

# 2018 AWARD RECIPIENTS



**Rheumatology  
Research Foundation**  
Advancing Treatment | Finding Cures

# CREATING A BRIGHTER FUTURE FOR RHEUMATOLOGY

The Rheumatology Research Foundation is committed to improving care for the more than 54 million Americans affected by arthritis or other forms of rheumatic disease. The Foundation's extensive awards program helps patients by increasing the number of rheumatology health professionals while also funding research advancements that lead to new treatments and cures.

For more than two decades, the Foundation has supported high-quality clinical and translational research as well as education and training programs. In the coming fiscal year (July 1, 2018 - June 30, 2019), the Foundation has committed to fund more than \$9.4 million to rheumatology research and training. About half of those awards will support efforts to recruit and train the next generation of rheumatology professionals, which decreases patient wait times and increases access to rheumatology care. The remaining funds will be awarded to advance research projects that lead to breakthroughs in treating people with rheumatic diseases.

In all, the Foundation has committed more than \$161 million to fund more than 3,400 awards since 1985, making it the largest private funding source of rheumatology research and training in the United States.

Congratulations to the Foundation's latest award recipients. Their work is vital to creating a brighter future for the field of rheumatology and for the people impacted by rheumatic disease.



## **BRYCE A. BINSTADT, MD, PHD** **CHAIR, SCIENTIFIC ADVISORY COUNCIL**

**ASSOCIATE PROFESSOR OF PEDIATRICS AND A DISTINGUISHED  
UNIVERSITY TEACHING PROFESSOR, DIVISION OF PEDIATRIC  
RHEUMATOLOGY, UNIVERSITY OF MINNESOTA MEDICAL SCHOOL**

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# INNOVATIVE RESEARCH AWARDS

The Innovative Research Award encourages independent investigators to conduct novel studies that generate new insights into the cause, progression, treatment and outcomes of rheumatic diseases.

# BRYCE A. BINSTADT, MD, PHD

UNIVERSITY OF MINNESOTA

## **MECHANISMS OF ARTHRITIS-ASSOCIATED CARDIOVASCULAR INFLAMMATION AND FIBROSIS**

Excess cardiovascular morbidity and mortality accompany many rheumatic diseases, including rheumatoid arthritis (RA). The mechanisms by which rheumatic diseases damage the cardiovascular system are not well understood.

Using a powerful mouse model, we are exploring the cellular and molecular events linking chronic rheumatic disease and cardiovascular injury. This project focuses specifically on the role of endothelial cells in this process. In the context of systemic inflammation, endothelial cells undergo endothelial-to-mesenchymal transition (EndoMT) to become pro-fibrotic and pathogenic myofibroblasts. We are studying the molecular mediators of EndoMT in a mouse model of co-existing inflammatory arthritis and cardiovascular inflammation and fibrosis.

Our goal is to define new therapeutic targets that will improve cardiovascular outcomes for patients with rheumatic diseases.



# KEITH A. ELKON, MD

UNIVERSITY OF WASHINGTON

## **CGAS-STING AND MITOCHONDRIAL DNA IN LUPUS**

This project seeks to identify proteins in an interferon pathway responsible for inflammation and associated with more severe disease activity in patients with SLE. We also propose to determine the source of activation and see whether we can show this pathway activated following a known trigger of lupus, ultraviolet light. If we succeed, several rational therapies, most already available for treatment of other diseases, can be tested for efficacy in selected patients with SLE (precision medicine).

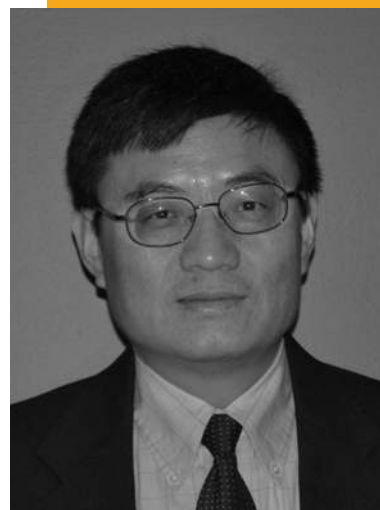


# XU FENG, PHD

UNIVERSITY OF ALABAMA AT BIRMINGHAM

## **DEVELOPMENT OF NOVEL THERAPEUTICS FOR BONE EROSION IN RHEUMATOID ARTHRITIS**

Bone erosion, a hallmark of RA, is a major factor contributing to the functional disability of RA patients. However, current drugs for RA bone erosion offer only moderate efficacy but often cause considerable side effects. Our long-term goal is to develop more efficacious and safer therapeutics for preventing and treating RA bone erosion. TNF- $\alpha$  (TNF) and IL-1 are two major factors implicated in bone erosion in RA. TNF/IL-1-mediated osteoclast formation requires RANK (the receptor activator of NF- $\kappa$ B) signaling, which is activated by RANKL (RANK ligand). Numerous studies showed that the blockade of RANKL-RANK interaction prevents bone loss in rodent RA models. However, this therapeutic strategy may cause various side effects since the RANKL/RANK system also plays important roles in the immune system function. Thus, a better targeting strategy would be to use small molecules that target RANK signaling pathways that are involved in osteoclast formation/RA bone erosion but not in immune system function. Our laboratory previously identified two RANK motifs which play crucial roles in osteoclast formation. We have generated knockin mice bearing inactivating mutations in these two RANK motifs, which develop osteopetrosis with impaired osteoclast formation. Importantly, inactivation of these two motifs does not affect the ability of RANK to activate the signaling pathways known to mediate immune cell development and function. Moreover, our preliminary studies using osteoclast precursors from the knockin mice show that the two RANK motifs are required for TNF/IL-1-mediated osteoclast formation in vitro. Based on these data, we hypothesize that specifically targeting these two RANK motifs has the potential to serve as an effective and safe strategy for preventing and treating RA bone erosion. We have also developed cell-based assay systems for identifying compounds targeting these two RANK motifs. High-throughput screens of several thousand compounds with the cell-based assay systems followed by counter screen assays identified numerous compounds that potently inhibited osteoclast formation in vitro. The objectives of this proposal are: a) evaluate the potential of the two RANK motifs as drug targets for RA bone erosion; and b) carry out an important proof-of-concept study to further support the novel targeting strategy.



# J. MICHELLE KAHLENBERG, MD, PHD

UNIVERSITY OF MICHIGAN

## **A NOVEL MECHANISM FOR PHOTSENSITIVITY IN SYSTEMIC LUPUS ERYTHEMATOSUS**

Increased photosensitivity is a hallmark of SLE and negatively impacts patients' quality of life, yet the mechanisms that drive it are not understood. Type I interferons (IFNs) are increased in cutaneous lupus erythematosus (CLE) lesions and contribute to disease pathogenesis, yet the role they play in photosensitivity is unknown. Keratinocytes are the primary source of IFN kappa ( $\kappa$ ), a type I IFN that is a genetic risk factor for cutaneous lupus, significantly upregulated in CLE skin lesions, and is produced more robustly from non-lesional SLE vs. control keratinocytes. We have now identified that autocrine IFN $\kappa$  primes keratinocytes for exaggerated responses to exogenous type I IFNs and that overexpression of IFN $\kappa$  is sufficient to increase keratinocyte apoptosis after UV light exposure. This project will evaluate the mechanisms by which IFN $\kappa$  contributes to enhanced cell death in lupus skin, including evaluation of reactive oxygen species formation and novel interferon-driven pro-apoptotic genes. We will utilize translational (studying control and lupus keratinocytes) and murine approaches to answer these questions. Completion of these studies will identify IFN $\kappa$  and its downstream mediators as novel targets for prevention of photosensitive reactions in SLE and consequently provide a mechanism by which UV-induced disease flares can be eliminated.





# TED R. MIKULS, MD, MSPH

UNIVERSITY OF NEBRASKA MEDICAL CENTER

## **DRIVING PERFORMANCE AND SAFETY IN RHEUMATOID ARTHRITIS**

Automobile driving is an instrumental activity of daily living of heightened importance among RA patients, who are especially reliant on driving for the preservation of health, well-being, and quality of life. Despite its importance, there have been no rigorously designed studies directly comparing driving performance in RA patients with other populations. As a result, determinants of driving risk, strategies to mitigate this risk and preserve this vitally important activity are unknown. In the first investigation of its kind, we will quantify and compare driving performance in RA patients and matched controls using a state-of-the-art high-fidelity driving simulator coupled with naturalistic in-car assessments. Our overarching hypothesis is that RA adversely affects driving performance and safety, posing a modifiable risk to patients. We will examine this hypothesis by addressing two study aims. In Specific Aim 1, we will systematically assess driving performance in RA patients and matched controls using a high-fidelity driving stimulator coupled with naturalistic in-car assessments leveraging instrumented vehicle technology. In Specific Aim 2, we will identify factors in RA patients associated with driving performance over a follow-up period 16 weeks. By using a longitudinal design, we will be able to specifically examine the how RA disease activity and corresponding physical function affect driving. Results from this study will fill critical gaps in RA management. By identifying the specific driving tasks that pose the greatest risk in this population, we will gather the information needed to guide future interventions aimed at improving safety and maintaining driving as a critical activity of daily living in RA patients. Importantly, this study will enable the future development of a driving assessment “toolkit” that could be leveraged by patients and arthritis providers alike to facilitate and improve patient-provider communications that, at present, appear to be grossly insufficient.



