DRINKING WATER FLUORIDATION: BONE MINERAL DENSITY AND HIP FRACTURE INCIDENCE

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Abstract from Bone 22 (3) 273-278 1998

The role of drinking water fluoride content for prevention of osteoporosis remains controversial. Therefore, we analyzed the influence of drinking water fluoridation on the incidence of osteoporotic hip fractures and bone mineral density (BMD) in two different communities in eastern Germany: in Chemnitz, drinking water was fluoridated (1 mg/L) over a period of 30 years; in Halle, the water was not fluoridated. BMD was measured in healthy hospital employees aged 20-60 years (Halle: 214 women, 98 men; Chemnitz: 201 women, 43 men respectively) using dual-energy X-ray absorptiometry. Hip fractures in patients ≥ 35 years admitted to the local hospitals in the years 1987-1989 were collected from the clinic registers. There was no difference in age, anthropometric, hormonal, or lifestyle variables between the two groups. Mean fluoride exposure in Chemnitz was 25.2 ± 7.3 years. No correlation was found between fluoride exposure and age-adjusted BMD. We found no significant difference in spinal or femoral BMD between subjects living in Halle and Chemnitz (lumbar spine: 0.997 ± 0.129 (g/cm²) vs 1.045 ± 0.171 (g/cm²), p = 0.08, for men; 1.055 ± 0.112 (g/cm²) vs 1.046 ± 0.117 (g/cm²), p = 0.47, for women). The fracture incidence showed an exponential increase with aging in men and women with an incidence about 3.5 times higher for women. In Chemnitz, we calculated an age-adjusted annual incidence of 142.2 per 100,000 for women; and 72.5 per 100,000 for men, respectively. In Halle the incidences were 178.5 per 100,000 for women and 89.2 per 100,000 for men. There was a lower hip fracture incidence after the age of 85 in women in Chemnitz (1391 per 100,000 in Chemnitz vs. 1957 per 100,000 in Halle, p = 0.006). Using the age-adjusted incidences, significantly fewer hip fractures occurred in Chemnitz in both men and women. In conclusion our study suggests that optimal drinking water fluoridation (1 mg/L), which is advocated for prevention of dental caries, does not influence peak bone density but may reduce the incidence of osteoporotic hip fractures in the very old.

Key Words: Bone mineral density; Drinking water fluoridation; Hip fracture incidence.

Reprints: Dr B Allolio, Medizinische Klinik der Universitat Wurzburg, Schwerpunkt Endokrinologie, Josef-Schneider-Str 2, 97080 Wurzburg, Germany.

COMMENT

The defects in the study are many and include the following.

Due to random variation, any two communities may have different fracture rates that have nothing to do with the factor being studied. This problem can be partially addressed by knowing the fracture rates at different ages in both communities prior to fluoridation of one of them. It may be, for instance, that the fracture incidence among elderly women in Halle has, for many years, been greater than that of the elderly women in Chemnitz. If so, one cannot argue that the difference observed here is due to fluoridation.

Total fluoride intake was not measured or even estimated. This could have been accomplished by urine fluoride tests. It is entirely possible that the total fluoride intake was essentially the same in the women with fractures in both communities.

Past or current estrogen therapy (ERT or HRT) was mentioned as part of the questionnaire obtained but not cited in its Table 2, showing characteristics of the study population. Estrogen is well known to be an anti-resorptive agent and thus a bone factor of some significance.
Water supply source was not identified. The difference between well water and river water used for drinking might be significant. Well water provides not only important minerals but these same minerals would bind to fluoride to reduce F-absorption. Also, if both of these industrial cities were on the same river and river water was their drinking water source, it would be important to know which city was down-stream of the other. Industrial pollutants in the river greatly affect general health and can react synergistically with fluoride.

Both communities are described as industrial cities. If the industrial activity involved coal burning, atmospheric fluoride would significantly nullify any difference in water fluoride intake in these communities.

Bone health involves many other variables, so few of which are considered in this study that no meaningful conclusion can be drawn. This absence of considering other variables is common in the pro-fluoride dental literature.

As the authors admit, fracture incidence in both of these communities is lower than that found in former West Germany. They assert this is due to "unexplained regional differences" which is no explanation at all but allows the inference that communities only 100 Km apart can also have "unexplained regional differences." It is likely, therefore, that other factors, such as diet, specific nutrients, environmental toxins, deaths from other causes, and other factors rather than water fluoridation affected the fracture incidence.

There is another curious thing about this study. Since the study was published in 1998 and the data were collected in 1989, one wonders why it took 9 years to write it? On the other hand, if the study was written in 1998, why choose only the 2-year time span of 1987-1989 instead of, say, a 5-year or 10-year time span for which data clearly were available. Given the known variability of fracture incidence, any given 2-year incidence can not be expected to be as representative as a 10-year mean incidence. Was there something the authors liked about this particular 2-year time span?

