Allergy and Immunology

Division Photo

First Row: N. Zimmermann, A. Assa'ad, M. Rothenberg; Second Row: A. Mishra, J.P. Abonia, L. Zuo, YH. Wang, K. von Tiehl, A. Munitz, S. Hogan, K. Risma, M. Lierl

Division Data Summary

Research and Training Details

Number of Faculty	14
Number of Joint Appointment Faculty	2
Number of Research Fellows	7
Number of Research Students	5
Number of Support Personnel	6
Direct Annual Grant Support	$5,036,336
Direct Annual Industry Support	$258,157
Peer Reviewed Publications	27

Clinical Activities and Training

Number of Clinical Staff	1
Number of Clinical Fellows	6
Number of Clinical Students	2
Number of Other Students	6
Inpatient Encounters	595
Outpatient Encounters	6,250

Significant Publications

Groschwitz KR, Ahrens R, Osterfeld H, Gurish MF, Han X, Abrink M, Finkelman FD, Pejler G, Hogan SP. Mast cells regulate homeostatic intestinal epithelial migration and barrier function by a chymase/Mcpt4-dependent mechanism. Proc Natl Acad Sci U S A2009 Dec 29;106(52):22381-6.

Altered intestinal barrier function is postulated to be a central predisposing factor to intestinal diseases, including inflammatory bowel diseases and food allergies. However, the mechanisms involved in maintaining homeostatic

intestinal barrier integrity remain largely undefined. A recent study by Groschwitz et al. identified a mechanism by which mast cells regulate homeostatic intestinal epithelial migration and barrier function. These findings further the basic understanding of intestinal barrier function, which is a necessary precursor to improved disease management and treatment.

Kottyan LC, Collier AR, Cao KH, Niese KA, Hedgebeth M, Radu CG, Witte ON, Khurana Hershey GK, Rothenberg ME, Zimmermann N. Eosinophil viability is increased by acidic pH in a cAMP- and GPR65-dependent manner. Blood2009 Sep 24;114(13):2774-82.

The microenvironment of the lung in asthma is acidic, yet the effect of acidity on inflammatory cells has not been well established. A recent study by Kottyan et al. demonstrated that acidity inhibits eosinophil apoptosis and increases cellular viability in a dose-dependent manner between pH 7.5 and 6.0, mainly via the G protein-coupled receptor 65 (GPR65). Notably, GPR65-deficient mice had attenuated airway eosinophilia and increased apoptosis in two distinct models of allergic airway disease. These animal model findings hint that reducing the increased eosinophil viability in the acidic microenvironment of the asthmatic lung may alleviate or lessen eosinophil-derived symptoms.

Abonia JP, Blanchard C, Butz BB, Rainey HF, Collins MH, Stringer K, Putnam PE, Rothenberg ME. Involvement of mast cells in eosinophilic esophagitis. J Allergy Clin Immunol2010 Jun 8.

Eosinophilic esophagitis is an emerging disorder with poorly understood pathogenesis. Whereas prior studies have primarily focused on the role of eosinophils in disease diagnosis and pathogenesis, a recent study by Abonia et al. investigated the involvement of mast cells. The investigators identified local mastocytosis and mast cell degranulation in the esophagi of patients with eosinophilic esophagitis, defined an esophageal mast cell-associated transcriptome that is significantly divergent from the eosinophil-associated transcriptome, and provided evidence for the involvement of KIT ligand in the pathogenesis of eosinophilic esophagitis. These findings unveil several of the further complexities of eosinophilic esophagitis pathogenesis in humans.

Blanchard C, Stucke EM, Burwinkel K, Caldwell JM, Collins MH, Ahrens A, Buckmeier BK, Jameson SC, Greenberg A, Kaul A, Franciosi JP, Kushner JP, Martin LJ, Putnam PE, Abonia JP, Wells SI, Rothenberg ME. Coordinate interaction between IL-13 and epithelial differentiation cluster genes in eosinophilic esophagitis. J Immunol2010 Apr 1;184(7):4033-41.

