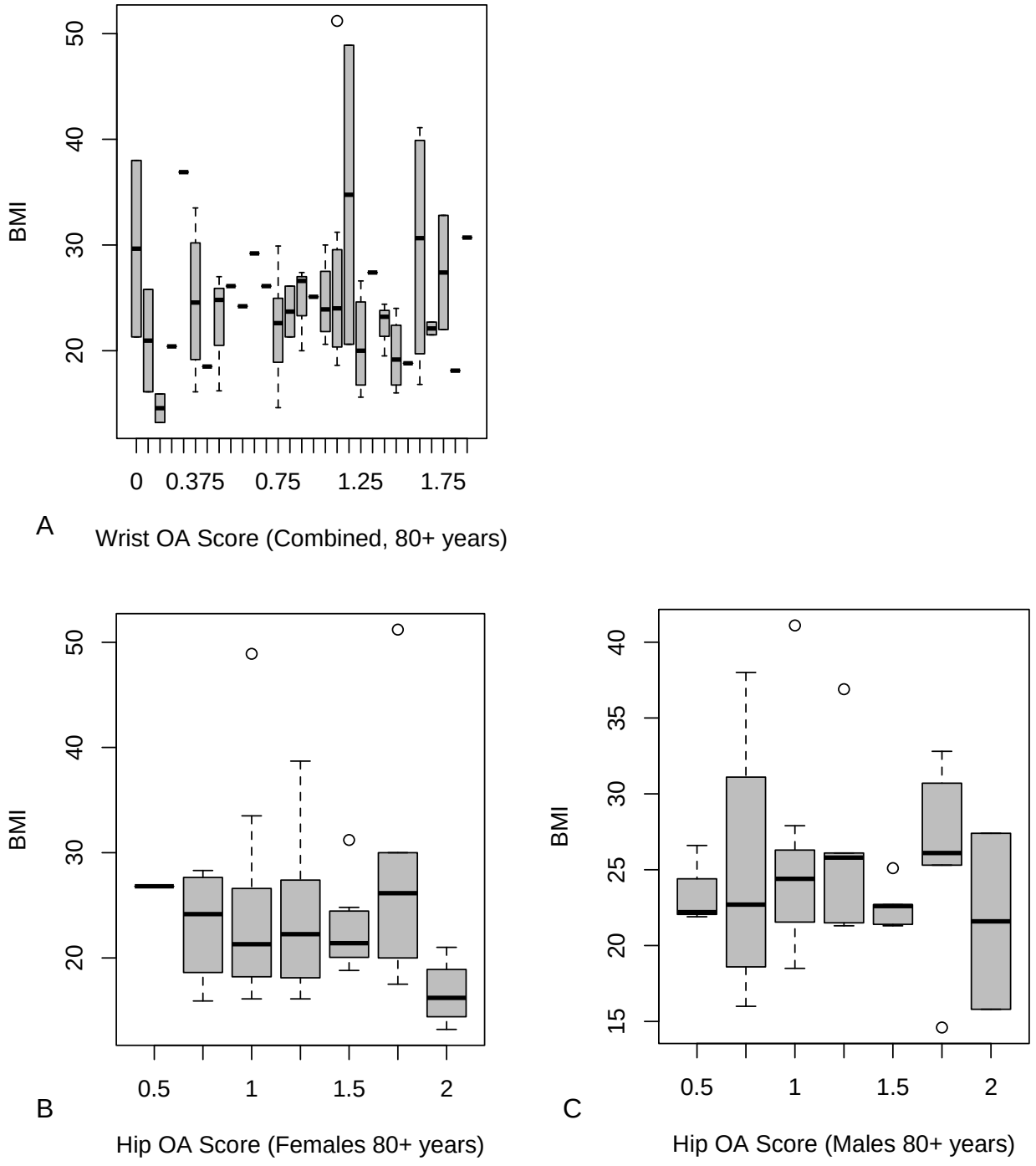


Figure 6-168. Hypothesis 4b: OA correlates positively with obesity; box-and-whisker plots for BMI value per OA score in females, males, and combined sample 80+ years. A) Combined wrist OA, B) Female hip OA, C) Male hip OA.

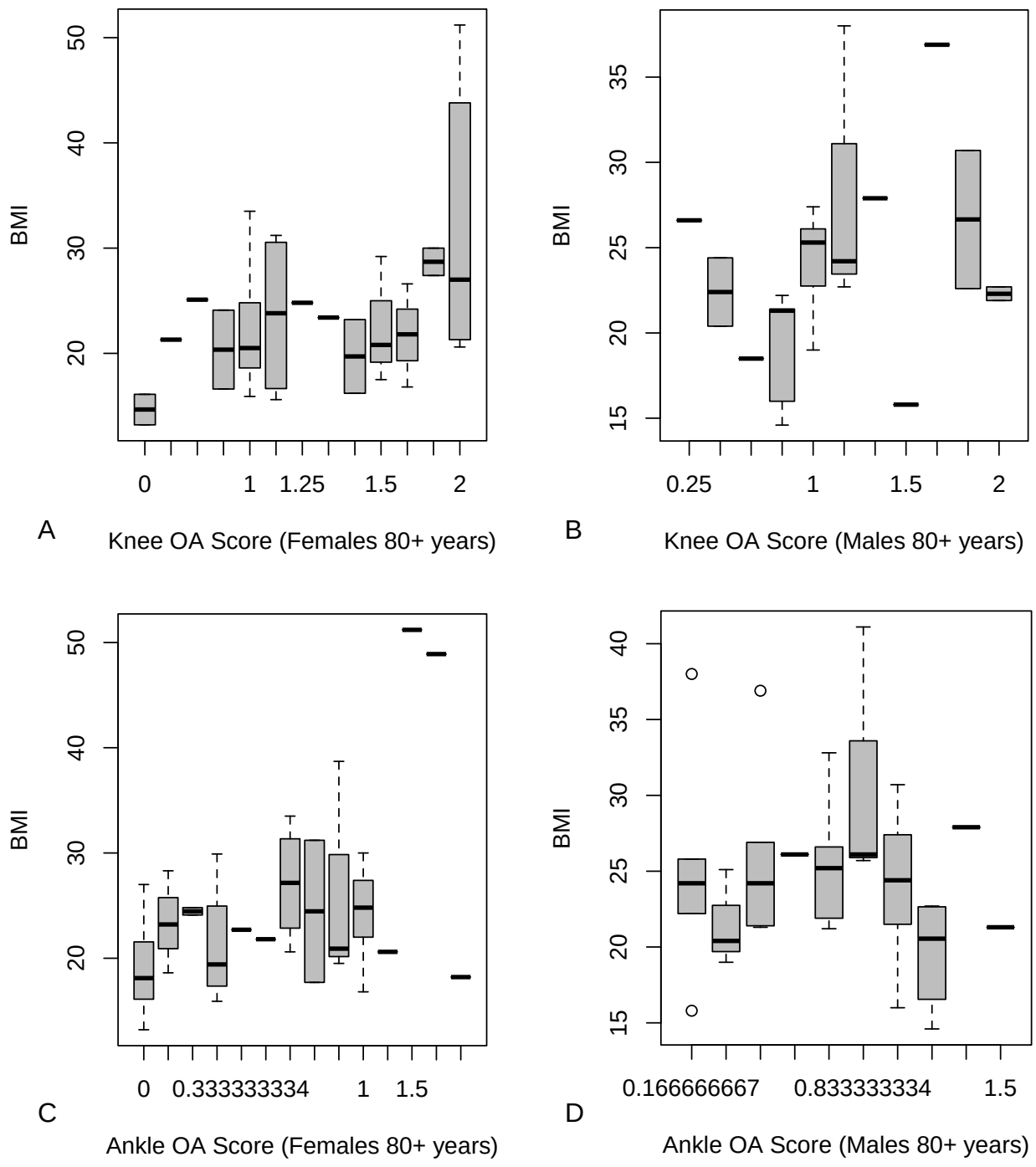


Figure 6-169. Hypothesis 4b: OA correlates positively with obesity; box-and-whisker plots for BMI value per OA score in females and males 80+ years. A) Female knee OA, B) Male knee OA, C) Female ankle OA, D) Male ankle OA.

Table 6-36. Spearman's rank-order correlations for BMI values and scores for OA in the various joints in the female, male, and combined-sex samples.

Group	*rho* (TMJ)	*rho* (Shoulder)	*rho* (Elbow)	*rho* (Wrist)	*rho* (Hip)	*rho* (Knee)	*rho* (Ankle)
Females <50 years	-0.154	NA	NA	NA	0.242	0.430	0.517*
Females 50-59 years	-0.075	NA	NA	NA	-0.047	0.498	0.508
Females 60-69 years	-0.100	NA	NA	NA	-0.170	0.206	0.091
Females 70-79 years	-0.127	NA	NA	NA	0.168	0.515	0.441
Females 80+ years	-0.143	NA	NA	NA	-0.112	0.363	0.375
Combined <40 years	NA	-0.015	0.285	0.159	NA	NA	NA
Combined 40-49 years	NA	0.381	0.112	-0.042	NA	NA	NA
Combined 50-59 years	NA	0.305	0.252	0.151	NA	NA	NA
Combined 60-69 years	NA	0.069	0.119	0.145	NA	NA	NA
Combined 70-79 years	NA	-0.111	0.101	0.125	NA	NA	NA
Combined 80+ years	NA	0.161	0.207	-0.019	NA	NA	NA
Males <40 years	-0.167	NA	NA	NA	-0.096	0.365	0.426
Males 40-49 years	-0.147	NA	NA	NA	0.119	0.308	0.028
Males 50-59 years	0.093	NA	NA	NA	0.375	0.473	0.336
Males 60-69 years	-0.124	NA	NA	NA	-0.171	0.377	0.369
Males 70-79 years	0.118	NA	NA	NA	0.288	0.390	0.276
Males 80+ years	-0.158	NA	NA	NA	0.082	0.323	-0.031

*Statistically significant at α=0.003 for females and the combined-sex sample (Bonferroni correction: α=0.05/20 and α=0.05/18, respectively); α=0.002 for males (Bonferroni correction: α=0.05/24).
NA indicates that tests were not conducted (the combined-sex sample was only used in testing when no sex differences were detected in OA distributions).

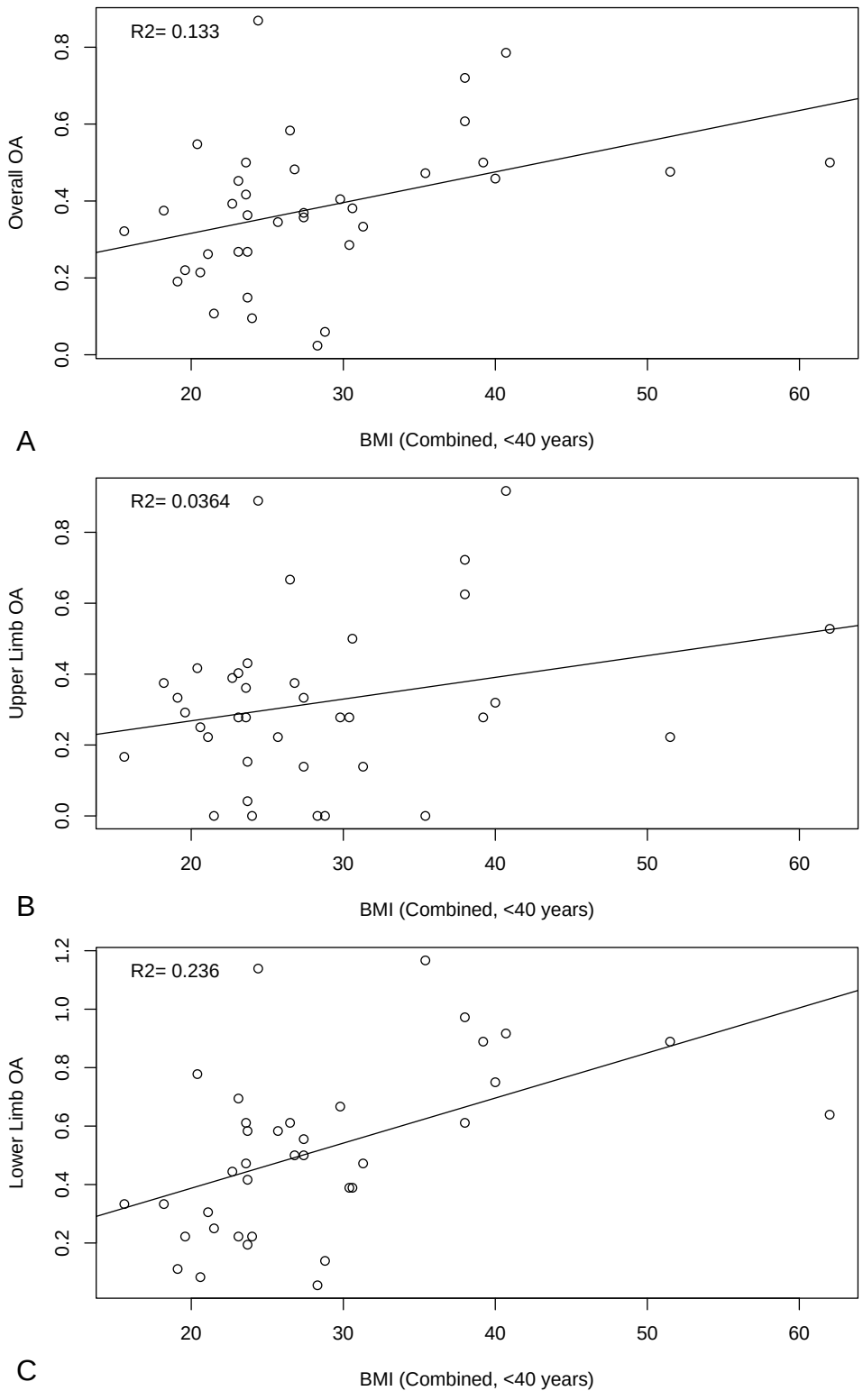


Figure 6-170. Hypothesis 4b: OA correlates positively with obesity; scatterplots for summary OA and BMI values in combined-sex individuals <40 years. A) Overall OA, B) Upper limb OA, C) Lower limb OA. Black line indicates linear regression model.

