

AN INVESTIGATION OF THE BIOMECHANICAL IMPLICATIONS
OF LOWER LIMB FRACTURES AND LEG LENGTH DISPARITY

by

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ABSTRACT

One of the primary goals of biological anthropology is to develop an accurate understanding of human anatomy, health, disease, and injury in both modern and archaeological populations. Paleopathological analyses are a fruitful means of identifying disease and injury in skeletal assemblages, but the individual long-term biomechanical effects associated with pathological conditions have not yet been adequately explored in the literature. Leg fractures are a common pathological condition in both modern and archaeological populations, the effects of which may alter the biomechanics of gait. A growing body of clinical literature demonstrates that abnormal ambulatory function may have far-reaching effects in the rest of the body.

To assess the long-term consequences of pathological conditions of the lower extremities, the relationship between lower limb long bone fracture occurrence, incidence of leg length disparity (LLD), and temporomandibular dysfunction (TMD) was analyzed. A total of 56 adult individuals (29 fractured, 27 unfractured) from the Hamann-Todd Osteological Collection (HTOC) at the Cleveland Museum of Natural History (curated between 1912 and 1938) were examined in this study. In total, the sample consisted of 37 males and 19 females (ages 25-76) of either black or white ancestry. LLD was assessed by taking standardized measurements of the lower limb long bones. TMD was analyzed by scoring the presence and severity of osteoarthritis of the temporomandibular joint (TMJ OA), dental attrition, and antemortem tooth loss.

Kendall's Tau correlation statistics were used to assess morphological integration between all unique pairwise combinations of lower limb and jaw measurements among unfractured and fractured groups. Results indicate that several measures of LLD and jaw dysfunction are correlated differently in the unfractured and fractured groups. Comparisons of

the All Unfractured and All Fractured groups most often showed higher absolute correlation values in unfractured individuals. Samples were also subdivided and compared based on known sex. Significant differences in patterns of morphological integration were observed between male and female sub-samples. Significant correlation values were almost always higher in the unfractured sample than in the fractured sample. Females, however, demonstrated both significant increases and significant decreases in absolute correlation values when comparing fractured and unfractured samples. Thus, patterns of significant differences in morphological integration between the lower limbs and jaw differ for males and females, with fairly consistent decreases in integration strength in the former and a mixed pattern of integration strength increases and decreases in the latter, when a leg fracture is involved. It is argued that these differences are explained by fundamental sexually dimorphic morphological and kinematic differences between males and females, such that fractures resulting in LLD affect the two sexes differently. Gendered lifetime social experiences and activity patterns may also explain the different male and female patterns identified in the analysis. These insights are applied to larger anthropological questions of social identity and the long-term injury experience.

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LIST OF ABBREVIATIONS

ANOVA	Analysis of Variance
HTOC	Hamann-Todd Osteological Collection
LLD	Leg Length Disparity
MANOVA	Multivariate Analysis of Variance
TEM	Technical Error of Measurement
TMD	Temporomandibular Dysfunction
TMJ	Temporomandibular Joint
TMJ OA	Osteoarthritis of the Temporomandibular Joint

CHAPTER 1: INTRODUCTION

The discipline of biological anthropology can be defined as the study of human biology as it relates to human behavior and culture. Biological anthropologists have long been concerned with understanding pathological conditions and the human injury experience. However, current paleopathological literature has tended to focus on the immediate medical effects of pathological conditions such that the long-term consequences of skeletal injury are understudied. An accurate understanding of the holistic effects of bodily injury contributes to improved paleopathological analyses of past human cultures, the identification and interpretation of victims in forensic contexts, and more sophisticated methods for addressing the medical treatment of skeletal injury in modern populations.

A holistic understanding of the entire injury experience in both the short and long term is an important goal of many biological anthropologists working at the intersection of anthropological and medical science. Since biological anthropologists are primarily interested in understanding how prehistoric and modern individuals have dealt with pathological conditions, it is important to consider the entire injury experience, from the moment a traumatic injury is sustained, throughout the healing process, and into the years following the injury. Depending on one's access to necessary medical care, an individual who sustains a traumatic injury may be able to mitigate the pain and physiological consequences such that the injury heals fully and the individual returns to normal function. In other cases, an individual may not have access to the appropriate medical care, or the injury may be too severe to heal effectively, resulting in permanently altered physiology and function. Individuals who experience long-term consequences of injury – such as diminished range of motion or locomotive capability – may be

unable to return to normal function and may alter their movement and behavior to compensate for the permanent condition. In certain cases, this may preclude them from participating in certain aspects of society, which may affect their social identity and status. Such cases have not yet been thoroughly explored in the literature and warrant further investigation.

Fractures are a common traumatic skeletal injury whose long-term effects have not yet been adequately explored. After a fracture occurs, the healing process and compensatory locomotor changes may result in permanently altered bone morphology. Because the human body is morphologically integrated, such an alteration to the integrated skeletal system may cause secondary pathological conditions to occur in regions outside of the local injury site. If fractures do in fact result in ancillary pathological conditions, fractured individuals (in both present and past populations) may undergo a more complex injury experience than was previously considered. Alterations in morphologically integrated structures can be quantified and explored using a correlation analysis (Olson and Miller, 1958).

Fractures of the lower extremity are a common pathological condition in both archaeological and modern populations, and their proximate physiological effects are well documented (Wedel and Galloway, 2014). Because the lower limb bones are instrumental in human ambulation, femoral, tibial, and fibular fractures represent a significant biomechanical disruption to the integrated skeletal system. The fracture healing process frequently results in a shortening of the affected bone, and in the case of leg fractures, such altered limb morphology may result in leg length disparity (LLD), which can cause abnormal gait (Hong and Bartlett, 2008; Perry, 1992; Kaufman et al., 1996). Therefore, shifts in posture resulting from altered weight distribution along the axial skeleton may have serious biomechanical consequences

extending to correlated regions of the skeleton, and these disruptions may manifest as ancillary pathological conditions.

The purpose of this thesis is to test a hypothesis and to determine the extent to which leg length disparity of the appendicular skeleton can cause secondary pathological conditions to occur in the mandibular region of the craniofacial complex. This analysis focuses on the relationship between leg length and the stomatognathic system by assessing the correlation between leg fractures and incidence of jaw dysfunction at the temporomandibular joint. This study addresses the following questions: 1) How are the leg bones and mandible morphologically integrated? 2) How are patterns of integration between these skeletal elements altered when lower limbs are fractured? I hypothesize that traumatic injuries disrupt patterns of morphological integration by altering limb length, resulting in cascading effects in other regions of the body such as the mandible, which are integrated with limb form. To assess these questions, a sample of modern adult fractured and unfractured individuals with known demographic information will be analyzed. Because the sample is comprised of individuals of known age and sex, it will be possible to control for these confounding factors.

Prior to testing this hypothesis, a brief theoretical and methodological background is given. Chapter 2 of this thesis reviews the bioarchaeological and clinical literature on injury, impairment, and disability and provides a brief overview of skeletal biomechanics. First, normal human ambulatory function is considered, followed by a discussion of pathological function and the implications of leg length disparity for the human gait. The concept of morphological integration is then discussed, with a particular focus on the holistic effects of pathological gait for the rest of the body. Clinical literature describing a link between the leg and jaw is reviewed and discussed. Chapter 3 outlines the materials used to test the research questions as well as the

methods of measurement and analysis. Chapter 4 describes the results of the analysis, including the incidence of lower limb fractures, LLD, and temporomandibular dysfunction. Special attention is paid to the statistically significant correlation patterns in all analyzed groups and the implications for patterns of morphological integration across samples. Chapter 5 provides a discussion of the patterns identified in the analysis, with particular emphasis on the differences observed between males and females. Possible explanations for the observed sex differences are enumerated, and their implications for modern and archaeological analyses of the long-term injury experience are discussed. The limitations of the study are also described. Chapter 6 provides a summary of the analysis and suggests directions for future analyses.

CHAPTER 2: LITERATURE REVIEW

A primary goal of those who study biological anthropology is to develop a thorough understanding of the human skeletal system and the role of the body in human behaviors and experiences. A major disciplinary focus is paleopathological analysis, which seeks to identify and understand human health, nutrition, disease, and injury and how pathological conditions may affect an individual's well-being and ways of life in both archaeological and modern populations. Questions pertaining to the long-term experience of injured individuals have recently come into focus within the bioarchaeological literature. In concert with the theoretical shift toward understanding past perceptions of social identity (Knudson and Stojanowski, 2008; Agarwal and Glencross, 2011), bioarchaeologists have begun to address the identification, interpretation, and treatment of injured individuals in prehistoric contexts.

Injury, Impairment, and Disability

Central to a discussion of the human injury experience are the concepts of physical impairment, disability, and care. These terms are often conflated in the literature and popular media, and it is important to define and distinguish between them for an accurate understanding of the long-term effects of skeletal trauma. The term "impairment" refers to a physical injury or other pathological condition (either temporary or long-term), which may preclude some aspect(s) of normal function. "Disability," on the other hand, is a social category used to describe injured or physically impaired individuals who are perceived to be unable to participate fully in society; the concept of "disability" therefore varies drastically from culture to culture and is difficult to identify archaeologically (Scheer and Gross, 1988). "Care" refers to the physical assistance given

to an impaired individual, such as medical treatment, physical therapy, or hygiene maintenance (Tilley, 2015:3).

Since the discipline's inception, bioarchaeologists have debated whether or not disability status and care can be identified in the archaeological record. Early disability studies of the 1970's and 1980's pointed to the long-term survival of seriously injured and impaired Neanderthals in the archaeological record as evidence of care and compassion in prehistoric societies. Rowlett and Schneider (1974:50), for example, analyzed the remains of the "Old Man" from La Chapelle-aux-Saintes and concluded that his physical ailments rendered him entirely incapable of taking care of himself, such that he would have been dependent on the help of others in his community until his death. Similarly, a number of studies have pointed to Shanidar I, an adult male Neanderthal from Iraq who survived several fractures and degenerative joint disease, as an example of a "crippled" individual who survived only because of the compassion and care of others (Solecki, 1971; Trinkaus, 1983). Similar arguments have been made about Romito 2, an individual with dwarfism from Calabria, Italy, and a young male individual with spina bifida from the early archaic site of Windover, Florida (Fruyer et al., 1987; Dickel and Doran, 1989).

While many studies have concluded that the survival of injured individuals in the archaeological record demonstrates compassion and care for disabled individuals, not all bioarchaeologists are convinced. Critics have pointed to the difficulties of distinguishing between physical impairment and disability, arguing that disability is a social construct that cannot be assessed through archaeological analysis. In response to Rowlett and Schneider's interpretation of the "Old Man" from La Chapelle-aux-Saintes, Tappen (1985) argued that the level of care that this individual would have required was overstated. After re-examining the

osteological evidence, he determined that the individual would have been capable of performing a number of tasks for himself (such as chewing his own food) and would not have relied as heavily on others as Rowlett and Schneider (1974) suggested. Dettwyler (1991) is perhaps the strongest critic of the compassion argument. In her seminal 1991 article “Can Paleopathology Provide Evidence for ‘Compassion’?” Dettwyler strongly condemns the practice of interpreting compassion and care from skeletal remains. She argues that archaeologists can draw conclusions about physical impairment from skeletal remains but that it is impossible to infer disability status and the extent to which an impaired individual could or could not contribute to society (Dettwyler, 1991).

More recently, however, this question has come to light again, with renewed interest in the concepts of disability, compassion, and care. Tilley (2011, 2013, 2015) has published extensively on what she calls the “bioarchaeology of care,” arguing that archaeological evidence of the long-term survival of injured individuals demonstrates that prehistoric populations showed compassion and care toward impaired members. Tilley argues that these situations are often overlooked in the archaeological literature and when they are addressed, they are analyzed as isolated case studies without consistent methodological or interpretive frameworks. To address this issue, Tilley has proposed the “bioarchaeology of care approach,” which delineates four stages for assessing disability and caregiving in archaeological assemblages: 1) description of an individual’s remains, pathological conditions, environment, and mortuary treatment, 2) determination of the pathological condition’s clinical impact on function and quality of life, 3) assessment of what care may have been administered to the individual based on the individual’s needs and the availability of resources in the environment, and 4) interpretation of the implications of the administered care for social practices, relations, and identity (Tilley, 2015:5-

6). Tilley and colleagues have also published this methodology in an online web application titled “The Index of Care” (www.indexofcare.org), which is designed to lead bioarchaeologists through the four stages for identifying and interpreting care in skeletal assemblages (Tilley and Cameron, 2014).

Tilley (2015) has recently responded to Dettwyler’s claims in detail, arguing that Dettwyler’s view is misguided and ultimately harmful to bioarchaeological disability studies, as it has discouraged research exploring disability, care, and identity. In light of this debate, it is clear that questions pertaining to the treatment and long-term prognosis of physically impaired individuals in past societies have not been sufficiently explored. Furthermore, issues relating to the diagnosis, treatment, and long-term prognosis of skeletal injury remain relevant today. Further research is needed to build a more accurate understanding of the proximate and ancillary effects of skeletal injury in the body and the experience and outlook of impaired and disabled individuals.

A recent study by Lovell (2016) demonstrates how current bioarchaeological analyses approach the assessment of injury, impairment, and social identity. Lovell (2016) analyzed the remains of an elderly man from the ancient Roman site of Erculam in Campania, Italy (1st-2nd century CE). The individual exhibited a healed femoral neck fracture that caused a shortening of the right leg. Osteological analysis of the lower limbs showed associated osteoarthritic lipping and eburnation of the right patella, first metatarsal, and first proximal phalanx. According to the author, these osseous changes indicate that the individual walked with a pronounced limp to compensate for the different lengths of his two legs. Furthermore, after determining that the individual was buried in a communal cemetery alongside and in the same manner as non-impaired individuals, Lovell determined that the individual was likely perceived to be mobility-

impaired during life, but was not considered “disabled” by his society (Lovell, 2016:94).

Lovell’s analysis demonstrates that although it may be impossible to determine an individual’s disability status based on skeletal analysis alone, by assessing multiple lines of evidence (e.g., osteological, mortuary, and clinical), bioarchaeologists can effectively determine an individual’s degree of impairment and make informed inferences about the impact of such an impairment on their social identity. This study also highlights an area of burgeoning research in bioarchaeology: the pathological condition of leg length disparity and its biomechanical implications.

Leg Length Disparity

Leg length disparity (LLD), or anisomelia, describes the condition in which the left and right lower limbs are of unequal length (Shapiro, 2001). According to the clinical literature, LLD is a relatively common condition, affecting as much as 90% of the population (Knutson, 2005). LLD can be categorized as two types: functional and anatomical. Functional LLD refers to a disparity between left and right leg length without osseous involvement, which may be caused by shortened soft tissues, muscular contracture or laxity, or axial misalignments (Brady et al., 2003:222). Anatomical LLD describes a shortening of one side of the lower limbs due to physical length differences between paired long bones (Brady et al., 2003), which can be observed and measured osteologically. The clinical significance of LLD is widely disputed in the literature, and as yet, there is no universal standard for distinguishing between normal and pathological asymmetry. However, a difference of 2 cm (20 mm) or more is generally considered to be worthy of clinical intervention (Thompson, 2014). LLD has been linked to a variety of musculoskeletal conditions, including lower back and hip pain, balance issues, osteoarthritis, stress fractures, and complications with walking and running (Gurney, 2002). Clinical treatment

of LLD generally involves the use of prosthetic lifts worn in the shoe to artificially increase the length of the shorter leg. Studies show that LLD of less than 30 mm can be partially or fully corrected through the use of shoe lifts (Reid and Smith, 1984). More severe cases of LLD may require one of several surgical techniques, including bone lengthening or shortening or epiphysiodesis (surgical closure of the growth plate of the longer leg) (Gurney, 2002:198).

LLD can be caused by a variety of factors, including environmental and genetic factors that influence an individual's ontogenesis (McCaw et al., 1991). A number of diseases, especially paralytic diseases such as tuberculosis, osteomyelitis, and polio, can also cause musculo-skeletal atrophy and result in LLD (Thompson, 2014). Anatomical LLD can also be caused by limb fractures. A fracture occurs when a bone is subjected to biomechanical stresses or forces beyond its elastic capacity, at which point the bone breaks (Wedel and Galloway, 2014). Fractures represent a disruption to the continuity of a bone; incomplete fractures do not extend fully through the diaphysis, while complete fractures break the diaphysis into at least two separate pieces (Lovell, 1997). Depending on the nature of the mechanical stresses leading to the injury, a fracture may take a variety of forms. Breaks resulting from direct trauma include transverse, penetrating, comminuted, and crush fractures, while indirect trauma causes oblique, spiral, greenstick, compression, impaction, and avulsion fractures (Lovell, 1997).

Lower Limb Fractures

Fractures of the lower limbs are common and most often involve the tibia and fibula. Tibia and fibula fractures occur most frequently at the ankle joint; clinically, the distal tibia and fibula are fractured more often than any other bone except the distal radius (Lovell, 1997). Distal tibia/fibula fractures may involve either the medial malleolus or lateral malleolus – rarely both –

and are generally caused by the adduction, abduction, and/or lateral rotation of the ankle joint (Adams and Hamblen, 1999; Lovell, 1997). Diaphyseal fractures of the lower leg usually involve both the tibia and fibula and result from either angular or rotational force on the limb. If the injury results from an angular force, the tibia and fibula will sustain transverse or oblique fractures at roughly the same level; in the event of an injury resulting from rotational force, the two bones will sustain spiral fractures that occur at different levels (Lovell, 1997:163). Because of the bone's close proximity to the skin surface, tibial fractures commonly result in open wounds which may lead to infection (e.g., osteomyelitis, periostitis) and complications to the bone union and healing process. In modern cases, these risks can be mitigated with rapid medical intervention and treatment with antibiotics; however, in pre-antibiotic populations, the risk of infection, malunion, and permanent alteration to the bone's morphology was much higher (Bartle and Keating, 2013; Lovell, 1997).

Femoral fractures are less common than tibia and fibula fractures and are bimodally distributed in frequency between young and elderly adults. The most common femoral fractures occur in elderly adults, especially females, and typically affect the femoral neck and trochanteric region (Lovell, 1997). The majority of fractures to this region are secondary to osteoporosis and result from minor, low-velocity trauma such as a stumble or fall (Lovell, 1997:162). Because of the bone's large size and dense composition, diaphyseal femoral fractures are uncommon and generally result from high-velocity trauma such as falls from significant heights, automobile accidents, and high-impact sports injuries. The fracture type and its location on the femoral diaphysis are dictated by the mechanism of injury; rotational forces generally result in spiral fractures, axial and transverse forces cause oblique fractures, and higher energy impacts result in segmented or comminuted fractures (Bartle and Keating, 2013). Dislocation of the hip is also

common and frequently occurs in conjunction with femoral shaft fractures (Bartle and Keating, 2013; Lovell, 1997).

Fracture Healing

After the initial injury, a fractured bone subsequently goes through four stages of healing: 1) formation of a hematoma through increased blood flow to the area, 2) stimulation of the periosteum prompting the formation of a fibrous soft callus, 3) mineralization of the soft callus forming a primary bony callus, and 4) the conversion of the woven bone to lamellar bone and the reduction and resorption of the bony callus (Lovell, 1997:144-145). If the two ends of a fractured bone are reunited and returned to their natural orientation and the affected limb is largely immobilized during the healing process, the healed bone may bear little or no permanent evidence of the fracture, and the individual may return to normal function (White and Folkens, 2005:48). However, if the two ends of a fractured bone are not properly reunited, the bone may heal with permanently altered morphology. Depending on the type and severity of the fracture, two broken portions of bone may heal together in an overlapped position, resulting in a shortening of the affected bone and limb (Lovell, 1997:147). In the case of leg fractures, this shortening results in a disparity between the lengths of the left and right legs (LLD) which may significantly affect ambulatory function (Hariga et al., 2011; Navascues, 2000).

Biomechanics of Ambulation

Normal Gait

Bipedal locomotion is one of humanity's defining evolutionary adaptations, and human anatomy is structured to accommodate the requirements of an upright gait. Human ambulation is

a delicate balance between maintaining a stable bipedal stance and propelling the body forward, and a normal gait is contingent upon the proper alignment and biomechanical movement of the lower extremities (Perry, 1992). A stride cycle can be subdivided into two categories: stance and swing, which work together to accomplish the tasks of weight acceptance, limb support, and limb advancement (Perry, 1992:9-10). In a non-pathological gait, the bones of the legs align evenly under the trunk of the body, with the pelvic girdle situated at a perpendicular axis to the rest of the body. The biomechanical forces of locomotion traverse normally from the point of impact of the advancing foot, through the knee and hip, and up the spine to the upper body. Weight is distributed evenly through the left and right sides of the body as the lower limbs alternately facilitate forward propulsion, stance stability, shock absorption, and energy conservation (Perry, 1992:22; Winter, 1989).

Pathological Gait and LLD

LLD between the left and right legs represents a significant disruption to locomotive function. When LLD occurs, the structural alignment of the skeleton is altered, resulting in shifts in weight distribution and a disruption of the normal biomechanical forces of human ambulation (Lovell, 1997:147). Depending on the severity of the LLD, a shortening of one side of the lower limbs may result in a concomitant drop in the affected side of the pelvis, resulting in increased tilt and torsion of the pelvic girdle (Kwon et al., 2015; Schuit et al., 1989). Increased pelvic obliquity may also result in lateral flexion of the lumbar vertebrae. To compensate for the lateral bending of the lower spine, the thoracic and cervical vertebrae must then bend in the opposite direction to maintain upright posture (Blunstein and D'Amico, 1985). Under these circumstances, certain areas of the body, such as the knees, hips, spine, shoulders, and neck may

experience abnormal stress, and affected individuals may be at a higher risk of developing pathological conditions in these regions (Figure 1). Depending on the degree of inequality between the left and right legs, individuals with LLD may be at risk of developing stress fractures, lower back pain, and osteoarthritis of affected joint surfaces (McCaw et al., 1991; Murray et al., 2015). Because the brain is always trying to maintain stability and balance, individuals with LLD may deal with the discrepancy between the longer and shorter leg by implementing a variety of compensation strategies such as increased pelvic obliquity, knee flexion of the longer leg, and plantarflexion of the foot (Walsh et al., 2000; Perttunen et al., 2004). However, while these strategies may help to overcome the effects of LLD in mild cases, moderate and severe LLD cases are likely to result in significant pathological conditions despite the adoption of compensation strategies. Furthermore, these strategies may themselves contribute to altering the biomechanical forces of the skeleton, resulting in further complications (Lovell, 1997:147).

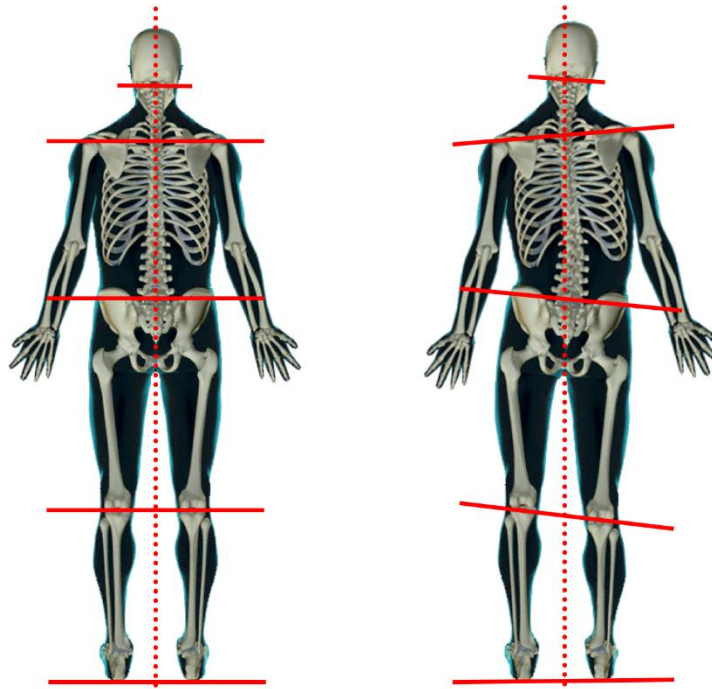


Figure 1: Comparison of anatomical alignment of an individual with equal leg lengths (left) and pathological alignment of an individual with LLD (right). Used with permission of Dr. Lana Williams.

Morphological Integration

Traditionally, gait analysis studies have subdivided the body into two discrete units during ambulation: the active locomotor unit, comprised of the feet, legs, and hips, and the inactive passenger unit, consisting of the trunk, arms, and head. Proponents of this school of thought have argued that during human locomotion, the upper body remains relatively motionless and contributes very little to ambulatory function (Perry, 1992:19-21). However, recent biomechanical studies have demonstrated that the pelvis and spine play a significantly more prominent role in ambulation than was previously realized (Needham et al., 2012). Gracovetsky (1985) highlights humanity's evolutionary history, pointing out that like all land-dwelling mammals, humans evolved from quadrupedal species for whom the spine played a

central role in locomotion. He argues that although humans have since shifted to bipedal locomotion, the spine continues to play an important role in human ambulation and should not be ignored in gait studies (Gracovetsky, 1985).

The increasing recognition of the role of the spine and upper body in human gait is part of a larger movement concerned with understanding how different anatomical structures develop and work together to produce a functional organism. The concept of morphological integration refers to the “tendency for structures to show correlated variation because they develop in response to shared developmental processes or function in concert with other structures” (Hallgrímsson et al., 2002:131). The concept was originally proposed by Olson and Miller (1958), who hypothesized that suites of traits with a developmental or functional relationship work together as complexes and evolve in concert with each other. This notion was largely overlooked until the 1990’s, when biological anthropologists began analyzing covariation patterns in the primate skull and limbs (Cheverud, 1995; Ackermann, 2005; Corner and Richtsmeier, 1991). In recent years, the number of publications concerned with morphological integration of hominin traits has increased; however, these studies have largely focused on covariation between traits of the cranium, mandible, and dentition (Richtsmeier et al., 1992; Bastir, 2008; Boughner and Hallgrímsson, 2008; Trainor and Richtsmeier, 2015), and assessments of integration between disparate regions of the body, such as the lower limbs and the jaw, have not yet been fully explored.

