Mother-daughter pairs: spinal and femoral bone densities and dietary intakes

Josephine Lutz and Rogene Tesar

ABSTRACT Bone mineral density (BMD) of the lumbar spine (L1–L4) and femur (femoral neck, Ward’s triangle, and trochanter) was measured in 37 healthy, white mother-daughter pairs by dual-photon absorptiometry. Mothers and daughters were aged 52 ± 7 and 25 ± 4 y (X ± SD), respectively. Three-day dietary intakes were evaluated. Significant correlations between mother-daughter pairs for BMD of all lumbar and femoral areas [except for L2 (r = 0.26, P = 0.054)] indicated familial resemblances in bone mineralization. Total calcium intake was significantly correlated with three BMD values for the daughters (L2, femoral neck, and trochanter) but not for the mothers. When mothers were classified as pre- (n = 20) or postmenopausal (n = 17), correlation coefficients for BMD were higher for premenopausal mothers and their daughters and lower for postmenopausal mothers and their daughters, except for the trochanter. The results suggest that the nature of inheritance of bone mass of women may have at least two components, one influencing the level of peak bone mass and one related to bone loss at menopause. Am J Clin Nutr 1990;52:872–7.

KEY WORDS Osteoporosis, hereditary diseases, genetics, health promotion, nutrition, bones, women, dietary calcium, diet

Introduction

Osteoporosis is the loss of cortical and trabecular bone to the extent that spontaneous fractures develop, usually of the vertebral column, hips, and wrists. The condition is a serious public health problem, especially for postmenopausal white women. As the population of elderly people increases, the prevalence of this disorder can be expected to increase unless preventive measures are instituted.

Fractures result in part from low bone mass (1). Although bone mass declines in all individuals with aging, it decreases more rapidly in women after menopause (1, 2). Although still speculative, researchers express concern that the most important risk factor for postmenopausal osteoporosis may be the amount of skeletal mass acquired during adolescence and the early adult years. A lower peak skeletal mass in early adulthood followed by a decrease in bone mass at menopause places these women at high risk for postmenopausal osteoporosis (2).

Postmenopausal osteoporosis is thought to be multifactorial in etiology, involving both hereditary and environmental factors. A family history of osteoporosis has often been cited as a risk factor despite little empirical evidence to support this view. Until recently the premise of a genetic contribution to osteoporosis was based on the study of the bone mineralization of the appendicular skeleton of twins (3–8) and of mother-daughter pairs (9, 10), and on racial differences in skeletal mass (11, 12). Although wrist fractures commonly occur in women with osteoporosis, these areas of the appendicular skeleton containing predominantly cortical bone are not primary sites of osteoporosis. With the development of techniques to measure bone mass of the axial skeleton, a primary site of osteoporosis and fractures, evidence to support a genetic contribution to bone mass of the spine and hip was published (8). Evans et al (13) found that relatives of osteoporotic patients had lower bone mineral in the os calcis and spine than did individuals with no family history of osteoporosis. Seeman et al (14) reported that daughters of women diagnosed as having postmenopausal osteoporosis had lower bone mineral content (BMC) of the lumbar spine and femoral neck and midshaft than did other women of the same age; the authors concluded that their lower amount of bone mineral may put these daughters at increased risk for fractures later in life.

The question has been asked as to whether genetic contributions to bone mass are systemic or site specific. Pollitzer and Anderson (15) recently reviewed studies in which genetic and environmental contributions to bone mass had been examined. They pointed out that although the number of studies has been surprisingly small, the similarities in correlations and estimates of heritability obtained in most studies suggested that the same genetic factors operate on both weight-bearing and nonweight-bearing bones. Pocock et al (8) concluded from their study of bone mineral density (BMD) of the lumbar spine, proximal femur, and forearm of twin pairs that one gene or a single set of genes determine bone mass at all skeletal sites. More evidence is needed to answer this question conclusively.

