Abstract

THE EFFECTS OF A 16 WEEK PHYSICAL ACTIVITY PROGRAM ON BONE MINERAL DENSITY IN LEAN AND OBESE PREPUBESCENT CHILDREN AGES 8-11.

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The current obesity epidemic has become a major health crisis to citizens around the United States. Obesity has been successfully linked to a plethora of different disease states including cardiovascular disease, diabetes, and stroke. Although many people might not recognize the trends, the obesity epidemic is putting children as young as 5 and 6 years of age at a health disadvantage compared to children of a healthier weight. With 17% of American’s children being classified as obese, it is important to uncover what detrimental influences obesity has on the childhood body. Specifically, with respect to bone health, there is a good amount of information supporting the concept that there is increased acquisition of bone mineral density (BMD) through exercise in children. However, none of these studies have investigated the effects of physical exercise on BMD in obese children compared to lean children. Previous studies show that overweight and obese children are at an increased risk of bone fracture due to low bone mass and bone area for weight. An increased response in BMD or bone mineral content (BMC) of obese children due to exercise could improve bone strength and decrease
fracture risk. The purpose of this study was to investigate whether there were differences in BMD acquisition in lean and obese children in response to a 16-week exercise intervention.

Dual energy X-absorptiometry (DEXA) was used to assess the change in BMD in children at week 0 and 16. Participants were grouped by body mass index percentile as obese (n=41) or lean (n=19) and then randomly assigned to exercise or control groups. The exercise protocol consisted of aerobic activities such as running, basketball, tennis, football, etc. Participants were required to meet a heart rate average of >140 beats per minute each one-hour exercise session. The protocol for the study was reviewed and approved by the East Carolina University Institutional Review Board. Both lean and obese exercise groups increased total body bone mineral density (+0.026±0.001g/cm², +0.028±0.001g/cm² respectively; p<0.05) from week 0 to 16. Total body BMC also increased in the lean and obese (+111± 4.4g, +106±4.3g respectively; p<0.05) exercise groups. There were no significant increases in BMD in controls groups. The increases in BMD and BMC of the exercise groups, suggests that bone metabolism responded similarly in both treatment groups. The results advocate that there is no difference in bone acquisition of lean and obese prepubescent children of different BMI percentiles.
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Chapter 1: Introduction

With recent budget cuts across the nation, schools have been forced to cut back on resource classes such as physical education, in order to save money. The elimination of physical education classes has a detrimental effect on the growing childhood obesity population; depriving children of physical activity over an eight hour day could have an additive negative effect on children’s ability to grow strong and healthy bones. Incidence of bone fracture is remarkably high in childhood as well as in adolescence. For this reason attaining peak bone mass in childhood is very important in prevention of fractures, as well as other bone diseases throughout life. It is theorized that every individual has a brief window during childhood, specifically puberty, to reach peak bone mass. Bone fractures in childhood can add to the healthcare cost of families so it is important for families and healthcare providers to understand what type of childhood exercise can be beneficial to build bone mass in children.

Peak bone mass can provide additive protection from diseases such as osteoporosis, and osteopenia, which normally arise at older ages (Kanis, John 1994; Rutherford, M. 1999). Recently it has been reported that the incidence of fracture has increased 30 percent in adolescent populations over the last 30 years (Kanis, John A. 1994). Though it is not completely understood as to what sparked such a dramatic increase in fractures among this specific population, it is certain that there is an increased incidence of obesity in children, and decreased activity patterns over the same 30-year time span (Ogden CL 2006; Ogden CL 2008). It is paramount to understand the mechanisms that drive the body to create bone so that proper preventive steps can be followed in order to reduce the risk of costly bone fractures and disease’s.
Hormonal factors, diet, and exercise have all been proven to affect biological processes that can manipulate bone metabolism. Many studies have been designed to investigate how these different factors manipulate bone development (L. H. Foo 2008; Bass, S. 1998). With regards to hormonal influences, it is unclear the exact mechanism to which hormones can increase bone density. Hormones such as parathyroid hormone, growth hormone, and vitamin D can contribute to osteoblast and osteoclast activity in the bones; however, more knowledge is needed before adequate recommendations can be made regarding the manipulation of these hormones to gain peak mass. Load bearing exercises, along with a diet supplemented with calcium have both been proven to increase bone mineral density. Load bearing activities such as running and jumping as few as 3 days a week have been shown to increase bone mineral density in boys and girls (Hind, K. 2007; MacKelvie, K.J. 2002).

Load bearing activities can have a positive influence on bone mineral density, however it is not as clear at what age bone mineral density can increase most readily. There is also some debate on whether children with more mass can derive skeletal benefits from constant weight bearing compared to individuals with less mass. Prior studies have shown that obese children have increased bone mass compared to healthy normal children (Leonard, Mary B. 2004; Manzoni, P. 1996;), while others suggest a low bone mass to body weight ratio exists in obese children (Goulding, A. 2000, Goulding, A. 2001). Goulding et al believed that if obese individuals display a low bone mass to body weight ratio, which would indicate structural instability. This instability would lead to a greater incidence of bone fracture because these children would be
carrying more loads compared to lean children. With conflicting evidence, there is still uncertainty as to if obese children have stronger bones compared to lean children.

The prepubertal stage of life represents a period of time where sex hormones such as testosterone and estrogen are found in lower concentrations than during and after puberty. The lack of steroids has led many investigators to believe that children are unable to produce adequate gains in bone density during the prepubertal years, whereas increases in bone mineralization during puberty can be far more superior. Sex hormones such as testosterone and estrogen can have an anabolic effect towards skeletal growth in children going through puberty (Behre, Hermann M. 1997). Growth hormone, Insulin like growth factor, parathyroid hormone, and vitamin D, are believed to contribute greatly to increases in bone mineral density in those children who have yet to reach puberty (Bass, S. 1998).

Purpose

Current literature lacks comprehensive data on how exercise can change bone mineral density in lean and obese prepubescent children. A study of exercise training, specifically in lean and obese children, could give a unique opportunity to understand how everyday activities, such as basketball, football, running, etc. can influence bone metabolism in children of two different weight categories. The current study examined the effects of a 16-week physical activity intervention program on bone mineral density in lean and obese prepubescent children ages 8-11. Furthermore, a sedentary lean and obese control group was used to determine if exercise had an effect.
Hypotheses

Based on the current and previous literature as well as pilot data seen in Figure 1 and Figure 2 below, I hypothesized that: bone mineral density would increase in the prepubescent lean children involved with the exercise intervention group when compared to the lean sedentary control group. Likewise, the prepubescent obese group would increase bone mineral density regardless of group.
Pilot data obtained from ongoing research suggested that a lean 16 week exercise (n=10) group had a greater gain in total body bone mineral density, when compared to a lean, sedentary, 16 week control (n=7) group.
Pilot Figure 2:

Change In Total Body BMD

Figure 2: Pilot data obtained from obese 16 week exercise (Group 1, n=15) and non-exercise (Group 2, n=8) groups suggested that both groups increase BMD values regardless of exercise.

Aim of the research:

1. To determine if there was an increase in total body bone mineral density and bone mineral content due to a 16 week exercise program in lean and obese prepubescent children. To accomplish this, bone mineral density was scanned using dual energy X-ray Absorptiometry before and after 16 weeks of either the exercise intervention (treatment groups) or a corresponding 16 weeks without intervention (control group).

Sub-Aims (Confounders of Bone Health in Children)
Sub aims were used to report supplemental data known to influence bone metabolism in prepubescent children. These sub-aims specifically report activity and dietary data collected from all participants.

1. Activity (steps) was monitored and reported as a 3-day step count average for all groups at weeks 0, 4, 8, 12, and 16. To accomplish this, activity monitors were worn by all participants every four weeks over the 16 week intervention.

2. Dietary information was monitored to determine if children were reaching recommended dietary allowances (RDA) for calcium (1300mg), phosphorus (1250mg), and adequate intake (AI) for vitamin D (5ug). This was accomplished by entering 3-day food records into a nutritional analysis program every four weeks over the 16 week intervention.

Limitations:

Limitations of the current study could include, but are not limited to, the following: 1. when recruiting potential subjects, physical activity is a major determinant of the child’s acceptance into the program. Being that only sedentary children are recruited, it is possible that the parents misconstrue the amount of physical activity the child actually gets so that they will be able to participate. 2. Without a controlled diet during the 16-week period, some participants could be receiving a greater amount of calcium and vitamin D, this ultimately could influence bone metabolism. 3. Limitation exists with the Dual energy X-ray absorptiometry when taking scans on children. Bone mineral density is often overestimated in smaller individuals.
Delimitations:

Delimitations found in the design of the current study include, but are not limited to, the following: 1. Only African American and Caucasian children are allowed to be participants. 2. Subjects involved with the exercise group are either classified as being lean or obese according to the CDC growth charts. 3. Recommendations generated from results are specific to children 8-11 years of age.
Chapter 2: Literature Review

Bone Mineral Density and Physical Activity.

Bone mineral density has been shown to increase through exercise, regardless of age and gender. While there is some scrutiny on the exact mechanism of how this increase occurs, there is evidence that a positive correlation does exist between the two factors. As shown throughout numerous experimental studies, increases of bone mineral density can be maximized through load bearing activities such as resistance training. (Bass, S. 1998; Hind, K. 2007). Although load bearing activities seem to be the “gold standard” with respect to bone mass increases, there is evidence that aerobic activities such as walking, running and school physical education programs designed for children can prevent bone loss, and even increase the body’s ability to synthesize bone at specific sites (MacKelvie, K J. 2002; MacKelvie, Kerry J. 2004). A few studies have been conducted to examine the effects of long term, longitudinal physical activity programs on bone mass (Bailey, D.A. 1999). While these particular studies provide good evidence of the effects of physical activity on bone density, it is still unclear as to what intensity an aerobic exercise protocol must be in order to entice the body to create more bone. Another key contributor to the mineralization of bone with regards to exercise is hormonal factors. Hormones such as growth hormone (GH), parathyroid hormone (PTH), insulin-like growth factor (IGF), and vitamin D all play an important role in the bone creation process. It has been observed that sedentary individuals can increase circulating levels of particular hormones (PTH) by simply exercising for 3 days, 30- 40 minutes a day at 50% of their VO$_{2}$max (Thorsen K 1997).
Age and Bone Mineral Density

Peak bone mass is very important to achieve with regards to preventing any type of bone fractures throughout life. Although with some controversy it has been suggested that children begin to build most of their bone mass by the age of 8 years old, and continue through adolescence. Many researchers believe that if a high bone density can be obtained successfully in younger years, it is likely to have a protective effect on bone fracture rate, and bone diseases such as Osteoporosis and Osteopenia (Kanis, John A. 1994; Rutherford, O M. 1999; Thorsen K 1997). While there is hard evidence that physical activity can increase bone mineral density in children involved with weight bearing physical activities, such as the ones provided in the physical education classrooms in school, the researchers debate at what age bone can most readily be manipulated to receive maximal bone density from such activities. The debate stems from the fact that young children, that have yet to reach puberty, don’t have sufficient ability to synthesize hormones needed to properly build bone. It is believed by some researchers that puberty marks the stages of increase bone accrual, for the simple fact that levels of hormones specific to bone metabolism increase at puberty, and this allows the bones to calcify more readily. It has also been shown that growing children have a better ability to absorb calcium through dietary means when compared to adults (Sareen G. 2007). This higher calcium absorption in children than adults could possibly explain why peak bone mass can be obtained throughout growing years.