Aiming to uncover molecular explanations for eosinophilic esophagitis pathogenesis, a recent study by Blanchard et al. compared epithelial responses between healthy patients and those with eosinophilic esophagitis. Their findings establish that the epithelial response in eosinophilic esophagitis involves a cooperative interaction between IL-13 and expression of epithelial differentiation complex genes.

Rothenberg ME, Spergel JM, Sherrill JD, Annaiah K, Martin LJ, Cianferoni A, Gober L, Kim C, Glessner J, Frackelton E, Thomas K, Blanchard C, Liacouras C, Verma R, Aceves S, Collins MH, Brown-Whitehorn T, Putnam PE, Franciosi JP, Chiavacci RM, Grant SF, Abonia JP, Sleiman PM, Hakonarson H. Common variants at 5q22 associate with pediatric eosinophilic esophagitis. Nat Genet2010 Apr;42(4):289-91.

Eosinophilic esophagitis is an allergic disorder characterized by the accumulation of eosinophils in the esophagus. A recent study by Rothenberg et al. identified the first association of eosinophilic esophagitis with a chromosomal locus, specifically with variants at 5q22. Gene expression data from esophageal biopsies from individuals with eosinophilic esophagitis compared with unaffected individuals implicate the 5q22 locus in the pathogenesis of eosinophilic esophagitis and identify TSLP as the most likely candidate gene in the region.

Division Highlights

Genetic Region Identified for a Children's Food Allergy

Division director Marc Rothenberg, MD, PhD, post-doctorate Joseph Sherill, PhD, and colleagues have identified the first major gene location responsible for eosinophilic esophagitis (EE), a severe, often painful type of food allergy that leaves its victims unable to eat a wide variety of foods (Rothenberg ME, et al. *Nature Genetics*. 2010;42(4):289-91). The genome-wide analysis studies implicated the 5q22 chromosomal locus in the pathogenesis of EE and identified thymic stromal lymphopoietin (TSLP) as the most likely candidate gene in the region with specific genetic variants in TSLP and its receptor specifically linking with EE compared with other allergic diseases (Sherrill JD, et al. *Journal of Allergy and Clinical Immunology*. 2010;126(1):160-165).

National Registry to Track Eosinophilic Disorders

A $1.6 million federal economic stimulus grant awarded by the National Institute of Diabetes and Digestive and Kidney Diseases has helped to launch the Registry for Eosinophilic Gastrointestinal Disorders (REGID), developed by division director Marc Rothenberg, MD, PhD, along with division faculty Pablo Abonia, MD, and CCHMC co-investigators James Franciosi, MD, and Keith Marsolo, PhD; see www.regid.org. The registry is the first of its kind for eosinophilic disorders and will allow participating centers around the nation to build a database of research-accessible clinical information for thousands of patients coping with eosinophilic disorders.

Interleukin 15 Involved in Eosinophilic Esophagitis

A recent study by associate professor Anil Mishra, PhD, research associate Xiang Zhu, PhD, and colleagues has reported the significance of the induced expression and protein levels of interleukin 15 (IL-15) in human and experimental eosinophilic esophagitis (EE). Notably, transcript levels of IL-15 strongly correlated with esophageal eosinophils in patients with active EE and significantly decreased in patients with improved treated EE, and mouse models of allergen-induced EE demonstrated that the IL-15 receptor, IL-15Rα, was necessary for the development of EE (Zhu X, et al. *Gastroenterology*. 2010;139(1):182-193.e7).

Division Co-director Supports CCHMC's National and Global Mission

During this past year, co-director Amal Assa'ad, MD, has exemplified the national and global mission of Cincinnati Children's Hospital Medical Center (CCHMC) through her far-reaching dedication and valued efforts to improve child health. She has represented CCHMC as an invited speaker at plenary sessions and international symposia at three national meetings in the USA and five international meetings in South America, Europe, Asia, and the Middle East. In addition, she has contributed as a reviewer of the first evidence-based guidelines for food allergy by the World Allergy Organization (WAO), the WAO Diagnosis and Rationale Against Cow Milk Allergy (DRACMA) Guidelines, and as a writer and writing section chair for the NIH Expert Panel on Food Allergy Guidelines.

