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Physical Activity and Exercise during Adolescence and Young Adulthood Can Act as Preventative Measures against the Development of Osteoporosis in Elderly People

Christine C. Center
Wayne State University, ck8856@wayne.edu

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Dr. Qin Lai

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Introduction

"It was the best of times, it was the worst of times, it was the age of wisdom, it was the age of foolishness, it was the epoch of belief, it was the epoch of incredulity, it was the season of Light, it was the season of Darkness, it was the spring of hope, it was the winter of despair, we had everything before us…" are the famous lines the begin Charles Dickson's *A Tale of Two Cities* (1895). These words may feel applicable to an individual at any stage of life, but none more than the college student. College experiences are filled with wonder, love, failure, triumph, and connections to students and Professors. The few (or many) years spent at a University are ones which contain much formation for students. Students discover who they are and how to thrive as who they have become in this complicated world. Careers and successful habits stem from this time of life.

It's easy to become very wrapped up into the "moment" of college and to only focus on the here and now. College may be preparing us for our future in regard to work and a career, but how is this time preparing students physically for the future? Advancements in media systems allow us to be informed of diseases and medical complications almost as soon as they are discovered. It is overwhelming as a female to hear about breast cancer, cervical cancer, colon, cancer, heart disease, obesity, diabetes, and hypertension. Avoid this food, don't smoke, and watch your midline and so many more pieces of advice to follow bombard us each day.

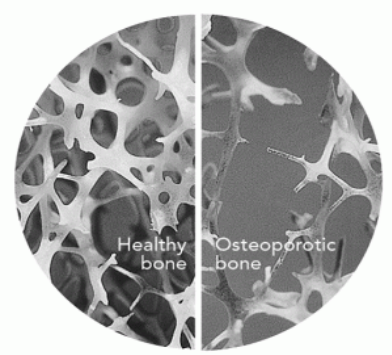
We can't possibly follow all the advice on how to live the perfect pain-free and disease-free life, but each day we can become a little more educated or change a small habit and thus make impactful changes in our lives.

As a female myself and a Kinesiology major, I have taken a special interest in one particular disease often called "the silent killer." This disease is osteoporosis. Through the course of this paper I will provide information on osteoporosis in general, methods of diagnosis, risk

factors, complications of, treatment methods, focus on the idea that physical activity and exercise during adolescence and young adulthood can act as preventative measures against the development of osteoporosis in elderly people, and conclude with information applicable to the college aged female.

Overview of Osteoporosis

To begin, what exactly is osteoporosis? Osteoporosis is the most common bone disease.[2] The word osteoporosis literally means "porous bones" .[1] A loss of bone density or mass results from the body breaking down bone faster than it is rebuilding it. This breakdown of bone mass is due to a disruption in osteoclasts and osteoblast activity. Bone formation involves work of osteoblast cells which lay down new bone and osteoclasts which reabsorb old bone.[7] This disturbance in bone formation leads to bone which is fragile and "porous." The picture below illustrates how bone looks in healthy bone compared to osteoporotic bone.



http://www.aont.org.au/osteoporosis/what-is-osteoporosis/

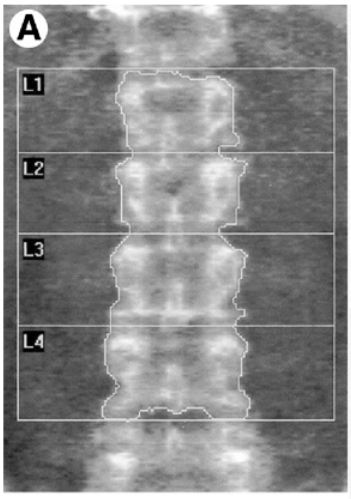
Diagnosing Osteoporosis

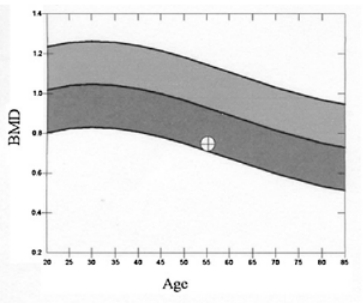
Osteoporosis quite often is not diagnosed until preventative measures are difficult to implement without further damage. How can osteoporosis be diagnosed? Osteoporosis can be diagnosed in multiple ways, typically through the evaluation of bone mass density (BMD). General radiographs of the lumbar spine were initially used to detect changes of bone density before bone densitometry testing was available.[4] Author Kenneth L. Bontrager shares on page

581 from his book *Textbook of Radiographic Positioning and Related Anatomy* "A loss of 30% to 50% of trabecular bone may produce the first visible changes on radiographs."[4] This method of detection identifies osteoporosis very late; progression of the disease is already in full swing. Today, the most common and criterion standard for assessing BMD is dual-energy x-ray absorptiometry (DEXA or DXA).[1,7] The ability to detect small percentages of bone loss while only exposing the patient to a very low radiation dose makes DEXA favorable.[1] Article *Osteoporosis Update* sates that a DEXA scan produces a radiation dose which is "90% less than a standard chest x-ray."[7]