Documenting the cascading effects of traumatic skeletal injury is a chief concern of anatomists, medical practitioners, and physical therapists and has recently been explored in the medical literature. Clinical studies have demonstrated correlations between the occurrence of LLD, gait abnormalities, and corresponding pathological conditions elsewhere in the axial and

appendicular skeleton. For example, Greenwood and colleagues (1997) discovered that individuals who had at some point experienced tibial fractures were more likely to suffer long-term knee and ankle joint pain than non-injured individuals.

An emerging trend in the clinical literature suggests that there is a strong relationship between the stomatognathic system and posture (Blum, 2008). While the lower limbs and jaw may seem like distant and unrelated anatomical structures, recent studies have demonstrated an apparent causal link between orthopedic misalignment of the axial skeleton and temporomandibular disorders (TMD), resulting in jaw pain, difficulty chewing, and dental malocclusion (Blum, 2008). Cuccia (2011) identified a correlation between temporomandibular dysfunction (TMD) and pathological conditions of the plantar arch by analyzing the gait function of subject groups with and without jaw abnormalities. Several studies have induced temporary LLD in otherwise healthy individuals and demonstrated a statistical correlation between LLD, shifts in lateral weight distribution during ambulation, and altered dental occlusion (Maeda et al., 2011; Ohlendorf et al., 2015; Park and Bae, 2014). Interestingly, the correlation between the leg and jaw appears to go both ways. Milani and colleagues (2000) artificially altered dental occlusion in a sample population by having participants wear mandibular orthopedic repositioning devices. The authors demonstrated that members of the test group experienced altered posture, which returned to normal after the removal of the splint (Milani et al., 2000). However, all of these clinical studies analyzed robust samples of living individuals, and it is not clear whether or not this trend is detectable in the absence of soft tissue data.

Not all studies have found a conclusive link between gait abnormalities and jaw dysfunction. Farella and Michelotti et al. (1999, 2005) have argued that the functional relationship between the tissues of the leg and jaw has been overstated and that the scientific

evidence for such a relationship is lacking (Farella et al., 2005). The authors argue that the correlation between leg and jaw dysfunction is spurious and caution medical practitioners to be skeptical of this relationship, especially when it comes to prescribing treatment (Michelotti et al., 1999; Farella et al., 2005). Hanke et al. (2007) are similarly unconvinced of the link between lower limb and dental maladies. After conducting an extensive review of the literature, the authors found that while studies pertaining to a potential association between orthopedic and dental abnormalities have progressively increased since the 1980's, most have utilized poor methodology. As a result, they argue that the "factual base is small" for determining a correlation between leg and jaw disorders (Hanke et al., 2007). Nonetheless, the trend identified in the clinical literature is compelling and suggests that our understanding of the correlation between injury patterns across disparate regions of the body can be improved with additional research.

By assessing the coincidence of TMD and LLD osteologically, the present analysis will provide additional insight into the relationship between the lower limbs and jaw, which has not yet been assessed in the bioarchaeological literature. Furthermore, a more accurate understanding of co-occurrence of lower limb fractures and jaw dysfunction will shed light on the long-term prognosis for individuals with fracture-induced LLD, which may help to improve our understanding of the long-term injury experience and how it relates to social identity.

CHAPTER 3: MATERIALS AND METHODS

Materials

The sample used in this analysis was derived from individuals housed in the Hamann-Todd Osteological Collection (HTOC) at the Cleveland Museum of Natural History in Cleveland, Ohio. The HTOC is one of the largest and most well-documented skeletal collections in the world, consisting of over 3,000 human skeletons amassed from unclaimed cadaver remains between the years 1912 and 1938 (Kern, 2006:10-11). The birth years of the cohort range between 1825 and 1910; thus, the collection is representative of a modern, industrialized urban society (Mensforth and Latimer, 1989). Since this time period predates the advent of many modern medical treatments such as antibiotics, hormonal therapies, and dietary supplements, such treatments would not have been available to mitigate the healing process in fractured individuals. Therefore, this collection represents an ideal sample for studying the biomechanical effects of fractures and LLD on the rest of the body without the confounding factor of modern medical intervention. Because the remains are the result of medical autopsy, the majority of remains were complete and well preserved, allowing for most of the necessary measurements to be taken confidently. While the HTOC houses remains of both adults and juveniles, this analysis focused on adult individuals in order to control for the confounding factors involved in skeletal growth and development. One individual (HTH 0542) was subsequently removed from the analysis, as this individual was a developing adult with unfused epiphyses of all major long bones.

The sample used in this study consisted of two groups: a test group comprised of individuals exhibiting at least one fracture of the lower limbs and a control group of non-

fractured individuals (see Appendix A). Individuals with lower limb fractures were identified and selected for the test group based on previous research that described fracture incidence and patterning in the HTOC (McNulty, 2009). Samples were randomly selected using a randomization function in Excel (=RANDBETWEEN) that generated random numbers between 1 and 10,000 associated with each individual in the entire sample. Columns of identifiers were sorted using randomly generated numbers, and the top 28 individuals were selected for measurement from both the fractured sample and the control group. One individual from the control group was found to have lower limb fractures and was moved to the test group. In total, the sample consisted of 56 adult individuals, 27 in the control group (18 males, 9 females) and 29 in the test group (19 males, 10 females). The number of males and females in each of the two groups was kept as consistent as possible to allow for robust statistical analysis of sex differences (Figure 2).

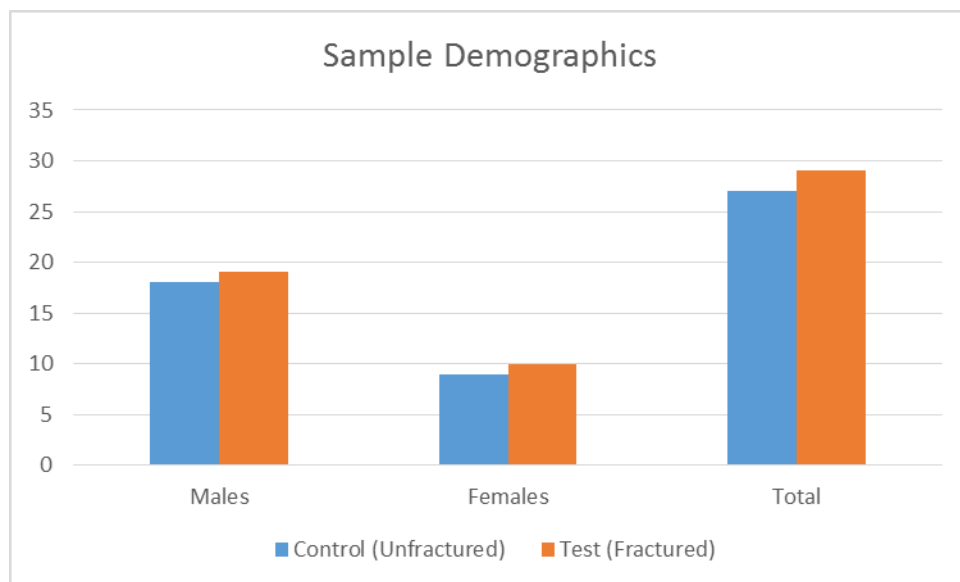


Figure 2: Histogram demonstrating the total sample size and the number of males and females in each sample group.

Methods

Individuals were inspected visually for the presence, location, and severity of lower limb fractures without the aid of radiographs. Observed fractures were scored as “unhealed” (0), “healing” (1), or “well healed” (2) according to published healing stages (Lovell, 1997). The maximum lengths of each individual’s femora, tibiae, and fibulae were measured using an osteometric board according to discipline conventions (Buikstra and Ubelaker, 1994) (Table 1). Substantial differences between left and right long bone pairs were noted as possible instances of LLD.

Table 1: Description and explanation of measurements used in the analysis.

Measurement Abbreviation	Explanation of Measurement	Citation/Method
Lfemur	Maximum length of left femur	Buikstra and Ubelaker, 1994
Rfemur	Maximum length of right femur	Buiktra and Ubelaker, 1994
LRFemurDiff	Difference between maximum length of left and right femur	Calculation in Microsoft Excel (Lfemur – Rfemur = LRFemurDiff)
Ltibia	Maximum length of left tibia	Buikstra and Ubelaker, 1994
Rtibia	Maximum length of right tibia	Buikstra and Ubelaker, 1994
LRTibiaDiff	Difference between maximum length of left and right tibia	Calculation in Microsoft Excel (Ltibia - Rtibia = LRTibiaDiff)
Lfibula	Maximum length of left fibula	Buikstra and Ubelaker, 1994
Rfibula	Maximum length of right fibula	Buikstra and Ubelaker, 1994
LRFibDiff	Difference between maximum length of left and right fibula	Calculation in Microsoft Excel (Lfibula – Rfibula = LRFibDiff)
LMandFossa	Osteoarthritis score on left mandibular fossa of the cranium (0-4 scale)	Rando and Waldron, 2012
RMandFossa	Osteoarthritis score on right mandibular fossa of the cranium (0-4 scale)	Rando and Waldron, 2012
LMandCondyle	Osteoarthritis score on left condyle of the mandible (0-4 scale)	Rando and Waldron, 2012

Measurement Abbreviation	Explanation of Measurement	Citation/Method
RMandCondyle	Osteoarthritis score on right condyle of the mandible (0-4 scale)	Rando and Waldron, 2012
RToothWearAvg	Average of tooth wear scores of all right teeth (#1-8, 25-32)	Turner et al., 1991
LToothWearAvg	Average of tooth wear scores of all left teeth (#9-24)	Turner et al., 1991
Right AMTL #	Total number of antemortem tooth loss of right teeth (#1-8, 25-32)	
Left AMTL #	Total number of antemortem tooth loss of left teeth (#9-24)	
Left/Right LLD	Total length difference of all left and right leg bones	Calculation in Microsoft Excel (LRFemurDiff + LRTibiaDiff + LRFibDiff = Left/Right LLD)
LRToothWearAvgDiff	Difference in average tooth wear scores for the left and right sides of the mouth	Calculation in Microsoft Excel (RToothWearAvg + LToothWearAvg = LRToothWearAvgDiff)
LRAMTLDiff	Difference in total number of teeth lost antemortem for the right and left sides of the mouth	Calculation in Microsoft Excel (Left AMTL # - Right AMTL # = LRAMTLDiff)

Jaw dysfunction was assessed by scoring the incidence of osteoarthritis of the temporomandibular joint (TMJ OA), patterns of mandibular and maxillary tooth wear, and antemortem tooth loss. TMJ OA is a pathological condition of the jaw characterized by pain and difficulty chewing, which can be observed skeletally in degenerative changes to the articular surfaces of the temporomandibular joint. The cranial mandibular fossae and mandibular condyles were scored as “absent” (0), “slight” (1), “moderate” (2), and “severe” (3) based on the degree of surface pitting, erosion, and eburnation (see Figure 3). The degree of wear of all 32 teeth was scored on a scale of 0 (no wear) to 4 (severe wear) based on the extent of dentin and pulp exposure (Turner et al., 1991). Antemortem tooth loss was also noted and included in the data, as it may reflect advanced tooth wear or jaw malocclusion. All measurements were recorded on

paper data collection sheets, entered into Microsoft Excel data spreadsheets, and cross-checked for accuracy.

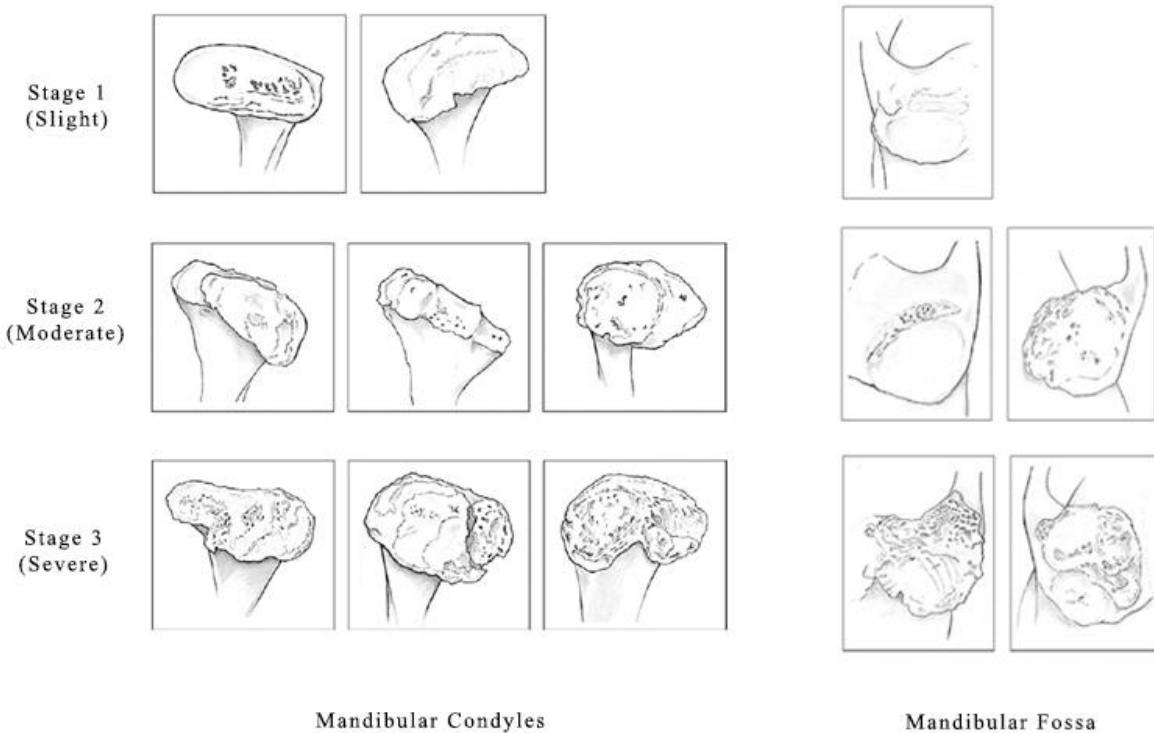


Figure 3: Stages of expression of TMJ OA on the mandibular condyles and articular eminence of the mandibular fossa used to score TMJ OA in the sample (adapted from Rando and Waldron, 2012).

Photographs were taken of all fractures, all instances and stages of TMJ OA, and discernable patterns of asymmetrical tooth wear. Remains were photographed with a Nikon D3000 DSLR camera against a black velvet background on a laboratory camera stand. A ring light was used to highlight important skeletal features otherwise not illuminated by the ambient lighting in the laboratory.

Analysis

LLD was assessed by calculating the total length differences between the left and right femora, tibiae, and fibulae of each individual. This was accomplished by subtracting the length of each right bone from the left, taking the absolute value of the result, and summing the values of all three limb bone differences. Total length differences of 20 mm or more were considered a clinically significant length disparity (Thompson, 2014).

Tooth wear scores for the left and right sides of the mouth were averaged by adding up all of the wear scores for each side and dividing by 16 (half the total number of teeth). Average score differences of 1.0 or more between the left and right side were considered possible cases of asymmetrical tooth wear. The total number of teeth lost antemortem was added for the left (#9-24) and right (#1-8, 25-32) sides. Substantial differences between left and right antemortem tooth loss counts were noted as possible cases of asymmetrical tooth loss.

Statistical analyses of the data were performed using SPSS v.23. In order to assess intraobserver error, approximately 18% of the sample (10 individuals) was measured and scored twice with more than 24 hours in between measurement sessions to avoid memory bias. Intraobserver error was then calculated using a Pearson's correlation test in SPSS v.23. Technical error of measurement (TEM) was calculated in Excel using the standard formula (Lewis, 1999). After initial data collection was complete, a post-hoc power analysis was conducted using G*Power v.3.1 to determine the achieved power of the study. To assess the influence of potentially confounding factors, such as age, sex, and ancestry on the measurements taken in this analysis, a MANOVA test with post-hoc ANOVAs was conducted.

Patterns of morphological integration between skeletal components of interest were assessed by calculating Kendall's Tau correlation values among pairs of traits measured from the

leg bones and mandible. Although Pearson's or Spearman Rho correlations are often used to assess continuous or categorical data, respectively, Kendall's Tau correlations are appropriate here because ties in ranks of categorical variables being compared were possible (Lomax and Hahs-Vaughn, 2012). However, Pearson's correlation tests are still appropriate for assessing intraobserver error, which is described in chapter 4 (Lomax and Hahs-Vaughn, 2012). The resultant values were compared to determine if correlation sign and strength were significantly different between all fractured and unfractured individuals and subsamples consisting of only males or only females. The interpretation of absolute correlation strength values varies between analyses; therefore, the thresholds for correlation strength were defined for this analysis as follows: 0.00-0.09 = No correlation (i.e., so weak as to be irrelevant), 0.10-0.29 = Weak correlation, 0.30-0.49 = Moderate correlation, 0.50-1.00 = Strong correlation. Because correlation values are not normally distributed, correlation values were converted to z-scores using a Fisher's Z transformation, and p-values were determined to assess the statistical significance of correlation differences among and between samples.

CHAPTER 4: RESULTS

Intraobserver reliability testing yielded a Pearson's correlation value of 1.0, indicating that data collection was consistent between measurement sessions and is therefore reliable. TEM calculations yielded a result of 0.23, which was less than 5% of the mean of all values measured in this study (5.66) and therefore is considered acceptably low. A post-hoc power analysis using G*Power software indicated that with the total sample size of 56 individuals, an effect size of 0.32, and a one-tailed test, achieved power (1- β error probability) was 0.81. Thus, this analysis has the ability to detect moderate to large significant effects in the samples compared.

Lower Limb Fractures

Leg fractures were observed in 29 of the 56 analyzed individuals (Table 2). The most commonly observed fractures were of the tibia and fibula, which frequently occurred simultaneously (Figure 4). As expected, femoral fractures were less frequent, although six were observed in the sample. The majority of fractures were well healed; however, four individuals exhibited fractures that were in the process of healing, and six individuals had unhealed perimortem fractures that were sustained at or near the time of death. Since most of the fractures observed in the sample were well healed, it is likely that any lasting secondary biomechanical consequences would have had sufficient time to manifest in the skeleton.

Table 2: Fracture incidence in entire sample by bone, location, side, and state of healing.

Individual Number	Involved Bone(s)	Location	Side	State of Healing
HTH 0204	Fibula	Distal	Right	Well-healed
HTH 0339	Tibia/Fibula	Medial	Right	Well-healed
HTH 0444	Femur	Proximal	Left	Healing
HTH 0459	Femur/Fibula	Medial/Distal	Left/Right	Well-healed
HTH 0479	Femur/Tibia/Fibula	Proximal	Right	Well-healed
HTH 0500	Tibia/Fibula	Distal/Proximal	Right	Well-healed
HTH 0534	Tibia/Fibula	Distal	Right	Well-healed
HTH 0354	Tibia	Medial	Right	Well-healed
HTH 0631	Fibula	Proximal	Right	Well-healed
HTH 0718	Tibia	Medial	Right	Well-healed
HTH 1552	Femur	Medial	Right	Well-healed
HTH 1592	Tibia/Fibula	Distal	Right	Healing
HTH 1647	Tibia/Fibula	Distal/Proximal	Right	Well-Healed
HTH 0543	Femur	Proximal	Left	Unhealed
HTH 0587	Tibia/Fibula	Medial	Right	Unhealed
HTH 0602	Tibia	Proximal/Distal	Right	Well-healed
HTH 0666	Fibula	Distal	Left	Well-healed
HTH 0742	Tibia	Medial	Right	Unhealed
HTH 0751	Fibula	Distal	Left	Well-healed
HTH 0868	Fibula	Proximal	Left	Well-healed
HTH 0974	Fibula	Distal	Right	Well-healed
HTH 1124	Tibia/Fibula	Distal	Left	Well-healed
HTH 1387	Fibula	Proximal	Left	Healing
HTH 1470	Tibia/Fibula	Medial/Proximal	Right	Unhealed
HTH 3091	Tibia	Distal	Left	Healing
HTH 3045	Fibula	Proximal	Right	Well-healed
HTH 1534	Tibia/Fibula	Proximal/Distal	Left	Unhealed
HTH 1903	Femur	Distal	Left	Unhealed
HTH 2761	Tibia/Fibula	Distal	Right	Well-healed



Figure 4: Simultaneous fractures of the left distal tibia and fibula of Individual HTH 1124 (anterior view).

Leg Length Disparity (LLD)

As expected, the overall prevalence of LLD was more common in the fractured group than in the unfractured group (Table 3). Five of the 29 individuals in the fractured group (17.24%) had an LLD of 20 mm or more, while LLD was present in only one individual in the unfractured group (3.7%).

Table 3: Overall LLD occurrence in the sample. LLD refers to the sum total of length discrepancy between the left and right femur, tibia, and fibula of each individual.

Individual Number	Sample Group	LLD (mm)
HTH 0500	Fractured	27.89
HTH 0602	Fractured	34.46
HTH 1552	Fractured	19.44*
HTH 1647	Fractured	28.78
HTH 2828	Unfractured	23.99
HTH 3045	Fractured	23.04

*Individual HTH 1552 falls just under, but approaches, the LLD threshold of 20 mm

The majority of fractured individuals with LLD of 20 mm or more exhibited complete, displaced fractures, which directly contributed to LLD by physically shortening the maximum length of the affected long bones (see example in Figure 5). Since the individual in the control group with LLD did not have any fractures, the disparity between the left and right legs must have been caused by other factors.



Figure 5: Simultaneous lower limb fracture and LLD occurrence in individual HTH 1552. (A) Healed complete, displaced fracture of right medial femur (lateral view; arrow indicates fracture location). (B) Comparison of maximum lengths of unfractured left (top) and fractured right (bottom) femur of individual HTH 1552 (anterior view). Note the considerable shortening of the fractured right femur compared to the unfractured left femur.

Temporomandibular Dysfunction

Instances of TMJ OA were roughly equivalent between the unfractured and fractured groups. In the fractured group, some degree of TMJ OA occurred in 57% (17/30) of the sample and was absent in the remaining 43% (13/30). The majority of cases of TMJ OA were slight and only involved one or two of the mandibular condyles or fossae. Two individuals exhibited moderate TMJ OA; there were no severe cases. Among the fractured individuals with LLD of 20 mm or more, three had slight TMJ OA, while two had none. In the unfractured group, 61% of the sample (17/28) exhibited some degree of TMJ OA, while 39% (11/28) had none. Most cases

were slight, although one individual had moderate TMJ OA; there were no severe cases. The one individual with LLD over 20 mm displayed slight TMJ OA with mandibular fossa involvement.

Dental attrition was approximately equivalent between the unfractured and fractured groups. In the fractured group, the minimum average wear score was 0 (no wear), and the maximum was 3.40 (severe wear). The average of all right tooth wear average scores was 1.01, and the left was 1.10. The average difference between left and right wear scores was 0.31. Among the individuals with LLD of 20 mm or more, the average of all left tooth wear score was 1.55, while the average of all right tooth wear scores was 1.82. The average difference between left and right tooth wear scores was 0.43. In the unfractured group, the minimum wear score was 0 (no wear), and the maximum was 2.50 (moderate wear). The average of all right tooth wear average scores was 1.05, and the average of all left tooth wear average scores was 0.87. The average difference between left and right wear scores was 0.45. The one unfractured individual with LLD was edentulous; thus, tooth wear scores could not be assessed.

Antemortem tooth loss counts were similar between the unfractured and fractured groups. In the fractured group, the minimum loss count was 0 (no antemortem tooth loss), and the maximum number was 32 (edentulous). The average of all right antemortem tooth loss counts was 6.28, and the average of all left antemortem tooth loss counts was 6.00. The average difference between left and right tooth loss counts was 1.66. Among the individuals with LLD of 20 mm or more, the average antemortem tooth loss count was 10.8 for the right side and 9 for the left, with an average left and right difference of 3. Individual #7 (HTH 0500) exhibited extremely asymmetrical antemortem tooth loss, with 12 teeth lost antemortem on the right and 1 tooth lost on the left. In the unfractured group, the minimum antemortem tooth loss count was 0

(no tooth loss) and the maximum antemortem tooth loss count was 32 (edentulous). The average tooth loss count was 7.56 for the right side and 7.97 for the left, with an average difference of 1.07. The one individual with LLD was edentulous; thus, antemortem tooth loss asymmetry could not be assessed as all teeth were lost antemortem.

Patterns of Morphological Integration

Comparisons of all three samples (All Unfractured vs. Fractured, Male Unfractured vs. Fractured, and Female Unfractured vs. Fractured) demonstrated patterns of differences in morphological integration that were unique to each two-sample comparison. Comparisons of correlation directionality among all three sample groups demonstrated an overall trend of positive correlation values in unfractured groups and negative values in the fractured groups (Figure 6). The second most common occurrence was for values to be consistently positive between unfractured and fractured groups, followed by negative correlation values in the unfractured group and positive values in the fractured group. The least common pattern was for correlation values to be consistently negative between the unfractured and fractured groups of all analyzed samples.

