Immunobiology

Division Details

RESEARCH AND TRAINING DETAILS	
Faculty	**13**
Research Fellows and Post Docs	**18**
Research Graduate Students	**16**
Total Annual Grant Award Dollars	**$4,953,470**
Total Annual Industry Award Dollars	**$121,391**
Total Publications	**124**

Row 1: K McLay, E Janssen, Y Rochman, C Chougnet

Row 2: S Morris, S Divanovic, J Katz, C Kinder, I Castro, T Alenghat

Row 3: A Herr, K Hoebe, HL Grimes, I Lewkowich, F Finkelman, K Cole, D Hildeman, H Singh

Research Highlights

Research Advances

Faculty in the division continue to generate new insights into cellular and molecular mechanisms underlying protective and pathogenic immune responses. The Jordan lab has demonstrated that patients with a mutation in the LRBA gene which result in an autoimmune syndrome, can be successfully treated with Abatacept, a CTLA4-Ig fusion protein. In collaboration with the Lenardo lab (NIH), they revealed that LRBA controls the intracellular trafficking and surface expression of CTLA4 thereby providing a mechanistic basis for the efficacy of CTLA4-Ig. This is a powerful demonstration of moving research from the bedside to the bench, thereby enabling clinical investigations to reveal fundamental cell biological mechanisms operative in the immune system. The Grimes lab has generated a new mouse model of AML which is rapidly-lethal, completely-penetrant, transplantable and importantly displays a normal karyotype. It is based on Flt3-internal-tandem duplication (Flt3ITD) and inducible deletion of Dnmt3a. This is a powerful translational model for karyotypically normal human AML involving mutations in Flt3 and Dnmt3a genes. The Grimes and Singh labs are exploiting single cell transcriptional profiling to analyze normal hematopoiesis. This has led to the discovery of unusual mixed-lineage intermediates in myelopoiesis. The Singh lab has uncovered a regulatory module involving cross antagonism between the transcription factors IRF4 and IRF8 that controls affinity maturation of B cells and plasma cell differentiation. This work could enable the discovery of new biomarkers and drug targets for enhancing vaccine responses. The Chougnet lab is attempting to understand the effect of prematurity and inflammation, on the developing immune system. They have established that in utero exposure to IL-1 mediated inflammation affects the balance between fetal regulatory and effector T cells. Such dysregulation could explain the increased pulmonary morbidity in infancy that follows exposure to chorioamnionitis. While exploring T cell homeostasis, the Hildeman lab has shown that aged mice accumulate thymically derived regulatory T cells (Treg) having effector phenotype. IL-6 and ICOS signaling promote the accrual of effector Tregs,

defining a novel feedback mechanism whereby inflammatory stimuli provoke a cellular anti-inflammatory response. Work in the Lewkowich lab is examining how the cytokine IL-17 functions in conjunction with IL-13 to induce more severe allergic asthma. Notably they have shown that IL-17 enhances IL-13-induced gene expression in asthma-relevant murine models and human epithelial cells via a mechanism involving more robust activation of Stat6. These findings represent the first mechanistic explanation of how IL-17 may directly contribute to the pathogenesis of IL-13-driven allergic asthma. The Herr lab in collaboration with the Farndale lab (University of Cambridge) has solved the crystal structures of the immune receptor OSCAR (osteoclast-associated receptor) alone and bound to its target, a collagen triple helix. This is the first structural data illustrating how an immune receptor in this family recognizes a collagenous ligand, and it has important implications for therapeutic intervention in osteoclast-associated disease states such as osteoporosis and rheumatoid arthritis.

Inter-Disciplinary Collaborations

Dr. Claire Chougnet, PhD, successfully spearheaded an inter-disciplinary collaborative proposal focused on perinatal infection and inflammation selected by the Academic Research Committee (ARC) for support. It involves a collaboration with colleagues in other divisions including Drs. Suhas Kallapur, Alan Jobe and Sing Sing Way.

Faculty Recruitment

Drs. Harinder Singh and David Hildeman conducted and led faculty searches in systems immunology and transplantation immunology, respectively. This led to the identification of two faculty candidates in systems immunology for recruitment, Wendy Huang (NYU) and Emily Miraldi (NYU). Dr. Virginia Miraldi will be joining the Division of Immunobiology as a faculty member in January 2017.

