Rubor (redness), tumor (swelling), calor (heat), and dolor (pain) are the classical signs of inflammation. These features are obvious in the skin, where injury or disease causes flare, wheal, and painful burning sensations. Vasodilatation underlies the flare and heat, plasma exudation the swelling, and activation of sensory nerves relays pain. In chronic conditions, skin biopsies show inflammatory cell infiltrate. Inflammation is not unique to the skin and contributes to disease and repair processes in other organ systems in the body. From the viewpoint of this volume, lung inflammation is now recognized as central to the pathophysiology of a number of severe respiratory conditions, the two most common being asthma and chronic obstructive pulmonary disease (COPD). In asthma, and to a lesser extent COPD, there is evidence of vasodilatation, with congestion of blood vessels accompanied by reddening of the airway mucosa, and of plasma exudation, leading to swelling of the airway wall. Similarly, although less pronounced than in the skin, there is evidence of pain, for example, the unpleasant chest sensations associated with asthma attacks. Understanding the pathogenesis of airway inflammation will enable rational design of drugs to effectively treat conditions such as asthma and COPD. However, whereas immediate access to the skin facilitates investigation of disease processes, the lung, although “open to atmosphere,” is much less accessible. Consequently, the investigation of lung inflammation is usually indirect. Thus, a wide variety of research techniques are used.

Human Airway Inflammation: Sampling Techniques and Analytical Protocols attempts to draw together many of the important methodologies and protocols for assessing inflammation in human airways. We start with techniques for the collection of samples. These can be such airway liquids as sputum or exhaled gases as nitric oxide (NO). Once collected, the samples can be used for the isolation and characterization of cells or for the measurement of markers of inflammation. Techniques to isolate and analyze all of the major inflammatory cells associated with lung inflammation are given. Similarly, protocols to measure many of the inflammatory mediators and enzymes released during lung inflammation are detailed herein. In an attempt to be inclusive and to attract both experienced researchers and the novice, we have included specialized chapters (for example, on tracing intracellular mediator storage), mixed with
more general chapters (for example, on Northern blotting). We wanted our volume to have something for everyone interested in assessing lung inflammation.

To achieve our aim, we are indebted to our authors for sharing their methodological secrets. As we all know, following the published “recipe” does not always (some would say ever) reproduce the data presented. To that end, the “Methods” series includes the authors “Notes.” These are the tips and nuances that convert abject failure into a successful experiment. Our contributors have been most forthcoming on their methods, most of which were painstakingly developed over long periods of time. Another factor that increases the impact of the book is the inclusion of color figures in some of the chapters. We are most grateful to Astra Zeneca and Boehringer Ingelheim, UK for sponsoring color reproduction. Finally, we wish to thank John Walker and Thomas Lanigan for inviting us to put this volume together—it has been an invaluable learning experience for us all.

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