THESIS

BONE DENSITY IN COMPETITIVE CYCLISTS: A LONGITUDINAL ASSESSMENT ACROSS THE CYCLING SEASON

Submitted by

Breanne S. Baker

Department of Health and Exercise Science

In partial fulfillment of the requirements

For the Degree of Master of Science

Colorado State University

Fort Collins, Colorado

Spring 2015

Master's Committee:

Advisor: Raoul F. Reiser II

Raymond Browning
Christian Puttlitz

Copyright by Breanne Sloan Baker 2015

All Rights Reserved

ABSTRACT

BONE DENSITY IN COMPETITIVE CYCLISTS: A LONGITUDINAL ASSESSMENT ACROSS THE CYCLING SEASON

The purpose of this study was to investigate in a relatively large group of competitive cyclists how sex, competition level and type of racing influenced bone mineral density (BMD) and bone mineral content (BMC) at the beginning of the season and changes that occurred during the season. In total, 42 participants (22 males and 20 females) completed the study. Subjects were stratified by sex, USA Cycling Category and racing type. At the beginning of the season in February, participants were asked to complete a health history questionnaire, four day dietary log and a DXA scan. After a mean of 180 days participants completed another visit.

At the beginning of the season significant differences were found between the groups. Pre-season sex differences were seen for height, Body Mass, Body Fat %, Lean Mass %, Lower Body (LB) BMCg, Upper Body (UB) BMCg, Shank BMD and estimated number of pre-season training ($p \leq 0.015$). Differences between Cat. 1 and Cat. 4 riders were observed for age and UB BMCg ($p \leq 0.019$). The number of years' experience cycling and racing and the estimated number of races were significant pre-season difference between type of racing ($p=0.019$).

BMD T Score was not significantly different between sexes, Cat. or type of racing and did not significantly increase over the season ($p \geq 0.053$). Further analysis shows a wide variety of positive and negative correlates of skeletal health that deserve further investigation such as age, body composition measures, diet and time spent cycling. This study suggests that cycling is not detrimental to BMD over a competitive season.

ACKNOWLEDGMENTS

Many people made this research possible; however, a few parties are deserving of special recognition. Thank you to my committee members Dr. Reiser, Dr. Browning and Dr. Puttlitz, without your guidance and patience this project would have never become a reality. There were many supporting characters that are also deserving of acknowledgment such as Dr. Braun, Dr. Israel, Dr. Hickey and Dr. Butki and HDPP staff. Last but not least, I am sincerely grateful to my co-workers, friends and family who have supported me along this journey.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER I

INTRODUCTION

Low bone mineral density (BMD) and osteoporosis are serious public health issues in the United States and abroad (Nagle & Brooks, 2011). Globally, osteoporosis causes more than 8.9 million fractures per year, resulting in an osteoporotic fracture every three seconds (Johnell & Kanis, 2006). Fortunately, bone is a living tissue within the body that, like other tissues, has specific roles, needs and dynamic regenerative capabilities. Studies have shown that physical activity (PA) can help to increase bone deposition by increasing osteoblastogenesis through mechanotransduction (Barry & Kohrt, 2008b; Cheung & Giangregorio, 2012; Medelli, Shabani, Lounana, Fardellone, & Campion, 2009). Within reasonable limits, the greater the magnitude and frequency of the mechanical load placed on the bones the more bone formation will occur, resulting in a greater BMD and bone mineral content (BMC) (Frost, 1997). Therefore, PA is often prescribed as a treatment for low BMD and osteoporosis (Rector, Rogers, Ruebel, Widzer, & Hinton, 2009).

While PA in general is good for increasing BMD and BMC, weight bearing PA tends to be superior to non-weight bearing PA. Non-weight bearing PA, such as swimming and cycling, are thought to provide insufficient stimuli to promote osteogenesis even though they incorporate significant muscular loading of the long bones (Warner, Shaw, & Dalsky, 2002). Many studies have shown that highly competitive cyclists have significantly lower BMD than their sedentary matched controls (Barry & Kohrt, 2008a; Medelli, Lounana, Menuet, Shabani, & Cordero-MacIntyre, 2009; Smathers, Bemben, & Bemben, 2009). For example, Tour de France cyclists were shown to have 10% lower lumbar spine BMD as compared to a control group with their

lumbar spine BMD decreasing 25% during the three week race ("Rapid bone loss in high-performance male athletes," 1996; Sabo, Bernd, Pfeil, & Reiter, 1996). Additionally, the femoral neck BMD of healthy, competitive cyclists has been shown to be up to 18% less than matched controls (Campion et al., 2010). However, it is possible that BMD is better preserved at the hip than the lumbar spine in cyclists due to greater hip joint-reaction forces produced by muscle contractions during cycling (Barry & Kohrt, 2007). The available literature largely describes lower levels of total body BMD and BMC in cyclists of all ages and both sexes (Olmedillas, Gonzalez-Aguero, Moreno, Casajus, & Vicente-Rodriguez, 2012). Low BMD might be a contributing factor to shoulder fractures which are the most common traumatic injury for cyclists (Silberman, 2013). These injuries may be attributed to the long periods of time cyclists spend on the bicycle and the limited time spent performing weight-bearing exercises (Stewart & Hannan, 2000). Considering that the bones of competitive cyclists should be exposed to at least the same levels of mechanical loading, if not greater due to muscle forces, than sedentary matched controls, suggests that their PA alters the remodeling process.

Besides insufficient loading, there are other factors associated with competitive cycling that may lead to reduced BMD such as mineral deficiencies and dietary restrictions. Elite level cycling demands a high work load for long periods of time, which is thought to disrupt Calcium homeostasis (Barry & Kohrt, 2007; Silberman, 2013). Dermal Calcium lost in sweat may trigger the resorption of Calcium from bone mass to maintain serum Calcium levels (Guillemant, Accarie, Peres, & Guillemant, 2004). Increased dietary Calcium and Vitamin D have been shown to have mixed results in the attenuation of bone loss (Barry et al., 2011; Gomez-Bruton et al., 2013; Sutton & MacDonald, 2003). Many competitive cyclists also battle with low energy availability during exercise (Ihle & Loucks, 2004). However, it has been shown that if cyclists

can remain in energy balance then skeletal mass is protected (Hinton, Rolleston, Rehrer, Hellemans, & Miller, 2010). This suggests that bone loss in cyclists is a very complex issue.

These factors alone make understanding how to predict and treat low BMD difficult. However, even more confounders exist with sex, age, body mass index (BMI), cycling experience and competitive level potentially playing a role. There is a sex difference as women reach a lower peak BMD and struggle to maintain that mass over the lifespan more than their male counterparts, but how this difference translates to cyclists is not well characterized (Olmedillas et al., 2012). Increasing age is a predictor of low BMD in sedentary and athletic populations (Warming, Hassager, & Christiansen, 2002). However, the relationship between cycling and age is debated. Cyclists often have low BMI's, which can result in lower BMD (Warner et al., 2002). Approximately two-thirds of cyclists with enough experience to be categorized as professional or master road cyclists can be diagnosed with osteopenia (Medelli, Shabani, et al., 2009). Elite cyclists are at a greater risk than recreational cyclists as they may spend up to 40 hours per week training, only exacerbating these risk factors (Hinton et al., 2010). To date, no longitudinal studies have investigated BMD in cyclists of multiple different competition levels. Considering that the previously performed studies have been focused on professional/elite cyclists, their results may not be translated to other populations of cyclists. Many cross-sectional studies have been performed with the aim to assess bone health at one period of time throughout the season; however, BMD is a complicated and intertwined health issue that deserves careful evaluation over time (Hinrichs, 2010). Studies have shown that bone mass may be improved or preserved across a competitive season in other non-weight bearing athletes, specifically collegiate rowers and swimmers (Bemben, Buchanan, Bemben, & Knehans, 2004; Harley, Hind, & O'Hara, 2011; Morgan & Jarrett, 2011; Snow, Williams, LaRiviere,

Fuchs, & Robinson, 2001; Young, 2014). Only a few studies have assessed BMD in professional/elite cyclists over time, most of which concluded that BMD and BMC decrease from the beginning of the season to the end of the season (Barry & Kohrt, 2008a; Nagle & Brooks, 2011; J. F. Nichols & Rauh, 2011; Sherk et al., 2014). However, the homogeneity of participants, both by sex and competition level, selected for these studies are limitations to consider. Furthermore, there currently is great debate on which aspects of the competitive season are so detrimental to cyclist's skeletal health and if these risks are of concern for all riders from professionals to recreational racers.

The purpose of this investigation was to examine how sex, competition level and bicycle type influenced BMD and BMC at the beginning of the season and how these variables affected changes that occurred during the season in a relatively large group of competitive cyclists ranging from entry level (USA Cycling Category 1) to elite (Category 4). Included within these analyses were potential contributing effects from age, body composition, cycling experience, pre-season training mileage and diet. We hypothesized that whole group BMD and BMC would be negatively correlated with increased age, cycling and racing experience and insufficient nutrition while being positively correlated with BMI at the start of the season and across the season. Furthermore, we hypothesized that females would have lower BMD and BMC than their male counterparts, USA Cycling Category 1 racers would have lower BMD and BMC than the less competitive Category 4 racers and lastly, Road racers would have lower BMD and BMC than Multiple Bicycle racers at the start of the season and be more affected across the season. These differences would affect the strength of the relationships with age, cycling experience, body composition and nutrition for all groups.

CHAPTER II

LITERATURE REVIEW

Since the early 1990's bone metabolism research began to investigate the associations between weight bearing and non-weight bearing sports and low BMD. Evidence began mounting that suggested athletes who reported consistently cycling as a primary form of physical activity (PA) had lower BMD. Over time many factors have been identified that may affect the patterns of bone loss and formation such as age, sex, diet and choice of PA. This section will outline the significance and mechanisms of low BMD while also examining many of the factors affecting BMD that have been considered in past research.

Osteoporosis

Our bodies can remodel approximately 10% of the skeletal mass each year (Manolagas, 2000). When this intricate balance of bone resorption and rebuilding is disturbed the most common known side effect is the increased risk of osteoporosis (Sutton & MacDonald, 2003). This disease is characterized by low BMD and deterioration of bone tissue with a consequent increase in bone fragility and susceptibility to fracture (Rizzoli, Bianchi, Garabedian, McKay, & Moreno, 2010). Osteoporosis is defined as men and women with a BMD T-score of the lumbar spine, total hip, or femoral neck of 2.5 standard deviations below their age and sex adjusted BMD means (Beck & Snow, 2003). There are four categories of bone density: normal, osteopenia, osteoporosis and severe osteoporosis as shown below in Table 2.1.

Table 2.1. T-score ranges for BMD categories as measured by DXA.

Category	T-score range	% Rank
Normal	T-score ≥ −1.0	85%
Osteopenia	−2.5 < T-score < −1.0	14%
Osteoporosis	T-score ≤ −2.5	0.60%
Severe osteoporosis	T-score ≤ −2.5 w/ fragility fracture	N/A

Adapted from (Kanis et al., 1994)

Most of the nearly nine million worldwide fractures caused by osteoporosis each year involve the lumbar vertebrae, hip, and wrist (Kanis et al., 1994). Aside from major trauma, the occurrence of many limb fractures in those over age of 50 years is explained by a fall. Those with low BMD are at increased risk of fracture as a result of a fall (Kaptoge et al., 2005). Osteoporosis in America costs approximately $22 billion each year in direct and indirect costs (Navarro et al., 2013). This loss is a significant burden upon the United States health care system and warrants research regarding its mechanisms, outcomes and prevention. A greater understanding of the intricacies behind bone resorption might yield therapeutic modalities to combat low BMD in all populations.

Bone Physiology and Remodeling

Bone is composed of 50 to 70% mineral, 20 to 40% organic matrix, 5 to 10% water, and <3% lipids (Clarke, 2008). There are two types of bone, trabecular and cortical. The average healthy adult human skeleton is composed of 80% cortical bone and 20% trabecular bone (Thomsen, Ebbesen, & Mosekilde, 2002). Cortical bone is dense, solid and provides bone the structural integrity to serve as the body's framework. Whereas trabecular bone is composed of a honeycomb-like network of plates and rods interspersed in the bone marrow and is responsible for many metabolic processes including the formation of red blood cells (Clarke, 2008). The

bone remodeling process is an intricate interaction between osteoblasts, osteoclasts and a wide variety of signaling molecules and stimuli.

Modeling is the process by which bones change their overall shape in response to internal physiologic influences or mechanical forces (Clarke, 2008). Bones may widen or change axis by removal or addition of bone material via osteoblasts and osteoclasts in response to biomechanical forces. Osteoblasts are responsible for bone formation and osteoclast stimulation results in bone resorption. A dynamic tissue, bone adapts to the associated mechanical stress, such as exercise, that are placed on it (Skerry, 2008). Bone formation occurs when Calcium phosphate matrix is deposited faster than it is resorbed. Osteoblasts produce enzymes and osteoid for the Calcium phosphate to bind to, thus creating the matrix (Silverthorn, 2007). Osteoclasts are responsible for mobilizing Calcium so that it can be removed from the matrix and absorbed into the body where it will be used for other metabolic processes. Almost 99% of Calcium in the body is found in the bones (Silverthorn, 2007). This delicate balance of bone formation and resorption is tightly regulated and when disrupted can lead to disease. Mechanotransduction is thought to be a primary stimulus of bone formation.

Mechanotransduction is the process of cells converting mechanical stimuli into chemical action; it is by this process that bones respond to force (Hughes & Petit, 2010; Katsumi, 2003; Tenforde & Fredericson, 2011). Almost twenty years ago Duncan and Turner divided the process of mechanotransduction into four steps: (1) mechanocoupling, (2) biochemical coupling, (3) transmission of signal, and (4) effector cell response; the result can be either bone resorption or deposition (R. L. Duncan & Turner, 1995).

In mechanocoupling, external or internal mechanical loads were thought to cause deformations in bone; including the stretching of bone cells and bone matrix which would result in fluid movement within the canaliculae (R. L. Duncan & Turner, 1995). Bone cells stimulated by this fluid flow or mechanical stretch produced second messengers that were assumed to lead to the production of a signal. Although this was an accurate portrayal of the basic process, the intricacies of how second messengers produced a biochemical signal were only recently discovered. During dynamic loading of bone, fluid is pressed through the osteocyte canaliculi, this fluid shear stress stimulates osteocytes to produce signaling molecules. Although both tissue strain, and fluid shear stress cause cell deformation, these stimuli could excite different signaling pathways that are still under great exploration (Mullender et al., 2004).

In 1995 the intricacies of biochemical coupling and subsequent signal transmission of bone cells had not been well characterized. Known contributors to this process included force transduction through the integrin-cytoskeleton-nuclear matrix structure and stretch-activated cation channels within the cell membrane. G protein-dependent pathways and linkage between the cytoskeleton and the phospholipase C or phospholipase A pathways also play key roles in signal transduction (Lloyd & Donahue, 2010). Over time the interaction between all of these pathways began to point to intercellular communication and mechanosensor capabilities via the osteoblasts and osteocytes. Various physiological mechanisms, including nerve communication, hormones, and cytokines play an important role in this process. Bonewald demonstrated in 2007 that gap junctions between these cells served as the bridge of information making intercellular communication possible. It has been demonstrated that osteocytes, osteoblasts and bone lining directly sense mechanical stimulus, translate it into biochemical signals and direct that signal to other cells; however, this exact mechanism is still being researched (Bonewald, 2007).

What we do know is that within minutes of the osteocytes and osteoblasts sensing particular fluid disruptions, a variety of cytokines and biochemical signals such as nitric oxide (NO), prostaglandins (PGE_2 and PGI_2), insulin-like growth factors (IGF-1) and adenosine triphosphate (ATP) are secreted (Scott, Khan, Duronio, & Hart, 2008; Zeng, Bai, & Han, 2014). The exact influences these particular molecules have on biochemical signaling within bone tissue is still unclear. This year alone, investigation of ATP's role in bone resorption signaling discovered that ATP is released load-dependently from osteocytes from the onset of mechanical stimulation (Kringelbach, Aslan, Novak, Schwarz, & Jorgensen, 2014). However, particular Calcium channel blockers have been shown to prevent the secretion of certain prostaglandins in the presence of mechanical stimulation (Ajubi, Klein-Nulend, Alblas, Burger, & Nijweide, 1999). This intricate balance between osteoblast and osteoclast proliferation verses apoptosis is mediated by a very messy web of the above mentioned biochemical signals, Calcium channel blockers, sclerostin and Wnt/β and Nfκβ signaling. What is known with the utmost certainty is after mechanical stimulation a signal is sent which leads to the cell response of either proliferation or apoptosis.

The cell response is dependent upon the magnitude, duration, and rate of the applied mechanical load. This idea is often associated with Frost's Mechanostat Theory of bone formation which was his mechanism for Wolff's Law. Wolff's law describes the observation that long bones change shape to accommodate to the stresses placed on them; however, Frost provided the first detailed theory of how this adaptation occurs (Clarke, 2008). He theorized that two thresholds existed for bone formation and bone resorption. When mechanical stress was below a certain threshold the body would shed unnecessary bone mass; when that stress was greater than average the bone would add mass in order to adapt to the greater load (Frost, 1987).

Frost also proposed the strain applied to the bone mass via the force magnitude was the key factor; however, it is now known that strain rate, the frequency of loading cycles, the amount of rest between loading cycles, and the distribution of strain within the bone are all important components of the bone's adaptation profile (Skerry, 2006). Different types of physical activity present different strain characteristics and thus could result in either bone formation or resorption.