Finally, one must look at the one age period in which a statistically significant fracture incidence difference exists between the two communities. Why would women over age 85 experience a higher fracture incidence in Halle than in fluoridated Chemnitz? When fluoridation was started in Chemnitz, in 1959, the 85-year old women (in 1957-1989) were post-menopausal, i.e., when bone remodelling is much less active, weaker bones at age 85 suggest weaker bones prior to menopause. These women were in their late 30's during the war. Was there something about the war experience or the post-war recovery period that adversely affected Halle women more than Chemnitz women? Is this the cause of their increased hip fracture incidence fifty years later? This study provides no information to answer this question. There exists no known mechanism by which fluoridated water could reduce fracture incidence in the very elderly without some sign of the same benefit in earlier age groups. Therefore, it is likely that this isolated divergence of fracture incidence has no relation to water fluoridation. The only finding of this study that seems sturdy is the fact that fluoridation produced no apparent gain in bone mineral density at any age, contrary to the claims of many fluoridation proponents.

The evidence of this study strongly supports the null hypothesis, namely, that water fluoridation had no effect on bone mineral density or fracture incidence.

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[Readers are invited to submit comments on papers published in the professional and scientific literature. Often, assessments of full papers are more helpful than the published abstracts. - Editors]
EFFECT OF CHRONIC FLUORIDE EXPOSURE IN UREMIC RATS
A Dunipace, E Brizendine, M Wilson, W Zhang, C Wilson, B Katz, A Kafrawy and G Stokey
Abstract from Nephron 78 (1) 96-103 1998

This study was conducted to test the hypothesis that the margin of safe fluoride exposure is narrowed in rats that are physiologically compromised by renal dysfunction. The study objective was to determine whether increases in fluoride retention and tissue fluoride levels in rats with surgically induced renal insufficiency result in toxic fluoride effects not ordinarily observed in healthy animals. Uremic and sham-operated control rats received 0 pgiml, 5 (0.26 mmol/l), 15 (0.79), or 50 pgiml (2.63 mmol/l) of fluoride in their drinking water for 3 or 6 months. Fluoride retention was monitored, and, following euthanasia, tissue fluoride and biochemical markers of tissue function were analyzed. Selected tissues were saved for histology, and bone marrow cells were harvested for determining the frequency of sister chromatid exchange, a marker of genetic damage. In spite of significantly higher levels of fluoride in the tissues of the animals with renal insufficiency, there were no clinically adverse, fluoride-induced, extraskeletal, physiological, biochemical, or genetic effects of chronic exposure to common levels of fluoride in these rats.

Key words: Fluoride metabolism; Fluoride toxicity; Rats; Renal insufficiency.
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COMMENT

Many studies from the same source have supported the safety of fluoridated water. The authors’ position is made clear in the first sentence of their Introduction, which states: “Fluoride is widely used for its beneficial effect in reducing dental caries, . . .” and then states of 1 ppm fluoridated water, citing a 1993 review of the US National Research Council: “under normal conditions it does not cause adverse effects.” After reading the full study, I make the following observations:

1) The experiment occupied periods of 3 and 6 months, so is hardly relevant to human populations consuming fluoridated water, as the authors claim.

2) There was a high mortality rate (up to 45%) among the treated groups, clearly a result of the surgery to which the rats were subjected. Although the authors concluded that “there was no association between the level of fluoride exposure and mortality rate”, the possibility that the rats more susceptible to surgical trauma might also, had they survived, been more susceptible to non-skeletal effects of fluoride, does not appear to have been considered.

3) The brain tissue of the rats was not examined. Possible neurotoxic effects were not looked for.

It is interesting to compare these authors’ findings with those of other studies, e.g. the Brazilian one the abstract of which was published in the last issue of Fluoride (31 100-101 May 1998). Over 40 years ago both skeletal and non-skeletal adverse effects on rats fed low levels of sodium fluoride were reported (Ramseyer WF, Smith CAH, McCay CM. Effect of sodium fluoride administration on body changes in old rats. Journal of Gerontology 12 (1) 14-19 1957), but the experiment occupied a longer period of time (520 days) than the Dunipace et al study discussed above.

John Colquhoun
THE ROLE OF THE PUBLIC IN WATER FLUORIDATION: PUBLIC HEALTH CHAMPIONS OR ANTI-FLUORIDATION FREEDOM FIGHTERS?

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Glasgow, Scotland and Newcastle upon Tyne, England.
Abstract from British Dental Journal 184 (1) 39-41 1998

Objective: Using the opportunity of pretesting a leaflet for the general public on water fluoridation, their views were also sought on the issue as a whole.
Design: Qualitative research using focus group discussions led by an experienced moderator.
Setting: Among the general public living in north east England.
Subjects: Members of the public living in both fluoridated and non-fluoridated areas in three age bands (20-35, 36-50 and 50+) and by social class.
Results: The study found: the low priority given to dental health; how emotive the subject of water is; the variable knowledge of fluoride in relation to dental and general health; and the desire for information if new water fluoridation schemes are planned.
Conclusions: The research confirmed public support for water fluoridation but highlighted the place of public health professionals in championing water fluoridation because of public apathy.

