Pathway for Junior Scientist Program

A partnership between Rutgers New Jersey Medical School and Rutgers Newark.

Lab: Ahn
Investigator: Dr. Hyung Ahn
Research Summary: Research Summary: Our laboratory studies cerebrovascular pathology in Alzheimer's disease and cerebral amyloid angiopathy (CAA), focusing on how amyloid deposition in brain blood vessels contributes to neuroinflammation and blood–brain barrier dysfunction. Using genetically engineered rodent models, we examine interactions among vascular amyloid, microglia, and the brain vasculature. Undergraduate students in the lab will gain hands-on experience in histology, immunohistochemistry, microscopy, and data analysis, while learning experimental design and responsible research practices.
Project: Cerebral amyloid angiopathy (CAA), a condition where β-amyloid (Aβ), a peptide that contributes to Alzheimer's disease (AD), accumulates within the blood vessels of the brain, is a major contributor of brain circulatory dysfunction of AD patients. There are rare familiar forms of CAA, called hereditary cerebral amyloid angiopathy (HCAA), wherein patients display exaggerated CAA pathology and a severe clinical course of strokes. Furthermore, they suffer from early-onset neurological dysfunction, dementia, and ultimately death. Because molecular mechanisms underlying CAA formation and subsequent severe brain circulatory pathology in both AD and HCAA patients are unclear, we want to study pathogenic mechanisms of CAA.

Lab: Astrof
Investigator: Dr. Sophie Astrof
Research Summary: Dr. Astrof's research program employs genetic, cell biological, and biophysical approaches to decode the fundamental principles of cardiovascular morphogenesis, with the goal of illuminating the etiology of congenital heart disease (CHD). A central theme of this work is investigating how cell-extracellular matrix (ECM) interactions and mechanotransduction, particularly through integrins and fibronectin, orchestrate the behavior of progenitor cells during embryonic development. By generating and analyzing sophisticated mouse genetic models, our lab has elucidated how ECM signaling integrates with developmental pathways to regulate the cell migration, proliferation, and differentiation of cardiovascular progenitors, thereby ensuring the proper morphogenesis of the heart and major arteries. Mechanistic insights gained from basic developmental processes provide a critical foundation for understanding the pathogenesis of CHD.

Lab: Bergsbaken
Investigator: Dr. Tessa Bergsbaken
Research Summary: Research in the Bergsbaken laboratory is focused on understanding the development of adaptive immune responses in the intestinal tissue during local bacterial and

viral infection. We are particularly interested in CD8+ tissue-resident T (Trm) cells, as they are poised at barrier surfaces and play a critical role in protective immunity by direct killing of infected cells, recruitment of additional immune cells, and induction of tissue-wide changes that improve pathogen resistance. Ongoing research projects in the laboratory are aimed at understanding the plasticity of intestinal Trm subsets and the unique contributions of these T cell subsets during secondary infection. We are also interested in local signals that regulate Trm function, allowing for control of infection while preventing unwanted tissue destruction.
Projects for undergraduate students are designed to both help them train in basic laboratory techniques and teach them critical aspects of T cell biology. Undergraduate trainees are paired with experienced graduate students or postdoctoral fellows with overlapping research interests to facilitate their training; however, trainees are given independent research questions to pursue to foster ownership of their research. Current available project for incoming undergraduate students include: 1) examining the impact of Trm reactivation on other immune (macrophages, ILC3s) and non-immune cells (enterocytes) in the tissue using flow cytometry, and 2) generating CRISPR gene knockouts of potential Trm regulators (identified in preliminary studies) to examine alterations in Trm differentiation in vitro.

Lab: Bartlett
Investigator: Dr. Paula J. Bartlett
Research Summary: My research focus is to understand the mechanisms which elicit and maintain hormone-induced Ca2+ oscillations in the liver. Hepatocytes display stimulus-strength regulated Ca2+ oscillations and waves when challenged with hormones, such as vasopressin and glucagon, which couple to PLC-linked GPCRs. My research utilizes single cell imaging techniques and photorelease of caged IP3 to dissect the mechanisms regulating oscillations. I have demonstrated multiple proteins involved in the Ca2+ signaling pathway are regulated by PKC which provides both positive and negative feedback mechanisms to shape Ca2+ responses. More recently my research has focused on Ca2+ dysfunction and the role this plays in the pathogenesis of disease states such as alcoholic and non-alcoholic fatty liver disease. Many primary functions of the liver are regulated by signaling mechanisms that manifest as cytosolic Ca2+ ([Ca2+]c) oscillations, including glucose metabolism, mitochondrial physiology and gene expression. I have shown, chronic alcohol consumption sensitizes hepatocytes to PLC-linked hormones, potentiating Ca2+ signals and increasing cellular oxidative stress. My current research aims to delineate the effects of high fat diet on hormone-dependent calcium signaling in hepatocytes, in particular how these contribute to the dysregulation glucose homeostasis and the role this plays in the development of hepatic insulin resistance and diabetes.

