University of Northern Iowa UNI ScholarWorks

Dissertations and Theses @ UNI

Student Work

1992

A descriptive study of bone health risk factors in females ages 13 to 16 years

Anita M. Johnson University of Northern Iowa

Let us know how access to this document benefits you

Copyright ©1992 Anita M. Johnson

Follow this and additional works at: https://scholarworks.uni.edu/etd

Part of the Orthopedics Commons, and the Pediatrics Commons

Recommended Citation

Johnson, Anita M., "A descriptive study of bone health risk factors in females ages 13 to 16 years" (1992). *Dissertations and Theses @ UNI*. 696. https://scholarworks.uni.edu/etd/696

This Open Access Thesis is brought to you for free and open access by the Student Work at UNI ScholarWorks. It has been accepted for inclusion in Dissertations and Theses @ UNI by an authorized administrator of UNI ScholarWorks. For more information, please contact scholarworks@uni.edu.

Offensive Materials Statement: Materials located in UNI ScholarWorks come from a broad range of sources and time periods. Some of these materials may contain offensive stereotypes, ideas, visuals, or language.

A DESCRIPTIVE STUDY OF BONE HEALTH RISK FACTORS IN FEMALES AGES 13 TO 16 YEARS

An Abstract of a Thesis

Submitted

In Partial Fulfillment

of the Requirements for the Degree

Master of Arts

Anita M. Johnson University of Northern Iowa

July 1992

ABSTRACT

The purpose of this study was to describe the known risk factor profile for bone health in an apparently healthy female adolescent population. A risk profile of nine factors, compiled from the current research literature included the following risks: (a) Female gender, (b) family history of osteoporosis, (c) Caucasian or Oriental race, (d) slender body frame/underweight, (e) dietary intake less than 1500 mg calcium/day, (f) weight-bearing physical activity less than 60 minutes three times a week, (g) amenorrhea or irregular menses associated with strenuous physical training, (h) smoking, and (i) alcohol use.

A three-day diet record was used to determine average daily intake of calcium, phosphorus, total protein, and total calories. A personal health questionnaire provided an estimate of physical activity levels, menstrual history, family history of osteoporosis, as well as history of cigarette and alcohol usage. Measurement of height, weight, and two skinfolds yielded a body mass index and estimates of body composition which were used to determine desirable weight for height.

Sixty female 8th, 9th, and 10th grade students attending Malcolm Price Laboratory School, ages 13 to 16 years participated in the study. Three of the nine risk factors dominated the bone health profile: Female gender (100%), Caucasian or Oriental race (94%), and low calcium intake (92%). The shortage of calcium found in these diets was possibly compounded by a high protein intake in 82% of participants. The frequent pattern of a Moderate to Very Active lifestyle (98%) is thought to be beneficial to bone health, while minimal smoking (2%), and low alcohol usage (7%) are potentially protective factors. Individuals with a small body frame or underweight for height (15%) were noted in each age group, while the factor of < 2 menses per year (15%), exhibited by 13-, 14-, and 15-year-olds, was most likely associated with delayed menarche. Additionally, 30% of participants reported some irregularity of menses during active sports seasons.

Thirty-seven (62%) of participants recorded three risk factors, 16 (27%) had four factors, and five subjects (8%) accumulated five risk factors. Individual combinations of multiple risk profiles were quite diverse.

A DESCRIPTIVE STUDY OF BONE HEALTH RISK FACTORS IN FEMALES AGES 13 TO 16 YEARS

A Thesis

Submitted

In Partial Fulfillment

of the Requirements for the Degree

Master of Arts

Anita M. Johnson

University of Northern Iowa

July 1992

This study by: Anita M. Johnson

Entitled: A DESCRIPTIVE STUDY OF BONE HEALTH RISK FACTORS IN FEMALES AGES 13 TO 16 YEARS

has been approved as meeting the thesis requirement for the Degree of Master of Arts

$$\frac{6-24-92}{\text{Date}}$$

$$\frac{10-24-92}{\text{Date}}$$

ii

TABLE OF CONTENTS

			Pł	\GE
LIST	OF	TABLES	. 、	7
LIST	OF	FIGURES	. 、	/i
CHAP	FER			
I.	IN	TRODUCTION	•	1
		Statement of the Problem	•	6
		Significance of the Study	•	6
		Delimitations	•	7
		Limitations	•	8
		Assumptions	•	8
		Definitions of Terms	•	9
II.	RE	VIEW OF RELATED LITERATURE	. 1	1
		Bone Structure	. 1	L 2
		Bone Physiology	. 1	L 3
		Adolescent Bone Growth	. 1	6
		Peak Bone Mass Concept	. 1	7
		Factors Affecting Peak Bone Mass	. 1	9
		Genetic Endowment	. 1	.9
		Nutrition and Bone Health	. 2	22
		Physical Activity	. 3	11
		Hormonal Factors	. 3	16
		Slender Body Frame	, 4	3
		Lifestyle Factors	. 4	5
		Adolescent Risk Factor Profile	, 4	5
		Summary	. 4	17
III.	ME	THODOLOGY	4	9

1	Resea	arch Design	49
5	Subje	ect Selection	49
I	Resea	arch Apparatus	50
	Tł	nree-day Dietary Intake Record	50
	Qı	lestionnaire	50
	Aı	nthropometric Data	52
]	Proce	edures	53
I	Data	Description	56
I	Data	Analysis	57
IV. RESU	JLTS	AND DISCUSSION	59
I	Demog	graphic Description	59
I	Dieta	ary Intake of Nutrients	61
1	Physi	ical Activity	67
1	lenst	trual Status	68
I	Body	Frame Size	70
1	Famil	ly History of Osteoporosis	74
C	Cigar	rette Smoking and Alcohol Consumption	74
I	Risk	Factor Profile	75
2	Summa	ary	79
V. CONC	CLUSI	IONS AND RECOMMENDATIONS	80
c	Concl	lusions	80
I	Recor	nmendations	81
REFERENCI	Es		84
APPENDIX	A:	Three-day Diet Record	98
APPENDIX	в:	Questionnaire1	00
APPENDIX	с:	Informed Consent1	03
APPENDTY	р:	Nomograph for Body Mass Index	05

LIST OF TABLES

TABLE	PAGE
1. Age and Race of Participants	
 Dietary Intake of Calories, Calcium, and Protein 	Phosphorus,
3. Physical Characteristics of Subjects.	
 Body Mass Index, Sum of Skinfolds, ar Percentage Estimates 	nd Body Fat
5. Occurrence of Multiple Risk Factors i Population	in Sample
6. Intercorrelations of Risk Factors	

LIST OF FIGURES

FIGURE

1.	Median calcium intake for United States females compared to National Institute of Health calcium intake recommendations	30
2.	Average and low bone mass concentrations, their relationship to fracture susceptibility, and the effects of calcium intake, genetics, physical activity, and hormones over a lifetime	46
3.	Average caloric intake of participants, ages 13 to 16 years compared to recommended caloric intake	62
4.	Mean calcium intakes as percentage of recommended intake	63
5.	Average hours per week of weight-bearing activity and incidence of irregular menses at each level	67
6.	Menses per year and reported irregular menses with sports activities	69
7.	Frequency of risk factors in the sample population	75

CHAPTER 1

INTRODUCTION

The age-related process of bone loss is a serious public health problem for many populations (Garn, Rohman, & Wagner, 1967; Newton-John & Morgan, 1970; Nordin, 1966; Riggs & Melton, 1986). A gradual demineralization and subsequent thinning imperceptibly erodes bone of volume and mineral density, making it more susceptible to fracture. This phenomenon, known as osteoporosis, affects 25 million persons in the United States alone; nearly 50% of women over age 45 and 25% of men (National Osteoporosis Foundation, 1991). The resultant 1.3 million fractures are estimated by the National Institute of Health to cost \$7-10 billion annually in health care and reduced productivity (Windom, 1989). The impact on quality of life due to disability and pain is immeasurable.

The focus of osteoporosis research has been directed toward slowing the age-related loss of bone and preventing fractures in the elderly female. Encouraging advances have been made in understanding the disease, identifying persons at risk, and improving treatment. Increasingly, post-menopausal women have shown benefit from exogenous estrogen to maintain or slow the loss of their bone mass (Heaney, Recker, & Saville, 1978; Johnston, 1989; Lindsay, et al., 1978). Yet there is no cure, and current treatment of osteoporosis often provides some maintenance of bone density but proffers minimal reconstruction of bone tissue (Heaney, 1983; Matkovic, Fontana, Tominac, Goel, & Chesnut, 1990).

A growing emphasis has been on primary prevention of osteoporosis by minimizing the rate of bone loss after menopause and, even more critically, augmenting the peak bone mass (PBM) at skeletal maturity (Matkovic et al., 1990). It is this initial ingathering of bone density that defines the greatest potential mass and provides a defense against microdamage and fractures associated with diminshing bone mass (Burckhardt & Michel, 1989; Garn et al., 1967; Newton-John & Morgan, 1970; Smith, Nance, Kang, Christian, & Johnston, 1973). This early acquisition of bone has piqued the interest of researchers recently, stimulating a new focus for osteoporosis prevention: adolescence.

Childhood and adolescence is characterized by rapid growth patterns. As much as 50% of the adult skeletal mass formation happens during the adolescent years alone (Kreipe & Forbes, 1990). Likewise, bone remodeling is very active with the replacement rate in children as much as 10 times greater than in adults (Mazess & Cameron, 1971). Although the physiology of bone loss has been extensively described (Garn et al., 1967; Newton-John & Morgan, 1970), much less is known about mechanisms that influence the magnitude of the peak bone mass. As yet, there have been no longitudinal studies completed that confirm the effects of various factors on PBM. Recent studies of young female amenorrheic athletes as well as females with anorexia nervosa have shown

significantly reduced bone mass, reaffirming that bone structure is sensitive to its nutritional and hormonal milieu (Ayers, Gidwani, Schmidt, & Gross, 1984; Bachrach, Guido, Katzman, Litt, & Marcus, 1990; Biller et al., 1989; Drinkwater et al., 1984; Kreipe & Forbes, 1990).

There is no doubt that the formation of PBM is multifactorial. The quantity and quality of bone mass at any age is the consequence of complex interactions of several variables. While genetic heritage apparently establishes the level of potential bone density (Goldsmith, Johnston, Picetti, & Garcia, 1973; Smith et al., 1973), endocrine function, nutrition, and exercise patterns regulate the realization of this potential (Brewer, Meyer, Keele, Upton, & Hagan, 1983; Matkovic et al., 1990; Nilsson & Westlin, 1971; Smith & Gilligan, 1989). Determining the ideal contributions of these factors is the current challenge.

Five categories of interest that influence peak bone mass are: genetics, nutrition, physical activity, hormones, and lifestyle factors. These categories parallel the most widely accepted risk factors for development of osteoporosis in the adult female. Genetically determined risk factors include the female gender, Caucasian or Oriental race, family history of osteoporosis, and a slender body frame.

Nutritionally, the intake of calcium, Vitamin D, protein, and phosphorous have been known to have a critical influence on the development of bone (Committee on

Nutrition, 1978; Garn, 1970; Leighton & Clark, 1929; Leitch & Aitken, 1959; Matkovic, Fontana, Tominac, Lehman, & Chesnut, 1986; Matkovic et al., 1979; Orr, 1928). Yet, considerable evidence points to frequent dieting and abnormal eating habits in adolescents. According to surveys, as high as 70% of female adolescents label themselves as "fat" (Nylander, 1971; Shapiro, Hampton, & Huenemann, 1967), 66% were preoccupied with weight and dieting (Casper & Offer, 1990), 30% are dieting (Crowther, Post, & Zaynor, 1985; Dwyer, Feldman, & Mayer, 1967), 79% binge (Crowther et al., 1985; Hawkins & Clement, 1980), and 5-10% are bulimic or anorexic (Joyce, Warren, Humphries, Smith, & Coon, 1989). If these studies are externally valid, it should not be surprising that the National Health and Nutrition Evaluation Survey (NHANES) I, 1971-1974 (Abraham, Carroll, Dresser, & Johnson, 1977) and NHANES II, 1976-1980, (Carroll, Abraham, & Dresser, 1983) found that, as early as age 10, the intake of calcium was below the 1983 Recommended Daily Allowance (RDA) standards of 1200 mg/day for adolescents and was chronically less than 800 mg/day during the adult years.

Physical activity is important to bone health, as is evidenced by the immediate and drastic loss of density with immobilization or, as discovered with the American Apollo astronauts, the weightless environment of space (Krolner & Toft, 1983; Rambaut, Mack, & Vogel, 1975). Paradoxically, athletic training to the point of amenorrhea has produced decreases in bone density (Buchanan, Myers, Lloyd, Leuenberger, & Demers, 1988; Davee, Rosen, & Adler, 1990; Kanders, Dempster, & Lindsay, 1988) suggesting that there may be an upper limit to the beneficial influences of physical activity on bone, not as a result of physical activity alone, but due to hormonal fluctuations. This concurrent action of hormones on determinants of bone mass is echoed throughout the adult stages of bone life. Even a low weight for height, especially during puberty, correlates with a lower skeletal mass (Kreipe & Forbes, 1990)

The lifestyle factors of alcohol consumption and cigarette smoking are known to have a negative effect on bone density. Although the mechanisms are not fully understood, the life-long consequences are unquestionably detrimental (Lindsay, 1981; MacMahon, Trichopoulos, Cole, & Brown, 1982; National Institute of Arthritis, Musculoskeletal, and Skin Diseases [NIAMS], 1986).

Prevention begins with an awareness of the risks within the targeted population. The prevalence of dieting and variable eating habits in adolescence is well documented and the subsequent intake of calcium repeatedly found to be lower than the RDA. There is a lack of information, however, regarding the status of other bone risk factors in the healthy adolescent population, particularly those related specifically to the formation of a maximal peak bone mass. When evaluating skeletal mass, many interactive factors need to be considered in order to form a composite of probable bone health. These genetic, nutritional, physical activity, hormonal, and lifestyle indicators are valuable when planning health education curriculum.

Statement of the Problem

The purpose of this study was to describe the known risk factor profile for bone health in an apparently healthy female adolescent population.

Significance of the Study

"Primary prevention consists of actions taken in the absence of signs or symptoms" (Green & Kreuter, 1991, p. 133). It is this early prevention approach that seems to be the best available method to avoid the crippling and painful results of osteoporosis; accumulating as much bone tissue as possible before age and menopause-related demineralization begins. During the adolescent years, almost half of the adult bone mass is laid down, providing an opportunity to optimize bone density during a metabolically active stage.

Since it is not practical nor probably desirable to routinely measure bone mass densities among adolescent females, a second line of prevention would be to determine the health of bone by assessment of risk factors. Although there are numerous factors mentioned in the literature, the most widely accepted are: (a) female gender, (b) Caucasian or Oriental race, (c) family history of osteoporosis, (d) slender body frame, (e) chronic low calcium intake, (f) lack of physical activity, (g) alcohol use, and (h) cigarette smoking (National Institute of Health, 1986). For adolescents, an added concern would be amenorrhea or exercise-induced amenorrhea and its harmful hypoestrogenic effects (Drinkwater, Bruemner, & Chesnut, 1990; Drinkwater et al., 1984; Drinkwater, Nilson, Ott, & Chesnut, 1986; Fisher, Nelson, Frontera, Turksoy, & Evans, 1986).

The assessment of these risk factors in a specific population serves a dual purpose: (a) Provides a profile of factors that may indicate specific areas of need for intervention by a health professional and, (b) promotes awareness in adolescents of the many interactions of diet, exercise, hormones, and lifestyle to skeletal health. Identifying those individuals at risk for a lowered PBM is essential in the adolescent years when bone mass can be significantly increased through healthy dietary and physical activity adaptations (Kelsey, 1989).

Delimitations

This study was delimited to:

 Female 8th, 9th, and 10th grade students, 13 to 16 years, attending Malcolm Price Laboratory School, Cedar Falls, Iowa, the Spring semester of 1992.

2. Dietary intake of nutrients as determined by a three-day diet record and subsequent evaluation by "The Food Processor" computer program (ESHA Research, Inc., 1986).

3. Weight measured to the nearest 0.5 pound on a level balance beam scale.

4. Standing, barefoot, height measurements to the nearest centimeter using a Physician's platform scale with vertical bar, steel measuring tape and horizontal bar attached to indicate height.

5. Body composition as measured by the sum of the triceps and mid-calf medial skinfold thicknesses.

6. Participants responses to a questionnaire concerning their own health history, use of vitamins, physical activity levels, and family history of osteoporosis.

Limitations

The following limitations of this study are noted:

1. Results of this study were limited to the responses received from female 8th, 9th, and 10th grade students attending Malcolm Price Laboratory School in the Spring semester of 1992. Results, therefore, may be suggestive for females in a similar setting, but are not generalizable to other populations.

2. The results presented in this study are based on the responses of 60 students out of a total of 78 invited to participate in the study. The self-exclusion of 18 females may distort the total picture of bone health risk factors for this population.

