

*Division of Allergy and  
Immunology*

*Department of Internal Medicine*

*Joy McCann Culverhouse Airway  
Disease Research Center*

*Morsani College of Medicine*

*University of South Florida*

*James A. Haley Veterans' Hospital*

*Tampa, Florida*



2011  
*Annual Report*

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# *I. Introduction*

*University of South Florida  
Morsani College of Medicine,  
Department of Internal Medicine,  
Division of Allergy and Immunology*

The late Samuel C. Bukantz, M.D., founded the University of South Florida Morsani College of Medicine, Department of Internal Medicine, Division of Allergy and Immunology in 1972. Richard F. Lockey, M.D. succeeded Dr. Bukantz in 1983 and is the current Director of the Division. Mrs. Joy McCann Culverhouse endowed the Division in 1997 and The Joy McCann Culverhouse Airway Disease Research Center was dedicated in February 1998. In 1998, Mabel and Ellsworth Simmons endowed the Division with a grant for education and research. The goals of the Division are: first, to provide care to patients with allergic and immunologic diseases at the University of South Florida College of Medicine, Tampa General Hospital, James A. Haley V.A. Medical Center, All Children's Hospital, and H. Lee Moffitt Cancer Center; second, to train students, residents, and fellows in the subspecialty of allergy and immunology; and third, to conduct basic and clinical research in allergy, asthma, and immunology. Individuals interested in collaborating with members of the Medicine Division may contact Richard F. Lockey, M.D. or any faculty member at (813) 972- 7631 (e-mail: rlockey@health.usf.edu). John W. Sleasman, M.D. or any other faculty member in the Pediatric Division may be contacted at 727-553-3533 or jsleasma@health.usf.edu.



## II. FACULTY AND STAFF

### Core Faculty

**Richard F. Lockey, M.D.**, Professor of Medicine, Pediatrics, and Public Health; University Distinguished Health Professor; Joy McCann Culverhouse Chair of Allergy and Immunology, Division Director

**Roger W. Fox, M.D.**, Professor of Medicine, Pediatrics and Public Health

**Dennis K. Ledford, M.D.**, Professor of Medicine and Pediatrics

**Mark C. Glaum, M.D., Ph.D.**, Associate Professor of Medicine and Pediatrics

**Michael N. Teng, Ph.D.**, Associate Professor of Medicine

**Narasaiah Kolliputi, Ph.D.**, Assistant Professor of Medicine

**Jia-Wang Wang, Ph.D.**, Assistant Professor of Medicine

**Glenn Whelan, Pharm.D.**, Assistant Professor of Medicine

### Joint Faculty

**John W. Sleasman, M.D.**, Professor of Pediatrics and Medicine; Robert A. Good Professor of Immunology; Chief, Division of Allergy and Immunology, Department of Pediatrics, University of South Florida, All Children's Hospital

**Gary W. Litman, Ph.D.**, University Distinguished Health Professor, Andrew and Ann Hines Chair in Pediatrics, Professor of Pediatrics and Medicine

**Noorbibi Day, Ph.D.**, Professor of Pediatrics and Medicine Professor of Medicine

**Sandra Gompf, M.D.**, Associate Professor of Medicine

**Michel J. Seleznick, M.D.**, Associate Professor Medicine

**Morna Dorsey, M.D., M.M.Sc.**, Associate Professor of Pediatrics and Training Program Director, Pediatric Division

**Soichi Haraguchi, Ph.D.**, Assistant Professor Pediatrics

**Elena E. Perez, M.D., Ph.D.**, Associate Professor of Pediatrics

**Panida Sriaroon, M.D.**, Assistant Professor Pediatrics

**Mandel R. Sher, M.D.**, Clinical Professor of Pediatrics

### Clinical Faculty

**Robert E. Windom, M.D.**, Clinical Professor Medicine

**Enrique Fernandez-Caldas, Ph.D.**, Clinical Professor Medicine

**Nathan Tang, M.D.**, Clinical Professor of Pediatrics

**Monroe J. King, D.O.**, Adjunct Clinical Associate Professor of Medicine and Pediatrics

**Brett E. Stanaland, M.D.**, Clinical Associate Professor of Medicine

**G.Edward Stewart II, M.D.**, Clinical Associate Professor of Medicine

**Hugh Windom, M.D.**, Clinical Associate Professor Medicine

**Michel Alkhalil, M.D.**, Clinical Assistant Professor of Medicine

**Rosa Codina, Ph.D.**, Clinical Assistant Professor of Medicine

**Mary L. Jelks, M.D.**, Clinical Assistant Professor Medicine

**Dennis Kim, M. D.**, Clinical Assistant Professor of Medicine

## **Research Staff Members**

**Lakshmi Galam**, Ph.D., Instructor  
**Mahesh Kumar Abbengula**, M.S., M.D., Research Fellow  
**Jutaro Fukumoto**, M.D., Ph.D., Postdoctoral Fellow  
**Sam Jalali**, Research Assistant  
**Kunyu Li**, Technical and Para Professional  
**Prasanna T. Parthasarathy**, M.S., Research Technician  
**Gurukumar K. Ramanathan**, Ph.D., Postdoctoral Fellow  
**Hassan Syed**, M.D.,  
**Kim Teng, M.S.**, Research Scientist  
**Rajan Babu Venugopal**, Ph.D., Postdoctoral Fellow

