

Meeting abstract

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Investigation of genotoxic effects of inhalative occupational exposure to vanadium: results of a multiple endpoint study

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Animal experiments showed that inhalative exposure to vanadate causes lung cancer. To assess DNA damage in exposed workers (EW, n = 52) of a metal factory, we monitored DNA migration and formation of micronuclei in blood cells of EW, who are exposed to vanadium pentoxide via inhalation and in a matched control group (CG, n = 54). Median level of vanadium serum concentrations of EW (2.23 µg/l) was found to be 7-fold increased compared to CG (0.31 µg/l). In the standard single cell gel electrophoresis (SCGE) assay with leucocytes, no differences were detected between EW and CG, but increased levels of oxidised DNA bases (detected with FPG and ENDO III) were found ($p < 0.05$). Pretreatment of leucocytes with bleomycin resulted in a higher extent of DNA migration (27%, $p < 0.001$) and reduced capacity of DNA repair (by 42%, $p < 0.001$) in EW. Furthermore, the cytochalasin blocked micronucleus (CBMN) assay was carried out with peripheral lymphocytes (n = 24 per group). EW showed a 2.5-fold higher MN frequency as CG and a 7-fold increase of nucleoplasmic bridges (which reflect formation of dicentric chromosomes) was seen. Nuclear buds (attributable to gene amplification events) were 3-fold higher in EW ($p < 0.001$ for all events). Apoptosis rates did not differ significantly between the two groups, whereas necrosis rates doubled in EW ($p < 0.001$). Taken together, our results show increased genetic damage in

individuals which inhale vanadium dust, possibly indicating increased risk for cancer.

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