Review: calcium supplementation has a small positive effect on bone mineral density but not fractures in postmenopausal women


Clinical impact ratings GP/FP/Primary care ★★★★★ IM/Ambulatory care ★★★★★ Geriatrics ★★★★★
Rheumatology ★★★★★★

In postmenopausal women, what is the effect of calcium supplementation on bone mineral density (BMD) and fractures?

**METHODS**

**Data sources:** Cochrane Controlled Trials Register (2001), Medline (January 1966 to April 2001), EMBASE/Excerpta Medica (January 1966 to April 2001), Current Contents (review of the 6 mo prior to April 2001), bibliographies of relevant articles, experts, and proceedings of international meetings.

**Study selection and assessment:** randomised controlled trials (RCTs) that compared calcium supplementation (≥400 mg/d) with usual calcium intake where both groups received a maintenance dose of vitamin D <400 IU/day and a vitamin D loading dose <300 000 IU; examined women >45 years who were postmenopausal (absence of menses of ≥6 mo); reported fractures or BMD of the total body, vertebral spine, hip, or forearm; and had follow up for ≥1 year. Three independent reviewers assessed methodological quality including allocation concealment, blinding, and losses to follow up.

**Outcomes:** BMD and fractures.

**MAIN RESULTS**

15 studies (1806 participants, 953 of whom received calcium supplementation) met the selection criteria. 13 studies reported using allocation concealment. Calcium supplementation did not reduce vertebral fractures but had a small positive effect on all BMD measures except BMD at the lumbar spine at 3–4 years (table).

**CONCLUSION**

In postmenopausal women, calcium supplementation has a small positive effect on bone mineral density but does not reduce vertebral fractures.

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### Comment

The meta analysis by Shea et al shows that calcium supplementation slows bone loss and suggests that calcium supplementation modestly reduces the risk of fractures. These moderate effects and limited evidence, however, may be sufficient to support public health efforts to increase calcium intake because calcium is inexpensive and safe.

When people think of bone, they reflexively think of calcium and may believe that osteoporosis is largely a deficiency of calcium that can be prevented by increasing calcium intake. Of course, bone strength is controlled by a much more complex array of factors, including other nutritional factors, genetic programming, locally produced cytokines, circulating hormones, and mechanical influences such as weight and exercise. It is reasonable to expect that, by itself, calcium would have a rather modest effect on risk of fracture. It is also reasonable to expect that calcium may play a supporting role for other factors.

Recommendations on calcium intake should consider the important interplay between this mineral and vitamin D. By itself, vitamin D also reduces the risk of fractures in older adults. Trials have shown that the combination of calcium and vitamin D reduces the risk of fractures in elderly patients with osteoporosis and those in institutional care. A combination of calcium (≥500 mg/d) and vitamin D supplementation has also been the foundation of treatment in all randomised trials of effective pharmacological treatments, and reasonable doubt exists that potent bisphosphonates or parathyroid hormone would work as safely and effectively in patients who have deficient vitamin D or calcium intake. Although the evidence is modest that calcium alone benefits everyone, calcium plus vitamin D (ideally ≥800 IU/d) remains the cornerstone of prevention of fractures in elderly people and patients with osteoporosis. Evidence about the value of vitamin D and calcium for all postmenopausal women and for prevention of conditions other than fracture will soon be forthcoming from the Women’s Health Initiative.

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### Table: Calcium supplementation v placebo on fractures and bone mineral density (BMD) loss in postmenopausal women*

<table>
<thead>
<tr>
<th>Outcomes (follow up)</th>
<th>Number of trials (sample size)</th>
<th>Weighted event rates</th>
<th>BMD site</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Intervention</td>
<td>Control</td>
</tr>
<tr>
<td>Vertebral fractures (2–4 y)</td>
<td>5 (576)</td>
<td>14%</td>
<td>18%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total body (2–4 y)</td>
<td>4 (358)</td>
<td>2.05% (0.24 to 3.86)</td>
<td></td>
</tr>
<tr>
<td>Lumbar spine (2 y)</td>
<td>9 (843)</td>
<td>1.66% (0.72 to 2.39)</td>
<td></td>
</tr>
<tr>
<td>Lumbar spine (3 or 4 y)</td>
<td>2 (218)</td>
<td>1.13% (–0.11 to 2.38)</td>
<td></td>
</tr>
<tr>
<td>Combined hip (2–4 y)</td>
<td>8 (830)</td>
<td>1.64% (0.70 to 2.57)</td>
<td></td>
</tr>
<tr>
<td>Distal radius (2–4 y)</td>
<td>6 (615)</td>
<td>1.91% (0.33 to 3.50)</td>
<td></td>
</tr>
</tbody>
</table>

*Abbreviations defined in glossary; event rates, RRR, and CI calculated from data in article using a fixed effects model.
†Not significant.

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