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A Comparison of Human Femoral Neck Cortical Bone: Walkers vs. Non-Walkers

Meghan M. Moran
Western Michigan University

A COMPARISON OF HUMAN FEMORAL NECK CORTICAL BONE: WALKERS VS. NON-WALKERS

by

Meghan M. Moran

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A COMPARISON OF HUMAN FEMORAL NECK CORTICAL BONE: WALKERS VS. NON-WALKERS

Meghan M. Moran, M.A.

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This empirical project examines human inferior femoral neck cortical bone and the response in this region to mechanical loading in association with bipedalism. It is suggested that habitual activity induces cortical bone hypertrophy. A radiographic analysis of femoral neck cortical bone was completed using two samples of individuals. One group following a normal developmental trajectory of walking was compared to another who has never walked as a result of cerebral palsy (CP) or spina bifida (SB). Two research questions were addressed: (1) Is the amount of femoral neck inferior cortical bone equal to or different from that seen in the superior femoral neck in individuals who have experienced different histories of biomechanical loading? (2) Is this trait a phenotypically plastic trait?

Measurements were taken of the femoral neck inferior and superior cortical borders and compared across the two samples. Unpaired t-tests and descriptive statistics were conducted to identify significant differences between the two groups. The results demonstrate that non-walkers exhibit more uniform superior and inferior cortex distribution than normal walkers; normal walkers exhibit the expected uneven femoral neck cortical bone distribution. The femoral neck inferior cortical hypertrophy as a phenotypically plastic trait is not supported from the data. Though the difference between the two groups is statistically significant, it is not large enough to apply this trait to the fossil record with accuracy in identifying bipedalism.

TABLE OF CONTENTS

Table of Contents—continued

LIST OF TABLES

LIST OF FIGURES

CHAPTER I

INTRODUCTION

The aim of my thesis research is to examine the shape and distribution of cortical bone at the inferior and superior borders in the human femoral neck and how this region responds to different patterns of mechanical loading. Specifically, it has been noted that the inferior portion of the human femoral neck exhibits more cortical bone relative to the superior border as a response to habitual bipedalism (Currey 2002; Lovejoy 1988; Martin et al. 1998; Ruff n.d.; Stern and Susman 1991). In order to understand the response of bone tissue with respect to the biomechanical loads associated with bipedalism, a radiographic analysis of femoral neck cortical bone has been undertaken. This study samples individuals who have followed a normal developmental trajectory of walking and individuals who have never walked as a result of cerebral palsy (CP) or spina bifida (SB). As strength, rigidity, and robusticity of the femur changes depending on the stress applied, amount of bending in the bone, and the point of loading on the femur, the development and the distribution of cortical bone is affected (Rafferty 1998; Ruff 2003, n.d.).

The goal of this project is to address one research question:

1. *Is the amount of femoral neck inferior cortical bone equal to or different from that seen in the superior femoral neck in individuals who have experienced different histories of biomechanical loading (i.e. individuals with no voluntary*

mobility in their lower limbs versus normal bipedal walkers)? It is expected that individuals who have never walked will not exhibit a well-developed femoral neck inferior cortex relative to the habitually bipedal sample, as the non-ambulatory patients have not experienced the effects of mechanical loading in their lower limbs. Therefore, this trait is a phenotypically plastic trait (i.e. a trait influenced predominantly by environmental conditions rather than a trait under tight genetic control). It is expected that the bone cortex of the inferior femoral neck will be thicker in individuals who have walked normally throughout life relative to those afflicted with CP or SB.

The exploration of this research question aims to further our understanding of factors that influence the production of femoral neck cortical bone and, therefore, has implications for skeletal biology. This project may also aid paleoanthropology by providing another diagnostic feature to bipedal identification (i.e. hypertrophy of inferior femoral neck cortical bone).

To more fully understand the hypertrophy of the femoral neck cortical bone and phenotypically plastic skeletal features in normal walkers and non-ambulators, an in depth literature review was undertaken in Chapter 2. The literature review is a discussion of skeletal biology and biomechanics described in non-primates, non-human primates, fossil hominins, and modern humans in regard to different locomotor patterns. The two disorders (cerebral palsy and spina bifida) that cause permanent immobility in individuals is further addressed; followed by a discussion on how paralysis affects skeletal biology. This background provides a foundation for the

empirical study conducted to examine cortical bone in ambulators and non-ambulators (Chapters 3 and 4). Finally, Chapter 5 discusses the implications of this project within the field of biological anthropology.

CHAPTER II

SKELETAL BIOLOGY AND BIOMECHANICS OF THE HOMINID LOWER LIMB

Biomechanics is the "application of mechanics theory to biological systems" (Ruff and Runestad 1992:407). The blend of biology and mechanics has been positively documented when studying the morphology and function of the lower limbs in hominin movement through skeletal remains (Currey 2002; Lieberman 1997; Ruff and Runestad 1992; Ruff 2000b, n.d.). The addition of biomechanics to biological anthropology furthers our understanding of how the mechanical forces of locomotion interact with bone through formation and resorption (Demes and Gunther 1989).

Aspects of normal cortical bone skeletal biology in non-primates, non-human primates, modern humans, and fossil hominins will be presented here. In addition, a discussion of diseases and abnormal skeletal biology related to cortical bone in non-ambulators will be used to contextualize the hypertrophy of the inferior cortical border of the femoral neck as a phenotypically plastic trait in ambulators and non-ambulators.

Normal Skeletal Biology and Biomechanical Theory

Bone is a dynamic, well-organized tissue composed of fibrous collagen and calcium phosphate crystals that form organic and inorganic components, respectively (Carter and Beaupre 2001; Currey 2002). The organic component determines the structure and biomechanical properties of the tissue while the inorganic phase provides bone with rigid properties (Carter and Beaupre 2001; Currey 2002; Einhorn 1996). By combining the organic and inorganic phases, bone is able to record the effects of various aspects of directional loading. Bone is, therefore, considered an anisotropic and biphasic material (Lieberman 1997; Nordin and Frankel 2001) with elastic and ultimate characteristics in response to applied forces. Elastic behavior is non-permanent change and ultimate characteristics are the permanent responses to external forces (Reilly and Burstein 1975). By understanding how bone reacts to applied forces, the habitual behavior of an individual during life can be assessed from the bone morphology (Currey 2002; Lieberman 1997; Martin and Burr 1989; Martin et al. 1998). In part, these responses comply with Wolff's Law in that bone modifies internally and externally in response to the environment or function (Currey 2002; Lieberman 1997; Martin et al. 1998; Ribble et al. 2001). Specifically, bone hypertrophies where more mechanical support is needed in reaction to these stresses (Einhorn 1996; White and Folkens 2000).

The structural integrity and stiffness of a long bone on a microscopic level is partially determined by the osteons that comprise cortical bone, which can be seen in Figure 1. In long bones, osteons are arranged in cylinders and oriented parallel to the

Figure 1. An SEM Image of Osteons (Bone Cells) (after Einhorn 1996:10)

long axis of the bone (Einhorn 1996). Osteons are arranged in two spiral systems winding from the periosteal to endosteal surfaces. The osteon spirals twist around each other forming a rope-like structure that is highly durable. This arrangement accounts for the biomechanical properties of this solid, dense bone and its ability to resist strain (Currey 2002; Einhorn 1996; Martin and Burr 1989).

Throughout life, bone constantly remodels as a response to the forces generated by habitual locomotion. Remodeling replaces old bone with new bone and through this process bone can change size and shape (Currey 2002; Einhorn 1996; Lanyon and Rubin 1985; Martin and Burr 1989; Raab et al. 1991). The product of

remodeling is lamellar bone, or mature bone, which best resists force applied parallel to the collagen fibers (Currey 2002; Einhorn 1996; Lanyon and Rubin 1985; Raab et al. 1991).

Normal remodeling occurs everywhere in the bone and continuously takes place over an individual's lifetime (Raab et al. 1991; Rafferty 1998). Bone remodeling often reflects the mechanical stress associated with weight bearing physical activities, such as bipedalism. This bony remodeling is in agreement with Wolff's Law, which is argued to be a key diagnostic feature of the femoral neck as the femur undergoes the most biomechanical stress during bipedalism (Currey 2002; Lieberman 1997; Ruff and Runestad 1992; Ruff 200b, n.d.).

