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# Does Lactose Intolerance Predispose to Low Bone Density? A Population-Based Study of Perimenopausal Finnish Women

R. HONKANEN,[1] P. PULKKINEN,[2] R. JÄRVINEN,[2] H. KRÖGER,[3] K. LINDSTEDT,[2] M. TUPPURAINEN,[4] and M. UUSITUPA[2]

[1] *Institute of Community Medicine, University of Tromsø, Norway*
[2] *Department of Clinical Nutrition, University of Kuopio, Finland*
[3] *Department of Surgery and* [4]*Department of Gynecology, University Hospital of Kuopio, Finland*

**The relationship of lactase malabsorption to osteoporosis is unclear. We examined the relationship of self-reported lactose intolerance (LI) to bone mineral density (BMD) in perimenopausal Finnish women. A random population sample of 2025 women aged 48–59, who underwent spinal and femoral BMD measurement with dual X-ray absorptiometry in Kuopio, Finland during 1989–1991 formed the study population. Out of these women, 162 women reported LI. The mean dairy calcium intake was 558 mg/day in women with LI and 828 mg/day in other women ($p < 0.0001$). The mean spinal BMDs were 1.097 and 1.129 g/cm$^2$ (–2.8%) ($p = 0.016$) and the mean femoral BMDs were 0.906 and 0.932 g/cm$^2$ (–2.8%) ($p = 0.012$) for the LI and other women, respectively. After adjusting for weight, age, years since menopause, and the history of hormone replacement therapy, these differences changed to –2.7% ($p = 0.016$) for the spinal and –2.4% ($p = 0.012$) for the femoral BMD, respectively. Dairy calcium intake was an independent determinant of femoral BMD. The addition of calcium intake variables into the multivariate model did not affect the spinal BMD difference, but weakened the femoral BMD difference to –1.9% ($p = 0.075$). Our results suggest that LI slightly reduces perimenopausal BMD, possibly through reduced calcium intake. (*Bone 19:23–28; 1996*)**

**Key Words:** Lactose intolerance; Lactose malabsorption; Bone density; Osteoporosis; Calcium intake; Menopause.

## Introduction

The relationship of lactose malabsorption to osteoporosis is unclear. Some studies have reported an association,[2,8,19,20] while the others have not.[1,12,13,24,26] Only the study by Slemenda et al. was large enough to exclude the effects of chance on study results.[24] They concluded that it would be more productive to query calcium intake rather than lactase deficiency as a risk factor for osteoporosis. In fact, a recent study of postmenopausal Italian back pain patients suspected of having osteoporosis demonstrated that only symptomatic lactose malabsorption was related to low calcium intake and low bone density.[5] The prevalence of lactose malabsorption in adult Italians is high (51%–71%).[3] None of the above-mentioned studies was based on a representative population sample, and only in one study[24] were major adjustments for confounding carried out.

In Finland, four-fifths of dietary calcium is obtained from milk products.[17] Thus, lactase deficient Finns —17% of the adults[23] —might have a calcium intake much below the Finnish average. These individuals might be good candidates for studies testing the hypothesis presented by several authors[2,5,20,24] that low calcium intake, and not the lactase deficiency per se, causes osteoporosis in persons with lactose malabsorption.

The purpose of the present study was to investigate the relationship between self-reported lactose malabsorption, i.e., lactose intolerance (LI), calcium intake, and BMD in a population sample of perimenopausal Finnish women.