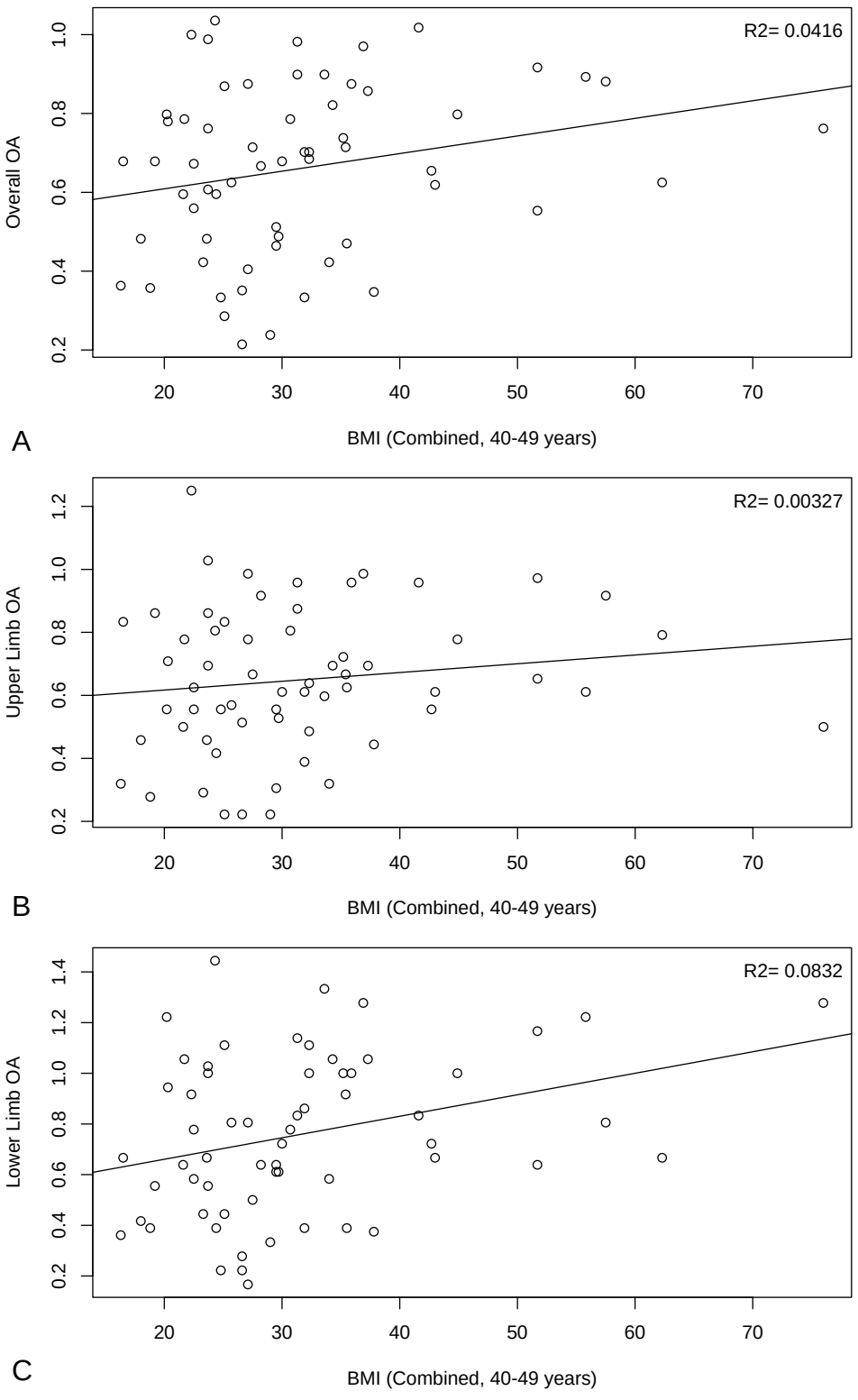


Figure 6-171. Hypothesis 4b: OA correlates positively with obesity; scatterplots for summary OA and BMI values in combined-sex individuals 40-49 years. A) Overall OA, B) Upper limb OA, C) Lower limb OA. Black line indicates linear regression model.

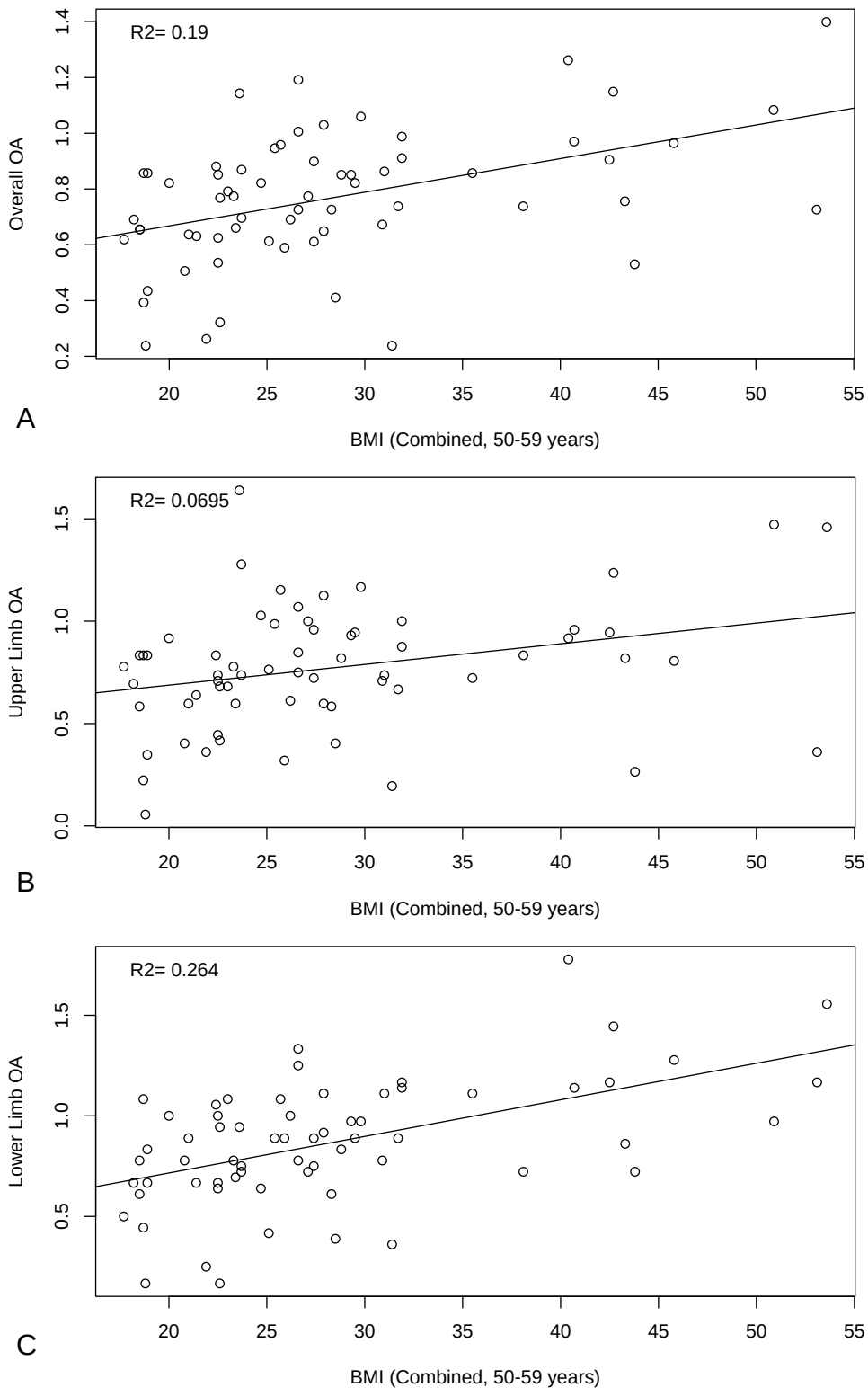


Figure 6-172. Hypothesis 4b: OA correlates positively with obesity; scatterplots for summary OA and BMI values in combined-sex individuals 50-59 years. A) Overall OA, B) Upper limb OA, C) Lower limb OA. Black line indicates linear regression model.

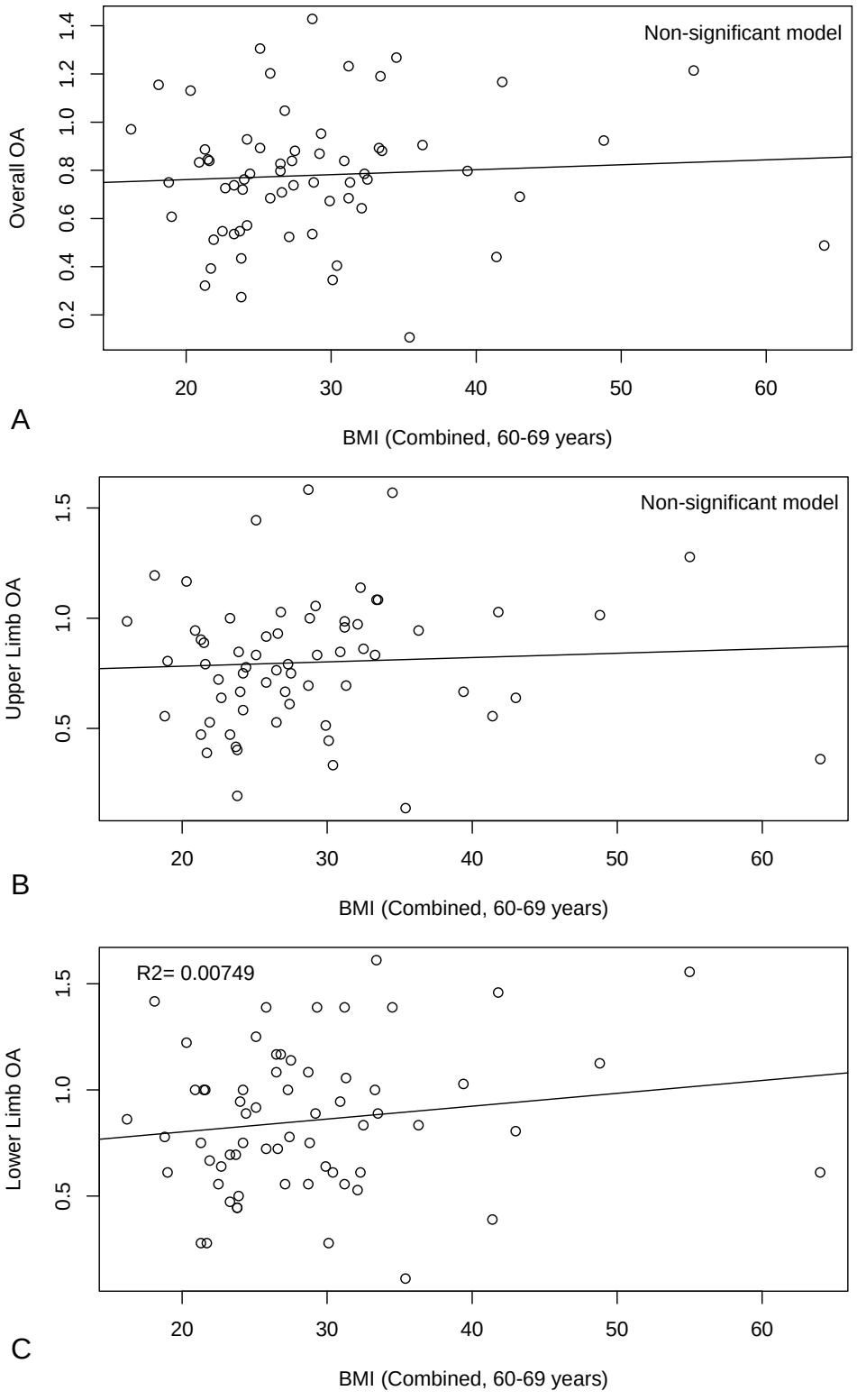


Figure 6-173. Hypothesis 4b: OA correlates positively with obesity; scatterplots for summary OA and BMI values in combined-sex individuals 60-69 years. A) Overall OA, B) Upper limb OA, C) Lower limb OA. Black line indicates linear regression model.

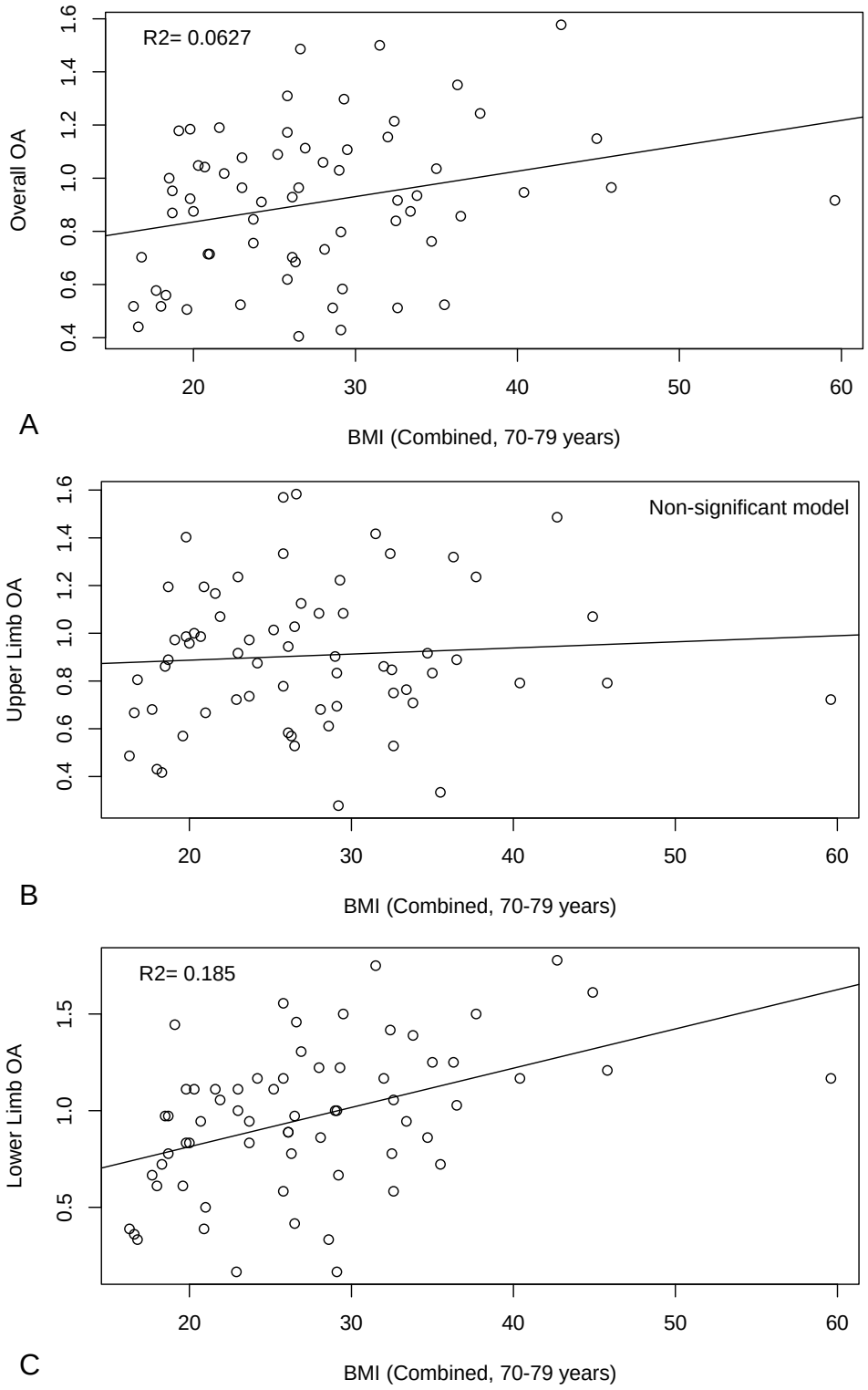


Figure 6-174. Hypothesis 4b: OA correlates positively with obesity; scatterplots for summary OA and BMI values in combined-sex individuals 70-79 years. A) Overall OA, B) Upper limb OA, C) Lower limb OA. Black line indicates linear regression model.