The Biomechanics of Bone

There are three types of biomechanical force applied to the skeleton that account for bony response, especially in association with bipedalism (Carter and Beaupre 2001; Currey 2002; Martin et al. 1998; Ruff 2003a). They are: (1) internal forces from tissue growth, (2) external forces from the environment, and (3) joint, tendon, and ligamentous forces due to muscle contractions. The combination of these forces makes it difficult to understand which force is directly responsible for bone hypertrophy.

This study will focus on how external forces affect cortical bone hypertrophy. The relationship between applied force due to locomotion and bone reaction is graphically illustrated in the modulus of elasticity (Figure 2). A linear relationship of

stress and strain measures the rigidity of the bone. This relationship exists between applied stress, resulting strain, and the magnitude of possible deformation (Carter and Beaupre 2001; Einhorn 1996; Martin and Burr 1989). Graphically, the linear

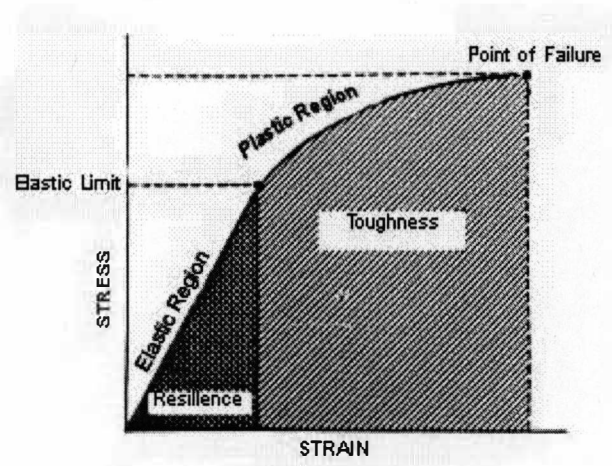

Figure 2. The Modulus of Elasticity Showing the Different Stages Bone Undergoes When Force is Applied

relationship of force and hypertrophy shows applied loads to bone will result in temporary bony deformation. Once the load is removed, the bone will return to the original shape within the elastic region shown on the graph. The steeper the slope in the elastic region, the greater the resistance to stress and deformation. However, there is an elastic limit to bone and beyond this point permanent deformation and damage will occur depending on the plasticity level or toughness of the bone (Einhorn 1996; Martin and Burr 1989). It has been suggested that the modulus of elasticity is proportional to the strain rate of the bone with the strain rate dependent upon the amount and direction of applied stress (Currey 2002; Lanyon and Rubin 1985).

From the modulus of elasticity and Hooke's Law, which states that stress and strain are proportional to each other (Backman 1957; Einhorn 1996; Lieberman 1997; Martin and Burr 1989), it can be deduced that bone is strongest when loaded under compressional forces. Bone will, therefore, hypertrophy with regular application of compressional force.

Biomechanical Beam Model Analysis of Bone as a Structure

When using a biomechanical beam model to study the structure of long bones, assumptions are made regarding the homogeneity of bone composition and static reference planes within the bone element (Carter and Beaupre 2001; Huiskes 1982; Martin and Burr 1989; Ruff 1989, 2000a). In mechanical beam analysis, specific properties, such as the moments of area, are measures of resistance to bending and torsional loading. These properties can be calculated and used to predict the mechanical strength of hollow beams, such as long bones, as well as assess the competence of bone under a variety of stresses (Ruff 2000a; Stock and Pfeiffer 2001). For example, the diaphysis of the femur is comparable to an engineering beam as it is more inflexible due to its shape under bending and twisting stresses during walking. Measurements of the amount of cortical bone in a cross-section can provide information on its resistance to compressive and tensile stresses (Ruff 2000a). The thickness of bony walls determines the amount of stress a long bone is capable of withstanding; thicker walls suggest that greater stress can be withstood (Currey 2002).

Several factors make it problematic to apply beam theory to the femoral neck. The beam model can only be accurately applied to the diaphysis of a long bone and not to the femoral neck or trochanteric area because the moment areas of these regions are dissimilar to those in the diaphysis. This is because the femoral neck and trochanteric area are not composed of the uniform cortical bone as seen in the diaphysis, and the locomotor force distribution is different when moving from the femoral neck down to the femoral diaphysis (Currey 2002; Ruff 1989; Rybicki et al. 1972; Valliappan et al. 1977).

Bone Theory in Regard to Applied Force

Thus far, it has been stated that force applied to bone will initiate cortical bone hypertrophy. Multiple theories exist to explain why and how this occurs. Martin (2000) raises one such theory on the uneven distribution of cortex, which focuses on how bone reacts to use and disuse through a trigger-type reaction within an osteocyte. The osteocyte is thought to signal the periosteum of the bone to begin active remodeling. The remodeling is elevated when the signal is decreased by reduction of the loading stress. As bone contains sensory cells that can monitor the amount of load applied, the osteocytes will produce a signal equivalent to the strength of the loading. The result of this reaction is bone hypertrophy.

Marotti (1996) and Marotti et al. (1992) hold a different point of view on remodeling. These researchers suggest that in order for a buried osteocyte in the bone matrix to stay in contact with the periosteal surface, it sends an inhibitory signal to the

osteoblasts. It is this inhibitory signal that is the same signal other researchers claim to be produced by mechanical loading.

Another explanation for bone hypertrophy is Frost's (1990a, b) mechanostat theory, which states that increased mechanical loading directly increases bone modeling and decreases bone remodeling, thereby increasing the amount of cortical bone at the point of increased loading. This is also the basis for the focus of biomechanics in non-primate and avian experimentation and research (Goodship et al. 1979; Kay and Condon 1987; Rubin and Lanyon 1984; Raab et al. 1991). While it is not yet known what induces bone remodeling, these theories provide the basis for assumption and further research.

Skeletal Biology and Non-Primate Experiments

Cortical bone response to mechanical loading has been studied experimentally in a number of animals (Goodship et al. 1979; Kay and Condon 1987; Rubin and Lanyon 1984 Raab et al. 1991). One experiment focused on the cortical bone response to increased exercise by placing seven sows on treadmills and subjecting them to increased regimes of mechanical loading (Raab et al. 1991). After a 20-week regime, the sows displayed an increase in the femoral cortical bone at the periosteal border relative to the control group that did not engage in an increased exercise regime. In another study, Goodship et al. (1979) focused on cortical bone thickness in the radii of immature pigs, which were surgically isolated by removing a section of the ulna, thereby increasing the mechanical load imposed upon the radius. The

increase in deposition of bone was directly associated with the increased strain; it was suggested that a 50% increase in the robustness of the radius resulted under active stress.

An avian study by Rubin and Lanyon (1984) focused on adult roosters in which the ulna was surgically loaded via pins. After six weeks of exposure to induced stress, the roosters exhibited a significant increase in ulnar periosteal and endosteal cortical bone formation.

Lastly, an experiment conducted on rats studied the effects of bipedalism and associated mechanical forces on bone. The upper limbs of the sixteen male rats were amputated to produce forced bipedalism (Kay and Condon 1987). After 115-123 days, the lower limb bones of the bipedal rats were weighed and measured. It was seen that the proximal femur diameter was 12% larger in the bipedal group than in the control group. This strongly suggests that the bipedal forces caused the lower limb bones of the rats to become more robust due to increased mechanical loading.

Non-primate experiments, such as these presented here, strongly support the idea that bone hypertrophy is a response to the demands of mechanical loading. Each study presents a case for the role of increases in active remodeling as exhibited under increased physical stress; these results form the basis for understanding mechanical loading in humans.

The Effects of Body Mass and Body Size

To fully understand the mechanical loading in humans, body mass must be controlled. Mechanical loading is directly associated with body mass and the amount of applied stress, both of which affect bone density in the femur. The axial loading and bending/torsional loading of the femur are proportional to body weight (Ruff 2000a).

The Biomechanics and Cortical Bone Distribution of Bipedalism

The movement of bipedalism has been said to be "a series of catastrophes narrowly averted" (Adrian and Cooper 1989:279). This potential mishap has been accurately compared to a pendulum as the legs are allowed to swing smoothly at the pelvis with each step (Carrier 1984; Ruff and Runestad 1992). Bipedalism is a fantastic feat for humans considering our anatomical build has a high center of gravity near the sacrum and a narrow support base at the feet (Adrian and Cooper 1989). Through this pendulum analogy, it is thought that cortical bone at the femoral inferior neck hypertrophies to withstand the typical loads associated with bipedalism (Nordin and Frankel 2001).