<u>Assumptions</u>

In carrying out this study, the following assumptions were made:

1. The three-day diet record was a reliable and valid representation of the average daily intake of the participant.

2. The questionnaire as a tool for data collection was reliable and valid.

3. The participants were accurate in their answers.

4. The participants were able to read and understand the questions as the researcher intended, thereby eliciting accurate information.

5. The questions were answered in accordance with the instructions given.

6. The height, weight, and body composition values and measurement procedures were valid and reliable.

Definitions of Terms

The following terms were operationally defined for this study:

<u>Amenorrhea</u>. Less than two menses per year or an absence of menses (Baker & Demers, 1988; Lloyd, Myers, Buchanan, & Demers, 1988).

Body Mass Index (BMI). A ratio that describes body mass as weight in kilograms/height in meters² (Keys, Fidanza, Karvonen, Kimura, & Taylor, 1972).

Bone Density. The amount or mass of bone present in a certain volume of bone.

Bone Mass. The total amount of bone in the body. Overall bone mass increases from birth and reaches a peak in the third and fourth decade and subsequently declines with age (Notelovitz & Ware, 1982).

Bone Mineral Content (BMC). The crystalline component of bone which gives bone strength; calcium and phosphorous in the form of hydroxyapatite are the principal components of bone mineral. Its concentration is measured in grams/centimeter².

<u>Cortical Bone</u>. The dense form of bone that makes up the shafts of long bones and the outer walls of other bones.

Eumenorrhea. Regular, cyclical menstrual function with 8 to 13 menses per year (Baker & Demers, 1988; Lloyd et al., 1988).

Oligomenorrhea. The occurrence of three to seven menses per year (Baker & Demers, 1988; Lloyd et al., 1988).

Osteoporosis. A reduction in overall bone mass characterized by increased porosity and thinning of the bone tissue to the point that microscopic or more obvious fracturing has occurred (Garn et al., 1967; Notelovitz & Ware, 1982).

<u>Peak Bone Mass</u> (PBM). The highest value of quantity and density of bone reached in life (Burckhardt & Michel, 1989).

<u>Risk Factor</u>. A condition that increases the likelihood that an individual will develop a particular health problem.

<u>Trabecular Bone</u>. The spongy-appearing form of bone that fills the ends of long bones and the interior of the vertebrae.

CHAPTER II

REVIEW OF RELATED LITERATURE

Even though the pathophysiology of osteoporosis has been extensively described, there is a wide variation in the pattern and amount of bone lost in individual women (Culliton, 1987; Garn et al., 1967; Guyton, 1976; National Institute of Health [NIH] Osteoporosis Consensus Conference, 1984; Newton-John & Morgan, 1970; Notelovitz & Ware, 1982; Riggs & Melton, 1986). Extensive studies of this process, particularly during the peri-menopausal sequence, have revealed numerous hormonal and dietary interactions that have significant consequences on the health and equilibrium of the bone matrix. These physiologic interactions that increase vulnerability to accelerated bone loss are described clinically as risk factors that may reduce or enhance the probability of an effective bone structure. By assembling what is known of these many complex interactions, an environment that would stimulate a strong peak bone mass during adolescence is suspected, though not completely verified.

This review of the current research focuses initially on the normal anatomy and physiology of bone tissue, then explores the peak bone mass concept and five areas of influence: genetics, nutrition, physical activity, hormones, and lifestyle habits. Finally, a composite of recommendations for optimal bone mass, derived from current literature is proposed for an adolescent female population.

Bone Structure

Mammalian bone is a complex and highly adaptable organ. Its unique functions require both rigidity and flexibility. As bone provides a framework for the attachment of muscles, it also allows for a vast variety of movement and maintains the general shape of the body. The skull, rib cage, vertebra, and pelvis provide protection for soft tissues, while red blood cells are manufactured deep within the bone marrow. Bone tissue is a storage site for 99% of the body's supply of calcium. This not only strengthens the bone, but maintains the blood calcium concentration at a steady level by moving calcium ions between the bone matrix and the extracellular fluid as needed by the tissues. This homeostasis is controlled by the parathyroid hormone (PTH) and the thyroid hormone, calcitonin (Hancox, 1972).

There are two types of tissue in every bone. The solid, dense area of the external envelope is called cortical or compact bone and the central core adjacent to the bone marrow is made up of a spongy, cancellous, trabecular bone. These two types of bone are found throughout the skeleton in varying proportions. The vertebrae and skull and thorax of the axial skeleton are primarily trabecular bone surrounded with a thin cortical layer. The long bones of the appendicular skeleton consist mostly of cortical bone with areas of trabecular bone at both ends. These amounts differ due to specific requirements for strength or lightness (Hancox, 1972).

There are three surfaces on which bone formation takes place. The periosteum forms an envelope around the outer-most surface while the endosteum envelope lines the marrow cavities and trabecula. It is thin and irregular, resulting in a large surface area. Within the dense cortex are the vast Haversian and Volkmann canal systems which create a large intracortical surface area. The Haversian system consists of a central canal for the passage of extracellular fluids, ions, and small blood vessels, surrounded by concentric circles of bone matrix called lamellae. Multidirectional layers of collagen fibers within the lamellae become the framework for calcium and phosphate deposition in the form of hydroxyapatite crystals. The fibers have a great tensile strength and the calcium crystals add compressional strength which together provide a tough rigid structure. Guyton (1976) states that the tensile and compressional strength of healthy bone is very similar to that of reinforced concrete.

Bone Physiology

The continual renewal and replenishing of cells is ongoing in all body tissues and especially so within bone. This process is known as bone remodeling. Its function is twofold: to repair microfractures and to preserve an adequate amount of metabolically young bone that is able to maintain the homeostasis of essential minerals (Parfitt, 1981). Heaney and Barger-Lux (1988) describe this energetic self-renewal process to be capable of replacing an entire

skeleton once every 8 to 16 years. The remodeling process is repeated at millions of sites which were described first by Frost (1982) as Basic Multicellular Units (BMU). Each remodeling unit in a normal adult removes and replaces 0.1 cubic millimeter of bone. The five successive stages are: (a) Resting phase (quiescence), when the outer layer of lining cells are intact, (b) resorption of the bone surface by the dissolving action of the osteoclasts on the matrix below, (c) completed resorption results in the creation of small crevices on the surface, (d) repairs are made within the crevices by osteoblasts, structuring a new layer of bone, (e) the bone returns to a resting phase before beginning the cycle again (Peck & Avioli, 1988).

Interestingly, the trabecular bone is active at approximately 900 sites/day, while cortical bone has about 100 active sites/day, this translates to an annual turnover rate of nearly 2-3% in cortical bone, and 25% in trabecular areas (Shane, 1988). Thus, changes in bone quality and quantity will be apparent in trabecular sites earlier than cortical sites.

In children, one complete sequence of a BMU is accomplished in 1 to 2 months. A young adult BMU cycles in 3 to 6 months, while the mechanism may require as long as 2 years in the elderly (Frost, 1982). Heaney and Barger-Lux (1988), have also noted evidence of incomplete remodeling activity in older persons.

The balance between resorption and formation is controlled by calcitonin and PTH, with formation proceeding more quickly than resorption in children, equally in adults and then, resorption dominates formation in the pattern of age-related bone loss. It is this cumulative imbalance that is responsible for the thinness and porosity of osteoporosis. Even a 10% loss of BMD may double or triple the risk of fracture (Kreipe & Forbes, 1990).

Cooper (1824), was the first to suggest that the increased frequency of fractures in the elderly population could be due to thinner bones. Albright, Smith, and Richardson (1941), identified generalized osteoporosis as a product of excessive bone loss, and suspected estrogen as an important ingredient in this process. In 1970, Newton-John and Morgan hypothesized that age-related bone loss occurs in all adults and that the most important determinant of osteoporosis is the amount of bone density accumulated at skeletal maturity. Clinically, this is important as the person that inherits a thin, light-weight skeletal frame, has age-related bone loss, or has lost density as a side-effect of corticosteroids may experience more bending and twisting of bones with ordinary work or exercise resulting in more microfractures than in a dense bone. Repairing these cracks is imperative, particularly if bone is thin and fragile, in order to maintain a functional skeleton and to avert further stress and fractures (Heaney & Barger-Lux, 1988).

Adolescent Bone Growth

Bone mass increases rapidly throughout infancy, childhood, and adolescence. Bone mineral content in children from 6 to 14 years increases by about 8.5% each year (Mazess & Cameron, 1971). Then, during the adolescent growth spurt, linear growth may be as much as 0.25 mm per day, reflecting a very metabolically active stage. The epiphyseal growth plates increase and longitudinal growth continues until epiphyseal closure at about age 18 years in most populations (Gilsanz, Gibbens, Roe, et al., 1988). Concurrently, the density of bone increases throughout childhood and adolescence. Muscle mass also continues to develop and its peak is thought to coincide with that of the peak skeletal mass. Nevertheless, patterns of age-related bone density vary considerably, from person to person, depending on the site measured and the method of measurement (Garn & Wagner, 1969). Dequeker, Geusens, Wielandts, Nijs, and Verstraeten (1984) reported the range of density in young adults to vary as much as $\pm 20\%$ in the trabecular bone of the vertebrae.

Even when longitudinal growth is complete, the cortex and trabecular plates are still thin and porous partly because the growth spurt occurs faster than bone can absorb calcium and formulate a dense matrix. Heaney and Barger-Lux (1988), state that consolidation of bone matrix may continue in some adults through the twenties and into the

mid-thirties and is primarily a densification of cortical tissue.

The Peak Bone Mass Concept

The peak bone mass (PBM) attained by any individual is the highest quantity and density of bone reached in life and is the result of many complex influences. This amount is of great importance because the quantity and quality of bone mass at any age in adult life is determined by the size of the PBM and the individual sequence of bone loss (Buchanan, Myers, Lloyd, & Greer, 1988; Burckhardt & Michel, 1989; Kanders et al., 1988). For example, a high PBM with an average rate of loss provides the best protection against fracture and even with a rapid bone loss pattern, a high PBM delays the fracture risk significantly. In comparison, a low PBM becomes vulnerable to fracture more quickly with an average age-related loss and quite rapidly with the precipitous loss of estrogen accompanying surgical menopause (Notelovitz & Ware, 1982). Since the normal range of total bone density extends over 250 mg/ml, and the PBM values vary as much as ± 100 mg/ml, there can be great differences among individuals' bone mineral content (Cann, Genant, Kolb, & Ettinger, 1985; Firooznia, Golimbu, Rafii, Schwartz, & Alterman, 1984; Gilsanz, Gibbens, Carlson, et al., 1988).

There has been considerable interest and some controversy about the age at which bone density peaks. One frequently cited study by Genant, Cann, Puszzi-Mucelli, and Kanter (1983), reported vertebral density to be at its height in the late thirties, but the study involved a large number of older women, few younger than 30 years, and only one younger than 20 years.

Mazess (1982) concluded the PBM in cortical bone is reached during the mid-thirties, while trabecular tissue peaks much earlier, probably before age 20. Awbrey et al. (1984) found no additional increases in BMC of the distal (trabecular) or the mid-radial (cortical) sites in subjects after age 20. In 1988, Gilsanz, Gibbens, Carlson, et al. compared cross-sectionally a much younger group of females whose ages ranged from 14 to 35 years. They observed bone density accrual to increase during adolescence and then to plateau between 14 years and 19 years. The results of that study suggested that vertebral density (trabecular) peaked much sooner, possibly near the end of longitudinal growth and closure of the epiphyses in the late teens or early This reaffirms the findings of Marcus, Kosek, twenties. Pfefferbaum, and Horning (1983), Riggs, Wahner, et al. (1986), and Pacifici, Susman, Carr, Birge, and Avioli (1987).

There is, as yet, no consensus about the sequence of peak density. Most disagreement centers on the exact timing of a peak mass attainment which seems to differ in trabecular tissue, depending on the site used for measurement. Furthermore, Buchanan, Myers, Lloyd, Leuenberger, et al. (1988), state that "no single study has

encompassed a sufficiently broad age range of subjects to include both the bone accretion that precedes the peak and the bone loss that follows it" (p. 674). Therefore, the exact timing of the PBM remains elusive. Even if the precise sequence is not known, the concept of the peak is extremely important because at any adult age, the existing bone density is a product of this peak and factors which are influencing it (Buchanan, Myers, Lloyd, & Greer, 1988; Evans et al., 1988; Goldsmith et al., 1973). It is this peak which has become the focus of preliminary research and preventative measures.

Factors Affecting Peak Bone Mass

Several interactive factors are known to influence bone metabolism. Genetic endowment, hormonal patterns, nutritional intake, and physical activity during growth and early adult life, as well as the lifestyle habits of smoking and alcohol use are thought to be the dominant determinants. Each has a distinct role in the development and maintenance of the skeletal system.

Genetic Endowment

Individual differences in bone tissue seem to vary substantially among ethnic groups. This phenomenon was noted by Herodotus, the Greek historian, in 430, B.C., as he described a long-deserted battlefield: "if you strike the Persian skulls, even with a pebble, they are so weak that you break a hole in them; but the Egyptian skulls are so strong that you may smite them with a stone and you will scarcely break them in" (Herodotus, 430, B.C./1947, p. 215). Epidemiological studies today also note vast differences in bone density and subsequent age-related loss patterns.

Bone density studies of North American Indians (Evers, Orchard, & Haddad, 1985) and St. Lawrence Island Eskimos (Harper, Laughlin, & Mazess, 1984) showed an average BMC lower than Caucasian women, while dark-skinned Caucasians of Spanish, Italian, Greek, or Middle Eastern heritage are less likely to develop osteoporosis (Farndon, 1985).

It is well established that Blacks average approximately 10% higher bone mass values than Caucasians, who generally have a greater bone mass than Orientals at maturity, even allowing for wide variation among individuals within each group (NIH Osteoporosis Consensus Conference Statement, 1984). These potentially genetic variations correlate with the observation that osteoporosis is much more common in Caucasian and Oriental women and rare in Black women (Smith et al., 1973).

Recognizable family and racial characteristics of bone structure and physiology suggest that there are significant genetic influences on the development of bone mass. While some individuals have large, heavy bones, others are dainty and light in skeletal structure. Smith et al. (1973) state that a person's genetic inheritance probably determines the highest achievable bone density, but several other factors mediate the attainment of that potential. It is postulated that Blacks may have denser bones due to increased resistance to the action of parathyroid hormone (PTH) which results in a higher level of circulating PTH, greater absorption of calcium from the diet, and conservation of renal calcium. The interaction of hormones is one possible way that genetic differences are expressed (Heaney & Barger-Lux, 1988). There may be concurrent environmental influences from nutritional patterns, prevalence of obesity, and the effect of a large muscle mass (Cohn et al., 1977).

Early studies of Garn (1963; 1970) with parent/offspring combinations and monozygous twins (Dequeker, Nijs, Verstraeten, Geusens, & Gevers, 1987) concluded that skeletal density seems to be predominantly a genetic determination. And in 1987, Pocock et al. suggested that a single gene or set of genes determines bone mass at all sites.

An ongoing study of 31 healthy Caucasian females, initiated at age 14, suggests a high correlation between potential PBM levels and the bone densities of both parents, rather than the mother or father alone. Furthermore, the BMC of the adolescents by age 14 years was already 90% of their premenopausal mother's BMC values and height (Matkovic & Chesnut, 1987) and 90-97% of their mother's BMC values by age 16 (Matkovic et al., 1990).

Using mother-daughter pairs, Lutz (1986) and Tylvansky, Bortz, Hancock, and Anderson (1989) also found a high correlation between parent and offspring bone densities. In contrast, Sowers, Burns, and Wallace (1986) found minimal correlation with mother/daughter pairs. This study however was based on a single, mid-radial, and primarily cortical site for bone mineral content analysis.

An interesting study from Australia tested relatives of persons known to have osteoporosis. The mean bone mass in these apparently healthy young adults was lower than the controls with no family history of osteoporosis (Evans et al., 1988).

Inheritance is thought to affect the development of osteoporosis in two ways: (a) Heredity determines the basic skeletal blueprint of bone size and density and thus its potential PBM at maturity and, (b) the rate of bone loss, possibly through hormonal influences, determines the amount of osteoporosis that occurs with the aging process (Smith et al., 1973). The combination of heredity and environment, as in the tendency towards obesity, highlights the complex interactions of numerous variables.

Nutrition and Bone Health

The impact of nutrition on bone health centers around the essential element, calcium. The functions and physiology of this mineral are extensively described, although data pertaining specifically to adolescence is scarce. While 99% of the available calcium is stored in the bones and teeth, another 1% circulates in the serous fluid, continuously supplying the calcium ions necessary for the

transmission of nerve impulses, blood clotting mechanisms, and muscle contraction, including the rhythmic cardiac contraction. It is also required in the synthesis of acetylcholine, absorption of Vitamin B_{12} , and in the function of numerous enzymes. Homeostasis of the serum calcium level is fundamental, therefore, if there is a deficit of dietary calcium due to inadequate intake or limited intestinal absorption, calcium is withdrawn from the bone matrix and used to maintain the serum level, resulting in a negative bone balance. It is this dissipation of stored calcium that is partially responsible for the loss of density in the adult (Sandler et al., 1985).