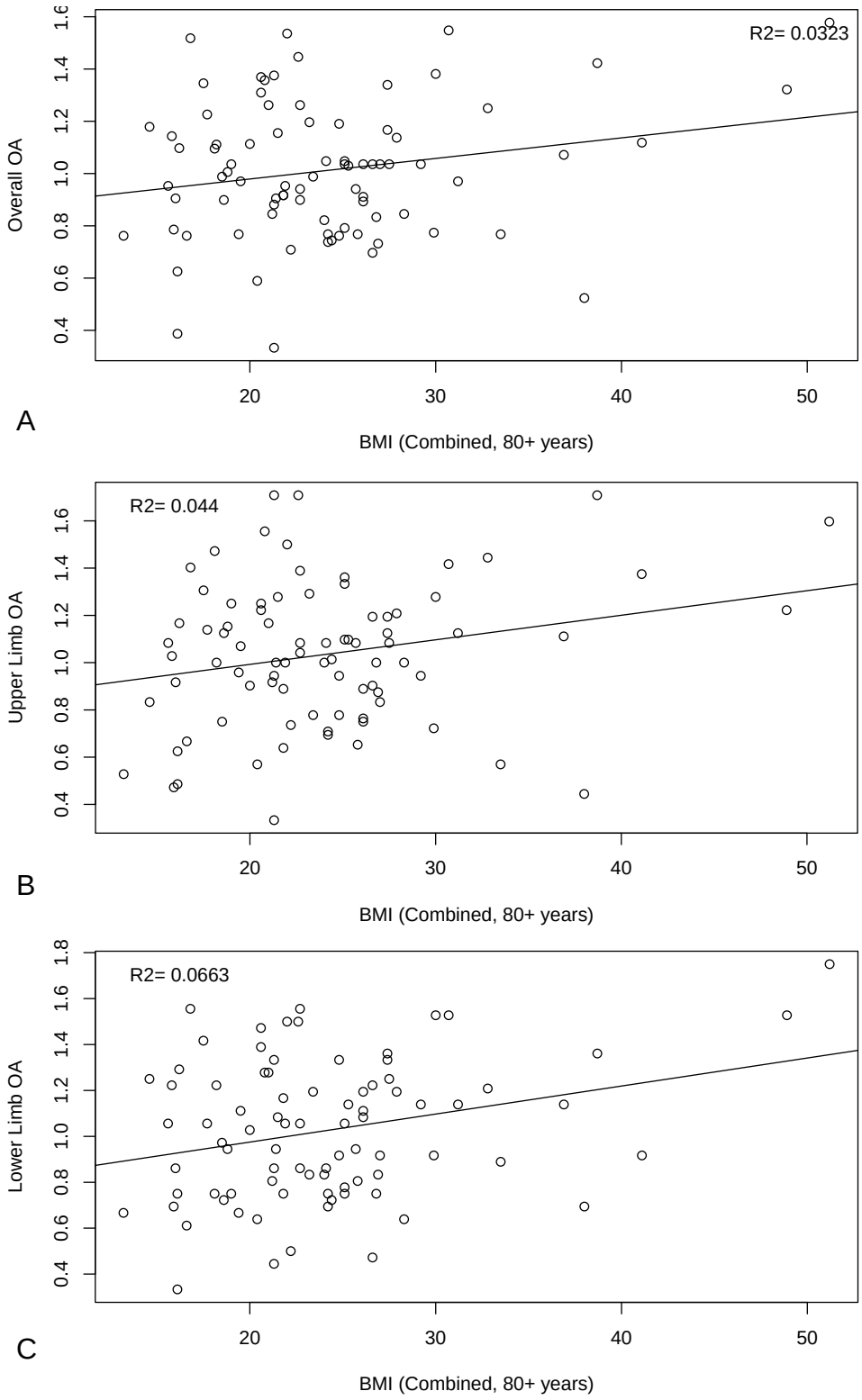


Figure 6-175. Hypothesis 4b: OA correlates positively with obesity; scatterplots for summary OA and BMI values in combined-sex individuals 80+ years. A) Overall OA, B) Upper limb OA, C) Lower limb OA. Black line indicates linear regression model.

Table 6-37. Spearman's rank-order correlations for summary OA and BMI values in the combined sample.

Group	*rho*
<40 years overall OA	0.434
<40 years upper limb OA	0.110
<40 years lower limb OA	0.542*
40-49 years overall OA	0.241
40-49 years upper limb OA	0.168
40-49 years lower limb OA	0.304
50-59 years overall OA	0.451*
50-59 years upper limb OA	0.310
50-59 years lower limb OA	0.498*
60-69 years overall OA	0.118
60-69 years upper limb OA	0.141
60-69 years lower limb OA	0.154
70-79 years overall OA	0.269
70-79 years upper limb OA	0.087
70-79 years lower limb OA	0.470*
80+ years overall OA	0.089
80+ years upper limb OA	0.151
80+ years lower limb OA	0.184

*Statistically significant at α=0.003 (Bonferroni correction: α=0.05/18).

Table 6-38. Regression results for summary OA and BMI values in the combined sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	0.156	0.093	1.672	0.103
<40 years overall OA	0.008	0.003	2.557	0.015
Intercept	0.146	0.119	1.220	0.230
<40 years upper limb OA	0.006	0.004	1.536	0.133
Intercept	0.079	0.132	0.596	0.555
<40 years lower limb OA	0.015	0.004	3.478	0.001*
Intercept	0.520	0.079	6.579	1.39e-08*
40-49 years overall OA	0.004	0.002	1.898	0.063
Intercept	0.561	0.085	6.567	1.46e-08*
40-49 years upper limb OA	0.003	0.003	1.094	0.278
Intercept	0.491	0.112	4.372	5.07e-05*
40-49 years lower limb OA	0.009	0.003	2.539	0.014
Intercept	0.427	0.090	4.740	1.29e-05*
50-59 years overall OA	0.012	0.003	3.973	1.88e-04*
Intercept	0.485	0.125	3.869	2.65e-04*
50-59 years upper limb OA	0.010	0.004	2.389	0.020
Intercept	0.352	0.111	3.170	0.002*
50-59 years lower limb OA	0.018	0.003	4.856	8.46e-06*
Intercept	0.720	0.121	5.96	1.3e-07*
60-69 years overall OA	0.002	0.004	0.51	0.612
Intercept	0.744	0.133	5.601	5.2e-07*
60-69 years upper limb OA	0.002	0.004	0.441	0.661
Intercept	0.680	0.150	4.546	2.6e-05*
60-69 years lower limb OA	0.006	0.005	1.215	0.229
Intercept	0.644	0.119	5.422	9.91e-07*
70-79 years overall OA	0.010	0.004	2.298	0.025
Intercept	0.836	0.131	6.407	2.13e-08*
70-79 years upper limb OA	0.003	0.005	0.562	0.576
Intercept	0.408	0.147	2.769	0.007
70-79 years lower limb OA	0.020	0.005	3.936	2.1e-04*
Intercept	0.822	0.102	8.041	6.25e-12*
80+ years overall OA	0.008	0.004	1.932	0.057
Intercept	0.785	0.119	6.582	4.28e-09*
80+ years upper limb OA	0.010	0.005	2.185	0.032
Intercept	0.731	0.117	6.233	1.95e-08*
80+ years lower limb OA	0.012	0.005	2.612	0.011

*Statistically significant at α=0.003 (Bonferroni correction: α=0.05/18).

Table 6-39. Multiple regression results for age, habitual activities, BMI, and CAS in the female sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	3.987	2.756	1.446	0.153
Age	0.054	0.043	1.263	0.211
BMI	0.060	0.086	0.706	0.483
Habitual activities	0.108	0.676	0.160	0.873
Age:BMI	-0.001	0.001	-0.712	0.479
Age:Habitual activities	-0.004	0.011	-0.310	0.758
BMI:Habitual activities	-0.015	0.024	-0.639	0.525
Age:BMI:Habitual activities	0.000	0.000	0.654	0.516

*Statistically significant at the α=0.05 level.

Table 6-40. Multiple regression model simplified by backwards-stepwise selection (criterion: AIC) for age, habitual activities, BMI, and CAS in the female sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	4.579	0.607	7.543	1.61e-10*
Age	0.041	0.009	4.592	1.99e-05*

*Statistically significant at the α=0.05 level.

Table 6-41. Multiple regression results for age, occupational activities, BMI, and CAS in the female sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	-0.051	2.842	-0.018	0.986
Age	0.112	0.044	2.532	0.012*
BMI	0.116	0.091	1.271	0.206
Occupational activities	1.386	1.246	1.112	0.268
Age:BMI	-0.002	0.002	-1.276	0.204
Age:Occupational activities	-0.024	0.019	-1.274	0.205
BMI:Occupational activities	-0.040	0.041	-0.963	0.337
Age:BMI:Occupational activities	0.001	0.001	1.098	0.274

*Statistically significant at the α=0.05 level.

Table 6-42. Multiple regression model simplified by backwards-stepwise selection (criterion: AIC) for age, occupational activities, BMI, and CAS in the female sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	4.051	0.340	11.930	<2e-16*
Age	0.046	0.005	9.248	<2e-16*

*Statistically significant at the α=0.05 level.

Table 6-43. Multiple regression results for age, habitual activities, BMI, and CAS in the male sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	3.288	3.793	0.867	0.390
Age	0.063	0.059	1.072	0.288
BMI	-0.015	0.154	-0.095	0.924
Habitual activities	-0.723	0.925	-0.781	0.438
Age:BMI	0.000	0.002	0.111	0.912
Age:Habitual activities	0.006	0.014	0.456	0.650
BMI:Habitual activities	0.027	0.037	0.729	0.469
Age:BMI:Habitual activities	-0.000	0.001	-0.472	0.639

*Statistically significant at the α=0.05 level.

Table 6-44. Multiple regression model simplified by backwards-stepwise selection (criterion: AIC) for age, habitual activities, BMI, and CAS in the male sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	1.518	0.662	2.291	0.025*
Age	0.066	0.007	9.796	2.78e-14*
BMI	0.053	0.017	3.186	0.002*

*Statistically significant at the α=0.05 level.

Table 6-45. Multiple regression results for age, occupational activities, BMI, and CAS in the male sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	4.641	2.613	1.776	0.078
Age	0.037	0.044	0.839	0.402
BMI	-0.033	0.096	-0.350	0.727
Occupational activities	-0.716	0.782	-0.916	0.361
Age:BMI	0.000	0.002	0.250	0.803
Age:Occupational activities	0.008	0.013	0.633	0.528
BMI:Occupational activities	0.027	0.028	0.945	0.346
Age:BMI:Occupational activities	-0.000	0.001	-0.596	0.552

*Statistically significant at the α=0.05 level.

Table 6-46. Multiple regression model simplified by backwards-stepwise selection (criterion: AIC) for age, occupational activities, BMI, and CAS in the male sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	3.900	0.752	5.190	5.82e-07*
Age	0.050	0.004	11.431	<2e-16*
BMI	-0.010	0.024	-0.407	0.685
Occupational activities	-0.224	0.184	-1.218	0.225
BMI:Occupational activities	0.010	0.007	1.498	0.136

*Statistically significant at the α=0.05 level.

Table 6-47. Multiple regression results for age, habitual activities, BMI, and overall OA in the combined-sex sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	0.323	0.422	0.767	0.445
Age	0.007	0.007	1.050	0.296
BMI	-0.002	0.015	-0.128	0.898
Habitual activities	-0.058	0.107	-0.540	0.590
Age:BMI	0.000	0.000	0.214	0.831
Age:Habitual activities	-0.000	0.002	-0.175	0.861
BMI:Habitual activities	0.000	0.004	0.021	0.983
Age:BMI:Habitual activities	0.000	0.000	0.623	0.535

*Statistically significant at the α=0.05 level.

Table 6-48. Multiple regression model simplified by backwards-stepwise selection (criterion: AIC) for age, habitual activities, BMI, and overall OA in the combined-sex sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	0.038	0.135	0.285	0.776
Age	0.011	0.001	8.912	3.87e-15*
BMI	0.002	0.004	0.604	0.547
Habitual activities	-0.062	0.028	-2.189	0.030*
BMI:Habitual activities	0.002	0.001	1.900	0.060

*Statistically significant at the α=0.05 level.

Table 6-49. Multiple regression results for age, habitual activities, BMI, and upper limb OA in the combined-sex sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	0.402	0.486	0.827	0.410
Age	0.007	0.007	0.931	0.354
BMI	-0.004	0.017	-0.234	0.815
Habitual activities	-0.071	0.123	-0.579	0.564
Age:BMI	0.000	0.000	0.074	0.941
Age:Habitual activities	-0.000	0.002	-0.237	0.813
BMI:Habitual activities	-0.000	0.005	-0.020	0.984
Age:BMI:Habitual activities	0.000	0.000	0.804	0.423

*Statistically significant at the α=0.05 level.

Table 6-50. Multiple regression model simplified by backwards-stepwise selection (criterion: AIC) for age, habitual activities, BMI, and upper limb OA in the combined-sex sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	0.342	0.244	1.398	0.165
Age	0.008	0.003	2.711	0.008*
BMI	-0.003	0.004	-0.585	0.560
Habitual activities	-0.150	0.058	-2.568	0.011*
Age:Habitual activities	0.001	0.001	1.450	0.150
BMI:Habitual activities	0.003	0.001	2.600	0.010*

*Statistically significant at the α=0.05 level.

Table 6-51. Multiple regression results for age, habitual activities, BMI, and lower limb OA in the combined-sex sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	0.293	0.554	0.528	0.598
Age	0.005	0.009	0.581	0.562
BMI	0.009	0.020	0.456	0.649
Habitual activities	-0.036	0.140	-0.259	0.796
Age:BMI	0.000	0.000	0.046	0.963
Age:Habitual activities	-0.000	0.002	-0.159	0.874
BMI:Habitual activities	-0.002	0.005	-0.325	0.746
Age:BMI:Habitual activities	0.000	0.000	0.669	0.505

*Statistically significant at the α=0.05 level.

Table 6-52. Multiple regression model simplified by backwards-stepwise selection (criterion: AIC) for age, habitual activities, BMI, and lower limb OA in the combined-sex sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	-0.164	0.143	-1.148	0.253
Age	0.010	0.002	6.006	1.73e-08*
BMI	0.015	0.003	5.465	2.22e-07*

*Statistically significant at the α=0.05 level.

Table 6-53. Multiple regression results for age, occupational activities, BMI, and overall OA in the female sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	-0.198	0.624	-0.316	0.752
Age	0.011	0.010	1.099	0.273
BMI	-0.006	0.020	-0.305	0.761
Occupational activities	-0.025	0.274	-0.093	0.926
Age:BMI	0.000	0.000	0.649	0.517
Age:Occupational activities	0.000	0.004	0.204	0.839
BMI:Occupational activities	0.005	0.009	0.583	0.560
Age:BMI:Occupational activities	-0.000	0.000	-0.580	0.562

*Statistically significant at the α=0.05 level.

Table 6-54. Multiple regression model simplified by backwards-stepwise selection (criterion: AIC) for age, occupational activities, BMI, and overall OA in the female sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	-0.333	0.120	-2.785	0.006*
Age	0.013	0.001	11.110	<2e-16*
BMI	0.007	0.002	3.935	1.22e-04*
Occupational activities	0.032	0.023	1.410	0.161

*Statistically significant at the α=0.05 level.

Table 6-55. Multiple regression results for age, occupational activities, BMI, and upper limb OA in the female sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	-0.007	0.683	-0.010	0.992
Age	0.009	0.011	0.813	0.417
BMI	-0.013	0.022	-0.607	0.545
Occupational activities	-0.090	0.300	-0.302	0.763
Age:BMI	0.000	0.000	0.690	0.491
Age:Occupational activities	0.002	0.005	0.363	0.717
BMI:Occupational activities	0.007	0.010	0.678	0.496
Age:BMI:Occupational activities	-0.000	0.000	-0.554	0.580

*Statistically significant at the α=0.05 level.

Table 6-56. Multiple regression model simplified by backwards-stepwise selection (criterion: AIC) for age, occupational activities, BMI, and upper limb OA in the female sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	-0.388	0.131	-2.967	0.004*
Age	0.013	0.001	10.654	<2e-16*
BMI	0.005	0.002	2.438	0.016*
Occupational activities	0.052	0.025	2.130	0.035*

*Statistically significant at the α=0.05 level.

Table 6-57. Multiple regression results for age, occupational activities, BMI, and lower limb OA in the female sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	-0.639	0.826	-0.774	0.440
Age	0.014	0.013	1.054	0.293
BMI	0.017	0.027	0.443	0.658
Occupational activities	0.238	0.362	0.657	0.512
Age:BMI	0.000	0.000	0.256	0.798
Age:Occupational activities	-0.002	0.005	-0.382	0.703
BMI:Occupational activities	-0.002	0.012	-0.182	0.856
Age:BMI:Occupational activities	-0.000	0.000	-0.015	0.988

*Statistically significant at the α=0.05 level.

Table 6-58. Multiple regression model simplified by backwards-stepwise selection (criterion: AIC) for age, occupational activities, BMI, and lower limb OA in the female sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	-0.232	0.141	-1.646	0.102
Age	0.011	0.002	7.290	1.16e-11*
BMI	0.013	0.002	5.438	1.87e-07*

*Statistically significant at the α=0.05 level.

Table 6-59. Multiple regression results for age, occupational activities, BMI, and overall OA in the male sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	0.308	0.610	0.504	0.615
Age	0.004	0.010	0.351	0.726
BMI	-0.014	0.022	-0.643	0.521
Occupational activities	-0.152	0.183	-0.830	0.408
Age:BMI	0.000	0.000	0.850	0.396
Age:Occupational activities	0.003	0.003	0.821	0.413
BMI:Occupational activities	0.008	0.007	1.249	0.213
Age:BMI:Occupational activities	-0.000	0.000	-1.03	0.303

*Statistically significant at the α=0.05 level.

Table 6-60. Simplified multiple regression model for age, occupational activities, BMI, and overall OA in the male sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	-0.220	0.106	-2.075	0.039*
Age	0.011	0.001	10.326	<2e-16*
BMI	0.009	0.002	4.166	4.86e-05*
Occupational activities	0.034	0.013	2.734	0.007*

*Statistically significant at the α=0.05 level.

