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Title: *Saturated Fat Intake in Midwestern Older Adults and its Effect on Bone Mineral Density: A Correlational Study*

The accompanying research report is submitted to the University of Wisconsin-Stout, Graduate School in partial completion of the requirements for the

Graduate Degree/ Major: MS Food and Nutritional Sciences

Research Advisor: Karen Ostenso, R.D., Ph.D.

Submission Term/Year: Summer 2014

Number of Pages: 59

Style Manual Used: American Psychological Association, 6th edition

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Mathews, Tyler J. *Saturated Fat Intake in Midwestern Older Adults and its Effect on Bone Mineral Density: A Correlational Study*

Abstract

Over 52 million people in the United States had osteoporosis or low bone mass in 2005, accounting for over 17 billion dollars in health care and rehabilitation services from osteoporotic fractures. While nutrients such as calcium and vitamin D have been shown to play a role in bone health, other nutrients such as saturated fat intake have also been linked to bone health. The purpose of this study was to determine whether dietary saturated fat intake influences bone mineral density in older adults. The secondary data was collected from a previous UW-Stout Collaboration Study conducted from September 2012 to May 2013, which included food frequency data and spine and pelvic bone mineral density measurements. Though not significant, a correlation analysis indicated that there was a positive relationship between saturated fat intake and spine and pelvic bone mineral density. The sample size was a limiting factor of this study; therefore future studies may require a larger sample size in order to find significance. Saturated fat may influence bone health by impacting bone mineral density, however its true relationship to bone mineral density remains uncertain. Therefore it is important to continue research on saturated fat and its relationship to bone mineral density.

Acknowledgments

First and foremost, I would like to thank my thesis advisor, Karen Ostenso. Without her involvement, encouragement, and patience throughout this process, this research would be non-existent. I cannot thank her enough for the opportunities she provided for me to succeed throughout my time at UW-Stout. Lastly, I would like to thank all my family and friends who believed in me and supported me throughout this research, as well as all those who have helped me throughout this writing process.

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Chapter I: Introduction

Osteoporosis is a common problem in older adults that occurs when bone strength is compromised, increasing the risk of fracture in older adults (Ahmadiéh & Arabi, 2013). Both osteoporosis and osteopenia (low bone mass), are major concerns in the United States that lead to billions of dollars in health care expenditures every year (National Osteoporosis Foundation, 2014f; Diab & Watts, 2013; Nayak, Roberts, & Greenspan, 2008). According to the National Osteoporosis Foundation (NOF), in 2005, 52 million Americans had either low bone mass or osteoporosis which led to over 17 billion dollars in health care and rehabilitation service costs from osteoporotic fractures (NOF, 2014f; Nayak et al., 2008). The Markov state-transition model of osteoporosis is used to simulate the natural history of osteoporosis through fracture events and costs as patients transition across different health states (healthy, fracture, post-fracture, and death) caused naturally or from a hip or pelvic fracture (Burge et al., 2007). Burge et al. (2007) used this model to estimate that health care costs, in 2005, would be around 16.9 billion in the United States, along with 2.05 million osteoporotic fractures. By 2025, health care costs and the amount of osteoporotic fractures are expected to increase 48% (Burge et al., 2007).

Looker, Melton III, Harris, Borrud, and Shepherd (2010) investigated the prevalence of low bone mineral density using the National Health and Nutrition Examination Survey (NHANES) 2005-2006 questionnaire. Over five million adults, 50 years and older, were estimated to have osteoporosis at the femur neck, while 0.3 million were estimated to have it in the hip. This data is congruent with Burge et al.'s (2007) estimation of 0.29 million hip fractures among males and females, while femur estimates were unknown. The study by Looker et al. (2010) found that age was negatively associated with femoral and total hip bone mineral density, identifying age as a risk factor for osteoporosis.

The high prevalence of osteoporosis is not only associated with an increase in annual health care costs, but with a decrease in quality of life. Bianchi et al. (2005) identified postmenopausal women with osteoporosis as having a significant reduction in physical function, social function, and general health perception. Pain was found in half the subjects, with 26% of the subjects experiencing pain for over ten hours each day (Bianchi et al., 2005). Other characteristics associated with osteoporosis included depression or the development of a poor sense of well-being (Bianchi et al., 2005).

Hallberg et al. (2004) found a significant reduction in quality of life (physical function, bodily pain, and social function) in older women (55 to 75 years) within the first 83 days of developing a forearm, humerus, vertebral, or hip fracture. After a two year follow up, those who experienced two or more fractures showed a greater reduction in their overall quality of life, compared to the subjects who only experienced one fracture (Hallberg et al., 2004). Hallberg et al. (2004) further found a relationship between bone mineral density and quality of life. Subjects classified as osteoporotic based on their T-score (<-2.5) indicated a reduction in physical function and vitality (Hallberg et al., 2004; Diab & Watts, 2013). Hip, vertebral, and wrist fractures resulting from osteoporosis were further associated with a significant reduction in quality of life in older adults (Borgstrom et al., 2006). Of the three fracture sites, Borgstrom et al. (2006) identified hip and vertebral fractures to have the greatest impact on quality of life right after the onset of the fracture. Similarly, Hallberg et al. (2004) also found a greater reduction in quality of life directly after a hip or vertebral fracture. The studies by Borgstrom et al. (2006) and Hallberg et al. (2004) both indicate the severity a hip or vertebral fracture has on the individual's quality of life.

There are many risk factors associated with osteoporosis and they are similar among men and women, risk factors are considered as either uncontrollable or controllable (NOF, 2014a). Uncontrollable risk factors for osteoporosis, listed by the National Osteoporosis Foundation include: gender, being over the age of 50, family history of disease, low body weight, the loss of height, and broken bones (NOF, 2014a; NOF, 2014d). Controllable risk factors include: poor calcium and vitamin D intake, poor fruit and vegetable intake, high consumption of protein, sodium and caffeine, inactive lifestyle, smoking, high alcohol consumption, and weight loss (NOF, 2014a; NOF, 2014d). Low testosterone and estrogen levels have also been linked to the development of osteoporosis in both men and women (NOF, 2014d). Similarly, Machlaughlin, Sleeper, McNatty, and Raehl (2006) indicated that physical frame, genetics, lifestyle, hormonal changes, and medication effects should all be taken into account as factors that can increase risk for fractures in the elderly.

Many of the controllable risk factors are directly associated with the diet, such as calcium and vitamin D intake. Twenty-nine reviewed random trials from 1978 to 2006 showed that calcium supplementation, with or without an added vitamin D supplementation, was an effective treatment for preventing osteoporosis (Tang, Eslick, Nowson, Smith, & Bensoussan, 2007). Despite the vast amount of evidence linking calcium and vitamin D to having a beneficial role for the prevention of osteoporosis, it still remains an issue for millions of Americans leading to over billions of dollars in health care costs (NOF, 2014f; Nayak et al., 2008). While most research has examined calcium and vitamin D and its relationship to bone loss, the diet has not been studied in its entirety (Macdonald, New, Golden, Campbell, & Reid, 2004).

Research has suggested a possible relationship between of dietary fats and their impact on bone health from high fat diets, unsaturated fats, and saturated fats. The evidence varies

between types of fat studied as well as the total amount of fat. Kato et al. (2000) found that an intake of ≥ 75 g of fat was associated with a 23% greater risk of all fracture types in postmenopausal women. Animal studies identified that a high fat diet was linked to a reduction in bone mineral density (Bielohuby et al., 2010; Parhami et al., 2001).

Dietary unsaturated fats have also been shown to negatively affect bone health. Martinez-Ramirez, Palma, Martinez-Gonzalez, Delgado-Martinez, de la Fuente and Delgado-Rodriguez (2007) and Macdonald et al. (2004) found a negative relationship between dietary polyunsaturated fatty acids (PUFA) and bone health in older adults. Macdonald et al. (2004) further identified a negative effect on femoral neck bone mineral density from monounsaturated fatty acids (MUFA), while Hogstrom, Nordstrom, P., and Nordstrom, A. (2007) found a similar negative relationship for total body bone mineral density. However, findings indicating negative effects from unsaturated fats on bone health are not unanimous throughout research.

There is also evidence that conflicts with the findings of Martinez-Ramirez et al. (2007), Macdonald et al. (2004), and Hogstrom et al. (2007), showing that unsaturated fats may positively influence bone health. Elderly women showed an improvement in bone mineral density at the lumbar spine and total body when dietary PUFA intake increased (Jarvinen et al., 2012). Likewise, Hogstrom et al. (2007) identified a positive relationship between peak total body and spine bone mineral density and PUFA concentrations among young, healthy men.

