Cellular Function in Occupational Lung Disease

New Directions

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Studies of cellular function in occupational lung disease have mainly focused on alveolar defense mechanisms and the response to injury of the parenchymal lung cells. Additionally, these studies have been influenced by the prevalence of diseases caused by fibrogenic “dusts” such as silica and asbestos. While much remains to be explored in these areas, we suggest that two new directions of research in the epidemiology and the cellular pathology of lung disease will become progressively more important.

AIRWAY DISEASE

Occupational asthma is becoming more commonly recognized. The precision with which the specific causal agent may be recognized has improved as bronchial challenge tests have become more widely used. However, there is an additional airway disease which requires more intensive study, namely “industrial bronchitis.” This entity is poorly defined and there are difficulties in distinguishing between bronchitis of occupational origin from bronchitis resulting from cigarette smoking. Likewise, any potential synergism between cigarette smoke and inhaled materials present in the workplace will require considerable precision in its definition.

These aspects of airway disease have also been considered in the relationship between lung disease and air pollution. In an extensive and careful study, Bouhuys and colleagues looked at airway function and the prevalence of “bronchial catarrh” (cough and sputum) in several different populations exposed to significantly different levels of air pollution in the urban and rural environments. These workers took careful cognizance of the effects of age, sex, race and smoking habits. They then described differences in the prevalence of cough and sputum in these populations, but could find no physiologic differences between these groups in spite of a detailed analysis of carefully performed expiratory flow-volume curves. The absence of physiologic differences made some environmentalists distinctly unhappy! The differential prevalence of cough and sputum gave the environmentalists some support! Does the increased prevalence of the latter mean air pollution causes “disease but not disability”? Does the absence of airway functional derangement reflect insufficient differences in pollution levels? These and other questions are either not yet answered, or alternatively the answers depend on one’s subjective value judgments. There is a slight increase in reported cough and sputum—a “disease without disability”? Is it a fair price to pay for society’s industrialization?

The problem of indoor pollution in the workplace may affect fewer persons than those potentially subjected to general environmental perturbations; however, these problems are in some ways more susceptible to precise analysis. The levels of “dust,” and the relatively specific nature of components of such indoor pollutants (cutting oils, coolants, metal dusts, polycyclic hydrocarbons and materials derived from the polymer industry) are more definable. The levels within the workplace air are likely to be quantitatively greater than those present even in the worst external urban environments. As such, the answers derived from the studies of “industrial bronchitis” could assume a broad ecologic significance. We would suggest that one major development in the study of occupational lung disease will focus on “bronchitis.”

Aside from the epidemiologic approaches, there is potential in examining this problem at the cellular level. Tracheal organ culture is a well-established technique. Materials suspected, on epidemiologic grounds, to be causes of industrial bronchitis, can be utilized to define several types of mucosal response. Such responses include glandular hyperplasia, mucus hypersecretion and both hyperplastic and metaplastic changes in bronchial epithelia. The
latter, of course, have implications of carcinogenesis. An exciting recent publication has demonstrated metaplastic changes occurring in the trachea exposed \textit{in vitro} to amphibole asbestos \cite{4}. More importantly, Mossman and colleagues \cite{4} have shown such effects to be prevented by the incorporation of vitamin A analogs into the culture medium. Thus, bronchial responses to at least one airborne hazard can be defined, and more importantly, prevented.

\textbf{Metals and Lung Disease}

The study of the effects of mineral dusts on lung cell function have become a tradition! However, review of the literature on the effects of metals on cellular function shows that while much is known of the systemic effects of lead, cadmium and mercury, remarkably little is known about the effects of specific metals and alloys on both nasal and lung function. The involvement of beryllium in sarcoid is well recognized and more recently the sequestration of insoluble beryllium salts in macrophage lysosomes has been described \cite{5}. We suggest that the fibrogenic responses of the lung to such metal mixtures as tungsten carbide/cobalt as used in high-speed steels, is another disease ripe for study at the cellular level. Similarly, is beryllium the only metal capable of causing a granulomatous reaction in the lung? What is it that makes inhaled iron so relatively non-toxic to the lung, whereas the high-speed steels appear to be injurious? Research on this aspect of “lung metallurgy” could well follow similar lines used to elucidate the relative toxicity of various forms of asbestos. We suspect that this aspect of occupational lung disease will prove to be worthy of exploration.

\textbf{References}

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