A B S T R A C T

Potential determinants of bone mass were investigated in a group of 70 young females (mean age 26.6 years), daughters of women studied in premenopause. Nutritional data, leisure physical activity level, lifestyle habits as well as familial similarities were assessed. The daughters' bone mineral density (BMD), measured by dual-energy absorptiometry, was significantly correlated with their body mass index (BMI) ($r = 0.22$), dietary vitamin D intake ($r = 0.19$) and their mothers' BMD ($r = 0.44$). Multiple regression analysis indicated that only the mothers' BMD remained an independent predictor of bone mass. Mother-daughter correlations were also observed for body weight ($r = 0.24$), height ($r = 0.39$), BMI ($r = 0.29$), dietary calcium intake ($r = 0.20$), and calcium ($r = 0.20$) or vitamin D ($r = 0.25$) intakes from dairy products. Hence, these observations support the evidence that mothers' BMD is the strongest predictor of bone mass of young women in their third decade.

A B R É G É

Nous avons évalué les principaux déterminants de la masse osseuse chez 70 jeunes femmes (âge moyen : 26,6 ans) dont les mères avaient été étudiées avant la ménopause. Notre évaluation a porté sur les apports nutritionnels, le niveau d'activité physique durant les loisirs, le mode de vie et les antécédents familiaux. Nous avons mesuré la densité osseuse de la colonne lombaire (DO) par densitométrie biphotonique. Afin de déterminer l'influence de l'hérédité et de l'environnement, ces paramètres ont été comparés à ceux des mères. La DO des filles était en relation significative avec celle de leur mère ($r = 0,44$), l'indice de masse corporelle (IMC) ($r = 0,22$) et l'apport alimentaire en vitamine D ($r = 0,19$). Par régression multiple, seule la DO des mères a été associée à la DO des filles. Des corrélations mère-fille ont été observées pour le poids ($r = 0,24$), la taille ($r = 0,39$), l'IMC ($r = 0,29$), l'apport en calcium ($r = 0,20$) et les apports en calcium ($r = 0,20$) ou en vitamine D ($r = 0,25$) provenant des produits laitiers. Ces observations suggèrent que la DO de la mère est le principal déterminant de la masse osseuse chez les jeunes femmes dans la trentaine.

Familial Resemblance of Bone Mineral Density Between Females 18 Years and Older and Their Mothers

Daniel Picard, MD, MSc,¹ Andrée Imbach, PhD,² Marielle Couturier, MSc,² Raymond Lepage, PhD,³ Michel Picard, MD¹

The risk of osteoporosis is determined later in life by the peak bone mass achieved in adulthood and the importance of subsequent bone loss. Understandably, much research has been focused on prevention of age- and menopause-related bone loss, but less attention has been given to the achievement of peak skeletal mass.

Longitudinal studies on peak bone mass reveal that while peak bone mass in the hip is reached in women in their early 20s,^{1,2} consolidation of bone mass continues toward the end of the third decade in the lumbar spine and forearm.³⁻⁶ Therefore, the identification of factors affecting women's bone density in the third decade is an important aspect of attempts to prevent osteoporosis. Peak bone mass is influenced by the complex and ongoing interaction between genetics and environmental factors, with genetics being considered one of the major determining factors. Studies of intrapair monozygotic and dizygotic twins suggest that the estimated heritability of bone mass at different sites varies between 57% and 90%.⁷⁻¹²

Investigations on familial resemblance of bone mass have yielded conflicting results. Premenopausal daughters of women with osteoporosis or hip fractures present lower bone mass than premenopausal daughters without a family history of osteoporosis.^{13,14} Between parents and children, correlations for bone mass at different skeletal sites have been observed to vary between

0.30 and 0.70.¹⁵⁻²⁰ Three-generation studies too have illustrated familial similarities in bone mass with correlations varying between 0.30 and 0.62.^{21,22} However, some authors have found no consistent evidence of significant bone mass resemblance between mothers and daughters.²³ It has been shown that genetic similarities of bone mass decrease with the increasing age of family members.²⁴ Furthermore, familial bone mass resemblance is not necessarily due entirely to the genetic transmission of traits and may be partially explained by common lifestyle habits of family members. Calcium intake, physical activity, smoking, consumption of caffeine and alcohol are a few of the numerous lifestyle factors associated with variability in bone density and rates of bone loss.²⁵⁻³⁰

Most studies in young women have not examined all potential factors that can influence bone mass. The aim of this investigation was to evaluate the main determinants of bone mass in a group of young females aged over 18, daughters of women already studied in premenopause, and to assess mother-daughter similarities in bone mass, anthropometric data as well as environmental factors such as calcium and vitamin D intakes.