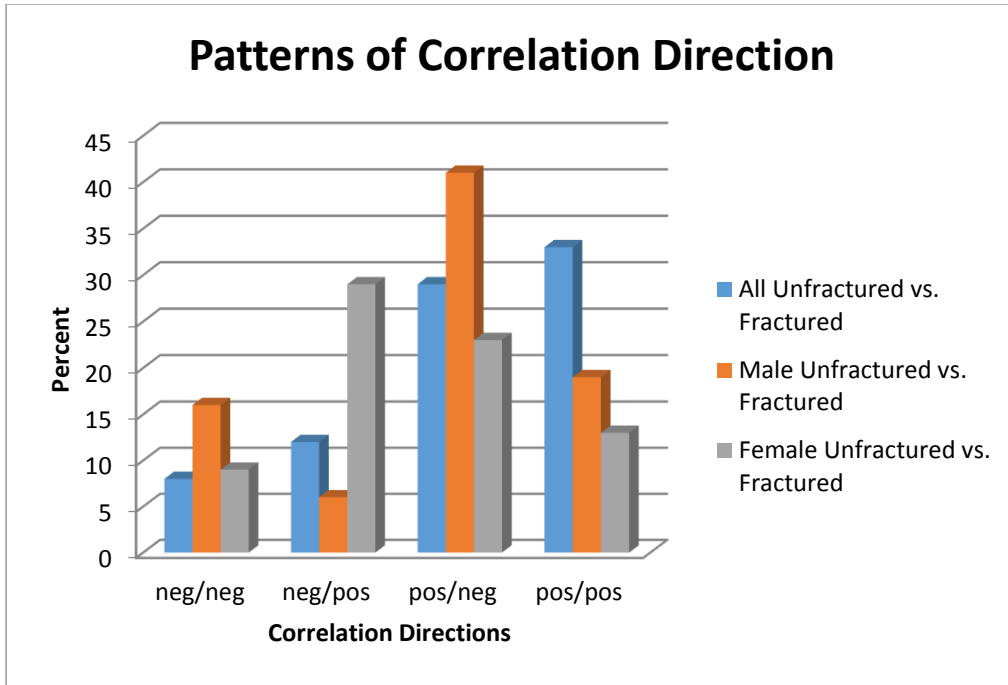


Figure 6: Overall correlation directionality between the three sample groups demonstrating patterns of morphological integration.

Differences in correlation strength between the unfractured and fractured samples varied considerably across the analyzed groups but demonstrated interesting and unique trends for each two-sample comparison. The most common occurrence was for correlation strength to be consistently weak between unfractured and fractured groups (Figure 7). The second most common occurrence was no correlation strength in the unfractured group and a weak correlation in the fractured group.

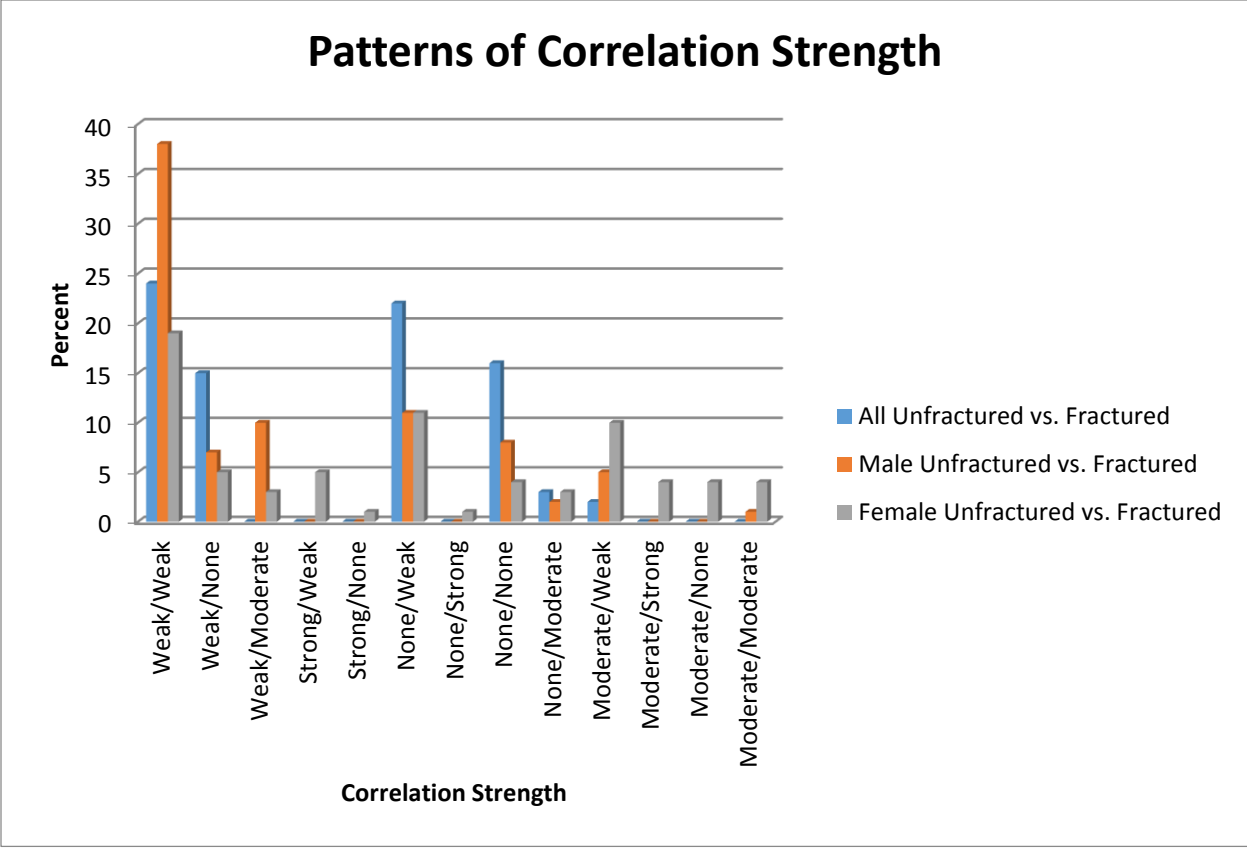


Figure 7: Overall correlation strength across all three sample groups demonstrating patterns of morphological integration.

All Unfractured vs. Fractured

Correlation direction was consistent (i.e., negative/negative, positive/positive) between unfractured and fractured groups in 50% of cases and was different (i.e., positive/negative, negative/positive) in 50% of cases (Figure 8). Approximately 40% of correlation values were positive for both unfractured and fractured individuals, while in 10% of cases, correlation values were negative for both unfractured and fractured individuals. In 35% of cases, correlation values were positive for unfractured individuals but negative for fractured individuals. In 15% of cases, correlation values were negative for unfractured individuals and positive for fractured individuals.

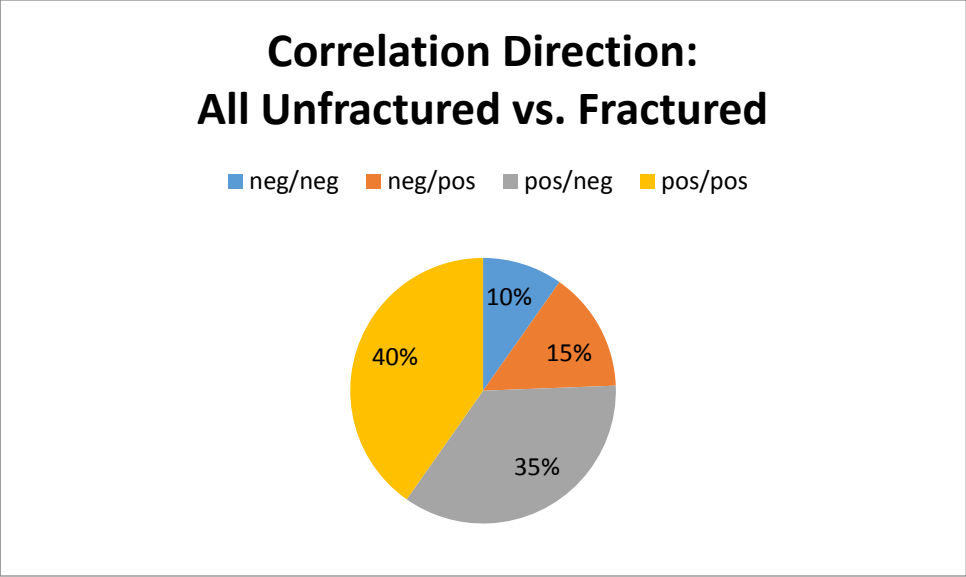


Figure 8: Correlation direction patterns between all unfractured and all fractured individuals.

Correlation strength was different (i.e., weak/none, none/moderate, moderate/weak) between unfractured and fractured groups in 51% of cases and was consistent (i.e. weak/weak, none/none, moderate/moderate) in 49% of cases (Figure 9). Correlation strength was lower for fractured individuals in 20% of cases and higher in 31% of cases. Outside of the general trends observed for all of the analyzed groups, clear differences can be seen between males and females. The female group demonstrated the greatest variety in correlation strength values as well as the strongest correlation values (moderate, strong) observed in the sample. Because of this, the sample was sub-divided into male and female groups and the differences between them analyzed in greater detail and assessed for statistical significance.

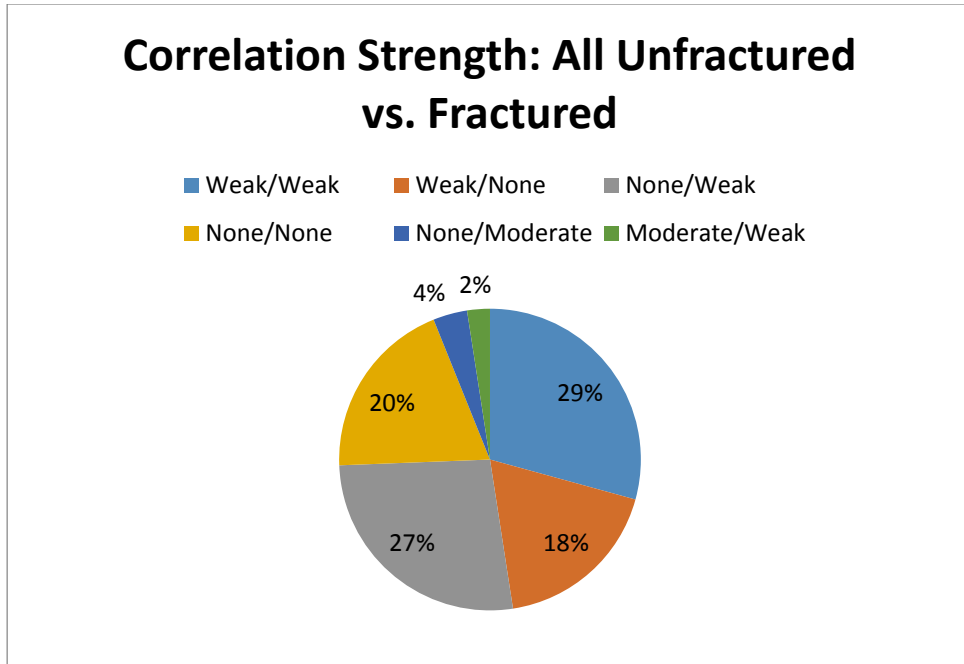


Figure 9: Correlation strength patterns between all unfractured and fractured individuals.

Male Unfractured vs. Fractured

Analysis of differences in correlation direction between the unfractured and fractured male sample yielded a similarly interesting pattern. In the majority of cases (50%), unfractured males exhibited a positive correlation between relevant leg and jaw measurements, whereas the homologous pair of measurements had a negative correlation in the fractured group (Figure 10). Approximately 43% of correlation directions were the same (i.e., negative/negative; positive/positive), while 7% of cases saw negative leg-jaw correlations in the unfractured group and positive correlations in the fractured group, thereby exhibiting differences in correlation directionality.

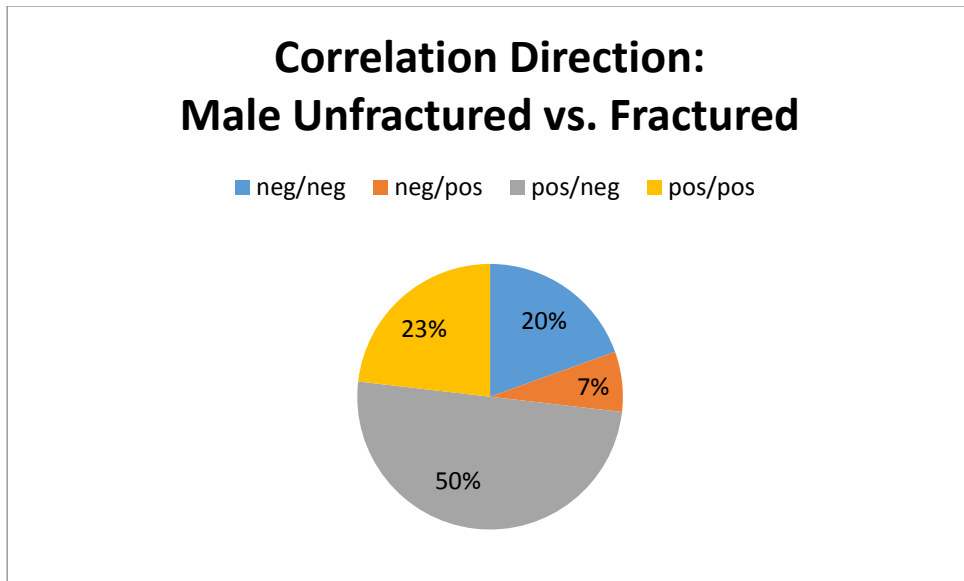


Figure 10: Correlation direction patterns between all male unfractured and fractured individuals.

Correlation strength was consistent (i.e., weak/weak, none/none, moderate/moderate) between unfractured and fractured males in the majority (57%) of cases (Figure 11). Correlation strength was lower in the fractured group in 15% of cases (i.e., moderate/weak, weak/none) and higher in the fractured group in the remaining 28% (i.e., none/weak, weak/moderate, none/moderate).

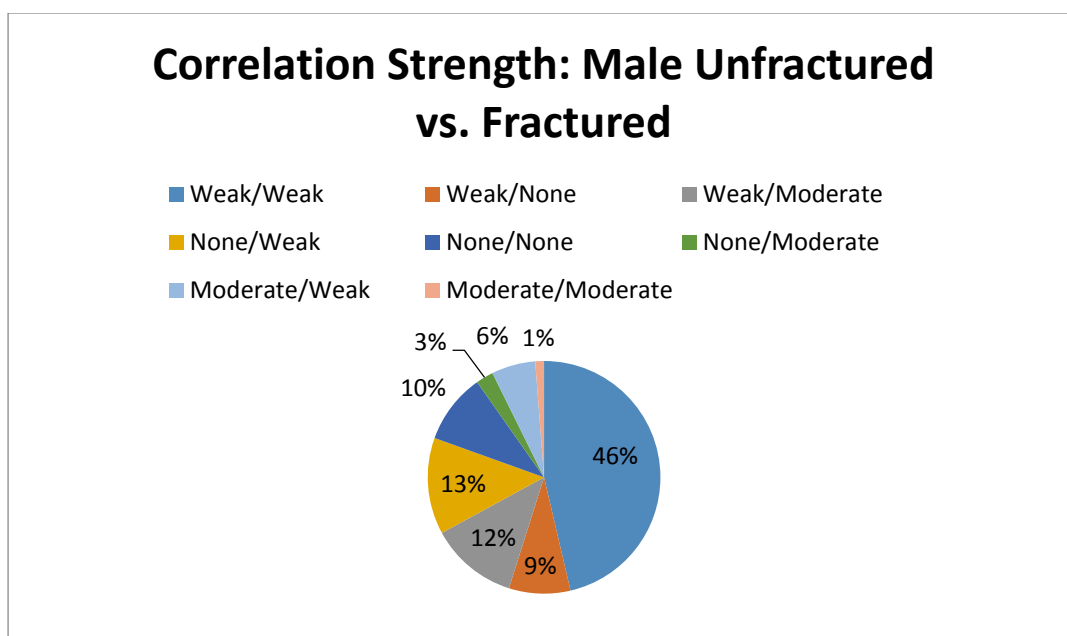


Figure 11: Correlation strength patterns between all male unfractured and fractured individuals.

Female Unfractured vs. Fractured

Correlation direction was different between the female unfractured and fractured groups in 70% of cases (i.e., negative/positive, positive/negative) and was consistent in 30% (i.e., negative/negative, positive/positive; Figure 12). For 39% of homologous measurement comparisons in the female sample, the correlation was negative in the unfractured group and positive in the fractured group. However, for 31% of comparisons the opposite trend occurred, with the correlation being positive in the unfractured group and negative in the fractured group.

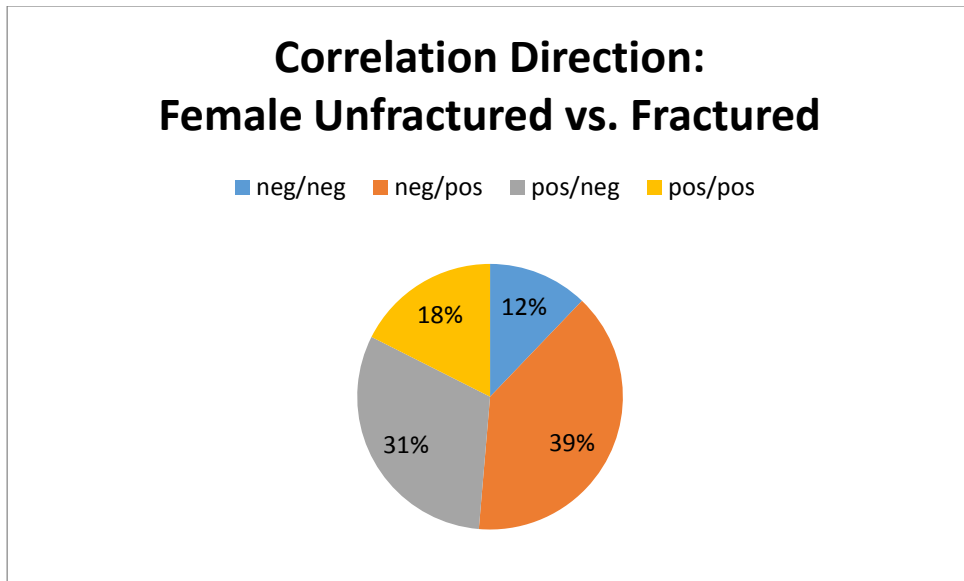


Figure 12: Correlation direction patterns between all female unfractured and fractured individuals.

Differences in correlation strength were highly variable in the female sample, with no clear pattern identified between the unfractured and fractured groups. It is worth emphasizing that the different combinations of correlation strength when comparing homologous measurements in the female samples spans 13 different types of combinations (see Figure 13), whereas only 8 and 6 combinations were present in the male and entire (all) sample comparisons, respectively. In 37% of female cases, correlation strength was consistent between the unfractured and fractured samples (i.e., weak/weak, moderate/moderate) while in the remaining 64% correlation strength was different (Figure 13). Within the altered correlations, 38% of cases saw higher correlation strength in the unfractured group, while in 26% of cases, correlation strength was higher in the fractured group. The female sample group also exhibited a greater frequency of moderate correlations and the only strong correlation values observed in the analysis.

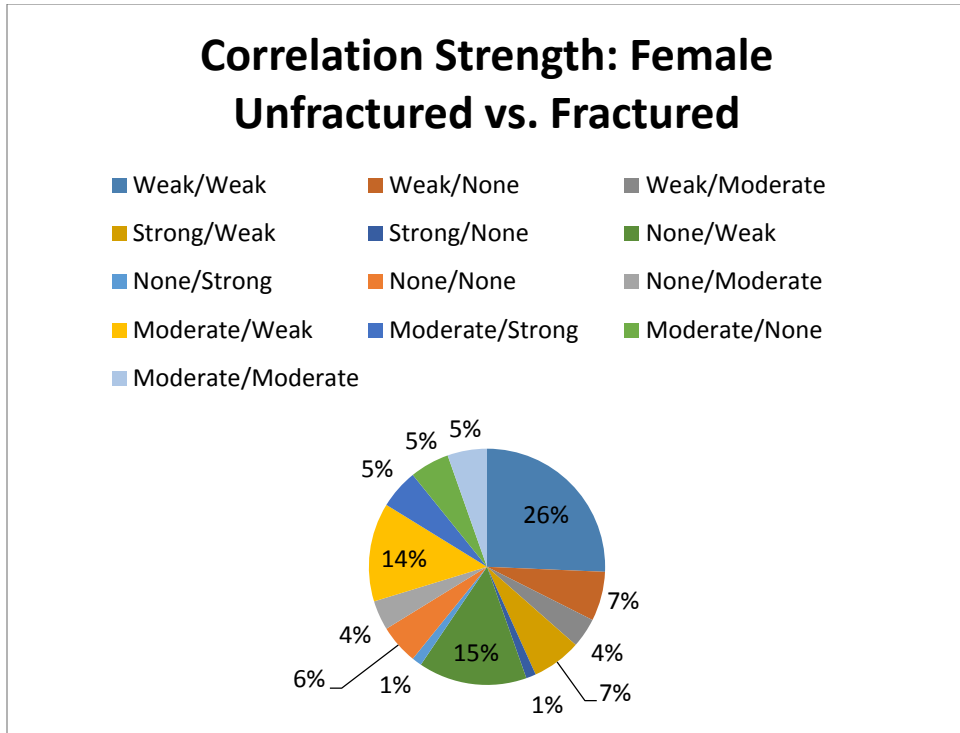


Figure 13: Correlation strength patterns between all female unfractured and fractured individuals.

Long Bone Comparisons

Comparisons of correlations between long bone lengths and all jaw measurements highlighted differences between the combined and sub-divided sample groups. In all cases, patterns of correlation between the All Unfractured vs. Fractured group and the Male Unfractured vs. Fractured group were largely consistent. The Female Fractured vs. Unfractured group, on the other hand, deviated considerably from the overall sample and the male sub-sample (Figures 14-23). Due to the small size of the female sub-sample and several cases of unobservable measurements, several correlations were undefined; these missing correlation values are represented by gaps in the line graphs generated for the female sample group.

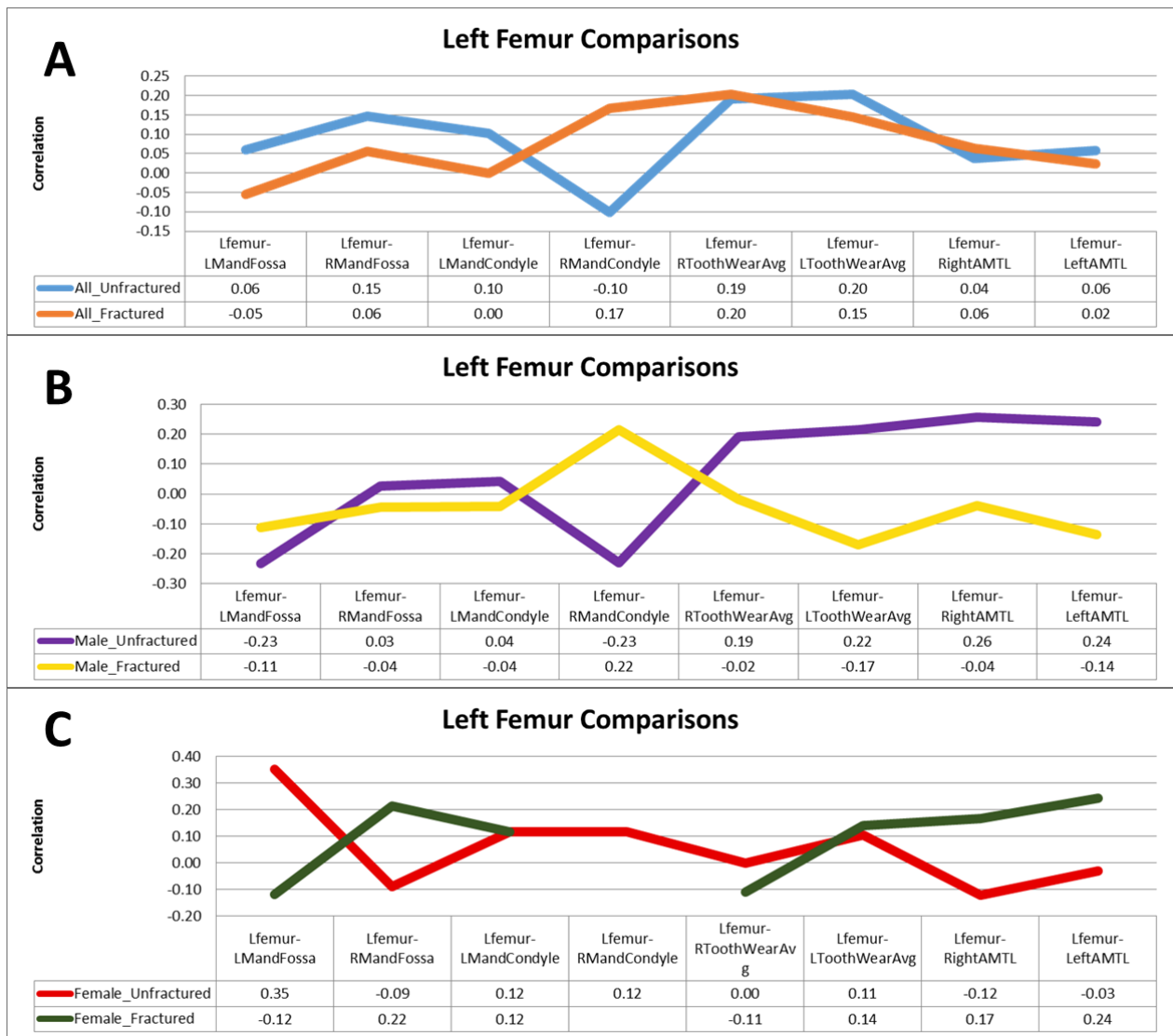


Figure 14: Comparison of all left femur correlation values in the All Unfractured vs. All Fractured (A), Male Unfractured vs. Fractured (B), and Female Unfractured vs. Fractured (C) sample groups.