Calcium
From muscle contractions to blood clotting calcium serves an important function in various biological systems throughout the body. Calcium is very important in children when it comes to their ability to build strong bones. If blood calcium levels are low in children the body will use calcium stores (bone) in order to meet its own needs, thus decreasing the amount of calcification that can occur in the bones. With this in mind researchers have been trying to determine what variables can potentially increase calcium levels in the blood, while at the same time increasing bone absorption of this extra calcium. Exercise has long been associated with its influence to help facilitate movement of calcium into the bone for absorption. Load bearing activities seem to gain most of the recognition for maximal increases in bone mineral density. While it is good to have load bearing types of activities for adults, who have already attained maximal bone growth, some investigators are worried about the negative effects improperly performed load bearing activities can have on growing children. In order to eliminate the possibility of injury in children due to load bearing activities it has been shown that increases in bone mineral density can be attained in children performing daily aerobic exercise routines that incorporate some level of low impact load bearing activity such as jumping. Studies have shown increases of BMD from school physical education programs that have implemented aerobic jumping routines into their curriculum (Hind, K. 2007; MacKelvie, Kerry J. 2004). Other studies have tried to detect how blood biomarkers such as calcium and parathyroid hormone change in the presence of different types of exercise protocols, and exercise intensities. From these studies it has been shown that serum calcium levels can increase slightly during aerobic exercise in road cyclists, while parathyroid hormones increase significantly.
Tanner Stage and Bone Mineral Density

Not all children grow and physically develop at the same rate, thus limitations exist in any study designed to understand what effect maturation has on children. In order to successfully categorize children the Tanner scale of physical development is used. This scale is useful when grouping males and females into similar groups according to sex specific traits. The Tanner scale is a way to classify what stage of puberty a particular participant falls into, this can sometimes be misinterpreted due to the ambiguity of measurement methods. With regards to bone mineral density, the Tanner scale can be useful when determining a participant’s stage of puberty.

Obesity and Bone Mineral Density

Childhood obesity carries an increased risk of contracting diseases such as diabetes, dyslipidemia, and hypertension. Obesity can promote a cascade of altered biological processes, as seen with Type 2 diabetes, so the likelihood that obesity can have a negative impact on bone formation cannot be ignored. It has been shown that overweight and obese children have lower bone mineral content and bone area when compared to their normal weight peers (Goulding, A. 2000). It has been theorized that adipocyte’s can collect within the bone alongside bone marrow in overweight and obese individuals. This increase in adipocytes within the bone can prevent osteoblast production, and without proper osteoblast production, bone can become weak and thin. Findings such as these could suggest that obese children are at a metabolic disadvantage in their ability to reach their maximum potential at forming bone. Obese individuals seem to have a decreased ability to synthesize some hormones. Specifically...
growth hormone, and insulin-like growth factor I, have been shown to be decreased in obese individuals when compared to lean counterparts. Kanaley et al. has shown that growth hormone was suppressed in obese individuals during aerobic exercise, while non-obese participants could significantly increase growth hormone secretion during an aerobic bout of exercise (Kanaley J A, 1997). Results such as these continue to suggest that obese individuals are structurally limited with regards to bone integrity given the data that supports growth hormones positive effect on bone formation. Suppression of hormones such as growth hormone, in obese children, could be one reason why an obese population could be at risk of not reaching maximal bone mineralization.

**Hormonal Influences on Bone Mineral Density**

Several studies have investigated how different hormonal biomarkers can be affected by physical activity. Vitamin D, also known as cacitriol, 1, 25-hydroxyvitamin D (25[OH] D), in its active form is a major contributor to bone metabolism. When in the blood, cacitriol works with parathyroid hormone to control serum calcium levels. If concentrations of calcium are low in the blood, parathyroid hormone is activated and signals are sent to the kidneys and intestinal cells to begin the process of absorbing calcium from the urine or food, cacitriol absorbs the food from the digestive track. If blood calcium concentrations are found to be too high, calcitonin, another hormone that regulates bone metabolism, is secreted from parafollicular cells of the thyroid to aid in the mineralization of calcium into the bones. Vitamin D, calcitonin, and parathyroid hormone act on different bone cells that can build bone or break down bone such as osteoblasts and osteoclasts. Primarily these hormones are used when calcium is found
in low or high concentration. Osteoblasts are bone cells that increase the mineralization process, Osteoclast’s are therefore the opposite in that they aid in the breakdown of bone so that calcium can be used for other bodily processes in times of inadequate supplementation.

**Parathyroid Hormone and Bone**

Parathyroid hormone (PTH) is secreted by the parathyroid gland, and is of large importance to bone building processes found throughout the body. PTH has influence on the kidneys and the bones; it is a major regulator of calcium concentrations throughout the body. When the concentrations of calcium decrease in extracellular fluid, PTH will send signals to the bone, and the kidneys to increase bodily resorption of calcium. Bone resorption occurs by increasing activation of osteoclasts found in the bone, which in turn aid in breaking down calcium stores and releasing this calcium into the extracellular fluid so that concentrations can return to normal levels. In the kidneys, PTH works to retain calcium via the kidney tubules. Once signaling begins, the kidney acts to prevent calcium from being lost during urination, thus the body can actively recycle its own calcium. It is hypothesized that PTH can act directly on local growth factors found in the bone. Insulin-like growth factor is thought to work directly with PTH to stimulate bone formation, although the exact mechanism for this process is still being assessed (Wuster C 1993; Yakar S, 2002). Studies involved with parathyroid supplementation have successfully determined that supplementation can increase bone mineral density of the spine, hip and total-body bone mineral.

**Growth Hormone and Bone Metabolism**
Growth hormone is secreted from the pituitary gland, and is instrumental in the bodies’ ability to stimulate growth and cellular reproduction; in its natural form, growth hormone is also referred to as somatotropin. Due to its anabolic properties, growth hormone is believed to create a signaling cascade involving insulin-like growth factor (IGF-1) to increase osteoblast activity which in turn can increase bone growth. While this process is a secondary, or indirect effect of growth hormone, its secretion is needed in order to fully accommodate bodily processes that use growth hormone for stimulation. Growth hormone secretion can be influenced by a variety of different factors. Stress, nutrition, exercise and sleep can all have an influence on the secretion patterns of this hormone. Growth hormone is secreted from the anterior pituitary gland; it is regulated primarily by hormones that are secreted from the hypothalamus and the stomach. Using a negative feedback loop with IGF-1, growth hormone is signaled to be secreted when blood levels of IGF-1 fall. Once growth hormone increases in the blood, the liver will begin to process more IGF-1 to be circulated to target tissues. It has been seen that obese individuals have lower secretion rates of growth hormone compared to lean peers (Scacchi, M. 1999). Using mathematical formulas, Iranmanesh et al. determined that GH secretion rate can be decreased by 6% for every body mass index (BMI) unit increase. Exercise training bouts have been shown to increase growth hormone secretion in non-obese individuals when compared to obese individuals (Iranmanesh A. 1991). This decrease in release of GH could be one of the factors that limit obese children from being able to increase bone mineral density, when compared to lean children.
IGF-1 and Bone Metabolism

Insulin-like growth factor 1 has been recognized as a facilitator for stimulation and growth of many cellular processes. Increase in the size of muscle and bone are directly related to increased levels of IGF-1. Produced in the liver, IGF-1 helps to regulate the amount of growth hormone found in the bloodstream, if GH is being inhibited due to some metabolic abnormality, such as found in obese individuals, it is likely that IGF-1 will be found in minimal concentrations due to the impaired releasing mechanism. Studies on osteoporotic patients have determined that lower concentrations of IGF-1 can play a big part in the weakening of bones. Wuster et al. showed that IGF concentrations were decreased in patients suffering from osteoporosis (WÜSTER, C. 1993). In prepubescent children IGF-1 plays an important role in facilitating longitudinal growth. Dwarfism can result in the case where an individual cannot biologically create ample amounts of IGF (Laron syndrome). It has also been discovered that knockout mice, which have been deprived of the IGF-1 gene have reduced skeletal development as well as reduced growth patterns (Yakar S 2002). It is certain that IGF-1 plays an important role in regulating cellular processes; it is less certain how an intervention such as exercise could inhibit or increase concentrations in children. As seen in previous studies it is clear that GH can be increased through exercise, this increase could indirectly facilitate increases of IGF-1, but lab testing would be needed to confirm this suspicion.

3 Day Food Records

Self-reported food records have been determined to be an accurate and inexpensive method to assess nutrient intake. With regards to children, these records have been shown to
be a very good measure of nutritional status (Domel, S B 1994). Children as young as eight years old have been reported to maintain accurate food records over a 3 day period (Crawford 1994; Livingstone 2000).

**Calcium Intake**

The increase in soft drink usage among all populations has created concern for whether children and adults are consuming enough calcium products such as milk in their daily diets. Fleming et al. reported that a large proportion of the U.S. population is receiving substantially less calcium than what RDA requirements have suggested (Fleming, Heimbach 1994). Current NHANES III data has supplemented those claims while investigating the trends of dietary intake in the United States. It was reported that in the year 2000 only 30% of population ages 2 and older were receiving the recommended amount of calcium (Briefel, Johnson 2004).

**Phosphorus Intake**

Phosphorus intake in children has stayed about the same among the United States population over the past 30 years (Enns, C.W 2002). Phosphorus in the diet can be obtained from a variety of sources including milk, yogurt, cheese and tuna. With the consumption of milk down over the past 10 years, it has been shown that soft drinks are providing a good majority of the dietary phosphorus being consumed in the United States. In fact soft drink consumption has increased 48% from the time span of 1978-1998 (French 2003). This dramatic increase in soft drink consumption supports the idea that soft drink consumption has surpassed consumption of milk.
Vitamin D Intake

The adequate intake for vitamin D is set at 5 international units per day. According to nationally represented data such as the Third National Health and Nutrition Examination Survey, and the Continuing Survey of Food Intakes report that vitamin D dietary intake has stayed within recommended levels over the past thirty years (Moore 2004). It is believed that fortification of foods such as cereal, breads, and milk help children reach the adequate levels.
Chapter 3 Methods

Methods

The data from this study was obtained in cooperation with a current ongoing study being conducted at East Carolina University. All methods are approved by the East Carolina University Institute Review Board.

