



RICHARD KING MELLON FOUNDATION INSTITUTE FOR PEDIATRIC RESEARCH

Mission

The Richard King Mellon Foundation Institute for Pediatric Research fosters an environment of scientific excellence in which multidisciplinary teams of physicians and scientists work together on cutting-edge solutions in medical science.

The core goals of the institute are:

- To support a state-of-the-art laboratory unit for research in molecular and cellular biology
- To enable the recruitment of the most innovative and capable pediatric scientists by supporting their most promising work
- To develop a competitive edge in recruiting and retaining scarce clinical and research talent
- To strengthen the educational program for future pediatric leaders

FACULTY AND STAFF

Jay K. Kolls, MD

Director, Richard King Mellon Foundation Institute for Pediatric Research
Professor of Pediatrics and Immunology
Vice Chair for Translational Research
Interim Director, T32 Fellowship Training Award, Division of Rheumatology

Scott Canna, MD

Mellon Scholar, Richard King Mellon Foundation Institute for Pediatric Research
Assistant Professor of Pediatrics

Kong Chen, PhD

Assistant Professor of Pediatrics

Timothy W. Hand, PhD

Mellon Scholar, Richard King Mellon Foundation Institute for Pediatric Research
Assistant Professor of Pediatrics

Bernhard Kühn, MD, FACC

Mellon Scholar, Richard King Mellon Foundation Institute for Pediatric Research
Associate Professor of Pediatrics
Director of Research in Cardiology, Department of Pediatrics

Pawan Kumar, PhD

Research Instructor

Amanda C. Poholek, PhD

Assistant Professor of Pediatrics

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OVERVIEW

The pursuit of bold new advances for the treatment of some of the world's most debilitating childhood illnesses defines the work of the Richard King Mellon Foundation Institute for Pediatric Research.

Established through a groundbreaking gift from the Richard King Mellon Foundation, the institute is an incubator for research that challenges conventional wisdom, leading to paradigm shifts in pediatric medicine. This kind of high-risk, high-impact investigation is not typically funded through government or conventional sources, placing Children's Hospital of Pittsburgh of UPMC in a unique realm of pediatric research centers.

Located within the John G. Rangos Sr. Research Center at Children's Hospital, the institute's faculty and programs are a part of the University of Pittsburgh School of Medicine. The Richard King Mellon Foundation Institute was led by its director, Jay Kolls, through the end of June 2017. Bernhard Kühn leads the Richard King Mellon Foundation Institute as interim director effective July 2017. The institute supports the exceptionally talented Mellon Scholars and their teams by encouraging them to pursue their most innovative ideas. The result is a profound effect on understanding of the causes and treatment of pediatric diseases.



RESEARCH AND OTHER SCHOLARLY ACTIVITIES

Jay K. Kolls, MD**RESEARCH**

The major goal of Jay Kolls' research is to investigate mechanisms of lung host defenses in normal and immunocompromised hosts. Kolls' team is investigating how interleukin (IL)-23 and IL-17 regulate neutrophil recruitment in response to infectious stimuli in the lung. Team researchers study cellular sources of IL-17A, IL-17F, and IL-22 in the lung and liver, as well as their signaling in response to pulmonary infection or hepatitis. The team also has long-standing interests both in determining whether Th17 cells and their cytokine products contribute to airway destruction in cystic fibrosis (CF) and in understanding cytokine biology in the lung. The lung immunology work focuses on CD4+ T cells and their subsets in regulating mucosal immunity to extracellular pathogens in the lung.

Improved Therapeutics and Diagnostics for Pneumocystis Pneumonia. The long-term goal of this project is to use the recently released pneumocystis genomes to advance treatment and diagnostics of this critical fungal infection.

T Cells and Pneumocystis Carinii Pneumonia. The aims of this project are to understand mechanisms by which CD4+ T cells provide host defense against *Pneumocystis* infection.

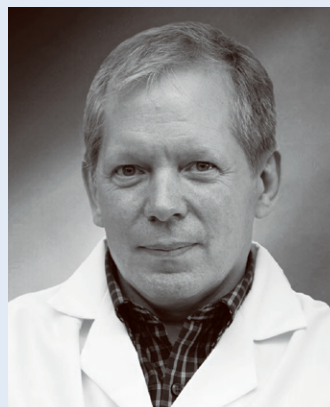
Tb17 Cytokines and Lung Immunity. The aims of this project are to understand the role of Th17 cytokines in mucosal immunity against *Klebsiella pneumoniae* and *Staphylococcus aureus* in the lung.