Calcium is a common element, occuring in abundance environmentally, yet, its bioavailability is dependent on three factors that influence any one individual's daily requirement: (a) Absorption efficiency by the intestinal tract, (b) the effect of calcium-regulating hormones, and (c) the natural obligatory loss function (Heaney & Barger-Lux, 1988). Absorption of calcium ranges from 10% to 50% of intake and is characteristically inefficient, depending on the amount available from the diet, individual metabolic patterns, and partially on the presence of Vitamin D. There is some evidence, however, that absorption rates may be higher during adolescence. Based on a study of 31, 14-year-old females, Matkovic et al. (1990), noted that their subjects were retaining higher amounts of calcium than is usually noted in adult subjects.

Whether ingested with food or manufactured in epithelial cells as a result of exposure to sunlight, Vitamin D is absorbed into the blood stream, converted to the active metabolite calcitriol, and acts on the small intestine to increase calcium absorption efficiency and on bone to enhance the resorption phase of remodeling. It also guards the extracellular fluid calcium balance (Heaney & Barger-Lux, 1988). A deficiency of Vitamin D (calcidiol levels between 15 ng/ml and 25-30 ng/ml) is thought to compromise calcium homeostasis, interestingly, supplementation with calcidiol boosts calcium absorption efficiency at levels of 30 ng/ml, but not at levels higher than this (Heaney, 1987).

As with other minerals, there are unavoidable obligatory losses of calcium in the urine, feces, and sweat. The amount of calcium excreted is influenced by the presence of other nutrients, so, even with a large calcium intake, eventually, only part becomes bioavailable.

Along with obligatory losses, an excess of protein has been shown to increase urinary calcium excretion. One of the natural by-products of the breakdown of protein is sulfuric acid which increases urinary calcium output (Heaney & Barger-Lux, 1988). Therefore, although protein is a vital nutrient, a diet high in protein, does require a higher calcium intake (Heaney, 1987). With the RDA of protein for adolescents set at 0.8 gm/kg of body weight, the average teen with a weight of 55 kg would require approximately 44 gm of protein/day. Heaney and Barger-Lux (1988) suggest that if daily protein was increased to 88 gm/day, the higher requirement would be approximately 200-300 mg of additional calcium/day. Since numerous bone surfaces are constantly remodeling and rejuvenating their structure, replacement minerals are necessary daily, making calcium repeatedly linked to the topic of bone health.

Numerous studies of calcium balance collected from the United States and Europe over the last 50 years suggested that the average daily requirement of calcium was approximately 400-600 mg/day for adults. Nutritionists subsequently set the Recommended Dietary Allowance (RDA) at 800 mg/day which was presumed to be above the actual requirement of 95% of the population and allowed for individual variations (Amschler, 1985; Heaney, 1987; Nordin & Heaney, 1990). But opinions surrounding the calcium issue have been subject to wide variation during the past 25 In the 1960s, calcium's connection with osteoporosis years. was not even mentioned in some major medical textbooks or was treated as minimally important. But, as new research trickled in, indications were that calcium was potentially very important (Marcus, 1987).

From 1982 to 1986, professional and public awareness of osteoporosis brought about a "calcium craze" (The Calcium Craze, 1986). The public was bombarded with information and advertising claims about the incredible benefits of calcium and a flurry of new studies investigated many aspects of

osteoporosis. Then, in 1984, due to this increased knowledge and a broader age-range of test subjects, the National Institute of Health recommended that the adult minimum daily intake of calcium be increased from 800 mg to 1000 mg, while children, adolescents, and pregnant women should consume 1,500 mg per day. Persons with osteoporosis, or at increased risk for developing osteoporosis, were also recommended to consume at least 1,500 mg of calcium per day. Subsequently, supplemental calcium sales increased from less than \$20 million a year in 1982 to over \$240 million in 1986 (Toufexis, 1987).

One of the pivotal reasons for this renewed interest in calcium was the study done by Matkovic et al., in 1979. Two Yugoslavian districts with population of similar genetic and cultural backgrounds but with an approximate twofold difference in average calcium intakes (500 mg/day vs. 1100 mg/day) were cross-sectionally compared. The district with the lower calcium intake had lower metacarpal density and more hip fractures than the higher calcium group. Significant differences in BMC were apparent even in 30-year-olds, but the two groups did not diverge further as age progressed which suggested calcium had an impact on the peak bone mass density in early adulthood.

This landmark study has been proposed as an indication that calcium intake early in life may have a long-term effect. From this data, Matkovic et al. (1979) suggested
that a high calcium intake may bolster the development of a higher peak bone mass but does not, by itself, prevent age-related bone loss. Interpretation of this cross-sectional study is hampered by the unknown factors of individual physical activity levels and hormonal variations.

A similar study was designed by Sowers, Wallace, and Lemke in 1985 utilizing two small demographically similar communities in northeast Iowa. Their water supplies were known to vary by approximately 315 mg/liter of elemental calcium, yearly, since 1938. A positive association between calcium intake and bone mass was found with both current and general calcium intakes significantly correlated with a higher mean forearm bone mass.

In Heaney's (1987) expansive review of nutrition and osteoporosis, out of 31 studies, (17 cross-sectional, 5 longitudinal, and 9 controlled studies) 21 showed a statistically positive correlation between bone density and calcium intake. Ten of these studies did not demonstrate a significant positive relationship, but, just as important, none of the studies showed a negative correlation. These investigations utilized primarily an early post-menopausal female population.

Despite the strong connection of calcium to bone mass formation, the National Aeronautics and Space Administration's bedrest studies showed that even a high calcium intake does not interrupt the bone loss of immobilization (Hantman et al., 1973). Furthermore, the

characteristic bone loss of the post-menopausal period occurs even with a high calcium intake; a persistant reminder that determinants of bone density are complex and interactive with exercise and hormones.

Nutritionists stress that an optimal diet includes a variety of foods that maintain a balanced intake of calcium (Ca) and phosphorus (P). A Ca:P ratio of 1:1 is considered ideal, not to exceed 3:1 or 1:3 (Finn, 1987). Albanese (1983) expressed concern that a persistent intake of high phosphorus foods may cause electrolyte imbalances that stimulate the skeleton to release stored calcium in order to maintain serum calcium homeostasis. Conversely, Heaney (1987) maintains that as the intake of phosphorus increases, the kidneys and intestine conserve calcium which preserves a positive calcium balance. Despite concerns from various researchers about possible phosphorus excesses due to the frequent use of soft drinks in the American diet, a conference called by the Federal Drug Administration (FDA) in 1981 reviewed available studies and concluded that excess phosphorus consumption becomes a problem only when these soft drinks replace dairy products and eliminate a prime source of calcium intake (FDA, cited in Heaney & Barger-Lux, 1988).

The actual daily calcium requirement for adolescents remains controversial. Even though the value of a high calcium intake has been documented, understanding the physiology of calcium is still tentative, particularly for

children and adolescents, due to the limitations of testing with this age group. Peacock (1991) documented the relationship between calcium intake, absorption, and the obligatory losses of calcium in sweat, feces, and urine, noting that even with an intake of 800 mg Ca/day, actual absorption may only provide 300 mg Ca/day. Coupling this with obligatory losses of 50-400 mg Ca/day in sweat, feces, and urine, a potential deficiency exists, particularly during the adolescent growth spurt. Furthermore, Miller, Kimes, Hui, Andon, and Johnston (1991) found that adolescents absorbed calcium at the same rates as adults, even though their requirements were much greater. It is again stressed by Peacock (1991), that direct measurement of calcium absorption, obligatory losses, and bone accretion rates of children and adolescents are tentative.

Confusing this issue is the continuing study by Matkovic et al. (1990) with 14-year-old females that reports a positive calcium balance was maintained with an intake of only 600 mg Ca/day, suggesting that the absorptive efficiency of the intestine is variable in response to the requirements of adolescence. It must be noted that this sample consisted of only 31 females. Even Heaney and Barger-Lux (1988) advise that individual variations can be quite diverse for calcium intake and obligatory loss.

The cross-sectional NHANES I, 1971-74 (Abraham et al., 1977) and NHANES II, 1976-80 (Carroll et al., 1983) surveys reported calcium intakes by women to be alarmingly low. The findings of NHANES I and II are superimposed on a graph of the NIH recommended amounts in Figure 1.



Figure 1. Median calcium intake for United States females compared to the National Institute of Health calcium intake recommendations (Adapted from NHANES I, 1971-74, Abraham et al., 1977 and NHANES II, 1976-1980, Carroll et al., 1983).

Not only were 75% of all the adult women surveyed ingesting less than 800 mg/day, 25% were getting less than 300 mg/day. "This latter figure is not enough to maintain calcium balance by any stretch of the imagination" (Heaney, 1987, p. 841). Ongoing studies by Matkovic et al. (1986) with adolescents showed calcium intakes ranging from 296 mg/day to 1801 mg/day. When compared to either the Food and Nutrition Board and National Academy of Sciences Recommended Dietary Allowances (RDA), or the NIH recommended amounts, a calcium deficiency possibly exists in a large portion of the adolescent population.

The best and most popular source of calcium in the United States is dairy products (NIAMS, 1984). The NHANES I (Abraham et al., 1977) and II studies (Carroll et al., 1983) found that soda often replaces dairy products in a teen-age diet which eliminates a valuable source of calcium. The evidence is strong that a high calcium intake may reduce the risk of a low peak bone mass, therefore, the current recommendation is that dietary calcium should be increased to the higher recommended amount of 1500 mg/day during puberty, in order to provide an environment that could augment the PBM significantly (Heaney, 1989).

Physical Activity

As early as the 17th century, Galileo discovered that there was relationship between mechanical forces and the shape of bone, noting that body weight and activity were related to bone size. In 1892, Wolff reported in <u>The Law of</u> <u>Bone Transformation</u> that a change in the function of bone resulted in predictable alterations in the internal and external architecture (Wolff, cited in Treharne, 1981). The mechanism responsible is thought to be piezoelectricity, a unique property of bone that allows mechanical energy to be converted to electrical energy. As bone is mechanically stimulated, a positive charge is produced which seems to

control the adaptive formation and resorption patterns (Bassett & Becker, 1962).

Dalsky (1990) presents four "concepts" regarding physical activity and its mechanical loading effect on bone (p. 281):

Concept 1. There is evidence that mechanical loading of the skeletal system contributes to an increase in bone mass (Dalsky, 1987; Drinkwater et al., 1984; Martin & McCulloch, 1987; Smith & Gilligan, 1989). This adaptation is illustrated by the increased bone mass of the playing arm of athletes in sports such as baseball and tennis. Athletes in weight-training have bone densities up to 40% higher in those sites being trained (Smith & Gilligan, 1989). Cross-country skiers and marathon runners have been noted to have higher bone densities than age-matched sedentary controls (Aloia et al., 1978). There are numerous crosssectional studies of bone mass in varying populations as well as several longitudinal studies that clearly demonstrate the positive relationship between weight-bearing exercise and increased bone density. When post-menopausal women were trained for 1-3 years, a smaller, yet positive effect of 3-8% increase in BMC was demonstrated (Dalsky, 1987).

<u>Concept 2</u>. The absence of physical activity results in a loss of bone density. Krolner and Toft (1983) observed 34 adults confined to bed and found a bone loss of 0.9% per week. When muscle activity is limited by immobilization

casting, mineralization is lost in the casted limb, but the unaffected limbs maintain their density (Jenkins & Cochran, 1969). A dramatic illustration of atrophy was noted in the astronauts of Gemini IV, V, and SkyLab, who, in an environment of weightlessness, lost the equivalent of 1-2% of their cortical bone mass a month, primarily from the lower extremities (Smith, Reddan, & Smith, 1981). This loss has been attributed to the lack of gravitational pull and minimal exercise. Likewise, non-weight-bearing simulations that reproduced the physical forces and hydrostatic pressures of walking did not reverse the negative calcium balance. While weight-bearing activity with large muscle involvement prevents mineral losses, weight-bearing without muscle activity apparently does not (Tipton, 1982).

Immobilization studies have shown an even greater loss of trabecular bone, about 1% per week, particularly from the vertebrae, but also from the os calcis. These depreciation rates are similar to those experienced by astronauts in the weightlessness of space. Fortunately, the reduced bone density can be regained in approximately 15 weeks (Krolner & Toft, 1983).

<u>Concept 3</u>. Mechanical loading of the skeleton during weight-bearing exercise stimulates new bone formation if it is a greater than average stimuli for the area. Since changes in bone mass are based on the daily, repeated stimulus to the bone, as in the tennis and baseball players' bones described previously, bone dynamically responds to

these forces and adapts its mass and shape in response to strain and activity. But, the reverse also happens, functional levels of bone mass are preserved only with a persistant stimulus (Lanyon, 1986).

Nilsson and Westlin (1971) ranked the order of distal femur density in a cross-section of athletes and non-athletes and found weight-lifters to have the highest density, then throwers, runners, soccer players, swimmers, exercising controls, and sedentary controls had the least. This emphasizes further that weight-bearing is essential to bone health.

Chesnut (1989) is cautionary with his comment that "while exercise would presumably have a positive effect on PBM attainment, no data are available to substantiate such a presumption in the adolescent female group" (p. 52). At the same time, mechanical stresses on the young maturing skeleton may be more important than on an older skeleton, potentially yielding the greatest positive effect in the young adult group (Kelsey, 1989). This sensitive response is thought to occur directly with mechanical loading and indirectly with an enhanced secretion of growth hormone (Heaney, 1984).

It has been noted that extreme amounts of physical activity in young people is associated with a higher bone mass as long as the activity in women does not result in amenorrhea. The altered hormone balance may lead to bone loss particularly in trabecular bone (Aloia et al., 1978;

Drinkwater, 1990; Drinkwater et al., 1984; Drinkwater et al., 1990; Huddleson, Rockwell, Kuland, & Harrison, 1980; Jones, Ravinikar, Tulchinsky, & Schiff, 1985; Joyce et al., 1989).

<u>Concept 4</u>. The adaptation of bone to mechanical strain is influenced by the hormonal, nutritional, and systemic conditions surrounding the bone tissue. While increased mechanical loading of physical activity stimulates mineralization of the skeleton, an adequate supply of nutrients, especially calcium, must be present in order for matrix formation and mineralization to take place (Heaney, 1989).

It is known that exercise-induced amenorrhea in young women is associated with decreased bone mineral content. Anorexic and bulimic women also have shown a decreased bone mass (Rigotti, Nussbaum, Herzog, & Neer, 1984). Until 1982, athletic amenorrhea was considered harmless and willfully reversible, usually to restore fertility. Encouraged by the understanding that physical activity would increase BMC (Smith et al., 1981), female athletes continued to exercise despite amenorrhea. When Gonzalez (1982) and Cann, Martin, Genant, and Jaffe (1984) reported a decrease in BMC in six long distance runners who were also amenorrheic, many were dubious. This began a flood of investigation with amenorrheic athletes and raised a whole new area of concern.

Drinkwater et al. (1984) repeated the 1984 study by Cann et al. and documented significantly less vertebral density in 14 amenorrheic runners than 14 eumenorrheic runners. The mean densities of the amenorrheic athletes were compared to lifetime average bone densities and found to be similar to those of a 50-year-old menopausal female.

Although low estrogen levels are initially thought to be responsible for amenorrheic bone loss, the systemic influences of inadequate nutrition, weight loss, low body fat percentage, energy drain, and chronic stress (glucocorticoid excesses) complicate this scenario. There is no doubt that the interactions are enigmatic, complex, and not well understood.

Hormonal Factors

The entire process of bone formation and resorption is controlled by a complex network of hormonal activity. The thyroid and parathyroid trigger the action of the osteoclasts while estrogen and calcitonin restrain the action of the osteoclasts as remodeling begins. Testosterone and progesterone are thought to enhance the kinetics of bone building. In fact, the characteristic growth spurt of adolescence is initiated by the gonadal hormones which are also thought to be responsible for the simultaneous acceleration of bone mineralization (Krabbe, Christiansen, Rodbro, & Transbol, 1979). This remodeling process is governed by a series of hormonal reactions that guard against a dangerous and precipitous loss (Heaney & Barger-Lux, 1988). An imbalance of any one of several parameters leads to an altered remodeling sequence. Whether the imbalance results from disuse (as in immobilization or weightless environments), inadequate calcium intake, estrogen deficiency, or late menarche, the result is a greater sensitivity of the bone matrix to PTH with a resultant increase in the number of active BMU sites. The overall effect is an increased rate of bone turnover, which, in the presence of a calcium or estrogen deficiency may lead to a gradual loss of bone (Dalsky, 1990). Additionally, a low serum estrogen causes increased urinary calcium loss and lower intestinal absorption, necessitating an increase in the daily calcium requirement (Nelson et al., 1986).