Lutz (9) previously reported significant correlations for ra-

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TABLE 1
Ages, heights, and weights of premenopausal and postmenopausal mothers and their daughters*

<table>
<thead>
<tr>
<th></th>
<th>Premenopausal (n = 20)</th>
<th>Postmenopausal (n = 17)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mothers</td>
<td>Daughters</td>
<td>Mothers</td>
</tr>
<tr>
<td>Age (y)</td>
<td>47 ± 4</td>
<td>57 ± 6</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>164.2 ± 6.4</td>
<td>166.2 ± 7.2</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>61.4 ± 9.1</td>
<td>68.6 ± 9.1</td>
</tr>
</tbody>
</table>

* x ± SD.
† Significantly different from weight of premenopausal mothers or daughters, P < 0.02.

dius BMC of 26 mother-daughter pairs (r = 0.400, P < 0.05). The major objective of the present study was to determine whether familial resemblances could also be demonstrated for BMD of the lumbar spine and femur of healthy mother-daughter pairs. The question asked was whether mothers with lower BMD for their age-group average tended to have daughters with lower BMD compared with their age-group average. In addition, 3-d dietary intakes were evaluated to assess the influence of diet and environment on bone mass.

Subjects and methods

Subjects

Healthy, white mother-daughter pairs were recruited for the study through announcements placed in community and university bulletin boards. The study was restricted to white women to provide homogeneity in sampling; black women reportedly have higher bone mass than white women and a lower incidence of osteoporosis (12). The age of the daughters was restricted to 20–35 y. Selection was based on medical histories. Thirty-seven of 150 pairs who applied for the study met the criteria for participation. Exclusion criteria included factors reported to affect bone: surgery (bilateral oophorectomy and gastrectomy), chronic illnesses (hormonal dysfunctions, malignancies, hepatic or renal diseases, malabsorption, and rheumatoid arthritis), and pharmacologic therapy (anticonvulsants, corticosteroids, diuretics, and vitamin D). Eight mothers had received estrogen-replacement therapy for 6 mo, and eight mothers and three daughters had smoked for 3 y; others had not taken estrogen and had never smoked. Criteria for selection included a moderate level of physical activity and acceptable weight-for-height limits based on Metropolitan Life Insurance Company weight and height tables (16). No subject was pregnant or lactating. No premenopausal women were amenorrheic and none had experienced a nontraumatic fracture or had been diagnosed as having osteoporosis. The mothers and daughters were well educated, both having had an average of 4 y of education past high school. The study was approved by university and multinationals human-subjects review boards, and voluntary, informed consent was obtained from all participants.

Relevant information about the participants is presented in Table 1. The mothers were aged 41–68 y, the daughters 20–34 y. Twenty mothers were still menstruating and were considered to be premenopausal, and 17 were postmenopausal; the average age time since menopause was 6.1 ± 4.2 y. Postmenopausal mothers were on average 10 y older than the premenopausal mothers; there was no significant difference in age between the two groups of daughters. There were no significant differences in height among the four groups. The weights of premenopausal mothers and their daughters were not significantly different. Postmenopausal mothers weighed significantly more than their daughters and the premenopausal mothers. The premenopausal mothers had had an average of 4.2 pregnancies and 3.2 term births; the postmenopausal mothers averaged 4.2 pregnancies and 3.7 term births. Thirteen daughters had been pregnant, with an average 1.8 pregnancies and 1.2 term births.

Methods

BMC of the lumbar spine and of the right proximal femur was measured by dual-photon absorptiometry (DPA) (17). Transmission scanning was done by use of the two energy peaks (7 and 16 Jeff) from a 153Gd source. This allowed computation of the BMC of bone independent of soft tissue. The standard protocol supplied by the manufacturer (Lunar Corp, Madison, WI) for automated analysis was used. All measurements were made by one person. The instrument was calibrated daily with a standardized three-step block bone phantom supplied by the manufacturer. Bone density readings randomly selected on 17 d over the 10-mo period of the study showed average CVs of 0.42%, 0.53%, and 0.53% for the large, medium, and small bone phantoms, respectively.

BMC of L1–L4 vertebrae was measured. BMC of all combinations of these vertebrae was determined by the computer program. The three regions of the proximal femur measured were the femoral neck, Ward’s triangle, and the trochanter. Ward’s triangle is the focus of aging bone loss and is critical to the strength of the proximal femur (18).

