Lactose malabsorption and rate of bone loss in older women

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Abstract

Objectives: to study the prevalence of lactose malabsorption with increasing age and to determine whether lactose malabsorbers consume less dietary calcium, have lower bone mineral density or display faster bone loss than lactose absorbers.

Design: 80 healthy Caucasian women aged 40–79 years (20 per decade) were studied for 1 year.

Methods: breath hydrogen exhalation was measured for 3 h after a 50 g oral lactose challenge. Bone density was assessed in the radius, femoral neck, lumbar spine and total body by dual energy x-ray absorptiometry and dietary calcium intake was estimated by 4-day diet records and food-frequency questionnaires.

Results: lactose malabsorption rose with age (15% in those aged 40–59 years versus 50% in those aged 60–79; \( P < 0.01 \)). Malabsorbers aged 70–79 years consumed significantly less calcium than lactose absorbers of this age (\( P < 0.05 \)). Baseline total body calcium values were lower in lactose malabsorbers (\( n = 26 \)) than in lactose absorbers (\( n = 54 \)) but age-adjustment eliminated this difference. Bone change (% per year) was correlated with dietary calcium intake at the femoral neck and trochanter (\( P < 0.05 \)) but was not statistically greater in malabsorbers than in absorbers.

Conclusions: the ability to absorb lactose declines in the 7th decade. This may contribute to decreased dietary intakes of milk products and calcium in elderly women. However, lactose malabsorption without reduction in calcium intake has little effect on bone mineral density or the rate of bone loss.

Keywords: bone loss, dietary calcium, lactose malabsorption, osteoporosis

Introduction

Impaired ability to absorb lactose, the main sugar in milk, has long been considered a risk factor for osteoporosis [1]. The view that lactose malabsorption is associated with a reduced bone density is supported by four lines of evidence. First, an adequate intake of calcium is needed to build and to conserve good bone mass [2–6], and dietary calcium intake (especially from milk) is often considerably lower in lactose malabsorbers than in lactose absorbers [7, 8]. Secondly, some groups have reported a higher prevalence of lactose malabsorption in osteoporotic than in non-osteoporotic populations [9, 10]. Thirdly, lactose may enhance alimentary calcium absorption in lactose absorbers but reduce this in malabsorbers [11]. Lastly, lower bone density values at the hip and spine have been observed in symptomatic lactose absorbers, the osteopenia being ascribed to reduced dietary calcium consumption as a consequence of milk avoidance [8, 12, 13].

On the other hand, many lactose malabsorbers maintain good calcium intakes [14], osteoporotic and non-osteoporotic groups may show similar prevalence of lactose malabsorption [15], the alimentary absorption of calcium is not always diminished in malabsorbers [10, 16], and bone density values in lactose absorbers and malabsorbers may be similar [10, 14, 17, 18]. Thus there no consensus as to whether lactose malabsorption affects bone density adversely.

We recently observed [15] that lactose malabsorption was significantly higher in older women than in young adults (60% prevalence versus 12%). The present prospective study was undertaken to determine in what decade age group the prevalence of lactose malabsorption increased in older New Zealand women. Our study objectives were to evaluate the effects of
advancing age on lactose malabsorption and to
determine whether lactose malabsorbers consume
less dietary calcium, have lower bone mineral density
(BMD) or display faster bone loss than lactose
absorbers.

Methods
Eighty healthy women volunteers aged 40–79 years
(20 per decade) recruited from the community by
advertisement, who were not taking any therapy
affecting bone, were enrolled in a 12-month study.
We were unaware of their customary milk consump-
tion or calcium intakes and no subject had previously
been tested for lactose intolerance. We were careful to
exclude subjects who had undergone gastrointestinal
surgery, radiotherapy, a hip replacement or were
known to have malabsorption syndromes, hyperthy-
oridism, hyperparathyroidism, Cushing’s syndrome or
any known history of bone or stone disease. We also
checked that no antibiotics had been taken within 4
weeks before lactose tolerance testing.

The protocol was approved by our hospital ethics
committee. At the first hospital visit, we tested lactose
absorption, obtained a medical history, estimated
dietary calcium using a food-frequency questionnaire
[15], measured biochemical markers of bone formation
(osteocalcin) and bone resorption (hydroxyproline and
deoxypyridinoline), determined height and weight,
and measured BMD. Subjects were asked to continue
their customary diets and 12 months later the same
food-frequency questionnaire was administered, 4-day
diet records obtained and bone density re-measured.