# MIRIAM A. SHELEF, MD, PHD

UNIVERSITY OF WISCONSIN, MADISON

## **PEPTIDYLARGININE DEIMINASE 2 AND CITRULLINATION OF IGG IN IMMUNITY AND RHEUMATOID ARTHRITIS**

Anti-citrullinated protein antibodies (ACPAs) are a hallmark of rheumatoid arthritis, but the role of citrullination and the citrullinating enzyme, peptidylarginine deiminase 2 (PAD2), is unclear. PAD2 may impact RA in part through citrullinating antigens ultimately targeted by ACPAs and in part through regulating immune cells, but its complete role is unknown. Identifying the mechanisms by which PAD2 and citrullination impact immunity and arthritis is critical to define fundamental immunologic pathways, understand aberrant pathways in RA, and intelligently guide the design of PAD inhibitors for RA treatment. The objective of this project is to discover the mechanisms by which PAD2 regulates plasma cells and antibodies in a normal immune response as well as autoantibodies in RA. The successful completion of this project will inform the development of novel diagnostics and therapeutics to improve the health of people with RA and rheumatologic diseases.



# HAN-RONG WENG, MD, PHD

UNIVERSITY OF GEORGIA

## **MOLECULAR MECHANISMS AND TREATMENT OF PAIN IN SYSTEMIC LUPUS ERYTHEMATOSUS**

Chronic pain in SLE significantly impairs the quality of life and productivities of patients due to the lack of potent and safe painkillers. There is a highly unmet need for the development of novel analgesics. Surprisingly, no animal studies had been conducted to investigate mechanisms underlying chronic pain in lupus until our recent study. In MRL lupus-prone (MRL/lpr) mice, we found that these animals exhibit robust spinal neuroinflammation and activation of microglial macrophage colony-stimulating factor-1 receptors in the spinal dorsal horn plays a crucial role in generation of chronic pain in MRL/lpr mice. Our study demonstrated that glial-mediated spinal inflammation is critical in the genesis of chronic pain in lupus, and pathways regulating this process are exciting approaches for the treatment of pain in lupus. GPR109A was newly identified in different immune cell types, and its activation produces anti-inflammatory effects. Whether and how spinal GPR109A regulates spinal neuroinflammation and the genesis of pathological pain (including chronic pain in SLE) is unknown. Our overarching hypothesis is that activation of the anti-inflammatory receptor GPR109A in spinal microglia attenuates lupus-induced chronic pain and microglial production of pro-inflammatory mediators, in part, through suppressing microglial N-type calcium channel activity. This hypothesis will be tested in 3 specific aims using lupus-prone (MRL/lpr) mice: (1) to test the hypothesis that chronic pain in MRL/lpr mice is reduced by activation of spinal GPR109A; (2) to test the hypothesis that activation of GPR109A suppresses microglial N-type  $\text{Ca}^{2+}$  channel activity in MRL/lpr mice with chronic pain; (3) to test the hypothesis that microglial activation and production of pro-inflammatory mediators are regulated by N-type  $\text{Ca}^{2+}$  channels and GPR109A. This study is the first to uncover analgesic effects of the GPR109A activator and its underlying mechanisms. Given that spinal neuroinflammation is a common feature shared by many chronic pain conditions, the results collected in the study will also provide a base for the use of GRP109A agonists to treat other chronic pain conditions.



# JULIE ZIKHERMAN, MD

UNIVERSITY OF CALIFORNIA, SAN FRANCISCO

## EXPLOITING NUR77 TO SELECTIVELY RESTRAIN AUTOREACTIVE B CELLS

Existing therapeutics for SLE and related autoimmune diseases generally target some component of normal immune cell function, leaving patients susceptible to infection. The 'holy grail' of autoimmune disease therapeutics would be to enhance tolerance mechanisms of antigen-specific autoreactive lymphocytes and thereby limit or even eliminate off-target immunosuppression. The Nr4a1, 2, and 3 genes encode a small family of orphan nuclear receptors, Nur77, Nurr1, and Nor1 respectively, that share significant structural similarities in their DNA and ligand binding domains. Nr4a1-3 are among a small set of primary response genes (PRGs) that are rapidly and robustly induced in response to antigen receptor stimulation in lymphocytes. We find that Nur77 negatively regulates BCR-induced B cell proliferation and imposes a negative feedback loop downstream of antigen stimulation by repressing transcription of other PRGs. In addition to dynamic induction of Nur77 with strong BCR stimuli, we have identified selective upregulation of Nur77 at steady state in two independent mouse models of B cell anergy, and in naturally occurring self-reactive B cells. Moreover, we find that Nur77 deficiency is sufficient to break tolerance on a genetic background characterized by autoreactive B cells and is permissive for production of DNA-specific auto-antibodies. We thus have evidence to suggest that the orphan nuclear receptor Nur77 is upregulated selectively in auto-reactive B cells at steady state, where it imposes a novel layer of immune tolerance and therefore represents an exciting new therapeutic target in lupus. The goals for our proposal are to define the cellular and transcriptional mechanisms by which Nur77 imposes B cell tolerance and to test the feasibility of manipulating Nur77 function with a small molecule agonist ligand in order to re-establish B cell tolerance in murine models of lupus. In doing so, we will validate Nur77 as a new clinical target in lupus and establish efficacy of a small molecule agonist in animal models, laying the groundwork for development of a novel therapy for lupus and related autoimmune diseases.



# CAREER DEVELOPMENT RESEARCH AWARDS

Establishing a productive research career in rheumatology is largely dependent upon the availability of major research funding.

Increasing concerns over the decline in federal funding for rheumatology research have forced many investigators to reconsider their careers, which leads to fewer researchers making important discoveries necessary to advance treatments and find cures.

The Career Development Research Awards are designed to encourage early and mid-career investigators to continue vital research into the cause, prevention, and treatment of rheumatic diseases.

## CAREER DEVELOPMENT BRIDGE FUNDING AWARD: K BRIDGE

THE K BRIDGE AWARD ENCOURAGES JUNIOR INVESTIGATORS TO CONTINUE CONDUCTING RESEARCH INTO NOVEL IDEAS WHILE REAPPLYING FOR AN NIH OR VA CAREER DEVELOPMENT AWARD.

### JUDITH ASHOURI, MD

UNIVERSITY OF CALIFORNIA, SAN FRANCISCO

#### **SIGNALING AND REPERTOIRE DIFFERENCES IN ARTHRITOGENIC T CELLS**

Rheumatoid arthritis affects millions globally and is without cure. T cells are known to play a key role in RA pathogenesis. Paradoxically, CD4 T cells from patients with RA that are hyporesponsive to T cell receptor (TCR) engagement are able to hyperproliferate and differentiate into pathogenic effector cells. The mechanism of this TCR-signaling paradox is unknown. Identification of Ag-activated T cells in RA would allow investigation into the contribution of both early TCR signaling events and their autoreactive repertoire to RA pathogenesis. However, difficulty in isolating the relevant TCR-activated T cells (as opposed to those activated by the inflammatory milieu) has limited the field's understanding of this apparent paradox. Dr. Ashouri and colleagues have pioneered a novel strategy to overcome this limitation. In this proposal, she builds on preliminary data that demonstrate CD4 T cells in both a mouse model of RA (SKG mice) and humans with RA respond to joint-specific Ags, and that, in vivo, TCR signaling strength in the SKG mice correlates with their ability to cause arthritis. Dr. Ashouri proposes to address two key questions in RA: (a) the mechanism of the TCR signaling paradox in disease pathogenesis and (b) the identification of the autoreactive TCR repertoire in arthritogenic T cells. Successful completion of the proposed studies will provide new mechanistic insights into RA pathogenesis that will hold promise for improved therapeutic targets.



## CAREER DEVELOPMENT BRIDGE FUNDING AWARD: K SUPPLEMENT

THE K SUPPLEMENT AWARD ENCOURAGES JUNIOR INVESTIGATORS TO EXPAND PROMISING RESEARCH BY PROVIDING ADDITIONAL SUPPORT TO COVER RESEARCH COSTS AND HELP INVESTIGATORS BECOME INDEPENDENT.

