CARBORUNDUM PNEUMOCONIOSIS

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INTRODUCTION

Silicon carbide (SiC) also called carborundum is a universally used abrasive produced by the fusion of high-grade silica and finely ground carbon in electric furnace at 2400°C. A recent review of the subject in Parkes textbook[6] led to the conclusion that there was no evidence that exposure to silicon carbide dust gave rise to a pneumoconiosis.

Nonetheless, pneumoconiosis has been reported in long term workers engaged in the manufacturing of SiC and it was suspected that sick workers had been previously exposed to other dust hazards or to quartz dust in the raw materials of the manufactures. One pathological report raised the possibility of a carborundum pneumoconiosis.[4]

Recent investigations in the Quebec carborundum industry have documented an excess of radiographic abnormalities compatible with pneumoconiosis, particularly in older workers of the industry.[7] In view of these findings, collaboration with the industry was established and a thorough multidisciplinary investigation was initiated.

Pathological studies of available lung tissue from long term workers of the SiC industry were analyzed in detail.[5] Recent chest radiographs of 1984 and 1979 of some 128 workers were reviewed.[3] Evaluation of occupational exposures in the industry was conducted and reported in detail.[1,2] A review of the occupational hygiene led to the preparation and characterization of reference samples of respirable particles collected in the SiC production industry for use in this experimental research.

The objectives of this study were to evaluate the in vivo lung biologic activity of the mineral dusts found in the carborundum manufacturing industry, to identify the offending dust in order to establish appropriate control of dust level in the workplace.

MATERIALS AND METHODS

Experimental Design

Seventy-two sheep were used in this study. The flock was divided into 9 groups of 8 sheep. Following pre-exposure (control) studies, the sheep tracheal lobe was exposed to:

- 100 ml saline = Sa group (control);
- 100 mg latex beads in 100 ml saline = latex group;
- 100 mg graphite in 100 ml saline = graphite group;
- 100 mg SiC raw particles in 100 ml saline = SiCp group;
- 100 mg SiC ashed particles in 100 ml saline = SiCpa group;
- 100 mg Minusil-5 in 100 ml saline = Si group;
- 100 mg crocidolite fibers in 100 ml saline = Cro group;
- 100 mg SiC raw fibers in 100 ml saline = SiCf group;
- 100 mg SiC ashed fibers in 100 ml saline = SiCfa group.

Exposure to the tracheal lobe was carried out via bronchoscopic infusion of the suspension in the lobe. The animals were studied prior to exposure and post-exposure at month 2, 4, 6, and 8 by BAL and by histopathological methods at month 8.

The Minerals

The materials for exposure were obtained from the following sources: Latex beads from Sigma Chemical Co., St-Louis, MO; these particles are uniform in size with a mean diameter of 1 μ. Minusil-5 from Pennsylvania Glass Co., Pittsburgh, PA; the silica particles are well characterized with 99.9% of diameter $< 5\,\mu$, and 95% $< 1\,\mu$. Crocidolite fibers from the Union Internationale Contre le Cancer (UICC); these fibers have a known diameter of $0.17 \pm .01\,\mu$ with an average length of $3.9 \pm 0.2\,\mu$ 95% of fibers with a length $< 10\,\mu$, 82% $< 5\,\mu$.

All other samples were obtained from the Quebec SiC industry, prepared and characterized. Briefly, these materials were collected from the production sites in the Acheson furnaces of two Quebec SiC plants. The non-fibrous SiC was collected from the center of large lumps of produced materials. The SiC fibers were collected mainly from the outside part of the main cylindrical lump produced by the process. The graphite was extracted from the core of a fired Acheson furnace. The raw particulate and fibrous SiC were, as expected, contaminated with graphite flakes on surface, which at least in theory, could alter biological activity. To eliminate these contaminants, reference samples of fibrous and particulate SiC were ashed. Graphite particles were 98.8% $< 5\,\mu$, particulate SiC raw or ashed were 99.5% $< 5\,\mu$. For the SiC fibers, seventy percent of fibers were less than 5 μ, with some longer than 20 μ. The fibrous SiC raw or ashed were of an average of $0.27\,\mu \pm 0.27$ diameter with an average length of $6.8 \pm 11.2\,\mu$. These morphometric data were considered in the

selection of the asbestos fiber crocidolite, for comparison in these experiments.

Assessment of Lung Reaction

To evaluate the disease process induced by exposure to these respirable minerals, we looked at lung lavage cellularity and biochemistry as biologic indicators of alveolitis. The severity of lung tissue damage was evaluated at autopsy, 8 months after exposure, by histopathology. To assess interstitial lung matrix changes we looked at the glycosaminoglycan accumulation in BAL fluid. We also measured the production of fibronectin by BAL cells in culture.

Lung lavage fluid was analyzed for the presence of molecules capable of enhancing fibroblast proliferation.

Histopathology

At month 8 of the study, all sheep were sacrificed and the lungs removed from the chest cavity. The tracheal lobe was identified and 9 samples of the lobe of each sheep were obtained each time for microscopic examination.