Systems Immunology Workshop

A workshop focused on systems immunology was co-organized by Drs. Singh and Herr. Accomplished postdoctoral fellows, from leading labs, made up the majority of invited speakers.. These next-generation scientists shared advances they are making in the analysis of gene regulatory and signaling networks; tracking of human immune responses using tools drawn from systems biology, and engineering of immune cells and immune receptors. The workshop elicited considerable interest from biotech and pharma companies, and was partially funded by AbbVie and Lycera. The workshop was a tremendous success and generated considerable institutional excitement for this emerging field that will greatly impact Immunology.

SAC Review

The division underwent a highly successful external review of its research and training programs. The research advances made by the faculty, along with the newly established inter-disciplinary collaborations, as well as the recruitment of faculty in systems immunology, were laudable. The two graduate training programs, Immunology (Cincinnati Children's and University of Cincinnati) and International Research Training Program (IRTG) (University of Lubeck) are regarded as excellent, with the latter noted for spawning international research collaborations.

Significant Publications

Meyer SE, Qin T, **Muench DE**, Masuda K, Venkatasubramanian M, **Orr E**, Suarez L, Gore SD, Delwel R, Paietta E, Tallman MS, Fernandez H, Melnick A, Le Beau MM, Kogan S, Salomonis N, Figueroa ME, **Grimes HL. DNMT3A Haploinsufficiency Transforms FLT3ITD Myeloproliferative Disease into a Rapid, Spontaneous, and Fully Penetrant Acute Myeloid Leukemia**. *Cancer Discov*. 2016 May;6(5):501-15.

Mutations in the FLT3 and DNMT3A genes are frequently found in the same Acute myelogenous leukemia (AML). In Cancer Discovery, a research team demonstrated that combining mutations in the corresponding murine genes produces a spontaneous model of human AML. Notably, DNMT3A haploinsufficiency results in reversible epigenetic alterations that transform FLT3-mutant myeloproliferative neoplasm into AML. Dissection of the cellular architecture of the AML model using single-cell RNA-Seq, flow cytometry and colony assays identified clonogenic subpopulations that differentially express genes that are sensitive to the methylation of nearby genomic loci, and varied in response to Dnmt3a levels.

Xu H, Chaudhri VK, Wu Z, Biliouris K, **Dienger-Stambaugh K, Rochman Y**, **Singh H**. **Regulation of bifurcating B cell trajectories by mutual antagonism between transcription factors IRF4 and IRF8**. *Nat Immunol*. 2015 Dec;16(12):1274-81.

Upon recognition of antigen, B cells undertake a bifurcated response in which some cells rapidly differentiate into plasmablasts that secrete low affinity antibodies while others undergo affinity maturation in germinal centers (GCs) to generate plasma cells that produce high affinity antibodies. The Singh lab has identified a double-negative feedback loop between the transcription factors IRF4 and IRF8 that regulates the bifurcated humoral immune response. IRF8 dampens signaling via the B cell antigen receptor (BCR), facilitates antigen-specific interaction with helper T cells, and promotes affinity maturation while antagonizing IRF4-driven differentiation of plasmablasts. These regulatory factors are used as predictive biomarkers and also targeted to enhance vaccine responses.

Hall SL, Baker T, Lajoie S, **Richgels PK, Yang Y, McAlees JW**, van Lier A, Wills-Karp M, Sivaprasad U, Acciani TH, LeCras TD, Myers JB, Kovacic MB, **Lewkowich IP**. **IL-17A enhances IL-13 activity by enhancing IL-13-induced signal transducer and activator of transcription 6 activation**. *J Allergy Clin Immunol*. 2016 Jun 11. pii: S0091-6749(16)30430-4.