### KRISTINE A. KUHN, MD, PHD

UNIVERSITY OF COLORADO, DENVER

#### TRAFFICKING OF BACTERIA-SPECIFIC T CELLS IN MURINE IBD-SPA

My research interests center upon the gut-joint hypothesis for the development of spondyloarthritis (SpA). The topic of my K08 award focuses upon how resident intestinal bacteria influence the function of colon intraepithelial lymphocytes, T cells that reside within the epithelium. Now in the second year of my K08 award, I have been very productive, having recently published on the role of specific intestinal bacteria in affecting intraepithelial lymphocyte function that improves epithelial barrier protection. As I become an independent investigator, my aim is to apply these skills towards designing studies that will elucidate how bacterial dysbiosis described in SpA stimulates inflammatory processes that lead to disease. My specific hypothesis is that intestinal dysbiosis during SpA leads to unique commensal-specific T cells that traffic to the joint where they stimulate arthritis. In this K Supplement, I will generate key preliminary data demonstrating the feasibility of identifying bacteria-specific T cells in the mucosal immune system of mice using TNFΔARE/+ mice with spontaneous inflammatory bowel disease (IBD) and SpA and comparing to healthy littermate controls. In addition, I will expand our ongoing preliminary studies of lymphocyte trafficking between the colon and joint by evaluating trafficking of lymphocytes from the small intestine to the joint. Such preliminary data will be essential to propose the planned aims of my R01 application: (1) Define the TCR repertoire of bacteria-specific T cells in mice with IBD-SpA relative to healthy mice; (2) Determine the localization of bacteria-specific T cells in mice with IBD-SpA relative to healthy mice; and (3) Identify the function of T cells of intestinal origin in the joint. The goal of the proposed studies is to define a novel mechanism for the development of SpA that can be utilized for translational studies in humans in which better diagnostic and therapeutic options can be developed.



## CAREER DEVELOPMENT BRIDGE FUNDING AWARD: R BRIDGE

THE R BRIDGE AWARD ENCOURAGES ESSENTIAL RHEUMATOLOGY RESEARCH BY SUPPORTING PROMISING INVESTIGATORS WHO ARE AT RISK OF RUNNING OUT OF RESEARCH FUNDING AND ARE REVISING OUTSTANDING NIH R01 OR VA RCS/ORD AWARD APPLICATIONS.

### BENJAMIN CHONG, MD, MSCS

UNIVERSITY OF TEXAS SOUTHWESTERN MEDICAL CENTER  
**OPTIMIZATION OF CLINICAL TRIAL DESIGN IN CUTANEOUS LUPUS**

To enhance the quality of clinical trials in cutaneous lupus erythematosus (CLE), this proposal will characterize disease activity courses of patients with CLE and benchmark outcome measures that adequately demonstrate treatment efficacy. We have collected longitudinal clinical and quality of life data from over 650 patients with CLE. However, our data's applicability for clinical trial design is restricted by lack of standardized time points. Thus, we propose a rigorous 24-week observational study with four visits spaced eight weeks apart to address this limitation. Our first aim will define disease activity courses over a 24-week period in patients with CLE. We will follow the patients' Cutaneous Lupus Erythematosus Activity and Severity Index (CLASI) activity scores over time. We hypothesize that patients on standard-of-care treatments for CLE will mostly demonstrate improvement in disease activity, thus making the data suitable for controls and helpful in planning for target recruitment numbers. Our second aim is to establish a benchmark for the percentage of improvement in CLASI activity score that is sufficient for treatment efficacy. We propose comparing percentage changes of CLASI activity scores with physician and patient assessments of disease activity change. We hypothesize that attaining at least 50% improvement in CLASI activity scores can be used as a benchmarked endpoint to demonstrate treatment efficacy. Our third aim will focus on changes in patient-reported outcome measure scores in patients with CLE as endpoints for therapeutic efficacy. We will compare changes in scores of patient-reported outcome measures, such as the SKINDEX-29+3 questionnaire, at weeks 0 and 24. We hypothesize that a decrease of at least 10 in SKINDEX-29+3 scores can be a benchmarked patient-reported outcome measure. Thus, CLE clinical trials may use these data as guidelines to determine whether patients experience therapeutic benefit.





## CECILIA P. CHUNG, MD, MPH

VANDERBILT UNIVERSITY

### **A PERSONALIZED MEDICINE APPROACH TO IMPROVE THE PREDICTION OF AZATHIOPRINE TOXICITY**

Azathioprine is an immunosuppressive drug widely used for the treatment of rheumatic and other inflammatory conditions. However, it has a narrow therapeutic index and the frequency of clinically significant side effects associated with its use is approximately 50%. This project will examine the role of genetic variants to improve the prediction of two of the most serious adverse effects of azathioprine: myelosuppression and pancreatitis.

We submitted an R01 proposal to conduct genetic and gene expression association analyses, leveraging two large practice-based biobanks: (1) Vanderbilt's BioVU, one of the largest practice-based biobanks in the U.S., and (2) the Million Veteran Program, currently enrolling, collecting clinical data from, and genotyping U.S. veterans. The study will include 6,625 patients exposed to azathioprine at Vanderbilt University Medical Center (discovery cohort) and 6,000 patients exposed to azathioprine at the VA (replication cohort), for whom we have clinical information and DNA available for testing. The research plan includes a multistep approach that combines candidate gene analyses, genome-wide association studies, gene expression association analysis, and novel statistical techniques to build models to predict myelosuppression and pancreatitis associated with azathioprine use.

In this Foundation bridge award, we will demonstrate the feasibility of our R01 proposal. We will: (1) construct cohorts of patients taking azathioprine, (2) develop and validate an algorithm to identify concurrent use of azathioprine during episodes of myelosuppression, (3) review charts to identify patients with pancreatitis induced by azathioprine use, (4) select candidate genes for the study, and (5) develop statistical models to incorporate clinical and genetic data into the prediction models of myelosuppression and pancreatitis.

This project coheres with the Foundation's mission to "improve the health of people with rheumatic diseases", in particular patients with systemic lupus erythematosus, systemic vasculitis, and other inflammatory conditions receiving azathioprine. Furthermore, by constructing two genetic models that will predict serious and frequent side effects of the commonly used drug azathioprine, our goals are also concordant with the Precision Medicine Initiative's goal that seeks to deliver "the right drug, at the right dose, to the right patient."





# INVESTIGATOR AWARD

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THE INVESTIGATOR AWARD ENCOURAGES JUNIOR INVESTIGATORS TO CONTINUE CONDUCTING INNOVATIVE RESEARCH THAT WILL BE COMPETITIVE FOR MORE SIGNIFICANT FUNDING WHILE THEY ESTABLISH THEMSELVES AS INDEPENDENT INVESTIGATORS.

# BRENDAN ANTIOCHOS, MD

JOHNS HOPKINS UNIVERSITY

## **THE RETROELEMENT LONG INTERSPERSED ELEMENT-1 (LINE-1) AS A SOURCE OF AUTOANTIGENS AND IMMUNOSTIMULATORY DNA IN SYSTEMIC LUPUS ERYTHEMATOSUS AND SJÖGREN'S SYNDROME**

SLE and Sjögren's syndrome (SS) are systemic rheumatic diseases that share phenotypic and immunologic features. Two particularly notable shared characteristics of these diseases have been studied in detail: the presence of autoantibodies that target a group of nucleic acid-binding proteins and activation of elements of the innate immune system in the form of an "interferon signature" in affected blood and tissues. Retroelements have recently been identified as stimuli which may contribute to features of autoimmunity through several mechanisms. In this study, we will address the hypothesis that the retroelement LINE-1 is an important mechanistic link between these two pathological observations in some patients with lupus and SS. As endogenous sources of immunostimulatory nucleic acid, retroelements have the capability to drive pathogenic immune activation and interferon production. Unique among human retroelements, LINE-1 also encodes two proteins: ORF1p and ORF2p. This project will determine (1) whether LINE-1 generates antigens that are targeted by autoantibodies in SLE and SS and (2) whether cytoplasmic DNA sensing machinery plays a role in the detection and regulation of LINE-1 in a manner that contributes to the interferon signature in these diseases.



# MEDHA BARBHAIYA, MD, MPH

HOSPITAL FOR SPECIAL SURGERY

## **METHODOLOGICAL APPROACHES TO ANTIPHOSPHOLIPID SYNDROME CLASSIFICATION AND EPIDEMIOLOGY**

Antiphospholipid syndrome (APS) is a systemic rheumatic disease whose pathogenesis is likely multifactorial, resulting from yet poorly understood genetic and environmental factors. According to the 2006 Revised Sapporo APS Classification Criteria, APS is characterized by thrombosis and/or pregnancy morbidity in patients with persistent antiphospholipid antibodies (aPL). We demonstrated that >90% of worldwide APS experts consider the current classification system to be inadequate by not capturing the full spectrum of clinical and laboratory manifestations of disease, distinguishing APS from other conditions, weighting criteria, or including strong evidence basis. Current APS studies are limited by small sample sizes, varying aPL/APS definitions, heterogeneous populations, unstandardized laboratory tests, and failure to consider other autoantibodies and non-thrombotic manifestations. Thus, given the major morbidity and mortality related to APS, there is an urgent need for stringent, high-performing classification criteria. The overall objective of this study is to develop and validate new APS classification criteria, employing a balance of expert- and data- driven methods. We will utilize bias reduction strategies and include a core group of American and European APS experts, as well as perform systematic literature reviews and develop large, multi-center patient datasets. We hypothesize that the newly developed APS Classification Criteria set will demonstrate excellent face, discriminant, and construct validity; and the sensitivity, specificity, and positive predictive value will be increased compared to expert diagnosis or current classification criteria. A secondary objective is to leverage the large, multi-center APS patient datasets to investigate associations between potentially modifiable environmental factors for risk of APS. We expect these aims to lead to improved understanding of the natural history of APS, identification of modifiable risk factors for prevention, and testing of treatment options in relatively homogeneous APS populations.



