Rheumatology Research Foundation

Advancing Treatment | Finding Cures

2023 AWARD RECIPIENTS

MESSAGE FROM LEADERSHIP

Dear Foundation friends and supporters,

WELCOME TO OUR NEW AWARD RECIPIENTS

The Foundation is pleased to announce the 2023 class of Award Recipients. Their work is vital to creating a brighter future for the field of rheumatology and for people impacted by rheumatic disease.

We are proud to commit $13,118,575 in fiscal year 2024 (July 1, 2023 - June 30, 2024) to fund this new class of award recipients. Roughly a quarter of these awards will support efforts to recruit and train the next generation of rheumatology professionals, which will reduce patient wait times and increase access to rheumatology care. The remaining funds will be awarded to advance research projects that will lead to breakthroughs in treating people with rheumatic disease.

Central to its mission, the Foundation's leadership has worked to build strong programming. These awards support the gamut of rheumatology by funding recruitment, education and training, research, and career development. In all, the Foundation has committed more than $218 million to the field of rheumatology through more than 4,406 individual research and training awards.

AWARDS PORTFOLIO UPDATES

We are excited to share upcoming changes to our awards portfolio. The Foundation's 4th Portfolio Review Panel (PRP) was convened in 2022 to evaluate the awards program and provide recommendations through the lens of impact, cost, feasibility, and relevance to the mission to the Board of Directors. With their approval, several exciting changes are being incorporated in the request for applications (RFAs) this year and implemented throughout the next year.

MESSAGE FROM LEADERSHIP (CONTINUED)

- Fellowship Training Awards have increased from one year of funding to two years for adult programs and three years for pediatric programs.
- Recipients of the Clinician Scholar Educator award (CSE) will see an increase of funding from $180,000 to $210,000 for the three-year term.
- Expanded award eligibility for individuals holding a faculty position residing in the U.S., regardless of their immigration status.
- New opportunities for awards targeting undergraduate students and those in post-baccalaureate training interested in rheumatology.

Additional recommendations by the Portfolio Review Panel will be considered and implemented over the next three years.

One final exciting program addition in 2023, is our new FOREUM/RRF Partnership Award to support international (U.S. and Europe) research collaboration among investigators who wish to pursue a synergistic, joint project in rheumatic disease research. This new program is based around the Foundation's strategic goal to increase collaborative research.

There has never been a brighter time for the future of rheumatology. This future would not be possible without the continued dedication and support of our donors, to whom we are most grateful. I am pleased to share with you the latest class of award recipients.

Sincerely,

Ted Mikuls, MD, MSPH
Chair, Scientific Advisory Council
Rheumatology Research Foundation

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Innovative Research Award

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.

INNOVATIVE RESEARCH AWARD

Aileen Chang, MD

The George Washington University

Determination of the Human Mechanisms of Interleukin-2 Therapy for Chronic Post-Chikungunya Viral Arthritis

Chikungunya virus (CHIKV) causes persistent arthritis in one-fourth of patients and there is currently no standard treatment. In this proposal, we will determine the mechanisms of novel interleukin (IL)-2 therapies for CHIKV arthritis using samples banked from CHIKV arthritis patients. Our preliminary data suggest that decreased regulatory T cell (Treg) function in CHIKV arthritis may be ameliorated by novel low-dose IL-2 therapies. Our main goal is to examine how several potential IL-2 therapies, including low-dose IL-2, IL-2 monoclonal antibody (mAb) and IL-2/IL-2 mAb complex, affect Treg number and function ex-vivo in samples derived from CHIKV-induced chronic arthritis patients in order to define the human mechanisms of IL-2 therapy in this population. Our central hypothesis is that treatment with IL-2/IL-2mAb complex will significantly increase Treg immunosuppressive function compared to IL-2 or IL-2 mAb alone or control in samples in CHIKV arthritis and that the mechanism is through enhanced FOXP3-mediated CTLA-4 suppression of Teff cells.

In Aim 1, we will determine the frequency of T cell subsets and the best IL-2 therapy to optimize the percentage of cells expressing Treg functional markers in peripheral blood mononuclear cells (PBMCs) from CHIKV arthritis patients using an in vitro model. In Aim 2, we will determine the best IL-2 therapy to optimize Treg function in CHIKV arthritis patient samples in an in vitro T cell suppression assay. In Aim 3, we will determine the change in immunosuppressive cytokines expressed by Tregs from CHIKV arthritis patients before and after incubation with IL-2 therapies vs. control and identify potential cytokine biomarkers that can predict response to IL-2 therapies. Impact: This work will define for the first time the human mechanisms of IL-2 therapies in chronic CHIKV arthritis providing valuable preclinical data for a novel therapy for viral arthritis and identification of specific endpoints for clinical trial study.

INNOVATIVE RESEARCH AWARD

Christina Charles-Schoeman, MD

University of California, Los Angeles

Investigating the Role of Paraoxonase-1 in Rheumatoid Arthritis

Our understanding of what drives the gradual development of rheumatoid arthritis (RA) from the first triggering of autoimmunity and the development of anti- citrullinated protein antibodies ACPA) to the robust immune response and clinical arthritis activity is limited. Better understanding of this process would allow not only more accurate biomarkers for early detection, but also novel, alternative preventative approaches and treatments. Smoking is the strongest known environmental risk factor for RA, particularly in ACPA positive patients, and recent data also strongly implicates air pollution in the development and flares of RA. Both of these environmental triggers promote oxidative stress, which is defined as an imbalance between the production and accumulation of free radicals and reactive metabolites, generally known as “reactive oxygen species” (ROS) in cells and tissues. Paraoxonase-1 (PON1) is a major high-density lipoprotein (HDL) associated protein, which normally neutralizes inflammatory, oxidized lipids, and thereby facilitates HDL’s role in regulation of systemic oxidative stress.

We hypothesize that suppression of the PON1 activity, which can occur from known RA triggers, drives the acceleration of systemic oxidative stress in the “pre-RA” stage, triggering clinical disease development. This project aims to study 1) the association of PON1 activity with the development of RA in a prospective cohort of ACPA positive patients, 2) the distribution of the PON1Q192R polymorphism in ACPA positive patients who develop RA compared to ACPA positive patients who do not develop RA, and 3) the ability of an oral PON1-inducing agent to reduce RA onset and arthritis severity in an animal model. The goal of this project is to understand the role of PON1 in the development of RA, which may provide novel biomarkers for RA risk assessment as well as novel targets for preventive and therapeutic approaches.

INNOVATIVE RESEARCH AWARD

Monica Guma, MD

University of California, San Diego

Bile Acid Profiling Predicts Response to an Anti-inflammatory Diet in Patients with Osteoarthritis

Osteoarthritis (OA) is the most common form of arthritis. Currently, there is no effective medical disease modifying therapy available for OA. A diet-induced weight loss intervention improved physical function and pain. Yet, losing and specially maintaining weight loss is challenging. We propose that a novel, distinct nutritional intervention to decrease pain independent of weight loss might represent a more realistic dietary approach for those suffering from OA.

Increasing evidence indicates that OA progression is mediated not simply by progressive degeneration of cartilage, but also by associated low-grade synovial inflammation that is associated with increased symptoms in knee OA (KOA). Prior randomized controlled clinical trial (RCT) evidence supported beneficial effects of anti-inflammatory micronutrients on pain and physical function in KOA. Here, we will take on major unmet needs for interventional trials of anti-inflammatory diet without weight loss in OA, and longitudinally assess functional and mechanistic endpoints to identify patients with KOA that are most likely to respond to dietary changes. Recent paradigm-shifting studies suggest the gut microbiome-derived pro- and anti-inflammatory metabolites contribute to pain and inflammation in KOA. Our scientific premise is that gut microbiome changes via a specific anti-inflammatory (ITIS) diet intervention will diminish articular inflammation and pain symptoms in KOA by modulating circulating gut microbiome-derived bile acids (BA).

In this new application, we will conduct an evaluator blinded RCT in patients with KOA to test the central hypothesis that the ITIS diet, even without weight loss, will significantly improve both clinical and functional outcomes in KOA. This RCT will randomize 72 patients with unilateral KOA with a visual analogue pain knee score of between 20-80 during the last 7 days to either receive control diet (Dietary Guidelines for Americans), or ITIS diet intervention for 12 weeks. We will collect blood and fecal samples at multiple visits to understand the anti-inflammatory effects mechanistically over time from a microbiome and metabolome perspective. We hypothesize that introduction of the ITIS diet will significantly improve pain scores and physical function after 3 months of intervention compared to the control group, and that changes in gut microbiome-derived BA will contribute to the clinical changes.

INNOVATIVE RESEARCH AWARD

S. Reza Jafarzadeh, PhD

Boston University

Novel Bayesian Approaches to Identify Persons with Osteoarthritis in Administrative Data in the Absence of a Reference Standard

Accurate characterization of osteoarthritis, the most prevalent form of arthritis and a leading cause of disability, in administrative health data will enable researchers to study its public health burden, economic impact, cost of care, inequities in care, adherence of care to guidelines, and attendant comorbidities. Existing case-finding algorithms for osteoarthritis have had limited success, often missing more than half of cases, mainly due to widespread under-reporting of osteoarthritis diagnostic labels and the fact that guidelines discourage acquisition of imaging that provides a definitive diagnosis.

This research project introduces novel Bayesian approaches to accurately identify persons with osteoarthritis, using multiple data elements from electronic health records and administrative health data. Our goal is to bring new insights into osteoarthritis research by facilitating observational and data-driven studies of disease risk factors and treatments that ultimately will benefit persons affected by osteoarthritis.

INNOVATIVE RESEARCH AWARD

J. Michelle Kahlenberg, MD, PhD

University of Michigan

The Role of Melanocytes in Cutaneous and Systemic Lupus

The mechanisms that regulate immune activation in the skin of systemic lupus erythematosus (SLE) patients are not well understood. Our preliminary data has revealed that one of the most dramatically affected cell populations in lesional and non-lesional skin of SLE patients is the melanocyte. Melanocytes reside in the basal epidermis, and following UV exposure, they utilize various stress response pathways to increase the synthesis of melanin and promote cellular survival. However, in SLE skin, our data support education of melanocytes by type I interferons (IFN) and upregulation of major histocompatibility complex (MHC) class I, class II, and CD74, suggesting that melanocytes in SLE may function as antigen presenting cells. Similar findings in melanocytes primed with IFN? have been shown in vitiligo in which melanocytes can present antigen to CD4+ T cells with CD2 and ICAM-1 help. However, unlike vitiligo, we do not find increased IFN?-regulated CXCR3 expression in lesional or non-lesional lupus melanocytes, suggesting that melanocytes are not undergoing apoptosis and that this phenotype may be driven by type I IFNs, which are produced by keratinocytes in non-lesional SLE skin. We thus hypothesize that keratinocyte-derived type I IFNs educate melanocytes in SLE skin to become pro-inflammatory and capable of T cell activation, and this can be exacerbated by UV light exposure which provides a source of antigen. This project will thus investigate two aims to address this hypothesis.