Stress applied to the femoral neck through locomotion is resisted, and the subsequent strain changes the shape of the bone producing a thicker inferior cortex (Lovejoy 1988; Marcus et al. 1996; Nordin and Frankel 2001; Ohman et al. 1997; Stern and Susman 1991). The amount of cortical bone in a cross-section can be seen

as a measure of resistance to compressive and tensile stresses associated with bipedalism (Ruff 2000a; Stern and Susman 1991) (Figure 3).

Derived skeletal features are physical examples of adapted behavior to the physical terrain in which an individual is in contact (Ruff n.d.; Ward 2002). This is directly visible in the thinner superior border of the femoral neck where minimal net stress is situated, and in the thicker inferior border where an increased amount of stress is withstood (Ohman et al. 1997).

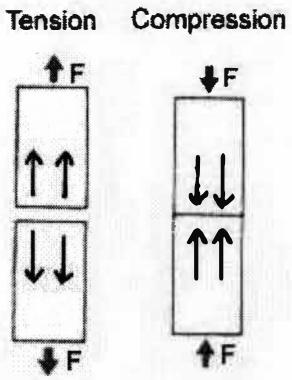

Figure 3. Tension and Compression Forces are Directionally Opposite Forces (F=Applied Force) (after Einhorn 1996:17)

Compression and tensile stresses are also associated with the contraction of the gluteus minimus and medius muscles, which are both abductor muscles and are, therefore, partially responsible for the uneven distribution of cortical bone in modern humans (Aiello and Dean 2002; Lovejoy 1988; Stern and Susman 1991). The gluteus maximus is a hip extensor and rotator muscle (Figure 4), which accounts for the stronger compressional forces on the inferior portion of the femoral neck and smaller tensile forces on the superior portion of the femoral neck (Stern 1972; Stern and

Susman 1981) (Figure 5). It is this relationship between muscular forces and bone that induces cortical bone hypertrophy on the inferior border of the femoral neck (Carter and Beaupre 2001; Lovejoy 1988; Ohman et al. 1997; Stern 1972; Stern and Susman 1991).

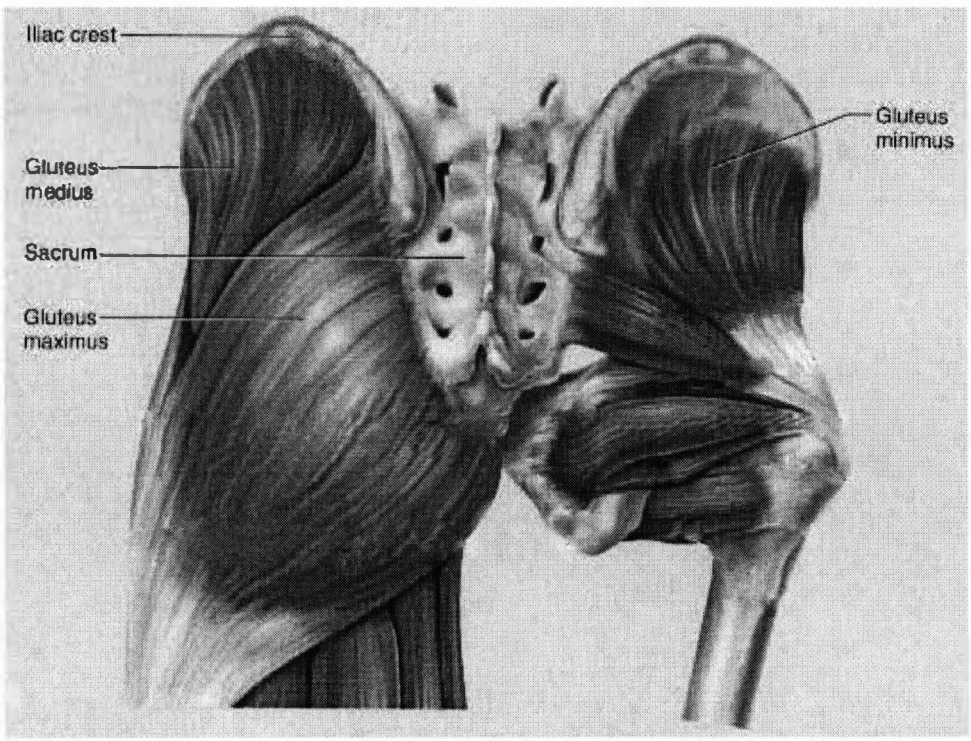

Figure 4. The Gluteal Muscles: Gluteus Maximus, Gluteas Medius, Gluteas Minimus (after Saladin 2005:331).

In human bipedalism, the gluteus medius holds the body erect during the swing phase of walking. This is essential in order to not fall over toward the stance-side hip while taking a step (Stern and Susman 1981). This is an issue as rapidly shifting from a two-legged stance, to swing phase, and to a one-legged stance repeatedly changes the location of the center of mass by altering the inclination of the

pelvis, which must be accounted for with this muscle contraction (Nordin and Frankel 2001).

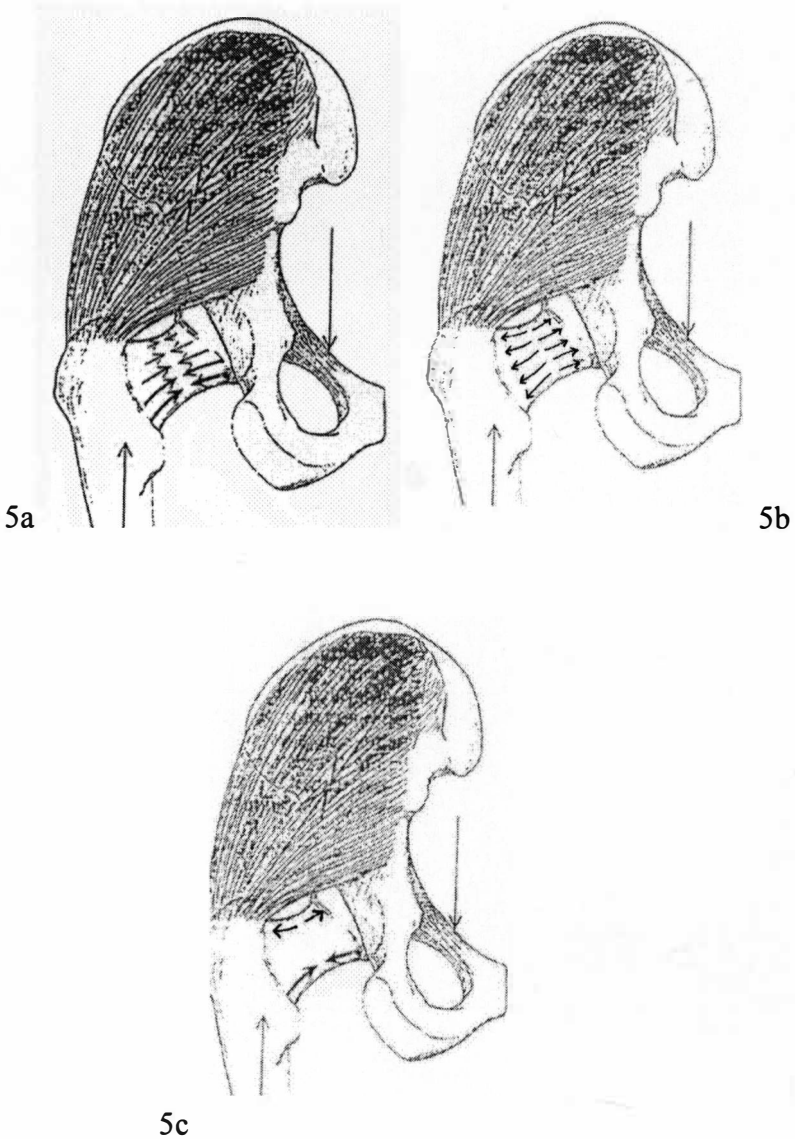

Figure 5. Compression (5a), Tension (5b), and the Combination of Compression and Tension Forces (5c) in the Femoral Neck: Ground Reaction Force Comes Up Through the Femur and Body Weight Bears Down Through the Pelvis (after Lovejoy 1988:124)

Quadrupedalism and Cortical Bone Distribution of Non-Human Primates

Those forces associated with bipedalism are also applied to quadrupedal locomotion. In quadrupedalism, all four limbs are used in locomotion and support of the body (Allen 1973). The magnitude and direction of force associated with patterns of locomotion affect the distribution of cortical bone at the femoral neck and at the midshaft, which vary between species (Demes et al. 1991).