A full-page background image of a female scientist in a white lab coat, focused on her work. She is using a Fisherbrand pipette to transfer liquid into a multi-well plate. The laboratory setting includes various equipment like a water bottle, a container, and other labware. The image has a blue tint.

# SCIENTIST DEVELOPMENT AWARD

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THE SCIENTIST DEVELOPMENT AWARD ENCOURAGES  
RHEUMATOLOGISTS AND RHEUMATOLOGY HEALTH  
PROFESSIONALS TO PURSUE INNOVATIVE RESEARCH IDEAS.

# STEPHEN J. BALEVIC, MD

DUKE UNIVERSITY

## **PHARMACOKINETICS AND PHARMACODYNAMICS OF HYDROXYCHLOROQUINE AND AZATHIOPRINE IN PREGNANT WOMEN WITH SYSTEMIC LUPUS ERYTHEMATOSUS**

Active SLE in pregnancy results in devastating neonatal outcomes. These outcomes include preterm birth, low birth weight, and critical care hospitalization. To control maternal disease activity and prevent poor outcomes, over 70% of pregnant women with lupus receive hydroxychloroquine (HCQ) or azathioprine (AZA) during pregnancy and postpartum. In spite of treatment, more than half of pregnant women with lupus continue to experience active disease and poor neonatal outcomes. This could be due, in large part, to inadequate dosing of HCQ and AZA that does not account for the physiologic changes throughout pregnancy that affect drug disposition. However, the presence, timing, and magnitude of the effect of pregnancy changes on drug disposition, and the implications on dosing in pregnancy are unknown, resulting in an urgent, unmet public health need. In this proposal, we will use drug levels to perform pharmacokinetic/pharmacodynamic modeling and dosing simulations to determine the optimal dose of HCQ and AZA throughout pregnancy and postpartum.





# JENNIFER L. BARNAS, MD, PHD

UNIVERSITY OF ROCHESTER

## THE CONTRIBUTION OF RENAL EPITHELIAL CELLS TO INNATE INTERFERON PATHWAYS IN LUPUS NEPHRITIS

Type I interferon production from nucleic acid-sensing pathways drives SLE pathogenesis according to current dogma. However, type III interferons (IFN- $\lambda$ ) can stimulate a similar gene expression profile. IFN- $\lambda$  is detected in the blood, kidneys, and skin of lupus patients. The contribution of IFN- $\lambda$  to lupus and lupus nephritis pathogenesis is currently unknown. The objective of this proposal is to identify innate interferon pathways operative in the human renal microenvironment of lupus nephritis. The main hypothesis is that renal tubule epithelial cells promote lupus nephritis via the production of IFN- $\lambda$ . It is hypothesized that IFN- $\lambda$  is the dominant interferon expressed in lupus nephritis renal tubule epithelial cells and that its expression level correlates with disease severity. It is also hypothesized they will produce IFN- $\lambda$  with stimulation of nucleic acid and oxidative stress sensing pathways. Immunohistochemistry and laser microdissection of patient renal biopsies will be used to assess in situ protein and RNA expression of innate interferons and their gene signatures. One working hypothesis is that nucleic acid sensors, IFN- $\lambda$ , and IFN- $\lambda$  stimulate innate interferon production in human renal tubule epithelial cells. In addition, oxidative stress is an independent pathway by which IFN- $\lambda$  can be produced via intracellular sensors like mitochondrial antiviral sensing protein (MAVS). As IFN- $\lambda$  production also utilizes MAVS, another working hypothesis is that oxidative stress triggers IFN- $\lambda$  release by epithelial cells. Production of innate interferons will be measured in cultured human primary renal tubule epithelial cells after stimulation via oxidative stress, nucleic acid pattern recognition receptors or innate interferon pathways. The expected outcome is that the innate interferon most clinically relevant to lupus nephritis will be identified and pathways stimulating renal tubule epithelial cell production of innate interferon will be delineated. Long-term goals include establishing the functional role of IFN- $\lambda$  in lupus and the renal microenvironment including its contribution to renal fibrosis and end-stage kidney disease. Understanding how innate interferon pathways interconnect will allow for their therapeutic calibration for improved lupus patient outcome as well as in other autoimmune and viral diseases.



# MARGARET CHANG, MD, PHD

BOSTON CHILDREN'S HOSPITAL

## TISSUE RESIDENT MEMORY T CELLS IN RECURRENT SYNOVITIS

Treatment of RA and juvenile idiopathic arthritis often requires lifelong therapy with considerable cost and morbidity, yet episodic flares remain commonplace, highlighting the need for specific therapeutic strategies for sustained disease control.

Arthritis flares exhibit a strong tendency to recur in the same joints, varying in a highly individualized manner from patient to patient. Tissue resident memory (TRM) T cells are a subset of memory cells that take up long-term residence in peripheral tissues and mediate recurrent site-specific inflammation. As our research group recently identified cells with TRM-like phenotype in human synovial samples taken from patients with RA, we hypothesize that synovial TRM cells represent the pathophysiologic basis for joint-specific memory in inflammatory arthritis.

To study this, we generated a murine model of recurrent, joint-specific arthritis. Our preliminary data in this system implicate TRM cells in arthritis flares, providing a tractable experimental model to explore the development and function of synovial TRM cells in vivo.

Our project aims to show how synovial TRM cells play a key role in the pattern of arthritis flares and to identify TRM cells as a promising new therapeutic target in order to attenuate recurrent arthritis.





# BRYANT R. ENGLAND, MD

UNIVERSITY OF NEBRASKA MEDICAL CENTER AND VA  
NEBRASKA-WESTERN IOWA HEALTH CARE SYSTEM

## **EPIDEMIOLOGY OF MULTIMORBIDITY IN RHEUMATOID ARTHRITIS**

Multimorbidity—the presence of two or more chronic conditions—poses a tremendous threat to health by increasing the risk of hospitalization, disability, and mortality. Although associated with similarly poor long-term outcomes and predisposing to the development of many individual chronic diseases, there has been little study of multimorbidity in the context of RA. Thus, while strategies to reduce multimorbidity development and progression in RA are needed, significant knowledge gaps limit the development of effective management approaches. In this proposal, we will conduct a comprehensive epidemiologic study of multimorbidity in RA. In Specific Aim 1, we will use large administrative and electronic health record enabled datasets to characterize the prevalence, incidence, and progression of multimorbidity compared to matched general population and diseased controls, with an emphasis on multimorbidity risk surrounding the period of RA onset. In Specific Aim 2, we will use novel bioinformatics methods to identify multimorbidity clusters in RA patients and compare their prognostic value for important health-related outcomes: hospitalization, mortality, and healthcare utilization. Together, this will establish a requisite foundation of knowledge referent to multimorbidity in RA including the derivation of clinically informative RA multimorbidity clusters. The results will inform future multimorbidity outcome measure development as well as pharmacoepidemiologic and comparative effectiveness studies in the multimorbid RA population. Coupled with a robust research training plan, the proposed investigation will be completed under the guidance of a highly productive team of senior investigators and will support the applicant's development into an independent investigator with the necessary skillset to lead future clinical and translational research efforts.



# JOYCE HUI-YUEN, MD, MSC

COHEN CHILDREN'S MEDICAL CENTER

## **EPIGENETIC INFLUENCES ON PEDIATRIC LUPUS**

SLE is an autoimmune disease that can affect major organs of the body, and primarily affects women of child-bearing age. Treatment is challenging, as what causes lupus remains unknown and there are few standard protocols. Often, a patient's blood gives us critical clues to identifying the types of cells and possible genetic factors that are important in the disease process. My proposed project addresses sampling of blood from children with lupus, and those who do not, to explore the relationship between genes and the environment on the development of disease, and the ways in which genetic variations can alter the human immune response and result in disease. This information will be crucial to the design of new treatments that may be safer and more effective than current therapies.