First, we will examine the antigen presentation phenotype of SLE melanocytes and the role of type I IFNs and other keratinocyte-mediated factors in their activation. Second, we will define the impact of UVB on melanocyte populations in SLE and HC skin in vivo. When completed, the proposal will identify melanocytes as a relevant and important population with immune function in SLE skin, possibly serving as a bridge to T cell activation, especially after UV exposure. Further, this award will give us new insights into what causes the skin to be so prone to inflammation in systemic lupus erythematosus (SLE) patients. These insights will help us to find new and better treatments to prevent skin and systemic inflammation in SLE.

INNOVATIVE RESEARCH AWARD

Deepak Kumar, PhD*

Boston University

A Multimodal High-Tech/High-Touch Intervention to Address Racial Inequalities in Management of Chronic Knee Pain and Depressive Symptoms

Black adults with knee osteoarthritis (OA) experience more severe pain and greater mobility impairments compared to non-Hispanic White adults. Black adults with knee OA also experience greater depressive symptoms which partly underlie the disparity in the pain experience. Underutilization of first line exercise interventions and mental health interventions is a key reason for poor management of OA pain in minority populations. Positive Minds-Strong Bodies (PMSB) is a culturally and linguistically appropriate, exercise and mental health intervention for minoritized older adults to improve mood and mobility. However, PMSB was not designed to address the needs of individuals with chronic musculoskeletal pain.

This study proposed to modify PMSB to address the needs of Black adults with knee pain and create PMSB-OA. PMSB-OA will include a community health worker-delivered mental health intervention, physical therapist-delivered telehealth exercise and pain education program, and digital health tools to foster engagement and social support.

Aim 1 will include focus group studies to identify potential barriers to PMSB-OA and modifications of PMSB to create PMSB-OA including optimization of the digital health platform. Aim 2 is a parallel-arm feasibility randomized controlled trial (n=40) of PMSB-OA. Participants will be recruited from Boston neighborhoods that have large minority populations. Participants will be randomized to a 3-month PMSB-OA intervention or usual care arm. Feasibility and acceptability of PMSB-OA including recruitment, adherence, and retention rates will be examined to inform the design of a larger efficacy study. Outcomes related to pain, mood, function, etc. as well as information on social determinants of health (SDOH) will also be collected. This study will inform further modifications to PMSB-OA guided by feedback from participants and interventionists, as well as strategies for sustainability and scalability.

* These funds are available in partnership with AbbVie and UCB, with additional support provided by Bristol Myers Squibb.

INNOVATIVE RESEARCH AWARD

Richard Loeser, MD

University of North Carolina at Chapel Hill

Small Molecule Discovery for Osteoarthritis Disease Modification

The goal of this project is to discover small molecules that target specific disease pathways to slow or halt the progression of structural damage in OA. The pathobiological processes that promote OA result from activation of cell signaling pathways and subsequent changes in gene transcription mediated by a host of factors. The complexity of OA explains why a single target approach for disease modification has met with limited success. We propose that use of small molecules to target pathways that regulate expression of multiple OA mediators will represent a viable and more promising approach. We have established a robust cell-based high throughput functional screening (HTS) assay using normal human chondrocytes treated with a matrikine relevant to OA progression. We found this system to be a valid in vitro model of the OA chondrocyte phenotype that allows us to investigate multiple pathways and targets, providing a distinct advantage over single target analysis.

Our aims are: Aim 1 Identify OA targets through high throughput cell-based compound screening and network analysis of genetic and genomic datasets. Aim 2 Determine the ability of target-selected compounds to restore the catabolic and anabolic balance in joint tissues; and Aim 3 Perform pre-clinical testing of selected compounds in a mouse model of injury-induced OA. At the completion of this project, we expect to have identified one or more compounds that: 1) restore the balance in the dysregulated chondrocyte catabolic and anabolic activity seen in OA; 2) inhibit production of OA mediators by chondrocytes, synovial fibroblasts, meniscal and potentially other joint tissue cells; 3) act as novel chemical probes to interrogate OA pathways; and 4) demonstrate efficacy in preclinical models of OA. These compounds and their targets would serve as the basis for lead optimization necessary to further develop drugs that would then be tested in future early phase human clinical trials.

INNOVATIVE RESEARCH AWARD

Kaleb Michaud, PhD

University of Nebraska Medical Center

The Cannabis Rheumatology KAP (Knowledge, Attitudes and Practice) and PEACE (Pain, Exercise and Cannabis Experience) Survey

There is an unmet need of finding an adjunct therapy to address chronic pain, disordered sleep, and general well-being in patients with rheumatic diseases – symptoms for which many patients are already taking cannabis and cannabinoids, which preclinical studies have shown the potential to reduce pain. Our study will address this unmet need by examining cannabis patterns of use, benefits, and adverse effects in patients as well as knowledge and attitudes about cannabis in patients and their rheumatologists, which guide behavior and practice. Our study has the two following arms:

1) determine the knowledge, attitudes, and practice (patient interactions) regarding cannabis among members of the American College of Rheumatology (ACR Cannabis Knowledge, Attitudes, Practice (KAP) Survey);

2) determine cannabis use patterns, cannabis subjective effects (positive and adverse), and knowledge and attitudes regarding cannabis in people with rheumatic diseases identified through rheumatology clinics, the Forward Databank registry, and rheumatic disease patient foundations (The Rheumatology Pain, Education, Attitudes and Cannabis Experience (PEACE) Survey).

The results of this study will help uncover physician and patients' biases toward cannabis. In addition, the information gleaned from this study will enable evidence-based prescribing of cannabis medicine to patients by maximizing benefits while reducing harm (i.e. adverse effects). The results of this study will guide the creation of evidence-based educational programs that can be used by physicians to direct patients on if and how to use cannabis safely, thereby improving overall health of people with rheumatic diseases.

INNOVATIVE RESEARCH AWARD

Anne Satterthwaite, PhD

University of Texas Southwestern Medical Center

Contribution of B-1a Cells to Lupus Autoimmunity

Systemic lupus erythematosus (SLE) is an autoimmune disease characterized by autoantibodies (autoAbs) against nucleic acid containing antigens. Immune complexes formed by these autoAbs promote inflammation and potentially fatal organ damage. B cell targeting therapies have been only modestly effective in lupus, perhaps due to the presence of both pathogenic and protective B cell subsets. Therapeutic approaches that target the former but spare the latter are likely to be more efficacious. Defining these opposing B cell subsets is thus of great clinical significance. B-1 cells are a long-lived innate like B cell subset with the potential to play either a protective or a pathogenic role in lupus. They produce natural IgM which promotes clearance of apoptotic cells, a source of self-antigen in lupus, and secrete the anti-inflammatory cytokine IL-10.

However, their repertoire is skewed toward polyreactive and low affinity self-reactive specificities, suggesting that their inappropriate activation may be pathogenic in lupus. Indeed, they are increased in several lupus models, are prone to plasma cell differentiation, can be induced to produce anti-dsDNA IgG, and are efficient antigen presenters that skew T cell responses towards the pro-inflammatory Th1 and Th17 subsets.

Numerous genetic manipulations that increase B-1 cell numbers also result in the development of lupus-like autoimmune disease. To date, however, there has been no direct demonstration of either a pathogenic or protective role of B-1 cells in lupus due to the previous lack of available reagents to track the fate of, deplete, or delete genes in this subset. Here we will take advantage of two newly available mouse strains that now facilitate these manipulations of B-1 cells. We will use these mice to track the fate of activated B-1 cells, determine their contribution to the autoreactive plasma cell pool, and define their role in the autoimmune phenotype of two lupus models.

INNOVATIVE RESEARCH AWARD

Sangeeta Sule, MD, PhD

Children's Research Institute

Characterizing Lupus Activity Using an Innovative Technology

Systemic lupus erythematosus (SLE), like many other rheumatologic conditions, is a complex disease involving many organ systems and can be associated with a variety of symptoms. The existing assessment tools and laboratory tests may not reliably reflect patient reported symptoms. Furthermore, these biomarkers may be unable to predict the onset of a flare state. Studies have shown that patients with well managed lupus accumulate less organ damage and demonstrate improved outcomes over time.

There is an unmet clinical need for reliable biomarkers that reflect disease activity to facilitate the treatment of a specific SLE disease activity phenotype and severity. This measure would help to guide the adequacy of immunosuppressive therapy or indicate a need to treat other symptoms. Regular monitoring of such an endpoint has the potential to predict a flare in activity, which would enable early, preemptive interventions and improve patient outcomes. The planned study proposes to enroll 20 SLE patients and follow them for a period of one year. During each clinic visit, assessment of a patient's SLE disease activity will be performed using the SLEDAI-2K clinical assessment tool and our device. We will establish the initial validation of the SLE Index as a measure of disease activity by assessing sensitivity, specificity, and reliability of the SLE Index.

Ultimately, this tool has the potential to serve as a monitor of SLE disease activity, enabling early detection of flares or management of symptoms not responsive to immune suppressing drugs. The application of this technology has the potential to be used as an objective clinical trial endpoint for the development of new therapeutics in SLE.

INNOVATIVE RESEARCH AWARD

Henri Tiedge, PhD

The Research Foundation of SUNY

Therapeutic Prospects in Neuropsychiatric Systemic Lupus Erythematosus

Systemic lupus erythematosus (SLE) is an autoimmune disorder of poorly understood etiology. The brain is impacted in neuropsychiatric lupus (NPSLE, a common subtype of SLE), and phenotypic manifestations often include seizures and cognitive impairment. As NPSLE pathogenesis remains underexplored, treatment options are typically limited to non-specific (e.g. immunosuppressive) interventions.