Participants

Sixty healthy children ages 8-11 were recruited from the Pitt County School Systems. A flyer was sent to the schools and handed out to grades 3, 4, and 5. Participants were screened over the phone with a questionnaire to ensure that they are currently sedentary and not involved in any type regular physical activity (i.e. gymnastics, football, basketball). Participants were excluded from the study if they fell between the 80th-95th BMI percentiles for age, if they were too physically active, or if they were currently taking medications that could affect central or peripheral circulation. Any participant who had prior medical history including diabetes, hypertension, congestive heart failure, angina, or peripheral vascular disease were also excluded from the study. A Tanner Stage questionnaire was used in order to determine if the children were prepubescent, Tanner stage 1 or 2. All participants were randomly assigned to either exercise or a sedentary control group. Random assignment was determined using a computer-based random number generator. Participants assigned to the experimental group exercised for a 16-week period. Participants assigned to the non-exercise group, followed the same testing protocol.
Procedures

Following recruitment, an initial meeting was scheduled with both the child and the parent. During this initial meeting, parameters of the study and any risks that might be involved with participation were explained to the participants. The child and parent both signed a consent and child assent before beginning any participation into the study. A medical history form was given to the parents to fill out in order to screen for any medications, or illnesses the child has experienced. Initial testing of body composition and VO2 performance were assessed in two later meetings. Exercise and non-exercise groups performed activities according to their respective groups. After the sixteen week period a final body composition was performed.

Training Protocol

Over the 16 week period participants exercised for three days a week for an hour each session. An average heart rate minimum of 140 beats per minute was obtained for each one hour exercise session. Exercise was performed on the campus of East Carolina University. All exercise was supervised by either a graduate student or exercise physiology undergraduate student. Activities were based on preference of the participant; aerobic activities such as running, basketball, tennis, football, etc. will be used to maintain proper heart rate. The heart rate of >140 beats per minute, was set from a 65% of heart rate max.

Testing
At the beginning of the study all participants were tested via a DEXA scan in order to obtain total bone mineral content values. After 16 weeks of intervention, all participants perform another DEXA scan.

**Activity Monitoring**

Activity patterns were measured at 4-week intervals throughout the 16-week study using a Yamax pedometer (Yamax, Japan) and a CSA accelerometer (Computer Science Applications, Inc.). Both groups received activity monitors 5 days prior to beginning of any intervention. These accelerometers and pedometers were worn for three week days (i.e. Wednesday, Thursday, and Friday) as well as for the weekend prior to starting intervention. In order to track activity during the 16 week program, children took home accelerometers and pedometers every four weeks (week 4, 8, 12); at this time children wore accelerometers and pedometers for 3 days (a program, non-program, and weekend day). On week 16 children again took home an accelerometer and pedometer for a final time, in which they wore it for five days, as performed on their pre-intervention week.

**Nutritional Monitoring**

A diet recall was used to track calcium intake levels among participants. When children picked up activity monitors they also took home a 5-day (pre and post weeks), or 3-day (weeks 4, 8, 12) diet log. Children recorded everything they ate during these days. The days recorded corresponded with the day’s activity monitors were worn. The children were given examples of how to complete dietary logs and were shown how to record proper measurements of food
intake. Parents were also shown all dietary information and were tutored on the importance of measurement collection. Upon completing diet logs and activity monitoring, records were reviewed with the child and parent to ensure greater accuracy. Nutritionist Pro was used for analysis of dietary intakes.

Statistics

A student T-test will be used to compare the means for BMD and BMC of lean treatment, lean control, obese treatment, and obese control from pretest to post test. 2 way repeated measures ANOVA will be used to test the difference between the groups (lean versus obese treatment) with respect to BMD and BMC values at pre and post testing. Significance will be accepted at the p<0.05.
Chapter 4: Results

Descriptive characteristics of both obese and lean participants are shown in table 1 as the mean plus or minus the standard deviation. The samples included in this study include 11 lean treatment participants, 8 lean control participants, 29 obese treatment participants, and 12 obese control participants. Unless otherwise noted all graphs are reported as the mean ± SEE.

Table 1: Descriptive Statistics (All Subjects)

<table>
<thead>
<tr>
<th></th>
<th>Lean Treatment (n=11)</th>
<th>Lean Control (n=8)</th>
<th>Obese Treatment (n=29)</th>
<th>Obese Control (n=12)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>9.0 ± 1.0</td>
<td>9.4 ± 1.2</td>
<td>9.7 ± 1.1</td>
<td>10.0 ± 1.1</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>35.4 ± 5.9</td>
<td>34.6 ± 7.8</td>
<td>58.7 ± 11.8</td>
<td>54.0 ± 12.3</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>144.7 ± 8.8</td>
<td>144.3 ± 11.4</td>
<td>147.8 ± 7.0</td>
<td>146.0 ±6.4</td>
</tr>
<tr>
<td>BMI %</td>
<td>51.9 ± 21.5</td>
<td>46.1 ± 24.3</td>
<td>97.0 ± 0.3</td>
<td>96.0 ± 2.3</td>
</tr>
</tbody>
</table>

Note: Children >95th percentile according to BMI are classified as obese. Children <80th percentile are classified as lean. Reported as mean plus or minus the standard deviation.

Bone Mineral Density

All participants performed a DEXA scan in the beginning and at the end of 16 weeks.

Table 2 shows descriptive data of bone mineral density (g/cm²) of obese participants. Table 2b. includes descriptive data of bone mineral density (g/cm²) of lean participants.
Table 2a. Bone Mineral Density (Obese)

<table>
<thead>
<tr>
<th>DEXA Scan</th>
<th>Obese Treatment</th>
<th>Percent Change</th>
<th>Obese Control</th>
<th>Percent Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>TB (Pre/Post)</td>
<td>0.983±0.01</td>
<td>1.01±0.15*</td>
<td>+2.67%</td>
<td>0.968±0.02</td>
</tr>
<tr>
<td>Arms (Pre/Post)</td>
<td>0.734±0.01</td>
<td>0.752±0.01</td>
<td>+2.39%</td>
<td>0.730±0.02</td>
</tr>
<tr>
<td>Legs (Pre/Post)</td>
<td>1.071±0.02</td>
<td>1.097±0.02*</td>
<td>+2.37%</td>
<td>1.075±0.04</td>
</tr>
<tr>
<td>Pelvis (Pre/Post)</td>
<td>0.993±0.03</td>
<td>1.023±0.02*</td>
<td>+3.02%</td>
<td>0.975±0.03</td>
</tr>
<tr>
<td>Spine (Pre/Post)</td>
<td>0.846±0.02</td>
<td>0.862±0.02*</td>
<td>+1.86%</td>
<td>0.823±0.03</td>
</tr>
</tbody>
</table>

Note: Bone mineral density in (g/cm²) scan pre to post testing; * P<0.05 pre vs pos; Reported as the mean ± SEE

Table 2b.: Bone Mineral Density (Lean)

<table>
<thead>
<tr>
<th>DEXA Scan</th>
<th>Lean Treatment</th>
<th>Percent Change</th>
<th>Lean Control</th>
<th>Percent Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>TB (Pre/Post)</td>
<td>0.9326±0.02</td>
<td>0.958±0.02*</td>
<td>+2.61%</td>
<td>0.929±0.02</td>
</tr>
<tr>
<td>Arms (Pre/Post)</td>
<td>0.620±0.06</td>
<td>0.677±0.02</td>
<td>+8.42%</td>
<td>0.682±0.02</td>
</tr>
<tr>
<td>Legs (Pre/Post)</td>
<td>0.944±0.04</td>
<td>0.985±0.04*</td>
<td>+4.16%</td>
<td>0.958±0.05</td>
</tr>
<tr>
<td>Pelvis (Pre/Post)</td>
<td>0.881±0.03</td>
<td>0.891±0.03</td>
<td>+1.12%</td>
<td>0.876±0.03</td>
</tr>
<tr>
<td>Spine (Pre/Post)</td>
<td>0.723±0.02</td>
<td>0.742±0.2*</td>
<td>+2.56</td>
<td>0.739±0.02</td>
</tr>
</tbody>
</table>

Note: Bone mineral density in (g/cm²) scan pre to post testing; * P<0.05 pre vs pos; Reported as the mean ± SEE
Total Body Bone Mineral Density

The change in total body bone mineral density (g/cm$^2$) for the obese treatment group, from week zero to week sixteen is displayed below (Figure 1). A student’s T test between the mean changes in bone mineral density shows a significant (P<0.001) increase from pre testing (0.982 ± .014 g/cm$^2$) to post testing (1.01 ± 0.015 g/cm$^2$).

Figure 1 shows the change of Total Body BMD in the obese treatment group from pre to post testing. N=29

The change in total body bone mineral density (g/cm$^2$) for the obese control group, from week zero to week sixteen is displayed below(Figure 2). A student’s T test between the mean...
changes in bone mineral density shows no significant (P>0.05) increase’s from pre testing (0.968 ± 0.029 g/cm$^2$) to post testing (0.984 ± 0.030 g/cm$^2$).

**Figure 2**

![Total Body Bone Mineral Density (Obese Control Group)](image)

Figure 2 shows the change of Total Body BMD in the obese control group from pre to post testing. N=12

The change in total body bone mineral density (g/cm$^2$) for the all obese groups, from week zero to week sixteen is displayed below (Figure 3).

**Figure 3**
The change in total body bone mineral density (g/cm²) for the lean treatment group, from week zero to week sixteen, is displayed below (Figure 4). A student’s T test between the mean changes in bone mineral density shows a significant (P<0.01) increase from pre testing (0.932 ± .021 g/cm²) to post testing (0.958 ± 0.020 g/cm²).

Figure 3 shows the change of Total Body BMD in the obese treatment and control group at week 0 and 16. N= 29, OT; 12, OC
The change in total body bone mineral density (g/cm$^2$) for the lean control group, from week zero to week sixteen is displayed below (Figure 5). A student’s T test between the mean changes in bone mineral density shows no significant (P>0.05) increase’s from pre testing (0.930 ± .023 g/cm$^2$) to post testing (0.942 ± 0.023 g/cm$^2$).
The change in total body bone mineral density (g/cm²) for the all lean groups, from week zero to week sixteen (Figure 6).

Figure 6

Figure six: The change of Total Body BMD in the lean treatment and control group at week 0 and 16. N=11, LT; 8, LC
Arm Bone Mineral Density

The change in arm bone mineral density (g/cm$^2$) for all lean groups, from pre to post testing is displayed below. Treatment subjects had no significant (P>0.05) increase in bone mineral density from pre testing to post testing (Figure 7). Control subjects significantly (P<0.05) increased arm BMD from pre testing (0.682±0.02 g/cm$^2$) to post testing (0.723±0.02 g/cm$^2$).

Figure 7
The change in arm bone mineral density (g/cm²) for all obese groups, from pre to post testing is displayed below (Figure 8). Treatment and control groups did not significantly increase bone mineral density from pre testing to post testing.