The *Textbook of Radiographic Positioning and Related Anatomy* explains on page 583 that DEXA incorporates energy pulsing and rare earth filters to distribute a high and low x-ray energy beam to access values of bone within soft tissue. A bone mineral report is created after exposure. Bone mineral reports include the image created by radiation exposure, BMD values compared to age, sex, and ethnicity of the individual, and quality control data.[4] Below is a sample report from a DEXA examination which includes a scan of the lumbar spine, BMD plotted compared to a reference range, BMD values of each vertebrae, and T and Z-scores.[5]

Dual-energy x-ray absorptiometry





DXA Results Summary:

Region	Area (cm^2)	BMC (g)	BMD (g/cm^2)	T - score	PR (%)	Z - score	AM (%)
L1	11.67	7.68	0.658	-2.4	71	-1.5	80
L2	12.77	9.98	0.782	-2.2	76	-1.2	86
L3	15.55	12.16	0.782	-2.7	72	-1.6	81
L4	16.79	12.67	0.754	-3.3	68	-2.1	76
Total	**56.78**	**42.49**	**0.748**	**-2.7**	**71**	**-1.6**	**81**

Results from bone densitometry tests provide a Z-score and a T-score. Z-scores and T-scores represent the number of standard deviations (SD) of the subjects' BMD from that of the respective reference population.[6] "The Z-score is based on the difference between the individuals' measured BMD and the average BMD of a population that is the same sex, age, and ethnicity as the individual." [7] Premenopausal women less than 50 years old typically utilize the Z-score for assessment of bone density.[7] T-score is popular for postmenopausal women. "The T-score represents the number of standard deviations from peak bone mass that is achieved in early adulthood."[7] The World Health Organization (WHO) suggested in 1994 that BMD should be used to diagnosis osteoporosis.[4] WHO created a table which categorizes osteoporosis and osteopenia into four different diagnoses based on T-scores.

TABLE 1

World Health Organization criteria for diagnosing osteoporosis using bone density measurements

CATEGORY	T SCORE
Normal	Not more than 1.0 standard deviations (SD) below the young adult mean
Osteopenia	Between 1.0 and 2.5 SD below the young adult mean
Osteoporosis	More than 2.5 SD below the young adult mean
Severe or established osteoporosis	More than 2.5 SD below the young adult mean with a fracture

http://www.clevelandclinicmeded.com/medicalpubs/ccjm/Jan06/watts.htm

Osteopenia is referred to in this table and thus is owed a brief description. The Greek words "osteo" and "penia" means "bone and poverty" when combined. Weakened bone with a lower BMD than normal places someone in the osteopenia category. Individuals with osteopenia can be considered on their way to osteoporosis; they are an increased risk of fracture due to their low BMD, but they may just naturally have this low BMD and not actually be losing BMD.[3]

Risk Factors of Osteoporosis

Besides tests to determine BMD, studies and research has been performed in order to compile a list of factors which affect an individual's risk of having osteoporosis. These risk factors typically are placed in two categories; modifiable risks and fixed risks. Modifiable risk factors, as their name implies, are factors which an individual can change because they tend to be related to lifestyle decisions. A majority of modifiable risks can progress to a decrease in BMD, or they increase the chance of fracture risk in a non-biological fashion.[1] Some of these modifiable risk factors include alcohol consumption, smoking, vitamin D deficiency, eating disorders, low calcium intake, and poor nutrition.[1] Alcohol consumption typically is not the first risk factor which may come to mind when discussing osteoporosis, but interestingly enough "alcohol intake is detrimental to bone health and increases the risk of falling."[6] Smoking may also increase an individual's risk for osteoporosis by decreasing calcium absorption and possible lowering estrogen levels.[7]

Risk factors related to osteoporosis which cannot be changed are termed fixed risks. These risks include gender, age, family history of osteoporosis, ethnicity, and personal history of fracture.[1] Although osteoporosis is a concern for men, women are four times as likely to develop osteoporosis than men.[9] Women typically start with a lower bone mass, as compared to men, and experience a sudden drop in estrogen at menopause; osteoporosis classically results from this estrogen upset. Estrogen is a key player in bone turnover; it inhibits bone reabsorption.[7] As gonadal function diminishes with aging, estrogen supplies decrease which leads to the body reabsorbing in excess which leads to accelerated bone loss.

Osteoporosis seems to be caused simply by getting older. Bontrager on page 581 states "By the age of 20 years, the average woman has acquired 98% of her skeletal mass. Therefore,

building bone mass during child and teenage years can be the best defense against this disease as an adult. Typically by the age of 35, more bone is removed than replaced, resulting in a gradual decrease in bone."[4] Bone mass built during younger years can delay the onset of osteoporosis.