Exercise and Mechanotransduction

In healthy individuals normal everyday locomotion such as walking is low frequency and results in minimal ground reaction forces, only a few loading directions and small amounts of shear, tension and compression forces applied to the long bones of the leg (VanSwearingen & Studenski, 2014). This minimal mechanical stimuli would only induce very small changes in fluid, thus resulting in little to no change in the bone mass. When a body experiences a force greater than one gravitational unit (+1*g*) the strain is more profound (Popovtzer, 1997). PA and participation in sport promote bone health across all ages among different populations by exposing the participant's skeletal system to a variety of forces (Gomez-Cabello, Ara, Gonzalez-Aguero, Casajus, & Vicente-Rodriguaz, 2012; Goulet et al., 2011). Examples of this positive relationship between sport and bone mass are abundant.

Weight bearing sports, or activities that subject the skeleton to greater than 1*g* normally have an osteogenic effect. Physical contact endured during a football tackle can be over 8.5*g* of force which has been shown to have an osteogenic effect (Vicente-Rodriguez et al., 2003). The use of plyometrics, has demonstrated an ability to increase total body and lower extremity bone mass (Witzke & Snow, 2000). Recreational running can increase BMD; however, there is

evidence that some long distance runners have low BMD (Beck & Snow, 2003; Hetland, Haarbo, & Christiansen, 1993). Ultra runners might suffer from bone loss during races; due to sustained energy deficit, or too much mechanical stimuli without enough rest (Kerschan-Schindl et al., 2009). The magnitude of the strain doesn't only come from gravitational forces but large muscle forces created from quick changes in direction can build BMD (Judex, Gross, & Zernicke, 1997).

Sports often lead to strain from many directions also known as odd-impacts, which have been shown to increase BMD; however, whether gravitational forces or muscle contractile forces are of greater importance is still debated (Judex & Carlson, 2009; Narra, Nikander, Viik, Hyttinen, & Sievanen, 2013). Soccer players are capable of generating three to four times their normal standing GRF values when cutting during match play and are subject to a variety of odd-impacts from locomotion to physical contact (Cowley, Ford, Myer, Kernozek, & Hewett, 2006). A study from England concluded that even a decade after retirement from professional soccer the athlete's BMD was significantly greater than matched controls (Uzunca, Birtane, Durmus-Altun, & Ustun, 2005). Professional mogul and slalom skiers deal with extreme odd-impact forces and substantial muscle contractile forces respectively. The mogul skier is a perfect model to study the influence of high repetition and large magnitude odd-impact forces on bone development. Studies show that professional mogul skiers can have on average 13% greater BMD (Nikander, Sievanen, Heinonen, Karstila, & Kannus, 2008). The slalom skiers endure odd-impacts but not as severe as the mogul skier; however, in combination with large muscle contractile forces BMD was 19% higher and the distal tibia BMD was 60% greater than matched controls (Nikander et al., 2008). The rate and intensity at which a load is applied is also important, but tight physiological limits are not yet established due to the multifaceted nature of the bone formation process.

Many sports provide sufficient mechanical loading to stimulate bone remodeling; however, other factors can influence whether bone is added of resorbed. Research shows that running, swimming and cycling can result in bone formation or resorption depending on the intensity and frequency of activity, energy expenditure during participation and environmental factors (Scofield & Hecht, 2012). It is also possible that simultaneously engaging in these three training regimens can mask distinct advantages or disadvantages of impact verses non weight-bearing regimens on BMD as observed in triathletes (McClanahan et al., 2002). Professional cyclists suffer from bone loss during races; which might be due to sustained energy deficit, or too little mechanical stimuli paired with little rest (Campion et al., 2010; Hinton et al., 2010). The variety of factors affecting this can be demonstrated by evidence of ballet dancers, who often have a reduced BMD because they train for long hours, have very restrictive diets and begin competing before the skeleton can fully mature (Valentino et al., 2001). However, gymnasts, who also have restrictive diets and begin very strenuous training at a young age have high BMD's (D. L. Nichols et al., 1994). Basketball, a contact sport, should increase BMD in athletes; but a study of elite collegiate basketball players observed a 3.3% decrease in BMD over four months (Klesges et al., 1996). Rest is a vital component of microfracutre repair, so when a force is applied with a great magnitude repeatedly, without rest, injury and bone loss is to be expected. Just this year, a study found that high frequency, high force impact which would damage trabecular bone was mediated by Ibuprofen consumption. The anti-inflammatory drug was shown to reduce osteoclasts and bone inflammatory cytokines, while improving muscle contractile forces on bones due to reduced nerve inflammation (Jain et al., 2014). In general it has been thought that high loading activities would produce higher BMD but human and animal studies have demonstrated that load magnitude (Rubin & Lanyon, 1985), frequency, rate

(Mosley & Lanyon, 1998), and gradient (Judex et al., 1997) all affect bone's adaptations to exercise while also being mediated by a host of other environmental factors. Most of the above mentioned sports offer sufficient mechanical stimuli to generate bone formation; however, non-weight bearing exercise is thought to rarely produce health benefits for the skeletal system.

Non-Weight Bearing Exercise

Exercise is generally thought to benefit bone health and is often recommended for both the prevention and treatment of low BMD; however, non-weight bearing exercise may attribute to low BMD (Kohrt et al., 2004). Skeletal unloading has been associated with a conversion of stromal cells to adipocytes rather than osteoblasts, leading to reduced bone formation (Ahdjoudj, Lasmoles, Holy, Zerath, & Marie, 2002). The exact magnitude of loading required to build skeletal mass is still debated. Several cross-sectional studies have concluded that children, adolescents and young adults who are competitive swimmers, a non-weight bearing activity, have lower BMD at several sites as compared to athletes that engaged in impact loading sports such as weight lifting and contact sports (Risser et al., 1990). Swimming has been shown to reduce BMD and negatively impact bone geometry (Ferry et al., 2011). Competitive cycling combines endurance training and non-weight-bearing exercise, two factors that are often associated with low BMD (Campion et al., 2010). Research performed on Tour de France cyclists suggested that two-thirds of the field could be classified as osteopenic (Medelli, Shabani, et al., 2009). Campion and colleagues assessed 30 professional road cyclists and their BMD verses matched controls. Professional cycling appears to negatively affect BMD in young healthy and highly active males, the femoral neck being one of the most affected cites with a 18% difference between experimental subjects and controls (Campion et al., 2010). Some

suggest that the lumbar spine is the most affected area for cyclists BMD as opposed to the femoral neck. It is possible that BMD is better preserved at the hip than the lumbar spine in cyclists due to greater hip joint-reaction forces produced by muscle contractions during cycling (Barry & Kohrt, 2007). Very small forces are transmitted through the bicycle as Farrell and colleague's findings concluded that cyclist's foot-pedal pressure is only 18% of that experienced when running (Farrell, Reisinger, & Tillman, 2003).

Despite a few studies, the available literature largely describes lower levels of total body BMD and BMC in cyclists of all ages, and both sexes (Olmedillas et al., 2012; Scofield & Hecht, 2012). In fact many recent meta-analysis papers have investigated the relationship between competitive cycling and BMD and have concluded that a negative association exists (Kelley, Kelley, & Kohrt, 2013; J. F. Nichols, Palmer, & Levy, 2003; Silberman, 2013). Some literature states that intensity and type of cycling play a role in bone loss (Hinton et al., 2010; Warner et al., 2002). Road endurance cycling at a highly competitive level could be more detrimental to bone mass than performing this activity recreationally, or worse than performing other disciplines such as cross-country cycling or mountain biking (Warner et al., 2002). New studies aim to identify the differences between types of cycling such as road racing or mountain biking racing on BMD in professional riders. It was recently demonstrated that radius geometry and BMD was superior in professional mountain bikers as compared to matched road cyclists (McVeigh, Meiring, Cimato, Micklesfield, & Oosthuyse, 2014). This might be due to the greater impact mountain bikers experience in their upper extremities; however, an interesting follow up study would be to assess the lower extremity BMD in these groups. Most elite mountain bikers use a full suspension bicycle which absorbs the GRF's before they are translated through their skeleton unlike road and cyclocross riders who use a bicycle called a hard tail, which is void of

any suspension system. Another possible contributor to this issue, that is independent of all previously stated confounders, is time.

Does time exacerbate the negative effects cycling might have on BMD? The research does appear to reveal that low BMD is more prominent in elite level cyclists' verses recreational riders; however, very little research has been conducted on less competitive riders (Campion et al., 2010; Warner et al., 2002). Future studies would be wise to include many different levels of cyclists to tease out possible effects of competition level and experience. If cycling is a negative risk factor for low BMD then it would stand to reason that elite cyclists, with the most experience, are at the greatest risk as they undoubtedly spend the most amount of time training; both in terms of hours per day and combined years (Hinton et al., 2010). The vast majority of literature regarding BMD and cycling is cross-sectional in nature; however, as previously illustrated BMD is a complicated issue that would be better suited with a longitudinal assessment. Most of the available longitudinal studies on BMD and cycling portray a clear decrease in BMD; however, all three of the mentioned studies study a different length of time ranging from six months to seven years (Barry & Kohrt, 2008a; Nagle & Brooks, 2011; J. F. Nichols & Rauh, 2011). Just this year a study was published that followed female road cyclists for one full year to assess body composition changes and concluded that while fat mass and lean mass did not significantly change, site specific BMD decreases were observed (Sherk et al., 2014). Depending on when these scans occurred they could tell many different stories. Some studies only follow participants for six months, with no clear indication of why those six months were chosen. The time at which cyclists are studied is important due to off season differences in frequency, type and intensity of training which could increase or decrease BMD independently of cycling. Future studies should try to characterize BMD over each stage of the year, pre-

season, competitive season, post-season and off-season. Some studies show that cycling is not a risk factor for low BMD; however, it is important to note that most of the next cited studies are cross-sectional.

Rico et al., using a cross sectional design, found no difference in the total or regional BMC between male adolescent cyclists and matched controls (Rico, Revilla, Hernandez, Gomezcastresana, & Villa, 1993). Duncan et al. concluded that adolescent female cyclists also had no difference in the total or regional BMD compared to age matched runners, swimmers, traiathletes and controls (C. S. Duncan et al., 2002). One major limitation to Duncan and his colleague's study design is that they did not control for time of season. It would have been important to collect data from each of these groups at the same time in their respective seasons instead of all at once. Beshgetoor et. al measured BMD in middle aged women who either were sedentary, runners or cyclists. Results showed no difference between the groups BMD at the baseline visit (Beshgetoor, Nichols, & Rego, 2000). Wilks and colleagues demonstrated that sprint cyclists and distance cyclists had greater tibia and radius bone strength than the controls, with tibial bone measures being well preserved with age in all groups. And further suggested that competitive cycling is actually beneficial in preserving bone strength into old age in men (Wilks, Gilliver, & Rittweger, 2009). One study with 16 mountain bikers, 14 road cyclists and 15 controls concluded that road cycling is not any more beneficial to skeletal health than recreational activity in healthy men. Higher BMD in the mountain bicycle riders may suggest that this type of racing provides an osteogenic stimulus that is not inherent to road cycling (Warner et al., 2002). Rico et al. (1993) suggested when BMD was adjusted for body weight, such as whole body BMC%, no significant changes were observed in the cycling subjects. They go on to argue that the majority of the early studies that didn't adjust for body weight were not

reliable sources, especially because they were cross-sectional in design. A logical next progression in the research field is to account for past short comings in research methodology and identify what aspects of cycling are most detrimental to skeletal health such as age, sex, diet, and other factors.

Age

The skeleton is constantly growing in size until the epiphyseal plates close, this process adds length and width to the skeleton's long bones and is measured in height. A person's genetics and hormonal profile influence when these plates close. However, most people have a fully mature skeleton by the age of 25 years, including cyclists. This mature skeleton will not add length but it will continue to add mass. Most males and females reach their peak BMD at 30-35 years of age; however, environmental factors can increase this range (Wilmore, Costill, & Kenney, 2008). By about the fifth decade of life, a process of inevitable bone loss begins in both men and women, as bone resorption outpaces formation (S. A. Brown & Rosen, 2003). Maximizing bone mineral mass during adolescents and early adulthood may help to prevent fractures at an older age (Rizzoli et al., 2010).

Sex hormones along with growth hormones are key factors in building and maintaining the skeleton; however, when these hormones are not in balance bone mass can be negatively affected. Testosterone and estrogen play a key role in Calcium homeostasis. Levels of these hormones decrease with age; however exercise can help to negate the loss of bone mass due to declining levels of hormones. Premenopausal women were included in an 18 month training program consisting of endurance and callisthenic exercises. All subjects experienced decreased BMD; however, the group that exercised lost less bone mass than controls (Heinonen, Oja, Sievanen, Pasanen, & Vuori, 1998).

In addition Maimoun et al. (2004) stated that age, among other factors, could influence BMD in people that practice sports including cycling. BMD was shown to be 17% lower at the femoral neck of adult cyclists when compared to matched controls. This same study was conducted in young adults, 18-25 years, where BMD was significantly lower when compared to matched controls. They concluded that cycling during adolescence may compromise the acquisition of bone mass and have negative effects into adulthood (Olmedillas, Gonzalez-Aguero, Moreno, Casajus, & Vicente-Rodriguez, 2011). It appears that it is beneficial to build as much mass before sex hormone levels decrease; however, this process is different between sexes and warrants more research.

Sex

Osteoporosis and low BMD affects more than 14 million men and women in the United States, however, it does not affect sexes equally (Olszynski et al., 2004). It is widely known that women have lower BMD than men throughout all stages of life and are at higher risk of suffering from osteoporosis and low BMD (Olmedillas et al., 2012). As previously stated menopause is a main contributor to the great discrepancy of low BMD incidence in women. Women's estrogen levels drastically decrease at menopause which directly affects the skeleton's ability to maintain mineral homeostasis. Men also have decreasing levels of testosterone as they age, however, the effects are less pronounced. Most women assume that low BMD is not a health concern until after menopause; however, bone loss often begins approximately at the age of 35 years and should be addressed as early as possible. As diet and exercise are often recommended to build or maintain bone mass, it is important to know what impacts cycling may have on skeletal health before this PA is included in the patient's prescription.

Diet

A proper diet provides the recommended daily amounts (RDA's) of nutrients required for the body to function efficiently; when the diet is not providing sufficient quantities of certain macro and trace nutrients health can deteriorate. A study showed that a fat-rich diet was correlated to a higher BMD in cyclists than other carbohydrate-rich diets over a period of 12 weeks (R. C. Brown, Cox, & Goulding, 2000). As important as the macro nutrients, fat, carbohydrate and protein, are to total body health, two primary trace nutrients are of key interest in regards to bone health. Calcium and Vitamin D have been described as being the most important nutritional factors related to peak bone mass acquisition (Gomez-Bruton et al., 2013). Vitamin D stimulates osteoblasts to differentiate and deposit calcified matrix (Owen et al., 1991). Vitamin D's most important derivative is 1,25-dihydroxyvitamin or D_3 which acts in conjunction with Parathyriod hormone (PTH) to tightly regulate plasma Calcium levels, which is pertinent to maintaining skeletal mineralization homeostasis (Sutton & MacDonald, 2003). When Vitamin D is not being adequately ingested or synthesized within the body hypocalcaemia can result and lead to decreased structural integrity of the skeletal system. Vitamin D_3 is also responsible for intestinal absorption of Calcium; however, even with adequate amounts of Vitamin D_3 only about 30% of Calcium ingested is absorbed into the bone (Silverthorn, 2007). Much research as has been performed in the past in regards to supplementation of both of these trace nutrients and performance; however, the results are equivocal.

A study in 2010 showed that professional cyclists ingested adequate Calcium and Vitamin D_3 and still had major decreases in BMD over the season (Campion et al., 2010). Other studies also indicate that there are no observable effects in consuming higher amounts of Calcium to reduce the supposed detrimental effects of cycling on bone mass (Gomez-Bruton et

al., 2013; Olmedillas et al., 2012). Medelli and colleagues demonstrated that there was no difference between medium Calcium and high Calcium for any BMC or BMD parameters in 73 professional and elite level cyclists (Medelli, Shabani, et al., 2009). Some authors have suggested that due to a higher caloric expenditure cyclists have an increased Calcium and Vitamin D requirement as compared to controls (Medelli, Shabani, et al., 2009). However, Werner et al. did not find a difference in Calcium or Vitamin D intake between road and mountain cyclists and controls (Warner et al., 2002). Barry and colleagues revealed that even with adequate Calcium supplementation, BMD and PTH levels did not improve in a group of competitive cyclists over a year (Barry & Kohrt, 2008a). As age is a common confounder for BMD, adolescent cyclists were studied in 2013 by Gomez-Bruton. Dr. Gomez-Bruton and his colleagues stated that nutritional aspects might partially explain differences regarding bone mass in adolescent cyclists and should be taken into account in bone mass analysis as important confounders (Gomez-Bruton et al., 2013). Energy expenditure is often only viewed in terms of kcals; however, with cycling the use of certain minerals might be increased due to profuse sweating.

Sweating is one of the body's natural mechanisms to maintain thermal homeostasis. As our bodies exercise heat is generated, this heat must dissipate in order to reduce the risk of hyperthermia. Barry and Kohrt hypothesize that the decrease in plasma Calcium levels might be due to dermal Calcium loss through sweating. Total plasma Calcium levels will decrease during an intense bout of exercise which can lead to an increase in PTH (Barry & Kohrt, 2007). PTH is a potent stimulator of bone resorption (Bouassida et al., 2003) and within minutes of an increase in PTH secretion, Calcium is mobilized from bone to protect Calcium homeostasis (Rasmusse.H, 1971). Barry et al. (2011) recently conducted a study on pre-exercise Calcium ingestion and its

effects on Calcium homeostasis. They discovered that supplementation attenuated the disruption of PTH; however, this attenuation had minor effects on performance. More research needs to be performed to examine the interaction between bone mass and exercise-nutritional factors (Gomez-Bruton et al., 2013).

