Original Article

Nutrient intake risk factors of osteoporosis in postmenopausal women

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This study was conducted to assess the association between dietary nutrient intake and osteoporosis risk in postmenopausal women. Bone mineral density was measured at the lumbar spine by dual-energy X-ray absorptiometry and a structured questionnaire was administered by a trained interviewer, which included information on sociodemographics, medical and reproductive history, and dietary intake. The study sample included 134 osteoporotic and 137 non-osteoporotic subjects between the ages of 52 and 68. Nutrient variables were classified into tertiles. Odds ratios and 95% confidence intervals (CI) were calculated which compared the highest tertile with the lowest tertile as a reference group. Odds ratios for osteoporosis were 1.47 (95% CI: 1.03 – 2.05) for total protein, 1.62 (95% CI: 0.51 – 3.92) for animal protein, and 2.98 (95% CI: 1.42 – 4.23) for sodium. Odds ratios for osteoporosis in the highest tertile were: 0.42 (95% CI: 0.23 – 0.83) for vegetable protein, 0.72 (95% CI: 0.51 – 0.90) for Ca, and 0.65 (95% CI: 0.49 – 0.88) for Fe, relative to the respective lowest tertile. These findings suggest that adequate nutrient intake may be important for prevention of osteoporosis in postmenopausal women.

Key Words: nutrient, diet, osteoporosis, menopause, risk

INTRODUCTION

Osteoporosis is one of three major chronic diseases found in the aged, including postmenopausal women. Menopause is associated with a reduction of estrogen secretion in women, resulting in decreased bone density that can lead to severe osteoporosis.[1] Consequently, changes in appetite may occur due to menopausal factors such as gustatory changes as well as physiological and psychological changes such as depression. As a result, the nutrition status of postmenopausal women may be impaired specifically with regard to a micronutrients.[2] It has been reported that micronutrients, such as the metalloenzymes of certain proteins involved in collagen synthesis and skeletal structure formation, are essential for bone density maintenance.[3]

Osteoporosis is associated not only with menopause, but also other factors such as ovariectomy, smoking, a lean body type, lack of exercise, deficient calcium intake, and excess intake of: animal protein, phosphorus, sodium, caffeine and alcohol.[4] Bone density is linked to nutrients, including certain minerals.[5] To date, there are several published studies analyzing the correlation between bone density and dietary patterns in postmenopausal women. The findings included the correlation of blood minerals such as calcium, phosphorus, magnesium, copper, zinc, and manganese with bone density.[6,7] A recent review article included a definition, etiologies, incidence of osteoporosis, and suggested possible dietary strategies for optimal bone health.[8]

The aim of our study was to investigate the adequacy of nutrition in the health of the skeletal system and reduction of bone loss after menopause. This cross-sectional study of postmenopausal women was conducted with measurements and analysis of the dietary intake of each subject to investigate the association between nutrients and bone density loss.

METHODS

Participants

The participants were postmenopausal women recruited during a medical visit to a University-affiliated clinic between September 2003 and February 2004. Exclusion criteria included early menopause before 40 years of age and current medication use that may affect bone density, including hormones, vitamins and mineral supplements. Women were also excluded if important risk factor or outcome data were not obtained from either their medical histories or questionnaires. The protocol and consent forms were approved by the institutional review board of the participating institution. Women were enrolled after informed consent and recommendations from their physician.

We used the World Health Organization Task Force on Osteoporosis definition of bone mineral density of at least 2.5 standard deviations below the average for the young

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healthy female population,[9] to identify 134 women for the osteoporosis group (cases) and 137 for the control group.

Physical Measurement and Bone Densitometry

Body height and weight were measured and body mass index (BMI) was calculated. Body fat percentage, lean body mass (LBM), and total body water were calculated by bio-electrical impedance analyzer on the basis of age and height. Bone density was measured at the lumbar spine (L2-L4) by dual energy X-ray absorptiometry. Trained and quality-monitored personnel performed the measurements.

Socio-demographics and Lifestyle Factors

A trained researcher conducted interviews using a structured questionnaire to obtain socio-demographics, family history, marital status, education, occupation, monthly household income, reproductive histories (age at menarche, menopause, full-term pregnancy, and last delivery), and smoking, drinking, and exercise habits.

