Author's response to reviews

Title: No association between the aluminium content of trabecular bone and bone density, mass or size of the proximal femur in elderly men and women

Authors:

Hans-Olov Hellstrom (hans.lov.hellstrom@akademiska.se)
Bengt Mjoberg (bengt.mj@telia.com)
Hans Mallmin (hans.mallmin@akademiska.se)
Karl Michaelsson (karl.michaelsson@akademiska.se)

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Author's response to reviews: see over
Dear Dr Sandra Lee

Regarding your concern about potential duplication of data in the manuscript “No association between the aluminium content of trabecular bone and bone density, mass or size of the proximal femur in elderly men and women” you can see below my clarifications. I have also done a clarification in the manuscript (discussion) regarding this. We also thank professor Karlsson and professor Arokoski for their reviews.

We do not regard this as a duplication of data when the result from previous paper in Osteoporosis International and the data presented in this paper are concerning different outcomes, i.e in previous paper hip fracture and accumulation of aluminium and in present BMD/BMC and bone areas and its association to aluminium. Furthermore are the previous data cited in the article and consequently not concealed.

Please also see the previous comment to Professor Karlsson (below).

Clarification in the manuscript:

Our recent larger case-control study displayed no significant increased risk of hip fracture with high bone levels of aluminium as well as it was an exponential increase in aluminium content in bone with age{{Hellstrom et al., 2005}}. Since age is also an important covariant in our present analysis with BMD and BMC as outcomes we have chosen to also mention the positive association with aluminium content in bone with age.

Magnus K Karlsson
We had several reasons not to include the DXA results in the prior publication published in Osteoporosis International. That study had a focus on hip fractures cases with and without dementia since the pilot study by Mjoberg et al published in Calcified Tissue International some years ago indicated that specifically demented hip fracture cases had high aluminium levels. There has been a debate on the importance of aluminium on the occurrence of dementia and theoretically aluminium might both cause dementia and fractures by effects on the neurons, i.e., the fractures occur by a balance disorder and not by negative effects on bone. The causes of hip fracture and low BMD/BMC/bone areas are not necessarily the same. We also, in that paper, graphically described the variation of aluminium content of bone by age. Our purpose to present some results of the association between aluminium and age in the present manuscript was to motivate for the reader why age was an important confounder since the significant association between aluminium and the DXA variables disappeared after age adjustment. This was also the explanation why we mentioned dementia and osteoarthritis as confounders in the results of the present manuscript. Nevertheless, in an order not to emphasize the age effect, we have now excluded these results from the abstract and from the last paragraph of the discussion. We also thought that it should be too many results in the same manuscript if we included data on age, hip fracture cases with and without dementia and controls, as well as the DXA-measurements. Last, we had not analyzed the DXA results when writing the hip fracture manuscript.)

Kind regards,

Hans-Olov Hellström
Department of Orthopaedics
Uppsala University Hospital
S-751 85 Uppsala
Sweden
Phone +46 18 6114479
E-mail: hans.lov.hellstrom@akademiska.se