While pathogenesis of allergic asthma is widely regarded as mediated by Th2 cells and production of Th2-associated cytokines like IL-13, emerging evidence in both mouse and humans suggest that allergic asthma with a mixed Th2/Th17 inflammatory profile is typically more severe. As severe asthmatics are often refractory to commonly available therapeutics, and are at elevated risk of hospitalization or death as a result of their disease, we need a greater understanding of the unique processes governing more severe forms of disease. Work from the Lewkowich lab examining the role of IL-17A, a factor produced by Th17 cells associated with more severe disease begins to identify how Th2 and Th17-derived factors may interact to cause more severe disease. Compared to mice given intratracheal IL-13 alone, those exposed to IL-13 and IL-17A displayed augmented airway hyperreactivity (AHR), mucus production, airway inflammation and IL-13-induced gene expression. In vitro, IL-17A enhanced IL-13-induced gene expression in asthma-relevant murine and human cells. In contrast to the exacerbating influence of IL-17A on IL-13-induced responses, co-exposure to IL-13 inhibited IL-17A-driven antimicrobial gene expression in vivo and in vitro. Mechanistically, in both primary human and murine cells, IL-17A-driven elevation of IL-13-induced gene expression is associated with enhanced IL-13-driven STAT6 activation. These data represent the first mechanistic explanation of how IL-17A may directly contribute to the pathogenesis of IL-13-driven pathology.

Raynor J, Karns R, **Almanan M**, **Li KP**, **Divanovic S**, **Chougnet CA**, **Hildeman DA**. **IL-6 and ICOS Antagonize Bim and Promote Regulatory T Cell Accrual with Age**. *J Immunol*. 2015 Aug 1;195(3):944-52.

Age-related immune suppression contributes to morbidity and mortality in aged individuals. In addition to intrinsic defects in lymphocytes, in collaboration with Dr. Chougnet, we have found that aged mice and humans have substantially increased levels of an immune-suppressive population of cells, so-called regulatory T cells (Treg). In this paper, we found that the Treg that accumulate with age are predominantly thymically derived. Interestingly, their accumulation relies on IL-6 signaling and the inducible co-stimulatory molecule, ICOS. Thus, these data illustrate a novel feedback mechanism, whereby pro-inflammatory signals (e.g. IL-6) provoke a negative feedback loop which engages an anti-inflammatory cell population that likely contributes to age-driven immune suppression.

Rueda CM, Moreno-Fernandez ME, Jackson CM, Kallapur SG, Jobe AH, **Chougnet CA**. **Neonatal regulatory T cells have reduced capacity to suppress dendritic cell function**. *Eur J Immunol*. 2015 Sep;45(9):2582-92.

Regulatory T cells (Treg cells) are essential to maintain immune homeostasis. They exert their function in multiple targets, but their control of antigen-presenting cells is the most critical functionally. However, the functionality of neonatal Treg cells remains poorly characterized. We have developed an in vitro model in which Treg cell function are measured at a physiological ratio of Treg cells, conventional T cells and antigen-presenting cells. Using this model, we showed that neonatal Treg cells were only partially efficient, but this defect was not a reflection of the increased proportion of naïve Treg cells in infants. Prematurity did not appear to further affect Treg cell functionality. In addition, we identified CTLA-4 and cyclic AMP as the main suppressive molecules used by neonatal Treg cells. Altogether, our data imply a developmentally regulated maturation of Treg cell function. Furthermore, the decreased capacity of neonatal Treg cells to control antigen-presenting cell activation may contribute to the exacerbated inflammatory diseases seen in neonates, particularly in the context of in utero exposure to severe chorioamionitis.

Division Publications

1. Ambroggio L, Sucharew H, Rattan MS, O'Hara SM, Babcock DS, Clohessy C, Steinhoff MC, Macaluso M, Shah SS, Coley BD. **Lung Ultrasonography: A Viable Alternative to Chest Radiography in Children with Suspected Pneumonia?** *J Pediatr*. 2016; 176:93-98 e7.

2. Ambroggio L, Test M, Metlay JP, Graf TR, Blosky MA, Macaluso M, Shah SS. **Beta-Lactam Versus Beta- Lactam/Macrolide Therapy in Pediatric Outpatient Pneumonia.** *Pediatr Pulmonol*. 2016; 51:541-8.

3. Ambroggio L, Test M, Metlay JP, Graf TR, Blosky MA, Macaluso M, Shah SS. **Comparative Effectiveness of Beta-Lactam Versus Macrolide Monotherapy in Children with Pneumonia Diagnosed in the Outpatient Setting.** *Pediatr Infect Dis J*. 2015; 34:839-42.