In neurons, Brain Cytoplasmic (BC) RNAs regulate local mRNA translation, and thus protein synthesis, at the synapse. Absence of BC RNAs from synapto-dendritic sites of function causes seizure susceptibility and cognitive impairment. BC RNAs are transported to such sites by RNA transport factors that interact with noncanonical structural motifs in BC RNA dendritic targeting elements (DTEs).

Autoimmune reactivities against BC RNAs were detected in a subset of patients with NPSLE. SLE anti-BC RNA autoantibodies (SLE anti-BC abs) displace transport factors from BC RNA DTEs, as a result interfering with BC RNA delivery to synapto-dendritic sites.

Here it is proposed to investigate, at three levels, the role of SLE anti-BC abs in molecular-cellular mechanisms contributing to NPSLE pathogenesis. (1) Molecular mechanism will be dissected that underlie the ability of SLE anti-BC abs to displace RNA transport factors from BC RNA DTEs. BC RNA decoys will be designed to deny SLE anti-BC abs access to these DTEs. (2) It is posited that SLE anti-BC ab displacement of transport factors from regulatory BC RNAs will result in their inadequate synapto-dendritic delivery. Decoys will be examined for their ability to rescue BC RNA transport and localization. (3) SLE anti-BC abs are hypothesized to cause seizure susceptibility and cognitive dysfunction. BC RNA decoy strategies will be used to intercept pathogenic SLE anti-BC abs and rescue BC RNA neuronal functionality in vivo.

It is thus the project goal to establish the utility of BC RNA decoys in target-specific therapies of NPSLE.

INNOVATIVE RESEARCH AWARD

Ernest Vina, MD*

University of Arizona

Mobilizing Hispanics with Knee Osteoarthritis through Live Video Consultations: A Dyad-Based Approach

Osteoarthritis (OA) is a disabling disease that greatly affects Hispanics living in the US. Hispanics, compared to non-Hispanic whites, are less likely to exercise to treat OA and are more likely to be physically inactive. Evidence suggests that social support may increase healthy behaviors and positive outcomes. The primary objective of the study is to carry out the activities necessary for the planning, design, and documentation of a clinical trial to demonstrate the efficacy of a culturally acceptable treatment program to increase exercise and regular physical activity (PA) among Hispanics with knee OA. The long-term goal is to maximize the utilization of exercise and regular PA that would reduce ethnic disparities in the clinical outcomes of patients with OA.

The first aim of the study is to conduct interviews to explore the acceptability of an intervention to promote exercise and regular PA with a family member/close friend among Hispanics with knee OA. Results of the first aim will be used to refine a culturally tailored video-administered educational, muscle strengthening, and PA program. The second aim of the study is to conduct a pilot study to determine the acceptability and feasibility of the developed intervention and its potential impact on OA symptoms.

Hispanic patients with knee OA paired with a chosen family member/close friend (i.e., dyads) will be recruited for study participation. Study dyads will be interviewed, and the qualitative data will be analyzed to identify variables that may affect exercise and PA participation on a regular basis. A pilot randomized controlled trial will also be conducted. An intervention will be administered by video consultations and will include educational sessions about OA, a muscle strengthening program, and a program that will aim to promote regular physical activity involving dyads. Feasibility of the intervention will be based on the number of dyads that will be recruited during the study and on attendance to the intervention sessions, and acceptability will be based on quantitative acceptability (e.g., program satisfaction) and qualitative measures. The potential effects of the intervention (compared to an OA educational only control) on OA symptoms will also be determined.

* These funds are available in partnership with AbbVie and UCB, with additional support provided by Bristol Myers Squibb.

INNOVATIVE RESEARCH AWARD

Shouan Zhu, PhD

Ohio University

Defining the Role of Chondrocyte de Novo Lipogenesis in Obesity Associated Osteoarthritis Development

Most older adults (~70 years of age) have some signs of osteoarthritis (OA) in their joints. Development of OA can also be accelerated by ~20 years in individuals who are obese. This phenomenon does not stem solely from greater mechanical loading, obesity also increases OA risk in non-weight-bearing hand joints. It is well-known that obesity causes excessive lipid deposition in non-adipose tissue, which leads to lipotoxicity and tissue dysfunction. Yet, it is still unknown how obesity promotes lipids accumulation locally in cartilage that may contribute to OA development. Identifying the mechanisms by which lipid accumulates in cartilage during obesity could inform effective interventions and therapeutic strategies that reduce the incidence and impact of OA, especially in the context of an increasing prevalence of obesity.

This project will investigate how chondrocyte de novo FA biosynthesis plays a role in lipid accumulation in cartilage tissue and promoting joint inflammation. Our preliminary data shows that chondrocytes accumulate proinflammatory ?-6 fatty acids during obesity, and this is associated with upregulation of acetyl-coA carboxylase 1 (ACC1) during obesity to synthesize FA. We also showed in vitro that blocking ACC1 activity effectively inhibits FA intracellular accumulation in chondrocytes. Yet how do ACC1 and its regulation of FA biosynthesis play a role in obesity associated OA development in vivo? Guided by our preliminary data and the literature, we will investigate this question via two specific aims: Aim 1. Determine the role of ACC1 mediated FA biosynthesis in obesity associated OA development; Aim 2. Determine the mechanism by which FA synthesis regulates cartilage catabolism following a pro-inflammatory challenge. Well-established mouse models of diet-induced obesity and OA will be used in combination with genetically modified mouse models that allow conditional deletion of ACC1 in cartilage. Mouse OA phenotyping will be used to examine the consequences of abolishing FA biosynthesis on OA pathology. In vivo and in vitro metabolic profiling methods will be leveraged to determine the effects of ACC1 mediated FA biosynthesis on inflammation and cartilage degeneration. We also will use advanced lipidomics technology to identify the specific FA or lipid species in cartilage. Successful completion of this research is expected to provide comprehensive understanding of how obesity increases FA accumulation in joint cells and promotes OA development, offering the potential to provide new therapeutic targets for OA treatment.

Norman B. Gaylis, MD Clinical Research Award

Established with a generous commitment to the Foundation from Dr. Gaylis, the Norman B. Gaylis, MD Clinical Research Award provides funding for research that will impact community practice. Studies may include, but are not limited to, international collaborations, health services research, outcome studies, practice supply and demand, and/or clinician–patient communication.

NORMAN B. GAYLIS, MD CLINICAL RESEARCH AWARD

Erin Bauer, MD

Virginia Mason Medical Center

Increasing Lung Cancer Screening Rates amongst CCP+ Rheumatoid Arthritis Patients

We plan to implement a multi-disciplinary quality improvement project within our community-based tertiary referral center to increase screening rates for lung cancer in Rheumatology patients. Lung cancer is the second most common cancer and leading cause of cancer death in the United States. Screening is critical in high-risk populations to improve prognosis. Rheumatoid arthritis (RA) patients have a two-fold increase in lung cancer and are more likely to be diagnosed at advanced stages. In 2018, the US Preventive Services Task Force updated its lung cancer screening recommendation: annual low dose CT screening in 50-80 year olds with 20 pack year smoking history who currently smoke or quit within the last 15 years.

Only 3-4% of eligible adults nationally are estimated to have had recommended lung cancer screening. Centers with known low lung cancer screening rates have demonstrated improvement after implementing more accurate smoking history documentation.

In 2022 we started an IRB approved pilot study to determine how many eligible CCP+ RA patients at VM have received recommended screening per USPSTF. Using a database of 600 CCP+ RA patients maintained by VM Benaroya Research Institute, we identified 37 individuals eligible for screening. Only 3 were up to date. Most individuals did not have enough information to determine eligibility as they were missing quit dates and/or pack year history.

Our goals: 1. Expand our pilot study to a larger CCP+ RA patient population. VM is one of the largest rheumatology practices in Washington state and serves a large geographic region with substantial population from rural/underserved areas and of ethnic populations with higher smoking rates. 2. Preform root cause analysis and conduct a PDSA to improve screening rates. We'd collaborate with VM primary care and pulmonary to improve screening metrics. We'd develop education for rheumatologists across the State to increase awareness around lung cancer screening.

Career Development Research Awards

Increasing concerns over the decline in federal funding for rheumatology research have forced many investigators to reconsider their careers, resulting in fewer researchers making the important discoveries necessary to advance treatments and find cures. The Career Development Research Awards are designed to encourage early and midcareer investigators to continue vital research into the cause, prevention, and treatment of rheumatic diseases.

Career Development Bridge Funding Award: R Bridge

The R Bridge Award encourages essential rheumatology research by supporting promising investigators who are revising outstanding NIH R01 or VA RCS/ORD award applications.

CAREER DEVELOPMENT BRIDGE FUNDING AWARD: R BRIDGE

Maureen Dubreuil, MD, MSc

Boston University

Back Pain Phenotypes: Imaging, Genetics and Outcome Associations Among Rural Residents of the United States

The R01 application to NIH/NIAMS, “Back Pain Phenotypes: Imaging, Genetics and Outcome Associations among Rural Residents of the United States” proposes studies chronic back pain among rural Americans, with a focus on midback pain. We will assess for associations of midback pain with key clinical outcomes that have been demonstrated in low back pain but never studies in relation to the midback. This knowledge will help guide individualized back pain treatment strategies and inform equitable implementation of back pain treatment among subgroups that are disproportionately affected back chronic back pain. Secondly, we will test for associations between chronic midback pain and thoracic spine pathologic patterns on CT thorax studies (disc disease, facet joint osteoarthritis, DISH, axial spondyloarthritis and scoliosis).

We hypothesize that midback pain will have stronger symptom-structure associations than exist for low back pain, thus supporting treatment of specific conditions of the thoracic spine. Finally, we will test for associations of back pain with polygenic risk scores for chronic back pain, disc disease and axial spondyloarthritis. Findings from this aim will support specific back pain treatment strategies and also aid understanding of the genetic underpinning for spinal conditions across gender, race/ethnicity and ancestry subgroups.

CAREER DEVELOPMENT BRIDGE FUNDING AWARD: R BRIDGE

Beatriz Hanaoka, MD, MSc

The Ohio State University

Mechanisms Underpinning Obesity Driven Rheumatoid Arthritis Disease Activity

The goal of this study is to address the mechanism(s) by which obesity and ketone ester (KE) supplementation affect the production of immune cells that sustain chronic inflammation in rheumatoid arthritis (RA). We will test the hypothesis that inflamed adipose tissue (AT) stimulates production of inflammatory monocytes that migrate into affected joints, aggravate inflammation, and contribute to treatment resistance. Although obesity is a significant predictor of refractory disease in RA, the underlying molecular mechanisms remain unknown. Furthermore, although dietary modification is one of the cornerstones of obesity treatment, a prescriptive dietary intervention approach for RA patients who are obese has not been rigorously tested. Emerging research suggests the ketone body beta-hydroxybutyrate (BHB) suppresses the NLRP3 inflammasome, which could translate into a therapeutic option for RA patients with obesity.