Future Endeavors

A thorough review of the literature shows that the relationship between cycling and low BMD is multifaceted and full of contradicting evidence. This review of literature highlighted important factors that may influence BMD in athletes, such as age, sex, diet, type and intensity of exercise and perspiration during those exercises. We know that cycling at a competitive level is less effective at improving bone mass when compared with weight-bearing sports; however, the data is inconsistent (Nagle & Brooks, 2011). According to McClanahan and collaborators future studies specific to cyclists should use more than 20 participants, extend follow up to longer training periods, such as a full competitive season, and include the assessment of bone sites high in trabecular content, such as the hip and spine (McClanahan et al., 2002). The proposed study meets all three of those goals for quality future research.

Conclusion

As previously elucidated changes in skeletal health take time and can be affected by a variety of factors (Hinrichs, 2010). Research has shown that cycling doesn't produce sufficient mechanical stimuli to have an osteogenic effect (Barry & Kohrt, 2008a; Campion et al., 2010; Sherk et al., 2014). Certain aspects of cycling, in addition to being a weight-supported activity, have been identified as possible contributors to low BMD such as diet, cycling experience and

competition level and type of bicycle raced; however, these factors have not been well characterized and need further investigation (Gomez-Bruton et al., 2013; Hinton et al., 2010; McVeigh et al., 2014). The current research is mixed regarding if additional Vitamin D and Calcium supplementation are necessary for cyclists or if it is a function of energy balance (Barry et al., 2011; Gomez-Bruton et al., 2013; Hinton et al., 2010). Elite cyclists are thought to be at an increased risk for low BMD as compared to recreational cyclists because of their relatively large weekly training loads; while highly experienced cyclists who have been cycling for many years would also have been exposed to these risk factors for long periods of time (Barry & Kohrt, 2008a; Hinton et al., 2010). Currently, there is little to no data on differing competition levels of cyclists and BMD. Certain types of bicycles and courses will provide different load profiles and could be more or less detrimental to low BMD and deserve more consideration (McVeigh et al., 2014). Future studies should begin to study these auxiliary factors of low BMD in cyclists and determine their level of contribution to this complicated issue.

The timing of when these measurements are taken are also of great importance, as the year can be divided into four seasons; pre-season, competitive season, post-season and off-season. These times are defined by Barry and Kohrt (2008) as January and February, marking the start of pre-season and September, marking the end of the season. Many cross-sectional studies do not discuss when they took DXA measurements of their cycling participants which is a major limitation. Bone mass as has been characterized in road cyclists over a competitive year and the trends show that BMD in January and February is at its highest and then steadily declines throughout the season (Barry & Kohrt, 2008a; Sherk et al., 2014). What is most unfortunate is that in both of these investigations bone mass was not recovered for most cyclists during the off season which could lead to a continuous decline in bone mass over a cyclist's career (Barry &

Kohrt, 2008a; Sherk et al., 2014). Future studies must be cognizant of the aforementioned auxiliary factors, timing of the study and differences between age, sex, BMI and other factors to yield insightful data.

CHAPTER III

METHODOLOGY

Subjects

A total of 49 subjects were enrolled in the study; however, four were removed for incomplete data and three were removed for conflicting medical issues. Forty one had complete data while a single male, a USAC Category (Cat.) 1, Multiple Bicycle racer, only had DXA results, failing to complete the food logs. In total, 42 subjects completed both data collections and were included in the analysis. Subjects were consented (Appendix I) and asked to complete a four day dietary recall and DXA scan at the beginning and end of the study. The first visit occurred in the February and the second in September, these times defined the pre and post season. All participants were aged between 18-49 years, were healthy and without any history of metabolic bone disorders including amenorrhea or had suffered major bone injuries in the past six months according to their Health and History Questionnaires (Appendix II). Participants also had to be considered a Cat. 4 racer or better as defined by USA Cycling. Riders reported either racing road bicycles only or a variety of bicycles in addition to their road bicycles, such as mountain, track or cyclocross. These groups were identified as Road Only and Multiple Bicycle, respectively. Multiple Bicycle racers who reported racing in multiple USAC Categories were classified by their highest Cat. The exclusion criteria included current pregnancy, irregular menstruation cycles, health history conflicts and commitment issues. Two male participants with a BMD T-score indicative of osteoporosis (-2.5g/cm^2) at the beginning of the study were excluded. Subjects were recruited using word of mouth, social media and fliers (Appendix III). Interested subjects were then contacted and explained the protocol before consenting (Appendix

IV). The project was approved by the Colorado State University Institutional Review Board (Appendix V).

DXA Protocol

A DXA machine (Hologic, Bedford, MA, version 3.4) was used to obtain whole body radiologic measurements. The in vitro precision value for this machine is 0.234% with a SD of 0.002 g/cm^2, this yields a least significant change value of 0.006 g/cm^2 for intra-individual scans. Participants were scanned in the supine position as per manufacturer instructions. Lean Mass (g), Fat Mass (g), and regional BMC (g) were calculated from the whole body scan (Olmedillas et al., 2011). Using whole Body Mass (g), Lean Mass (g), Fat Mass (g) and regional BMG (g) the following variables were calculated: Body Fat %, Lean Mass %, Whole Body (WB) BMC %, Upper Body (UB) BMCg, Lower Body (LB) BMCg, Pelvis BMD, Thigh BMD, Shank BMD and Lumbar BMD. Pelvis BMD, Thigh BMD, and Shank BMD were calculated by averaging the left and right sides. The top of the pelvis served as the dividing line between UB BMCg and LB BMCg. The knee joint served as the dividing line between the thigh and the shank. Lastly, the femoral neck served as the dividing line between the pelvis and the thigh regions. T and Z scores were calculated from whole body BMC g/cm^2. All scans and analyses were performed by a student investigator after completing all mandated training protocols and obtaining all necessary certifications.

Dietary Recall Protocol

Each participant was required to record a four day dietary log at the beginning and end of the study. Participants were given instruction regarding serving sizes, how to record vitamins,

minerals, supplements and beverage consumption. The logs were analyzed by the same student investigator using the Nutritionist Pro software (Axxya Systems, Stafford, TX version 5.4). This program was used to calculate participant's total Kcal, Vitamin D (ug), Calcium (mg), iron (mg), phosphorus (mg), magnesium (mg) and manganese (mg) intake; however, for this study only Kcal, Vitamin D and Calcium were of interest.

Statistical Analysis

Statistical analysis of data was conducted using SPSS version 22 (International Business Machines, Armonk, New York). Prior to any statistical comparisons being performed, each variable was examined for outliers. All extreme outliers using box plot analysis were removed (i.e. those greater than three box lengths from the end of the box). All variables were examined for normality using the Shapiro-Wilks test. Repeated measures T-tests were used to assess whole group pre-to-post season differences. Standard T-tests were used to investigate pre-season differences between males vs. females, Cat.1's vs. Cat.4's and Road Only racers vs. Multiple Bicycle racers. If variables were not normally distributed then the Mann Whitney U test was utilized instead on the nonparametric data. 2x2 repeated measures ANOVA's were ran on each of these group analyses to assess the effects of time, subgroup, and potential interaction between time and subgroup. If the interaction was significant, post-hoc T-tests were ran to assess differences existing between groups pre versus post-season. Pearson correlations were performed on pre-season variables as well as the change in variables across the season (Δ) to establish relationships to BMC and BMD markers. Finally, stepwise linear regressions were performed to determine variables of most importance for BMC and BMD markers. Significance was set at $p \leq 0.05$ for all statistical measures.

CHAPTER IV

RESULTS

Subject Characteristics

A total of 42 subjects completed both data collections and were included in the analysis. Forty one had complete data while a single male, a USAC Cat. 1 Multiple Bicycle racer, only had DXA results, failing to complete the food logs. Statistical outliers were removed from the following pre-season variables (number of subjects in parentheses): Body Mass (1), BMI (1), Pelvis BMD (2), Lumbar BMD (1), Kcal (1), Vitamin D ingestion (4), years racing experience (1), estimated pre-season mileage (1), and estimated number of races (1). Post-season variables that had outliers removed included: UB BMCg (1), Kcal (1), Vitamin D ingestion (3) and the actual number of races participants competed in (1).

A total of 20 participants competed at Cat. 1, three at Cat. 2, three at Cat. 3 and 16 competed at the Cat. 4 level. There were 16 Road Only racers and 26 Multiple Bicycle racers (Table 4.1). The whole group was 31.2±8.5 years of age (mean±standard deviation), 176.1±8.1cm tall and had 180.6±11.9 days between their first DXA scan and their last (Table 4.2). T-tests explored differences between sexes (male versus female), USAC Cat. (1 versus 4), and type of racing (Road Only versus Multiple Bicycle) at the beginning of the season (Table 4.2). The sex comparison revealed eight out of the 22 variables were significantly different. Of those variables that were different males had a greater value for every variable except Body Fat %. Category comparison only showed two variables of significance. Cat. 4's had greater values for age and UB BMCg. When investigating differences between racing type the only significant differences were within cycling variables. Reported years' experience cycling and racing and the estimated number of races to be competed in. It is important to note that Multiple Bicycle

racer's T Score was trending lower than Road Racers and approached statistical significance (p=0.053). Pre-season correlates for all groups can be found in Appendix VI. No consistent variables were correlates for T Score across all groups. Stepwise linear regressions were ran to determine which variables were most influential for important bone variables. Whole group analysis showed that LB BMCg was strongly predicted by the combination of Body Mass and Body Fat % (R=0.811). For Cat. 4 racers the T Score was best predicted by Lean Mass % plus Fat Mass % and BMI (R=0.946).

When analyzing the whole group over the season, Figure 4.1 shows that T Score did not significantly increase (+0.080%Δ). Table 4.3 provides the mean and standard deviations for the changes that occurred across the season for all whole group variables. Correlation tables in Appendix VI show that no whole group seasonal change variables were strongly correlated to the change in T Score over the season. However, Δ WB BMC% presented several negative correlates with the sole positive association coming from Δ Lean Mass. From the stepwise linear regressions, the change in BMI was the strongest predictor of the Δ WB BMC% (R=0.880). When stratified by sex, Figure 4.2 shows that both males and females increase their T Score by 0.60%Δ and 0.02%Δ respectively; however, neither of these values represented a significant change. Two variables significantly changed, Δ Lean Mass % and Δ Lumbar BMD, both of which males experienced the greater increase. Further analysis of T Score changes over time within the two different categories can be observed in Figure 4.3. Cat'1 did start and end the season with a lower T Score than the Cat. 4's. Each group did see an increase in these values but not significant with a 0.06%Δ for Cat. 1's and a +0.6%Δ for Cat. 4's. Only two variables significantly changed for category. Cat. 4's increased their Body Mass and BMI more than Cat. 1's (table 4.3). Lastly, racing type was examined, Figure 4.4 shows the change in T Score for

each group. Road racers represented the only group that had a positive T Score at the end of the season with a 0.57%Δ increase. Multiple Bicycle racers did increase their T Scores from -0.508 to -0.485, a +0.05%Δ; however, this too was not significant. Multiple Bicycle riders experienced a greater increase in their Δ Thigh BMD than Road Racers (Table 4.3). This data suggests that regardless of sex, competition level or racing type BMD is not negatively affected over a six month season and that a wide variety of factors play an important role in this multifaceted issue.

Table 4.1 Participant Subcategories

Sex	Type of Racing	Cat. 1	Cat. 2	Cat. 3	Cat. 4
Males	**Road**	0	2	1	3
N=22	**Multiple**	10	0	2	4
Females	**Road**	4	0	0	6
N=20	**Multiple**	6	1	0	3

Table 4.2 Pre-Season Participant Characteristics					
	Whole Group		Sex	Cat.	Race Type
Variable	Mean	SD	p value	p value	p value
Age (yrs)	31.2	8.5	.176	.019 **C4**	.406
Height (cm)	176.1	8.1	<.001 **M**	.304	.523
Body Mass (kg)	67.7	7.5	<.001 **M**	.065	.553
BMI (kg/m²)	21.8	1.7	.590	.221	.830
Body Fat %	18.3	6.7	<.001 **F**	.857	.436
WB BMC%	3.5	0.4	.055	.751	.358
Lean Mass %	77.0	6.3	<.001 **M**	.911	.465
LB BMCg	1133.4	171.2	<.001 **M**	.670	.490
UB BMCg	1281.7	407.7	.007 **M**	.002 **C4**	.328
T Score	-0.293	0.921	.146	.413	.053
Z Score	-0.319	0.895	.248	.323	.337
Ave. Pelvis BMD (g/cm²)	1.088	0.092	.511	.234	.332
Ave. Thigh BMD (g/cm²)	1.370	0.146	.054	.223	.571
Ave. Shank BMD (g/cm²)	1.010	0.115	.011 **M**	.751	.419
Lumbar BMD (g/cm²)	1.109	0.134	.238	.743	.267
Years Experience	8.8	6.2	.211	.178	<.001 **MB**
Years Racing Experience	5.7	5.1	.245	.478	<.001 **MB**
Estimated Mileage	361.4	307.9	.015 **M**	.956	.626
Estimated # of Races	16.9	11.2	.596	.814	.019 **MB**
Kcal	2730.7	925.4	.114	.514	.628
Vitamin D (ug)	11.6	22.3	.251	.420	.167
Calcium (mg)	1140.0	524.3	.158	.278	.306

SD=Standard Deviation

BMC %= Bone Mineral Content % body mass

LB BMC= Lower Body BMC

UB BMC= Upper Body BMC

M or **F** shows which sex had the greater value.

C1 or **C4** shows which category had the greater value.

MB shows that this group had the greater value.

Table 4.3 Participant Characteristics as they changed over time Δ.					
	Whole Group		Sex	Cat.	Race Type
Variable	Mean	SD	p value	p value	p value
Δ Body Mass (kg)	0.19	2.05	.885	.006 **C4**	.278
Δ BMI (kg/m²)	0.03	0.61	.885	.010 **C4**	.656
Δ Body Fat %	3.63	2.15	.757	.481	.234
Δ WB BMC%	0.03	0.11	.354	.325	.165
Δ Lean Mass %	-3.60	1.82	.0413 **M**	.259	.376
Δ LB BMCg	4.44	37.67	.710	.735	.422
Δ UB BMCg	18.40	31.01	.142	.061	.721
Δ T Score	0.03	0.26	.612	.926	.911
Δ Z Score	0.02	0.26	.439	.742	.843
Δ Ave. Pelvis BMD (g/cm²)	0.00	0.06	.992	.853	.872
Δ Ave. Thigh BMD (g/cm²)	0.00	0.02	.218	.167	.016 **MB**
Δ Ave. Shank BMD (g/cm²)	0.00	0.04	.993	.370	.555
Δ Lumbar BMD (g/cm²)	0.00	0.07	.031 **M**	.424	.750
Δ Number of Races	-4.00	7.79	.113	.487	.721
Δ Kcal	-395.98	685.21	.186	.346	.340
Δ Vitamin D (ug)	-5.45	14.79	.420	.167	.511
Δ Calcium (mg)	-134.87	545.95	.061	.069	.065

SD=Standard Deviation

LB BMC= Lower Body BMC

BMC %= Bone Mineral Content % body mass

UB BMC= Upper Body BMC

M indicates that males had the greater increase in the variable.

C4 indicates that the Cat. 4 racers had a greater increase in the variable.

MB indicates that the Multiple Bicycle racers had a greater increase in the variable.

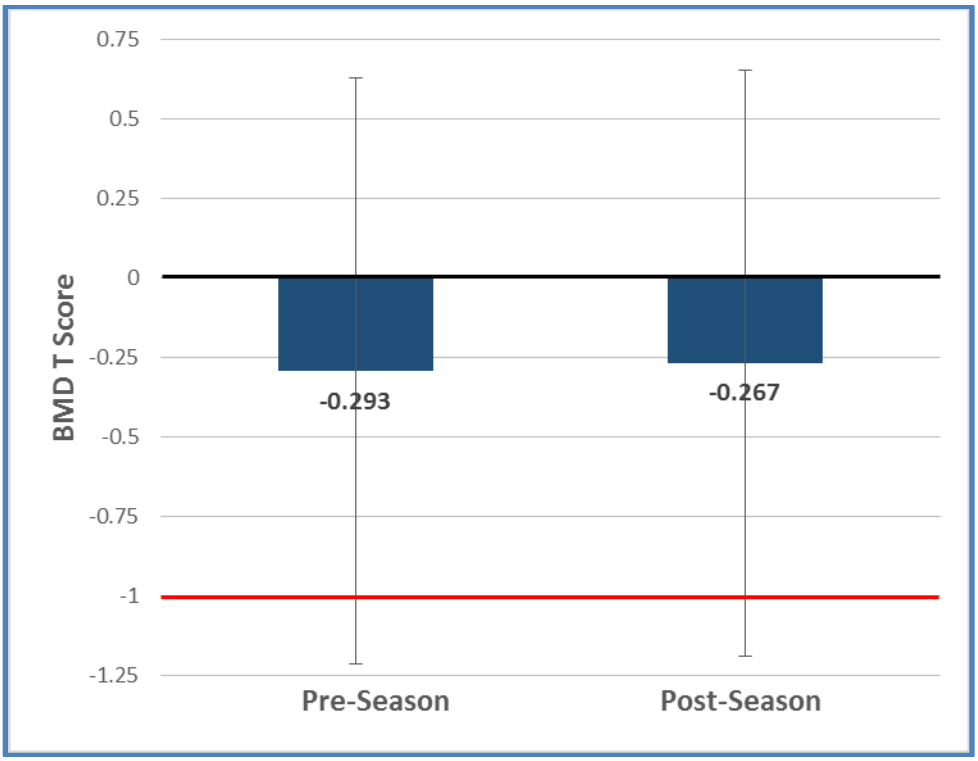

Figure 4.1 Whole Group T Score as it changed +0.08% over time. Error bars represent 1 SD.