4. Appelberg R, Moreira D, Barreira-Silva P, Borges M, Silva L, Dinis-Oliveira R, Resende M, Correia-Neves M, Jordan M, Ferreira N. **The Warburg Effect in Mycobacterial Granulomas Is Dependent on the Recruitment and Activation of Macrophages by Interferon-Gamma.** *Immunology*. 2015; 145:498-507.

5. Aronson PL, Williams DJ, Thurm C, Tieder JS, Alpern ER, Nigrovic LE, Schondelmeyer AC, Balamuth F, Myers AL, McCulloh RJ, Alessandrini EA, Shah SS, Browning WL, Hayes KL, Feldman EA, Neuman MI, Febrile Young Infant Research Collaborative. **Accuracy of Diagnosis Codes to Identify Febrile Young Infants Using Administrative Data.** *J Hosp Med*. 2015; 10:787-93.

6. Auger KA, Davis MM. **Pediatric Weekend Admission and Increased Unplanned Readmission Rates.** *J Hosp Med*. 2015; 10:743-5.

7. Auger KA, Jerardi KE, Simmons JM, Davis MM, O'Toole J, Sucharew HJ. **It's Complicated: Overcoming Statistical Challenges of Group Learning in Medical Education Research.** *J Grad Med Educ*. 2015; 7:326-30.

8. Auger KA, Kahn RS, Davis MM, Simmons JM. **Reply: To Pmid 25241184.** *J Pediatr*. 2015; 167:213-4.

9. Auger KA, Mueller EL, Weinberg SH, Forster CS, Shah A, Wolski C, Mussman G, Ipsaro AJ, Davis MM. **A Validated Method for Identifying Unplanned Pediatric Readmission.** *J Pediatr*. 2016; 170:105-12 e1-2.

10. Balamuth F, Weiss SL, Hall M, Neuman MI, Scott H, Brady PW, Paul R, Farris RW, McClead R, Centkowski S, Baumer-Mouradian S, Weiser J, Hayes K, Shah SS, Alpern ER. **Identifying Pediatric Severe Sepsis and Septic Shock: Accuracy of Diagnosis Codes.** *J Pediatr*. 2015; 167:1295-300 e4.

11. Beck AF, Florin TA, Campanella S, Shah SS. **Geographic Variation in Hospitalization for Lower Respiratory Tract Infections across One County.** *JAMA Pediatr*. 2015; 169:846-54.

12. Bednar KJ, Tsukamoto H, Kachapati K, Ohta S, Wu Y, Katz JD, Ascherman DP, Ridgway WM. **Reversal of New-Onset Type 1 Diabetes with an Agonistic Tlr4/Md-2 Monoclonal Antibody.** *Diabetes*. 2015; 64:3614-26.

13. Brower L, Schondelmeyer A, Wilson P, Shah SS. **Testing and Empiric Treatment for Neonatal Herpes Simplex Virus: Challenges and Opportunities for Improving the Value of Care.** *Hosp Pediatr*. 2016; 6:108-11.

14. Cappelletti M, Della Bella S, Ferrazzi E, Mavilio D, Divanovic S. **Inflammation and Preterm Birth.** *J Leukoc Biol*. 2016; 99:67-78.

15. Chen XL, Serrano D, Ghobadi F, Mayhue M, Hoebe K, Ilangumaran S, Ramanathan S. **Tcr and Il-7 Signaling Are Altered in the Absence of Functional Gtpase of the Immune Associated Nucleotide Binding Protein 5 (Gimap5).** *PLoS One*. 2016; 11:e0151837.

16. Chen X-L, Serrano D, Mayhue M, Hoebe K, Ilangumaran S, Ramanathan S. **Gimap5 Deficiency Is Associated with Increased Akt Activity in T Lymphocytes.** *Plos One*. 2015; 10:e0139019.

17. Chougnet C, Thacker R, Shehata H, Hennies C, Lehn M, Lages C, Janssen E. **Loss of Phagocytic and Antigen Cross-Presenting Capacity in Aging Dendritic Cells Is Associated with Mitochondrial Dysfunction.** *J Immunol*. 2015; 195:2624-32.