Mast Cells Regulate Homeostatic Intestinal Epithelial Migration and Barrier Function

A recent study by associate professor Simon Hogan, PhD, graduate student Katherine Groschwitz, and colleagues has identified a chymase / mast cell protease 4 -dependent mechanism by which mast cells regulate homeostatic intestinal epithelial migration and barrier function (Groschwitz et al. *Proceedings of the National Academy of Science of the United States of America*. 2009;106(52):22381-6).

Eosinophil Viability Increased in Acidic Microenvironment

A recent study by associate professor Nives Zimmermann, MD, graduate student Leah Kottyan, and colleagues has demonstrated that acidity inhibits eosinophil apoptosis and increases cellular viability in a dose-dependent manner between pH 7.5 and 6.0, mainly via the G protein-coupled receptor 65 (GPR65). Notably, GPR65-deficient mice had attenuated airway eosinophilia and increased apoptosis in two distinct models of allergic airway disease (Kottyan et al. *Blood*. 2009;114(13):2774-82).

Allergic Reaction to Mecasermin

A recent case report by division co-director Amal Assa'ad, MD, clinical fellow Kelly Metz, MD, and colleagues details the second case of cutaneous and systemic allergic reaction to mecasermin, a recombinant human insulin-like growth factor 1 (IGF-1) approved by the Food and Drug Administration for treatment of growth failure in children with severe primary IGF-1 deficiency (Metz et al. *Annals of Allergy, Asthma & Immunology*. 2009;103(1):82-3.

C-C chemokine receptor type 3 Promising Target for Age-related Macular Degeneration

A recent collaborative research study by division director Marc Rothenberg, MD, PhD, and adjunct assistant professor Ariel Munitz, PhD, has shown promising results for age-related macular degeneration (AMD). Choroidal neovascularsation, the major cause of blindness from AMD, was more effectively reduced by blockade of C-C chemokine receptor type 3 than by blockade of vascular endothelial growth factor A blockade, which is in present clinical use. Additionally, blockade of C-C chemokine receptor type 3 was also less toxic to the retina (Takeda et al. *Nature*. 2009;460(7252);225-30).

T cell Subsets in Experimental Eosinophilic Esophagitis

A recent study by associate professor Anil Mishra, PhD, research associate Xiang Zhu, PhD, and colleagues has demonstrated an imbalance of esophageal effector and regulatory T cell subsets in a mouse model of eosinophilic esophagitis. Esophageal effector T cells increased whereas regulatory T cells decreased in allergen-challenged mice, suggesting that interaction of these T cell subsets may be required for protective and pathogenic immunity in eosinophilic esophagitis (Zhu et al. *American Journal of Physiology – Gastrointestinal and Liver Physiology*. 2009;297(3):G550-8).

Involvement of Mast Cells in Eosinophilic Esophagitis

Whereas prior studies have primarily focused on the role of eosinophils in disease diagnosis and pathogenesis of eosinophilic esophagitis, the involvement of mast cells was investigated in a recent study by division director Marc Rothenberg, MD, PhD, assistant professor J. Pablo Abonia, MD, and colleagues. The investigators identified local mastocytosis and mast cell degranulation in the esophagi of patients with eosinophilic esophagitis, defined an esophageal mast cell-associated transcriptome that is significantly divergent from the eosinophil-associated transcriptome, and provided evidence for the involvement of KIT ligand in the pathogenesis of eosinophilic esophagitis (Abonia et al. *Journal of Allergy and Clinical Immunology*. 2010;126(1):112-119).

Coordinate Interaction Between IL-13 and Epithelial Differentiation Cluster Genes in Eosinophilic Esophagitis

Aiming to uncover molecular explanations for eosinophilic esophagitis pathogenesis, a recent study by division director Marc Rothenberg, MD, PhD, instructor Carine Blanchard, PhD, and colleagues compared epithelial responses between healthy patients and those with eosinophilic esophagitis. Their findings establish that the epithelial response in eosinophilic esophagitis involves a cooperative interaction between IL-13 and expression of epithelial differentiation complex genes (Blanchard et al. *Journal of Immunology*. 2010;184(7):4033-41).