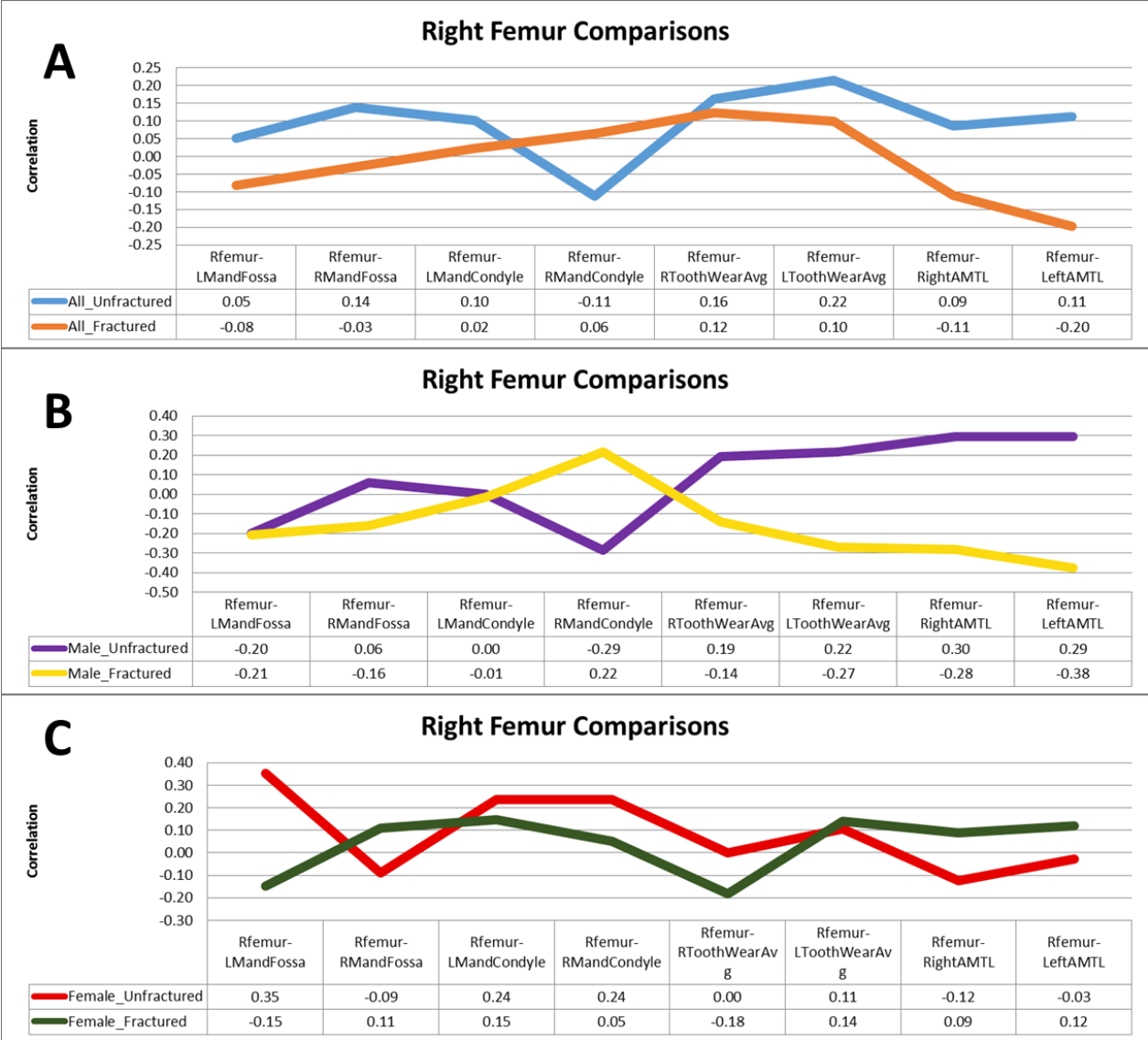


Figure 15: Comparison of all right femur correlation values in the All Unfractured vs. All Fractured (A), Male Unfractured vs. Fractured (B), and Female Unfractured vs. Fractured (C) sample groups.

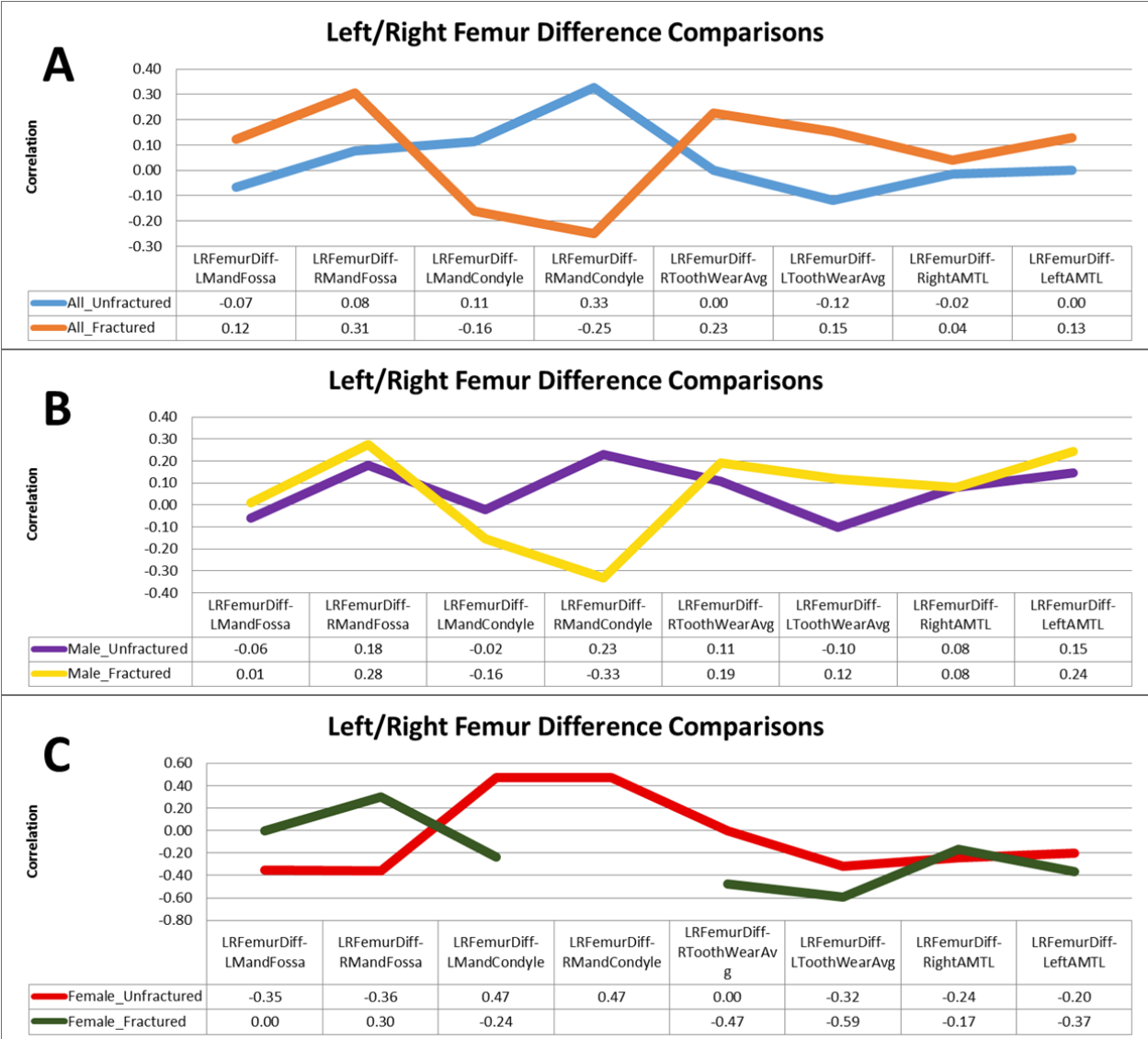


Figure 16: Comparison of all left/right femur difference correlation values in the All Unfractured vs. All Fractured (A), Male Unfractured vs. Fractured (B), and Female Unfractured vs. Fractured (C) sample groups.

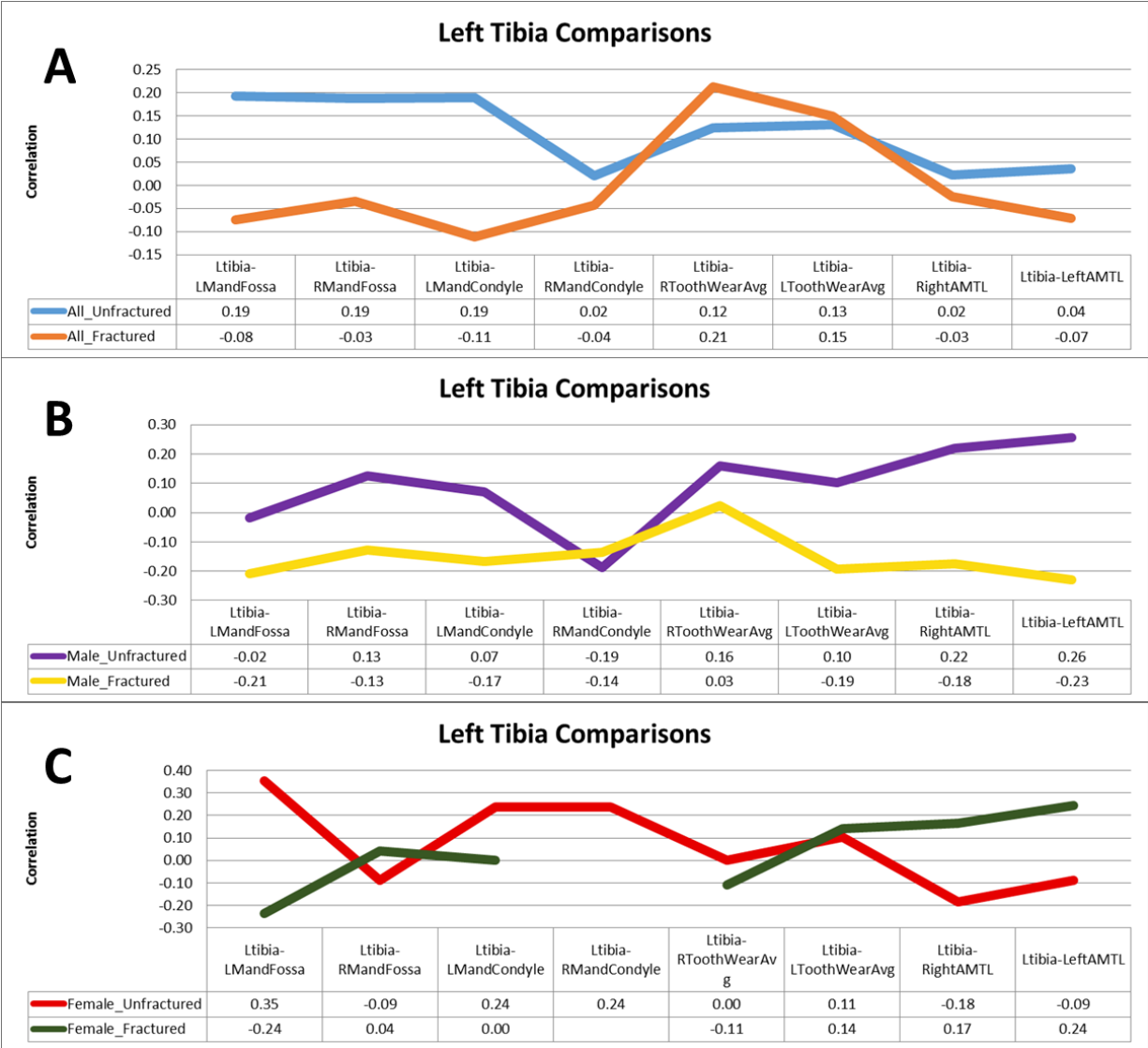


Figure 17: Comparison of all left tibia correlation values in the All Unfractured vs. All Fractured (A), Male Unfractured vs. Fractured (B), and Female Unfractured vs. Fractured (C) sample groups.

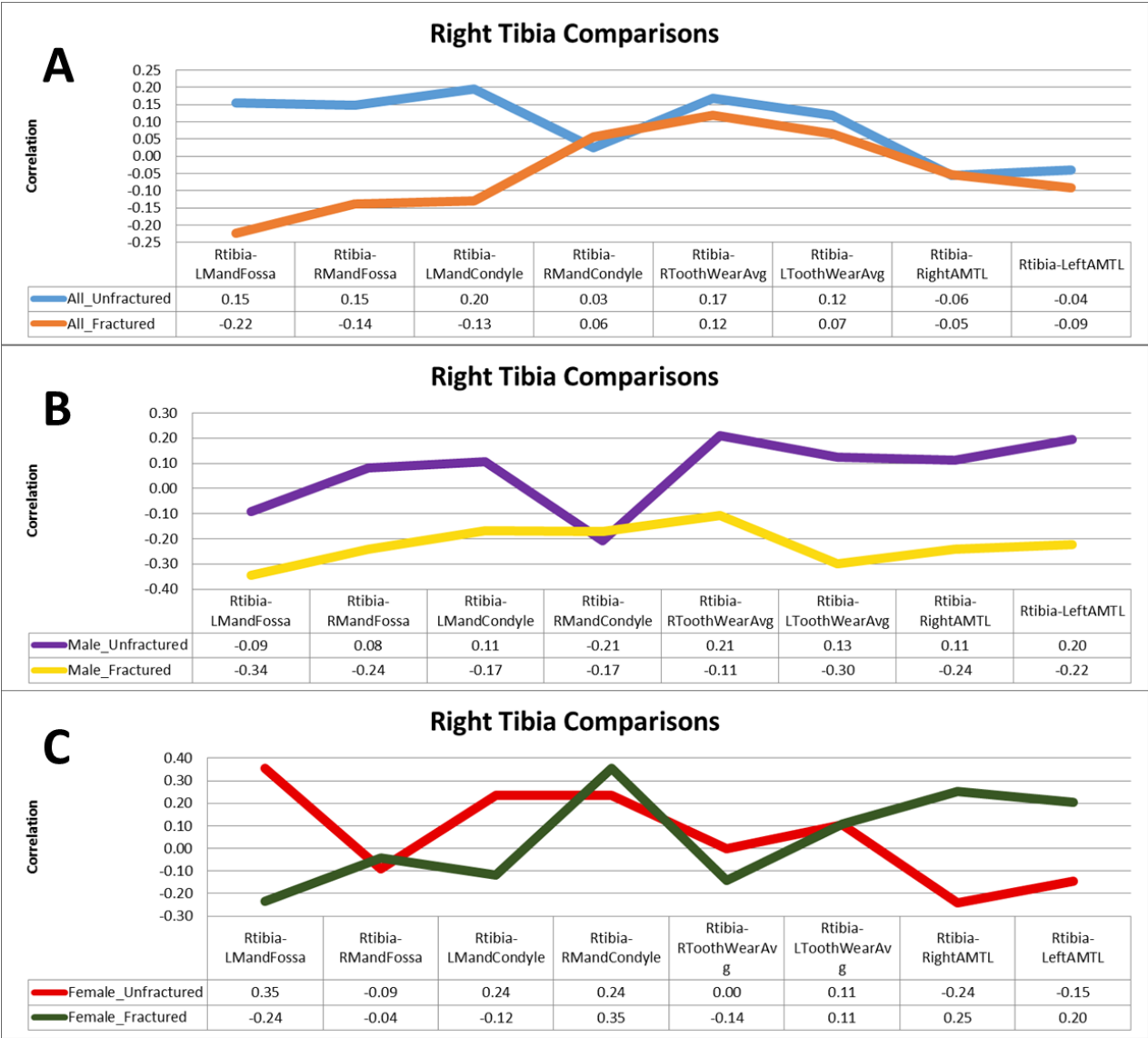


Figure 18: Comparison of all right tibia correlation values in the All Unfractured vs. All Fractured (A), Male Unfractured vs. Fractured (B), and Female Unfractured vs. Fractured (C) sample groups.

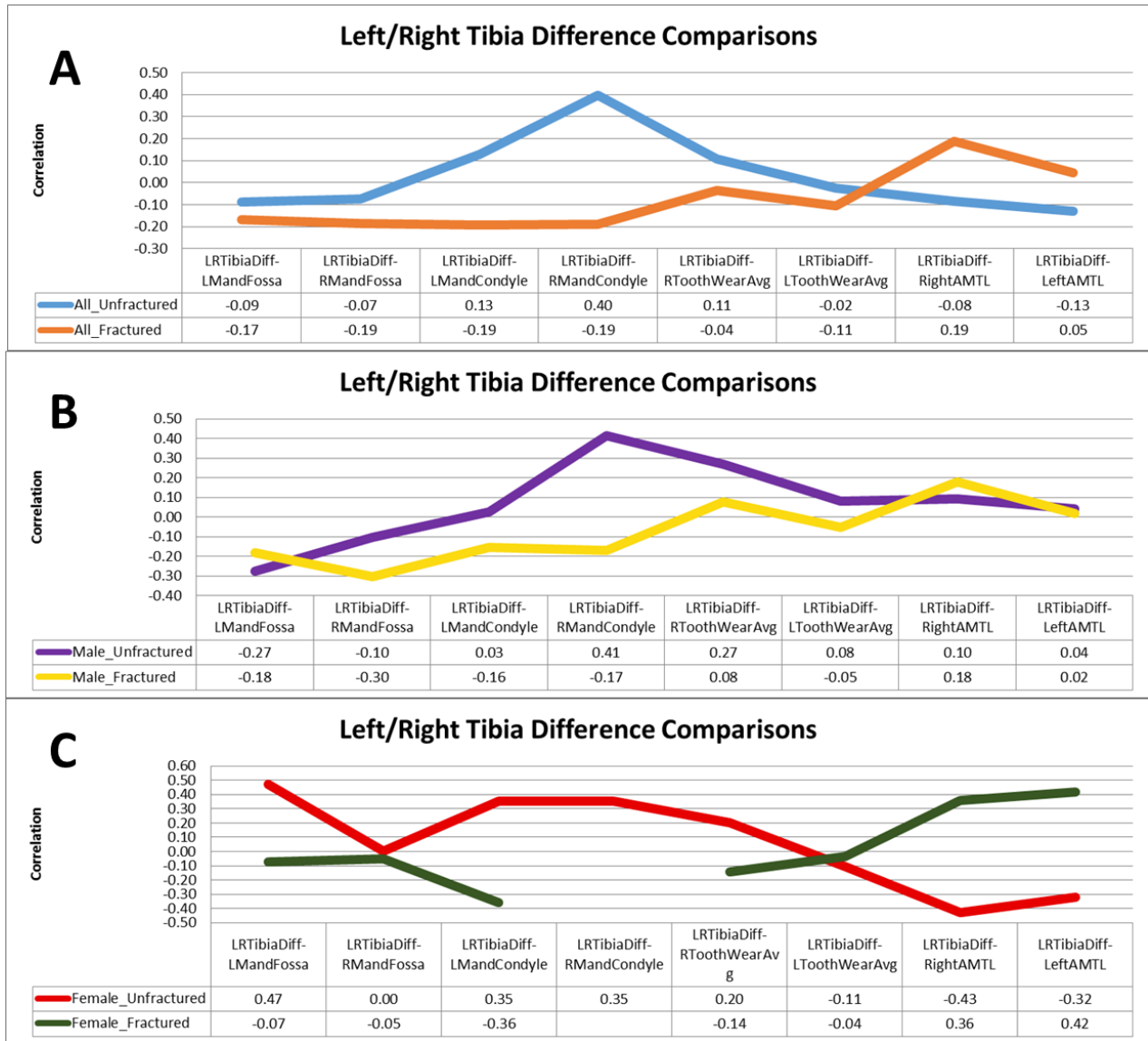


Figure 19: Comparison of all left/right tibia difference correlation values in the All Unfractured vs. All Fractured (A), Male Unfractured vs. Fractured (B), and Female Unfractured vs. Fractured (C) sample groups.

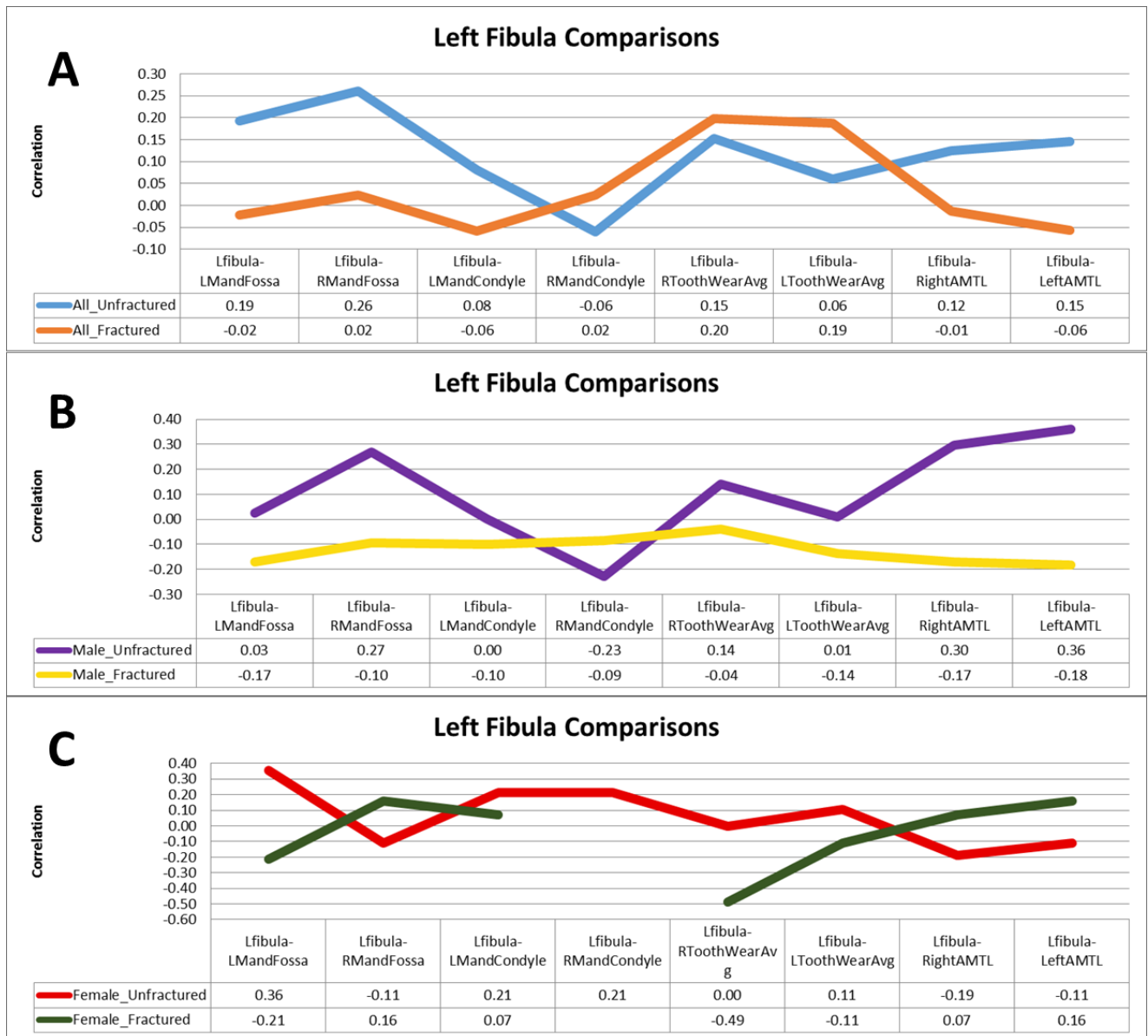


Figure 20: Comparison of all left fibula correlation values in the All Unfractured vs. All Fractured (A), Male Unfractured vs. Fractured (B), and Female Unfractured vs. Fractured (C) sample groups.

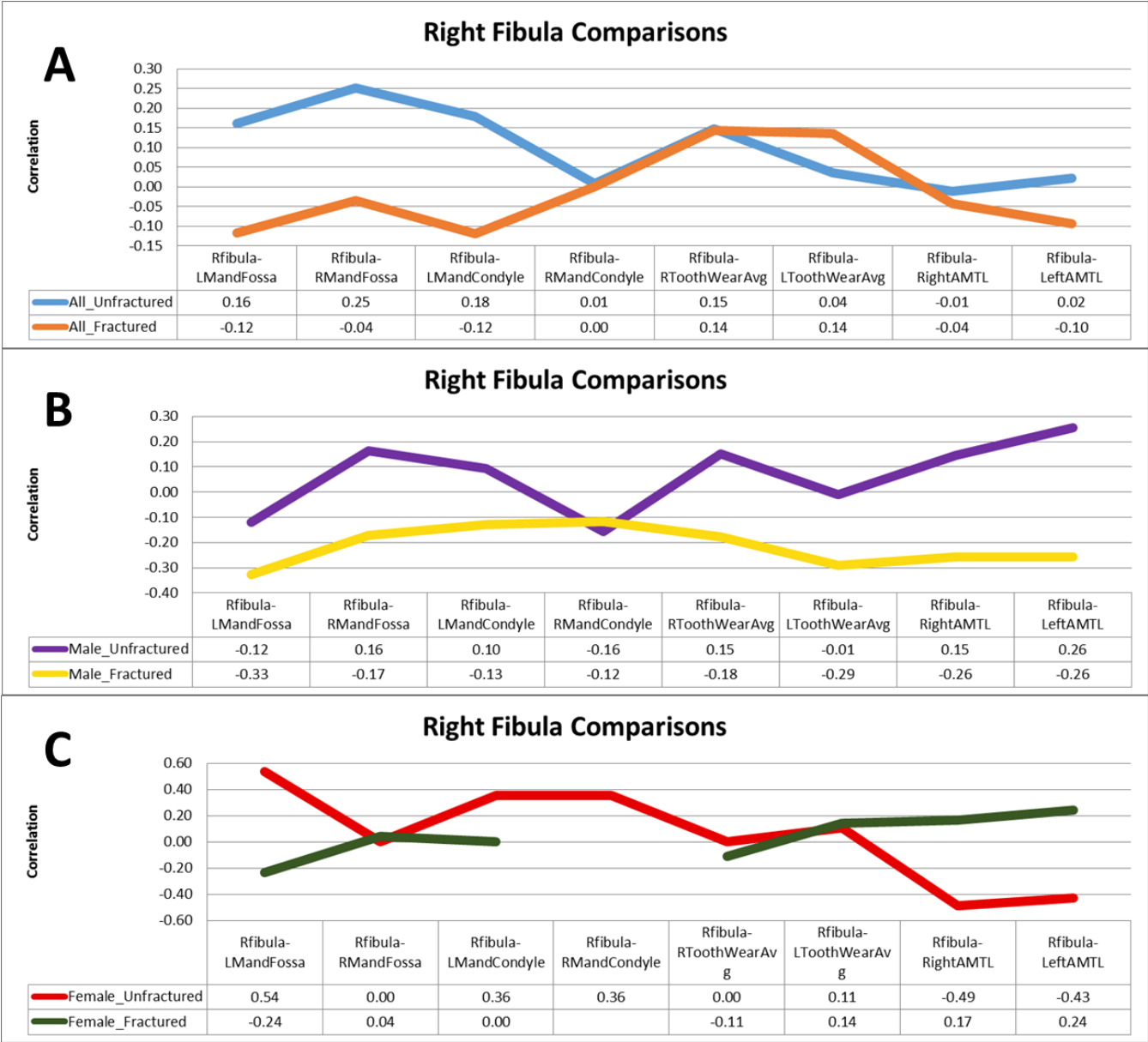


Figure 21: Comparison of all right fibula correlation values in the All Unfractured vs. All Fractured (A), Male Unfractured vs. Fractured (B), and Female Unfractured vs. Fractured (C) sample groups.