## **2011-2012 Fellows-in-Training**

### **2<sup>nd</sup> Year Fellows**

**David Fitzhugh**, M.D., Chief Fellow  
**Ahmed Butt**, M.D.  
**Jim Parkerson**, D.O.

### **1<sup>st</sup> Year Fellows**

**Salman Aljubran**, M.D.  
**Susan Culverhouse**, M.D.,  
**Neetu Talreja**, M.D.

## **Students and Visiting Research Scholars**

**Beth Coughlin**, Research Assistant  
**Ruan Cox**, Graduate Student  
**Sara Garcia**, Research Student  
**Matthew Ho**, Research Student  
**Amanda Hodgkins**, Research Assistant/Student  
**Bao Huynh**, Research Assistant/Student  
**Michelle Kaminsky**, Research Assistant/Student  
**Anu Stephen**, Research Student  
**Tran Tran**, Graduate Student  
**Jillian Whelan**, Graduate Student

## **Administrative Personnel**

### **Assistant to the Division Director**

**Michelle Grandstaff-Singleton**, B.H.Sc., LPN, Clinical Research Administrator and Administrative Assistant to the Division Director

### **Division of Allergy and Immunology and The James A. Haley V.A. Medical Center**

**Peggy Hales**, Program Assistant  
**Rebecca Carter**, Administrative Secretary  
**Geeta Gehi**, Administrative Secretary

### **Clinical Research Unit**

**Michelle Grandstaff-Singleton**, BHSc, L.P.N., Clinical Research Administrator  
**Diana Miller**, B.A., Clinical Research Coordinator  
**Rebecca McCrery**, Administrative Secretary

### **All Children's Hospital**

**Amy Baldwin**, Administrative Assistant to Dr. Sleasman  
**Marjorie Peak**, Allergy and Immunology Fellowship Coordinator (Pediatrics)



### III. Joy McCann Culverhouse Airway Disease Research Center

#### A. Basic Research Projects

##### 1. Akt as a therapeutic target for respiratory syncytial virus

(R01 AI081977)

We currently are exploring two aspects of Akt activity in RSV infection. First, we have identified RSV P as a target for Akt phosphorylation and are determining the role of this phosphorylation on viral replication. Second, we are in the process of determining the viral factors responsible for Akt activation during RSV infection. We anticipate that these two lines of research will result in publications within the next 6 – 12 months. These studies are being done in collaboration with Dr. Biao He (UGA).

##### 2. Structural determinants of NS2 for pathogenic functions

(R01 submitted)

We have previously published that NS2 blocks interferon induction by binding to RIG-I. In addition NS2 appears to have additional functions associated with viral pathogenesis, including NFkB induction and STAT2 degradation. We are trying to separate these activities by mutagenesis to understand how NS2 accomplishes each function. Our focus is on differentially altering the functions to develop an attenuated RSV vaccine candidate that maintains its immunogenicity.

##### 3. Mechanism of RSV temperature sensitivity due to cis-acting sequences

(R21 submitted)

Previous studies have found that a single nucleotide change in the M2 transcription start sequence is sufficient to confer temperature sensitivity to recombinant RSV. We are determining the mechanism by which this mutation affects RSV replication and transcription at non-permissive temperatures.

##### 4. Anti-G responses in immunity to RSV

(P01 to be re-submitted)

This is a long-term collaboration between us and Dr. Ralph Tripp (University of Georgia). Dr. Tripp has found that antibodies against the central conserved domain of RSV G can ameliorate vaccine-enhanced disease. We are currently testing the hypothesis that directing antibody responses against this domain can decrease the severity of RSV disease upon subsequent infection.

## 5. RSV M protein trafficking and virus assembly

This is a long-term collaboration between us and Drs. David Jans and Reena Ghildyal (Monash University, Melbourne, AUS). We are determining the role of M protein trafficking in RSV morphogenesis and the importance of nuclear translocation in M function.

