

PREVENTING OSTEOPOROTIC FRACTURES

I seem to have reached the point where all I have to risk is just my bones

William Faulkner

OSTEOPOROSIS and its associated fractures constitute a huge public health problem. The nationwide cost for direct treatment (inpatient, outpatient and nursing home costs) of osteoporosis-related fractures in 1995 alone was estimated at \$13.8 billion.¹ The cost in Oregon in 2003 for hospital care of osteoporotic hip fractures was \$90 million—and this does not include nursing home costs, outpatient costs, caregiver expenses, lost productivity, or fractures in other sites.

In addition to the economic impact, there are significant human costs. In 2003, 4978 hospitalizations in Oregon were for treatment of hip fractures—91% of these occurred in patients ≥ 45 years and 83% were in those ≥ 65 years old. Using national data we estimate that 77–81% of all Oregon hip fractures are associated with osteoporosis, and 84–88% of hip fractures in patients ≥ 45 were due to osteoporosis.

The estimated lifetime risk of an osteoporotic fracture in a 50-year-old postmenopausal white female is nearly 40%, and her lifetime risk of a hip fracture is 14%. Many fractures in elderly men and other ethnic populations are due to osteoporosis. About 20% of patients with hip fractures die within the first year, and only 1/3 regain pre-fracture functional levels.²

Fracture risk is a function of decreased bone strength and exposure to trauma (e.g., falls). In this *CD Summary* we focus on factors associated with preventing osteoporosis and maintaining bone strength. Practitioners should recognize, however, that preventing falls is an additional effective strategy to minimize fracture risk. (See www.cdc.gov/ncipc/factsheets/falls.htm)

PREVENTION STRATEGIES

The burden of osteoporotic fractures rests primarily on the elderly. Current practices to minimize the morbidity due to osteoporosis include treatments to maintain or restore bone density, e.g. calcium, vitamin D, bisphosphonates, and until recently, hormone replacement therapy. However, the osteoporosis literature suggests prevention strategies that can be considered throughout a patient's lifetime.

BUILD STRONG BONES EARLY!

In addition to targeting the elderly, a reasonable (but less proven) strategy to reduce osteoporosis morbidity is to increase peak bone mass during childhood and adolescence. Higher peak bone mass is associated with a lower fracture risk later in life.³ As the figure below suggests, individuals achieving a higher peak bone mass in childhood and adolescence (solid line) may defer osteoporosis and the associated fracture risk compared to individuals achieving a lower peak bone mass (dotted line).

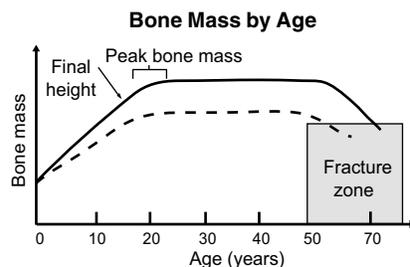


Figure reproduced with permission from BMJ publishing group *Arch Dis Child* 2005; 90:374.

Gains in peak bone mass occur most rapidly during adolescence. Genetic factors account for 60–80% of the variability in peak bone mass, while diet, physical activity, and hormonal status act as important modifiers. Low calcium intake during childhood and adolescence is associated with a lower BMD at age 31,⁴ and lower milk consumption in childhood is associated

with an increased fracture risk.⁵

In Oregon only 10% of 11th grade boys and a mere 4–5% of 11th grade girls estimated their milk consumption over the previous 7 days at 4 or more glasses per day (which supplies 1200 mg of the recommended 1300 mg of daily calcium). Even more alarming, 16% of 11th grade girls reported that they drank *no* milk in the last 7 days.

High-impact physical activity, such as weight lifting or running, increases bone mass in children and teens. In Oregon, only 30% of 11th grade boys report 20 minutes of vigorous activity each day and only 20% of 8th grade girls and 12% of 11th grade girls report this level of activity.

While there is no “smoking gun” linking efforts to increase bone mass in adolescence with decreased fracture risk later in life, the Oregon data suggest that the time spent by health care providers to encourage adequate dietary calcium intake and physical activity in adolescents and children would be time well spent.

WHAT ABOUT SCREENING?

Bone density accounts for about 70% of bone strength, and is a function of peak bone mass and bone loss. Bone mass levels off after the early 20s, and then begins to decline in the early 50s especially in postmenopausal women. The US Preventive Services Task Force recommends that providers routinely screen all women ≥ 65 years old for osteoporosis, and screen those women ≥ 60 years who have additional risk factors for osteoporosis.⁶ This recommendation is based on evidence that bone densitometry accurately predicts the risk for fracture, and that treating asymptomatic women with osteoporosis reduces their risk for fracture.

