5-1992

Characteristics Related to the Incidence of Osteoporosis in Two Distinct Female Populations

Deborah A. Pyke
Utah State University

CHARACTERISTICS RELATED TO THE INCIDENCE OF OSTEOPOROSIS IN TWO DISTINCT FEMALE POPULATIONS

by

Deborah A. Pyke

A thesis submitted in partial fulfillment
of the requirements for the degree

of

MASTER OF SCIENCE

in

Nutrition and Food Science

Approved:

UTAH STATE UNIVERSITY
Logan, Utah

1992

ACKNOWLEDGEMENTS

I would like to thank my major professor, Dr. Georgia Lauritzen, for all your patience as well as knowledge and encouragement throughout this project. Thank you for your endearing sense of humor, and for believing I really would finish this someday.

I greatly appreciate my other committee members, Dr. Deloy Hendricks and Dr. Brian Pitcher. Thank you for reviewing this thesis and for your helpful comments and insights along the way.

To Dr. Kathe Gabel, thanks for getting me started on this project, but most of all thank you for being such a good friend.

A special thanks goes to Noreen Schvaneveldt because you have done so many special things for me during my graduate studies. You have provided jobs and recommended me for scholarship funds. You helped me through some extremely turbulent times and encouraged me not to give up. If I have ever had a mentor in the field of dietetics, it is you.

To my family, David, Jason, and Rhian, I dedicate this thesis. Thank you, Jason and Rhian, for your patience and love while I was a graduate student. Dave, I can never thank you enough for all your help on this. I couldn't have done it without you, and I will always consider this thesis as an accomplishment of Pyke and Pyke. You must love me a lot.

Deborah A. Pyke

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

ABSTRACT

Characteristics Related to the Incidence of Osteoporosis in Two Distinct Female Populations

by

Deborah A. Pyke, Master of Science

Utah State University, 1992

Major Professor: Dr. Georgia C. Lauritzen
Department: Nutrition and Food Sciences

Osteoporosis is responsible for approximately 1.3 to 1.5 million fractures per year in the United States. The risk of osteoporosis increases with age, especially among postmenopausal women, and with lifestyle factors such as the use of certain drugs, heavy alcohol consumption, cigarette smoking, high caffeine intake, and sedentary living. Genetic factors that may influence susceptibility to osteoporosis include a positive family history of the disease, or a low weight-for-height ratio. In some cases, race and geographic location may increase or decrease the risk of osteoporosis. Known protective factors include obesity, estrogen replacement therapy, weight bearing exercise, and possibly calcium, fluoride, and Vitamin D. Although several studies have examined multiple factors in single populations, few comparisons have been made between populations within the same country.

Recent epidemiological studies have shown that the Utah population has lower rates of some chronic diseases than the national average. In this study, we used a questionnaire approach to relate the incidence of osteoporotic fracture to 28 lifestyle, dietary, physical, and geographic factors in postmenopausal women between a Utah population and a combined population from North Dakota, South Dakota, and Colorado.

Logistic regressions were used to determine the probability of osteoporosis for these two populations and to determine which factors significantly ($P \leq 0.05$) increased or decreased the incidence of osteoporosis. The following factors were significantly related to the occurrence of osteoporosis: age, race, arthritis, cortisone, and fluoridated water. Dietary factors, including calcium, were not significantly related to osteoporosis in this study. The model successfully predicted the occurrence of an osteoporotic event in 72% of the cases. (90 pages)

INTRODUCTION

Osteoporosis is the most common metabolic bone disease in elderly women in the United States (1). Osteoporosis is characterized by a reduction in bone mass, microarchitectural deterioration of bone tissue, increased bone porosity, and a subsequent increased susceptibility to fracture. It is considered a major public health problem in the United States and is estimated to affect 15-20 million people (2). Osteoporosis is responsible for approximately 1.3 to 1.5 million fractures in the United States alone (3, 4). The estimated annual direct cost of osteoporosis is $7 billion, yet the inclusion of indirect costs increases the estimated amount to $10 billion per year (4). In addition to medical costs, fractures may cause prolonged disability and premature death. As the elderly population grows, it is anticipated that osteoporosis will become even more prevalent and will account for a still greater proportion of the health care bill in the United States.

Osteoporosis increases the risk of fracture at three primary sites: the hip, the vertebrae, and the distal forearm (Colles' fracture). Sixty-six percent of women age 65 have radiologic evidence of osteoporosis (5). Hip fracture has the greatest degree of debilitation and is fatal in 12 to 20% of cases (4, 5). Approximately 250,000 hip fractures occur yearly, and much of the expense of osteoporosis is related to this type of injury (4). Twenty-five percent of the patients with hip fracture are confined to long-term care in a nursing

home, and 50% of hip fracture patients are not able to walk independently, nor live independently afterward (4, 5). The lifetime risk for hip fracture for white women is approximately 15% (4); however, women who live to be 90 years of age have a 33% risk of fracturing a hip (6).

Vertebral fractures can occur spontaneously or with very minimal trauma, such as a cough. Riggs estimates 650,000 vertebral fractures occur each year (4). One third of women age 65 or older experience these fractures (4, 7). Vertebral fractures may or may not cause back pain, but once vertebrae are deformed, they never regain normal shape. An accumulation of these deformities can lead to "Dowager's hump," loss of stature, and to limitations in reaching, bending, or in other activities of daily living. Each complete compression fracture will cause a loss of 1 cm in height (8). Vertebral fractures account for approximately 160,000 physician's office visits annually (9). The economics, as well as disabilities attributable to vertebral fractures, is virtually unknown.

Fractures of the distal radius (Colles' fractures) are very common among postmenopausal women, resulting in approximately 200,000 Colles' fractures per year (4). They are the most common fractures reported among white women in the United States before age 75, after which they are surpassed by hip fractures (8). Colles' fractures are rarely fatal and cause much less disability than hip fractures. Less than 20% of these fractures require hospitalization, and

the total medical care cost is estimated to be $140 million annually (10).

Epidemiological studies have revealed several risk factors for osteoporosis as well as several factors which are considered protective (e.g., 8, 11, 12, 13). These studies have shown that the risk of osteoporotic fractures increases with age, especially among postmenopausal women and among women who have undergone bilateral oophorectomy. The risk of osteoporosis can increase due to lifestyle factors, such as use of certain drugs, heavy alcohol consumption, cigarette smoking, high caffeine intake, sedentary living, and even possibly a high protein intake. Genetic factors that may influence susceptibility to osteoporosis include a positive family history of the disease, or a low weight-for-height ratio. Race and geographic location can increase risk for osteoporosis or be protective against it. Factors which are considered protective include obesity, estrogen replacement therapy, weight bearing exercise, and possibly calcium, fluoride, and Vitamin D.

Several studies have evaluated various lifestyle risk factors and the incidence of fracture (14, 15, 16, 17, 18). Other studies have investigated the incidence of osteoporosis variation among race (19, 20, 21, 22) and geographic location (23, 24, 25). A few studies have looked at the incidence of osteoporosis among different geographic locations within a country or state (26, 27, 28). Interviews and questionnaires have been used frequently to help assess a person's risk for

osteoporosis; however, they are usually used along with other measures of assessment, such as bone mineral density (e.g., 17, 18, 29, 30, 31, 32, 33).

A few surveys have used written questionnaires as their only tool of assessment. Some of these have been used to assess fracture risk according to various lifestyle factors (14, 16). A few others have used postal surveys to assess women's views regarding hormone replacement therapy in osteoporosis and to evaluate risk of falling with incidence of forearm fractures (34, 35). No reported studies have used a written questionnaire alone to determine factors related to incidence of osteoporosis among different geographic locations within a country.

Purpose and Objectives

The purposes of this study include:

1. To assess by mailed questionnaire, participants' dietary, physical, and demographic characteristics related to osteoporosis,
2. To relate those characteristics to the incidence of osteoporotic fracture, and
3. To assess any differences between two distinct female populations (Utah and non-Utah), in the incidence of osteoporotic fracture, both geographically and according to lifestyle.

LITERATURE REVIEW

As early as 1824, Astley Cooper noted the relationship between aging and skeletal fragility in his Treatise on Dislocations and on Fractures of the Joints (cited in 19). Fifty years ago, Albright et al (36) suggested estrogen deficiency as the etiologic factor of osteoporosis in postmenopausal women. Today most investigators agree that osteoporosis is a very complex and multifactorial bone disorder.

Age and Sex

Age is probably the most powerful predictor of bone mass, and bone loss is considered a universal phenomenon of aging. Bone mass peaks by about age 35, with 90% accumulating before age 20 (37). Bone mass decreases after the fourth or fifth decade in all populations that have been studied (10). The most rapid rate of bone loss in women occurs for approximately 5 years after menopause, when there can be a negative calcium balance of 40-120 mg/day (38). Approximately 70% of all hip fractures occur after age 65 (8).

Two distinct patterns emerge when the incidence of fracture is plotted as a function of age. First, fractures at sites which contain large amounts of trabecular bone increase in women shortly after menopause (4). These are usually fractures of the distal forearm and vertebral fractures. Colles' fractures (distal forearm) continue to

increase until about age 65, then plateaus; vertebral fractures continue to rise (4, 8, 37). The second pattern is associated with sites containing similar amounts of cortical and trabecular bone. Most important is fracture of the hip, which increases slowly with aging and reaches a peak incidence between 70 and 90 years of age in both men and women (4, 38).

Osteoporotic fractures are more common in women than men. Seventy-five to 80% of hip fractures affect women and approximately 50% of these fractures are among women 80 years of age or older (8, 12). Studies from medical records show that hip fracture is two to three times higher in women than men, and incidence rates for fracture of the wrist, proximal humerus, and pelvis are six to eight times higher in women than in men (4, 24).

Some of the reasons attributed to the higher fracture rate among women include a lower peak bone mass compared to men, the marked reduction in estrogen levels after menopause, and possibly a lower habitual dietary calcium intake (38).

Geography and Race

Incidence of osteoporosis varies considerably from one geographic area to another and among races. Fracture rate tends to be higher among whites than non-whites regardless of geographic location (37). It has long been known that blacks have greater bone mass and vertebral density, thicker bone cortex and fewer fractures than whites (20, 22, 39). Hip

fracture rates are twice as high in white American women as black American women (8, 22). Bollet et al (40) studied black and white populations in one city (Charlottesville, Virginia) where environmental factors should be less variable than in a cross-cultural study. The age-adjusted fracture rate for whites was approximately twice that of blacks in this single community study.

The United States has the highest fracture rate (8, 41), and the Bantu of South Africa have the lowest recorded incidence of fracture (20, 37). The Moari in New Zealand also have a very low incidence of fracture (37). Conversely, fracture rates are high among Indians in Singapore, which otherwise has a low incidence (19, 37). American and European-born Jewish women living in Jerusalem have higher hip fracture rates than women in that city who are native-born Israelis or of African or Asian descent (12, 42). Smith (21) has shown that Puerto Rican women have fewer osteoporotic fractures than their American counterparts, and Mazess (43) found North Alaskan eskimos have a lower bone mineral content than white Americans.

Lewinnek et al (12) compared several studies on the incidence of hip fracture in various countries to the latitude of the city studied. Lower rates of fracture occurred within studies in tropical cities and higher rates generally occurred in Northern European cities. He also compared the degree of industrialization, specifically motor vehicles in use per capita among various cities and

countries. A positive correlation was demonstrated between motor vehicles in use and fracture rate.

Nutrients

Several nutrients, notably calcium, have been suggested to influence the incidence of osteoporosis. Calcium is necessary for normal bone growth and for attainment of peak bone mass. Although the Recommended Dietary Allowance for calcium had been set at 800 mg/day for adults for several years, considerable debate persists concerning optimal levels. The intake of calcium during the years of bone mineralization appears to be related to peak bone mass. It is still uncertain at what age peak bone mass is attained; however, generally it is thought to be by age 35 and probably not before age 25 (44). The most current edition of the RDA recommends an extra calcium allowance through age 24 to permit more adequate mineral deposition (45). This allowance of 1200 mg/day had, in previous editions, only been recommended through age 18.

The 1984 Consensus Committee on Osteoporosis suggested a calcium intake of 1500 mg/day before menopause may reduce the risk of osteoporosis later in life (46). Heaney et al (47) estimated the average calcium intake of postmenopausal women in the United States to be 475-575 mg/day, and suggested the RDA of 800 mg/day may be insufficient for many of these women to maintain a zero calcium balance. He estimated that postmenopausal women who are not estrogen

treated have a calcium requirement of 1540 mg/day (48). Calcium balance depends on dietary intake of calcium, intestinal absorption, and obligatory calcium loss in sweat, urine, and feces. The intestine becomes less efficient in absorption with age, especially after age 70, and coupled with a decline in production of 1,25-dihydroxyvitamin D, which aids calcium absorption, calcium requirements may increase with age (4, 9, 47, 49).