**Figure 8**

![Change in Arm BMD](image)

Figure 8 displays the change in arm bone mineral density (g/cm²) for all obese groups, from pre to post testing. N= 29, OT; 12, OC

**Leg Bone Mineral Density**

The change in leg bone mineral density (g/cm²) for all lean groups, from pre to post testing, is displayed below. Treatment subjects significantly (P<0.001) increased leg BMD from pre testing (0.944±0.04 g/cm²) to post testing (0.985±0.04 g/cm²) (Figure 9). Control increased leg BMD from pre testing (0.958±0.05 g/cm²) to post testing (0.982±0.04 g/cm²).
The change in leg bone mineral density (g/cm$^2$) for all obese groups, from pre to post testing, is displayed below. Treatment subjects significantly (P<0.001) increased leg BMD from pre testing (1.071±0.02 g/cm$^2$) to post testing (1.097±0.02 g/cm$^2$) (Figure 10). Control subjects significantly (P<0.05) increased leg BMD from pre testing (1.075±0.05 g/cm$^2$) to post testing (1.100±0.04 g/cm$^2$).
Pelvis Bone Mineral Density

The change in pelvis bone mineral density (g/cm$^2$) for all lean groups, from pre to post testing, is displayed (Figure 11). Treatment subjects significantly (P>0.05) increased pelvis BMD from pre testing (0.881±0.03 g/cm$^2$) to post testing (0.891±0.03 g/cm$^2$) (Figure 11). Control subjects significantly (P>0.05) increased leg BMD from pre testing (0.876±0.03 g/cm$^2$) to post testing (0.898±0.03 g/cm$^2$).
The change in pelvis bone mineral density (g/cm$^2$) for all obese groups, from pre to post testing, is displayed (Figure 12). Treatment subjects significantly (P<0.001) increased pelvis BMD from pre testing (0.993±0.02 g/cm$^2$) to post testing (1.023±0.02 g/cm$^2$). Control subjects significantly (P=<0.001) increased pelvis BMD from pre testing (0.975±0.03 g/cm$^2$) to post testing (1.007±0.04 g/cm$^2$).
Spine Bone Mineral Density

The change in spine bone mineral density (g/cm$^2$) for all lean groups, from pre to post testing, is displayed (Figure 13). Treatment subjects significantly ($P<0.01$) increased spine BMD from pre testing (0.723±0.02 g/cm$^2$) to post testing (0.742±0.02 g/cm$^2$). Control subjects increased pelvis BMD from pre testing (0.739±0.03 g/cm$^2$) to post testing (0.746±0.02 g/cm$^2$).
The change in spine bone mineral density (g/cm²) for all obese groups, from pre to post testing, is displayed (Figure 14). Treatment subjects were able to significantly (P<0.01) increase spine BMD from pre testing (0.846±0.02 g/cm²) to post testing (0.862±0.02 g/cm²). Control subjects were unable to significantly (P>0.05) increase pelvis BMD from pre testing (0.823±0.03 g/cm²) to post testing (0.838±0.02 g/cm²).
Bone Mineral Content

Bone mineral content expressed in grams are displayed in table 3.

Table 3a: Bone Mineral Content (Obese)

<table>
<thead>
<tr>
<th>DEXA Scan</th>
<th>Obese Treatment</th>
<th>Percent Change</th>
<th>Obese Control</th>
<th>Percent Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>TB (Pre/Post)</td>
<td>1758±73.4</td>
<td>1864±76.6*</td>
<td>+5.69%</td>
<td>1726±142.9</td>
</tr>
<tr>
<td>Arms (Pre/Post)</td>
<td>185.7±8.73</td>
<td>198.8±9.39*</td>
<td>+6.59%</td>
<td>175.4±15.7</td>
</tr>
<tr>
<td>Legs (Pre/Post)</td>
<td>720.9±35.1</td>
<td>777.1±36.7*</td>
<td>+6.46%</td>
<td>685.8±64.4</td>
</tr>
<tr>
<td>Pelvis (Pre/Post)</td>
<td>202.2±17.5</td>
<td>221.9±14.4*</td>
<td>+8.88%</td>
<td>189.0±21.9</td>
</tr>
<tr>
<td>Spine (Pre/Post)</td>
<td>172.9±10.6</td>
<td>173.7±9.0</td>
<td>+0.46%</td>
<td>153.8±13.8</td>
</tr>
</tbody>
</table>

Note: Bone mineral content in grams (g) scan pre test and post test; * P<0.05 pre vs pos; Reported as the mean ± SEE
Table 3b: Bone Mineral Content (Lean)

<table>
<thead>
<tr>
<th>DEXA Scan</th>
<th>Lean Treatment</th>
<th>Percent Change</th>
<th>Lean Control</th>
<th>Percent Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>TB (Pre/Post)</td>
<td>1332±78.7</td>
<td>1443±87.7*</td>
<td>+7.69%</td>
<td>1376±133.2</td>
</tr>
<tr>
<td>Arms (Pre/Post)</td>
<td>139.8±14.3</td>
<td>152.7±16.1*</td>
<td>+8.45%</td>
<td>152.1±23.9</td>
</tr>
<tr>
<td>Legs (Pre/Post)</td>
<td>505.0±43.3</td>
<td>549.5±49.3*</td>
<td>+8.10%</td>
<td>541.1±70.0</td>
</tr>
<tr>
<td>Pelvis (Pre/Post)</td>
<td>141.1±11.9</td>
<td>146.5±11.9*</td>
<td>+3.69%</td>
<td>155.2±16.5</td>
</tr>
<tr>
<td>Spine (Pre/Post)</td>
<td>101.9±8.97</td>
<td>109.6±5.05</td>
<td>+7.03%</td>
<td>105.7±9.98</td>
</tr>
</tbody>
</table>

Note: Bone mineral content in grams (g) scan pre test and post test; *P<0.05 pre vs post; Reported as the mean ± SEE

Total Body Bone Mineral Content

The change in total body bone mineral content (grams) for all obese groups, from pre to post testing, is displayed (Figure 15). Treatment subjects significantly (P<0.001) increased total body BMC from pre testing (1758±73.4g) to post testing (1864±76.5g). Control subjects significantly (P<0.05) increased total body BMC from pre testing (1726±142.9g) to post testing (1794±139.4 g/cm²).
The change in total body bone mineral content (grams) for all lean groups, from pre to post testing, is displayed (Figure 16). Treatment subjects significantly ($P<0.001$) increased total body BMC from pre testing ($1332\pm 78.6g$) to post testing ($1443\pm 87.7g$). Control subjects significantly ($P<0.001$) increased total body BMC from pre testing ($1376\pm 133.2g$) to post testing ($1449\pm 137.8 \text{ g/cm}^2$).
Arm Bone Mineral Content

The change in arm bone mineral content (grams) for all obese groups, from pre to post testing, is displayed (Figure 17). Treatment subjects significantly (P<0.001) increased arm BMC from pre testing (185.7±8.73g) to post testing (198.8±9.39g). Control subjects significantly (P<0.007) increased arm BMC from pre testing (175.4±15.7g) to post testing (190.8±18.0g).
The change in arm bone mineral content (grams) for all lean groups, from pre to post testing, is displayed (Figure 18). Treatment subjects significantly (P<0.05) increased arm BMC from pre testing (139.8±14.3g) to post testing (152.7±16.1g). Control subjects significantly (P<0.05) increased arm BMC from pre testing (152.1±23.9g) to post testing (168.7±24.9g).
Leg Bone Mineral Content

The change in leg bone mineral content (grams) for all obese groups, from pre to post testing, is displayed (Figure 19). Treatment subjects significantly (P<0.001) increased leg BMC from pre testing (720.9±35.1g) to post testing (777.1±36.7g). Control subjects significantly (P<0.01) increased leg BMC from pre testing (685.8±64.4g) to post testing (738.6±68.7g).
The change in leg bone mineral content (grams) for all lean groups, from pre to post testing is displayed (Figure 20). Treatment subjects significantly (P<0.001) increased leg BMC from pre testing (505.0±43.3g) to post testing (549.5±49.3g). Control subjects significantly (P<0.01) increased leg BMC from pre testing (541.1±70.0g) to post testing (567.4±70.6g).
Figure 20 displays the change in leg bone mineral content (grams) for all lean groups, from pre to post testing. N= 11, LT; 8, LC

**Pelvis Bone Mineral Content**

The change in pelvis bone mineral content (grams) for all obese groups, from pre to post testing is displayed (Figure 21). Treatment subjects significantly (P<0.05) increased pelvis BMC from pre testing (202.2±17.5g) to post testing (221.9±14.4g). Control subjects significantly (P<0.01) increased pelvis BMC from pre testing (189.0±21.9g) to post testing (191.0±21.1g).
The change in pelvis bone mineral content (grams) for all lean groups, from pre to post testing, is displayed (Figure 22). Treatment subjects were able to significantly (P<0.05) increase pelvis BMC from pre testing (134.0±10.0g) to post testing (146.5±11.8g). Control subjects increased pelvis BMC from pre testing (155.2±16.5g) to post testing (156.5±18.9g).
Spine Bone Mineral Content

The change in spine bone mineral content (grams) for all obese groups, from pre to post testing is displayed (Figure 23). Treatment subjects significantly (P>0.05) increased spine BMC from pre testing (172.8±10.6g) to post testing (173.7±8.98g). Control subjects significantly (P>0.05) increased spine BMC from pre testing (153.8±13.8g) to post testing (160.6±14.2g).
The change in spine bone mineral content (grams) for all lean groups, from pre to post testing, is displayed (Figure 24). Treatment subjects increased spine BMC from pre testing (172.8±10.6g) to post testing (173.7±8.98g). Control subjects significantly (P>0.05) increased spine BMC from pre testing (153.8±13.8g) to post testing (160.6±14.2g).
Aim 2: Activity Monitoring

Three day step count was measured at weeks 0, 4, 8, 12, and 16 using a ymax accelerometer.

Average 3-day step counts are displayed in Table 4.

Table 4: Average 3-day Step Count

<table>
<thead>
<tr>
<th></th>
<th>Week 0 Steps</th>
<th>Week 4 Steps</th>
<th>Week 8 Steps</th>
<th>Week 12 Steps</th>
<th>Week 16 Steps</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Obese Treatment</strong></td>
<td>6902.76±403.1</td>
<td>7228.65±610.6</td>
<td>8697.34±594.3</td>
<td>8574.63±558.7</td>
<td>7183.72±580.9</td>
</tr>
<tr>
<td><strong>Obese Control</strong></td>
<td>8164.40±814.2</td>
<td>7142.33±950.7</td>
<td>10710.25±3406.8</td>
<td>8526.57±1223.8</td>
<td>6499.44±510.0</td>
</tr>
<tr>
<td><strong>Lean Treatment</strong></td>
<td>8609.33±640.1</td>
<td>10118.11±1613.9</td>
<td>10684.88±1254.9</td>
<td>7506.75±618.4</td>
<td>8017.50±682.9</td>
</tr>
<tr>
<td><strong>Lean Control</strong></td>
<td>7533.58±979.6</td>
<td>7185.49±741.6</td>
<td>9703.79±1027.0</td>
<td>8550.60±1171.7</td>
<td>6841.58±603.3</td>
</tr>
</tbody>
</table>

Average 3-day step counts are displayed in Table 4. Reported as the mean ± SEE;
The average number of steps was computed by averaging the total step counts for a three day period. These days included an exercise program day, non-program day, and a weekend day. For non-exercisers these days included two week days, and a weekend day. (Figure 25).

**Figure 25**

Figure 25 displays the average number of steps taken in a three day period at weeks 0, 4, 8, 12, and 16. N= 29, OT; 12, OC; 11, LT; 8, LC

**Aim 3: Nutritional Monitoring**

Three day food records were kept by participants at weeks 0, 4, 8, 12, and 16. These records were then entered into Nutritionist Pro, a computer software program, to be analyzed for some substrates known to effect the growth of bone. Table 5a.-5c. reflects the data obtained from 3 day food records. Figures 26, 27, and 28 graphically display these data along with the recommended dietary allowance (RDA) for each respective nutrient.