With the loss of ovarian function, whether by natural menopause or oophorectomy, there is a drop in natural circulating estrogen. The effects of this on bone are well documented (Albright et al., 1941; Garn et al., 1967; NIH Osteoporosis Consensus Conference, 1984; Notelovitz & Ware, 1982). In the first few years after menopause, a rapid loss of bone mass, as much as 2-3% per year, is common. After approximately 10 years, this rate slows to 1% per year and to 0.5% by age 70 (Heaney, 1987).

One of the more effective therapies for delaying or preventing this post-menopausal bone loss is exogenous estrogen. Its substantial benefits are offset by the potential risk of endometrial cancer in some women. When used in combination with progestin, this risk is allayed, but the protective effect estrogen has on the vascular system is negated (Barrett-Connor, 1989).

This beneficial effect of exogenous estrogen is documented also with young women using oral contraceptives (OC). Goldsmith and Johnston (1975), found an increase in cortical bone mass with OC use. Likewise, Kanders, Lindsay, and Dempster (1984) and Lindsay, Tohm, and Kanders (1986), found a significantly higher lumbar vertebral density in women using OCs, and estimated a 1% gain per year of usage.

The principle source of serum estrogen is from the conversion of androstenedione in adipose tissue in the body's periphery. This accounts for the positive correlation between body fat and serum estrogen levels in post-menopausal women (Bunt et al., 1990). It may also be responsible for the low circulating estrogen levels in very lean athletes.

Normal estrogen levels act to increase trabecular bone, and conversely, a low serum estrogen level, associated with menstrual dysfunction, results in reduced progesterone, reduced estradiol (E₂), and reduced bone density. Eumenorrheic athletes are noted by Lloyd et al. (1988), to have bone densities of approximately 197 mg/ml. Oligomenorrheic athletes (missed 50% or more of expected menses) often have densities that are much lower. This lower value becomes important as these women age because atraumatic vertebral fractures are known to occur frequently and easily in a trabecular bone with < 70 mg/ml of density.

It is ironic that weight-bearing, repetitive exercise so beneficial to bones, can also contribute to the hormone imbalance of menstrual dysfunction and loss of trabecular bone. This decrease in ovarian function appears to hinder the positive effect of exercise on bone metabolism (Baker, 1981; Cook et al., 1987). A review of studies involving BMC of adolescents and adults with anorexia nervosa hypothesized that the effect of the absolute level of estrogen on BMC was not as important as the length of time this value was below normal levels. Furthermore, the age at the onset of these dysfunctions may be critical to the amount of subsequent damage (Salisbury & Mitchell, 1991).

Another concern is the even more common type of overuse injury, the stress fracture. This is identified with repetitive, weight-bearing activities and encompasses a broad range of maladies, from sore and swollen feet to the more serious injuries with radiographic evidence of fractured and remodelled bone. The incidence of these injuries apparently is increased in females with irregular or absent menses (Jones, Harris, Vinh, & Rubin, 1989; Lloyd et al., 1986).

Lloyd et al. (1986), found radiologically confirmed fractures in 9% of young female athletes with regular menses but in 24% of oligomenorrheic or amenorrheic women (p < 0.025). Furthermore, Drinkwater et al. (1984) found that 2 out of 14 young amenorrheic runners had vertebral bone mineral densitites below the fracture threshold level,

likewise, 2 out of 19 oligomenorrheic runners were below the fracture threshold as reported in Lloyd et al. (1986).

In an attempt to determine the incidence of menstrual dysfunction during strenuous exercise, 28 untrained women with an average age of 22 years, were trained noncompetitively for eight weeks. Half of the subjects (Group I) maintained their initial weight and the other half (Group II) were on a restricted diet, and subsequently lost an average of four pounds. In Group I, 25% had no menstrual irregularities, 58% had abnormal bleeding and 8% had delayed In Group II, 6% had no menstrual irregularities, menses. 44% had abnormal bleeding, and 75% experienced delayed Despite these abnormalities, all subjects returned menses. to regular menstrual cycles within six months of the cessation of training, reaffirming the reversibility of this hormonal variation (Bullen et al., 1985).

The reversability of decreased bone mineral content is suspected in the young adult age group, but this phemonenon is minimally documented. Lindberg, Powell, Hunt, Ducey, and Wade (1987) reported "early osteopenia associated with exercise-induced menstrual dysfunction improved when runners reduced their running distance, gained weight, and became eumenorrheic" (pp. 218-219). Drinkwater et al. (1986) "concluded that the resumption of menses was the primary factor for the significant increase in the vertebral bone mineral density of the formerly amenorrheic athletes" (p. 380). Their sample of seven amenorrheic runners experienced a mean increase of 6% bone density in the lumbar spine during 15 months of resumption of menses. Also, Treasure and Russell (1987) found some early evidence that females with anorexia nervosa were able to restore bone mineral content as they regained body weight.

Exercise-induced menstrual dysfunction ranges from 10% to 40% of young female runners and is often concurrent with a low body fat percentage, more intense and greater distance running, low weight for height, as well as a history of menstrual dysfunction (Dale, Gerlach, & Wilhite, 1979; Feicht, Johnson, Martin, Sparkes, & Wagner, 1978; Schwartz et al., 1981; Shangold, 1980; Shangold, Freeman, Thysen, & Gatz, 1979). Amenorrhea is also reported in 44% of ballet dancers (Calabrese et al., 1983), 25% of non-competitive runners, and 12% of swimmers and cyclists (Sanborn, Martin, & Wagner, 1982). This contrasts with a 5% rate of menstrual dysfunction in a sedentary population (Speroff, 1982).

During amenorrhea, low concentrations of serum estrogens are well-documented. Nelson et al. (1986) found the average concentration of estradiol in amenorrheic women (< 20 pg/ml) to be 33% of the level found in eumenorrheic women and proposed that this low concentration of estrogen over a long period of time during adolescence may hinder the formation of a large PBM. Nevertheless, even though there is documentation of a low BMC in the lumbar vertebrae in amenorrheic athletes, a sedentary female with a low estrogen has even less BMC at this site (Dalsky, 1990). It is further postulated by Nelson et al. (1986) that amenorrhea may be a functional adaptation to a negative energy balance, either as a result of a low calorie intake alone or an increased energy expense in combination with a low calorie intake. This same process may be the cause of amenorrhea in anorexia nervosa. Bachrach et al. (1990) studied vertebral and whole body bone mineral density in female adolescents with anorexia nervosa (AN). Twelve out of the 18 subjects were found to have bone mineral densities two standard deviations less than values for age-matched controls. Therefore, Bachrach et al. (1990) stress the need for evaluation of bone composition in females with AN, especially if weight is low in comparison to height.

Since teenage girls are often obsessed with weight loss, fad diets, or exercise (Maloney, McGuire, Daniels, & Specker, 1989), Kreipe and Forbes (1990) have expressed concern about general growth and development in this age group. With Bachrach et al. (1990) finding a significant correlation of weight for height with BMD in both the studied population and controls, an appropriate weight gain during adolescence appears desirable for the development of a high PBM. Henderson (1991) asks "whether the positive effect of exercise or the negative effects of delayed menarche or other menstrual irregularities are in some way different during this period of skeletal development"

(p. 28), the answer is, as yet, unknown. Bullen et al. (1985) noted that in most amenorrheic athletes, moderation of training and weight gain prompts the return of menses. Again, the restoration of lost bone density with AN is reported in the study by Treasure & Russell (1987), in amenorrheic runners by Lindberg et al. (1987), and Drinkwater et al. (1986).

<u>Slender Body Frame</u>

A well-accepted risk factor for osteoporosis is the morphology of a small, thin body frame or being underweight for height (NIH, 1986). Several investigators have studied the positive association between slender body frame and low BMC and suggest two possible explanations: (a) The reduced mechanical loading on the skeletal frame from a small muscle mass stimulates less bone formation, therefore, a small female may not only have a lesser biological potential for bone mass, but also less mechanical stress during weightbearing exercise (Dalsky, 1990; Drinkwater et al., 1984; Martin & McCulloch, 1987; Smith & Gilligan, 1989); and (b) the lack of fat tissue results in less production of the female hormone, estrogen, which may result in a lower bone density (Drinkwater, Bruemner, & Chesnut, 1987; 1990).

In 1990, Drinkwater et al. found bone density to be significantly correlated with body weight at seven anatomical sites measured for BMC. However, an interesting interplay was noticed in that, "normal estrogen levels seemed to override any negative effect of decreased body

weight. As menstrual irregularities increased in severity, weight became a more important factor" (p. 548).

Body fat percentage. This indirect measure of body composition is often estimated by the sum of several skinfolds and applied to an equation developed for a specific population. In the healthy adolescent female, a 17% body fat is generally considered to be necessary to activate the onset of menstruation, while a normal, cyclical pattern requires a higher, 22% body fat in females over age 16 (Frisch & McArthur, 1974). In contrast to these established norms, individual patterns vary widely, as many female athletes with body fat less than 17% do report normal menstrual cycles throughout training (Shangold, 1980; Shangold et al., 1979).

Body mass index. The body mass index (BMI) is a widely used method of evaluating body composition in general and obesity in particular. Also known as Quetelet's Index, this ratio was devised by Quetelet in 1833 from an observation that in adults of normal size, weight is proportional to height squared (W/H²). This ratio was first named body mass index by Keys et al., in 1972. The use of BMI as a predictor of BMC has been tested by several groups (Bachrach et al., 1990; Davee et al., 1990; Mazess & Cameron, 1971; Rundgren, Eklund, & Jonson, 1984; Tylvansky et al., 1989). Its usefulness appears to improve somewhat with younger adult age groupings as stated by Tylvansky et al. (1989) who found BMI to be the only significant predictor of BMC values, when measured at mid and distal radial sites. Davee et al. (1990) also determined BMI to be the best predictor of BMC in a group of 20- to 30-year-old females. Likewise, Bachrach et al. (1990) discovered that in teens with anorexia nervosa, BMI alone emerged as positively associated with BMC. Moreover, the BMI of their age-matched controls correlated significantly with BMC using lumbar, radial, and whole body measurements.

Lifestyle Factors

Smoking and alcohol consumption both have a deleterious effect on bone health. The heavy use of cigarettes appears to reduce estrogen production (Lindsay, 1981; MacMahon et al., 1982; NIAMS, 1986), while alcohol may depress bone formation with a direct effect on osteoblasts (NIAMS, 1986). The regular consumption of alcohol is also known to alter the healthy nutritional routines which potentially affects calcium balance. Although these factors have not been researched in an adolescent population, considering what is known about the adult female, an association between alcohol, smoking and bone density is biologically plausable.

Adolescent Risk Factor Profile

The concept of risk factors presents two possibilities: (a) The risk factor denotes a predisposition to develop a specified pathology, but may or may not be a causative factor, or (b) the risk factor suggests causation or pathogenesis. Therefore, the goal of prevention is to reduce the exposure to the factor.

Over a lifetime, several factors lend their influence during specific times on the skeletal system. Figure 2 illustrates the timeline of these effects.



Figure 2. Average and low bone mass concentrations, their relationship to fracture susceptibility, and the effects of calcium intake, genetics, physical activity, and hormones over a lifetime (Adapted from Heaney, 1984).

Heaney (1984) refers to the parameters of bone health as a combination of habits and habitus. Habitus reflects the predisposition an individual may have to a disease through genetic inheritance, while habits are lifestyle choices that may create an increased risk for the development of a disease. Since prospective studies are not yet available, it is difficult to evaluate the adolescent risk factors and particularly to determine the relative importance of each potential factor (Heaney, 1984). Yet, there are strong indications that certain behaviors increase risk of poor bone metabolism and that other, healthier behaviors can make a significant contribution to the enhancement of bone matrix during the unique sequence of maturation. The impact of these adolescent behaviors may last a lifetime. The current available literature thus suggests the following factors to be of importance for adolescent bone maturation:

1. Female gender

2. Family history of osteoporosis

3. Caucasian or Oriental race

4. Slender body frame/underweight

5. Dietary intake of less than 1500 mg calcium/day.

6. Weight-bearing physical activity less than 60 minutes three times a week or strenuous training leading to amenorrhea

7. Amenorrhea or irregular menses

8. Smoking

9. Alcohol use

Summary

The stage of adolescence is a time of vast change. As the body matures into an adult form, the effects of early and adequate nutrition are imprinted on cellular systems as well as on lifestyle habits formulating patterns that will probably repeat in adult years. Heaney (1989) emphasizes that a high peak bone mass provides the most effective protection against osteoporosis at any stage in life. Knowledge of the determinants of a healthy bone mass provide a basis for adolescents to assess their current behaviors and personal risk factor profile. Since the peak bone mass formation occurs within a critical timeframe, a limited opportunity exists to maximize the individuals' genetic potential. Informed choices of healthy lifestyle behaviors may protect and enhance the skeletal system and with this knowledge teens should be able to develop a lifetime strategy that would work for varying lifestyles.

CHAPTER III

METHODS

The purpose of this study was to describe the known risk factor profile for bone health in an apparently healthy female adolescent population. Specifically, the study was designed to determine: (a) The mean dietary intake of calcium, phosphorous, calcium:phosphorous ratio, total protein, and total caloric intake; (b) physical activity level; (c) history of menstrual function; (d) family history of osteoporosis; (e) sum of triceps and calf skinfold thicknesses, from which percent fat was estimated; (f) height and weight, from which body mass index (BMI) was calculated; (g) cigarette use; and (h) alcohol use, all of which form a profile of risk factors important for optimal bone health in the adolescent.

<u>Research Design</u>

The design of this study was descriptive in nature. A three-day diet study was obtained to elicit nutritional information followed by a questionnaire that addressed personal behaviors and family history of osteoporosis. Anthropometric measures of weight, height and skinfolds were determined to assess frame size and body composition.

Subject Selection

Subjects for this study consisted of apparently healthy female adolescents in the 8th, 9th, and 10th grade classes at Malcolm Price Laboratory High School during the 1992

Spring semester ($\underline{N} = 60$). This convenience sample ranged from 13 to 16 years of age.

<u>Research Apparatus</u>

The following instruments were used to implement this study:

Three-day Dietary Intake Record

A three-day dietary intake record analyzed with "The Food Processor" computer program (ESHA Research, Inc., 1986) on an Apple IIc computer (see Appendix A).

Questionnaire

A 15-item questionnaire, divided into four sections was constructed to elicit information from each participant about their activity level, history of menstrual function, lifestyle behaviors, and family history of osteoporosis (see Appendix B). The content and format was prepared in consultation with two members of the graduate faculty, a registered dietition, and a registered nurse/health educator. The questionnaire was pilot-tested with 25 female adolescents from the Dike School District to assess it's effectiveness with females of a similar age and modified for clarity and format.

Physical activity. Section A was devised to assess the level of physical activity of each subject utilizing a table of common leisure and sports activities available to teens. Subjects were asked to indicate their participation in each of 14 different weight-bearing activities, and to indicate which months of the year they were involved in the sport or exercise, how many times per week and how many minutes a day were spent in the activity. The inventory format used was an adaptation of the Minnesota Questionnaire for the Assessment of Leisure Time Physical Activities (Taylor et al., 1978). If a subject exercised for a minimum of three times per week with 45-60 minutes of weight-bearing activity, they were classified as Moderately Active, as recommended by the guidelines of the National Osteoporosis Foundation (1991). If the activity was less than Moderately Active, the subject was classified as having a Low level of activity. If a subject participated in 45 minutes to one hour of moderate to strenuous weight-bearing activity six times a week or more, they were classified as Very Active.

<u>History of menstrual function</u>. Section B of the questionnaire dealt with the subject's history of menstrual function including age of menarche and number of menses per year. If a subject had two periods or less per year, they were classified as amenorrheic. If they had three to seven menses per year, they were classified as oligomenorrheic. Those subjects with eight to thirteen menses per year were considered eumenorrheic. This classification was used previously by Baker and Demers (1988), Lloyd et al. (1987), and Lloyd et al. (1988). Furthermore, participants were asked if they noted any irregularities in their monthly cycles while they were very active in a sports season.

Family history of osteoporosis. Section C addressed the possible family history of osteoporosis or the tendency

to have bones that break easily. A picture profile was included on the questionnaire showing the most common physical feature of adult osteoporosis: the dowager's hump.

<u>Personal behavior</u>. Section D documented the use of medicines, including vitamins, minerals, and calcium supplements. Subjects were asked to specify brand name, frequency, and duration of usage. Questions were also included on the number of cigarettes smoked per day, number of alcoholic drinks per day, as well as their pattern of dieting during the past year.

Anthropometric Data

<u>Weight</u>. A level balance-beam scale was used to measure the weight of the subject to the nearest 0.5 pound.

<u>Height</u>. A level platform with a vertical bar attached was equipped with a steel measuring tape and used to measure height. A horizontal bar attached perpendicularly to the vertical bar and was placed firmly on the subject's head, then a standing, barefoot, height was recorded to the nearest centimeter.

Body mass index (BMI). The metric values of height and weight were used to compute BMI. The ratio used to determine BMI was Quetelet's index: weight in kilograms/height in meters².

<u>Triceps and calf skinfold thicknesses</u>. The triceps and medial calf skinfold thicknesses were determined using a Lange caliper, as described by Lohman, Roche, and Martorell (1988). The sum of these two thicknesses was compared to a percent body fat estimate devised by Lohman and Slaughter (cited in McSwegin, Pemberton, Petray, & Going, 1989).