Glucocorticoid-regulated Genes in Eosinophilic Esophagitis

A recent study by division director Marc Rothenberg, MD, PhD, post-doctorate Julie Caldwell, PhD, and colleagues provides evidence that swallowed glucocorticoid treatment directly affects esophageal gene expression in patients with EE. In particular, increased esophageal FK506-binding protein 5 (FKBP51) transcript levels identify glucocorticoid exposure in vivo and distinguish patients with EE who responded to fluticasone propionate treatment from untreated patients with active EE and patients without EE, suggesting that esophageal FKBP51 levels may have diagnostic and prognostic significance in patients with EE (Caldwell et al. *Journal of Allergy and Clinical Immunology*. 2010;125(4):879-88 e8).

Polymorphisms in Sialic Acid-binding Immunoglobulin-like Lectin-8 Associated with Asthma Susceptibility

A recent collaborative research study by division director Marc Rothenberg, MD, PhD, and associate professor Nives Zimmermann, MD, has identified a significant association of polymorphisms in the sialic acid-binding immunoglobulin-like

lectin-8 gene with susceptibility to asthma in diverse populations (Gao et al. *European Journal of Human Genetics*. 2010;18(6):713-9).

Arginase I Suppresses Intestinal Inflammation During Acute Schistosomiasis

A recent collaborative research study by division director Marc Rothenberg, MD, PhD, and associate professor Nives Zimmermann, MD, has identified that macrophage-derived arginase I protects hosts from excessive tissue injury caused by worm eggs during acute schistosomiasis by suppressing interleukin 12 / interleukin 23 p40-driven intestinal inflammation (Herbert et al. *Journal of Immunology*. 2010;184(11):6438-46).

Cationic Amino Acid Transporter 2 Regulates Lung Fibrosis in Allergic Airway Inflammation

Using mouse models of allergic airway inflammation and pulmonary fibrosis, a recent study by associate professor Nives Zimmermann, MD, research assistant Kathryn Niese, and colleagues has identified cationic amino acid transporter 2 as a regulator of fibrotic response in the lung (Niese KA et al. *Respiratory Research*. 2010;11(1):87).

Differential Involvement of Interleukin 9/Interleukin 9 Receptor Pathway in Systemic and Oral Antigen-induced Anaphylaxis

Using mouse models of parenteral and oral antigen-induced anaphylaxis, a recent study by associate professor Simon Hogan, PhD, research assistant Heather Osterfeld, and colleagues has identified that parenteral antigen-induced systemic anaphylaxis is mediated by immunoglobulin G and immunoglobulin E -dependent pathways that can occur independently of interleukin 9 / interleukin 9 receptor signaling, whereas oral antigen-induced anaphylaxis is strictly immunoglobulin E-mediated and requires the interleukin 9 / interleukin 9 receptor signaling pathway (Osterfeld H et al. *Journal of Allergy and Clinical Immunology*. 2010;125(2):469-476.e2).

Persistent Rotavirus Vaccine Shedding in Severe Combined Immunodeficiency: A Reason to Screen

A recent case report by division director Marc Rothenberg, MD, PhD, clinical fellow Burcin Uygungil, MD, and colleagues details a case of persistent rotavirus vaccine shedding in a child with previously undiagnosed severe combined immunodeficiency and highlights the need for neonatal screening measures for severe combined immunodeficiency as this case is not an isolated incident (Uygungil et al. *Journal of Allergy and Clinical Immunology*. 2010;125(1):270-1).

Local B Cells and Immunoglobulin E Production in the Esophageal Mucosa in Eosinophilic Esophagitis

A recent study by division director Marc Rothenberg, MD, PhD, post doctorate Maria Vicario-Perez, PhD, and colleagues has demonstrated the heretofore unproven occurrence of both local immunoglobulin class switching to immunoglobulin E and immunoglobulin E production in the esophageal mucosa of patients with eosinophilic esophagitis. Sensitization and activation of mast cells involving local immunoglobulin E may therefore critically contribute to disease pathogenesis (Vicario et al. *Gut*. 2010;59(1):12-20).