Dietary Intake

We previously developed the food frequency questionnaire (FFQ) used in this study. The questionnaire's reliability and validity of food and nutrient intake were estimated and previously reported.[10-13] The average daily nutrient intake for each participant was calculated from a sum of 98 food items, after calculating each value in consideration of food intake by item and nutrient contents per 100g of food. We defined nine categories of food intake frequency as follows: never or rare, once a month, twice or three times a month, once or twice a week, three or four times a week, five or six times a week, once a day, twice a day, three times a day. Food portion was defined by three categories, small, medium, and, large, which were obtained from the FFQ. Daily intake of calories, proteins (animal protein, vegetable protein), carbohydrates, fats, cholesterol, and minerals (calcium, phosphorus, iron, sodium, potassium, zinc) were calculated. The nutritional intake data were analyzed with a new computer software program developed by the authors to analyze the intake of each nutrient obtained from the FFQ.

Data Integrity

Personal identifiers were encoded for confidentiality. Outlier and inconsistent data were identified and verification was attempted by reviewing of the original questionnaire or follow-up questioning of the participants.

Data Analysis

Comparisons between means were made using Student's t-test, and comparisons with categorical variables were made using the chi-square test. Nutrient intakes were categorized into high, middle, and low thirds of the range for each participant.

We estimated risk for osteoporosis with an odds ratio and 95% confidence interval (CI) using the lowest tertile for each value as the reference. Variables that were identified as potential confounding variables included age, smoking, alcohol drinking, BMI, exercise, family history of osteoporosis, and energy intake. Adjusted osteoporosis risk was estimated with multiple logistic regression analysis and Mantel-extension test was used to test for trends. All results were considered significant if $p < 0.05$.

Nutrient intake was calculated from a software program developed by the authors and statistical analysis was performed using SPSS version 12.0 (SPSS Inc., Chicago, IL, USA).

RESULTS

Comparisons of Bone Density, Age, Body Mass Index, Body Fat, Lean Body Mass, and Total Body Water by Osteoporosis status

Means and standard deviations for age, body mass index, body fat, lean body mass, and total body water measurements by osteoporosis status are shown in Table 1.

Average bone density T-scores for cases and controls were -2.91±1.28 and -0.58±0.61, respectively. There were no significant differences between cases and controls for ages at time of study, menarche, menopause, and at first full-term pregnancy.

The case group had an average of 15.9 years since start of menopause, significantly longer compared to controls (mean duration 13.6 years, $p < 0.05$). The average age of osteoporotic women at their last birth was 36.7 years, significantly higher than the average age of 33.4 years among controls ($p < 0.05$). The average body mass index (BMI) among the women with osteoporosis was 22.7±2.4

Table 1. Comparison of Mean Bone Density, T-scores, Age, Body Mass Index, Body Fat, Lean Body Mass and Total Body Water by Osteoporosis Status

Variables	Cases(n=134) Mean ± SD	Controls(n=137) Mean ± SD	*p*
BMD (T-score) ‡	-2.91 ± 1.28	-0.58 ± 0.61	0.002
Age (yrs)	62.6 ± 6.2	61.9 ± 5.8	0.263
Age at menarche (yrs)	17.1 ± 1.9	16.2 ± 1.3	0.350
Age at menopause (yrs)	47.5 ± 3.1	48.2 ± 3.8	0.638
Years since menopause (yrs) †	15.9 ± 8.3	13.6 ± 7.1	0.031
Age at the 1st full-term pregnancy (yrs)	24.4 ± 4.3	23.8 ± 3.9	0.429
Age at the last delivery (yrs) †	36.7 ± 4.6	33.4 ± 5.2	0.028
Body Mass Index (kg/m^2) ‡	22.7 ± 2.4	24.9 ± 3.1	0.009
Body fat (%)§	30.9 ± 5.6	35.8 ± 6.2	<0.001
Lean Body Mass (%)†	68.9 ± 5.9	64.2 ± 6.1	0.027
Total Body Water (%)†	50.3 ± 4.2	47.1 ± 3.9	0.028

$^{†, ‡, §}$ Values with different letters in a row means statistically significant differences at $\alpha = 0.05, 0.01, 0.001$ respectively by t-test.

kg/m^2, which was significantly lower compared to controls (24.9±3.1 kg/m^2, p = 0.009). The percentages of body fat, lean body mass, and total body water were each significantly different between the two groups (Table 1).

