Innate Immunity and Airway Inflammation

In this issue of the *Journal of Innate Immunity*, we present a focus on innate immunity and airway inflammation that will highlight the state of the art in this exciting new area of immunological research. First, an authoritative overview by Lanckacker et al. [1] critically discusses how the innate immune system of the lung deals with the onslaught of 'real-world' exposures to environmental atmospheric pollutants in which innate immunity is evolutionary ill prepared to respond to such challenges. Second, an equally authoritative review by Horner [2] discusses how the innate immune system senses and responds to the indoor ambient environment of households. Horner challenges current hypotheses of immune tolerance and protective immunity in allergic inflammation and instead proposes a new paradigm by which ligands occupying airborne pattern recognition receptors influence the generation of allergic diseases of the lung. In a related article, Eisen [3] critically analyzes how deficiency in mannose-binding lectin (an important innate immune pattern recognition receptor) enhances host susceptibility to lower respiratory tract infections. In addition, three

key research articles discuss novel systems and mechanisms of innate immune defense against respiratory infections and immunoregulation. In the first of these articles, Williams et al. [4] describe a novel role for the transmembrane Muc1 mucin receptor in innate immunity. Genetic deletion of Muc1 promoted a heightened functional response of dendritic cells in response to Toll-like receptors TLR4 and TLR5 signaling pathways, suggesting a previously underappreciated role for Muc1 in regulating innate immune responses of conventional myeloid pulmonary dendritic cells. In the following article, Pesce et al. [5] provide new information on the role of CpG-containing oligonucleotides as highly effective mucosal adjuvants in the immune therapy of respiratory infections, which they propose to be partly dependent on the de novo recruitment of dendritic and natural killer cells. Finally, Bober et al. [6] describe collagen VI as a novel target structure for adherence of bacterial pathogens that play important roles in airway infections.

Marc A. Williams, Research Triangle Park, N.C., USA

References

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