*Address for correspondence and reprints:* Dr. Risto Honkanen, Institute of Community Medicine, University of Tromsø, 9037 Tromsø, Norway. E-mail: risto@ism.uit.no

## Subjects and Methods

Subjects were recruited from the Kuopio Osteoporosis Risk Factor and Prevention (OSTPRE) Study.[10,11] In 1989 the OSTPRE "baseline postal enquiry" was sent to the 14,220 women between the ages of 47 and 56 in the Kuopio Province, Eastern Finland. Thirteen thousand and one hundred (92%) women responded. The study population consisted of the random sample of 2,025 women who underwent densitometry from 1989–1991.[16] The second questionnaire about current and past risk factors, such as the use of dairy products, coffee, and alcohol as well as body weight, was administered at the time of densitometry. Information about menopause, oophorectomy, and hormone replacement therapy were checked and updated by interview during densitometry. One hundred and sixty-two (8.0%) of these women had reported LI in 1989. Valid BMD measurements of the spine and hip were obtained from 1835 and 2010 women, respectively.

Finally, 210 LI women out of the whole densitometry sample[16,25] responded to the third enquiry about the time and method of lactose malabsorption diagnosis. Most (82%) of these 210 LI cases reported that their LI had been verified with the lactose tolerance test (serial blood glucose determinations after a 50 g oral dose of lactose). Abdominal symptoms during the test were reported by 79% of the women. A majority (63%) of these diagnoses had been made during the 1980s, and the LI women had started to reduce their use of milk in 1982, on average. Use of supplemental calcium in 1989 was calculated for these 210 LI women and for a random subsample of 210 control women.[25]

Bone mineral density (BMD) was measured at the lumbar spine (L2–4) and femoral neck using dual-energy X-ray absorptiometry (DXA) (Lunar DPX).[15,16]

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Dairy calcium intake was estimated with two questions: (1) "How many deciliters of milk products (such as milk, buttermilk, processed sour milk, and yogurt) do you use daily on average?," and (2) "How many slices of cheese do you use daily on average?." Dairy calcium intake was computed as the sum of calcium derived from fluid milk products (120 mg/dL) and cheese (87 mg/slice) based on the NUTRICA, a PC-program for nutritional data developed by the Social Insurance Institution of Finland.[14] The validity of the questionnaire was tested against a 4-day food record completed by 76 women (39 LI and 37 control women) in 1990.[18] Dairy calcium covered 72%–79% of the dietary calcium intake. The correlation coefficient for the dairy calcium from the questionnaire vs. the dietary calcium from food records was 0.50 and that for the dairy calcium calculated from the 1989 vs. 1990 questionnaires was 0.64.

History of hormone replacement therapy (HRT) by the time of densitometry was used both as a dichotomous (0–5/6+ months) and continuous (years) variable. The premenopausal women were those with less than 6 months since the last menstruation, who had never used HRT. The postmenopausal women were those with 12 months or more since the last menstruation.

Statistical analyses were carried out with the SPSS program. The statistical significance of differences was tested with the chi-square test for categorical data and with the Student's $t$-test for continuous data. The nonparametric Mann–Whitney test was used to test the difference between calcium intake from supplements and from diet. Linear and multiple regression was used to examine the associations between BMD and calcium intake. All the variables in **Table 1** were introduced in forced entry regression models. Variables which were significantly related to BMD were retained in final models (**Table 2**). The association between LI and BMD was examined with the analysis of covariance (**Tables 3 and 4**).

## Results

Table 1 compares characteristics of women with and without LI. Current dairy calcium intake was lower among LI women than controls. There was also a marked difference in the recalled dairy calcium intake for the year 1980 [710 (SD 373) vs. 880 (368) mg/day] ($p < 0.001$), and a slight difference for 1970 [840 (372) vs. 906 (379) mg/day] ($p = 0.050$), but no statistically significant difference for the year 1960 [861 (406) vs. 891 (396) mg/day]. The lower dairy calcium intake of LI women was due to their reduced consumption of fluid milk products (**Figure 1**). On the other hand, the cheese consumption of LI women was similar to that of other women, in 1990 it was even higher [347 vs. 300 mg calcium from cheese per day, respectively ($p = 0.013$)].