Rafferty (1998) conducted osteological analyses on the femoral necks of twenty-one primate species. Her research focused on strepsirhine primates and the distribution of trabecular and cortical bone in the femoral neck. It was found that strepsirhine primates exhibit thicker inferior cortices when compared to superior cortices, but this has been associated with the bundling of trabeculae in this region. Humans have a unique distribution of bone compared to monkeys. Since monkeys are mostly arboreal, they do not have one stereotypical style to their movements while humans are committed bipeds.

Some apes, such as *Pan troglodytes*, *Pan paniscus*, and *Hylobates*, display a thicker inferior cortex than superior cortex at the femoral neck due to the locomotor stress distribution, but this is variable (Rafferty 1998). This may be due in part to the fact that both species of *Pan* and, to a larger extent, the *Hylobates* walk bipedally when on the ground. However, cortical bone distribution is affected by the placement of the gluteal muscles, which differs in apes relative to humans. The muscles work as rotators and not as abductors in apes and, therefore, may not apply the same amount

or pattern of stress to the femoral neck. The uneven cortical bone distribution is almost non-existent in orangutans, with the difference between femoral cortical borders being only 0.03 mm across individuals. Their unique locomotor pattern allows movement of their upper and lower limbs in multiple directions for an equal buildup of cortex. This supports activity as the primary cause of bone hypertrophy. These primate osteological studies are applied to the fossil record and to modern humans in order to identify habitual locomotor hominin patterns.

Demes et al. (2000) studied cortical bone distribution in strepsirhine primates. This study focused on the superior and inferior cortices in the femoral neck of strepsirhines during frontal bending. The inferior cortex was shown to always be thicker than the superior cortex at the femoral neck due to the loading of the lower limbs during arboreal activity. Strepsirhine primates exhibit a thicker inferior than superior cortical border at the femoral neck, though this is highly variable. Specifically, vertical clingers and leapers display a cortical bone distribution at the femoral neck similar to humans. This is due to the habitual use of the lower limbs in this mode of locomotion, though there are multiple skeletal features, which can differentiate vertical clingers and leapers from habitual bipeds.

Related Bipedal Applications of Skeletal Biology and Biomechanics

Cortical Bone in Modern Humans

In modern humans, long bones are subjected to mechanical loads through specific habitual physical activities. The femoral neck cortical bone distribution is consistent with a thick inferior border and thin superior border (Ohman et al. 1997). This distribution of cortex is visible in the femur as the superior cortex is about one quarter of the inferior border in modern humans. It is also exhibited in the humerus with a thicker medial cortex and a thinner lateral cortex.

An example of modern human cortical bone distribution is seen in two foraging groups, the first from the South African Late Stone Age (LSA). These foragers habitually moved through irregular terrain (Stock and Pfeiffer 2001). The second group is the tribal Andaman Islander (AI) from the 19th century. The AI participate in swimming and paddling canoes as habitual physical activities. The LSA group exhibited more robust femora and the AI presented more robust humeri. These areas of hypertrophy in the femur and humerus are directly correlated to the characteristic activities of each group supporting the hypothesis that behavior can be predicted from the hypertrophy of long bones and that strenuous habitual activity causes bone to hypertrophy. This cortical bone increase is also seen in the cross-section of humeri in ocean-rowing populations, such as the Alaskan Aleuts who apply

a large amount of strain to their upper bodies by habitually paddling in the open ocean (Weiss 2003).

Throughout human evolution, the gracilization trend in cortical bone distribution has been well documented but poorly understood, as there are multiple factors that can contribute to this feature. The environment of the Last Glacial Maximum, around 21 kya, generated a major change in the mobility of humans resulting in a decrease in foraging. This directly correlated with the way humans exploited their surroundings for subsistence (Holt 2003). This environmental shift can be inferred from the diaphyseal robustness of human long bones. The cross-sectional robusticity of the proximal femur and midshaft were found to have decreased during the Upper Paleolithic suggesting a decrease in locomotor activity and, therefore, a decrease in active subsistence mobility.

This trend in cortical bone distribution is further seen in a later group at the border between the Late Woodland and the Mississippian Periods around 1050 AD. The Native American group from the Illinois River Valley, Dickson Mounds, displays an uneven distribution of cortical bone at the femoral neck-shaft junction with thicker inferior and thinner superior cortical borders (Moran 2003). There is evidence that this uneven distribution manifests itself at the age of walking and continues to increase with age; therefore, supporting the claim that habitual bipedalism is a catalyst for inferior cortical bone hypertrophy in the femoral neck.

More evidence for an increase in cortical bone hypertrophy is exhibited in a tennis player's arm where the humerus hypertrophies as a response to the force of

habitually hitting a tennis ball. This is illustrated in a cross-section of the 'hitting' humerus where it is up to 35% thicker in males and 28% thicker in females than their non-playing arm (Lanyon and Rubin 1985; Lieberman 1997; Ruff et al. 1994).

Cortical Bone of Extinct Hominids

The reconstruction of bipedal function from the morphology of the proximal femur has been problematic due to the fragmentary fossil hominin record. Information collected from skeletal and muscular studies of primates and modern humans can help fill in blanks of the fossil record in the study of hominin behavior (Lovejoy 1975).

The morphology of the lower limb has undergone major changes in order to accommodate bipedal walking. Yet, the presence of a specific trait in an individual does not imply the trait has been maintained for the original function. This idea should be applied to all fossil remains and confuses the issue of identifying hypertrophic cause (Ward 2002). The femur has changed with regard to the cortex of the femoral neck and diaphysis and both locations of cross-sections will be discussed in the chronological context of fossil remains (Lovejoy 1975).

Orrorin tugenensis

Difficulty in defining the origin of bipedalism has been highlighted by the disagreement concerning the femoral anatomy of the 6 million year old *Orrorin tugenensis*; specifically, whether it was bipedal (Gee 2001; Senut et al. 2001). If so,

this would help to confirm it's status as a hominin and it may be one of the earliest known bipeds (Gee 2001; Senut et al. 2001; Ward 2002). The more complete femoral fragment (BAR 1002'00) is preserved from the proximal end to the midshaft and exhibits a short femoral neck similar to modern humans (Aiello and Collard 2001; Klein 1999; Senut et al. 2001) (Figure 6).

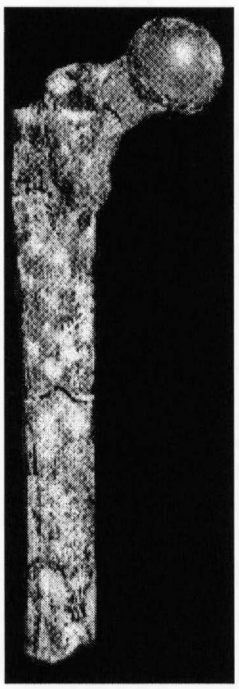

Figure 6. Proximal Femur of *Orrorin tugenensis* (BAR 1002'00) (after Senut et al. 2001:141)

Preliminary research on BAR 1002'00 using computerized tomographic (CT) scans suggest that *O. tugenensis* has a thicker inferior border and thinner superior border at the femoral neck, which would suggest that *O. tugenensis* participated in a bipedal locomotor pattern as this is also seen in modern humans (Pickford et al. 2002).

Australopithecines and Paranthropines

Unlike *O. tugenensis*, australopithecines are considered to have been fully bipedal though they exhibit a combination of arboreal and bipedal traits throughout the skeleton. One *Australopithecus afarensis* specimen in excellent condition is the 3.2 million year old Maka femur (MAK-VP-1/1) (Figure). It is considered to be a habitual biped through analyses of the femoral neck cortex (Lovejoy et al. 2002). The Maka femur displays thicker inferior cortex and thinner superior cortex at the neck-shaft junction.

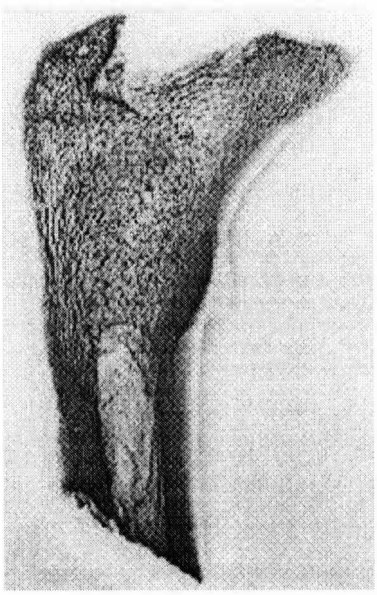

Figure 7. The Maka Femur (after Lovejoy e al. 2002:46)

Other *A. afarensis* specimens, including Lucy (AL 288-1) and AL 211-1, which was naturally broken at the neck-shaft junction, also display thicker inferior and thinner superior cortices at the femoral neck (Lovejoy et al. 2002; Ohman et al. 1997).

