

Effetti renali di basse dosi di mercurio

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KEY WORDS

Mercury; nephrotoxicity; proteinuria; autoantibodies

SUMMARY

«Renal effects associated with low exposure to mercury». Objectives: *The present study was aimed at investigating early markers of renal damage and dysfunction in subjects exposed to low doses of mercury from different sources. Different groups of subjects were examined with urinary Hg excretion (HgU) ranging from 0.1 to 35.0 µg/g creatinine: 122 occupationally exposed workers, 22 subjects living in a non-polluted area, but consuming large amounts of tuna and sword fish, and 197 controls.* Methods: *Several markers of renal changes were measured in urine (albumin, fibronectin, β₂-microglobulin, retinol-binding protein, tubular antigens, N-acetyl-β-D-glucosaminidase activity) and serum (β₂-microglobulin and cystatin C). Serum autoantibodies towards collagen, laminin and tubular antigens were assessed in subjects with abnormal renal markers. The role of glutathione-S-transferases GSTT1 and GSTM1 polymorphisms in the inter-individual variability of biological response to Hg was also investigated.* Results: *Renal markers were not correlated with HgU. None of such markers differed significantly between exposed workers and controls, except for urinary β₂-microglobulin, which was decreased in Hg-exposed workers (GM=55.8 vs 86.6 µg/g creatinine), in the absence of any changes in serum concentration. Subjects usually eating tuna and sword fish showed an increased urinary excretion of β₂-microglobulin, albumin and fibronectin. Serum titres of auto-antibodies did not differ between the groups. Neither in controls nor in exposed workers were the observed differences modified by the GSTM1 and GSTT1 genotypes.* Conclusion: *The present study did not provide evidence of any changes in kidney integrity and function in subjects exposed to very low levels of inorganic Hg resulting in urinary Hg lower than 35 µg/g creatinine. Nor did we obtain evidence of Hg-induced autoimmunity towards kidney components. The potential modifying role of GST polymorphisms could not be clarified in the absence of effects associated with exposure to the risk factor, i.e., to inorganic Hg. Preliminary data suggesting nephrotoxic effects of organic Hg from a diet rich in large fish resulting in increased levels of both blood and urinary Hg – which however did not exceed 20 µg/g creatinine – deserves further investigation.*

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