Research regarding saturated fat intake and bone health has provided conflicting results, which is similar to research conducted on unsaturated fats. Corwin, Hartman, Maczuga, and Graubard (2006) found that saturated fats reduced bone mineral density in humans, while Orchard et al. (2010) associated saturated fat intake with a higher risk of hip fracture. However, Macdonald et al. (2004) and Martinez-Ramirez et al. (2007) both found no known association

between saturated fat and bone health. The inconsistent study results on the relationship between dietary fats and bone health indicate the need for further research in this area.

Statement of the Problem

Osteoporosis is a growing problem around the world. Management of osteoporosis has been difficult. More research needs to be conducted in order to better understand effective methods to manage osteoporosis. Of interest to this research is the investigation of dietary management of osteoporosis, specifically related to saturated fat intake. Recently, there have been a number of research studies investigating the link between dietary fats and bone health (Corwin et al., 2006). Research has shown that both mice and humans with osteoporosis have more adipocytes in their bone marrow compared to osteogenic cells (Parhami et al., 1999). A number of researchers have looked at the relationship between high fat diets and its impact on bone mineral density, while few have specifically looked at the impact of saturated fat.

Research has indicated that a diet high in saturated fat is linked to an increased risk in bone disease (Corwin et al., 2006). Postmenopausal women developed a higher risk of hip fracture when a higher saturated fat intake was consumed compared to a lower intake (Orchard et al., 2010). A higher saturated fat intake was negatively associated with a decrease in certain bone mineral density sites in men (Corwin et al., 2006). Conversely, women showed no association between saturated fat intake and bone mineral density (Corwin et al., 2006). The high prevalence of osteoporosis in the United States along with the high costs of management, diminished quality of life, and the limited research on saturated fat and its impact on bone health, points to the need for further examination of saturated fat's role in bone health.

Purpose of the Study

The purpose of this study was to determine whether dietary saturated fat intake influences bone mineral density in older adults. This study used secondary data from a UW-Stout collaboration study, which included: demographic data, de-identified food frequency questionnaire data files, and bone mineral density measurements taken at the spine and pelvis. This study examined the relationship between bone mineral density and saturated fat intake, and asked whether a high saturated fat intake influences bone mineral density at the spine and pelvis in order to provide data for future research in this area of study. The research question asked was:

What impact does total dietary saturated fat intake have on bone mineral density in older adults?

Assumptions of the Study

This study was based on a number of assumptions. It was assumed that the food frequency questionnaires were answered accurately, and that bone mineral density scores were measured properly from a calibrated source. Finally, all subjects involved were assumed to be healthy individuals, and that bone mineral density data was not influenced by any known medical conditions.

Definition of Terms

Adipocytes. Fat cells (Parhami et al., 1999)

Bone mineral density (BMD). The National Osteoporosis Foundation defines bone mineral density as the “average concentration of minerals in your bones” (National Osteoporosis Foundation, 2014b).

C57BL/6 mice. Type of mouse that is sensitive to atherosclerosis (Parhami et al., 2001).

Fracture risk assessment tool (FRAX) or FRAX algorithm. Ten year absolute fracture risk assessment tool created by the World Health Organization. FRAX risk factors include: weight, history of fracture, parental hip fracture, cigarette smoking, current cortisone/prednisone use, secondary osteoporosis and alcohol use (Siris et al. 2011; McCloskey, 2009)

Interlukin-1. A protein made by white blood cells, primarily macrophages, that helps fight infections and helps leukocytes pass through blood vessels (National Cancer Institute, 2014a).

Low bone mass. Also called osteopenia (Diab & Watts, 2013).

Low-energy fracture. “Fracture produced by a same-level fall”, such as from slipping, tripping or falling while standing up (Martinez-Ramirez et al., 2007; Information Systems Security Association, 2014)

National Health and Nutrition Examination Survey (NHANES). The NHANES assesses large cross-sectional samples for health and nutritional status (Looker et al., 2010).

Odds ratio (OR). Used in the study by Martinez-Ramirez et al. (2007) to determine the outcome of a nutrient’s effect on fracture risk.

Osteopenia. Low bone mass. T-score between -1.0 and -2.5 (Diab & Watts, 2013).

Oxidatively modified LDL cholesterol (MM-LDL). A product of lipid and lipoprotein oxidation (Parhami et al., 1999).

Perimenopausal. The period of time when a woman is transitioning into menopause (Macdonald et al., 2004).

Population attributable for risk (PAR). Formula used to find the overall average risk, by dividing both the exposed and unexposed population’s average risk with the average risk of the unexposed (Levine, 2007). The formula that was used by Orchard et al. (2010): $PAR = p(HR -$

$1)/1+p(HR-1)$, where p represented the prevalence of the risk factor, and HR to represent the hazard ratio.

T-score. The T-score compares a person's bone mineral density score, using standard deviations, to a younger individual with normal bone mineral density. To be classified as having normal bone health, an individual must have a T-score of -1 and above. A T-score of -1 to -2.5 is classified as osteopenia, while a score of -2.5 or lower is classified as osteoporotic (Lee et al., 2013; Diab & Watts, 2013).

Women's Health Initiative. The Women's Health Initiative is a study conducted on postmenopausal women that looks to prevent and control common diseases, such as coronary artery disease, breast and colorectal cancers, and osteoporotic fractures, that contribute to illness or potential death (Orchard et al., 2010)

Limitations of the Study

Findings of the study cannot be generalized to the entire older adult population, as the secondary data was obtained from a convenience sample from a small geographic location that was not ethnically diverse. Another potential limitation of the study could have come from self-reporting of the nutritional data, due to the Hawthorne effect. The subject's nutritional intake may have been modified on the food frequency questionnaire, so the subjects could appear to have consumed a healthier diet. Lastly, because secondary data was used, it is unknown whether or not the bone mineral density scores were influenced by medications, hormone replacement therapy or supplementation, if any.

Methodology

Data used in this research is secondary from a previous UW-Stout Collaboration study, which had obtained IRB approval and followed IRB protocols. The data consisted of 26 de-

identified records of a sample population from Menomonie, Wisconsin, consisting of adults 50-80 years old. Nutritional data was originally obtained from the 26 Diet History Questionnaire II, past month without portions survey. This tool is a validated instrument developed by the National Cancer Institute used for nutrient estimates (National Cancer Institute, 2014b). The Diet History Questionnaire II survey results were processed using Diet Calc 1.5 software into datafile compatible with SPSS. The dietary datafile included saturated fat intake from the study participants. Bone mineral density measurements, at the spine and pelvis, were obtained using a dual energy X-ray absorptiometry scanner.

Chapter II: Literature Review

In this chapter, a background of bone metabolism and osteoporosis is provided followed by a review of literature on dietary fats and their impact on bone health. The review of the literature specifically examines total fat, unsaturated and saturated fat intake and their impact on bone mineral density. Finally, this chapter addresses potential causations as to how dietary fats may affect bone health. This chapter provides evidence for the need for further research on saturated fat intake and the potential effect it has on bone health.

Bone Metabolism Overview

To maintain optimal control of calcium homeostasis and strengthen bones, bone remodeling is a necessity to the human body to preserve a healthy skeleton (Henriksen, Bollerslev, Everts, & Karsdal, 2011). During normal bone health, bones are constantly being remodeled within our body through three different bone cell types: osteoblasts, osteoclasts, and osteocytes (Caetano-Lopes, Canhao, & Fonesca, 2007; Henriksen et al., 2011). Osteoblast cells play a key role in bone tissue formation by creating and maintaining skeletal architecture (Caetano-Lopes et al., 2007). During bone formation, osteoblasts send out signals to stem cells located in bone marrow to secrete collagen and other matrix proteins (Mahan, Escott-Stump, & Raymond, 2012). Once collagen is in place, it forms a mature triple-stranded fiber that gets crystalized into hydroxyapatite from calcium and phosphate salts precipitating onto the collagen fibers. Along with bone formation, osteoblasts also play a large role in bone remodeling with osteoclasts, which shows how bone remodeling and bone formation are in sync with one another (Mahan et al., 2012). After a while, bone formation reaches a point where it becomes stable, this period is called peak bone mass (Langman & Trippe, 2010).

