The Pulmonary Response to Fibrous Dusts of Diverse Compositions

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ibrous quartz, chrysotile asbestos, and tremolite talc dust, all of respirable particle ze, injected intratracheally, produced polypoid proliferative inflammations with- maller air-conducting tubes as well as more peripherally. With time, the inflam- ory tissue became converted into collagenous scars which often caused permanent deformities of bronchi and bronchioles. After intratracheal injection of a fibrous dust such as synthetic chrysotile, ceramic aluminum silicate, silicon carbide, glass, or brucite, the main pulmonary response was a macrophage reaction with minimal stromal participation. In addition, within 4 days after the injection, there were foci of polypoid proliferative inflammation but limited to the more peripheral respiratory bronchiole and alveolar ducts. Because these polypoid lesions did not collagenize and did not destroy the anatomic integrity of the air spaces, and because the lesions were reversible, the dusts calling forth this type of response must be classed as biologically "inert." Furthermore, the polypoid lesions are believed to be artifactual in the sense that their production is determined by the method of introducing the dust into the lungs, since such lesions are not seen in animals *inhaling* high concentrations of the same dusts.

Introduction

WITH THE INCREASING production and use of fibrous materials, both the naturally occurring and those industrially produced, the dust created by their fragmentation is becoming more prevalent. We know that one type of naturally occurring fibrous dust, namely asbestos, is biologically active and is capable of causing extensive and fatal scarring of the lungs, and some kinds of this mineral have been associated with the production of cancer.

Inasmuch as it is not known exactly what is about the asbestos dust particle that is responsible for its pathogenicity, the simplest explanation which seemed to be attractive to many in the past, was that the pathogenicity of asbestos, and, therefore, of all fibrous dusts, was related to the fibrous shape of the particles. According to this theory, when the fibers are inhaled, their sharp ends traumatize the cells they contact, and fibrosis results from the multiple traumata.

Although some years ago we had investigated the pulmonary response to one industrially produced fibrous dust, namely ceramic aluminum silicate fibers, and found it to be biologically "inert"[1] in recent years the needle-like character of fibrous dust has again been implicated as the pathogenic factor. This has occurred in connection with fibrous glass dust, the pathogenic potential of which has been questioned in spite of the fact that nonfibrous glass dust has been found to be biologically "inert."[2]

This paper is concerned with a study of the pathogenic potential of fibrous dusts not heretofore documented and with the pathologic

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