Horwitz Lab - Principal Investigator

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B.Sc., University of California, Davis, (1982);
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Research Interests

Laboratory of Viral Immunopathology

During viral infection, the symptoms experienced are either a direct result of the virus infecting and damaging cells and tissue or a consequence of the activated inflammatory host immune response that was generated to pursue and remove the virus. Typically, it is a combination of both. As the virus is eliminated, the immune response subsides and recovery from disease is observed as symptoms subside. Furthermore, many viruses have been associated and identified as the triggers of more complex diseases. These diseases involve chronic autoimmunity, immunosuppression, haemorrhagic fever and meningitis. In these instances, chronic disease develops as a result of improper control and regulation of the viral-induced immune response. Understanding the involvement and regulation of the immune system in response to viral infection is central to the discovery of mechanisms designed at prevention and treatment of viral-induced complex disorders.

Our laboratory is interested in identifying, characterizing and determining the mechanisms of viral-induced immune disease in a variety of complex chronic disorders. These include, but are not limited to autoimmune diseases like diabetes, autoimmune myocarditis and multiple sclerosis, immunosuppression induced by viruses such as HIV and Measles, haemorrhagic fevers as observed following Dengue fever virus infection, and meningitis induced by viruses like West Nile Virus.

Specifically, the primary goal of our program is to interconnect the changes effecting the ability of the immune system to respond to infection with its ability to develop immune dysfunction leading to disease. Ongoing studies are aimed at describing and controlling the mechanisms of viral-
induced autoimmune disease in at least two different mouse models. Both insulin-dependent Diabetes Mellitus and autoimmune myocarditis can be induced in mice and man by a common pathogen of childhood, coxsackievirus. Dependent on the strain of virus and the strain of mice, different outcomes of acute coxsackieviral infection can lead to the induction of chronic autoimmune diabetes, heart disease or no disease. Our laboratory has taken advantage of this model to study the specific components that regulate disease induction.