# SARAH B. LIEBER, MD

HOSPITAL FOR SPECIAL SURGERY

## **FRAILITY IN SYSTEMIC LUPUS ERYTHEMATOSUS: A NOVEL PARADIGM TO ENHANCE UNDERSTANDING OF DISEASE ACTIVITY AND DAMAGE**

Frailty, a well-defined syndrome in the geriatric literature reflecting decreased homeostatic reserve, has been linked to increased morbidity and mortality in multiple chronic conditions, but very little is known about the effect of frailty on patients with SLE. Frailty may be underappreciated in lupus patients, as these patients are often young and do not have the cachectic phenotype often associated with the frail elderly. In addition, whether frailty is associated with inflammatory and metabolic biomarkers, body composition, disease activity, disease damage, and patient-reported outcome measures (PROMs) and whether frailty is a risk factor for poor outcomes in patients with lupus are not clear. The aims of this study are to determine the prevalence of frailty in a prospective cohort of patients with lupus, as well as to evaluate the cross-sectional association of frailty with imaging and metabolic biomarkers, PROMs, and sarcopenia as determined by dual-energy x-ray absorptiometry scan. In addition, this study will determine the longitudinal association of frailty with disease activity, damage, and PROMs in patients with lupus. We hypothesize that the prevalence of frailty in this cohort of lupus patients will be comparable to the prevalence of frailty in community-dwelling elderly. We also hypothesize that frailty will be significantly associated with inflammatory and imaging biomarkers, as well as worse disease activity and damage, after controlling for potential confounders, both cross-sectionally and longitudinally. Frailty may reflect a distinct phenotype that captures a subset of patients with lupus whose vulnerability is not fully explained by evaluation of disease activity and damage and is a potentially modifiable risk factor that could be a target for non-pharmacologic, or even pharmacologic, therapies, especially in patients for whom immunosuppressive therapies are inappropriate or contraindicated.



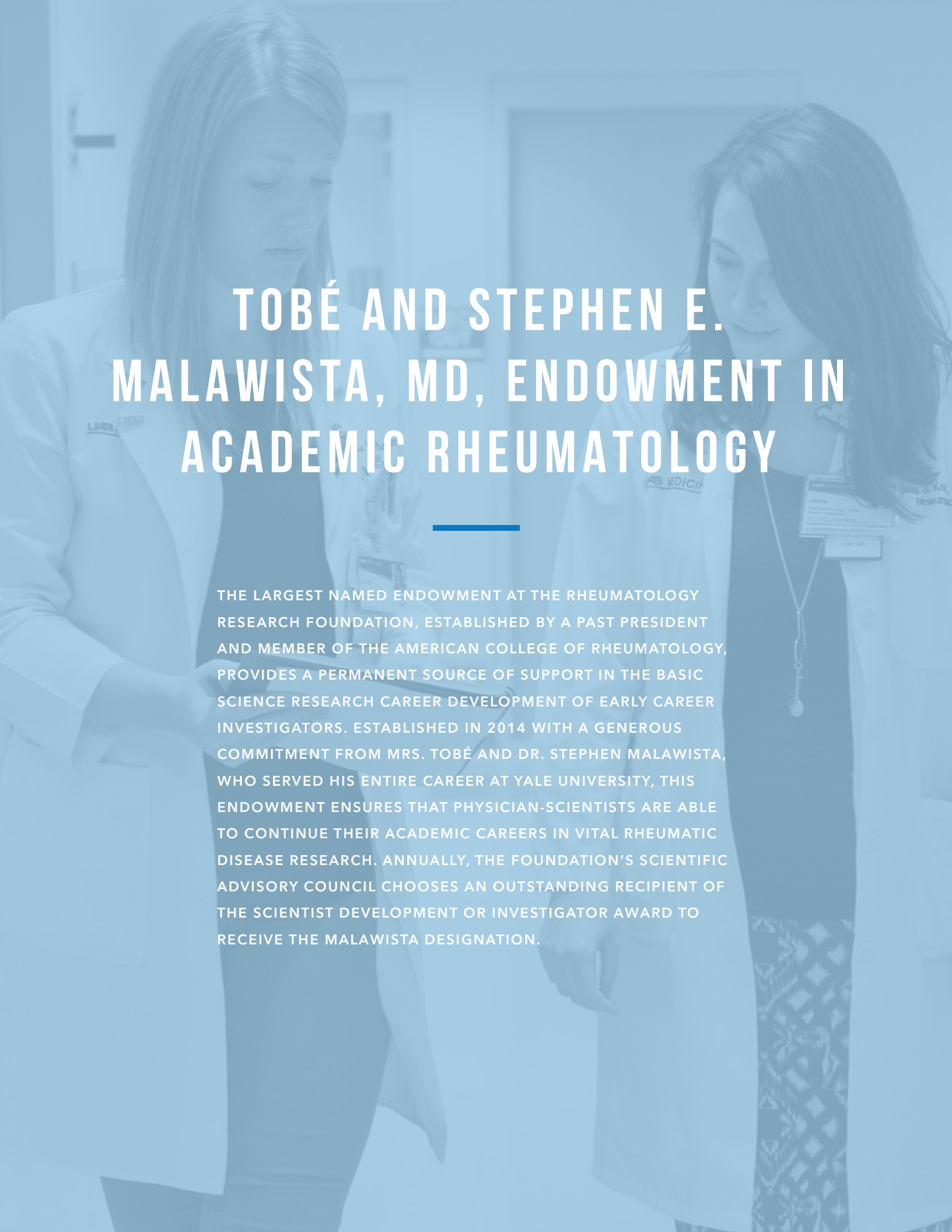
# KATHERINE D. WYSHAM, MD

UNIVERSITY OF CALIFORNIA, SAN FRANCISCO

## **DETERMINING THE ROLE OF DISEASE ACTIVITY ON BONE MASS IN A LONGITUDINAL COHORT OF PATIENTS WITH RHEUMATOID ARTHRITIS**

Patients with RA demonstrate reduced bone mineral density (BMD) and increased risk of osteoporotic fracture. Osteoporotic fractures produce morbidity and, especially in the case of hip fractures, loss of independence and increased mortality. The contribution of RA disease activity vs. other traditional risk factors for osteoporosis (OP) (e.g., estrogen deficiency, glucocorticoid therapy, inactivity) has not been determined. Such knowledge would enhance the ability of rheumatologists to identify and target those patients at highest risk for fracture and intervene effectively, with lifestyle and pharmacologic interventions, as well as to consider tailoring therapy for the underlying RA. Using the UCSF RA Cohort, the proposed study will test the hypothesis that increased RA disease activity negatively impacts bone mass, thereby increasing fracture risk. Analyses will determine the associations between clinical and biochemical measures of disease activity and BMD and identify potential biomarkers for low BMD in RA patients. A strength of the proposal is that we will use three validated measures of disease activity assessed over time, comparing them to BMD measurements in a large well-characterized patient cohort. Although prior studies have evaluated the association between disease activity and BMD in RA patients, none has controlled for the dynamic nature of disease activity over time as we will do. Another novel aspect of this proposal is the use of a biochemical measure of disease activity, the Vectra-DA, which has never been evaluated in this context. Analyses will also explore the association between the individual serum biomarkers that comprise the Vectra-DA and BMD. This study will set the foundation for future studies to develop and test models for predicting fracture risk. Improved risk prediction will help reduce morbidity and mortality due to OP fractures in this high-risk population.





# TOBÉ AND STEPHEN E. MALAWISTA, MD, ENDOWMENT IN ACADEMIC RHEUMATOLOGY

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THE LARGEST NAMED ENDOWMENT AT THE RHEUMATOLOGY RESEARCH FOUNDATION, ESTABLISHED BY A PAST PRESIDENT AND MEMBER OF THE AMERICAN COLLEGE OF RHEUMATOLOGY, PROVIDES A PERMANENT SOURCE OF SUPPORT IN THE BASIC SCIENCE RESEARCH CAREER DEVELOPMENT OF EARLY CAREER INVESTIGATORS. ESTABLISHED IN 2014 WITH A GENEROUS COMMITMENT FROM MRS. TOBÉ AND DR. STEPHEN MALAWISTA, WHO SERVED HIS ENTIRE CAREER AT YALE UNIVERSITY, THIS ENDOWMENT ENSURES THAT PHYSICIAN-SCIENTISTS ARE ABLE TO CONTINUE THEIR ACADEMIC CAREERS IN VITAL RHEUMATIC DISEASE RESEARCH. ANNUALLY, THE FOUNDATION'S SCIENTIFIC ADVISORY COUNCIL CHOOSES AN OUTSTANDING RECIPIENT OF THE SCIENTIST DEVELOPMENT OR INVESTIGATOR AWARD TO RECEIVE THE MALAWISTA DESIGNATION.

# EVAN D. NAIR-GILL, MD, PHD

UT SOUTHWESTERN MEDICAL CENTER

## **ROLE OF PHOSPHOFURIN ACIDIC CLUSTER SORTING PROTEIN-1 (PACS-1) IN B CELL FUNCTION**

Signals from the B cell receptor (BCR) guide B cell selection and determines their functionality, longevity, and self-tolerance. Identifying new genes that control BCR signaling informs B cell behavior in normal and autoimmune responses and provides potential targets for treating immune-related diseases. Using forward genetic screening, we identified phosphoacidic cluster sorting protein-1 (Pacs-1) as a new regulator of B cell homeostasis in mice. Targeted deletion of Pacs-1 (Pacs-1 KO) results in peripheral B cell deficiency, and Pacs-1 KO mice have evidence of a developmental block at the pro- to pre-B transition. Mature B cells show blunted calcium flux after BCR crosslinking and demonstrate attenuated activation of Syk, a key transducer of proximal BCR signals. Pacs-1 is a Golgi sorting protein that connects membrane cargo to the intracellular trafficking machinery. Its role in lymphocyte function and antigen receptor signaling is currently unknown. We hypothesize that (A) Pacs-1's role in intracellular trafficking is important for organizing the proximal BCR signaling complex and (B) that the signaling defect in Pacs-1 KO B cells leads to defective humoral responses to weak antigens. We will test these hypotheses with the following experimental specific aims: (1) Define the signaling defect in Pacs-1 KO B cells by measuring early events after BCR stimulation, including BCR clustering and phosphorylation of downstream signaling proteins. (2) Identify Pacs-1 cargo in B cells using both candidate screening and exploratory approaches and validate that putative cargo participate in BCR signaling in a Pacs-1-dependant manner. (3) Employ a unique BCR-transgenic system on the Pacs-1 KO background that enables antigenic titration to dissect Pacs-1's role in humoral responses to strong versus weak antigens. These studies will provide insight into a novel control mechanism of antigen receptor signaling and may reveal new therapeutic targets to treat abnormal B cell activation in autoimmunity.