Peak bone mass is reached between the ages of 18 to 30 years (National Osteoporosis Foundation (NOF), 2010; Mahan et al., 2012). Prior to reaching peak bone mass, bone mass continues to build bone strength and density eventually reaching its maximum potential (National Institutes of Health, 2012). As individuals age, their bone mass begins to decrease, increasing the likelihood of developing osteoporosis (Diab & Watts, 2013). Adult bone health is determined based on peak bone mass attained during youth, maintenance of bone mass during adulthood, and by reducing the rate of bone loss in older adulthood (Lanham-New, 2009).

To compensate for changing lifestyle factors that occur with aging, bones go through remodeling in order to maintain extracellular fluid calcium concentrations and to repair microscopic fractures (Mahan et al., 2012). Osteoclasts are the cells responsible for degrading both the inorganic calcium matrixes and the organic collagen matrixes (Henriksen et al., 2011). Bone remodeling begins with the activation of preosteoclastic cells by interleukin-1 and other cytokines released from inactive osteoblasts. The preosteoclast cells eventually make their way to the bones surface, where they mature into osteoclast cells (Chernoff, 2014). After maturation, the osteoclast cells cover the appropriate bone tissues and release acids and proteolytic enzymes to form cavities at remodeling sites (Chernoff, 2014). Osteoblasts than “resorb” back into the cavities and secrete new collagens and matrix constituents leading to bone formation (Chernoff, 2014). After the bone remodeling process, the embedded osteoblast cells undergo a transformation, becoming osteocytes (Chernoff, 2014). This transformation occurs at the end of the bone formation phase (Chernoff, 2014). Osteocytes function as mechanosensors, regulating bone turnover in the body through osteoclast and osteoblast activity (Chernoff, 2014; Henriksen et al., 2011). As a person ages, a decrease in bone formation occurs from a reduction in osteoblasts, activity, and life span; while bone resorption decreases from a reduction in sex

hormones (Marie, 2010). As the bone formation and bone resorption processes decline, bone mass decreases and the risk for developing a fracture increases (Marie, 2010).

Osteoporosis

Osteoporosis is a skeletal disorder where bone strength is weakened, leading to an increased risk for developing a fracture (Ahmadiéh & Arabi, 2013). Osteoporosis is considered an increasing health concern that could lead to early disability or mortality (Tucci, 2006). According to Cawthon (2011), osteoporosis is typically thought of as a “woman’s disease” due to the prevalence and rate of fractures that are higher in postmenopausal women compared to older men. Men over 50 years of age are considered more likely to break a bone from osteoporosis than develop prostate cancer, while women have an equal chance of breaking a hip and developing breast, ovarian, and uterine cancer combined (NOF, 2014a).

Methods of diagnosis. Various methods can be used to diagnose osteoporosis, including: bone mineral density test, FRAX algorithm, various blood and urine laboratory tests, medical history, and a physical examination (NOF, 2014e). A bone mineral density test is commonly performed using a dual energy x-ray absorptiometry (DEXA), which measures bone density, and with that can be used to determine the chance for a potential fracture (NOF, 2014e). DEXA is considered the “gold standard” for diagnosing osteoporosis, which is why it is used in the current study (OrthoGeorgia, 2014). A DEXA scan resulting in a T-score value of ≤ -2.5 in the hip, spine, or forearm indicates osteoporosis (Diab & Watts, 2013). According to Blake and Fogelman (2007), the preferred testing measurement site to assess the risk of osteoporosis is at the lumbar spine and hip. The National Osteoporosis Foundation explains that a fracture is more likely to occur at the hip or spine for someone who is considered osteoporotic, and the outcomes associated with these types of fractures are more severe (extended recovery period, greater pain,

and disability) (NOF, 2014c). Hip bone mineral density was identified to be the best measurement site to predict whether osteoporosis is present for elderly subjects, as opposed to the spine, in the study by Arabi et al. (2007).

Prevalence and impact of osteoporosis. Arlot, Sornay-Rendu, Garnero, Vey-Marty, and Delmas (1997) studied the effect of age on women (31 to 89 years) with bone loss, using a DEXA scan for total body, hip, distal radius and lumbar spine. DEXA measurements found that 22.5% of females between 50 and 59 years and 54% of females aged 60 and 69 years were considered osteoporotic at one of the measured sites (Arlot et al., 1997). Females 70 years and older, indicated that 71% were osteoporotic at one of the sites. Based on all four sites, 6.2% of females between 50-59 years were found to have osteoporosis at all four of the measured sites, while 7.6% of the females over 70 indicated the same finding (Arlot et al., 1997).

Based on DEXA measurements for elderly women, hip bone mineral density indicated that 70.5% were considered osteoporotic and 44.6% were osteoporotic at the spine (Arabi et al., 2007). Elderly males also were found to have a higher prevalence of osteoporosis at the hip (72.6%) compared to the spine (28.6%). Arabi et al. (2007) identified that, had the bone mineral density measurements been taken from just the spine, the majority of male and female subjects who were considered osteoporotic would have gone unnoticed. Not only was hip bone mineral density identified as one of the best measurement site for diagnosing osteoporosis, but it also was strongly related to vertebral fractures in the elderly compared to spine measurements (Arabi et al., 2007). Bone mineral density measurements are considered risk factors used to identify the risk of fracture from osteoporosis, however there are other factors that are used to identify the risk of osteoporosis (Arabi et al., 2007).

Risk factors. Risk factors associated with osteoporosis are either controllable or uncontrollable (NOF, 2014a). Gender is an important uncontrollable risk factor for osteoporosis. In the United States, 80% of those who have developed osteoporosis are female (NOF, 2014g). For the year 2005, the Markov state-transition model estimated that women would experience 71% of total fractures, accounting for 76% of total health care costs resulting from osteoporotic fractures (Burge et al., 2007). The National Osteoporosis Foundation states the two main reasons women are more prone to develop osteoporosis are because women naturally have a thinner bone structure compared to men, and from estrogen levels decreasing once menopause is reached (NOF, 2014g).

The most important influential factor in the development of osteoporosis in women is menopause (Jagtap, Ganu, & Nagane, 2011; Lee et al., 2013; NOF, 2014g; NOF, 2014a). Women undergo an abrupt decrease in bone mineral density between the ages of 50 to 59 years, with the odds of osteoporosis increasing by ten to 23 times in women 80 and older (Lee et al., 2013). The National Osteoporosis Foundation further indicated that five to seven years after menopause, women can undergo a 20% decrease in bone mineral density (NOF, 2014g). During menopause, estrogen levels decrease from the loss of ovarian function (Mahan et al., 2012). The lack of estrogen stops the absorption and use of calcium, leading to a decrease in skeletal mass through an imbalance between bone resorption and formation (Jagtap et al., 2011).

Another common risk factor associated with osteoporosis is calcium and vitamin D intake. A cohort study by Cumming et al. (1997) found that out of 9,704 women over the age of 65, 75% of them had a dietary calcium intake of 400mg or more a day (mean intake of 714mg/d) (Cumming et al., 1997). A more recent study by Zhong, Okoro, and Balluz (2009), using data from a 1999-2002 National Health and Nutrition Examination Survey (NHANES) report on 2006

postmenopausal women, also found that 75% of the women consumed ≥ 400 mg/d of total calcium with a mean total intake of 791mg/d. Although the mean calcium intake identified in the studies by Cumming et al. (1997) and Zhong et al. (2009) were similar, they were both under National Research Council's (2006) recommendations for postmenopausal women (1,200mg/d). Although calcium and vitamin D supplementation are known to have preventative effects on osteoporosis, other dietary factors have come to light that may also play a role in bone health, specifically of interest to this study are dietary fats, particularly saturated fats (Cumming et al., 1997; Corwin et al., 2006).

Dietary Fats and Their Relationship to Bone Mineral Density

Research has indicated that dietary fats can affect bone health (Corwin et al., 2006). Available research on dietary fat and bone health have primarily focused on either dietary total fat intake, unsaturated fat (i.e., poly- and monounsaturated fats), or saturated fat intake. The available research on dietary fats and their relationship with osteoporosis remains limited (Martinez-Ramirez et al., 2007). Various studies have shown that saturated and unsaturated fats have adverse effects on bone health (Corwin et al., 2006; Orchard et al., 2010; Martinez-Ramirez et al., 2007; Macdonald et al., 2004). While research on unsaturated fats has also indicated a potential beneficial role on bone health (Jarvinen et al., 2012; Hogstrom et al., 2007; Lau et al., 2010; Orchard et al., 2010). Similar to research findings on saturated and unsaturated fats, showing a negative interaction on bone health, there are also a number of studies showing a negative association between a high fat diet and bone health (Kato et al., 2000; Schuit et al., 2004; Bielohuby et al., 2010; Parhami et al., 2001).