Immune Airway-Epithelial Interactions in Steroid-Refractory Severe Asthma. The purpose of this proposal is to establish a new paradigm for severe asthma based on which novel therapeutics could be developed in the future using cutting-edge immunological, cellular, and RNA sequencing techniques.

Scott W. Canna, MD**RESEARCH**

Inflammation is a core pathogenic mechanism in virtually every disease process. Systemically, this culminates in the systemic inflammatory response syndrome (SIRS), identified since ancient times as sepsis. Blocking inflammation in SIRS has been largely disappointing, conferring neither broad benefit nor harm. Scott Canna's research uses genetic and functional insights from patients and model

Generation of Novel Human Monoclonal Antibodies for Lung Disease. This project aims to clone memory B cells from the lungs of patients with CF to characterize the B-cell response and generate a panel of human monoclonal antibodies from the CF lung.



Jay Kolls, MD
Director, Richard King Mellon
Foundation Institute
for Pediatric Research

EDITORSHIPS

- Deputy editor, *Journal of Immunology*
- Consulting editor, *Journal of Clinical Investigation*
- Associate editor, *American Journal of Respiratory and Critical Care Medicine*
- Advisory editor, *Journal of Experimental Medicine*

PROFESSIONAL AFFILIATIONS/SOCIETY MEMBERSHIPS

- Southern Medical Association
- Associate, American College of Physicians
- American College of Chest Physicians
- American Thoracic Society
- American Society for Microbiology
- American Association for the Advancement of Science
- American Society of Gene and Cell Therapy
- Society for Immunotherapy of Cancer
- Southern Society for Clinical Investigation
- American Society for Clinical Investigation
- Association of American Physicians
- Member, F1000 Immunology

systems to find ways to subtype patients with SIRS/sepsis in diagnostically and therapeutically meaningful ways.

Patients found to have monogenic defects causing excessive innate immune responses have been particularly helpful. Most "autoinflammatory" patients have chronic organ-specific or systemic inflammation but not typically SIRS. The dramatic response of many "autoinflammatory" patients

to inhibition of the inflammasome-activated cytokine IL-1 has reinvigorated the quest for anti-inflammatory targets in SIRS and reinforced the therapeutic potential of targeting the inflammasome and related innate immune pathways.

Canna's group studies the intersections of hyper- and auto-inflammation. In particular, the group studies two related disorders, hemophagocytic lymphohistiocytosis (HLH) and macrophage activation syndrome (MAS), because they typify the concept of hyperinflammatory SIRS. Whereas the pathogenesis of HLH clearly includes the inflammatory effects of defects in granule-mediated cytotoxicity, the mechanisms at work in MAS are less clear. The researchers combine clinical insights from rheumatology and innate immunity with basic models of overwhelming systemic inflammation to define new disease subtypes and disease activity biomarkers, to flesh out mechanisms of inflammatory disease, and to test promising therapeutic strategies.

STUDY SECTIONS

- Abstract/grant review, Scientific Review Committee, Childhood Arthritis and Rheumatology Research Alliance
- Peer reviewer, Basic Science Section, Rheumatology Research Foundation Innovative Research Award
- Abstract reviewer, annual scientific meeting, American College of Rheumatology
- Peer reviewer, Fondation Innovations en Infectiologie

PROFESSIONAL AFFILIATIONS/SOCIETY MEMBERSHIPS

- American Academy of Pediatrics
- American College of Rheumatology
- Childhood Arthritis and Rheumatology Research Alliance Histiocyte Society

EDITORSHIPS

- Editorial Board, *Arthritis and Rheumatology*
- Editorial Board, *Frontiers in Immunology*

MAJOR LECTURESHIPS AND SEMINARS

- "IL-18 at the Intersection of Auto- and Hyperinflammation," University of Pittsburgh Department of Immunology Seminar Series, Pittsburgh, Pa., December 2016
- "Monogenic Inflammation: From Inflammasomes to SIRS," Pediatric Academic Society annual meeting, Baltimore, Md., May 2016
- "IL-18 at the Intersection of Auto- and Hyperinflammation," invited presentation, KFO 249 Symposium: Defects of the Innate Immune System in Autoinflammation and Autoimmunity, Dresden, Germany, August 2017
- "IL-18 at the Intersection of Auto- and Hyperinflammation," invited presentation, Federation of

Clinical Immunology Societies annual meeting, National Institutes of Health (NIH) Immunology Interest Group Symposium, Chicago, Ill., June 2017