Hegsted (41) has noted a sharp contrast between recommendations of the Consensus Conference on Osteoporosis and epidemiological data. The susceptible populations consume more calcium than those populations with low incidence of osteoporosis. Unfortunately, studies which measure bone density have shown inconsistent results from increasing calcium intake (eg, 8, 50, 51). This may be due in part to variability in dietary assessment.

A recent review of controlled clinical trials of calcium supplementation has revealed some valuable insight. There appears to be different responses to supplementation of calcium in women depending on their menopausal age (52). During early menopause, especially the first five years, bone loss from the radius is attenuated, but not arrested by calcium supplementation (52, 53). Doses of 1000 mg/day seem to produce the maximal effect. The spine does not have this response to calcium supplementation during this early postmenopausal age (52). Older postmenopausal women have been studied less extensively than younger postmenopausal

women, especially women 80 years of age and older. Current findings in this age group of women indicate that bone loss from the appendicular skeleton, particularly the radius, is attenuated by increased calcium intake (52). Bone loss from the spine may be retarded in older women who have typically had a low calcium diet, by increasing calcium intake to current RDA levels (52, 54). Unfortunately, it is still inconclusive whether calcium supplementation alters hip density in postmenopausal women.

While the role of dietary calcium in the etiology of osteoporosis remains controversial, there is growing evidence of its effect on peak bone mass and possible preventative therapeutic value (2, 38, 55, 56) Matkovic et al (26) studied cortical bone mass density and prevalence of hip fracture in two areas of Yugoslavia. The areas differed greatly in their consumption of calcium (450 mg/day vs 1200 mg/day), while other variables were similar. Maximal bone density was significantly higher by age 20 and fracture rate lower among people in the high calcium district. A difference in bone mass of approximately 7% was responsible for more than a 50% difference in fracture rate. Adult bone status and fracture risk were established by late adolescence and were due to differences in peak bone mass.

Vitamin D, as previously stated, aids in absorption of calcium. Vitamin D is first metabolized to 25-hydroxyvitamin D, or calcidiol, in the liver and then hydroxylated in the kidney to either 1,25-dihydroxyvitamin D (1,25(OH)2 D), or to

24,25-dihydroxyvitamin D (24,25(OH)2 D). 1,25(OH)2 D, or calcitriol, is the most potent form with wide-ranging effects, including modulation of intestinal absorption of calcium and bone remodeling (57). Various studies have been conducted using Vitamin D analogues in patients with osteoporosis. Riis (58) showed that treatment of osteoporosis with 24,25(OH)2 D had no prophylactic effect on postmenopausal bone loss. Riggs et al (59) and Gallagher et al (60) treated osteoporotic patients with calcitriol. Calcium absorption normalized in both studies, which supports the hypothesis that inadequate production of calcitriol is the primary cause of decreased calcium absorption in postmenopausal osteoporosis.

Vitamin D may have a more profound effect on trabecular bone than on cortical bone. Bikle et al (61) infused rats with 1,25(OH)2 D to determine if there were varying responses between trabecular and cortical bone. The proximal tibia and shaft were examined for bone mass, calcium accumulation, and density distribution of bone particles. In the proximal tibia, vitamin D decreased calcium accumulation; however, there was an increase in bone mass and a shift in the particle distribution to more mineralized fractions. In the shaft there was no change in total bone mass and no decrease in calcium accumulation. There was, however, a redistribution of bone to less mineralized fractions. Bone resorption was not directly measured in this study, but the

net result was an increase in bone mass and bone density of trabecular bone that was not found in cortical bone.

The use of fluoride in the treatment of osteoporosis is still experimental. Fluoride stimulates osteoblastic activity with a subsequent production of osteoid tissue. The fluoride ion is deposited in the crystal lattice of hydroxyapatite in place of the hydroxyl ions, resulting in a mineral phase which is more resistant to resorption (62). Fluoride doses of 1 ppm do not appear to have an effect on incidence of fracture; however, Iskrant (63) found that mortality from falls in elderly white women is lower at intakes of 4 ppm or greater. In 1966, an epidemiological study of two populations in North Dakota showed a high fluoride intake (4 to 5.8 ppm) reduced the number of osteoporotic fractures (28). An autopsy study showed cancellous bone strength was greater in immobilized women who drank fluoridated water than those whose water was not fluoridated (64). More recent studies have indicated that fluoride treatment of osteoporosis has a very narrow therapeutic window (3, 65). Its effect on incidence of fracture remains controversial and is probably dose dependent. Riggs (4) notes that fluoridic bone has increased crystallinity, but also has decreased elasticity, and, therefore, may be structurally abnormal with decreased bone strength. A recent study failed to demonstrate a decrease in vertebral fracture rate after 4 years of treatment with 75 mg of sodium fluoride per day, in spite of an increase in

trabecular bone mass of the spine (66). Pak (65) states that fluoride can protect against vertebral fracture, but not alone or in high dosages. He recommends that calcium be given with a slow release form of sodium fluoride to allow for adequate mineralization of bone and minimum risk for fracture.

Experimental increases in protein increase urinary calcium excretion and may have adverse effects on calcium balance. Hegsted (41) suggested that a high osmotic load on the kidney, due to the high protein diets of affluent western societies, might eventually impair the kidney's ability to regulate calcitriol levels. Epidemiological data show hip fractures and protein are positively related (41). Mazess (43) has shown North Alaskan Eskimos have lower bone mineral content than United States whites and suggested their high protein diet (200-400 g/day) as a contributing factor.

Physical Activity and Body Build

Decreased physical activity often accompanies the aging process and has been implicated as a risk factor in osteoporosis. Prolonged inactivity causes atrophy in muscle and bone tissue, and immobilization is known to reduce bone mineral content (67). Athletes are known to have greater bone densities than non-athletes. Brewer et al (32) compared the skeletal status of marathon runners and sedentary premenopausal women, 30-49 years of age. Bone mineral content and density were greater in the distal radius of the

marathon runners than the sedentary women. Pocock et al (68) studied the relationship between physical fitness and bone mass in the femoral neck, lumbar spine, and forearm in eighty-four pre and postmenopausal women. In the postmenopausal women, fitness was the only significant predictor of femoral neck bone density, whereas weight and fitness were predictors in lumbar spine density. Increased fitness may increase bone density at clinically important fracture sites in osteoporosis. Heinrich et al (33) compared bone mineral content at four skeletal sites among young female adults who regularly performed either weight lifting, running, swimming, or were inactive. Body builders had greater bone mineral content than runners, swimmers, or controls. Due to the results of their study, they suggested that weight training may provide more stimulus for increasing bone mineral content than nonresistance endurance exercise.

Thin women have less cortical bone mass and are at greater risk for osteoporosis than obese women (8). Williams et al (69) found the risk of hip fracture was elevated in thin postmenopausal women, and Hemenway et al (14) found the combination of thinness and alcohol consumption of more than 15 g/day substantially increased the incidence of fracture. Obesity is considered protective against osteoporosis. Most of the estrogen produced postmenopausally is from the conversion of androstenedione to estrone and much of this conversion takes place in adipose tissue. Obese women are therefore likely to produce more estrone than thin women (8).

In addition, greater body weight may be associated with greater peak bone mass in early adulthood.

Alcohol and Smoking

The relation between lifestyle variables such as alcohol and smoking and risk for fracture is not well understood. Daniell (70) noted that women who were smokers lost an average rate of 1.02% of cortical bone per postmenopausal year compared with 0.69% per postmenopausal year for nonsmokers. The age of menopause was earlier for smokers than for nonsmokers (47.6 vs 49.1) in this study. He concludes that the lower frequency of obesity in the women who smoked contributed significantly to the greater loss of cortical bone than was seen in the nonsmoking subjects studied.

In a study conducted by Williams et al (69), postmenopausal women were interviewed to determine which characteristics influence the occurrence of fracture. Subjects were women ages 50-74 who had sustained either hip or forearm fractures. Estrogen use, height and weight status, and smoking history were evaluated. The risk of hip fracture was greater among thin women who smoked, especially if they did not use estrogen. Smoking did not increase the risk of forearm fractures in estrogen users, but among nonusers of estrogen, smoking did increase the risk, especially in those who were thin.

Conversely, a study on fracture and lifestyle characteristics by Hemenway et al (14) found that smoking was not a risk factor for hip or forearm fracture. This study, involving registered nurses, used a mailed questionnaire to assess fracture risk. A limitation of the survey was the age group of the women surveyed. Only 37% were in an age group that could be considered near menopause or postmenopausal. The population studied may have been too young to determine the effects of smoking on fracture risk.

Decreased bone mass has been noted in patients who abuse alcohol (71, 72). Several explanations have been proposed to account for this. These include nutritional deficiencies of calcium and Vitamin D, malabsorption of calcium and Vitamin D secondary to pancreatic or liver disease, abnormalities in metabolism of Vitamin D due to cirrhosis of the liver, abnormal parathyroid function or secretion induced by ethanol directly or indirectly, or a toxic effect of ethanol on calcium absorption. There is also a possible effect of ethanol on bone remodeling (71).

Caffeine and Drugs

Caffeine has been a reported risk factor for osteoporosis, particularly because of its effect on calcium excretion. The average intake of caffeine in the American population is about 250 mg/day. Massey and Wise (73) and Massey and Berg (74) studied the effect of dietary caffeine on urinary excretion of several nutrients including calcium.

Subjects fasted for 10 hours prior to receiving 0, 150, or 300 mg caffeine in beverage form. Total 3-hour urinary excretion of calcium increased significantly after caffeine intake. Urinary concentrations of calcium nearly doubled after ingestion of the higher (300 mg) caffeine dose.

Heaney and Recker (75) studied the effects of different levels of nitrogen, phosphorus, and caffeine on calcium balance in premenopausal women. Caffeine consumption correlated with a decreased calcium balance in their subjects by increasing both urinary and intestinal calcium losses.

Chronic use of certain drugs may increase bone loss and predispose their users to fracture. These include certain diuretics, heparin, antacids containing aluminum, excessive doses of thyroid hormone, and corticosteroids. Of these, corticosteroids have been the best documented. Although the anti-inflammatory and immunosuppressive effects of glucocorticoids is extremely therapeutic, adverse effects include the development of osteopenia with an increased propensity to bone fracture (76). There are approximately five million individuals in the United States who take steroid medication, and many of these are over age 65 (76, 77). These people would already be prone to osteoporosis, as well as to age-dependent decreases in bone mass and intestinal calcium absorption. Additional glucosteroid effects on the skeleton in this age group are obviously undesirable. A study done by Reid and Heap (78) addressed the impact of glucocorticoid therapy on vertebral mineral

density. There was a significant relationship not only in the duration of steroid treatment and vertebral mineral density but also in the total cumulative steroid dose. The authors cautioned that effort should be made to minimize dosage and duration when using glucocorticoid therapy.

Reproductive History and Heredity

Childbearing and breastfeeding have also been studied in respect to changes in bone mass and fracture risk. Calcium demands during pregnancy and breastfeeding are accompanied by changes in levels of Vitamin D, calcitonin, and parathyroid hormone. These changes lead to alterations in bone mineral metabolism such as increased gastrointestinal absorption and increased renal conservation of calcium. Some studies indicate pregnancy and breastfeeding are associated with a decreased risk of postmenopausal hip fracture or bone loss (77, 30). Wyshak (77) collected data from nursing home records to examine the relationship between hip fracture and reproductive history. She found a sharp decline in incidence of hip fracture among women of high parity of four or more living children. In contrast, Alderman et al (29) did not find any significant relationship among parity, breastfeeding, and risk of hip or forearm fracture. Aloia et al (30) examined the relationship of various factors to bone mass in early postmenopausal women. Breastfeeding and pregnancy were associated in this study to higher bone mass.

There is a definite direct relationship between estrogen loss and osteoporosis. Bone loss occurs whenever estrogen levels are reduced, whether by natural menopause or by oophorectomy (79). Evidence exists that trabecular bone loss may begin as estradiol production rates start to decline, even prior to cessation of menses (80). Many studies have shown that estrogen replacement therapy prevents or greatly retards bone loss in postmenopausal women as long as the therapy is continued (81, 82, 83). The recommended dose is 0.625 mg of conjugated estrogen; adding a progestogen does not interfere with skeletal response to estrogen and appears to decrease the risk of unopposed estrogen therapy and endometrial cancer (3). Estrogen therapy given early in menopause for at least five years reduces hip and Colles' fractures by approximately 50%, and vertebral fractures by 90% (3). However, the effect of estrogen is reversible. When therapy stops, the accelerated phase of bone loss will occur again (77). This does not mean the benefits of estrogen are lost. If, for example, a woman uses estrogen therapy for 10 years, her bone mass "age" will be at least 10 years younger than her actual age when she discontinues the therapy (77).