METHODS

Daughters, aged 18 to 35 years, of French Canadian women who had already been studied 10 years ago while in premenopause³¹ and subsequently in perimenopause³² were invited to participate in the present study. Of 95 eligible daughters who were enrolled, 70 completed the study. The exclusion criteria were actual pregnancy or breast feeding, history of cancer, abnormal thyroid function, chronic anovulation, early menopause or oophorectomy, myalgic encephalomyelitis and drug

1. Department of Nuclear Medicine, Hôpital Saint-Luc and Université de Montréal, Montreal, QC
2. Department of Physiology, Faculty of Medicine, Université de Montréal
3. Department of Biochemistry, Hôpital Saint-Luc This project was supported by the Dairy Bureau of Canada.

Correspondence and reprints: Dr. Daniel Picard, Department of Nuclear Medicine, CHUM – Research Centre, Hôpital St-Luc, 1058 St-Denis, Montreal, QC H2X 3J7, Tel: 514-281-2121, ext. 6229.

abuse. Fifty-eight mothers with an average age of 44 years old when studied in premenopause, were involved in the comparison. The group of daughters included 16 pairs of sisters. All participants had given their written informed consent.

General data, as well as those relative to lifestyle habits, were collected by an interviewer with the use of a questionnaire. Some data referred to the actual state of the subject such as age, civil status, general state of health, smoking habits, consumption of alcohol, caffeine and use of vitamin and mineral supplements. Other data referred to lifetime history such as medical history, use of medication, age at first menarche and history of amenorrhea.

Dietary assessment was done with a 5-day food record (4 weekdays and 1 weekend day), mailed with proper instructions to each participant, brought to the interview and reviewed with a dietician. Food records were coded and intakes calculated with the Nutrient Analysis Program.³³ The distribution of calcium and vitamin D intakes per food group and meal was also analyzed.³⁴ Moreover, intakes of calcium and vitamin D were assessed with a food frequency questionnaire.

The level of leisure physical activity was determined using a questionnaire including the most popular social and sport activities (dancing, aerobics, golf, bowling, skating, etc.), specifying for each the frequency of practice, duration in minutes per session, intensity in METS according to a recent Compendium of physical activity.³⁵ The daily energy expenditure (kcal/day) was then calculated by multiplying the METS by the body weight.

Height (m) was measured with the subjects in stocking feet, using a vertical wall scale, and weight (kg) was measured without shoes and heavy clothing on a hospital platform scale. Body mass index (BMI) was then calculated in kg/m². For mothers, the values measured 10 years before (in premenopause) were used.³¹

Serum ionized calcium (adjusted to pH 7.40) was measured within an hour after sampling on an ICA 1. Serum creatinine, total calcium, inorganic phosphorous, albumin and alkaline phosphatase were measured on the day of sampling by automated colorimetry on a Paramax analyzer

TABLE I
Anthropometric Measurements

	Daughters (n = 70)	Mothers (n = 58)
Age (years)	26.6 ± 4.7*	44.1 ± 2.7
Weight (kg)	59.7 ± 8.6	59.3 ± 7.5
Height (m)	1.63 ± 0.06	1.59 ± 0.05
BMI (kg/m ²)	22.5 ± 2.8	23.1 ± 2.5

* Mean ± SD

TABLE II
Daily Intake of Nutrients Noted from 5-day Food Records

Nutrient	Mean ± SDM	Range	RNI
Energy (kcal)	1906 ± 399	(1152-3039)	
Protein (g)	74.6 ± 16.5	(45.1-113.0)	51*
Carbohydrate (g)	236 ± 61	(134.0-450)	
Fat (g)	73.6 ± 20.3	(36.0-142.0)	
Cholesterol (mg)	231 ± 68	(94.4-382.0)	
Calcium (mg)	941 ± 352	(340-1846)	700
Phosphorus (mg)	1241 ± 363	(599-2441)	850
Iron (mg)	12.8 ± 6.0	(6.7-43.0)	13
Zinc (mg)	10.1 ± 7.0	(4.2-48.4)	9
Copper (mg)	1.2 ± 0.9	(0.6-5.4)	
Sodium (mg)	2652 ± 779	(1352-5078)	
Potassium (mg)	2771 ± 836	(1370-6355)	
Magnesium (mg)	256 ± 113	(115-877)	203†
Vitamin A (RE)	1569 ± 1462	(343-7768)	800
Vitamin D (µg)	4.8 ± 4.9	(0.1-28.4)	2.5
Thiamin (mg)	2.2 ± 3.9	(0.7-32.7)	0.8
Riboflavin (mg)	1.8 ± 1.0	(0.9-8.1)	1
Niacin (NE)	33.5 ± 15.0	(14.8-125.0)	14
Vitamin B6 (mg)	1.7 ± 1.1	(0.6-8.2)	1.1‡
Vitamin B12 (µg)	4.3 ± 5.2	(1.1-42.1)	1
Folate (µg)	257 ± 167	(106-1091)	185¶
Vitamin C (mg)	133 ± 84	(37-413)	30
Total Fiber (g)	12.4 ± 8.2	(2.6-51.3)	