Division Collaboration

Collaboration with Gastroenterology; Pathology; Nutrition; Social Work

Collaborating Faculty: Drs. Mitchell Cohen, Philip Putnam and James Franciosi; Dr. Margaret Collins
Cincinnati Children's Center for Eosinophilic Disorders (CCED)

Faculty Members

Marc E. Rothenberg, MD, PhD, Professor ; *Division Director*
Research Interests: Elucidating the mechanisms of allergic responses especially in mucosal tissues such as the lung and the gastrointestinal tract

J. Pablo Abonia, MD, Research Assistant Professor
Research Interests: The role of mast cells in eosinophilic esophagitis

Amal H. Assa'ad, MD, Professor Clinical ; *Clinical Director*
Research Interests: The occult effect of allergic sensitization to foods on the bronchial hyper-responsiveness seen in asthmatic and the genetic basis of food allergy

Carine Blanchard, PhD, Research Instructor
Research Interests: To study food allergy; eosinophilic esophagitis; asthma

Thomas J. Fischer, MD, Adjunct Professor Clinical
Research Interests: The pharmacologic management of asthma, immune deficiency diseases

Simon P. Hogan, PhD, Assistant Professor
Research Interests: To study allergies, food allergies, eosinophil biology & gastrointestinal inflammation

Michelle B. Lierl, MD, Adjunct Associate Professor
Research Interests: To reduce environmental tobacco smoke exposure in children with asthma

Anil Mishra, PhD, Research Assistant Professor
Research Interests: Understanding the mechanism of aeroallergen-induced allergic responses in the lung and lower gastrointestinal tract

Kimberly A. Risma, MD, PhD, Research Assistant Professor
Research Interests: The molecular and cellular bases of primary disorders of immune deficiency and dysregulation, especially as it relates to lymphocyte cytotoxicity

Yui-Hsi Wang, PhD, Research Assistant Professor

Nives Zimmermann, MD, Research Associate Professor
Research Interests: The molecular understanding of eosinophil survival in allergic inflammation and asthma

Li Zuo, MD, Instructor Clinical
Research Interests: To understand the molecular pathogenesis involved in food allergy related disorders.

Joint Appointment Faculty Members

Gurjit Khurana Hershey, MD, Professor
Asthma Research

Alexandra Filipovich, MD, Professor
Hematology/Oncology Diagnostic Laboratory
Primary immunodeficiencies; BMT for primary immunodeficiencies; Hemophagocytic lymphocytosis; Post-BMT immune reconstruction

Clinical Staff Members

- **Kalra Harpinder, MD**

Trainees

- **Gerald Lee, MD,** PL-7, Saint Vincent's Catholic Medical Centers, New York
- **Kelly Metz, MD,** PL-6, University of Cincinnati, Ohio
- **Charles DeBrosse, MD,** PL-5, Ohio State University, Ohio
- **Muthuvel Arumugam, PhD,** University of Madras, Chennai, India
- **Julie Caldwell, PhD,** University of Cincinnati, Ohio
- **Chen Chun-Yu, PhD,** University of Rochester, New York
- **Carlos Fernandez Gimenez, MD,** Clinical Hospital of Salamanca, Spain
- **Eun Jin Lim , PhD,** University of Kentucky, Kentucky
- **Ariel Munitz, PhD,** Hebrew University of Jerusalem, Israel
- **Joseph Sherrill, PhD,** University of Cincinnati, Ohio
- **Zeenath Unnisa, PhD,** Osmania University, Hyderabad, India
- **Ramon Urrea-Moreno, PhD,** Hospital Gregorio Maranon, Madrid, Spain
- **Ting Wen, PhD,** Rutgers University/UMDNJ, New Jersey
- **Katherine Groschwitz, ,** Xavier University, Ohio
- **Hongyan Zhu, ,** Hubei College of Traditional Medicine, China
- **Amanda Beichler, ,** Ohio Northern University, Ohio
- **Tom Lu, ,** University of Cincinnati, Ohio
- **Leah Kottyan, ,** Huntingdon College, Alabama
- **, ,**

Significant Accomplishments

Gene region identified for severe food allergy

Director Marc Rothenberg, MD, PhD, and colleagues identified the first major gene location (5q22.1) responsible for susceptibility to eosinophilic esophagitis (EE), a severe food allergy that leaves individuals unable to eat a wide variety of foods. Using a genome-wide gene variation analysis, they identified overexpression of the TSLP gene at this chromosomal location. These data implicate the 5q22 locus in the pathogenesis of EE and identify TSLP as the most likely candidate gene.