The use of calcium supplements in 1989 was more common among LI women than other women in the total study population (Table 1). The daily dose information was available for the sample of 420 (210 LI cases and 210 controls), with the following case/control percentile values: 60th 140/0 mg, 70th 224/0 mg, 80th 381/75 mg, 90th 612/278 mg, and 100th 1376/1170 mg (Mann–Whitney $p < 0.0001$). However, the total (dairy + supplemental) calcium intake was lower in LI cases than controls (Mann–Whitney $p = 0.0001$), with the following percentile values for cases/controls: 10th 242/414 mg, 50th 703/852 mg, and 90th 1284/1477 mg. LI cases and controls who used supplements in this sample of 420 showed the following mean (SD) calcium intakes, respectively: (a) supplemental: 365 (310) and 284 (245) mg ($p = 0.098$), and (b) total: 875 (444) and 1155 (520) mg ($p = 0.0005$).

*Calcium intake and bone density.* The association between dairy calcium intake and BMD (during the 1989–1991 period) was investigated with regression analysis (Table 2). In linear regression, the intake in 1980 explained 0.1% ($p = 0.154$) and 0.4% ($p = 0.007$) of the respective BMD variations. The explanatory power of other past intake variables was even lower. The dairy intakes in 1989 and in 1980 together explained 0.4% ($p = 0.053$) of the variation of the spinal and 0.9% ($p = 0.0003$) of that of the femoral BMD. The use of supplements in 1989 as a dichotomous variable was slightly inversely related to BMD, explaining 0.2% of the variation of both spinal ($p = 0.046$) and femoral ($p = 0.050$) BMD. The dairy intakes in 1989 and 1980

**Table 1.** Characteristics of the 2025 women by lactose intolerance status

| Characteristic | Lactose intolerants (N = 162) | Others (N = 1863) | $p$ value |
|---|---|---|---|
| (A) Means (SDs) of continuous variables | | | |
| Age, years | 53.7 (2.9) | 53.5 (3.0) | 0.612 |
| Years since menopause | 2.80 (3.82) | 3.06 (3.75) | 0.391 |
| Weight, kg | 68.0 (10.5) | 69.4 (12.2) | 0.143 |
| Height, cm[a] | 161.5 (4.9) | 161.1 (5.2) | 0.377 |
| Dairy calcium intake, mg/day[a] | 558 (362) | 828 (378) | <0.0001 |
| Use of coffee, cups/day | 3.8 (2.2) | 4.3 (2.1) | 0.004 |
| Number of health disorders (excl. LI)[a] | 1.51 (1.40) | 1.20 (1.20) | 0.002 |
| Spinal bone density, g/cm$^2$ | 1.097 (0.145) | 1.129 (0.158) | 0.016 |
| Femoral bone density, g/cm$^2$ | 0.906 (0.126) | 0.932 (0.127) | 0.012 |
| (B) Proportions (%) of categorical variables | | | |
| Postmenopausal, 12+ months | 52.5 | 54.2 | 0.669 |
| Bilateral oophorectomy[a] | 6.8 | 8.2 | 0.524 |
| Hormone replacement therapy, ever | 59.9 | 46.3 | 0.0009 |
| Smoking, ever[a] | 14.2 | 17.6 | 0.279 |
| Use of alcohol, >36 g/week | 17.7 | 13.3 | 0.125 |
| Physically inactive[a,b] | 16.7 | 19.9 | 0.318 |
| Use of calcium supplements[a] | 61.6 | 40.5 | <0.0001 |
| Reported health disorders (excl. LI)[a] | 71.6 | 66.5 | 0.186 |

[a]Information from baseline postal enquiry in 1989. Other information collected during densitometry in 1989–1991.
[b]Light or no work and no regular leisure activity.