## 6. NALP-3 inflammasome silencing attenuates ceramide induced transepithelial permeability

The hallmark of acute lung injury (ALI) is the influx of proinflammatory cytokines into lung tissue and alveolar permeability that ultimately leads to pulmonary edema. However, the mechanisms involved in inflammatory cytokine production and alveolar permeability are unclear. Recent studies suggest that excessive production of ceramide has clinical relevance as a mediator of pulmonary edema and ALI. Our earlier studies indicate that the activation of inflammasome promotes the processing and secretion of proinflammatory cytokines and causes alveolar permeability in ALI. However, the role of ceramide in inflammasome activation and the underlying mechanism in relation to alveolar permeability is not known. We hypothesized that ceramide activates the inflammasome and causes inflammatory cytokine production and alveolar epithelial permeability. To test this hypothesis, we analyzed the lung ceramide levels during hyperoxic acute lung injury in mice. The effect of ceramide on activation of inflammasome and production of inflammatory cytokine was assessed in primary mouse alveolar macrophages and THP-1 cells. Alveolar transepithelial permeability was determined in alveolar epithelial type-II cells (AT-II) and THP-1 co-cultures. Our results reveal that ceramide causes inflammasome activation, induction of caspase-1, IL-1 $\beta$  cleavage and release of proinflammatory cytokines. In addition, ceramide further induces alveolar epithelial permeability. Short hairpin RNA silencing of inflammasome components abrogated ceramide-induced secretion of proinflammatory cytokines in vitro. Inflammasome silencing abolishes ceramide-induced alveolar epithelial permeability in AT-II. Collectively, our results demonstrate for the first time that ceramide-induced secretion of proinflammatory cytokines and alveolar epithelial permeability occurs through inflammasome activation. (*J. Cell Physiol.* 2011 Dec 14. doi: [10.1002/jcp.24026](https://doi.org/10.1002/jcp.24026). [Epub ahead of print])



## 7. **Inflammasome inhibition suppresses alveolar cell permeability through retention of NRG-1**

The inflammasome is a newly discovered molecular platform required for the caspase-1 activation and maturation of IL-1 $\beta$ . IL-1 $\beta$  is the most active cytokine in acute lung injury patients. Alveolar cell permeability is induced through neuregulin (NRG)-1 and human epidermal receptor (HER)-2 signaling. However, the signaling mechanism in alveolar cell permeability is unknown. Our previous results suggest that inflammasome mediates hyperoxia-induced alveolar cell permeability. It was hypothesized that inflammasome activates alveolar cell permeability through NRG-1/HER-2 signaling. To test this hypothesis, inflammasome was activated or inhibited in THP-1 cells and supernatants from these cells were added to A549 cells, human primary small airway epithelial cells (HPSAEC) or human pulmonary artery endothelial cells (HPAEC). The protein expression of NRG-1 and phospho-HER-2 were measured by western blot analysis. Epithelial permeability using was measured using Lucifer yellow dye. Alveolar permeability in A549 and HPSAEC was increased when treated with supernatants of inflammasome activated THP1 cells. Alveolar permeability is significantly suppressed when treated with supernatant of inflammasome inhibited THP1 cells. Inflammasome mediated permeability is decreased when A549 and HPSAEC are pretreated with IL-1 $\beta$  receptor antagonist (RA). In addition HER-2 kinase inhibitor AG825 or NRG-1 inhibitor TAPI also inhibits inflammasome mediated permeability in A549 and HPSAECs demonstrating a critical role IL-1 $\beta$ , NRG1 and HER-2 in inflammasome-mediated alveolar permeability. These findings suggest that inflammasome induced alveolar cell permeability is mediated by NRG-1/HER-2 through IL-1 $\beta$ . **(Submitted to The Journal of Immunology)**

## 8. **Resolvin inhibits the Cryopyrin/Nalp3 inflammasome**

The inflammasome is a novel protein complex that stimulates caspase-1 activation to promote the processing and secretion of IL-1 $\beta$ , a pro-inflammatory cytokine, which is among the most biologically important inflammatory mediators in the airspace of patients with early acute lung injury (ALI). Among the various types of inflammasomes, Cryopyrin/NALP3 has been suggested to be involved in sensing ROS stress response, extracellular ATP, danger-associated molecular patterns (DAMPs), and crystals. Recently inappropriate NALP3 inflammasome activity in ALI, various acute and autoimmune diseases has been reported. Therefore, inhibitors of the NALP3 inflammasome offer considerable therapeutic promise. Recently Omega-3 fatty acid derivatives termed resolvins have shown to alter the effects of pro-inflammatory cytokine storm seen in sepsis mediated acute injury. However the ability of resolvins to modulate the effects of inflammasome activation has not been studied. We investigated whether resolvin treatment inhibits inflammasome activation