Through A-P radiographic analysis, the MLD 46 proximal femur specimen of *A. africanus* from Makapansgat, South Africa exhibits a thicker medial cortex and a thinner lateral cortex at the diaphysis and thicker inferior cortex and thinner superior cortex at the femoral neck (Ohman et al. 1997; Reed et al. 1993).

Paranthropines, such as *Paranthropus boisei* and *P. robustus*, are considered bipeds (Klein 1999; Ruff et al. 1993). A cross-section of the 1.8-1.2 mya SK 82 and the SK 97 proximal femur specimens tentatively attributed to *P. robustus* suggests the inferior border of the femoral neck is thicker than the superior border (Klein 1999; Ruff et al. 1999). This similar bony distribution of australopithecines and paranthropines to modern humans can be attributed to similar stress absorption at the femoral neck and diaphysis through comparable pelvic morphology, gluteal muscle placement, and locomotor pattern (Aiello and Dean 2002).

Pleistocene *Homo*

Most forms of early *Homo* tend to exhibit a thicker cortex at the medial diaphysis (Ruff et al. 1993). This diaphyseal cortex distribution is due to the longer femoral neck displayed in *Homo* and the increased medial-lateral bending of the femoral diaphysis (Ruff 1995). The Middle Pleistocene femoral specimen from Berg

Aukas in Northern Namibia presents a thicker inferior border at the femoral neck, which continues into the subtrochanteric cortex border in cross-section and in anterior-posterior radiographs (Grine et al. 1995). Archaic *Homo* exhibits thicker medial cortex than lateral cortex at the femoral midshaft similar to the distribution of cortex in modern humans (Aiello and Dean 2002). This uneven distribution is exhibited in Neandertals, *Homo erectus*, and other Upper and Lower Pleistocene hominins, such s the KNM-ER 999 specimen (Aiello and Dean 2002; Trinkaus 1993). This distribution of cortex suggests that these early hominins had a locomotor behavior similar to modern humans. This femoral cortical bone hypertrophy has been formed by either a lower level of resorption or by a higher level of deposition as a phenotypically plastic response to the habitual stresses to the lower limb through bipedalism (Abbot et al. 1996; Ruff et al. 1993).

Phenotypically Plastic Bony Features

Phenotypically plastic traits are features affected by the environment (Scheiner 1993). For example, in humans the femoral bicondylar angle, or the valgus knee, and the femoral neck-shaft angle are two skeletal traits that result from habitual bipedalism during development. Due to selection for habitual bipedalism, humans express a number of unique skeletal morphological features, such as: the sinusoidal vertebral curvature (including lumbar lordosis); a short pelvis (wider in breadth than in height); laterally flaring iliac blades; a broad sacrum (wide medial-laterally); a short femoral neck; a large femoral head size; and an adducted hallux (Aiello and

Dean 2002; Demes et al. 2000; Lovejoy et al. 2002; Ohman et al. 1997; Rafferty 1998; Ruff 2003b; Ward 2002). These skeletal features in association with habitual bipedalism will be discussed in relation to ambulators and non-ambulators (Aiello and Dean 2002; Lovejoy et al. 2002; Ward 2002).

Bipedal traits that are phenotypically plastic are revealed through their absences in non-ambulatory individuals. The femoral bicondylar angle (FBA) is measured through the femoral diaphysis to the infracondylar plane, which is located perpendicular to the knee joint (Shefelbine et al. 2002). The FBA begins formation around 1 year of age when most children begin to walk. The medial side of the femur grows faster than the lateral portion, forcing the knee joint inward toward the centerline of the body (Shefelbine et al. 2002; Tardieu 1999). The FBA increases dramatically in the first years of life with the acquisition of walking; by the age of 8, it is between 8 and 10 degrees where it will remain, with regular application of bipedal forces, for life. Humans fall between the FBA ranges of fossil hominins and chimpanzees (Tardieu 1999). Australopithecines and early *Homo* have FBA's of 12 to 15 degrees due to the presence of a longer femoral neck and broader pelvis. Chimpanzees have only about a 1 to 2 degree angle because they are considered quadrupedal (Ruff 1995; Shefelbine et al. 2002; Stern Susman 1981). Paralyzed individuals have an FBA of 0 degrees suggesting that the FBA is a phenotypically plastic trait induced by habitual bipedal locomotor forces (Shefelbine et al. 2002; Tardieu 1999).

The valgus knee is associated with the FBA as it is the 'knock-kneed' appearance of the knee joint that is not exhibited in newborn human babies. In these individuals, the diaphysis is perpendicular to the infracondylar plane because they have not yet learned to walk (Tardieu 1999; Ward 2002). An example presented by Tardieu (1999) is of child who was a non-ambulatory until the age of 7 when he underwent rehabilitation; after which, his FBA increased to 1.5 degrees. By age 10, it increased to about 5 degrees, which supports the contention that the FBA and the valgus knee are phenotypically plastic traits altered by habitual bipedal locomotion.

The femoral neck shaft angle, which is the inclination of the femoral neck to the femoral diaphysis in the frontal plane, is the last phenotypically plastic skeletal feature discussed here (Nordin and Frankel 2000). It is 0 degrees in paralyzed individuals as they have never loaded their hips with mechanical bipedal forces (Duren and Lovejoy 1997). Human adults who follow a normal trajectory of walking have an average femoral neck shaft angle of 121 to 133 degrees and reach this degree by the age of three years (Duren and Lovejoy 1997; Nordin and Frankel 2000; Stern and Susman 1983). These three phenotypically plastic traits can be associated with the inferior thickening of the femoral neck cortex in that all four traits develop during childhood growth and are induced by bipedal locomotion (Shefelbine et al. 2002; Tardieu 1999). It is when a biological problem arises, such as non-ambulation due to a disorder that the normal growth and development of an individual does not continue.

Skeletal Biology of Paralysis

Paralysis due to neurological disorders result in bone atrophy or immobilization osteopenia due to a lack of biomechanical loading (Carter and Beaupre 2001; Frey-Rindova et al. 2000; Kiratli 1996; Mazees and Whedon 1983; Nishiyama et al. 1986; Shaw et al. 1994). Bone atrophy is caused by an increase in bone resorption without concomitant bone deposition, and can range from insignificant to extensive depending on the severity of immobilization (Anselme et al. 2000; Martin et al. 1998). Under normal bone growth, changes in size and shape will occur but paralysis affects the structural dimension of the long bone (Bertram et al. 1997). In a study on chicks, arrested growth was exhibited in paralyzed long bones relative to non-paralyzed long bones. It is likely that muscle or other tissues influence bone growth.

A lack of muscle activity and an inability to bear weight on the limbs below the spinal cord injury are common problems for paralyzed individuals (Kiratli 1996). It has been shown that osteopenia in a paralyzed individual exceeds the level of osteopenia in normal walking individuals (Frey-Rindova 2000; Mazees and Whedon 1983; Tsuzuku et al. 1999). Most of this bone loss takes place in the first year after the initial spinal cord damage (Chantraine et al. 1986; Kiratli 1996). Such extreme osteoporosis has been seen as soon as six weeks after the initial spinal cord trauma (Chantraine et al. 1986). It has been suggested that much of this bone loss can occur at the femoral neck (Tsuzuku et al. 1999), though it is possible to keep some degree of bone mineral density up through continual use of a manual wheelchair. However,

quadriplegics cannot independently operate a manual wheelchair and are, therefore, not able to maintain such levels of bone mineral density.

Disorders and Skeletal Biology of Non-Ambulators

Cerebral palsy (CP) is an acquired, neurological disorder that affects cognitive, sensory and communicative abilities, as well as the posture and movement of an individual by impairing the motor control system (Dabney et al. 1998; Eicher and Batshaw 1993; Lundy et al. 1998; Unnithan et al. 1998). It is caused by an injury at the time of birth, or shortly after birth, to the immature brain, specifically the motor cortex (Dabney et al. 1998; Eicher and Batshaw 1993; Gage 1991). CP usually affects 1.5 out of 1000 live births in this country and is also associated with low infant birth weight (Dabney et al. 1998; Eicher and Batshaw 1993).