Our published data demonstrated that chronically inflamed AT sustains low-grade chronic inflammation in obesity by increasing the production of myeloid cells in the bone marrow (BM) and increasing circulating monocytes, via activation of the NLRP3 inflammasome and secretion of IL-1b. Therefore, we hypothesize that a KE intervention will have a favorable impact on AT inflammation and disease activity of RA patients with obesity via inhibition of the NLRP3 inflammasome. Our preliminary data demonstrated that there is a robust expansion of CD14+CD16+ intermediate monocytes in the blood of obese RA patients compared to non-obese RA. A higher frequency of these monocytes in either peripheral blood or the synovial lining is associated with worse RA disease activity. Although the precise molecular mechanism is not known, intermediate monocytes could produce IL1-b and other key cytokines (IL-6, IL23) required for induction/ expansion of proinflammatory Th17 cells and aggravate the disease.

Therefore, we will determine cellular and molecular markers of AT inflammation in RA individuals with obesity that correlate with disease activity; the ability of CD14+CD16+ cells sorted from RA patients with obesity to induce pathogenic Th17 cells from CD4 T cells in vitro using co-culture studies; and whether a KE intervention has a favorable impact on cellular and molecular indices of inflammation in AT and peripheral blood in RA.

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.

CAREER DEVELOPMENT BRIDGE FUNDING AWARD: K BRIDGE

Isaac Harley, MD, PhD, MS

University of Colorado, Denver

Defining the Molecular Basis of MicroRNA-146A Risk Allele Effects in Systemic Lupus Erythematosus

Systemic Lupus Erythematosus (SLE) arises from an incompletely defined mix of genetic and environmental factors. We identified a causal genetic variant for SLE, rs2431697 and applied genome-editing to show that this variant disrupts an enhancer of MIR146A gene expression. In vitro data suggested a type I interferon mechanism. Contradictory in vivo SLE patient data suggests a type I Interferon activity independent mechanism. Subsequent data point to a mechanism involving anergy or peripheral tolerance of autoreactive B-cells to self-antigens. First, rs2431697 is an eQTL for MIR146A in B cells. Second, the transcription factor, BHLHE40, exhibits allele-dependent physical interaction with the SLE risk variant in B cells. Third, our data highlights a role for both genes in B cell anergy.

These results prompt our hypothesis: the MIR146A SLE risk enhancer confers SLE risk by modulating B cell anergy development. Thus, we aim to: Aim 1) define the cellular locus of MIR146A dysregulation in vivo. To do this we will define whether the molecular correlates of our model are present in diverse immune cell populations from healthy individuals and those with SLE analyzed immediately ex vivo We will use a combination of genotyping, RNA-Flow and spectral flow cytometry of PBMC immunophenotyping test our model. Aim 2) define the functional relationship between BHLHE40 and MIR146A in B cell anergy.We will determine whether MIR146A expression regulates autoreactive B-cell tolerance (Aim 2a) ex vivo in primary cells, (Aim 2b) in vitro in cell lines and (Aim 2c) in vivo in mice. Aim 3) define whether global down regulation of microRNA metabolism occurs in anergic B cell. Most microRNAs undergo decay during acute antigen receptor stimulation. Since chronic antigen receptor simulation is the major mechanism of B-cell anergy, global microRNA decay may enforce anergy. To interpret our results in context, we must know whether this is true. This will also advance Dr. Harley's career goals.

CAREER DEVELOPMENT BRIDGE FUNDING AWARD: K BRIDGE

Sahar Lotfi-Emran, MD, PhD

University of Minnesota

Resident Memory CD8+ T Cells Trigger Joint Specific Inflammation in Rheumatoid Arthritis

Understanding the tissue resident immune environment of synovial joints is key to addressing the critical need for new, targeted, and safer therapies for rheumatoid arthritis (RA). At a systemic level, the pathophysiology of RA is driven by CD4+ T cells and B cells generating anti-citrullinated antibody (ACPA), but these are processes that can predate by decades the clinical diagnosis of RA, made at the onset of joint inflammation. CD8+ TRM are a subset of memory T cells that populate non-lymphoid tissues following infection, persist after resolution of infection, and, unlike their central memory counterparts, do not recirculate in quiescent periods. TRM serve as tissue sentinels with sensing and alarm functions that prevent and control re-infection but can also contribute to pathologic processes.

I hypothesize that following viral infection, resident memory CD8+ T cells (TRM) localize to specific joints where they can promote arthritogenesis. I will utilize a mouse model of viral infection as well as a more holistic natural microbial exposed collagen induced arthritis mouse model to determine if TRM sensing and alarm functions recruit immune cells to the joint, activate neutrophil NETosis at the joint which generates citrullinated targets, increase permeability of the joint to ACPAs, and support ectopic germinal center formation.

I will define a joint-specific signature that identifies CD8+ TRM in both mice and humans via comparative immunology and single cell RNA sequencing and analysis. Overall, this proposal will clarify the contribution of CD8+ TRM to joint inflammation, establish a pathoimmunologic link between prior infectious exposure and the development of RA and RA joint flares, and identify an innovative, local target for therapy.

Investigator Award

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.

INVESTIGATOR AWARD

Sebastian Bruera, MD*

Baylor College of Medicine

Implementation of a Structured Telemedicine Program in Patients with Systemic Lupus Erythematosus

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease that requires clinical and laboratory evaluation quarterly. However, work schedules, childcare, or difficulties with transportation may affect adherence to these clinic and laboratory visits. Under-resourced patients with SLE have no-show rates upwards of 40%. Furthermore, no-shows to clinic appointments are associated with increased morbidity. Strategies are needed to decrease no-show clinic appointments in patients with SLE.

The coronavirus 2019 (COVID-19) pandemic resulted in the widespread emergence of telemedicine as a modality for clinic visits. Telemedicine may provide an opportunity to improve clinical outcomes, including a decreased no-show rate, in patients with SLE – especially those that are under-resourced. However, very few studies have been conducted exploring the role of telemedicine in patients with rheumatic diseases – and there is especially a paucity of knowledge as it pertains to SLE.

This proposal seeks to understand the benefits of telemedicine visits for SLE patients, particularly those from under-resourced populations in the United States. The overall goal for this proposal is to implement and evaluate a structured video telemedicine program designed for patients with low socioeconomic status living with SLE. We hypothesize this will increase access to care, while maintaining high patient satisfaction and quality of care. Furthermore, we will establish the validity of SLE disease activity indices (SLEDAI) and damage indices (SDI) for video telemedicine visits.

* These funds are available in partnership with AbbVie and UCB, with additional support provided by Bristol Myers Squibb.

INVESTIGATOR AWARD

Ali Duarte-Garcia, MD

Mayo Clinic

Developing an Evidence Base for Glucocorticoid Use in Lupus Nephritis

Patients with systemic lupus erythematosus (SLE) have increased morbidity and mortality compared to the general population. This may be due, at least in part, to the use of glucocorticoids. Glucocorticoids remain one of the main therapies for SLE, particularly for lupus nephritis. Yet, there is a lack of evidence regarding the safest dose and duration of glucocorticoid regimens for lupus nephritis treatment. This proposal aims to develop evidence-based standards for glucocorticoid use in lupus nephritis by analyzing practice variation across the US and comparing the efficacy and safety of different glucocorticoid regimens.

In this study, we will use a diverse nationwide database containing de-identified claims for over 150 million persons linked to pharmacy and laboratory data to identify patterns of glucocorticoid prescribing in patients with lupus nephritis. We will also perform an individual patient data meta-analysis of published and unpublished randomized clinical trials to evaluate the effect of different glucocorticoid regimens on response rates and infections among patients with lupus nephritis.

The ultimate goal of this research is to fill critical knowledge gaps in the management of lupus nephritis and improve the effectiveness and safety of glucocorticoid regimens. The study will also inform randomized controlled trials to prospectively test different glucocorticoid regimens, which will be natural extensions of the proposed work.

The study has a unique opportunity to identify evidence-based glucocorticoid regimens for lupus nephritis with the lowest effective doses and exposure, which will reduce infections, glucocorticoid-associated comorbidities, and may reduce mortality. The findings from this novel research will provide the necessary fundamental information to inform contemporary practice and the design of clinical trials to define evidence-based glucocorticoid regimens.

INVESTIGATOR AWARD

Lesley Jackson, MD

University of Alabama at Birmingham

Barriers and Facilitators to COVID-19 Vaccinations in Patients with Autoimmune Arthritis

This Investigator Award project will develop a behavioral intervention to address COVID-19 vaccine/ booster uptake among people with autoimmune arthritis in the Deep South. Vaccine hesitancy is of key clinical and public health importance because of the added COVID-19 risk related to some treatments for autoimmune arthritis and the critical need to increase vaccine uptake in this at-risk population. Telehealth may address a gap in care by offering a mechanism to answer vaccine-related questions and to address patient concerns, screen for eligibility, and connect patients to a place where they can receive the vaccine. In addition, telehealth approaches may be preferred by those individuals in rural, high-need and traditionally underserved areas.

In Aim 1, I will test the hypothesis that, compared to vaccinated or boosted people, unvaccinated people with autoimmune arthritis will have higher rates of hospital admissions, intensive care unit admissions, mechanical ventilation need, and longer length of hospital stay. I will use the extensive National COVID Cohort Collaborative (N3C) database to conduct this assessment. For Aims 2 and 3, participants will be recruited from University of Alabama at Birmingham (UAB) rheumatology clinics. In Aim 2, I will evaluate decision-making behaviors that affect vaccine hesitancy among people with autoimmune arthritis with focus groups and a discrete choice experiment. I hypothesize that this approach will generate actionable barriers/ facilitators to COVID-19 vaccine uptake as key vaccine attributes (e.g., fear of disease flare, influence of immune suppression on efficacy, or risk for adverse events). In Aim 3, I will develop a multimodal intervention with telehealth components tailored to the needs of patients with autoimmune arthritis from the Deep South addressing COVID-19 vaccine/ booster uptake.