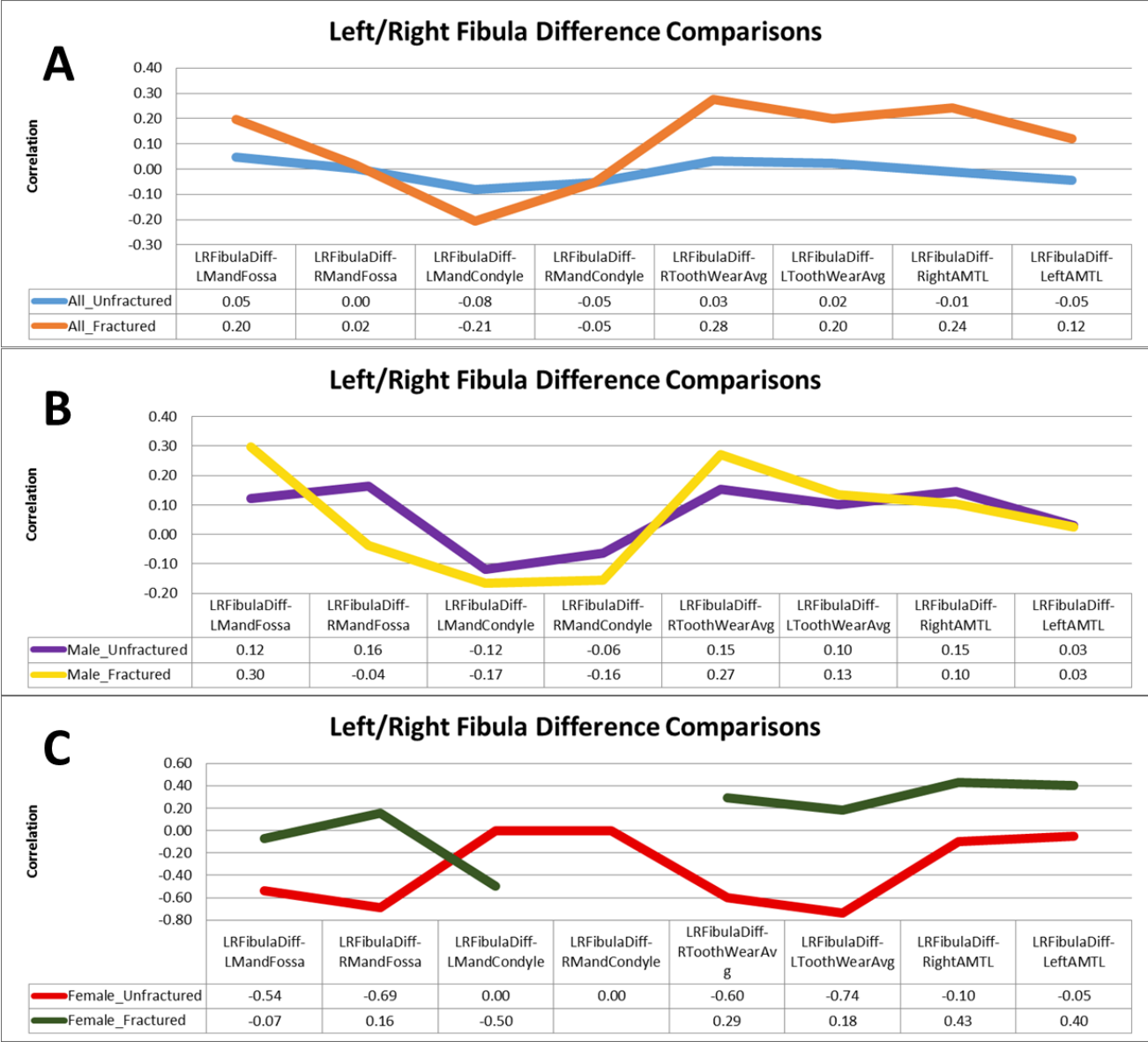


Figure 22: Comparison of all left/right fibula difference correlation values in the All Unfractured vs. All Fractured (A), Male Unfractured vs. Fractured (B), and Female Unfractured vs. Fractured (C) sample groups.

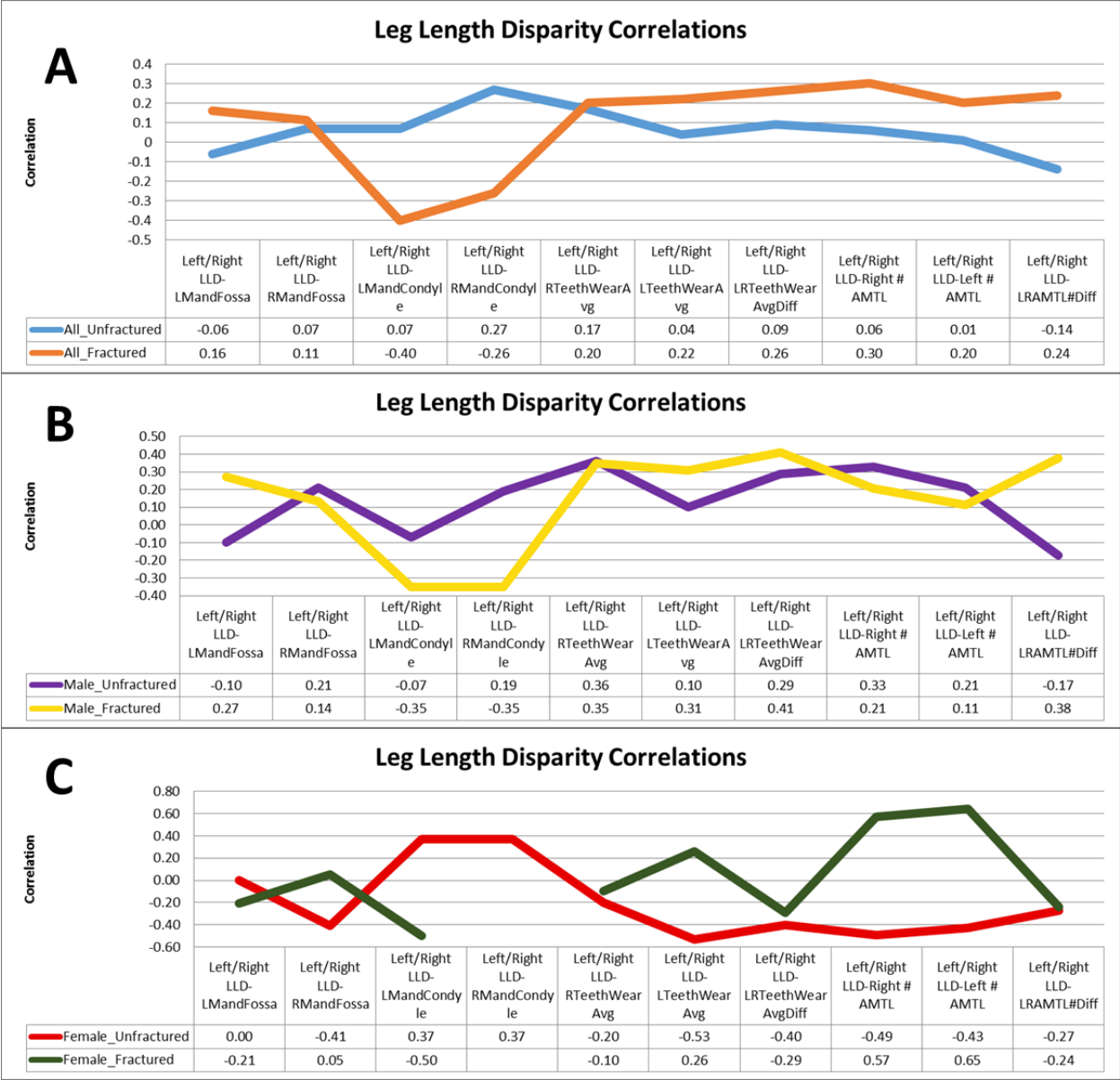


Figure 23: Comparison of all leg length disparity correlation values in the All Unfractured vs. All Fractured (A), Male Unfractured vs. Fractured (B), and Female Unfractured vs. Fractured (C) sample groups.

Statistically Significant Correlation Patterns

All Fractured vs. Fractured

Comparisons of correlation patterns between unfractured and fractured individuals in the entire sample demonstrated slight differences between the two groups. In 59% of cases, correlation values between lower limb and jaw measurements were higher for unfractured individuals, whereas in 41% of cases, correlation values between homologous variables were higher for fractured individuals. Due to the small sample size (which was then further subdivided for sex-based analyses), an alpha level of 0.10 was selected for statistical analyses and assessment of correlation patterns. Significant correlation differences were observed for measurements between the LRFemurDiff-RMandCondyle ($p=0.02$), Rtibia-LMandFossa ($p=0.09$), LRTibiaDiff-RMandCondyle ($p=0.02$), Left/Right LLD-LMandCondyle ($p=0.04$), Left/Right LLD-RMandCondyle ($p=0.03$), and Left/Right LLD-LRAMTL#Diff ($p=0.09$) (Figure 24). However, since all individuals were statistically compared two times, a Bonferonni adjustment for multiple testing was carried out, thereby reducing the initial alpha value from 0.10 to 0.05 for the results reported here and for correlation results reported for males and females below. As a result, several of the reported statistical differences can no longer be considered significant.

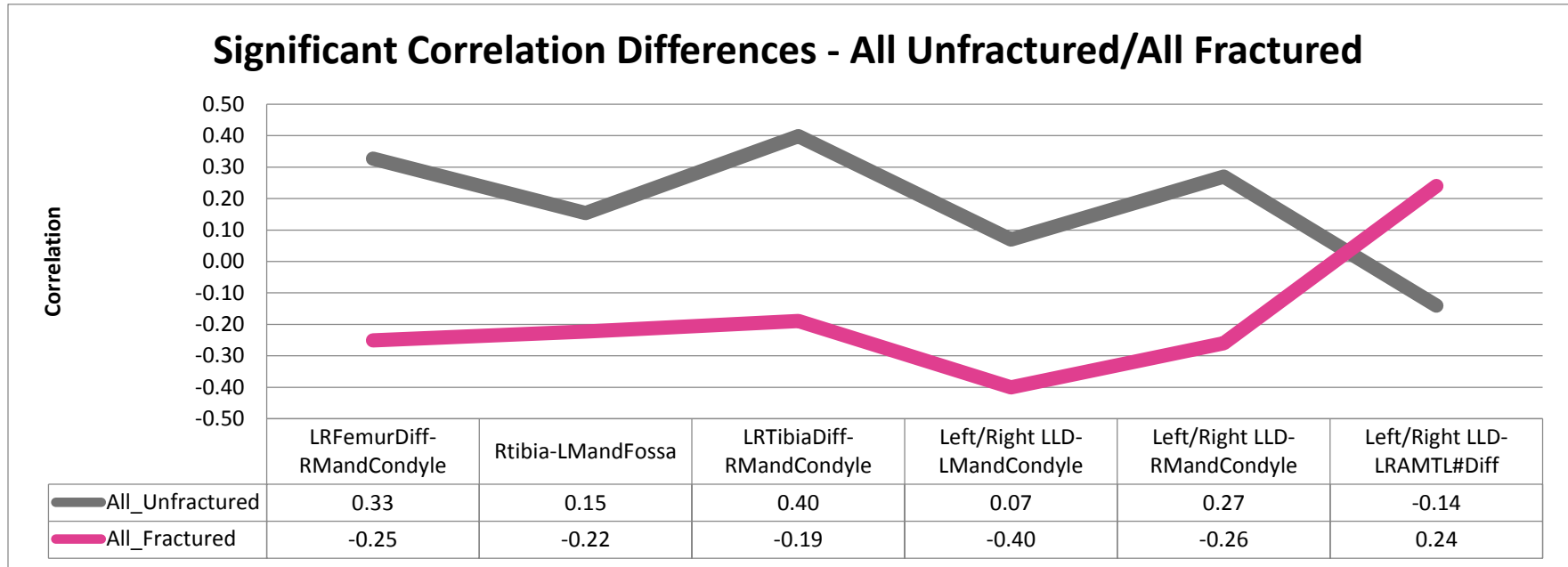


Figure 24: Correlation values that significantly differ between the All Fractured and All Unfractured samples.

Male Unfractured vs. Fractured

When the unfractured and fractured sample groups were separated by sex, analysis of the male sample yielded a more striking pattern than was observed in the combined sample. In 74.4% of cases in the male sample, correlation values between lower limb and jaw measurements were higher for unfractured individuals than for fractured individuals (25.6%). Significant correlation differences were observed for measurements between the Rfemur-RMandCondyle ($p=0.08$), Rfemur-LToothWearAvg ($p=0.08$), Rfemur-RightAMTL ($p=0.05$), Rfemur-LeftAMTL ($p=0.03$), LRFemurDiff-RMandCondyle ($p=0.05$), Ltibia-LeftAMTL ($p=0.04$), LRTibiaDiff-RMandCondyle ($p=0.09$), Lfibula-RightAMTL ($p=0.06$), Lfibula-LeftAMTL ($p=0.06$), Rfibula-LeftAMTL ($p=0.07$), Left/Right LLD-RMandCondyle ($p=0.06$), and Left/Right LLD-LRAMTL#Diff ($p=0.06$) (Figure 25).

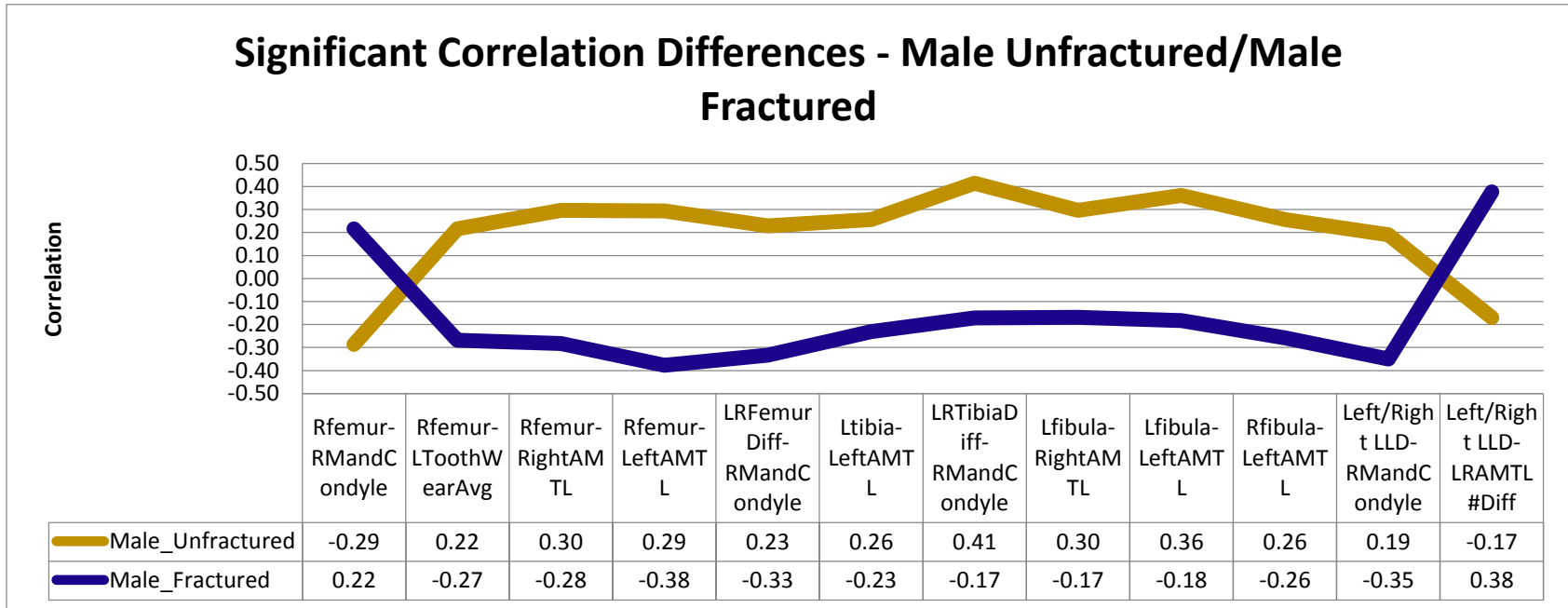


Figure 25: Correlation values that significantly differ between male unfractured and fractured samples.

Female Unfractured vs. Fractured

Analysis of correlation differences between unfractured and fractured females demonstrated a distinct pattern from the male group. For the female group, Kendall's Tau correlation values were only higher in the unfractured group 45% of the time, while in 55% of cases correlation values were higher for fractured individuals. Significant correlation differences were observed for measurements between the LRFemurDiff-LMandCondyle ($p=0.09$), RTibiaDiff-LMandCondyle ($p=0.09$), LRTibiaDiff-RightAMTL ($p=0.07$), LRTibiaDiff-LeftAMTL ($p=0.08$), Rfibula-LMandFossa ($p=0.07$), LRFibulaDiff-RMandFossa ($p=0.04$), LRFibulaDiff-RToothWearAvg ($p=0.04$), LRFibulaDiff-LToothWearAvg ($p=0.02$), Left/Right LLD-LMandCondyle ($p=0.05$), Left/Right LLD-LToothWearAvg ($p=0.06$), Left/Right LLD-RightAMTL# ($p=0.02$), and Left/Right LLD-LeftAMTL# ($p=0.01$) (Figure 26).

Significant Correlation Differences - Female Unfractured/Female Fractured

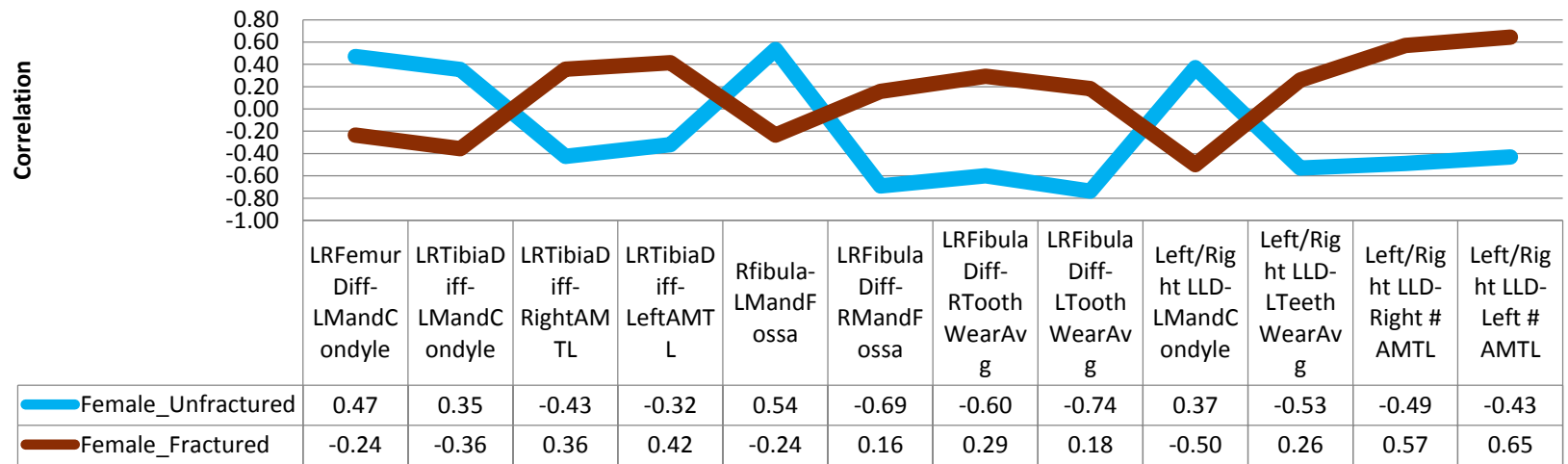


Figure 26: Correlation values that significantly differ between the female unfractured and fractured samples.

A MANOVA analysis ($\alpha = 0.10$) determined that all four analyzed factors (Unfractured vs. Fractured, Sex, Age, and Ancestry) were significant (Table 4). However, post-hoc ANOVA analyses of the four factors demonstrated that for the Unfractured vs. Fractured and Age factors, individual dependent variables were not significant (Tables 5 and 6). Post-hoc ANOVA results for Sex, on the other hand, showed significant values for the Lfemur and Rfemur measurements ($p=0.01$), LToothWearAvg ($p=0.02$), and Ltibia and Rtibia ($p=0.09$) (Table 7). Results for the Ancestry factor also yielded significant values for the Ltibia ($p=0.05$) and Rtibia ($p=0.07$). After a Bonferonni adjustment for multiple testing (one MANOVA and four post-hoc ANOVAs) the alpha value was reduced to 0.02, and several of these values can no longer be considered significant.

Table 4: MANOVA results for the Wilks' Lambda test. Statistically significant values are starred and highlighted in bold.

Factor	Partial Eta ²	F	Observed Power	P-value
Unfractured vs. Fractured	1.00	498.10	0.85	0.04*
Sex	1.00	30376.74	1.00	<0.01*
Age	0.93	3.08	1.00	<0.01*
Ancestry	1.00	23978.17	1.00	0.01*

* Significant at $\alpha = 0.05$

** Significant at $\alpha = 0.10$

Table 5: Post-hoc ANOVA results for Unfractured vs. Fractured. Statistically significant values are starred and highlighted in bold.

Dependent Variable	Partial Eta²	F	Observed Power	P-value
Lfemur	0.00	0.00	0.05	0.97
Rfemur	0.00	0.01	0.05	0.91
LRFemurDiff	0.01	0.09	0.06	0.77
Ltibia	0.00	0.01	0.05	0.93
Rtibia	0.01	0.11	0.06	0.75
LRTibiaDiff	0.17	2.45	0.30	0.14
Lfibula	0.00	0.04	0.05	0.85
Rfibula	0.00	0.02	0.05	0.89
LRFibDiff	0.14	1.90	0.25	0.19
Left/Right LLD	0.13	1.83	0.24	0.20
LMandFossa	0.04	0.54	0.10	0.48
RMandFossa	0.02	0.21	0.07	0.65
LMandCondyle	0.02	0.28	0.08	0.61
RMandCondyle	0.15	2.06	0.26	0.18
RToothWearAvg	0.00	0.02	0.05	0.89
LToothWearAvg	0.16	2.32	0.29	0.15
LRToothWearAvgDiff	0.08	0.97	0.15	0.34
Right AMTL #	0.01	0.15	0.07	0.71
Left AMTL #	0.02	0.23	0.07	0.64

Dependent Variable	Partial Eta²	F	Observed Power	P-value
LRAMTL#Diff	0.6	0.70	0.12	0.42

* Significant at $\alpha = 0.05$

** Significant at $\alpha = 0.10$

Table 6: Post-hoc ANOVA results for Age. Statistically significant values are starred and highlighted in bold.

Dependent Variable	Partial Eta²	F	Observed Power	P-value
Lfemur	0.61	0.85	0.29	0.65
Rfemur	0.64	0.96	0.33	0.55
LRFemurDiff	0.75	1.62	0.56	0.19
Ltibia	0.48	0.51	0.18	0.92
Rtibia	0.49	0.51	0.18	0.92
LRTibiaDiff	0.75	1.66	0.58	0.18
Lfibula	0.36	0.31	0.12	0.99
Rfibula	0.37	0.32	0.12	0.99
LRFibDiff	0.57	0.72	0.25	0.76
Left/Right LLD	0.64	0.99	0.34	0.53
LMandFossa	0.68	1.13	0.39	0.43
RmandFossa	0.72	1.42	0.50	0.27
LmandCondyle	0.40	0.36	0.13	0.98
RmandCondyle	0.29	0.22	0.10	1.00

Dependent Variable	Partial Eta ²	F	Observed Power	P-value
RtoothWearAvg	0.76	1.69	0.59	0.17
LtoothWearAvg	0.77	1.82	0.62	0.14
LRToothWearAvgDiff	0.66	1.06	0.37	0.48
Right AMTL #	0.52	0.59	0.20	0.86
Left AMTL #	0.65	0.99	0.34	0.53
LRAMTL#Diff	0.29	0.22	0.10	1.00

* Significant at $\alpha = 0.05$

** Significant at $\alpha = 0.10$

Table 7: Post-hoc ANOVA results for Sex. Statistically significant values are starred and highlighted in bold.

