PI/Lab Name	Rotation Semster(s)	Rotation Project(s)
Alcorn	Fall and Spring	(1) Immune mechanisms of influenza-induced susceptibility to secondary bacterial pneumonia in mice (2) Influenza-induced persistent alveolitis during lung repair in mice (3) Development of Influenza, Streptococcus pneumoniae super-infection model in mice (4) Influenza vaccine driven PBMC gene expression and antibody responses in human
Ambrose	Fall and Spring	(1) Imaging host cell factors and/or innate immune responses involved in HIV capsid uncoating, reverse transcription, and/or nuclear entry (2) Characterization of HIV drug resistance mutations after novel antiretroviral prevention or treatment therapies (3) In vivo imaging of HIV disseminatation in a murine model
Bina	All	Bacterial pathogenesis and antimicrobial resistance in the human pathogen Vibrio cholerae.
Biswas	All	Mechanisms of IL-17 mediated antifungal immunity
Byersdorfer	Fall and Spring	The Byersdorfer lab studies in vivo T cell metabolism, during both graft-versus-host disease (GVHD), a common complication following allogeneic blood and marrow transplantation, and in T cells driving anti-leukemia responses. The lab utilizes mouse models, gene targeting, and multiple metabolic analyses to pinpoint the role of specific genes and proteins during in vivo metabolic reprogramming, to find targets whose inhibition might mitigate disease while protecting or enhancing immune reconstitution. Projects for rotating students include functionally characterizing specific metabolic proteins in the pathogenesis of GVHD, testing metabolic compounds in vivo for their ability to mitigate GVHD while preserving anti-leukemia effects, and enhancing immunotherapeutic approaches by enhancing the in vivo persistence of anti-leukemia T cells.

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Canna	All	(1) Macrophage Activation Syndrome and Pulmonary alveolar proteinosis. We have discovered a novel and deadly phenomenon: some patients with a systemic inflammatory disorder called Systemic Juvenile Idiopathic Arthritis develop a life-threatening complication called Pulmonary Alveolar Proteinosis. The etiology of this complicaiton is unknown, but clinical and biomarker evidence suggest dysregulation of three cytokines key to the pathogenesis of SJIA: IL-1beta, IL-6, and IL-18. This project will involve evaluating the effects of excess or neutralization of these cytokines on chemical based models of PAP in mice. (2) Does IL-18 affect ILCs and promote immunodeficiency? We found that both Rag1-ko mice and mice with a gain-of-function mutation in the NLRC4 inflammasome both modestly overproduce IL-18, but together they synergize to produce a lot. Serendipitously, we found that Rag1-/-;NLRC4-KI mice developed a spontaneous lung infection, while littermate Rag1-/- did not. This project would involve a collaboration with John Alcorns lab. First, phenotyping the ILCs of these mice to see if chronic IL-18 changes the number and character of lung and gut ILCs. Second, we will challenge the mice with oropharyngeal aspiration to see if they develop more severe lung infection. (3) We have data showing a variety of circumstances wherein systemic or local inflammation leads to a slow, but profound upregulation of MHC-II on intestinal epithelia. We have generated mice specifically lacking MHC-II on their intestinal epithelia. This project would entail inducing EAE in these mice to see if the absence of intestinal MHC-II exacerbates or prevents the development of distant autoimmunity. (4) We found that mice that constitutively produce IL-18 have a severe systemic reaction to an otherwise mild viral infection (LCMV). The other genotype in which this occurs is deficiency of perforin, and it so happens that excess IL-18 and perforing deficiency are the two known host susceptibility factors for hyperinflammatory SIRS (Systemic Inflammatory Response Syndrome). This project involves assessing CD8 T-cells in these mice by flow, RNAseq, and killing assays. (5) Patients with Familial Mediterranean Fever (FMF) rarely develop features of sepsis. When they do, they tend to highly upregulated IL-18. We have transcriptional evidence showing that systemic Type I IFN induces transcription of both Il18 and Mefv (the gene mutated in FMF). This project would involve adminstering Type I IFN-induicng stimuli, such as Poly I:C and Influenza A infection, in mice carrying gain-of-function mutations in MEFV and seeing if they develop features of hyperinflammation, particularly induction of Il18 and IFNgamma.
Cooper	Fall and Spring	Evolutiuon of antimicrobial resistance in biofilms or dynamics within host-associated microbiomes
Coyne	No answer	No answer
Culyba (pending approval to grad faculty)	Spring	No answer