- "Chronic IL-18 of Diverse Origins Defines and Drives the Hyperinflammatory Macrophage Activation Syndrome," Keystone Pattern Recognition Signaling Symposium, Banff, Alberta, Canada, March 2017
- "Cytokines in Systemic Inflammation: Following the Monogenic Breadcrumb Path," Children's Hospital of Pittsburgh Molecular Medicine Research Seminar, Pittsburgh, Pa., January 2017

Kong Chen, PhD

RESEARCH

Kong Chen is studying memory Th17 responses using mouse models of *Klebsiella pneumoniae*. Chen's previous work (*Immunity*, 2011;35(6):997-1009) demonstrated that immunization-induced Th17 cells provide serotype/antibody-independent protection against a variety of strains of *K. pneumoniae*, including the recently described multidrug-resistant New Delhi metallo-beta lactamase strain. These Th17 cells recognized conserved *Klebsiella* outer-membrane proteins (OMPs), and immunization with OMPs also conferred serotype/antibody-independent protection. Current work includes understanding the mechanism of generation of these memory Th17 cells, cloning the highly conserved OMPs from *K. pneumoniae*, and testing their immunogenicity as Th17-based vaccine candidates. Successful vaccine strategy can be used not only in prevention of multidrug-resistant bacteria outbreaks, but also in prophylactic treatment for immunocompromised patients.

Additionally, Chen is studying memory Th17 responses using macaque models of simian immunodeficiency virus (SIV) and *Streptococcus pneumoniae*. Both *K. pneumoniae* and *S. pneumoniae* are extracellular pathogens with polysaccharide capsules. The data from a mouse model with *K. pneumoniae* suggest that Th17 cells can provide clade-specific immune protection regardless of capsular serotypes. Chen found that Th17 responses are critical for pulmonary host defense against *S. pneumoniae* in rhesus macaques. Th17 cell frequency is increased in the antiretroviral therapy-treated SIV-infected macaques, which is associated with improved antibacterial immune responses after pulmonary *S. pneumoniae*. Ongoing research using next-generation sequencing will improve the understanding of the impacts of SIV, antiretroviral therapy, and bacterial infection on the lung transcriptome and identify biomarkers of mucosal immunity in the lung that may assist in disease management of individuals infected with human immunodeficiency virus.

Chen also studies pathogenic Th17 cells, especially their inflammatory roles leading to emphysema and lung cancer. He found that cigarette smoke is a strong Th17 adjuvant in the lung and that the Th17 pathway is involved in smoke-induced emphysema in mouse models. Th17 cell frequency is increased in lung cancer animal models. Carefully phenotyping the T cells in this model will increase the understanding of Th17 pathways in cancer and provide new therapeutic targets in lung cancer.

Chen is highly interested in cutting-edge, next-generation sequencing technology; he has developed several applications and is seeking NIH and CF Foundation support. Based on the preliminary data that the production of proinflammatory chemokines and cytokines from CF epithelial cells was elevated and that this elevated production can be abolished by BET inhibitor, a small molecule that inhibits epigenetic enhancement of gene transcription, Chen hypothesizes that the enhanced production of proinflammatory mediators is, in part, controlled by an epigenetic regulatory program that will be revealed in the chromatin accessibility landscape analysis by ATAC-seq, a newly established assay for transposase-accessible chromatin using next-generation sequencing, which provides information on nucleosome positioning, chromatin accessibility, and transcription factor binding simultaneously. This study is currently supported by the CF Foundation.

STUDY SECTIONS

- Reviewer for grants, Biotechnology and Biological Sciences Research Council

PROFESSIONAL AFFILIATIONS/SOCIETY MEMBERSHIPS

- American Association of Immunologists
- American Thoracic Society

EDITORSHIPS

- Lead guest editor, *Mediators of Inflammation*

HONORS AND AWARDS

- Outstanding Research Award, Pittsburgh-Munich International Lung Conference, 2016
- American Thoracic Society Abstract Scholarship, 2016

MAJOR LECTURESHIPS AND SEMINARS

- “Epithelial IL-17R Is Required for Pulmonary Host Defense Against *K. Pneumoniae*,” Gordon Research Conference on Biology of Acute Respiratory Infection, 2016

- “Anti-Inflammatory Effects of Bromodomain and Extra-Terminal Domain Inhibition in Cystic Fibrosis Lung Inflammation,” American Thoracic Society international conference, 2016
- “Updates on Research Projects Supported by the CF Foundation,” CF Center Seminar, University of Pittsburgh, Pittsburgh, Pa., January 2017

Timothy W. Hand, PhD

RESEARCH

The major goal of Timothy W. Hand’s research is to describe the role of immune response in intestinal disease. The laboratory is focused on the following projects.