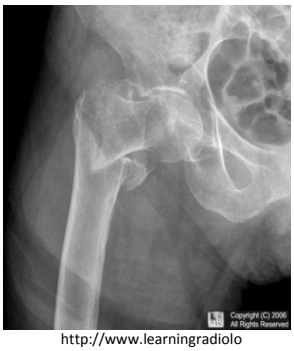
Family history of osteoporosis and fractures is a very important risk factor. An in-depth look is being taken at "the potential significance of genetics on the pathogens of the disease.[6] This and the rest of the risk factors mentioned are evaluated by risk factor assessment tools and algorithms so that clinicians can approximate an individual's fracture risk. Website www.shef.ac.uk/FRAX provides the algorithm known as FRAX which is an acronym for "Fracture Risk Assessment Tool."[6] World Health Organization developed FRAX which is one of the newest and most recognized risk assessment algorithms.[6] The most complete evaluation for osteoporosis would be a combination of BMD with determined risk factors.

Complications of Osteoporosis

We have briefly reviewed what osteoporosis is, how it is diagnosed, and risk factors; but what makes osteoporosis a topic of which to be concerned? The major concern of osteoporosis is the number of fractures that are a product of this serious disease. Every three seconds someone in the world is fractured as a result of osteoporosis. The International Osteoporosis Foundation enlightens that "osteoporosis causes more than 8.9 million fractures annually." After the age of 50, one in three women will suffer from osteoporotic fractures.[1] These fractures are costly. The cost of osteoporosis-related fractures was approximately seven billion in 2005 and is extrapolated to reach at least $25 billion by the year 2025.[6]

Osteoporosis is the culprit responsible not only for fractures, but also an increase in morbidity and mortality. Levine explains "Hip fractures have been shown to result in 10-20

percent excess mortality within 1 year, and also lead to a 2.5-fold increase in risk of future fractures."[6]



http://www.learningradiolo

Treatment for Osteoporosis

A brief review of osteoporosis, how it is diagnosed, risk factors, and concerns for osteoporosis victims leads next to the question of what can be done to prevent or treat osteoporosis. One method of treatment for osteoporosis is medication (drug-therapy). The goal of medications intake is to decrease the amount of fractures. History of fracture, T-scores $\leq$ -2.5, and the presence of multiple risk factors would typically be prompts to a physician to consider medication for a patient.[7] However, medications as a method of treatment should only be considered after the possible underlying causes of osteoporosis, such as lifestyle factors, have been considered as well as other treatment options attempted have been deemed unsuccessful. [7]

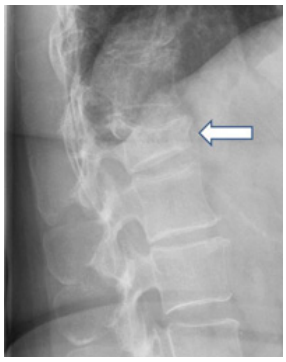
Four medication types are typically involved with osteoporosis treatment including bisphosphonates, Estrogen therapy, parathyroid hormone, and Calcitonin. Entire papers could be written on each type of medication mentioned, but I will only provide a brief description of each type. Bisphosphonates have proven to be effective, safe, and inexpensive and thus is considered a first-line medication choice for osteoporosis treatment.[7] Bisphosphonates bind to calcium and then are consumed by osteoclasts, causing inactivation of these "bone-breakdown" cells. Bone mineralization improvement, bone remodeling decrease, and the structure of trabecular and cortical bone is maintained are some positive results credited to bisphosphonates.[7]

Bisphosphonates, as well as estrogen hormone therapy, are considered anti-resorptive agents because they preserve bone mass density by slowing bones natural reabsorption process.[1] Estrogen also blocks osteoclast doings. Benefits of estrogen therapy include decreased fracture rate by approximately 30%.[7] Hormone replacement therapy, although a first-line treatment, is in the process of being researched and improved because hormone therapy comes with its fair share of risks and side effects including breast cancer and coronary heart disease.[7]

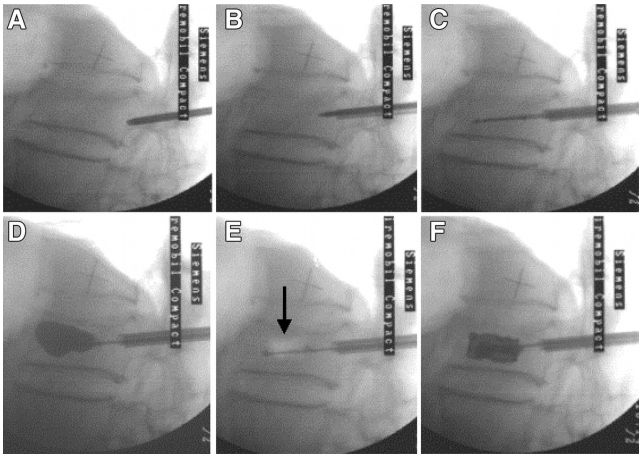
Parathyroid hormone therapy is another medication type. Osteoblasts are stimulated by the parathyroid hormone, thus improving bone mass density. This increase in bone mass density due places the parathyroid hormone in the category of anabolic agents.[1] The last mentioned medication is Calcitonin therapy which can reduce bone pain.[9] Calcitonin prevents bone reabsorption by weakening osteoclast activity. Parathyroid hormone and Calcitonin are both not used as a first-line of treatment because other medications have been found to be more effective in treating osteoporosis.[8] All medications involve some side effect; constant reassessment from a physician should occur to re-evaluate medication needs.

