Introduction
John E. Repine, M.D., Chairman, 25th Aspen Lung Conference

This year’s Aspen Lung Conference focused on new advances in research into mechanisms of lung defense, injury and repair with a special emphasis on determining the contribution of alveolar macrophages and neutrophils to these processes. It became clear that important similarities and differences underlie many of the mechanisms which are involved in lung defense, such as in pneumonia; acute lung injury, such as the adult respiratory distress syndrome (ARDS), and more chronic lung injury, such as emphysema. Moreover, the potential contribution of variations caused by differences in repair mechanisms in various conditions became apparent and signaled the need for more research in this new and exciting area.

Owing to the highly focused theme of this year’s conference, each session, including the extra session held on Friday night, was heavily attended. State-of-the-art lectures given by Drs. Peter Henson, Dale Hammerschmidt, Joe McCord, Aaron Janoff, Kenneth Brigham and Ronald Crystal were uniformly outstanding and together provided a comprehensive and thoughtful overview of research on this topic. Their careful preparation and efficient chairing of the various sections was an important feature of the success. Many of the individual papers were exciting and provocative. These state-of-the-art addresses and brief minipapers are provided in this publication.

A special highlight of this year’s conference was the comments and summary given by Thomas P. Stossel, M.D., editor of The Journal of Clinical Investigation and a faculty member of the Massachusetts General Hospital. Because of his training in hematology rather than pulmonary research, and his unusual analytical skills, Tom was able to provide a highly critical summary with broad perspective. Dr. Stossel’s enthusiasm for the future importance for some of the basic research in pulmonary disease was very encouraging. His concluding comments are summarized in this publication.

As this year’s Chairman, I wish to acknowledge the excellent support of a number of individuals. Tom Petty served as co-chairman and Dallas Pierson of the American Lung Association of Colorado provided outstanding assistance with ground transportation, the programs and name tags. Local arrangements, including the traditional picnic, were organized in superb fashion by Virginia Carpenter and Louise Nett. Indeed, special appreciation is extended to Virginia Carpenter who initiated and managed a hospitality suite which was greatly enjoyed by all of the participants. I also thank the section chairman and the members of the Local Steering Committee who helped review abstracts and plan the overall program.

Lung Defense
The Paradox of Inflammation*
Gary L. Larsen, M.D.†; Debra A. Parrish, M.D.‡; and Peter M. Henson, Ph.D.§

The mechanisms through which the lung protects itself against air or blood-borne insults are many, and range from mechanical barriers such as the cells lining the respiratory tract and pulmonary circulation to proteins including immunoglobulin and complement found within alveoli and the blood stream. These diverse mechanisms are reviewed in detail in several recent publications. We would like to concentrate on just one of these pulmonary defense mechanisms, the inflammatory response. While the process of inflammation may be generally thought of as beneficial, as stated by Metchnikoff in 1891 when he described the process as a "...salutary reaction against some injurious influence," recent experimental and clinical observations have pointed out that in the lung, inflammation may also be harmful to the host. For example, in patients with idiopathic interstitial pneumonitis, circulating immune complexes and intrapulmonary deposition of IgG and C3 were found in patients with histologically active disease, suggesting immune complexes are related to disease pathogenesis as either cause or effect. The adult respiratory distress syndrome (ARDS) has also been suggested to occur as a result of the lung becoming the target organ of an inflammatory reaction triggered by intravascular complement activation.

Realizing that pulmonary inflammation may be either beneficial or harmful to the host, attempts to define when a potentially helpful response becomes deleterious, why the response is harmful, and how this can be altered to benefit the host are important questions for chest physicians and other specialists who care for such patients. It is hoped that this article will contribute to these scientific questions by reviewing the recent literature, emphasizing the advances made during the past year, and focusing on the role of inflammation in the pathogenesis of pulmonary disease.

*From the Department of Pediatrics, National Jewish Hospital and Research Center/National Asthma Center, and Departments of Pediatrics, Pathology and Medicine, School of Medicine, University of Colorado Health Sciences Center, Denver; Supported by Grants No. HL-21565 and No. HL-27063 from the National Heart, Lung and Blood Institute.
†Assistant Professor of Pediatrics.
‡Pediatric Pulmonary Research Fellow.
§Professor of Pathology and Medicine.
Reprint requests: Dr. Larsen, Department of Pediatrics, National Jewish Hospital/NAC, Denver 80206

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