I will evaluate the feasibility and acceptability of implementing this novel multimodal intervention in a pilot implementation study of 20 people with autoimmune arthritis following their in-person clinic visits. I will gain expertise in mixed methods research, behavioral intervention design and implementation while adding to our understanding of the factors contributing to COVID-19 vaccine hesitancy among people with autoimmune arthritis.

INVESTIGATOR AWARD

Maximilian Konig, MD

Johns Hopkins University

Antigen-Specific T Cell Therapies for Antiphospholipid Antibody Syndrome

Antiphospholipid syndrome (APS) is an autoimmune disease characterized by arterial and venous thrombosis, recurrent fetal loss, and microangiopathic organ damage. Pathogenic antibodies in APS target the phospholipid-binding protein beta-2-glycoprotein I (ß2GPI), and inhibition of anti- ß2GPI domain I antibodies prevents disease in mice. As sources of these disease-causing autoantibodies, anti-ß2GPI B cells are ideal therapeutic targets in APS. Selectively eliminating these autoreactive B cells, while preserving normal B cells, has the potential to prevent and treat APS without increasing the risk of infection. Current treatment strategies for APS, however, do not address this underlying immunopathology and often fail.

Engineered T cell therapies have revolutionized the treatment of cancer over the past decade and can be curative. Conventional T cell therapies targeting pan-B cell antigens, such as CD19-targeted CAR-T cells, have the potential to achieve complete remission in patients with refractory autoimmune diseases, but their widespread adoption will be limited by infection risk. To fully leverage the potency of engineered T cell therapies for the prevention and treatment of autoimmune diseases, there is a critical need for precision cellular immunotherapies that can selectively eliminate disease-causing B cells. In this proposal, we aim to develop such a precision therapy for the treatment of APS.

We hypothesized that T cells can be reprogrammed to selectively eliminate anti-ß2GPI B cells in an antigen-dependent manner, thereby treating APS without impairing overall B cell immunity. In preliminary studies, we showed that this can be achieved by expressing a chimeric autoantigen-T-cell receptor (CATCR) on engineered T cells, introducing autoantigenic sequences of ß2GPI as part of the T-cell receptor (TCR)-CD3 protein complex. CRISPR/Cas-edited CATCR-T cells acquired the ability to bind and kill autoreactive anti-ß2GPI B cells in APS via the perforin–granzyme pathway, while not depleting other B cells. In this proposal, we will develop different CATCR-T cell therapies designed to prevent and treat APS. The therapeutic potency and specificity of these precision therapies will be systematically tested in vitro using several orthogonal human cellular model systems, developed in this proposal, and in vivo for APS-induced fetal loss. Collectively, these studies aim to advance a new class of precision cellular immunotherapies to treat autoimmune rheumatic diseases without impairing normal immune function.

INVESTIGATOR AWARD

Sahar Lotfi-Emran, MD, PhD

University of Minnesota

Localizing Systemic Autoimmune Processes: CD8+ T Cells Trigger Joint Specific Inflammation in Rheumatoid Arthritis

Many patients with recalcitrant autoimmune arthritis, including Rheumatoid Arthritis, have not benefited from recent advances in therapeutics that have dramatically improved patient quality of life. The goal of this proposal is to determine the joint specific mechanisms by which infectious exposure in people at risk for autoimmune arthritis leads to development of disease and recurrent joint flares.

Understanding the tissue resident immune environment at the joint is key to addressing the critical need for new, joint targeted, and safer therapies. To facilitate these goals, this project builds prior work demonstrating virus specific CD8+ T cells traffic to joints during and after resolution of infection to fully assess the potential mechanisms by which CD8+ T cells contribute to joint inflammation. This project also evaluates the effects of natural microbial exposure on susceptibility to induced autoimmune arthritis in mice. Thus, through deterministic and naturalized models, this proposal seeks to understand the contribution of infectious exposure on shaping and misshaping the immune system in the setting of health and autoimmune disease.

Scientist Development Award

The Scientist Development Award encourages early career rheumatologists and rheumatology health professionals to pursue innovative research ideas.

SCIENTIST DEVELOPMENT AWARD

Rachel Elam, MD

Augusta University Research Institute

Fracture Risk Assessment in Persons with Rheumatoid Arthritis

Rheumatoid arthritis (RA) is a central risk factor for osteoporosis. Persons with RA have up to two times higher risk of osteoporotic fracture, which are associated with significant morbidity and excess mortality. Despite the burden of fractures in RA, many persons with RA at high risk of fracture are not taking one of several widely available pharmacologic therapies to treat osteoporosis. Concerns about rare side effects (i.e. atypical femoral fractures, osteonecrosis of the jaw) of bone heath medications, particularly bisphosphonates, are prevalent.

The most commonly used fracture risk assessment tool in persons with RA is FRAXTM, which produces 10-year risk probabilities of major osteoporotic fracture (MOF; hip, clinical spine, forearm or humerus fracture) and hip fracture. When shared decision-making discussions regarding bone health medications are framed in terms of long-term (i.e. 10-year) fracture risk, persons often perceive that potential medication side effects or other health problems take precedence over possible fracture prevention over such a long horizon. However, fracture risk is not stable over time. Imminent (i.e. 2-year) fracture risk may be of greater immediate personal significance to persons with RA considering initiation of pharmacologic therapy for fracture prevention.

We hypothesize that we can determine how well the 10-year MOF and hip fracture risk probabilities generated from U.S. FRAX capture imminent MOF and hip fracture risk in persons with RA age 65 years and older. We will use the Rheumatology Informatics System for Effectiveness (RISE) registry in conjunction with linked Medicare claims data to assess the discriminative ability of U.S. FRAX to predict incident imminent and intermediate horizon (i.e. 4-year) risk of MOF and hip fracture.

We further hypothesize that imminent fracture risk prognostication in persons with RA can be improved by incorporating RA-specific characteristics (i.e. seropositivity for rheumatoid factor (RF) and/or anti-cyclic citrullinated peptide antibody (ACPA), RA disease activity level, and RA therapy). To assess this, we will determine if the addition of RA-specific characteristics to models already incorporating the individual risk factors in U.S. FRAX improves model predictive performance for imminent and intermediate horizon risk of MOF and hip fractures in our older RA cohort from RISE.

SCIENTIST DEVELOPMENT AWARD

Guy Katz, MD

Massachusetts General Hospital

Complement Activation and Damage in IgG4-Related Disease

IgG4-related disease (IgG4-RD) is a systemic immune-mediated disease described in the early 2000's that leads to inflammation and tumor-forming lesions that can affect nearly any organ or anatomic site. Although effective treatments exist, many patients with IgG4-RD experience substantial, irreversible damage as a result of their disease. However, study of damage in the disease has been limited by the lack of a validated instrument by which damage can be described and quantified. In addition, although early findings have suggested a potential role of complement activation in the inflammatory infiltrate of IgG4-RD, the precise nature of complement activation and its relationship to disease activity and damage are not yet known.

In the proposed project, we aim to fill these knowledge gaps by leveraging the large clinical database and biorepository of patients with IgG4-RD at the Massachusetts General Hospital (MGH) as well as previously established international, multidisciplinary collaborations of IgG4-RD experts. First, we plan to develop a damage index for the disease by identifying salient manifestations of damage, applying multicriteria decision analysis, and performing an initial validation of the damage index. Second, we will characterize complement activation in patients with IgG4-RD by measuring complement activation products (CAPs) in a large, cross-sectional cohort and a smaller, longitudinal cohort of patients with IgG4-RD. In the cross-sectional cohort derived from the MGH IgG4-RD biorepository, we will measure and compare CAPs from samples obtained during active disease and remission. In the longitudinal cohort, we will identify patients with active IgG4-RD and planned treatment with rituximab, a highly effective treatment for the disease, and we will measure CAPs prior to and periodically after the initiation of treatment. We hypothesize that CAPs will be present during active disease and correlate with disease activity.

The findings from the proposed project will provide a much-needed tool to assess damage in the disease, which can be implemented into clinical trials and used to better understand the mechanisms and risk factors leading to damage in IgG4-RD. Furthermore, a better understanding of the role of the complement system in IgG4-RD may identify novel therapeutic targets for the disease.

SCIENTIST DEVELOPMENT AWARD

Maxime Kinet, MD, PhD

University of California, San Francisco

Inflammatory Imprinting of Th2-Supporting Fibroblasts

Patients with rheumatic disease can exhibit relapsing courses. Understanding mechanisms driving relapse will improve treatments for these patients. Tissue-resident lymphocytes may drive some of these relapsing tendencies; however, these cells can be resistant to eradication. Targeting stromal niches that promote lymphocyte persistence in tissues may help control disease. Despite fibroblasts being key components of tissue immune niches, we do not know whether peripheral tissue fibroblasts can possess memory of previous inflammation that persists after the initial inflammatory stimulus resolves in vivo.

We recently characterized a novel fibroblast-Th2 cell niche in skin. Through their production of IL-33, these fibroblasts, which we termed Th2-interacting fascial fibroblast (TIFFs), sustain tissue-resident Th2 cells. In turn, Th2 cells activate TIFFs through type 2 cytokines. In mice, transiently activating this mutually supportive TIFF-lymphocyte niche triggers histologic changes strikingly reminiscent of eosinophilic fasciitis. When inflammation resolves, normal skin architecture returns. However, challenge with a second stimulus results in a more robust TIFF response. Thus, memory of previous inflammation may be imprinted in TIFFs. I expect to find that this memory modulates important and complementary TIFF functions, including lymphocyte niche provision and tissue repair, by changing chromatin accessibility at key genes, which in turn alters subsequent transcriptional responses. I will use novel, fibroblast-subset-specific mouse genetic tools to address the following aims:

Aim 1. Investigate the molecular mechanisms of inflammatory imprinting on TIFF immune niche formation

Aim 2. Investigate the functional consequences of inflammatory imprinting on TIFF-mediated tissue repair.

The overall goal of this proposal is to investigate the fundamental biology of a novel subset of tissue stromal cells and their role in facilitating inflammation.

SCIENTIST DEVELOPMENT AWARD

Vanessa Kronzer, MD, MSCI

Mayo Clinic

Respiratory Interactions and Genetic Variants for Rheumatoid Arthritis Prediction and Prevention

Dr. Vanessa Kronzer is a rheumatology fellow at Mayo Clinic who aims to become an independent investigator in the genetic epidemiology of rheumatoid arthritis (RA). She has previously led investigations finding that upper airway diseases increase RA risk. In addition, whole-exome sequencing has revealed low-frequency and rare genetic variants that strongly influence risk of other diseases. Our central hypothesis is that gene by respiratory interactions and low-frequency/rare variants drive RA pathogenesis and thereby improve disease prediction.

