



### **Helene F. Rosenberg, M.D., Ph.D.**

Helene F. Rosenberg, M.D., Ph.D. is a Senior Investigator and Section Chief in the Laboratory of Allergic Diseases of the National Institute of Allergy and Infectious Diseases (NIAID) of the National Institutes of Health, Bethesda, Maryland, USA. She graduated from Brandeis University in 1979 with a combined B.A. and M.A. in Biochemistry and the Medical Scientist Training Program (MSTP) of the Rockefeller University / Cornell University Medical College, New York (now known as the Tri-Institutional Program) in 1984 and 1985, respectively. After post-doctoral studies at Harvard Medical School, she joined NIAID in 1991 and was granted tenure in 1998. She has trained numerous students and has been a member of the Editorial Board of Journal of Leukocyte Biology since 1996.

Dr. Rosenberg's research program focuses on inflammation and specifically highlights the inflammatory responses elicited by acute respiratory virus infection. While therapeutic developments in this field have focused on the development of new and ever more complex antiviral agents, Dr. Rosenberg and her group have shown that virus-induced inflammation that is insensitive to these agents contributes profoundly to airway dysfunction and death. Her studies include natural mouse pathogen models, most notably, pneumonia virus of mice (PVM; family *Pneumoviridae*). Via an ongoing exploration of lethal PVM infection, her group has defined the inflammatory responses that are characteristic of natural respiratory virus infections *in vivo*. They have also shown that manipulation of the inflammatory response can convert a lethal infection into one that is functionally benign.

Dr. Rosenberg's group has also made sizable contributions to our understanding of the biology of human and mouse eosinophilic leukocytes. While eosinophils are perhaps best known for their contributions to the functional pathophysiology of Th2-driven asthma, Dr. Rosenberg has pointed out that evolution tells us that the ability to induce pathology cannot be a *raison d'être* for any existing cell lineage. Several distinct lines of evidence have led her group to consider the unrecognized impact of eosinophils on respiratory viruses and virus-induced acute inflammation.