**Table 2.** Relations of current dairy calcium intake to spinal and femoral bone density according to linear and multiple regression analyses.

| | Regression coefficient (beta) | SE (beta) | *T* ratio | *p* value |
|---|---|---|---|---|
| (A) Spinal bone density, mg/cm$^2$ (N = 1829) | | | | |
| Calcium, mg/day | 0.022 | 0.010 | 2.34 | 0.020 |
| Intercept | 1108.0 | 8.52 | 130.1 | <0.0001 |
| $R^2$ = 0.30% | | | | |
| Calcium, mg/day | 0.016 | 0.009 | 1.86 | 0.063 |
| Weight, kg | 3.753 | 0.295 | 12.7 | <0.0001 |
| Years since menopause | −9.634 | 1.116 | −8.63 | <0.0001 |
| Age, years | −6.637 | 1.405 | −4.72 | <0.0001 |
| Hormone replacement therapy (N/Y) | 14.78 | 6.835 | 2.16 | 0.031 |
| Intercept | 1230.9 | 75.37 | 16.33 | <0.0001 |
| $R^2$ = 17.3% | | | | |
| Calcium, mg/day | 0.013 | 0.009 | 1.42 | 0.157 |
| Weight, kg | 3.748 | 0.294 | 12.74 | <0.0001 |
| Years since menopause | −9.703 | 1.115 | −8.70 | <0.0001 |
| Age, years | −6.580 | 1.404 | −4.69 | <0.0001 |
| Hormone replacement therapy (N/Y) | 15.69 | 6.839 | 2.30 | 0.022 |
| Lactose intolerance (N/Y) | −28.27 | 12.52 | −2.26 | 0.024 |
| Intercept | 1233.2 | 75.29 | 16.38 | <0.0001 |
| $R^2$ = 17.5% | | | | |
| (B) Femoral bone density, mg/cm$^2$ (N = 2004) | | | | |
| Calcium, mg/day | 0.028 | 0.007 | 3.87 | 0.0001 |
| Intercept | 906.6 | 6.56 | 138.2 | <0.0001 |
| $R^2$ = 0.74% | | | | |
| Calcium, mg/day | 0.017 | 0.006 | 2.57 | 0.010 |
| Weight, kg | 4.852 | 0.212 | 22.91 | <0.0001 |
| Years since menopause | −4.112 | 0.809 | −5.09 | <0.0001 |
| Age, years | −5.713 | 1.028 | −5.56 | <0.0001 |
| Hormone replacement therapy (N/Y) | 17.29 | 4.996 | 3.46 | 0.0006 |
| Intercept | 890.6 | 55.13 | 16.15 | <0.0001 |
| $R^2$ = 24.8% | | | | |
| Calcium, mg/day | 0.014 | 0.007 | 2.12 | 0.034 |
| Weight, kg | 4.848 | 0.212 | 22.92 | <0.0001 |
| Years since menopause | −4.156 | 0.808 | −5.14 | <0.0001 |
| Age, years | −5.675 | 1.027 | −5.24 | <0.0001 |
| Hormone replacement therapy (N/Y) | 17.99 | 5.002 | 3.60 | 0.0003 |
| Lactose intolerance (N/Y) | −20.15 | 9.269 | −2.17 | 0.030 |
| Intercept | 892.3 | 55.09 | 16.20 | <0.0001 |
| $R^2$ = 25.0% | | | | |

Categorization of dichotomous variables: 0 = no, 1 = yes.

together with the use of supplements explained 0.6% ($p$ = 0.020) of the variation of spinal and 1.1% ($p$ = 0.0002) of that of the femoral BMD. In multiple regression (Table 2), only the intake in 1989 was independently associated with femoral BMD. In multiple regression adjusted for weight, years since menopause, age, and use of HRT, the introduction of calcium intake variables (dairy intakes in 1989 and in 1980 + use of supplements) into the model increased the proportion of the BMD variation explained by 0.4% at both the spine and hip.