The Role of Antibodies in Preventing the Development of Necrotizing Enterocolitis and Pediatric Crohn’s Disease. In collaboration with Mike Morowitz and Kevin Mollen, Hand is looking at whether monoclonal antibodies can be used to block the development of these diseases. In addition, he is investigating whether deficits in secreted antibody contribute to the development of disease.

Mucosal T-Cell Memory. Hand is looking at the factors necessary for the development of an effective memory T-cell response in the gastrointestinal tract and how these may be affected by chronic gastrointestinal conditions.

T-Cell Regulation and Inflammatory Bowel Disease. Hand is looking at the cellular requirements to prevent immunopathology in the gastrointestinal tract driven by T-cell responses against the commensal microbiota.

Immune Dysfunction in the Intestine of CF Patients. Using intestine-specific CF transmembrane conductance regulator knockout mice, the Hand laboratory is investigating whether loss of control over the microbiota by a dysfunctional immune response contributes to distal intestinal obstruction syndrome and cirrhosis.

PROFESSIONAL AFFILIATIONS/SOCIETY MEMBERSHIPS

- American Association of Immunologists
- Society for Mucosal Immunology
- Faculty of 1000

STUDY SECTIONS

- Grant reviewer, Wellcome Trust (United Kingdom)
- Grant reviewer, Deutsche Forschungsgemeinschaft (Germany)

MAJOR LECTURESHIPS AND SEMINARS

- “Establishing a New Gnotobiotic Facility: Education, Missions, and Accommodating Success,” Institute

for Laboratory Animal Research Workshop on Gnotobiotics, National Academy of Sciences, Washington, D.C., 2016

- “Long-Term Immune Consequences of Gastrointestinal Infection,” Molecular Medicine Research Seminar, Children’s Hospital of Pittsburgh of UPMC, Pittsburgh, Pa., 2016
- “A Holistic Approach to Immune-Bacterial Interactions in the Gastrointestinal Tract,” joint immunology/microbiology departmental retreat, Pittsburgh, Pa., 2016
- “Chronic Effects of Acute Gastrointestinal Infection,” Senior Vice Chancellor Seminar Series, Pittsburgh, Pa., 2016
- “Staying Frenemies—The Love/Hate Relationship Between the Microbiota and the Immune System,” Science 2016 Game Changers, Pittsburgh, Pa., 2016
- “Immunity to Intestinal Bacterial Colonization,” Louisiana State University Veterinary College, Baton Rouge, La., 2017
- “Maternal Antibodies, the Neonatal Microbiota, and Necrotizing Enterocolitis,” American Society for Investigative Pathology, Pittsburgh Pa., 2017
- “Maintenance of the Host/Microbiome Relationship in the Intestine,” Microbiology and Molecular Genetics Seminar, University of Pittsburgh, Pittsburgh Pa., 2017
- “Maintenance of the Host/Microbiome Relationship in the Intestine,” Molecular Medicine Research Seminar, Children’s Hospital of Pittsburgh of UPMC, Pittsburgh, Pa., 2017
- “Maintenance of the Host/Microbiome Relationship in the Gastrointestinal Tract,” plenary speaker, Cold Spring Harbor Laboratory meeting: Fundamental Immunology and Its Therapeutic Potential, Cold Spring Harbor, N.Y., 2017

Bernhard Kühn, MD, FACC

RESEARCH

The research laboratory that Bernhard Kühn directs has three interconnected goals: to understand the mechanisms of growth and regeneration in the heart; to provide mechanistic explanations for the huge differences in regenerative activity that exist in biology; and, drawing on the answers to these two fundamental questions, to conduct translational research for the diagnosis and treatment of heart muscle diseases. Prior to Kühn’s work, it was commonly thought that heart muscle cells, cardiomyocytes, are in irreversible proliferative arrest after birth and that myocardial regeneration cannot be increased in mammals. Physicians and

scientists were skeptical that it would be possible to stimulate cardiomyocyte proliferation after birth, let alone that this mechanism would regenerate myocardium. Kühn is credited with demonstrating that the postnatal mammalian heart has cardiomyocytes that can be stimulated to divide and that this process gives rise to myocardial regeneration. Kühn has developed an approach of using extracellular factors to stimulate cardiomyocyte proliferation. A peptide of periostin, a component of the extracellular matrix, stimulates cardiomyocyte proliferation and myocardial regeneration in a rat model of myocardial infarction. This work was published in *Nature Medicine* in 2007. Kühn followed up this seminal study with large-animal experiments published in 2012.