Lactose absorption was assessed by measuring end-
alveolar breath hydrogen exhalation at 15 min intervals
for 3 h following an oral challenge of 50 g lactose in
200 ml water after overnight fasting, by methods
described previously [15]. Subjects with a rise of
>10 ppm H2 above baseline 60–180 min after consum-
ing the lactose load were diagnosed as malabsorbers.
Serum osteocalcin concentration was measured with a
radioimmunoradiometric assay (Nichols Institute, CA,
USA). Urinary hydroxyproline was determined color-
imetrically [19] and urinary deoxypyridinoline was
estimated with a Pyrilinks-D EIA kit (Metra Biosystems
Inc., CA, USA). Bone density was measured by dual
x-ray absorptiometry (Lunar DPX-L) at the radius, hip,
spine and total body [20]. The precision for BMD
measurements in our laboratory (coefficients of varia-
tion) is: 1.61% for ultradistal radius, 1.52% for 33%
radius, 1.42% for neck of femur, 0.67% for L2–4 and
0.7% for total body.

Statistical analysis
Results are displayed as means and standard errors for
the four 10-year age groups and for lactose absorbers
and malabsorbers. Simple Pearson correlations illus-
trate the associations between age and total body
calcium and between percentage change in BMD and
dietary calcium. We used multiple regression to
investigate the effect of adjusting for age, body
weight, menopausal status and calcium intakes on
the differences between absorbers and malabsorbers at
baseline, and the effect of adjusting for differences at
baseline, age and body weight on the differences
between absorbers and malabsorbers at the end of the
study.

The results of eight subjects were omitted from our
statistical modelling of annual bone change because
during the study two women commenced corticoster-
oid therapy, five started hormone replacement therapy
and one developed malignancy. Only one of the
women excluded was a lactose malabsorber.

Results
Breath hydrogen determinations revealed that 26
women were lactose malabsorbers, while 54 were
lactose absorbers (Figure 1). Twenty-six women were
pre-menopausal (five malabsorbers, 21 absorbers) and
54 were post-menopausal. The prevalence of malab-
sorption rose sharply and significantly in the 7th
decade and remained high and similar in the 7th and
8th decades (Table 1).

Malabsorbers aged 70–79 years had lower calcium
intakes than absorbers, but calcium intakes did not
differ in other age groups and values were high overall.
Malabsorbers over 70 also obtained less of their total
dietary calcium from milk than absorbers (45% versus
58%, P<0.05). Reported frequency of milk consump-
tion was lower in adulthood than in childhood and this
did not differ between malabsorbers and absorbers.
Among malabsorbers, 15% reported previous symptoms

Figure 1. Breath hydrogen exhalation values in lactose
absorbers and malabsorbers after a 50g oral lactose
challenge.
of gastrointestinal discomfort associated with milk drinking, versus 11% of the absorbers (not significant). Calcium intakes remained stable during the study. Mean intakes estimated by the food-frequency questionnaire at baseline and 1 year were not statistically different. There was also good agreement between final dietary calcium estimations by food-frequency questionnaire and 4-day diet records ($r = 0.714$).

Malabsorbers were older and shorter than absorbers, with a significantly lower total body content of both calcium and bone mineral (Figure 2). However, their unadjusted baseline bone densities did not differ at any site. Age-adjustment alone eliminated the differences between lactose absorbers and malabsorbers in total body calcium and bone mineral, showing that raw data values differed solely because malabsorbers were older than absorbers. After adjusting for age, body weight, menopausal status and dietary calcium, no significant effects of lactose malabsorption were found for any bone variable (Table 2). In our study population 35% of malabsorbers and 37% of absorbers had osteopaenia by World Health Organisation criteria (below $−1$ SD of young normal BMD values) while 35% of malabsorbers and 30% of absorbers had overt osteoporosis (below $−2.5$ SD of young normal BMD values) [21].

Analyses of biochemical markers of bone formation and resorption gave similar results in the two groups: the mean levels of serum osteocalcin, urinary hydroxyproline/creatinine and deoxypyridinoline/creatinine in absorbers were $6.38 \pm 0.42$ ng/ml, $0.017 \pm 0.001$ mmol/mmol and $4.05 \pm 0.27$ nmol/mmol, while the mean levels in malabsorbers were $6.22 \pm 0.39$ ng/ml, $0.017 \pm 0.001$ mmol/mmol and $4.85 \pm 0.38$ nmol/mmol.

During the study, average BMD values fell at every measured site except the lumbar spine (Figure 3). We attribute this rise in spinal density to osteoarthritis [22]. In the whole study population, change in BMD (unadjusted data) at the neck of femur ($r = 0.254$, $P < 0.03$, data not illustrated) was significantly correlated with dietary calcium intake (4-day record). Change in BMD at other skeletal sites did not show this relationship.