Lab: Bhanot
Investigator: Dr. Purnima Bhanot
Research Summary: Malaria remains one of the world's most serious health challenges, with hundreds of millions of cases each year. Our lab is working to change that.
We study the malaria parasite *Plasmodium* and its development in the liver and red blood cells. Using cutting-edge techniques in cell biology, biochemistry, advanced microscopy, tissue

culture, and molecular genetics (CRISPR, gene knockout/knockdown), we aim to uncover how the parasite infects and grows inside human cells.
Our Goal: To identify parasite proteins essential for infection and collaborate with chemists to design new molecules that block these pathways—paving the way for next-generation malaria prevention drugs. Be part of the fight against malaria and make an impact in biomedical research!

Project: The student scholar may participate in drug discovery studies assessing the effect of new compounds on the parasite's replication. Other projects investigate how the immune system responds to Plasmodium's infection of the liver and identifying parasite proteins that play key roles in the parasite's development within hepatocytes. We use tools of molecular genetics (CRISPR/Cas9 gene editing, gene knockouts, conditional knockdowns), cell biology and microscopy. Why Join Us?

- Hands-on experience with advanced research tools
- Learn CRISPR and molecular genetics techniques
- Contribute to global health solutions

Lab: Carcea
Investigator: Dr. Ioana Carcea
Research Summary: Neuromodulation and social behavior.
Projects: The quality and quantity of social interactions are some of the best predictors for mental health and for longevity in humans. However, the biological mechanisms by which these interactions can have such profound effects on our brain and behavior remain largely a mystery. In our lab, we use animal models to investigate these mechanisms at the behavioral, electrophysiological and neurochemical levels. One of the circuits we probe in this context is the oxytocinergic system in mice. We approach this from three different angles.
Three projects include: 1. Maternal behavior: We investigate neural substrates for some key maternal behaviors. In particular, we are interested in understanding how maternal mice recruit the help of other adult mice in order to provide optimal care for offsprings. In this context, we investigate sensory processing of pup-related cues, and how it might result in activating the oxytocinergic system.
2. Social buffering: Our research will investigate the neural circuits by which social contexts modulate auditory processing and behavior. We will use electrophysiological, functional imaging, optogenetic and pharmacogenetic techniques in mice to study the role of cortical and subcortical circuits in social modulation of auditory representations.
3. Social Learning and Memory: We investigate whether oxytocin can modulate neuronal activity in major brain structures important for memory formation (frontal cortex, hippocampus, amygdala, etc). To address the potential beneficial role of social learning in dementia, we will investigate oxytocin activity in aged and Alzheimer’s Disease rodent models.

Lab: Christakos
Investigator: Dr. Sylvia Christakos

Research Summary: Research in Dr. Christakos' lab has examined the role of vitamin D deficiency in bone loss as well as in breast cancer and in autoimmune diseases. She has made important discoveries into the relationship between vitamin D and its receptor on the expression of genes responsible for the maintenance of calcium balance, inhibition of the growth of breast cancer cells and inhibition of autoimmune inflammation associated with multiple sclerosis. Her research has focused on how the vitamin D receptor, together with vitamin D and co-regulatory proteins, can alter the structure of the chromosome at the control regions for these genes to turn them on and off. Project: The undergraduate research project will focus on how intestinal effects of vitamin D change across the lifespan, leading to intestinal resistance in older adults. State of the art genomic technology will be used to understand how intestinal resistance develops and to test strategies to promote intestinal calcium absorption to prevent adult and age-related bone loss.

Lab: Kim
Investigator: Dr. So-youn Kim
Research Summary: Our laboratory focuses on women's reproductive health, with a particular emphasis on ovarian biology and fertility preservation in female cancer patients undergoing anticancer therapies. Our research aims to elucidate the molecular and cellular mechanisms governing therapy-induced oocyte death and to identify targeted strategies that preserve the ovarian reserve and long-term reproductive and endocrine function.

Lab: Lee
Investigator: Dr. Men Jean Lee
Research Summary: Maternal-Fetal Medicine

Lab: Parveen
Investigator: Dr. Nikhat Parveen
Research Summary: The projects involve study of the molecular basis of Lyme disease/syphilis and tick-borne coinfections.
Project: In vitro studies of spirochetes (syphilis causing Treponema pallidum and Lyme disease causing Borrelia burgdorferi) and tick-borne parasite, Babesia microti. The project will offer opportunity to learn and carry out molecular biological experiments, see real-time PCR procedure carried out by postdocs in the lab, microscopic examination of microbial pathogens, determination of parasitemia, protein purification and analysis by SDS-PAGE, Western blotting, ELISA techniques.

Lab: Rodriguez
Investigator: Dr. Gloria Marcela Rodriguez
Research Summary: Pathogenesis on tuberculosis
Project: A student scholar accepted in my lab could work in recombinant DNA technologies and molecular biology in the context of investigating M. tuberculosis virulence determinants.