and regulates the functional effects of inflammasome mediated by IL-1 $\beta$  secretion. In our study the inflammasome was activated by ATP and H<sub>2</sub>O<sub>2</sub> (known inflammasome activators in acute lung injury) in the absence or presence of D series resolvins, resolvin D1 and resolvin D2. Inflammasome activation was assessed by analyzing IL-1 $\beta$  release (end product of inflammasome activation) and caspase-1 cleavage (indicator of inflammasome activation) in THP-1 cells. Further inflammasome was activated in the presence or absence of resolvin in THP-1 cells, and supernatants from these cells were added to A549 cells and human primary small airway epithelial cells (HPSAEC) to study inflammasome mediated functional effects. Our results indicate that resolvin treatment ameliorates inflammasome activation as indicated by decreased caspase-1 activity and IL-1 $\beta$  release. In addition resolvin treatment inhibits inflammasome mediated epithelial cell activation as indicated by suppressed IL-8 release and decreased ICAM expression. These novel findings suggest that resolvins can be used to modulate the inflammasome activity as well as blunt the effects of the IL-1 $\beta$  mediated cytokine storm stemming from inflammasome activation. These results may offer a therapeutic approach to diseases such as acute lung injury, where uncontrolled pro-inflammatory cytokine secretion exacerbates the disease pathology.

#### 9. Role of Forkhead Transcription Factor FOXO3a in acute lung injury

Acute lung injury (ALI) is a devastating clinical problem involving the key events of inflammation and alveolar epithelial cell death. The fundamental mechanism of this serious condition evolves from an imbalance between pro-inflammatory and anti-inflammatory cytokines finally resulting in oxidative stress induced cell death. FOXO transcription factors are important regulators of cell survival in response to a variety of stress stimuli, among which are oxidative stress, DNA damage, and nutrient deprivation. However, the role of FOXO3a in acute lung injury and epithelial cell death is not known. It was hypothesized that FOXO3a protects alveolar epithelial cells from H<sub>2</sub>O<sub>2</sub> induced cell death. To test this hypothesis, the effects of H<sub>2</sub>O<sub>2</sub> on subconfluent human alveolar epithelial cell (A549) cultures were tested. A549 cells were transfected with vector containing wild type FOXO3a cDNA (FOXO3a-WT) or vector containing mutant FOXO3a cDNA (FOXO3a-M) and treated with or without H<sub>2</sub>O<sub>2</sub> (1hour) to induce oxidative stress. The results showed that H<sub>2</sub>O<sub>2</sub> induced a significant alveolar epithelial cell necrosis and apoptosis. In contrast epithelial cells transfected with FOXO3a-WT inhibited H<sub>2</sub>O<sub>2</sub> induced cell necrosis and apoptosis. However cells transfected with a mutant, FOXO3a-M did not alter H<sub>2</sub>O<sub>2</sub> mediated epithelial cell apoptosis and necrosis. Together these results suggest that FOXO3a protects alveolar epithelial cells from oxidative stress induced cell death. (*FASEB J* March 17, 2011 25:)

#### 10. **MicroRNAs as biomarkers and therapeutics for asthma**

MicroRNAs (miRs) are ~22 nucleotides long non-coding RNAs that inhibit mRNA translation by the base pairing rule at the accuracy of one base. So far 2154 human mature miRs are discovered. As each miR may regulate hundreds of genes, and each gene may be regulated by multiple miRs, it is believed that most human genes and the entire spectrum of biological pathways are tightly and delicately controlled by the miRNome. Deregulation of miRs may contribute to various diseases. We have been working on inflammation regulators responsive to lipopolysaccharides (LPS) stimulation (Science. 2002 15;295(5562):2094-7, J Immunol. 2001 Apr 1;166(7):4586-95, Oncogene. 2004 May 20;23(23):4089-97). We found that SH2-containing inositol phosphatase (SHIP) plays an important role in two processes that limit the success of allogeneic marrow transplantation: graft rejection and graft-versus-host disease using Cre/loxP conditional knockout mouse models, and discovered a novel gene: LPS-responsive beige-like anchor (Lrba) that may be involved in leading intracellular vesicles to activated receptor complexes. Currently, we are interested in studying the mechanism underlying miR regulations of the immunity and developing miR biomarkers and therapeutics for inflammatory diseases such as asthma using cell culture and mouse models.

## **B. Clinical Research Projects Conducted at the Division's Clinical Research Unit**

### **1. Repeated nasal challenge in skin prick-puncture negative, intradermal positive dust mite allergic rhinitis patients**

Skin prick-puncture testing is a specific test to determine whether or not an individual is allergic. The primary goal of this study is to evaluate the clinical usefulness of intradermal skin testing when prick-puncture tests are negative. Intradermal skin testing is more sensitive but less specific than prick-puncture testing. There is little evidence-based data to support the clinical relevance of a negative prick-puncture test with a positive intradermal test result. The hypothesis of this study is that subjects, who have a clinical history of perennial rhinitis symptoms associated with dust exposure or not associated with other perennial allergens, will have a positive challenge with *Dermatophagoides pteronyssinus* when they have a positive intradermal test and a negative prick-puncture test. Subjects who are prick-puncture negative and intradermal skin test positive to *Dermatophagoides pteronyssinus* will be challenged with nasal sprays containing either placebo or *Dermatophagoides pteronyssinus* extract solution seven separate days under observation in the research office (no more than 3 days between visits). This will be followed by a one month washout period, then patients will crossover and return to the search office for the administration of the other nasal spray under observation, again on seven separate days. Both subjects and investigators will be blinded to the spray being used. Recruitment for this project is ongoing at this time.