Table 6-61. Multiple regression results for age, occupational activities, BMI, and upper limb OA in the male sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	0.336	0.716	0.469	0.639
Age	0.003	0.012	0.215	0.830
BMI	-0.011	0.026	-0.425	0.671
Occupational activities	-0.221	0.214	-1.031	0.304
Age:BMI	0.000	0.001	0.579	0.563
Age:Occupational activities	0.004	0.004	1.087	0.279
BMI:Occupational activities	0.009	0.008	1.128	0.261
Age:BMI:Occupational activities	-0.000	0.000	-0.895	0.372

*Statistically significant at the α=0.05 level.

Table 6-62. Simplified multiple regression model for age, occupational activities, BMI, and upper limb OA in the male sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	-0.363	0.124	-2.920	0.004*
Age	0.012	0.001	9.840	<2e-16*
BMI	0.010	0.003	3.769	2.24e-04*
Occupational activities	0.059	0.015	4.034	8.19e-05*

*Statistically significant at the α=0.05 level.

Table 6-63. Multiple regression results for age, occupational activities, BMI, and lower limb OA in the male sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	0.562	0.755	0.744	0.458
Age	-0.001	0.013	-0.045	0.965
BMI	-0.021	0.028	-0.761	0.447
Occupational activities	-0.188	0.226	-0.832	0.407
Age:BMI	0.001	0.001	1.017	0.311
Age:Occupational activities	0.003	0.004	0.758	0.450
BMI:Occupational activities	0.010	0.008	1.266	0.207
Age:BMI:Occupational activities	-0.000	0.000	-1.032	0.304

*Statistically significant at the α=0.05 level.

Table 6-64. Simplified multiple regression model for age, occupational activities, BMI, and lower limb OA in the male sample.

Variable	Estimate	Std. Error	t value	Probability
Intercept	-0.187	0.131	-1.420	0.157
Age	0.009	0.001	7.417	4.95e-12*
BMI	0.014	0.003	4.842	2.81e-06*
Occupational activities	0.031	0.016	2.030	0.044*

*Statistically significant at the α=0.05 level.

Table 6-65. Observed instances of traumatic injury or surgical intervention in the various left and right joints in females and males for which acetabular variable and/or OA scores were available*.

Joint	Trauma/surgery (females)	Trauma/surgery (males)
L TMJ	3	2
R TMJ	2	3
L shoulder	9	4
R shoulder	8	5
L elbow	7	8
R elbow	2	3
L wrist	16	12
R wrist	14	9
L hip	18 (20)	17
R hip	22 (26)	8 (11)
L knee	10 (16)	12 (18)
R knee	12 (16)	11 (17)
L ankle	5	10
R ankle	6	14

*Additional instances of trauma/surgery (i.e., complete prosthetic joint replacements) were observed in the hip and knee. While these additional instances of trauma/surgery could not be tested for acetabular changes or OA, they increased female and male hip and knee totals to the higher numbers noted in parentheses above.

Table 6-66. Probability (*p*) that acetabular changes are unaffected by trauma/surgery in the left and right hip joints of females and males (based on repeated resampling of unaffected individuals and comparisons with acetabular score medians for affected individuals).

Joint	*p* (females)	*p* (males)
L Variable 1	0.166	0.006
R Variable 1	0.109	0.146
L Variable 2	0.054	0.040
R Variable 2	0.022	0.178
L Variable 3	0.047	0.014
R Variable 3	0.000*	0.164
L Variable 4	0.177	0.068
R Variable 4	0.326	0.086
L Variable 5	0.162	0.095
R Variable 5	0.097	0.002*
L Variable 6	0.000*	0.133
R Variable 6	0.000*	0.263
L Variable 7	0.000*	5e-04*
R Variable 7	0.000*	0.052

*Statistically significant at α=0.003 (Bonferroni correction: α=0.05/14).

Table 6-67. Probability (*p*) that OA is unaffected by trauma/surgery in the various left and right joints in females and males (based on repeated resampling of unaffected joints and comparisons with OA score medians for affected joints).

Joint	*p* (females)	*p* (males)
L TMJ	0.748	0.135
R TMJ	0.021	0.002*
L shoulder	1e-04*	0.093
R shoulder	0.050	0.005
L elbow	0.000*	3e-04*
R elbow	0.045	9e-04*
L wrist	0.000*	0.000*
R wrist	1e-04*	0.001*
L hip	0.050	0.000*
R hip	0.000*	0.052
L knee	0.020	0.013
R knee	0.059	0.003
L ankle	0.027	9e-04*
R ankle	6e-04*	0.000*

*Statistically significant at α=0.003 (Bonferroni correction: α=0.05/14).

Table 6-68. Mean and median OA scores in the various joints in females and males.

Joint	Mean (female)	Median (female)	Mean (male)	Median (male)
TMJ	0.643	0.5	0.476	0.5
Shoulder	0.970	1	0.910	1
Elbow	0.688	0.667	0.783	0.833
Wrist	0.609	0.5	0.710	0.625
Hip	1.080	1	0.968	1
Knee	1.008	1	0.869	1
Ankle	0.481	0.333	0.629	0.667

Table 6-69. Pelvic joint preservation: mean and median preservation scores for the pubic symphysis, auricular surface, and acetabulum in combined-sex individuals of various ages.

Joint	Mean preservation score	Median preservation score
Pubic symphysis <80 years	0.654	0.5
Pubic symphysis 80+ years	0.971	1
Auricular surface <70 years	0.483	0.5
Auricular surface 70-79 years	0.757	1
Auricular surface 80+ years	1.174	1
Acetabulum <70 years	0.404	0.5
Acetabulum 70-79 years	0.581	0.5
Acetabulum 80+ years	0.641	0.75

Table 6-70. Pelvic joint preservation: results of Wilcoxon rank-sum testing of preservation scores for the pubic symphysis (PS), auricular surface (AS), and acetabulum (Ac) in combined-sex individuals of various ages.

Joint	PS <80	PS 80+	AS <70	AS 70-79	AS 80+	Ac <70	Ac 70-79	Ac 80+
PS <80 years		4.501e-08*	1.551e-04*	0.276	4.989e-13*	6.539e-08*	0.429	0.864
PS 80+ years	4.501e-08*		2.736e-13*	0.006	0.014	<2.2e-16*	1.602e-06*	2.395e-06*
AS <70 years	1.551e-04*	2.736e-13*		0.003	<2.2e-16*	0.179	0.123	0.004
AS 70-79 years	0.276	0.006	0.003		7.897e-05*	9.221e-05*	0.143	0.379
AS 80+ years	4.989e-13*	0.014	<2.2e-16*	7.897e-05*		<2.2e-16*	1.22e-08*	9.376e-10*
Ac <70 years	6.539e-08*	<2.2e-16*	0.179	9.221e-05*	<2.2e-16*		0.013	4.394e-05*
Ac 70-79 years	0.429	1.602e-06*	0.123	0.143	1.22e-08*	0.013		0.384
Ac 80+ years	0.864	2.395e-06*	0.004	0.379	9.376e-10*	4.394e-05*	0.384	

*Statistically significant at α=0.002 (Bonferroni correction: α=0.05/28).

CHAPTER 7
DISCUSSION

With the development of improved healthcare standards, the mean age of the world population is rising. Demographic research indicates that the life expectancies of healthy human populations increase regularly and linearly from year to year (Vaupel, 2004). Accurate and precise age estimates for elderly adults are notoriously difficult to achieve, and most age estimation techniques lump the elderly into broad categories such as “50+” (e.g., Todd, 1920) or “60+” (e.g., Lovejoy et al., 1985). Diseases like obesity and OA are epidemic in the modern U.S., potentially complicating age-at-death estimation in older individuals. In the forensic setting, imprecise age estimates for the elderly reduce the likelihood of identifying the remains of unknown individuals of advanced age. In the bioarchaeological setting, imprecise estimates of older adult age contribute to an incomplete understanding of past mortality profiles. Acetabular age estimation methods have been found to provide good age discrimination for the elderly (Rissech et al., 2006). However, a more sensitive understanding of the relationship between acetabular changes and OA—and the influences of age, activity, and obesity on both—is warranted. Scientific knowledge of the biology underlying acetabular changes is necessary before acetabular age estimation can continue, as this knowledge has the potential to validate or invalidate, refine or reject methods of age estimation based on the acetabulum.

Discussion of Hypotheses

Hypothesis 1: Acetabular Changes Correlate Positively with OA

Supported. This suggests a link between acetabular age changes and the progression of OA. In essence, acetabular changes are not metamorphic, but related to a primarily degenerative disease.

CAS. Testing revealed statistically significant positive correlations between CAS and OA in all individual joints in both sexes; *rho* values were particularly high in the hip (the 95% confidence intervals for female and male hip OA did not contain the *rho* values of any other joints). Regression of CAS on overall OA, upper limb OA, and lower limb OA revealed statistically significant positive associations between these composite acetabular and OA scores in both females and males. Thus, the processes of OA seem to be linked with the aging processes observed and scored in the acetabulum.

Acetabular variables. Many statistically significant positive correlations were present between the individual acetabular variables and OA in the individual joints, suggesting a link between acetabular aging processes and OA. Further, the fact that both female and male hip OA correlated statistically significantly positively with all acetabular variables argues for similarities between the acetabular variables and the processes of hip OA. If the changes being scored in the acetabulum (the variables of Rissech et al., 2006) were metamorphic, this strong relationship with OA would not be expected.

There may be a functional component to this correlation. Higher *rho* values were always seen between the individual acetabular variables and overall and lower limb OA (compared with upper limb OA); in many cases, differences were marked enough that the 95% confidence intervals for the overall and lower limb *rho* values did not contain

the upper limb *rho* value. In linear regression analyses of CAS, r^2 values were also higher for lower limb OA than upper limb OA in both females and males, as would be expected if the regular stresses of bipedal locomotion were causing both acetabular age change and lower limb OA.

However, there may also be a systemic component to the positive relationship between the acetabular variables and OA. While female and male hip OA correlated significantly with all acetabular variables, female and male knee and ankle OA did not. In the ankle, and especially in females, associations with acetabular variables were particularly weak. In contrast, combined-sex shoulder, elbow, and wrist OA had statistically significant positive correlations with all acetabular variables. This indicates a systemic cause for both OA and acetabular changes, rather than a purely biomechanical etiology. Hereditary predisposition (not assessed in the current study) or age effects (i.e., senescence, see below discussion of *Hypothesis 2*) are two likely systemic influences. However systemic acetabular changes might be, however, they do not seem to be strongly linked with the processes of TMJ OA in either sex (only Variables 1 and 3 had statistically significant positive correlations with TMJ OA).

Hypothesis 2: Age

Hypothesis 2a: Acetabular changes correlate positively with age

Supported. In general, the current findings support the use of the acetabulum for age estimation. While acetabulum-specific aging processes seem linked with OA, acetabular changes may involve some age-related processes not implicated in the normal progression of OA in the hip and other joints.

Age estimates. In most cases, the age estimates generated by the acetabular aging method (Rissech et al., 2006) correlated with the documented ages of the sample

individuals, indicating the applicability of the method for estimating age in both females and males and in both younger and older adults. However, estimating age in the sample's youngest females proved problematic.

In males of all ages, point age estimates correlated statistically significantly with known ages. In females, however, only the age estimates for older and combined-age individuals correlated statistically significantly with known ages, and the youngest females (<60 years) also displayed high percentages of prediction error and low percentages of correct classification into age groups. This poor performance of the age estimation method on younger individuals was unexpected, as it counters previous findings indicating that young adult age is estimated with higher precision and accuracy than older adult age (Aykroyd et al., 1999; Nawrocki, 2010; Winburn and Brown, 2010; 2011). However, the high error rates of the younger female acetabular age estimates are likely due in large part to the underrepresentation of young females in the current sample. The IDADE2 program depends upon sufficient representation of acetabular scores from each age group in the estimating distribution (in this case, a randomly sampled, age-stratified subgroup of the study sample); when certain age groups are not well represented, age estimation accuracy decreases in those age groups. By contrast, larger numbers of young male individuals were present in both the estimating distribution and test sample, likely leading to higher percentages of correctly classified <60-year males compared with <60-year females.

However, percentages of prediction error were similar between females and males of the various age groups, and they were always lowest in the oldest age groups—just 16% for both 60+ females and 60+ males. At first examination, this low

error in the oldest individuals seems counterintuitive. Yet, this trend may be due to the IDADE2 software's creation of broader confidence intervals in older individuals in order to account for increasing acetabular variability with increasing age—at least in this sample's males. In males, confidence intervals for <60-year-old individuals (mean=24 years) were statistically significantly narrower than confidence intervals for 60+ individuals (mean=30.4 years; p=0.005; Wilcoxon rank-sum test). These broader confidence intervals in the elderly reflect the increasing uncertainty often associated with older adult age estimation (Aykroyd et al., 1999; Nawrocki, 2010), even as they result in higher probabilities of correct classification for older individuals. This is a trend also seen in pubic symphysis and auricular surface aging methods, and probabilities of misclassifying older adults into an age group have been found to be lower than probabilities of misclassifying younger adults using the Suchey-Brooks (1990), Lovejoy and colleagues (1985), and Osborne and colleagues (2004) methods (Winburn and Brown, 2010; 2011).

In contrast, however, confidence intervals for 60+ individuals were nearly identical in breadth to confidence intervals for <60-year-old individuals in this sample's females (<60-year-old mean=30.5 years; 60+ mean=31.2 years; p=0.871; Wilcoxon rank-sum test). Unlike in males, rather than reflecting the broader confidence intervals that account for increased uncertainty in elderly adult aging, the IDADE2 program truly seemed to estimate age more accurately in older females than in younger females. This could be due to the fact that older females were better represented in this sample than younger females, whereas the male age distribution was more balanced. Regardless, the fact that the acetabular aging method showed no obvious decrease in

age estimation power for older versus younger females supports previous research indicating its potential to estimate age accurately in elderly individuals (Rissech et al., 2006), even where well-established methods like that of Brooks and Suchey (1990) fail.

In general, the acetabular aging method seemed to perform slightly better on males than on females: while *rho* values for combined-age female and male age estimates were similar (based on overlapping 95% confidence intervals), the *rho* value for combined-age male age estimates was higher than the *rho* values for both <60 and 60+ female age estimates, as indicated by a 95% confidence interval that did not include the *rho* values for either female group. This may be due to the fact that the method was originally developed on an all-male sample (Rissech et al., 2006). The earlier and more rapid onset of skeletal dysfunction evidenced in females compared with males—and a corresponding increase in acetabular variation—may also be contributing to slightly lower *rho* values in females. Still, the statistically significant correlations between known and estimated ages in 60+ and combined-age females, in addition to males of all ages, supports previous research indicating the applicability of the method to individuals of both sexes (San-Millán et al., 2016; 2017).

When the IDADE2 software was used to estimate age in ten female and ten male test subsamples, and 95% confidence intervals of "fit" values were bootstrapped from 10,000 resamples of the resulting distributions, the model produced fairly consistent age estimation errors from run to run. While there were some exceptions, most observed "fit" means were included within the 95% confidence intervals bootstrapped from other runs. This indicates that the model is relatively stable. Stability does not equal certainty, however. The IDADE2 program authors have suggested that "fit" values

greater than 10 are associated with distributions that either exhibit high variance or low variance centered around an age class that is far from the actual age at death—both of which can lead to poor age estimations (Rissech et al., 2006). In all female and male subsamples, “fit” means were greater than 10, though the overall lower male “fit” means again suggested better performance of the model on males compared with females.

Finally, it should be noted that even given the error discussed above, the tautological age estimation analysis employed in this study (wherein acetabular scores from the *Bass Collection* sample were used to estimate age in individuals from the same sample) likely resulted in lower error than would be observed in an analysis in which the reference and test samples were independent. The major potential benefit of a Bayesian age estimation method is also its major potential pitfall: the user attempts to choose a relevant reference distribution; but age estimates will suffer if that reference distribution proves inappropriate for the analysis at hand.