Various studies have shown a relationship between heredity and bone mass. Genetic studies of twins show strong associations between bone mineral content and bone density (55, 84). Mother-daughter pairs have also been studied, and frequently a significant relation between bone mass and

genetics has been noted (55, 85). Lutz and Tesar (85) suggest that inheritance of bone mass may have two components, one which influences development of peak bone mass and one related to loss of bone at menopause. Pollitzer and Anderson (55) reviewed ethnic and genetic factors in bone mass and indicated that the bone mass accumulated by age 20 is highly associated with maternal bone mass, but beyond menopause other factors of environmental nature seem to dominate.

METHODOLOGY

Subject Selection and Rationale

The population sample was chosen from female postmenopausal subjects in Utah, and three Great Plain states, North Dakota, South Dakota, and Colorado (Non-Utah). This allowed for subjects that came from two distinct geographical regions of the United States, and from populations that differ in their predominate religious preference.

Some studies have indicated that lifestyles associated with certain religious preferences lend themselves to lower rates of some chronic diseases (86, 87, 88). The majority of Utah's population (70%) are members of the Church of Jesus Christ of Latter-Day Saints (Mormons). The Mormon church teaches abstinence from alcohol, caffeine, and smoking. It promotes a healthy lifestyle, including a prudent diet and regular exercise. Mormons typically have large families, giving Utah the highest birth rate in the United States. All of these factors have been implicated in osteoporosis and, therefore, may give many Utah women added protection against bone loss.

Recent surveys have shown that Utah has lower rates of some chronic diseases than the national average. *Morbidity and Mortality Weekly Report* (89) shows Utah to have the third lowest mortality rate from coronary heart disease in the United States. Some behavioral risk factors associated with

many diseases are less prevalent in Utah than other states surveyed. These factors include obesity, sedentary lifestyle, smoking, and alcohol abuse (90). Utah women, including those who are postmenopausal, have a higher calcium intake than average when compared to the USDA Household Food Consumption Survey (91).

Utah's population is 1,461,037 and is composed of 94% Caucasian and approximately 4% Chicano. Four-fifths of the population of Utah live in urban areas. Utah's water supply is primarily non-fluoridated.

Colorado has a population of 2,889,964 of which approximately 89% is Caucasian. The remaining population is primarily Black, Japanese, and American Indian. Eighty percent of the population are in urban areas. The predominate religions are Roman Catholic, Methodist, and Baptist.

North Dakota's population is 652,717, and it is still a rural state with urban residents in only a few metropolitan areas. Its population is primarily Caucasian with about 5% of the remaining population being American Indian, Black and Asian. The predominate religions are Lutheran and Roman Catholic.

South Dakota has a population of 690,768 and is approximately 54% rural. Its population is 6% American Indian, 3% Black, and the rest is mostly Caucasian. Lutheran, Roman Catholic, Methodist, and Presbyterian are the most common religious faiths.

The non-Utah states more commonly have fluoridated water with some regions in Colorado and North Dakota having greater than 4 ppm.

Questionnaire Design and Implementation

A written questionnaire was designed and implemented using standard techniques (92, 93). Special attention was given in developing the questionnaire for the target population which was an elderly group of postmenopausal women. This included the design of the cover page, large print throughout the questionnaire, and ease of completion. The questionnaire is found in Appendix A. Questions pertaining to lifestyle, nutrient intake, medical history, and demographics were asked.

Lifestyle factors included the evidence of weight bearing exercise, amount of smoking, and the ingestion of caffeine and alcohol. Nutrient intake focused primarily on calcium content of the diet; however, use of vitamin supplements was also evaluated. The medical history gathered information regarding family history of osteoporosis, incidence of fracture of the hip, vertebrae or wrist, use of medications, evidence of chronic disease, surgeries such as hysterectomy, use of estrogens, and age of menopause. Demographic data included the subject's age, height, weight, number of children, race, religious preference, residence, and fluoridation of the water supply.

The questionnaire was pretested on a small sample of postmenopausal women as a portion of a separate study (pers. comm., Dr. G. Lauritzen and Dr. D. Hendricks, Utah State University, Logan). This study examined risk factors relating to osteoporosis and also required the participants to undergo Computed Tomography at Logan Regional Hospital. Participants had no difficulty in completing the questionnaire, and it appeared to be ready for use on a larger scale.

The questionnaire was distributed to 600, 200, 200, and 200 postmenopausal women in Utah, North Dakota, South Dakota, and Colorado, respectively. The surveys were mailed in bulk to Registered Dietitians working in the Division of Aging and Adult Services in each of the four states. The dietitians then distributed the surveys to area managers of senior citizen centers throughout their state. The surveys were distributed more heavily in urban areas; however, both urban and rural areas were covered. Women at the senior citizen centers were asked to fill out the questionnaire. Each questionnaire was coded with a four digit identification number in the upper right hand corner. The first digit corresponds to the state to which it was sent. A cover letter was attached to each questionnaire explaining the purpose of the survey and requesting the assistance of each participant. A self-addressed stamped envelope was attached to each questionnaire for return of the questionnaire. A follow-up letter was mailed to the previously mentioned

dietitians in each state for distribution to the area managers of the senior citizen centers. These served as a thank-you to all the participants and a reminder to non-responders to return their questionnaires. Due to limited funding for this research project, a second mailout was not attempted.

Upon return of the questionnaires, data were coded using standard categorical coding procedures. A copy of the codes is included in Appendix B. When participants did not respond to any given question, they were placed in a no response category.

For the purposes of this study, participants were classified as osteoporotic or non-osteoporotic. A participant was defined as having osteoporosis when at least one of the following criteria was met:

1. A participant was diagnosed by a physician as having osteoporosis (Question #1 = yes).
2. A participant had a fracture in any of the three major osteoporotic sites (wrist, hip, or spine), and the fracture occurred at or after the age of menopause (Question #3 = yes, and Question #22 = yes).
3. A participant had a fracture in any of the three major osteoporotic sites and did not respond to the question concerning age of menopause (Question #3 = yes, and Question #22 = no response), yet the

fracture occurred at or greater than age 55 (Question #3a $\geq$ 55).

The average age of menopause in the United States is 51. A women was defined as postmenopausal if she recorded her age as 55 or greater, allowing a reasonable margin for individual variation.

If a participant was not diagnosed by a physician to have osteoporosis, and age of menopause as well as current age was not recorded by the participant (Questions #22 and 25), then osteoporosis status was put in a no response category.

If questionnaires were not adequately completed, they were rejected from the sample. For example, if the participant failed to answer critical questions to the study, particularly questions regarding medical history and demographic information, they were rejected. If a participant failed to give only selected information, such as current weight, this was simply coded as a no response category.

Calcium intake was calculated for each participant in mg calcium/month from the food frequency information found in the questionnaire (Questions 5-11). Calcium content for the serving size given for each food in the questionnaire was calculated from food tables in USDA Handbook #8- Composition of Foods. Calcium content of milk was calculated using the value for 2 % milk (297 mg/cup). The calcium contents of yogurt (311 mg/8 ounces), hard cheese (200 mg/ounce), cottage

cheese (73 mg/0.5 cup), ice cream and frozen yogurt (88 mg/0.5 cup), and bread (23 mg/slice) were calculated by averaging their various respective types. If the participant answered "never" to any of the questions regarding consumption of a high calcium food, the calcium intake for that food was 0 mg/month. If the participant answered "monthly" (1-3 days/month), "weekly" (1-4 days/week), or "daily" (5-7 days/week) to any of the food consumption questions, an average of 2 days/month, 8 days/month, or 28 days/month was calculated for monthly calcium intake of that food.

Caffeine intake was calculated for each participant in mg caffeine/month. Caffeine content of coffee and tea was calculated as mg/cup and as mg/12 ounces for cola.

Alcohol servings per month were also calculated for each participant. One serving equals 12 ounces of beer, 4 ounces of wine, or 1 ounce of hard liquor.

Participants were asked to indicate weight bearing exercise in which they were engaged (Question #24). They recorded the number of times weekly or monthly they participated in each activity and also the typical duration (minutes/day). From this information, the participant's total minutes/month of weight bearing activity were calculated.

Statistical Analysis

Significant factors related to the occurrence of osteoporosis were determined using a hierarchical log-linear modeling approach that led to a final logit model (94). The occurrence of osteoporosis was the dependent factor while demographic, lifestyle, and geographic parameters were the independent factors in the logit model. Significant logits were transformed to provide a probability of osteoporosis. Descriptive variables that were measured as continuous data (eg, height, height-loss) were tested between the two groups using an analysis of variance.

RESULTS

Out of a total of 1200 surveys, 723 were returned, giving a response rate of 60%. Depending on the variable examined, and the analysis method, the total useable number of respondents varied from 584 to 655. Caucasian was the predominant race in Utah and non-Utah respondents (92% and 97% respectively), with the remainder being divided among Asians and Blacks in similar proportions to those found in the general population.

Differences in the predominant religious preference between the Utah and non-Utah respondents were anticipated. The religious preference of Utah respondents was 67% LDS, 16% Protestant, and the remainder divided among other religions, or no religious preference. The religious preference of non-Utah respondents was 67% Protestant, 25% Catholic, and the remainder divided among other religions, or no religious preference.

There was a slight, but significant difference in age between osteoporotic and non-osteoporotic ($P < 0.01$, 73.4 vs. 70.6 years) and between Utah and non-Utah ($P = 0.03$, 70.6 vs. 72.6) respondents. There was no significant difference in weight between osteoporotic and non-osteoporotic ($P = 0.43$) and between Utah and non-Utah ($P = 0.27$) respondents (Figure 1). There was also no significant difference in height between osteoporotic and non-osteoporotic ($P = 0.74$) and between Utah and non-Utah ($P = 0.88$) respondents (Figure 2).

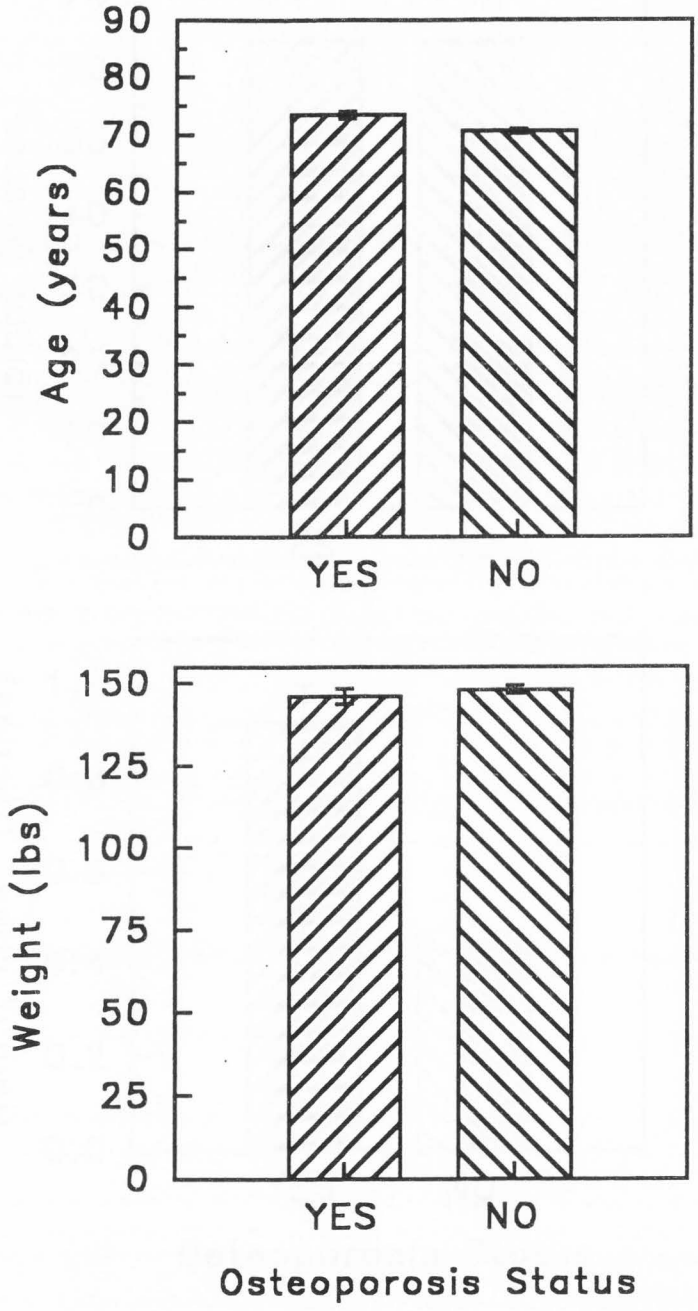

FIG 1. Mean (± 1 S.E.) age and weight of respondents with and without osteoporosis.

