



ERIC FREED

Zirus Science Board

Dr. Eric Freed received his Ph.D. in 1990 in the laboratory of Dr. Rex Risser at the University of Wisconsin-Madison and did postdoctoral work with Dr. Howard Temin at UW-Madison in 1991. He joined the Laboratory of Molecular Microbiology at the National Institute of Allergy and Infectious Diseases (LMM/NIAID) in 1992, where he worked with Dr. Malcolm Martin on a variety of topics relating to virus assembly and entry/post-entry events in the HIV-1 replication cycle. In 1997 he was appointed as a Tenure-Track Investigator in NIAID, and he was promoted to a tenured Senior Investigator position in 2002. Dr. Freed joined the HIV Drug Resistance Program in 2003 as Chief of the Virus-Cell Interaction Section. He was an organizer of the 2004 Cold Spring Harbor Retroviruses conference and the 2006 ASCB Conference on the Cell Biology of Retroviruses. He currently serves on the Editorial Boards of Journal of Virology, Virology, Open Virology Journal, Retrovirology, and Advances in Virology and is the Editor-in-Chief of Viruses. Dr. Freed also serves as an adjunct Associate Professor in the Department of Cell Biology and Molecular Genetics at the University of Maryland, College Park and is a member of the University of Maryland Virology Program.

Dr. Freed's research focuses on several areas of HIV-1/retroviral replication: 1) Retroviral Gag trafficking, envelope glycoprotein incorporation into virus particles, and virus assembly. Work in this area is particularly focused on defining the host cell machinery involved in retroviral Gag trafficking to the site of particle assembly. 2) Retrovirus budding and endosomal sorting, with particular emphasis on the role of the cellular endosomal sorting machinery (the ESCRT pathway) in virus budding. 3) Lipid microdomains and HIV-1 replication. Dr. Freed's lab has previously defined a role for cholesterol-enriched lipid rafts in HIV-1 assembly and over the past several years has been studying the effect of a cholesterol-binding compound, amphotericin B methyl ester (AME) on both early and late phases of the HIV-1 replication cycle. 4) Inhibition of HIV-1 maturation. These studies have involved defining the mechanism of action of the maturation inhibitor bevirimat (PA-457 or dimethyl succinyl betulonic acid) and characterizing resistance pathways by which HIV-1 escapes this inhibitor. Bevirimat is currently in phase II clinical trials.

Dr. Freed will assist the work being done at Zirus by serving as a scientific advisor on issues related to host cell factors involved in HIV-1 and other viral replication, particularly those that function in late stages of the viral life cycle. Dr. Freed's lab will also collaborate with Zirus to conduct characterization of the trapped genes in terms of their potential roles in HIV-1 replication. Dr. Freed's expertise in molecular biology, cell biology, biomolecular imaging, and virology will be brought to bear on studies that develop from the Zirus gene trap technology.