BMD was calculated by dividing the BMC by the projected area of the region scanned and was expressed as g/cm². The results were compared with age- and sex-specific normal values that were based on multicenter values incorporated into the computer program (19). Individual values were compared by the computer with normative values for white women of the same age and expressed as a percent of age-matched control values. The individual values were also compared with normative values for young, white women and expressed as a percent of young normal values. These latter values are used to assess an individual’s risk of fracture.

Three-day dietary records were obtained for each mother-daughter pair for the same 3 d. This consisted of a 24-h recall taken on the day the bone measurements were made, followed by food-intake records kept by the participants for 2 subsequent days. The women were instructed so as to ensure accurate recording of the quantity and nature of foodstuffs consumed. The nutrient intakes were coded and evaluated by one registered dietitian, by use of the N-Squared Nutritionist III computer program (N-Squared Computing, Silverton, OR). Total intakes of nutrients were calculated as dietary plus supplemental nutrient intake; these were compared with the recommended dietary allowance (RDAs) (20).

Statistical analysis

After the data were plotted and determined to approximate linearity, Pearson product-moment correlation coefficients
TABLE 2  
Bone mineral density of the lumbar spine and femur for mothers and daughters

<table>
<thead>
<tr>
<th>Area*</th>
<th>Mothers (n = 37)</th>
<th>Daughters (n = 37)</th>
<th>Correlation coefficient</th>
<th>t test</th>
</tr>
</thead>
<tbody>
<tr>
<td>L1</td>
<td>1.129 ± 0.200†</td>
<td>1.242 ± 0.166</td>
<td>0.385 (0.009)</td>
<td>-3.34 (0.002)</td>
</tr>
<tr>
<td>L2</td>
<td>1.225 ± 0.192</td>
<td>1.345 ± 0.181</td>
<td>0.268 (0.054)</td>
<td>-2.32 (0.003)</td>
</tr>
<tr>
<td>L3</td>
<td>1.250 ± 0.206</td>
<td>1.367 ± 0.167</td>
<td>0.390 (0.008)</td>
<td>-3.42 (0.002)</td>
</tr>
<tr>
<td>L4</td>
<td>1.260 ± 0.184</td>
<td>1.336 ± 0.166</td>
<td>0.416 (0.004)</td>
<td>-2.34 (0.020)</td>
</tr>
<tr>
<td>L1-L2</td>
<td>1.177 ± 0.192</td>
<td>1.294 ± 0.172</td>
<td>0.337 (0.021)</td>
<td>-3.39 (0.002)</td>
</tr>
<tr>
<td>L1-3</td>
<td>1.204 ± 0.193</td>
<td>1.321 ± 0.166</td>
<td>0.365 (0.013)</td>
<td>-3.48 (0.001)</td>
</tr>
<tr>
<td>L1-4</td>
<td>1.221 ± 0.187</td>
<td>1.325 ± 0.161</td>
<td>0.399 (0.007)</td>
<td>-3.29 (0.002)</td>
</tr>
<tr>
<td>L2-3</td>
<td>1.238 ± 0.195</td>
<td>1.356 ± 0.169</td>
<td>0.345 (0.018)</td>
<td>-3.43 (0.002)</td>
</tr>
<tr>
<td>L2-L4</td>
<td>1.246 ± 0.186</td>
<td>1.348 ± 0.163</td>
<td>0.401 (0.007)</td>
<td>-3.21 (0.003)</td>
</tr>
<tr>
<td>L3-4</td>
<td>1.256 ± 0.190</td>
<td>1.350 ± 0.163</td>
<td>0.434 (0.004)</td>
<td>-3.02 (0.005)</td>
</tr>
<tr>
<td>FN</td>
<td>0.870 ± 0.128</td>
<td>1.001 ± 0.142</td>
<td>0.501 (0.001)</td>
<td>-5.85 (0.000)</td>
</tr>
<tr>
<td>WT</td>
<td>0.735 ± 0.165</td>
<td>0.930 ± 0.179</td>
<td>0.590 (0.000)</td>
<td>-7.60 (0.000)</td>
</tr>
<tr>
<td>T</td>
<td>0.735 ± 0.114</td>
<td>0.791 ± 0.128</td>
<td>0.382 (0.010)</td>
<td>-2.54 (0.015)</td>
</tr>
</tbody>
</table>

* L, lumbar vertebrae; FN, femoral neck; WT, Ward's triangle; and T, trochanter.
† X ± SD.