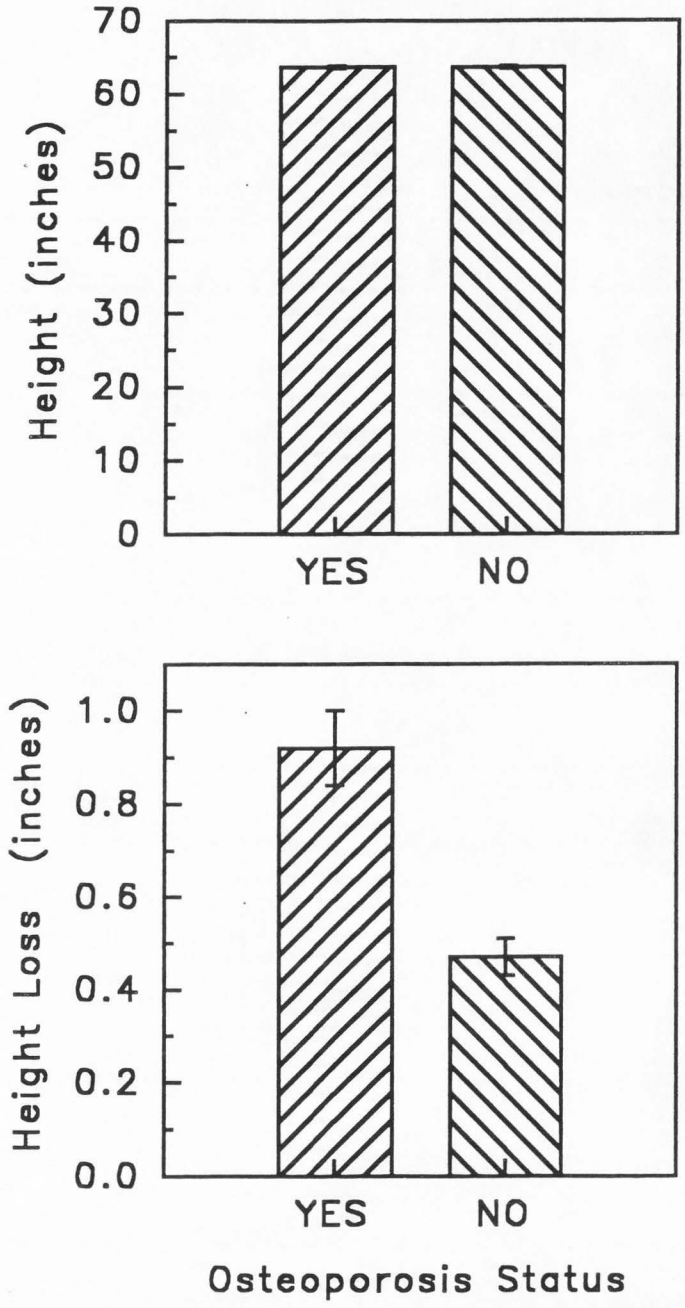

FIG 2. Mean (± 1 S.E.) height of respondents with and without osteoporosis and height loss of respondents from age 21 to present with and without osteoporosis.

Participants were asked to record their current height, as well as their height at age 21. Height loss from early adulthood to current postmenopausal age was calculated. There was a significant difference in height loss between the two osteoporosis groups ($P < 0.01$); however, the two state groups did not differ ($P = 0.67$). Height loss in the non-osteoporotic group was 0.47 inches, while the height loss in the osteoporotic group was 0.92 inches (Figure 2).

Caffeine intake (mg/month) was calculated for Utah and non-Utah respondents with and without osteoporosis. Mean intake of caffeine in non-Utah respondents without osteoporosis was 434 mg/day, while non-Utah respondents with osteoporosis was 389 mg/day. As anticipated, the Utah respondents had a lower mean caffeine intake. The Utah group without osteoporosis had a mean caffeine intake of 206 mg/day, and the osteoporotic group had a mean intake of 145 mg/day. There was a significant difference between caffeine intake and various religions ($P < 0.01$). Catholics had a greater caffeine intake than all other religions, while Mormons had the lowest caffeine intake. Other religions were intermediate in their caffeine intake.

There was no significant difference in dietary calcium intake between Utah and non-Utah ($P = 0.34$, 1014 vs 1010 mg/day) and between osteoporotic and non-osteoporotic ($P = 0.39$, 1047 vs 999 mg/day) respondents. There was also no significant difference in calcium intake between various races ($P = 0.27$).

The total number of children breastfed and the duration of breastfeeding for each participant was calculated. There was a marginal difference in number of children breastfed between osteoporotic and non-osteoporotic participants ($P = 0.052$, 1.6 vs 1.9 children).

There was a significant difference in alcohol servings between Utah and non-Utah respondents ($P < 0.01$, 1.13 vs 0.81) and between religious preferences ($P < 0.01$). Respondents who indicated their preference as "other" consumed more alcohol than those who were LDS, and those who indicated no religious preference consumed more than those who were LDS.

For exercise, there was a significant interaction between osteoporosis and state ($P = 0.04$). For the Utah population, the osteoporotic group exercised more than the non-osteoporotic group (113 vs 93 minutes/month), whereas the reverse was true for the non-Utah population (63 vs 83 minutes/month).

The probability of postmenopausal women having osteoporosis was related significantly to the following factors: arthritis, age, cortisone, fluoridated water, and race (Table 1). The model successfully predicted the occurrence of an osteoporotic event in 72% of the cases. A woman between the ages of 50-59 has approximately a 7% probability of having osteoporosis. This increases to about 18% by the eighth decade of life, or approximately 2.5-fold

Table 1.

The best-fit logit model for the occurrence of osteoporosis (O_Y = osteoporotic; O_N = non-osteoporotic) and the maximum likelihood estimates for the significant explanatory variables.

$$\log\left(\frac{O_Y}{O_N}\right) = -6.64 + 0.37F + 1.13C + 1.24A + 0.04AC - 0.56R$$

Explanatory Variable	Parameter Estimate	Chi-square	P > Chi-square
Intercept	-6.64	43.57	0.0001
Fluoride in Water (F)	0.37	8.89	0.0029
Cortisone (C)	1.13	12.76	0.0004
Arthritis (A)	1.24	35.13	0.0001
Age Class (AC)	0.04	11.96	0.0005
Race (R)	-0.56	3.93	0.0474

increase risk of osteoporosis (Figure 3). If a woman between the ages of 50-59 also has arthritis, her risk for having osteoporosis increases to 20%. By the time a woman is 80 years old and has arthritis, her risk of having osteoporosis increases to 43%. Adding cortisone to the previous risk factors of age and arthritis nearly doubles the probability of having osteoporosis in each age category. Adding fluoride to the model does not cause a dramatic increase in the probability of having osteoporosis; however, there is a 7% increase by age 80.

Geographic region (Utah vs. non-Utah) was not a significant explanatory variable in the logistic regression. Therefore, the region in which people lived did not contribute to the likelihood of having osteoporosis. Dietary factors, including dietary and supplemental calcium, were not significantly related to osteoporosis in this study.

Osteoporotic risk by race is illustrated in Figure 4. As age class increases among Caucasian as well as Asian races, the probability of having osteoporosis increases. The Asian population studied was small (n = 23) in comparison to the Caucasian population. In the age class of 50-59 years among Caucasians, there is approximately a 7% probability of having osteoporosis. This increases to 18% in the eighth decade of life, or again, approximately a 2.5-fold increase risk of having osteoporosis. For Asians, at ages 60-69 there is only a 1% probability of having osteoporosis, which increases to a 2% probability by age 80.

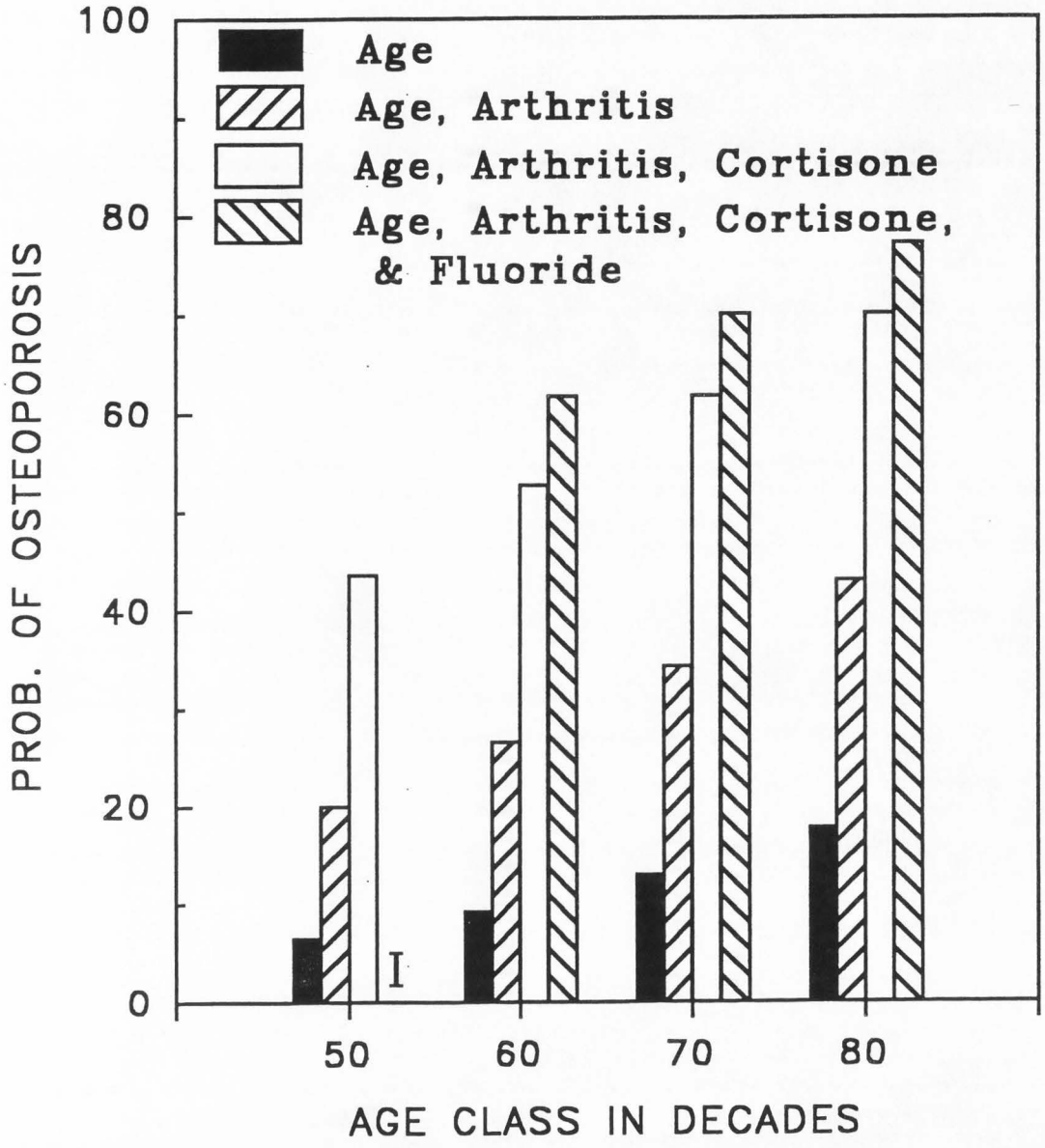

FIG 3. The probability of having osteoporosis with increasing age and with the cumulative effect of three risk factors (arthritis, cortisone, and fluoride). Columns represented by the letter I indicate insufficient numbers of individuals to estimate the probability of that category.