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21. Florin TA, Aronson PL, Hall M, Kharbanda AB, Shah SS, Freedman SB, Alpern ER, Mistry RD, Simon HK, Berry J, Coley BD, Neuman MI. **Emergency Department Use of Computed Tomography for Children with Ventricular Shunts.** *J Pediatr*. 2015; 167:1382-8 e2.

22. Forster CS, Jerardi KE, Herbst L, Brady PW. **Right Test, Wrong Patient: Biomarkers and Value.** *Hosp Pediatr*. 2016; 6:315-7.

23. Gallagher P, Shah S, Windle M, Carter B, Rosenkrantz T, Springer S. **Omphalitis**. New York: Medscape; 2016 [updated January 2, 2016].

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25. Giacomin P, Moy R, Noti M, Osborne L, Siracusa M, Alenghat T, Liu B, McCorkell K, Troy A, Rak G. **Epithelial-Intrinsic Ikk Alpha Expression Regulates Group 3 Innate Lymphoid Cell Responses and Antibacterial Immunity.** *J Exp Med*. 2015; 212:1513-28.

26. Giles DA, Moreno-Fernandez ME, Stankiewicz TE, Cappelletti M, Huppert SS, Iwakura Y, Dong C, Shanmukhappa SK, Divanovic S. **Regulation of Inflammation by Il-17a and Il-17f Modulates Non-Alcoholic Fatty Liver Disease Pathogenesis.** *PLoS One*. 2016; 11:e0149783.

27. Gisslen T, Alvarez M, Wells C, Soo MT, Lambers DS, Knox CL, Meinzen-Derr JK, Chougnet CA, Jobe AH, Kallapur SG. **Fetal Inflammation Associated with Minimal Acute Morbidity in Moderate/Late Preterm Infants.** *Arch Dis Child Fetal Neonatal Ed*. 2016.

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43. Kinnear B, O'Toole JK. **Care of Adults in Children's Hospitals: Acknowledging the Aging Elephant in the Room.** *JAMA Pediatr*. 2015; 169:1081-2.

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Grants, Contracts, and Industry Agreements

Annual Grant Award Dollars

Investigator	Title	Sponsor	ID	Dates	Amount
Theresa Alenghat	Epigenomic Integration of Microbiota-derived Signals in the Pathogenesis of IBD	Crohn's & Colitis Foundation of America	Alenghat CCFA 2015	10/1/2015 - 9/30/2018	$100,000
Theresa Alenghat	Epigenomic Regulation of Host-microbiota Interactions	The Pew Charitable Trusts	Alenghat PEWS 15-16	8/1/2015 - 7/31/2019	$60,000
Theresa Alenghat	Epigenomic Regulation of the Host-commensal Relationship	Burroughs Wellcome Foundation (University of Cincinnati)	Alenghat UC/BWF	9/1/2014 - 8/31/2019	$140,000
Theresa Alenghat	Epigenetic Regulation of Intestinal Homeostasis	National Institutes of Health	K08 DK093784	9/19/2014 - 3/31/2017	$150,444
Julio Aliberti, PHD	Lipoxins and Control of Inflammation during Cerebral Malaria	National Institutes of Health	R21 AI118302-01	7/1/2015 - 6/30/2017	$234,000
Claire A Chougnet, PHD Louis Muglia	Maternal Temperament, Stress, and Inflammation in Preterm Birth	National Institutes of Health	R01 HD078127	9/1/2013 - 8/31/2017	$310,298
Claire A Chougnet, PHD	Host-microbe Cross-talk and	Burroughs Wellcome	1012534	6/1/2013 -	$150,000