In this proposal, we seek (aim 1) to identify key gene-respiratory disease interactions that increase RA risk, and (aim 2) to identify low-frequency and rare coding variants associated with RA risk. To accomplish these aims, we will leverage the Mayo Clinic Biobank, Mayo Clinic Tapestry study, and Mass General Brigham Biobank. These datasets contain clinical, genome-wide association study (GWAS), and whole-exome sequencing data for over 211,000 individuals (3,000 with RA).

We expect this proposal to identify novel gene-respiratory interactions (aim 1) and risk genes/variants (aim 2) that have a positive impact on RA prediction and prevention. It will also strengthen Dr. Kronzer's collaborations and provide training in key domains necessary to become an independent investigator, including genetic epidemiology, next-generation sequencing, and research methodology.

Overall, the proposed work will position Dr. Kronzer to submit competitive NIH K/R01 applications toward the prediction, prevention, and treatment of RA. It will thereby improve the health of people with rheumatic disease.

SCIENTIST DEVELOPMENT AWARD

Amit Lakhanpal, MD, PhD

Hospital for Special Surgery

T Cell Receptor Repertoire and Somatic Mutations Of Synovial CD4 T Cells in Rheumatoid Arthritis

Rheumatoid arthritis (RA) is the most common autoimmune disorder encountered in rheumatology practice. Despite the exciting development of multiple targeted biologic therapies, the burden of late remission due to serial medication trials – or even lack of response to any available therapy – is a major unmet need for which progress would have a significant impact on patients' well-being. Understanding the mechanistic underpinnings of the observed clinical diversity of RA phenotypes, including responsiveness to specific therapies, is likely to be an important component of addressing this need.

In RA there is evidence for a pathologic contribution of T cells in general, and CD4 T cells in particular, within certain patient subsets. There is also evidence for a role of acquired somatic mutations in autoinflammatory and autoimmune diseases associated with age, such as RA. Utilizing synovial tissue and peripheral blood from RA patients, we will pursue a study designed to accentuate properties of CD4 T cells that are more likely to be pathogenic at the single-patient level. We aim to (i) study the contrast between individual subjects' CD4 T cells in the synovium versus those of CD4 T cells circulating in the peripheral blood with respect to T cell receptor (TCR) features and somatic mutation burden, and (ii) identify commonalities between CD4 T cells residing in different joints of the same patient.

Specifically, we will employ single-cell RNA sequencing to precisely identify TCR properties in synovial and blood CD4 T cells, testing the hypothesis that they differ between synovium and blood in individual patients. We will also utilize targeted amplification and sequencing to query whether there are synovium-enriched somatic mutations, even within a limited subset of CD4 T cells, that could drive synovial pathology. Further, we will compare the clonal CD4 populations of distinct synovial sites within individual patients, in order to understand to what degree a patient's RA in one joint is the same as in another. We hope that the combination of contrasts between RA synovium and blood, and comparisons across different synovial sites, will generate insight into actually pathologic features that may be targetable in a clinical setting.

SCIENTIST DEVELOPMENT AWARD

Yiming Luo, MD

Columbia University

Rare Genetic Variations in Takayasu's Arteritis: A Whole-Exome Sequencing Study

Takayasu's arteritis (TAK) is a rare inflammatory disease affecting large arteries, primarily the aorta and its major branches. There is an unmet need for better clinical management of TAK due to limited understanding of the disease biology. Substantial evidence has supported a genetic component contributing to the etiopathogenesis of TAK. Genetic association analysis is a powerful approach to understand the casual molecular alterations and disease heterogeneity. Previous studies using genome-wide association studies (GWAS) approach is limited by modest effect size and explaining only a minority of heritability.

Due to evolutionary selection pressure, deleterious variants tend to be rare, particularly for early-onset debilitating diseases that potentially affect reproductive fitness. Thus, we hypothesized that TAK is a genetically complex disease driven in part by rare variants with relatively large effect size. The objective of this study is to discover novel rare genetic variants associated with TAK on gene-, pathway-, and protein interaction network-level by leveraging data from whole exome sequencing (WES) of 160 patients with TAK and 3000 general population controls.

Our study will use state-of-the-art statistical methods and bioinformatic tools which allows for system-level investigation of disease biology. Gene-level collapsing analysis and multiple pathway-level analyses will be performed. Mantis-ml, a novel bioinformatic program based on machine-learning approach will be used to further prioritize potential causal genes in TAK. We will then evaluate and visualize top ranked genes in protein interaction networks and perform functional enrichment analysis using Metascape. Overall, we anticipate that this project will expand our understanding of the genetic landscape of TAK. This project will also be a critical step for my long-term goal toward improving personalized clinical management of rheumatic diseases based on advanced research in genomic medicine.

SCIENTIST DEVELOPMENT AWARD

Gregory McDermott, MD

Brigham & Women's Hospital

Identifying Genetic and Clinical Risk Factors for Fibrotic Rheumatoid Arthritis-Associated Interstitial Lung Disease

Rheumatoid arthritis-associated interstitial lung disease (RA-ILD) is a serious extra-articular disease complication that can affect up to 1 in 7 patients with rheumatoid arthritis and results in significantly increased morbidity and mortality. Although typically studied as a single entity, RA-ILD consists of multiple subtypes with different pathogenesis, prognosis, and potential treatment options. The most devastating subtype is the usual interstitial pneumonia (UIP) subtype, which is characterized by lung fibrosis, accounts for approximately half of RA-ILD cases, and has a particularly high mortality.

This proposal seeks to deconstruct RA-ILD disease heterogeneity and identify genetic and clinical risk factors for the fibrotic subtype of RA-ILD. We will investigate the impact of pulmonary fibrosis risk genes on RA-ILD using two RA cohorts and also examine the impact of recurrent respiratory infections on the development of fibrotic RA-ILD. To accomplish these aims, we will leverage genetic and clinical data from several sources. We will investigate the impact of pulmonary fibrosis risk genes on RA-ILD risk using genetic data from two large RA-ILD case-control studies. We will calculate a pulmonary fibrosis polygenic risk score and test the association of this score with RA-ILD by comparing RA-ILD cases to RA controls without lung disease. To investigate the impact of respiratory infections on fibrotic RA-ILD, we will use a large RA cohort with detailed clinical data from electronic health records to identify patients with the UIP subtype of RA-ILD. We will compare the rate of preceding respiratory infection in patients with the UIP pattern of RA-ILD to RA-ILD patients with non-UIP pattern. We anticipate that our findings will demonstrate that a pulmonary fibrosis polygenic risk score and history of recurrent respiratory infections will help to identify RA patients at high risk for RA-ILD, and the fibrotic UIP subtype of RA-ILD, in particular.

The findings from this proposal will improve understanding of genetic and clinical risk factors for fibrotic RA-ILD and help identify high risk patients who may benefit from enhanced screening or novel therapeutic strategies.

SCIENTIST DEVELOPMENT AWARD

Rachel Wallwork, MD

Johns Hopkins University

Interstitial Lung Disease Trajectory Prediction in Systemic Sclerosis

Interstitial lung disease (ILD) is a major cause of morbidity and mortality in systemic sclerosis (Scleroderma, SSc). While up to 90% of patients with SSc have evidence of ILD on high-resolution computed tomography (HRCT) scans of the chest, only 15-25% of patients develop progressive lung disease requiring treatment. Unfortunately, our ability to predict an individual patient's progression risk is limited. Risk factors for progressive lung disease at a population level, such as baseline forced vital capacity (FVC), Black race, diffuse skin involvement and anti-topoisomerase 1 antibody positivity, are well established, but how the risk factors interact and synergize in an individual patient is unknown.

Active debate surrounds when it is appropriate to initiate SSc-ILD treatment. Some providers only initiate medical therapy in patients with severe or progressive disease, while others treat all patients with any imaging evidence of ILD. The former treatment approach delays treatment until a patient demonstrates clear progression, at which point there may be irreversible damage and lower likelihood of treatment response. The latter strategy risks exposing patients at low likelihood of progression to significant side effects, including gastrointestinal symptoms, infections, and malignancy. Therefore, improving early prognostication of worsening ILD is a major unmet need.

Our overarching goal is to lay the foundation to develop an evidence-based, quantitative methods for evaluating a patient's risk of developing clinically meaningful ILD at an early stage of the disease. By analyzing lung function trajectories from patients in the Johns Hopkins Scleroderma Center Research Registry's prospective cohort, we will identify baseline clinical and serologic biomarkers that enable prediction of FVC and diffusion capacity for carbon monoxide (DLCO) trajectory and examine the association with poor clinical outcomes. Our Registry is one of the largest SSc registries internationally with over 4,207 patients and 22,238 pulmonary function tests (PFTs). Our overarching hypothesis is that by harnessing patient and population-level data we can predict which patients are at highest risk of ILD progression.

SCIENTIST DEVELOPMENT AWARD

Monica Yang, MD

University of California, San Francisco

Cell Specific Molecular Profiling of Scleroderma Associated Interstitial Lung Disease Subtypes

Interstitial lung disease (ILD) is present in up to 90% of systemic sclerosis (SSc) patients and is the leading cause of SSc-related mortality. SSc-ILD is classified pathologically in two subtypes: non-specific interstitial pneumonia (SSc-NSIP), characterized by diffuse septal thickening, lymphocytic inflammation, and is the most common pattern of SSc-ILD (75% of cases), and usual interstitial pneumonia (SSc-UIP), characterized by subpleural fibrosis and fibroblast foci adjacent to normal lung in a pattern hisptopathologically synonymous with idiopathic pulmonary fibrosis (IPF). Epidemiologic studies have shown SSc-UIP has a significantly worse prognosis than SSc-NSIP, does not respond to immunomodulating therapies, and has minimal treatment options outside of lung transplant. However, despite these clinical differences, little is known regarding the molecular pathways of these two SSc-ILD subtypes, making biomarker discovery and treatment development challenging and limiting precision medicine approaches in the field.