Results

The BMD values of the lumbar vertebrae and femoral areas for the mothers and daughters are presented in Table 2 along with correlation coefficients and t-test values. All correlation coefficients were significant except for L2 BMD (r = 0.268, P = 0.054). The paired t-test indicated that mean BMD values of the mothers were all significantly lower than those of the daughters.

The BMD values of the lumbar vertebrae of the mothers averaged 98% of the values for young control subjects and 107% of age-matched control values. BMD values of the femoral neck, Ward's triangle, and trochanter for the mothers were 87%, 78%, and 91% of the values for young control subjects, respectively, and 100%, 99%, and 103% of age-matched control values. For the daughters the BMD values of all areas measured averaged 102% of the values for young control subjects.

Age of the mothers was negatively and significantly correlated with BMD values, except for the trochanter. For the mothers (n = 37), correlation coefficients between age and the weighted average of BMD of vertebrae L2, L3, and L4 (L2-L4), the femoral neck, Ward's triangle, and trochanter were -0.500 (P < 0.005), -0.345 (P < 0.025), -0.433 (P < .005), and 0.186 (NS), respectively. For the daughters (n = 37), the correlation coefficients between age and BMD of the lumbar vertebrae and trochanter were not significant but were significant for the femoral neck (r = -0.284, P < 0.05) and Ward's triangle (r = -0.343, P < 0.03).

Height and weight of the mothers were not significantly correlated with their BMD values. Daughters' heights correlated significantly with BMD of L2-L4 (r = 0.324, P < 0.05) but not with BMD of the femoral areas. Daughters' weights correlated with BMD of L2-L4 (r = 0.382, P < 0.025), femoral neck (r = 0.341, P < 0.025), and Ward's triangle (r = 0.288, P < 0.05), but not with that of the trochanter (r = 0.206, P = 0.221).

The mothers were divided into two groups, premenopausal (n = 20) and postmenopausal (n = 17), to assess the effect of maternal menstrual status on familial resemblances for BMD. The mean BMD values for the two groups of mothers and their daughters are given in Table 3, along with correlation coefficients for the association between mother and daughter values and the paired t-test values. There were no statistically significant differences in age, height, weight, or any of the BMD values between the daughters of the two groups. Paired and independent t-tests indicated no significant differences in BMD of the trochanter within the mother-daughter groups. BMD values of premenopausal mothers were significantly lower than values of their daughters for the femoral neck and Ward's triangle but not for L2-L4. BMD values of L2-L4, femoral neck, and Ward's triangle of the postmenopausal mothers were significantly lower than values for their daughters. In addition, BMD values of postmenopausal mothers for L2-L4, and Ward's triangle were significantly lower than those of premenopausal mothers. However, the differences in the correlation coefficients for the two mother-daughter groups were not significant, as determined by the Fisher r-to-Z transformation test.

When the BMD values were compared with those of normative values for women of the same age, all values for both pre- and postmenopausal mothers were appropriate, 95% to 100% of age-matched control values. However, when the BMD was expressed as a percent of normative values of young women, the mean values for postmenopausal mothers were lower than those of premenopausal mothers. The BMD of the femoral neck, Ward's triangle, and the trochanter, expressed as a percent of normal values for young women, were 83%, 72%, and 88%, respectively, for the postmenopausal mothers and 90%, 84%, and 93% for the premenopausal mothers.