COMMENT

The British Dental Journal's acceptance of the above paper, which merely describes canvassing of opinions about artificial water fluoridation and pretesting of a propaganda poster, is surprising, particularly because the conclusions drawn are based on a non-random selected sample size of between 48 and 64 people. The use of the Social Marketing Department of the University of Strathclyde, where three of the authors work, is a novel approach to the controversial issue of water fluoridation, which one could reasonably expect to be settled by scientific discussion.

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List of some other publications

Arnold CM, Bailey DA, Faulkner RA et al. The effect of water fluoridation on the bone mineral density of young women. Canadian Journal of Public Health (Revue Canadienne de Sante Publique) 88 (6) 388-391 1997. (A positive effect is suggested, from a comparison of small atypical samples from two communities.) Reprints: C M Arnold, University of Saskatchewan, School of Physiotherapy, Saskatoon, SK S7N 0W3 Canada.
Lane JM. Osteoporosis - Medical prevention and treatment. Spine 22 (24 Suppl) S32-37 1997. (Suggests fluoride therapy for low bone turnover cases.) Reprints: J M Lane, Hospital for Special Surgery, Osteoporosis Prevention Center, 535 E 70th St, New York, NY 10021, USA.
Lawrence HP, Sheiham A. Caries progression in 12- to 16-year-old schoolchildren in fluoridated and fluoride-deficient areas in Brazil. Community Dentistry & Oral Epidemiology 25 (6) 402-411 1997. (A non-blind study reporting slower progression in 183 from fluoridated central Rio de Janeiro than in 107 from other, "fluoride-deficient", districts on the outskirts. In a further, similar, study on pp 412-418 of the same journal, the authors report radiographic detection of "subtle differences" in progression in the two areas.) Reprints: H P Lawrence, University of North Carolina School of Dentistry, CB 7450 Chapel Hill, NC 27599, USA.

Fluoride 31 (3) 1998
TWO UNANSWERED LETTERS

The following letter received no reply or acknowledgment:

October 15, 1997
Dr. Bruce Alberts, President
National Academy of Sciences
2101 Constitution Avenue, NW
Washington, DC 20418

Dear Dr. Alberts:

As you may be aware, the Dietary Reference Intakes report on calcium, magnesium, phosphorus, vitamin D, and fluoride prepared by the Institute of Medicine of the National Academy of Sciences and scheduled for publication this month, contains a number of recommendations concerning fluoride that are cause for grave concern over their validity for setting public health policy. This concern has been heightened by statements made by speakers and panel members and their responses to queries at the recent September 23rd workshop on the report held at the National Academy of Sciences.

We, the undersigned, regard the problem as so serious that we are requesting you to take immediate steps to delete the fluoride section of the report and to have it re-addressed by a panel that includes members of the scientific community who are not committed to promoting or supporting fluoride use. What follows is a brief summary of the basis for our concern.

At the heart of the matter is whether fluorine, as fluoride (F⁻), should be ranked with Ca, Mg, P, and vitamin D as an essential nutrient. In fact, there is no known essential biochemical role for fluoride in any animal, including humans. The formation of sound, decay-resistant and caries-free teeth as well as strong, sturdy bones, whether in animal or human populations, does not require fluoride, or at least not in more than minuscule, trace amounts. As acknowledged by sources cited in the report, even when a mother’s fluoride intake is elevated, her milk is extremely low in fluoride, but owing to prenatal accumulation, her baby excretes more fluoride than it ingests from her milk. This fact clearly indicates that any natural physiological need for fluoride, if indeed any exists, must be exceedingly small and certainly far below that being recommended in the report.

At the September 23rd workshop, as recorded on videotape, fluoride was repeatedly regarded by speakers and panel members as an essential nutrient. But, toward the end, when challenged on this key issue, Dr. Vernon R. Young, Chair of the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes, bluntly stated that fluoride should not be considered as an “essential” component of the diet. Instead, without clarifying the distinction, he insisted that it should be viewed as only a “beneficial element.”

The fact that fluoride is incorporated into the mineral matrix of bones and teeth does not make it an essential nutrient. Other elements hardly considered essential, such as lead and cadmium, also accumulate in bones and teeth, and they are not regarded as beneficial. Obviously, if fluoride is not essential in human nutrition, any consideration of it in terms of an “adequate intake” is clearly not appropriate and should not be part of a “dietary reference intakes” report.

An association of fluoride with reduction in dental caries is cited as the basis for recommending the various intakes of fluoride. At the same time, the report acknowledges that most of the anti-caries effect attributed to fluoride occurs by topical exposure, not through systemic ingestion. Moreover, large, whole-population studies not cited in the report (e.g., in New Zealand) show that declines in tooth decay over the last 40-50 years...