Nephrol Dial Transplant (2007) 0: 1–1
doi: 10.1093/ndt/gfm663
Advance Access publication xx xx xxxx, 2007

NDT
Nephrology Dialysis Transplantation

Letter to the Editor

Fluoridation of drinking water and chronic kidney disease: absence of evidence is not evidence of absence

Sir,

Ludlow *et al*. [1] only confirmed that our knowledge of the potential adverse effects of chronic low fluoride supplementation of drinking water on normal or diseased kidneys is insufficient. More than 60 years after water fluoridation, there is no high-level evidence as most published studies are small, have methological deficiencies or are otherwise flawed.

There are two areas of concern regarding the nephrotoxic potential of fluoride. A small and inclusive amount of research suggests that fluoridation of community water actually causes kidney disease. Kidney damage to tubular function and structure, and reduction in glomerular filtration rate occurred in residents of endemic fluoride areas [2] and anecdotal cases of fluoride intoxication [3] suggested a causal relationship between fluoride intake and renal failure. Ludlow *et al.* are correct that no evidence of an increased frequency of kidney disease or tubular dysfunction has been observed in early US epidemiological studies, comparing non-fluoridated areas (0.3 mg/dl) to up to 8 mg/l fluoride in drinking water. None of these studies described renal function of the participants or serial changes in simple urinalysis. Of interest, the data of a recently published study suggested that drinking water contains fluoride levels over 2.0 mg/l—half of the fluoride concentration deemed safe by the US Environmental Protection Agency (EPA)—could cause damage to renal tubular structures in children. This conclusion is based on an investigation of 210 children living in areas of China with varying levels of fluoride in the community water (0.6–5.7 ppm). Children drinking water with more than 2 ppm fluoride were found to have increased levels of NAG and yGT in their urine—both markers of renal tubular damage [4].

It may be stated that there are no known adverse effects associated with the ingestion of relatively low levels of fluoride (1–2 ppm in drinking water) on a chronic basis. However, the actual levels of intake have to include fluoride not only in water, but also in the diet and in other fluoride containing products.

Moreover, a fairly substantial body of research indicates that patients with chronic renal insufficiency are at an increased risk of chronic fluoride toxicity. Patients with reduced glomerular filtration rates have a decreased ability to excrete fluoride in the urine. These patients may develop skeletal fluorosis even at 1 ppm fluoride in the drinking water [5]. Whether or not the body burden of fluoride may further damage the diseased kidneys is unknown. The National Kidney Foundation in its 'Position Paper on Fluoride—1980' as well as the Kidney Health Australia express concern about fluoride retention in kidney patients. They caution physicians to monitor the fluoride intake of patients with advanced stages of kidney diseases. However, a number of reasons will account for the failure to monitor fluoride intake in patients with stages 4 and 5 of chronic kidney diseases and to detect early effects of fluoride retention on kidneys and bone. The safety margin for exposure to fluoride by renal patients is unknown, measurements of fluoride levels are not routine, the onset of skeletal fluorosis is slow and insidious, clinical symptoms of this skeletal disorder are vague, progression of renal functional decline is multifactorial and physicians are unaware of side effects of fluoride on kidneys or bone.

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Received for publication: 22.8.07
Accepted in revised form: 28.8.07