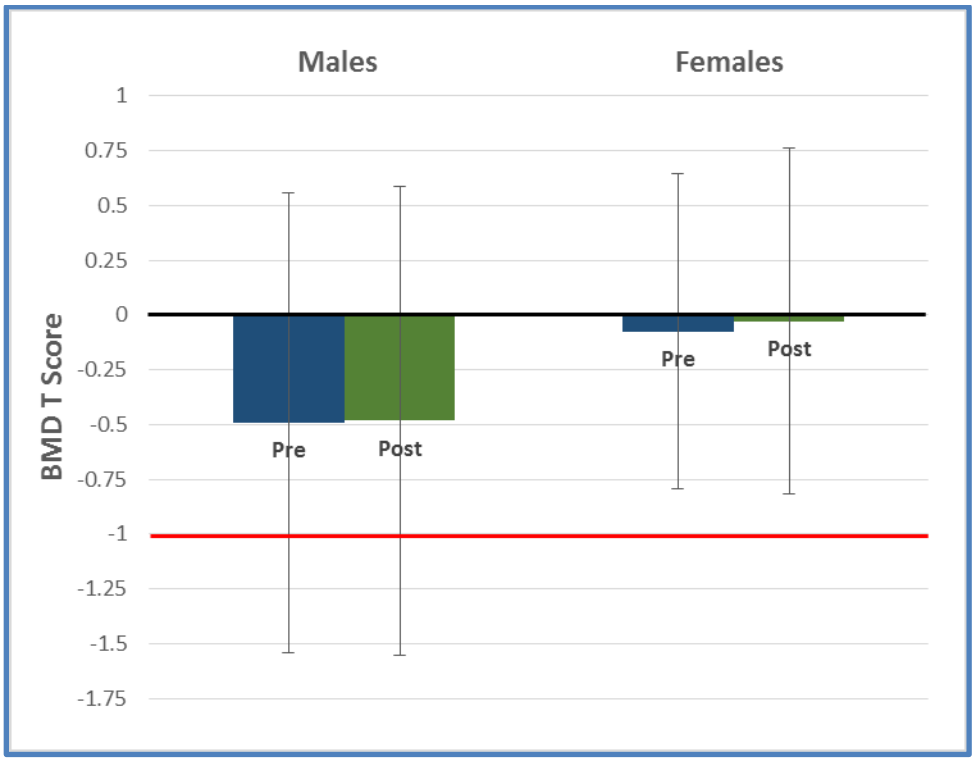

Figure 4.2 Sex comparison of T Score as it changed +0.02% for males and +0.6% for females over time. Error bars represent 1 SD.

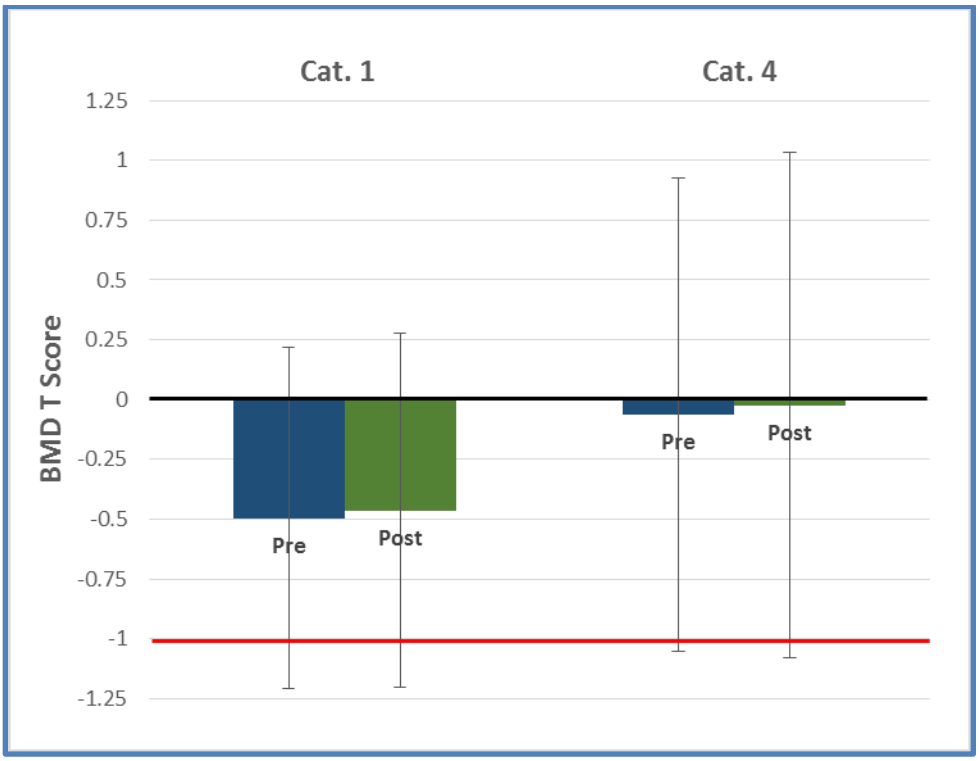

Figure 4.3 USAC Category comparison of T Score as it changed +0.06% for Cat. 1's and +0.6% for Cat. 4's over time. Error bars represent 1 SD.

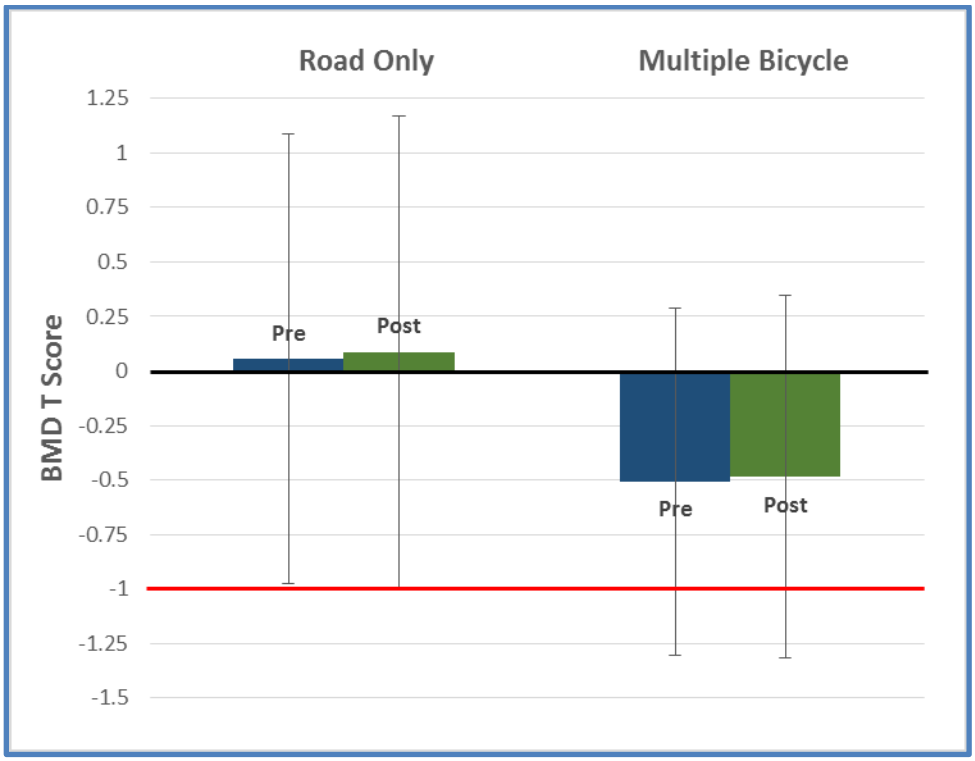

Figure 4.4 Type of racing comparison of T Score as it changed +0.57% for Road racers and +0.05% for Multiple Bicycle racers over time. Error bars represent 1 SD.

CHAPTER V

DISCUSSION

The purpose of this investigation was to examine how sex, competition level and type of racing influenced BMD and BMC at the beginning of the season and how these variables affected the changes that occurred during the season in a large group of competitive cyclists. We hypothesized that whole group BMD and BMC would be negatively correlated with increased age, cycling and racing experience and insufficient nutrition while being positively correlated with BMI at the start of the season and across the season; however, our data do not support this hypothesis. Next, we hypothesized that females would have lower BMD and BMC than their male counterparts at the beginning of the season and that they would be more affected across the season, which was also not supported. We correctly hypothesized that USA Cycling Cat. 1 racers would have lower BMD and BMC than the less competitive Cat. 4 racers at the beginning of the season. Lastly, our results did not support the hypothesis that Road Only racers would have lower BMD and BMC as compared to the Multiple Bicycle racers. Overall, while BMD and BMC were not compromised at the beginning of the season or across the season, factors within/between sexes, Categories, and racing type affect their BMD and BMC such that they should be considered when assessing an individual.

Whole Group

As a whole, the cyclists of this study had normal BMI's and BMD T Scores at the beginning and end of the study. Kcals ingested were above average and Calcium was sufficient according to RDA's. However, average Vitamin D intake was only around 40% of RDA's. It is important to note that we did not control for environmental Vitamin D. Zero correlates of T

Score and Z Score specifically emerged from the whole group analysis. Also, the lack of pre-season mileage and years' experience of cycling and racing correlates were counterintuitive. These results might be difficult to observe with such a diverse group of participants that were included in the whole group analysis.

Whole Group analysis revealed no change in the mean T Score and all regional BMD's throughout the season. Age was positively correlated with BMD measures at the beginning of the season just not over time. This relationship may have still been present at the end of the season but age effects might have been masked by the gain in Fat Mass %. BMI and Body Mass remained unchanged over the season but the body composition of these racers did not; Lean Mass % decreased and Fat Mass % increased for most individuals. This change in body composition may be due to tapering, as many cyclists in this study reported the greatest number of training miles were logged in the weeks prior to pre-season. Certain tapering protocols have been shown to increase Fat Mass and decrease Lean Mass while not affecting performance significantly (Garcia-Pallares, Sanchez-Medina, Perez, Izquierdo-Gabarren, & Izquierdo, 2010; Neary, Martin, & Quinney, 2003). Many studies have found that fat mass can increase axial BMD via secretion of hormones such as estrogen and leptin which may explain the results observed here (Karsenty, 2006; Nouvenne et al., 2013; Reid et al., 1992; Syed & Khosla, 2005).

Vitamin D and Calcium have not been consistently associated with increased or decreased BMD and BMC in cyclists; however, total caloric intake has been. Hinton and colleagues suggest that maintaining energy balance is one of the most important factors for protecting skeletal mass over a competitive season. Unfortunately, without resting metabolic rate measurements and more accurate training logs it is impossible to estimate caloric demands of each participant and thus conclude if they were in either energy surplus or deficit. It could be

assumed that participants were in energy surplus because of the increase in Body Fat %, which may partially explain why bone mass increased over the season.

Sex

The relationship between sex and BMD is not clear cut. Physical differences that may attribute to the findings that females actually had higher T Scores than males include female's significantly greater Fat Mass % to start and end the season and a lesser change in body composition by retaining more Lean Mass and gaining less Fat Mass than males. The change in BMI was a very strong predictor of the change in WB BMC% for both males and females with R=0.899 and R=0.912. Despite the greater Kcal intake males were also deficient in Vitamin D, receiving only about 42% of their RDA's from dietary sources, females received about 40%. A noteworthy finding was that females reported more supplementation of Vitamin D and Calcium than males. Males also reported cycling more miles regardless of Cat. as compared to females at the beginning of the season. These data suggest that maybe differences in T Score and regional BMD's are more associated with time spent cycling at a high level than sex or diet, and body composition's relationship to bone mass deserves more attention.

Category

When stratifying the whole group by category we see the younger, more experienced Cat. 1's also have significantly lower UB BMCg compared to Cat. 4's; however, no statistical difference was observed in the change of T Score over the season. An important consideration is that although Cat. 1's and Cat. 4's increased T Score by similar amounts, 0.030 and 0.038 respectively, these are drastically different percentages of pre-season values. The Cat. 1's mean

T Score increased by only 6%; while the Cat. 4's T Score increased by over 60% of the pre-season values. The greater increase in T Score observed in Cat. 4 racers coincides with 25% greater gain in Fat Mass %. Lean Mass %, Fat Mass % and BMI best predicted T Score and Z Score (R=0.946 and 0.934 respectively) in Cat. 4 racers. Cat.1 reported almost double the years' experience riding and nearly four times as much racing experience as Cat. 4 riders. Years' experience riding was a negative correlate of both T Score and Z Score for Cat. 1 riders. While it goes against the common perspectives relative to attempting to maintain BMD and BMC, more competitive riders might actually benefit from low BMD as maintaining a low body mass is important for peak power in cycling. In summary category data suggests that total time cycling at high intensities may play a larger role than changes in body composition in competitive cyclists.

Racing Type

Evidence has suggested that there are differences between the types of bicycle racing and bone health (McVeigh et al., 2014; Silberman, 2013; Wilks et al., 2009). At the pre-season Multiple Bicycle racers reported more years' experience cycling and racing and more estimated races. They also appear to have trending lower T Scores and Z Scores ($p \geq 0.053$). Road Only racers increased their T Score by 57% from pre-season values; while Multiple Bicycle racers only increased their T scores by just over 4%. Our data suggests that as male cyclists continue to move up categories, they also begin to race an assortment of bicycles which may be predictive of lower BMD. Body composition changes provided many strong correlations and prediction equations for bone variables; however, few were consistent for both groups. Kcal was not a significant predictor of any variables but Road racers ingested almost 20% more Vitamin D than

Multiple Bicycle racers, which may be linked to the amount of bone mass gained over the season. Multiple Bicycle racers reported 61% more years' experience riding and 68% more years' experience racing. This suggests that the difference between BMD's might not be due to the type of bicycle raced but more because of the total time spent cycling at a competitive level and the associated changes in body composition.

Limitations/Delimitations

The results of this study are not consistent with those of previous studies showing a decreased BMD in cyclists (Barry & Kohrt, 2008a; Campion et al., 2010; J. F. Nichols et al., 2003; Sherk et al., 2014). This study is among the first to investigate such a wide range of age, fitness, racing volume and caliber, anthropometric characteristics and dietary intake which limits the ability of direct comparisons between studies that assess more homogenous populations.

There are several possible reasons why no changes in whole body BMD were found in this study. Before the season even began, as part of our exclusion criteria, two Cat. 1, Multiple Bicycle males were excluded for T Scores indicative of osteoporosis. It is possible that these racer's exclusion could have created a bias towards a healthier data set. Secondly, skeletal acclimatization has been observed in athletes who have been engaging in similar training patterns for many years. This suggests that highly competitive racers with lower BMD, like Cat. 1, Multiple Bicycle male participants might only experience negative BMD changes if significant alterations to their training patterns are imposed (McClanahan et al., 2002). Since a control group was not used, we cannot dismiss the possibility that factors unrelated to the participant's cycling season might have contributed to changes. We also relied on self-reported measures of training logs and dietary intake instead of more sensitive measures. We did not

control for the frequency and type of off-season training which may have confounded this data set and should also be considered in future endeavors. The number of participants was not evenly split between the Road Only and the Multiple Bicycle groups, which may have skewed results. Lastly, because we did not track training mileage or have resting metabolic rate data, true caloric demands and expenditure is impossible to accurately quantify. It is also important to look at a full year of cycling because different types of racing start and end at different times throughout the year. Despite these limitations, this study assessed a very assorted group of cyclists and suggested that there are no negative effects on BMD or BMC during a competitive cycling season.

Conclusions

The results of this study suggest that participation in competitive cycling might not have deleterious effects on BMD and BMC as previously demonstrated. T Score, Z Score and regional BMD measurements did not significantly decrease across the season for any group; however, certain variables such age, body composition measures, cycling and racing experience and insufficient nutrition are deserving of further investigation. BMD and BMC in cyclists is a very multifaceted issue, this can be demonstrated by the fact that in each subgroup men and women did not share the same strength of relationships with the same variables. Differences were not consistent for highly competitive rider's verses less experiences racers. And, body composition and time spent cycling seem to be very important regardless of what type of racing one chooses. More importantly, we must work to understand when, where and why does cycling shift from a safe osteogenic activity, as demonstrated by this study, to a possibly dangerous osteoporotic sport, as demonstrated by many others.

REFERENCES

Ahdjoudj, S., Lasmoles, F., Holy, X., Zerath, E., & Marie, P. J. (2002). Transforming growth factor beta 2 inhibits adipocyte differentiation induced by skeletal unloading in rat bone marrow stroma. *Journal of Bone and Mineral Research, 17*(4), 668-677. doi: DOI 10.1359/jbmr.2002.17.4.668

Ajubi, N. E., Klein-Nulend, J., Alblas, M. J., Burger, E. H., & Nijweide, P. J. (1999). Signal transduction pathways involved in fluid flow-induced PGE2 production by cultured osteocytes. *Am J Physiol, 276*(1 Pt 1), E171-178.

Barry, D. W., Hansen, K. C., Van Pelt, R. E., Witten, M., Wolfe, P., & Kohrt, W. M. (2011). Acute Calcium Ingestion Attenuates Exercise-Induced Disruption of Calcium Homeostasis. *Medicine and Science in Sports and Exercise, 43*(4), 617-623. doi: Doi 10.1249/Mss.0b013e3181f79fa8

Barry, D. W., & Kohrt, W. M. (2007). Acute effects of 2 hours of moderate-intensity cycling on serum parathyroid hormone and calcium. *Calcif Tissue Int, 80*(6), 359-365. doi: 10.1007/s00223-007-9028-y

Barry, D. W., & Kohrt, W. M. (2008a). BMD decreases over the course of a year in competitive male cyclists. *Journal of Bone and Mineral Research, 23*(4), 484-491. doi: Doi 10.1359/Jbmr.071203

Barry, D. W., & Kohrt, W. M. (2008b). Exercise and the preservation of bone health. *Journal of Cardiopulmonary Rehabilitation and Prevention, 28*(3), 153-162.