**Table 5a. Dietary Calcium Intake in Obese and Lean prepubescent children undergoing either a 16-week physical activity treatment or a control period of 16 weeks without treatment.**
Table 5b. Dietary Phosphorus Intake in Obese and Lean prepubescent children undergoing either a 16-week physical activity treatment or a control period of 16 weeks without treatment.
Lean Control  |  1283.8±262.6  |  598.6±90.2  |  663.1±78.7  |  801.2±155.8  |  1175.5±187.7  
RDA          |  1,250         |  1,250       |  1,250       |  1,250        |  1,250         

Note: Phosphorus is recorded as a three day average in milligrams (mg); Reported as the mean ± SEE

Table 5c. Dietary Vitamin D Intake in Obese and Lean prepubescent children undergoing either a 16-week physical activity treatment or a control period of 16 weeks without treatment.
Table 6 displays the total body BMC to weight, total body BMC to lean tissue, and total body BMC to fat tissue ratios as mean plus or minus the standard deviation. The Obese treatment group increase total body BMC/weight ratio significantly (p<0.05) pre (0.031±0.003 BMC/kg) to post testing (0.032±0.004 BMC/kg). There was also a significant increase in total body BMC/fat tissue ratio from pre testing (0.057±0.010 BMC/kg) to post testing (0.058±0.010 BMC/kg) in the obese treatment group.
<table>
<thead>
<tr>
<th>Table 6.</th>
<th>TB BMC/Weight</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>P value</td>
<td></td>
</tr>
<tr>
<td>Lean</td>
<td>0.038±0.004 BMC/kg</td>
<td>0.039±0.004 BMC/kg</td>
<td>p=0.12</td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obese</td>
<td>0.031±0.003 BMC/kg</td>
<td>0.032±0.004 BMC/kg</td>
<td>p=0.03*</td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lean</td>
<td>0.039±0.004 BMC/kg</td>
<td>0.039±0.004 BMC/kg</td>
<td>p=0.40</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>0.041±0.015 BMC/kg</td>
<td>0.040±0.012 BMC/kg</td>
<td>p=0.22</td>
<td></td>
</tr>
<tr>
<td>Obese</td>
<td>0.041±0.015 BMC/kg</td>
<td>0.040±0.012 BMC/kg</td>
<td>p=0.22</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>TB BMC/Lean Tissue</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>p value</td>
<td></td>
</tr>
<tr>
<td>Lean</td>
<td>0.051±0.005 BMC/kg</td>
<td>0.052±0.004 BMC/kg</td>
<td>p=0.33</td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obese</td>
<td>0.057±0.010 BMC/kg</td>
<td>0.058±0.010 BMC/kg</td>
<td>p=0.70</td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lean</td>
<td>0.050±0.003 BMC/kg</td>
<td>0.051±0.003 BMC/kg</td>
<td>p=0.85</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>0.055±0.009 BMC/kg</td>
<td>0.055±0.008 BMC/kg</td>
<td>p=0.70</td>
<td></td>
</tr>
<tr>
<td>Obese</td>
<td>0.055±0.009 BMC/kg</td>
<td>0.055±0.008 BMC/kg</td>
<td>p=0.70</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>TB BMC/Fat Tissue</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>p value</td>
<td></td>
</tr>
<tr>
<td>Lean</td>
<td>0.051±0.005 BMC/kg</td>
<td>0.052±0.004 BMC/kg</td>
<td>p=0.74</td>
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</tr>
<tr>
<td>Treatment</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obese</td>
<td>0.057±0.010 BMC/kg</td>
<td>0.058±0.010 BMC/kg</td>
<td>p=0.01*</td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lean</td>
<td>0.024±0.068 BMC/kg</td>
<td>0.024±0.101 BMC/kg</td>
<td>p=0.97</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>0.090±0.014 BMC/kg</td>
<td>0.087±0.014 BMC/kg</td>
<td>p=0.15</td>
<td></td>
</tr>
</tbody>
</table>

N= 29, OT; 12, OC; 11, LT; 8, LC

*Indicates significant difference from pre to post testing.
Chapter 5 Discussion

The current obesity epidemic has become a major health crisis to citizens around the United States. The increase in vast amounts of cheap calorie enriched foods is wreaking havoc on a very unstable healthcare system through the added cost of health related sickness brought about by obesity. Obesity has been successfully linked to a plethora of different disease states including cardiovascular disease, diabetes, and stroke. Although many people might not recognize the trends, the obesity epidemic is also putting children as young as 5 and 6 years of age at a health disadvantage compared to children of a healthier weight (Andersen 2006).

Physical activity can be one way to combat obesity. Increases in caloric expenditure through physical activity, can create a situation where fat stores are reduced. Promotion of physical activity programs in children can decrease cholesterol levels, insulin levels and increase lean tissue, HDL cholesterol and bone density (Raitakan 1994; MacKelvie 2004). With regard to bone health in particular, there is a good amount of information supporting the acquisition of bone mineral density (BMD) through exercise, in both adults and children. However, no studies to date have investigated how a sixteen week physical exercise program can influence bone acquisition in lean and obese children. Previous studies have shown that overweight and obese children are at an increased risk of bone fracture due to low bone mass and bone area for weight (Goulding 2000; Goulding 2001), but this information does not suggest whether exercise could increase bone mass in these obese children.

Data obtained through the current study shows total body BMD, pre testing to post testing, increased in both lean and obese groups regardless of treatment. There is a slightly
larger response of bone density in experimental groups when compared to control groups. Over a sixteen week period the obese experimental group increased total body bone mineral density by 2.6%. The lean group expressed an increase in total body BMD of 2.6%. Previous longitudinal exercise studies have shown increases in BMD in children, but these studies are unlike the current study in that the current study gave emphasis on reaching and maintaining a heart rate above 140 beats per minute or 65 percent of maximal heart rate. It is apparent from total body BMD results obtained in this study that prepubescent children 9-11 years of age can experience a small percentage increase in total BMD by simply exercising for three hours a week, for sixteen weeks.

Exercise has been shown to be very site specific when it comes to new bone formation (Bradney 1998; Bass 1998). For this reason, along with total body measurements, the current study also reports descriptive data on how bone density of the legs, arms, pelvis and spine, changed from pretest to post testing in both treatment and control groups. Of the different sites analyzed there was a significant increase of at the spine of the obese treatment group, while no such significance is found for the obese control group. A previously reported study reports no differences in obese childhood spinal bone mineral density compared to control subjects (Schepper, 1995).

When analyzing the data from lean children there are several different regions that increase in bone density for the treatment group, but not in the control group. Total-body, legs and spinal density all increase in the lean treatment group, but not in the control groups. These findings agree with similar findings in prepubertal gymnasts that significantly increase spine,
leg, and total body BMD over time, compared to control groups (Bass 1998). The most substantial increase occurs in the spine, where the treatment group experienced an increased BMD of 2.56% versus a control group that only increased 0.94%.

Classifying participants by BMD exclusively can fail to account for the change in bone thickness (Katzman 1991). To get a better understanding of bone thickness, it is important to consider the BMC measure by extracting the area component from the density formula. With regards to BMC, it is worth mentioning first that the participants with a higher BMI percentile (>95th) started at baseline with higher BMC values than the lower BMI percentile (<85th) (p<0.05). This is believed to be correlated to the greater amount of weight bearing these children perform in everyday activities due to their larger mass. When classified by the change in bone mineral content, pre testing to post testing, the change in BMC was found to be higher at all sites in both lean and obese treatment groups compared to controls. In the obese category, children increase total body BMC in the treatment group by 106 grams, or 5.69% (p<0.05). In the obese control group there was only a total body increase of BMC by 68 grams or 3.79%. Interestingly, lean treatment participants were able to increase total body BMC by 111 grams, or 7.69% (p<0.05), while lean control participants increased total body BMC by 73 grams, or 5.04%. It is likely that the higher amounts of BMC, expressed by the lean and obese treatment groups, could have a positive effect on bone fracture prevention. Previous evidence shows that the stress placed on the bone during exercise allows for more calcium deposits to build up, these deposits can increase the strength of the lattice structure specific to bone tissue. If this lattice structure is indeed strengthened, the treatment children could have greater
protection from fall related fractures. Cortical bone is the most abundantly found skeletal tissue throughout the body, comprising eighty percent of the skeletal tissue. It is therefore likely that a good majority of the bone that is acquired through exercise is indeed cortical bone. Cortical bone is found on the outside layers of bone tissue and can be protective against bone fractures due to its strength, and has also been found to increase in size at specific sites through sporting events such as tennis (Krahl 1994).

It is evident from the results that BMC is changed very rapidly over a sixteen week period in prepubertal children regardless of group. Obese treatment children increased their BMC values 6.63 grams per week, while the obese control group was only increasing BMC by 4.25 grams per week. The treatment group had over a 2 gram net gain per week, that amount can create a substantial effect on bone enhancement annually. If current conditions were held constant for both groups and BMC continued to accrue at this rate over a year’s period, treatment participants would continue to increase their total body BMC by 344.5 grams, which would be the equivalent to almost \( \frac{1}{5} \)th of their pretest bone mineral content. The obese control children’s total body BMC, if current conditions were held constant, would increase annually by 221 grams, or \( \frac{1}{8} \)th of pretest values. Lean children increased total body BMC as well, and if current conditions were held constant for a year’s period and the lean treatment group continued to gain 6.94 grams per week they would be able to increase total body BMC by 360.8 grams annually. 360.8 grams comes to be over \( \frac{1}{4} \)th of pretest BMC value or an increase of BMC one standard deviation from pretest values. Lean control subjects gaining 4.56 grams per week would only be able to increase total body BMC values by 237.3 grams annually, or
almost \( \frac{1}{6} \)th of pretest values. A review of controlled trials on prepubertal children and bone formation through exercise, show an increase in total body bone mineral values of 1.1-4.5\% (Hind, 2007). Our current study corresponds with these percentage increases for exercisers, but what is most significant about these trials, compared to ours, is the program time. The exercise intervention used with our study was only 4 months long (16 weeks), while the studies mentioned in the review were implemented for at least 7 months or longer. The approach used with this studies exercise protocol was different compared to previous investigations. A target heart rate of >140 beats per exercise session was used with all training participants as opposed to a set routine as explored in the other studies. The results and the subsequent gains in BMD and BMC in the lean and obese treatment participants gives solid evidence that games such as the ones played in this study that induce an exercise heart rate >140 beats per minute can indeed increase bone mineral accrue. Due to the limited amount of data concerning the growth patterns of bone during prepubertal childhood, it is very hard to predict if the current rate of growth experienced with our participants would indeed continue or if some physiological element would further enhance, or limit biological processes of bone growth. Judging from this data these children, who have yet to experience major levels of steroid hormone interaction as seen in puberty, have increased bone mineral accrual over sixteen weeks, which could add to an even greater net gain of BMD and BMC when with children actually hit puberty. It appears that experimental group in this population is at a slight physiological advantage of BMC accrual versus the control groups.
Other studies have indicated that obese children are at more risk to fracture due to a higher load placement on the skeleton from extra weight (Goulding 2000, Goulding 2001). With our study these findings are confirmed in Table 6. Results show that obese children have a poor BMC to body weight ratio compared to lean children, suggesting that the obese children’s bones are in fact weaker, relative to weight. A promising finding within the obese population is that the obese children improved their BMC to weight ratio by performing the exercise protocol prescribed in this study (p<0.05). Improving the ratio of BMC to weight can provide the obese children greater protection from fracture, as well as other bone abnormalities. Obese children also had a higher total body BMC/fat tissue ratio than lean children at baseline (p<0.001), indicating that this population has a greater amount of fat per gram of bone than the lean children. Through exercise intervention obese children increased its BMC/fat tissue ratio as well.