### **2. Effects of Pine Cone Extract (PCE) on IgE levels in patients with allergic rhinitis**

Pine cones and their aqueous extracts (PCE) have been known to have medicinal properties in Japanese populations as far back as 2000 years ago. Anecdotal reports have suggested that use of PCE improves allergic rhinitis symptoms and in the mouse model has been shown to significantly reduce serum IgE levels. The purpose of this study will be to determine if oral PCE extract administered in a double blind fashion will significantly reduce IgE levels in patients with evidence of perennial allergies. We are currently enrolling subjects at this time.

**3. Pollen and mold counts and immunochemical quantification of outdoor allergens**

Particles, other than pollen, which transport aeroallergens, have been described. The Division, which houses the Pollen and Mold Counting Station for Tampa, has two collectors adapted to collect both pollen and pollen aeroallergens. The collectors are located on the roof of the James A. Haley V.A. Medical Center Research Building. Pollen counts are performed twice weekly, disseminated to local media once weekly and to the Internet twice weekly. Dr. Mary Jelks reads and interprets the slides.

**4. Prevalence of food allergy in adult patients with eosinophilic esophagitis**

Food allergies are known to play a significant role in children with eosinophilic esophagitis. Little is known about the prevalence of food allergies in adult patients with eosinophilic esophagitis. The purpose of this study is to determine the prevalence of food allergies in a cohort of adult patients with eosinophilic esophagitis. These results will be compared to findings in adult patients with gastroesophageal reflux disease. We are currently enrolling subjects for this study.

**5. Effect of oxymetazoline hydrochloride in combination with nasal glucocorticoid on the Apnea Hypopnea Index (AHI)**

“Nocturnal Oxyhemoglobin Saturation, Snoring, and Sleep Quality in Subjects with Persistent Nasal Congestion. A Double Blinded, Placebo Control, Cross Over Prospective Trial.” To evaluate the effectiveness of the addition of oxymetazoline to intranasal ICS (Mometasone) on the apnea /hypopnea index (AHI), and on other sleep parameters in subjects with persistent nasal congestion secondary to allergic or non-allergic rhinitis despite treatment with the highest recommended doses of intranasal ICS, Mometasone. This project is approved by the USF Institutional Review Board and is in the start up phase.

**6. A study of function of respiration and cognition in the elderly (The FORCE study)**

In this pilot study, our first aim is to determine which diagnostic tool (IOS or spirometry) is more feasible for the elderly population. We hypothesize that older adults will be significantly more likely to successfully complete the IOS diagnostic tool. Second, we want to determine if cognitive performance is related to the ability to perform spirometry and IOS. We hypothesize that lower cognitive functioning will decrease the likelihood of performing spirometry correctly. Lastly, we will explore the relationship between biomarkers, cognitive performance, and asthma diagnosis. We predict that higher levels of pro-inflammatory biomarkers will be related to lower levels of cognitive functioning and higher incidences of asthma. We are currently recruiting subjects for this study.



**7. Evaluation of calcium and vitamin D intake in children on inhaled or intranasal corticosteroids compared to normal children**

The specific aims of this project are to evaluate the dietary calcium intake of asthmatic children (4-17 years) who are receiving long-term treatment with inhaled or intranasal corticosteroids and compare it with healthy controls. 100 asthmatic/ chronic rhinitis children based on inclusion and exclusion criteria will be selected. A validated food frequency questionnaire will be used to assess their calcium intake. This questionnaire will be provided to them on their first clinic visit. 100 control healthy subjects with similar age distribution will be selected. The same questionnaire will be provided to them for review of their calcium intake. Written informed consent will be obtained from both the groups. These results of the survey will be compared and assessment will be made if the calcium intake of asthmatic/ chronic rhinitis children (on inhaled corticosteroid or intranasal corticosteroids) is adequate and if their consumption of calcium is more or less than the control population. Institutional Review Board approval is pending for this study.

**8. A comparison of microRNA samples in patients with nasal polyps and aspirin exacerbated respiratory disease and those with nasal polyps only.**

The prevalence of aspirin exacerbated respiratory disease may be as high as 22% in those with nasal polyps. A leading hypothesis for the cause of AERD is the Inhibition of Cox-1 by NSAID decreases the production of PGE2. The increased production of the cysteinyl leukotriene LTC4 is proposed as the primary cause of bronchospasm in AERD. LTC4 synthase has several allelic variations and Type C may be more prevalent in AERD5 suggesting there are genetic differences between those with AERD and polyps and those with polyps only. An additional explanation for the variations in gene expression in AERD could be differences in regulatory miRNA. During this study, nasal epithelial tissue will be collected from the Inferior nasal turbinates of subjects with nasal polyps and AERD disease and from the inferior turbinates of subjects with nasal polyps but without AERD. These samples will be analyzed by miRNA assay to determine if there is a difference in microRNA expression between the two subject groups. We are currently recruiting subjects for this study.