<u>Procedures</u>

Permission was sought and granted by the Human Subjects Review Board of the University of Northern Iowa to proceed with this study. Also, this research was approved by the principal of Malcolm Price Laboratory High School and permission was granted to collect data during regularly scheduled instructional time. Parental consent for non-invasive research studies are on file in each student's permanent record.

All female students in the 8th, 9th, and 10th grade physical education classes were invited by the researcher to participate in this study which was described as consisting of a three-day diet record, a questionnaire relating to personal exercise, health habits, menstrual history, and measures of height, weight, and two skinfolds. Students were informed that this nutritional study was being conducted for research purposes only and participants needed to describe as accurately as possible their typical diet. The topic of bone health was not presented at this time so that students' typical choices of food intake would not be biased. The tone of the presentation was carefully constructed to avoid any sense of evaluation or criticism of dietary practices, stressing that the purpose of the study was merely to describe the current practices of this group of teens. Students who volunteered to participate were

asked to sign an informed consent statement (see Appendix C) indicating their awareness of the nature and extent of their participation. It was stressed that participation in this study would not affect the grade received for this course and, although the diet record and questionnaire needed to be coded in order to match them for statistical analysis, the results would be confidential.

Initially, participants were asked to record three days of their dietary intake. Specific instructions were given for recording their food intake for two weekdays and one weekend day. Sample beverage glasses and serving sizes were demonstrated to increase accuracy and consistency in recording. Subjects were told that the study was to determine an average daily diet intake but were not informed of the specific parameters being studied. Participants were asked to include in their record the brand names of any vitamins, minerals, or supplements regularly ingested. Thoroughness and accuracy were stressed by the researcher and reinforced by the instructors of the class. Diet record sheets were provided as well as a completed sample sheet for their reference. Colorful pens were distributed with the record sheets as an incentive and reminder to complete the record.

Three days later, the diet studies were collected and a questionnaire was presented for their completion with the supervision of the researcher. A verbal presentation was given by the reseacher, of the symptoms characteristic of osteoporosis, including frequent incidence of hip, vertebral, and wrist fractures, as well as the most common and conspicuous expression of osteoporosis, the dowager's hump. A picture profile of the dowager's hump was included on the questionnaire.

Each participant was weighed in their clothes, without shoes, using a platform balance-beam scale that recorded to the nearest 0.5 pound and converted to kilograms. For height measurements, subjects, wearing no shoes, were instructed to stand with their feet together and their backs and heels against the upright bar of the height scale. Participants were told to "look straight ahead" so that their heads were in the Frankfort horizontal plane. There were asked to "stand up real straight" so they were standing The horizontal bar was lowered, firmly on the erect. subject's head. With the observer's eye-level in the same plane as the horizontal measuring bar, height was recorded to the nearest centimeter and converted to meters.

As described by Lohman et al. (1988), the site of the triceps skinfold was determined by measuring, on the right side, with a metal measuring tape, the midline point of the distance between the tip of the shoulder and the tip of the elbow, when the elbow was flexed at a 90° angle. The midline was marked posteriorly, directly over the triceps muscle. The site of the medial calf skinfold was determined at the point of greatest girth of the calf muscle.

The procedure used to measure skinfolds involved grasping the skin and underlying fat away from the muscle in a vertical fold using the thumb and forefinger one centimeter above the marked measuring site. A Lange caliper was used with tension applied slowly. A reading was taken approximately two seconds after full tension was reached. The vertical fold was maintained until after the thicknesses were measured. Three determinations were made at each site, alternately, and recorded to the nearest millimeter. The average thickness at each site was calculated and the two scores were added to obtain the sum of the skinfolds.

Once the data were gathered and evaluated, a presentation of the results of the study was given by the researcher to all participants. A profile of risk factors was made available to each participant describing the group as a whole, and each participant individually. In addition, a summary of the average daily consumption of 26 nutrients from "The Food Processor" computer program (ESHA Research, Inc., 1986) based on their three-day record was offered to each participant.

Data Description

The data collected in this study consisted of the following measurements:

1. Weight in kilograms

2. Height in meters

3. Body Mass Index (Quetelet's Index of weight in kilograms/height in meters²)

4. Sum of the triceps and medial calf skinfold thicknesses

5. Average daily intake of calcium, phosphorous, calcium:phosphorous ratio, total protein and total calories

A physical activity level assessment of Low,
Moderate, or Very Active

7. A menstrual history of amenorrhea, oligomenorrhea or eumenorrhea

8. Incidence of cigarette-smoking and alcohol use

9. Positive or negative response for known family history of osteoporosis

Data Analysis

The three-day diet records were coded, entered and evaluated by "The Food Processor" computer program on an Apple IIc computer for the average daily intake of calcium, phosphorus, total protein, and total calories (ESHA Research, Inc., 1986). All data were coded and entered on the VAX410 computer system at the University of Northern Iowa. <u>The Statistical Package for the Social Sciences</u> (SPSS) was employed for the analysis of the data (Nie, Hull, Jenkins, Steinbrenner, & Bent, 1975). Descriptive statistics were used to describe the mean and standard deviation of anthropometric data from the sample population. Body mass index was calculated for each subject using the ratio of weight (kg) to height (m²). Frequencies within each risk factor category were described for the sample as a

whole, then were converted to percentages to create a descriptive profile of the sample. A measure of small, slender frame was determined by comparing the BMI to the nomogram devised by Thomas, McKay, and Cutlip (1976) for determination of an overweight, desirable weight, or underweight classification (see Appendix D). Additionally, a Pearson product-moment coefficient of correlation was calculated for all variables.

CHAPTER IV

RESULTS & DISCUSSION

The purpose of this study was to describe the known risk factor profile for bone health in an apparently healthy female adolescent population. A profile of nine factors for bone health was established from a review of current literature. The following variables were considered in the assessment of these risk factors: (a) race, (b) family history of osteoporosis, (c) dietary calcium intake, (d) dietary phophorous intake, (e) calcium to phosphorous ratio, (f) total calories, (g) total protein, (h) physical activity level, (i) menstrual status, (j) irregularity of menses with sports activities, (k) height, (l) weight, (m) sum of triceps and calf-skinfold thickness, (n) use of cigarettes, and (o) use of alcohol. This chapter presents data and discussion for each risk factor assessed, then describes the sample population as a whole.

Demographic Description

This study is based on a volunteer sampling of adolescent females attending Malcolm Price Laboratory School. The purpose and requirements of this project were introduced to 78 students during 8th, 9th, and 10th grade physical education classes. A total of 60 participants completed all three phases of the study: a three-day diet record, questionnaire, and anthropometric measurements resulting in a response rate of 77%. Table 1 specifies the ages and race of the sample.

Table 1

Age and Race of Participants

Variable	Subjects	Percent
Age (years)		
13	9	15
14	19	32
15	26	43
16	6	10
Race		
Caucasian	55	92
Asian	1	2
Mixed	2	3
Black	2	3

<u>Note.</u> $\underline{N} = 60$.

As noted in Table 1, this sample was predominantely Caucasian (92%), with 2% of Asian ancestry, 3% of mixed race, and 3% Black. The mean age of the sample was 14.5 years with a standard deviation of .9 and a range of 13 to 16 years. All subjects appeared to be healthy at the time of testing; further documentation of individual medical histories was not part of this study.

Dietary Intake of Nutrients

Initially, participants were asked to record three days (two weekdays and one weekend day) of their food intake on the record sheets provided. Sixty completed records were returned, coded, and evaluated by "The Food Processor" software program for nutritional components (ESHA Research, Inc., 1986).

Table 2

Dietary Intake of Calories, Calcium, Phosphorous, and Protein

Variable	М	SD	Range
Total calories	1674.7	472.2	823-3455
Calcium (mg)	981.5	398.2	197-2034
Phosphorous (mg)	1176.2	358.1	582-2135
Total protein (gm)	60.7	15.4	32-105

<u>Note</u>. $\underline{N} = 60$.

Average daily intake was determined from the total of three days intake for calories, calcium, phosphorous, and protein. With the recommended daily allowance for 11- to 14-year-olds being 1200-3000 calories/day, and 1500-3000 calories/day for 15- to 18-year-olds (Select Committee on Nutrition and Human Needs, 1977), the nutritional data obtained suggest that most of the adolescents consumed the recommended amount of calories. The range of caloric intake (823-3455 calories) was quite broad. Five subjects ingested less than 1200 calories/day and one subject consumed an average over 3000 calories/day. Figure 3 illustrates further the pattern of caloric intake for this sample. In this figure, individual totals were rounded to the nearest hundred.



Figure 3. Average caloric intake of participants, ages 13 to 16 years compared to recommended caloric intake $(\underline{N} = 60)$.
Of all responders, 15 (25%) reported a variety of weight reduction diet experiences during the past year. The length of the diets ranged from one week to seven months, with the average length being two months. Weight loss ranged from zero to 20 pounds while the average weight change was a loss of seven pounds. Compared to the definition of Thomas et al. (1976) using the body mass index nomogram, five of those dieting were "overweight," one was "underweight," and nine were currently of "desirable weight for height" (see Appendix D).

As indicated in Table 1, the mean calcium intake was only 981.5 mg, Figure 4 further illustrates the range of calcium intake for this sample.



Figure 4. Mean calcium intakes as percentage of NIH (1984) recommended intake.

These amounts are far below the National Institute of Health's (NIH) recommended intake of 1500 mg/day (NIH Consensus Conference on Osteoporosis, 1984), and also below the Recommended Dietary Allowance (RDA) of 1200 mg/day as established by the Food and Nutrition Board of the National Research Council (1989). These findings continue the documentation that calcium intake of adolescent females is notoriously low (Abraham et al., 1977; Carroll et al., 1983; Heaney, 1989; Matkovic et al., 1986). A total of 55 (92%) of the sample averaged less than 1500 mg of calcium per day. Of these, nine (15%) averaged 75%-100% of the recommended amount, 25 (42%) consumed 50-74%, 16 (27%) ingested 25-49%, and three (5%) took in less than 25% of the recommended calcium requirement.

Ideally, the calcium to phosphorus ratio (Ca:P) is 1:1, and in this sample the average Ca:P ratio was 1:1.3. While 52 (87%) met the standard of 1:1, 7 (12%) exhibited a ratio closer to 1:2 while one was a more atypical 1:3. Further notation of that diet record revealed that the calcium consumed was an unusally low 197 mg, while the phosphorous was a more moderate level of 597 mg, creating the 1:3 ratio. Total caloric intake for this subject was 1161 calories. Overall, despite the apparent balance between calcium and phosphorus in the majority of the subjects, the absolute values for both of these minerals was markedly below recommended levels.

Another influence on calcium utilization is the amount of protein present in the diet. Due to the increase in obligatory urinary calcium loss with an elevated protein intake, an inflated calcium requirement is subsequently necessary to compensate for these losses. The National Research Council (1989), has recommended that females 11-14 years consume 46 gms of protein/day and females 15-18 years consume 44 gms of protein/day. In this sample, 49 (82%) averaged a protein intake greater than their RDA and 9 (15%) averaged less than their RDA. Protein intake ranged from 32 to 105 grams with a mean of 60.7 and a standard deviation of 15.4. This represents a protein intake that is markedly higher than the amount considered necessary for tissue function and repair. Therefore, the amount of calcium that is bioavailable may be even lower than the noted intake.

Looking further at specific subjects with high protein intakes, the highest report of 105 grams of protein occurred with the largest intake of 3455 calories. The accompanying calcium intake of 2034 grams provides some cushion for the suspected increase in urinary loss in this participant. Other reports included: 87 grams of protein with 804 grams of calcium, 83 grams of protein with 1383 grams of calcium, and 82 grams of protein with 1061 grams of calcium. These higher, almost double the recommended amounts of protein reportedly increase the calcium requirement by 200-300 mg/day (Heaney & Barger-Lux, 1988), which may exacerbate an already insufficient supply.

Participants were asked to list vitamins and supplements on the diet record and again on the questionnaire. Of 60 participants, 16 (27%) reported the use of a daily vitamin supplement and five indicated occassional usage of a vitamin supplement. Seven responders recorded the brand name of their vitamin and none of these were found to contain the mineral calcium. Only one subject reported the use of a calcium supplement, Tums®, which, taken daily, supplied an additional 200 mg of elemental calcium.

The methodology of a three-day diet study yields a record of current food intake that may or may not be reflective a long-term habitual intake. Some researchers consider this kind of data too "soft" and unreliable for long-term assessment (Wylie-Rosett, Wassertheil-Smoller, & Elmer, 1990). Yet, the impact of calcium on bone health is a variable of such importance that an attempt to document a pattern of its consumption is imperative. The documentation process alone is often an enlightening experience for the participant.

It was the subjective observation of the researcher that most participants made a serious and thorough effort to record food intake accurately. If records were unclear or unfinished, participants were queried further in order to complete the record. A potential source of bias were the 18 students (23%) who were introduced to the study but failed

to return the diet record. Their exclusion may distort the total picture of dietary patterns for this population.

Physical Activity

An estimate of average hours per week of physical activity was obtained from a survey of weight-bearing sports and leisure activities (see Appendix B). According to the National Osteoporosis Foundation (1991), less than three hours per week of exercise is considered a Low activity level. Three to six hours per week is a Moderate level and greater than six hours indicates a Very Active level. Using this criterion, Figure 5 illustrates the activity pattern for this sample.



Hours of Weight-Bearing Activity/Week

Figure 5. Average hours per week of weight-bearing activity and incidence of irregular menses at each level.

The mean number of hours spent in weight-bearing activity per week was 6.8 hours with a standard deviation of 2.9 and a range of 2.5-17 hours. Overall, these results suggest this sample is a very active group. The activities reported were primarily school-sponsored sports with limited seasons during the school year as well as daily required physical education classes. None of the subjects reported involvement in a long-term training program. This activity level reflects seasonal activity, rather than a long-term program and is a very general measure of activity. Variation in type and intensity of exertion was not part of this documentation.

The high rate of Very Active patterns (58%) and Moderate activity (40%) provides the gravitational stimulation required for bone growth. Only one subject reported involvement in exercise or activity less than three hours a week. These findings indicate that this sample may be benefitting from the positive influence of weight-bearing activity on bone health.

Menstrual Status

Interestingly, 18 (30%) of the subjects reported their menstrual cycles were irregular or more irregular than usual during participation in a sports season. As noted in Figure 5, six (10%) in the moderately active group experienced irregular menses, while 12 (20%) of the very active group noted disturbances in their cycles. This phenomenon may or may not be related to activity as there are many other

variables in the life of a teenager that may be contributing to this variance including stress, dieting patterns, and youthful hormonal fluctuations.

Section B of the questionnaire provided information on the participant's age of menarche and the number of menses during the past year. Amenorrhea is defined as less than two menses per year, oligomenorrhea describes 3-7 menses per year and eumenorrhea is regular cycling 8-13 times a year. In this sample, the average age of menarche was 12 years and the mean number of menses was 8.9 per year with a standard deviation of 4.0 and a range of 0-16 per year. Figure 6 describes the pattern of menses for this sample.



<u>Figure 6</u>. Menses per year and reported irregular menses with sports activites.

This sample of 60 participants represents an early adolescent female population. Nine (15%) of the sample retained primary amenorrhea, while 13 (22%) were experiencing their first year of menses. Repeated in Figure 6 are the findings of irregular menses with sports activities. Within the eumenorrheic group, 12 noted irregularities while 4 of the oliogomenorrheic group and 2 of the amenorrheic group reported they had no menses during an active sports season. These findings are consistent with other reports of menstrual dysfunction during vigorous exercise (Bullen et al., 1985; Dale et al., 1979; Feicht et al., 1978; Schwartz et al., 1981; Shangold et al., 1979; Speroff, 1982). It is not within the scope of this study to suggest a causal relationship between activity and menses, nevertheless, the interactive nature of hormones and activity warrants documentation of the occurrence of fluctuations in menstrual patterns and examination of the possible negative effects of a low estrogen environment on peak bone mass formation.

Body Frame Size

An indication of body frame size was determined by the calculation of body mass index (BMI), using the formula:

BMI = weight(kg)/height²(m)

This index was then compared to the nomogram of Thomas et al. (1976) describing desirable weight for height, with a value < 19 classified as "underweight," indices 19-24 were considered "desirable weight" for height and a value > 24 was designated as "overweight" (see Appendix D). The mean BMI for this sample was 21.9, with a standard deviation of 3.7. The indices ranged from 16 to 34 as shown in Table 3.

Table 3

Physical Characteristics of Subjects

Parameter	м	SD	Range
Height (inches)	64.6	21	60-69
Weight (pounds)	130.2	23.5	93-214
Body mass index	21.9	3.7	16-34
Sum of skinfolds (mm)	33.5	10.4	17-58

<u>Note</u>. <u>N</u> = 60.