Total fat intake. According to the National Research Council (2006), no estimated average requirements (EAR) or adequate intake (AI) for total fat intake is set for individuals over

the age of one, due to insufficient data indicating a consumption level that can assist with the prevention of diseases. Calories from dietary fats come from saturated, poly- and monounsaturated, and trans-fats combined (National Research Council, 2006). A high fat diet is known to increase a person's weight, and body weight influences bone health (Centers for Disease Control and Prevention, 2011). According to Hsu et al. (2006), body weight is a strong predictor of bone mass. Lau et al. (2010), further believes that a larger body weight may be related to an increase in bone mineral density as well as certain measures of bone strength. Beck et al. (2001) found that by increasing the skeletal load at the femoral neck and shaft, the bone strength index also increased. Unfortunately, studies by Macdonald et al. (2004) and Kato et al. (2000) failed to show a positive association between body weight and bone health. However, research by Kato et al. (2000) has indicated that total dietary fat intake may actually increase the risk for fracture.

A marginally significant association ($p=0.066$) between a higher total fat intake and hip and wrist fractures was found in postmenopausal women when examined for the risk of fracture associated with dieting (Kato et al., 2000). The research found a 23% greater risk in all fracture types for postmenopausal women who consumed ≥ 75 g of fat. As fat levels increase from <57.2 g to ≥ 74 g, the number of wrist and hip fractures steadily increases from 33 to 44. Kato et al. (2000) claim that high fat intake may increase fracture risk in postmenopausal women. Although the study by Kato et al. (2000) did not directly look at bone mineral density, a study by Schuit et al. (2004) found that low bone mineral density is highly related a fracture in the hip or wrist.

Similar research conducted on mice, that were fed high fat diets, further provided evidence indicating bone loss. In a study by Bielohuby et al. (2010), mice were given one of two

Low Carbohydrate-High Fat (LC-HF) diets or a control diet to identify any changes in bone growth and quality. Both the LC-HF-1 (66% fat, 33% protein, and 1% carbohydrate (CHO)) and the LC-HF-2 (94.5% fat, 4.2% protein, and 1.3% CHO) groups showed a significant reduction in bone mineral density at the left tibiae, compared to the control group (9% fat, 33% protein, and 58% CHO). Rats fed with either of the LC-HF diets, were also found to have more fat cells located in their bone marrow (Bielohuby et al., 2010).

Another study comparing a high fat diet (1.25% cholesterol, 15.8% fat, and 0.5% cholate) to a control diet (6% total fat) in C57BL/6 rats, found a significantly lower bone mineral density in three out of eight femur slices after consuming the high fat diet for four months (Parhami et al., 2001). All three of the samples, that indicated a reduction in bone mineral density, were from the mid-section of the femur (Parhami et al., 2001). A more recent study conducted by Ionova-Martin et al. (2011) found no changes in whole body and femoral bone mineral density in adolescent and adult C57BL/6 mice fed both a high fat diet (60% fat, 20% CHO, and 20% protein) and low fat diet (10% fat, 70% CHO, and 20% protein) after 16 weeks. However, an 18% reduction in spinal bone mineral density was observed in the adult high fat group compared to the adult low fat group, while a 9% decrease was observed in the adolescent high fat group compared to the adolescent low fat group (Ionova-Martin et al., 2011).

The body of research conducted on mice indicates that a high fat diet can reduce bone mineral density (Bielohuby et al., 2010; Parhami et al., 2001; Ionova-Martin et al., 2011). Ionova-Martin et al. (2011) also suggest that high fat diets increase the risk for spinal fracture in mice during adulthood and adolescence.

A number of studies have shown a negative association between a high fat diet impacting bone health (Kato et al., 2000; Schuit et al., 2004; Bielohuby et al., 2010; Parhami et al., 2001;

Ionova-Martin et al., 2011). However, it is not known what the composition of fat was in these studies. Therefore, it is not clear whether the type of fat in a high fat diet has a specific impact on bone health. It is also unclear as to whether providing different types of fat, in high fat diet research protocols, would impact bone health differently. Research has also been conducted on different classifications of fats to further investigate each of their impacts on bone health, such as unsaturated fats.

Unsaturated fatty acids. There are two classifications of unsaturated fatty acids, polyunsaturated fatty acids (PUFA) and monounsaturated fatty acids (MUFA) (Glowacki, Manson, & LeBoff, n. d.). “Poly” and “Mono” indicates how many double bonds are found on the unsaturated fatty acids (Poly=two or more; Mono=one) (Glowacki et al., n. d.). Unsaturated fatty acids with a double bond at the n-3 or n-6 position are considered essential, due to mammalian cells not being able to produce the double bond at the respected position (Glowacki et al., n. d.). According to the National Research Council (2006), no EAR or AI is set for dietary PUFA’s or MUFA’s, due to a lack of evidence indicating health benefits. Similar to the research conducted on high fat diets by Kato et al. (2000), Bielohuby et al. (2010), Parhami et al. (2001), and Ionova-Martin et al. (2011), studies have indicated adverse effects to bone health from dietary PUFA intake (Martinez-Ramirez et al., 2007; Macdonald et al., 2004).

Dietary PUFA’s and bone health. Elderly patients (≥ 65 years), who sustained a low-energy fracture 6 to 24 months prior to the study, were examined to determine whether different types of fat would predict the risk of fracture (Martinez-Ramirez et al., 2007). Martinez-Ramirez et al. (2007) considered a low-energy fall to be fracture resulting from a same-level fall. PUFA intake that was assessed using a food frequency questionnaire indicated that a high intake was correlated with an increased risk of osteoporotic fracture (Martinez-Ramirez et al., 2007). After

PUFA intake was categorized into quartiles, the odds ratio (OR) indicated that the top two quartiles had the strongest statistically significant ($p=0.011$) increase in risk of fracture (Q3:15-17g/OR=3.59; Q4: \geq 18g/OR=5.88). While Martinez-Ramirez et al. (2007) found that a high intake of PUFA can increase the risk for fracture, PUFA intake has also been linked to a decrease in bone mineral density.

A study conducted by Macdonald et al. (2004) also identified a high PUFA intake to be negatively associated with a change in bone mineral density at the femoral neck in perimenopausal women, but not at the lumbar spine. Once adjusted for numerous confounders (i.e. age, weight, smoking status, menopausal status, hormone replacement therapy, etc.), the negative association was significant. PUFA's were further associated with a significant negative change in bone mineral density when total calcium intake was low ($r=-0.213$, $p<0.001$), as well as when calcium intake from diet only ($r=-0.201$, $p=0.001$) was low. The research suggested that women around the age of menopause may experience more adverse effects from PUFA's when their dietary calcium intake is low (Macdonald et al., 2004).

Unlike the Macdonald et al. (2004) study, which found a negative correlation between PUFA intake and bone mineral density, numerous other studies have identified PUFA intake as having a beneficiary role to bone health. Jarvinen et al. (2012) found a positive relationship between total PUFA intake, linoleic and linolenic acids, total n-3 and n-6 fatty acids with bone mineral density at the lumbar spine and total body in elderly women (mean age: 67.9 ± 1.9), while no associations were found between PUFA intake and bone mineral density at the femoral neck. In the study by Jarvinen et al. (2012), total PUFA intake consisted of n-3 (linolenic acid, eicosapentaenoic acid (EPA), and docosahexaenoic acid (DHA)) and n-6 (linoleic acid and arachidonic acid) fatty acids combined. Food records revealed that total n-3 PUFA's were

largely represented by linolenic acid, and they were significantly associated with a higher total body and lumbar spine bone mineral density (Jarvinen et al., 2012). Likewise, Hogstrom et al. (2007) identified a significant positive correlation, using serum phospholipid fraction, between total n-3 fatty acids and DHA for total body (n-3 fatty acids: $r=0.27$, $p=0.02$; DHA: $r=0.32$, $p=0.004$) and spine (n-3 fatty acids: $r=0.25$, $p=0.02$; DHA: $r=0.30$, $p=0.008$) bone mineral density in a younger male sample (mean age: 16.7 years). Similar to Jarvinen et al. (2012), total n-3 fatty acids represented in the study by Hogstrom et al. (2007) included linolenic acid, EPA, and DHA; while Hogstrom et al. (2007) also included docosapentaenoic acid (DPA). Although both studies found favorable effects on total body and spine bone mineral density, despite the slight difference in total n-3 fatty acids composition, other various aspects of the studies differed.