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D'Cruz	All	(1) Determine the fuction of the transcriptional regulator Id3 in adipose Tregs. We have generated a new mouse model in which we can constitutively express Id3 in regulatory T cells. The rotation project will involve characterizing this mouse model. We have also generated Id3 Treg deficient lines and Id3-GFP reporters to further determine how Id3 can enforce a central Treg phenotype. (2) Determine the compensatory function of the transcriptional regulator Id2 in adipose T cell differentiation. We have generated a novel mouse line in which we have knocked the Id2 gene into the Id3 locus. Using this animal model, we can determine if loss of Id3 and expression of Id2, perturbs T cell development and differentiation in the thymus, lymphoid and adipose tissue.
DeLuca	All	(1) Virus-cell interactions affecting transcription. Herpesvirus have evolved multiple mechanisms to utilize the host cell RNA polII machinery for the robust transcription of their genomes. They also have profound effects on the transcription of cellular genes. Ongoing projects address the mechanisms underlying both processes and their significance. Projects involve viral genetics, protein biochemistry, genomics, and proteomics. (2) Triggering and inhibition of the innate immune response to herpes simplex virus. Component(s) of the virus particle trigger the expression of antiviral molecules in cells. HSV has also involved multiple mechanism to inhibit this response. Ongoing projects address the mechanisms underlying both processes and their significance. Projects involve viral genetics, protein biochemistry, genomics, and proteomics.
Dutta	All	Pulled this from the attachment Dr. Dutta sent to me: "One of our current research interests focuses on immunology of cardiovascular disease. We are investigating how myeloid cells, such as monocytes and macrophages, induce inflammation in metabolic disease such as type II diabetes and cardiovascular disease such as myocardial infarction."
Ferris	No answer	No answer
Flynn	No answer	No answer
Gaffen	Fall and Spring	Centered around IL-17 or Th17 function in either signal transduction or in anti-fungal immunity
Gao (pending approval to grad faculty)	Fall and Spring	No answer
Gottschalk	Fall and Spring	(1) Elucidating tissue-specific macrophage signaling (2) Quantifying age-associated tuning of inflammatory thresholds (3) Analyzing macrophage signaling heterogeneity using live single-cell reporters
Klimstra	All	Investigation of glycoprotein conformational rearrangements that occur during alphavirus infection. Development of a flaring arthritis animal model for Chikungunya virus related to changes in efficacy of the type I interferon response. Investigation of the evolving tropism of eastern equine encephalitis virus for myeloid cells during infection of vertebrates. Development of vaccines against the encephalitic and arthritogenic alphaviruses.

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Lakdawala	Fall and only Spring 1	Examining influenza virus assembly in human lung organoids using microscopy techniques.
Lee	Fall and Spring	Next-generation Sequencing to Study Viral Non-coding RNA Function
Mathers	All	My lab is interested in understanding cutaneous inflammation. Specifically, we are interested in the role the alarmin, ATP, has in the pathogenesis of autoimmune psoriasis. In our current publication we have determined that ATP signaling through the P2X7 receptor can induce an acute psoriasiform dermatitis in mice. Moving forward, we have several exciting projects that students can pick from that are looking into the mechanisms of how ATP/P2X7R induces psoriasis and if co-morbidities are also induced, such as diabetes, vascular disease, and psoriatic arthritis. By understanding the mechanisms we can begin to design more refined therapeutics to treat this devastating disease. We utilize mouse models (we are currently having a mouse developed that overexpresses the P2X7R in the skin), human to mouse skin xenotransplants, human skin explants, and some in vitro cell culture models to answer our questions.
McElroy	All	(1) Evaluating the role of cellular tropism in Rift Valley fever virus pathogenesis: The rotation project would involve the generation of recombinant viruses that contain cell specific host miRNA incorporations and evaluating these viruses for their ability to replicate in the various cell types. The long-term goal of the project is to evaluate these same viruses in vivo in the mouse model to understand how infection of a specific cell type contributes to disease. (2) Endothelial dysfunction in hemorrahagic fever virus infection: The rotation project would involve the generation of tagged clones of Ebola and Rift Valley fever virus glycoproteins and assessment of interactions of these proteins with thrombomodulin, a protein that is shed from the endothelial surface during infection. Elevated levels of thrombomodulin are associated with disease severity and death from these two hemorrhagic fever viruses and thrombomodulin contains C-type lectin domains that could mediate direct virus binding. If an interaction is established, therapeutics that could block this interaction would be explored as novel treatment. (3) Developing a better mouse model for RVFV. Most inbred mice are exquisitely susceptible to acute RVFV infection. This makes the mouse model difficult to use for studies of pathogenesis. The rotation project would involve infection of mouse or human macrophages with WT RVFV and a transcriptomic approach to define innate immune response differences that could contribute to the susceptibility of the mouse. This data would be used to evaluate various genetic mouse models with the goal of identifying a better mouse model for studies of RVFV pathogenesis.
Poholek	All	(1) Identify Blimp-1 expressions in antigen-specific T cell responses in a model of asthma. (2) Explore the role of Blimp-1 in effector Treg differentiation in the lung and gut.
Reed	Fall and Spring	(1) Isolate and phenotype rabbit peripheral blood leukocytes, response to mitogens (proliferation, cytokine production) (2) Culture and pheynotype rabbit macrophages and dendritic cells; infect with Francisella (3) Apoptosis/pyroptosis in Francisella infection. Heat shock protein release by Francisella-infected cells (4) Opsonization of Francisella by rabbit antibody (functional assay, using LVS and clpB)

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Sarkar	All	We are broadly interested in the role of innate immune signaling in antiviral and antitumor host response. The student will have the flexibility to pick from a number of available projects in this broad area, where initial feasibility studies have been done.
Shlomchik, M	Spring	Project related to systemic autoimmune disease and B cell biology
Shlomchik, W	Fall and Spring	This would be dependent on the student
Shuda	Spring	Search for the new mechanisms of small DNA tumor virus (MCV and HPV)-induced carcinogenesis.
Vignali	Any is ok, but Spring is best	Inhibitory mechanisms in cancer and autoimmunity, using mouse models
Williams	All	Our lab studies human metapneumovirus (HMPV), a leading cause of lower respiratory infection. We use in vitro and in vivo approaches to investigate the pathogenesis and immunity of HMPV. Specific areas for rotation projects include: a) mechanisms by which HMPV alters host innate immunity, and the effect of this modulation upon pathogenesis; b) mechanisms of T cell impairment by HMPV; and c) HMPV protein interactions with host cell proteins. The skills students can expect to learn include cell culture, viral culture and titration, mouse research, flow cytometry, and other immunologic techniques. We have a collegial lab that currently includes graduate students, postdoctoral fellows, and research assistants.