Surgical interventions are also options for treating osteoporosis. Spinal-fusion by metal-rods, vertebroplasty, and kyphoplasty are common procedures performed. "About 700,000 cases of compression fractures due to osteoporosis occur each year in the United States."[10] These compression fractures my go unnoticed at first because they do not occur as a results of trauma, they do not injure the spinal cord, and they may not be painful.[9] Compression fractures of multiple vertebrae can lead to decreased height and deformity of the back which resembles a hunchback. This hunch back appearance is referred to as a dowager's hump, or more commonly kyphosis.[9] Vertebral bodies which should appear square, now appear angled on x-ray.

As listed earlier, kyphoplasty is one method to correct kyphosis. Surgeons gain access into the fractured vertebra and place a balloon inside. This balloon is then inflated with decompressed the vertebra. Bone cement is next poured into the vertebra. This procedure can be thought of as an internal cast; vertebral height is restored which reduces deformity and any pain of the individual.[10] X-ray images below demonstrate compression fractures of vertebrae, and the regained vertebral body height after kyphoplasty is performed.[11] Other surgery methods differ in approach, but share the same goal of pain reduction and height retained.



http://orthoinfo.aaos.org/topic.cfm?topic=A00538



Nutrition also plays a role in promoting bone health. It would be a challenge to find an article, study, or textbook about osteoporosis which didn't mention calcium and vitamin D. Calcium slows the rate of bone reabsorption by osteoclast, which helps to maintain bone mass density.[7] Fruits, vegetables, supplements and fortified foods are all sources of calcium. Vitamin D works closely w calcium assisting with absorption.[6] Vitamin D is obtained through sunlight, fortified foods, and supplements. The third and final method of prevention of osteoporosis will be the focus of this paper.

Exercise and Osteoporosis

Physical activity and exercise during adolescent and young adult ages can act as preventative measures against the development of osteoporosis in elderly people. The terms "physical activity" and "exercise" tend to be stated in health and medical literature and conversation as substitutable terms, however, these terms do in fact have distinct differences. The American College of Sports Medicine provides definitions for these terms. Physical activity is first described as "any bodily movement produced by the contraction of skeletal muscles that result in a substantial increase over resting energy expenditure."[12] Next, exercise is defined as "a type of physical activity consisting of planned, structured, and repetitive bodily movement done to improve or maintain or more components of physical fitness."[12] This definition of exercise leads to the need of defining physical fitness; "a set of attributes or characteristics that people have or achieve that relates to the ability to perform physical activity."[12] So although the term "physical activity" is often used in reference to normal activities of daily living and "exercise" acts as a subset relating towards sports or activates consciously chosen to receive physiological and physiological benefits; for the sake of this paper, the terms will be used interchangeably unless otherwise indicated.

The phrase "exercise prevents osteoporosis" is commonly quoted among the sport, exercise, and health communities, but how exactly does this relationship work? Physiologically, how does exercise impact bone growth? Exercise effects bone growth directly and indirectly. Bones contain cells and tissues which are living and interacting and complex.[13] Bone growth occurs by three cellular processes: hyperplasia, hypertrophy, and accretion.[14] Hyperplasia refers to an increase in the amount or quantity of cells, hypertrophy refers to the increase in the size of the cells, and accretion entails an increase in intercellular materials.[14]

Exercise can directly influence bone growth by stimulating osteogenesis. Compressive forces generated from exercise are essential for osteogenesis. Strain on bone, as a result of exercise, also helps to increase bone strength and cortical bone enlargement.[14] Benefits of exercise on bone include increased mineralization, higher bone density, and enlarged bones which are less likely to fracture .[14] Exercise is "…fairly efficient in promoting bone acquisition…"[14] Bones adaption to mechanical stress placed on them from exercise is explained by the mechanostat theory. Our bodies are constantly adjusting to meet new demands. After initial bone growth which occurs during normal development of human beings, bones remain ready-for-action against mechanical tension or compressive forces which cells must organize and grow and adapt to resist. These forces on bones encourage bone modeling and remodeling processes of osteoclast and osteoblast cells which than influence bone strength according to the mechanostat theory.[14] Osteogenesis stimulation results from this mechanostat-related mechanics. Increasing the demand on bones with exercise leads to bone modeling. Bone modeling "increase mechanical bone resistance through the osteoblastic deposit and mineralization of bone, as well as improving the cortical and trabecular (internal) architecture with a new gain of bone mass.[14]