Lifestyle Risk Factors by Osteoporosis Status

Table 2 presents the odds ratios estimating risk of osteoporosis by lifestyle factors. The referent group for estimating educational level risk factors was less than six years of education. Compared to this referent group, the odds ratio (OR) for women with osteoporosis and six to nine years, 10-12, and 13 years or more of education decreased significantly with increasing level of education (respective OR and 95% confidence interval [CI] for each educational level were 0.77 and 0.66 – 0.91, 0.67 and 0.56 – 0.81, 0.52 and 0.29 – 0.89, p = 0.037).

In addition, the odds ratio was higher for osteoporosis among current smokers (OR 1.84, 95% CI: 1.15 – 2.94) compared to a referent group of non-smokers (p = 0.047). Alcohol consumption, family history, and coffee consumption was not significantly different between the two groups. Additionally, the osteoporosis risk for women with a habit of exercise was lower compared to those who did not exercise (p < 0.001).

Comparison of Nutrient Intakes between the Case Group and Control Group

The odds ratios related to nutrient intake risk factors between the cases and control groups are shown in Table 3. The intake of total protein, animal protein, and sodium were significant risk factors for osteoporosis in this study population. Relative to lowest tertile, the odds ratios for the highest tertile were 1.47 (95% CI: 1.03 – 2.05; p for trend: 0.004) for total protein, 1.62 (95% CI: 1.03 – 3.92; p for trend: 0.03) for animal protein, and 2.98 (95% CI: 1.42 – 4.23; p for trend: 0.005) for sodium. Some nutrients, such as vegetable protein, calcium, and iron, appeared to be protective dietary factors against osteoporosis. The odds ratios for the highest tertiles were 0.42 (95% CI: 0.23 – 0.83; p for trend: 0.011) for vegetable protein, 0.72 (95% CI: 0.51 – 0.90; p for trend: 0.003) for calcium, and 0.65 (95% CI: 0.49 – 0.88; p for trend: 0.002) for iron.

DISCUSSION

The objective of this study was to determine whether specific nutrients are associated with the development of osteoporosis in postmenopausal women. To date, many studies have assessed the relationship between bone density and protein intake. A high protein diet is known to aid skeletal growth during the growth period and to accelerate the degeneration of renal structure and function resulting in increased urinary elimination of calcium during aging.[14] A positive correlation between bone density and protein intake has been previously reported.[15] Calcium eliminated in the urine depends on both the intake quantity and the nature of the protein. Previous studies have reported that urinary calcium excretion increased with increased amounts of animal protein intake,[16,17] Another study found the rate of hip joint fractures increased with prolonged duration of large amounts of animal protein intake.[18] Isoflavone in soybean protein has recently been reported to function as a weak estrogen and to favorably affect bone metabolism.[19] Kaul et al.[20] reported a reduction in bone loss induced by aging in male rats when fed soybean protein instead of casein.

Previous findings also indicate that different sources of dietary protein may have different effects on bone

Table 2. Lifestyle Risk Factors by Osteoporosis status: numbers (n), percents, and odds ratios(OR) with 95% confidence intervals(CI)