*Lactose intolerance and bone density.* The mean crude and adjusted BMDs in the entire study population were 2%–3% lower among women who had reported LI than among other women (Table 3). LI explained 0.3% of the variation of both spinal and femoral BMD, while LI together with dairy calcium intake in 1989 explained 0.5% of the spinal and 0.9% of the femoral BMD variation, and together with dairy calcium intakes in 1989 and 1980 explained 0.6% of the spinal and 1.1% of the femoral BMD. The addition of calcium intake variables [dairy intakes in 1989 and in 1980 + use of supplements (no/yes)] in the analysis of covariance model with age, years since menopause, weight and use (No/Yes) of HRT as covariates increased the explanatory power of the model by 0.2% (spine) and 0.4% (hip). The addition of calcium intake variables in the model slightly decreased the LI effect (Table 3). The effects of calcium intake and LI on BMD did not significantly interact.

*Health status.* A subanalysis of those 349 women (including 14 LI women) who did not report health disorders, HRT or bilateral oophorectomy yielded BMD differences between LI and other women of −6.9% ($p$ = 0.065) at the spine and −10.9% ($p$ = 0.0020) at the hip.

*Estrogen repletion status.* LI did not relate to BMD in premenopausal women, but was related to BMD in postmenopausal women, especially if they had used HRT (Table 4). Bilaterally oophorectomized women had received HRT more often than naturally postmenopausal women (81% vs. 37%) ($p$ < 0.0001).

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**Table 3.** Crude and adjusted bone density means by lactose intolerance status

| | Bone density means, g/cm² | | | |
|---|---|---|---|---|
| | Lactose intolerants | Others | Difference % | *p* value ($R^2$) (%) |
| (A) Spinal bone density (N = 1835) | | | | |
| Crude | 1.097 | 1.129 | −2.8 | 0.016 (0.3) |
| Adjusted Ca | 1.104 | 1.131 | −2.3 | 0.079 (0.8) |
| Adjusted CF | 1.099 | 1.129 | −2.7 | 0.016 (16.5) |
| Adjusted Ca + CF | 1.101 | 1.131 | −2.7 | 0.030 (16.7) |
| (B) Femoral bone density (N = 2010) | | | | |
| Crude | 0.906 | 0.932 | −2.8 | 0.012 (0.3) |
| Adjusted Ca | 0.914 | 0.930 | −1.7 | 0.167 (1.3) |
| Adjusted CF | 0.909 | 0.932 | −2.4 | 0.012 (24.6) |
| Adjusted Ca + CF | 0.912 | 0.930 | −1.9 | 0.075 (25.0) |

$R^2$ = proportion (%) of bone density variance explained by the analysis of covariance model. Adjusted for Ca: calcium intakes (mg/day) in 1989 and in 1980 and current use of supplements (N/Y); CF: weight (kg), age (years), years since menopause and lifetime use of HRT (N/Y).

However, in other respects bilateral oophorectomy behaved as natural menopause. LI women on HRT used corticosteroids (tablets and inhaled) more often than other women on HRT (8.9% vs. 1.7% )($p$ = 0.016). The exclusion of corticosteroid users from HRT users decreased BMD differences (cf. Table 4) from −6.9% ($p$ = 0.0003) to −6.0% ($p$ = 0.014) at the spine and from −5.2% ($p$ = 0.012) to −4.3% ($p$ = 0.046) at the hip.

## Discussion

The purpose of this study was to examine the relationship of self-reported lactose intolerance (LI), calcium intake, and bone density (BMD) among perimenopausal Finnish women. The main findings were that LI women had a lower calcium intake and a slightly lower BMD than women without LI. Calcium intake was particularly related to femoral BMD.