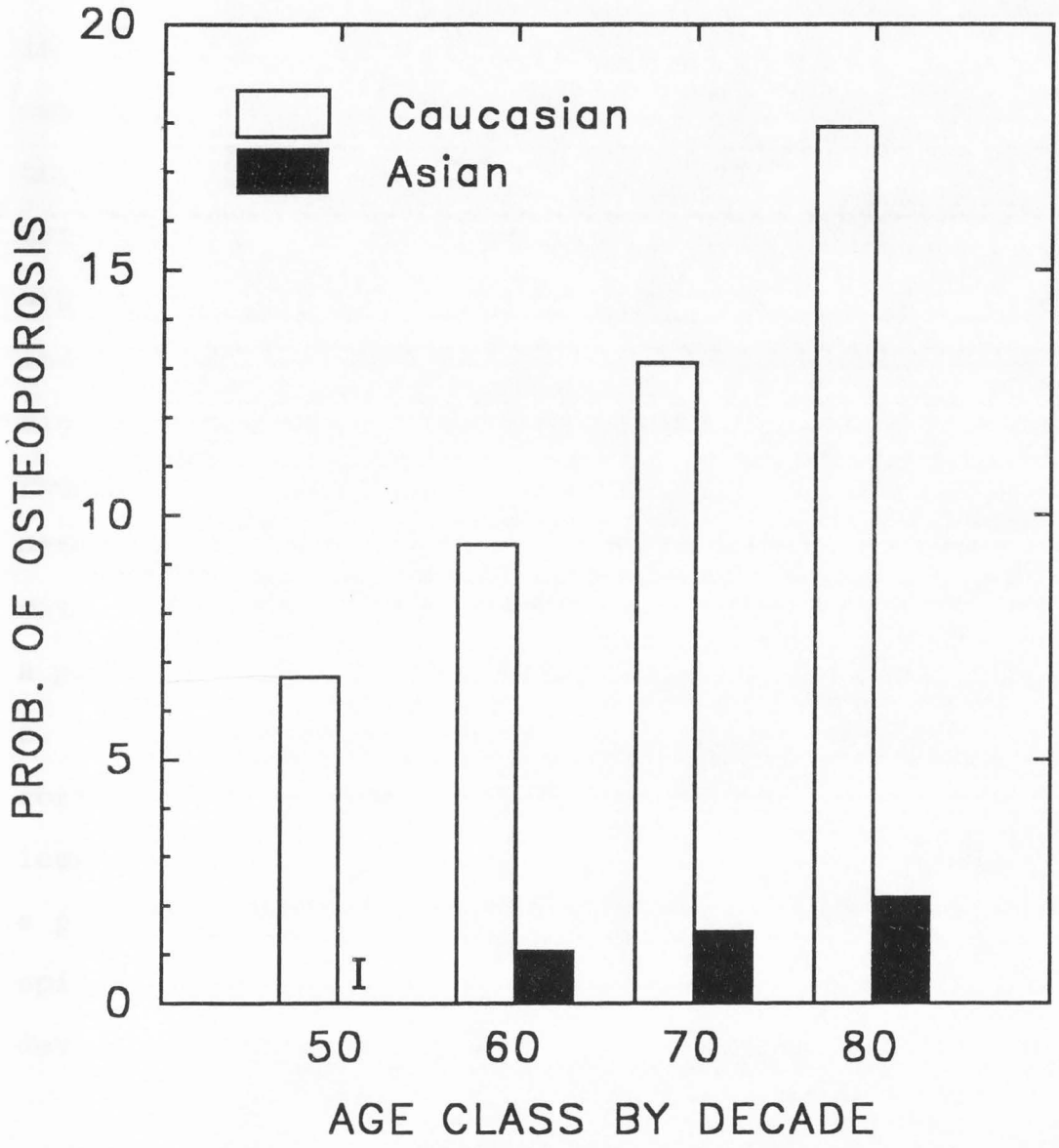

FIG 4. The probability of having osteoporosis with regard to race and increasing age. Columns represented by the letter I indicate insufficient numbers of individuals to estimate the probability of that category.

DISCUSSION

Osteoporosis is a disease primarily of the elderly and is rarely found in women prior to menopause. For osteoporosis to be present in a younger person, there must be unusual circumstances such as surgical menopause with no estrogen replacement therapy, Cushing's syndrome, excessive exposure to glucocorticoids, or bone loss from exercise-induced amenorrhea (7, 95). Age was the only factor significant in both analysis of variance and logistic regression. Because age-related bone loss is universal, much attention has been focused on attaining optimum peak bone mass during youth. One author stated that "osteoporosis is a pediatric disorder that presents to the geriatrician" (80, p. 45). Age-related bone loss begins around age 40 for cortical bone and 5-10 years earlier for trabecular bone loss; this continues into extreme old age (4). Age is such a powerful predictor of bone mass that bone density (lumbar spine or femoral neck) can be predicted with a standard deviation of only 10%, simply from a woman's age (4).

With increasing age, there is usually an increased propensity to fall. This may be due to a number of reasons including failing vision, arthritis and other diseases, drugs, and slower reflexes. Most authorities consider prevention of osteoporosis in terms of exercise, adequate calcium intake, and estrogen replacement therapy. Innovative

strategies for preventing falls in our increasing elderly population should also become part of prevention planning.

Both Caucasian and Asian races have been cited by numerous authors as risk factors for osteoporosis (eg, 3, 7, 8, 55). Caucasians have the greatest risk of all races; however, it has been noted that Asians have less cortical bone mass than whites (96). Nordin (23)did a worldwide survey of osteoporosis and found a significant relationship between osteoporotic radiographs of the spine and low calcium intake. In Japan, where calcium intake is low, he found spinal osteoporosis to be very common. However, hip fractures were much less common and only constituted 0.1 % of all admissions to the university hospital in Tokyo during a six-year period. The hip fracture rate per 100,000 women for the United States is 295.0, whereas for Hong Kong it is 72.4, and for the Chinese in Singapore, it is 50.3 (55). When dealing with race as a risk factor, environmental and dietary factors can be confounding. Although the Japanese and other Asian populations have lower incidence of hip fracture than Caucasian populations, lifestyle factors must be accounted for. Traditional Japanese women live close to the floor and frequently squat, rise, and sit again; they generally do not work outside the home and spend more time in traditional work within the home. The lower hip fracture rate is thought to result from the increased sitting and rising activities and the high incidence of vertebral osteoporosis from low calcium intake (55). As seen in Figure 4, Caucasians had a much

greater probability of having osteoporosis than did Asians in our survey. There was a desire in this study to evaluate more racial groups; however, the random sampling was 92-97% Caucasian with only 4% Asian. The Asian group was found primarily in one senior citizen center in Utah. Although the proportions of the race classifications are similar to those in the general public in these states, caution in interpreting these results must be used due to the small sample size of Asians.

Glucocorticoid use is associated with decreased intestinal calcium absorption and increased urinary calcium loss, diminished bone formation, and increased bone resorption (78, 97). Long-term use of glucocorticoids leads to osteoporosis. Chronic corticosteroid use is mentioned as a risk factor by numerous investigators (5, 7, 8, 78, 80). The results in this study confirm those of several authors who have demonstrated an increased risk of osteoporosis with the use of corticosteroids (78, 98, 99). Arthritis is also cited as a risk factor for osteoporosis by some investigators (e.g. 7, 8, 80). However, the precise mechanism involved is not known. Often people suffering from arthritis have limited ability to exercise and therefore have a sedentary lifestyle which is associated with bone loss. The data from the National Health and Nutrition Examination Survey (NHANES 1) shows a high prevalence of musculoskeletal impairments, including all types of arthritis, among elderly persons (100). The survey also found rheumatoid arthritis to be more

prevalent among women and among Whites compared to non-Whites. Cortisone is often prescribed for arthritis. Therefore, it is reasonable to suggest a relationship among women in their incidence of arthritis, use of cortisone, race, and age. All of these risk factors were significant in our survey.

Although fluoride did not add as much to the model as other factors, it was still significant. Since fluoride is generally thought of as therapeutic treatment for osteoporosis, one might conclude the results from this study are contrary to existing literature. However, recent research has mixed reviews of the therapeutic role fluoride plays in osteoporosis. Fluoride has been evaluated more widely than any other formation-stimulating regimen, yet its use in treatment of osteoporosis is very controversial in the United States and has not yet received United States Food and Drug Administration approval for use in osteoporosis. Pak (65) concluded there are several problems related to fluoride treatment for osteoporosis. Its narrow therapeutic window has made it difficult to maintain appropriate blood fluoride levels without exceeding the toxic threshold. The bone created may be mechanically defective, and fluoride in high concentrations may actually be toxic to osteoblasts. Another problem is that 25-30% of patients may not respond to fluoride treatment. In this survey, we assessed only whether the participant drank fluoridated water or not. Levels of fluoridation in the water supplies were not assessed;

however, we do know that some areas in Colorado and North Dakota have high fluoride content and that fluoridation in Utah is nearly nonexistent. Studies evaluating incidence of fracture and level of fluoridation in the water supply have had varying results. Leone et al (101) and Bernstein et al (28) both found decreased incidence of vertebral fracture in areas of high natural fluoridation compared to regions of low fluoridation. A more recent study found similar results in Finland, comparing low and high levels of fluoridation (102). Femoral neck fractures had a lower prevalence in high fluoride areas compared to low fluoride areas. However, Sowers et al (103) compared regions of high and low fluoridation and reported no difference in fracture rate. Likewise, the National Health Interview surveys in 1973 did not find a relationship between fluoride content of drinking water and incidence of hip fracture (104). The role of fluoride in regard to osteoporosis remains an area of further study and debate.

Vertebral fractures in osteoporosis include a combination of compression (collapse of the entire vertebral body), concavity (collapse of the endplates), or wedging (relative loss of anterior height). These can all reflect fracture (13). Definition of a new vertebral fracture requires a 15% reduction in vertebral height; however, changes in shape cannot be assessed in a survey of fracture occurrence such as ours (13). It is thought that by age 80, most white women have had a least one partial vertebral

deformity (8). Trabecular bone is concentrated in the vertebrae and is much more active metabolically and, therefore, more responsive to hormonal changes. In women, the accelerated bone loss due to estrogen deficiency at menopause results in a disproportionate loss of trabecular bone from the vertebrae. Loss of height is one of the symptoms of vertebral fracture (4). Our results show a greater height loss from early adulthood to old age in the osteoporotic group than the non-osteoporotic group. The loss of height in both groups was determined from the respondents' estimate of current height as well as height in their youth. A preferred method of estimating height loss would be actual measurements of participants; however, we believe our results reflect the presence of osteoporosis.

Although there was a significant difference in caffeine intake among religions, caffeine was not a significant factor in the probability of having osteoporosis in this study. Utah has lower mortality rates from some chronic diseases, and the predominance of the Mormon religion and the lifestyle it entails may contribute to this. However, in this case there was no protection from the occurrence of osteoporosis due to a lower intake of caffeine.

There was a marginal difference in the number of children breastfed between the osteoporotic respondents and the non-osteoporotic respondents, however,it was not part of the log linear model. When analysis of variance is used, there is a trend that those who were non-osteoporotic

breastfed more children than those with osteoporosis. These results are consistent with several other studies which indicate that breastfeeding may be a protective factor (11, 30, 55).

The question of whether to supplement calcium intake is frequently posed to health professionals by women of all ages. Calcium supplements are readily available, and the message that dietary or supplemental calcium may improve one's chances of avoiding osteoporosis has been well covered by the media. None of the sources of calcium were significant factors in this study. The mean intake of calcium in this study was higher than that of the two National Health and Nutrition Examination Surveys (1000 vs 500 mg/day) for women age 50 and older. Several investigators have found the food frequency to be the method of choice for a questionnaire or when assessing one nutrient in the diet (105, 106, 107). Food frequency questionnaires can be used to determine habitual intake and are especially applicable in relating diet to long-term effects (107). They are low in cost as no specialized training or equipment is needed to administer them. This method is limited due to difficulty in validation, as they cover longer periods of time than food diaries or recall methods (105). Food frequencies may overestimate actual intake (106, 107), and this may explain in part the high calcium intake calculated in this study. Current calcium intake may not correlate well with fracture risk, and previous intake can be difficult to

obtain with any accuracy. Our participants were free-living adults who may have changed their eating patterns with aging. Diets often simplify as family size decreases, or with the death of a spouse. Economic status may change with retirement, and physical ailments may increase. These can all affect food choices and preparation. We attempted to evaluate the usual intake of calcium, which included listing foods high in calcium as well as frequently eaten foods (cottage cheese, bread, milk in coffee) that contribute to the overall calcium content of the diet. Bone health as well as bone fragility is a complex issue, of which calcium is just a part. Calcium may exert more influence on peak bone mass achieved in youth than loss of bone mass in old age (108). Current bone health is the result of diet and lifestyle events that occurred decades earlier; therefore, the link of current nutritional practices to osteoporosis is difficult to establish.

Summary and Recommendations

The participants in this study were evaluated by mailout questionnaire, and their dietary, physical, and demographic characteristics were related to the incidence of osteoporotic fracture. Age, race, arthritis, cortisone use, and fluoridation of water were significantly related to the probability of having osteoporosis.

Very few differences were found between the two female populations studied. The Utah population exercised more and

consumed less caffeine than the non-Utah population; however, these factors were not significantly related to the occurrence of osteoporosis in this study.

There were certain limitations of this study which may have affected the outcome. This was not a random sample of elderly postmenopausal women. The participants represented the population most likely to participate at senior citizen centers. Although the racial demographics were similar to the general population, some groups such as Native Americans and Blacks were underrepresented and may indicate lack of participation at these centers. The segment of the population with severe or debilitating osteoporosis or other impairments were underrepresented, as most of these potential participants are either housebound to some extent, or are in long-term care facilities.

The return rate of this survey was acceptable; however, the non-respondents may have altered the results if they had chosen to participate.

Verification of the presence of osteoporosis by quantified computer tomography would enhance the use of this questionnaire.

Treatment and prevention of osteoporosis is advancing, but still needs improvement. Research needs to be done to evaluate the interaction among risk factors such as age, race, arthritis, and medications. This study indicates that treatments for other conditions, such as arthritis, may increase the risk for osteoporosis. We may need to further

evaluate the therapeutic dosage of drugs and nutrients, such as cortisone and fluoride, as well as others, to improve the risk relationship between them and osteoporosis.