Dependent Variable	Partial Eta ²	F	Observed Power	P-value
Lfemur	0.44	9.24	0.80	0.01*
Rfemur	0.41	8.42	0.76	0.01*
LRFemurDiff	0.01	0.06	0.06	0.82
Ltibia	0.22	3.47	0.40	0.09**
Rtibia	0.23	3.51	0.41	0.09**
LRTibiaDiff	0.05	0.64	0.11	0.44
Lfibula	0.19	2.74	0.33	0.12
Rfibula	0.18	2.65	0.32	0.13

Dependent Variable	Partial Eta ²	F	Observed Power	P-value
LRFibDiff	0.00	0.02	0.05	0.88
Left/Right LLD	0.00	0.05	0.05	0.84
LMandFossa	0.16	2.35	0.29	0.15
RMandFossa	0.02	0.26	0.08	0.62
LMandCondyle	0.00	0.03	0.05	0.86
RMandCondyle	0.00	0.04	0.05	0.84
RToothWearAvg	0.14	1.95	0.25	0.19
LToothWearAvg	0.37	6.96	0.68	0.02*
LRToothWearAvgDiff	0.14	2.03	0.26	0.18
Right AMTL #	0.03	0.33	0.08	0.58
Left AMTL #	0.08	1.03	0.16	0.33
LRAMTL#Diff	0.03	0.42	0.09	0.53

* Significant at $\alpha = 0.05$

** Significant at $\alpha = 0.10$

Table 8: Post-hoc ANOVA results for Ancestry. Statistically significant values are starred and highlighted in bold.

Dependent Variable	Partial Eta ²	F	Observed Power	P-value
Lfemur	0.08	1.08	0.16	0.32
Rfemur	0.08	1.03	0.16	0.33
LRFemurDiff	0.02	0.28	0.08	0.61

Dependent Variable	Partial Eta²	F	Observed Power	P-value
Ltibia	0.28	4.70	0.51	0.05**
Rtibia	0.25	3.99	0.45	0.07**
LRTibiaDiff	0.03	0.31	0.08	0.59
Lfibula	0.18	2.64	0.32	0.13
Rfibula	0.21	3.23	0.38	0.10
LRFibDiff	0.04	0.56	0.11	0.47
Left/Right LLD	0.00	0.00	0.05	0.97
LMandFossa	0.01	0.08	0.06	0.78
RMandFossa	0.00	0.01	0.05	0.93
LMandCondyle	0.01	0.15	0.07	0.71
RMandCondyle	0.05	0.57	0.11	0.47
RToothWearAvg	0.09	1.18	0.17	0.30
LToothWearAvg	0.11	1.47	0.20	0.25
LRToothWearAvgDiff	0.01	0.15	0.07	0.71
Right AMTL #	0.00	0.03	0.05	0.86
Left AMTL #	0.01	0.09	0.06	0.78
LRAMTL#Diff	0.01	0.07	0.06	0.80

* Significant at $\alpha = 0.05$

** Significant at $\alpha = 0.10$

CHAPTER 5: DISCUSSION

Descriptive statistical analysis between the combined unfractured and fractured samples yielded several trends. On average, LLD was more common in the fractured group, demonstrating that the healing process of lower limb fractures can cause a discrepancy in length between the left and right legs. In terms of co-occurrence of LLD and TMD, there were no apparent significant differences between the unfractured and fractured groups; however, this may be due to a number of sampling issues, such as small sample size, male bias, and unmatched age categories.

Significant Correlations

Significant Kendall's Tau correlation values in all of the analyzed groups demonstrated interesting patterns. When comparing the entire sample of unfractured and fractured individuals, significant correlation values relating to LLD were identified for the 1) difference between the left and right femur and the right mandibular condyle (LRFemurDiff-RMandCondyle), 2) difference between the left and right tibia and the right mandibular condyle (LRTibiaDiff-RMandCondyle), 3) leg length disparity and the left mandibular condyle (Left/Right LLD-LMandCondyle), 4) leg length disparity and the right mandibular condyle (Left/Right LLD-RMandCondyle), and 5) leg length disparity and the difference between left and right antemortum tooth loss (Left/Right LLD-LRAMTL#Diff). These correlation values indicate that the co-occurrence of LLD with TMJ OA and LLD with asymmetrical antemortem tooth loss were significantly different between the unfractured and fractured groups. Four out of five significant differences in correlation values demonstrated a difference in directionality that was positive in the unfractured group and negative in the fractured group. This suggests that

morphological integration of the lower limbs and jaw was more frequently disrupted in the fractured group than in the unfractured group. As a result, fractured individuals in the entire sample exhibited LLD associated with TMJ OA and asymmetrical antemortem tooth loss more often than members of the unfractured group.

In the Male Unfractured vs. Fractured group, significant correlation values for LLD were observed for the 1) difference between the left and right femur and the right mandibular condyle (LRFemurDiff-RMandCondyle), 2) difference between the left and right tibia and the right mandibular condyle (LRTibiaDiff-RMandCondyle), 3) leg length disparity and the right mandibular condyle (Left/Right LLD-RMandCondyle), and 4) leg length disparity and antemortem tooth loss asymmetry (Left/Right LLD-LRAMTL#Diff), indicating that LLD occurrence was associated with TMJ OA and asymmetrical antemortem tooth loss at different rates between the unfractured and fractured male groups. Ten out of twelve correlation values were positive in the unfractured group and negative in the fractured group, suggesting that morphological integration of the lower limbs and jaw was disrupted more frequently in the fractured group. Fractured males were therefore more likely to exhibit fracture-induced LLD associated with TMJ OA and asymmetrical antemortem tooth loss than unfractured males.

In the Female Unfractured vs. Fractured group, significant LLD correlation differences were observed for the 1) difference between the left and right femur and the left mandibular condyle (LRFemurDiff-LMandCondyle), 2) difference between the left and right tibia and the left mandibular condyle (LRTibiaDiff-LMandCondyle), 3) difference between the left and right tibia and right antemortem tooth loss (LRTibiaDiff-RightAMTL), 4) difference between the left and right tibia and left antemortem tooth loss (LRTibiaDiff-LeftAMTL), 5) difference between the left and right fibula and the right mandibular fossa (LRFibulaDiff-RMandFossa), 6)

difference between the left and right fibula and the right tooth wear average (LRFibulaDiff-RToothWearAvg), 7) difference between the left and right fibula and the left tooth wear average (LRFibulaDiff-LToothWearAvg), 8) leg length disparity and the left mandibular condyle (Left/Right LLD-LMandCondyle), 9) leg length disparity and the left tooth wear average (Left/Right LLD-LToothWearAvg), 10) leg length disparity and right antemortem tooth loss (Left/Right LLD-RightAMTL#), and 11) leg length disparity and left antemortem tooth loss (Left/Right LLD-LeftAMTL#). This group showed the largest number of significant correlation differences across the entire sample and sub-sample comparisons, and the correlation values suggest that the co-occurrence of LLD with TMJ OA, antemortem tooth loss, and heavy dental attrition were significantly different between the female unfractured and fractured groups. However, no clear pattern between the unfractured and fractured female groups could be identified. In fact, the pattern represented by correlation data is that significant changes in correlation strength and direction are inconsistent and erratic in the female sub-sample relative to comparisons of the entire sample and the male sub-sample. It is likely that the variability observed in the female sample is the result of a number of confounding factors relating to female anatomy and biology that obfuscate the patterns of morphological integration between the lower limbs and jaw in the unfractured and fractured groups.

Overall, the analysis of the significant correlation values suggests that patterns of morphological integration were more frequently disrupted in fractured individuals, which may have caused biomechanical forces to be altered, placing undue stress on affected areas of the body and increasing the likelihood of the development of ancillary pathologies. Taken as a whole, these values support the hypothesis that LLD resulting from fractures may cause ancillary pathological conditions of the jaw, such as TMJ OA, dental attrition, and antemortem tooth loss.

These findings are congruent with recent clinical literature that has identified a link between LLD and jaw dysfunction in living patients (Blum, 2008; Maeda et al., 2011; Park and Bae, 2014). This study suggests that given sufficient time, the pathological conditions identified in soft tissue clinical studies (e.g., dental malocclusion and TMD) may manifest skeletally and be detectable in osteological analyses.

The results of the MANOVA indicate that fracture status, sex, age, and ancestry all significantly influence correlation differences between samples; however, the post-hoc ANOVA analyses only demonstrated significance of individual dependent variables for ancestry and sex. In the ancestry analysis, there were two variables (Ltibia, Rtibia) with significant p-values and moderately strong observed power that drive the significant ancestry MANOVA effect. Based on the post-hoc ANOVA analysis by sex, a total of five variables (Ltibia, Rtibia, Lfemur, Rfemur, LToothWearAvg) were significant, with high observed power values, which drive the significant sex MANOVA effect. This suggests an interesting pattern for the sex-based analysis and indicates that sex may be an important factor driving the differences between the analyzed samples. The lack of significant post-hoc ANOVA variables for fracture status and age indicates that all measurements slightly contribute to the significant MANOVA effect, but none of the individual measurements alone are driving that significant effect.

Sex Differences

Musculoskeletal Anatomy and Endocrinology

The observed disparity in correlation patterns between the male and female groups may be explained by the sexually dimorphic characteristics of male and female skeletal anatomy. The pelvis is the skeletal element tied most closely to the requirements of childbirth and displaying

the most sexually dimorphic characteristics (Byers, 2007). The morphology of the human pelvis was shaped by evolution to accommodate a bipedal stance, but in females, the pelvis must also accommodate the demands of childbirth (Tague, 1986). Because the female pelvis must be wide enough to allow a fetus to pass through the birth canal, it is thus wider and shorter than the male pelvis.

The differences in pelvic morphology between males and females extend to the lower limb anatomy. In females, because the pelvic girdle is generally wider than it is in males, the femur joins the pelvis at a more lateral angle than it does in males. This affects both the anatomy and function of the lower limbs in males and females. The Quadriceps Angle, or Q-Angle, describes the angle of alignment of the patella with the femur and pelvis. The Q-Angle is measured from the centermost point of the patella to the anterior superior iliac spine of the pelvis and the center of the tibial tubercle (Figure 27). In males, a normal Q-angle range is 10° - 14° , while in females, it generally falls between 15° - 17° (Malik and Malik, 2015:112).

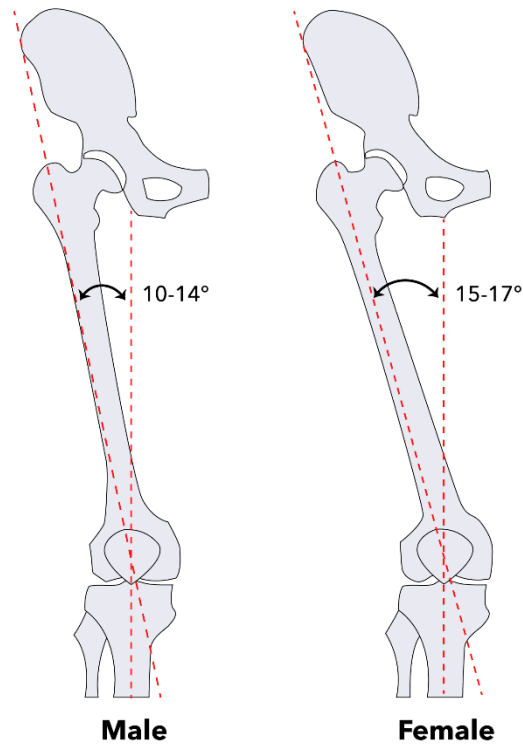


Figure 27: Differences in Q-Angle between males and females. Note the more lateral positioning of the femur in the female leg as opposed to the more upright positioning of the male (illustrated by Olivier Lacan, after Malik and Malik, 2015).

Because of the differences between male and female lower limb anatomy, the biomechanical forces of ambulation are somewhat different for the two sexes. An extensive body of clinical research has documented differences in gait kinematic patterns between males and females (Kerrigan et al., 1998; Smith et al., 2002) and a higher prevalence of sports and activity-related injuries in females than in males (Geraci and Brown, 2005). Studies have shown that females tend to exhibit greater pelvic obliquity, hip adduction, and internal rotation, as well as lower center of mass displacement than males in both walking and running gait (Smith et al., 2002; Ferber et al., 2003). Several explanations for these differences have been posited. Malik

and Malik (2015:112) attribute these differential gait and injury patterns to differences in male and female Q-Angle morphology, noting that “the greater the Q-angle, the greater the lateral force on the patella, and a Q-angle of more than 20° is a risk factor for patella subluxation and patellofemoral joint pain.” As a result, females, especially female athletes, are nearly twice as likely as males to develop knee injuries and joint pain (Geraci and Brown, 2005).

Differential sex hormone levels between males and females may also account for differences in musculoskeletal factors influencing gait kinematics. The male sex hormone testosterone serves a number of important functions in male development and is a primary driving force behind the differences between the male and female phenotypes. In addition to its role in male sexual maturation and reproduction, testosterone functions as an anabolic steroid, increasing the size and strength of skeletal and muscular tissues in both young and old men (Bhasin et al., 2003). Testosterone is also produced in females, but to a lesser degree than it is in males; on average, testosterone levels are 7-8 times higher in males than in females (Torjesen and Sandnes, 2004). As a result, males generally have greater total muscle mass than females. Lower limb musculature is considerably larger and stronger in individuals with higher testosterone levels; one study by Bhasin et al. (1996) demonstrated that individuals who did not exercise but were administered testosterone on a regular basis had significantly larger quadriceps regions after 10 weeks of treatment. Higher testosterone levels promote musculoskeletal stability of the legs and reduce the mechanical workload of joints during ambulation, resulting in distinct differences between male and female gait kinematics (Kerrigan et al., 1998). In particular, the difference in the size and strength of the quadriceps region drives differences between the male and female gait; in females, quadriceps activation during knee flexion is approximately 20% higher than in males, and fatigue occurs more rapidly (Wünschel et al., 2013). In conjunction

with the wider female Q-Angle, this results in lower joint stability and increased risk of injury relative to males.

Recent orthopedic and sports science studies have examined the role of the female sex hormone estrogen in the occurrence of lower limb injuries in females. It has been argued that estrogen increases laxity of the lower limb musculature, reducing the muscular reinforcement of the leg bones and predisposing females to injury and altered gait (Slauterbeck et al., 1999). This is thought to be the result of cyclically elevated estrogen levels during the menstrual cycle that reduce collagen content in soft tissue and decrease tensile strength of ligaments that act as anchors for the skeletal system (Shultz et al., 2005). Gray and colleagues (2016) investigated the correlation between the use of hormonal oral contraceptives and anterior cruciate ligament (ACL) injury. The authors analyzed groups of female soccer players with and without ACL injuries and found that injury occurrence was less common in the female athletes who were on birth control, suggesting that a reduction in natural estrogen levels decreases the risk of knee injury occurrence (Gray et al., 2016). Since the females in the present analysis lived and died in the early 20th century (before the advent of hormonal contraceptives), they would not have been able to regulate their estrogen levels with birth control; thus, they would most likely have experienced muscular laxity of the lower extremities during the menstrual cycle and been more susceptible to injury and altered gait.

The musculoskeletal mechanics of pregnancy and childbirth may also play a role in the perceived differences between male and female correlation patterns. During pregnancy, females experience a variety of hormonal and physiological changes that affect the lower limbs and gait kinematics. Because of their altered physiology, pregnant females tend to adjust their gait

pattern, walking at a slower velocity, with shorter average step length and increased duration of stance and support phases (Blaszczyk et al., 2016). The pregnancy hormone relaxin may also play a role in altering the physiology and gait of pregnant females. Relaxin is a hormone whose primary function is to increase laxity of the sacroiliac joint in order to facilitate childbirth (MacLennan, 1991). During pregnancy, relaxin levels spike to 10 times their normal levels, causing soft tissues to weaken, thereby increasing joint flexibility (Calguneri et al., 1982). The result is heightened laxity of the ligaments and other anchoring soft tissues of the lower extremities, which can cause lower extremity dysfunction and pain (Ponnapula and Boberg, 2010). Although there is no reliable method for determining maternity status osteologically, because the females in this sample were adult women during the early 20th century, it is likely that some or all of them experienced pregnancy and childbirth. Thus, they may have undergone considerable changes in gait kinematics, which may explain their deviation from the male correlation pattern. Because males do not produce large quantities of estrogen or undergo the hormonal changes involved in pregnancy and childbirth, they are less likely to experience lower limb instability or injury due to muscular laxity.

It is likely that the structural, hormonal, and biomechanical differences between male and female physiology account for the observed correlation differences between the male and female samples in this study. Since absolutely higher Kendall's Tau correlation values were observed in the male sample, it is possible that the biomechanical forces between the lower limbs and jaw travel more directly through the male body, whereas in females, they are disrupted somewhat by the more widely-set pelvis and femora. Females are also subject to a much greater range of factors influencing gait kinematics, which may account for the observed variability. Thus, male and female LLD patients may experience differential biomechanical consequences of the

disparity between their left and right lower limbs, and these differences should be taken into account in diagnoses of modern medical cases and in paleopathological analyses of past populations. Males may be more likely to develop clearer patterns of temporomandibular changes in the event of fracture-induced LLD, while females may not. The differential response of males and females to a discrepancy between lower limb lengths may indicate that males are more susceptible to developing ancillary pathological conditions that may impact their social status.

Social Experience

Another explanation for the observed differences in the male and female patterns may be different lifetime social experiences. The individuals housed in the HTOC represent a modern, industrialized population from late 19th and early 20th century Cleveland, Ohio. During the roughly 100-year period in which the individuals in this collection lived and died, Cleveland developed from a small trading province to a diverse, urban industrial center (Miller and Wheeler, 1997). The construction of an efficient railroad system in the mid-19th century spurred massive population growth through the immigration of both native and foreign-born citizens and led to the development of Cleveland's most lucrative industries, iron and steel manufacturing (Miller and Wheeler, 1997:70-71). As members of a late 19th and early 20th century urban industrial community, the men and women analyzed in this study lived according to the prescribed gender roles of the era which included a largely sex-based division of labor. In late 19th century Cleveland, the majority of employment opportunities consisted of hard manual labor in iron and steel production, construction, machining, oil refinement, shipping, and manufacturing. These industries employed the majority of the male workforce, although

wealthier males were often employed as doctors, lawyers, and business entrepreneurs (Miller and Wheeler, 1997). By the early 20th century, Cleveland's economy had expanded to include automobile manufacturing, slaughtering and meat packing, clothing production, printing and publication, and the paint and varnish industry (Miller and Wheeler, 1997:101).

During the early 19th century, women were largely absent from the paid workforce, occupying themselves instead with securing a marriage, bearing children, and maintaining a household (Rury, 1991). By 1880, however, nearly 18% of Cleveland workers were women, employed primarily as domestic servants, seamstresses, laundresses, teachers, and clerks; the garment production industry also employed primarily women in their factories (Miller and Wheeler, 1997:87). By 1930, female labor, especially in professional and clerical services, was increasingly common. However, even after women began entering the paid workforce, many returned to the domestic sphere after marriage (Rury, 1991:5).

Because of their different employment opportunities and social roles, the men and women analyzed in this study would have experienced different levels of physical exertion and occupational risk of injury during life. Working class men were considerably more likely to engage in physically demanding and/or risky manual labor and were therefore more likely to experience the long-term biomechanical consequences of fractures and LLD. While the women in the sample may have been formally employed, they would have occupied roles involving relatively low physical hardship and would thus not have been subject to the same degree of activity-related biomechanical stress as their male counterparts. It is also possible that some or all of the women in this sample were not employed in formal occupations and instead carried out primarily domestic and less physically demanding duties as homemakers. This may explain why LLD and TMD were correlated more significantly and consistently in the male sample than they

were in the female sample; the male sample may reflect a higher degree of biomechanical stress after lower limb fractures than was experienced by the female population, resulting in the increased likelihood of comorbidity identified in the male sample.

The differential male and female social experience of late 19th and early 20th century urban American society likely dictated different social consequences of lower limb fractures and LLD for the men and women of the era. During this time period, social perceptions of masculinity and femininity underpinned the gender-based division of labor between the public and private spheres; males were primarily employed outside the home and females within it – either in their own homes or in the homes of others (Laslett and Brenner, 1989). Men were the primary wage earners in the American family, and contemporary perceptions of masculinity emphasized a man’s physical prowess and ability to work in a gainful profession (Rotundo, 1983). In Cleveland’s turn of the century economy, able-bodied working class men employed in the manufacturing, construction, and shipping industries fulfilled social expectations of masculinity, while men of underdeveloped physique or diminutive stature were deemed physically deficient and unable to participate in physically demanding employment by society (Baynton, 2013). Lower limb fractures would have represented a considerable physical impairment to working class males who relied on ambulatory mobility and strength to carry out their duties as manual laborers and breadwinners. In the absence of modern medical intervention, fractures were considerably less likely to be properly reunited and immobilized during healing, increasing the likelihood of long-term or permanent complications such as LLD. If these complications prevented the individual’s return to the workforce and fulfillment of the requisite breadwinning duties of manhood, he may have been considered disabled by turn of the century Cleveland society. Men incapacitated by injury and unable to work were often confined to the

home, where they were cared for by their wives (Abel, 2000). Men in such situations were generally looked down upon by society, as productivity and willingness to work were highly valued, and unproductive individuals were seen as “burdens” to their families and society (Abel, 2000:161-162).

Women who were affected by lower limb fractures were likely at lower risk of being considered disabled than males. While women also provided for their families during this time period, female working opportunities were available both outside and within the home, as domestic duties were of chief importance to the feminine ideal (Laslett and Brenner, 1989). Because female work was generally less physically demanding, lower limb injuries were unlikely to prevent women from providing for their families and fulfilling the social expectations of womanhood. Women with long-term impairments such as fracture-induced LLD were thus more likely to avoid disability status and to maintain their sense of identity within turn of the century society.

Implications

This study has several important implications for the analysis and interpretation of social identity in both archaeological and modern populations. First, this analysis has demonstrated that several trends identified in the clinical literature are in fact detectable in osteological remains and should be considered in bioarchaeological analyses. As expected, fractured individuals were more likely to exhibit LLD, demonstrating that comorbidity can and does occur and should be considered a possibility in paleopathological analyses. In particular, fractures of the tibia and fibula were most commonly associated with LLD, although femoral fractures were also responsible for a lower proportion of the LLD observed in the sample. This suggests that while

any fracture of the lower limbs can result in LLD, tibial and fibular fractures are particularly likely to cause a disparity in leg length, as they are the most common clinically. In addition, clear differences between male and female physiology and gait were discussed in the literature, and different patterns of morphological integration were identified for males and females in this analysis. This suggests that increased sensitivity to differences in male and female lower limb biomechanics can improve our interpretations of past injury expression.

The correlation trend identified in the clinical literature between LLD and TMD is still pertinent today, as modern populations still currently experience these issues, and medical professionals need every tool at their disposal to effectively diagnose and mitigate the symptoms that a patient with LLD may experience. This is especially important to those populations most at risk of developing clinically significant LLD, such as young children with lower limb fractures, elder individuals with frail bones at risk of incurring fractures, and military personnel who may injure or lose appendages in combat. Medical professionals should be especially cognizant of the ancillary pathological conditions that may develop as a result of fracture-related LLD and be prepared to not only diagnose and treat the local injury site, but also to be vigilant of conditions possibly developing in correlated regions. Better treatment methods contribute to improved long-term outlook and may prevent long-term disability status.

The interpretation of the social identity of the men and women analyzed in this study highlights the debate surrounding bioarchaeological disability studies and sheds light on the question of whether or not bioarchaeologists can and should interpret social identity and disability status from pathological conditions. Despite their polarized stances, the arguments of both proponents and critics of archaeological disability studies have their merits. Dettwyler (1991) is right to advise caution in archaeological interpretations of care and compassion; as she

points out in her critique of several paleoanthropological studies of the 1970's, archaeologists can and often do overextend their interpretations to make claims about compassion and care that are not substantiated by the available evidence. As Tilley (2015) points out, however, Dettwyler's criticism of disability studies goes too far, discouraging bioarchaeologists from interpreting impairment and disability where it may be possible to do so. By employing appropriate methodology such as the Index of Care (2014), it is possible to determine not only the degree of impairment an injured individual experienced, but also how he or she was perceived by society and whether or not caregiving from other members of the community would have been required. As Lovell (2016) demonstrates in her analysis of an ancient Roman man with LLD, taking all of the osteological, archaeological, and historical evidence into account allows bioarchaeologists to draw informed, accurate conclusions about an individual's disability status in past society. The analysis presented here further supports the conclusion that holistic bioarchaeological analyses can provide insight into both past and present perceptions of the long-term injury experience and disability.

Limitations

There were several logistical constraints that limited the scope of this study. In the first place, the HTOC contains considerably more male remains than female, such that the sample used in this study was heavily biased toward males. This bias was reflected in the fractured sample, and the control group had to be similarly proportioned in order to allow for robust statistical analyses. Sample size was also an issue because time limitations prevented the measurement and analysis of a larger group of individuals. Furthermore, both the test group and

control group reflected a wide age range because time and logistical restrictions did not allow for the selection of consistent age ranges.

CHAPTER 6: CONCLUSION

In order for biological anthropologists to cultivate a holistic understanding of human life, health, and well-being, a more accurate understanding of the human injury experience is imperative. Morphological integration studies are a fruitful means of investigating correlations and covariance between related skeletal components and have the potential to provide crucial insight into the holistic effects of skeletal pathological conditions.