The main difference between the studies by Jarvinen et al. (2012) and Hogstrom et al. (2007) was the sample population. A sample population consisting of elderly women was studied by Jarvinen et al. (2012), while Hogstrom et al. (2007) studied younger male subjects. Another difference between both studies was the methods used to measure dietary PUFA's. Jarvinen et al. (2012) used a three day dietary recall, and found that elderly women had a mean daily intake of 8.8 ± 3.4 g total PUFA; whereas Hogstrom et al. (2007) used a non-fasting total plasma lipid profile to measure total PUFA intake, indicating levels to be 42.5 ± 2.1 g in the younger male sample. Regardless of the study design, both studies indicated that n-3 fatty acids may have beneficial effects to bone health, as shown through an increased change for total body and lumbar spine bone mineral density (Jarvinen et al., 2012; Hogstrom et al., 2007). Similarly, unsaturated fats have also shown beneficial properties to bone mineral density in animal studies.

Lau et al. (2010) distributed 40 rats into four groups, adding a different composition of dietary fat into the same diet given to each group. Two of the treatment groups were given either

a dietary fat source of n-3 PUFA's (20% flaxseed oil) or n-6 PUFA's (20% safflower oil). After being euthanized, bone mineral density measurements were collected from the left femur through a DEXA scan. The authors identified that rats fed the diets high in n-3 and n-6 PUFA's showed an increase in femoral bone mineral density compared to the control sample, but was not considered significant when unadjusted and adjusted for final body weight (Lau et al., 2010). Due to the lack of significance between the PUFA groups, compared to the control, Lau et al. (2010) concluded that diets high in PUFA's showed no negative effect on bone mineral density.

A study conducted by Orchard et al. (2010) looked at various fatty acids and their relationship to the risk of osteoporotic fractures for women in the Women's Health Initiative. Percent of energy received from PUFA intakes, indicated that the higher three PUFA intake groups, represented by quartiles (Q2:5.17 to 6.42%; Q3:6.43 to 7.89%; Q4:7.90 to 31.84%), were associated with a slightly lower risk for total fractures. No indication of fracture risks were identified based on total n-3 fatty acid consumption, despite a decrease in risk for total fracture when dietary n-3 fatty acid increased. The authors further identified that the highest n-3 fatty acid intake (5.64% of energy) was associated with the lowest risk for hip fracture (Orchard et al., 2010).

Dietary MUFA's and bone health. Like dietary PUFA's, research has indicated adverse effects from dietary MUFA's on bone health, as well as potential preventative effects (Macdonald et al., 2004; Hogstrom et al., 2007; Martinez-Ramirez et al., 2007; Orchard et al., 2010). Macdonald et al. (2004) identified a negative correlation between a high dietary MUFA intake with bone mineral density at the femoral neck ($r=-0.069$, $p<0.05$), while no statistical association was found at the lumbar spine. The negative association between dietary MUFA intake and bone mineral density at the femoral neck was only considered significant after

adjustment for confounders were made (Macdonald et al., 2004). The study by Hogstrom et al. (2007) also found a significant negative correlation ($p < 0.05$) between total MUFA intake and total body bone mineral density ($r = -0.25$, $p = 0.02$). The same negative correlation was found between a monounsaturated fatty acid, called oleic acid ($r = -0.26$, $p = 0.02$), and total body bone mineral density (Hogstrom et al., 2007). While Macdonald et al. (2004) found that a high MUFA intake had a negative effect on bone mineral density, studies have identified that a higher MUFA intake can be beneficial to fracture risks (Martinez-Ramirez et al., 2007; Orchard et al., 2010).

Based on the odds ratio (OR) for the daily intake of MUFA and risk of low-energy fractures, in a study conducted by Martinez-Ramirez et al. (2007), the quartile representing the lowest MUFA intake (Q1: $< 39\text{g}$, OR=1) was associated with the highest risk for fracture ($p = 0.247$) compared to the higher quartiles (Q2: -46g , OR=0.27; Q3: $47\text{-}54\text{g}$, OR=0.65; Q4: $\geq 54\text{g}$, OR=0.52). Unlike Macdonald et al. (2004), Martinez-Ramirez et al. (2007) found preventative effects from a higher MUFA intake in regards to osteoporotic fractures. A high intake of MUFA has also been suggested to lower total fracture risk in postmenopausal women (Orchard et al., 2010). Once total dietary MUFA intake was divided into quartiles, based on percent energy from dietary MUFA intake (Q1=lowest, Q4=highest), a dietary intake of 12.18 to 14.51% (Q3) and 14.52 to 48.5% (Q4) showed a slight significant reduction in total fractures after adjustments (Orchard et al., 2010). Research on unsaturated fats has indicated both a positive and adverse effect on bone health. This same conflicting tendency was observed among research on saturated fats and bone health.

Saturated fatty acids. Unlike unsaturated fatty acids, which are liquid at room temperature, saturated fatty acids have a solid consistency (Glowacki et al., n. d.). While

unsaturated fatty acids appear as a liquid, due to having one or more double bonds that “kinks” the molecules preventing them from packing together; saturated fatty acids are solid at room temperature due to the molecules being packed together with hydrogen atoms (Indiana University, 2014; Rustan & Drevon; 2005). Saturated fats can be made within the body and are used as a source of energy, as well as are needed for structural and physiological functions (National Research Council, 2006). There is no EAR or recommended dietary allowances (RDA) set for saturated fat intake by the National Research Council (2006), due to saturated fats not being considered essential in the diet, and from lacking any known preventative effects for chronic diseases.

Human research has shown that a high intake of saturated fat is linked to a reduction in bone mineral density in humans (Corwin et al., 2006). Saturated fats have also been linked to an increased risk of hip fractures, when consumed in high amounts (Orchard et al., 2010). Research on saturated fat intake potentially having a negative effect on bone health remains scarce and conflicting, yet promising at times (Corwin et al., 2006; Orchard et al., 2010; Macdonald et al., 2004; Martinez-Ramirez et al., 2007; Lau et al., 2010).

Adverse effects from saturated fat on bone health. Corwin et al. (2006) has identified that a high saturated fat intake can decrease bone mineral density among both females and males. Using the NHANES III national survey, Corwin et al. (2006) assessed total dietary fat and specific fatty acids and the effect they have on bone mineral density at the hip region for men and women (n=14,850). The specific bone mineral density sites analyzed were: femoral neck, trochanter, intertrochanter, and total hip. A negative association was found for both men and women at the femoral neck, where a 2.3% reduction (Q1:0.843g/cm²; Q5:0.824g/cm²) was

observed from the lowest to the highest saturated fat intakes (Q1: ≤ 12.4 g; Q5: 36.4 to 130.3g) (Corwin et al., 2006).

Additionally, men of all ages showed a more distinct negative relationship between saturated fat and bone mineral density compared to women (Corwin et al., 2006). Corwin et al. (2006) believed that the relationship, or lack thereof, between saturated fat intake and bone mineral density in the female subjects, could have resulted from a measurement error; specifically from female subjects underreporting saturated fat intake for the NHANES III survey. Among all men, a 2 to 4% decrease was found at the femoral neck (Q1: 0.897 g/cm²; Q5: 0.858 g/cm²), trochanter (Q1: 0.765 g/cm²; Q5: 0.824 g/cm²), and total hip (Q1: 1.028 g/cm²; Q5: 0.995 g/cm²) sites (Corwin et al., 2006).

Corwin et al. (2006) found that younger men (<50 years old) had a greater reduction in bone mineral density at the femoral neck (Q1: 0.963 g/cm²; Q5: 0.922 g/cm²) compared to older males (≥ 50 years) (Q1: 0.811 g/cm²; Q5: 0.782 g/cm²). Despite both male age groups indicating a reduction in bone mineral density, younger men had a significant negative association at the femoral neck site; while older men just showed negative tendencies that were not considered significant. Interestingly, women showed no associations between saturated fat intakes negatively influencing bone mineral density for any of the examined sites. Corwin et al. (2006) concluded that saturated fat intake may negatively affect bone mineral density at the respective sites, while men showed a stronger inverse association between saturated fat consumption and bone mineral density.

