



Rheumatology Research Foundation

Advancing Treatment | Finding Cures

ANNUAL REPORT 2012





David I. Daikh, MD, PhD

PRESIDENT'S MESSAGE

To fully appreciate the Foundation's present and properly prepare for its future, it's important to understand its past.

The American College of Rheumatology established the Research and Education Foundation in 1985 as a way to raise funds for those developing careers as rheumatologists and in research. In its early years, a modest portfolio of programs was funded at less than \$100,000 a year. That portfolio had grown to \$500,000 in 1993. In 2001, we finally crossed the \$1 million mark and from that point, the upward trend became remarkable. By 2005, our research funding was \$4 million; three years later, it was over \$10 million. Every year, it seems, we set a new record for research support. That's quite an accomplishment, but it's also a tremendous responsibility.

All of this success also prompted a look inward. As an organization had we reached the point of where we needed to be? The Foundation has grown into largest private funding source of rheumatology research and training programs in the U.S., yet it is clear that there remains a lot of work to do in these areas.

In response to those needs and the opportunities provided by all those who support the work of the Foundation, we are now focused on moving forward in new, exciting directions. In 2012, building on more than a quarter of a century of success, the ACR REF changed its name to the Rheumatology Research Foundation.

Our new name more clearly conveys our identity, allowing us to strengthen our bonds with the ACR community while offering exciting opportunities to introduce our mission, achievements and discoveries to a new audience—patients and families who care about uncovering the causes, advancing treatment and finding cures for rheumatic diseases.

I am proud to serve as the Foundation's first president—in a manner of speaking. We are not a new organization, our mission has not changed, but we are progressing quickly and nowhere is this progress more visible than in the \$60 million Journey to Cure campaign. You'll learn more about Journey to Cure and how it aims to advance patient care and accelerate discoveries in rheumatic disease research within this annual report.

Today, the Foundation is leading the way in a new era of rheumatology support that focuses on advancing research and training to improve the health of people with rheumatic diseases.

Now is a great time to be the Rheumatology Research Foundation. Perhaps the only thing that can surpass it is our future.

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“I have had a great experience with the Scientist Development Award. It provided exactly the support I needed to begin my career as a clinical investigator with tuition and research support in addition to salary support. I enjoyed and benefited greatly from my coursework which was uniquely relevant to me as a practicing clinician and clinical researcher. My courses have provided a valuable knowledge base that will act as a foundation for future grant proposals, research work and mentoring as I progress in my career in academic rheumatology. I do not think that I would have had such success in my early career without support from this award.”

– **Amanda Nelson, MD**

Assistant Professor, University of
North Carolina School of Medicine



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FOUNDATION AWARDS AND GRANTS



11/1/2018

PRECEPTORSHIPS

Medical Student Clinical Preceptorship

This preceptorship introduces students to the specialty of rheumatology by supporting a full-time clinical opportunity. Students receive a stipend and reimbursable travel funds to attend the ACR/ARHP Annual Meeting. Funding is made possible through the Abbott Endowment for Rheumatology Development.

Jeffrey Bartsch

Preceptor: Joel M. Hirsh, MD
Denver Health Medical Center

Binoy Bhatt

Preceptor: James Katz, MD
George Washington University

Cathryn Byrne-Dugan

Preceptor: Vladimir M. Ognenovski, MD
University of Michigan

Megan Q. Chan

Preceptor: Katherine K. Temprano, MD
St. Louis University

Lindsay M. Dittman

Preceptor: Philip L. Cohen, MD
Temple University

Elisheva Douglas Frankel

Preceptor: Murray H. Passo, MD, MEd
Medical University of South Carolina

Mithu Maheswaranathan

Preceptor: Diane Kamen, MD
Medical University of South Carolina

Courtney Kiersten Pollard

Preceptor: Robert W. Janson, MD
University of Colorado, Denver

Mia Taylor

Preceptor: Marcy B. Bolster, MD
Medical University of South Carolina

Brandon L. Todd

Preceptor: Charles R. Arkin, MD
Rheumatology & Osteoporosis
Center of Memphis, P.C.

Medical Student Research Preceptorship

This preceptorship introduces students to the specialty of rheumatology by supporting a full-time research experience. Students receive a stipend and reimbursable travel funds to attend the ACR/ARHP Annual Meeting. Funding is made possible through the Abbott Endowment for Rheumatology Development.