This study offers the first bioarchaeological analysis of morphological integration between leg fractures, LLD, and TMD and has demonstrated that these conditions often covary, or coexist. Based on this analysis, it appears that morphological integration between the lower limbs and jaw is frequently disrupted in fractured individuals, causing fractured individuals to be at a higher risk of developing TMD. However, this pattern is not consistent between the sexes; it is apparent from this analysis that significant differences exist between male and female lower limb biomechanics and that males and females have differential responses to lower limb fractures and LLD. Specifically, males are more likely to exhibit disrupted morphological integration of the lower limbs and jaw as measured by a significant reduction in absolute correlation strength and therefore may be more likely to develop TMD as an ancillary pathological condition. Females, on the other hand, may exhibit absolutely lower or absolutely higher statistically significant correlation strength differences in homologous measurements from fractured and unfractured samples. A clear conclusion of this study is that differences between male and female skeletal anatomy and lower limb function should be considered in biomechanical analyses, as males and females may experience differential risk of developing secondary pathological conditions. As a result, bioarchaeologists and modern medical

professionals should consider an individual's sex when determining an individual's long-term injury experience and disability status.

This study has also highlighted the importance of the debate surrounding the ability of bioarchaeologists to identify disability, caregiving, and social identity in the archaeological record. Although critics of disability studies are right to question the assumption that non-impaired individuals necessarily show compassion toward impaired group members, studies such as the one carried out by Lovell (2016) demonstrate that given sufficient information about a population's way of life, bioarchaeologists can interpret an individual's disability status based on how their injury would have been perceived by society. The present analysis further supports the argument that with appropriate caution and proper methodology, it is possible to infer an individual's degree of impairment and disability status from archaeological evidence. Such studies are important for the bioarchaeological discipline, as they shed light on ancient ways of life and contribute to our understanding of the human injury experience in both the past and the present.

Future Directions

Given this study's small sample size, the next step to furthering this analysis would be to collect data on additional individuals and repeat the steps of the analysis to increase statistical robusticity and determine if the patterns identified in this study remain consistent with a larger sample size. With more time and resources, it would also be possible to ensure equal male and female representation in the sample as well as a more consistent age range of analyzed individuals. Furthermore, with a larger sample size, it would be possible to sub-divide the sample into smaller groups and further explore the potential effects of age, ancestry, and fracture

location, affected bone, and state of healing on the correlation between LLD and jaw dysfunction.

Another potentially informative aspect of future studies would be to conduct a full osteological analysis of the individuals in the sample with the most serious LLD to determine if the altered biomechanical forces of an impaired gait manifested elsewhere in the body. Since LLD affects the alignment of the feet, knees, hips, and shoulders, there may be evidence of other pathological conditions in these areas of LLD occurrence, which would further support the argument that LLD results in ancillary pathological conditions outside of the lower limbs.

APPENDIX A: RAW SAMPLE DATA TABLES

Table 9: Raw data for entire control (unfractured) sample.

Individual	ID	Sex	Age (years)	Ancestry	Lfemur (mm)	Rfemur (mm)	LRFemurDiff (mm)	Ltibia (mm)	Rtibia (mm)	LRTibiaDiff (mm)	Lfibula (mm)	Rfibula (mm)	LRFibDiff (mm)	Left/Right LLD (mm)	LMandFossa (0-3)	RMandFossa (0-3)	LMandCondyle (0-3)	RMandCondyle (0-3)	RToothWearAvg (0-4)	LToothWearAvg (0-4)	LRToothWearAvgDiff (0-4)	Right AMTL #	Left AMTL #	LRAMTL#Diff	
1	HTH 0026	Male	40	White	449.71	447.24	2.47	368.96	372.3	3.34	375.79	370.88	4.91	10.72	1	1	0	0	0.75	0	0.75	3	3	0	
9	HTH 0311	Female	56	White	445.52	447.52	2	348.26	346.77	1.49	340.24	342.87	2.63	6.12	0	0	0	0				16	16	0	
13	HTH 0788	Male	57	White	476.07	475.3	0.77	396.34	397.8	1.46	395.84	397.07	1.23	3.46	0	0	0	0	1.18	1.19	0.01	1	1	0	
14	HTH 0895	Male	60	White	479.62	483.71	4.09	379.74	370.53	9.21	383.4	383.98	0.58	13.88	0	1	0	0				14	14	0	
18	HTH 1666	Male	57	Black	449.33	445.39	3.94	369.39	365.15	4.24	360.53	361.38	0.85	9.03	0	0	1	1	0.5	0	0.5	2	4	2	
19	HTH 1827	Male	54	Black	478.64	478.81	0.17	387.11	387.37	0.26	377.31	376.9	0.41	0.84	0	0	1	0	0.83	1.67	0.84	9	8	1	
20	HTH 2269	Female	58	Black	425.07	424.85	0.22	339.36	336.38	2.98					0	0	0	0				16	16	0	
21	HTH 2276	Female	38	Black	426.09	422.3	3.79	334.63	338.71	4.08	328.44	332.91	4.47	12.34	0	0	0	0	0.13	0.15	0.02	0	0	0	
22	HTH 2325	Male	75	White	459.95	457.65	2.3	364.99	361.95	3.04	362.45	357.7	4.75	10.09	1	1	0	0	2.5			3	5	2	
24	HTH 0584	Male	25	White	442.43	442.12	0.31	349.47	349.13	0.34	343.81	342.15	1.66	2.31	0	0	0	0	0	0	0	0	0	0	
31	HTH 0741	Male	63	White	440.39	436.34	4.05	362.72	365.26	2.54	357.38	360.41	3.03	9.62	1	1	0	0	3.25	2	1.25	2	6	4	
32	HTH 2395	Female	52	Black	464.53	464.17	0.36	385.02	389.81	4.79	382	381.88	0.12	5.27	1	1	0	0	1.31	0.69	0.62	3	4	1	
33	HTH 2531	Male	52	White	453.31	451.41	1.9	360.62	357.26	3.36	347.29	338.67	8.62	13.88	0	0	0	0	1.61	1.88	0.27	6	6	0	
34	HTH 2597	Female	34	Black	440.4	441.44	1.04	358.03	357.07	0.96	350.79	352.76	1.97	3.97	0	0	0	0	0	0.17	0.17		9	6	3
35	HTH 2646	Female	43	Black	467.81	465.65	2.16	389.12	390.99	1.87	388.35				0	0	0	0				16	16	0	
36	HTH 2749	Male	42	Black	476.47	476.47	0	407.85			386.25				2	2	0	0	1			15	16	1	
37	HTH 933	Female	38	Black	444.27	449.5	5.23	375.19	379.6	4.41	364.02	365.6	1.58	11.22	0	0	1	1	0.17	0.17	0	7	5	2	
42	HTH 2778	Male	40	White	437.96	434.38	3.58	349.34	355.17	5.83	336.18	339.43	3.25	12.66	0	0	1	1	2.18	1.5	0.68	4	5	1	
43	HTH 2828	Male	65	Black	499.72	493.32	6.4	421.74	413.11	8.63	411.34	402.38	8.96	23.99	0	1	0	0				16	16	0	
46	HTH 3111	Female	38	White	391.8	393.45	1.65	306.81	306.96	0.15	302.15	301.36	0.79	2.59	0	1	0	0				16	15	1	
49	HTH 3118	Female	54	White	397.61	400.65	3.04	308.48	308.77	0.29	307	305.51	1.49	4.82	0	0	0	0	2	1	1	12	12	0	

Individual	ID	Sex	Age (years)	Ancestry	Lfemur (mm)	Rfemur (mm)	LRFemurDiff (mm)	Ltibia (mm)	Rtibia (mm)	LRTibiaDiff (mm)	Lfibula (mm)	Rfibula (mm)	LRFibDiff (mm)	Left/Right LLD (mm)	LMandFossa (0-3)	RMandFossa (0-3)	LMandCondyle (0-3)	RMandCondyle (0-3)	RToothWearAvg (0-4)	LToothWearAvg (0-4)	LRToothWearAvgDiff (0-4)	Right AMTL #	Left AMTL #	LRAMTL#Diff
50	HTH 3242	Male	58	White	456.83	456.95	0.12	371.82	369.23	2.59	360.26	358	2.26	4.97	0	0	0	0	2.5	2.45	0.05	1	2	1
51	HTH 0385	Male	37	White	428.62	424.18	4.44	358.85	361.03	2.18	354.96	356.32	1.36	7.98	1	1	0	0	0.33	0.63	0.3	5	6	1
52	HTH 0440	Male	50	White	466.27	467.57	1.3	387.5	387.93	0.43	378.95	375.23	3.72	5.45	1	1	1	0	0.43	0.75	0.32	9	11	2
53	HTH 2683	Male	41	White	446.89	450	3.11	356.39	356.44	0.05	358.15	358.43	0.28	3.44	1	1	0	0	0.92	0.67	0.25	3	7	4
54	HTH 0344	Male	49	White	440.81	438.99	1.82	356.27	355.55	0.72	352.24	349.8	2.44	4.98	1	1	0	0	0.29	1.57	1.28	3	1	2
56	HTH 2207	Male	40	White	437.85	438.36	0.51	360.75	359.68	1.07	355.05	353.32	1.73	3.31	1	1	0	0	0.25	0	0.25	13	14	1

Table 10: Raw data for entire test (fractured) sample.

Individual	ID	Sex	Age (years)	Ancestry	Lfemur (mm)	Rfemur (mm)	LRFemurDiff (mm)	Ltibia (mm)	Rtibia (mm)	LRTibiaDiff (mm)	Lfibula (mm)	Rfibula (mm)	LRFibulaDiff (mm)	Left/Right LLD (mm)	LMandFossa (0-3)	RMandFossa (0-3)	LMandCondyle (0-3)	RMandCondyle (0-3)	RToothWearAvg (0-4)	LToothWearAvg (0-4)	LRToothWearAvgDiff (0-4)	Fracture Healing (0-2)	Right AMTL #	Left AMTL #	LRAMTL#Diff
2	HTH 0204	Female	27	White	386.15	388.94	2.79	303.55	302.39	1.16	302.4	300.01	2.39	6.34	0	0	0	0	0.46	0	0.46	2	0	1	1
3	HTH 0339	Female	38	White	444.74	444.13	0.61	347.64	349.37	1.73	339.04	340.03	0.99	3.33	0	0	1	0	0.59	0.56	0.03	2	2	4	2
4	HTH 0444	Male	45	White	444.16	444.46	0.3	345.23	345.85	0.62	339.14	342.11	2.97	3.89	1	0	0	0	0.6	0.87	0.27	1	0	0	0
5	HTH 0459	Male	40	White	438.76	441.07	2.31	354.01	362.43	8.42	349.69	353.42	3.73	14.46	1	1	0	0	2.06	1.93	0.13	2	0	1	1
6	HTH 0479	Male	58	White	454.69	446.5	8.19	368.25	367.87	0.38	368.05	362.99	5.06	13.63	1	1	0	0	2.33	2	0.33	2	8	6	2
7	HTH 0500	Male	40	White	465.62	467.51	1.89	383.89	369.33	14.56	383.68	372.24	11.44	27.89	1	0	0	0	1.33	2.15	0.82	2	12	1	11
8	HTH 0534	Male	68	White	449.31	451.53	2.22	364.31	364.24	0.07		359.01			2	2	0	0	0.58	2.13	1.55	2	1	5	4
10	HTH 0354	Male	48	White	473.51	478	4.49	398.5	398.97	0.47	388.46	387.94	0.52	5.48	0	1	0	0	2	2	0	2	5	5	0
11	HTH 0631	Female	36	White	448.12	446.43	1.69	353.18	358.08	4.9	340.49	342.44	1.95	8.54	0	0	0	0	0	0	0	2	1	3	2
12	HTH 0718	Male	39	Black	447.7	450.39	2.69	383.9	386.73	2.83	376.63	377.06	0.43	5.95	0	0	0	0	0.86	1	0.14	2	2	2	0
15	HTH 1552	Male	76	White	434.05	420.08	13.97	350.85	349.2	1.65	347.19	343.37	3.82	19.44	1	1	0	0	2	2.5	0.5	2	11	14	3

Individual	ID	Sex	Age (years)	Ancestry	Lfemur (mm)	Rfemur (mm)	LRFemurDiff (mm)	Ltibia (mm)	Rtibia (mm)	LRTibiaDiff (mm)	Lfibula (mm)	Rfibula (mm)	LRFibulaDiff (mm)	Left/Right LLD (mm)	LMandFossa (0-3)	RMandFossa (0-3)	LMandCondyle (0-3)	RMandCondyle (0-3)	RToothWearAvg (0-4)	LToothWearAvg (0-4)	LRToothWearAvgDiff (0-4)	Fracture Healing (0-2)	Right AMTL #	Left AMTL #	LRAMTL#Diff
16	HTH 1592	Male	68	White	457.46	457.74	0.28	358.84	357.99	0.85	351.91	354.35	2.44	3.57	0	0	1	1				1	15	14	1
17	HTH 1647	Female	63	White	387.95	388.48	0.53	322.62	310.28	12.34	316.02	300.11	15.91	28.78	0	0	0	0	1	1	0	2	14	14	0
23	HTH 0543	Male	38	Black	455.65	457.54	1.89	368.31	369.74	1.43	364.16	361.88	2.28	5.6	1	0	0	0	0.81	0.97	0.16	0	0	0	0
25	HTH 0587	Male	40	White	450.4	443.9	6.5	401.36			392.99				1	1	0	0	1.14	1.2	0.06	0	5	5	0
26	HTH 0602	Male	35	White	449.91	439.55	10.36	363.28	382.02	18.74	359.98	365.34	5.36	34.46	0	0	0	0	0	0.43	0.43	2	7	6	1
27	HTH 0666	Male	45	Black	490.32	488.06	2.26	425.49	427.72	2.23	415.28	418.88	3.6	8.09	0	0	0	0	1	0.63	0.37	2	8	7	1
28	HTH 0742	Female	50	White		437.79			373.13						0	0	0	1				0	7	10	3
29	HTH 0751	Female	65	Black	405.77	404.33	1.44	327.7	331.07	3.37	323.59	325.88	2.29	7.1	2	2	0	0	0.45	0.4	0.05	1.5	6	4	2
30	HTH 0868	Female	60	Black	467.49	464.22	3.27	376.44	371.56	4.88	372.74	368.08	4.66	12.81	0	1	0	0		0.33		2	15	12	3
38	HTH 0974	Male	45	Black	446.16	448.53	2.37	373.51	369.65	3.86	350.34	356.27	5.93	12.16	0	0	0	0	1.29	0.67	0.62	2	5	2	3
39	HTH 1124	Female	40	Black	413.49	414.78	1.29	361.94	355.74	6.2	350.85	351.94	1.09	8.58	0	0	0	0	0.15	0.13	0.02	2	3	4	1
40	HTH 1387	Male	65	White	396.38	394.11	2.27	335.61	332.97	2.64	318.91	321.5	2.59	7.5	1	1	0	0				1	16	16	0
41	HTH 1470	Male	65	White	431.14	428.05	3.09	362.79	364.24	1.45	356.59				0	0	1	0	0.55	1	0.45	0	3	5	2
44	HTH 3091	Male	54	White	420.68	420.2	0.48	342.66	337.34	5.32	338.92	338	0.92	6.72	0	0	0	0		2		1	14	13	1
45	HTH 3045	Male	66	White	432.9	426.48	6.42	379.71	377.28	2.43	369.45	355.26	14.19	23.04	1	0	0	0	3.4	3	0.4	2	10	10	0
47	HTH 1534	Female	45	Black	466.66	466.53	0.13	400.87	397.89	2.98		396.94			0	0	0	0	1.08	1.25	0.17	0	7	5	2
48	HTH 1903	Male	32	Black		536.54		440.38	439.44	0.94	428.36	425.31	3.05	3.99	0	0	0	0	0.14	0.32	0.18	0	0	1	1
55	HTH 2761	Female	46	Black	404.6	402.5	2.1	343.26	338.26	5	337.49	334.01	3.48	10.58	0	0	0	0	0.45	0.17	0.28	2	5	4	1

APPENDIX B: KENDALL'S TAU B CORRELATION TABLES

Table 11: Kendall's Tau correlation values, z-scores, and p-values for the All Unfractured and Fractured sample. Statistically significant values are starred and highlighted in bold.

Measurement	Kendall's Tau Correlation		Z-score	P-value
	Unfractured	Fractured		
Lfemur-LMandFossa	0.06	-0.05	0.40	0.34
Lfemur-RMandFossa	0.15	0.06	0.32	0.37
Lfemur-LMandCondyle	0.10	0.00	0.36	0.36
Lfemur-RMandCondyle	-0.10	0.17	-0.95	0.17
Lfemur-RToothWearAvg	0.19	0.20	-0.04	0.48
Lfemur-LToothWearAvg	0.20	0.15	0.21	0.42
Lfemur-RightAMTL	0.04	0.06	-0.09	0.46
Lfemur-LeftAMTL	0.06	0.02	0.12	0.45
Rfemur-LMandFossa	0.05	-0.08	0.47	0.32
Rfemur-RMandFossa	0.14	-0.03	0.59	0.28
Rfemur-LMandCondyle	0.10	0.02	0.28	0.39
Rfemur-RMandCondyle	-0.11	0.06	-0.63	0.27
Rfemur-RToothWearAvg	0.16	0.12	0.14	0.44
Rfemur-LToothWearAvg	0.22	0.10	0.42	0.34
Rfemur-RightAMTL	0.09	-0.11	0.69	0.24
Rfemur-LeftAMTL	0.11	-0.20	1.10	0.14
LRFemurDiff-LMandFossa	-0.07	0.12	-0.68	0.25
LRFemurDiff-RMandFossa	0.08	0.31	-0.84	0.20
LRFemurDiff-LMandCondyle	0.11	-0.16	0.97	0.17

Measurement	Kendall's Tau Correlation		Z-score	P-value
	Unfractured	Fractured		
LRFemurDiff-RMandCondyle	0.327	-0.251	2.11	0.02*
LRFemurDiff-RToothWearAvg	0.00	0.23	-0.82	0.21
LRFemurDiff-LToothWearAvg	-0.12	0.15	-0.96	0.17
LRFemurDiff-RightAMTL	-0.02	0.04	-0.20	0.42
LRFemurDiff-LeftAMTL	0.00	0.13	-0.46	0.32
Ltibia-LMandFossa	0.19	-0.08	0.95	0.17
Ltibia-RMandFossa	0.19	-0.03	0.79	0.22
Ltibia-LMandCondyle	0.19	-0.11	1.07	0.14
Ltibia-RMandCondyle	0.02	-0.04	0.22	0.41
Ltibia-RToothWearAvg	0.12	0.21	-0.32	0.37
Ltibia-LToothWearAvg	0.13	0.15	-0.06	0.47
Ltibia-RightAMTL	0.02	-0.03	0.17	0.43
Ltibia-LeftAMTL	0.04	-0.07	0.37	0.35
Rtibia-LMandFossa	0.154	-0.223	1.35	0.09**
Rtibia-RMandFossa	0.15	-0.14	1.02	0.15
Rtibia-LMandCondyle	0.20	-0.13	1.16	0.12
Rtibia-RMandCondyle	0.03	0.06	-0.11	0.46
Rtibia-RToothWearAvg	0.17	0.12	0.18	0.43
Rtibia-LToothWearAvg	0.12	0.07	0.19	0.43
Rtibia-RightAMTL	-0.06	-0.05	-0.01	0.50
Rtibia-LeftAMTL	-0.04	-0.09	0.18	0.43

Measurement	Kendall's Tau Correlation		Z-score	P-value
	Unfractured	Fractured		
LRTibiaDiff-LMandFossa	-0.09	-0.17	0.29	0.39
LRTibiaDiff-RMandFossa	-0.07	-0.19	0.41	0.34
LRTibiaDiff-LMandCondyle	0.13	-0.19	1.13	0.13
LRTibiaDiff-RMandCondyle	0.398	-0.189	2.16	0.02*
LRTibiaDiff-RToothWearAvg	0.11	-0.04	0.50	0.31
LRTibiaDiff-LToothWearAvg	-0.02	-0.11	0.29	0.39
LRTibiaDiff-RightAMTL	-0.08	0.19	-0.97	0.17
LRTibiaDiff-LeftAMTL	-0.13	0.05	-0.62	0.27
Lfibula-LMandFossa	0.19	-0.02	0.76	0.22
Lfibula-RMandFossa	0.26	0.02	0.86	0.20
Lfibula-LMandCondyle	0.08	-0.06	0.50	0.31
Lfibula-RMandCondyle	-0.06	0.02	-0.30	0.38
Lfibula-RToothWearAvg	0.15	0.20	-0.16	0.44
Lfibula-LToothWearAvg	0.06	0.19	-0.46	0.32
Lfibula-RightAMTL	0.12	-0.01	0.49	0.31
Lfibula-LeftAMTL	0.15	-0.06	0.72	0.24
Rfibula-LMandFossa	0.16	-0.12	0.99	0.16
Rfibula-RMandFossa	0.25	-0.04	1.03	0.15
Rfibula-LMandCondyle	0.18	-0.12	1.06	0.14
Rfibula-RMandCondyle	0.01	0.00	0.03	0.49
Rfibula-RToothWearAvg	0.15	0.14	0.01	0.49

Measurement	Kendall's Tau Correlation		Z-score	P-value
	Unfractured	Fractured		
Rfibula-LToothWearAvg	0.04	0.14	-0.35	0.36
Rfibula-RightAMTL	-0.01	-0.04	0.12	0.45
Rfibula-LeftAMTL	0.02	-0.10	0.41	0.34
LRFibulaDiff-LMandFossa	0.05	0.20	-0.54	0.30
LRFibulaDiff-RMandFossa	0.00	0.02	-0.06	0.48
LRFibulaDiff-LMandCondyle	-0.08	-0.21	0.46	0.32
LRFibulaDiff-RMandCondyle	-0.05	-0.05	0.00	0.50
LRFibulaDiff-RToothWearAvg	0.03	0.28	-0.89	0.19
LRFibulaDiff-LToothWearAvg	0.02	0.20	-0.64	0.26
LRFibulaDiff-RightAMTL	-0.01	0.24	-0.91	0.18
LRFibulaDiff-LeftAMTL	-0.05	0.12	-0.59	0.28
Fracture Healing-LMandFossa		-0.05	0.17	0.43
Fracture Healing-RMandFossa		0.10	-0.35	0.36
Fracture Healing-LMandCondyle		-0.18	0.66	0.26
Fracture Healing-RMandCondyle		-0.36	1.34	0.09**
Fracture Healing-RToothWearAvg		0.13	-0.47	0.32
Fracture Healing-LToothWearAvg		-0.01	0.05	0.48
Fracture Healing-RightAMTL		0.03	-0.12	0.45
Fracture Healing-LeftAMTL		-0.01	0.02	0.49

* Significant at $\alpha = 0.05$

** Significant at $\alpha = 0.10$

Table 12: Kendall's Tau correlation values, z-scores, and p-values for the Male Unfractured and Fractured sample. Statistically significant values are starred and highlighted in bold.