**9. Identification of plasma miRNAs as potential biomarkers in asthma exacerbation**

We hypothesize that there is a statistically significant difference in miRNA profiling and expression of subjects with asthma upon its exacerbation compared to patient's baseline level or following effective treatment of an exacerbation of asthma. Therefore, plasma miRNA profiling may provide noninvasive, highly specific and sensitive biomarkers for asthma exacerbation's detection and treatment follow-up. The primary objective of this study is to evaluate to effect of asthma exacerbation on miRNA profiling and expression. This study is in the start up phase.

**10. Differences in mold counts from January 1995 to December 2011**

Allergic diseases are complex interaction between genetic and environmental factors. Airborne mold and pollens are known to trigger allergic respiratory disease in sensitive individuals; yet little is known about the change in pollen and mold counts over the last 16 years in relation to global warming. The Division, which houses the pollen and mold counting station for Tampa and Sarasota, has two collectors adapted to collect pollen and mold aeroallergens. Pollen and fungal spores are performed daily and the data, between January 1995 to December 2010, in Sarasota is available to us. Weather data for Sarasota has been obtained from the National Climatic Data Center. The objective is to see if these pollen/mold counts can be correlated with meteorological parameters to track or predict allergic disease in the future.

## C. Clinical Research Unit

The University of South Florida, Asthma, Allergy and Immunology Clinical Research Unit was established in 1977 to improve the treatment of patients who suffer from asthma, allergic and immunologic diseases. The Clinical Research Unit is a segment of the Division of Allergy and Immunology, Department of Internal Medicine at the University of South Florida College of Medicine. The Division is affiliated with the H. Lee Moffitt Cancer Center, James A. Haley Veterans Administration Hospital and the University of South Florida Medical Clinics in Tampa. The Unit is also affiliated with All Children's Hospital and Bay Pines Veterans Hospital, both in St. Petersburg.

The Unit provides quality research in a variety of clinical areas which include the following: allergic conjunctivitis; allergen immunotherapy; allergen skin testing; allergic rhinitis; asthma; atopic eczema; bronchitis, acute and chronic; contact dermatitis; chronic obstructive pulmonary disease; exercise induced asthma; headache (migraine and tension); hereditary angioedema; immunodeficiency diseases; insect allergy; intravenous immunoglobulin; nasal polyps; sinusitis, acute and chronic; temporomandibular joint disease; urticaria and vasomotor rhinitis.

Studies funded by pharmaceutical companies are conducted at the Division's Clinical Research Unit (CRU). Funds from these studies support the Division's research and clinical training program. Seven studies were completed in 2010, seven in 2011 and ten additional studies will continue into 2012. To date, the CRU has agreements for eleven new studies.

The CRU is a member of the American Lung Association's Asthma Clinical Research Center network, one of 20 centers throughout the United States. The American Lung Association Clinical Research Center completed one study in 2010, one in 2011 and three are still in progress. The American Lung Association has several new protocols pending approval.

See page 17 for funding.

## *IV. Basic and Clinical Research Support*

### *Endowments*

Joy McCann Culverhouse Chair in Allergy and Immunology  
Mabel and Ellsworth Simmons Professorship for Asthma Research

### *Extramural Funding*

#### **Government Funding**

National Institutes of Health  
National Heart, Lung and Blood Institute

#### **Non-Profit Funding**

American Lung Association – National Institutes of Health

#### **Pharmaceutical Funding**

Almirall Pharmaceuticals  
Dyax Corporation  
Forest Laboratories  
Genentech Inc  
GlaxoSmithKline  
Jerini, US  
Merck and Co., Inc.  
MedImmune  
Novartis Pharmaceuticals  
Pfizer  
Pharming Inc  
Sanofi-Aventis Pharmaceuticals  
Schering-Plough Corporation  
Teva Pharmaceuticals  
Viropharma

# V. PUBLICATIONS

## PEER-REVIEWED PUBLICATIONS PUBLISHED OR IN PRESS: 2011

Aljubran SA, Cox R, Parthasarath PT, Gurukumar KR, Mohapatra SS, Lockey RF, Kolliputi N. Enhancer of Zeste Homolog 2 induces Pulmonary Artery Smooth Muscle Cell Proliferation, *PLoS One*, 2011 (In revision).

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Kim D, Phillips J, Lockey RF: Oral curcumin supplementation inpatients with atopic asthma. *Allergy & Rhinology*, in press.

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Kumar A, Glaum M, El-Badri N, Mohapatra S, Haller E, Park S, Patrick L, Nattkemper L, Vo D, Cameron DF. Initial Observations of Cell Mediated Drug Delivery to the Deep Lung. *Cell Transplantation*; 20(5): 609-18, 2011.