Median nutrient intakes (dietary plus supplemental) for the
MOTHER-DAUGHTER PAIRS

TABLE 3
Comparison of bone mineral densities of the lumbar spine and femur of pre- and postmenopausal mothers and their daughters

<table>
<thead>
<tr>
<th>Area</th>
<th>Premenopausal-mother group (n = 20 pairs)</th>
<th>Postmenopausal-mother group (n = 17 pairs)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mothers*</td>
<td>Daughters*</td>
</tr>
<tr>
<td>L2-L4</td>
<td>1.322 ± 0.142‡</td>
<td>1.359 ± 0.183</td>
</tr>
<tr>
<td>FN</td>
<td>0.905 ± 0.102</td>
<td>1.002 ± 0.132</td>
</tr>
<tr>
<td>WT</td>
<td>0.786 ± 0.136‡</td>
<td>0.944 ± 0.163</td>
</tr>
<tr>
<td>T</td>
<td>0.750 ± 0.107</td>
<td>0.806 ± 0.117</td>
</tr>
</tbody>
</table>

* P = ± SD.
†‡ Significantly different from value for postmenopausal mother (t test): †P < 0.001, ‡P < 0.05.
§ P < 0.001.
|| P < 0.01.

Thirteen mothers and 18 daughters were ingesting < 800 mg/d Ca; 6 mothers and 7 daughters ingested < 500 mg/d.

Mean nutrient intakes of mothers and daughters were not significantly different as evaluated by t test. Correlation coefficients between nutrient intakes of mothers and their daughters were not significant. Caffeine intake of mothers was higher than that of the daughters (t = 3.29, P = 0.002). For the mothers, there were no significant positive correlations between BMD values and intakes of energy, protein, phosphorus, caffeine, or total calcium (one-tailed significance test). For the daughters, correlations were significant between total calcium intake and the BMD of three bone sites: L2 (r = 0.286, P = 0.043), the femoral neck (r = 0.303, P = 0.034), and the trochanter (r = 0.312, P = 0.030); however, energy, protein, phosphorus, and caffeine intakes did not correlate significantly with BMD.

Discussion

The significant positive correlations between mothers and their daughters for BMD of the lumbar spine and femur (Table 2) support the existence of familial resemblances for BMD of these areas. This study confirms the results of previous studies, which provided evidence of familial resemblances for appendicular (3–8) and axial bone mass among twins (8) as well as between pairs of individuals with other types of relatedness (9, 13, 14).

Both environmental and genetic factors may have contributed to the positive correlations for BMD between mothers and their daughters. Current methods do not allow hereditary factors to be clearly differentiated from environmental factors (22). Maternal effects, similarities in lifestyle and habits acquired by daughters through emulation of their mothers, tend to increase correlations of physical traits of mother-daughter pairs. For this reason, these estimates of association are more correctly taken as measures of the degree of familial resemblances and not strictly as measures of the strength of the genetic component of that relationship.

The classification of the mothers as pre- and postmenopausal resulted in increased correlations for BMD of the premenopausal mother-daughter pairs and decreased correlations for

women of the study are given in Table 4 along with the RDAs. Median values are presented because some women had extreme intakes on both the high and the low side, median values being less affected by extreme scores. Twenty-three mothers and 19 daughters were taking vitamin and/or mineral supplements. Supplemental calcium intake by the mothers (n = 21) averaged 580 mg/d and by the daughters (n = 13), 340 mg/d.