One of the most important factors influencing a child’s ability to increase bone stores is diet. Johnston et al. showed that prepubertal twins receiving supplements of calcium to reach recommended dietary allowances can indeed significantly increase BMD values compared to controls subjects (Johnston 1992; Ruiz 1995). Likewise, studies have shown that children ingesting more than 1000 mg of calcium daily have a higher BMC than children consuming less (Chan 1991). With the current study, dietary calcium was obtained through a 3 day dietary recall in order to understand if recommended dietary allowances were being reached. Children in this study were not receiving intakes near the recommended 1300 mg/day. If these calcium poor diets continue longitudinally these children could be at risk of calcium deficiency fractures.
or diseases such as osteoporosis. Bakary et al. investigate populations of children severely deprived of calcium (<342mg/d) and found these children were subject to poor growth, and BMC values. After supplementation of calcium (1000mg/d) for a 12 month timeframe, children significantly increased BMC values (Bakary 2000).

The children in this study were found to ingest more phosphorus over the 16 week intervention than calcium. Bone building processes relay heavily on the maintenance of a certain calcium/phosphorus ratio. If phosphorus levels rise in the blood, calcium resorption will occur in the bone and gut to maintain proper ratio levels in the blood, thus minimal bone can be created and stored. Bone stores are increasing with the treatment groups despite the higher levels of dietary phosphorus compared to calcium. The increases in phosphorus through the diet can be correlated to the rise in soft drink intake among younger populations (HARNACK 1999; Nielsen 2004). The increase in soft drink consumption has been linked to a decrease in milk consumption, thus less calcium rich products such as milk are being used by younger populations.

Vitamin D aids absorption of calcium from the intestinal track. Though vitamin D is found to be very low among the groups in the current study, this measure can be an inaccurate indicator of true vitamin D status, because this hormone can be gained from contact with sunlight. 15 minutes of sunlight can provide the body with more than enough vitamin D needed for proper bodily function (Gartner 2003).

Our study does present some limitations. Biomarkers of bone turnover such as parathyroid hormone, and growth hormone were not measured in the participants. Without
this data no determination can be made as to what impact these hormones have on the growing body of prepubescent children. Blood values would also be valuable to obtain to make inferences concerning the dietary information. Levels of serum calcium, vitamin D, and phosphorus could allow us to make better comparisons between diet and bone metabolism.
Chapter 6: Conclusion

In conclusion the results of the current study show that children, ages 8-11, can increase bone mineral density, regardless of exercise load. In both instances (lean and obese) the treatment group had a slightly greater increase in both BMD and BMC accrue versus the control groups. The findings in this study are in agreement with findings of other studies that investigated BMD and BMC, in prepubescent children. This study shows that lean children have a higher BMC/weight ratio, and BMC/fat ratio at pre and post testing compared to obese children. Obese children carry more weight than lean children, so this population could be at more risk of bone fracture due to these poor ratios. Promisingly, the treatment protocol increased the BMC/fat ratio, and BMC/weight ratio in the obese children, from pretesting to post testing (Table 6). As mentioned previously, it is important to draw distinctions in this study compared to others that have studied prepubescent children so that proper exercise protocols can be established to efficiently increase both BMC and BMD in the target population. The exercise protocol used with the current study only required that the children keep a target heart rate of >140 beats per minute. This allowed the child to pick a variety of different activities during their exercise sessions, which promoted adherence for the program because the child would pick games that he/she felt comfortable performing. Increases in skeletal mass are shown to be very vital in the battle against osteoporosis, osteopenia, as well as reduced fracture risk; therefore, it is important to know that exercise can indeed help increase both BMD and BMC during prepubertal childhood. Further longitudinal research could be useful to understand if the changes in BMD and BMC continue.
References


Bass, S. (2000). The prepubertal years: A uniquely opportune stage of growth when the skeleton is most responsive to exercise. Sports Medicine, 30, 73-78.


Crawford, P. B., Obarzanek, E., Morrison, J., & Sabry, Z. I. (1994). Comparative advantage of 3-day food records over 24-hour recall and 5-day food frequency validated by observation of
9- and 10-year-old girls. *Journal of the American Dietetic Association, 94*(6), 626-630. doi:DOI: 10.1016/0002-8223(94)90158-9


APPENDIX A: IRB Approval

TO: Robert Hickner, PhD, Dept. of EXSS & Physiology, 363 Ward Sports Medicine Bldg., ECU
FROM: UMCIRB
DATE: July 19, 2010
RE: Full Committee Approval for Continuing Review of a Research Study
TITLE: Reduction in CVD Risk in Children Through Physical Activity
UMCIRB #05-0384

The above referenced research study was initially reviewed by the convened University and Medical Center Institutional Review Board (UMCIRB) on 9/21/05. The research study underwent a subsequent continuing review for approval on 6/23/10 by the convened UMCIRB. The UMCIRB deemed this NIH/NIDDK sponsored study more than minimal risk requiring a continuing review in 12 months. Changes to this approved research may not be initiated without UMCIRB review except when necessary to eliminate an apparent immediate hazard to the participant. All unanticipated problems involving risks to participants and others must be promptly reported to the UMCIRB. The investigator must submit a continuing review/closure application to the UMCIRB prior to the date of study expiration. The investigator must adhere to all reporting requirements for this study.

The above referenced research study has been given approval for the period of 6/23/10 to 6/22/11. The approval includes the following items:
- Continuing Review Form (dated 06/14/2010)
- Protocol (version date 07/26/2007)
- Protocol Summary
- Informed consent (version date 07/26/2007)
- Minor assent (version dated 07/26/2007)
- Flyer (dated 01/08/2009)
- Youth Risk Behavior Survey (dated 08/01/2005)
- Leisure Time Exercise Questionnaire (dated 08/01/2005)
- 30-Day Physical Activity Recall (dated 08/01/2008)
- 3-Day Food Record including Instructions for Keeping Your 3-Day Food Record
- Medical History Form
- Physical Activity Logs
- PedsQL 4.0
- Food Frequency Questionnaire
- Physical Self-Perception and Children’s Attraction Toward Physical Activity Scale

The following UMCIRB members were recused for reasons of potential for Conflict of Interest on this research study: R. Hickner & S. McCammon

NOTE: The following UMCIRB members with a potential Conflict of Interest did not attend this IRB meeting: None

The UMCIRB applies 45 CFR 46, Subparts A-D, to all research reviewed by the UMCIRB regardless of the funding source. 21 CFR 50 and 21 CFR 56 are applied to all research studies under the Food and Drug Administration regulation. The UMCIRB follows applicable International Conference on Harmonisation Good Clinical Practice guidelines.
APPENDIX B: Informed Consent Form

INFORMED CONSENT

Principal Investigator: Robert C. Hickner, Ph.D.
Institution: Human Performance Laboratory
Address: 371 Ward Sports Medicine Building
Telephone Number: (252) 328-4677

TITLE OF PROJECT: Reduction in CVD risk in children through physical activity

INTRODUCTION
Your child has been asked to participate in a research study being conducted by Robert C. Hickner and colleagues. This research is designed to determine the effect of physical activity on cardiovascular disease risk in children.

We will study lean and overweight preadolescent children. Studies will take place in the Human Performance Laboratory of East Carolina University and in Minges Coliseum.

PLAN AND PROCEDURES
Prior to testing, you, as a guardian(s) will read and sign this Informed Consent for research, as well as fill out a medical history questionnaire pertaining to your child.

Your child’s participation will involve:

- You will fill out a personal history form that pertains to your child. Your child will fill out forms consisting of a youth risk behavior survey, leisure time exercise questionnaire, a personal history form, a medical form, a 30-day physical activity recall, pediatric quality of life inventory, and a physical self-perception profile and children’s attraction toward physical activity scale.

- Determination of body composition using body mass index (BMI), waist-to-hip ratio (WHR), skinfolds, and a DEXA Scan will be conducted at the Human Performance Laboratory. To calculate BMI, height and weight will be measured. Circumference measures will be taken at the waist and hip to calculate WHR. Finally, skinfold thickness of the tricep, subscapular (shoulder blade), abdomen, thigh, supraillium (hip bone), and calf will be taken on the right side of the body, in duplicate, with a skinfold caliper. Your child will undergo a test of body composition called a DEXA scan. It is like an x-ray of your entire body. During this test your child will be asked to wear minimal clothing (e.g., swimsuit, or shorts and a shirt, or a gown), and to remove all jewelry. He/she will lie still on a padded table for the length of the scan (approximately 6 minutes). The table will move across and up and down to scan his/her body. Your child will not feel anything and can breathe normally during the scan. If your child has metal in his/her body, then your child will not be able to participate in the DEXA scan. Radiation exposure from a DEXA scan is approximately 0.04 mrem. The effective radiation exposure that your child would receive in this study is less
than 0.6% of the radiation exposure an individual receives from natural background sources in one year.

- **Determination of Aerobic Capacity**
  Two maximal treadmill tests will be completed to evaluate initial aerobic capacity. Two tests will be performed to assure that there is adequate effort by the children during the maximal treadmill test and to determine day-to-day variability in the test. If these two tests are not very similar, a third test may need to be conducted, so it is important that your child put out a maximal effort for this test. For this test, your child will walk or run on a treadmill for approximately 10-15 minutes. During this test, your child will wear a mouthpiece so the air they breathe out can be collected for analysis of oxygen. At first, your child will walk leisurely on the level treadmill, but the speed and level of hill climbing will become harder until your child can no longer continue.

You will be asked to complete the n-3 FFQ at baseline, 4 weeks, 8 weeks, 12 weeks, and 16 weeks. This will allow us to determine 1) what your child eats over time and 2) seasonal variations in your child's age group and population.

- Your child will complete a 3 or 4 day food record at baseline (before microdialysis), 4 weeks, 8 weeks, 12 weeks and 16 weeks.

- Your child will wear a **physical activity monitor** (RT3 Triaxial Accelerometer) and a pedometer (Yamax, Japan) five days prior to microdialysis and during the microdialysis portion of the study. Additionally, your child will wear the accelerometer and pedometer for 3 days at 4 weeks, 8 weeks, and 12 weeks.

- A **fasting blood sample** will be obtained from an arm or hand vein. This will involve one to three small needle sticks. The procedure will take place in the Human Performance Laboratory.

- Your child will be given a cotton swab to test for **salivary cortisol levels** and will be instructed to chew on it for 45-60 seconds. Samples will be collected at 7 a.m. (fasting), 1:45 (prior to a standardized 2 p.m. lunch), and 30, 45, and 60 minutes after lunch. Additional samples will be collected every hour on the hour from 9 a.m. to 3 p.m. The collection of samples will take place in the Human Performance Lab.

