CELLULAR EFFECTS OF PARTICLES – IMPACT OF DISSOLUTION ON TOXICITY OF MAN-MADE MINERAL FIBERS

K. Luoto1, M. Holopainen2, M. Perander3, K. Karppinen2, K. M. Savolainen1, 4
1Department of Toxicology, and
2Department of Environmental Epidemiology, National Public Health Institute, Kuopio
3Paroc Oy Ab, Parainen
4Department of Pharmacology and Toxicology, University of Kuopio, Kuopio, Finland

SUMMARY

The use of man-made vitreous fibers (MMVF) has grown rapidly because exposure to natural fibers, mainly asbestos, has proved harmful to humans. Biological activity of MMVF made of glass, rock, slag, or other minerals does not depend only on their respirability, but also on their chemical durability and persistency. In the use of MMVF, the goal is to decrease harmful effects of fibers by increasing their dissolution and removal from the lungs. The dissolution of Fe and Al from MMVF is more marked by rat alveolar macrophages (AMs) in culture than by mere medium, whereas medium is more effective than AMs in dissolving silicon (Si) from MMVF. Fe and Al content of the fibers correlate negatively with the fiber Si dissolution by the AMs. Scanning electron micrographs show that MMVF are readily phagocytized by rat AMs in culture. The phagocytosis begins within 30 min after the onset of the exposure and continues for a 96-h observation period. Short fibers, less than 20 μm in length, are readily phagocytized by the AMs whereas longer fibers are attacked with a large number of AMs. MMVF induce also non-lethal changes in the rat AM surface morphology. Before exposure the cells have continuous membranes. The exposed AMs produce extensions which fasten them to the fibers or to other cells to form clumps or clusters of cells and fibers, each cell engulfing part of a fiber. Over 70% of the exposed cells are viable after 96 h of exposure suggesting that MMVF are not acutely toxic rat AMs. MMVF also slightly damage cell membranes and increase the production of reactive oxygen species.

Key words: man-made vitreous fibers, fiber dissolution, rat alveolar macrophages, phagocytosis, cellular toxicity

Address for correspondence: K. M. Savolainen, Department of Toxicology, National Public Health Institute, P. O. B. 95, FI-70701 Kuopio, Finland