Response: Bone Lead Is Elevated in Renal Failure

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Dear Sir,

We thank Jorens et al. for their comments and interest.

A normal human being inhales about 14 µg lead per day and absorbs about twice that amount from food [1]. About 54–78% of the lead contained in the blood are excreted in the urine. The remainder is eliminated otherwise or dwells in the body [2], primarily in the skeleton, where lead is stored for a long time [3]. Individuals with an impaired capacity of eliminating ubiquitous contaminants are at risk of cumulating these substances in storage compartments (e.g. bone). Bone lead is measured indirectly by means of the EDTA test, which mobilizes lead in the storage tissue and thus causes an increase in blood lead. The chelate-lead complex is eliminated renally. Elevated blood lead levels in subjects with impaired renal function were reported by Szadowski et al. [4] in 1969.

Emmerson [5] and Behringer et al. [6] found a positive correlation between serum creatinine and lead excretion in urine after infusion of EDTA in patients with gout and known exposure to lead. A similar correlation was found by Behringer et al. [6] in patients without gout and known exposure to lead. In addition, they found slightly increased blood lead levels in patients with impaired renal function compared to control subjects without renal disease. These findings were confirmed by Koster et al. [7].

Martegani et al. [8] also concluded to a lead overload in patients with renal insufficiency. They found a linear correlation between serum creatinine and erythrocyte zinc protoporphyrin IX level, the increase of which may reflect a lead overload.

The aim of our study [9] was to gather information on the lead body burden of subjects without known lead exposure at various stages of renal impairment by direct measurement of the lead contents of the storage pool (bone). Neither the control group nor the patient groups had a known past exposure to lead (occupational or domestic); this has been ruled out by exact anamneses. All subjects were living in a rural area and thus had a low common environmental lead exposure. The mean bone lead (iliac crest biopsies) in our control subjects (n = 8) was 1.63 ± 1.02 µg/g wet weight (median 1.15 µg/g wet weight).

Grandjean et al. [10] found a median of 1.4 µg/g lead dry weight of iliac crest biopsies of cadavers in Denmark (n = 16).

The mean bone lead in our CRF patients was 2.18 ± 0.98 µg/g wet weight and in our hemodialysis patients 3.59 ± 2.3 µg/g wet weight (p = 0.034).

A former study examining the bone lead contents of dialysis patients comes from Van de Vyver et al. [11]. They used cadavers as controls (n = 8).

In the cadavers they found a mean iliac crest lead of $7.2 \pm 5.6 \mu\text{g/g}$ wet weight, an astonishingly high value compared with the results of Grandjean et al. [10] and with ours [9]. The values reported for bone lead in the control group by Van de Vyver [11] et al. are even slightly higher than in the dialysis patients: $5.5 \pm 4.6 \mu\text{g/g}$ wet weight.

From their results, they had to conclude that in individuals with normal exposure to lead renal insufficiency alone does not cause an increase in bone lead. However, considering the results of the studies of the other authors

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cited [5–8] and those of our study [9], we think the opposite is true. The mean bone lead of controls (cadavers) in the study by Van de Vyver et al. [11] is surprisingly high, perhaps as a result of unknown past exposure to lead. It is difficult in this case to elucidate the former history of occupational or domestic lead exposure, but a ‘*conditio sine qua non*’ for an exact comparison and for the right conclusions.

We remain convinced – as others – that renal failure plays an important role in the accumulation of lead.

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