REFERENCES

1. Chesnut CH. Treatment of postmenopausal osteoporosis. *Comprehensive Therapy*. 1984; 10:41-47.

2. Matkovic V, Fontana D, Tominac C, Goel P, Chesnut CH. Factors that influence peak bone mass formation: a study of calcium balance and the inheritance of bone mass in adolescent females. *Am J Clin Nutr*. 1990; 52:878-888.

3. Christiansen C. Consensus development conference: prophylaxis and treatment of osteoporosis. *Am J Med*. 1991; 90:107-110.

4. Riggs BL. Overview of osteoporosis. *West J Med*. 1991; 154:63-77.

5. Escher JE, Gambert SR. Osteoporosis: approaches to management. *The Female Patient*. 1989; 14:59-65.

6. Spector TD, Huskisson EC. A rational approach to the prevention and treatment of postmenopausal osteoporosis. *Drugs*. 1989; 37:205-211.

7. Bilezikian JP, Shane E. Osteoporosis: an update on risk factors and treatment options. *The Female Patient*. 1988; 13:31-45.

8. Cummings SR, Kelsey JL, Nevitt MC, O'Dowd KJ. Epidemiology of osteoporosis and osteoporotic fractures. *Epidemiol Rev*. 1985; 7:178-207.

9. Johnston CC. Osteoporosis- extent and cause of the disease. *Proceedings of the Society for Experimental Biology and Medicine*. 1989; 191:258-260.

10. Cummings SR. Epidemiology of osteoporotic fractures:selected topics. In:Roche AF, ed. *Osteoporosis: Current Concepts Report of the Seventh Ross Conference on Medical Research*. Columbus, Ohio:Ross Laboratories; 1987:3-8.

11. Kreiger N, Kelsey JL, Holford TR, O'Connor T. An epidemiologic study of hip fracture in postmenopausal women. *J Epidemiol*. 1982; 116:141-147.

12. Lewinnek GE, Kelsey J, White AA, Kreiger NJ. The significance and a comparative analysis of the epidemiology of hip fractures. *Clin Orthop Rel Res*. 1980; 152:35-43.

13. Melton JL, Kan SH, Frye MA, Wahner HW, O'Fallon WM, Riggs BL. Epidemiology of vertebral fractures in women. *Am J Epidemiol*. 1989; 129:1000-1011.

14. Hemenway D, Colditz GA, Willett WC, Stampfer MJ, Speizer FE. Fractures and lifestyle: effect of cigarette smoking, alcohol intake, and relative weight on the risk of hip and forearm fractures in middle-aged women. *Am J Public Health.* 1988; 78:1554-1558.

15. Mazess RB, Barden HS. Bone density in premenopausal women:effects of age, dietary intake, physical activity, smoking, and birth-control pills. *Am J Clin Nutr.* 1991; 53:132-142.

16. Hernandez-Avila M, Colditz GA, Stampfer MJ, Rosner B, Speizer FE, Willett WC. Caffeine, moderate alcohol intake, and risk of fractures of the hip and forearm in middle-aged women. *Am J Clin Nutr.* 1991; 54:157-163.

17. VanHemert AM, Vandenbroucke JP, Birkenhager JC, Valkenburg HA. Prediction of osteoporotic fractures in the general population by a fracture risk score. *Am J Epidemiol.* 1990; 132:123-135.

18. Kiel DP, Felson DT, Hannan MT, Anderson JJ, Wilson PW. Caffeine and the risk of hip fracture: the Framingham study. *Am J Epidemiol.* 1990; 132:675-684.

19. Wong PCN. Fracture epidemiology in a mixed southeastern asian community (Singapore). *Clin Orthop.* 1966; 45:55-61.

20. Solomon L. Osteoporosis and fracture of the femoral neck in the South African Bantu. *J Bone Joint Surg.* 1968; 50:2-11.

21. Smith RW, Rizek J. Epidemiologic studies of osteoporosis in women of Puerto Rico and Southeastern Michigan with special reference to age, race, national origin and to other related or associated findings. *Clin Orthop.* 1966; 45:31-47.

22. Farmer ME, White LR, Brody JA, Bailey KR. Race and sex differences in hip fracture incidence. *Am J Public Health.* 1984; 74:1374-1380.

23. Nordin BEC. International patterns of osteoporosis. *Clin Orthop.* 1966; 45:17-30.

24. Owen RA, Melton LJ, Johnson KA, Ilstrup DM, Riggs BL. Incidence of Colles' fracture in a North American community. *Am J Public Health.* 1982; 72:605-607.

25. Chalmers J, Ho KC. Geographical variations in senile osteoporosis. *J Bone Joint Surg.* 1970; 52:667-673.

26. Matkovic V, Kostial K, Simonovic I, Buzina R, Brodarec A, Nordin BEC. Bone status and fracture rates in two regions of Yugoslavia. *Am J Clin Nutr.* 1979; 32:540-549.

27. Alffram PA, Hernborg J, Nilsson BER. The influence of a high fluoride content in the drinking water on the bone mineral mass in man. *Acta Orthop Scandinav.* 1969; 40:137-142.

28. Bernstein DS, Sadowsky N, Hegsted DM, Guri CD, Stare FJ. Prevalence of osteoporosis in high- and low-fluoride areas in North Dakota. *JAMA.* 1966; 198:85-90.

29. Alderman BW, Weiss NS, Daling JR, Urr CL, Ballard JH. Reproductive history and postmenopausal risk of hip and forearm fracture. *Am J Epidemiol.* 1986; Vol:262-267.

30. Aloia JF, Vaswani AN, Yeh JK, Ross P, Ellis K, Cohn SH. Determinants of bone mass in postmenopausal women. *Arch Intern Med.* 1983; 143:1700-1704.

31. Dhuper S, Warren MP, Brooks-Gunn J, Fox R. Effects of hormonal status on bone density in adolescent girls. *J Clin Endocrin Metab.* 1990; 71:1083-1088.

32. Brewer V, Meyer BM, Keele MS, Upton J, Hagan RD. Role of exercise in prevention of involutional bone loss. *Med Sci Sports Exerc.* 1983; 15:445-449.

33. Heinrich CH, Going SB, Pamenterr RW, Perry CD, Boyden TW, Lohman TG. Bone mineral content of cyclically menstruating female resistance and endurance trained athletes. *Med Sci Sports Exerc.* 1990; 22:558-563.

34. Draper J,, Roland M. Perimenopausal women's views on taking hormone replacement therapy to prevent osteoporosis. *Br Med J.* 1990; 300:786-788.

35. Winner SJ, Morgan CA, Evans JG. Perimenopausal risk of falling and incidence of distal forearm fracture. *Br Med J.* 1989; 298:1486-1488.

36. Albright F, Smith PH, Richardson AM. Postmenopausal osteoporosis: its clinical features. *JAMA.* 1941; 116:2465-2474.

37. Avioli LV. *The Osteoporotic Syndrome.* Orlando, FL: Grune and Statton; 1987.

38. Allen LH. Calcium and age related bone loss. *Clin Nutr.* 1986; 5:147-152.

39. Trotter M, Broman GE, Peterson RR. Densities of bones of white and negro skeletons. *J Bone Joint Surg.* 1960; 42A:50-58.

40. Bollett AJ, Engh G, Parson W. Epidemiology of osteoporosis. *Arch Intern Med.* 1965; 16:191-194.

41. Hegsted DM. Calcium and osteoporosis. *J Nutr.* 1986; 116:2316-2319.

42. Levine S, Makin M, Menczel J, Robin G, Naor E, Steinberg R. Incidence of fractures of the proximal end of the femur in Jerusalem. *J Bone Joint Surg.* 1970; 52A:1193-1196.

43. Mazess RB, Mather W. Bone mineral content of North Alaskan eskimos. *Am J Clin Nutr.* 1974; 27:916-925.

44. Anderson JJB. Dietary calcium and bone mass through the lifecycle. *Nutrition Today.* 1990; 25:9-14.

45. Food and Nutrition Board. *Recommended Dietary Allowances.* 10th ed. Washington D.C.: National Academy of Sciences; 1989.

46. Anonymous. Osteoporosis:consensus conference. *JAMA.* 1984; 252:799-802.

47. Heaney RP, Gallagher JC, Johnston CC, Neer R, Parfitt AM, Chir B, Whedon GD. Calcium nutrition and bone health in the elderly. *Am J Clin Nutr.* 1982; 36:986-1013.

48. Heaney RP, Recker RR, Saville PD. Menopausal changes in calcium balance performance. *J Lab Clin Med.* 1978; 92:953-963.

49. Spencer H, Kramer L. Osteoporosis, calcium requirements and factors causing calcium loss. *Clin Ger Med.* 1987; 3:389-402.

50. Horsman A, Gallagher JC, Simpson M. Prospective trial of oestrogen and calcium in postmenopausal women. *Br Med J.* 1977; 2:789-792.

51. Riis B, Thomsen K, Christiansen C. Does calcium supplementation prevent postmenopausal bone loss? *N Engl J Med.* 1987; 316:173-177.

52. Dawson-Hughes B. Calcium supplementation and bone loss: a review of controlled clinical trials. *Am J Clin Nutr.* 1991; 54:274S-280S.

53. Riis B, Thomen K, Christiansen C. Does calcium supplementation prevent postmenopausal bone loss? A double-blind, controlled clinical study. *N Engl J Med.* 1987; 316:173-177.

54. Dawson-Hughes B, Dallal G, Tannenbaum S, Sahyoun N, Krall E. Effect of calcium supplements on postmenopausal bone loss. *J Bone Miner Res.* 1989; 4(suppl):1091.

55. Pollitzer WS, Anderson JJB. Ethnic and genetic differences in bone mass: a review with a heredity vs environmental perspective. *Am J Clin Nutr.* 1989; 50:1244-1259.

56. Matkovic V. Calcium metabolism and calcium requirements during skeletal modeling and consolidation of bone mass. *Am J Clin Nutr.* 1991; 54:245S-260S.

57. Parfitt AM, Chir B, Gallagher JC, Heaney RP, Johnston CC, Neer R, Whedon GD. Vitamin D and bone health in the elderly. *Am J Clin Nutr.* 1982; 36:1014-1031.

58. Riis BJ, Thomsen K, Christiansen C. Does 24R,25(OH)2-Vitamin D3 prevent postmenopausal bone loss? *Calcif Tissue Int.* 1986; 39:128-132.

59. Riggs BL, Nelson KI. Effect of long term treatment with calcitriol on calcium absorption and mineral metabolism in postmenopausal osteoporosis. *J Clin Endocrin Met.* 1985; 61:1676-1684.

60. Gallagher JC, Riggs,BL, Recker RR, Goldgar D. The effect of calcitriol on patients with postmenopausal osteoporosis with special reference to fracture frequency. *Proc Soc Exp Biol Med.* 1989; 191:287-292.

61. Bikle DD, Halloram BP, McGalliard-Cone C, Morey-Holton E. Different responses of trabecular and cortical bone to 1,25(OH)2 D3 infusion. *Am J Physiol.* 1990; 259:E715-722.

62. Jackson TK, Ullrich IH. Understanding osteoporosis: cornerstone of prevention and treatment. *Postgrad Med.* 1984; 75:119-125.

63. Iskrant AP. The etiology of fractured hips in females. *Am J Pub Health.* 1968; 58:485-490.

64. Alhava EM, Olkkonen H, Kauranen P. The effect of drinking water fluoridation on the fluoride content, strength and mineral density of human bone. *Acta Orthop Scand.* 1989; 51:413-420.

65. Pak CY. Fluoride and osteoporosis. *Soc Exp Biol Med.* 1989; 37:278-286.

66. Riggs BL, Hodgson SF, Wahner HW, Muhs J, O'Fallon WM. Effect on long-term fluoride therapy in bone metabolism, bone density, and fracture rate in type 1 (postmenopausal) osteoporosis: Final results of prospective, controlled, double-blind clinical trial. *J Bone Miner Res.* 1989; 4:S418-S422.

67. Oyster N, Morton M, Linell S. Physical activity and osteoporosis in postmenopausal women. *Med Sci Sports Exerc.* 1984; 16:44-50.

68. Pocock NA, Eisman JA, Yeates MG, Sambrook PN, Eberl S. Physical fitness is a major determinant of femoral neck and lumbar spine bone mineral density. *J Clin Invest.* 1986; 78:618-621.

69. Williams AR, Weiss NS, Ure CL, Ballard J, Daling JR. Effect of weight, smoking, and estrogen use on the risk of hip and forearm fractures in postmenopausal women. *Obst and Gyn.* 1982; 6:695-698.

70. Daniell HW. Osteoporosis of the slender smoker. *Arch Intern Med.* 1976; 136:298-304.

71. Seeman E, Melton LJ, O'Fallon WM, Riggs BL. Risk factors for spinal osteoporosis in men. *Am J Med.* 1983; 75:977-983.