National registry to track eosinophilic disorders

A $1.6 million federal economic stimulus grant from the National Institute of Diabetes and Digestive and Kidney Diseases helped us launch the Registry for Eosinophilic Gastrointestinal Disorders (REGID). It is the first registry to provide the infrastructure for understanding and treating eosinophilic disorders at a national – and ultimately international - level. Rothenberg developed REGID along with division faculty member Pablo Abonia, MD, and co-investigators James Franciosi, MD, and Keith Marsolo, PhD.

Interleukin 15 involved in eosinophilic esophagitis

Anil Mishra, PhD, and colleagues identified the significance of induced expression and protein levels of interleukin 15 (IL-15) in human and experimental EE. The researchers found that transcript levels of IL-15 strongly correlated with esophageal eosinophils in patients with active EE. Likewise, the levels significantly decreased in patients with improved, treated EE. Using mouse models, researchers showed the receptor for IL-15, IL-15Rα, is necessary for the development of EE. Elevated IL-15 levels in the blood samples of EE patients could potentially serve as a diagnostic biomarker.

Division Publications

1. :

Grants, Contracts, and Industry Agreements

Grant and Contract Awards — Annual Direct / Project Period Direct

Beichler, A

Graduate Student Award 2009
AGA Foundation for Digestive Health & Nutrition
07/01/09 - 06/30/11 — $20,000 / $40,000

Blanchard, C

Urplakin 1b: A Genetic Risk Marker for Eosinophilic Esophagitis
American Partnership for Eosinophilic Disorder
07/01/09 - 06/30/10 — $25,000 / $25,000

Caldwell, J

Molecular Mechanisms of Glucocorticoids and FKBP51 in Inflammation
American Heart Association
09POST2180041 — 07/01/09 - 06/30/11 — $43,000 / $88,000

DeBrosse, C

2009 Fellows Career Development Award
American Academy of Allergy, Asthma & Immunology
07/01/09 - 06/30/10 — $50,000 / $50,000

Groschwitz, K

Mast Cell-Mediated Intestinal Permeability
National Institutes of Health
F30 DK 082113 — 07/01/08 - 06/30/11 — $32,588 / $95,625

Hogan, S

Interleukin-9 in Experimental Intestinal Anaphylaxis
National Institutes of Health
R01 AI 073553 — 04/01/08 - 03/31/12 — $247,500 / $995,000

Intestinal IL-9 and Mast Cells in Food-Induced Anaphylaxis
Food Allergy and Anaphylaxis Network
02/02/09 - 01/31/11 — $124,773 / $249,546

Kucuk, Z

Immunology/Allergy Fellowship Training Grant
University of Cincinnati (National Institutes of Health)
T32 AI 060515 — 07/01/09 - 06/30/11 — $45,048 / $45,048

Mishra, A

Role of IL-15 in the Pathogenesis of Eosinophilic Esophagitis
National Institutes of Health
R01 AI 080581 — 07/22/09 - 06/30/11 — $250,000 / $500,000

Mechanistic Analysis of Eosinophilic Esophagitis
National Institutes of Health
R01 DK 067255 — 07/17/09 - 06/30/11 — $20,000 / $20,000

Moncrief, T

ACAAI Clinical Fellowship
American College of Allergy, Asthma & Immunology
07/01/09 - 09/30/10 $50,000 / $50,000

Munitz, A

Regulation of Eosinophils by PIR-B
American Heart Association - Ohio Affiliate
0825621D 07/01/08 - 06/30/10 $45,000 / $88,000