CAS. Testing revealed statistically significant positive correlations between CAS and age in both sexes, indicating the applicability of acetabular age estimation to both female and male individuals. This finding is consistent with recent research demonstrating the pattern of acetabular aging to be similar in both females and males (San-Millán et al., 2016; 2017), despite the fact that the original acetabular aging method was developed and tested on all-male samples (Rissech et al., 2006, 2007).

Acetabular variables. Statistically significant positive correlations were also noted between age and all of the individual acetabular variables in the combined-sex sample. These results are consistent with previous research by San-Millán and colleagues (2016) indicating the strong age correlation of the seven variables. Other

research, however, has indicated that acetabular fossa variables are less highly correlated with age than acetabular rim variables: Mays (2012) found that only Variables 1-4 were significantly correlated with age (Mays, 2012); and Calce (2012) found that Variables 1, 3, and 4 contributed most to age-related acetabular variation. This discrepancy could reflect population differences among the samples analyzed by Calce (2012; males from the early 20th-century *Grant Collection*, Toronto, Canada), Mays (2012; females and males from the 18th-to-19th-century *Spitalfields Collection*, London, U.K.), San-Millán and colleagues (2016; females and males from the 19th and 20th century Lisbon Collection, Portugal), and in the current research (females and males from the modern *Bass Collection*, Tennessee, U.S.A.).

However, discrepancies in the pattern of age correlations of the acetabular variables could also reflect difficulties of applying the acetabular aging method (Rissech et al., 2006), possibly representing an artifact of different observers' interpretations of the variables. For example, Mays (2012) reported that he modified the method protocol in his scoring of Variables 6 and 7; it is thus unsurprising that the modified scores do not correlate as strongly with age as the original authors reported (Rissech et al., 2006). Other authors have critiqued previously published descriptions of acetabular variables, stating that difficulties in interpreting the descriptions lead to observer subjectivity (Calce and Rogers, 2011; Stull and James, 2010). In the current researcher's experience, having been trained in the method by the original study's lead author (Carme Rissech) has been particularly helpful in the interpretation of acetabular fossa variables, which assess ambiguous skeletal traits like bone texture and difficult-to-differentiate micro- and macro-porosities. Still, during the current research, it was repeatedly noted that

Variables 6 and 7 of the acetabular fossa were the most difficult to score, due to overlapping descriptions referring to related skeletal aging processes (the loss of consistency in the region of the fossa; see *Problems with Acetabular Aging*, below). It is possible that fossa variables would have correlated more strongly with age in the research of Calce (2012) and Mays (2012) if the variable descriptions had been more clearly elucidated in the original method (Rissech et al., 2006). To this end, recent research has emended the existing descriptions of the acetabular variables (San-Millán et al., 2016), modifying Variables 5 through 7 for clarity and repeatability. Future method tests will determine whether these changes have improved the method sufficiently to reduce issues of intra- and inter-observer error.

Alternately, the lack of age correlation reported by previous authors for acetabular fossa variables could reflect biological differences in the aging processes taking place in the region of the acetabular fossa, compared with the more highly age-correlated acetabular rim. Previous research has indicated that marginal joint changes (e.g., osteophyte formation) are more highly correlated with age than changes to the joint's articular surfaces (Weiss and Jurmain, 2007). Recent reevaluations of acetabular age estimation methods consistently conclude that traits related to activity of the acetabular rim and apex correlate with age (Calce, 2012; Calce and Rogers, 2011; Mays, 2012). A recent geometric morphometric shape analysis of age-related changes to the acetabulum revealed that bone production along the borders of the lunate surface (i.e., the acetabular rim, apex, and outer edge of the acetabular fossa) is associated with age in both females and males (San-Millán et al., 2017). In the current study, the two acetabular variables with the highest *rho*-value correlations with age were Variables

3 and 4, and sometimes these differences in age correlation were marked (e.g., the 95% confidence interval for Variable 3 did not include the *rho* values for Variables 1, 2, and 6; the 95% confidence interval for Variable 4 did not include the *rho* values for Variables 1 and 6). Variables 3 and 4 describe porosity of the acetabular rim and osteophyte formation of the apex, respectively. Both of these strongly age-correlated variables describe marginal joint changes. Rim porosity, like osteophyte formation, may be linked more with age than OA, given the previous finding that porosity has no significant relationship with OA (Rothschild, 1997).

However, both acetabular rim porosity and rim shape (i.e., osteophyte formation) did correlate positively with OA in the current study, and osteophyte formation is a classic diagnostic characteristic of OA in both clinical and skeletal analyses (Jurmain, 1990; Kellgren and Lawrence, 1957). Links between these rim variables and OA seem clear, but the question remains whether these variables capture any information that is not already captured by general OA scores. This study indicates that they may. General hip OA had a weaker age correlation than did specific acetabular aging processes (as indicated by high *rho* values for CAS; see discussion of *Hypothesis 2b*, below). In essence, while hip OA and acetabular changes were highly correlated, hip OA was not as strongly correlated with age as were acetabulum-specific aging processes. This suggests that while OA represents a large component of the aging processes scored in the method of Rissech and colleagues (2006), “something extra” seems to be captured by the acetabular scores, contributing to their strong age correlation.

This may be due in part to the complexity of the acetabulum compared with other appendicular joints. Unlike the femoral head, for example, which (with the exception of the small fovea capitis) forms a smooth and unbroken articular surface, the skeletal acetabulum is comprised of multiple components: the lunate surface that articulates with the femoral head; the surrounding acetabular rim which supports the labrum during life; and the acetabular fossa, to which the stabilizing ligamentum teres attaches. Each region, in turn, is subject to its own age-related changes. For example: pitting and eburnation can occur on the lunate surface; osteophytes and porosity affect the margins of the rim; and the bone of the fossa becomes swollen and loses consistency with age. Thus, while acetabular traits are linked with OA, the sheer amount of variation they capture may be contributing to their stronger age correlation.

Further, certain of the acetabular variables score age-related processes not captured in general OA scores. The fossa variables, for example, deal with loss of consistency and density in the non-articular bone of the acetabular fossa. These changes may be linked with osteoporosis, a process that is itself age-related. Thus, while the problematic nature of the acetabular fossa variables has been discussed at length here and elsewhere, the age-prediction value they may be adding argues for their continued study in future acetabular age-estimation research.

Hypothesis 2b: OA correlates positively with age

Supported. In general, the current findings suggest that OA is also relevant for age estimation, despite its predominantly degenerative etiology.

Individual joints. Age had a statistically significant positive correlation with OA in all individual joints except the female ankle. Compared with acetabular variables, OA in most joints seemed to have a similar level of age correlation (based on similar *rho*

values and overlapping 95% confidence intervals). However, combined-sex shoulder OA had a stronger positive correlation with age than all acetabular variables and OA in most other joints (as indicated by a 95% confidence interval that did not contain any of the other *rho* values except that of male hip OA). In contrast, ankle OA had a particularly weak (in the case of females, absent) association with age (as indicated by *rho* values lower than all 95% confidence intervals except that of female knee OA).

In attempting to untangle the links between the aging process in the acetabulum and general OA, it is also relevant to compare correlations between age and CAS, and age and hip OA. Hip OA exhibited a weaker correlation with age in both females (hip OA *rho*=0.438, CAS *rho*=0.565) and males (hip OA *rho*=0.525, CAS *rho*=0.713); for both sexes, the 95% confidence interval of the higher CAS *rho* value did not include the lower OA *rho* value. While not conclusive, this suggests that CAS may incorporate additional age-related variation not captured by general OA scores.

Summary OA. Age had a statistically significant positive correlation with overall, upper limb, and lower limb OA. In comparing regression results with acetabular age analyses, however, lower limb OA and age had a lower r^2 value than CAS and age in both sexes. This highlights the possibility that acetabular age changes may include processes not implicated in the progression of normal lower limb OA.

Hypothesis 3: Activity

Hypothesis 3a: Acetabular changes correlate positively with activity

Rejected. The current findings do not support a positive correlation between acetabular changes and occupational or habitual physical activities. This provides support for the acetabulum as an age indicator, as it indicates that occupational and

habitual physical activities do not contribute significantly to progressive acetabular changes.

CAS. Composite acetabular score data did not support a positive correlation between acetabular changes and physical activities. No statistically significant correlations were detected between CAS and MET values for occupations or habitual physical activities in females or males of any age group.

Acetabular variables. Acetabular variable data did not support a positive correlation between acetabular changes and physical activities. No statistically significant positive correlations were detected between acetabular variables and MET values for occupations or habitual physical activities in females or males of any age group. Moreover, many variables exhibited (non-significant) negative associations. Exceptions included Variable 5 (in which a majority of associations with habitual physical activities were positive in the combined-sex sample) and Variable 7 (in which all associations with occupational activities were positive in both females and males)

Variables 5 and 7 are two of the acetabular fossa variables identified as problematic by multiple authors (see discussion of *Hypothesis 2a*, above, and *Problems with Acetabular Aging*, below). The fact that they were frequently positively (if not statistically significantly) associated with MET values for occupational and habitual physical activities indicates that in addition to the scoring issues noted below, these variables may capture components of acetabular change that are explained by variation in physical activity, not by age. It is also worth noting that the acetabular variables capturing changes to the acetabular rim (Variables 1 through 3) exhibited predominantly negative associations with occupational activity values in both females and males; the

variable addressing osteophytic changes to the apex (Variable 4) also had predominantly negative associations with occupational activity in male age groups. Similarly, all four of these variables exhibited predominantly negative associations with habitual physical activity. Previous researchers have suggested that changes observed on the margins of a joint (in this case, the acetabular rim and apex) are more highly correlated with age and less highly correlated with activity than changes to the articular surfaces themselves (Weiss and Jurmain, 2007). The limited evidence for negative associations with occupational activity in Variables 1 through 4 suggests that this may be the case in the acetabulum. This provides further evidence that Variables 1 through 4 may prove most informative for age estimation, as alleged by previous authors (Calce, 2012; Mays, 2012) and discussed above (see *Hypothesis 2a*). In contrast, the fossa variables (as typified by Variables 5 and 7) again emerged as problematic.

While not statistically significant, the negative associations noted between MET values and some of the acetabular variables are worthy of further discussion. This limited support for an inverse relationship between physical activity and acetabular changes is consistent with previous work by Mays (2012) on the documented 18th/19th-Century Spitalfields sample, in which acetabular changes were more advanced in individuals working non-manual trades than in individuals who had undertaken a lifetime of manual labor. It is possible that physical activity actually improves the health of this joint's skeletal tissue, in contrast with the intuitive assumption that the daily wear and tear of bipedal locomotion adversely affects the acetabulum. In the author's professional experience, most forensic anthropology practitioners dismiss acetabular age estimation without in-depth consideration, on the basis of this very assumption. This

research suggests that their criticism of the use of a diarthrodial lower-limb joint in age estimation may be unfounded. In rejecting an overt link between increased physical activity and increased acetabular degeneration, the current research adds to the body of evidence indicating that the pelvis does not bear the brunt of the degenerative changes associated with bipedal locomotion (e.g., Jurmain, 1989; 2000), but that other body regions (i.e., the spine) may play this role (see *Other Avenues for Future Research*, below).

Lower limb effects. No statistically significant differences were detected between CAS and acetabular variable means from individuals engaging in strenuous physical activities utilizing the lower limb compared with scores from individuals who did not engage in these rigorous activities. This indicates that variation in mechanical loading of the lower limb is not contributing significantly to acetabular variation.

Hypothesis 3b: OA correlates positively with activity

Rejected. The current findings do not support a positive correlation between OA and occupational or habitual physical activities. These findings contradict the intuitive assumption that increased physical activity leads to increased OA. Factors like age seem to have more influence on OA (see discussion of *Hypothesis 2b*, above).

Occupational activities. Data from the individual joints and summary regions (overall, upper limb, and lower limb OA) did not support a positive correlation between OA and occupational activities. No statistically significant correlations were detected between OA scores and MET values for occupations in females or males of any age group. The lack of significant correlations indicates that the effect of occupational activities on OA is not as direct or straightforward as has been previously posited.

Habitual physical activities. Data from the individual joints and summary regions (overall, upper limb, and lower limb OA) did not support a positive correlation between OA and habitual physical activities. No statistically significant correlations were detected between OA scores and MET values for habitual activities in combined-sex individuals of any age group. The lack of significant correlations indicates that the effect of habitual activities on OA is not as direct or straightforward as has been previously posited.

To examine this further, the pattern of activity associations displayed by the TMJ can be viewed as a sort of control for comparison with the other joints more clearly tied to gravitational loads. If an activity-based etiology for OA is assumed, the TMJ would be expected to exhibit associations with activity that are consistently lower than those seen in the other tested joints. However, when *rho* values for TMJ OA and occupational/habitual activity MET values are compared with *rho* values from other joints, no clear pattern emerges. In several female and male age groups, the *rho* values for TMJ OA and occupation indeed fall below the 95% confidence intervals for other joints. In other female and male age groups, the associations (or lack thereof) between TMJ OA and occupational activities mirror the associations seen in other joints, as indicated by 95% confidence intervals for these joints that include the *rho* values for TMJ OA. In several age groups within the combined-sex sample, *rho* values for TMJ OA and habitual activities actually fall *above* the 95% confidence intervals for other joints. These results do not show the expected pattern for this “control joint,” providing further evidence that a loading-based OA paradigm is overly simplistic.

Lower limb effects. No statistically significant differences were detected between lower limb joint OA score means (hip, knee, ankle and lower limb OA) from individuals engaging in strenuous physical activities utilizing the lower limb compared with scores from individuals who did not engage in these rigorous activities. This indicates that variation in mechanical loading of the lower limb is not contributing significantly to lower limb OA.

Along with the above findings from occupational and habitual activities analyses, these results undermine the long-held belief that physical activity contributes directly to the development of OA. This means that while there may be some connection between activity and OA, the use of OA as a proxy for activity—still common in bioarchaeology—is not justified. This view represents a holdover from the New Archaeology-era theory that a society's mortuary data can be used a proxy for the living society, in order to make assumptions about social complexity and intra-societal roles (Binford, 1971; Saxe, 1970). Processual bioarchaeological studies often used OA to reconstruct patterns of activity within societies, correlate those activities with status and social organization, and compare levels of complexity and quality of life among different societies (e.g., Bridges, 1991; Lallo, 1973; Larsen, 1982; Tainter, 1980). The post-processual critique of the Saxe-Binford approach showed flaws in the representationist perspective and urged a particularist consideration of the historical context of each society under study (Hodder, 1982; Parker Pearson, 1982). This movement inspired bioarchaeologists to probe historical documents, utilize spatial and material cultural data, and consider multiple biological processes acting on the human skeleton in order to understand the unique contexts that result in the differential health and status of deceased individuals

(e.g., Davidson et al., 2002; Sofaer Derevenski, 2000; Williams, 2005). Still, studies recognizing the multifactorial etiology of OA, the limitations of OA for interpreting activity, and the importance of supporting biological data with documentary, demographic, material cultural, and other contextual data remain the exception, not the norm. The evidence provided by the current study that the relationship between activity and OA is not a straightforward positive correlation argues for a rejection of the representationist perspective on OA once and for all.

Perhaps most importantly, the decoupling of activity and OA suggests new avenues for medical therapies, including the prescription of strength training and exercise in order to improve the quality of life of individuals suffering from OA. Exercise can improve the quality of articular cartilage (Urquhart et al., 2011), and strength training can decrease pain and increase joint function (Hunter and Eckstein, 2009; Tak et al., 2005). This study's results provide some support for the idea that moderate, non-injurious physical activity may in fact be beneficial, not detrimental, to joint health.

However, these activity findings must be interpreted with caution. The mechanism by which physical activity may improve skeletal evidence of OA remains unknown, as moderate exercise (of the sort undertaken by OA patients—typically elderly, and possibly with compromised mobility) is unlikely to exert sufficient joint forces to impact bone. It is in fact possible that the most dramatic exercise-induced improvement in the OA patient constitutes a change in emotional state rather than bone quality. While this is perhaps equally valuable in terms of pain mitigation, it could not be captured by research on skeletal remains. Other complications of the current activity analyses are discussed below (see *Problems with Activity Analyses*).

