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Biological effects of polyvinyl chloride industrial dust

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The object of the present investigations was to study the effects of dust arising from the production of polyvinyl chloride on the organism, with particular emphasis on the pulmonary parenchyma and the upper respiratory passages.

Animals (white rats) were subjected for four hours each day over a period of six months to the inhalation of dust from a polyvinyl chloride composition which is obtained by intensive mixing and subsequent heating at 120 °C of the following components: 25 parts w/w refined chalk, 4 parts w/w lead silicate, 2 parts w/w calcium stearate, 10 parts w/w dibutyl phthalate to 100 parts w/w PVC resin.* The dust concentrations in the chamber were 100-150 mg/m^3, with particle size distributions of 1-3 μm - 76 %, 3-5 μm - 16 %, >5 μm - 8 %. The dust from this PVC composition was virtually insoluble in water and biological media.

The fibrogenous effect on the animals was investigated with reference to the weight coefficient of the lungs (ratio of wet to dry tissue = drying coefficient, ratio of weight of wet lung in mg to weight of animal in g = lung index), and to the content of oxyproline in the lung tissue based on the Neumann/Logan method (1950) as modified by Khvapil (1960). The morphological changes in the lungs and upper respiratory passages were studied by staining the tissue sections with hematoxylin-eosin. The fibrous structures were investigated by means of the Van Heeson and Homor reaction and with fuchsiline using the Weigert method.

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* Recipe as used in the production of imitation leather based on PVC resin.

The functional condition of the pulmonary macrophages in white rats following the inhalation effects from the dust of the above PVC composition in concentrations of 80 mg/m^3 was studied on live animals by staining the macrophages with trephine blue using the method of E M Gramenitskii (1963).

The macrophage reactions were evaluated with reference to the absorption of the dye and its dispersion and concentration. It was found that the experimental animals and the animals in the control group differed with regard to the extent to which the functional condition of the pulmonary macrophages was affected. 72 hours after the introduction of the dye some macrophages were found to be stained dark blue in colour, indicating incipient destruction of the macrophages under the effect of the dust. After 120 hours the number of macrophages destroyed in this way increased, and the macrophages in which the dye appeared in the form of granules diminished considerably. In a number of cases the cytoplasm was observed to disintegrate into individual fragments, and the macrophages were found to "degranulate" and discharge granules into the intercellular material, which could only be taken to indicate destruction of the koniophages.

From the results of the macrophage reactions, therefore, it must be concluded that the PVC dust is responsible for the functional disruption of the macrophages and can even bring about their destruction. The dust must therefore be considered potentially hazardous from the fibrogenous point of view.

This was further substantiated by biochemical and morphological investigations carried out after prolonged inhalation effects.

After 6 months' exposure to the inhalation effects of the above dust no significant difference was found between the weights of the experimental and control groups of animals, although the dry weight of the lungs of the experimental animals was significantly greater than that of the animals in the control group.

The lung index of the experimental group of animals was 27 % greater than that of the control group. The drying coefficient was found to be less informative ($P > 0.05$).

After 6 months' testing the mean content of oxyproline in the lungs of the experimental animals was 69 % greater than that of the control group. The oxyproline content referred to 100 g weight of animal and 100 g dry lung tissue was also

found to have increased by 71 % and 40 % respectively compared with the control group.

The weight coefficient data and the results of the 6 months biochemical investigations thus indicate that the investigated dust is capable of instigating the growth of connective lung tissue.

During the morphological investigations of the trachea and the large bronchi, expressed changes of an inflammatory nature were found together with seats of destruction of their walls. A deep-seated destruction of the microcirculatory bed was observed in the lung tissue in the form of dilation of the plasmorhagia and centres of hemorrhage. An expressed perivascular sclerosis was observed; the walls of the fine and intermediate blood vessels were sclerosed and hyalised. The lumeni of some alveola were filled with a polymorphous cellular exudate.

The interalveolar walls were swollen from the proliferation of cell elements - histocytes, fibroblasts and fibrocytes. Seats of emphysematous dilation were found at various sites. The induced pulmonary process could be characterised as moderately expressed, diffusive, intercellular and productive.

Dystrophic changes in the cell elements of the parenchymatous organs were noted, in combination with an expressed varying degree of destruction of the microcirculatory bed. The liver tissue, for example, showed signs of plethora in the intralobular capillaries located around the periphery of the hepatone, together with some enlargement of the Disse regions. Most of the nuclei were seen to be in a state of increased mitotic activity. A deep-seated, clearly expressed dyscomplexation of the hepatocytes was observed, with vacuolar dystrophy of their cytoplasm.

The kidneys were likewise found to have an expressed disruption of the microcirculation; granular dystrophy of the epithelium of the proximal ducts was observed, whilst the lumeni of the ducts were in many cases filled with albuminous precipitates.

In the heart muscle the changes found were primarily in the microcirculatory bed, in the form of a weakly expressed pericapillary swelling, whilst lymphoid-histocyte infiltration was observed in the capillaries.

The chronic inhalation effects of the above PVC dust accordingly take the form of dystrophic changes in the upper respiratory passages, together, also, with a moderate fibrogenous effect on the lung parenchyma, mainly of a diffuse nature.

The inflammatory changes in the lung parenchyma and the formation of polymorphous cell exudation could be explained by the presence of toxic components in the dust of the PVC composition, whilst the morphological changes in the parenchymatous organs indicate the toxic effects of the investigated dust.

In comparing our data with the results obtained by other authors (A P Golovatyuk, 1967, et al) on the effects of dusts of pure PVC resins, the more expressed pneumonokoniotic and toxic effects should be noted. The 70 % increase in the oxyproline content of the lungs found by us (test of 6 months' duration, concentration: 100 - 150 mg/m^3) is 1.5 times greater than the increase in oxyproline content obtained by A P Golovatyuk from the effects of pure PVC dust (test duration: 6 months, dust concentration: 300 - 400 mg/m^3).

The much more expressed pathomorphological changes in the internal organs compared with A P Golovatyuk's results also testifies to the greatly increased toxic effects of the investigated dust.

The more intense pneumonokoniotic effects of the investigated dust are undoubtedly due to its lead silicate content, whilst the toxic effects must be attributed to the presence of the dibutyl phthalate.

In view of the foregoing, therefore, we consider the investigated dust to be more highly active than pure PVC dust, and we accordingly recommend that its MAC value be reduced.

Circulation

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