The Expression and Function of ILT-3/LIR-5 in Pediatric Eosinophilic Esophagitis
Thrasher Research Fund
06/01/09 - 05/31/11 $14,000 / $25,000

Risma, K

The Functional Consequences of Incomplete Perforin Processing
American Academy of Allergy, Asthma & Immunology
07/01/09 - 06/30/12 $100,000 / $300,000

Proteolytic Maturation of Perforin: Determining the Requirements for Cytotoxic Function in Patients with Hemophaglcytic Lymphohistiocytosis
Histiocytosis Association of America
01/01/10 - 12/31/10 $50,000 / $50,000

Rothenberg, M

IL-13 and Eosinophilic Esophagitis
National Institutes of Health
R01 DK 076893 09/01/07 - 06/30/12 $200.900 / $1,020,900

IL-13 and Eosinophilic Esophagitis
National Institutes of Health
R01 DK 076893 09/30/09 - 08/31/11 $1,042,081 / $1,042,081

Novel Genetic and Therapeutic Approaches Focusing on Siglec-8 for the Diagnosis and Treatment of Human Idiopathic Eosinophilic Disorders
The Dana Foundation
10/01/07 - 09/30/10 $100,000 / $300,000

The Role of CREB Binding Protein in Eosinophilic Esophagitis
Food Allergy and Anaphylaxis Network
02/02/09 - 01/31/11 $62,386 / $124,772

IL-13 Associated Eosinophil Lung Responses
National Institutes of Health
R01 AI 083450 08/20/09 - 07/31/14 $250,000 / $1,247,500

Regulations of Gastrointestinal Eosinophils
National Institutes of Health
R01 AI 045898 12/01/09 - 11/30/14 $225,000 / $1,125,000

Genetic Studies of Food Allergies Research Program
Department of Defense
W81XWH1010167 03/01/10 - 02/28/12 $246,436 / $492,872

A Multi-Center Clinical Trial of 1760 mcg of Daily Swallowed Fluticasone vs. Placebo
National Institutes of Health
U01 AI 088806 09/26/09 - 08/31/11 $409,241 / $612,185

A Multi-Center Clinical Trial of 1760 mcg of Daily Swallowed Fluticasone vs. Placebo
National Institutes of Health
U01 AI 088806 06/28/10 - 05/31/11 $90,000 / $90,000

Epithelial Genes in Allergic Inflammation - Project #2
National Institutes of Health
U19 AI 070235 09/15/06 - 07/31/11 $182,955 / $874,228

Epithelial Genes in Allergic Inflammation - Project #2
National Institutes of Health

U19 AI 070235 08/13/09 - 07/31/11 $100,000 / $100,000

Strauss, A / Rothenberg, M

Pediatric Center for Gene Expression and Development
National Institutes of Health
K12 HD 028827 04/01/07 - 11/30/11 $400,000 / $2,000,000

Wang,Y

CTSA KL2 Scholar Award
University of Cincinnati (National Institutes of Health)
KL2 RR 026315 07/01/09 - 03/31/10 $83,928 / $83,928

Regulation of TH2 Memory/Effector Cells During Allergic Inflammation
National Institutes of Health
R01 AI 090129 05/01/10 - 04/30/15 $250,000 / $1,250,000

Zimmermann, N

Role of Acidic Environment in Eosinophilic Inflammation
National Institutes of Health
R21 AI 079251 02/15/09 - 01/31/11 $148,500 / $273,500

Assessing Eosinophil Survival in Acidic Environments Modeling the Asthmatic Lung
University of Cincinnati
07/01/09 - 06/30/10 $3,000 / $3,000

Role of Acidity and GPR65 in Food Allergy
National Institutes of Health
R21 AI 088559 03/03/10 - 02/29/12 $125,000 / $275,000

Current Year Direct **$5,036,336**

Industry Contracts

Abonia, P

Ception Therapeutics, Inc. $ 211,579

Rothenberg, M

Novartis Pharmaceuticals $ 27,328

Merck & Co, Inc. $ 19,250

Current Year Direct Receipts **$ 258,157**

Total $5,294,493