Although malabsorbers tended to show somewhat higher bone loss than absorbers, group differences were not statistically different and, after adjusting for values at the beginning of the study, calcium intake, age and body weight, the effects of lactose malabsorption on bone variables 1 year later are small and non-significant (Table 3). This was also true if no adjustments were made for calcium intake. There was a tendency for change at most bone sites to be slightly more negative in malabsorbers than absorbers.

**Discussion**

We have confirmed that lactose malabsorption increases in frequency with advancing age in Caucasian New Zealand women [15]. Presumably this rising prevalence of malabsorption is due to declining lactase activity in the ageing intestinal mucosa. Our results establish that the main rise in malabsorption occurs in the 7th decade, 55% of subjects aged 60–69 years displaying poor absorption of lactose in comparison with 15% in women under 60. The prevalence of malabsorption we report is similar to that observed

<table>
<thead>
<tr>
<th>Table I. Prevalence of lactose malabsorption and dietary calcium intakes at baseline in different age groups</th>
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<tbody>
<tr>
<td>Mean value, by age group (years)</td>
</tr>
<tr>
<td>No. of subjects</td>
</tr>
<tr>
<td>No. (and %) of lactose malabsorbers</td>
</tr>
<tr>
<td>Mean calcium intake in mg/day (and SEM)</td>
</tr>
<tr>
<td>Lactose malabsorbers</td>
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<td>Lactose absorbers</td>
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</table>

a $P < 0.01$ versus younger age groups (Fisher’s exact test).
b $P < 0.05$ versus age-matched absorbers.
previously (12% in 50 women aged 17–30 years of age and 60% in 31 women over 60) [15]. Up to 30% of the population may have non-hydrogen producing colonic flora: breath hydrogen testing will underestimate the prevalence of lactose malabsorption in these subjects [23]. Our study design deliberately excluded individuals with malabsorption or previous intestinal surgery who frequently have lactose malabsorption. Thus, the true prevalence of malabsorption in a representative population sample of elderly New Zealand women could be even higher.

A decreased intake of milk, rather than total milk avoidance, generally occurs in lactose intolerance. Although lactose malabsorption was common in our study population, few subjects reported alimentary symptoms, and dietary calcium intakes were quite high. However, our oldest malabsorbers (those over 70 years of age) had both lower milk and lower calcium intakes than age-matched lactose absorbers. In old people, calcium intake and calcium absorption are often low [24]. Perhaps the mild gastrointestinal discomfort associated with the onset of reduced lactase activity may contribute to the decline in milk and dietary calcium intake, which is so widespread among elderly people [24–27].

Symptoms (bloating, diarrhoea, flatulence, pain) associated with colonic hydrogen production from lactose malabsorption can be diminished by altering nutritional habits [28–30]. We suggest that choosing

<table>
<thead>
<tr>
<th>Difference</th>
<th>Crude(^a)</th>
<th>Adjusted(^b)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean 95%CI</td>
<td>Mean 95%CI</td>
</tr>
<tr>
<td>Bone mineral density (g/cm(^2))</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ultradistal radius</td>
<td>0.004 –0.030 to 0.059</td>
<td>–0.023 –0.043 to 0.004</td>
</tr>
<tr>
<td>33% radius</td>
<td>0.031 –0.015 to 0.074</td>
<td>–0.007 –0.039 to 0.024</td>
</tr>
<tr>
<td>L2–4</td>
<td>0.076 –0.015 to 0.166</td>
<td>0.037 –0.049 to 0.122</td>
</tr>
<tr>
<td>Neck of femur</td>
<td>0.054 –0.059 to 0.107</td>
<td>–0.021 –0.081 to 0.039</td>
</tr>
<tr>
<td>Trochanter</td>
<td>0.058 –0.040 to 0.117</td>
<td>–0.009 –0.077 to 0.059</td>
</tr>
<tr>
<td>Total body</td>
<td>0.042 –0.013 to 0.097</td>
<td>–0.001 –0.046 to 0.045</td>
</tr>
<tr>
<td>Total body calcium (g)</td>
<td>77.9 7.3 to 148</td>
<td>19.1 –29.2 to 67.4</td>
</tr>
<tr>
<td>Total body bone mineral (g)</td>
<td>207.9 22.0 to 395</td>
<td>59.6 –67.5 to 186.7</td>
</tr>
</tbody>
</table>

\(^a\)Raw data.
\(^b\)Adjusted for age (years), body weight (kg), menopausal status (yes/no) and calcium intake (mg/day).

CI, confidence interval.

