Short Communication

EFFECTS OF GLASS FIBER EXPOSURE ON RAT LUNGS

Key words: glass fiber; inhalation exposure; rat lung; pulmonary inflammation; hydroxyproline

As is generally known, asbestos is physicochemically and economically suitable for building materials. However, respirable fine fibers of asbestos have been confirmed to cause asbestosis, bronchial cancer and pleural and peritoneal mesothelioma. As alternatives of asbestos, man-made mineral fibers such as glass fibers, ceramic fibers and others have been developed, but how they affect a person's health has not yet been fully clarified. The aim of this study was to ascertain whether respirable glass fibers are harmful to health. Experiments were designed to investigate the possible involvement of bronchoalveolar inflammation and pulmonary fibrosis in male rat lungs exposed to glass fibers. To achieve the aim, protein and neutral sugar contents in the lung lavage fluid and the content of hydroxyproline in the lung were investigated.

Materials and methods. Specific pathogen free, 5-week-old male Wistar rats were exposed to glass fibers for 6 h/d, 5 d/wk for 4 wk in stainless steel chambers of 480 l. Dust clouds of the glass fibers were generated by a special dust generator using a disintegrated binderless glass fiber filter (GB-100R, Toyo Roshi, Co., Japan), whose silicon content was 29%. Their mass median aerodynamic diameter measured by an Andersen sampler was 2.8 μm. Their mean diameter and length determined by a scanning electron microscope were 0.51 and 5.5 μm, respectively. The average exposure concentration was 10.8 ± 3.3 mg/m³. Some of the animals, usually 5 rats at each time, were sacrificed at 24 h after the scheduled exposure-time. The remaining rats were sacrificed at 1 wk, 6 months and 1 yr after the exposure. The animals were anesthetized with an intraperitoneal injection of sodium pentobarbital, and after opening the chest and obtaining blood from the heart, the rats were exsanguinated by severing the abdominal aorta. After exposing the trachea in situ, a slit was made just below the larynx and a catheter was inserted. Phosphate buffered saline (5 ml/g lung) was injected into the lungs and then withdrawn. Four additional lavages were done using fresh saline, after which all the lavage solutions were pooled. Cells were removed from the pooled lavage solution by centrifugation at 700 × g for 10 min, and the resulting supernatant was further centrifuged at 15,000 × g for 30 min at 4°C to remove crude pulmonary surfactant. The lung lavage fluid thus prepared was stored at −80°C. Protein was determined by the Lowry method using bovine serum albumin as standard. Neutral sugar was measured by the Anthrone-sulfuric acid method using glucose as standard. The lungs were homogenized in 50 mm phosphate buffer (pH 7.5) and centrifuged at 1,500 × g for 10 min. The precipitate was washed with distilled water and dissolved in 0.1 N NaOH at 95°C for 45 min. After centrifugation at 1,500 × g for 10 min, the resulting supernatant which contained dissolved collagen fibers was hydrolyzed with 6 N HCl at 116°C for 20 h. Hydroxyproline thus prepared was determined by the Berg method.

Results and discussion: After exposure, glass fibers were detected microscopically and chemically in the rat lungs and were shown to be respirable. The deposition and clearance rates of the inhaled glass fibers have been reported elsewhere. Effects of the glass fiber exposure on the content of protein in the lung lavage fluid of rats are shown in Fig. 1. Significant increases in the protein content were observed at 3 and 4 wk of exposure, but the observed increase was very slight. No significant difference between the exposed rats and control could be detected at any other time. A much longer exposure experiment might be needed to confirm such a weak effect, because Lee et al. reported reversible protein accumulation in alveoli by 3 months' exposure to fiberglass.

A significant increase and a decrease in the neutral sugar content in the lung lavage fluid of the rats were observed at 2 wk of exposure and 1 wk after exposure, respectively (data not shown).

It seems likely from the foregoing results on protein and sugar accumulation that the glass fiber exposure caused slight inflammation in the rat lungs.
Contents of hydroxyproline in the rat lungs are shown in Fig. 2. Although an increase in the content accompanied by growth was detected, no significant difference between the exposed and the control was observed during and after the exposure. This result was consistent with early inhalation experiments,8,9) but two studies10,11) have observed a relation between glass fiber exposure and fibrosis.

In conclusion, under the conditions of the present experiment, the glass fiber exposure resulted in slight pulmonary inflammation and little effect on pulmonary hydroxyproline content.

References


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