Variables	Cases(n = 134) N(%)	Controls(n = 137) N(%)	OR§	95%CI¶	p
Education (years) †					
< 6	38 (28.4)	21 (15.3)	1.0		0.037
6 – 9	37 (27.6)	42 (30.7)	0.77	0.66 – 0.91	
10 – 12	41 (30.6)	53 (38.7)	0.67	0.56 – 0.81	
> 12	14 (10.4)	19 (13.9)	0.52	0.29 – 0.89	
Smoking†					
Never	106 (79.1)	118 (86.1)	1.0		0.047
Past	13 (9.7)	11 (8.0)	1.38	0.88 – 2.16	
Current	14 (10.4)	8 (5.8)	1.84	1.15 – 2.94	
Alcohol					
No	28 (20.9)	131 (95.6)	1.0		0.240
Yes	105 (78.4)	90 (65.7)	1.57	0.94 – 2.61	
Exercise (Athletic activity) ‡					
No	126 (94.0)	105 (76.6)	1.0		<0.001
Yes	7 (5.2)	31 (22.6)	0.59	0.36 – 0.97	
Family History					
No	104 (77.6)	117 (85.4)	1.0		0.791
Yes	15 (11.2)	12 (8.8)	0.96	0.64 – 1.46	
Coffee					
Never	48 (35.8)	54 (39.4)	1.0		0.538
1-3 cups / day	63 (47.0)	58 (42.3)	1.29	0.57 – 2.42	
> 3 cups / day	18 (13.4)	23 (16.8)	1.17	0.63 – 2.17	

Adjusted osteoporosis risk was estimated with multiple logistic regression analysis and Mantel-extension test was used to test for trends.
†, ‡ Values with different letters in a row means statistically significant differences at α = 0.05, 0.001 respectively by ANOVA.
§ OR=Odds ratio, ¶95%CI=95% Confidence interval

Table 3. Odds ratios for osteoporosis risk by nutrient intakes from the diet

Nutrient	Cases (n = 134) Mean±SD	Controls (n = 137) Mean±SD	Lowest	Middle	Highest	*p* for trend
Energy	1567.3 ± 459.7	1525.0 ± 413.3	< 1310	1310 – 1750	> 1750	
(kcal)			1.00	1.34 (0.83-2.15)	1.36 (0.79-2.28)	0.920
Total Protein‡	62.8 ± 13.5	56.9 ± 14.7	< 48	48 – 76	> 76	
(g)			1.00	0.91 (0.68-1.21)	1.47 (1.03-2.05)	0.004
Animal protein†	23.7 ± 18.5	15.0 ± 11.9	< 15	15-24	>25	
(g)			1.00	1.21 (0.58-2.52)	1.62 (1.03-3.92)	0.030
Vegetable protein†	39.1 ± 20.2	41.9 ± 23.5	< 34	34-54	>54	
(g)			1.00	0.62 (0.31-1.23)	0.42 (0.23-0.83)	0.011
Fat	31.3 ± 17.6	33.9 ± 20.4	< 20	20 – 40	> 40	
(g)			1.00	0.87 (0.67-1.12)	0.92 (0.72-1.19)	0.537
Carbohydrate	245.9 ± 170.8	260.8 ± 158.2	< 220	220 – 270	> 270	
(g)			1.00	0.77 (0.59-1.01)	0.81 (0.63-1.05)	0.250
Fiber	5.30 ± 2.81	4.94 ± 3.12	< 4	4 – 6	> 6	
(g)			1.00	1.01 (0.75-1.36)	1.20 (0.89-1.64)	0.122
Cholesterol	240.7 ± 180.9	229.4 ± 161.2	< 151	151 – 290	> 290	
(mg)			1.00	1.15 (0.87-1.53)	0.97 (0.69-1.35)	0.538
Ca‡	409.3 ± 219.0	430.9 ± 258.8	< 330	330 – 510	> 510	
(mg)			1.00	0.93 (0.73-1.18)	0.72 (0.51-0.90)	0.003
P	832.5 ± 370.4	809.5 ± 395.7	< 645	645 – 1050	> 1050	
(mg)			1.00	1.28 (0.97-1.70)	1.07 (0.77-1.48)	0.529
K	1879.4 ± 490.8	2008.5 ± 524.9	< 1758	1758 – 2426	> 2426	
(mg)			1.00	1.24 (0.95-1.62)	0.98 (0.74-1.35)	0.260
Na‡	3785.9 ± 1988.0	3258.9 ± 2011.4	< 2628	2628 –4031	> 4031	
(mg)			1.00	1.35 (0.64-2.80)	2.98 (1.42-4.23)	0.005
Fe‡	8.4 ± 2.7	10.5 ± 3.9	< 8.5	8.5 – 13.2	> 13.2	
(mg)			1.00	0.83 (0.65-1.13)	0.65 (0.49-0.88)	0.002
Zn	7.59 ± 3.91	7.80± 3.24	< 6.1	6.1 – 9.2	> 9.2	
(mg)			1.00	0.80 (0.62-1.04)	0.83 (0.63-1.06)	0.272