- At the Human Performance Lab, the insertion of up to three small probes to determine glycerol levels and rates of lipolysis will take place. This probe, called a **microdialysis probe**, is a small flexible piece of plastic tubing (about an inch long and the width of a needle) that is inserted through the skin, and then through the subcutaneous fat about 1/8 to 1/4 inch below the skin of the stomach. First, to numb the area of insertion, a topical cold spray (ethyl chloride) or a numbing creme (Emla creme) will be applied to the skin. A needle surrounded by plastic tubing will be inserted into the subcutaneous fat of your child’s stomach. The needle will be taken out of the fat and replaced with the small piece of tubing. The tubing will not be located in a blood vessel but between fat cells. A Ringer solution (a
saline/salt-water solution) will be pumped through the piece of tubing to monitor blood flow and fat break down in the fat tissue. The pumped fluid will be harmless to your child since it is similar to the fluid already present between the cells of the body. The Ringer solution will be pumped at a rate of no more than 5 microliters per minute (equivalent to a very tiny drop). Your child will not feel the presence or effects of this solution. Samples will be collected every hour while your child is at ECU and at home for the remainder of that day until the following morning. Only one overnight sample will be collected when your child wakes up the following morning.

- After the microdialysis pump is set up, your child will participate in activities pre-planned and provided by the study investigators. Activities will take place in Minges Coliseum. Possible activities will include walking on a treadmill, riding a stationary cycle ergometer, roller-skating, and jump roping. Other activities will include watching movies and playing board games. All activities will be monitored by a trained exercise physiologist who is familiar with the usage and safety precautions for each activity. By no means will your child be limited in what he/she can do during the day, except for activities that involve rough physical contact (for example, football).

- The full day monitoring will take place on a day that the child is already out of school (i.e. vacation, weekend).

- If your child is randomized (similar to picking groups by flipping a coin) to the 16-week physical activity program, he/she will undergo all testing described above (preliminary measurements and a separate visit of approximately 8 hours for microdialysis in the lab) before and after the 16 week program. If your child is randomized to the control group that does not participate in the 16 week physical activity program, they will be required to undergo only the preliminary measurements and another visit (microdialysis) of approximately 8 hours in the lab.

RISKS AND DISCOMFORTS
There are certain risks and discomforts that may be associated with this research. They include:

- The total amount of blood drawn for fasting blood draw is negligible. There is an extremely small risk of local hematoma or infection associated with the needle stick.

- Insertion of the microdialysis probe is associated with mild discomfort, similar to that experienced during an intramuscular injection. Your child will not feel discomfort from the substances (for example, Ringer solution) pumped through the microdialysis probe. Risks associated with this procedure are small, and include hematoma (swelling and bruising) and infection. To minimize the risk of hematoma or infection associated with the insertion of the microdialysis probes into the subcutaneous adipose tissue, these procedures will be performed using sterile techniques. The probes are also made of biocompatible materials.

- There are some risks associated with physical activity such as bumps, bruising, scrapes and other injuries associated with active children.
• Risks associated with the maximal exercise are dizziness, ventricular arrhythmia (odd heart beats), and in very rare instances death. These risks are very small, with an incidence of fewer than 1 in 10,000 deaths in patients who are known to, or suspected of, having heart disease. The risk is expectedly much smaller than this in a group of young, healthy subjects. To further minimize the risk, faculty and students that have been extensively trained in administering maximal exercise tests will administer the assessments. If during a test a subject complains of dizziness, chest discomfort or other signs of exercise intolerance, the test will be promptly stopped. In the event of loss of consciousness, breathing or heart beat, appropriate CPR and AED administration will be initiated and Greenville Fire/Rescue will be notified via 911.

• Risks of the body composition assessment are those associated with exposure to low levels of radiation. Risks will be minimized by using an FDA-approved bone density machine (Prodigy, GE Lunar Corp., Madison, WI). This procedure involves a minimal amount of radiation. 1-3 microSiervets) that is within an acceptable range as provided by “North Carolina Regulations for Protection Against Radiation”. The amount of radiation (1-3 microSieverts) exposure of one procedure is quite minimal. For example, radiation exposure is approximately 80 microSieverts on a transatlantic airline flight of 8 hours, 50 microSieverts living in Denver, Colorado, at an elevation of 5,000 feet for approximately 4 weeks, or 30 to 40 microSieverts during a typical chest x-ray. There is a potential risk to unborn children for those who are pregnant; therefore, pregnant women must not undergo this procedure.

• Your child should be aware that there are unforeseen risks involved with this and all research studies.

POTENTIAL BENEFITS
Subjects will be able to participate in supervised physical activity and games. The risks are minimal relative to these benefits and the benefits of gaining knowledge with respect to the role of cortisol in childhood obesity.

TERMINATION OF PARTICIPATION
Your child’s participation in this research study may be terminated without your consent if the investigators believe that these procedures will pose unnecessary risk to your child. Your child may also be terminated from the participation if your child does not adhere to the study protocol.

COST AND COMPENSATION
Your child will be paid $50.00 as well as prizes worth $25 for his/her time and inconvenience for completion of each microdialysis procedure. Each family with child/children in the exercise group will be compensated $40 for transportation related expenses at 8 weeks.

The policy of East Carolina University does not provide for the compensation or medical treatment for subjects because of the physical or other injury resulting from this research activity.
However, every effort will be made to make the facilities of Brody School of Medicine, Pitt County Memorial Hospital available for treatment in the event of such physical injury.

CONFIDENTIALITY
Only the investigators associated with this study will have access to the data obtained. No identifying information will be released. Numeric coding, which only the primary investigator will have access to, will protect the identity of your child and other subjects. Data will be secured in a locked filing cabinet in the office of the primary investigator in the Human Performance Laboratory. The data will be kept for 7 years. Samples will be stored in freezers at the Human Performance Laboratory for a maximum of 7 years. Your child can request destruction (discarded into biohazard containers and disposed of by ECU biohazard personnel) of his/her samples at any time.

VOLUNTARY PARTICIPATION
Your child understands that his/her participation in this study is voluntary. Refusal to participate will involve no penalty or loss of benefits to which your child is otherwise entitled. Furthermore, your child may stop participating at any time he/she chooses without penalty, loss of benefits, or without jeopardizing his/her continuing medical care at this institution.

RESEARCH PARTICIPANT AUTHORIZATION TO USE AND DISCLOSE INFORMATION
Federal laws require that researchers and health care providers protect your identifiable health information. Federal laws also require that researchers get your permission to use collected health information for research. The identifiable information we will collect from subjects in this research project will include:
* General Medical History including: Family health history, medications, nutrition, physical activity levels and body weight history.
* Body composition information, adipose tissue blood flow and metabolism, blood levels of insulin, glucose, free fatty acids, and other compounds related to cardiovascular disease risk.

The members of our research team that will have access to your information will include the Principle investigator, co-investigators, as well as technical and nursing personnel involved in this project. Information about you will be used and released in such a way that will protect your identity as much as possible; however, confidentiality cannot be absolutely guaranteed. We will only share your information with those individuals listed above. If we need to share information with other individuals other than those listed, we will request your permission a second time.

You will be given a signed copy of your authorization to release medical information for your records. You can limit the amount and type of information that is shared and you must make this request in writing; however, the researcher is able to use any and all information collected prior to the request not to disclose information. Although you can limit the release of your medical information, withholding some information may cause you to become ineligible for this research project. Because research information continues to be looked at after a study is finished, it is difficult to say when the use of your information will stop. There is currently not an expiration date for the use and disclosure of your information for this study.
PERSONS TO CONTACT WITH QUESTIONS
If you have questions related to the sharing of information, please call Robert Hickner at 252-737-4677 (days) or 252-353-5556 nights or weekends or Joseph Garry, M.D. at 744-1953 (days) or 353-2825 (nights and weekends). You may also telephone the University and Medical Center Institutional Review Board at 252-744-2914. In addition, if you have concerns about confidentiality and privacy rights, you may phone the Privacy Officer at Pitt County Memorial Hospital at 252-847-6545 or at East Carolina University at 252-744-2030.

CONSENT TO PARTICIPATE
Your child certifies that he/she has read all of the above information, asked questions, and received answers concerning areas he/she did not understand, and have received satisfactory answers to these questions. Your child willingly consents for participation in this research study. (A copy of this consent form will be given to the person signing as the subject or as the subject’s authorized representative.)

Participant’s Name (Print)

Authorized Representative’s Name (Print) – Guardian #1

Signature of Authorized Representative – Guardian #1 Date

Authorized Representative’s Name (Print) – Guardian #2

Signature of Authorized Representative - Guardian #2 Date

AUDITOR WITNESS: I confirm that the contents of this consent/assent form were orally presented.

Auditor’s Name (Print)

Signature of Auditor Date

Principal Investigator’s Name (Print)

Signature of Principal Investigator Date
FUTURE TESTING OF BLOOD/MICRODIALYSIS SAMPLES

Upon termination of this study, the blood and urine samples collected for this study will be stored for up to 10 years to research scientific questions specifically related to cardiovascular disease risk in children. I will continue to be the owner of the samples and retain the right to have the sample material destroyed at any time during this study by contacting the study principal investigator. During this study the samples will be stored with number identifiers only; however, the number identifier will be linked to a specific name and will be kept on file in the possession of the principal investigator. The linked file will be stored password protected on the Principal Investigator’s computer with CD backup. No other individuals will have access to these identifying materials unless the principal investigator is required by law to provide such identifying information. Data will not be publicly available and participants will not be identified or linked to the samples in publication. If a commercial product is developed from this research project, I will not profit financially from such a product.

CONSENT TO PARTICIPATE IN FUTURE TESTING OF BLOOD SAMPLES

I certify that I have read all of the above, asked questions and received answers concerning areas I did not understand, and have received satisfactory answers to these questions. I willingly give my consent for participation in this research study. (A copy of this consent form will be given to the person signing as the subject or as the subject’s authorized representative.)

CONSENT TO PARTICIPATE

Your child certifies that he/she has read all of the above information, asked questions, and received answers concerning areas he/she did not understand, and have received satisfactory answers to these questions. Your child willingly consents for participation in this research study. (A copy of this consent will be given to the person signing as the subject or as the subject’s authorized representative.)

Participant’s Name (Print)

Authorized Representative’s Name (Print) – Guardian #1

Signature of Authorized Representative – Guardian #1 Date

Authorized Representative’s Name (Print) – Guardian #2

Signature of Authorized Representative - Guardian #2 Date

AUDITOR WITNESS: I confirm that the contents of this consent/assent form were orally presented.

Auditor’s Name (Print)
<table>
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<tr>
<th>Signature of Auditor</th>
<th>Date</th>
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<tbody>
<tr>
<td>Principal Investigator’s Name (Print)</td>
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</tr>
<tr>
<td>Signature of Principal Investigator</td>
<td>Date</td>
</tr>
</tbody>
</table>
APPENDIX C: Minor Assent Form

ASSENT DOCUMENT FOR CHILDREN

Principal Investigator: Robert C. Hickner, Ph.D.
Institution: Human Performance Laboratory. 371 Ward Sports Medicine Building
Telephone Number: (252) 737-4677

TITLE OF PROJECT: Reduction in CVD risk in children through physical activity

You have been asked by Dr. Robert Hickner and workers in the Human Performance Lab to be part of a research project at East Carolina University. In this project, you will do several different things.