RESULTS

The Particulates

In comparison to Sa group, all the particulate exposed groups had a slight and transient early increase in cellularity except for the Si group, which had an early 500% increase in cellularity which decreased to 250% at month 4, but remained elevated to the end of experiment. This was largely due to increase in the macrophage population, but increases in lymphocytes and neutrophils were also significant and sustained in the Si group. Similarly, in the biochemical and cell culture analyses, only the Si group had significant increases in BAL lactate dehydrogenase, glycosaminoglycan, and increased production of fibronectin and fibroblast growth activity. The lung morphology of the sheep was normal in groups Sa and latex. The lung tissue of sheep in the graphite group, SiCp and SiCpa groups contained accumulation of particles in alveoli and interstitium without cellular reaction. In the graphite exposed sheep, the morphologic changes are reminescent of the early simple pneumoconiosis of coal workers.[6] In the Si exposed sheep, the lung changes are characterized by a diffuse alveolitis with early nodular silicotic lesions as reported in our earlier studies. The pathological scores were 0 ± 0 for Sa group, latex group, graphite group, SiCp group and 2.9 ± 1.0 for the Si group ($p < 0.01$ for Si group vs others).

The Fibers

Briefly, cellularity of BAL was increased in all the fiber groups following exposure with a larger attenuation for the SiCf group. This effect was pancellular and was also seen in the pattern of response of LDH overtime. Fibronectin production was significantly increased, again with some attenuation for the SiCf group. Fibroblast growth activity was increased significantly in all fiber groups.

Pathologic analysis of lung tissue in the crocidolite exposed sheep revealed a peribronchiolar fibrosing alveolitis as previously reported in other asbestos exposure. In the fibrous carborundum exposed sheep, we found nodular lesions in the parenchyma which were not located around the bronchioles. These nodules were composed of multinucleated macrophages, monocytes and a few neutrophils and contained several carborundum fibers and "bodies." The intensity and profusion of lesions in the fiber groups were: 1.9 ± 0.25 for Cro group, 1.2 ± 0.21 for the SiCf and 1.6 ± 0.2 for the SiCfa group ($p > 0.05$ between the 3). The slightly lower score ($p > 0.05$) in the SiCf compared to SiCfa could suggest a partial inhibitory effect of the graphite on surface of the SiCf fibers.

DISCUSSION

In recent years, several epidemiological and clinical investigations have suggested that carborundum workers may have a specific occupational lung disease. This experimental study of the airborne dust particles and fibers in the carborundum industry provides significant new information on the pathogenesis of interstitial lung disease in workers of that industry. It documents that of all the non-fibrous particulate minerals encountered on site, only silica appears to be potentially responsible for some of the lung injury. In addition, this study documents clearly that fibrous SiC has significant biologic activity and can initiate a fibrosing lung disease. In SiC manufactures, SiC fiber inhalation can contribute to the genesis of an interstitial lung disease. Therefore, it is appropriate to recommend that surveillance of the work environment in that industry should include an assessment of airborne fiber levels.

REFERENCES

1. Dufresne, A., Lesage, J., Perrault, G.: Evaluation of Occupational Exposure to Mixed Dusts and Polycyclic Aromatic Hydrocarbons in Silicon Carbide Plants. *Am. Ind. Hyg. Assoc. J.* 48:160-166 (1987).
2. Dufresne, A., Perrault, G., Sébastien, P., Adnot, A., Baril, M.: Morphology and Surface Characteristics of Particulates from Silicon Carbide Industries. *Am. Ind. Hyg. Assoc. J.* 49: in press (1988).
3. Durand, P., Bégin, R., Samson, L., Cantin, A., Massé, S., Dufresne, A., Perrault, G., Laflamme, J.: Silicon Carbide Pneumoconiosis: Current Exposure Levels do not Change the Radiographic Assessment of Disease. Submitted for publication, 1988.
4. Funahashi, A., Schlueter, D.P., Pintar, K., Siegesmund, K.A., Mandel, G.S., Mandel, N.S.: Pneumoconiosis in Workers Exposed to Silicon Carbide. *Am. Rev. Respir. Dis.* 129:635-640 (1984).
5. Massé, S., Bégin, R., Cantin, A.: Pathology of Silicon Carbide Pneumoconiosis. *Modern Pathology* 1:104-108 (1988).
6. Parkes, W.R.: *Occupational Lung Disease*, 2nd Ed. Butterworth, London, U.K. (1982).
7. Peters, J.M., Smith, T.J., Bernstein, L., Wright, W.E., Hammond, S.K.: Pulmonary Effects of Exposures in Silicon Carbide Manufacturing. *Br. J. Ind. Med.* 41:109-115 (1984).

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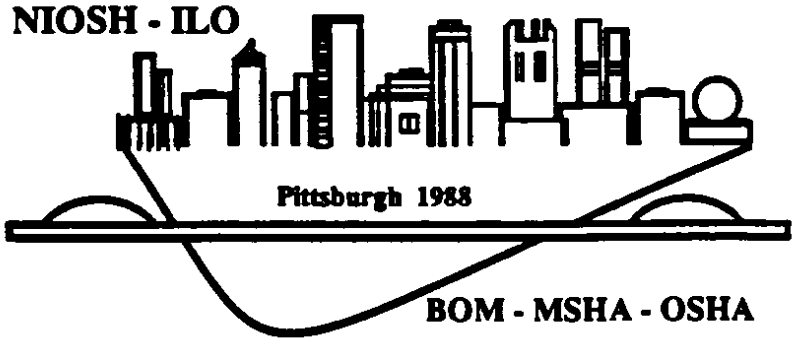

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