Adjusted osteoporosis risk was estimated with multiple logistic regression analysis and Mantel-extension test was used to test for trends.
†, ‡ Values with different letters in a row means statistically significant differences at $\alpha = 0.05$, 0.01 respectively.
Adjusted for age, smoking, alcohol drinking, BMI, exercise, family history of osteoporosis, and energy intakes

metabolism.[21] This was confirmed by the findings of the European Prospective Investigation into Cancer and Nutrition (EPIC)-Potsdam Study, which suggested that the animal-to-vegetal protein ratio, rather than the protein ratio, is most relevant for maintaining bone health.[22] It is presumed that animal foods tend to be net acid producing and vegetable foods net base producing.[23] However, associations between protein sources and bone mineral density do not generally support a dominant role for endogenous acid mechanism suggesting that other interactions with proteins and their sources.[24] Decreases in bone loss with increased vegetable and decreased animal protein requires confirmation in prospective studies and randomized clinical trials.

Further studies into mechanisms underlying the relationships between the amount and source of protein with bone health may help our understanding of epidemiological findings.

Heaney & Weaver[25] and Weaver & Plawecki[26] reported that low oxalate vegetables (i.e., broccoli, kale, cabbage, etc.) had a higher calcium content than milk. Phosphorus intake and phosphorus-supplying foods are important in terms of calcium. The optimal intake ratio of calcium to phosphorus is approximately 2:1[27] and with higher phosphorous intake, calcium absorption is hindered and bone loss occurs.[27] Another study found that the ratio of calcium to phosphorus is positively correlated with bone radius, while the intake of phosphorous over the recommended allowance is associated with a decrease in osteoid volume.[28] Bell et al.[29] reported that an increased phosphorous intake resulting in a calcium to phosphorous ratio less than 0.5 had a deleterious effect on the skeleton, and the risk of bone loss increased if the mineral intake continued. The results of our study are in agreement with these findings.

Calcium and sodium are major cations in the blood, sharing a reabsorption mechanism in the kidney. An excessive intake of sodium increases urinary elimination of calcium because of the close relationship between sodium and calcium transport in the kidneys.[30] Excessive sodium intake induces increases in urinary elimination of sodium, decreases in calcium reabsorption, and increases in urinary calcium excretion.[31,32] In a study of bone density changes in women with ages ranging from 22 to 70 years, after a week of consuming either a high sodium diet (300nmol/day) or a low sodium diet (50nmol/day), osteolysis occurred only in postmenopausal women consuming a high sodium diet.[33] Therefore, nutritional recommendations should target reductions in sodium intake for women affected by osteoporosis. Such a phenomenon seems to be caused by the eating habits of subjects preferring a salty taste and showing high salt intake, and by dietary patterns that center on vegetables such as kimchi (a Korean fermented cabbage seasoned with red pepper), pickled greens, and seasoned greens.

Smoliar[34] reported that iron-deficient diets caused retardation of collagen maturation in the femur, a calcium and phosphorus metabolic disorder, and that the intake of dietary iron influenced bone density. Conclusions from another study of the relationship between bone density and iron intake emphasized that iron was a positive marker of bone density in the femoral neck by Angus et al.[35] In conclusion, our study of postmenopausal women found associations between dietary factors and osteoporosis risk. Our findings suggest that diets rich in total protein, animal protein, or sodium are independent risk factors for osteoporosis, and that low intakes of vegetable protein, calcium, and iron appear to be protective against osteoporosis. Understanding the relationships among nutrients may be an important step toward the identification of dietary factors related to the prevention of age-related bone loss, especially in postmenopausal women.

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AUTHOR DISCLOSURES

Jeongseon Kim, Sun-Young Lim and Joo-Hak Kim, no conflicts of interest.

