TREATMENT OF POSTMENOPAUSAL OSTEOPOROSIS
WITH SLOW-RELEASE SODIUM FLUORIDE:
Critique of Final Report from CYC Pak et al

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This final report* of a randomized controlled trial of slow release fluoride given for osteoporosis, is a continuation of the study by Dr. Pak et al first described in 1989 and follows an interim report published in 1994. In this final report the authors describe the results of 54 postmenopausal osteoporotic patients receiving 25 mg of slow-release sodium fluoride twice daily (in cycles of 12 months of medication followed by 2 months not receiving it) and 400 mg of calcium citrate twice daily continuously, comparing their results with 56 similar patients receiving only the calcium citrate and a placebo capsule (without sodium fluoride) twice daily. The authors concluded that slow-release sodium fluoride and calcium citrate administered for 4 years inhibits new vertebral fractures (but not recurrent fractures), augments spinal and femoral neck bone mass, and is safe to use.

In my earlier comment (Fluoride 27 (4) 227-228 1994) I described this study as an exercise in controlled osteofluorosis (osteosclerosis), a well-known phenomenon of excess fluoride intake which augments bone mass and prevents compression deformities but at the risk of eventual spinal stiffness and the formation of imperfectly formed bone with loss of tensile (torsion) strength. Earlier studies of fluoride supplementation at similar dosages resulted in increased bone density but an unacceptable incidence of gastrointestinal problems and an eventual increase in hip fractures incidence.1-3 The slow-release fluoride avoided gastrointestinal problems but leaves open the possibility of eventual increase in hip fracture incidence. Dr Pak et al believe that the lower serum concentrations of fluoride obtained by their slow-release form of sodium fluoride achieve a "therapeutic window" which will also avoid the fluoride-induced damage to bone quality seen in these earlier studies. Since hip fractures produce more serious morbidity than vertebral compression fractures, this matter is of greater importance than merely demonstrating increased bone mass and a decrease in the more minor vertebral compression changes.

In the Pak et al study, the mean duration of treatment was 3.57 years. In the earlier fluoride studies, the increase in hip fracture incidence occurred after 3 years of treatment. Thus the present study may simply be a year or so short of seeing a similar rise in hip fracture incidence. It is likely that, given the lower serum fluoride

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levels, an increase of hip fractures is somewhat delayed by the slow-release form of sodium fluoride given. The authors agree that their treatment: (1) did not alter the rate of fractures occurring on already fractured vertebra (recurrent fractures); (2) “only marginally” affected spinal fracture rate in patients with severe bone loss; and (3) did not appear to inhibit non-spinal fractures. Thus, the jury is still out on the question of hip fractures, especially in women with fairly advanced osteoporosis.

It is clear from a considerable mass of scientific research that osteoporosis is not a disease of fluoride deficiency. Osteoporosis is a multi-factorial metabolic bone disorder involving improper nutrition, lack of exercise, and lack of appropriate hormones. In this latter regard, Pak et al found no discernible benefit from estrogen supplementation. In the US and other industrially advanced countries, estrogen supplementation is the cornerstone of postmenopausal osteoporosis treatment. This is a major error, as the Pak et al study and others demonstrated and is the primary reason why osteoporotic fractures occur at such a high rate in these countries. As I have described in previous reports, treatment that includes attention to nutrition, exercise, and progesterone supplementation results in bone mass improvement and fracture prevention at least as impressive as Pak’s slow-release fluoride treatment and without potential fluoride-induced deleterious side effects.

As is the case in medical problems, the most effective treatments result when the cause of the condition is corrected. Fluoride deficiency is not the cause of osteoporosis. In my opinion, fluoride treatment as Pak et al recommend should still be regarded as controlled osteofluorosis and the 3.5 year trial is insufficient to evaluate the potential increased risk of hip fracture. Further research along the lines I have pioneered is far more likely to achieve proper prevention and treatment of postmenopausal osteoporosis.

References
7 Lee JR. Natural Progesterone: the Multiple Roles of a Remarkable Hormone. BLL Publishing (PO Box 2068, Sebastopol CA 95473, USA), Sebastopol 1993.