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PULMONARY SURFACTANT SECRETION AS A BIOCHEMICAL ASSAY FOR NON-SPECIFIC LUNG INJURY CAUSED BY CHEMICAL AGENTS

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Whether inhaled, or carried to the lung by the blood following parental administration, chemicals may cause y which is expressed as impaired metabolic function as well as morphological change. Attempts to develop a biochemical as an indicator of the degree of chemical injury are limited by the cellular complexity of pulmonary tissue. There are 40 different cell types in the lung. Measurement of parameters such as nucleic acid synthesis generally yields data are difficult to associate with a particular cell type. Moreover increases in synthesis in one type of cell may be t by a reduction in synthesis in another, with the result that specific changes may be undetected. Many biochemical s are sensitive under laboratory circumstances where high to massive levels of chemical injury are employed. However, tion of levels of exposure toward that comparable with environmental circumstances may be concomitant with reduced bation of biochemical parameters to a point which precludes distinction between exposed and non-exposed animals.

Pulmonary surfactant is a chemically complex protein-lipid substance secreted into the alveolar spaces by the pneumocytes. The main function of surfactant is to decrease surface tension and to stabilize surface forces between and small alveoli during the inflation/deflation cycle. In our experiments, measurement of the rate of surfactant ion, which is a specific differentiated function of a single cell type, has proved to be of value as a biochemical tor of lung damage. The inhaled chemical agents investigated include pollutants such as petrol vapour and cigarette and toxins carbon tetrachloride and trichloroethylene. Ultrastructural examination of the tissues was carried out at ep in attempt to correlate biochemical perturbations with morphological alterations at the electron microscopic level.

Inhalatic of petrol vapour or cigarette smoke for up to 45 days reduced the surfactant yield to less than 50% of s. Although the initial decrease in surfactant level occurred during the first 15 days of exposure, the reduced level ntained throughout treatment. Injury evoked by carbon tetrachloride and trichloroethylene produced a similar massive on in surfactant secretion, with inhibition being consistently greater with carbon tetrachloride. The relative y of these agents is apparent in so far as similar depression of surfactant yield following daily exposure for 8 hours ol vapour and 30 minutes to carbon tetrachloride or trichloroethylene. Dose responses have been obtained, indicating nibition of surfactant secretion is proportional to the amount of petrol or carbon tetrachloride vapour administered. r trend appears to be the case following treatment with cigarette smoke. The injury evoked by these agents is d in the patterns of morphological response determined by electron microscopy. The classic trend of pulmonary injury very, exemplified after intratracheal instillation of 3-methylcholanthrene, is found to vary in intensity with the t agents tested. The present data support the concept that surfactant production is a significant target for the f toxic agents, and postulate the usefulness of surfactant production as a sensitive biochemical assay for ific lung damage.

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