Exercises' indirect influence of bone mass density relates to muscle mass. Increased muscle mass and muscle strength is a consequence of exercise. Bone acquisition can occur by hypertrophied muscles creating a greater force on bones at sites of attachment. Muscle mass growth has been found to be a good indicator of bone mass density independent of exercise.[14] This relationship of muscle growth and bone growth can also be understood by previously mentioned mechanostat theory: "bigger muscles exert higher tensile forces on the bones they attach."[14] Although research supports this method as a means of increasing bone mass density,

mechanical loading directly influencing bone development has a larger impact on bone mass density increase.[14]

Often exercise in post-menopausal women as a means of maintaining bone mass density is given much attention; but what about building bones mass density in younger years as a method of prevention? When is the most valuable time to incorporate exercise into one's lifestyle as an approach to increase bone mass density and thus, hopefully, deter osteoporosis in later years? Research agrees that maximizing bone mass density is ideal during childhood and adolescence, especially the two to three years surrounding puberty (typically ages 11-13 for girls and 12-14 for boys).[14,15,16] These particular years surrounding puberty consist of intense bone mineral accrual and contribute a large percentage of adult peak bone mass. It is agreed that much bone growth happens at this time, but the percentage of growth occurring varies immensely from study to study. One study stated at least 25%, Another claimed up to 60%, and other studies' data fell between these extremes.[14,15,16]

Research studies on Exercise and Osteoporosis

On May 28, 2013 The American Academy of Pediatrics published an article titled *A 4-year Exercise Program in Children Increases Bone Mass Without Increasing Fracture risk.*[17] This 4 year study included pre-pubertal boys and girls (ages 7-9 years) divided into an intervention group and a control group. The intervention group consisted of 446 boys and 362 girls, while the control group contained 807 boys and 780 girls. Baseline bone mass density was similar for all participants at the beginning of the study. Four elementary schools participated in this study, only one of the four schools was chosen as the intervention school. The four intervention schools provided school physical education 40 minutes per day (200 min per week) and the control schools continued their typical routine which was 60 minutes of physical

education total per week.[17] Climbing, jumping, running, and ball games were included in the physical education sessions and taught by teachers that taught at the schools.

DEXA was used to assess mineral content and bone width. A sampling of children from both the intervention and control groups were assessed before the study began, and then in that same month for 4 consecutive years of the study. Lifestyle choices related to diet and physical activity and other factors were reviewed by use of a questionnaire given to the children in both groups. Results from this study found that the intervention group, by participating during key years of growth in moderately intense exercise, could increase bone mass and size as compared to the control groups. Data of both girls and boys was provided, but only looking at data from the girls participating in the intervention group showed an increase of bone mass by 7.0% in the lumber spine, 4.1% in the femoral neck, 8.7% in the trochanter, and 0.7% in the third lumbar vertebra compared to the girls in the control group.[17]

TABLE 3 Baseline Data and Annual Changes in the Subsample of Girls and Boys Who Were Chosen for Measurements, Presented as Absolute Values, Evaluating the Effect of 4 Years of Exercise Intervention in Anthropometry, Bone Mineral Parameters, Bone Size, and HSA in the Exercise Intervention Group and the Control Group