Our results are in line with a few previous studies reporting greater prevalence of lactose malabsorption among osteoporotic subjects than controls.[2,8,19,20] All of these studies reported lower calcium intake among osteoporotic subjects than controls. However, studies which did not show any association between lactose malabsorption and osteoporosis,[1,12,13,24,26] recorded similar calcium intake in LI cases (or osteoporotics) and controls. Two recent studies in particular[5,24] showed that lactase deficiency per se does not significantly affect BMD. Corazza and co-workers demonstrated, in addition, that only symptomatic lactose malabsorption was related to low calcium intake and low BMD in postmenopausal Italian women suspected of having osteoporosis.[5]

Our study design was cross-sectional, though calcium intake and LI were asked approximately 1 year before the densitometry. The main strengths of our study were the large sample size, population-based design, BMD measurements with DXA, and adjustments for putative confounding factors. Weaknesses include postal enquiry of calcium intake and postal enquiry validation of lactose malabsorption. Calcium intake questions were validated against a 4-day food record, but the correlation between our instrument and the standard was modest ($r$ = 0.50).[20] Combining current and past dairy calcium intake or dairy calcium intake and supplements failed to improve the association between calcium intake and BMD significantly.

**Table 4.** Spinal and femoral bone density means by lactose intolerance (LI) and estrogen repletion statuses

| Subgroup | | | Mean (95% CI) g/cm² | Difference LI+ vs. LI− % | *p* value |
|---|---|---|---|---|---|
| (A) Spinal | | | | | |
| Premenopausal[a] at densitometry | | | | | |
| LI | No | (N = 313) | 1.188 (1.172–1.204) | | |
| LI | Yes | (N = 20) | 1.199 (1.150–1.249) | +0.9 | 0.745 |
| Postmenopausal[b] at densitometry | | | | | |
| LI | No | (N = 895) | 1.091 (1.081–1.101) | | |
| LI | Yes | (N = 77) | 1.042 (1.008–1.077) | −4.5 | 0.010 |
| Adjusted[c] | | | | | |
| LI | No | (N = 895) | 1.091 | | |
| LI | Yes | (N = 77) | 1.041 | −4.5 | 0.004 |
| Postmenopausal, HRT[d] | | | | | |
| LI | No | (N = 382) | 1.103 (1.087–1.118) | | |
| LI | Yes | (N = 40) | 1.027 (0.982–1.072) | −6.9 | 0.003 |
| Adjusted[e] | | | | | |
| LI | No | (N = 382) | 1.104 | | |
| LI | Yes | (N = 40) | 1.018 | −7.3 | 0.001 |
| Postmenopausal, no HRT | | | | | |
| LI | No | (N = 505) | 1.083 (1.070–1.096) | | |
| LI | Yes | (N = 36) | 1.058 (1.002–1.114) | −2.3 | 0.341 |
| (B) Femoral | | | | | |
| Premenopausal[a] at densitometry | | | | | |
| LI | No | (N = 341) | 0.969 (0.956–0.983) | | |
| LI | Yes | (N = 68) | 0.951 (0.896–1.006) | −1.9 | 0.522 |
| Postmenopausal[b] at densitometry | | | | | |
| LI | No | (N = 1001) | 0.911 (0.904–0.919) | | |
| LI | Yes | (N = 85) | 0.885 (0.856–0.914) | −2.9 | 0.057 |
| Adjusted[c] | | | | | |
| LI | No | (N = 1001) | 0.911 | | |
| LI | Yes | (N = 85) | 0.887 | −2.6 | 0.054 |
| Postmenopausal, HRT[d] | | | | | |
| LI | No | (N = 414) | 0.921 (0.910–0.933) | | |
| LI | Yes | (N = 45) | 0.873 (0.835–0.911) | −5.2 | 0.012 |
| Adjusted[c] | | | | | |
| LI | No | (N = 414) | 0.922 | | |
| LI | Yes | (N = 45) | 0.869 | −5.7 | 0.003 |
| Postmenopausal, no HRT | | | | | |
| LI | No | (N = 578) | 0.905 (0.896–0.915) | | |
| LI | Yes | (N = 39) | 0.900 (0.854–0.947) | −0.6 | 0.801 |

[a]Last menstruation within 6 months, HRT never.
[b]One year or more since last menstruation.
[c]Adjusted for age (years), years since menopause, weight (kg) and lifetime HRT use (N/Y).
[d]Hormone replacement therapy 6 months or more ever.
[e]Adjusted as above except that HRT was a continuous (years) variable.