Orchard et al. (2010) identified that a higher saturated fat intake was found to be significantly related to an increased risk for hip fracture in women. No association was found between saturated fat intake and total fracture risk, except at the highest intake of saturated fat

(Q4:12.78 to 36.7% of energy). The authors found that the population attributable for risk, based on consuming saturated fat within the range of highest quartile (Q4), resulted in a 5.5% chance for developing a hip fracture (Orchard et al., 2010). Orchard et al. (2010) determined that women were at a similar risk for developing a hip fracture, when saturated fat makes up 12.78 to 36.7% of total calories, compared to women who were current smokers (5.6%). The research conducted by Corwin et al. (2006) and Orchard et al. (2010), both found that a diet high in saturated fat can negatively influence bone mineral density at the hip as well as increase the risk of hip fracture; while other studies found no such relationship between saturated fat and bone health (Macdonald et al., 2004; Martinez-Ramirez et al., 2007; Lau et al., 2010).

No effect from saturated fat on bone health. Macdonald et al. (2004) and Martinez-Ramirez et al. (2007) both found no known associations between saturated fat intake and osteoporotic fractures or bone loss. Macdonald et al. (2004) speculated the lack of effect from saturated fat on the femoral neck or lumbar spine bone mineral density could have been from milk products consumed by the subjects, since milk products provide health benefits for bone health. Martinez-Ramirez et al. (2007) did not elaborate as to why saturated fat did not show any relationship to osteoporotic fractures, but did find that subjects consuming $\geq 34\text{g}$ of saturated had the most cases of low-energy fractures compared to the lower intakes and their corresponding controls.

While Macdonald et al. (2004) directly looked at bone mineral density, as opposed to fractures risk, Lau et al. (2010) found that rats given a diet with added saturated fat (18% coconut oil and 2% soybean oil) showed an increase in whole femur bone mineral density ($212.93 \pm 6.53 \text{ mg} \cdot \text{cm}^{-2}$) compared to the control ($200.96 \pm 12.15 \text{ mg} \cdot \text{cm}^{-2}$). Although a higher femoral bone mineral density was found in the high saturated fat diet group, the authors found no

significant difference between the saturated fat group and the control group (Lau et al., 2010). Therefore the researchers identified that saturated fat did not negatively affect bone mineral levels (Lau et al., 2010). Lau et al. (2010) found that both PUFA groups were linked to an increase in body weight, as was the saturated fat group. Again, this could possibly explain the higher bone mineral density found after 65 days of consuming a higher fat diet.

There are studies on high saturated fat intakes being linked to both a reduction in bone mineral density and an increase in risk of fracture, as well as studies indicating no relationship between saturated fat intake and bone health (Corwin et al., 2006; Orchard et al., 2010; Macdonald et al., 2004; Martinez-Ramirez et al., 2007; Lau et al., 2010). Inconsistent findings were also found unsaturated fats, as well as with high fat diets in this review on dietary fats and bone health. However, this may be due to the limited amount of research examining the effect of fat intake on bone mineral density, bone health, and fractures (Martinez-Ramirez et al., 2007).

Theories on Dietary Fats Influencing Bone Health

While Corwin et al. (2006) found a negative “effect” between bone mineral density and saturated fat intake among all men, research provided no clear “cause” indicating how saturated fat intake could have affected bone mineral density. Leading Corwin et al. (2006) to declare that the study showed no “cause/effect relation” between the two variables. This could be said of all the studies featured in this chapter, that found a negative association between dietary fats and bone health, associations were found but the reasons remain unclear. Research has shown a potential link to bone marrow being influenced through a high fat diet (Parhami et al., 1999; Parhami et al., 2001).

The study by Parhami et al. (1999) identified a repressed mineralization in marrow stromal cells in rats from both a high fat diet (7.5% cocoa butter, 1.25% cholesterol, and 0.5%

sodium cholate) and from an accumulation of oxidatively modified LDL cholesterol (MM-LDL) near preosteoblasts. MM-LDL, product of lipid and lipoprotein oxidation, is known to inhibit the differentiation and mineralization of preosteoblasts located in the bone marrow, which eventually could affect osteoblasts (Parhami et al., 1999). A similar study conducted by Parhami et al. (2001) found that mice given a high fat diet (1.25% cholesterol, 15.8% fat, and 0.5% cholate) for four to seven months, showed a decrease in bone mineral density. The bone marrow sample further suggested that osteoblast maturation is affected from the high fat diet (Parhami et al., 2001). Likewise, Bielohuby et al. (2010) also found more adipocytes in marrow, when rats were fed a Low Carbohydrate-High Fat diet. The studies by Parhami et al. (1999) and Parhami et al. (2001) provided insight as to how lipids may alter the regulation of bone metabolism, leading to a decrease in bone mineral density (Parhami et al., 1999; Parhami et al., 2001). While high lipid levels have been found to effect bone marrow resulting in a decrease in bone mineral density, saturated fatty acids have been identified to reduce calcium absorption (Parhami et al., 1999; Parhami et al., 2001; Bielohuby et al., 2010; Gacs & Barltrop, 1976).

Gacs and Barltrop (1976) investigated the availability of calcium absorption in rats from formed soaps induced by different fatty acids. The authors found that stearic acid, a saturated fatty acid, significantly increased Ca-soap formation in the intestines and in the feces, which led to a decrease in calcium absorption (Gacs & Barltrop, 1976). Rats fed one mg of Ca and 100 mg of stearic acid only absorbed 32% of the calcium compared to the control, which absorbed around twice the amount of calcium as did the other fat groups. This study may indicate that a normal diet may undergo a decrease in calcium absorption through the presence of stearic acid induced Ca-soap formation in the intestines (Gacs & Barltrop, 1976). While these results show that stearic acid may negatively influence calcium absorption, triglycerides showed little effect

on calcium absorption. The decrease in calcium absorption can result in calcium being “borrowed” from the bones to maintain a calcium balance, which overtime can lead to osteoporosis (Colman, 2014).

Another area of discussion, potentially explaining the reason why dietary fats effect bone mineral density, is that osteoporosis may be related to cardiovascular disease due to similar risk factors (den Uyl, Nurmohamed, van Tuyl, Raterman, and Lems (2011). A systematic review conducted by den Uyl et al. (2011), identified that cardiovascular disease is associated with an increased risk for fracture and bone loss. Major risk factors linking cardiovascular disease to osteoporosis include: age estrogen, deficiency, and inflammation (den Uyl et al., 2011). Although den Uyl et al. (2011) indicated a potential relationship between cardiovascular disease and osteoporosis; the relationship was considered inconclusive and was in need of more research.

Summary

Although the review of literature has looked at different classifications of dietary fats and their role on bone health, saturated fat was the primary variable chosen for the current research. Saturated fats were specifically chosen because a high intake can raise low-density lipoprotein cholesterol, potentially leading to the development of coronary heart disease or stroke (Centers for Disease Control and Prevention, 2012; American Heart Association, 2014). Older adults are at the greatest risk for developing osteoporosis, heart disease, or stroke (Ahmadiéh & Arabi, 2013; Mozaffarian et al., 2013). Since saturated fat already has a negative connotation with coronary heart disease and stroke in the elderly, and the limited research indicating saturated fat having an effect on bone health, research is necessary to further investigate whether saturated fat has an effect on bone mineral density on the elderly population (Mozaffarian et al., 2013).

In conclusion, the review of literature on saturated fat intake and bone health has found mixed results. Saturated fat intake was found to negatively influence bone mineral density and increase the risk of fracture, while also demonstrating no effect. The need for further research on saturated fat and bone health is essential, as research remains limited. In regards to bone health, research on saturated fat intake is of the importance. Osteoporosis shares similar risk factors with coronary heart disease. Despite the lack of evidence indicating saturated fat having a negative effect on bone health, the similarities between the two diseases should not go unnoticed. Chapter Three provides the methodology used in the study.

Chapter III: Methodology

The purpose the study was to determine whether dietary saturated fat intake influenced bone mineral density in older, Midwestern adults. This study used secondary data from a UW-Stout Collaboration Study, which consisted of 26 de-identified records including: demographic data, food frequency questionnaire data files, and bone mineral density measurements at the spine and pelvis. This chapter discusses and describes the subjects, how the data was obtained, collection procedures, and the limitations involved in the methodology.