Beck, B. R., & Snow, C. M. (2003). Bone health across the lifespan - Exercising our options. *Exercise and Sport Sciences Reviews, 31*(3), 117-122. doi: Doi 10.1097/00003677-200307000-00003

Bemben, D. A., Buchanan, T. D., Bemben, M. G., & Knehans, A. W. (2004). Influence of type of mechanical loading, menstrual status, and training season on bone density in young women athletes. *Journal of Strength and Conditioning Research, 18*(2), 220-226.

Beshgetoor, D., Nichols, J. F., & Rego, I. (2000). Effect of training mode and calcium intake on bone mineral density in female master cyclist, runners, and non-athletes. *Int J Sport Nutr Exerc Metab, 10*(3), 290-301.

Bonewald, L. F. (2007). Osteocytes as dynamic multifunctional cells. *Ann N Y Acad Sci, 1116*, 281-290. doi: 10.1196/annals.1402.018

Bouassida, A., Zalleg, D., Ajina, M. Z., Gharbi, N., Duclos, M., Richalet, J. P., & Tabka, Z. (2003). Parathyroid hormone concentrations during and after two periods of high intensity exercise with and without an intervening recovery period. *European Journal of Applied Physiology, 88*(4-5), 339-344. doi: DOI 10.1007/s00421-002-0721-2

Brown, R. C., Cox, C. M., & Goulding, A. (2000). High-carbohydrate versus high-fat diets: effect on body composition in trained cyclists. *Medicine and Science in Sports and Exercise, 32*(3), 690-694. doi: Doi 10.1097/00005768-200003000-00021

Brown, S. A., & Rosen, C. J. (2003). Osteoporosis. *Medical Clinics of North America, 87*(5), 1039-+. doi: Doi 10.1016/S0025-7125(03)00065-8

Campion, F., Nevill, A. M., Karlsson, M. K., Lounana, J., Shabani, M., Fardellone, P., & Medelli, J. (2010). Bone Status in Professional Cyclists. *International Journal of Sports Medicine, 31*(7), 511-515. doi: DOI 10.1055/s-0029-1243616

Cheung, A. M., & Giangregorio, L. (2012). Mechanical stimuli and bone health: what is the evidence? *Current Opinion in Rheumatology, 24*(5), 561-566. doi: Doi 10.1097/Bor.0b013e3283570238

Clarke, B. (2008). Normal bone anatomy and physiology. *Clin J Am Soc Nephrol, 3 Suppl 3*, S131-139. doi: 10.2215/CJN.04151206

Cowley, H. R., Ford, K. R., Myer, G. D., Kernozek, T. W., & Hewett, T. E. (2006). Differences in neuromuscular strategies between landing and cutting tasks in female basketball and soccer athletes. *Journal of Athletic Training, 41*(1), 67-73.

Duncan, C. S., Blimkie, C. J., Cowell, C. T., Burke, S. T., Briody, J. N., & Howman-Giles, R. (2002). Bone mineral density in adolescent female athletes: relationship to exercise type and muscle strength. *Med Sci Sports Exerc, 34*(2), 286-294.

Duncan, R. L., & Turner, C. H. (1995). Mechanotransduction and the Functional-Response of Bone to Mechanical Strain. *Calcified Tissue International, 57*(5), 344-358. doi: Doi 10.1007/Bf00302070

Farrell, K. C., Reisinger, K. D., & Tillman, M. D. (2003). Force and repetition in cycling: possible implications for iliotibial band friction syndrome. *Knee, 10*(1), 103-109.

Ferry, B., Duclos, M., Burt, L., Therre, P., Le Gall, F., Jaffre, C., & Courteix, D. (2011). Bone geometry and strength adaptations to physical constraints inherent in different sports: comparison between elite female soccer players and swimmers. *Journal of Bone and Mineral Metabolism, 29*(3), 342-351. doi: 10.1007/s00774-010-0226-8

Frost, H. M. (1987). Bone "mass" and the "mechanostat": a proposal. *Anat Rec, 219*(1), 1-9. doi: 10.1002/ar.1092190104

Frost, H. M. (1997). Why do marathon runners have less bone than weight lifters? A vital-biomechanical view and explanation. *Bone, 20*(3), 183-189. doi: 10.1016/s8756-3282(96)00311-0

Garcia-Pallares, J., Sanchez-Medina, L., Perez, C. E., Izquierdo-Gabarren, M., & Izquierdo, M. (2010). Physiological Effects of Tapering and Detraining in World-Class Kayakers. *Medicine and Science in Sports and Exercise, 42*(6), 1209-1214. doi: Doi 10.1249/Mss.0b013e3181c9228c

Gomez-Bruton, A., Gonzalez-Aguero, A., Olmedillas, H., Gomez-Cabello, A., Matute-Llorente, A., Julian-Almarcegui, C., . . . Vicente-Rodriguez, G. (2013). Do calcium and vitamin D intake influence the effect of cycling on bone mass through adolescence? *Nutricion Hospitalaria, 28*(4), 1136-1139. doi: DOI 10.3305/nh.2013.28.4.6490

Gomez-Cabello, A., Ara, I., Gonzalez-Aguero, A., Casajus, J. A., & Vicente-Rodriguaz, G. (2012). Effects of Training on Bone Mass in Older Adults. *Sports Medicine, 42*(4), 301-325.

Goulet, G. C., Halonen, N. R., Koch, L. G., Britton, S. L., Zernicke, R. F., & Kozloff, K. M. (2011). Osteoblast Response to Ovariectomy Is Enhanced in Intrinsically High Aerobic-Capacity Rats. *Calcified Tissue International, 88*(4), 325-335. doi: DOI 10.1007/s00223-010-9457-x

Guillemant, J., Accarie, C., Peres, G., & Guillemant, S. (2004). Acute effects of an oral calcium load on markers of bone metabolism during endurance cycling exercise in male athletes. *Calcified Tissue International, 74*(5), 407-414. doi: DOI 10.1007/s00223-003-0070-0

Harley, J. A., Hind, K., & O'Hara, J. P. (2011). Three-Compartment Body Composition Changes in Elite Rugby League Players during a Super League Season, Measured by Dual-Energy X-Ray Absorptiometry. *Journal of Strength and Conditioning Research, 25*(4), 1024-1029. doi: Doi 10.1519/Jsc.0b013e3181cc21fb

Heinonen, A., Oja, P., Sievanen, H., Pasanen, M., & Vuori, I. (1998). Effect of two training regimens on bone mineral density in healthy perimenopausal women: A randomized controlled trial. *Journal of Bone and Mineral Research, 13*(3), 483-490. doi: DOI 10.1359/jbmr.1998.13.3.483

Hetland, M. L., Haarbo, J., & Christiansen, C. (1993). Low Bone Mass and High Bone Turnover in Male Long-Distance Runners. *Journal of Clinical Endocrinology & Metabolism, 77*(3), 770-775. doi: Doi 10.1210/Jc.77.3.770

Hinrichs, T. E.-H., C.; Lehmann, R.; Allolio, B.; Platen, P. (2010). Bone Mineral Density in Athletes of Different Disciplines : a Cross-Sectional Study. *The Open Sports Sciences Journal, 3*, 129-133.

Hinton, P. S., Rolleston, A., Rehrer, N. J., Hellemans, I. J., & Miller, B. F. (2010). Bone formation is increased to a greater extent than bone resorption during a cycling stage race. *Applied Physiology Nutrition and Metabolism-Physiologie Appliquee Nutrition Et Metabolisme, 35*(3), 344-349. doi: Doi 10.1139/H10-025

Hughes, J. M., & Petit, M. A. (2010). Biological underpinnings of Frost's mechanostat thresholds: the important role of osteocytes. *J Musculoskelet Neuronal Interact, 10*(2), 128-135.

Ihle, R., & Loucks, A. B. (2004). Dose-response relationships between energy availability and bone turnover in young exercising women. *Journal of Bone and Mineral Research, 19*(8), 1231-1240. doi: Doi 10.1359/Jbmr.040410

Jain, N. X., Barr-Gillespie, A. E., Clark, B. D., Kietrys, D. M., Wade, C. K., Litvin, J., . . . Barbe, M. F. (2014). Bone loss from high repetitive high force loading is prevented by ibuprofen treatment. *J Musculoskelet Neuronal Interact, 14*(1), 78-94.

Johnell, O., & Kanis, J. A. (2006). An estimate of the worldwide prevalence and disability associated with osteoporotic fractures. *Osteoporosis International, 17*(12), 1726-1733. doi: DOI 10.1007/s00198-006-0172-4

Judex, S., & Carlson, K. J. (2009). Is bone's response to mechanical signals dominated by gravitational loading? *Med Sci Sports Exerc, 41*(11), 2037-2043. doi: 10.1249/MSS.0b013e3181a8c6e5

Judex, S., Gross, T. S., & Zernicke, R. F. (1997). Strain gradients correlate with sites of exercise-induced bone-forming surfaces in the adult skeleton. *Journal of Bone and Mineral Research, 12*(10), 1737-1745. doi: DOI 10.1359/jbmr.1997.12.10.1737

Kanis, J. A., Alexeeva, L., Bonjour, J. P., Burkhardt, P., Christiansen, C., Cooper, C., . . . Tosteson, A. (1994). Assessment of Fracture Risk and Its Application to Screening for Postmenopausal Osteoporosis - Synopsis of a Who Report. *Osteoporosis International, 4*(6), 368-381. doi: Doi 10.1007/Bf01622200

Kaptoge, S., Benevolenskaya, L. I., Bhalla, A. K., Cannata, J. B., Boonen, S., Falch, J. A., . . . Reeve, J. (2005). Low BMD is less predictive than reported falls for future limb fractures in women across Europe: results from the European Prospective Osteoporosis Study. *Bone, 36*(3), 387-398. doi: DOI 10.1016/j.bone.2004.11.012

Karsenty, G. (2006). Convergence between bone and energy homeostases: leptin regulation of bone mass. *Cell Metab, 4*(5), 341-348. doi: 10.1016/j.cmet.2006.10.008

Katsumi, A. O., A.W.; Tzima, E.; Schwartz, M.A. (2003). Integrins in Mechanotransduction. *Journal of Biological Chemistry, 279*(13), 12001-12004. doi: doi:10.1074/jbc.R300038200

Kelley, G. A., Kelley, K. S., & Kohrt, W. M. (2013). Exercise and Bone Mineral Density in Premenopausal Women: A Meta-Analysis of Randomized Controlled Trials (vol 2013, 741639, 2013). *International Journal of Endocrinology*. doi: Artn 583217

Doi 10.1155/2013/583217

Kerschan-Schindl, K., Thalmann, M., Sodeck, G. H., Skenderi, K., Matalas, A. L., Grampp, S., . . . Pietschmann, P. (2009). A 246-km continuous running race causes significant changes in bone metabolism. *Bone, 45*(6), 1079-1083. doi: 10.1016/j.bone.2009.07.088

Klesges, R. C., Ward, K. D., Shelton, M. L., Applegate, W. B., Cantler, E. D., Palmieri, G. M. A., . . . Davis, J. (1996). Changes in bone mineral content in male athletes - Mechanisms of action and intervention effects. *Jama-Journal of the American Medical Association, 276*(3), 226-230. doi: DOI 10.1001/jama.276.3.226

Kohrt, W. M., Bloomfield, S. A., Little, K. D., Nelson, M. E., Yingling, V. R., & American College of Sports, M. (2004). American College of Sports Medicine Position Stand: physical activity and bone health. *Med Sci Sports Exerc, 36*(11), 1985-1996.

Kringelbach, T. M., Aslan, D., Novak, I., Schwarz, P., & Jorgensen, N. R. (2014). UTP-induced ATP release is a fine-tuned signalling pathway in osteocytes. *Purinergic Signal, 10*(2), 337-347. doi: 10.1007/s11302-013-9404-1

Lloyd, S. A., & Donahue, H. J. (2010). Gap Junctions and Biophysical Regulation of Bone Cells. *Clin Rev Bone Miner Metab, 8*(4), 189-200. doi: 10.1007/s12018-011-9084-8

Manolagas, S. C. (2000). Birth and death of bone cells: Basic regulatory mechanisms and implications for the pathogenesis and treatment of osteoporosis. *Endocrine Reviews, 21*(2), 115-137. doi: Doi 10.1210/Er.21.2.115

McClanahan, B. S., Ward, K. D., Vukadinovich, C., Klesges, R. C., Chitwood, L., Kinzey, S. J., . . . Frate, D. (2002). Bone mineral density in triathletes over a competitive season. *Journal of Sports Sciences, 20*(6), 463-469. doi: Doi 10.1080/02640410252925134

McVeigh, J. A., Meiring, R., Cimato, A., Micklesfield, L. K., & Oosthuyse, T. (2014). Radial bone size and strength indices in male road cyclists, mountain bikers and controls. *Eur J Sport Sci*, 1-9. doi: 10.1080/17461391.2014.933881

Medelli, J., Lounana, J., Menuet, J. J., Shabani, M., & Cordero-MacIntyre, Z. (2009). Is Osteopenia a Health Risk in Professional Cyclists? *Journal of Clinical Densitometry, 12*(1), 28-34. doi: DOI 10.1016/j.jocd.2008.07.057

Medelli, J., Shabani, M., Lounana, J., Fardellone, P., & Campion, F. (2009). Low bone mineral density and calcium intake in elite cyclists. *Journal of Sports Medicine and Physical Fitness, 49*(1), 44-53.

Morgan, A. L., & Jarrett, J. W. (2011). Markers of bone turnover across a competitive season in female athletes: a preliminary investigation. *Journal of Sports Medicine and Physical Fitness, 51*(3), 515-524.

Mosley, J. R., & Lanyon, L. E. (1998). Strain rate as a controlling influence on adaptive modeling in response to dynamic loading of the ulna in growing male rats. *Bone, 23*(4), 313-318. doi: Doi 10.1016/S8756-3282(98)00113-6

Mullender, M., El Haj, A. J., Yang, Y., van Duin, M. A., Burger, E. H., & Klein-Nulend, J. (2004). Mechanotransduction of bone cells in vitro: mechanobiology of bone tissue. *Med Biol Eng Comput, 42*(1), 14-21.

Nagle, K. B., & Brooks, M. A. (2011). A Systematic Review of Bone Health in Cyclists. *Sports Health, 3*(3), 235-243. doi: 10.1177/1941738111398857

Narra, N., Nikander, R., Viik, J., Hyttinen, J., & Sievanen, H. (2013). Femoral neck cross-sectional geometry and exercise loading. *Clin Physiol Funct Imaging, 33*(4), 258-266. doi: 10.1111/cpf.12022

Navarro, M. D., Saavedra, P., Jodar, E., de Tejada, M. J. G., Mirallave, A., & Sosa, M. (2013). Osteoporosis and metabolic syndrome according to socio-economic status, contribution of PTH, vitamin D and body weight: The Canarian Osteoporosis Poverty Study (COPS). *Clinical Endocrinology, 78*(5), 681-686. doi: Doi 10.1111/Cen.12051

Neary, J. P., Martin, T. P., & Quinney, H. A. (2003). Effects of taper on endurance cycling capacity and single muscle fiber properties. *Med Sci Sports Exerc, 35*(11), 1875-1881. doi: 10.1249/01.MSS.0000093617.28237.20

Nichols, D. L., Sanborn, C. F., Bonnick, S. L., Benezra, V., Gench, B., & Dimarco, N. M. (1994). The Effects of Gymnastics Training on Bone-Mineral Density. *Medicine and Science in Sports and Exercise, 26*(10), 1220-1225.

Nichols, J. F., Palmer, J. E., & Levy, S. S. (2003). Low bone mineral density in highly trained male master cyclists. *Osteoporosis International, 14*(8), 644-649. doi: DOI 10.1007/s00198-003-1418-z

Nichols, J. F., & Rauh, M. J. (2011). Longitudinal Changes in Bone Mineral Density in Male Master Cyclists and Nonathletes. *Journal of Strength and Conditioning Research, 25*(3), 727-734. doi: Doi 10.1519/Jsc.0b013e3181c6a116

Nikander, R., Sievanen, H., Heinonen, A., Karstila, T., & Kannus, P. (2008). Load-specific differences in the structure of femoral neck and tibia between world-class moguls skiers and slalom skiers. *Scandinavian Journal of Medicine & Science in Sports, 18*(2), 145-153. doi: DOI 10.1111/j.1600-0838.2007.00643.x

Nouvenne, A., Ticinesi, A., Guerra, A., Folesani, G., Allegri, F., Pinelli, S., . . . Meschi, T. (2013). Influence of lean and fat mass on bone mineral density and on urinary stone risk factors in healthy women. *J Transl Med, 11*, 248. doi: 10.1186/1479-5876-11-248

Olmedillas, H., Gonzalez-Aguero, A., Moreno, L. A., Casajus, J. A., & Vicente-Rodriguez, G. (2011). Bone Related Health Status in Adolescent Cyclists. *Plos One, 6*(9). doi: ARTN e24841

DOI 10.1371/journal.pone.0024841

Olmedillas, H., Gonzalez-Aguero, A., Moreno, L. A., Casajus, J. A., & Vicente-Rodriguez, G. (2012). Cycling and bone health: a systematic review. *Bmc Medicine, 10*. doi: Artn 168

Doi 10.1186/1741-7015-10-168

Olszynski, W. P., Davison, K. S., Adachi, J. D., Brown, J. P., Cummings, S. R., Hanley, D. A., . . . Yuen, C. K. (2004). Osteoporosis in men: Epidemiology, diagnosis, prevention, and treatment. *Clinical Therapeutics, 26*(1), 15-28. doi: Doi 10.1016/S0149-2918(04)90002-1

Popovtzer, M. M. (1997). Gravitational forces and bone metabolism. *American Journal of Kidney Diseases, 30*(6), xxxiv-xxxvi.

Rapid bone loss in high-performance male athletes. (1996). *Medical Digest, 18*(20).