Individuals afflicted with CP usually present with osteopenia and multiple cognitive and motor problems, which include imbalanced muscular power, below normal muscular power, and below normal muscle endurance for both the upper and lower limbs (Anselme et al. 2000; Eicher and Batshaw 1993; Harcke et al. 1998; Lundy et al. 1998; Unnithan et al. 1998). It has been suggested that low bone mineral density is common in non-ambulatory individuals with spastic cerebral palsy in which weight bearing of the lower limbs is extremely problematic (Anselme et al. 2000; Henderson et al. 1995; King et al. 2003; Lin and Henderson 1996). Due to the lack of muscle control, dislocated hips are common among individuals with cerebral palsy (Lundy et al. 1998). Hip dislocation can be very painful and cause difficulty sitting.

Femoral osteotomies are one type of hardware and are often surgically implanted to prevent hip dislocations (Bielski 2003, personal communication; Dabney et al. 1998; Selva et al. 1998) (Figure 8).

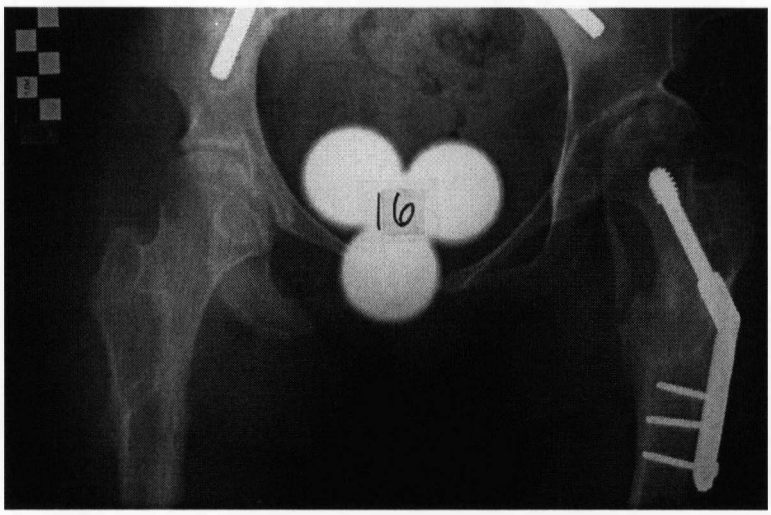

Figure 8. A Femoral Osteotomy on a 17 Year-Old Female Afflicted with Cerebral Palsy (1 cm scale) (photograph taken by author, 2003)

Myelomeningocele (MMC), commonly referred to as spina bifida (SB), is another disorder that causes non-ambulation; it is one type of neural tube defect (NTD), which manifests during the fourth gestational week (Barker et al. 2002; Feeley et al. 2003; Iborra et al. 1999; Walsh et al. 2001). It is a disease that features protruding meninges (the protective coating of the spinal cord) in a sac-like cyst that is filled with cerebral spinal fluid and abnormal neural tissue (Figure 9). This sac distends through the open, defective gap of the vertebral column. One or more

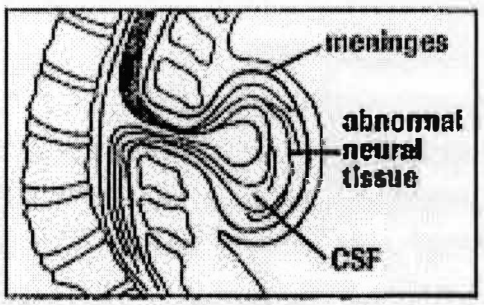

Figure 9. A Cross-Sectional Diagram of a Myelomeningocele (MMC) Spinal Cyst (after Barker et al. 2002:35)

vertebrae are malformed allowing for the exposure of the spinal cord (Aufderheide and Rodriguez-Martin 1998; Barker et al. 2002; Feeley et al. 2003; Iborra et al. 1999; Walsh et al. 2001). There are three types of spina bifida, which are listed from least to most severe and include spina bifida occulta, meningocele, and myelomeningocele (Aufderheide and Rodriguez-Martin 1998; Barker et al. 2002; Iborra et al. 1999; Walsh et al. 2001). It is one of the most common congenital birth defects and results in permanent disability including non-ambulation (Barker et al. 2002; Iborra et al. 1999; Walsh et al. 2001).

Individuals with spina bifida regularly exhibit lower limb sensory and motor deficits and some degree of mental retardation. Nerve damage causes these deficits and accounts for limited mobility and paralysis (Norrlin et al. 2003). The degree of paralysis is determined by the location of the protrusion and amount of associated nerve damage. This condition contributes to non-ambulation resulting in decreased bone density, osteoporosis, and susceptibility to fracture (Barker et al. 2002; Quan et al. 1998) (Figure 10). Orthopedic problems also complicate the lives of individuals

with spina bifida, which include scoliosis and hip dislocation (Barker et al. 2002; Norrlin et al. 2003).

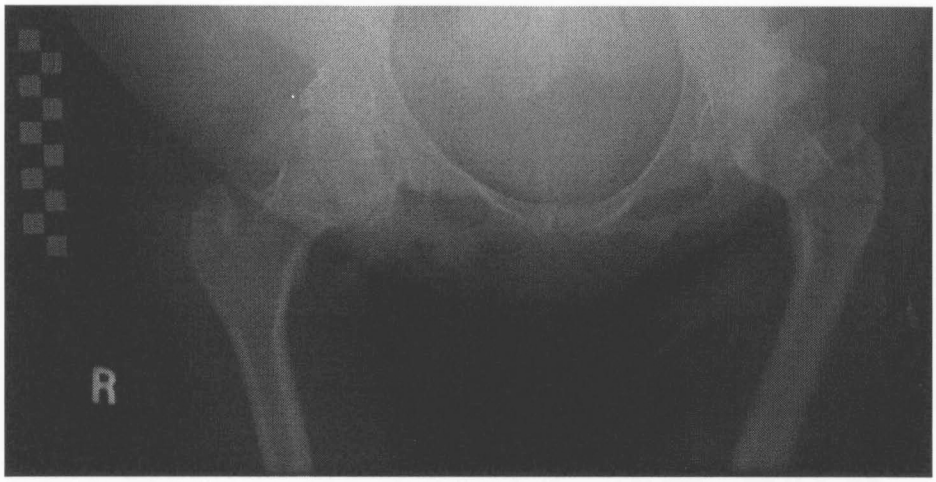

Figure 10. An Example of a 23 Year-Old Female Patient with Spina Bifida (1 cm scale) (photograph taken by author, 2003)

Summary

From the evidence presented in this chapter, it can be deduced that the size and shape of long bones are affected by activity. Skeletal biological research can provide one method for assessing habitual physical activities or lack thereof. Activity appears to be a predominant cause of increases in cortical bone. By understanding the skeletal biology and biomechanics of extant primates and modern humans, the changes in bone under applied force can be understood and classified as phenotypically plastic; this can then be applied to the hominin fossil record in deciphering locomotor patterns.

CHAPTER III

METHODS AND MATERIALS

In order to answer the two main research questions posed in this thesis, a radiographic analysis of cortical bone was completed. The two research questions are:

1. Is the amount of femoral neck inferior cortical bone equal to or different from that seen in the superior femoral neck in individuals who have experienced different histories of biomechanical locomotion?

2. Is the hypertrophy of the inferior femoral neck cortical bone as a trait phenotypically plastic?

For this study, clinical radiographs of 29 modern humans housed at Loyola University Medical Center in Maywood, IL were utilized. The radiographs represent two samples categorized as Non-walkers (n=8) and Normal walkers (n=21). The Non-walkers sample is composed of individuals with cerebral palsy (CP) and spina bifida (SB). In addition, none of the Non-walkers have undergone any surgery on their lower limbs to correct for hip dislocation. In this regard, the CP and SB individuals are similar enough to collapse into one sample.

Cerebral Palsy (CP), Spina Bifida (SB), and Normal Walkers

The CP and SB individuals sampled for this project have never been able to walk and, therefore, have never loaded their lower limbs with any of the mechanical forces characteristic of bipedalism. It is expected that a cross-section of the femoral neck of Non-walkers will display a more equal distribution of cortex.

The individuals with CP and SB are quadriplegic; therefore, both their upper and lower limbs are immobile (Gage 1991; Unnithan et al. 1998). Individuals with hip dislocation problems, common to CP, were not used for this study because many undergo femoral osteotomy surgery (Selva et al. 1998; Silver et al. 1985). Since nerve damage is common in spina bifida, these individuals usually do not undergo surgery to correct their lower limbs, as this is not painful to them (Barker et al. 2002; Bielski 2003, personal communication).