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Shaik R, Waxman AB, and Kolliputi N. ASK-1 regulates inflammasome complex formation in hyperoxic acute lung injury. *J. Immunol*, 2011 (In revision).

Wang JW, Li K, Zhang W, Liu G, Hellermann G, Mohapatra S, Lockey RF, Mohapatra SS: MiR-150 suppresses lung inflammation and cytokine production in allergic asthma. *Journal of Clinical Investigation*, submitted.

Zhang W, Cao X, Chen D, Wang JW, Yang H, Wang W, Mohapatra S, Hellermann G, Kong X, Lockey RF, Mohapatra SS. Plasmid-encoded NP73-102 modulates atrial natriuretic peptide receptor signaling and plays a critical role in inducing tolerogenic dendritic cells. *Genet Vaccines Ther*. 2011 Jan 10;9(1):3.

## **BOOK CHAPTERS PUBLISHED OR IN PRESS: 2011**

Fitzhugh DJ, Lockey RF: History of Immunotherapy: The First 100 Years. In: Immunology and Allergy Clinics of North America. Cox L (Guest ed). Alam R (Consulting ed). W. B. Saunders, Philadelphia, PA. Vol 31 (2), Chapter 1, pp. 149 – 157, 2011.

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## **ABSTRACTS**

Aljubran SA, Battula KK, Jalali S, Mohapatra S, Lockey RF, Kolliputi N: Enhancer of Zeste Homolog 2: A Pivotal Role in Pulmonary Artery Smooth Muscle Cell Proliferation? Submitted, American Thoracic Society (ATS) meeting, Denver, Colorado, May 13-18, 2011.

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Wang J, Li K, Hellermann G, Mohapatra SS, Lockey RF, Mohapatra S: MiR150 Transgenic Mice Exhibit Altered T Cell Response, Cytokine Profile and Lung Inflammation. 68<sup>th</sup> Annual American Academy of Allergy, Asthma & Immunology Meeting, Orlando, FL, March 2 – 6, 2012. *J Allergy Clin Immunol* 2012; 129(2): AB136 (# 517). (Featured poster)



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## **BOOKS**

Pawankar R, Canonica, GW, Holgate ST, Lockey, RF (eds) *World Allergy Organization (WAO) White Book on Allergy*. World Allergy Organization, 2011. Website: [www.worldallergy.org](http://www.worldallergy.org)

## **ELECTRONIC MEDIA**

### **1. World Allergy Organization Interactive Case Reports** **[http://www.worldallergy.org/interactive\\_case\\_reviews](http://www.worldallergy.org/interactive_case_reviews)**

#### **Persistent childhood snoring**

Posted: October 2011

Written by: Michel Alkhalil, MD; Robert Pesek, M.D.

Division of Allergy and Immunology, Department of Internal Medicine, University of South Florida, Morsani College of Medicine, and James A. Haley Veterans' Hospital, Tampa, Florida, USA

#### **Seizure in a neonate**

Posted: August 2011

Written by: James Parkerson, D.O.; Richard F. Lockey, M.D.

Division of Allergy and Immunology, Department of Internal Medicine, University of South Florida, Morsani College of Medicine, and James A. Haley Veterans' Hospital, Tampa, Florida, USA

#### **Urticaria and arthralgias in a nine year old with urinary tract infections**

Posted: July 2011

Written by: Ahmed Butt, M.D.; Daanish Rashid; Roger Fox, M.D.

Division of Allergy and Immunology, Department of Internal Medicine, University of South Florida, Morsani College of Medicine, and James A. Haley Veterans' Hospital, Tampa, Florida, USA

#### **A Patient with Facial Rash and Angioedema**

Posted: September 2010

Written by: Robbie Pesek, M.D.; Richard F. Lockey, M.D.

Division of Allergy and Immunology, Department of Internal Medicine, University of South Florida, Morsani College of Medicine, and James A. Haley Veterans' Hospital, Tampa, Florida, USA

## **Cough and Failure to Thrive**

Posted: August 2010

Written by: Dennis Kim, M.D.; Jennifer Kim, M.D.; Richard F. Lockey, M.D.  
Division of Allergy and Immunology, Department of Internal Medicine; University of South Florida, Morsani College of Medicine, and James A. Haley Veterans' Hospital, Tampa, Florida, USA

## **A Persistent Rash with Urticaria and Angioedema Following Penicillin Treatment**

Posted: In press

Written by: Ahmed Butt, M.D.; Daanish Rashid; Richard F. Lockey, M.D.  
Division of Allergy and Immunology, Department of Internal Medicine, University of South Florida, Morsani College of Medicine, and James A. Haley Veterans' Hospital, Tampa, Florida, USA

## **2. PLoS One**

**<http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0037712>**

Aljubran SA, Cox R, Parthasarath PT, Gurukumar KR, Mohapatra SS, Lockey RF, Kolliputi N. Enhancer of Zeste Homolog 2 induces Pulmonary Artery Smooth Muscle Cell Proliferation, *PLoS One*, 5/2012

## VI. Faculty and Staff Awards: 2009-2011

Roger W. Fox, M.D., Selected as one of “*The Best Doctors in America*®” Guide.