1. You will fill out forms about physical activity habits, including forms consisting of a youth risk behavior survey, leisure time exercise questionnaire, a medical form, a 30-day physical activity recall, HPCS, MSPCS, pediatric quality of life inventory, and a physical self-perception profile and children’s attraction toward physical activity scale

2. You will have your height, weight, and skinfolds and percent body fat measured. Skinfolds are measured by pinching different areas of fat on your body. You may feel a very light pinch. You will then go to another room where we will do a test called a DEXA Scan. It is like an x-ray of your entire body. During this test you will wear shorts and a shirt, or a gown, and you will take off any jewelry. You will then lie still on a padded table for about 6 minutes. The table will move across and up and down to scan your body, but you do not feel anything and can breathe normally during the scan.

3. You will come to the Human Performance Lab for a day (7 a.m. to 3 p.m.), where you will be able to play fun games, watch movies, and make new friends.

4. Someone at the lab will draw blood from a vein in your arm or hand. The needle stick will only hurt for a few seconds, although we may need to try up to three times if we do not get the blood on our first try.

5. You will have a small needle put into the fat under the skin of your stomach. You may feel a slight sting, but Dr. Hickner will try to make sure that this hurts as little as possible by spraying a cold spray or putting a cream on your stomach to numb your skin. A small plastic tube (as thin as a piece of thread) will be put through this needle under your skin. The needle will then be taken out after the plastic tube is in place. The plastic tube will then be hooked up to a little pump (smaller than a Walkman). You will have three of these needle sticks and plastic tubes put under the skin of the stomach. A liquid, called Ringer’s solution, will be pumped through the plastic tubes. This solution will help measure the break down of fat in your tissue. You will wear the pump on a belt while you are at ECU and while you are at home on this day until the next morning. You will have this test done before and after the 16-week physical activity program.
6. You will wear activity monitors, which looks like a pager, for 5 days prior to the day visit, and during the day visit. Additionally, you will wear the activity for 3 days during weeks 4, 8, and 12. You will wear the monitors on your belt or clothes.

7. You will participate in a maximal exercise test on the treadmill. For this test, you will walk or run on a treadmill for approximately 10-15 minutes. During this test, you will wear a mouthpiece so the air you breathe out can be collected. At first, you will walk on the level treadmill, but the speed and level of hill climbing will become harder until you can no longer continue. You will go through this test on two separate days. If these two tests are not very similar, a third test may be needed, so it is important that you put out a maximal effort for this test.

8. You will participate in a 16-week physical activity program where you will skate, ride bicycles, and play active games. You will need to come to the activity center 3 to 4 times per week for at least one hour per time.

9. You will be asked to drink a mixture of an amino acid (a component of protein that is found in many foods) and fruit juice before you go to bed. After this, you will be asked to collect your urine in a plastic container for the next 10 hours (each time you wake up). You will be asked to do this at start of the study, after 8 weeks of the activity program and at the end (approximately 16 weeks) of the study.

10. On a separate day, you will be asked to collect your urine in a jug we provide to you for a 24 hour period. This will occur at the start of the study, after 8 weeks of the activity program and at the end of the study. You will write down everything you eat or drink on the day in which you collect your urine.

11. You will be asked to complete a 3 or 4 day food records at baseline (before microdialysis), 4 weeks, 8 weeks, 12 weeks and 16 weeks.

12. Your personal information and samples collected will be kept private and safe in the Human Performance Lab. Only Dr. Hickner and co-workers will have access to your data. If you decide that you want you samples thrown out, your samples will be gotten rid of properly by workers at ECU.

______________________________    ________________________
Child's Name (print)               (Date)

______________________________    ________________________
Child's signature                 (Date)
PERSONS TO CONTACT WITH QUESTIONS
The investigators will be available to answer your (or your guardian’s) questions concerning this research, now or in the future. You or your guardian(s) may contact the investigators, Robert Hickner, Ph.D. at 737-4677 (days) or Joseph Garry, M.D. at 744-1953 (days) or 353-2825 (nights and weekends). Also, if questions arise about your rights as a research subject, you or your guardian(s) may contact the Chairman of the University and Medical Center Institutional Review Board at 252-744-2914 (days).

CONSENT TO PARTICIPATE
You certify that you have read all of the above information, asked questions, and received answers concerning areas you did not understand, and have received satisfactory answers to these questions. You willingly consent for participation in this research study. (A copy of this consent form will be given to the person signing as the subject or as the subject’s authorized representative.)

Authorized Representative Name (Print) – Guardian #1

Signature of Authorized Representative – Guardian #1 Date

Authorized Representative Name (Print) – Guardian #2

Signature of Authorized Representative – Guardian #2 Date

AUDITOR WITNESS: I confirm that the contents of this consent/assent form were orally presented.

Objective Third Party Witness Name (Print)

Signature of Objective Third Party Witness Date

Principal Investigator’s Name (Print)

Signature of Principal Investigator Date
APPENDIX D: 3 DAY DIET LOG

INSTRUCTIONS FOR KEEPING YOUR THREE DAY FOOD RECORD

1. Record EVERYTHING that you eat and drink for three days.

2. For accuracy, it is best to record each meal or snack immediately after it is eaten.

3. It is important to record the food accurately, regardless of when it was eaten. Be sure to include water, milk, juice, soda, etc.

4. If additional space is required for the same day, continue onto the back of the page, noting clearly that it is a continuation.

5. Record BRAND NAMES, if known.

6. If eating out, record foods eaten as accurately as possible, including the NAME OF THE ESTABLISHMENT and the SPECIFIC FOOD ITEM ORDERED.

7. Always specify METHOD OF PREPARATION.

Examples include: baked, broiled, fried, breaded, sautéed, etc.

8. Describe all foods as fully as possible.

For example: 3 oz. baked chicken thigh, no skin.
(NOTE: 3 oz. is approximately the size of a deck of cards.)

9. List ALL INGREDIENTS for sandwiches, casseroles, and other mixed dishes.

Example: Peanut butter sandwich – 2 pieces oat bran bread, 1½ Tbsp. chunky peanut butter.

A full recipe is not required.

10. Record EXACT AMOUNTS when known. Specify weight or volume or dimensions in inches (e.g., 1 piece banana bread, 1” by 2” by 4”). Use household measuring cups or spoons to estimate portions.

11. Include all ADDITIONS to food at the table, such as salt, sugar, or milk. Record each addition on a separate line.
<table>
<thead>
<tr>
<th>Meal</th>
<th>Time of day</th>
<th>Serving Size</th>
<th>Food Item</th>
<th>Prepared by</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breakfast</td>
<td>7:30 am</td>
<td>1 cup</td>
<td>Cheers</td>
<td>Mom</td>
</tr>
<tr>
<td></td>
<td></td>
<td>½ cup</td>
<td>2% milk</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 cup</td>
<td>Apple juice</td>
<td></td>
</tr>
<tr>
<td>Snack</td>
<td>10:00 am</td>
<td>1 medium</td>
<td>Banana</td>
<td>Teacher</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 cup</td>
<td>water</td>
<td></td>
</tr>
<tr>
<td>Lunch</td>
<td>12:00 pm</td>
<td>2 slices</td>
<td>Bread - hamburger bun</td>
<td>McDonalds</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 slice</td>
<td>Cheddar cheese</td>
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<td></td>
<td></td>
<td>1 patty</td>
<td>Hamburger</td>
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<td></td>
<td>1 supersized</td>
<td>French fries</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>1 16 ounce</td>
<td>Regular coke</td>
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<tr>
<td>Snack</td>
<td>3:30 pm</td>
<td>15</td>
<td>Crackers - Sociables</td>
<td>Mom</td>
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<tr>
<td></td>
<td></td>
<td>2 Tbsp</td>
<td>Peanut butter</td>
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<td></td>
<td>1 8 ounce</td>
<td>Juice box</td>
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<tr>
<td>Dinner</td>
<td>6:30 pm</td>
<td>5 ounces</td>
<td>Chicken -thigh - baked</td>
<td>Mom/Dad</td>
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<tr>
<td></td>
<td></td>
<td>1 ½ cups</td>
<td>rice</td>
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<td></td>
<td></td>
<td>½ cup</td>
<td>Broccoli</td>
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<td></td>
<td></td>
<td>1 cup</td>
<td>2% milk</td>
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<td></td>
<td></td>
<td>¼ cup</td>
<td>Mixed fruit - fruit cocktail with sauce</td>
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<tr>
<td>Snack</td>
<td>7:45 pm</td>
<td>1 ½ cups</td>
<td>Vanilla ice cream</td>
<td>Dad</td>
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<td></td>
<td></td>
<td>3 Tbsp</td>
<td>Chocolate sauce</td>
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</table>

Was this a typical day's intake? (Y/N. If no, please explain).

No, this was not a typical day's intake because I had a doctor's appointment and we went to McDonald's afterwards for lunch.
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Was this a typical day's intake? (Y/N. If no, please explain). ________________

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Was this a typical day's intake? (Y/N. If no, please explain).

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Was this a typical day's intake? (Y/N. If no, please explain). ________________________________

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APPENDIX E: ACTIVITY MONITORING LOG

Exercises and Sport Science
Call us at the ECU Department of
If you have any questions or equipment problems,

To

Recording Dates

Do not wear them when you
when you put on the activity monitors and when you like
Wear the activity monitors all day. Make sure to record the
Activity Monitor and Pedrometer. These are pictures in this
Activity Monitor and Pedrometer. These are pictures in this

Please read them carefully.

For completing the Logbook at the top of each page.
Complete one page at the end of each day.

This is your physical activity logbook for the next week.

Physical Activity Logbook

# Activity
# Ped.

A. V. You are not required to record
B. Press the yellow button to set the
C. At the end of the day record the number
D. Open the case
E. Close the case
F. Pedrometer

HOW TO WEAR THE MONITORS

HOW TO USE THE PEDROMETER
DAY 5


day.

Instructions: Fill this page out at the end of your

Physical Activity Logbook Instructions

Date

Day of the week: M W T H F SA SU

hawkations: How long did you do this activity?

Total number of steps taken:

You will keep track of four things:

Time activities were taken on:

Each day I will begin when you wake up and end when you go to bed. The physical activity logbook will be filled out the next day.

The number of steps you take throughout the day will be noted in the next column. Please fill in the circle of your choice each day.

What is the physical activity logbook for?

When did you start the physical activity logbook?

Who is the physical activity logbook owner?

When did you call the HPI (circle one)?

Yes

NO

Did you call the HPI (circle one)?

252-372-468

Time activities were taken on:

252-373-409

Number of steps taken on the end of the day

b) 30 min. 1 hr

(c) Soccer, dance, gymnastics, softball, FITT

What activity did you do the most of today?

How long did you do this activity? And how long is the physical activity logbook?
How long did you do this activity?

Total number of steps taken:

Time monitors were taken off:

Did you use the HPL? (check one)

Time monitors were put on:

Date:

Day of the week: M T W T H S F S A

Instructions: Place name at the end of your report.

Day 3

Day 2

Do the activities below sound appealing?

- Yoga, dance, gymnastics, softball, FITT

What activity did you do the most of today?

252-374-685

No

Yes

(Circle one)