

Early events of experimental exposure to amorphous and crystalline silica in the rat: time course of surfactant protein D

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SUMMARY

Pneumoconioses determined by chronic inhalation of different kinds of silica present with peculiar clinical and histopathological features. Silicosis, caused by crystalline silica, is characterized by typical fibrous parenchymal nodules. Less defined are pneumoconioses due to amorphous silica.

Aim of current experimental research on silicosis is to investigate the early events that lead to nodular fibrosis of the lung. A secretory component of the pulmonary environment, surfactant, seems to be involved in silica toxicity; surfactant protein D is a protein constituent, apparently involved in the homeostasis of the phospholipid component. We studied the behaviour of SP-D 2, 12 and 24 hours after treatment with 200 mg/kg crystalline silica or pumice powder suspended in 400 µl/kg saline solution and instilled intratracheally to rats. Both immunohistochemical localization and immunoblotting quantification demonstrated a sensible increase in intracellular SP-D, localized in alveolar type II cells and some bronchiolar epithelial cells, 2 hours after treatment. Increment appears less marked 12 hours after administration, reaching again levels comparable to control at 24 hours. The behaviour of SP-D after pumice instillation is similar, but with a significantly minor increment at 2 hours. These results indicate crystalline silica as responsible for a stronger acute injury of pulmonary tissue.