It is important to note that women with pre-existing atraumatic or minimally traumatic fractures were not



If you need this material in an alternate format, call us at 503/731-4024.

If you would prefer to have your *CD Summary* delivered by e-mail, zap your request to cd.summary@state.or.us. Please include your full name and mailing address (not just your e-mail address), so that we can effectively purge you from our print mailing list, thus saving trees, taxpayer dollars, postal worker injuries, etc.

Age	Provider actions
Childhood and adolescence	Counsel patients about importance of adequate dietary calcium and high impact exercise
Early fifties and postmenopause	Counsel adequate calcium and vitamin D, regular exercise and avoid smoking and excess alcohol
Women ≥60 years with risk factors	Screen for osteoporosis, treatment if appropriate
Women ≥65 years	Screen for osteoporosis, treatment if appropriate
All adults with minimal trauma fractures	Evaluate and treat for osteoporosis if appropriate

considered in the general screening population, because these women already meet the World Health Organization definition of osteoporosis. A pre-existing osteoporosis related fracture is a strong predictor of a future fracture. Consequently, the American National Osteoporosis Foundation emphasizes the need to initiate effective anti-resorptive therapy in women with such prevalent fractures irrespective of the patient's bone mineral density.⁷ Existing data suggest that this opportunity for secondary prevention is frequently missed.

WHAT ABOUT DIAGNOSIS?

A recent study of patients presenting to an emergency room with minimal trauma fractures of distal forearm, spine, pelvis, humerus, ankle and hip, demonstrated that only 1–3% received a diagnosis of osteoporosis.⁸ Fortunately, treatment rates were higher than diagnosis rates: follow-up of these patients showed that 30% received some form of osteoporosis treatment after the first fracture.

Nonetheless, most received only calcium supplements; bisphosphonates were rarely prescribed. Further, 70% of the patients with minimal trauma fractures received no osteoporosis treatment at all.

Closer to home, review of the 2003 Oregon hospital discharge data shows that only 13% of the admissions for hip fractures had a listed diagnosis code for osteoporosis, despite the fact that an estimated 80% of the fractures are attributable to osteoporosis. No data exist on the proportion of Oregon patients who received osteoporosis treatment following their hip fracture. Nonetheless, instituting appropriate osteoporosis therapy after a minimal trauma fracture is another opportunity for Oregon providers to prevent future fractures in high-risk patients.

SUMMARY

Osteoporosis is a disease of the elderly; however, providers may be able to minimize the fracture morbidity and mortality with timely intervention throughout life by: encouraging healthy diets and

physical activity in children and adolescents; counseling postmenopausal women to exercise regularly, avoid smoking, and maintain adequate calcium and vitamin D intake; screening appropriately and treating women ≥65 and those women ≥60 at increased risk; and evaluating and treating those patients who have already sustained a minimal trauma fracture in order to prevent a second osteoporotic fracture.

REFERENCES

1. Ray NF, Chan JK, Thamer M and LJ Melton Medical expenditures for the treatment of osteoporotic fractures in the United States in 1995. *J Bone Miner Res* 1997; 12:24–35.
2. NIH Consensus Statement Osteoporosis Prevention, Diagnosis and Therapy, 2000 <http://consensus.nih.gov/2000/2000Osteoporosis111html.htm>
3. Cooper C, Eriksson JG, Forsen T, et al Maternal height, childhood growth and risk of hip fracture later in life: a longitudinal study. *Osteoporos Int* 2001; 12:623–629.
4. Laitinen J, Kiukaanniemi K, Heikkinen J, et al Body size from birth to adulthood and bone mineral content and density at 31 years of age: results from the N. Finland 1966 birth cohort study. *Osteoporos Int* 2005; Mar 22, Epub (published on line only so far and available abstract on PubMed)
5. Kalkwarf HJ, Khoury JC, and BP Lanphear Milk intake during childhood and adolescence, adult bone density and osteoporotic fracture risk in US women. *Am J Clin Nutr* 2003; 77:257–65.
6. USPTF Screening recommendation for osteoporosis in postmenopausal women. *Ann Int Med* 2002; 137:526–28.
7. National Osteoporosis Foundation, Osteoporosis Clinical Practice Guideline, 1999, available online at <http://nof.org/professionals/clinical.htm>
8. Castel H, Bonneh DY, Sherf M, and Y Liel Awareness of osteoporosis and compliance with management guidelines in patients with newly diagnosed low impact fractures. *Osteoporos Int* 2001; 12:559–64