Lab: Sugimoto
Investigator: Dr. Katsunori Sugimoto
Research Summary: Genome integrity is constantly challenged by exogenous and endogenous insults that generate various types of DNA damage. DNA double-strand breaks (DSBs) are formed as a result of exposure to exogenous agents such as ionizing radiation or chemotherapeutic agents. DSBs are also generated as byproducts after DNA replication stress or as intermediates of recombination events during meiosis and lymphocyte development. Failure to repair DSBs can result in chromosome loss, and inappropriate repair can cause chromosome rearrangements and mutagenesis. Defects in DSB repairs are associated with human disorders such as infertility, immunodeficiency, and cancer. The mechanisms of how cells respond to DSB induction are in many respects almost completely conserved among eukaryotes. In our laboratory, we have been studying how cells recognize and process DSBs using yeast as a model system.
Project: Project 1. Activation mechanism of ATR-related checkpoint protein kinase Mec1
Cells activate checkpoint signaling in response to DSB induction. The protein kinase ATR plays an important role in triggering checkpoint signaling during DSB processing. In yeast, MEC1 encodes ATR.
The C-terminal half of ATR contains a kinase domain, whereas the N-terminal half consists of repetitive sequences of unknown function. It is not known whether the N-terminal region of ATR controls the C-terminal kinase domain. We hypothesize that the N-terminus of ATR plays a regulatory role for the C-terminal kinase domain. To test the hypothesis, we will perform genetic screening to isolate dominant hyperactive mutations at its N-terminus of ATR.
Mutagenesis will be carried out by error-prone PCR and hyperactive signaling will be monitored by Western blotting.
Project 2. Telomere maintenance by DSB repair proteins
Telomeres are the ends of linear chromosomes in eukaryotes. Telomeres cannot be fully copied during each round of DNA replication (the end-replication problem), resulting in gradual shortening of chromosomes and thus cell senescence. To prevent telomere shortening, eukaryotic cells add telomere repeats to the ends of their telomeres. Telomerase plays a major role in the addition of telomere repeats. However, in some cancer cells, DSB repair proteins extend telomere repeats independently of telomerase. Telomerase-independent telomere extension is called Alternative Lengthening of Telomeres (ALT). We established a system to generate short telomeres in telomerase-knocked out cells. Using this system, we will examine how telomeres elongate independently of telomerase. Knock-out of DSB repair genes will be carried out by a PCR-based method and telomere addition will be monitored by Southern blotting.

Lab: Tergas
Investigator: Dr. Ana Tergas
Research Summary: Health equity in cervical cancer screening implementation.
Projects: Our lab focuses on developing patient-centered approaches to improve cancer screening among underserved and high-risk populations. One project evaluates a mail-based HPV self-sampling program to increase cervical cancer screening among underscreened women in Newark by assessing feasibility, acceptability, and real-world implementation. Another project

incorporates trauma-informed care principles into cervical and colorectal self-sampling workflows in substance use disorder (SUD) clinics, using user-centered design to develop a scalable screening model. We also conduct a retrospective chart-review study examining cervical, breast, colorectal, and lung cancer screening rates, and follow-up after abnormal results, among individuals with SUD. Together, these projects work to expand access to evidence-based screening for populations facing significant structural and psychological barriers to care.

Lab: A. Wong
Investigator: Dr. Alex Wong, MD
Research Summary: Lymphedema is a progressive and irreversible condition characterized by impaired lymphatic drainage, chronic limb swelling, pain, and recurrent infections, affecting over 250 million individuals globally and more than 3 million in the United States. Despite its widespread impact and rising incidence in cancer survivors, no FDA-approved drug therapies exist to restore lymphatic function, and current treatment options—including compression garments and manual lymphatic drainage—are labor-intensive, lifelong, and fail to address the root cause of lymphatic vessel damage. To overcome this challenge, one of our aims is to a develop a biodegradable, drug-eluting nanofiber scaffold designed to deliver sustained doses of small molecules known to promote lymphangiogenesis.
Project: The researcher will perform In vitro assessment of 9-cis retinoic acid-eluting nanofiber tubular scaffolds on lymphatic endothelial cells (LECs) as well as assess the ability to prevent lymphedema by implantation of drug eluting scaffolds in our established murine models of lymphedema.

Lab: T. Wong
Investigator: Dr. Tania Wong
Research Summary: Our lab uses advanced omics approaches—including single-cell RNA-Seq, metabolomics, spatial metabolite imaging, and proteomics—to study how infection-driven metabolic changes influence immune responses and infection outcomes. Using both in vitro and in vivo models with clinical and laboratory bacterial strains, we focus on how pathogens exploit the host metabolism to persist in the lung.
Project: We aim to define how various multidrug-resistant airway pathogens alter host immunometabolism and explore ways to target these changes to enhance bacterial clearance.

Lab: Yang
Investigator: Dr. Jason Yang
Research Summary: System biology of infectious disease
Project: Two potential projects. The first focuses on studying human macrophages, an important immune cell that is involved in protection from infectious diseases and cancer. This project involves either performing metabolism experiments with human macrophages or computationally analyzing macrophage experimental data.
The other project focuses on experimentally studying antimicrobial resistance in important human pathogens such as Klebsiella pneumoniae or Staphylococcus aureus.