72. Bikle D, Genart HK, Cann C, Recker RR, Halloran B, Strewler GJ. Bone disease in alcohol abuse. *Ann Intern Med.* 1985; 103:42-48.

73. Massey LK, Wise KJ. The effect of dietary caffeine on urinary excretion of calcium, magnesium, sodium and potassium in healthy young females. *Nutr Res.* 1984; 4:43-50.

74. Massey LK, Berg TA. The effect of dietary caffeine on urinary excretion of calcium, magnesium, phosphorus, sodium, potassium, chloride and zinc in healthy males. *Nutr Res.* 1985; 5:1281-1285.

75. Heaney RP, Recker RR. Effects of nitrogen, phosphorus, and caffeine on calcium balance in women. *J Lab Clin Med.* 1982; 99:46-55.

76. Avioli LV. Glucocorticoids in medicine. In: Roche AF, ed. *Osteoporosis: Current Concepts Report of the Seventh Ross Conference on Medical Research.* Columbus, Ohio: Ross Laboratories; 1987:33-35.

77. Wyshak G. Hip fracture in elderly women and reproductive history. *J Gerontology*. 1981; 36:424-427.

78. Reid R, Heap SW. Determinants of vertebral mineral density in patients receiving long-term glucocorticoid therapy. *Arch Intern Med*. 1990; 150:2545-2548.

79. Ettinger B. Overview of the efficacy of hormonal replacement therapy. *Am J Obstet Gynecol*. 1987; 156:1298-1303.

80. Lindsay R. Osteoporosis: an updated approach to prevention and management. *Geriatrics*. 1989; 44:45-54.

81. Ettinger B, Genant HK, Cann CE. Long-term estrogen therapy prevents bone loss and fractures. *Ann Int Med*. 1985; 102:319-324.

82. Horsman A, Jones M, Francis R, Nordin BE. The effect of estrogen dose on postmenopausal bone loss. *N Engl J Med*. 1983; 309:1404-1407.

83. Sowers MR, Wallace RB, Lemke JH. Correlates of mid-radius bone density among postmenopausal women: a community study. *Am J Clin Nutr*. 1985; 41:1045-1053.

84. Smith DM, Nance WE, Kang KW, Christian JC, Johnston CC. Genetic factors in determining bone mass. *J Clin Invest*. 1973; 52:2800-2808.

85. Lutz J, Tesar R. Mother-daughter pairs: spinal and femoral bone densities and dietary intakes. *Am J Clin Nutr*. 1990; 52:872-877.

86. Beeson WL, Mills PK, Phillips RL, Fraser GE. Chronic disease among Seventh-Day Adventists, a low risk group: rationale, methodology and description of the population. *Cancer*. 1989; 64:570-581.

87. Phillips RL, Snowden DA. Dietary relationships with fatal colorectal cancer among Seventh-Day Adventists. *JNCI*. 1985; 74:307-317.

88. Snowden DA, Phillips RL. Does a vegetarian diet reduce the occurrence of diabetes? *Am J Pub Health*. 1985; 75:507-512.

89. MMWR. Chronic disease reports: coronary heart disease mortality- United States, 1986. *Morbidity and Mortality Weekly Report*. 1989; 38:285-288.

90. MMWR. Behavioral risk-factor surveillance- selected states, 1984. *Morbidity and Mortality Weekly Report.* 1989; 35:353-354.

91. Lauritzen G. Unpublished data.

92. Dillman DA. *Mail and Telephone Surveys: the Total Design Method.* New York, NY: John Wiley & sons, Inc.; 1978.

93. Converse JM, Presser S. *Survey Questions: Handcrafting the Standardized Questionnaire.* Beverly Hills, CA: Sage Publications, Inc.; 1986.

94. Fienberg SE. *The Analysis of Cross-Classified Categorical Data.* 2nd ed. Cambridge, Mass: MIT Press; 1985.

95. Kelly JB, Hatfield SL. Menstrual irregularities and bone loss in female athletes. *The Female Patient.* 1989; 14:35-39.

96. Garn SM, Pao EM, Rihl ME. Compact bone in Chinese and Japanese. *Science.* 1964; 143:1439-1440.

97. Reid IR, Ibbertson HK. Calcium supplements in the prevention of steroid-induced osteoporosis. *Am J Clin Nutr.* 1986; 44:287-290.

98. LoCascio V, Bonucci E, Imbimbo B. Bone loss after glucocorticoid therapy. *Calcif Tissue Int.* 1984; 36:435-439.

99. Adinoff AD, Hollister JR. Steroid-induced fractures and bone loss in patients with asthma. *N Engl J Med.* 1983; 309:265-268.

100. Cunningham LS, Kelsey JL. Epidemiology of musculoskeletal impairments and associated disability. *Am J Pub Health.* 1984; 74:574-579.

101. Leone NC, Stevenson CA, Hilbish TF, Sosman MC. A roentgenologic study of a human population exposed to high-fluoride domestic water: a ten year study. *Am J Roentgen.* 1955; 74:874-885.

102. Simonen O, Laitinen O. Does fluoridation of drinking water prevent bone fragility and osteoporosis. *Lancet.* 1985; 2:432-434.

103. Sowers MFR, Wallace RB, Lemke JH. The relationship of bone mass and fracture history to fluoride and calcium intake: a study of three communities. *Am J Clin Nutr.* 1986; 44:889-898.

104. Madans J, Kleinman JC, Cornoni-Huntley J. The relationship between hip fracture and water fluoridation: an analysis of national data. *Pub Health Briefs*. 1983; 73:296-298.

105. Woteki, CE. Dietary survey data: sources and limits to interpretation. *Nutr Rev*. 1986; May supplement:204-213.

106. Barrett-Connor, E. Nutrition epidemiology: how do we know what they ate? *Am J Clin Nutr*. 1991; 54:182S-187S.

107. Zulkifli SN, Yu SM. The food frequency method for dietary assessment. *J Am Diet Assoc*. 1992; 92:681-685.

108. Heaney RP. Nutritional factors in bone health in elderly subjects: methodological and contextual problems. *Am J Clin Nutr*. 1989; 1182-1189.

APPENDICES

Appendix A

Questionnaire used in collecting data for this study.

FIRST, WE WOULD LIKE YOU TO ANSWER A FEW QUESTIONS ABOUT OSTEOPOROSIS, BONE LOSS AND YOUR LIFESTYLE.

1. Have you ever been told by a doctor that you have osteoporosis? (circle number)
   1. NO
   2. YES

2. Do you know if your mother had osteoporosis? (circle number)
   1. NO
   2. YES
   3. DO NOT KNOW

3. Have you ever had any fractured or broken bones as an adult? (circle number)
   1. NO (If NO, go to question 4)
   2. YES

   → 3a. If YES, where was the fracture or broken bone and how old were you at the time? (circle all that apply)

	AGE
1. HIP	_____
2. WRIST or FOREARM	_____
3. SPINE	_____
4. OTHER(S) (please state body location)	_____

_____

4. Some people take vitamin and/or mineral supplements. Please check the supplements you use, give the brand name if you know it, and how often you use them.

Supplement	Brand Name	Daily	Weekly	Monthly
Calcium				
Vitamin D				
Fluoride				
Vitamin A				
Multi-Vitamin/ Mineral Tablet				

NEXT, WE WOULD LIKE TO ASK ABOUT YOUR DIET AND EATING HABITS.

5. How often do you drink milk? (circle number)

   1. NEVER
   2. MONTHLY (1 to 3 days a month)
   3. WEEKLY (1 to 4 days a week)
   4. DAILY (5 to 7 days a week)

   → 5a. If you drink milk DAILY, how many cups (8 ounces) do you usually have?

   _____CUPS EACH DAY

6. How often do you eat yogurt? (circle number)

   1. NEVER
   2. MONTHLY (1 to 3 days a month)
   3. WEEKLY (1 to 4 days a week)
   4. DAILY (5 to 7 days a week)

   → 6a. If you eat yogurt DAILY, how many cartons (6-8 ounces) do you usually have?

   _____CARTONS EACH DAY

7. How often do you eat hard cheeses, such as Cheddar, Colby or American? (circle number)

   1. NEVER
   2. MONTHLY (1 to 3 days a month)
   3. WEEKLY (1 to 4 days a week)
   4. DAILY (5 to 7 days a week)

   → 7a. If you eat cheese DAILY, how many thin slices (1 ounce) do you usually eat?

   _____SLICES EACH DAY

8. How often do you eat cottage cheese?

   1. NEVER
   2. MONTHLY (1 to 3 days a month)
   3. WEEKLY (1 to 4 days a week)
   4. DAILY (5 to 7 days a week)

8a. If you eat cottage cheese DAILY, how many (1/2 cup) servings do you usually have?

_____SERVINGS EACH DAY

9. How often do you you eat ice cream or frozen yogurt?

1. NEVER
2. MONTHLY (1 to 3 days a month)
3. WEEKLY (1 to 4 days a week)
4. DAILY (5 to 7 days a week)

→9a. If you eat ice cream or frozen yogurt DAILY, how many scoops (1/2 cup) do you usually have?

_____SCOOPS EACH DAY

10. How many slices of bread (any kind) do you usually eat each day?

_____SLICES EACH DAY

11. How many times each week do you eat cereal WITH MILK?

_____TIMES EACH WEEK

12. Please tell us how much and how often you drink the following beverages: (If you drink none of these, go to question 14)

BEVERAGE	DAILY	WEEKLY	MONTHLY	NEVER (✓)
Example: TEA	2 cups			
COFFEE				
TEA				
COLA BEVERAGES				
ALCOHOL				

13. Do you take milk (not cream or other substitute) in your coffee or tea? (circle number)

1. NO
2. YES

14. Do you drink fluoridated water? (circle number)

1. NO
2. YES
3. DO NOT KNOW

NOW WE WOULD LIKE YOU TO GIVE US SOME INFORMATION ABOUT YOUR MEDICAL HISTORY.

15. Please indicate if you regularly take or have taken any of the following medication: (circle all that apply)

1. DIURETICS (Water pills or blood pressure pills)
2. CORTISONE (Prednisone or Cortisol)
3. ISONIAZID (For treatment of tuberculosis)
4. ANTICONVULSANTS (Dilantin, Phenobarbitol or Mysoline)
5. COUMADIN (Anticoagulant or blood thinner)
6. THYROID MEDICINE

16. Please circle any of the following antacids you take regularly (more than once a week).

1. TUMS
2. ROLAIDS
3. MAALOX
4. MYLANTA
5. AMPHOGEL
6. GELUSIL
7. OTHER (please state name brand)_____

17. Do you smoke cigarettes? (circle number)

1. NO (If NO, go to question 18)
2. YES

→17a. If YES, how much do you smoke? (circle number)

1. LESS THAN 1/2 PACK A DAY
2. 1/2 TO 1 PACK A DAY
3. 1 1/2 TO 2 PACKS A DAY
4. MORE THAN 2 PACKS A DAY

17b. How long have you smoked?

_____YEARS

18 Do you have any of the following: (circle all that apply)

1. KIDNEY DISEASE
2. ARTHRITIS
3. MILK ALLERGY OR LACTOSE INTOLERANCE

19. Please list any surgeries you have had:

20. How many pregnancies have you had? _____

21. How many of your children did you breastfeed?

_____CHILDREN

→21a. If you breastfed any of your children, on the average how long did you breastfeed?

_____MONTHS

22. Have you experienced menopause? (circle number)

1. NO (If NO, go to question 23)
2. YES

→22a. If YES, about how old were you at menopause?

_____YEARS

22b. Do you use estrogen (hormone) replacement therapy? (circle number)
1. NO
2. YES

23. Have you had a hysterectomy? (circle number)

1. NO (If NO, go to question 24)
2. YES

→23a. If YES, how old were you when you had the hystectomy?

_____YEARS

24. Please circle any of the following activities you do. Tell us how many times a week or month you do the activity and about how many minutes you usually spend doing them. Do not include housework or gardening.

ACTIVITY	WEEKLY	MONTHLY	# MINUTES
EXAMPLE:			
WALKING	5 times		20 minutes
1. SWIMMING			
2. JOGGING			
3. WALKING			
4. EXERCISE BIKE/ OR BICYCLING			
5. SKIING/NORDIC TRACK			
6. ROWING/ROWING MACHINE			
7. AEROBICS CLASS			
8. SQUARE DANCING			
9. OTHER:			

FINALLY, WE WOULD LIKE TO ASK A FEW QUESTIONS ABOUT YOURSELF FOR STATISTICAL PURPOSES.