	Girls					
	Baseline			Annual Changes		
	Cases (n = 48)	Controls (n = 48)	P Value	Cases (n = 48)	Controls (n = 48)	P value
Age	7.7 (7.5–7.8)	7.9 (7.7–8.1)	.08	—	—	
Anthropometrics						
Wt, kg	27.8 (26.2–29.5)	27.3 (25.6–28.9)	.63	4.4 (3.9–4.8)	3.7 (3.3–4.0)	<.05[a]
Height, cm	128.3 (126.7–129.9)	129.1 (126.8–131.4)	.57	6.2 (5.9–6.5)	6.1 (5.9–6.4)	.81
Lean mass, kg	19.9 (19.2–20.6)	20.2 (19.4–21.0)	.54	2.6 (2.4–2.8)	2.5 (2.3–2.7)	.20
Fat mass, kg	5.5 (4.3–6.6)	5.2 (4.2–6.2)	.73	1.5 (1.3–1.8)	0.9 (0.7–1.1)	<.001[a]
Percentage body fat, %	18.0 (15.3–20.7)	17.8 (15.6–19.9)	.89	1.3 (1.0–1.7)	0.6 (0.2–1.1)	<.01[a]
BMC, g						
Total body	943.6 (899.4–987.8)	931.8 (877.9–985.7)	.73	179.6 (160.5–198.7)	166.4 (149.4–183.4)	<.05[a]
LS	29.4 (27.2–31.6)	29.7 (27.8–31.5)	.84	9.1 (7.9–10.3)	7.1 (6.1–8.0)	<.01[a]
FN	2.6 (2.4–2.8)	2.7 (2.5–2.9)	.60	0.39 (0.33–0.45)	0.28 (0.23–0.33)	<.01[a]
Trochanter	2.5 (2.2–2.7)	2.6 (2.3–2.8)	.67	0.92 (0.82–1.02)	0.72 (0.62–0.83)	<.01[a]
Bone width, cm						
Third lumbar vertebra	2.9 (2.8–2.9)	2.9 (2.8–3.0)	.48	0.13 (0.11–0.14)	0.11 (0.09–0.12)	<.01[a]
FN	2.4 (2.4–2.5)	2.5 (2.4–2.6)	.31	0.15 (0.12–0.17)	0.10 (0.08–0.13)	<.01[a]
HSA						
CSA, cm^2	0.92 (0.87–0.98)	0.89 (0.84–0.94)	.37	0.11 (0.10–0.13)	0.09 (0.07–0.11)	<.05[a]
Z, cm^3	0.28 (0.26–0.31)	0.27 (0.24–0.29)	.30	0.06 (0.05–0.06)	0.05 (0.04–0.06)	<.05[a]
CSMI, cm^4	0.35 (0.31–0.39)	0.34 (0.30–0.38)	.54	0.10 (0.08–0.11)	0.08 (0.07–0.09)	<.05[a]

Emphasis was placed on these findings not being accompanied by an increase fracture risk for those children who had increased their amount of physical activity. Bone size enlargement resulting from physical activity was also a highlighted finding: "[bone size] contributes to bone resistance to fracture, independently of bone mass."[17] This is an intriguing discovery because spine fractures tend to be present in women with smaller lumbar vertebra. [17]

A Finnish study titled *Long-Term Leisure-Time Physical Activity Has a Positive Effect on Bone Mass Gain in Girls* was published online November 23, 2009 by the American Society for Bone and Mineral Research.[18] This study was a longitudinal study of seven years which was dedicated on Finish girls. These girls were ages 10-13 years old at the beginning of the study, and totaled 202 in number. Over the course of the seven years, self-administered questionnaires were used to assess leisure time physical activity (LTPA). Based on results of the questionnaire, four groups were created which places the girls into consistently high activity (G_{HH}), consistently low activity (G_{LL}), changed from high to low activity (G_{HL}), or changed from low to high activity (G_{LH}) groups. Evaluation of physical performance was accomplished by measuring left knee extensors strength by way of dynamometer of isometric muscle strength.[18] A vertical counter movement jump was also used to assess explosive performance capacity of the lower limbs. DEXA was utilized to assess bone mineral content and bone mass density. At baseline no significant differences were seen between the four groups. At the conclusion of this seven year study, girls (now 18 years old) in the G_{HH} group were seen to have a higher amount of lean mass, higher maximal force in knee extension, higher jumping heights, as well as, higher BMC and BMD in comparison to girls within the other three groups. The study found that "long-term LTPA has a positive effect on peak bone mass gain of multiple bone sites in girls from the age of

11 to 18 years" and that "increasing physical activity from a low level during puberty promotes bone accrual at multiple bone sites."[18]

Table 1. Physical Characteristics of the Girls at 84-Month Follow-up According to Consistency of LTPA During Puberty

	High-high ($n=50$)	High-low ($n=48$)	Low-high ($n=51$)	Low-low ($n=53$)	ANOVA p	Post hoc					
						HH-HL	HH-LH	HH-LL	HL-LH	HL-LL	LH-LL
Age (years)	18.3 ± 1.1	18.3 ± 1.3	18.3 ± 1.1	18.5 ± 1.0	.493			N.S.			
Height (cm)	166 ± 5.5	165 ± 4.9	167 ± 6.1	165 ± 6.1	.051			N.S.			
Weight (kg)	60.0 ± 8.9	59.3 ± 9.5	61.4 ± 9.1	58.0 ± 8.5	.222			N.S.			
BMI (kg × m^{-1})	21.7 ± 2.9	21.9 ± 3.2	21.9 ± 2.6	21.3 ± 2.6	.804			N.S.			
Fat mass (kg)	18.4 ± 7.4	19.6 ± 7.0	18.7 ± 6.8	18.8 ± 5.9	.659			N.S.			
Lean mass (kg)	39.0 ± 3.3	36.7 ± 4.2	39.8 ± 3.9	36.4 ± 3.6	<.001	.012		.004	<.001		<.001
Bone Mass (kg)	2.5 ± 0.4	2.5 ± 0.4	2.5 ± 0.4	2.3 ± 0.4	.051			N.S.			
Menarche age (years)	13.1 ± 1.2	12.8 ± 1.1	13.0 ± 1.3	12.9 ± 1.3	.534			N.S.			
Maximum force KE (Nm)	404 ± 86.1	351 ± 93.1	408 ± 85.4	383 ± 87.9	<.01	.018			.009		
Jumping height (cm)	23.9 ± 5.2	21.0 ± 4.0	23.4 ± 5.7	21.3 ± 3.6	<.01	.022		.044			
LTPA score	123 ± 66.9	22.0 ± 13.0	137 ± 102.1	19.3 ± 13.7	<.001	<.001		<.001	<.001		<.001
PIA (h/day)	17.6 ± 3.0	17.8 ± 2.6	17.4 ± 2.9	18.1 ± 3.2	.587			N.S.			
Total Ca (mg/day)	1340 ± 521	1191 ± 511	1484 ± 623	1189 ± 494	.378			N.S.			