Subject Selection and Description

Prior to obtaining the secondary data, the UW-Stout Institutional Review Board (IRB) exempted the study from review for the Protection of Human Subjects due to use of data from a previous UW-Stout Collaboration Study (Appendix A). The secondary data was from a 32-week intervention study that began in the September of 2012 and ended in May 2013. The UW-Stout Collaboration Study used a convenience sample consisted of 26 male and female participants. Subjects ranged from 50 to 80 years old and were from the Dunn County, Wisconsin. In 2013, Dunn County had an estimated population of 44,122 (United States Census Bureau, 2014). Adults aged 65 and older represented 13.3% of the population in 2012, while Caucasians made up 95.1% of the population. The median household income from 2008 to 2012 was \$47,847 (United States Census Bureau, 2014).

Data Collection Procedures

The secondary data was de-identified prior to receiving it for this research. The secondary data included a National Cancer Institute's Diet History Questionnaire II, which contained dietary fat information including saturated fats. Spine and pelvic bone mineral density data was measured with a DEXA scanner, model DPX-IQ. Dietary saturated fat intake,

demographic data and pelvic and spine bone mineral density measurements were the primary data used for analysis.

Data Analysis

Statistical analyses were conducted using the Statistical Program for Social Sciences (SPSS), version 19.0. A descriptive statistics test was run to determine the mean, minimum and maximum age, total caloric intake, and total saturated fat intake for all subjects. Another descriptive statistics test was run on spine and pelvic bone mineral density measurements to determine minimum, maximum, mean, and the standard deviation intake among all subjects.

To investigate the relationship between total saturated fat intake and spine and pelvis bone mineral density, a two-tailed Pearson's Correlation statistical analysis were conducted. The two-tailed Pearson's Correlation test was run on total saturated fat intake and spine bone mineral density and again on total saturated fat intake and pelvic bone mineral density. Due to the small sample size of the study, mean total saturated fat intake (g) was used for the Pearson's Correlation test with mean bone mineral density scores (g/cm^2) for both males and females combined. Since dietary saturated fats have shown conflicting results on bone mineral density, a two-tailed test was used versus a one-tailed because a positive correlation was expected as equally as a negative correlation. Statistical significance was determined based on a p -value of <0.05 .

Limitations

Due to the nature of using secondary data, various limitations were present in this study. First, the small sample size was a limitation in this study. The sample size prevented the elimination of outliers during data analysis, as well as the analysis of individual gender due to vast differences between the two (females: $n=20$ /males: $n=6$). It is also possible that the

nutritional intake values were a limitation due to reporter bias, resulting in a Hawthorne effect. The subjects could have modified their nutrient intake to make it look like they consumed a healthier diet than they actually did. Another potential limitation of the study is that bone mineral density measurements were only from the spine and pelvis, limiting analysis only to these areas. This study is further limited from generalizing its findings due to the use of a convenience sample, which does not entirely represent older adults from other geographical locations.

Chapter IV: Results

The purpose of this study was to determine whether dietary saturated fat intake influences bone mineral density in older adults from the Midwest. This study analyzed secondary data obtained from a UW-Stout Collaboration Study. The data included dietary saturated fat intake and bone mineral density measurements at the spine and pelvis. I used the data to examine the relationship between saturated fat intake at both the spine and pelvis sites to address the following research question:

What impact does total dietary saturated fat intake have on bone mineral density in older adults?

Descriptive statistics and two-tailed Pearson's Correlation tests were used for data analysis using SPSS, version 19.0. This chapter addresses the statistical tests examined in relation to the study research question.

Subject Demographics

The study samples ($n=26$) age ranged from 50 to 80 years with a mean age of 63.69 (Table 1). Females represented the largest portion of the sample, accounting for 77% ($n=20$) of the sample with a mean age of 62.2. While males represented 23% ($n=6$) of the sample, with a mean age of 68.67.

Table 1

Descriptive Statistics for Gender and Age

	Age			
	<i>n</i>	Minimum	Maximum	Mean
All Subjects	26	50	80	63.69
Female	20	50	80	62.2
Male	6	59	80	68.67

Note. $n=26$ (females: $n=20$, males: $n=6$)

Total Caloric and Saturated Fat Intake

Descriptive statistics were run on the subject's total caloric and saturated fat intake, based off of their responses from the food frequency questionnaire, to determine the monthly average of total calorie intake and saturated fat intake among all subjects ($n=26$) (Table 2). The total calorie intake among all subjects ($n=26$) ranged from 961.16kcal to 2829.02kcal, with a mean intake of 1846.32kcal. Whereas total saturated fat intake for all subjects ranged between 9.02g to 45.68g, with a mean intake of 23.65g. Calories from mean saturated fat intake among all subjects represented 11.5% of total mean calories consumed (Table 3).

Female subjects ($n=20$) calorie intake ranged from 961.16 to 2599.09 kcal, with a mean intake of 1693.69 kcal. Saturated fat intake among women ranged from 9.02 to 45.68g, with a mean intake of 21.6515g. The mean saturated fat intake represented 11.5% of mean kcal consumed for women. Among the male subjects ($n=6$), the mean caloric intake ranged between 1802.57 to 2829.02kcal, with a mean of 2355.09kcal. Male subjects' saturated fat intake ranged from 18.33 to 38.28g, with a mean of 30.293g. Saturated fat represented 11.6% of total calories consumed for men.

Table 2

Descriptive Statistics for Total Caloric and Saturated Fat Intake

	<i>n</i>	Minimum	Maximum	Mean
Calorie Intake (kcal)	26	961.16	2829.02	1846.32
Female	20	961.16	2599.09	1693.69
Male	6	1802.57	2829.02	2355.09
Total Saturated Fat (g)	26	9.02	45.68	23.65
Female	20	9.02	45.68	21.6515
Male	6	18.33	38.28	30.293

Note. Females: $n=20$; Males: $n=6$

Table 3

Percent of Calories Consumed from Saturated Fat

	Mean Calorie Intake (kcal)	Mean Saturated Fat (g)	% of kcal From Saturated Fat
All Subjects	1846.32	23.65	11.50%
Female	1693.69	21.6515	11.50%
Male	2355.09	30.293	11.60%

Note. 1g of saturated fat=9 kcal; $n=26$ (females: $n=20$, males: $n=6$)

Bone Mineral Density

The results from the descriptive statistics test conducted on spine and pelvic bone mineral density measurements can be found in Table 4. Analysis of the bone mineral density measurements among all subjects indicated that the mean spine measurement was 1.20g/cm², while the pelvis' mean was 1.125g/cm². Female subjects further indicated a mean spine bone mineral density measurement of 1.21g/cm² and a pelvic measurement of 1.12g/cm² (Table 5). Male subjects had a mean spine bone mineral density measurement of 1.18g/cm² and a mean

pelvis measurement of 1.15g/cm². The mean spine bone mineral density measurement translates to a T-score of 0.2, while the pelvis translated into a T-score of 0.125 (Steel & Peel, 2011). A T-score of -1 or above is considered normal (Lee et al., 2013; Diab & Watts, 2013).

Table 4

Descriptive Statistics for Bone Mineral Density in All Subjects

BMD site	<i>n</i>	Minimum	Maximum	Mean	Standard Deviation
Spine (g/cm ²)	26	0.907	1.503	1.20	0.148
Pelvis (g/cm ²)	26	0.911	1.405	1.125	0.125

Note. BMD= bone mineral density; *n*=26

Table 5

Mean Bone Mineral Density Scores for Spine and Pelvis among Females and Males

	BMD Site (g/cm ²)	
	Mean Spine	Mean Pelvis
Female	1.21	1.12
Male	1.18	1.15

Note. BMD= bone mineral density; Females: *n*=20; Males: *n*=6

Total Saturated Fat Intake's Influence on Bone Mineral Density

The mean total saturated fat intake (Table 2) and bone mineral density scores (Table 4) were used in a two-tailed Pearson's Correlation test, to evaluate the relationship between the mean total saturated fat intake and spine and pelvis bone mineral density for all subjects (Table 6). Spine bone mineral density showed a positive, but not significant relationship ($r=0.289$, $p=0.152$) with total saturated fat intake. Similarly, pelvis bone mineral density showed a positive relationship with total saturated fat intake, but it was also not significant ($r=0.14$, $p=0.494$).