TABLE 4
Total nutrient intakes and Recommended Dietary Allowances (RDAs)*

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Mothers</th>
<th>Daughters</th>
<th>RDA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy (kcal)</td>
<td>1413 (712-2131)</td>
<td>1448 (639-2886)</td>
<td>1880, 2000†</td>
</tr>
<tr>
<td>Protein (g)</td>
<td>61.7 (30.1-98.0)</td>
<td>59.9 (14.5-217.0)</td>
<td>44</td>
</tr>
<tr>
<td>Calcium (mg)</td>
<td>1102 (172-2477)</td>
<td>818 (281-2366)</td>
<td>800</td>
</tr>
<tr>
<td>Phosphorus (mg)</td>
<td>1108 (403-1676)</td>
<td>1003 (368-2072)</td>
<td>800</td>
</tr>
<tr>
<td>Caffeine (mg)</td>
<td>197 (35-706)</td>
<td>83 (0-715)</td>
<td>10</td>
</tr>
<tr>
<td>Iron (mg)</td>
<td>17.4 (5.6-51.0)</td>
<td>14.8 (3.3-107.7)</td>
<td>10, 18‡</td>
</tr>
<tr>
<td>Magnesium (mg)</td>
<td>284 (64-767)</td>
<td>245 (60-780)</td>
<td>300</td>
</tr>
<tr>
<td>Zinc (mg)</td>
<td>10.1 (3.1-40.2)</td>
<td>7.9 (2.0-35.9)</td>
<td>15</td>
</tr>
<tr>
<td>Vitamin A (RE)</td>
<td>2144 (234-9436)</td>
<td>1841 (126-5742)</td>
<td>800</td>
</tr>
<tr>
<td>Vitamin C (mg)</td>
<td>147 (20-1640)</td>
<td>132 (20-1078)</td>
<td>60</td>
</tr>
<tr>
<td>Thiamin (mg)</td>
<td>2.1 (0.4-126.0)</td>
<td>1.7 (0.5-51.7)</td>
<td>1.0</td>
</tr>
<tr>
<td>Riboflavin (mg)</td>
<td>2.5 (0.6-125.8)</td>
<td>1.8 (0.6-52.0)</td>
<td>1.2</td>
</tr>
<tr>
<td>Niacin (mg)</td>
<td>30.9 (9.2-520.4)</td>
<td>23.6 (6.7-168.9)</td>
<td>13</td>
</tr>
</tbody>
</table>

* Medians; ranges in parentheses.
† RDAs for mothers and daughters, respectively.
‡ RDAs for menstruating and postmenopausal women, respectively.
the postmenopausal mother-daughter pairs (Tables 2 and 3). It is generally accepted that bone density depends to a large extent on the amount of bone developed during growth and its subsequent rate of loss during menopause and with aging (2, 6). The nature of inheritance of bone mass of women may, therefore, have at least two major components, one that influences peak bone mass attained during growth and a second set of genetic factors that regulates postmenopausal hormonal changes associated with bone loss. The two sets of genetic factors may be independent entities and are not necessarily passed on from mother to daughter as a unit. Differential environmental and genetic forces may influence each of the determinants to produce the end result—the degree of bone mass after menopause.

The correlations for BMD of the premenopausal mother-daughter pairs are interpreted to be a measure of familial resemblance in bone mass whereas the dilution of the correlation of BMD values for the postmenopausal mother-daughter groups is construed to be a consequence of bone loss experienced by the mothers during menopause. Perhaps the loss of bone by the postmenopausal mothers involved a second set of genetic factors not yet evident in their daughters. BMD of premenopausal mothers may therefore be a better predictor of iliac peak bone mass. Although the differences in the correlations for BMD values between pre- and postmenopausal groups were not statistically significant, it is felt that they might have been significant if there had been a larger sample. Larger cross-sectional studies of mothers and daughters, both of whom are postmenopausal, and longitudinal studies to evaluate how familial resemblances for BMD change over time, are necessary to confirm these suppositions.

These results suggest that menopausal status is an important consideration in the selection of subjects for studies related to genetic determinants of bone mass of women. In previous studies of twins and biologically related individuals, discrepancies between expected and obtained results were interpreted to mean that environmental factors were more important than genetic factors. Pocock et al (8) also found that estimates of heritability for forearm bone mass were higher for premenopausal women than for the group as a whole; they interpreted this to mean that environmental factors are increasingly important after the menopause and/or with advancing age. Sowers et al (10) found no consistent evidence of resemblance of distal radius bone mass among mother-daughter sets they studied and interpreted this as evidence of a stronger environmental rather than genetic influence on bone mass among women. These investigators (10) pointed out that given the differences in race and gender patterns of bone gain and loss, the amalgamation of dissimilar subgroups may obscure important relationships. The results of the present study suggest that homogeneity of subjects in terms of age and menopausal status is also an important consideration when studying women.

The BMD values for the women in this study compared favorably with those reported by Mazess et al (19) for women at their multicity center study. Average BMD values were slightly higher than those reported by Mazess et al (19); they were within one SD. All mean values were appropriate for women of their age. The women in the present study were selected to exclude medical, pharmaceutical, and lifestyle factors reported to affect BMD. Although none had been diagnosed as osteoporotic, a few mothers had BMD values below those considered normal (19, 23).

