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CMP Cellular and Molecular Pathology

Adam Bailey

Program of the Dissertation Defense Seminar
for the Degree of Doctor of Philosophy
in Cellular and Molecular Pathology

"Exploring the primate plasma virome: implications for human health and disease"

HEALTH SCIENCES LEARNING CENTER

Friday, June 11, 2015

3:00pm Room 1306

Research conducted in the lab of
David O'Connor, PhD
Department of Pathology & Laboratory Medicine

UNIVERSITY OF WISCONSIN – MADISON

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University of Wisconsin-Madison
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Colgate University, 2005-2009
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Molecular Biology with Honors

Research Experience

AIDS Vaccine Research Laboratory, University of Wisconsin - Madison. Dr. David O'Connor (August 2012-present): Discovery and characterization of novel primate viruses, including the development of animal models of infection.

Osborne Laboratory, University of Washington. Dr. William Osborne. (June 2009-June 2010): Construction of lentiviral gene therapy vectors for use in a rat model of diabetes and obesity.

Laboratory of Molecular Pathogenesis, Colgate University. Dr. Geoff Holm. (Jan 2009-Apr 2009): *In vitro* study of the innate immune response to reovirus infection.

Laboratory of Viral Diseases, National Institutes of Health. Drs. Jonathan Yewdell and Scott Hensley. (June-Dec 2008): Study of antigenic drift and cellular immune responses in a murine model of pulmonary influenza infection.

Dewhurst Laboratory, University of Rochester. Dr. Stephen Dewhurst. (June-Aug 2007): Study of adenoviral vectors for use in gene therapy.

DISSERTATION ABSTRACT

Wild primates serve as the immediate source and long-term reservoir of many human pathogens. However, with the exception of simian immunodeficiency viruses (SIVs), the viruses naturally harbored by these animals remain largely uncharacterized. Here, we present the discovery and characterization of several novel viruses within the *Flaviviridae* and *Arteriviridae* families referred to as GB virus C and simian arteriviruses (aka. simian hemorrhagic fever viruses), respectively. Together with SIV, it appears that these viruses are widespread and prevalent among African cercopithecoid (*i.e.* Old World) monkeys; thereby defining the "plasma RNA virome" of these animals. Each monkey species we examined harbors its own species-specific variant(s) of these viruses, which cause high-titer viremia in these hosts without inducing overt signs of disease. Infection studies in captive macaques and baboons infected with GBV-C and simian arteriviruses, respectively showed that these viruses are semi-persistent in these hosts but exhibit vastly different degrees of within-host genetic diversity and sequence evolution, suggesting that these viruses maintain persistence though different mechanisms. While infection of macaques with GBV-C did not elicit overt signs of disease, infection of macaques with simian arteriviruses induced high morbidity and mortality in this "non-natural" host. Given the several features of the simian arteriviruses -- vast genetic diversity, high titer and persistent infection, and the ability to transmit between primates of different species and cause disease, we believe this group of viruses merits further study and close surveillance as a zoonotic threat to human health. In contrast, our infection of macaques with baboon GBV-C created the first laboratory animal model of human GBV-C infection. With this model, we have begun to investigate the mechanism behind the widely cited but poorly understood phenomenon of GBV-C-associated protection from AIDS in HIV+ people.

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