Hypothesis 4: Obesity

Hypothesis 4a: Acetabular changes correlate positively with obesity

Rejected. The current findings do not support a positive correlation between acetabular changes and obesity. These findings lend support for the use of the acetabulum in age estimation, as age seems to have a stronger influence on acetabular changes than obesity (see discussion of *Hypothesis 2a*, above).

CAS and acetabular variables. Composite acetabular scores and acetabular variable data did not support a positive correlation between acetabular changes and obesity. No statistically significant correlations were detected between CAS/variable scores and BMI for female or male individuals of any age group.

These findings are interesting in light of the fact that the auricular surface, another pelvic age indicator, does seem to be affected by obesity, yielding statistically significantly less accurate and more biased age estimates for obese compared with normal-weight adults (Wescott and Drew, 2015). The pubic symphysis, in contrast, seems to be more resistant to obesity effects (Wescott and Drew, 2015). However, of eight commonly used age estimation methods using skeletal indicators throughout the body, the acetabulum emerges as one of the least biased and most reliable indicators for obese individuals (Merritt, 2017). This study's results support this conclusion, indicating that the acetabulum is resistant to the increasingly prevalent condition of obesity. However, the current results must be compared with those of Wescott and Drew (2015) and Merritt (2017) with caution, as the explicit effects of obesity on the acetabular age estimation method of Rissech and colleagues (2006) were not tested in the current study (see *Other Avenues for Future Research*, below).

Previous researchers have asserted that the hip is more resistant to the biomechanical effects (e.g., activity, obesity) than the more distal and exposed joints of the lower limb (Jurmain, 1977). In this study, acetabular variables indeed proved resistant to both activity and obesity. In analyses of general OA, the hip did not prove to be any more resistant to activity than the other joints of the leg; however, activity effects were limited in all joints (see discussion of *Hypothesis 3*, above). General OA analyses also revealed a less-conclusive relationship between obesity and hip OA than between obesity and the other joints of the leg (see discussion of *Hypothesis 4b*, below). Thus, this study provides evidence to support that the hip in general (and the acetabulum in particular) are resistant to activity and obesity effects. Indeed, at the completion of Hypothesis 4a analyses, the overall interpretation was that acetabular changes are more strongly correlated with age than either obesity or activity. This interpretation was tested explicitly in the Hypothesis 5a analysis (see below).

Hypothesis 4b: OA correlates positively with obesity

Supported by limited evidence. The majority of associations between BMI and OA were positive in most joints, though few were statistically significant. All associations between BMI and overall, upper limb, and lower limb OA were positive, and some were statistically significant. The strongest positive associations among the individual joints were observed in knee and ankle OA, and the strongest positive associations among the summary measures of OA were observed in the lower limb. This suggests a biomechanical mechanism for obesity-influenced OA (i.e., that high BMI leads to disproportionate loading of the joints of the lower limb). However, the more nebulous relationship between obesity and hip OA, coupled with the presence of

relatively strong BMI associations with OA in upper limb joints, suggest that this relationship is complicated, likely also including a systemic, metabolic component.

Individual joints. Almost no statistically significant correlations were observed between BMI and OA. However, one statistically significant positive correlation was present (female ankle OA in the <50-year group), and multiple additional positive correlations would have seemed statistically significant had a less-conservative testing strategy been employed. Most of these near-significant positive correlations were observed in the knee and ankle. Thus, while the evidence is far from conclusive, the knee and ankle emerged as the joints most likely to exhibit obesity-related OA in this study. Interestingly, this trend seemed to be exacerbated in females, with *p*-values for BMI and knee and ankle OA consistently lower than *p*-values for males in the same joints. This finding is consistent with previous research demonstrating that obesity has a particularly detrimental effect on the development of lower-limb OA in females (Coggon et al., 2001; Felson et al., 1988; McKean et al., 2007).

Relevant to the current acetabular research, obesity had no statistically significant correlation with hip OA. This contrasts with ankle OA and (somewhat surprisingly) shoulder OA, for which a BMI correlation approached statistical significance in 40-49-year-old combined-sex individuals. The finding of a positive association between BMI and shoulder OA suggests a systemic (i.e., metabolic) component to the relationship between obesity and OA: there seems to be more than a purely mechanical etiology for obesity-based degenerative change. The systemic effects of obesity on the development of OA may be limited, however. Where any relationships existed between TMJ OA and BMI, they were non-significant and consistently negative, indicating that metabolic obesity effects were not detrimentally impacting the health of this joint.

Summary OA. All associations between BMI and summary OA were positive. Statistically significant positive associations with BMI were observed in both overall and lower limb OA. This contributes to the interpretation (based on the above analyses of BMI and individual joint OA) that, while lower limb OA is more strongly influenced by obesity than OA in the rest of the body, obesity also has a limited effect on the joints of the upper limb. Based on the relatively strong relationship between BMI and knee and ankle OA (compared with the more tenuous relationship between BMI and hip OA), it is likely that these more distal joints are driving the positive association between lower limb OA and BMI. It is also noteworthy that in multiple regression analyses of OA's various contributing factors, BMI always remained (and was statistically significant) in minimal adequate models of the factors contributing to lower limb OA (see below discussion of *Hypothesis 5b*).

The limited evidence for a positive association between obesity and OA demonstrated in this study is consistent with recent biological anthropology findings indicating that obese individuals exhibit increased articular surface degeneration and marginal osteophytic lipping compared with non-obese individuals (Merritt, 2015). These findings are relevant to forensic practitioners, as obese, prematurely aged joints may complicate age estimation, potentially leading to the overestimation of age in obese individuals (Wescott and Drew, 2015).

These findings are also consistent with current medical interpretations of obesity as a major risk factor for joint disease (Coggon et al., 2001; Couchman, 2009; Felson et al., 1988; 2000; Felson and Zhang, 1998; Fransen et al., 2011; Mandl, 2007). Over 34% of U.S adults were classified as obese in 2011-2012; with obesity on the rise

worldwide, OA has the potential to become an epidemic (Ogden et al., 2014). The current results suggest that weight loss and exercise should be considered as important palliative interventions for OA in obese individuals.

In this study, the clearer BMI associations that emerged in the knee versus the hip are consistent with medical research indicating that there is more evidence for a relationship between increased body mass and increased risk of OA in the knee than in the hip (Felson and Zhang, 1998; Hochberg, 2004). This may be due to the lower joint forces in the hip compared with the knee (Huang et al., 2000) or the fact that the ball-and-socket joint of the hip is more anatomically protected than the musculoskeletally exposed knee and ankle and thus more shielded from dislocation or other traumatic injury. Regardless, this bodes well for the use of the hip joint in anthropological age estimation, as it appears to be relatively more resistant to the biomechanical effects of obesity than the other joints of the lower limb. Indeed, at the completion of Hypothesis 4b analyses, the overall interpretation was that OA is more strongly correlated with obesity than with activity, but more strongly correlated with age than either obesity or activity. This interpretation was tested explicitly in the Hypothesis 5b analysis.

Hypothesis 5: Relative contributions of age, activity, obesity

Hypothesis 5a: Of the above factors, age has the most influence on acetabular changes

Supported. After the process of model simplification based on the criterion of AIC, age remained in every minimal adequate linear model investigating the contribution of the various factors to CAS. Age was a statistically significant term in every minimal adequate model for both sexes. In the female sample, automated model simplification eliminated BMI, habitual activities, occupational activities, and all interaction terms from

both models, leaving age as the only remaining contributor. The resulting age-only models explained 23% and 33% of the variance in female acetabular change, respectively. In the male sample, automated model simplification eliminated habitual activities and all interaction terms from the habitual activities model, leaving age and BMI as the only contributing factors. In the male occupational activities model, automated model simplification eliminated most interaction terms, leaving age, BMI, occupation, and the BMI:occupation interaction term (only age was statistically significant). These multivariate models explained 60% and 42% of the variance in male acetabular change, respectively. Thus, age alone explained approximately one-quarter-to-one-third of the variance in CAS observed in females, and, along with BMI and other factors, age explained approximately half of the variance in CAS observed in males.

It is interesting to note, however, that over two thirds of the variance in female CAS was not explained by the female age-only models, and approximately half of male CAS variance was not explained by the male combined linear models. Additional contributing factors undoubtedly influence acetabular changes—they simply were not measured in the current study. It is tempting to speculate that the influences of heredity or hormones (particularly in the case of females) may explain some of this variance in CAS, but this is impossible to determine without additional research.

In order to illuminate the relationships among the variables further, the multiple regression data were examined graphically. When the full multiple regression models were examined via pairwise comparisons of variables, scatterplots showed strong linear relationships between CAS and age. Visualizing the linear regression models as trees indicated that age was the most important explanatory variable in every model. In

contrast, the other variables appeared to be contributing little more than noise. For example, in the male habitual activities analysis (in which both age and BMI remained in the simplified model), scatterplots showed an ambiguous relationship between BMI and CAS that appeared to be driven by outliers; in the male occupational activities analysis (in which age, BMI, occupation, and the BMI:occupation interaction remained in the simplified model), some relationships with occupation appeared to be non-linear. It is difficult to imagine how the interaction between BMI and occupation could have a significant influence on CAS when the relationships between CAS and the individual variables were so ambiguous; indeed, while this interaction term remained in the simplified occupation model, it was not statistically significant. Graphical examination suggests that some data may have been fitted in these models that do not meet the assumptions of linearity and normality; this was also indicated by the poor fit of linear models to associations tested in Hypotheses 3 and 4. Thus, there is a possibility that multiple regression is simply not an appropriate tool to analyze the current dataset.

Still, these results support the conclusions from Hypotheses 2a through 4a that, of the three factors investigated herein, age is always a consistent and significant contributor to acetabular changes. This lends support for the use of the acetabulum in age estimation.

Hypothesis 5b: Of the above factors, age has the most influence on OA

Supported by limited evidence. After the process of model simplification based on the criterion of AIC, age remained in every minimal adequate linear model investigating the contribution of the various factors to OA. In the combined-sex sample used for habitual activity analyses, as well as the female and male samples used for occupational activity analyses, age was a statistically significant term in every minimal

adequate model. However, habitual/occupational activities, and particularly BMI, also emerged through the process of model simplification as contributors to OA. Of the nine OA models (overall, upper limb, and lower limb for the combined-sex habitual activity sample and the female and male occupation samples), BMI remained (and was significant) in seven, and habitual or occupational activities remained (and were significant) in six.

In the combined-sex, habitual activities model for overall OA, automated model simplification eliminated most interaction terms from the model, leaving age, BMI, habitual activities, and the BMI:activity interaction term (only age and habitual activities were statistically significant). In the combined-sex, habitual activities model for upper limb OA, automated model simplification eliminated most interaction terms from the model, leaving age, BMI, habitual activities, and the age:activity and BMI:activity interaction terms (only age, activity, and the BMI:activity interaction term were statistically significant). In the combined-sex, habitual activities model for lower limb OA, automated model simplification eliminated habitual activities and all interaction terms from the model, leaving age and BMI as the only contributing factors. The overall and upper limb models explained over 33% the variance in OA, and the lower limb model explained 28% of the variance in OA.

In the all-female occupational activities model for overall OA, automated model simplification eliminated all interaction terms, leaving age, BMI, and occupational activities (only age and BMI were statistically significant). In the all-female occupational activities model for upper limb OA, automated model simplification eliminated all interaction terms, leaving age, BMI, and occupational activities as the contributing

factors. In the all-female occupational activities model for lower limb OA, automated model simplification eliminated occupational activities and all interaction terms, leaving age and BMI as the only contributing factors. The female overall and upper limb models explained over 40% of the variance in OA, and the female lower limb model explained 27% of the variance in OA.

In the three all-male occupational activities models (overall, upper limb, and lower limb OA), automated model simplification eliminated all interaction terms, leaving age, BMI, and occupational activities as contributing factors. The male overall and upper limb models explained approximately 40% of the variance in OA, and the male lower limb model explained 28% of the variance in OA.

As with the multiple regression analyses of CAS and its contributing factors, over half of the variance in OA remained unexplained by these combined linear models. This finding indicates that additional factors not measured in the current study contribute to OA. This is consistent with the current understanding of OA as a multifactorial disease. The presence of activity and obesity effects in the minimal adequate models—in addition to age alone—is also consistent with the multifactorial etiology of OA. It is interesting to note that in particular, BMI was a present and significant explanatory variable in every minimal adequate models explaining OA of the lower limb. This is consistent with the above results (see discussion of *Hypothesis 4b*) suggesting a largely biomechanical mechanism for the influence of obesity on OA. However, for every subsample tested (combined-sex habitual activities; female and male occupations), r^2 values were lower in the models explaining lower limb OA than in the models for overall and upper limb OA, indicating that much variance remained unexplained.

In spite of these seemingly intuitive results, the same caveats noted above for multiple regression analyses of CAS also apply to the OA regression analyses. Specifically, graphical examinations of the multiple regression data revealed strong relationships between age and OA in the various regions, but weak or problematic relationships with the other variables, raising the possibility that some data fitted in these models do not meet the assumptions of linearity and normality. Multiple regression may not be the most appropriate analytical tool for the current dataset.

Certainly, OA has a significant and consistent positive association with age, but these multiple regression results tentatively suggest that activity and obesity may also play roles in the etiology of the disease. This is consistent both with the multifactorial etiology posited by recent OA research and the strong age correlations demonstrated for OA by Hypothesis 2b of the current research. Osteoarthritic changes may be useful for age estimation, but activity and obesity likely have more of an influence on generalized OA than they do on the specific changes occurring in the acetabulum.

Ancillary Research Goals

Trauma

Scores in most acetabular variables were unaffected by previous instances of traumatic injury/surgical intervention. The fossa variables, however, proved to be exceptions to this trend. In females, Variables 6 and 7 were statistically significantly influenced by trauma to the left and right hips; in males, Variable 5 (right side) and Variable 7 (left side) showed statistically significant hip trauma effects. Since it is well established that acetabular aging methods should never be applied to hip joints showing evidence of skeletal trauma (Rissech et al., 2006), these limited trauma effects should be both unsurprising and easily avoided. Right acetabula can be substituted for left in

cases of obvious skeletal trauma, surgery, or pathology (Rissech et al., 2006), or the joint can be avoided as an age indicator altogether. Still, the apparent resistance of most acetabular variables to traumatic injuries and/or surgical interventions argues in favor of this age indicator, as there are instances in which hip trauma would be indiscernable on skeletal remains (e.g., muscular, cartilage, and connective-tissue injuries). In particular, Variables 1, 2, and 4 were resistant to trauma effects in this sample, providing further evidence that the variables of the acetabular rim and apex are more relevant to age estimation analyses than the problematic variables of the acetabular fossa.

In contrast with the acetabular variable analyses, previous instances of trauma/surgery had a statistically significant effect on OA in most joints. This finding is consistent with previous research indicating that trauma is one of the major risk factors for OA (Coggon et al., 2001; Couchman, 2009; Felson and Zhang, 1998; Fransen et al., 2011; Neyret et al., 1993; Zhang et al., 1996). While untestable in this skeletal sample, it is probable that additional traumatic injuries not directly involving the skeletal tissue (e.g., muscular, cartilage, and connective-tissue injuries) also had an effect on the development of OA.

Notably, in several tests of the shoulder, hip, and knee, OA score medians showed no statistically significant differences between affected and unaffected individuals (female right shoulder, left hip, and left and right knee; male left and right shoulder, right hip, and left and right knee), whereas in the elbow, wrist, and ankle, nearly all differences were statistically significant. This finding is particularly interesting in light of the fact that some of the highest frequencies of trauma/surgery were noted in

the hip and knee, in both females and males. This could suggest that more anatomically protected joints (e.g., shoulder, hip) are protected from the detrimental effects of trauma/surgery relative to their more anatomically exposed counterparts (e.g., wrist, ankle). However, this phenomenon is more likely due to the elimination of individuals with total prosthetic hip and knee replacements from the trauma/surgery test sample. Because of the complete nature of their joint replacement surgeries, the individuals most likely to exhibit high levels of OA could not actually be scored for OA. This may have caused hip and knee injuries and surgeries to appear less influential on the development of OA than they truly are. However, it is also possible that some features of hip degeneration are relatively resistant to the effects of trauma/surgery (see above discussion of acetabular variables and trauma).