Kühn has shown that administration of neuregulin, a growth factor produced by endothelial cells in the heart muscle, stimulates cardiomyocyte proliferation and myocardial regeneration in animals. This paper was published in 2009 in *Cell*, the most prestigious journal for basic laboratory investigations. The work is especially significant because it raises the possibility of using subcutaneous administration of recombinant neuregulin in human patients.

Kühn has shown that both factors activate the same cellular mechanism, which is proliferation of a subpopulation of mononucleated cardiomyocytes. He has identified the receptors and intracellular pathways by which periostin peptide and neuregulin act on cardiomyocytes.

Kühn has developed a cellular growth chart of the human heart. His model shows that cardiomyocyte proliferation and enlargement contribute to developmental myocardial growth between birth and adulthood. This growth model significantly advances the conventional model, which had been the basis for medical textbooks since the 1950s. Kühn published this paper in *Proceedings of the National Academy of Sciences* in 2013. Kühn’s growth model of the human heart led him to make three important predictions. First, young humans may be able to regenerate heart muscle. Second, cardiomyocyte proliferation may be a mechanism that is altered in myocardial diseases. Third, it raises the possibility to stimulate cardiomyocyte proliferation therapeutically in children with the goal of promoting myocardial regeneration. Kühn’s current research efforts are based on these three advances.

STUDY SECTIONS

- Exploratory Grant Review Committee, Maryland Stem Cell Program
- Special Emphasis Panel, Pathway to Independence Awards, NIH
- Special Emphasis Panel, Program Project Parent Review Committee, National Heart, Lung, and Blood Institute, NIH
- Ad hoc reviewer, French Muscular Dystrophy Association
- Cardiac Contractility, Hypertrophy, and Failure Study Section, NIH
- Grant Review Committee, Translational Research Program, Boston Children's Hospital
- Biomaterials and Biointerfaces Study Section, NIH
- Special Emphasis Panel ZRG1 CVRS E, NIH
- Ad hoc reviewer, Research Foundation Flanders (Belgium)
- Ad hoc reviewer, Telethon Italy
- Ad hoc reviewer, Israel Science Foundation
- Basic Cell GE2 Study Section, American Heart Association

HONORS

- American Society of Clinical Investigation, 2016

PROFESSIONAL AFFILIATIONS/SOCIETY MEMBERSHIPS

- American Academy of Pediatrics
- American College of Cardiology
- American Association for the Advancement of Science
- Elected member, American Society of Clinical Investigation

MAJOR LECTURESHIPS AND SEMINARS

- “Postnatal Mammalian Cardiomyocyte Proliferation—When and How Much?” invited speaker and panelist, Weinstein Cardiovascular Development and Regeneration Conference, 2016
- “Growth Factor Injection for the Treatment of Heart Failure Cardiovascular Outcomes,” McGowan Institute for Regenerative Medicine Retreat, University of Pittsburgh, 2016
- “From Current Understanding to New Regenerative Concepts,” invited plenary speaker, Third Munich Conference on Cardiac Development, 2016
- “Genetic and Genomic Models of Polyploidy,” invited speaker and workshop, Allied Genetics Conference, Genetics Society of America, 2016
- Invited plenary speaker, Victor Chang Cardiac Research Institute 17th International Symposium, Sydney, Australia, 2016
- “Neuregulin Stimulation of Human Myocardium Reveals a Therapeutic Window,” invited plenary speaker, American Heart Association annual scientific sessions, 2016

- “Targeting Cardiomyocyte Proliferation for Heart Regeneration,” research seminar, John A. Burns School of Medicine, University of Hawaii at Manoa, 2016
- “Generation of Heart Muscle Cells in Humans,” research seminar, Pediatric Cardiology, Children's Hospital of Philadelphia, 2017
- “Heart Muscle Regeneration in Mammals,” Developmental and Stem Cell Biology Colloquium, Duke University, 2017
- “Heart Muscle Regeneration in Mammals,” research seminar, McAllister Heart Institute and Integrative Program for Biological and Genome Sciences, University of North Carolina Chapel Hill, 2017
- “A Transcriptional Map of Human Heart Muscle Cell Differentiation at the Single Cell Level,” Single Cell User Group Meeting, University of Pittsburgh Medical School, 2017