Values are given as mean ± SD. PIA = physical inactivity; KE = knee extension.

Table 1 shows no difference of data within the participants of the four groups, and differences of the groups when compared to each other. Many studies have been conducted to examine exercise' impact on bone growth, however, this particular study mentioned is significant because of its length (seven years) and due to the fact that it was conducted in a longitudinal manner.

Best prevention method for Osteoporosis

So far the terms "physical activity" and "exercise" have been referred to very vaguely; obviously these terms may be used in reference to a wide variety of pass times or sports. Is there a particular type of exercise or physical activity which proves to be superior in BMD, structure, or mineral content improvement?

Boys and girls that are regularly active can achieve 9-17% higher bone mineral content than boys and girls that are inactive.[16] Article *Osteoporosis Update* provides a table of exercises which have been proven to positively influence BMD; these exercise types include high-impact

loading activities, changing, diverse or novel loading angle, weight-bearing activities, and activities which directly impact bone.[7]

Table 4. Exercise Categories and Examples

Category of Exercise	Example
High impact, rapid forceful loading	Running, jumping, gymnastics, volleyball
Changing, diverse, or novel loading angles	Ball sports, gymnastics
Weight-bearing, high forces	Dancing and weight lifting
Direct impact of the bone of interest	Dominant arm of tennis players

Classified as high-impact activities are many of the sports children participate in such as running, ice-skating, volleyball, ballet, gymnastics, soccer, swimming.[16] These activities have been found to somewhat improve BMD. Running for example, although one would assume benefits from its high-impact nature, tends to produce athletes with lower BMD as compared to athletes involved in other sports.[16]

Weight-bearing activities tend to "trump" other categories of exercise when it comes to BMD enhancement. "weight-bearing" is the term most often tied to exercise interventions for osteoporosis. For clarification of what is specifically meant by this term, the May Clinic defines weight-bearing activities as "aerobic exercises on your feet with your bones supporting your weight."[19] Examples of weight-bearing activities include dancing, jogging, brisk walking, and resistance training. Compared to high-impact loading activities, greater BMD increases of 1.2-5.6% have been seen in participants of weight-bearing activities.[16] "Although the most suitable sporting activity for maximal bone growth remains unknown, compelling evidence has accumulated to suggest that participation in weight-bearing sports is fairly efficient in promoting bone acquisition in prepubertal children".[14]

http://detroit.cbslocal.com/2011/12/31/12-ways-to-get-fit-with-your-kids-in-2012/

Nonweight-bearing activities will not aid in BMD increases as compared to weight-bearing activities. The Lumbar spine and lower extremities of nonweight-bearing athletes will be lower than weight-bearing activity athletes.[16] Swimming is an example of a nonweight-bearing activity. During the key BMD development adolescent years, promotion of swimming as a means of osteoporosis prevention for post-menopausal years does provide significant benefits. Swimming need not been totally discredited however, this activity can be used for elderly individuals with severe osteoporosis to improve aerobic capacity when high-impact or weight-bearing actives of too much intensity could be more of a risk than a benefit in regard to osteoporotic fractures.

Mentioned very rarely in connection with osteoporosis is flexibility activity for children. Although flexibility activities, stretching and yoga for example, do not increase BMD, they decrease chances of muscle injury.[19] A muscle injury could detain exercise for periods of time and prevent opportunities for BMD improvement, thus flexibility can be thought of as an accessory to increase MBD.

Use it or lose it?

The connections between exercise and osteoporosis prevention has been analyzed and it is evident that exercise during childhood does in fact improve MBC, BMD, and bone strength. What if now as college students we have not participated in forms of exercise in a few years? Are the bone benefits gained from childhood exercise still applicable, or can positive BMD changes gained be lost over time? Although many studies focus only on short term exercise

interventions and their positive effects and do not provide answers to this question; a few studies have been conducted over a long term basis.