A brief discussion of the identification of pathological hip conditions is warranted, given the possibility for DDH and other congenital conditions to affect this joint. The method of Rissech and colleagues (2006) specifically stipulates that pathological joints be eliminated from analyses. Indeed, in the current sample, any acetabula with signs of trauma or surgical intervention were omitted from age estimation analyses. However, not a single instance of DDH, Legg-Calvé-Perthes disease, or other congenital hip condition was noted in the current sample of 409 European-American individuals. This lends support to previous claims that truly pathological instances of developmental hip disorders are rare in modern populations (Bialik et al., 1999; Shefelbine and Carter, 2004). Further, while one theory of OA etiology holds that all forms of hip OA are secondary to underlying, possibly undiagnosed, hip defects (Ganz et al., 2008; Murray, 1965; Solomon, 1976), another holds that age-related change in the acetabulum as a

whole is not affected by such biomechanical factors as hip dysplasias, which tend to occur only in areas of localized hyperpressure (Rougé-Maillart et al., 2004). Concerns about DDH and other pathological conditions complicating the use of acetabular age estimation are likely largely unfounded.

Sex

Several different sex-related aging patterns emerged in the current study. No statistically significant sex differences were detected for the individual acetabular variables, supporting previous research indicating that acetabular aging processes are similar in both sexes (San-Millán et al., 2016; 2017). This indicates that forensic anthropologists need not pursue sex-specific age-estimation standards when using this joint, in contrast to other joints of the pelvis (i.e., the pubic symphysis) in which aging processes are sex-dependent (Brooks and Suchey, 1990).

However, patterns of OA did differ in females and males—typically, in the direction of higher frequencies of female OA. Females exhibited statistically significantly more OA in the TMJ and knee; the higher observed frequencies of female hip OA also approached statistical significance. Males exhibited more OA than females only in the ankle. While not expressly included in the research questions of the proposed study, biological sex likely plays an important role in joint degeneration. Previous research has also indicated the same sex disparity in OA, with females disproportionately affected (Coggon et al., 2011; McKean et al., 2007), especially when the condition is exacerbated by obesity (Felson et al., 1988) or advanced age (Felson and Zhang, 1998). In particular, previous claims of disproportionately high female knee OA (Felson et al., 1988; McKean et al., 2007) are supported by the current research. Many factors have been posited to contribute to this disparity, including differences in

anatomy, hormones, and, in the case of obese individuals, the disproportionate loading of small female joint surfaces (Weiss and Jurmain, 2007). Age further exacerbates an already-dangerous loading situation in the hips of females, for whom even normal walking produces the elevated hip joint contact stresses that can contribute to OA (Boyer et al., 2008). Indeed, the higher OA prevalence in females vs. males only increases with age (Felson and Zhang, 1998). Female knee OA can result in biomechanical gait changes not seen in male OA patients, potentially creating a feedback loop leading to abnormal loading, more OA, and further gait alterations (McKean et al., 2007). The high levels of knee and, to a lesser extent, hip OA seen in this sample's females contribute to the growing body of medical research indicating that sex-specific OA therapies are warranted.

It is important to note, however, that in both females and males, the highest frequencies of OA in this sample were observed in the hip (female mean=1.08; male mean=0.97). This finding stands in contrast with that of previous researchers, who have demonstrated that frequencies of hip OA are consistently lower than frequencies of knee OA in multiple geographically and temporally distinct populations, including North American hunter-gatherers and agriculturalists from the Archaic through Mississippian periods (Bridges, 1991; Larsen, 1982; Jurmain, 1980; 1990) as well as modern and historic U.S. individuals (Felson and Zhang, 1998; Fransen et al., 2011; Watkins, 2010). In those samples dating to pre-Contact contexts, low levels of hip OA may be due in part to the relative lack of obesity as a contributing factor to OA. In the current study, the hip proved more resistant to the effects of obesity than the other joints of the leg, yet differences in adiposity between pre-Contact and modern populations may partially

explain this hip-OA disparity. Yet, the same discrepancy in hip OA frequencies is present between this study (high levels of hip OA) and studies of other modern populations (low levels of hip OA). This could be due to the fact that the most skeletally observable characteristics of hip OA (primarily, osteophytes) do not in fact correlate well with the symptoms reported in order to generate OA frequencies for *in vivo* studies. Unlike in the knee, where osteophytes tend to impede movement of the relatively flat articular surfaces of the distal femur and proximal tibia, the ball-and-socket configuration of the hip is little affected by osteophytic activity around the joint margins. These osteophytes could, in effect, extend the labrum and enhance the joint's stability. Thus, it is possible that the high levels of hip OA observed skeletally in this sample's females and males do not correspond with actual antemortem experiences of hip disease, pain, or disability. Marginal osteophyte formation is often highly correlated with age (Calce, 2012; Calce and Rogers, 2011; Mays, 2012; San-Millán et al., 2017; Weiss and Jurmain, 2007), and it may even be part of the healthy, normal aging of this ball-and-socket joint (Solomon, 1976).

Finally, it is interesting to note that sex differences were present in the distributions for occupation MET values, with the male mean (MET=3.35) statistically significantly higher than the female mean (MET=2.47). However, no sex differences were present in the distributions for habitual physical activity MET values. It seems that while females and males in the present sample engaged in similarly vigorous recreational activities, their occupational activities still reflect a modern U.S. gender divide in which male occupations are more physically demanding.

Preservation

As expected, acetabular damage scores were lower than pubic symphysis and auricular surface scores—often statistically significantly so. These results support previous claims that the acetabulum is the best-surviving pelvic joint (Powanda, 2008; Rouge-Maillart et al., 2004; Rissech et al., 2006). Unexpectedly, however, the allegedly robust auricular surface exhibited the highest damage scores of the three joints in the oldest age group (80+ years), showing statistically significantly more damage in these elderly individuals than even the friable pubic symphysis. The acetabulum, meanwhile, proved relatively resistant to age-related changes in preservation quality. These results indicate that the acetabulum is a useful age indicator for the oldest individuals, proving statistically significantly more resistant to damage than both the pubic symphysis and auricular surface in individuals 80+ years of age at death.

The durability of the acetabulum compared with the other pelvic joints argues for its relevance to skeletal analyses—even those investigating archaeological and other detrimental depositional contexts. However, it should be noted that, with the exception of the auricular surface in individuals older than 80 years (mean=1.174), mean preservation scores were relatively low (ranging between 0.4 and 1.0) in all joints and age groups. A score of “1” in the preservational scoring system outlined in Table 5-4 indicates slight postmortem damage, with most or all of the joint’s age-related variables still observable. This indicates that in spite of statistically significant differences in preservation, age estimation analyses could proceed using the majority of the observed joint surfaces. Still, even this low-level damage has the potential to complicate age estimation. For example, an inexperienced observer might score an eroded auricular surface as an older-than-warranted age phase, confusing taphonomic porosity for the

macroporosity and irregularity characteristic of an aged joint surface. These issues of taphonomic vs. biological porosity also have more of an impact in the finer-grained, relatively fragile pubic symphysis and auricular surface than they do in the robust acetabulum, where postmortem damage is easily distinguished from the porosity sometimes associated with age (e.g., on the acetabular rim). The auricular surface, with its relatively high rates of damage in elderly individuals, may be particularly susceptible to such taphonomic damage. Thus, even low-level instances of postmortem damage can negatively impact analyses of the more fragile pelvic joints.

Study Limitations and Future Directions

Problems with Acetabular Aging

Scoring difficulties were noted for some of the acetabular rim variables (Variables 1-3). When scoring acetabular rim shape (Variable 2), in cases when osteophytes seemed to originate within the acetabular groove rather than the rim itself, it was difficult to determine where to place the calipers to measure them. When scoring acetabular rim porosity (Variable 3), there appeared to be instances when porosity occurred in this region for reasons unrelated to the breakdown of the rim. Thus, a low score might be assigned to this variable in order to address the dense and well-structured rim; but if porosity were present elsewhere in the region (e.g., below the anterior inferior iliac spine), it would not be captured. Alternately, if the presence of this non-rim porosity resulted in a higher score for Variable 3, this would not accurately portray the bone quality of the rim.

Even considering these scoring difficulties, however, the variables of the acetabular fossa (Variables 5-7) remained subjectively more difficult to score than the variables of the rim (see the discussion of *Hypothesis 2a*, above). In particular, there is

some overlap in the acetabular changes being described by Variables 6 and 7, making the scoring of one dependent on the scores assigned to the other. For example, in both Variables 6 and 7, state 5 concerns the loss of consistency in the acetabular fossa. At times, Variable 6 might appear to warrant a lower score based on other characteristics of the fossa, but if Variable 7 is scored as a 5 (indicating loss of consistency), the score for Variable 6 is also pulled up to a 5 (as lower scores for this variable explicitly state that consistency is maintained). These variables should score independent processes within the acetabular fossa, but instead, they both address fossa consistency. Another issue arises with fossa consistency (Variable 6) and the scoring of the acetabular crest (Variable 5). Often, the fossa crest resembles a dense osteophyte growing between the acetabular fossa and lunate surface. However, in some cases, it seems to "bubble" upward from the fossa with a porous appearance. It might be argued that the presence of any type of crest growing upward from the fossa pulls the score of Variable 6 up to a 5 (as the outgrowth of the crest indicates loss of fossa consistency). Yet, if the crest is formed of dense bone, this indicates that consistency is maintained. Further, in older individuals with porous, brittle bone, it can be difficult or even damaging to palpate the crest between the acetabular fossa and lunate surface—a protocol that is warranted when the crest cannot be easily seen. While most traits of the acetabulum are robust and resistant to damage, this fossa variable seems to degrade easily in older individuals with compromised bone quality. In summary, the scoring of all three fossa variables is problematic.

Recent research has emended the descriptions of the acetabular fossa variables to include clarified descriptions with additional detail (San-Millán et al., 2016), but the

descriptions for both Variables 6 and 7 still discuss fossa consistency. Future tests of this revised method will demonstrate whether the clarified descriptions are sufficient to allow independent scoring of the fossa variables. Alternately, emphasis could be placed instead on the OA-related variables of the acetabular rim—though it would be unfortunate if the unique (if poorly understood) processes occurring in the acetabular fossa had to be eliminated from the method.

Perhaps the most pressing problems with the current acetabular aging methodology concern the IDADE2 statistical program. The program's difficult-to-master, non-user-friendly interface was noted by Calce in 2012, and in the intervening years, an additional problem has arisen. The program no longer works on most computers, having been designed in 2004 for older operating systems. This makes the program even more inaccessible and inapplicable to the normal forensic or archaeological analyst. Further, although the program allows for the incorporation of prior distributions of acetabular variable scores obtained from relevant reference samples, no database of reference samples is currently available. Thus, unless the user has previously scored large numbers of acetabula from a population independent of the one currently under study, the user must undertake a tautological age estimation analysis (like the one described herein) or decide not to use the program. If the program is not updated to be compatible with modern computer software and to include acetabular scores from multiple skeletal samples, the method will become obsolete, and all of its Bayesian potential for age estimation informed by relevant prior distributions will be lost. In the future, the researcher hopes to adapt the IDADE2 code into a freely accessible *R* script and plans to share the current acetabular variable data with all

potential users. It is hoped that this work can be accomplished in collaboration with some of the original authors of the acetabular aging method (Rissech et al., 2006).

Problems with OA Analyses

Only two instances of ankylosis were noted in the sample of 409 individuals—in both cases, ankle fusion secondary to surgical intervention. This means that only two out of 5,726 analyzed joints received a score of "3" in the Jurmain (1990) ordinal scoring system (Table 5-3 and Figure 5-3). It seems that a revision of the scoring system designed to capture more variation within non-ankylosed joints would be a better use of the limited ordinal scores within the system. For example, score "2" could be revised to comprise severe OA without eburnation, score "3" could represent severe OA with eburnation, and an additional category "4" could be added to capture the rare instances of joint ankylosis. This might allow improved discrimination among OA patients of varying severities.

Alternately, a continuous scoring system could be developed (e.g., incorporating measurements of osteophyte height, pore diameter, or the dimensions of a region of eburnation). Continuous data are easier to analyze statistically, while discretization forces data into categories that may not adequately describe them. That one score in the four-score ordinal system of Jurmain (1990) is largely useless speaks to the larger problem in age estimation of attempting to analyze fine-grained biological processes using gross-grained analytical techniques. The sensitivity of ordinal scores to identifying clinical markers of OA and the correspondence of those scores with actual OA symptoms in living patients remain unknown. In absence of the articulated-joint radiographs used in clinical diagnoses of OA (Kellgren and Lawrence, 1957), biological anthropologists rely on the presence of macroscopically observable osteophytes,

eburnation, and porosity to diagnose OA in a deceased individual. Yet, the correlation of these indicators with OA symptoms is not clear even in the clinical setting, where radiographic evidence of the disease does not always correlate with actual symptoms of disability (Loeser, 2007; Rothschild, 1997). Still, skeletal methods that better approximate the clinical assessment of OA could be explored, in order to enable comparisons between the anthropological and medical literature. For example, in lieu of observing and scoring the separate skeletal components of a joint, an anthropologist could approximate the joint's articulation, take a radiograph, and apply a modified version of the Kellgren and Lawrence (1957) system (i.e., focusing on skeletal indicators like sclerosis and bone contour deformity).

Problems with Activity Analyses

The activity coding system employed in the current study is subject to limitations similar to the OA scoring systems discussed above. Simply, it is unclear how well (or poorly) averaged MET values from the Compendium of Physical Activities reflect the actual energetic exertions of the deceased individuals in the *Bass Collection*. The Compendium of Physical Activities was designed for use in self-reported activity surveys in which participants quantify their daily activities in a standardized fashion. In contrast, the occupational and habitual activity data tested in the current study were non-standardized—captured by open-ended prompts on the *Bass Collection* skeletal donor forms titled, "usual (life-long) occupation" and "habitual activities (i.e., jogging, repetitive motions, life-long occupation activities, etc.)," respectively.

It is also difficult to assess whether averaged MET values can differentiate among real, *in vivo* activity differences to a degree that would be meaningful biomechanically and in terms of skeletal response. Metabolic equivalency values reflect

energy exertion, not skeletal biomechanics, so the use of MET data as a proxy for the actual joint forces to which an individual's joints were regularly subjected may be questioned. This is particularly true for the habitual activities dataset. Unlike occupational activities (which can be interpreted as subjecting joints to activity-related stresses for an average of eight hours per day), even strenuous, high-intensity habitual activities (e.g., running) were likely only undertaken for an hour or less each day. Further, reported activity data can be subjective and inaccurate. It is possible to imagine a scenario in which a donor family recalls a loved one's military service and reports his habitual activities as "physical training," disregarding the fact that the individual has lived a sedentary lifestyle since his retirement 30 years previously. It is hoped that the relatively large sample size employed herein provides resolution sufficient to swamp such problematic scenarios with valid data points; however, the actual resolution of this activity analysis remains unknown.

It should also be stated that the steps taken in the current study to control for the effects of age in analyses of activity (restricting testing to within age-matched subsamples) reduce the statistical power of those analyses. This increases the chances of achieving results that support the null hypotheses of no occupational or habitual activity effects. In turn, this makes the results of these analyses less directly comparable with the analyses of Hypotheses 1 and 2, which include larger sample sizes. The study design has, in effect, stacked the deck in favor of rejecting activity effects. For all of these reasons, this study's conclusions about physical activity (i.e., its lack of correlation with acetabular changes and OA) should be considered tentative.

Problems with Obesity Analyses

Like the occupational and habitual activities discussed above (see *Problems with Activity Analyses*), BMI data for the sampled individuals were largely self-reported. For most individuals in the *Bass Collection* sample (*n*=375), BMI was calculated from reported height and weight data. However, for a minority of individuals, BMI could also be calculated from measured cadaver weights (*n*=25). In 12 of these 25 individuals, BMI category changed when the measured body weight figures were added to the calculation. This illustrates the fact that an individual's BMI might change (either positively or negatively) between the time of completion of skeletal donation paperwork and their actual time of death. This introduces a level of uncertainty into the analysis of BMI associations. However, it could be argued that the donor individuals' reported BMI likely reflects their obesity status during the majority of their life, whereas their cadaver weight reflects their obesity status at death, often after long-term illnesses with wasting effects. Thus, the use of reported, presumably *in-vivo*, BMI rather than time-of-death BMI can be justified.