Table 6

Relationship between Mean Total Saturated Fat Intake and Bone Mineral Density for All

Subjects

	BMD Site			
	Spine		Pelvis	
	Correlation	<i>p</i>	Correlation	<i>p</i>
Mean Total Saturated Fat Intake	0.289	0.152	0.14	0.494

Note. BMD= bone mineral density; *n*=26

*Correlation significant at the 0.05 level (two-tailed)

Chapter V: Discussion

This study, sought to determine whether total dietary saturated fat intake influenced spine and pelvic bone mineral density. This chapter includes a discussion of the limitations of the study, followed by the findings and conclusions. Concluding this chapter are the recommendations for future research.

Limitations

This study had a number of limitations. One limitation was the small sample size ($n=26$). The small sample size makes it difficult to generalize these findings to a larger population, which could lead to sample error occurring from a small sample being compared to the entire population of older adults (Institute for Work & Health, 2008). Another limitation resulting from the small sample size was that no outliers from the data could be removed. This is problematic because outliers have the potential to distort the overall findings from the sample population (Cousineau & Chartier, 2010). A larger sample size would have benefited the study by improving the precision of the findings based on the population, reducing the margin of error (Institute for Work & Health, 2008). The small sample size also decreased the power of the study, lowering the probability of finding any significance in saturated fat affecting bone mineral density (Institute for Work & Health, 2008). This could have potentially led to a false-negative error. Had the sample been larger, any significant findings would have been easier to detect (Banerjee, Chitnis, Jadhav, Bhawalkar, & Chaudhury, 2009).

Since the subjects were all from the same small community (Menomonie, Wisconsin) it limits the study's findings from being generalized to a larger population, where dietary intake patterns may be different. Gender distribution also was a limitation of the study. Statistical analysis could not be conducted individually due to the subject's difference in gender (females:

$n=20$; males: $n=6$). The current study's gender distribution limits the comparison of its findings to other research that looks at a specific gender.

Recall bias may have been another limitation in the study. The subjects may not have accurately recalled their diet of the previous month correctly on the food frequency surveys. However, the food frequency questionnaire used to obtain nutritional intake has been validated and found to be accurate (National Cancer Institute, 2014b). Most likely, the Hawthorne effect was the issue in the present study, due to the subjects modifying their dietary intakes.

Conclusions

The purpose of this study was to determine whether total dietary saturated fat intake influences bone mineral density, specifically at the spine and pelvis. Research looking at the relationship between saturated fat intake and bone health remains conflicting, as well as limited, while even fewer studies have specifically investigated the relationship between saturated fat intake and its effect on bone mineral density.

The current study found a non-significant positive relationship between mean saturated fat intake and bone mineral density measurements at the spine and pelvis in older adults, when saturated fat represented 11.5% of total calories consumed. These results were consistent with the findings by Macdonald et al. (2004), based on a two-tailed Pearson's Correlation analysis. Macdonald et al. (2004) indicated a slight, positive relationship between saturated fat intake and lumbar spine (unadjusted: $r=0.058$; adjusted: $r=0.044$) and femoral neck (unadjusted: $r=0.017$; adjusted: $r=0.037$) bone mineral density. Although the current study examined pelvic bone mineral density, as opposed to the femoral neck, the present findings also found a positive correlation between saturated fat intake and spine ($r=0.289$, $p=0.152$) and pelvic ($r=0.14$, $p=0.484$) bone mineral density. Despite the similarities, the study by Macdonald et al. (2004)

featured only female subjects (baseline: 47.5 ± 1.5 years; follow-up: 53.9 ± 1.6 years), whereas the present study analyzed both males and females combined (50 to 80 years).

Interestingly, Corwin et al. (2006) and Macdonald et al. (2004) found similar effects on bone mineral density from saturated fat intake based on individual gender. Corwin et al. (2006) found a 2.3% decrease in femoral neck bone mineral density based on the lowest to the highest saturated fat intake for both males and females combined (lowest: ≤ 14.4 g, 0.843g/cm^2 ; highest: 36.4 - 130.3 g, 0.824g/cm^2). Individual gender analysis indicated that the negative association was more pronounced in men compared to women. This is believed to have heavily influenced the findings by Corwin et al. (2006), since females showed no statistically significant relationship to bone mineral density at any of the examined sites (femoral neck, trochanter, intertrochanter, and total hip). Although the current study combined males and females for analysis, due to the small sample size ($n=26$), females predominately accounted for the majority of the sample ($n=20$, $\sim 77\%$). It is possible that the current findings are also heavily influenced by the female subjects. The findings in this study, along with the findings by Corwin et al. (2006) and Macdonald et al. (2004), may indicate that bone mineral density is less likely to be negatively influenced from saturated fat intake in females. However, at this point in time, it remains as speculation for future research to address. In conclusion, the current research indicates that total dietary saturated fat intake may positively influence spine and pelvic bone mineral density in older adults.

Recommendations

Although this research failed to provide any significant results, the inconsistencies among research findings on saturated fat and bone health should not be overlooked. Osteoporosis continues to be a growing issue throughout the world, and remains a common problem among

older adults (Ahmadiéh & Arabi, 2013). Research should continue to investigate the role of saturated fats on bone health to fully understand the relationship between the two.

There is a limited amount of research on saturated fat intakes effect on bone health, while even less research has been specifically conducted on saturated fat intakes effect on bone mineral density. Further research investigating the relationship between saturated fat and bone mineral density could help determine an explanation for the interaction between the two variables, while studies investigating the risk for fracture make it more difficult to distinguish a “cause”.

Hogstrom et al. (2007) found that dietary PUFA’s intake was associated with a positive correlation for total body and spine bone mineral density, while a negative correlation was found between MUFA intake and total body bone mineral density. PUFA and MUFA are both unsaturated fats, yet they had a different effect on bone mineral density. It is possible that the lack of evidence on saturated fat intake and bone health is due to specific saturated fats contradicting the effects of one another. Gacs and Barltrop (1976) found that stearic acid, a saturated fatty acid, significantly increased Ca-soap formation in the intestines and in the feces, which led to a decrease in calcium absorption. Future research may benefit from researching specific saturated fatty acids, since the relationship between saturated fat and bone health is still unclear.

Research on dietary fats has predominately focused on females, which is not surprising since women have a higher risk of developing osteoporosis than men (National Osteoporosis Foundation, 2014g). Since Corwin et al. (2006) identified a negative relationship between saturated fat intake and bone mineral density in men, as opposed to women, more research should be conducted on males to further investigate the relationship between saturated fat and bone mineral density.

Future research would benefit more from a longitudinal study design, to better observe a change in bone mineral density based on saturated fat intake over a period of time, rather than from one point in time. Finally, it is also recommended that a larger sample size is utilized for future research. This would not only help with gender diversity, but it would increase the chance of finding statistically significant results.

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Appendix A: Institutional Review Board Approval

April 30, 2014

Tyler Mathews
Food and Nutrition
UW-Stout

RE: The association between dietary fat intake and bone mineral density in older adults in the Midwest

Dear Tyler:

The IRB has determined your project, "*The association between dietary fat intake and bone mineral density in older adults in the Midwest*", is **Exempt** from review by the Institutional Review Board for the Protection of Human Subjects. The project is exempt under **Category 1** of the Federal Exempt Guidelines and holds for 5 years. Your project is approved from **April 30, 2014** through **April 29, 2019**. Should you need to make modifications to your protocol or informed consent forms that do not fall within the exemption categories, you will need to reapply to the IRB for review of your modified study.

If your project involved administration of a survey, please copy and paste the following message to the top of your survey form before dissemination:

This project has been reviewed by the UW-Stout IRB as required by the Code of Federal Regulations Title 45 Part 46


If you are conducting an **online** survey/interview, please copy and paste the following message to the top of the form:

"This research has been reviewed by the UW-Stout IRB as required by the Code of Federal Regulations Title 45 Part 46."

Informed Consent: All UW-Stout faculty, staff, and students conducting human subjects research under an approved "exempt" category are still ethically bound to follow the basic ethical principles of the Belmont Report: 1) respect for persons; 2) beneficence; and 3) justice. These three principles are best reflected in the practice of obtaining informed consent from participants.

If you have questions, please contact Research Services at 715-232-1126, or foxwells@uwstout.edu, and your question will be directed to the appropriate person. I wish you well in completing your study.

Sincerely,


Susan Foxwell
Research Administrator and Human Protections Administrator,
UW-Stout Institutional Review Board for the Protection of Human Subjects in Research (IRB)

CC: Karen Ostenso