Rasmusse.H. (1971). Ionic and Hormonal Control of Calcium Homeostasis. *American Journal of Medicine, 50*(5), 567-&. doi: Doi 10.1016/0002-9343(71)90113-6

Rector, R. S., Rogers, R., Ruebel, M., Widzer, M. O., & Hinton, P. S. (2009). Lean Body Mass and Weight-Bearing Activity in the Prediction of Bone Mineral Density in Physically

Active Men. *Journal of Strength and Conditioning Research, 23*(2), 427-435. doi: Doi 10.1519/Jsc.0b013e31819420e1
Reid, I. R., Ames, R., Evans, M. C., Sharpe, S., Gamble, G., France, J. T., . . . Cundy, T. F. (1992). Determinants of total body and regional bone mineral density in normal postmenopausal women--a key role for fat mass. *J Clin Endocrinol Metab, 75*(1), 45-51. doi: 10.1210/jcem.75.1.1619030
Rico, H., Revilla, M., Hernandez, E. R., Gomezcastresana, F., & Villa, L. F. (1993). Bone-Mineral Content and Body-Composition in Postpubertal Cyclist Boys. *Bone, 14*(2), 93-95. doi: Doi 10.1016/8756-3282(93)90233-Z
Risser, W. L., Lee, E. J., Leblanc, A., Poindexter, H. B., Risser, J. M. H., & Schneider, V. (1990). Bone-Density in Eumenorrheic Female College Athletes. *Medicine and Science in Sports and Exercise, 22*(5), 570-574. doi: Doi 10.1249/00005768-199010000-00005
Rizzoli, R., Bianchi, M. L., Garabedian, M., McKay, H. A., & Moreno, L. A. (2010). Maximizing bone mineral mass gain during growth for the prevention of fractures in the adolescents and the elderly. *Bone, 46*(2), 294-305. doi: DOI 10.1016/j.bone.2009.10.005
Rubin, C. T., & Lanyon, L. E. (1985). Regulation of Bone Mass by Mechanical Strain Magnitude. *Calcified Tissue International, 37*(4), 411-417. doi: Doi 10.1007/Bf02553711
Sabo, D., Bernd, L., Pfeil, J., & Reiter, A. (1996). Bone quality in the lumbar spine in high-performance athletes. *Eur Spine J, 5*(4), 258-263.
Scofield, K. L., & Hecht, S. (2012). Bone health in endurance athletes: runners, cyclists, and swimmers. *Curr Sports Med Rep, 11*(6), 328-334. doi: 10.1249/JSR.0b013e3182779193
Scott, A., Khan, K. M., Duronio, V., & Hart, D. A. (2008). Mechanotransduction in human bone: in vitro cellular physiology that underpins bone changes with exercise. *Sports Medicine, 38*(2), 139-160.
Sherk, V. D., Barry, D. W., Villalon, K. L., Hansen, K. C., Wolfe, P., & Kohrt, W. M. (2014). Bone loss over 1 year of training and competition in female cyclists. *Clin J Sport Med, 24*(4), 331-336. doi: 10.1097/JSM.0000000000000050
Silberman, M. R. (2013). Bicycling injuries. *Curr Sports Med Rep, 12*(5), 337-345. doi: 10.1249/JSR.0b013e3182a4bab7
Silverthorn, D. U. (2007). *Human physiology : an integrated approach* (4th ed.). San Francisco: Pearson/Benjamin Cummings.
Skerry, T. M. (2006). One mechanostat or many? Modifications of the site-specific response of bone to mechanical loading by nature and nurture. *J Musculoskelet Neuronal Interact, 6*(2), 122-127.
Skerry, T. M. (2008). The response of bone to mechanical loading and disuse: Fundamental principles and influences on osteoblast/osteocyte homeostasis. *Archives of Biochemistry and Biophysics, 473*(2), 117-123. doi: DOI 10.1016/j.abb.2008.02.028
Smathers, A. M., Bemben, M. G., & Bemben, D. A. (2009). Bone Density Comparisons in Male Competitive Road Cyclists and Untrained Controls. *Medicine and Science in Sports and Exercise, 41*(2), 290-296. doi: Doi 10.1249/Mss.0b013e318185493e
Snow, C. M., Williams, D. P., LaRiviere, J., Fuchs, R. K., & Robinson, T. L. (2001). Bone gains and losses follow seasonal training and detraining in gymnasts. *Calcified Tissue International, 69*(1), 7-12. doi: DOI 10.1007/s00223-001-0014-5
Stewart, A. D., & Hannan, J. (2000). Total and regional bone density in male runners, cyclists, and controls. *Medicine and Science in Sports and Exercise, 32*(8), 1373-1377. doi: Doi 10.1097/00005768-200008000-00003

Sutton, A. L. M., & MacDonald, P. N. (2003). Vitamin D: More than a "Bone-a-Fide" hormone. *Molecular Endocrinology, 17*(5), 777-791. doi: Doi 10.1210/Me.2002.0363

Syed, F., & Khosla, S. (2005). Mechanisms of sex steroid effects on bone. *Biochem Biophys Res Commun, 328*(3), 688-696. doi: 10.1016/j.bbrc.2004.11.097

Tenforde, A. S., & Fredericson, M. (2011). Influence of Sports Participation on Bone Health in the Young Athlete: A Review of the Literature. *Pm&R, 3*(9), 861-867. doi: DOI 10.1016/j.pmrj.2011.05.019

Thomsen, J. S., Ebbesen, E. N., & Mosekilde, L. (2002). Static histomorphometry of human iliac crest and vertebral trabecular bone: a comparative study. *Bone, 30*(1), 267-274.

Uzunca, K., Birtane, M., Durmus-Altun, G., & Ustun, F. (2005). High bone mineral density in loaded skeletal regions of former professional football (soccer) players: what is the effect of time after active career? *Br J Sports Med, 39*(3), 154-157; discussion 154-157. doi: 10.1136/bjsm.2003.011494

Valentino, R., Savastano, S., Tommaselli, A. P., D'Amore, G., Dorato, M., & Lombardi, G. (2001). The influence of intense ballet training on trabecular bone mass, hormone status, and gonadotropin structure in young women. *Journal of Clinical Endocrinology & Metabolism, 86*(10), 4674-4678. doi: DOI 10.1210/jc.86.10.4674

VanSwearingen, J. M., & Studenski, S. A. (2014). Aging, Motor Skill, and the Energy Cost of Walking: Implications for the Prevention and Treatment of Mobility Decline in Older Persons. *J Gerontol A Biol Sci Med Sci*. doi: 10.1093/gerona/glu153

Vicente-Rodriguez, G., Jimenez-Ramirez, J., Ara, I., Serrano-Sanchez, J. A., Dorado, C., & Calbet, J. A. (2003). Enhanced bone mass and physical fitness in prepubescent footballers. *Bone, 33*(5), 853-859.

Warming, L., Hassager, C., & Christiansen, C. (2002). Changes in bone mineral density with age in men and women: A longitudinal study. *Osteoporosis International, 13*(2), 105-112. doi: DOI 10.1007/s001980200001

Warner, S. E., Shaw, J. M., & Dalsky, G. P. (2002). Bone mineral density of competitive male mountain and road cyclists. *Bone, 30*(1), 281-286. doi: Doi 10.1016/S8756-3282(01)00704-9

Wilks, D. C., Gilliver, S. F., & Rittweger, J. (2009). Forearm and Tibial Bone Measures of Distance- and Sprint-Trained Master Cyclists. *Medicine and Science in Sports and Exercise, 41*(3), 566-573. doi: Doi 10.1249/Mss.0b013e31818a0ec8

Wilmore, J. H., Costill, D. L., & Kenney, W. L. (2008). *Physiology of sport and exercise* (4th ed.). Champaign, IL: Human Kinetics.

Witzke, K. A., & Snow, C. M. (2000). Effects of plyometric jump training on bone mass in adolescent girls. *Med Sci Sports Exerc, 32*(6), 1051-1057.

Young, K. C. K., K.L.; Patterson, K.M.; Pandya, P.D.; Fairman, C.M.; Smith, S.M. (2014). Rowing Performance, Body Composition, and Bone Mineral Density Outcomes in Collegiate-Level Rowers Following a Season of Concurrent Training. *Int J Sports Physiol Perform, 9*(2).

Zeng, H., Bai, D., & Han, X. L. (2014). [The biological function of osteocytes]. *Sheng Li Ke Xue Jin Zhan, 45*(2), 100-104.

APPENDIX I

BONE DENSITY IN COMPETITIVE CYCLISTS: A LONGITUDINAL ASSESSMENT ACROSS THE SEASON

INFORMED CONSENT

Consent to Participate in a Research Study
Colorado State University

TITLE OF STUDY: **Bone Density in Competitive Cyclist: A longitudinal Assessment Across the Season**

PRINCIPAL INVESTIGATOR: Dr. Raoul Reiser, Ph.D., Department of Health and Exercise Science. Director of the Clinical Biomechanics Laboratory. Contact at (970) 491-6958 or Raoul.Reiser@ColoState.edu.

CO-PRINCIPAL INVESTIGATOR: Dr. Ray Browning Ph.D., Department of Health and Exercise Science. Director of the Physical Activity Energetics/Mechanics Laboratory. Contact at (970) 491-5868 or Ray.Browning@Colostate.edu.

CO-INVESTIGATOR: Bree Baker, Graduate Student, Department of Health and Exercise Science. Contact at (719) 429-2690 or Bree Baker at Bree.Baker@ColoState.edu.

WHY AM I BEING INVITED TO TAKE PART IN THIS RESEARCH? You are a perfect candidate for our research study because of your commitment to cycling at a competitive level and interest in personal health.

WHO IS DOING THE STUDY? This research is being conducted by three primary members, Dr. Reiser, Dr. Browning and Bree Baker. Dr. Reiser is interested in musculoskeletal biomechanics. Dr. Browning is interested in the development of physical activity equipment, interventions and monitoring tools for the prevention and treatment of obesity. Both Dr. Reiser and Dr. Browning have performed many cycling studies in the past. Bree Baker is a graduate student interested in bone metabolism within competitive athletes.

WHAT IS THE PURPOSE OF THIS STUDY? The purpose of this study is to assess bone mineral density changes in competitive cyclists over a nine month season and to identify possible reasons for bone mineral density change such as age, gender, body composition, dietary calcium and vitamin D intake.

WHERE IS THE STUDY GOING TO TAKE PLACE AND HOW LONG WILL IT LAST? All aspects of the study will take place in the Human Performance Clinical/Research Laboratory on the Colorado State University campus, 910 Moby Drive, Fort Collins, CO 80523. Two visits will be required. Each will include you submitting your food log and a DEXA scan. These visits will occur near the beginning and end of the season, January/February and August/September. Both visits will take about 30 minutes to complete. Over the season a total of about 60 minutes of your time will be spent on the CSU campus.

WHAT WILL I BE ASKED TO DO? In January/February you will show up for your first visit. Before your visit you will be asked to complete a four day dietary recall and bring this information with you. This visit will begin with a DEXA scan. The DEXA is a machine that will use x-rays to determine your body composition and bone mineral content. The DEXA scan requires you to lie quietly on a padded table while a small probe gives off low-level x-rays and sends them over your entire body. This test gives very accurate measurements of your body fat

and bone mineral density. After the scan is completed the visit will be over. In August/September you will be asked to do a DEXA scan and dietary recall again.

ARE THERE REASONS WHY I SHOULD NOT TAKE PART IN THIS STUDY? If you are under the age of 18 or over the age of 49 you are not eligible to participate. If you have had any history of metabolic bone disorders or have suffered a broken or fractured bone in the last six months you cannot participate. You must be at least a Category 4 racer as defined by USA Cycling. If there are any reasons that you can foresee limiting your ability to make both visits or race the whole season you should not participate in the study. For female participants, if you are pregnant or have not had a normal menstrual cycle the past six months you are prohibited from participation. You will also not be able to participate if we find an abnormally low bone density (z score 2.5 below average) from your first DEXA scan.

WHAT ARE THE POSSIBLE RISKS AND DISCOMFORTS? The risks associated with the DEXA are very low. The maximum radiation dose you will receive per scan is less than 1/3000th of the federal and state occupational whole body dose limit allowed to radiation workers. Put another way, you will receive less than 1.3 mrem from this scan and you already receive approximately 450 mrem per year from normal background radiation doses in Colorado. However, the more radiation you receive over the course of your life, the more the risk increases of developing a fatal cancer or inducing changes in genes. The radiation dose you receive from this scan is not expected to significantly increase these risks, but the exact increase in such risks is not known. There are no discomforts associated with this procedure. It is not possible to identify all potential risks in research procedures, but the researchers have taken reasonable safeguards to minimize any known and potential, but unknown, risks.

ARE THERE ANY BENEFITS FROM TAKING PART IN THIS STUDY? There are no direct benefits to you; however, you will have the benefit of receiving full body composition testing via DEXA and nutritional analysis for free. Your bone mineral density results will be discussed with you after each DEXA scan however, all body composition and food log results won't be released to you until the end of the study.

DO I HAVE TO TAKE PART IN THE STUDY? Your participation in this research is voluntary. If you decide to participate in the study, you may withdraw your consent and stop participating at any time.

WHO WILL SEE THE INFORMATION THAT I GIVE? We will keep private all research records that identify you, to the extent allowed by law. For this study, we will assign a code to your data, example you might be identified as A1Y. The only place your name will appear in our records is on the consent, a contact information page, and in our data spreadsheet which links you to your code. Only the research team will have access to the link between you, your code, and your data. The only exceptions to this are if we are asked to share the research files for audit purposes with the CSU Institutional Review Board ethics committee, if necessary. In addition, for funded studies, the CSU financial management team may also request an audit of research expenditures. For financial audits, only the fact that you participated would be shared, not any research data.

CAN MY TAKING PART IN THE STUDY END EARLY? If you fail to appear for both visits or if you lose the ability to train, compete or meet any of the exclusion criteria at any time throughout the season you may be removed from the study.

WILL I RECEIVE ANY COMPENSATION FOR TAKING PART IN THIS STUDY? You will not receive any financial compensation for involvement.

WHAT HAPPENS IF I AM INJURED BECAUSE OF THE RESEARCH? The Colorado Governmental Immunity Act determines and may limit Colorado State University's legal responsibility if an injury happens because of this study. Claims against the University must be filed within 180 days of the injury.

WHAT IF I HAVE QUESTIONS? Before you decide whether to accept this invitation to take part in the study, please ask any questions that might come to mind now. Later, if you have questions about the study, you can contact Dr. Raoul Reiser at Raoul.Reiser@Colostate.edu or Bree Baker at Bree.Baker@ColoState.edu. If you have any questions about your rights as a volunteer in this research, contact Janell Barker, Human Research Administrator at 970-491-1655. We will give you a copy of this consent form to take with you.

Your signature acknowledges that you have read the information stated and willingly sign this consent form. Your signature also acknowledges that you have received, on the date signed, a copy of this document containing 2 pages.

_____ _____

Signature of person agreeing to take part in the study Date

_____

Printed name of person agreeing to take part in the study

_____ _____

Name of person providing information to participant Date

_____

Signature of Research Staff

APPENDIX II

BONE DENSITY IN COMPETITIVE CYCLISTS: A LONGITUDINAL ASSESSMENT ACROSS THE SEASON

HEALTH & HISTORY QUESTIONNAIRE

Subject ID: _____

Cyclist's BMD Study Health Screening - Coded Cover Sheet

(Separate from the coded screening form, store separately)

Project Title: ***Bone Mineral Density in Cyclists over a Competitive Season***

Name (Last, First): _____

Address: _____ .
_____
_____

Phone number:(_____)_____-_____

Email: _____

Screened by:_____

Acceptable Subject: Yes or No

Willing to be contacted for future studies: Yes or No

Notes_____
_____
_____
_____
_____

(This page intentionally blank)

- Phone Screening Section-

Screener's initials: _____ Screening date:_____ Approved: Y or N

Subject ID:_____ Sex: **O** M **O** F DOB:_____ Age:_____

Cycling Information

Please estimate the average training mileage you cycle over the last 4 weeks? _____

How many races do you plan on attending this season from January-September? ____

Please explain what physical activity you engage in other than cycling? Please include how many times per week and for how long you participate in these activities.

_____
_____
_____

Please describe your cycling habits over the course of a year.

_____
_____
_____

Years of cycling experience _____ Years of racing experience _____

Please list any race categories you compete in (example Cat-4 Mountain Bike) and how long you have been in that category.

_____
_____

Please estimate how many non-training miles per week you cycle for purposes such as recreation or commuting needs _____

What type(s) of bike do you ride (check all that apply)? O Mtn O Road O CX O Other

What type(s) of bike do you race (check all that apply)? O Mtn O Road O CX O Other

Personal Health Information

List any medications you are currently taking:

_____
_____

List any vitamins and/or supplements you are currently taking:

_____

_____

Are you currently pain and injury free? O Yes O No

If not please explain

_____

Have all prior injuries healed at least 4 months ago? O Yes O No

If not please explain

_____

_____

Do you have any bone disorders that affect your ability to exercise? O Yes O No

If so please explain

_____

_____

Female Participants Only

Have you had a regular menstrual cycle over the past 6 months? O Yes O No

Are you experiencing pre-menopausal symptoms? O Yes O No

Are you pregnant or trying to become pregnant over the next 9 months? O Yes O No

Study Requirements

Are you available for a DEXA scan in Jan/Feb, and Aug/Sept? O Yes O No

Are you able to provide your own transportation to and from CSU? O Yes O No

Please list any reasons that you might not be able to participate in both visits:

Example: My wife and I might be moving this summer or last year I didn't finish the season because of work conflicts.

_____

_____

Are you currently involved in any other research studies? O YesO No

If yes please explain

_____

How did you find out about this study?

_____

Are there any other special notes or considerations you think the researchers should know about prior to the study?