Measurement	Kendall's Tau Correlation		Z-score	P-value
	Unfractured	Fractured		
Lfemur-LMandFossa	-0.23	-0.11	-0.34	0.37
Lfemur-RMandFossa	0.03	-0.04	0.19	0.42
Lfemur-LMandCondyle	0.04	-0.04	0.24	0.41
Lfemur-RMandCondyle	-0.23	0.22	-1.26	0.10
Lfemur-RToothWearAvg	0.19	-0.02	0.59	0.28
Lfemur-LToothWearAvg	0.22	-0.17	1.09	0.14
Lfemur-RightAMTL	0.26	-0.04	0.84	0.20
Lfemur-LeftAMTL	0.24	-0.14	1.06	0.14
Rfemur-LMandFossa	-0.20	-0.21	0.03	0.49
Rfemur-RMandFossa	0.06	-0.16	0.62	0.27
Rfemur-LMandCondyle	0.00	-0.01	0.04	0.49
Rfemur-RMandCondyle	-0.29	0.22	-1.43	0.08**
Rfemur-RToothWearAvg	0.19	-0.14	0.94	0.17
Rfemur-LToothWearAvg	0.22	-0.27	1.38	0.08**
Rfemur-RightAMTL	0.30	-0.28	1.66	0.05**
Rfemur-LeftAMTL	0.29	-0.38	1.95	0.03*
LRFemurDiff-LMandFossa	-0.06	0.01	-0.19	0.42
LRFemurDiff-RmandFossa	0.18	0.28	-0.27	0.39
LRFemurDiff-LmandCondyle	-0.02	-0.16	0.37	0.35

Measurement	Kendall's Tau Correlation		Z-score	P-value
	Unfractured	Fractured		
LRFemurDiff-RmandCondyle	0.23	-0.33	1.61	0.05**
LRFemurDiff-RToothWearAvg	0.11	0.19	-0.23	0.41
LRFemurDiff-LToothWearAvg	-0.10	0.12	-0.62	0.27
LRFemurDiff-RightAMTL	0.08	0.08	0.00	0.50
LRFemurDiff-LeftAMTL	0.15	0.24	-0.28	0.39
Ltibia-LMandFossa	-0.02	-0.21	0.53	0.30
Ltibia-RMandFossa	0.13	-0.13	0.71	0.24
Ltibia-LMandCondyle	0.07	-0.17	0.67	0.25
Ltibia-RMandCondyle	-0.19	-0.14	-0.15	0.44
Ltibia-RToothWearAvg	0.16	0.03	0.38	0.35
Ltibia-LToothWearAvg	0.10	-0.19	0.83	0.20
Ltibia-RightAMTL	0.22	-0.18	1.12	0.13
Ltibia-LeftAMTL	0.26	-0.23	1.38	0.08**
Rtibia-LMandFossa	-0.09	-0.34	0.74	0.23
Rtibia-RmandFossa	0.08	-0.24	0.91	0.18
Rtibia-LmandCondyle	0.11	-0.17	0.77	0.22
Rtibia-RmandCondyle	-0.21	-0.17	-0.10	0.46
Rtibia-RtoothWearAvg	0.21	-0.11	0.89	0.19
Rtibia-LtoothWearAvg	0.13	-0.30	1.20	0.11
Rtibia-RightAMTL	0.11	-0.24	1.00	0.16
Rtibia-LeftAMTL	0.20	-0.22	1.19	0.12

Measurement	Kendall's Tau Correlation		Z-score	P-value
	Unfractured	Fractured		
LRTibiaDiff-LmandFossa	-0.27	-0.18	-0.28	0.39
LRTibiaDiff-RmandFossa	-0.10	-0.30	0.58	0.28
LRTibiaDiff-LmandCondyle	0.03	-0.16	0.51	0.31
LRTibiaDiff-RmandCondyle	0.41	-0.17	1.71	0.04*
LRTibiaDiff-RToothWearAvg	0.27	0.08	0.55	0.29
LRTibiaDiff-LToothWearAvg	0.08	-0.05	0.37	0.36
LRTibiaDiff-RightAMTL	0.10	0.18	-0.24	0.40
LRTibiaDiff-LeftAMTL	0.04	0.02	0.06	0.47
Lfibula-LMandFossa	0.03	-0.17	0.55	0.29
Lfibula-RMandFossa	0.27	-0.10	1.03	0.15
Lfibula-LMandCondyle	0.00	-0.10	0.28	0.39
Lfibula-RMandCondyle	-0.23	-0.09	-0.41	0.34
Lfibula-RToothWearAvg	0.14	-0.04	0.50	0.31
Lfibula-LToothWearAvg	0.01	-0.14	0.41	0.34
Lfibula-RightAMTL	0.30	-0.17	1.32	0.09**
Lfibula-LeftAMTL	0.36	-0.18	1.57	0.06**
Rfibula-LMandFossa	-0.12	-0.33	0.60	0.27
Rfibula-RmandFossa	0.16	-0.17	0.95	0.17
Rfibula-LmandCondyle	0.10	-0.13	0.63	0.27
Rfibula-RmandCondyle	-0.16	-0.12	-0.11	0.46
Rfibula-RtoothWearAvg	0.15	-0.18	0.93	0.18

Measurement	Kendall's Tau Correlation		Z-score	P-value
	Unfractured	Fractured		
Rfibula-LtoothWearAvg	-0.01	-0.29	0.80	0.21
Rfibula-RightAMTL	0.15	-0.26	1.14	0.13
Rfibula-LeftAMTL	0.26	-0.26	1.46	0.07**
LRFibulaDiff-LMandFossa	0.12	0.30	-0.51	0.30
LRFibulaDiff-RMandFossa	0.16	-0.04	0.56	0.29
LRFibulaDiff-LMandCondyle	-0.12	-0.17	0.13	0.45
LRFibulaDiff-RMandCondyle	-0.06	-0.16	0.26	0.40
LRFibulaDiff-RToothWearAvg	0.15	0.27	-0.34	0.37
LRFibulaDiff-LToothWearAvg	0.10	0.13	-0.09	0.46
LRFibulaDiff-RightAMTL	0.15	0.10	0.12	0.45
LRFibulaDiff-LeftAMTL	0.03	0.03	0.01	0.50
Fracture Healing-LMandFossa		0.08	-0.22	0.41
Fracture Healing-RMandFossa		0.21	-0.58	0.28
Fracture Healing-LMandCondyle		-0.38	1.12	0.13
Fracture Healing-RMandCondyle		-0.21	0.58	0.28
Fracture Healing-RToothWearAvg		0.42	-1.24	0.11
Fracture Healing-LToothWearAvg		0.29	-0.84	0.20
Fracture Healing-RightAMTL		0.12	-0.32	0.37
Fracture Healing-LeftAMTL		0.09	-0.26	0.40

* Significant at $\alpha = 0.05$

** Significant at $\alpha = 0.10$

Table 13: Kendall's Tau correlation values, z-scores, and p-values for the Female Unfractured and Fractured sample. Statistically significant values are starred and highlighted in bold.

Measurement	Kendall's Tau Correlation		Z-score	P-value
	Unfractured	Fractured		
Lfemur-LMandFossa	0.35	-0.12	0.88	0.19
Lfemur-RMandFossa	-0.09	0.22	-0.55	0.29
Lfemur-LMandCondyle	0.12	0.12	0.00	0.50
Lfemur-RMandCondyle	0.12		0.21	0.42
Lfemur-RToothWearAvg	0.00	-0.11	0.20	0.42
Lfemur-LToothWearAvg	0.11	0.14	-0.07	0.47
Lfemur-RightAMTL	-0.12	0.17	-0.52	0.30
Lfemur-LeftAMTL	-0.03	0.24	-0.50	0.31
Rfemur-LMandFossa	0.35	-0.15	0.93	0.17
Rfemur-RMandFossa	-0.09	0.11	-0.36	0.36
Rfemur-LMandCondyle	0.24	0.15	0.16	0.44
Rfemur-RMandCondyle	0.24	0.05	0.34	0.37
Rfemur-RToothWearAvg	0.00	-0.18	0.33	0.37
Rfemur-LToothWearAvg	0.11	0.14	-0.07	0.47
Rfemur-RightAMTL	-0.12	0.09	-0.38	0.35
Rfemur-LeftAMTL	-0.03	0.12	-0.27	0.39
LRFemurDiff-LMandFossa	-0.35	0.00	-0.67	0.25
LRFemurDiff-RMandFossa	-0.36	0.30	-1.23	0.11
LRFemurDiff-LMandCondyle	0.47	-0.24	1.35	0.09**

Measurement	Kendall's Tau Correlation		Z-score	P-value
	Unfractured	Fractured		
LRFemurDiff-RMandCondyle	0.47		0.92	0.18
LRFemurDiff-RtoothWearAvg	0.00	-0.47	0.92	0.18
LRFemurDiff-LtoothWearAvg	-0.32	-0.59	0.64	0.26
LRFemurDiff-RightAMTL	-0.24	-0.17	-0.14	0.44
LRFemurDiff-LeftAMTL	-0.20	-0.37	0.32	0.38
Ltibia-LmandFossa	0.35	-0.24	1.10	0.14
Ltibia-RmandFossa	-0.09	0.04	-0.24	0.41
Ltibia-LmandCondyle	0.24	0.00	0.43	0.33
Ltibia-RmandCondyle	0.24		0.43	0.33
Ltibia-RtoothWearAvg	0.00	-0.11	0.20	0.42
Ltibia-LtoothWearAvg	0.11	0.14	-0.07	0.47
Ltibia-RightAMTL	-0.18	0.17	-0.64	0.26
Ltibia-LeftAMTL	-0.09	0.24	-0.60	0.27
Rtibia-LmandFossa	0.35	-0.24	1.10	0.14
Rtibia-RmandFossa	-0.09	-0.04	-0.08	0.47
Rtibia-LmandCondyle	0.24	-0.12	0.65	0.26
Rtibia-RmandCondyle	0.24	0.35	-0.23	0.41
Rtibia-RtoothWearAvg	0.00	-0.14	0.26	0.40
Rtibia-LtoothWearAvg	0.11	0.11	-0.01	0.50
Rtibia-RightAMTL	-0.24	0.25	-0.91	0.18
Rtibia-LeftAMTL	-0.15	0.20	-0.63	0.26

Measurement	Kendall's Tau Correlation		Z-score	P-value
	Unfractured	Fractured		
LRTibiaDiff-LmandFossa	0.47	-0.07	1.05	0.15
LRTibiaDiff-RmandFossa	0.00	-0.05	0.09	0.46
LRTibiaDiff-LmandCondyle	0.35	-0.36	1.34	0.09**
LRTibiaDiff-RmandCondyle	0.35		0.67	0.25
LRTibiaDiff-RtoothWearAvg	0.20	-0.14	0.62	0.27
LRTibiaDiff-LtoothWearAvg	-0.11	-0.04	-0.12	0.45
LRTibiaDiff-RightAMTL	-0.43	0.36	-1.49	0.07**
LRTibiaDiff-LeftAMTL	-0.32	0.42	-1.39	0.08**
Lfibula-LmandFossa	0.36	-0.21	1.06	0.14
Lfibula-RmandFossa	-0.11	0.16	-0.48	0.32
Lfibula-LmandCondyle	0.21	0.07	0.26	0.40
Lfibula-RmandCondyle	0.21		0.39	0.35
Lfibula-RtoothWearAvg	0.00	-0.49	0.96	0.17
Lfibula-LtoothWearAvg	0.11	-0.11	0.39	0.35
Lfibula-RightAMTL	-0.19	0.07	-0.47	0.32
Lfibula-LeftAMTL	-0.11	0.16	-0.49	0.31
Rfibula-LmandFossa	0.54	-0.24	1.51	0.07**
Rfibula-RmandFossa	0.00	0.04	-0.08	0.47
Rfibula-LmandCondyle	0.36	0.00	0.67	0.25
Rfibula-RmandCondyle	0.36		0.67	0.25
Rfibula-RtoothWearAvg	0.00	-0.11	0.20	0.42

Measurement	Kendall's Tau Correlation		Z-score	P-value
	Unfractured	Fractured		
Rfibula-LtoothWearAvg	0.11	0.14	-0.07	0.47
Rfibula-RightAMTL	-0.49	0.17	-1.26	0.10
Rfibula-LeftAMTL	-0.43	0.24	-1.27	0.10
LRFibulaDiff-LmandFossa	-0.54	-0.07	-0.95	0.17
LRFibulaDiff-RmandFossa	-0.69	0.16	-1.81	0.04*
LRFibulaDiff-LmandCondyle	0.00	-0.50	0.99	0.16
LRFibulaDiff-RmandCondyle	0.00		0.00	0.50
LRFibulaDiff-RtoothWearAvg	-0.60	0.29	-1.79	0.04*
LRFibulaDiff-LtoothWearAvg	-0.74	0.18	-2.03	0.02*
LRFibulaDiff-RightAMTL	-0.10	0.43	-1.00	0.16
LRFibulaDiff-LeftAMTL	-0.05	0.40	-0.85	0.20
Fracture Healing-LMandFossa		-0.35	0.65	0.26
Fracture Healing-RMandFossa		-0.15	0.28	0.39
Fracture Healing-LMandCondyle		0.21	-0.38	0.35
Fracture Healing-RMandCondyle		-0.56	1.13	0.13
Fracture Healing-RToothWearAvg		-0.32	0.60	0.28
Fracture Healing-LToothWearAvg		-0.48	0.94	0.17
Fracture Healing-RightAMTL		-0.35	0.65	0.26
Fracture Healing-LeftAMTL		-0.27	0.49	0.31

* Significant at $\alpha = 0.05$

** Significant at $\alpha = 0.10$

LIST OF REFERENCES

- Abel EK. 2000. *Hearts of wisdom: American women caring for kin, 1850-1940*. Cambridge: Harvard University Press.
- Ackermann RR. 2005. Ontogenetic integration of the hominoid face. *J Hum Evol* 48:175–197.
- Adams JC, Hamblen DL. 1992. *Outline of fractures, including joint injuries*. Edinburgh: Churchill Livingstone.
- Agarwal SC, Glencross BA. 2011. *Social bioarchaeology*. Malden: Wiley-Blackwell.
- Bartle D, Keating J. 2013. Femoral and tibial fractures. *Surgery* 31(9):460-465.
- Bastir M. 2008. A systems-model for the morphological analysis of integration and modularity in human craniofacial evolution. *J Anthropol Sci* 86:37-58.
- Baynton DC. 2013. Disability and the justification of inequality in American history. In: Davis LJ, editor. *The disability studies reader*. 4th ed. New York: Routledge. p 17–33.
- Bhasin S, Woodhouse L, Storer TW. 2003. Androgen effects on body composition. *Growth Horm IGF Res* 13(Suppl A):S63–S71.
- Bhasin S, Storer TW, Berman N, Callegari C, Clevenger B, Phillips J, Bunnell TJ, Tricker R, Shirazi A, Casaburi R. 1996. The effects of supraphysiologic doses of testosterone on muscle size and strength in normal men. *N Engl J Med* 335:1–7.
- Blaszczyk JW, Opala-Berdzik A, Plewa M. 2016. Adaptive changes in spatiotemporal gait characteristics in women during pregnancy. *Gait Posture* 43:160-164.
- Blum C. 2008. The relationship between the pelvis and stomatognathic system: A position statement. *Sacro Occipital Tech Organ*:40–43.
- Blunstein SM, D’Amico JC. 1985. Limb length discrepancy: Identification, clinical significance, and management. *J Am Podiatr Med Assoc* 75(4):200-206.
- Boughner JC, Hallgrimsson B. 2008. Biological spacetime and the temporal integration of functional modules: A case study of dento-gnathic developmental timing. *Dev Dyn* 237(1):1-17.
- Buikstra JE, Ubelaker DH. 1994. *Standards for data collection from human skeletal remains*. Fayetteville: Arkansas Archaeological Survey.
- Brady RJ, Dean JB, Skinner TM, Gross MT. 2003. Limb length inequality: Clinical implications for assessment and intervention. *J Orthop Sports Phys Ther* 33(5):221-234.

- Byers SN. 2007. Attribution of sex. In: Introduction to forensic anthropology, 3rd ed. New York: Pearson. p 176-201.
- Calguneri M, Bird HA, Wright V. 1982. Changes in joint laxity occurring during pregnancy. *Ann Rheumat Dis* 41:126-128.
- Cheverud JM. 1995. Morphological integration in the saddleback tamarin (*Saguinus fuscicollis*) cranium. *Am Nat* 145:63-89.
- Corner BD, Richtsmeier JT. 1991. Morphometric analysis of craniofacial growth in *Cebus apella*. *Am J Phys Anthropol* 84(3):323-342.
- Cuccia AM. 2011. Interrelationships between dental occlusion and the plantar arch. *J Bodyw Mov Ther* 15:242-250.
- Dettwyler KA. 1991. Can paleopathology provide evidence for “compassion”? *Am J Phys Anthropol* 84:375–384.
- Dickel DN, Doran GH. 1989. Severe neural tube defect syndrome from the early archaic of Florida. *Am J Phys Anthropol* 80:325-334.
- Farella M, Michelotti A, Pellegrino G, Giani U, Martina R. 2005. Interexaminer reliability and validity for diagnosis of temporomandibular disorders of visual leg measurements used in dental kinesiology. *J Orofac Pain* 19:285–290.
- Ferber R, Davis IM, Williams III DS. 2003. Gender differences in lower extremity mechanics during running. *Clin Biomech* 18:350-357.
- Frayner DW, Horton WA, Macchiarelli R, Mussi M. 1987. Dwarfism in an adolescent from the Italian late Upper Paleolithic. *Nature* 330:60-62.
- Geraci MC, Brown W. 2005. Evidence-based treatment of hip and pelvic injuries in runners. *Phys Med Rehab Clin North Am* 16:711– 747.
- Gracovetsky S. 1985. An hypothesis for the role of the spine in human locomotion: A challenge to current thinking. *J Biomed Eng* 7:205–216.
- Gray AM, Gugala Z, Baillargeon JG. 2016. Effects of oral contraceptive use on anterior cruciate ligament injury epidemiology. *Med Sci Sports Exerc* 48(4):648-654.
- Greenwood D, Muir K, Doherty M, Milner S, Stevens M, Davis TRC. 1997. Conservatively managed tibial shaft fractures in Nottingham, UK: Are pain, osteoarthritis, and disability long-term complications?. *J Epidemiol Community Heal* 51:701–704.
- Gurney B. 2002. Leg length discrepancy. *Gait Posture* 15:195-206.

- Hallgrímsson B, Willmore K, Hall BK. 2002. Canalization, developmental stability, and morphological integration in primate limbs. *Yearb Phys Anthropol* 45:131–158.
- Hanke BA, Motschall E, Turp JC. 2007. Association between orthopedic and dental findings: What level of evidence is available? *J Orofac Orthop* 68:91–107.
- Hariga H, Mousny M, Docquier PL. 2011. Leg length discrepancy following femoral shaft fracture in children: Clinical considerations and recommendations. *Acta Orthop Belg* 77:782–787.
- Hong Y, Bartlett R. 2008. *Routledge handbook of biomechanics and human movement science*. New York: Routledge.
- Kaufman KR, Miller LS, Sutherland DH. 1996. Gait asymmetry in patients with limb-length inequality. *J Pediatr Orthop* 16:144–150.
- Kern KF. 2006. T. Wingate Todd: Pioneer of modern American physical anthropology. *Kirtlandia* 55(1):1-42.
- Kerrigan DC, Todd MK, Della Croce U. 1998. Gender differences in joint biomechanics during walking: Normative study in young adults. *Am J Phys Med Rehabil* 77:2–7.
- Knudson KJ, Stojanowski CM. 2008. New directions in bioarchaeology: Recent contributions to the study of human social identities. *J Archaeol Res* 16:397-432.
- Knutson GA. 2005. Anatomic and functional leg-length inequality: A review and recommendation for clinical decision-making. Part I, anatomic leg-length inequality: Prevalence, magnitude, effects and clinical significance. *Chiropr Osteopat* 13:11.
- Kwon YJ, Song M, Baek IH, Lee T. 2015. The effect of simulating a leg-length discrepancy on pelvic position and spinal posture. *J Phys Ther Sci* 27:689–91.
- Laslett B, Brenner J. 1989. Gender and social reproduction: Historical perspectives. *Annu Rev Sociol* 15:381-404.
- Lewis SJ. 1999. Quantifying measurement error. In: Anderson S, editor. *Current and recent research in osteoarchaeology 2: Proceedings of the 4th, 5th and 6th meetings of the osteoarchaeological research group*. Oxford: Oxbow Books. p 54-55.
- Lomax RG, Hahs-Vaughn DL. 2012. *An introduction to statistical concepts*. New York: Routledge.
- Lovell NC. 1997. Trauma analysis in paleopathology. *Yrbk Phys Anthropol* 40:139-170.
- Lovell NC. 2016. Tiptoeing through the rest of his life: A functional adaptation to a leg shortened by femoral neck fracture. *Int J Paleopathol* 13:91-95.

- MacLennan AG. 1991. The role of the hormone relaxin in human reproduction and pelvic girdle relaxation. *Scand J Rheumatol Scand* 88:7-15.
- Maeda N, Sakaguchi K, Mehta NR, Abdallah EF, Forgione AG, Yokoyama A. 2011. Effects of experimental leg length discrepancies on body posture and dental occlusion. *J Craniomandib Pract* 29:194–203.
- Malik SS, Malik SS. 2015. *Orthopaedic biomechanics made easy*. Cambridge: Cambridge University Press.
- McCaw ST, Bates BT. 1991. Biomechanical implications of mild leg length inequality. *Br J Sp Med* 25(1):10-13.
- McNulty SL. 2009. Pattern and distribution of fractures in the William M. Bass and Hamann-Todd Osteological Collections. Master's Thesis, University of Tennessee.
- Mensforth R, Latimer B. 1989. Hamann-Todd Collection aging studies: Osteoporosis fracture syndrome. *Am J Phys Anthropol* 80:461-479.
- Michelotti A, Manzo P, Farella M, Martina R. 1999. Occlusion and posture: Is there evidence of correlation? *Minerva Stomatol* 48:525-534.
- Milani RS, Deville De Perière D, Lapeyre L, Pourreyron L. 2000. Relationship between dental occlusion and posture. *Cranio* 18:127–133.
- Miller CP, Wheeler RA. 1997. Cleveland: A concise history, 1796-1996. 2nd ed. Bloomington: Indiana University Press.
- Murray KJ, Azari MF. 2015. Leg length discrepancy and osteoarthritis in the knee, hip and lumbar spine. *J Can Chiropr Assoc* 59:226–37.
- Navascues J. 2000. Premature physeal closure after tibial diaphyseal fractures in adolescents. *J Pediatr Orthop* 20:193–196.
- Needham R, Chockalingam N, Dunning D, Healy A, Ahmed E, Ward A. 2012. The effect of leg length discrepancy on pelvis and spine kinematics during gait. *Stud Heal Technol Inf* 176:104-107.
- Ohlendorf D, Himmelreich M, Mickel C, Groneberg DA, Kopp S. 2015. Does a temporary leg length discrepancy have an influence on upper body posture and lower jaw position in competitive athletes? *Sportverletzung Sportschaden* 29(3):157-63.
- Olson CE, Miller RL. 1958. *Morphological integration*. Chicago: University of Chicago Press.

- Park Y, Bae Y. 2014. Change of range of motion of the temporomandibular joint after correction of mild scoliosis. *J Phys Ther Sci* 26:1157–60.
- Perry J. 1992. *Gait analysis: Normal and pathological function*. Thorofare: Slack.
- Perttunen J, Anttila E, Sodergard J, Merikanto J, Komi P. 2004. Gait asymmetry in patients with limb length discrepancy. *Scand J Med Sci Sport* 14:49–56.
- Ponnappula P, Boberg JS. 2010. Lower extremity changes experienced during pregnancy. *J Foot Ankle Surg* 49(5):452-458.
- Rando C, Waldron T. 2012. TMJ osteoarthritis: A new approach to diagnosis. *Am J Phys Anthropol* 148:45-53.
- Reid DC, Smith B. 1984. Leg length inequality: A review of etiology and management. *Physiother Can* 36:177–82.
- Richtsmeier JT, Cheverud JM, Lele S. 1992. Advances in anthropological morphometrics. *Annu Rev Anthropol* 21:283-305.
- Rotundo AE. 1983. Body and soul: Changing ideals of American middle-class manhood 1770-1920. *J Soc Hist* 16(4):23-38.
- Rowlett RM, Schneider MJ. 1974. The material expression of Neanderthal childcare. In: Richardson M, editor. *The human mirror*. Baton Rouge: Louisiana State University Press. p 41–58.
- Rury JL. 1991. *Education and women's work: Female schooling and the division of labor in urban America, 1870-1930*. Albany: State University of New York Press.
- Scheer J, Groce N. 1988. Impairment as a human constant: Cross-cultural and historical perspectives on variation. *J. Social Issues* 44:23-37.
- Schuit D, McPoil TG, Mulesa P. 1989. Incidence of sacroiliac joint malalignment in leg length discrepancies. *J Am Podiatr Med Assoc* 79:380–3.
- Shapiro F. 2001. *Pediatric orthopedic deformities: Basic science, diagnosis, and treatment*. San Diego: Academic Press.
- Shultz SJ, Sander TC, Kirk SE, Perrin DH. 2005. Sex differences in knee joint laxity change across the female menstrual cycle. *J Sports Med Phys Fitness* 45(4):594–603.
- Slauterbeck J, Clevenger C, Lundberg W, Burchfield DM. 1999. Estrogen level alters the failure load of the rabbit anterior cruciate ligament. *J Orthop Res* 17(3):405–8.

- Smith LK, Lelas JL, Kerrigan DC. 2002. Gender differences in pelvic motions and center of mass displacement during walking: Stereotypes quantified. *J Womens Health Gend Based Med* 11:453–458.
- Solecki RS. 1971. *Shanidar: The first flower people*. New York: Alfred A. Knopf.
- Tague RG. 1986. *Obstetric adaptations of the human bony pelvis*. Ph.D. Thesis, Kent State University, Ohio.
- Tappen NC. 1985. The dentition of the “Old Man” of La Chapelle-aux-Saints and inferences concerning Neanderthal behavior. *Am J Phys Anthropol* 67:43-50.
- Tilley LA. 2013. *Towards a bioarchaeology of care: A contextualised approach for identifying and interpreting health-related care provision in prehistory*. PhD Dissertation, Australian National University, Canberra.
- Tilley LA. 2015. *Theory and practice in the bioarchaeology of care*. New York: Springer.
- Tilley LA, Cameron T. 2014. Introducing the Index of Care: A web-based application supporting archaeological research into health-related care. *Int J Paleopathol* 6:5-9.
- Tilley LA, Oxenham MF. 2011. Survival against the odds: Modeling the social implications of care provision to seriously disabled individuals. *Int J Paleopathol* 1:35-42.
- Thompson AR. 2014. Differential diagnosis of limb length discrepancy in a 19th century burial from southwest Mississippi. *Int J Osteoarchaeol* 24:517–530.
- Torjesen PA, Sandnes L. 2004. Serum testosterone in women as measured by an automated immunoassay and a RIA. *Clin Chem* 50(3):678-679.
- Trainor PA, Richtsmeier JT. 2015. Facing up to the challenges of advancing craniofacial research. *Am J Med Genet A* 167(7):1451-1454.
- Trinkaus E. 1983. *The Shanidar Neanderthals*. New York: Academic Press.
- Turner CG, Nichol CR, Scott GR. 1991. Scoring procedures for key morphological traits of the permanent dentition: The Arizona State University dental anthropology system. In: Kelley MA, Larsen CS, editors. *Advances in dental anthropology*. New York: Wiley-Liss. p 13-31.
- Walsh M, Connolly P, Jenkinson A, O’Brien T. 2000. Leg length discrepancy – An experimental study of compensation changes in three dimensions using gait analysis. *Gait Posture* 12:156–161.
- Wedel VL, Galloway A. 2014. *Broken bones: Anthropological analysis of blunt force trauma*. Springfield: Charles C Thomas.

White TD, Folkens PA. 2005. The human bone manual. Burlington: Elsevier.

Winter DA. 1989. Biomechanics of normal and pathological gait: Implications for understanding human locomotor control. *J Mot Behav* 21(4):337-355.

Wünschel M, Wülker N, Müller O. 2013. Gender differences in tibio-femoral kinematics and quadriceps muscle force during weight-bearing knee flexion in vitro. *Knee Surg Sports Traumatol Arthrosc* 21:2557–2563.