* Protein: 0.86 g/kg x average weight of group (59.7 kg)

† Magnesium: 3.4 mg/kg x average weight of group (59.7 kg)

‡ Vitamin B6: 0.015 mg/g protein

¶ Folate: 3.1 µg/kg x average weight of group (59.7 kg)

(Dade). Serum for all other tests was kept at -20°C and thawed only once before batch measurement with commercially available kits. When doubtful, the state of ovarian function was determined measuring FSH and LH, as well as estradiol. Thyroid function was assessed in all participants by measuring TSH, T3 and total thyroxine. Osteocalcin (Immunotopics, Nichols Institute Diagnostics, San Juan Capistrano, CA; between assay CV: 6.7%) and 25-hydroxy Vitamin D (Incstar, Stillwater, MN; between assay CV: 10.5%) were measured in duplicate using commercial radioimmunoassays. Serum 1,25 dihydroxy Vitamin D was determined by a calf-thymus radioreceptor assay after separation by HPLC.³⁶ The between assay CV for this measurement was 3.7%. Serum PTH was evaluated with an in-house radioimmunoassay (between assay CV =

11.6%) recognizing large carboxyl terminal fragments of PTH.^{37,38}

Bone mineral content of the lumbar spine (BMC_L) was evaluated in all subjects by dual photon absorptiometry using Novo LAB-22B equipment. Measurements were taken on the L₂, L₃ and L₄ vertebrae. The results are expressed in grams of hydroxyapatite (gHA) for the total BMC of L₂ to L₄ vertebrae, corrected for the bone silhouette area of L₂ to L₄ (BMC_L/cm²). Short-term precision error in non-osteoporotic human subjects was 2.5%.³¹ The same method of measurement was used for mothers when they were studied in premenopause and perimenopause. For the purpose of this study, we compared daughters' BMD to mothers' BMD in premenopause since the latter has not been subjected to perimenopausal bone loss associated with a decline in estrogens.

TABLE III
Biochemical Parameters

		Normal Values
Ionized calcium (mmol/L)	1.26 ± 0.04*	1.19 - 1.34
Total calcium (mmol/L)	2.37 ± 0.07	2.23 - 2.60
Phosphorus (mmol/L)	1.12 ± 0.16	0.70 - 1.30
Albumin (g/L)	42 ± 3	37 - 51
Alkaline phosphatase (U/L)	53 ± 14	25 - 97
Parathormone (pmol/L)	5 ± 2	2.5 - 10.0
Osteocalcin (ng/ml)	6.5 ± 2.3	1.2 - 10.5
25-OH Vitamin D (nmol/L)	82.6 ± 19.3	30 - 115
1-25 (OH) ₂ Vitamin D (nmol/L)	102.7 ± 29.5	72 - 120

* Mean ± SD

TABLE IV
Simple Correlation Coefficients of Bone Density to Other Variables

Variable	R	P
BMI	0.22	0.03
Dietary vitamin D intake	0.19	0.05
Mothers' premenopausal bone density	0.44	< 0.0001

TABLE V
Stepwise Regression Analysis of Bone Density in Daughters*

Variable	Beta	P	%R ²
Mothers' premenopausal bone density	0.433	0.0002	19
Cholesterol intake	- 0.221	0.0454	4.8
Age	0.059	0.5935	0

* R² = 0.23819 (24%), adjusted R² = 0.20458, F = 6.87854, P = 0.0004

Statistical analysis

Descriptive statistics (means, standard deviations and ranges) were determined for all variables. Pearson correlation analysis was performed to examine the linear relationship of the daughters' bone mass to anthropometric, dietary and other factors as well as mother-daughter similarities.

After evidence was obtained of the normality of criterion variables distributions and following critical review of the intercorrelation patterns, the daughters' bone mineral density (BMD) was submitted to hierarchical stepwise multiple regression. All analyses were interpreted using an uncorrected 2-tailed alpha level of 0.05.

RESULTS

Anthropometric data

Table I presents anthropometric data on the groups studied. Most subjects (73%) were at their ideal body weight, as expressed by a BMI between 20 and 25.

Nutritional data

Table II presents energy and the mean daily intakes of 22 nutrients for the daughters. Most intakes were in accordance with Canadian recommended nutrient intakes (RNI) except for calcium, iron, zinc, vitamin A, vitamin D and folate, for which 6%, 14%, 13%, 13%, 26% and 4% of subjects respectively did not meet 2/3 of the RNI. The habitual calcium intake as calculated from a food frequency questionnaire was 892 mg ± 354/day. There was a strong correlation between calcium and vitamin D intakes calculated by the food records and food frequency questionnaires (0.89 p<0.0001 for calcium and 0.91 p<0.0001 for vitamin D).