The objective of this proposal is to define the specific cell types and molecular pathways underpinning SSc-NSIP and SSc-UIP while comparing to those of IPF, a more common progressive form of ILD driven by telomere dysfunction and senescence reprogramming of epithelial cells. To establish a detailed understanding of SSc-ILD subtypes, we will leverage single nuclei RNA-seq on archived lung tissue to define the shared and distinct cell-specific molecular pathways of each subtype. We will then use RNAscope and immunohistochemistry to validate, and spatially localize within lung tissue the key cell types and transcripts of interest. Finally, we will probe aging and senescence related mechanisms, given their role in IPF pathogenesis, by measuring known genetic signatures, cell-specific telomere length, and circulating telomere-associated autoantibodies. Our central hypothesis is that the SSc-ILD subtypes will demonstrate shared pathways with IPF, including increased senescence and DNA damage, while also having distinct pathways that are SSc specific, such as enhanced inflammatory signature, that will together contribute to unique molecular profiles for each SSc-ILD subtype. We expect these innovative studies will have significant scientific and clinical impact by advancing our understanding of SSc-ILD classification, application of existing therapeutics, and discovery of new therapeutic targets.

Tobé and Stephen E. Malawista, MD Endowment in Academic Rheumatology

Established with a generous commitment from Mrs. Tobé and Dr. Stephen Malawista, this endowment ensures that physician scientists can continue their academic careers in vital rheumatic disease research. The Tobé and Stephen E. Malawista, MD Endowment in Academic Rheumatology provides a permanent source of support in basic science research career development for early career investigators.

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

Nikhil Jiwrajka, MD

University of Pennsylvania

Investigation of a Novel Epigenetic Mechanism Contributing to Female-Biased Disease in Systemic Sclerosis

Systemic sclerosis (SSc) is highly female-biased, but the molecular origins of this female bias are unclear. Sex hormonal and genetic factors have been insufficient to fully explain this female bias. Studies in individuals with sex chromosome aneuploidies have demonstrated a positive association between X chromosome dosage and susceptibility to female-biased rheumatic diseases, including SSc. These findings suggest that epigenetic dysregulation of the X chromosome may contribute to female-biased pathology in SSc. X-Chromosome Inactivation (XCI) is an X-chromosome-specific mechanism of epigenetic regulation that equalizes X-linked gene dosage between XX and XY individuals via the action of XIST RNA, an X-linked noncoding RNA that coats and transcriptionally silences the inactive X chromosome (Xi) in XX individuals. Though XCI was previously thought to be maintained in all female somatic cells via the static enrichment of XIST RNA on the Xi, our laboratory discovered that XCI maintenance is a dynamic process in T cells. In “dynamic XCI maintenance” (dXCIm), resting, naïve T cells from healthy XX humans and mice surprisingly lack focal enrichment of XIST RNA on the Xi. However, upon cellular activation, XIST RNA dynamically relocalizes to the Xi. Notably, the X chromosome contains several proinflammatory and profibrotic genes that are aberrantly overexpressed in circulating and/or skin-infiltrating T cells from patients with SSc.

I have therefore hypothesized that dXCIm is impaired in T cells from females with SSc, and that this impairment confers the observed female bias of SSc via an abnormal dosage of pathologic X-linked transcripts. In Aim 1, I am using high-dimensional spectral cytometry and single-cell imaging to determine whether dXCIm is impaired in relevant T-cell subsets from females with SSc, and then conducting allele-specific transcriptomic studies to determine the contribution of the Xi to X-linked gene expression. In Aim 2, I am inducing scleroderma-like disease in a novel mouse model of impaired XCI maintenance to determine how impaired dXCIm in T cells may contribute to pathology relevant to SSc. This work will improve our understanding of how biological sex contributes to SSc pathogenesis and may lead to the identification of new pathologic X-linked transcripts implicated in disease susceptibility and progression.

Education & Training Awards

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

EDUCATION & TRAINING AWARDS

Clinician Scholar Educator Award

The Clinician Scholar Educator Award supports educators dedicated to developing new and improved programs to enhance education in musculoskeletal and rheumatic diseases for future doctors and rheumatology health professionals.

CLINICIAN SCHOLAR EDUCATOR AWARD

Anisha Dua, MD, MPH

Northwestern University

Rheum2Teach: A Medical Educators Program for Rheumatology Fellows

Rheum2Teach will be designed to teach rheumatology fellows how to enhance their skills as educators using asynchronous video lectures, learning prompts, and a synchronous workshop which will provide opportunities for practice, re-enforcement and feedback. After completion of this curriculum, the fellow will:

1. Develop skills in presentation delivery and design,

2. Identify features of good feedback and practice applying those skills in a safe environment with opportunities for structured mentoring and critical appraisal

3. Identify the characteristics of a positive learning environment and demonstrate the ability to establish one

4. Practice creating and writing goals and objectives

5. Identify basic foundational principles in adult learning theory and curriculum design

As interest in clinical education among our trainees increases, I anticipate that interest in this curriculum will be high. I have attained the educational background and experience to develop such a curriculum for rheumatology fellows-in-training (FITs). Rheum2Teach will allow me to leverage my experience in medical education, including the application of adult teaching methodologies, to create an innovative curriculum that will help train the next generation of educators.

CLINICIAN SCHOLAR EDUCATOR AWARD

Sonam Kiwalkar, MBBS

Good Samaritan Foundation

Impact of Redesigning Rheum2Learn: A Comparison of Knowledge, Confidence, and Clinical Reasoning in Residents Using Virtual Patients vs. Text-Based Modules in a Randomized Controlled Trial

According to the American College of Rheumatology's (ACR) 2015 Rheumatology Workforce Study, it is estimated that the U.S. will need an additional 4,729 adult rheumatologists by 2030 to meet the growing patient demand. One of the ways to bridge this gap is to increase interest and exposure to rheumatology among residents, future front-line physicians, who will be diagnosing, managing, and referring patients with rheumatologic diseases. Consequently, in 2012, ACR created fifteen text-based modules called Rheum2Learn (R2L) targeted to meet the needs of residents. Using virtual patients (VPs) as the instructional strategy, under the leadership of Dr. Kiwalkar, these text-based modules are currently being redesigned and updated to Rheum2Learn 2.0.

Previous studies have shown that VPs foster clinical reasoning, but there is a paucity of studies in graduate medical education that compare the efficacy of VPs to text-based modules. The purpose of this study is to examine the educational impact on the knowledge, confidence, and learning of clinical reasoning, with VP modules (from R2L 2.0) compared to text-based modules (from existing version of R2L) in a multi-site, randomized cross-over study design.

Successful implementation of this study will establish a scientific foundation for the future creation and utility of VP modules in graduate medical education. This, in turn, will support the efforts of the ACR to fund and complete the creation of future VP modules for Rheum2Learn 2.0 project. This project has the potential to ensure that the current generation of residents in training is adequately prepared to graduate into a clinical practice environment where they can confidently diagnose, manage and refer patients with rheumatologic conditions. The Rheum2Learn 2.0 project will be a durable resource that can be used by other learners in the future and support the Rheumatology Research Foundation's mission of improving the care of patients with rheumatic disease and advancing rheumatology as a subspecialty.

EDUCATION & TRAINING AWARDS

Fellowship Training Award for Workforce Expansion

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 or that has created a new training slot. It can also be awarded to a new training program.

Augusta University
Icahn School of Medicine at Mount Sinai
Indiana University
University of Alabama at Birmingham
University of Utah
UPMC Children's Hospital of Pittsburgh
Virginia Commonwealth University
Yale University

Funding for these awards is provided in part by the Andrejeski Fund for Fellowship Training.

EDUCATION & TRAINING AWARDS

Fellowship Training Award

The Fellowship Training Award supports the training of a rheumatology fellow to provide a more robust and highly trained workforce to care for people with rheumatic disease.

Baylor College of Medicine (Pediatric)
Beth Israel Deaconess Medical Center
The Children's Hospital of Philadelphia
Cincinnati Children's Hospital Medical Center
Duke University (Adult)
Duke University (Pediatric)
Georgetown University
Johns Hopkins University
Larkin University
Louisiana State University Health Sciences
Massachusetts General Hospital
New York University
Northwestern University
Oregon Health & Science University
Stanford University
Tufts Medical Center
University of Alabama at Birmingham
University of Arizona
University of California, San Diego
University of California, San Francisco
University of California, Los Angeles
University of Chicago (Adult)
University of Chicago (Pediatric)
University of Michigan
University of Minnesota (Adult)
University of Minnesota (Pediatric)
University of Nebraska Medical Center
University of Pennsylvania

Funding for these awards is provided in part by Amgen, Inc.

EDUCATION & TRAINING AWARDS

Paula de Merieux Fellowship Training Award

The Paula de Merieux Fellowship Training Award provides support for the training of a promising rheumatology fellow who is an under-represented minority or a woman.

Washington University in St. Louis

EDUCATION & TRAINING AWARDS

State of Texas Association of Rheumatologists (STAR) Fellowship Training Award Endowment

Established in 2022, the State of Texas Association of Rheumatologists (STAR) Fellowship Training Award Endowment will cover the cost of one Fellowship Training Award received by an adult rheumatology fellowship training program in the state of Texas per year.

Baylor College of Medicine

EDUCATION & TRAINING AWARDS

Health Professional Online Education Grant

The Health Professional Online Education Grant covers the cost of registration for either the Fundamentals of Rheumatology Course or the Advanced Rheumatology Course to increase the knowledge and skills of rheumatology health professionals to ensure that they are better equipped to meet the needs of a growing rheumatology patient population.

Angela Hackney, RN, BSN

Margaret Coyle, DNP, FNP-BC

Mariah Hamilton, PA-C

Payton Nance, PA-C

EDUCATION & TRAINING AWARDS

Mentored Nurse Practitioner/ Physician Assistant Award for Workforce Expansion

The Mentored Nurse Practitioner/Physician Assistant Award for Workforce Expansion provides tailored training for nurse practitioners or physician assistants who are new to the field of rheumatology and who are in geographically underserved areas.