![Figure 3](image3.png)
Figure 3. Annual change in bone mineral density (BMD) at various skeletal sites in lactose absorbers and malabsorbers (raw data). UD, ultradistal radius; 33% rad, 33% radius; NOF, neck of femur.

![Figure 4](image4.png)
Figure 4. Annual change in bone mineral density (BMD) at the neck of femur (NOF) in relation to dietary calcium intake (4-day record) in lactose absorbers and malabsorbers (raw data).
dairy foods which are low in lactose (including cheeses and lactase-treated milks), consuming yoghurt rather than milk [31–34] and taking milk in small regular portions rather than in large single amounts [28] may ameliorate gastrointestinal discomfort in women suffering from lactose malabsorption. This advice could help many elderly women to conserve bone by enabling malabsorbers with gastrointestinal symptoms to keep up their consumption of dairy products sufficiently to cater for their body calcium needs. Calcium supplements should be considered for those with severe intestinal symptoms and low intakes of dairy products.

Since most of our subjects maintained reasonably high calcium intakes, it is perhaps not surprising that, like Slemenda and co-workers [14], we failed to detect any adverse effect of lactose malabsorption on current bone density. However, calcium intakes were reduced only in the oldest malabsorbers and their malabsorption was presumably of relatively recent origin. A detrimental effect of low calcium intake on bone density may therefore take some years to develop. Evaluation of even older malabsorbers could be rewarding in this regard. Men also warrant study.

We have shown that after adjusting for age, weight and dietary calcium malabsorbers did not show either lower baseline bone density values and/or faster change in bone density than absorbers. This is the first time that rate of bone change has been studied in relation to lactose absorptive status. We evaluated change in BMD for only 1 year—a longer follow-up would be useful. However, biochemical markers of bone formation and resorption were also similar in absorbers and malabsorbers, providing further support for the view that bone turnover was similar in absorbers and malabsorbers. Our findings strongly suggest that alimentary absorption of calcium is not seriously impaired in lactose malabsorbers.

Any adverse effects of lactose absorption on bone density are likely therefore to be due to reductions in calcium intake, via milk product avoidance, rather than to impaired absorption of ingested calcium. The fact that the change in bone density was modestly correlated with dietary calcium intake at two sites in the hip supports the view that low calcium intakes accelerate bone loss in elderly women. Perhaps lactose malabsorption acts as a risk factor for osteoporotic bone loss in symptomatic individuals with true lactose intolerance and very low calcium intakes [12, 15]. By contrast, lactose malabsorption unaccompanied by a reduction in dietary calcium appears to have little impact on the pathogenesis of osteoporosis.

Acknowledgements

We thank the Dairy Advisory Board of New Zealand, the Health Research Council and the NZ Lottery Foundation for support.

Key points

- The prevalence of lactose malabsorption rises with advancing age, increasing sharply in the 7th decade.
- Lactose malabsorbers aged 70–79 years consume less dietary calcium and less milk than lactose absorbers, suggesting that lactose malabsorption may contribute to decreased dietary calcium intakes in elderly women.
- Lactose malabsorption without reduction in calcium intake has little effect on bone mineral density or the rate of bone loss.
- Change in bone density per year at the femoral neck correlated with dietary calcium intake.
- Lactose malabsorbers who avoid milky foods should be advised to consume low-lactose high-calcium alternatives such as cheese and yoghurt.

References


Table 3. Differences between lactose absorbers and lactose malabsorbers after 12 months, adjusted for measurements at the beginning of the study, calcium intake, age and body weight

<table>
<thead>
<tr>
<th>Bone mineral density (g/cm²)</th>
<th>Difference</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ultradistal</td>
<td>−0.0025</td>
<td>−0.007 to 0.002</td>
</tr>
<tr>
<td>33% radius</td>
<td>−0.0018</td>
<td>−0.009 to 0.005</td>
</tr>
<tr>
<td>L–4</td>
<td>−0.0054</td>
<td>−0.023 to 0.013</td>
</tr>
<tr>
<td>Neck of femur</td>
<td>0.0015</td>
<td>−0.014 to 0.017</td>
</tr>
<tr>
<td>Trochanteric</td>
<td>0.0052</td>
<td>−0.019 to 0.026</td>
</tr>
<tr>
<td>Total body</td>
<td>−0.0004</td>
<td>−0.010 to 0.009</td>
</tr>
<tr>
<td>Total body calcium (g)</td>
<td>−8.04</td>
<td>−25.5 to 9.44</td>
</tr>
<tr>
<td>Total body bone mineral (g)</td>
<td>−50.04</td>
<td>−73.4 to 13.35</td>
</tr>
</tbody>
</table>

CI, confidence interval.


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