REFERENCES

1. Hadji P, Gottschalk M, Ziller V, Kalder M, Jackisch C, Wagner U. Bone mass and the risk of breast cancer: the influence of cumulative exposure to oestrogen and reproductive correlates. Results of the Marburg breast cancer and osteoporosis trial (MABOT). Maturitas. 2007;56:312-321.
2. Bales CW, Steinman LC, Freeland-Graves JH, Stone JM, Young RK. The effect of age on plasma zinc uptake and taste acuity. Am J Clin Nutr. 1986;44:664-669.
3. Eaton-Evans J. Osteoporosis and the role of diet. Br J Biomed Sci. 1994;51:358-370.
4. Ilich JZ, Kerstetter JE. Nutrition in bone health revisited: a story beyond calcium. J Am Coll Nutr. 2000;19:715-737.
5. Chen YM, Ho SC, Woo JL. Greater fruit and vegetable intake is associated with increased bone mass among postmenopausal women. Br J Nutr. 2006;96:745-751.
6. Mongiorgi R, Gnudi S, Moroni A, Bertocchi G, Galliani I, Benfenati L. Bone mineral alterations and Mg content in aging. Boll Soc Ital Biol Sper. 1990; 66:623-630.
7. Sojka JE, Weaver CM. Magnesium supplementation and osteoporosis. Nutr Rev. 1995;53:71-74.
8. Cashman KD. Diet, nutrition, and bone health. J Nutr. 2007; 137:2507S-2512S.
9. Genant HK, Gooper C, Poor G, et al. Interim report and recommendations for the World Health Organization Task-Force for Osteoporosis. Osteoporosis Int. 1999;10:259-264.
10. Kim J, Ahn YO, Paik HY, Hamajima N, Inoue M, Tajima K. Calibration of a food frequency questionnaire in Koreans. Asia Pac J Clin Nutr. 2003;12:251-256.
11. Kim J, Kim DH, Ahn YO, Tokudome Y, Hamajima N, Inoue M, Tajima K. Reproducibility of a food frequency questionnaire. Asian Pac J Cancer Prev. 2003;4:253-257.
12. Kim J, Kim Y, Ahn YO, Paik HY, Ahn Y, Tokudome Y, Hamajima N, Inoue M, Tajima K. Development of a food frequency questionnaire in Koreans. Asia Pac J Clin Nutr. 2003;12:243-250.
13. Kim J, Kim YJ, Ahn YO, Paik HY, Ahn Y, Tokudome Y, Hamajima N, Inoue M, Tajima K. Contribution of specific foods to fat, fatty acids, and cholesterol in the development of a food frequency questionnaire in Koreans. Asia Pac J Clin Nutr. 2004;13:265-272.
14. Amanzadeh J, Gitomer WL, Zerwekh JE, Preisig PA, Moe OW, Pak CY, Levi M. Effect of high protein diet on stone-forming propensity and bone loss in rats. Kidney Int. 2003; 64:2142-2149.
15. Kamel HJ. Postmenopausal osteoporosis: etiology, current diagnostic strategies, and nonprescription interventions. J Manag Care Pharm. 2006;12:S4-S9.
16. Calvo MS, Bell RR, Forbes RM. Effect of protein-induced calciuria on calcium metabolism and bone status in adult rats. J Nutr. 1982;112:1401-1413.
17. Barzel US, Massey LK. Excess dietary protein can adversely affect bone. J Nutr. 1998;128:1051-1053.
18. Abelow BJ, Holford TR, Insogna KL. Cross-cultural association between dietary animal protein and hip fracture: a hypothesis. Calcif Tissue Int. 1992;50:14-18.
19. Spence LA, Lipscomb ER, Cadogan J, Martin B, Wastney ME, Peacock M, Weaver CM. The effect of soy protein and soy isoflavones on calcium metabolism in postmenopausal women: a randomized crossover study. Am J Clin Nutr. 2005;81:916-922.
20. Kaul DN, Masoro EJ, Yu BP, Hardin RR, Hollis BW. Modulation of age-related hyperparathyroidism and senile bone loss in fisher rats by soy protein and food restriction. Endocrinology. 1988;122:1847-1854.
21. Sellmeyer Dr, Stoen KL, Sebastian A, Cummings SR. A high ratio of dietary animal to vegetable protein increases the rate of bone loss and the risk of fracture in postmenopausal women. Study of Osteoporotic Fractures Research Group. Am J Clin Nutr. 2001;73:118-122.
22. Weikert C, Walter D, Hoffmann K, Kroke A, Bergmann MM, Boeing H. The relation between dietary protein, calcium and bone health in women: results from the EPIC-Potsdam cohort. Ann Nutr Metab. 2005;49:312-318.
23. Sebastian A, Sellmeyer DE, Stone KL, Cummings SR. Dietary ratio of animal to vegetable protein and rate of bone loss and risk of fracture in postmenopausal women. Am J Clin Nutr. 2001;74:411-412.
24. Promislow JH, Goodman-Gruen D, Slymen DJ, Barrett-Connor E. Protein consumption and bone mineral density in the elderly: the Rancho Bernardo Study. Am J Epidemiol. 2002;155:636-644.
25. Heaney RP, Weaver CM. Calcium absorption from kale. Am J Clin Nutr. 1990;51: 656-657.
26. Weaver CM, Plawecki KL. Dietary calcium: adequacy of a vegetarian diet. Am J Clin Nutr. 1994;59:1238-1241S.
27. Teegarden D, Lyle RM, McCabe GP, McCabe LD, Proulx WR, Michon K, Knight AP, Johnston CC, Weaver CM. Dietary calcium, protein, phosphorus are related to bone mineral density and content in young women. Am J Clin Nutr. 1998;68:749-754.
28. Metz JA, Anderson JJ, Gallagher PN Jr. Intakes of calcium, phosphorus, and physical activity level are related to radial bone mass in young adult women. Am J Clin Nutr. 1993;58: 537-542.
29. Bell RR, Draper HH, Tzeng DY, Shin HK, Schmidt GR. Physiological responses of human adults to foods containing phosphate additives. J Nutr. 1977;107:42-50.
30. Harrington M, Cashman KD. High salt intake appears to increase bone resorption in postmenopausal women but high potassium intake ameliorate this adverse effect. Nutr Rev. 2003;61:179-183.
31. Castenmiller JJ, Mensink RP, van der Heijden L, Kouwenhoven T, Hautvast JG, de Leeuw PW, Schaafsma G. The effect of dietary sodium on urinary calcium and potassium ex-