Calcium and/or vitamin D supplements were consumed regularly by 10% of the sample. In those taking supplements, mean calcium intake from this source was 425 ± 360 mg and 11.8 ± 5.6 µg for vitamin D. It should be noted that 57% of their mothers were also taking such supplements at the time of the study. Dairy products provided 62% of calcium and 50% of dietary vitamin D.

Ovarian function and lifestyle-related data

All participating daughters had normal ovarian function. Mean age at the onset of first menarche was 13.0 ± 1.7 years.

Smoking was a daily habit in 38.6% of the sample, who smoked 0.5 ± 0.4 packs per day. They started smoking at age 16.3 ± 3.1 years and smoked for 9.6 ± 6.2 years. The young women also consumed 144 ± 177 mg of caffeine, the equivalent of 1.5 regular cups of coffee, and 5.7 ± 8.1 g of alcohol daily which is less than half a drink.

In these otherwise active women, energy expenditure from leisure physical activity amounted to 236 ± 189 kcal/day (987 ± 791 kJ), corresponding to 1 hour of walking at a moderate pace of 3.5 mph, ½ hour running at 5 mph or 20 minutes of squash for a woman of 60 kg. None of the women were engaged in competitive sports.

Biochemical parameters

All of the biochemical parameters measured were within normal range (Table III). TSH, T₃, T₄, FSH, LH and estradiol levels were only used to establish the normality of the sample and are not reported in this table.

Correlation with bone density

Mean BMD was 0.95 ± 0.10 gHA/cm² in daughters, compared to 0.93 ± 0.10 gHA/cm² in their mothers measured in premenopause. BMD of the daughters correlated positively with their BMI, their dietary intake of vitamin D (not including supplements), as well as their mothers' premenopausal BMD (Table IV). There was no significant correlation between BMD and calcium intake in these young women.

Predictors of bone mass in daughters

Predictors of the daughters' BMD were determined by stepwise multiple regression analysis (Table V). Two factors explain 24% of the daughters' BMD, the mothers' BMD being the strongest predictor and cholesterol intake. In this group, age did not appear to be a predictor of bone density.

Mother-daughter correlations

Besides the strong correlation observed between mother-daughter BMD, weight, height, ensuing BMI, calcium intake, calci-

um and vitamin D from dairy products as well as alcohol and caffeine intakes were also positively correlated (Table VI).

Correlations between sisters

Among the study subjects, there were 16 pairs of sisters from 10 different families. These sisters were alike in bone density and morphologic parameters such as weight, height and BMI (Table VII), indicating a genetic relationship. On the other hand, factors suggesting the influence of the environment, such as the level of physical activity, calcium and vitamin D intakes bore no significant resemblance.

DISCUSSION

Our results provide evidence that in the period of bone mass consolidation, mothers' BMD is the main determinant of bone mass in their daughters. In fact, we found that in this group of young women, mostly in their third decade of life, mothers' BMD can explain as much as 19% of variance in BMD observed in agreement with other studies.^{17,18,22,39} Besides genetic contribution, familial similarities in bone mass may be partly explained by common mother-daughter environmental factors. Ulrich et al.²⁰ reported a positive correlation between lifetime milk consumption of mother-daughter pairs. We found mother-daughter correlations for caffeine, alcohol, dietary calcium intake and calcium or vitamin D from dairy products. The mothers had taken part in an earlier study on the factors influencing bone mass in premenopause. This study may have heightened the awareness of these women to the benefits of adequate nutritional habits for the prevention of osteoporosis since their calcium intake increased by 50% in 10 years.³² Furthermore, our mother-daughter correlations suggest that an awareness program in mothers may have beneficial effects on the dietary habits of their daughters.

We observed a strong bone mass correlation between sisters with a coefficient of 0.68. Few studies have addressed the relation between bone mass in sisters.^{18,40} The correlation observed between siblings reflects, in a certain way, the influence of both parents. It is suggested that a daughter

TABLE VI
Mother-daughter Correlations

	R	P
Bone density (lumbar spine)*	0.44	<0.0001
Weight*	0.26	0.014
Height*	0.39	0.0004
BMI*	0.29	0.007
Dietary calcium intake	0.26	0.015
Calcium intake from dairy products	0.20	0.05
Vitamin D intake from dairy products	0.25	0.017
Alcohol consumption	0.24	0.02
Caffeine consumption	0.33	0.003