# EDUCATION AND TRAINING AWARDS

Building the rheumatology workforce in attempts to satisfy the growing demand for rheumatologists and rheumatology health professionals requires robust education and training opportunities. The Education and Training Awards help cultivate future generations of rheumatology professionals and ensure that people with rheumatic diseases have access to the care they need.



A background image of a scientist with long hair and a beard, wearing a white lab coat and black gloves, is shown in profile. He is focused on his work, using a pipette to transfer liquid into a small vial. The laboratory setting is visible in the background, with various pieces of equipment and glassware. The entire image has a light blue overlay.

# CLINICIAN SCHOLAR EDUCATOR AWARD

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THE CLINICIAN SCHOLAR EDUCATOR AWARD SUPPORTS EDUCATORS DEDICATED TO DEVELOPING NEW AND IMPROVED PROGRAMS TO ENHANCE EDUCATION IN MUSCULOSKELETAL AND RHEUMATIC DISEASES FOR FUTURE PHYSICIANS AND RHEUMATOLOGY HEALTH PROFESSIONALS.



# JANE S. KANG, MD, MS

COLUMBIA UNIVERSITY

## **AN INTERACTIVE, CASE-BASED, ONLINE ETHICS CURRICULUM FOR RHEUMATOLOGY FELLOWS: ISSUES IN INDUSTRY INTERACTIONS AND INDUSTRY FUNDED TRIALS**

Ethical issues are a concern of the American College of Rheumatology (ACR) membership, and there is a need for educational programs that will help rheumatologists address these ethical matters. Since rheumatologists feel they need more knowledge and tools to assess bioethical issues they encounter, improving their knowledge of bioethics in order to assess ethical considerations is essential. In particular, physician-industry relationships are a significant concern of ACR members.

Rheumatology fellows need ethics education to prepare them for industry-related interactions after fellowship. And yet, during fellowship, formal education on these particular ethical dilemmas in rheumatology is usually absent or minimal. We should give our trainees the tools to organize their thoughts within a bioethical framework, so they have a systematic, focused, and informed way of approaching ethical issues pertaining to industry-related interactions and industry-funded clinical research.

The aim for this project is to develop an interactive case-based online ethics curriculum for rheumatology fellows, so they can (1) describe and recall major bioethical principles and (2) apply these principles to analyze and assess bioethical issues in (a) industry-related interactions, (b) industry-funded rheumatology clinical research, and (c) their own clinical cases and research projects. Specifically, industry-related interactions and industry-funded research in rheumatology brings to light certain bioethical considerations such as the goals of clinical research vs. patient care, conflicts of interest, the use of placebo and subtherapeutic dosing, and the recruitment of disadvantaged or vulnerable human research subjects.

This curriculum would be the first formalized ethics curriculum for rheumatology fellows. To encourage knowledge retention and application, the curriculum supports active learning through the use of an interactive, case-based, online ethics curriculum. The curriculum starts with knowledge acquisition, with later modules requiring fellows to recall these principles to analyze and evaluate bioethical matters in rheumatology.



# JAY MEHTA, MD

CHILDREN'S HOSPITAL OF PHILADELPHIA

## **CONSTRUCTING A CAREER IN PEDIATRIC RHEUMATOLOGY: RESEARCH INFORMING PRACTICE**

Very little is known about how early career pediatric rheumatologists make decisions about their careers, which makes it difficult to develop programs to advise and support them. Broadly, clinical rotations and mentors are influential to those who decide to pursue rheumatology. However, pediatric rheumatology remains an unpopular field, contributing to a worsening national shortage of pediatric rheumatologists. Additionally, early career pediatric rheumatologists often suffer from a lack of mentorship and local role models, adding to their baseline concerns about job stability. Some data suggest that satisfaction with a career in rheumatology is decreasing. A major obstacle to reversing some of these trends is that workforce data derived from national surveys only provide general patterns but do not provide insight into the decision-making process that individual early career physicians go through. For example, just knowing there is a lack of mentorship does not tell us what kind of mentorship an individual resident or fellow might find most helpful. Another obstacle is a lack of formal training in career counseling for most fellowship training directors.



To further our understanding of these issues, I will use a qualitative research methodology, known as constructivist grounded theory, and conduct a series of in-depth, semi-structured interviews with pediatrics residents, pediatric rheumatology fellows, and early pediatric rheumatology attendings in order to understand what factors are influential in the career decision-making process and group these into a set of life themes based on career construction theory. Ultimately, I aim to shed light on individual and social factors that impact how early career rheumatologists navigate their careers. The long-term goal of this work is to use these findings to inform recruitment strategies for medical students and residents and to create a framework for counseling early career pediatric rheumatologists. While qualitative methodologies have been used extensively in other career counseling settings, there is little published on this approach in medical specialties. I anticipate this work serving as a model for career advisement in adult rheumatology, as well as other pediatric subspecialties and will allow me to gain expertise as a medical education qualitative researcher.

# REBECCA ELI SADUN, MD, PHD

DUKE UNIVERSITY

## **MIND THE GAP: TRAINING RHEUMATOLOGY FELLOWS IN TRANSITION AND TRANSFER SKILLS TO ENHANCE THE CARE OF ADOLESCENT AND YOUNG ADULT PATIENTS**

The transition from pediatric to adult healthcare is a vulnerable time for adolescents and young adults (AYA) with chronic conditions, including those with pediatric-onset rheumatologic diseases. Education innovation can improve AYA care by equipping rheumatologists with transition and transfer best practice skills and teaching rheumatologists how to use existing transition resources. Dr. Sadun's proposal involves the design, implementation, and evaluation of three curricula for use in training both adult and pediatric rheumatology fellows in key transition and transfer skills. Specifically, a longitudinal and experiential, a series of interactive web-based modules, and a stand-alone lecture will all be developed, piloted, and rigorously evaluated for their ability to increase rheumatology fellows' confidence in transition skills, performance on an objective structured clinical examination (OSCE) transition station, and feedback on 360-degree evaluations. The OSCE and other transition assessment tools herein described are all novel tools for the evaluation of physicians' transition skills and will be validated as part of this project. Ultimately, this suite of educational materials will enable rheumatologists to better utilize the ACR's "transition toolkit," improve the healthcare received by young adult rheumatology patients, and enable rheumatology to lead the way in AYA transition care education.





# FELLOWSHIP TRAINING AWARD FOR WORKFORCE EXPANSION

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THE FELLOWSHIP TRAINING AWARD FOR WORKFORCE EXPANSION SUPPORTS THE TRAINING OF A RHEUMATOLOGY FELLOW AT AN INSTITUTION THAT HAS PREVIOUSLY BEEN UNABLE TO FILL ALL OF THEIR ACGME-APPROVED SLOTS DUE TO FUNDING CONSTRAINTS IN AN EFFORT TO ENSURE AN ADEQUATE SUPPLY OF RHEUMATOLOGY PROVIDERS IN ALL AREAS OF THE COUNTRY.

2018 AWARD RECIPIENT

**VANDERBILT UNIVERSITY**

# FELLOWSHIP TRAINING AWARDS

THE FELLOWSHIP TRAINING AWARD SUPPORTS THE TRAINING OF RHEUMATOLOGY FELLOWS TO PROVIDE A MORE ROBUST AND HIGHLY TRAINED WORKFORCE TO CARE FOR PEOPLE WITH RHEUMATIC DISEASES.