_____

_____

APPENDIX III

BONE DENSITY IN COMPETITIVE CYCLISTS: A LONGITUDINAL ASSESSMENT ACROSS THE SEASON

RECRUITING FLIER

Bone Mineral Density Study in Cyclists

If you are:

- **18-49 years old**
- **Cat 1-4 or professional/elite racer for USCA**
- **Competing in the 2014 road or mountain racing season**

The Department of Health and Exercise Science at Colorado State University is studying the changes of bone mineral density in cyclists from January to September, 2014.

Requirements:

- ***2 visits lasting 30 min each***
- ***Have your body composition, nutrition and physical activity analyzed. A $400 value FREE!***
- ***You are ineligible if you have suffered any bone injuries or lost significant time on the bike in the last 6 months***
- ***Women must not be pregnant and menstruating normally***

If interested, please call the lab of Raoul F. Reiser II, Ph.D. ask to speak with a Research Coordinator.

491-7980

Colorado State University

Don't Delay!

Spring 2014

APPENDIX IV

BONE DENSITY IN COMPETITIVE CYCLISTS: A LONGITUDINAL ASSESSMENT ACROSS THE SEASON

PHONE SCRIPT

Hello,

This is Bree from the Health and Exercise Science Department at CSU. I spoke with you at a group meeting the other day and you expressed an interest in helping us out with our bone mineral density study. Is this a good time to talk?

Let me start by describing the study to you again. We are looking at bone mineral density (BMD) changes associated with cycling. Recent publications suggest that cyclists have a decreased BMD as compared to other athletes. Possible reasons for the observed difference may be age, gender, body composition, training habits, recovery techniques and dietary calcium and vitamin D intake. We are looking to work with both male and female competitive cyclists between the ages of 18-49 years from the area to further understand why cyclists tend to have low BMD. There are a few additional criteria that we will get to if you are interested, but let me tell you a few more things first. The study requires two thirty minute visits to the laboratory in Moby on the west side of the CSU campus. In January/February you will show up for your first visit. Before your visit you will be asked to complete a dietary recall and bring this information with you. This visit will mainly contain a DEXA scan. The DEXA is a machine that uses low dose x-rays to determine your body composition and bone mineral content. Once we have gone through your information bone mineral density results the visit will be over. In August/September you will be asked to do a DEXA scan and dietary recall again. There are no invasive procedures, needles or blood draws required for this study. We will provide you with your BMD results immediately after each scan. All other results will be provided to you at the end of the study. The total value of services provided is $400; however, these are free to you. Are you still interested?

To further verify if you are eligible we have a series of questions relative to your health history.

[go through the Health History Questionnaire with them]

Based on your answers:

a) It does not look like you qualify
b) We would like to schedule your first visit

As we look to schedule the first visit, we want to remind you that if you don't think you will be able to attend both visits for any reason, please tell us now.

What day and times might work for you?

APPENDIX V

BONE DENSITY IN COMPETITIVE CYCLISTS: A LONGITUDINAL ASSESSMENT ACROSS THE SEASON

IRB APPROVAL LETTER

Colorado State University
Knowledge to Go Places

Research Integrity & Compliance Review Office
Office of the Vice President for Research
321 General Services Building - Campus Delivery 2011 Fort Collins, CO TEL: (970) 491-1553
FAX: (970) 491-2293

NOTICE OF APPROVAL FOR HUMAN RESEARCH

DATE: November 11, 2014

TO: Reiser, Raoul, Health & Exercise Science

Braun, Barry, Baker, Bree, 1582 Dept Hlth & Exer Sci, Browning, Ray, 1582 Dept Hlth & Exer Sci

FROM:	Swiss, Evelyn, Coordinator, CSU IRB 1	
PROTOCOL TITLE:	Bone Density in Competitive Cyclists	
FUNDING SOURCE:	NONE	
PROTOCOL NUMBER:	13-4687H	
APPROVAL PERIOD:	Approval Date: November 11, 2014	Expiration Date: January 09, 2015

The CSU Institutional Review Board (IRB) for the protection of human subjects has reviewed the protocol entitled: Bone Density in Competitive Cyclists. The project has been approved for the procedures and subjects described in the protocol. This protocol must be reviewed for renewal on a yearly basis for as long as the research remains active. Should the protocol not be renewed before expiration, all activities must cease until the protocol has been re-reviewed.

If approval did not accompany a proposal when it was submitted to a sponsor, it is the PI's responsibility to provide the sponsor with the approval notice.

This approval is issued under Colorado State University's Federal Wide Assurance 00000647 with the Office for Human Research Protections (OHRP). If you have any questions regarding your obligations under CSU's Assurance, please do not hesitate to contact us.

Please direct any questions about the

IRB's actions on this project to: IRB

Office - (970) 491-1553;

RICRO_IRB@mail.Colostate.edu
Evelyn Swiss, IRB Coordinator - (970) 491-1381; Evelyn.Swiss@Colostate.edu

Swiss, Evelyn

Evelyn Swiss

Swiss, Evelyn

Administrative change to update DEXA risks to consent per IRB approved language and updated personnel section.

Approval Period:	November 11, 2014 through January 09, 2015
Review Type:	EXPEDITED
IRB Number:	00000202

APPENDIX VI

BONE DENSITY IN COMPETITIVE CYCLISTS: A LONGITUDINAL ASSESSMENT ACROSS THE SEASON

CORRELATION TABLES

A. I. 1 Pre-season correlations with all variables for the whole group.

Variable	WB BMC%	LB BMCg	UB BMCg	T score	Z score	Pelvis BMD	Thigh BMD	Shank BMD	Lumbar BMD
Age	.318*	.214	.421**	.179	.174	.156	.164	-.091	.008
Body Mass	.022	.719**	.566**	.091	.117	.080	.490**	.207	-.083
BMI	-.343*	.143	.149	.223	.221	.175	.276	.024	.007
Body Fat %	-.507**	-.523**	-.330*	.127	.091	.136	-.274	-.386*	.188
Lean Mass %	.485**	.517**	.320*	-.146	-.108	-.144	.258	.381*	-.184
Years Experience	.039	.059	.193	-.203	-.197	-.228	-.151	-.082	-.249
Years Racing Experience	-.059	.099	.172	-.180	-.185	-.252	-.029	-.021	-.262
Estimated Mileage	.032	.257	.086	-.022	-.018	-.004	.222	.220	-.006
Kcal	.152	.300	.188	-.024	-.026	-.034	.192	.269	-.076
Vitamin D	-.016	-.170	-.069	-.028	-.008	.014	-.193	-.062	.077
Calcium	.026	.209	.236	-.137	-.126	-.230	.128	.122	-.284

*. Correlation is significant at the 0.05 level (2-tailed).
**. Correlation is significant at the 0.01 level (2-tailed).
BMI=Body Mass Index (kg/m²)
WB BMC%= Whole Body Bone Mineral Content % body mass
LB BMCg= Lower Body Bone Mineral Content in grams
UB BMCg= Upper Body Bone Mineral Content in grams
BMD= Bone Mineral Density

A. I.2 Whole group correlations with all variables as they changed over time Δ.

Variable	Δ WB BMC%	Δ LB BMCg	Δ UB BMCg	Δ T score	Δ Z score	Δ Pelvis BMD	Δ Thigh BMD	Δ Shank BMD	Δ Lumbar BMD
Age	.103	.225	-.193	.139	.112	.014	.168	-.064	-.175
Δ Body Mass	-.833**	.102	-.104	-.169	-.276	-.103	-.088	.207	-.022
Δ BMI	-.860**	.059	-.145	-.213	-.325*	-.094	-.088	.165	-.058
Δ Body Fat %	-.516**	.028	-.110	.003	-.057	-.169	-.054	.131	.115
Δ Lean Mass %	.588**	.048	.110	.034	.112	.158	.028	-.135	-.143
Δ Kcal	-.052	.184	.134	.108	.072	.247	.106	-.091	-.184
Δ Vitamin D	-.375*	.068	.193	-.318	-.404*	.056	.049	-.003	-.179
Δ Calcium	.152	-.038	.160	-.162	-.130	.134	-.079	-.119	-.374*

*. Correlation is significant at the 0.05 level (2-tailed).
**. Correlation is significant at the 0.01 level (2-tailed).
BMI=Body Mass Index (kg/m²)
WB BMC%= Whole Body Bone Mineral Content % body mass
LB BMCg= Lower Body Bone Mineral Content in grams
UB BMCg= Upper Body Bone Mineral Content in grams
BMD= Bone Mineral Density

A. I. 3 Pre-season correlations with all variables for males.

Variable	WB BMC%	LB BMCg	UB BMCg	T score	Z score	Pelvis BMD	Thigh BMD	Shank BMD	Lumbar BMD
Age	.434*	.290	.465*	.367	.374	.208	.263	-.206	-.004
Body Mass	.316	.727**	.661**	.485*	.477*	.316	.545**	-.192	.185
BMI	.249	.205	.458*	.464*	.466*	.372	.395	-.146	.400
Body Fat %	-.194	-.301	.045	-.035	-.036	.008	-.084	-.323	.331
Lean Mass %	.115	.202	-.101	-.055	-.053	-.060	-.016	.276	-.344
Years Experience	.034	-.101	.137	-.069	-.067	-.122	-.217	-.259	-.034
Years Racing Experience	-.123	-.156	.086	-.115	-.126	-.117	-.154	-.227	-.085
Estimated Mileage	-.102	.091	-.057	.057	.056	.158	.214	.131	.147
Kcal	.116	.161	.077	.138	.128	.086	.137	.124	.220
Vitamin D	-.068	.224	-.037	.019	.014	-.192	-.044	.232	-.196
Calcium	-.221	-.084	.103	-.068	-.069	-.429	-.018	-.104	-.336

*. Correlation is significant at the 0.05 level (2-tailed).
**. Correlation is significant at the 0.01 level (2-tailed).
BMI=Body Mass Index (kg/m²)
WB BMC%= Whole Body Bone Mineral Content % body mass
LB BMCg= Lower Body Bone Mineral Content in grams
UB BMCg= Upper Body Bone Mineral Content in grams
BMD= Bone Mineral Density

A. I. 4 Pre-season correlations with all variables for females.

Variable	WB BMC%	LB BMCg	UB BMCg	T score	Z score	Pelvis BMD	Thigh BMD	Shank BMD	Lumbar BMD
Age	.073	-.192	-.040	-.059	-.137	.146	-.206	-.155	.094
Body Mass	-.617**	.434	.081	.058	.057	.003	.263	.258	-.049
BMI	-.458*	.502*	.091	.206	.137	.236	.463*	.473*	.197
Body Fat %	-.621**	.133	-.108	-.174	-.168	.150	-.055	-.020	-.025
Lean Mass %	.604**	-.120	.094	.186	.185	-.143	.058	.030	.043
Years Experience	-.160	-.228	-.165	-.331	-.355	-.388	-.332	-.083	-.421
Years Racing Experience	-.245	.040	-.057	-.153	-.189	-.493*	-.085	.097	-.392
Estimated Mileage	-.047	.044	-.092	.060	.023	-.163	.030	.068	-.009
Kcal	-.075	-.131	-.328	-.123	-.178	-.204	-.074	.183	-.300
Vitamin D	.044	-.295	-.052	-.122	-.071	.095	-.246	-.230	.125
Calcium	.028	-.063	-.076	-.040	-.068	.242	-.037	.095	-.156

*. Correlation is significant at the 0.05 level (2-tailed).
**. Correlation is significant at the 0.01 level (2-tailed).
BMI=Body Mass Index (kg/m²)
WB BMC%= Whole Body Bone Mineral Content % body mass
LB BMCg= Lower Body Bone Mineral Content in grams
UB BMCg= Upper Body Bone Mineral Content in grams
BMD= Bone Mineral Density

A. I. 5 Male correlations with all variables as they changed over time Δ.

Variable	Δ WB BMC%	Δ LB BMCg	Δ UB BMCg	Δ T score	Δ Z score	Δ Pelvis BMD	Δ Thigh BMD	Δ Shank BMD	Δ Lumbar BMD
Age	.127	.326	-.285	.580**	.544**	.256	.088	.337	.259
Δ Body Mass	-.866**	.179	.123	-.171	-.270	.446*	-.197	-.131	-.078
Δ BMI	-.872**	.153	.128	-.192	-.297	.420	-.194	-.165	-.092
Δ Body Fat %	-.657**	-.069	.188	-.200	-.261	.469*	.158	-.308	-.051
Δ Lean Mass %	.599**	.043	-.164	.169	.214	-.459*	-.222	.283	.016
Δ Kcal	-.102	.170	-.143	.041	.003	-.087	.365	.156	-.395
Δ Vitamin D	-.435	-.092	-.158	-.118	-.188	.211	-.112	-.346	-.373
Δ Calcium	.144	-.150	-.047	.016	.076	-.218	.211	-.400	-.146

*. Correlation is significant at the 0.05 level (2-tailed).
**. Correlation is significant at the 0.01 level (2-tailed).
BMI=Body Mass Index (kg/m²)
WB BMC%= Whole Body Bone Mineral Content % body mass
LB BMCg= Lower Body Bone Mineral Content in grams
UB BMCg= Upper Body Bone Mineral Content in grams
BMD= Bone Mineral Density

A. I. 6 Female correlations with all variables as they changed over time Δ.

Variable	Δ WB BMC%	Δ LB BMCg	Δ UB BMCg	Δ T score	Δ Z score	Δ Pelvis BMD	Δ Thigh BMD	Δ Shank BMD	Δ Lumbar BMD
Age	.146	-.009	.213	-.260	-.252	-.426	-.034	-.075	-.019
Δ Body Mass	-.898**	-.142	-.259	-.204	-.349	.149	-.489*	-.303	-.137
Δ BMI	-.899**	-.145	-.262	-.251	-.390	.125	-.486*	-.290	-.153
Δ Body Fat %	-.373	.119	-.090	.190	.143	.352	-.004	-.151	-.163
Δ Lean Mass %	.577**	.123	.140	-.109	-.018	-.339	.151	.333	.134
Δ Kcal	-.045	.276	-.280	.198	.123	-.007	.134	.136	-.188
Δ Vitamin D	-.396	.139	-.661*	-.368	-.455	.059	.216	.017	-.358
Δ Calcium	.113	.171	-.183	-.363	-.369	-.119	.368	-.067	-.558*

*. Correlation is significant at the 0.05 level (2-tailed).
**. Correlation is significant at the 0.01 level (2-tailed).
BMI=Body Mass Index (kg/m²)
WB BMC%= Whole Body Bone Mineral Content % body mass
LB BMCg= Lower Body Bone Mineral Content in grams
UB BMCg= Upper Body Bone Mineral Content in grams
BMD= Bone Mineral Density

A. I. 7 Pre-season correlations with all variables for the USAC Category 1 racers.

Variable	WB BMC%	LB BMCg	UB BMCg	T score	Z score	Pelvis BMD	Thigh BMD	Shank BMD	Lumbar BMD
Age	.214	.042	.234	-.240	-.256	-.343	-.299	-.152	-.176
Body Mass	-.094	.585**	.485*	-.032	.041	.340	.426	.329	.003
BMI	-.367	-.029	.034	.186	.188	.166	.164	-.028	.175
Body Fat %	-.763**	-.573**	-.609**	.193	.174	-.089	-.280	-.482*	-.077
Lean Mass %	.777**	.585**	.617**	-.179	-.155	.120	.284	.477*	.113
Years Experience	.119	.103	.200	-.555*	-.534*	-.354	-.315	-.161	-.346
Years Racing Experience	.025	.215	.124	-.376	-.374	-.156	-.027	-.055	-.263
Estimated Mileage	.300	.257	.200	.186	.195	.193	.339	.368	.267
Kcal	.410	.432	.313	.007	.020	.047	.375	.608**	.026
Vitamin D	.013	.241	.217	-.081	-.073	-.251	.155	.301	-.099
Calcium	.166	.292	.290	-.060	-.041	-.111	.175	.395	-.081

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

BMI=Body Mass Index (kg/m²)

WB BMC%= Whole Body Bone Mineral Content % body mass

LB BMCg= Lower Body Bone Mineral Content in grams

UB BMCg= Upper Body Bone Mineral Content in grams

BMD= Bone Mineral Density

A. I. 8 Pre-season correlations with all variables for the USAC Category 4 racers.

Variable	WB BMC%	LB BMCg	UB BMCg	T score	Z score	Pelvis BMD	Thigh BMD	Shank BMD	Lumbar BMD
Age	.240	.347	.456	.286	.263	.523*	.216	.279	-.009
Body Mass	-.011	.870**	.667**	.052	.059	-.009	.621*	.644**	-.159
BMI	-.020	.502*	.344	.447	.401	.466	.678**	.546*	.252
Body Fat %	-.114	-.562*	-.475	.196	.133	.283	-.399	-.577*	.438
Lean Mass %	.042	.524*	.422	-.254	-.193	-.321	.362	.547*	-.461
Years Experience Cycling	.094	.076	.153	-.006	-.011	.281	-.084	.108	-.176
Years Racing Experience	.328	.236	.383	.169	.176	.024	.363	.558*	-.354
Estimated Mileage	-.322	.461	.199	-.308	-.299	-.177	.351	.272	-.463
Kcal	-.245	.148	.190	-.204	-.216	-.149	.102	.136	-.407
Vitamin D	-.226	-.498	-.272	-.097	-.071	.000	-.474	-.359	.123
Calcium	-.317	.127	-.055	-.318	-.323	-.423	.070	.178	-.579*

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

BMI=Body Mass Index (kg/m²)