* Refers to data in premenopausal mothers

TABLE VII
Correlations Between Sisters

	R	P
Bone density (lumbar spine)	0.68	0.001
Weight	0.65	0.003
Height	0.56	0.012
BMI	0.64	0.003

n = 16 pairs

ter has a 5.1 times greater risk of low BMD if her mother has a low BMD and this risk increases to 34.4 if both parents have a low BMD.⁴¹

No association was observed between calcium intake and bone mass. However, we only evaluated actual intakes; long-term intakes may have greater effects on bone mass. On the other hand, the group's mean calcium intake was approximately 900 mg, which is higher than those reported in other Canadian studies.^{42,43} Hence, calcium intakes might have been too high to observe any significant relationship with bone mass. Kanders et al.⁴⁴ observed that vertebral BMD does not appear to increase with calcium intake above 800-1000 mg, implying a threshold effect. In the literature, there is inconclusive evidence on the relationship between calcium intake and bone mass in premenopausal women. While some studies support the finding that calcium has a positive impact on bone mass,^{27,45,46} others have shown no benefit of dietary calcium on bone mass.⁴⁷⁻⁵⁰ It is suggested that regions with high trabecular content, such as the spine, appear to be stable after growth and until menopause unless hormonal changes occur, and are less influenced by calcium intake.⁵¹

Many studies have reported a correlation between BMD and weight, height or BMI.^{13,17,20,27,41,52-54} In our study, the

daughters' BMI was correlated with BMD in single correlation analysis, but not retained as a predictor variable by multiple regression analysis. On the other hand, there were mother-daughter correlations for BMD, weight, height and BMI. In the regression, the mothers' BMD was the strongest predictor of the daughter's BMD. This independent factor represents both environmental and genetic factors. Hence, in daughters, the influence of weight and/or height on their BMD could be part of the genetic inheritance estimated through their mothers' BMD.

No correlation between BMD and the level of physical activity was observed in our study subjects, and this was the case in other studies.^{19,45,50,53,54} This factor probably assumes greater importance later in life as some women remain active and others become sedentary.

Surprisingly, cholesterol intake was retained as a significant predictor of BMD by multiple regression analysis. To the best of our knowledge, there has been no previous report of such a correlation. Michaëlsson et al.⁵⁵ noted an inverse correlation between dietary fat and BMD while others demonstrated a positive association between dietary animal fat and fractures.⁵⁶ The mechanism suggested is the formation of insoluble soaps by the interaction of fat with calcium in the gut, impairing calcium absorption.

Dietary vitamin D intake was weakly correlated with BMD but was not retained as a predictor variable by multiple regression analysis. Vitamin D is one of the important factors implicated in the maintenance of healthy bone mass. Besides dietary intake, skin exposure to solar light can generate vitamin D. In our group of young active women, the sun may have provided an important quantity of vitamin D. There was no relation between 25-OH vitamin D – a best marker of the vitamin D status – and vitamin D intake. Since our subjects were all evaluated during the summer period, their 25-OH vitamin D level might have been higher than their average yearly level.

In conclusion, this study shows that mothers' BMD is the strongest predictor of bone mass of young women in their third decade. This familial resemblance is suggestive of a genetic contribution to bone mass but may be partly attributed to common lifestyle factors since there was a familial influence on dietary habits such as dietary calcium intake, calcium and vitamin D from dairy products, as well as alcohol and caffeine consumption. Therefore, measures of osteoporosis prevention in mothers may have a beneficial impact on the dietary habits of their daughters.

ACKNOWLEDGEMENTS

The authors thank Dr. Marc Dumont of COREV group for the statistical analyses and Dr. Marielle Gascon-Barré for the assessment of vitamin D serum measurements. We acknowledge the editorial work done on this manuscript by Mr. Ovid DaSilva of the Research Support Office, Research Centre, Centre hospitalier de l'Université de Montréal.