**Baylor College of Medicine**  
Fellowship Training Award

**Brigham and Women's Hospital**  
Fellowship Training Award

**Case Western Reserve University**  
Fellowship Training Award

**Cincinnati Children's Hospital Medical Center**  
Fellowship Training Award

**Duke University**  
Fellowship Training Award

**Georgetown University**  
Amgen Fellowship Training Award

**Johns Hopkins University**  
Paula de Merieux Fellowship Training Award  
*Funding for this award is made possible through an endowment from the estate of Paula de Merieux, MD*

**Massachusetts General Hospital**  
Amgen Fellowship Training Award

**New York University**  
Amgen Fellowship Training Award

**Oregon Health and Science University**  
Amgen Fellowship Training Award

**Stanford University**  
Fellowship Training Award

**The Children's Hospital of Philadelphia**  
Amgen Fellowship Training Award

**Tufts Medical Center**  
Amgen Fellowship Training Award

**University of Alabama at Birmingham**  
Amgen Fellowship Training Award

**University of California, Los Angeles**  
Fellowship Training Award

**University of California, San Diego**  
Amgen Fellowship Training Award

**University of California, San Francisco**  
Amgen Fellowship Training Award

**University of Chicago**  
Fellowship Training Award

**University of Michigan**  
Fellowship Training Award

**University of Pennsylvania**  
Fellowship Training Award

**University of Washington/Seattle Children's Hospital**  
Fellowship Training Award

**Washington University in St. Louis**  
Amgen Fellowship Training Award

# PRECEPTORSHIPS

Preceptorships encourage students and residents to learn more about rheumatology and pursue careers in the field by supporting a one-on-one, real-world learning experience.

## RHEUMATOLOGY FUTURE PHYSICIAN SCIENTIST AWARD

THIS AWARD SUPPORTS MD/PHD STUDENTS WHO DEMONSTRATE OUTSTANDING POTENTIAL AND SIGNIFICANT COMMITMENT TO A CAREER IN RHEUMATOLOGY RESEARCH IN AN EFFORT TO SUPPORT THE NATION'S TOP EMERGING PHYSICIAN-SCIENTISTS IN THE FIELD OF RHEUMATOLOGY.

### NICK HUANG

SUNY UPSTATE MEDICAL UNIVERSITY

#### ENDOCYTIC CONTROL OF T-CELL DEVELOPMENT

SLE is an autoimmune disease of unknown etiology that affects 0.1% of the US population with a 10% mortality in 5 to 10 years. The current state of inadequate therapies stems from critical gaps in our understanding of disease pathogenesis and lack of mechanistic therapeutic targets. This project aims to develop new, fundamental knowledge of metabolic pathways that control pro-inflammatory cell type-specific development and identify checkpoints of pathogenesis for targeting new pharmaceutical interventions in lupus.

Metabolism has become pivotal to our understanding of immunological development in the last decade. HRES-1/Rab4A, hereon referred to as Rab4A, is a GTPase that has strong implications in endosomal trafficking, recycling of receptors, and autophagy. Overexpressed in lupus T cells, Rab4A has been linked to disease susceptibility with pulmonary and renal organ involvement. Rab4A's ability to target the mitochondrial fission factor dynamin-related protein 1 for lysosomal degradation establishes itself as a regulator of mitophagy. Preliminary data suggests deficiency in Rab4A drives the mTORC1-dependent expansion of pro-inflammatory TH17 cells and altered mitochondrial metabolism. Additionally, in a pristane model of induced autoimmunity, Rab4A protects from diffuse alveolar hemorrhage, a rare but fatal complication of lupus, and glomerulonephritis. Blocking activation of mTORC1 with rapamycin and N-acetylcysteine shows clinical efficacy in patients with lupus. Therefore, revealing how of Rab4A controls the metabolic pathways and mTORC1 activation will not only provide insight into disease etiologies and pathogenesis but also establish the foundations for therapeutic drug development.



## EPHRAIM P. ENGLEMAN ENDOWED RESIDENT RESEARCH PRECEPTORSHIP

THE EPHRAIM P. ENGLEMAN ENDOWED RESIDENT RESEARCH PRECEPTORSHIP ENCOURAGES RESIDENTS TO CONSIDER A CAREER IN RHEUMATOLOGY RESEARCH BY SUPPORTING A FULL-TIME, IN-DEPTH RESEARCH EXPERIENCE WITH AN ESTABLISHED RHEUMATOLOGY PROFESSIONAL. FUNDING FOR THIS AWARD IS MADE POSSIBLE THROUGH AN ENDOWMENT FROM DR. EPHRAIM P. ENGLEMAN.

### JULIE M. CHASE, MD, PHD

PRECEPTOR: LESLIE J. CROFFORD, MD  
VANDERBILT UNIVERSITY MEDICAL CENTER

#### **ENHANCING IMMUNE THERAPY FOR SLE THROUGH METABOLIC CONDITIONING**

SLE is a life-threatening systemic autoimmune illness affecting nearly every organ system. While disease-related mortality has greatly improved with treatment advances, significant morbidity persists due in part to increased infection from systemic immune suppression and treatment-associated drug toxicities. There remains a continued need for more effective therapeutic regimens, and one attractive strategy is that of conditioning immune cells to increase their sensitivity to existing therapies. This approach could in theory render previous treatment failures successful and may allow for decreased immunosuppression, potentially reducing incidence of infection and treatment-related toxicity.

In considering synergistic strategies for the treatment of lupus, it has recently been discovered that lupus effector T cells (Teff) exhibit hyperactive metabolism. We hypothesize that this hyperactive metabolism contributes to resistance to current immune therapies and may account for some treatment failure. This hypothesis is supported by emerging data from the field of immunometabolism, which indicate that metabolic dysregulation of T cells fosters the pathogenesis of autoimmune disease and that normalization of metabolic pathways via administration of agents such as metformin and 2-DG results in prevention of disease in a murine lupus model.

In the current project, we seek to determine the therapeutic potential for synergism between metabolic conditioning and traditional immunosuppressive therapies, specifically utilizing a well-established murine lupus model system (SLE.123). We hypothesize that normalization of Teff metabolism will render these cells more sensitive to immune suppression, increasing the efficacy of currently available treatments including mycophenolate mofetil, cyclosporine A, and anti-CTLA4 (abatacept). These studies will illuminate the interaction between metabolism and immune therapy and will extend our knowledge on the role of metabolism in the generation of durable tolerance. Furthermore, this research will evaluate a novel, feasible and clinically relevant alternative therapeutic strategy for the treatment of lupus.





## LAWREN H. DALTROY HEALTH PROFESSIONAL PRECEPTORSHIP

THE LAWREN H. DALTROY AWARD WAS DESIGNED TO IMPROVE PATIENT-CLINICIAN INTERACTIONS THROUGH THE DEVELOPMENT OF A MORE QUALIFIED AND TRAINED HEALTH PROFESSIONAL WORKFORCE. FUNDING FOR THIS AWARD IS MADE POSSIBLE IN PART THROUGH THE RHEUMATOLOGY RESEARCH FOUNDATION AND THROUGH AN ENDOWMENT FROM RHEUMINATIONS, INC.

## KATHLEEN C. MADARA, DPT

PRECEPTOR: JOSEPH ZENI, PT, PHD  
UNIVERSITY OF DELAWARE

### MEASURING EXPECTATIONS IN PATIENTS AWAITING TOTAL KNEE REPLACEMENT

Total knee arthroplasty (TKA) has become the standard procedures to manage end-stage knee osteoarthritis, but research into the expectations, rehabilitation, and outcomes of younger patients is lacking. The purpose of this study is to test the reliability of a new expectation questionnaire and evaluate expectations in a contemporary sample of patients awaiting total knee replacement. 100 subjects will be recruited from the Christian Care Center for Advanced Joint Replacement. Patients will complete expectation questionnaires at the pre-operative and 6-month post-operative time point. The first questionnaire measures patient's expectation of recovery after surgery. The second questionnaire is a satisfaction questionnaire, which will measure patient satisfaction across multiple domains 6 months after surgery. In this study, we will (1) assess the test-retest reliability of the expectation questionnaire, (2) identify factors that correlate to higher expectations, and (3) evaluate if satisfaction at 6 months after surgery is related to pre-operative expectations. Results from this study will provide important insight into the expectations of patients awaiting TKA and provide clinicians with a unique and easy tool to evaluate pre-operative expectations.

There is a disconnect between patient needs, patient expectations, clinical recommendations, and patient education for individuals with osteoarthritis. This project will elucidate factors related to patient expectations before and after joint replacement in order to improve patient-clinician communication and ultimately patient outcomes.



A person wearing a white lab coat and white gloves is using a pipette to transfer liquid into a small vial. The background is a blurred laboratory setting with various equipment and containers. The entire image has a light blue overlay.

# RESIDENT RESEARCH PRECEPTORSHIP

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THE RESIDENT RESEARCH PRECEPTORSHIP INTRODUCES  
RESIDENTS TO THE SPECIALTY OF RHEUMATOLOGY AND  
ATTRACTS PROMISING PHYSICIAN-SCIENTISTS TO THE FIELD BY  
SUPPORTING A FULL-TIME RESEARCH EXPERIENCE.

# EMILY BOWERS, MD

PRECEPTOR: KRISTEN DEMORUELLE, MD  
UNIVERSITY OF COLORADO SCHOOL OF MEDICINE

## **THE INFLUENCE OF RESPIRATORY INFECTION ON ANTI-CCP GENERATION IN THE LUNG**

It is well established that anti-citrullinated protein antibodies (ACPA) can be elevated in the blood years prior to the onset of inflammatory arthritis in rheumatoid arthritis during a 'preclinical' period of RA development. The presence of systemic ACPA prior to the onset of RA suggests that ACPA are initially generated at a site outside of the joints, and several lines of evidence support that the lung may be one potential site where ACPA originates. Our group has previously demonstrated through induced sputum testing that ACPA, as characterized by anti-cyclic citrullinated peptide (CCP) antibodies, are generated in the lung in a portion of subjects determined to be 'at risk' for future RA. However, it is unknown what factors in the lung could trigger the generation of these sputum antibodies.