cretion in normotensive men with different calcium intake. Am J Clin Nutr. 1986;41:52-60.
32. Shortt C, Madden A, Flynn A, Morrissey PA. Influence of dietary sodium intake on urinary calcium excretion in selected Irish individuals. Eur J Clin Nutr. 1988;42:595-603.
33. Evans CE, Chughtai AY, Blumsohn A, Giles M, Eastell R. The effect of dietary sodium on calcium metabolism in premenopausal and postmenopausal women. Eur J Clin Nutr. 1997;51:394-399.
34. Smoliar VI. Effect of iron-deficient diets on the formation of bone tissue. Vopr Pitan. 1984;5:55-59.
35. Angus RM, Sambrook PN, Pocock NA, Eisman JA. Dietary intake and bone mineral density. Bone Miner. 1988;4:265-277.

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影響更年期女性骨質疏鬆的營養攝取危險因子

本研究評估停經後期女性飲食中營養素攝取與骨質疏鬆危險性之關係。骨質密度測量方式，在腰椎部位利用雙能量 X 光吸收儀測量獲得。問卷部分，請受過訓練的訪視者以有架構的問卷進行訪談，其中包含的資訊為：社會人口學變項、用藥及生育史、飲食攝取。研究樣本收集 134 位骨質疏鬆與 137 位沒有骨質疏鬆之受試者，年齡為 52 歲至 68 歲之間。各營養素攝取量劃分為四分位。以最低攝取量組當作參考組，將最高組與最低組相比較，計算出勝算比(odds ratio)及信賴區間。對骨質疏鬆的發生，總蛋白質攝取量之勝算比為 1.47，動物性蛋白質為 1.62，鈉為 2.98。反之，植物性蛋白質之勝算比為 0.42，鈣為 0.72，鐵為 0.68。本研究的發現指出在停經後女性中，適當的營養素攝取可能對於預防骨質疏鬆是重要的。

關鍵字：營養素、飲食、骨質疏鬆、停經、危險因子