Pawan Kumar, PhD**RESEARCH**

Pawan Kumar is studying the role of IL-17 in regulating commensal microbiota colonization. Kumar found that enteric IL-17R signaling is critical for regulating colonization of segmented filamentous bacteria. His recent work suggests that enteric IL-17A/F signaling regulates the gut microbiota by regulating the levels of α -defensins, Nox1, and pIgR expression and that abrogation of this signaling pathway leads to gut dysbiosis, dysregulated expansion of Th17 cells, and an increased predisposition to autoimmunity. Kumar studies the role of IL-17R signaling in secretory and paneth cell development and function. Kumar's research will provide insights into IL-17-dependent regulation of host defense pathways and thus the interaction among the microbiome, intestinal immune cells, and enteric and autoimmune inflammation.

Kumar studies the role of enteric IL-17R signaling in regulating autoimmune hepatitis. IL-17R knockout mice are resistant to autoimmune hepatitis; Kumar's group found that IL-17RA knockout mice have expanded IL-22 responses in the serum. IL-22 has a protective role in the liver. Furthermore, intestinal IL-17R signaling regulates commensal microbiota-dependent IL-22 generation. Kumar discovered that intestinal IL-17R knockout mice have expanded IL-22 generation. Thus, IL-17R-dependent, gut-derived IL-22 could play a dominant role in hepatocyte regeneration and protection. A concanavalin-mediated liver-injury model will be used to investigate intestinal

IL-17R-signaling-dependent regulation of IL-22 in hepatocyte regeneration.

Kumar is also interested in studying the metabolic pathway in regulating Th17 cells, especially the role of Aryl hydrocarbon receptor (Ahr), Nrf2, and SOD3 axis in modulating metabolic programming of T cells. He found that Ahr-dependent regulation of Nrf2 and SOD3 regulates Th17 differentiation *in vitro*.

PROFESSIONAL AFFILIATIONS/SOCIETY MEMBERSHIPS

- American Association of Immunologists

Amanda C. Poholek, PhD

RESEARCH

The goal of the research program in Amanda C. Poholek's laboratory is to understand how transcription factor networks are regulated in a cell-type-specific manner to control the differentiation and function of immune cells. The Poholek laboratory is focused on the regulation and function of the transcriptional repressor Blimp-1 across multiple immune cell types. Blimp-1 is associated with constraining T-cell-mediated autoimmune disease and is highly expressed in exhausted T cells present in chronic viral infection and the tumor microenvironment. In addition, Blimp-1 is the master regulator of plasma cell formation in B cells. Therefore, Blimp-1 has wide-ranging functions depending on cell type, suggesting that its role in disease is context-dependent. Currently, the team is focused on T cells to understand the factors that regulate expression of Blimp-1 in CD4 and CD8 T cells, as well as to identify the non-coding genetic regulatory elements such as enhancers that are critical for cell-type-specific expression of Blimp-1.

The Poholek laboratory is also exploring the genes regulated by Blimp-1 in CD4 T-cell subsets using next-generation sequencing technologies such as ChIP-seq, RNA-seq, and ATAC-seq to understand the function of Blimp-1 in T cells. In addition, the team is extending *in vitro* observations to clinically relevant diseases such as allergic asthma to understand the functional consequences of Blimp-1 in disease settings.

Regulation of Blimp-1 in Immune Cells. The long-term goal is to identify the extracellular signals and genetic elements that control Blimp-1 expression in various immune cell types to understand how the regulation of Blimp-1 expression constrains T-cell-mediated autoimmunity or promotes allergic asthma.

Function of Blimp-1 in Immune Cells. The aim of this project is to identify the genes regulated by Blimp-1 both directly and indirectly in various immune cell types to understand the context-dependent and cell-type-specific functions of Blimp-1 in various disease settings.

Identify Blimp-1-Mediated Allergic Asthma Disease. The aim of this project is to understand why the selective absence of Blimp-1 in T cells results in reduced lung inflammation and development of allergic airway disease.

PROFESSIONAL AFFILIATIONS/SOCIETY MEMBERSHIPS

- American Association of Immunology

MAJOR LECTURESHIPS AND SEMINARS

- "IL-10 Induces an Autoregulatory Loop in T Cells That Promotes Blimp-1 Restriction of Cell Expansion," platform presentation, Cold Spring Harbor Laboratory meeting: Gene Expression and Signaling in the Immune System, Cold Spring Harbor, N.Y., April 2016

THREE-YEAR BIBLIOGRAPHY

2015

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