Correspondence

Dietary calcium deficiency & rickets

Sir,

In the March 2008 issue of the Journal, Teotia and Teotia reviewed their extensive experience of nutritional bone disease in India gained through over 40 yr of research in the field. In one section, they discuss the role of dietary calcium deficiency in the pathogenesis of rickets in children and state categorically that “our observations provide scientific proof of evidence that calcium deficiency alone does not produce rickets” and then go on to suggest that the reports of dietary calcium deficiency are in fact “the syndromes of calcium deficiency and fluoride interactions”. We wish to refute their arguments. In South Africa, where the first cases of rickets caused by low dietary calcium intakes were described in otherwise healthy children, water fluoride was measured from a number of boreholes and surface water in the community of Driefontein from which the children came and all had levels ranging from 0.05-0.1 ppm. It is unclear from where Teotia and Teotia obtained the figures of 5.5 to 14.5 ppm, which they suggest was the water fluoride content in the area of Driefontein. It is possible they are confusing the study with another of ours where we did in fact report on the presence of rachitic like lesions in children living in another part of South Africa where endemic fluorosis was evident and water fluoride concentrations ranged from 8-12 ppm. A number of reports of dietary calcium deficiency causing rickets in Nigeria have also been published. Once again it is unclear where the Teotias obtained their quoted fluoride values (2.4-4.5 ppm) from, as in the Jos region where extensive work on dietary calcium deficiency rickets has been reported, water fluoride levels are undetectable or very low (S. Porter, personal communication). In Bangladesh, dietary calcium deficiency rickets has been reported from the Chakaria region. Again the Teotias quote fluoride levels of 2.9-5.5 ppm, yet measured fluoride levels in the Chakaria region averaged 0.5 ppm with many water samples having undetectable levels (J. Arnaud, personal communication). Thus we have been unable to find elevated fluoride levels in any of the sites where we have described dietary calcium deficiency as a cause of rickets, and thus believe that endemic fluorosis has no part to play (emphasis ours) in the pathogenesis of the bone disease we have reported. However, we also believe that where high water fluoride levels are present, these combine with low dietary calcium intakes to exacerbate rachitic like bone deformities and biochemical abnormalities in children.

Teotias are correct in saying that dietary calcium deficiency probably does not act alone in most situations in the pathogenesis of nutritional rickets. We have suggested that genetic factors, other dietary constituents such as oxalates and phytates, relative boron deficiency and vitamin D insufficiency might all play variable roles. In contrast, the association between aluminium excess and calcium deficiency has been excluded. What is clear, however, is that the bone disease and biochemical perturbations respond completely to an increase in the dietary calcium content alone, indicating that whatever secondary role other factors might be playing, low dietary calcium intakes are primarily responsible.

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Authors’ response

We refute Pettifor’s arguments based on our 40 years of systematized scientific research, observations and conclusions that provide the scientific evidence that calcium deficiency alone does not cause rickets. We are summarizing the errors in Pettifor’s, studies (South Africa) and from Nigeria and Bangladesh.

(i) In 1983 Pettifor presented a paper on calcium deficiency rickets in the conference “Clinical disorders of bone & mineral metabolism” at Detroit, Michigan. I opined that all his patients are of endemic skeletal fluorosis and calcium deficiency interaction syndromes and not of calcium deficiency rickets.

(ii) In 1988 at the same conference, Pettifor presented a poster on rickett skeletal deformities caused by low dietary calcium intake and fluoride synergistic actions in South Africa in areas with water fluoride 8-12 ppm, perhaps due to my comments in 1983 as referred above.

The errors in Pettifor’s, Nigerian and Bangladesh studies include:

(i) Patients reported from S. Africa and Nigeria were only hospital-based and no epidemiological surveys on calcium deficiency rickets in the representative populations to serve as controls. The emerging cross-sectional picture would have provided not only (a) the prevalence of rickets; and (b) the extent to which the rickets is likely to relate to low calcium intakes.

(ii) No study has been undertaken to measure the fluoride content of the drinking water and its aetio-pathological relationship in the causation of rickets in calcium deficient and normal children.

(iii) In our experience, as little as 2 ppm fluoride in drinking water taken continuously for more than six months can produce skeletal fluorosis. Fluoride accumulates faster and greater in the metabolically active bones of the children, inhibits mineralization and causes rickets which is more severe and complex in calcium deficient children.

(iv) Calcium deficiency rickets in S. Africa has been reported around Johannesburg (Southern Transval, South-eastern Transval, Northern Natal, Driefontein, Piet-Retief, Kenhardt) in rural Blacks consuming drinking water from ponds and superficial wells. The concentration of fluoride in the drinking water in these areas ranged from 5.5-14.5 ppm, estimated using

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specific fluoride ion electrode and PHM 64 radiometer during personal surveys of the above areas. The concentration of fluoride in South Africa varied from 0.3-35 ppm to as high as 58 ppm.

(v) Similarly children reported from Nigeria with calcium deficiency rickets were drinking ground water with a fluoride content 2.4-4.5 ppm.

(vi) My visit to Bangladesh (Chakaria region) revealed that the children living in the home for the disabled, developed vitamin D deficiency due to lack of exposure to sunlight (UVR 290-315 nm) and diets severely deficient in calcium and other nutrients. They were drinking water from the superficial uncovered wells and the hand pumps with the fluoride content of 2.9-5.6 ppm. Bone disease and rickets in these children resulted due to the combined effects of calcium deficiency, vitamin D deficiency and fluoride toxicity interactions.

(vii) We are surprised that Pettifor has mentioned the very low (0.05-0.1ppm) water fluoride values in his letter to the editor. The fluoride values had never been mentioned in any of his earlier publications from South Africa and from Nigeria or Bangladesh. Now only he has mentioned the water fluoride values in his letter to the editor.

(viii) Improvement reported by Pettifor in hospitalized children, only on calcium supplementations is not correct. In fact in these children improvement could have occurred due to (a) on hospitalization the exposure of children to endemic fluoride had ceased; and (b) calcium intake and diet had improved. Calcium is the most effective antagonist of fluoride toxicity and inhibits the toxic effects of fluoride on bone and bone mineral metabolism.

(ix) Also, Jackson from Pettifor’s areas of study, reported Kenhardt bone disease (named after the village studied) in rural Blacks and subsequently attributed it to calcium deficiency and fluoride interactions.

(x) There is thus a strong epidemiological and scientific proof that all the children reported as calcium deficiency rickets by Pettifor (South Africa) and from Nigeria and Bangladesh were exposed to high intakes of endemic fluoride and in fact cases of the syndromes of calcium deficiency and fluoride interactions. Jackson’s research and our studies had resolved the existing “Stirred controversy on calcium deficiency rickets” to change medical practice. Each case of calcium deficiency presenting as rickets should be investigated for lack of exposure to sunlight (vitamin D deficiency) and over exposure to endemic fluoride (also fluoridated water) or some combinations.

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References