REFERENCES

- Melton LJ. Epidemiology of fractures. In: Riggs BL, Melton LJ (Eds.), *Osteoporosis: Etiology, Diagnosis and Management*, 2nd ed. Philadelphia: Lippincott-Raven, 1995;225-47.
- Eddy D, Johnston C, Cummings S, et al. Osteoporosis: Review of the evidence for prevention, diagnosis and treatment and cost-effectiveness analysis. *Osteoporos Int* 1998;8(S4):S1-S88.
- Matkovic V, Jelic T, Wardlaw GM, et al. Timing of peak bone mass in Caucasian females and its implication for the prevention of osteoporosis. *J Clin Invest* 1994;93:799-808.
- Rodin A, Murby B, Smith MA, et al. Premenopausal loss in the lumbar spine and neck of femur: A study of 225 Caucasian women. *Bone* 1990;11:1-5.
- Sowers MR, Clark MK, Hollis B, et al. Radial bone density in pre- and peri-menopausal women: A prospective study of rates and risk factors for loss. *J Bone Miner Res* 1992;7:647-57.
- Recker RR, Davies M, Hinders S, et al. Bone gain in young adult women. *JAMA* 1992;268:2403-08.
- Nguyen TV, Howard PJ, Kelly PJ, et al. Bone mass, lean mass, and fat mass: Same genes or same environments? *Am J Epidemiol* 1998;147:3-16.
- Dequeker J, Nijs J, Verstraeten A, et al. Genetic determinants of bone mineral content of the spine and the radius: A twin study. *Bone* 1987;8:207-9.
- Pocock NA, Eisman JA, Hopper JL, et al. Genetic determinants of bone mass in adults: A twin study. *J Clin Invest* 1987;80:706-10.
- Christian JC, Wu P-L, Slemenda CW, et al. Heritability of bone mass: A longitudinal study in aging male twins. *Am J Hum Genet* 1989;44:429-33.
- Slemenda CW, Christian JC, Williams CJ, et al. Genetics determinants of bone mass in adult women: A reevaluation of the twin model and the potential importance of gene interaction on heritability estimates. *J Bone Miner Res* 1991;6:561-67.
- Arden NK, Baker J, Hogg C, et al. The heritability of bone mineral density, ultrasound of the calcaneus and hip axis length: A study of postmenopausal twins. *J Bone Miner Res* 1996;11:530-34.
- Seeman E, Hopper JL, Bach LA, et al. Reduced bone mass in daughters of women with osteoporosis. *N Engl J Med* 1989;320:554-58.
- Seeman E, Tsalamandris C, Formica C, et al. Reduced femoral neck bone density in the daughters of women with hip fractures: The role of low peak bone density in the pathogenesis of osteoporosis. *J Bone Miner Res* 1994;9:739-43.
- Lutz J. Bone mineral, serum calcium, and dietary intakes of mother/daughter pairs. *Am J Clin Nutr* 1986;44:99-106.
- Tylavsky FA, Bortz A, Hancock R, et al. Familial resemblance of radial bone mass between premenopausal mothers and their college-age daughters. *Calcif Tissue Int* 1989;45:265-72.
- Lutz J, Tesar R. Mother-daughter pairs: Spinal and femoral bone densities and dietary intakes. *Am J Clin Nutr* 1990;52:872-77.
- Hansen MA, Hassager C, Jensen SB, et al. Is heritability a risk factor for postmenopausal osteoporosis? *J Bone Miner Res* 1992;7:1037-43.
- Krall EA, Dawson-Hughes B. Heritable and lifestyle determinants of bone mineral density. *J Bone Miner Res* 1993;8:1-9.
- Ulrich CM, Georgiou CC, Snow-Harter CM, et al. Bone mineral density in mother-daughter pairs: Relations to lifetime exercise, lifetime milk consumption, and calcium supplements. *Am J Clin Nutr* 1996;63:72-79.
- Kahn SA, Pace JE, Cox ML, et al. Osteoporosis and genetic influence: A three-generations study. *Postgrad Med J* 1994;70:798-800.
- Mckay HA, Bailey DA, Wilkinson AA, et al. Familial comparison of bone mineral density at the proximal femur and lumbar spine. *Bone Miner* 1994;24:95-107.
- Sowers MR, Burns TL, Wallace RB. Familial resemblance in bone mass in adult women. *Genet Epidemiol* 1986;3:85-93.
- Slemenda CW, Christian JC, Williams CJ, et al. Genetic determinants of bone mass in adult women: A reevaluation of the twin model and the potential importance of gene interaction on heritability estimates. *J Bone Miner Res* 1991;6:561-67.
- Laitinen K, Välimäki M. Alcohol and bone. *Calcif Tissue Int* 1991;49:S70-S73.
- Bassey EJ, Ramsdale SJ. Increase in femoral bone density in young women following high-impact exercise. *Osteoporos Int* 1994;4:72-75.
- Ramsdale SJ, Bassey EJ, Pye DJ. Dietary calcium intake relates to bone mineral density in premenopausal women. *Br J Nutr* 1994;71:77-84.
- Harris SS, Dawson-Hughes B. Caffeine and bone loss in healthy postmenopausal women. *Am J Clin Nutr* 1994;60:573-78.
- Hopper JL, Seeman E. The bone density of female twins discordant for tobacco use. *N Engl J Med* 1994;330(6):387-92.
- Flicker L, Hopper JL, Rodgers L, et al. Bone density determinants in elderly women: A twin study. *J Bone Miner Res* 1995;10:1607-13.
- Picard D, Ste-Marie LG, Coutu D, et al. Premenopausal bone mineral content relates to height, weight and calcium intake during early adulthood. *Bone Miner* 1988;4:299-309.
- Picard D, Imbach A, Couturier M, et al. Longitudinal study on the evolution of bone density and its determinants in women in peri or early menopause. *Calcif Tissue Int* 2000;67:356-60.
- Warwick E. *Nutrient Analysis Program*. Cornwall, P.E.I.: 1991.
- Couturier M, Imbach A. Calcium and vitamin D intakes of women and their daughters: Distribution by food groups and meals. *J Can Diet Assoc* 1997;58:77-83.
- Ainsworth BE, Haskell WL, Leon AS, et al. Compendium of physical activities: Classification of energy costs of human physical activities. *Med Sci Sports Exerc* 1993;25:71-80.
- D'Amour P, Gilbert F, Gascon-Barré M, et al. Late increase in serum 1,25-dihydroxyvitamin D one month after surgery for adenomatous hyperparathyroidism. *Clin Endocrinol* 1986;24:349-58.
- D'Amour P, Labelle F, Lazure C. Comparison of four different carboxylterminal tracers in a radioimmunoassay specific to the 68-84 region of human parathyroid hormone. *J Immunoassay* 1984;5:183-204.
- D'Amour P, Labelle F, Wolde-Giorghis R, et al. Immunological evidences for the presence of small late carboxylterminal fragment(s) of human parathyroid hormone (PTH) in circulation in man. *J Immunoassay* 1989;10:191-205.
- Henderson NK, Price RI, Cole JH, et al. Bone density in young women is associated with body weight and muscle strength but not dietary intakes. *J Bone Miner Res* 1995;10:384-93.
- Sowers MR, Boehne M, Jannausch ML, et al. Familiality and partitioning the variability of femoral bone mineral density in women of child-bearing age. *Calcif Tissue Int* 1992;50:110-14.
- Jouanny P, Guillemin F, Kuntz CJ, et al. Environmental and genetic factors affecting bone mass. *Arthritis Rheumat* 1995;38:61-67.
- Mailhot M, Ghadirian P, Parent M-E, et al. Patterns of calcium intake among French-Canadians living in Montreal. *Can J Public Health* 1994;85:351-56.
- Santé Québec, Bertrand L. *Les Québécoises et les Québécois mangent-ils mieux? Rapport de l'enquête québécoise sur la nutrition, 1990*. Ministère de la Santé et des Services sociaux, Gouvernement du Québec: Montréal, QC, 1995.
- Kanders B, Dempster DW, Lindsay R. Interaction of calcium nutrition and physical activity on bone mass in young women. *J Bone Miner Res* 1988;3:145-49.