Additionally, skinfold measurements from the triceps and medial calf sites were summed to estimate body composition. The mean sum of skinfolds was 33.5 mm with a standard deviation of 10.4 mm. Sums ranged from 17-58 mm. These sums were converted to percent body fat estimates as determined by Lohman and Slaughter, (cited in McSwegin et al., 1989). This sample ranged from 15% to 41% body fat. Thirty-four (57%) of the total sample were in the optimal range, 18 (30%) were in the moderately high range, 2 (3%) were in the high range, while 6 (10%) were calculated as very high. Table 4 summarizes the calculations of BMI, sum of skinfolds, and body fat percentage estimates.

Table 4

Body Mass Index, Sum of Skinfolds, and Body Fat Percentage Estimates

Variable	Underweight	Average	Overweight		
Body mass index	<19	19-24	>24		
	9 (15%)	40 (67%)	11 (18%)		
Sum of skinfolds	<16	16-35	>35		
	0	34 (57%)	26 (43%)		
Body fat percentage	<15	16-27	>27		
	0	34 (57%)	26 (43%)		

<u>Note</u>. $\underline{N} = 60$.

The sum of skinfolds appear negatively skewed in comparison to the BMI. This may be due to the choice of a medial calf site for measurement. In an active population with a very muscular calf area, measurement of skinfold at the medial site may yield a falsely high score. A Pearson product-moment correlation was employed to assess the relationship between BMI and sum of skinfolds and was found to be significantly correlated ($\underline{r} = .86$, $\underline{p} = .01$). Further correlations emerged showing the triceps skinfold alone to be significantly correlated with the body mass index ($\underline{r} = .83$, $\underline{p} = .01$) as well as the calf alone ($\underline{r} = .83$, $\underline{p} = .01$).

Since an underweight status with a low weight for height is a well-established risk factor for low bone mass development, this factor becomes highlighted with the addition of amenorrhea or irregular menses and its potential hypoestrogenic effects. As illustrated in the dramatic example of anorexia nervosa, endocrine changes can have a major effect on bone metabolism. At the same time, caloric intake is of particular note in individuals with a low weight for height.

A closer look at the range of BMI in this group reveals nine (15%) to be considered "underweight." Although their corresponding body fat percentages were not considered below desirable range, four were amenorrheic (three 13-year-olds and one 15-year-old) and one 14-year-old was oligomenorrheic. All but the 15-year-old was estimated to be Very Active. The measure of BMI and menses correlated with moderate significance ($\underline{r} = .31$, $\underline{p} = .05$). Eight of this group also had a low calcium intake although only one had a low calorie intake.

The measure of BMI reoccurs as a positive predictor of bone mineral content in several studies; with anorexics and their control subjects (Bachrach et al., 1990), and in young adult females (Pocock et al., 1987). This suggests that the BMI could potentially be used as a simple, easily obtained measure of body size and body composition. This would be valuable as part of a risk factor screening process.

Family History of Osteoporosis

Following a verbal explanation by the researcher of symtoms characteristic of osteoporosis, including frequent incidence of hip, vertebral, and wrist fractures, as well as a description of a dowager's hump, participants were asked to respond to Section D of the questionnaire. There were six positive responses for a family history of osteoporosis. One of these positive responses noted that "almost everyone" had osteoporosis. An additional nine subjects (15%) made "possibly positive" responses and several of these commented that they knew of hip and wrist fractures experienced by their grandparents, but were not sure if osteoporosis was The remaining 45 (75%) had no known family history present. of osteoporosis. All positive and possibly positive responses were from subjects of Caucasian descent.

Cigarette Smoking and Alcohol Consumption

Section C of the questionnaire addressed the use of cigarettes and alcohol, noting the frequency of use. One subject reported the regular use of cigarettes, but declined to state the frequency of use. There were four reported users of alcohol with frequencies of one drink per week,

0-2 drinks per week, 0-6 drinks per week, and 0-10 drinks per week. This low reported use of cigarette and alcohol products represents two protective factors in the total picture of bone health for the study population.

Risk Factor Profile

The incidence of each of the nine reported risk factors is reflected in Figure 7.



Risk Factors

<u>Figure 7</u>. Frequency of risk factors in the sample population.

The risk factor of female gender encompassed the entire sample and 93% of participants were Caucasian or Oriental. Supplementing these dominant factors, is the low calcium intake described by 92% of the sample. These three factors comprise the most common combination of risk factors in this population.

A family history of osteoporosis was noted by 9 (15%) of the participants while a small or underweight body frame was calculated for 9 (15%). Another 9 (15%) reported < 2 menses per year, and only 4 (7%) described using alcohol. One subject (2%) was a smoker. Furthermore, in this group, only one participant was estimated to have a low level of activity.

Since a risk factor profile is additive, the cumulative number of risk factors each subject experiences is individually important. The occurrence of multiple risk factors are reflected in Table 5.

Table 5

Occurrence of Multiple Risk Factors in Sample Population

Risk Factors	Subjects Percent		
1	0	0	
2	2	3	
3	37	62	
4	16	27	
5	5	8	
Total	60	100	

Again, the most frequent amalgam of risk factors is female gender, Caucasian race, and low calcium intake, which occured in 85% of this sample. Additional multiple risk factors occurred most frequently in the youngest group of 13-year-olds who reported 6 (67%) of the 9 low number of menses or delayed menarche. Three of these also had a low BMI which is probably most reflective of the young adolescent stage of these females. These subjects' profile may change significantly in the next year.

Two subjects, ages 14 and 15, accumulated five risk factors; female gender, Caucasian race, low calcium, low BMI, and low number of menses. This particular combination of factors, at this age, represents a bone health profile that may be missing a prime time of peak bone mass formation. Other subjects with risk factors that occurred in multiples presented a wide variety of combinations.

Pearson product-moment correlations were calculated for all variables. Strong associations were found among anthropometric measures, as may be expected. Menses was moderately correlated with age, weight, BMI, and sum of skinfolds. Of note, also, is a trend within this sample to decrease activity with increasing age. Further correlations of the proposed risk factors are shown in Table 6.

Table 6

Intercorrelations of Risk Factors

		1	2	3	4	5	6	7	8
1.	Age (years)								
2.	Height (inches)	.17							
3.	Weight (pounds)	.24	.38**						
4.	Body mass index	.16	.02	.91**					
5.	Skinfold sum (mm)	.24	.05	.81**	.86**	ł		·	
6.	Calcium · (mg)	01	.41**	.16	.01	.00			
7.	Activity level (hours/week)	44**-	 05 ·	04 -	01	11	.22		
8.	Menstrual history (menses/year)	.33**-)	01	.29*	.31*	.27*	.02	07	
9.	Irregular menses with sports	.01	.16	.05	.03	03	02	.07	.05

<u>Note.</u> $\underline{N} = 60$.

- ****** = significant at the .01 level.
- * = significant at the .05 level.

Summary

This research investigated the incidence of bone health risk factors among an adolescent female population. Variables affecting each of nine risk factors were assessed revealing three of the nine factors strikingly prevalent. Female gender, Caucasian race, and low calcium intake dominated the risk factor profile. On the positive side, the moderate to very active lifestyle, minimal smoking, and low alcohol usage presumably provide protective benefits for bone health. The factor of small body frame/underweight occurred in each age group, while < 2 menses per year, exhibited by 13-, 14-, and 15-year-olds, were probably associated with delayed menarche. Individual combinations of multiple risk profiles were guite diverse.

CHAPTER V

CONCLUSIONS AND RECOMMENDATIONS

Conclusions

The purpose of this study was to describe the nature and extent of known risk factors for bone health in an apparently healthy female adolescent population. From the findings of this study, it can be concluded that substantial areas of risk as well as factors that are potentially protective exist within this sample. Specifically, these findings lead to the following conclusions:

1. The calcium intake of the sample was substantially lower than recommended for this adolescent age group.

2. For 82% of the sample, the risk of a low calcium intake was aggravated by a high protein intake.

3. The average level of activity was quite high, albeit characteristic for this age group, necessitating an adequate calcium supply to support the kinetics of bone remodeling.

4. The sample represented a very active adolescent population with 98% participation in regular weight-bearing activities throughout the year which may have a positive influence on bone health.

5. The reported infrequent use of cigarettes and alcohol demonstrated another protective factor for bone density in this sample.

6. Thirty percent of adolescent females in this group reported irregularity of menstrual cycles during active sports seasons.

7. Six (10%) of responders knew of a positive family history of osteoporosis. This number reflects some awareness of this disease process which may increase interest in lowering individual risk factors.

These results and subsequent conclusions refer only to the population studied and may not be generalizable to all adolescent females.

Recommendations

The assessment of risk factors is a popular and proven method of stimulating awareness, increasing knowledge, and presenting choices for risk modification (Green & Krueter, 1991). Based on the findings of this study, the following actions are recommended:

1. Adolescent females need to become more aware of the essential role of calcium in the formation of a peak bone mass. Daily recommended amounts and sources of the calcium nutriture should be emphasized, as well as the corollary effect of a high protein diet. Effective sources of calcium supplementation should be presented as an option for those unable to consume enough calcium through diet alone.

2. Teenage girls should be cognizant of all the risk factors for optimal bone health, and be guided in their assessment of changeable and non-changeable risks in order to increase their peak bone mass and subsequently delay the sequence of age-related bone loss.

3. Information concerning bone health risk factors needs to be provided also to parents, health educators, and

coaches of female athletes to further promote awareness of changeable factors and reinforce lifestyles conducive to a high peak bone mass.

4. The female adolescent population should be notified of the potential impact of both minimal and maximal levels of physical activity on hormonal balance.

The potential benefit of reducing risk to bone health early in life while opportunities are available to significantly increase bone density is substantially important to both the individual and to a society generally at risk for osteoporosis later in life. Based on the findings within this study, the following recommendations for further study are presented:

1. Further studies on bone mass concentrations utilizing an adolescent population are critically needed to assess the relative contributions of known risk factors separately, and in interactive combinations.

2. It may be instructive to repeat this study using a sample from a more diverse population, to describe their risk profile pattern. This would be particularly useful for planning educational objectives.

3. Repeating this study utilizing a college-age female population involved in more vigorous sports would provide data to further assess the incidence of menstrual dysfunction during vigorous exercise. 4. In order to enhance the reliability of this profile, the Low, Moderate, and Very Active classifications of physical activity should be validated with $\dot{v}O_{2max}$ studies.

5. Ideally, a measurement of bone mineral content is necessary to further explore and substantiate the relative contributions of known bone health risk factors.

6. The development of a risk factor profile chart valid for use with adolescents would facilitate the screening process and assist in the instruction of teens concerning their individual bone health profile.

7. Further studies with coaches, teachers, and health educators to determine their knowledge and understanding of adolescent bone health risk factors would assist in the development of curriculum and continuing education programs for these professionals.

REFERENCES

- Abraham, S., Carroll, M. D., Dresser, C. M., & Johnson, C. L. (1977). <u>Dietary intake findings, United States 1971-1974</u> (HEW Publication No. HRA 77-1647). Hyattsville, MD: National Center for Health Statistics.
- Albanese, A. A. (1983). Calcium nutrition throughout the life cycle. <u>Bibliography of Nutrition and Dieta</u>, <u>33</u>, 80-99.
- Albright, F., Smith, P. H., & Richardson, A. M. (1941). Postmenopausal osteoporosis. <u>Journal of the American</u> <u>Medical Association</u>, <u>116</u>, 2465-2474.
- Aloia, J. F., Cohn, S. H., Tabu, T., Abesamis, C., Kalici, N., & Ellis, K. (1978). Skeletal mass and body composition in marathon runners. <u>Metabolism</u>, <u>27</u>(12), 1793-1796.
- Amschler, D. H. (1985). Calcium intake: A lifelong proposition. <u>Journal of School Health</u>, <u>55</u>(9), 360-363.
- Awbrey, B. J., Jacobson, P. C., Grubb, S. A., McCartney, W. H., Vincent, L. M., & Talmage, R. V. (1984). Bone density in women: A modified procedure for measurement of distal radial density. <u>Journal of Orthopedic Research</u>, <u>2</u>(4), 314-321.
- Ayers, J. W. T., Gidwani, G. P., Schmidt, I. M. V., & Gross, M. (1984). Osteopenia in hypoestrogenic young women with anorexia nervosa. <u>Fertility and Sterility</u>, <u>41</u>(2), 224-228.
- Bachrach, L. K., Guido, D., Katzman, D., Litt, I. F., & Marcus, R. (1990). Decreased bone density in adolescent girls with anorexia nervosa. <u>Pediatrics</u>, <u>86</u>(3), 440-447.
- Baker, E. R. (1981). Menstrual dysfunction and hormonal status in athletic women: A review. <u>Fertility & Sterility</u>, <u>36</u>, 691-696.
- Baker, E., & Demers, L. (1988). Menstrual status in female athletes: Correlation with reproductive hormones and bone density. <u>Obstetrics and Gynecology</u>, <u>72</u>(5), 683-687.
- Barrett-Connor, E. L. (1989). The risks and benefits of long-term estrogen replacement therapy. <u>Public Health</u> <u>Reports</u>, <u>9-10</u>(Suppl.), 62-65.
- Bassett, C. A., & Becker, R. O. (1962). Generation of electric potentials by bone in response to mechanical stress. <u>Science</u>, <u>137</u>, 1063-1064.

- Biller, B. M. K., Saxe, V., Herzog, D. B., Rosenthal, D. I., Holzman, S., & Klibanski, A. (1989). Mechanisms of osteoporosis in adult and adolescent women with anorexia nervosa. <u>Journal of Clinical Endocrinology and Metabolism</u>, <u>68</u>(3), 548-554.
- Brewer, V., Meyer, B. M., Keele, M. S., Upton, S. J., & Hagan, R. D. (1983). Role of exercise in prevention of involutional bone loss. <u>Medicine and Science in Sports and Exercise</u>, <u>15</u>(6), 445-449.
- Buchanan, J. R., Myers, C., Lloyd, T., & Greer, R. B. (1988). Early vertebral trabecular bone loss in normal premenopausal women. <u>Journal of Bone and Mineral Research</u>, <u>3</u>(5), 583-587.
- Buchanan, J. R., Myers, C., Lloyd, T., Leuenberger, P., & Demers, L. M. (1988). Determinants of peak trabecular bone density in women: The role of androgens, estrogen, and exercise. <u>Journal of Bone and Mineral Research</u>, <u>3</u>(6), 673-680.
- Bullen, B. A., Skrinar, G. S., Beitins, I. Z., Mering, G. von, Turnbull, B. A., & McArthur, J. W. (1985). Induction of menstrual disorders by strenuous exercise in untrained women. <u>New England Journal of Medicine</u>, <u>312</u>(21), 1349-1353.
- Bunt, J. C., Going, S. B., Lohman, T. G., Heinrich, C. H., Perry, C. D., & Pamenter, R. W. (1990). Variation in bone mineral content and estimated body fat in young adult females. <u>Medicine and Science in Sports and Exercise</u>, <u>22</u>(5), 564-569.
- Burckhardt, P., & Michel, C. H. (1989). The peak bone mass concept. <u>Clinical Rheumatology</u>, <u>8</u>(2), 16-21.
- Calabrese, L. H., Kirkendall, D. T., Floyd, M., Rapoport, S., Williams, G. W., Weiker, G. G., & Bergfeld, J. A. (1983). Menstrual abnormalitites, nutritional patterns and body composition in female classical ballet dancers. <u>Physicians</u> <u>and Sports Medicine</u>, <u>11</u>(2), 76-98.

The Calcium Craze. (1986, January, 26). Newsweek, pp. 48-52.

- Cann, C. E., Genant, H. K., Kolb, F. O., & Ettinger, B. (1985). Quantitative computed tomography for prediction of vertebral fracture risk. <u>Bone</u>, <u>6</u>, 1-7.
- Cann, C. E., Martin, M. C., Genant, H. K., & Jaffe, R. B. (1984). Decreased spinal mineral content in amenorrheic women. <u>Journal of the American Medical Association</u>, <u>251</u>, 626-629.