MENTORED NURSE PRACTITIONER/PHYSICIAN ASSISTANT AWARD FOR WORKFORCE EXPANSION

Advanced Rheumatology of Houston
Tamar Brionez, MD
Kimberly Louangpho, MSN, APRN, FNP-C

Allergy ARTS
Nicole Davey-Ranasinghe, MD
Abby Schlumpf, MPAS, PA-C

Arthritis Clinic of Cypress and Katy, PA
Aman Kugasia, MD
Aracely Kundmueller, APRN, MSN

Arthritis Consultants of Tidewater
Tatiana Keck, MD, MBA
Norjan Faye Tomawis, FNP-C

Asheville Arthritis and Osteoporosis Center
Sunil Abraham, MD, FACR
Haley Ballard, PA-C

Capital Rheumatology
Priyank Chaudhary, MD
Rebecca Unger, PA-C

Geisinger Medical Center
Kirsten Koons, MD, RhMSUS
Celia Prince, CRNP

Integrative Rheumatology of South Texas
Naiara Alvarez, MD
Belinda Garza, DNP, FNP

Loma Linda University Health
Karina Torralba, MD, MACM
Alpha Gonzalez, FNP-BC

Massachusetts General Hospital
Minna Kholer, MD, RhMSUS
Christopher Estes, MSN, FNP-BC, CNRN, GERO-BC

New Haven Rheumatology
Robert Schoen, MD, MBA
Mary Elliott, PhD, MMSc

Oregon Health & Science University
Atul Doedhara, MD, MRCP
Eliza Thompson, FNP

Penn State Health Rheumatology
Nancy Olsen, MD
Hongli Shi, MSN, BSN, RN

Peterson Specialty Care
Jammie Barnes, MD
Lindsey Neel, FNP

South Charlotte Rheumatology
Firas Kassab, MD, FACR
Caleb Huffman, FNP, MS

Texas Children's Hospital
Eyal Muscal, MD, MS
Shelby Brooks, MSN

The Feinstein Institutes for Medical Research
Beth Gottlieb, MD, MS
Nina Skaria, RN, CPNP-PC

The Ohio State University
Alex Meara, MD, MS
Mary Caldwell, MSN

MENTORED NURSE PRACTITIONER/PHYSICIAN ASSISTANT AWARD FOR WORKFORCE EXPANSION

UCLA - Olive View Medical Center
Sunica Volkov, MD
Chloe Shin, FNP

University of Alabama at Birmingham
Matthew Mullen, MD
Elizabeth Dye, MSN

University of Alabama at Birmingham
Emily Smitherman, MD
Bethany Walker, CRNP

University of Kentucky Research Foundation
Kristine Lohr, MD, MS
Hannah Brugger, MSN, BSN

University of Utah Pediatric Rheumatology
Karen James, MD, MSCE
Kate Thompson, MSN, APRN, FNP-C

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.

PRECEPTORSHIPS

Rheumatology Future Physician Scientist Award

The Rheumatology Future Physician Scientist Award supports MD/PhD students who demonstrate outstanding potential and significant commitment to a career in rheumatology research, in order to support the nation’s top emerging physician scientists in the field of rheumatology.

RHEUMATOLOGY FUTURE PHYSICIAN SCIENTIST AWARD

Emily Balczewski, BA

University of Michigan

Characterizing Diagnostic Delay in Systemic Lupus Erythematosus and Antiphospholipid Syndrome

Reducing time from the presenting symptom to diagnosis is a key area of need in SLE and APS. Prompt diagnosis and treatment can reduce these diseases' often substantial burden and improve long-term outcomes. Significant diagnostic delay of months to years has been shown in several studies for both SLE and APS, but the factors which contribute to this delay are poorly understood. The objective of this project is to use the electronic health record (EHR) to a) evaluate the extent of diagnostic delay in US populations, b) identify important features (such as number and type of healthcare visits) that contribute to this delay, and c) assess the ability of these important features to predict future diagnosis of SLE and APS.

To evaluate the extent of diagnostic delay, we will construct a timeline for each patient in two EHR databases from initial presenting symptom of SLE and/or APS to diagnosis. These timelines will include EHR-derived diagnosis codes, laboratory tests, medication changes, and clinical encounters. The length of a patient's timeline which results in SLE/APS diagnosis is their diagnostic delay. To identify important EHR features which may contribute to diagnostic delay, we will cluster these timelines to group patients with similar paths to diagnosis or non-diagnosis; by interrogating the differences between these groups, we can begin to identify important factors which may accelerate or delay diagnosis. To further investigate these important factors, we will build a discrete-time survival model (DTSM)—a type of logistic regression model used for longitudinal data—to predict time-to-diagnosis from initial presenting symptom. This model can tell us a) how well we can predict diagnosis from EHR factors and b) what factors are most important to this prediction. Finally, we will select individuals with a high predicted probability of diagnosis who do not have a current diagnosis and evaluate the possibility of a missed diagnosis through expert chart review.

We hope that the insights generated by this project will surface ideas for future study and highlight possible interventions to accelerate diagnosis and improve the health of patients with SLE and APS.

Resident Research Preceptorship

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.

Alysson Covello, MD
Michael Pillinger, MD, FACP (Preceptor)
New York University School of Medicine

Sarah Smith, MD
Paula Ramos, PhD, MSc (Preceptor)
Medical University of South Carolina

PRECEPTORSHIPS

Lawren H. Daltroy Preceptorship in Health Communication

The Lawren H. Daltroy Preceptorship in Health Communication was established with the aim of improving patient-clinician interactions and communications.

Cindy Chiu, DO
Jenna Thomason, MD, MPH (Preceptor)
University of Washington

Medical & Graduate Student Preceptorship

The Medical and Graduate Student Preceptorship encourages medical and graduate students to consider a career in rheumatology by supporting a clinical or research mentorship with an established rheumatology professional.

MEDICAL & GRADUATE STUDENT PRECEPTORSHIP

Maya Adams*
Benjamin Chong, MD (Preceptor)
University of Texas Southwestern Medical Center

Justin Arnold*
Rosalind Ramsey-Goldman, MD, DrPH (Preceptor)
Northwestern University

Caroline Bauchiero
Joerg Ermann, MD (Preceptor)
Brigham and Women's Hospital

Iagn Nino Kenji Cabahug
Noelle Rolle, MBBS, FACR (Preceptor)
The Medical College of Georgia at Augusta University

Paula Caras*
Shanthini Kasturi, MD (Preceptor)
Tufts Medical Center

Anna Deck
Shanthini Kasturi MD, MS (Preceptor)
Tufts Medical Center

Kaila Fennell
Patrick Corrigan, PT, DPT, PhD (Preceptor)
Saint Louis University

Sabrina George, MS
James Jarvis, MD (Preceptor)
Jacobs School of Medicine & Biomedical Sciences University at Buffalo

Rachael Hart
Bharat Kumar, MD, MME, FACP, RhMSUS (Preceptor)
University of Iowa

Celestine He
Chrisanna Dobrowolski, MD (Preceptor)
Icahn School of Medicine, Mount Sinai

Brendan Hughes, MS
Daniel White, PT, ScD, MSc (Preceptor)
University of Delaware

Jessie Jarrell
JoAnn Zell, MD (Preceptor)
University of Colorado Denver, AMC and DC

Heejo Keum
Heidi Jacobe, MD, MSCS (Preceptor)
University of Texas Southwestern Medical Center

Aleksandra Kostic
Amanda Nelson, MD, MSCR, RhMSUS (Preceptor)
The University of North Carolina at Chapel Hill

Meisui Liu***
Marcy Bolster, MD (Preceptor)
Massachusetts General Hospital

Faye Megaris*
Kyriakos Kirou, MD (Preceptor)
The Hospital for Special Surgery

Sarah Novroski
Louise Thoma, PT, DPT, PhD (Preceptor)
University of North Carolina at Chapel Hill

Andrew Pechstein, PhD
Daniel White, PT, ScD, MSc (Preceptor)
University of Delaware

** Funding for these awards is provided by the Marc R. Chevrier, MD, PhD, FACR, Lupus Research Memorial Fund.*

**** Funding for these awards is provided by the Majithia Family Endowment.*

MEDICAL & GRADUATE STUDENT PRECEPTORSHIP

Austin Pan
Jennifer Stichman, MD (Preceptor)
University of Colorado Denver, AMC and DC

Samantha Price, DPT
Patrick Corrigan, DPT, PhD (Preceptor)
Saint Louis University

Natalie Schanzer
Joshua Baker, MD, MSCE (Preceptor)
University of Pennsylvania

Nirali Shah, MPT
Deepak Kumar, PhD (Preceptor)
Boston University

Rohan Sharma
Ram Singh, MD (Preceptor)
University of California, Los Angeles

Hayley Smitheman, DPT**
Karin Gravare Silbernagel, PT, ATC, PhD (Preceptor)
University of Delaware

Kimberly Tran
Debendra Pattanaik, MD (Preceptor)
University of Tennessee Health Science Center

Elise Travis, MA***
Bryce Binstadt, MD, PhD (Preceptor)
University of Minnesota

Rathnam Venkat**
Jeffrey Sparks, MD, MSSc (Preceptor)
Brigham and Women's Hospital

David Werner, DPT**
Elizabeth Wellsandt, PT, DPT, PhD, OCS (Preceptor)
University of Nebraska Medical Center

Claire Yang***
Yongdong (Dan) Zhao, MD, PhD (Preceptor)
Seattle Children's Hospital

Emma Zeng
Julie Paik, MD, MHS (Preceptor)
Johns Hopkins University School of Medicine

*** Funding for these awards is provided by the Daniel J. Wallace, MD Graduate Student Preceptorship Endowment.*

**** Funding for these awards is provided by the Majithia Family Endowment.*

ABSTRACT AWARDS AND SCHOLARSHIPS

Cultivating an interest in rheumatology is essential to ensuring students, residents and fellows pursue careers in the field and help fill shortages, both academically and geographically. The Abstract Awards and Scholarships recognize scholarship among aspiring rheumatology professionals at the ACR Convergence and Pediatric Rheumatology Symposium (PRSYM).

Pediatric Rheumatology Symposium (PRSYM) Abstract Award

The purpose of the Pediatric Rheumatology Symposium Abstract Award is to provide outstanding students, residents and fellows the opportunity to attend and present an abstract at the 2023 Pediatric Rheumatology Symposium.

Jonathan Li, MD
University of Pittsburgh

David McDonald
Baylor College of Medicine

Kristina Nasto
Baylor College of Medicine

Stephanie Wood
Baylor College of Medicine

Claire Yang
University of Washington

The Rheumatology Research Foundation is able to fund cutting-edge research and the training of the next generation of rheumatology professionals because of the generous support of our donors, thank you.

If you would like to learn more about how you can make an impact at the Rheumatology Research Foundation, please contact us at foundation@rheumatology.org.

RheumResearch.org

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