45. Fehily AM, Coles RJ, Evans WD, et al. Factors affecting bone density in young adults. *Am J Clin Nutr* 1992;56:579-86.
46. Metz JA, Anderson JJB, Gallagher PN. Intakes of calcium, phosphorus, and protein, and physical-activity level are related to radial bone mass in young adult women. *Am J Clin Nutr* 1993;58:537-42.
47. Riggs BL, Wahner HW, Melton LJ, et al. Dietary calcium intake and rates of bone loss in women. *J Clin Invest* 1987;80:979-82.
48. Desai SS, Baran DT, Grimes J, et al. Relationship of diet, axial, and appendicular bone mass in normal premenopausal women. *Am J Med Sci* 1987;293(4):218-20.
49. 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;53:132-42.
50. Armamento-Villareal R, Villareal DT, Avioli LV, et al. Estrogen status and heredity are major determinants of premenopausal bone mass. *J Clin Invest* 1992;90:2464-71.
51. Nieves JW, Golden AL, Siris E, et al. Teenage and current calcium intake are related to bone mineral density of the hip and forearm in women aged 30-39 years. *Am J Epidemiol* 1995;141:342-51.
52. Lindsay R, Cosman F, Herrington BS, et al. Bone mass and body composition in normal women. *J Bone Miner Res* 1992;7:55-63.
53. Parsons TJ, Prentice A, Smith EA, et al. Bone mineral mass consolidation in young British adults. *J Bone Miner Res* 1996;11:264-74.
54. Young D, Hopper JL, Nowson CA, et al. Determinants of bone mass in 10- to 26-year-old females: A twin study. *J Bone Miner Res* 1995;10:558-67.
55. Michaëlsson K, Holmberg L, Mallmin H, et al. Diet, bone mass, and osteocalcin: A cross-sectional study. *Calcif Tissue Int* 1995;57:86-93.
56. Whyshak G. Dietary animal fat intake, calcium intake, and bone fractures in women 50 years and older. *J Women's Health* 1993;2:329-34.

Received: January 24, 2000

Accepted: February 21, 2001

COMING EVENTS ACTIVITÉS À VENIR

To be assured of publication in the next issue, announcements should be received by **November 15, 2001** and valid as of **December 31, 2001**. Announcements received after **November 15, 2001** will be inserted as time and space permit. Pour être publiés dans le prochain numéro, les avis doivent parvenir à la rédaction avant le **15 novembre 2001** et être valables à compter du **31 décembre 2001**. Les avis reçus après le **15 novembre 2001** seront insérés si le temps et l'espace le permettent.