Mark C. Glaum, M.D., Ph.D., Selected as one of “*The Best Doctors in America*®”.

Mark C. Glaum, M.D., Ph.D., promoted to Associate Professor, August 2010.

Mark C. Glaum, M.D., Ph.D., Platinum Dean’s Recognition Award for achievements and leadership in education and research /scholarly activity, December 2010.

Sandra G. Gompf, M.D., Selected by Infectious Disease Subspecialty peers for inclusion in “*Best Doctors in America*®”.

Narasaiah Kolliputi, PhD, Society for Experimental Biology and Medicine Young Investigator Award Maywood, New Jersey 2009

Dennis K. Ledford, M.D., President of the American Academy of Allergy, Asthma and Immunology, 2011 to 2012.

Dennis K. Ledford, M.D., Selected as one of “*The Best Doctors in America*®”.

Richard F. Lockey, M.D., President of the World Allergy Organization, 2010 - 2012.

Richard F. Lockey, M.D., Recognition Award, WAO WISC, Dubai. December 6 – 8, 2010.

Richard F. Lockey, M.D., 40 years Department of Veterans Affairs Service Award, 2011.

Richard F. Lockey, M.D., Florida Super Doctors 2009, special advertising supplement to the Tampa Tribune, Gulf Coast Edition, January 17, 2009.

Richard F. Lockey, M.D., Selected as one of “*The Best Doctors in America*®”.

Jia-Wang Wang, PhD, University South Florida SIPAID travel grant, 2011

## ***VII. VISITING PROFESSOR*** ***EDUCATIONAL PROGRAM*** ***2010-2011***

**Mario Sánchez-Borges, M.D.**, Professor of Allergy and Clinical Immunology Faculty of Medicine Central University of Venezuela, Director of Allergy and Clinical Immunology Department, Centro Médico-Docente La Trinidad, Caracas, Venezuela "Atopy, Aspirin, and Mites". December 20, 2010

**Mingnan Chen, Ph.D.**, Center for Biologically Inspired Materials & Material Systems, Department of Biomedical Engineering, Duke University, Durham, North Carolina. "Engineering Polypeptide-Based Vaccines and Drug Carriers for Cancer Therapy" November 19, 2010.

**Robert Naclerio, M.D.**, Professor of Surgery, Section Chief, Otolaryngology – Head and Neck Surgery, University of Chicago, Pritzker School of Medicine, Chicago, Illinois. "Informal roundtable discussion on state of the science and pathophysiology of allergic rhinitis" and "Treatment of Nonallergic Rhinitis" October 27, 2010.

**David B. Weiner, Ph.D.**, Professor, Department of Pathology & Laboratory Medicine, Chair, Gene Therapy & Vaccine Program, University of Pennsylvania School of Medicine, Philadelphia, PA SIPAIIID Distinguished Lecture Series, "DNA Vaccines: Rise of the Phoenix", June 15, 2010.

**Georgio Walter Canonica, M.D.**, Professor of Allergy and Respiratory Diseases Chairman of the Allergy and Respiratory Diseases Clinic, Director of the Specialty School of Pulmonary Diseases, Genoa University, Genoa, Italy. "Sublingual Immunotherapy 2010 or SLIT after the WAO Position" May 14, 2010.

**Chen Dong, Ph.D.**, Professor, Immunology, MD Anderson Cancer Center, Houston, Texas SIPAIIID Distinguished Lecture Series. "Molecular Pathways Leading to Allergic Diseases". April 15, 2010.

**Giovanni Piedimonte, M.D.**, Wyeth Research Scholar, Professor & Chairman in the Department of Pediatrics, West Virginia University School of Medicine, Physician-in-Chief, West Virginia University Children's Hospital. "State of the Science: Emerging Issues in Basic Asthma Research" March 5, 2010.

**David Skoner, M.D.**, Director, Allegheny General Hospital, Division of Allergy, Asthma and Immunology, Vice Chair, Allegheny General Hospital Clinical Research, Division of Allergy & Immunology, Professor, Pediatrics, Drexel University College of Medicine, Pittsburgh, Pennsylvania. “Hot Topics in Respiratory Disease” May, 2011

**Brian Lipworth, M.D.**, Professor of Allergy and Respiratory Medicine, Asthma and Allergy Research Group, Division of Medicine & Therapeutics, Ninewells Hospital & Medical School, Dundee, Scotland. “Cornerstone of Asthma: Treating Large and Small Airways” February, 2011