- Carroll, M. D., Abraham, S., & Dresser, C. M. (1983). <u>Dietary intake source data: United States 1976-1980 National</u> <u>Center for Health Statistics, vital and health statistics</u> <u>series 2</u> (DHHA Publication No. PHS 83-1681). Washington, DC: U. S. Government Printing Office.
- Casper, R. C., & Offer D. (1990). Weight and dieting concerns in adolescents, fashion or symptom? <u>Pediatrics</u>, <u>86(3)</u>, 384-390.
- Chesnut, C. H. (1989). Is osteoporosis a pediatric disease? Peak bone mass attainment in the adolescent female. <u>Public</u> <u>Health Reports</u>, <u>9-10</u>(Suppl.), 50-54.
- Cohn, S. H., Abesamis, C., Yasumura, S., Aloia, J. F., Zanzi, I., & Ellis, K. J. (1977). Comparative skeletal mass and radial bone mineral content in black and white women. <u>Metabolism</u>, <u>26</u>, 171-178.
- Committee on Nutrition. (1978). Calcium requirements in infancy and childhood. <u>Pediatrics</u>, <u>62</u>, 826-834.
- Cook, S. D., Harding, A. F., Thomas, K. A., Morgan E. L., Schnurpfeil, K. M., & Haddad, R. J. (1987). Traebecular bone density and menstrual function in women runners. <u>The</u> <u>American Journal of Sports Medicine</u>, <u>15</u>(5), 503-507.
- Cooper, A. (1824). <u>Treatise on dislocation and fractures of</u> <u>the joints</u> (4th ed.). London: Longman.
- Crowther, J. H., Post, G., & Zaynor, L. (1985). The prevalence of bulimia and binge eating in adolescent girls. <u>International Journal of Eating Disorders</u>, <u>4</u>, 29-42.
- Culliton, B. J. (1987). Osteoporosis reexamined: Complexity of bone biology is a challenge. <u>Science</u>, <u>235</u>, 833-834.
- Dale, E., Gerlach, D. H., & Wilhite, A. L. (1979). Menstrual dysfunction in distance runners. <u>Obstetrics and Gynecology</u>, <u>54</u>(1), 47-53.
- Dalsky, G. P. (1987). Exercise: Its effect on bone mineral content. <u>Clinical Obstetrics and Gynecology</u>, <u>30(4)</u>, 820-832.
- Dalsky, G. P. (1990). Effect of exercise on bone: Permissive influence of estrogen and calcium. <u>Medicine and Science in</u> <u>Sports and Exercise</u>, <u>22</u>(3), 281-285.
- Davee, A. M., Rosen, C. J., & Adler, R. A. (1990). Exercise patterns and trabecular bone density in college women. <u>Journal of Bone and Mineral Research</u>, 5(3), 245-250.

- Dequeker, J., Geusens, L., Wielandts, J., Nijs, J., & Verstraeten, A. (1984). Lumbar BMC skeletal size nomogram. In C. Christiansen, C. D. Arnaud, B. E. C. Nordin, A. M. Parfitt, W. A. Peck, & B. L. Riggs (Eds.), <u>Osteoporosis:</u> <u>Proceedings of the Copenhagen International Symposium on</u> <u>Osteoporosis</u> (pp. 341-344). Copenhagen, Denmark: Department of Clinical Chemistry, Glostrup Hospital.
- Dequeker, J., Nijs, J., Verstraeten, A., Geusens, P., & Gevers, G. (1987). Genetic determinants of bone mineral content at the spine and radius: A twin study. <u>Bone</u>, <u>8</u>(4), 207-209.
- Drinkwater, B. L. (1990). Physical exercise and bone health. Journal of American Medical Women's Association, 45(3), 91-97.
- Drinkwater, B. L., Bruemner, B., & Chesnut, C. H. (1987). Menstrual history and bone demineral density in female athletes. <u>Medicine and Science in Sports and Exercise</u>, <u>19</u>, (Abstract No. S12).
- Drinkwater, B. L., Bruemner, M. S., & Chesnut, C. H. (1990). Menstrual history as a determinant of current bone density in young athletes. <u>Journal of the American Medical</u> <u>Association</u>, <u>263</u>, 545-548.
- Drinkwater, B. L., Nilson, K., Chesnut, C. H., Bremner, W. J., Shainholtz, S., & Southworth, M. B. (1984). Bone mineral content of amenorrheic and eumenorrheic athletes. <u>The New</u> <u>England Journal of Medicine</u>, <u>311</u>(5), 277-281.
- Drinkwater, B. L., Nilson, K., Ott, S., & Chesnut, C. H. (1986). Bone mineral density after resumption of menses in amenorrheic athletes. <u>Journal of the American Medical</u> <u>Association</u>, <u>256</u>(3), 380-382.
- Dwyer, J. T., Feldman, J. J., & Mayer, J. (1967). Adolescent dieters: Who are they? Physical characteristics, attitudes and dieting practices of adolescent girls. <u>American Journal</u> of Clinical Nutrition, 20, 1045-1056.
- ESHA Research, Inc. (1986). <u>The Food Processor</u> [Computer program, Version 2.0]. Salem, OR: Author.
- Evans, R. A., Marel, G. M., Lancaster, E. K., Kos, S., Evan, M., & Wong, S. Y. P. (1988). Bone mass is low in relatives of osteoporotic patients. <u>Annals of Internal Medicine</u>, 870-873.
- Evers, S. E., Orchard, J. W., & Haddad, R. G. (1985). Bone density in postmenopausal North American Indian and Caucasian females. <u>Human Biology</u>, <u>57</u>(4), 719-726.

- Farndon, D. F. (1985). <u>Osteoporosis</u>. New York: Macmillan Publishing Company.
- Feicht, C. B., Johnson, T. S., Martin, B. J., Sparkes, K. E., & Wagner, W. W. (1978). Secondary amenorrhea in athletes. <u>Lancet</u>, 2(8100), 1145-1146.
- Finn, S. (1987). Osteoporsis: A nutritionist's approach. <u>Health Values</u>, <u>11(4)</u>, 20-23.
- Firooznia, H., Golimbu, C., Rafii, M., Schwartz, M. S., & Alterman, E. R. (1984). Quantitative commuted tomography assessment of spinal trabecular bone in osteoporotic women with and without vertebral fractures. <u>Journal of Computed</u> <u>Tomography</u>, <u>8</u>, 99-103.
- Fisher, E. C., Nelson, M. E., Frontera, W. R., Turksoy, R. N., & Evans, W. J. (1986). Bone mineral content and levels of gonadotropins and estrogens in amenorrheic running women. <u>Journal of Clinical Endocrinology and Metabolism</u>, <u>62</u>(6), 1232-1236.
- Frisch, R. E., & McArthur, J. W. (1974). Menstrual cycles: Fatness as a determination of minimum weight for height necessary for their maintenance or onset. <u>Science</u>, <u>185</u>, 949-951.
- Frost, H. M. (1982). Review article: Mechanical determinants of bone remodeling. <u>Metabolism & Bone Diseases and Related</u> <u>Research</u>, <u>4</u>, 217-229.
- Garn, S. M. (1963). Human biology and research in body composition. <u>Annals of the New York Academy of Science</u>, <u>110</u>, 429-437.
- Garn, S. M. (1970). <u>The earlier gain and later loss of</u> <u>cortical bone</u>. Springfield, IL: Charles C. Thomas.
- Garn, S. M., Rohman, C. G., & Wagner, B. (1967). Bone loss as a general phenonenon in man. <u>Federal Proceedings</u>, <u>26</u>, 1729-1736.
- Garn, S. M., & Wagner, B. (1969). The adolescent growth of the skeletal mass and its implications to mineral requirements. In F. P. Heald (Ed.), <u>Adolescent nutrition</u> <u>and growth</u> (pp.139-161). New York: Appelton-Century Crofts.
- Genant, H. K., Cann, C. E., Puszzi-Mucelli, R. S., & Kanter, A. S. (1983). Vertebral mineral determination and quantitative computed tomography: Clinical feasibility and normative data. <u>Journal of Computed Assisted Tomography</u>, <u>7</u>, 554-559.

- Gilsanz, V., Gibbens, D. T., Carlson, M., Boechat, M. I., Cann, C. E., & Schulz, E. E. (1988). Peak trabecular vertebral density: A comparison of adolescent and adult females. <u>Calcified Tissue International</u>, <u>43</u>, 260-262.
- Gilsanz, V., Gibbens, D. T., Roe, T. F., Carlson, M., Senac, M. O., Boechat, M. I., Huang, H. K., Schulz, E. E., Libanti, C. R., & Cann, C. C. (1988). Vertebral bone density in children: Effect of puberty. <u>Radiology</u>, <u>166</u>(3), 847-850.
- Goldsmith, N. F., & Johnston, J. O. (1975). Bone mineral: Effects of oral contraceptives, pregnancy, and lactation. Journal of Bone and Joint Surgery, <u>57-A</u>, 657-668.
- Goldsmith, N. F., Johnston, J. O., Picetti, G., & Garcia, C. (1973). Bone mineral in the radius and vertebral osteoporosis in an insured population. <u>Journal of Bone and</u> <u>Joint Surgery</u>, <u>55-A</u>(6), 1276-1293.
- Gonzalez, E. R. (1982). Premature bone loss found in some non-menstruating sportswomen. <u>Journal of the American</u> <u>Medical Association</u>, <u>248</u>, 513-514.
- Green, L. W., & Kreuter, M. W. (1991). <u>Health promotion</u> <u>planning: An educational and environmental approach</u>. Mountain View, CA: Mayfield Publishing Company.
- Guyton, A. C. (1976). <u>Textbook of medical physiology</u>. Philadelphia, PA: W. B. Saunders Company.
- Hancox, N. M. (1972). <u>Biology of bone</u>. Cambridge, MA: University Printing House.
- Hantman, D. A., Vogel, J. M., Donaldson, C. L., Friedman, R., Goldsmith, R. S., & Hulley, S. B. (1973). Attempt to prevent disuse osteoporosis by treatment with calcitonin, longitudinal compression and supplementary calcium and phosphate. <u>Journal of Clinical Endocrinology and</u> <u>Metabolism</u>, <u>36</u>, 845-858.
- Harper, A. B., Laughlin, W. S., & Mazess, R. B. (1984). Bone mineral content in St. Lawrence Island eskimos. <u>Human</u> <u>Biology</u>, <u>56</u>(1), 63-77.
- Hawkins, R. C., & Clement, P. F. (1980). Development and construct validation of a self-report measure of binge eating tendencies. <u>Addictive Behavior</u>, 5(3), 219-226.
- Heaney, R. P. (1983). Prevention of age-related osteoporosis in women. In L. V. Avioli (Ed.), <u>The osteoporotic syndrome</u>, <u>detection</u>, <u>prevention & treatment</u> (pp. 123-144). Orlando, FL: Greene & Stratton, Inc.

- Heaney, R. P. (1984). Risk factors in age-related bone loss and osteoporosis fracture. In C. Christiansen, C. D. Arnaud, B. E. C. Nordin, A. M. Parfitt, W. A. Peck, & B. L. Riggs (Eds.), <u>Osteoporosis: Proceedings of the Copenhagen</u> <u>Symposium on Osteoporosis June 3-8</u> (pp. 245-251). Copenhagen, Denmark: Department of Clinical Chemistry, Glostrop Hospital.
- Heaney, R. P. (1987). The role of nutrition in prevention and management of osteoporosis. <u>Clinical Obstetrics and</u> <u>Gynecology</u>, <u>50</u>(4), 833-846.
- Heaney, R. P. (1989). The calcium controversy: Finding a middle ground between the extremes. <u>Public Health</u> <u>Supplement</u>, <u>9-10</u>, 36-46.
- Heaney, R. P., & Barger-Lux, M. J. (1988). <u>Calcium and</u> <u>common sense</u>. New York: Doubleday.
- Heaney, R. P., Recker, R. R., & Saville, P. D. (1978). Menopausal changes in calcium balance performance. <u>Journal</u> of Laboratory & Clinical Medicine, <u>92</u>(6), 953-963.
- Henderson, R. C. (1991). Bone health in adolescence. <u>Nutrition Today</u>, pp. 25-29.
- Herodotus. (1947). <u>The Persian Wars, Book III</u>. (G. Rawlinson, Trans.). New York: The Modern Library.
- Huddleson, A. L., Rockwell, D., Kuland, D. N., & Harrison, R. B. (1980). Bone mass in lifetime tennis athletes. <u>Journal</u> <u>of American Medical Association</u>, <u>244</u>, 1107-1109.
- Jenkins, D. P., & Cochran, T. H. (1969). Osteoporosis: The dramatic effect of disease of an extremity. <u>Clinical</u> <u>Orthopedics</u>, <u>64</u>, 128-134.
- Johnston, C. C. (1989). Treatment of osteoporotic patients. <u>Public Health Reports</u>, <u>9-10</u>(Suppl.), 75-77.
- Jones, B. H., Harris, J. M., Vinh, T. N., & Rubin, C. (1989). Exercise-induced stress fractures and stress reactions of bone: Epidemiology, etiology and classifications. <u>Exercise</u> <u>and Sport_Science Review</u>, <u>17</u>, 379-422.
- Jones, K. P., Ravinikar, V. A., Tulchinsky, D., & Schiff, I. (1985). Comparison of bone density in amenorrheic women due to athletics, weight loss, and premature menopause. <u>Obstetrics and Gynecology</u>, <u>66</u>(1), 5-8.

- Joyce, J. M., Warren, D. L., Humphries, L. L., Smith, A. J., & Coon, J. S. (1989). Osteoporosis in women with eating disorders: Comparison of physical parameters, exercise, and menstrual status with SPA and DPA evaluation. <u>Journal of</u> <u>Nuclear Medicine</u>, <u>31</u>(3), 325-331.
- Kanders, B., Dempster, D. W., & Lindsay, R. (1988). Interaction of calcium nutrition and physical activity on bone mass in young women. <u>Journal of Bone and Mineral</u> <u>Research</u>, <u>3</u>(2), 145-149.
- Kanders, B., Lindsay, R., & Dempster, D. W. (1984).
 Determinants of bone mass in young healthy women. In C.
 Christiansen, C. D. Arnaud, B. E. C. Nordin, A. M. Parfitt,
 W. A. Peck, & B. L. Riggs (Eds.), <u>Osteoporosis: Proceedings</u> of the Copenhagen Symposium on Osteoporosis June 3-8 (pp. 337-340). Denmark: Department of Clinical Chemistry, Glostrop Hospital.
- Kelsey, J. L. (1989). Risk factors for osteoporosis and associated fractures. <u>Public Health Reports</u>, <u>9-10</u>(Suppl.), 14-20.
- Keys, A., Fidanza, F., Karvonen, M. J., Kimura, N., & Taylor, H. L. (1972). Indices of relative weight and obesity. <u>Journal of Chronic Diseases</u>, <u>25</u>, 329-343.
- Krabbe, S., Christiansen, C., Rodbro, P., & Transbol, I. (1979). Effect of puberty on rates of bone growth and mineralization. <u>Archives of Disease in Childhood</u>, <u>54</u>, 950-953.
- Kreipe, R. E., & Forbes, G. B. (1990). Osteoporosis: A "new" morbidity for dieting female adolescents? <u>Pediatrics</u>, <u>86(3)</u>, 478-480.
- Krolner, B., & Toft, B. (1983). Vertebral bone loss: An unheeded side-effect of therapeutic bed rest. <u>Clinical</u> <u>Science</u>, <u>64</u>, 537-540.
- Lanyon, L. E. (1986). Biomechanical factors in adaptation of bone structure to function. In H. K. Uhthoff (Ed.), <u>Current</u> <u>concepts of bone fragility</u> (pp.18-34). New York: Spingerverlag.
- Leighton, G., & Clark, M. L. (1929). Milk consumption and the growth of school-children. <u>Lancet</u>, 40-43.
- Leitch, I., & Aitken, F. C. (1959). The estimation of calcium requirement: A reexamination. <u>Nutrition Abstract Review</u>, 29, 393-411.

- Lindberg, J. S., Powell, M. R., Hunt, M. M., Ducey, D. E., & Wade, C. E. (1987). Increased vertebral bone mineral in response to reduced exercise in amenorrheic runners. <u>Western Journal of Medicine</u>, <u>146</u>(1), 39-42.
- Lindsay, R. (1981). The influence of cigarette smoking on bone mass and bone loss. In H. F. DeLuca, H. Frost, W. S. S. Jee, C. C. Johnston, & A. M. Parfitt (Eds.), Osteoporosis: Recent advances in pathogenesis and treatment (p. 481). Baltimore, MD: University Park Press.
- Lindsay, R., MacLean, A., Kraszewski, A., Hart, D. M., Clark, A. C., & Garwood, J. (1978). Bone response to termination of estrogen treatment. <u>Lancet</u>, <u>6</u>, 1325-1327.
- Lindsay, R., Tohm, J., & Kanders, B. (1986). The effect of oral contraceptive use on vertebral bone mass in pre- and post-menopausal women. <u>Contraception</u>, <u>34</u>, 333-340.
- Lloyd, T., Buchanan, J. R., Bitzer, S., Waldman, C. J., Myers, C., & Ford, B. (1987). Interrelationships of diet, athletic activity, menstrual status, and bone density in collegiate women. <u>American Journal of Clinical Nutrition</u>, <u>46</u>, 681-684.
- Lloyd, T., Myers, C., Buchanan, J. R., & Demers, L. (1988). Collegiate women athletes with irregular menses during adolescence have decreased bone density. <u>Obstetrics and</u> <u>Gynecology</u>, <u>72</u>(4), 639-642.
- Lloyd, T., Traintafyllou, S. J., Baker, E. R., Houts, P. S., Whiteside, J. A., Kalenak, A., & Stumpf, P. G. (1986). Menstrual disturbance in women athletes: Association with increased skeletal injuries. <u>Medicine and Science in Sports</u> and Exercise, <u>18</u>, 374-349.
- Lohman, T. G., Roche, A. F., & Martorell, R. (1988). <u>Anthropometric standardization reference manual</u>. Champaign, IL: Human Kinetics Books.
- Lutz, J. (1986). Bone mineral, serum calcium, and dietary intakes of mother/daughter pairs. <u>American Journal of</u> <u>Clinical Nutrition</u>, <u>44</u>, 99-106.
- MacMahon, B., Trichopoulos, D., Cole, P., & Brown, J. (1982). Cigarette-smoking and urinary estrogens. <u>New England</u> <u>Journal of Medicine</u>, <u>307</u>, 1062-1065.
- Maloney, M. J., McGuire, J., Daniels, S. R., & Specker, B. (1989). Dieting behavior and eating attitudes in children. <u>Pediatrics</u>, <u>84</u>(3), 482-489.