Normal walkers in this study are individuals who presumably began to walk during the average range of 13 and 15 months of age (Eisenberg et al. 1989). From the commencement of walking, it was assumed that these individuals followed a normal trajectory of walking with locomotor skills developing during late infancy and early childhood and continuing until the child was able to walk unassisted with increasing speed (Bogin 1999; Eisenberg et al. 1989). This bipedal pattern continued through the date of their radiograph. These individuals had been radiographed and received medical treatment at Loyola University Medical Center as the result of a traumatic event. All of these individuals are at least 16 years of age and older.

Methodology

The inferior and superior cortical bone at the femoral neck of the Non-walking sample (n=8) is compared to the Normal walking sample (n=210. All individuals included here are 16 years of age and older. Measurements taken for this study include the width of the inferior femoral neck cortical bone and the width of the superior femoral neck cortical bone (Table 1). The location of these measurements is shown in Figure 11. These measurements were recorded from either the left or right sides, or both when available. Measurements of the Non-walkers were taken using digital calipers; measurements for the Normal walkers were made using PACS (Picture Archiving Communication Systems). Additional information recorded for each individual included age and sex, which was clinically noted with each radiograph.

Table 1.

Measurements Collected from Each Individual

Measurements	**Definitions of Measurements**
Width of inferior cortex at the femoral neck	Width of cortex from periosteal to medullary border
Width of the superior cortex at the femoral neck	Width of cortex from periosteal to medullary border

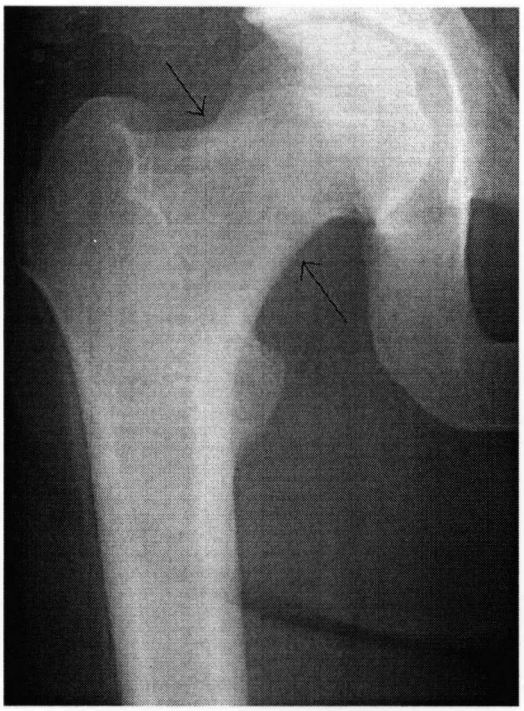

Figure 11. The Location of Inferior and Superior Cortical Bone Measurements Designated by Arrows on a Normal Walking 18 Year-Old Male (photograph taken by author, 2003)

When collecting metric data from humans, body size needs to be controlled. To do this, the superior cortical bone width was divided by the inferior cortical bone width and multiplied by 100 to create a ratio (Equation 1). This provides the percent difference of cortical bone in the femoral neck and inherently controls for body size within each individual.

$$\frac{\text{Superior Cortex} \times 100}{\text{Inferior Cortex}} = \%\ \text{Difference in Cortex Thickness}$$

Equation 1. Formula Used to Figure the Percent Difference in Cortical Bone Thickness

Radiographs

The Non-walking data was taken from film-based analog radiographs. Measurements were taken using digital calipers with a sheet of acetate laid over the radiograph for protection; after which the radiograph was photographed using a digital camera mounted on a tripod. The mounted digital camera was set 53 cm away from the front of the lightbox and stood 155 cm from the floor. The 2.1-mega pixel digital camera was set to a tungsten-white balance setting specific for fluorescent lighting. Two photos of each radiograph were taken to ensure clarity, one at a fine resolution and one at a superfine resolution.

Radiographs are commonly associated with parallax distortion (Ortner and Putschar 1985). To control for parallax distortion, the nametags found on each radiograph of the Non-walking group were measured directly from the radiograph and from the actual radiographic film cassette using digital calipers. A formula was employed to determine the percentage of parallax distortion. This process allows for the percentage of parallax distortion associated with each radiograph to be calculated, and ensures greater accuracy. All the radiographs yielded a percentage of parallax distortion of 1% or less. Therefore, no adjustments of the measurements were necessary.

$$\frac{\text{Measure of actual image}}{\text{Measure of radiographic image}} = \%\text{ of parallax distortion}$$

Equation 2. Formula Used to Figure the Percent of Parallax Distortion in Radiographs

All radiographs of the Normal walkers were digital radiographs. The analog data taken at the time of the x-ray was transferred through a computer process to a digital image on a computer screen. Digital imaging of radiographs provides immediate display of the image, possible image enhancement, and easy storage and retrieval. The Normal walker radiographs were viewed and measured using the computer program PACS. This computer program is capable of calibrating the 5 cm scale present in each radiograph and allowed each radiograph to be accurately measured assuring minimal parallax distortion and a true image.

The PACS measuring method consisted of clicking on the two locations that encompass the distance to be measured. Each measurement was repeated three times and the average was recorded. Copies of the radiographs of the walking sample, without personal information, were printed by the physician and given to this primary researcher as a record of data.

Data Analysis and Statistical Methods

Unpaired t-tests and descriptive statistics for each of the two samples were performed to identify significant differences between the two sample groups. The unpaired to-tests were run on the left, right, and average measurement values that have been controlled for body size and parallax distortion using a two group categorical label of 'NON' and 'WALKERS'.

A critical alpha value of 0.05 serves as an arbitrary value set for significant results. The *p*-value of 0.05 was chosen because this is the amount of type1 error

accepted for this project. Unpaired t-tests were run on the two categorical groupings by combining the two non-walking samples into one group (NON, WALKERS). The results and discussion of the statistical testing are presented in the following chapter.

CHAPTER IV

RESULTS AND DISCUSSION

The results of the statistical testing using unpaired t-tests and descriptive statistics on Normal walkers and Non-walkers are presented here, as well as a discussion of their implications. Table 2 provides the descriptive statistics for the two sample groups (Non-walkers and Walkers), including measurements for left side, right side, and the average of the left and right side. As mentioned in Chapter 3, the superior cortex was divided by the inferior cortex for each individual to produce a percent difference value for cortical bone. The percent difference controls for body size within each individual.

Table 3 provides the actual percentage of cortical bone difference for the Normal walkers and the Non-walkers. As the percentage approaches 1.0, the more similar the superior and inferior cortical borders are in width, meaning the cortex is more uniform and more equal in cross-section.

If the percentage were larger than 1.0, the superior cortex would be thicker than the inferior cortex; and the further the percentage is away from 1.0, the greater the difference between the superior and inferior cortices. Therefore, as the value approaches 0, this indicates the inferior cortex is thicker than the superior cortex.

The Walkers have a 47.2% difference in cortical bone, which is less than the 57.8% displayed in the Non-walkers. This means that the Non-walkers exhibit a

Table 2.

Descriptive Statistics for Absolute Values (mm)

	Left superior/Inferior	Right Superior/Inferior	Average Superior/Inferior
Non-Walkers:			
Mean	0.64	0.516	0.578
Median	0.606	0.429	0.499
S.D.	0.154	0.213	0.16
S.E.	0.054	0.075	0.057
Minimum Value	0.486	0.339	0.412
Maximum Value	0.893	0.926	0.802
Count	8	8	8
Walkers:			
Mean	0.439	0.505	0.472
Median	0.399	0.486	0.484
S.D.	0.122	0.152	0.094
S.E.	0.027	0.033	0.02
Minimum Value	0.274	0.279	0.28
Maximum Value	0.718	0.931	0.63
Count	21	21	21

Table 3.

Values for Percent Difference in Cortical Bone

	Walkers (NORM)	Non-Walkers (NON)
% Difference in Cortical Bone	47.20	57.80

superior cortex and an inferior cortex that are more similar in thickness than the femoral neck cortices of the Normal walkers. The Normal walkers, therefore, exhibit the expected cortical bone distribution at the femoral neck with a thicker inferior border and a thinner superior border. The difference between the Non-walkers and the Normal walkers is significant (p-value = 0.0345) (Table 4).

Table 4.