	Pregnancy Outcomes	Foundation (University of Cincinnati)		5/31/2017	
Claire A Chougnet, PHD David A Hildeman, PHD	Homeostasis and Function of Regulatory T cells in Aging	National Institutes of Health	R01 AG033057	8/1/2012 - 4/30/2016	$578,960
Senad Divanovic, PHD	Immunopathogenesis of Non-alcoholic Fatty Liver Disease	National Institutes of Health	R01 DK099222	9/5/2013 - 7/31/2016	$420,135
Senad Divanovic, PHD	Immune Mechanisms of Inflammation-induced Preterm Birth	Burroughs Wellcome Foundation (University of Cincinnati)	Senad BWF 1015032	6/1/2015 - 3/31/2019	$300,000
Harry Leighton Grimes, PHD	Genetic Dissection of Cytogenetically Normal AML	Alex's Lemonade Stand Foundation	Grimes ALS POST Awar	6/6/2016 - 7/29/2016	$5,000
Harry Leighton Grimes, PHD	Understanding Induction Chemotherapy Failure	Cancer Free Kids	Grimes CFK	7/1/2015 - 6/30/2017	$45,000
Harry Leighton Grimes, PHD	Mechanisms of Granulocyte Homeostasis	National Institutes of Health	R01 HL122661	7/1/2015 - 4/30/2019	$780,000
Harry Leighton Grimes, PHD	Developing Novel STAT5 Protein Inhibitors for Treatment of Leukemias	National Institutes of Health	R21 CA186945	9/1/2014 - 8/31/2016	$178,315
David A Hildeman, PHD	PEARL: Pathway Exploration and Analysis in Renal Lupus	National Institutes of Health (Feinstein Institute for Medical Research)	UH2 AR067688 Hildeman	6/1/2015 - 5/31/2017	$49,346
Edith Janssen, PHD	Induction of Anti-tumor Responses by MRI-guided High Intensity Ultrasound	Cancer Free Kids	Janssen CFK	7/1/2015 - 6/30/2017	$45,000
Edith Janssen, PHD	CD244 Targeting Therapeutics in SLE	Lupus Research Institute	LRI_Janssen	1/1/2015 - 12/31/2017	$100,000
Edith Janssen, PHD	Effect of Different MRgHIFU Approaches on Anti-tumor Responses	National Institutes of Health	R03 CA201918	1/19/2016 - 12/31/2017	$78,000
Michael B Jordan, MD David A Hildeman, PHD	Exploiting the DNA Damage Response to Selectively Sculpt the T Cell Repertoire	National Institutes of Health	R01 AI109810	7/15/2014 - 3/31/2018	$390,000
Durga Krishnamurthy, PHD	Rapid Suppression of Food Allergy with Anti-FceRIa Antibody	Intermountain Medical Center (University of Cincinnati)	Finkelman/Krishnamur	7/22/2014 - 9/30/2017	$82,858
Durga Krishnamurthy, PHD	Induction of Food Allergy in Mice by Allergen Inhalation	Department of Defense (Cincinnati Educ & Res for Veterans Fdn)	W81XWH-13-1-0497	9/30/2013 - 9/29/2016	$131,972
Ian P Lewkowich	Prenatal HDM Exposure and	National Institutes of	P30 ES006096	8/1/2015 -	$15,000

	Offspring Asthma Severity	Health (University of Cincinnati)		3/31/2016	
Ian P Lewkowich	Mechanisms of IL-17A-mediated Enhancement of Asthma Severity	National Institutes of Health	R01 HL122300	5/1/2014 - 2/28/2019	$390,000
Ian P Lewkowich	Study of Activity-Dependent Sympathetic Sprouting	National Institutes of Health (University of Cincinnati)	R01 NS045594 - Zhang	2/1/2016 - 1/31/2021	$8,332
Alexey Porollo, PHD	Suppression of IgE-Mediated Disease by Polyclonal Rapid Desensitization	National Institutes of Health (University of Cincinnati)	R01 AI113162	7/15/2014 - 6/30/2018	$87,170
Harinder Singh, PHD	Distinct Functional Outcomes of BCR/TLR7 and BCR/TLR9 Co-Engagement	National Institutes of Health (Univ of Massachusetts Medical School)	R01AR066808	3/12/2015 - 2/29/2020	$68,640
Heping Xu, PHD	Interplay of IRF4 and IRF8 in Orchestrating Cell Fate Dynamics of Germinal Center B Cells	The Leukemia and Lymphoma Society	Xu LLS CDP	7/1/2015 - 6/30/2018	$55,000
Total Annual Grant Award Dollars					**$4,953,470**

Annual Industry Award Dollars

Investigator	Industry Sponsor	Amount
Andrew Herr, PHD	Airway Therapeutics & Steve Linberg	$121,391
Total Annual Industry Award Dollars		**$121,391**