HIV and Viral Immunity

Jody Baron [1]

Our experiments seek to use and extend our transgenic model of primary HBV infection to identify the mechanisms involved in acute and chronic hepatitis. Our long-term goal is to develop a more comprehensive understanding of the role of both innate and adaptive immunity in HBV clearance and virus-induced liver damage.

Other Research in Dr. Baron's Lab:


Warner Greene [4]

My laboratory investigates how powerful intrinsic antiviral factors, the APOBEC3's, achieve their effects, how viruses mount counterstrikes against the APOBEC3's, and the potential role of the APOBEC3G as a regulator of endogenous retroelement retrotransposition. A second line of study seeks to define the molecular basis for HIV latency and to derive new approaches for purging HIV from the latent reservoir, a requirement if a "cure" for HIV infected patients is ever to be realized. A third area of study explores the biology of HIV in dendritic cells to understand the molecular underpinnings of HIV transmission.

Other Research in Dr. Greene's Lab:

Inflammation [2]

Lewis Lanier [5]

NK cells mediate innate immunity against virus-infected cells. They are particularly important in immunity against cytomegalovirus (CMV) and other double-stranded DNA viruses. In mice, members of the Ly49 family of NK receptors recognize cytomegalovirus and cause the activation of NK cells, resulting in the secretion of cytokines and the killing of CMV-infected cells. We have shown that the Ly49H receptor directly recognizes a glycoprotein, m157, encoded by mouse cytomegalovirus (Arase et al., Science. 2002 [6]). In response to selective pressure imposed by NK cells, CMV has evolved genes encoding viral protein that evade NK cell recognition. For example, we have identified two mouse CMV genes, m152 and m155, that prevent NK cell recognition of CMV-infected cells (Lodoen et al., J Exp Med. 2003 [7] and Lodoen et al., J Exp Med. 2004 [8]). Our lab focuses on the role of NK cells in immune responses to CMV and other viruses.
Featured Articles:

Arase et al., Science. 2002 [9]

Other Research in Dr. Lanier’s Lab:


Mike McCune [14]

Research in the McCune lab focuses on the definition of pathogenic mechanisms of viral diseases, particularly HIV-1 disease. This focus has spanned a range of fields, from understanding critical structural determinants of infectivity, to devising a small animal model (the SCID-hu Thy/Liv mouse) to study HIV pathogenesis and to prioritize antiretroviral compounds against HIV, to studying mechanisms of T cell depletion and repletion in vivo. This body of work has engaged in hypothesis-driven, patient-oriented research that has involved collaborative teams of basic scientists, translational researchers, and clinicians. Most recently, ever more attention is being devoted to understanding the correlates of protective immunity against HIV, with the specific intent to work with others to develop an effective vaccine. This change of focus has now been materialized at UCSF by the creation of the Division of Experimental Medicine, of which Dr. McCune is the Chief.

Find out more about Dr. McCune's research [14]

Melanie Ott [15]

We study why and how HIV infection induces a state of chronic immune activation in infected individuals. This state is highly critical for HIV to cause AIDS and remains at lower levels even after effective antiretroviral therapy. We found in the past that HIV itself causes immune activation in infected CD4+ T cells and identified the viral protein Tat as one mediator of this activation. We find that Tat through inhibition of the cellular protein deacetylase SIRT1 induces hyperacetylation of key transcription factors such as NF-kappa B, a process associated with enhanced reactivity of the cells to T cell activation. The lab is interested in the new role of SIRT1 in T cell function and the connection between nonhistone protein acetylation and immune activation. We are also interested in the premature aging syndrome emerging in long-term HIV-infected people and the link to chronic immune activation.

Other Research in Dr. Ott’s Lab:

Diabetes and Autoimmunity [12]
Marion Peters [16]

Host-viral interactions in Hepatitis C B infection. This project evaluates the role of inflammatory cytokines and their receptors using DNA polymorphism analysis and mRNA gene profiling. We assess the effect of the host response on severity of disease and response to therapy including the effects of alcohol, obesity and HIV co-infection.

Other Research in Dr. Peters' Lab:


Nadia Roan[17]

Research in the Roan lab focuses on understanding how the tissue microenvironment can affect immunity. We study how soluble and cellular factors in the genital mucosa and other tissues can influence the susceptibility of CD4+ T cells to HIV infection, and how HIV can alter the properties and functions of CD4+ T cells. By analyzing the properties of HIV-infected CD4+ T cells (from ex vivo infection models or patients) using a variety of approaches including single-cell phenotyping (mass cytometry and flow cytometry) and global gene expression analysis, we have gained insights into the mechanisms of HIV transmission, persistence, and pathogenesis.

Other Research in Dr. Roan’s Lab:


Shomyseh Sanjabi [19]

We utilize genetically modified humanized mice and induced pluripotent stem cell technology to address the role of host genetics in HIV transmission and pathogenesis.

Other Research in Dr. Sanjabi’s Lab:


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Links
[2] https://immunology.ucsf.edu/inflammation