25. What is your age?

_____YEARS

26. What is your weight?

_____POUNDS

27 How tall are you?

_____FEET _____INCHES

28. How tall were you at age 21?

_____FEET _____INCHES

29. What city/town do you live in? _____

30. How long have you lived there?

_____YEARS

31. If less than 5 years, where did you live previously?

CITY_____STATE _____

32. Is your race or ethnic origin: (circle number)

1. CAUCASION/WHITE
2. BLACK
3. LATIN, SPANISH OR MEXICAN AMERICAN
4. AMERICAN INDIAN
5. ASIAN/PACIFIC ISLANDER
6. OTHER: _____

33. Do you have a religious preference? (circle number)

1. NO
2. YES

→33a. If YES, what? (circle number)

1. PROTESTANT
2. CATHOLIC
3. LDS
4. OTHER: _____

YOUR TIME AND EFFORT ON THIS SURVEY IS GREATLY APPRECIATED.

Appendix B

Coding of questionnaire responses used in computer entry.

Question	Item Abbreviation	Code	Response	Frequency
0	IDNUM	1000-1600	UTAH	381
		2000-2200	COLORADO	52
		3000-3200	NORTH DAKOTA	119
		4000-4200	SOUTH DAKOTA	103
1	DR	1	NO	560
		2	YES	89
		8	OTHER	0
		9	NO RESPONSE	6
2	HX	1	NO	583
		2	YES	65
		3	DON'T KNOW	0
		8	OTHER	0
		9	NO RESPONSE	0

Appendix B continued

3	FRACTURE	1	NO	420
		2	YES	270
		8	OTHER	0
		9	NO RESPONSE	25
3A1	HIP	1	NO	647
		2	YES	40
		9	NO RESPONSE	28
3A2	AGE_HIP	00	NO FRACTURE	*
		01-98	AGE	
		99	NO RESPONSE	
3A3	WRIST	1	NO	582
		2	YES	105
		9	NO RESPONSE	28
3A4	AGEWRIST	00	NO FRACTURE	*
		01-98	AGE	
		99	NO RESPONSE	
3A5	SPINE	1	NO	662
		2	YES	25
		9	NO RESPONSE	28
3A6	AGESPINE	00	NO FRACTURE	*
		01-98	AGE	
		99	NO RESPONSE	

Appendix B continued

3A7	OTHERFRC	1	NO	520
		2	YES	167
		9	NO RESPONSE	28
3A8	AGEOTHER	00	NO FRACTURE	*
		01-98	AGE	
		99	NO RESPONSE	
4A	CALCIUM	1	NO	393
		2	YES	262
		9	NO RESPONSE	0
4B	CA_OFTEN	0	NEVER	*
		1	DAILY	
		2	WEEKLY	
		3	MONTHLY	
		8	OTHER	
		9	NO RESPONSE	
4C	VIT_D	1	NO	572
		2	YES	83
		9	NO RESPONSE	0

Appendix B continued

4D	D_OFTEN	0	NEVER	*
		1	DAILY	
		2	WEEKLY	
		3	MONTHLY	
		8	OTHER	
		9	NO RESPONSE	
4E	FLUORIDE	1	NO	690
		2	YES	16
		9	NO RESPONSE	9
4F	FL_OFTEN	0	NEVER	*
		1	DAILY	
		2	WEEKLY	
		3	MONTHLY	
		8	OTHER	
		9	NO RESPONSE	
4G	VIT_A	1	NO	601
		2	YES	54
		9	NO RESPONSE	0

Appendix B continued

4H	A_OFTEN	0	NEVER	*
		1	DAILY	
		2	WEEKLY	
		3	MONTHLY	
		8	OTHER	
		9	NO RESPONSE	
4I	MULTI	1	NO	429
		2	YES	226
		9	NO RESPONSE	0
4J	M_OFTEN	0	NEVER	*
		1	DAILY	
		2	WEEKLY	
		3	MONTHLY	
		8	OTHER	
		9	NO RESPONSE	
5	MILK	1	NEVER	102
		2	MONTHLY	61
		3	WEEKLY	82
		4	DAILY	398
		9	NO RESPONSE	12
5A	MILKCUPS	00-09	CUPS	*
		99	NO RESPONSE	

Appendix B continued

6	YOGURT	1	NEVER	305
		2	MONTHLY	196
		3	WEEKLY	107
		4	DAILY	22
		9	NO RESPONSE	25
6A	YOGCARTS	00-09	CARTONS	*
		99	NO RESPONSE	
7	CHEESE	1	NEVER	78
		2	MONTHLY	262
		3	WEEKLY	267
		4	DAILY	36
		9	NO RESPONSE	12
7A	CHEESESL	00-09	SLICES	*
		99	NO RESPONSE	
8	COT_CH	1	NEVER	68
		2	MONTHLY	366
		3	WEEKLY	190
		4	DAILY	18
		9	NO RESPONSE	13
8A	COT_CHSV	00-09	SERVINGS	*
		99	NO RESPONSE	

Appendix B continued

9	ICE_CR	1	NEVER	68
		2	MONTHLY	307
		3	WEEKLY	215
		4	DAILY	40
		9	NO RESPONSE	25
9A	ICE_CRSC	00-09	SCOOPS	*
		99	NO RESPONSE	
10	BREAD	00-98	SLICES	*
		99	NO RESPONSE	
11	CEREAL	0-7	TIMES/WEEK	*
		9	NO RESPONSE	
12A	COFFEE	1	NO	220
		2	YES	423
		9	NO RESPONSE	12
12B	COFOFTEN	0	NEVER	220
		1	DAILY	402
		2	WEEKLY	13
		3	MONTHLY	8
		9	NO RESPONSE	12
12C	COF_CUPS	00-98	CUPS	*
		99	NO RESPONSE	

Appendix B continued

12D	TEA	1	NO	456
		2	YES	185
		9	NO RESPONSE	14
12E	TEAOFTEN	0	NEVER	456
		1	DAILY	113
		2	WEEKLY	47
		3	MONTHLY	25
		9	NO RESPONSE	14
12F	TEA_CUPS	00-98	CUPS	*
		99	NO RESPONSE	
12G	COLA	1	NO	398
		2	YES	244
		9	NO RESPONSE	13
12H	COLOFTEN	0	NEVER	398
		1	DAILY	113
		2	WEEKLY	90
		3	MONTHLY	41
		9	NO RESPONSE	13
12I	COLA_CAN	00-98	12 OUNCE CAN	*
		99	NO RESPONSE	
12J	ALCOHOL	1	NO	588
		2	YES	62
		9	NO RESPONSE	3

Appendix B continued

12K	ALCOFTEN	0	NEVER	588
		1	DAILY	5
		2	WEEKLY	48
		3	MONTHLY	9
		9	NO RESPONSE	5
12L	ALC_SV	1-8	SERVINGS	*
		9	NO RESPONSE	
13	MILKCOF	1	NO	*
		2	YES	
		9	NO RESPONSE	
14	FLH2O	1	NO	281
		2	YES	206
		3	DON'T KNOW	138
		9	NO RESPONSE	30
15.1	DIURETIC	1	NO	348
		2	YES	303
		9	NO RESPONSE	4
15.2	CORTISONE	1	NO	595
		2	YES	56
		9	NO RESPONSE	4
15.3	ISON	1	NO	707
		2	YES	3
		9	NO RESPONSE	5

Appendix B continued

15.4	ANTICON	1	NO	702
		2	YES	8
		9	NO RESPONSE	5
15.5	COUMADIN	1	NO	608
		2	YES	43
		9	NO RESPONSE	4
15.6	THYROID	1	NO	543
		2	YES	108
		9	NO RESPONSE	4
16.1	TUMS	1	NO	556
		2	YES	95
		9	NO RESPONSE	4
16.2	ROLAIDS	1	NO	661
		2	YES	49
		9	NO RESPONSE	5
16.3	MAALOX	1	NO	674
		2	YES	36
		9	NO RESPONSE	5
16.4	MYLANTA	1	NO	660
		2	YES	50
		9	NO RESPONSE	5

Appendix B continued

16.5	AMPHOGEL	1	NO	708
		2	YES	2
		9	NO RESPONSE	5
16.6	GELUSIL	1	NO	704
		2	YES	6
		9	NO RESPONSE	5
16.7	OTHERANT	1	NO	662
		2	YES	48
		9	NO RESPONSE	5
17	CIG	1	NO	594
		2	YES	44
		8	OTHER	0
		9	NO RESPONSE	21
17A	CIG_AMT	0	NONE	*
		1	<.5 PACK/DAY	
		2	.5-1	
		3	PACK/DAY	
		4	1.5-2	
		9	PACKS/DAY	
			>2 PACKS/DAY	
			NO RESPONSE	

Appendix B continued

17B	YR_SMOKE	00 01-98 99	NONE YEARS NO RESPONSE	*
18.1	KIDNEY	1 2 9	NO YES NO RESPONSE	682 16 17
18.2	ARTHRIT	1 2 9	NO YES NO RESPONSE	301 342 12
18.3	LACTOSE	1 2 9	NO YES NO RESPONSE	597 46 12
19	SURG_INT	1 2 9	NO YES NO RESPONSE	678 20 17
20	PREG	00-98 99	PREGNANCIES NO RESPONSE	*
21	BF	00-98 99	CHILDREN NO RESPONSE	*
21A	BF_MONTH	00-98 99	MONTHS NO RESPONSE	*

Appendix B continued

22	MENOPAUS	1 2 9	NO YES NO RESPONSE	0 485 170
22A	AGE_MENO	00-98 99	YEARS NO RESPONSE	*
22B	ESTROGEN	1 2 8 9	NO YES OTHER NO RESPONSE	446 118 0 91
23	HYSTEREC	1 2 9	NO YES NO RESPONSE	340 270 45
23A	AGE_HYST	00-98 99	YEARS NO RESPONSE	*
24A	SWIM	1 2 9	NO YES NO RESPONSE	664 31 20
24B	SWIMXMO	00-98 99	TIMES/MONTH NO RESPONSE	*
24C	SWIM_MIN	0000- 9998 9999	MINUTES NO RESPONSE	*

Appendix B continued

24D	JOG	1 2 9	NO YES NO RESPONSE	640 2 13
24E	JOGXMON	00-98 99	TIMES/MONTH NO RESPONSE	*
24F	JOG_MIN	0000- 9998 9999	MINUTES NO RESPONSE	*
24G	WALK	1 2 9	NO YES NO RESPONSE	192 450 13
24H	WALKXMON	00-98 99	TIMES/MONTH NO RESPONSE	*
24I	WALK_MIN	0000- 9998 9999	MINUTES NO RESPONSE	*
24J	BIKE	1 2 9	NO YES NO RESPONSE	523 119 13
24K	BIKEXMON	00-98 99	TIMES/MONTH NO RESPONSE	*

Appendix B continued

24L	BIKE_MIN	0000- 9998 9999	MINUTES NO RESPONSE	*
24M	SKI	1 2 9	NO YES NO RESPONSE	635 7 13
24N	SKIXMON	00-98 99	TIMES/MONTH NO RESPONSE	*
24O	SKI_MIN	0000- 9998 9999	MINUTES NO RESPONSE	*
24P	ROW	1 2 9	NO YES NO RESPONSE	635 7 13
24Q	ROWXMON	00-98 99	TIMES/MONTH NO RESPONSE	*
24R	ROW_MIN	0000- 9998 9999	MINUTES NO RESPONSE	*
24S	AEROBICS	1 2 9	NO YES NO RESPONSE	610 32 13

Appendix B continued

24T	AEROXMON	00-98 99	TIMES/MONTH NO RESPONSE	*
24U	AERO_MIN	0000- 9998 9999	MINUTES NO RESPONSE	*
24V	DANCE	1 2 9	NO YES NO RESPONSE	633 9 13
24W	DANCXMON	00-98 99	TIMES/MONTH NO RESPONSE	*
24X	DANC_MIN	0000- 9998 9999	MINUTES NO RESPONSE	*
24Y	OTHER_EX	1 2 9	NO YES NO RESPONSE	594 101 20
24Z	OTHERXMO	00-98 99	TIMES/MONTH NO RESPONSE	*
24AA	OTHR_MIN	0000- 9998 9999	MINUTES NO RESPONSE	*

Appendix B continued

25	AGE	00-98 99	YEARS NO RESPONSE	*
26	WT	000- 998 999	POUNDS NO RESPONSE	*
27	HT	00.0- 99.8 99.9	INCHES NO RESPONSE	*
28	HT_21	00.0- 99.8 99.9	INCHES NO RESPONSE	*
29	YRSLIVED	00-98 99	YEARS NO RESPONSE	*
30	PREV_ST	00-51 99	STATE NO RESPONSE	*
31	RACE	1 2 3 4 5 6 9	CAUCASIAN BLACK MEXICAN AMERICAN INDIAN ASIAN OTHER NO RESPONSE	594 14 0 0 23 0 24

Appendix B continued

32	REL_PREF	1	NO	37
		2	YES	609
		9	NO RESPONSE	9
32A	RELIGION	0	NONE	37
		1	PROTESTANT	245
		2	CATHOLIC	90
		3	LDS	255
		4	OTHER	19
		9	NO RESPONSE	9

*Either non-categorical data or not used in analysis.