WB BMC%= Whole Body Bone Mineral Content % body mass

LB BMCg= Lower Body Bone Mineral Content in grams

UB BMCg= Upper Body Bone Mineral Content in grams

BMD= Bone Mineral Density

A. I. 9 USAC Category 1 correlations with all variables as they changed over time Δ.

Variable	Δ WB BMC%	Δ LB BMCg	Δ UB BMCg	Δ T score	Δ Z score	Δ Pelvis BMD	Δ Thigh BMD	Δ Shank BMD	Δ Lumbar BMD
Age	.124	.259	.026	.291	.299	.085	.003	.459*	-.089
Δ Body Mass	-.805**	-.087	-.402	.016	-.098	.282	-.233	-.260	.126
Δ BMI	-.790**	-.080	-.382	-.003	-.125	.268	-.250	-.290	.117
Δ Body Fat %	.162	.140	.152	.425	.462*	.325	.243	-.135	-.119
Δ Lean Mass %	.171	.122	-.176	-.395	-.353	-.324	-.092	.277	.066
Δ Kcal	-.248	.011	-.104	.034	-.001	-.201	.417	.037	-.245
Δ Vitamin D	-.221	.051	-.582*	-.250	-.206	.083	.171	-.269	.296
Δ Calcium	-.212	-.149	-.045	-.302	-.304	.188	.200	-.567*	-.090

*. Correlation is significant at the 0.05 level (2-tailed).
**. Correlation is significant at the 0.01 level (2-tailed).
BMI=Body Mass Index (kg/m²)
WB BMC%= Whole Body Bone Mineral Content % body mass
LB BMCg= Lower Body Bone Mineral Content in grams
UB BMCg= Upper Body Bone Mineral Content in grams
BMD= Bone Mineral Density

A. I. 10 USAC Category 4 correlations with all variables as they changed over time Δ.

Variable	Δ WB BMC%	Δ LB BMCg	Δ UB BMCg	Δ T score	Δ Z score	Δ Pelvis BMD	Δ Thigh BMD	Δ Shank BMD	Δ Lumbar BMD
Age	.083	.361	-.169	.023	.009	-.068	-.029	.089	-.108
Δ Body Mass	-.877**	.301	-.725**	-.228	-.309	.398	-.130	.018	-.354
Δ BMI	-.909**	.168	-.609*	-.317	-.399	.297	-.155	-.064	-.361
Δ Body Fat %	-.659**	.025	-.473	-.106	-.178	.365	.015	-.206	-.069
Δ Lean Mass %	.649**	-.024	.458	.134	.201	-.341	-.027	.211	.082
Δ Kcal	.146	.617*	-.404	.269	.248	.279	.188	.464	-.309
Δ Vitamin D	-.512	.160	-.679*	-.397	-.530	.096	.053	.051	-.380
Δ Calcium	.141	.040	.344	-.074	-.095	-.501	.360	-.011	-.707**

*. Correlation is significant at the 0.05 level (2-tailed).
**. Correlation is significant at the 0.01 level (2-tailed).
BMI=Body Mass Index (kg/m²)
WB BMC%= Whole Body Bone Mineral Content % body mass
LB BMCg= Lower Body Bone Mineral Content in grams
UB BMCg= Upper Body Bone Mineral Content in grams
BMD= Bone Mineral Density

A. I. 11 Pre-season correlations with all variables for Road Only racers.

Variable	WB BMC%	LB BMCg	UB BMCg	T score	Z score	Pelvis BMD	Thigh BMD	Shank BMD	Lumbar BMD
Age	.327	.091	.658**	.571*	.537*	.449	.642**	-.221	.136
Body Mass	-.056	.569*	.658**	.005	.012	-.162	.360	-.151	-.189
BMI	-.201	.225	.311	.355	.295	.312	.465	.005	.125
Body Fat %	-.359	-.530*	-.307	.166	.097	.287	-.279	-.245	.572*
Lean Mass %	.339	.571*	.300	-.168	-.094	-.278	.290	.257	-.533*
Years Experience Cycling	.022	-.072	.401	.182	.171	.085	.259	-.218	-.149
Years Racing Experience	.028	-.067	.363	.177	.174	.016	.153	-.174	-.174
Estimated Mileage	-.140	.307	.106	-.043	-.015	.027	.147	-.031	.140
Kcal	.125	.511	.345	.049	.076	-.020	.294	.116	-.313
Vitamin D	-.001	.022	-.069	-.056	-.010	-.050	-.037	.168	-.125
Calcium	-.103	.427	.278	-.260	-.232	-.453	.171	.132	-.675**

*. Correlation is significant at the 0.05 level (2-tailed).
**. Correlation is significant at the 0.01 level (2-tailed).
BMI=Body Mass Index (kg/m²)
WB BMC%= Whole Body Bone Mineral Content % body mass
LB BMCg= Lower Body Bone Mineral Content in grams
UB BMCg= Upper Body Bone Mineral Content in grams
BMD= Bone Mineral Density

A. I. 12 Pre-season correlations with all variables for Multiple Bicycle racers.

Variable	WB BMC%	LB BMCg	UB BMCg	T score	Z score	Pelvis BMD	Thigh BMD	Shank BMD	Lumbar BMD
Age	.365	.229	.373	.019	.027	.035	-.108	-.032	-.027
Body Mass	.116	.780**	.654**	.233	.275	.332	.625**	.565**	.000
BMI	-.044	.284	.301	.271	.283	.296	.344	.179	.345
Body Fat %	-.677**	-.515**	-.597**	.039	.028	.005	-.298	-.514**	-.046
Lean Mass %	.646**	.486*	.577**	-.081	-.068	-.028	.261	.492*	.022
Years Experience Cycling	.130	.050	.229	-.298	-.288	-.323	-.347	-.085	-.232
Years Racing Experience	-.073	.121	.138	-.315	-.326	-.347	-.107	.030	-.256
Estimated Mileage	.156	.233	.196	.055	.042	.025	.301	.380	-.026
Kcal	.199	.236	.218	-.022	-.040	-.022	.171	.374	.017
Vitamin D	-.034	-.266	-.128	-.025	-.021	.047	-.335	-.269	.170
Calcium	.049	.175	.171	-.214	-.208	-.169	.078	.214	-.184

*. Correlation is significant at the 0.05 level (2-tailed).
**. Correlation is significant at the 0.01 level (2-tailed).
BMI=Body Mass Index (kg/m²)
WB BMC%= Whole Body Bone Mineral Content % body mass
LB BMCg= Lower Body Bone Mineral Content in grams
UB BMCg= Upper Body Bone Mineral Content in grams
BMD= Bone Mineral Density

A. I. 13 Road bike only correlations with all variables as they changed over time Δ.

Variable	Δ WB BMC%	Δ LB BMCg	Δ UB BMCg	Δ T score	Δ Z score	Δ Pelvis BMD	Δ Thigh BMD	Δ Shank BMD	Δ Lumbar BMD
Age	.035	-.201	-.564*	-.004	-.094	-.368	-.102	-.514*	.097
Δ Body Mass	-.870**	-.188	.215	-.343	-.454	.350	-.418	-.410	-.408
Δ BMI	-.903**	-.212	.177	-.379	-.498*	.309	-.446	-.410	-.468
Δ Body Fat %	-.565*	.017	.251	-.037	-.120	.265	-.240	-.102	-.510*
Δ Lean Mass %	.700**	.218	-.179	.120	.237	-.229	.274	.216	.508*
Δ Kcal	.085	-.181	-.161	.160	.094	.166	.253	-.273	.024
Δ Vitamin D	-.503	.038	-.437	-.419	-.570*	.101	-.002	-.117	-.268
Δ Calcium	-.078	-.212	-.382	-.014	-.049	-.271	.141	-.375	-.287

*. Correlation is significant at the 0.05 level (2-tailed).
**. Correlation is significant at the 0.01 level (2-tailed).
BMI=Body Mass Index (kg/m²)
WB BMC%= Whole Body Bone Mineral Content % body mass
LB BMCg= Lower Body Bone Mineral Content in grams
UB BMCg= Upper Body Bone Mineral Content in grams
BMD= Bone Mineral Density

A. I. 14 Multiple Bicycle correlations with all variables as they changed over time Δ.

Variable	Δ WB BMC%	Δ LB BMCg	Δ UB BMCg	Δ T score	Δ Z score	Δ Pelvis BMD	Δ Thigh BMD	Δ Shank BMD	Δ Lumbar BMD
Age	.136	.295	.042	.242	.241	.047	-.034	.503**	.181
Δ Body Mass	-.696**	.476*	-.355	.110	.018	.435*	-.062	.127	.370
Δ BMI	-.710**	.421*	-.315	.050	-.046	.415*	-.042	.032	.345
Δ Body Fat %	-.630**	.002	-.241	.055	-.001	.493*	.073	-.288	.377
Δ Lean Mass %	.597**	.004	.195	-.052	.004	-.457*	-.056	.272	-.364
Δ Kcal	-.085	.274	-.140	.091	.071	-.085	.448*	.270	-.370
Δ Vitamin D	-.004	.131	-.431	-.078	-.039	.106	.151	-.046	-.511*
Δ Calcium	.278	-.042	.160	-.258	-.206	-.133	.288	-.153	-.568**

*. Correlation is significant at the 0.05 level (2-tailed).
**. Correlation is significant at the 0.01 level (2-tailed).
BMI=Body Mass Index (kg/m²)
WB BMC%= Whole Body Bone Mineral Content % body mass
LB BMCg= Lower Body Bone Mineral Content in grams
UB BMCg= Upper Body Bone Mineral Content in grams
BMD= Bone Mineral Density

APPENDIX VII

BONE DENSITY IN COMPETITIVE CYCLISTS: A LONGITUDINAL ASSESSMENT ACROSS THE SEASON

STEPWISE LINEAR REGRESSION TABLES

A. VII. 1 Pre-season stepwise linear regression with all variables for the whole group.

Variable	WB BMC% (N=34)		LB BMCg (N=33)		UB BMCg (N=34)		T Score (N=34)		Z Score (N=34)		Pelvis BMD (N=32)		Thigh BMD (N=34)		Shank BMD (N=34)		Lumbar BMD (N=34)	
Rank	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value
1	Body Fat %	0.361	Body Mass	0.763	Body Mass	0.626	BMI	0.348					Body Mass	0.532			Body Fat %	0.367
2			Body Fat %	0.811									Cat.	0.608				

A. VII. 2 Whole group stepwise linear regressions with all variables as they changed over time Δ.

Variable	Δ WB BMC% (N=31)		Δ LB BMCg (N=30)		Δ UB BMCg (N=29)		Δ T Score (N=31)		Δ Z Score (N=31)		Δ Pelvis BMD (N=29)		Δ Thigh BMD (N=31		Δ Shank BMD (N=30)		Lumbar BMD (N=3	
Rank	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value
1	Δ BMI	0.880			Δ Vitmain D	0.419	Δ BMI	0.360	Δ BMI	0.461			Δ Kcal	0.369	Δ BMI	0.406	Δ Calcium	0.378
2													Δ Bike	0.521	Δ Bike	0.536		

A. VII. 3 Pre-season stepwise linear regression with all variables for females.

Variable	WB BMC% (N=17)		LB BMCg (N=17)		UB BMCg (N=17)		T Score (N=17)		Z Score (N=17)		Pelvis BMD (N=15)		Thigh BMD (N=17)		Shank BMD (N=17)		Lumbar BMD (N=17)	
Rank	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value
1	Cat.	0.601	BMI	0.571									BMI	0.532	BMI	0.508	Kcal	0.590
2													Cat.	0.747	Lean Mass %	0.693		
3													Lean Mass %	0.857				
4													Yrs. Exp.	0.910				

A. VII. 4 Females stepwise linear regressions with all variables as they changed over time Δ.

Variable	Δ WB BMC% (N=15)		Δ LB BMCg (N=15)		Δ UB BMCg (N=13)		Δ T Score (N=15)		Δ Z Score (N=15)		Δ Pelvis BMD (N=13)		Δ Thigh BMD (N=15		Δ Shank BMD (N=15)		Lumbar BMD (N=1	
Rank	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value
1	Δ Body Mass	0.912			Δ Vitamin D	0.684			Δ BMI	0.525			Δ Cat.	0.562				
2									Δ Body Mass	0.784								

A. VII. 5 Pre-season stepwise linear regression with all variables for males.

Variable	WB BMC% (N=17)		LB BMCg (N=16)		UB BMCg (N=17)		T Score (N=17)		Z Score (N=17)		Pelvis BMD (N=17)		Thigh BMD (N=17)		Shank BMD (N=17)		Lumbar BMD (N=17)	
Rank	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value
1			Body Mass	0.598	Body Mass	0.734	Body Mass	0.539	Body Mass	0.529			Body Mass	0.563				

A. VII. 6 Males stepwise linear regressions with all variables as they changed over time Δ.

Variable	Δ WB BMC% (N=16)		Δ LB BMCg (N=15)		Δ UB BMCg (N=16)		Δ T Score (N=16)		Δ Z Score (N=16)		Δ Pelvis BMD (N=16)		Δ Thigh BMD (N=16		Δ Shank BMD (N=15)		Lumbar BMD (N=1	
Rank	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value
1	Δ BMI	0.889					Age	0.524			Δ Fat Mass %	0.544			Δ Lean Mass %	0.541		
2											Δ Kcal	0.783						

A. VII. 7 Pre-season stepwise linear regression with all variables for Cat.1 racers.

Variable	WB BMC% (N=16)		LB BMCg (N=16)		UB BMCg (N=16)		T Score (N=16)		Z Score (N=16)		Pelvis BMD (N=15)		Thigh BMD (N=16)		Shank BMD (N=16)		Lumbar BMD (N=16)	
Rank	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value
1	Lean Mass %	0.700	Sex	0.800	Sex	0.788	Yrs. Racing	0.597	Yrs. Racing	0.549			Body Mass	0.534	Sex	0.615		
2	Yrs. Racing	0.834			Bike	0.085									Yrs. Racing	0.741		
3					Yrs. Racing	0.941												

A. VII. 8 Cat. 1 racer's stepwise linear regressions with all variables as they changed over time Δ.

Variable	Δ WB BMC% (N=14)		Δ LB BMCg (N=14)		Δ UB BMCg (N=14)		Δ T Score (N=14)		Δ Z Score (N=14)		Δ Pelvis BMD (N=13)		Δ Thigh BMD (N=14		Δ Shank BMD (N=14)		Lumbar BMD (N=14	
Rank	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value
1	Δ Body Mass	0.877			Δ Vitamin D	0.653	Age	0.588			Δ Kcal	0.576			Δ BMI	0.772	Δ Calcium	0.617
2	Age	0.919									Δ BMI	0.804						

A. VII. 9 Pre-season stepwise linear regression with all variables for Cat.4 racers.

Variable	WB BMC% (N=12)		LB BMCg (N=12)		UB BMCg (N=12)		T Score (N=12)		Z Score (N=12)		Pelvis BMD (N=11)		Thigh BMD (N=12)		Shank BMD (N=12)		Lumbar BMD (N=16)	
Rank	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value
1			Body Mass	0.799			Lean Mass %	0.654	Lean Mass %	0.603			BMI	0.724	BMI	0.644	Yrs. Racing	0.731
2							Fat Mass %	0.863	Fat Mass %	0.864			Yrs. Exp.	0.856				
3							BMI	0.946	BMI	0.934			Bike	0.934				

A. VII. 10 Cat. 4 racer's stepwise linear regressions with all variables as they changed over time Δ.

Variable	Δ WB BMC% (N=11)		Δ LB BMCg (N=11)		Δ UB BMCg (N=9)		Δ T Score (N=11)		Δ Z Score (N=11)		Δ Pelvis BMD (N=10)		Δ Thigh BMD (N=11		Δ Shank BMD (N=11)		Lumbar BMD (N=1	
Rank	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value
1	Δ Body Mass	0.950			Δ Vitamin D	0.676			Δ BMI	0.666					Δ Bike	0.628		
2															Sex	0.846		

A. VII. 11 Pre-season stepwise linear regression with all variables for Road Only racers.

Variable	WB BMC% (N=13)		LB BMCg (N=12)		UB BMCg (N=13)		T Score (N=13)		Z Score (N=13)		Pelvis BMD (N=13)		Thigh BMD (N=13)		Shank BMD (N=13)		Lumbar BMD (N=13)	
Rank	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value
1			Sex	0.662	Age	0.712	Age	0.565					Age	0.670			Calcium	0.717
2					Lean Mass %	0.872												

A. VII. 12 Road Only racer's stepwise linear regressions with all variables as they changed over time Δ.

Variable	Δ WB BMC% (N=14)		Δ LB BMCg (N=13)		Δ UB BMCg (N=12)		Δ T Score (N=14)		Δ Z Score (N=14)		Δ Pelvis BMD (N=14)		Δ Thigh BMD (N=14		Δ Shank BMD (N=13)		Lumbar BMD (N=1	
Rank	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value
1	Δ BMI	0.904			Age	0.774			Δ Vitamin D	0.599								
2	Δ Body Mass	0.937																

A. VII. 13 Pre-season stepwise linear regression with all variables for Mixed Bike racers.

Variable	WB BMC% (N=21)		LB BMCg (N=21)		UB BMCg (N=21)		T Score (N=21)		Z Score (N=21)		Pelvis BMD (N=19)		Thigh BMD (N=21)		Shank BMD (N=21)		Lumbar BMD (N=21)	
Rank	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value
1	Fat Mass %	0.503	Body Mass	0.852	Body Mass	0.755							Body Mass	0.652	Fat Mass %	0.572		
2	Lean Mass %	0.774											Yrs. Racing	0.827	BMI	0.690		

A. VII. 14 Mixed Bike racer's stepwise linear regressions with all variables as they changed over time Δ.

Variable	Δ WB BMC% (N=17)		Δ LB BMCg (N=17)		Δ UB BMCg (N=17)		Δ T Score (N=17)		Δ Z Score (N=17)		Δ Pelvis BMD (N=15)		Δ Thigh BMD (N=17		Δ Shank BMD (N=17)		Lumbar BMD (N=17	
Rank	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value	Predictor	R Value
1	Δ Body Mass	0.780									Δ Kcal	0.520	Δ Kcal	0.492	Δ BMI	0.629	Cat.	0.497
2																	Δ Kcal	0.709