Bacterial and viral infections have long been speculated to play a role in the development of RA. However, the role of infection on ACPA generation in the lung has not been well studied. Therefore, our central hypothesis is that acute lung infection is one mechanism that can trigger ACPA generation in the lung. To study this hypothesis, we will collect sputum samples from adults without RA who are hospitalized for acute pulmonary infection (both viral and bacterial) as well as controls. We will test sputum for anti-CCP antibodies and compare sputum antibody levels between subjects with pulmonary infection and controls, as well as between subjects with viral and bacterial pulmonary infections. Our working hypothesis is that sputum anti-CCP levels will be higher in subjects with acute pulmonary infection. These findings will provide further insight into potential mechanisms by which inhaled factors could lead to autoimmunity in RA.



# NICOLE DECREDICO, MD

PRECEPTOR: MONIQUE HINCHCLIFF, MD, MS  
NORTHWESTERN UNIVERSITY - MCGAW MEDICAL CENTER  
**INTERSTITIAL LUNG DISEASE AND SYSTEMIC SCLEROSIS**

Interstitial lung disease (ILD) is present in  $\geq 90\%$  of systemic sclerosis (SSc) patients and is the leading cause of SSc-related mortality. Our study aims to group pulmonary function test (PFT) trajectories in a large number ( $>700$ ) of SSc patients and identify their associated clinical covariates. Specifically, we will look at patient characteristics (age, sex, body mass index, disease subtype, etc) as well as the presence of autoantibodies and inflammatory markers to determine if these characteristics are predictive of a specific PFT trajectory. This knowledge will inform ILD screening and treatment protocol development.

In addition, we will examine PFT trajectories between SSc patients with normal versus reduced forced vital capacity (FVC) and diffusion capacity of carbon monoxide (DLCO) at baseline, adjusting for important clinical covariates including immune suppression. Studies exist demonstrating the effect of immune suppression, including mycophenolate mofetil and cyclophosphamide on preventing decline and/or improving PFT trajectory in patients with SSc. However, immunosuppressive treatments are often not initiated in SSc patients until clinically evident ILD is apparent as assessed by FVC decline. We hypothesize that early initiation of immune suppression may improve/stabilize lung function in patients with normal lung function at the baseline visit. Patients with SSc in our cohort will be dichotomized into those with normal vs. reduced PFT ( $FVC > 80$  vs  $FVC \leq 80$  and  $DLCO > 60\%$  vs  $\leq 60\%$ ) and between those who are prescribed, and are not prescribed, immune suppression, paying careful attention to clinical indication for immune suppressive therapy. We will examine PFT trajectory among these subgroups. This analysis will determine if early initiation of immune suppression prior to development of abnormal pulmonary function tests is beneficial in patients with SSc.



# AYESHA JALEEL, MD

PRECEPTOR: KENNETH SAAG, MD, MSC  
BAPTIST BROOKWOOD HEALTH

## **PRACTICAL USE OF BONE TURNOVER MARKERS (BTMS) IN OSTEOPOROSIS: PATTERNS AND PREDICTORS FOR OSTEOPOROSIS MANAGEMENT AND BISPHOSPHONATE DRUG HOLIDAYS**

Bone homeostasis is a delicate balance between osteoclast and osteoblast activity, and biochemical markers of bone turnover (BTMs) measure the products generated during the formation and resorption of bones. BTMs can be used to monitor treatment efficacy and patient adherence before BMD changes would be available via bone density. I will be conducting a retrospective study to analyze how physicians in a multi-disciplinary osteoporosis clinic at University of Alabama in Birmingham used BTMs in real-world clinical practice and how it impacted clinical decision making and patient outcomes (re-initiation of therapy, BMD, and fractures). The BTMs to be examined include either urine N-terminal (NTX) cross-linking telopeptides, serum C-terminal cross-linking telopeptides, or bone alkaline phosphatase. I will determine the range of the BTM (low, normal, or high), and subsequent osteoporosis treatment patterns, BMD results, and fracture history obtained following the diagnosis. Those who have BTMs measured will constitute the “exposed” group and they will be compared to a similarly defined osteoporosis patient group (based on the ICD 9/ICD10 and therapy use) “unexposed to turnover makers”. I will see whether there is any relationship between the BTMs and the subsequent BMD and fractures in the in “exposed” patients. Patients will be further divided into IV vs oral therapy and compared based on age, sex, BMDs on DXA, and FRAX score. Patients with stage IV or V chronic kidney disease, liver disease, active cancer, Paget’s disease, and those who had to stop bisphosphonate therapy due to side effects will be excluded. Time to re-initiate osteoporosis therapy after drug holiday will be our primary outcome and will be measured in both groups. A biostatistician will guide the ACCESS or Redcap database build process, provide oversight on data analysis, and contribute to scientific report preparation. This preceptorship will allow me to gain valuable experience working with EHR data, performing clinical data acquisition from medical records, managing research data, analyzing, and preparing reports.



# RENÉE PETERKIN-MCCALMAN, MD, MPH

PRECEPTOR: LAURA CARBONE, MD

MEDICAL COLLEGE OF GEORGIA AT AUGUSTA UNIVERSITY

## **ASSOCIATION OF NIACIN AND NICOTINAMIDE USE WITH SKIN CANCER AND OSTEOPOROSIS IN RHEUMATOID ARTHRITIS**

Patients with RA are at increased risk for skin cancer, particularly those using biologics, with rates for melanoma, squamous cell, and basal cell cancers all increased. Despite this, because these drugs are so effective, many patients with a history of nonmelanomatous skin cancers are still prescribed biologics. Additionally, patients with RA are at an increased risk for osteoporotic fractures relative to similarly matched controls without RA.

Recently, nicotinamide, a derivative of niacin, has shown efficacy for preventing squamous cell, basal cell cancer, and actinic keratosis in patients with a prior history of nonmelanomatous skin cancer. Early data in renal transplant patients, a high-risk population for these cancers, also looks promising. However, studies of its use in RA patients, including those prescribed biologics, have not been done. Outside of clinical trials, nicotinamide is not widely used. Niacin, though, is prescribed for hyperlipidemia, a prevalent comorbidity in patients with RA.

The relationship of niacin or nicotinamide to osteoporotic fractures has not previously been reported. Importantly, these compounds can reduce inflammatory cytokines and it has been reported that patients with pellagra are at risk for osteoporosis.

The purpose of this epidemiologic study is to examine the association of niacin/nicotinamide use with the actinic keratosis and nonmelanomatous skin cancers and fractures in patients with RA. If study findings are positive, this would support my future plans for a randomized controlled trial using these agents to prevent both skin cancer and osteoporosis in patients with RA.



# ISAAC DAVID SMITH, MD

PRECEPTORS: MARCY B. BOLSTER, MD, AND HYON CHOI, MD, DRPH  
MASSACHUSETTS GENERAL HOSPITAL

## **THE IMPACT OF ALCOHOL USE ON MYOCARDIAL INFARCTION AND ALL-CAUSE MORTALITY IN PATIENTS WITH RHEUMATOID ARTHRITIS**

RA is a systemic inflammatory condition associated with an increased risk of cardiovascular disease and premature mortality. Many prospective population studies have consistently found that moderate alcohol consumption is associated with a 25-40% reduced risk for coronary heart disease and death. As such, the American Heart Association suggests that “if you drink alcohol, do so in moderation.” These potential cardiovascular and survival benefits of alcohol consumption in moderation may be highly relevant for patients with RA. However, to date there have been no studies addressing the potential cardioprotective effects of alcohol in this patient population.

Alcohol use has been largely discouraged for patients with RA taking methotrexate (MTX), the dominant first-line therapy for RA, due to potentially enhanced hepatotoxicity risk. However, a recent population-based study found that it may be safe for patients with RA taking MTX to consume a moderate amount of alcohol without a significantly increased risk of hepatotoxicity. Because patients with RA are at increased risk of CHD and premature mortality, it is important to elucidate the potential effects of alcohol use on cardiac disease in this population. The goal of our study is to examine the relationship between alcohol intake and the risk of CHD and all-cause mortality among RA patients, both in a general population context and in those patients with RA who are taking MTX.

We hypothesize that moderate alcohol consumption may be associated with a lower risk of CHD and mortality in both the general RA population and in patients using MTX. We will conduct a cohort study to examine the potential impact of alcohol use on CHD and survival in patients with RA using data from an electronic medical record database (The Health Improvement Network [THIN] database) representative of the UK general population from 1995-2017. The study population will include RA patients enrolled in THIN (N>43,000), including patients with RA taking MTX (N>22,000). Data on alcohol consumption is self-reported by patients at primary care visits, and the endpoints will be incident cases of acute myocardial infarction and all-cause mortality.



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