Calcium intake relates only modestly to BMD.[6,21,22] One of the main reasons why the verification of this association has been difficult is the lack of accuracy of calcium intake measurements.[7] Self-reported LI, as an indicator of long-term low calcium intake, might, therefore, help to detect this slight bone effect better than food frequency enquiry alone. According to our results, LI increased the proportion dairy calcium intake (in 1989 and in 1980 together) explained of the variation of the spinal BMD from 0.4% to 0.6% and that of the femoral BMD from 0.9% to 1.1%.

The slightly lower BMD (−3%) among our LI women than controls was probably due to their lower calcium intake; at the time of the study, our LI women used 33% less dairy calcium

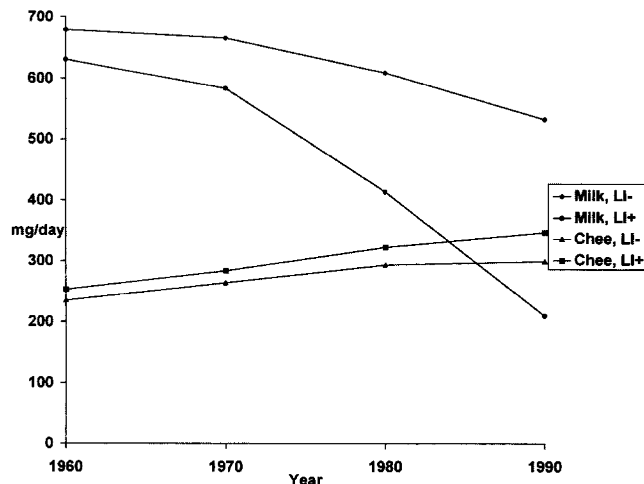


**Figure 1.** Calcium intake (mg/day) from milk and cheese during 1960–1990 as reported by lactose intolerant (LI+) and other (LI–) women in 1990.

than other women. The addition of calcium intake (which was related to BMD) to the multivariate model slightly decreased the LI effect. This suggests that low calcium intake was the actual cause of the low BMD among the LI women. The incomplete accuracy of the food frequency method and the perimenopausal age of our women may have been reasons why this mechanism could not be more clearly shown in our study. One may claim that these LI women were served less milk during growth than other women, resulting in low peak BMD. However, their self-reported milk consumptions at the age of 11–17 were similar (3.7 vs. 3.6 glasses/day, respectively) (Honkanen, Unpublished data). The hypothesis that lactase deficiency impairs calcium absorption from the bowel[4] requires further confirmation.

The prevalence of symptomatic LI in our study population was 8.0%, which is half of the true prevalence (17%) of lactase deficiency in the Finnish population.[23] Most of our LI women (82%) had undergone a tolerance test. It can be assumed that nonsymptomatic lactase deficiency does not affect calcium intake or BMD. Half of our LI women had a total calcium intake from dairy products and supplements of 700 mg or less. Thus, the size of the group at risk of low BMD due to symptomatic LI is not large in the Finnish population.

Our perimenopausal LI women reported a lowered calcium intake during only the last 8 years, on average. Thus, their LI-related bone loss seems to be quite recent. One could expect, therefore, that long-term bone effects due to low calcium intake would be more clearly seen in them later, as bone is more susceptible to calcium deficiency post- than premenopausally.[9] This was also in accordance with our result that the BMD difference between LI women and controls could be seen more clearly post- than premenopausally. Our finding that LI women on HRT had lower BMD than other women on HRT also supports the radical hypothesis that HRT does not prevent postmenopausal bone loss in lactase deficient women. However, it is more likely that such a finding in our study was due to selection. The verification of this radical hypothesis would require a prospective study.

## Conclusion

In conclusion, our results suggest that lactose intolerance predisposes perimenopausal women to a slightly lowered bone density, possibly through diminished calcium intake.

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