Statistical Values from the Unpaired t-test

	Mean Diff.	DF	t-value	*p*-value		Count	Mean	Variance	SD	SE
NON, NORM	0.106	27	2.226	0.0345	NON	8	0.578	0.026	0.16	0.057
					NORM	21	0.472	0.009	0.094	0.02

The final comparison is illustrated in the boxplot in Figure 12. From this boxplot, the relationship between the Non-walkers and Normal walkers is shown. The small amount of overlap between the two samples suggests a significant difference. The Non-walkers have slightly higher vales of cortical bone difference supporting the idea that the Non-walkers have more evenly distributed cortical bone at the femoral neck when compared to Normal walkers.

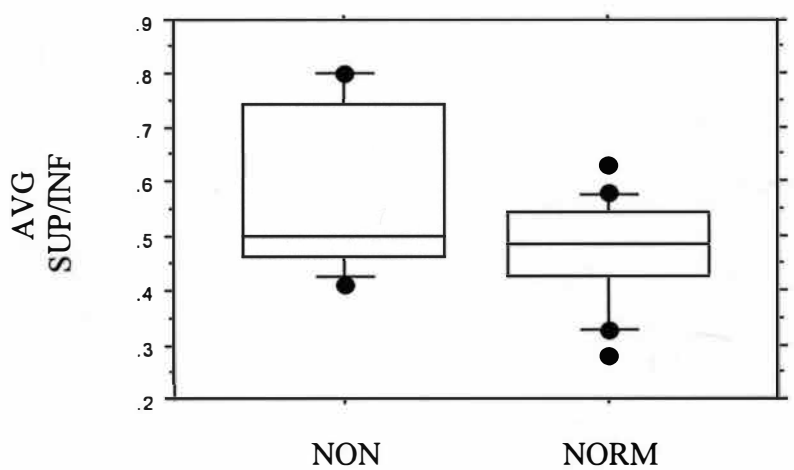

Figure 12. A Boxplot of the Average Superior/Inferior Values for Non-Walkers and Normal Walkers

Based on the hypothesis that stress is associated with locomotor patterns, such as bipedalism, Normal walkers will exhibit a thicker inferior cortical border relative to the superior border (Carter and Beaupre 2001; Currey 2002; Ruff 2003a). Those afflicted with CB or SB (Non-walkers) are not expected to show these distributions but rather to exhibit a more equal width of the femoral cortices. Therefore, the results shown in Table 3 support the idea that Non-walkers show a more equal distribution of the femoral neck cortices than the Normal walkers.

In response to the research question posed for this project: Is the amount of femoral neck inferior cortical bone equal to or different from that exhibited in the superior femoral neck in individuals who have experienced no biomechanical loading relative to walkers? Yes, there is a difference in femoral neck cortical bone displayed in Non-walkers and in Normal walkers. In Non-walkers, the distribution of femoral neck cortex, a thicker inferior cortex and thinner superior cortex, is not exhibited to the same extent as in Normal walkers. This is shown in the percent difference in cortical bone thickness. The Non-walkers displayed 47.2%, which is less than 57.8% exhibited by the Normal walkers. The smaller percentage supports the claim that there is less difference between the inferior and superior cortices at the femoral neck. Therefore, the Normal walkers show a large difference between femoral neck cortical borders; a thicker inferior and thinner superior cortex.

Is the inferior cortical hypertrophy at the femoral neck a phenotypically plastic trait? The data does not support this question thoroughly. From the literature review in Chapter 2, it can be assumed that habitual activity induces cortical bone

hypertrophy. There is a larger difference present between the inferior and superior cortical borders at the femoral neck in Normal walkers than in Non-walkers. However, this is not definitively stating that in cross-section the femoral neck cortical bone is equal. This trait cannot be thought of in terms of black and white; instead it is more complex. Therefore, more research is necessary to state if this is a phenotypically plastic trait.

CHAPTER V

CONCLUSION

The aim of the current research was to examine the distribution of femoral neck cortical bone in humans in order to understand how this area reacts to different patterns of mechanical loading, specifically non-ambulation versus normal bipedal walking. It has been suggested that the inferior border of the human femoral neck has more cortical bone relative to the superior border as a specific response to habitual bipedalism (Currey 2002; Lovejoy 1988; Martin et al. 1998; Ruff, n.d.; Stern and Susman 1991). To explore the question of cortical bone reaction to bipedalism, the femoral neck region of two samples were used for empirical radiographic analysis. One sample is composed of Normal walkers; the second sample is composed of individuals who have never walked as a result of cerebral palsy (CP) or spina bifida (SB). Such a comparison will help us to understand if the hypertrophied inferior femoral neck cortex is a trait exhibited in Non-walkers; therefore, is this trait phenotypically plastic?

The comparative analyses indicate that the Non-walkers displayed a more even cortical bone distribution in comparison to their Normal walking counterparts. The inferior hypertrophy of the femoral neck cortical bone is affected to an extent by activity, specifically bipedalism. This adds further support to the evidence discussed in Chapter 2 in that bone morphology is affected by activity, and the relationship

between activity and skeletal biology does cause hypertrophy of the inferior femoral neck cortical border (Carter and Beaupre 2001; Einhorn 1996; Goodship et al. 1979; Lanyon and Rubin 1985; Martin and Burr 1989; Ruff 1989, 2000a).

The hypertrophy of the inferior femoral neck cortex cannot be defined as a phenotypically plastic skeletal feature at this time. Not surprisingly, defining this trait as a phenotypically plastic trait is more complex than expected. One possible explanation is a combination of the genetic baseline and phenotypic plasticity that produces the thicker inferior cortex and the thinner superior cortex as this distribution is seen in some non-ambulators, but to a much lesser extent when compared to Normal walkers. This is the result of the non-ambulators not exhibiting this trait like the Normal walkers do. They have never loaded their lower limbs with any environmentally induced, locomotor-based mechanical forces, but statistically the differences between the two samples are too similar to definitely state this trait as a phenotypically plastic trait.

This genetic baseline may have more basis today for the femoral neck cortical bone distribution. It is possible that hominins have been bipedal for nearly six million years. Because early hominin bipedalism was a new adaptation, the environment may have played a more extensive role in the functional morphology of the femur, including the distribution of femoral neck cortical bone. With the increasingly sedentary lifestyle of modern humans, extreme activity affects the cortical bone distribution; but how does an individual who is physically lazy compare to an extreme

athlete when studying cortical bone distribution? This leaves room for plenty of future research.

Impacts on Biological Anthropology

This project supports the idea that environmentally-based activity may be one factor in attempting to identify locomotor patterns in the human evolutionary record, but this problem is more complex than once thought. Skeletal biology can draw a great deal of information from this project in understanding bony reaction to applied force.

From the results of this project, it would be more accurate to utilize more than one skeletal trait when attempting to identify the locomotor patterns of fossil hominins, as the inferior femoral neck cortex is not one that is independently trustworthy in paleoanthropology.

Limits of Study

Limits of this study include the finite amount of individuals who have never walked in their lives. Today, if a child is born with a birth defect or congenital disease, it is medically and surgically corrected. Therefore, most individuals have had some type of surgery to correct the medical problem. Also, it is difficult to match the ages of individual between the two samples. The Normal walkers will only be radiographed in the case of an emergency or traumatic event, while the individuals

with cerebral palsy or spina bifida would have radiographs taken at regular intervals to follow growth and development.

Future Research

This study could be expanded by adding another sample, such as a prehistoric group, specifically the Illinois River Valley Native American group from Dickson Mounds. It has been previously shown that this population exhibits the hypertrophic cortical bone distribution beginning at the age of walking (Moran 2003). A comparison could then be made between a prehistoric and a post-industrial sample, which may show differences in cortical bone distribution due to different levels of physical activity.

Furthermore, a comparison of femoral neck cortical bone distributions can be expanded to include a group of Normal walking individuals who have become paralyzed later in life. If possible, comparing the before paralysis and after paralysis radiographs would further our understandings of bone atrophy.

Turning this project into an ontogenetic study to follow individuals from the onset of walking for twenty years at two-year intervals would provide the researcher with a great deal of information to further understand how cortical bone develops or changes with age and bipedalism.

A final possibility for future research would be to compile three samples, one of normally active individuals, a group of sedentary but still movable individuals, and a group of extremely active individuals to compare the width and distribution of

femoral neck cortical bone between these groups. Additionally, the inclusion of fossil hominin remains can be used to compare the different levels of activity in modern humans.

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