8th Canadian Conference on International Health (CCIH)/ 8^{ème} Conférence canadienne sur la santé internationale (CCSI)

Child and Youth Health: Action, Research and Advocacy/ Pour un plan d'action, de recherche et de promotion sur la santé de l'enfant

18-21 November/novembre 2001 Ottawa, ON

Organized by the Canadian Society for International Health, with funding support from the Canadian International Development Agency, and the Pan-American Health Organization

Confirmed speakers/ Conférenciers confirmés : Lieutenant-General Romeo Dallaire, Dr. Ciro De Quadros

Contact/ Contacter :

CCIH Secretariat/ Secrétariat de la CCSI

Tel: 1-877-722-4140

Outside/ Hors Canada: 1-613-722-4140, ext. 224

E-mail/ Courriel : ccih@ag-cdn.com

http://www.csih.org/index_e.html ou

http://www.csih.org/index_f.html

2001: A TB Odyssey

Sponsored by the Ontario Ministry of Health

5-6 December 2001 Toronto, ON

Contact:

Michelle Wilson

Eventives Conference and Event Management

E-mail: eventives@hotmail.com

Fax: 905-619-1761

The Early Years: Building Family Capacity/Building Supportive Communities

Sponsored by Interprofessional Continuing Education, University of British Columbia and BC Association of Infant Development Consultants

31 January-2 February 2002 Vancouver, BC

Contact:

Elaine Liau, Director

Interprofessional Continuing Education, UBC

Tel: 604-822-4965 Fax: 604-822-4835

E-mail: elaine@cehs.ubc.ca

Better Breathing 2002

The Ontario Thoracic Society's Annual Conference on Respiratory Health

1-2 February 2002 Toronto, ON

Contact:

The Ontario Thoracic Society

Tel: 416-864-9911 Fax: 416-864-9916

E-mail: julianaq@on.lung.ca www.on.lung.ca

8th Annual Qualitative Health Research Conference
Sponsored by International Institute for Qualitative Methodology (IIQM) and University of Alberta
4-6 April 2002 Banff, AB

Keynote speakers: Jeanie Kayser-Jones, Margaret Lock

Contact:

IIQM

Tel: 780-492-9041 Fax: 780-492-9040

E-mail: qualitative.institute@ualberta.ca

www.ualberta.ca/~iiqm/qhr2002/

ALCOA Forum 2002: A Lifelong Odyssey - Active Living for Older Adults

Saskatchewan Seniors Mechanism

9-12 May 2001

Regina, SK

Contact:

ALCOA office 1-800-549-9799

and/or the website:

<http://www.alcoa.ca> or the

Saskatchewan Seniors Mechanism

Tel: 306-359-9956

6^e Conférence mondiale sur la Prévention et contrôle des traumatismes / 6th World Conference on Injury Prevention and Control

Traumatismes, suicide et violence : construire un savoir, des politiques et des pratiques pour promouvoir un monde en sécurité / Injuries, Suicide and Violence: Building Knowledge, Policies and Practices to Promote a Safer World

12-15 mai/May 2002

Montréal, QC

Contact / Contacter :

Congress Secretariat

511, place d'Armes, # 600

Montréal, QC H2Y 2W7

Tel: 514-848-1133 or

1-877-213-8368 (Canada & US)

Fax: 514-288-6469

E-mail: trauma@coplanor.qc.ca

www.trauma2002.com

Travel & Epidemics

IIIrd European Conference on Travel Medicine

Sponsored by WHO and CDC Atlanta

15-18 May 2002

Florence, Italy

Contact:

Prof. Walter Pasini

Director, WHO CC for Travel Medicine

Tel. +39-0541-24301 Fax. +39-0541-25748

E-mail: wpasini@rimini.com

**CALL FOR ABSTRACTS
DEMANDE DE RÉSUMÉS**

Our Environment, Our Health

Notre environnement, notre santé

CPHA 93rd Annual Conference

93^e Conférence annuelle de l'ACSP

Co-sponsored by the Northwest Territories/Nunavut Branch, CPHA / Co-parrainée par la Division des Territoires du Nord-Ouest et du Nunavut, ACSP

7-10 July/juillet 2002 Yellowknife, NWT/TN-O

Contact / Contacter :

CPHA Conference Department

Département des conférences de l'ACSP

Tel/Tél : 613-725-3769, ext. 126

Fax/Téléc : 613-725-9826

E-mail/Courriel : conferences@cpha.ca

www.cpha.ca

Deadline for Abstracts

Date limite de soumission : 7 January/janvier 2002