- Marcus, R. (1987). Calcium intake and skeletal integrity: Is there a critical relationship? <u>Journal of Nutrition</u>, <u>117</u>, 631-635.
- Marcus, R., Kosek, J., Pfefferbaum, A., & Horning, S. (1983). Age-related loss of trabecular bone in premenopausal women: A biopsy study. <u>Calcified Tissue International</u>, <u>35</u>, 406-409.
- Martin, A. D., & McCulloch, R. G. (1987). Bone dynamics: Stress, strain, and fracture. Journal of Sports Science, 5, 155-163.
- Matkovic, A. D., & Chesnut, C. (1987). Genetic factors and acquisition of bone mass. Journal of Bone & Mineral <u>Research</u>, 2(Suppl.1, Abstract No. 329).
- Matkovic, A. D., Fontana, D., Tominac, C., Goel, P., & Chesnut, C. H. (1990). Factors that influence peak bone mass formation: A study of calcium balance and the inheritance of bone mass in adolescent females. <u>American</u> <u>Journal of Clinical Nutrition</u>, <u>52</u>, 878-888.
- Matkovic, A. D., Fontana, D., Tominac, C., Lehman, G., & Chesnut C. H. (1986). Influence of calcium on peak bone mass: A pilot study. <u>Journal of Bone and Mineral Research</u>, <u>1</u>(Suppl., Abstract No. 168).
- Matkovic, A. D., Kostial, K., Simonovic, I., Buzina, R., Brodarec, A., & Nordin, B. E. C. (1979). Bone status and fracture rates in two regions of Yugoslavia. <u>The American</u> <u>Journal of Clinical Nutrition</u>, <u>32</u>, 540-549.
- Mazess, R. B. (1982). On aging bone loss. <u>Clinical</u> <u>Orthopedics</u>, <u>165</u>, 239-252.
- Mazess, R. B., & Cameron, J. R. (1971). Skeletal growth in school children: Maturation and bone mass. <u>American Journal</u> of Physical Anthropology, <u>35</u>, 399-408.
- McSwegin, P., Pemberton, C., Petray, C., & Going, S. (1989). <u>Physical best: The AAHPERD guide to physical fitness</u> <u>education and assessment</u>. Reston, VA: AAHPERD.
- Miller, J. Z., Kimes, T., Hui, S., Andon, M. B., & Johnston, C. C. (1991). Nutrient intake variability in a pediatric population: Implications for study design. <u>Journal of</u> <u>Nutrition</u>, <u>121</u>, 265-274.
- National Institute of Arthritis, Musculoskeletal, and Skin Diseases (NIAMS). (1986). Osteoporosis: Cause, treatment, prevention. <u>Orthopaedic Nursing</u>, 5(6), 29-38.

- National Institute of Health. (1986). <u>Osteoporosis: Cause,</u> <u>treatment, and prevention</u> (NIH Publication No. 86-2226). Washington DC: U.S. Government Printing Office.
- National Institute of Health (NIH) Osteoporosis Consensus Conference Statement. (1984). <u>Osteoporosis</u> (Publication No. 421-132: 4652). Washington, DC: U.S. Government Printing Office.
- National Osteoporosis Foundation. (1991). <u>Changing the shape</u> of your future. Washington, DC: Author.
- National Research Council, Sub-Committee on the 10th Edition of the RDA, Food & Nutrition Board, Commission on Life Sciences. (1989). <u>Recommended Dietary Allowance</u> (10th ed.). Washington DC: National Academy Press.
- Nelson, M. E., Fisher, E. C., Catsos, P. D., Meredith, C. N., Turksoy, R. N., & Evans, W. J. (1986). Diet and bone status in amenorrheic runners. <u>The American Journal of</u> <u>Clinical Nutrition</u>, 43, 910-916.
- Newton-John, H. F., & Morgan, B. D. (1970). The loss of bone with age, osteoporosis, and fractures. <u>Clinical</u> <u>Orthopedics</u>, <u>71</u>, 229-252.
- Nie, N. H., Hull, C. H., Jenkins, J. G., Steinbrenner, K., & Bent, D. H. (1975). <u>Statistical Package for the Social</u> <u>Sciences(SPSS)</u>. New York: McGraw-Hill.
- Nilsson, B. E., & Westlin, N. E. (1971). Bone density in athletes. <u>Clinical Orthopaedics and Related Research</u>, <u>77</u>, 179-182.
- Nordin, B. E. C. (1966). International patterns of osteoporosis. <u>Clinical Orthopaedics and Related Research</u>, <u>45</u>, 17-30.
- Nordin, B. E. C., & Heaney, R. P. (1990). Calcium supplementation of the diet: Justified by present evidence. <u>British Medicine Journal</u>, <u>300</u>, 1056-1060.
- Notelovitz, M., & Ware, M. (1982). <u>Stand tall! Every woman's</u> <u>guide to preventing osteoporosis</u>. New York: Bantam Books.
- Nylander, I. (1971). The feeling of being fat and dieting in a school population: An epidemiologic interview investigation. <u>Acta Sociomedicine Scandia</u>, <u>3</u>, 17-26.
- Orr, J. B. (1928). Milk consumption and the growth of school-children. <u>The Lancet</u>, 202-203.

- Pacifici, R., Susman, N., Carr, P. L., Birge, S. J., & Avioli, L. V. (1987). Single and dual energy tomographic analysis of spinal trabecular bone: A comparative study in normal and osteoporotic women. <u>Journal of Clinical Endocrinology</u>, <u>64</u>, 209-214.
- Parfitt, A. M. (1981). Integration of skeletal and mineral homeostasis. In H. F. DeLuca, H. Frost, W. Jee (Eds.), <u>Osteoporosis: Recent advances in pathogenesis and treatment</u> (pp. 115-126). Baltimore, MD: University Park Press.
- Peacock, M. (1991). Calcium absorption efficiency and calcium requirements in children and adolescents. <u>American</u> <u>Journal of Clinical Nutrition</u>, <u>54</u>, 2615-2655.
- Peck, W. A., & Avioli, L. V. (1988). <u>Osteoporosis-the silent</u> <u>thief</u>. Glenview, IL: Scott, Foresman, & Company.
- Pocock, N. A., Eisman, J. A., Hopper, J. L., Yeates, M. G., Sambrook, P. N., & Eberl, S. (1987). Genetic determinants of bone mass in adults: A twin study. <u>Journal of Clinical</u> <u>Investigation</u>, <u>80</u>(3), 706-710.
- Rambaut, P.C., Mack, P. B., & Vogel, J. M. (1975). Skeletal response. In R. S. Johnston, L. F. Dietlein, & C. A. Berry (Eds.), <u>Biomedical results of Apollo</u> (NASA SP-368) (pp. 303-322). Washington, DC: Scientific and Technical Information Office.
- Riggs, B. L., & Melton, L. J. (1986). Involutional osteoporosis. <u>New England Journal of Medicine</u>, <u>314</u>(26), 1676-1686.
- Riggs, B. L., Wahner, H. W., Melton, L. J., Richelson, L. S., Judd, H. L., & Offord, K. P. (1986). Rates of bone loss in the appendicular and axial skeletons of women. <u>Journal of</u> <u>Clinical Investigation</u>, <u>77</u>, 1487-1491.
- Rigotti, N. A., Nussbaum, S. R., Herzog, D. B., & Neer, R. M. (1984). Osteoporosis in women with anorexia nervosa. <u>New</u> <u>England Journal of Medicine</u>, <u>311</u>, 1601-1606.
- Rundgren, A., Eklund, S., & Jonson, R. (1984). Bone mineral content in 70- and 75-year-old men and women: An analysis of some anthropometric background factors. <u>Age and Ageing</u>, <u>13</u>, 6-13.
- Salisbury, J. J., & Mitchell, J. E. (1991). Bone mineral density and anorexia nervosa in women. <u>American Journal of</u> <u>Psychiatry</u>, <u>148</u>(6), 768-774.
- Sanborn, C. F., Martin, B. J., & Wagner, W. W. (1982). Is athletic amennorhea specific to runners? <u>American Journal</u> of Obstetrics and Gynecology, 143(8), 859-861.

- Sandler, R. B., Slemenda, C. W., LaPorte, R. E., Cauley, J. A., Schramm, M. M., Barrese, M. L., & Kriska, A. M. (1985). Postmenopausal bone density and milk consumption in childhood and adolescence. <u>The American Journal of Clinical</u> <u>Nutrition</u>, <u>42</u>, 270-274.
- Schwartz, B., Cumming, O. C., Riordan, E., Selye, M., Yen, S. S., & Rebar, R. W. (1981). Exercise-associated amenorrhea: A distinct entity? <u>American Journal of Obstetrics &</u> <u>Gynecology</u>, <u>141</u>(6), 662-670.
- Select Committee on Nutrition and Human Needs. (1977). <u>Dietary goals for the United States</u> (2nd ed.). Washington, DC: U.S. Government Printing Office.
- Shane, E. (1988). Osteoporosis. In S. C. Manolagas & J. M. Olefsky (Eds.), <u>Metabolic and bone disorders</u> (pp. 151-192). New York: Churchill Livingstone.
- Shangold, M. M. (1980). Sports and menstrual function. <u>The</u> <u>Physician & Sportsmedicine</u>, <u>8</u>(8), 66-72.
- Shangold, M. M., Freeman, R., Thysen, B., & Gatz, M. (1979). The relationship between long-distance running, plasma progesterone, and luteal phase length. <u>Fertility &</u> <u>Sterility</u>, <u>31</u>(2), 130-133.
- Shapiro, L. R., Hampton, M. C., & Huenemann, R. L. (1967). Teenagers: Their body size and shape, food, and activity. Journal of School Health, 37, 166-170.
- Smith, E. L., & Gilligan, C. (1989). Mechanical forces and bone. <u>Bone and Mineral Research</u>, <u>6</u>, 139-173.
- Smith, E. L., Nance, W. E., Kang, K. W., Christian, J. C., & Johnston, C. C. (1973). Genetic factors in determining bone mass. <u>The Journal of Clinical Investigations</u>, <u>52</u>, 2800-2808.
- Smith, E. L., Reddan, W., & Smith, P. E. (1981). Physical activity and calcium modalities for bone mineral increase in aged women. <u>Medicine and Science in Sports and Exercise</u>, <u>13</u>(1), 60-64.
- Sowers, M. R., Burns, T. L., & Wallace, R. B. (1986). Familial resemblance of bone mass in adult women. <u>Genetic</u> <u>Epidemiology</u>, <u>3</u>, 85-93.
- Sowers, M., Wallace, R. B., & Lemke, J. H. (1985). Correlates of forearm bone mass among women during maximal bone mineralization. <u>Preventive Medicine</u>, <u>14</u>, 585-596.

- Speroff, L. (1982). Impact of exercise on menstruation and reproduction. <u>Contemporary Obstetrics & Gynecology</u>, <u>19</u>, 54-78.
- Taylor, H. L., Jacobs, D. R., Schucker, B., Knudsen, J., Leon, A. S., & Debacker, G. (1978). A questionnaire for the assessment of leisure time physical activities. <u>Journal of</u> <u>Chronic Diseases</u>, <u>31</u>, 741-755.
- Thomas, A. E., McKay, D. D., & Cutlip, M. B. (1976). A nomograph method for assessing body weight. <u>American</u> <u>Journal of Clinical Nutrition</u>, <u>29</u>, 302-304.
- Tipton, C. M. (1982). Considerations for exercise prescriptions in future space flights. <u>Medicine and Science</u> <u>in Sports and Exercise</u>, <u>15</u>(5), 441-444.
- Toufexis, A. (1987, February 23). Going crazy over calcium. <u>Time</u>, <u>129</u>, pp. 88-89.
- Treasure, J. L., & Russell, G. F. M. (1987). Reversible bone loss in anorexia nervosa. <u>British Medical Journal</u>, 295, 474-475.
- Treharne, R. W. (1981). Review of Wolff's Law and its proposed means of operation. <u>Orthopaedic Review</u>, <u>10</u>, 35-47.
- Tylvansky, F. A., Bortz, A. D., Hancock, R. L., & Anderson, J. J. B. (1989). Familial resemblance of radial bone mass between premenopausal mothers and their college-age daughters. <u>Calcified Tissue International</u>, <u>45</u>, 265-272.
- Windom, R. E. (1989). FDA special topic conference on osteoporosis [Foreward]. <u>Public Health Reports</u>, <u>9-10</u>(Suppl.), iii-iv.

Wylie-Rosett, J., Wassertheil-Smoller, S., & Elmer, P. (1990). Assessing dietary intake for patient education planning and evaluation. <u>Patient Education and Counseling</u>, <u>15</u>, 217-227.

APPENDIX A

Three-day Diet Record
Student #_____ Date:____

FOOD INTAKE DIARY

Instructions: Please record <u>all</u> foods, beverages, candy, and medication ingested. Include amounts and brand names, if possible, for each item.

Code	Food Description	# of servings	size of serving	Brand Name
	•			
		· · · · · · · · · · · · · · · · · · ·		
			-	-
				-
			-	

List any medicines	or food supplements	ingested today:	
Name:	Amount:	Brand Name:	
Name:	Amount:	Brand Name:	

APPENDIX B

Questionnaire

Code #

Adolescent Health Survey

Section A: Physical Activity Inventory

1. Please check the activities in which you participated during the last year.

Activity	No	Yes	Month of Activity					Average	Min							
			J	F	м	A	м	J	J	A	s	0	N	D	of times per week	each day
Walking for pleasure or exercise																
Walk to and from school																
Jog/Run																
Dancing or Aerobic Exercise																
Physical Education Classes																
Cheerleading																
Volleyball																
Basketball																
Track/cross country																
Softball																
Exercises at home			,													
Weight-lifting																
Tennis																
Other form of exercise																

Section B: Menstrual history.

5

F

Γ

1.	How old were you when your monthly periods started?	
2.	How many periods have you had during the past year? (Feb. 1991 - Feb. 1992)	аг.
3.	Are your periods irregular during sports season, or when you have been very active? No	Yes
4.	Do you take oral contraceptives (the Pill)? No	Yes

Section C: Personal Behaviors.

1.	Do you smoke? No Yes How many cigarettes per day?
2.	Do you drink alcohol? No Yes How many drinks per week?
з.	Have you been on a low calorie diet during the past year?
	NoYesHow long were you dieting?
	How much weight did you lose?
4.	Do you take vitamins? No Yes How many times a week?
	Do you use Tums/Rolaids? No Yes How often?
	Do you take any other medicine? No Yes How often?
	What is the name of the medicine?

Section D: Family history of osteoporosis.

1.	Do any of your blood relatives (grandparents, aunts,	uncles,
	cousins) have osteoporosis or bones (like hip	
	or wrist fractures) that seem to	
	break easily? NoYes	(H)
	If yes, how many?	$\langle - \rangle \rangle$
2.	Do any of your blood relatives have a curved spine	
	that looks similar to this figure?	\prod
	No Yes	
	· · · ·	<u> </u>

Thank you for your time and effort in answering these questions!

APPENDIX C

Informed Consent

Informed Consent

The data gathered from this diet study, questionnaire, and physical measurements will be used for my research study that is part of the Masters of Arts in Health Education at the University of Northern Iowa.

Your participation in this study will involve three parts: (a) a three-day diet diary, (b) a questionnaire about personal health behaviors, and (c) height, weight, two skinfold measurements, and an elbow and wrist diameter measurement. Your responses will be confidential. An identification number will be used to match the three parts of the study, but this number will not be part of the analysis. Personal information will not be released to anyone other than the investigators without your permission. Your accurate and honest responses will be essential for the completion of this study.

Your participation in this study is voluntary. Your choice of whether or not to participate in this research will not affect the grade you receive for this course.

If you have any questions about the research or your rights as a subject, you may contact the UNI Graduate College office at 273-2748.

I am fully aware of the nature and extent of my participation in this project as stated above and the possible risks arising from it. I hereby agree to participate in this project. I acknowledge that I have received a copy of this consent statement.

(signature of subject)

(date)

(printed name of subject)

(signature of investigator)

Anita M. Johnson Graduate student

Faculty office: 273-6479 Department office: 273-2141

APPENDIX D

Nomograph for Body Mass Index

NOMOGRAPH FOR BODY WEIGHT

NOMOGRAPH FOR BODY MASS INDEX (KG/M^2)



Thomas, A. E., McKay, D. D., & Cutlip, M. B. (1976). A nomogram method for assessing body weight. <u>American Journal</u> of Clinical Nutrition, 29, 302-304.