Respiration 1992;59(suppl 1):12

P.H. Godard

Clinique des maladies respiratoires
Montpellier, France

Airway Macrophages and Bronchial Asthma

Key Words
Alveolar macrophages
Broncho-alveolar lavage
Mediators

Abstract
Bronchial asthma has been defined as a bronchial disease inducing an airway obstruction. Recently a renaming was proposed: *eosinophilic desquamative chronic bronchitis*. This is very interesting as it outlines the importance of inflammation in the pathogenesis of the disease. Until recently it was assumed that the narrowed airways were preferentially located centrally, that is to say in the large bronchi. However, evidence now exists that deep lung is also involved in bronchial asthma: 1) indeed it is well known that many aeroallergens are able to reach the alveoli; 2) assessment of flow volume curves and of ventilation/perfusion abnormalities showed the airway obstruction is often located on peripheral airways, especially in the chronic stages of the disease; 3) bronchoalveolar lavage (BAL) has been used extensively in asthmatics and results support this hypothesis.

Alveolar macrophages (AM) are the principal resident phagocytes in the bronchoalveolar lumen, but these cells have been observed in more central airways by segmental bronchial wash and bronchial biopsy using conventional staining or monoclonal antibodies; we propose to name them airway macrophages. They play a major role in local defence against environmental agents. In a very elegant and important piece of work, R. Patterson demonstrated that bronchoalveolar cells obtained by BAL in sensitized monkeys and injected into the trachea of unsensitized syngenic monkeys were able to promote bronchial asthma after specific inhalation challenge. Since AM accounted for about 90 to 95% of the cells recovered by BAL, potential role for AM could be suggested.

We would like to overview the lines of evidence which suggest that macrophages play an important role in the pathophysiology of human bronchial asthma. 1) AM are able to synthesize and release chemical mediators involved in the pathophysiology of bronchial asthma; they can be activated by an IgE dependent mechanism, phagocytosed particles (zymosan and opsonized zymosan) or soluble stimuli such as a chemoattractant peptide (fMLP) or a potent activator of protein kinase C (PMA); and these mechanisms appear to be relevant to bronchial asthma; 2) AM are activated in vivo in asthmatic patients and their activation correlates with the severity of the disease (as assessed by either the Aas' clinical score, or airway obstruction or nonspecific airway reactivity); the concept of releasability seems to be very important; 3) AM play a central role in the immune response; they act in cooperation with lymphocytes in allergic asthmatics, AM suppressive activity is decreased and interleukin-1 (Il-1) production is increased.

P.H. Godard
Clinique des maladies respiratoires
Avenue Major Flandre, F-34059 Montpellier, France

0025–7931/92/0597–0012
$ 2.75/0