The International Osteoporosis Foundation published an article online January 9, 2007 titled *The reduction of physical activity reflects on the bone mass among young females: a follow-up study of 142 adolescent girls.*[20] This study involved 142 female participants that varied in age from 9-15 years old. At baseline, basic physical measurements of age, height, weight and BMI were obtained, as well as, more sophisticated measurements such as BMD via use of a DEXA scanner. Over the course of this seven year study, measurements were again obtained at the three year mark and then again at the seven year mark.

Table 1 Characteristics of the study population (n=142) at baseline, in the 3-year follow-up and in the 7-year follow-up measurements and the baseline values of drop outs (n=49). Results are expressed as unadjusted mean values (SD), MET values are medians (first, third quartile)

	Baseline, Drop-outs	Baseline	3 years	7 years
Age, y	12.8 (1.7)	13.0 (1.8)	16.0 (1.8)	20.0 (1.8)
Height, cm	156.4 (10.2)	158.2 (9.2)	164.6 (6.5)	166.2 (6.3)
Weight, kg	45.1 (10.5)	47.4 (9.8)	55.3 (8.3)	60.0 (8.7)
BMI, kg/m^2	18.4 (2.7)	18.8 (2.5)	20.4 (2.5)	21.7 (2.8)
Tanner stage: 1	12	23	0	0
2	19	86	11	0
3	18	33	131	142
MET-index	32.0 (10.3, 68.0)	42.5 (12.4, 75.0)	25.5 (12.0, 75.0)	20.0 (8.8, 40.0)
Calcium intake, mg	1667.3 (573.3)	1550 (642)	1523 (577)	1116 (482)
Vitamin-D intake, µg	4.3 (2.1)	4.4 (2.1)	4.0 (2.2)	5.4 (2.6)
BMC_{LS}, g	38.9 (12.6)	41.11 (14,55)	54.67 (11.01)	59.45 (10.82)
BMD_{LS}, g/cm^2	0.808 (0.129)	0.832 (0.168)	0.965 (0.122)	1.002 (0.107)
BMC_{FN}, g	3.46 (0.75)	3.64 (0.85)	4.29 (0.74)	4.36 (0.70)
BMD_{FN}, g/cm^2	0.790 (0.116)	0.824 (0.153)	0.918 (0.147)	0.927 (0.143)

Tanner stages are presented in frequencies (1=prepubertal girls, 2=pubertal girls, 3=mature girls). There were no significant differences between drop outs and study participants.
MET=ratio of work metabolic rate. The MET-index was calculated by multiplying frequency, mean duration and mean intensity of weekly physical activity and divided by 60.
BMC=bone mineral contents
BMD=bone mineral density
FN=femoral neck
LS=lumbar spine

Data from this study found that girls must continue to be involved in exercise to ensure preservation of acquired bone mass even during adolescence![20] The article declares that "It is confirmed that exercise induced bone gain is not permanent without a sustained physical activity level during later life."[20] Physical activity reduction equal or greater than 50% in females studied showed to result in loss of MBC and MBD even before they turned 25 years old.[20] This is significant because the average female tends to not start losing bone mass through natural

aging processes until reaching 35-years of age.[4] So, in fact, research does show that we must remain activite, because if we don't "use it" we will "lose it."

Conclusion

Osteoporosis: the silent killer. Through the course of this paper I provided information on osteoporosis in general, methods of diagnosis, risk factors, complications of, treatment methods, and focused on the idea that physical activity and exercise during adolescence and young adulthood can act as preventative measures against the development of osteoporosis in elderly people. Much information was presented about osteoporosis, and. although we can't heed every piece of advice given in an effort to enhance our lives, each day we can become a little more educated or change a small habit and thus make impactful changes in our lives.

So what can we take away from this paper in regard to college-aged females and osteoporosis? Although the effects of osteoporosis may not be experienced until post-menopausal years, we must begin today in our quest to not be another statistic of an osteoporosis fracture. Beginning exercise interventions during adolescence to achieve increased BMD is key, as well as, maintaining an active lifestyle. If you didn't have the most active childhood, start now. Benefits from exercise will not be as outstanding in college years as they are during childhood, but they are still present so go and start exercising!

My Thoughts

When faced at the beginning of this semester with the daunting task of writing a twenty-page paper, it seemed unachievable. And yet, here we are at the end of it. Microsoft Word kept track that 83.5 hours were spent editing this paper along; that doesn't include hours of brainstorming, researching and meetings with Advisors and Librarians. I learned so much about

the topic of osteoporosis. As a female, Kinesiology Major, and Radiologic Technologist, the topic of osteoporosis seems the perfect fit for my research, and I found this topic of great interest. My appreciation for libraries increased, especially in regard to searching databases for references for this paper. This time also reminded me how far I have come during my Wayne State University journey, and about how much I can accomplish when I apply myself and put forth my best effort. Dr. Qin Lai, my adviser, was very reassuring during my frequent visits to his office this semester. During the process of writing this paper he was encouraging, affirming, and supportive and I would like to especially thank him.

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