Finally, the same statistical testing problem reported above for activity analyses holds true for this study's obesity analyses. Restricting testing to within age-matched subsamples makes the current obesity results less comparable with the larger-sample-size analyses of Hypotheses 1 and 2 and increases the chances of rejecting obesity effects. The fact that the study provides some evidence in favor of obesity effects on OA suggests that associations are being detected even given the smaller sample sizes; yet, future studies could attempt to replicate these results across larger, age-matched samples that are more comparable with the overall sample size of the current study.

Other Avenues for Future Research

The primary goals of the proposed research were to illuminate the nature of the progressive changes observed in the human acetabulum, to determine their relationship with OA, and to identify the ways in which three factors—age, activity, and body mass—contribute to the progression of OA in general and acetabular changes in particular. As noted above, however, OA is a multifactorial disease, and other factors (e.g., heredity, nutrition, bone density, biological sex) undoubtedly contribute to its development and progression. These other factors were outside the scope of the current study. Specifically, the effects of ancestry (as a proxy for genetic predisposition) could not be studied in the current research, due to the makeup of the documented *Bass Collection* sample (see discussion in Chapter 5). Populations of different ancestral affinities also may progress through the acetabular aging variables (Rissech et al., 2006) slightly differently, or even express different patterns of acetabular change (Rissech et al., 2007). The absence of ancestry data is a limitation of the current study, one that should be remedied in future studies of OA and acetabular change.

Future studies should also test for correlations among acetabular changes and vertebral OA. Like the synovial joints of the appendicular skeleton, vertebral synovial joints (i.e., superior and inferior facet articulations) are subject to the development of OA. Superior and inferior vertebral centra can also develop a form of OA known as vertebral osteophytosis (VOP). Differentiated from normal OA due to the fact that vertebral centrum articulations are fibrocartilagenous, not synovial joints, VOP nonetheless has a similar etiology to OA *sensu stricto* (Jurmain, 1990). Vertebral OA data are key to investigations of possible correlations between activity and OA: evidence from primate and other mammal studies suggests that the spine, not the

pelvis, suffers the majority of the degenerative changes associated with bipedal locomotion (Jurmain, 1989; 2000), and this should be tested in future research.

The link between age and shoulder OA demonstrated by the current study is consistent with recent work indicating a strong positive correlation between shoulder-joint OA and age at death (Brennaman et al., 2016). These authors have suggested that incorporating OA scores into multifactorial methods of age estimation may be a productive future research avenue (Brennaman et al., 2016). The positive age correlation of OA with most joints in this study (except the ankle) indicates that this may be the case. Acetabular changes may be degenerative, but OA is also strongly correlated with age. Future research could refine current multifactorial methods to include multiple skeletal indicators of OA—perhaps focusing on the shoulder and hip. Previous research has indicated that factors like obesity disproportionately influence OA in relatively anatomically exposed joints like the knee (Felson et al., 1988), whereas the protected ball-and-socket joints of the hip and shoulder are more shielded from the adverse effects of injury and instability (Jurmain, 1980; Jurmain and Kilgore, 1995). In keeping with these findings, the current research indicates that the shoulder and hip may be particularly productive regions of focus for OA-related age estimation.

This study highlights the need articulated by Boldsen and colleagues (2002) for further investigation into the skeletal changes associated with advanced age. Accurate and precise elderly age estimates, while desirable, simply may not reflect the biology underlying the variable processes of skeletal degenerative change. However, forensic anthropology methods must keep pace with ever-increasing modern human lifespans, and paleodemographers must also combat the notions of short life expectancies

conveyed by the previous application of flawed age-estimation methods (Howell, 1982; Lucy et al., 2002). Work toward better skeletal age estimates for elderly individuals is warranted. Explicit tests of the effects of obesity on the accuracy of acetabular age estimates should also be considered (*sensu* Wescott and Drew, 2015).

Finally, a caveat about the current study protocol should be noted. While this research demonstrated no evidence for a metamorphic etiology of acetabular age change, it remains to be seen whether the current testing protocol (i.e., comparing acetabular changes with OA in other regions of the body) is sufficient to differentiate metamorphic from degenerative changes. This lack of certainty is due to the fact that the protocol has never been applied to joints where known metamorphic changes occur. If the same protocol were applied to the pubic symphysis, where the ventral rampart is an acknowledged site of skeletal metamorphosis, it is possible that the results would be similar—also showing a positive correlation with OA. Future research could score pubic symphyseal changes in the same sample of 409 individuals and apply the same testing protocol in order to ascertain if its resolution is sufficient to detect metamorphic change in skeletal articulations.

Final Thoughts about Metamorphic and Degenerative Change

One of the main goals of this dissertation was determining whether the progressive changes of the acetabulum constituted metamorphic or degenerative change. Skeletal metamorphoses, later-in-life skeletal changes that have been shown to correlate highly with age (e.g., the formation of the pubic symphysis ventral rampart) are typically considered to be more informative for age estimation than degenerative changes (e.g., OA), which are believed to be subject to multiple other influences that preclude a straightforward age correlation. This study has indicated that in the case of

OA, one of these influences (activity) has no discernable impact on the progression of the disease, and another (obesity) has a limited positive correlation. In contrast, however, the positive correlation between age and OA is strong.

This suggests that the often-touted dichotomy between metamorphic and degenerative change (and their probative vs. non-probative value for age estimation) may be a false one. If both metamorphic and degenerative changes are strongly correlated with age, why should the former be given preference in age estimation studies? After all, while some regions of metamorphic change enable reliable age predictions (at least in younger individuals—e.g., the pubic symphysis), others have been discounted as age-relevant for decades (e.g., the cranial sutures; Stewart, 1979). In the pubic symphysis, the ventral rampart forms in middle adulthood—a small buttress of bone along one side of the symphyseal face. Perhaps it is the fact that this ramp-shaped formation represents bone *production*, rather than *destruction*, that makes it seem more relevant to aging. Yet, there is an anabolic component to "degenerative" OA as well; abnormal subchondral bone production, sclerosis, and the formation of marginal osteophytes are integral facets of the disease in addition to the destructive processes of cartilage degradation and bone erosion. There seems no legitimate basis on which to tout "metamorphic" bone production as "good" and dismiss "degenerative" bone production as "bad."

Further, the very assumption that bone production represents a beneficial process and bone destruction a maladaptive process must be questioned. If bone is building a structure (e.g., a ventral rampart or osteophyte) where one is not functionally necessary, it could be argued that this process is maladaptive regardless of whether the

formation is classified as metamorphic or degenerative. For the purposes of age estimation, this dichotomous distinction may be meaningless. The fact remains that metamorphic change is a normal part of the aging of the human skeleton, while OA is a pathological condition for which age is a major predisposing factor. However, if this pathological process is as highly age correlated as this study suggests, then it too should be considered in skeletal estimates of age.

Indeed, it seems imprudent to dismiss degenerative processes like OA as "ancillary (if not a last resort)" to other skeletal indicators of age, as biological anthropologists have done for decades (Aykroyd et al., 1999, p. 59). Both metamorphic and degenerative skeletal changes are highly age correlated. Both should be incorporated into skeletal age estimation methods. A potential venue for this incorporation might be the multifactorial Bayesian age estimation methods that are currently gaining support among forensic anthropologists. Recent research indicates that degeneration-based age estimation may indeed be productive (e.g., Brennaman et al. 2016; Falys and Prangle, 2015). Certainly, if ambiguous age indicators like the auricular surface continue to receive attention in the field, OA also deserves its due.

Summary

This chapter contextualized the study results with a discussion of their implications. While acetabular changes were found to be degenerative, they proved to be highly correlated with age and relatively resistant to the effects of activity and obesity. In other joints of the body, OA was also highly correlated with age and relatively resistant to the effects of activity, though limited evidence emerged for a positive association with obesity. In general, these findings provided support for the use of the acetabulum in age estimation, despite the similarities between acetabular

changes and the degenerative processes of OA. In the medicolegal context, these results validate the utility of acetabular age estimation methods and argue for their continued refinement. In the bioarchaeological context, the lack of correlation between OA and physical activity argues for the emendment of an overly simplistic paradigm of OA that has long been used to link observations of OA with interpretations of subsistence and other activities. This chapter also considered limitations of the current study and suggested directions for future research—among them, a rejection of the metamorphic-degenerative dichotomy and an incorporation of OA into multifactorial methods of age estimation. The next chapter summarizes the dissertation's conclusions.

CHAPTER 8
CONCLUSIONS

Some biological anthropologists consider the acetabulum to be a robust and age-informative joint that exhibits unique metamorphic changes. For others, it is "just another diarthrodial joint"—its utility for age estimation undermined by its involvement in bipedal locomotion and body mass support, exhibiting degenerative changes that merely constitute osteoarthritis. This study endeavored to reveal the nature of acetabular changes, their relevance to age estimation, and their relationship with factors including OA, activity, and obesity.

In this sample of 409 European-American females and males, progressive acetabular changes (as indicated by individual variables and overall CAS) correlated positively with OA in most joints and body regions. This indicates that a metamorphic origin for acetabular age changes is unlikely, and that they are instead linked with the largely degenerative processes of OA. However, there seem to be systemic (i.e., senescence-related) rather than purely functional (i.e., biomechanical) components to the age-progressive processes occurring in the hip, and the strong age correlations revealed by subsequent testing suggest that the metamorphic vs. degenerative dichotomy may be a false one.

In this study, age estimates generated with the acetabular aging method of Rissech and colleagues (2006) correlated positively with age in both sexes and all age groups—with the exception of the youngest female individuals, for whom low representation in the reference sample may have hindered correct age classifications. This indicates the general applicability of the method for estimating age, even in the traditionally problematic category of elderly individuals. Individual acetabular changes

also correlated positively with age, indicating their relevance for age estimation despite their primarily degenerative etiology. In particular, acetabular rim and apex Variables 1 through 4 (Rissech et al., 2006) seemed to be valid age indicators.

General OA in various joints and body regions also correlated positively with age, but slightly stronger age associations with acetabulum-specific measures versus general hip OA suggest that there may be some age-related variation scored by these measures that is not captured by normal OA scoring. Even within the realm of general OA, the hip and shoulder emerged as highly age-correlated joints, while the other joints of the lower limb exhibited weaker age correlations.

Occupational and habitual physical activities did not have a significant effect on acetabular changes in this sample—even when those physical activities expressly involved the lower limb. Rather than supporting the idea that the daily wear and tear of bipedal locomotion causes premature acetabular aging, acetabular variables appeared resistant to mechanical loading. However, these activity findings must be considered tentative, due to the difficulties of assessing the metabolic and biomechanical impacts of occupational and habitual physical activities in a skeletal sample of donated remains.

Results of analyses investigating OA and physical activity also diverged from the direct correlation once posited by biological anthropologists and medical researchers between activity and OA. No statistically significant correlations with occupational and habitual physical activities were detected for OA in any joints—including the lower limb joints involved in bipedal locomotion. Further, engaging in strenuous physical activities with the lower limb was not associated with statistically significant differences in scores for hip OA, knee OA, ankle OA, or summary measures of lower limb OA. While these

activity findings must also be considered tentative, they are consistent with recent medical research on the benefits of physical activity for aging individuals. These results suggest that the interactions between activity and OA are more complicated than previously thought.

Obesity did not have a significant effect on acetabular changes in this sample. This indicates the relevance of the acetabulum for age estimation even in today's increasingly obese populations.

In terms of OA, however, there was some evidence for a general correlation between increased BMI and increased OA. This trend was particularly pronounced in the lower limb (more strongly so in the knee and ankle than in the hip). This indicates a predominantly biomechanical etiology for the influence of obesity on OA, though a systemic, metabolic component of obesity may be contributing to OA in the joints of the arm. Frequencies of OA were also generally higher in females than in males, suggesting that sex-specific OA therapies are warranted.

This study demonstrated the robusticity of the acetabulum, showing it to be more resistant to postmortem damage than the other pelvic joints commonly utilized in skeletal age estimation—particularly in individuals of advanced age. It indicated that most acetabular changes are resistant to the effects of previous injury and surgical intervention—with the exception of the variables of the acetabular fossa, which proved more problematic than variables of the acetabular rim and apex in multiple analyses. This study also indicated that instances of developmental pathologies rarely complicate the process of acetabular age estimation.

The degenerative age changes of the acetabulum proved resistant to activity and obesity effects, arguing for their utility in age estimation. General hip OA also proved more resistant to the effects of obesity than the other joints of the lower limb, suggesting that even those acetabular variables capturing general hip OA processes may be useful for age estimation. In multiple regression analyses, age was the major contributing factor explaining both acetabular changes and OA, though BMI made a statistically significant contribution to acetabular changes in one minimal adequate model, and multiple other contributors remained in the simplified models for OA. These findings are weakened by the fact that multiple regression may not be well suited to analyze this particular dataset. Still, the tentative conclusion from multiple regression analyses is that OA—and particularly acetabular changes—are predominantly age-related phenomena.

The above findings undermine the metamorphic-degenerative dichotomy in skeletal aging and validate the acetabulum as an age indicator. In essence, this research suggests that the hip is not "just another diarthrodial joint." If an analyst is choosing an age indicator among the mobile joints in the human body, the hip may be the best choice. This study has also highlighted the influence of obesity on OA (particularly in the knee and ankle), indicating that when forensic anthropologists use generalized OA (rather than specific acetabular analyses) to estimate age, they should take into account the potential for obesity to complicate their age estimates. Traumatic injuries and surgical interventions also had a positive relationship with the development of OA in this sample, indicating that anthropologists should use caution when interpreting age based on joints showing evidence of antemortem trauma.

This dissertation research has provided the biological anthropology community with a more complete understanding of the age-related changes occurring in the acetabulum, their relationship with OA, and the impact of major contributing factors. All of these contributions advance the science of adult age estimation, in accordance with the recommendations of the NAS (NRC, 2009) and the *Daubert* decision (Daubert v. Merrell Dow Pharmaceuticals, 1993). This research also opens the door for continued testing and refinement of acetabular aging methods for additional (i.e., non-European) populations, which will ultimately lead to the successful medicolegal identification of unknown adult individuals.

However, it is hoped that this more nuanced understanding of the relationships among acetabular changes and OA, age, activity, and obesity will contribute not only to forensic anthropology, but also to bioarchaeological research and the practice of medicine. This research has confirmed that the etiology of OA is indeed multifactorial. This study demonstrated flaws in the traditional bioarchaeological use of OA as a proxy for habitual and occupational physical activities. Future bioarchaeological studies of OA should consider the effects of age, trauma, and, where relevant, obesity, in order to portray the lifestyles of study populations accurately. The correlations shown in this study between obesity and lower limb OA also represent a valuable contribution to medical understandings of OA development and progression: with an increasingly obese human population, OA has the potential to become an epidemic. However, this study's results suggest attainable interventions for both obesity and OA, in the form of weight loss and exercise.

A better understanding of the roles played by age, activity, and obesity in the degeneration of the hip has the potential not only to benefit the identification and interpretation of the dead, but also to improve health outcomes and interventions for the living.

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BIOGRAPHICAL SKETCH

Allysha Winburn holds a B.A. in archaeological studies from Yale and an M.A. in anthropology from New York University. She served as a lecturer in anthropology at the University of New Hampshire from 2016-2017, and she has also taught multiple anthropology courses at the University of Florida (UF) as a graduate instructor. She will begin a position as an assistant professor in anthropology at the University of West Florida in fall 2017. She worked previously as a forensic anthropology analyst at UF's C.A. Pound Human Identification Laboratory (CAPHIL) and as a GS forensic anthropologist at the Joint POW/MIA Accounting Command (JPAC). Her laboratory managerial experience includes serving as Quality Assurance Coordinator at the CAPHIL and interim project manager of the JPAC's "K-208 Project" (a massively commingled assemblage of human remains dating to the Korean War). Her research has focused on pelvic methods of age estimation for nearly a decade. Other research interests include ethics, error, and objectivity in forensic anthropology, and the medicolegal interpretation of ritual remains used in African diaspora religious practices.