Significant negative correlations between age and BMD were obtained for L2–L4 for the mothers but not for the daughters, and for the femoral neck and Ward's triangle for both the mothers and the daughters, which provides further evidence that the loss of BMD in the femoral neck and Ward's triangle starts in young adulthood in women. Similar results were reported for the multicenter study (19); for women aged 20–39 y, spinal BMD was stable whereas the correlations of age and femoral BMD were low but significant, and for women aged 40–69 y, spinal and femoral BMD declined. In the present study, BMD values of L2–L4 and Ward's triangle were significantly lower for postmenopausal mothers than for premenopausal mothers (Table 3).

That median calcium intakes of mothers and daughters met the RDA for calcium was unexpected because surveys indicate that median calcium intakes for adult women in the United States approximate 500 mg/d (24). However, since the surveys, information on postmenopausal osteoporosis and the possibility of offsetting the development of osteoporosis by increasing calcium intake has been widely publicized (25). Many women have increased their consumption of foods high in calcium and their use of calcium supplements (25). In the present study, 21 mothers and 13 daughters were taking calcium supplements. Although there were significant correlations between calcium intakes and the BMD of three bone sites for the daughters, the importance of this finding is questionable. Three-day dietary records were evaluated in this study. These may or may not have been representative of long-term intakes. Because a number of different dietary and physiologic factors interacting over a long time determine bone density, it seems unlikely that BMD could be a reflection of a single factor, the 3-d dietary intake.

Lifelong deficiency of calcium intake or absorption is considered to be a factor in the development of osteoporosis. Bone weight and size increase rapidly during the preadolescent and pubertal years, and nutrition profoundly affects skeletal growth and bone mineralization. There is evidence that dietary calcium in early life influences peak skeletal mass. Matkovic et al (26) reported that a rural population in Yugoslavia with lifelong calcium intakes of 1000 mg/d had higher bone mass after age 30 y than did a neighboring population with calcium intakes of 500 mg/d. More recently, Halioua and Anderson (27) assessed present and past calcium intakes of premenopausal women; they concluded that habitual calcium intakes approximating the RDA were sufficient to achieve maximal BMD of the distal and midshaft radius. The benefits of calcium supplementation for BMD of the axial skeleton have not been conclusively demonstrated for adult and postmenopausal women in prospective, controlled studies (28, 29). Positive relationships were obtained when food frequency questionnaires were used to assess longer-term calcium intakes (30).

BMD did not correlate with caloric, protein, phosphorus, or caffeine intakes. Although it has been reported that these components affect calcium nutireture and possibly bone density (24), the effects of some of them have not been studied sufficiently in real-life situations.

Striking features of the dietary assessments were the low-energy intakes and the extremes in intakes of other nutrients. The risk of nutritional deficiencies of other nutrients increases when energy intakes are substantially below the RDA (20). However, 62% of the mothers and 51% of the daughters were taking vita-
min and/or mineral supplements. Surveys indicate that nutrition-supplement use is common among white, well-educated women (31). In the present study some women were consuming nutrient supplements greatly in excess of the RDAs. This is of concern because extremely high intakes of certain vitamins and minerals are potentially toxic.

The results of the present study have implications for the prevention of postmenopausal osteoporosis. Older women are more likely to be concerned about osteoporosis and to seek medical assessment for this disorder. Young women are also increasingly concerned about their own potential risk, especially upon learning of their mothers’ diagnoses of osteoporosis. Maternal findings could be used to identify daughters at risk. Young women could take advantage of strategies for maximizing that portion of bone mineralization open to environmental manipulation, which include ingesting 800 mg Ca/d, maintaining a program of moderate physical activity, avoiding smoking, and moderating alcohol use. On the other hand, major differences in lifestyle and habits within a mother-daughter pair could invalidate risk assessment based on the mother’s BMD. For example, smoking (32), anorexia nervosa (33), and intensive exercise training (34) are reported to be associated with menstrual dysfunctions, which may lead to bone demineralization. Such differences could affect the accuracy of the prediction.

This study provides further evidence of the strength of bone density similarities between healthy mothers and their daughters in areas of the skeleton most susceptible to osteoporosis.

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References