

Introduction

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Cells communicate by cell-cell contact and by the release of mediators which may originate either from tissues remote from their sites of action, such as hormones, or locally. Over the last decade there has been a great increase in knowledge of the local factors—the cytokines—that may be produced by cells within the local tissue microenvironment. It is now clear that cytokine effects can be tightly regulated spatially within the inflammatory response: products of one cell may influence the cell itself (autocrine action), or cells in close proximity (paracrine action), as well as exerting more distant systemic effects. Cytokines may be classified into groups with a common function such as growth factors or interferons and trigger target cells through specific receptors, resulting in the stimulation of the target cell to up-regulate the synthesis of one or more gene products. Adding to the complexity of cytokine mediated responses is the more recent discovery of soluble antagonists of cytokines such as inhibitors of interleukin (IL)-1 and tumour necrosis factor (TNF)- α which compete for receptor binding.

The almost infinite complexity of possible cytokine networks has made the interpretation of studies of cytokines difficult to apply to human disease and has resulted in confusion and frustration for the non-specialist

who is trying to interpret their role in lung pathology. As a result of this, and because of the undoubted key roles played by cytokines in the pathogenesis of chronic inflammatory lung diseases, this series has brought together reviews from four groups who have contributed significantly to our knowledge of cytokines in lung disease.

The introductory article by Nicod discusses how cytokines influence the function of individual cell types present in normal and inflamed lung, and the subsequent three reviews are devoted to the role of cytokines in coordinating mechanisms of inflammation. Strieter and colleagues address lung inflammation with particular regard to neutrophil recruitment and lung injury; Robinson and coworkers summarise the networks involved in chronic airway inflammation with specific focus on the role of T cells; and Gauldie *et al* conclude the series with a discussion of cytokines and fibrogenesis. The aim of each author has been to illustrate the important issues in a format which is “user friendly” to the non-expert. While it is impossible to disguise completely the vast repertoire of this potent group of effector molecules, the contributors have succeeded admirably in dissecting out those issues of relevance to the lung physician.

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