Jeffrey G. Clark

Preceptor: Steven J. Spalding, MD
Cleveland Clinic Foundation

Kristen E. Graeber

Preceptor: Nancy J. Olsen, MD
Pennsylvania State MS Hershey Medical Center

Lindsey E. Harward

Preceptor: Megan E.B. Clowse, MD, MPH
Duke University

Sarah M. Jacks

Preceptor: Virginia D. Steen, MD
Georgetown University

Boram Kim

Preceptor: Anne Davidson, MBBS
Feinstein Institute for Medical Research

Eric J. Lee

Preceptor: Kathleen M. O'Neil, MD
University of Oklahoma Health Sciences Center

Nicki Nabavizadeh

Preceptor: James N. Jarvis, MD
University of Oklahoma Health Sciences Center

Irina Perjar

Preceptor: Joanne Jordan, MD, MPH
University of North Carolina
at Chapel Hill

Atousa Sobhi

Preceptor: Susan A. Boackle, MD
University of Colorado, Denver

Neil Tailor

Preceptor: James C. Oates, MD
Medical University of South Carolina

Xue (Rose) Tian

Preceptor: Natasha M. Ruth, MD, MS
Medical University of South Carolina

Darneesh Thornton-Johnson, MS

Preceptor: Daniel A. Albert, MD
Dartmouth-Hitchcock Medical Center

Stephanie J. Wilburn

Preceptor: Andrea Lynn Sestak, MD, PhD
University of Oklahoma Health
Sciences Center

Health Professional Research Preceptorship

This preceptorship introduces students to rheumatology-related health care by supporting full-time research by a graduate student in the area of rheumatic diseases. Students receive a stipend and reimbursable travel funds to attend the ACR/ARHP Annual Meeting. Funding for this award is made possible through the Abbott Endowment for Rheumatology Development.

Alexandra H. Antonioli

Preceptor: V. Michael Holers, MD
University of Colorado, Denver

Erin Arthurs

Preceptor: Brett D. Thombs, PhD
McGill University

Amanda B. Feinstein, MS

Preceptor: Sampath Prahalad, MD
Emory Children's Center

Ilya Razykov

Preceptor: Brett D. Thombs, PhD
McGill University

Roopa Akkineni

Preceptor: Daniel A. Albert, MD
Dartmouth-Hitchcock Medical Center

Jennifer M.P. Woo

Preceptor: Deborah McCurdy, MD
University of California, Los Angeles

Ephraim P. Engleman Endowed Resident Research Preceptorship

This preceptorship introduces residents to the specialty of rheumatology by supporting a full-time, mentored research experience. The award is funded for one year at \$15,000. Funding is made possible through the generous financial support of the Ephraim P. Engleman Endowment.

Tamara Augustin

Preceptor: John Stone, MD
North Shore Medical Center

EDUCATION and TRAINING

Clinician Scholar Educator Award

This award recognizes and supports rheumatologists who are dedicated to providing a high-quality clinical educational experience to future rheumatologists. It is funded for three years at up to \$60,000 annually.



Juliet Aizer, MD, MPH
Hospital for Special Surgery

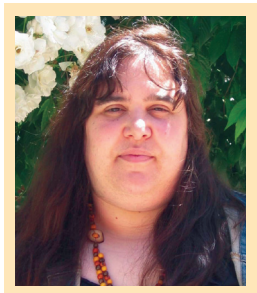
Development of curricula in metabolic bone disease

Available curricular materials for metabolic bone education in rheumatology are lacking in scope or structure.

This project specifies four specific aims: (1) to implement a unified, interactive curriculum in metabolic bone education for rheumatology trainees, residents and students; (2) to construct a set of instruments for measuring learner attitudes, knowledge, competence and performance regarding metabolic bone disease; (3) to enhance teaching skills of rheumatology fellows; and (4) to increase medical student and resident interest in rheumatology.

Rheumatology fellows (n=13) will participate in six interactive sessions on metabolic bone disease. Fellows will be mentored to lead sessions for medical residents and students on metabolic bone disease. Data will be collected at baseline and after completion of the curriculum on fellow attitudes, knowledge, competence and performance, and student and resident attitudes.

After implementation of this metabolic bone curriculum, investigators expect that rheumatology fellows will show greater awareness of metabolic bone issues in their rheumatology outpatients, as evidenced by documentation in the chart of appropriate evaluation, treatment and counseling. Fellows will report enhanced comfort in handling metabolic bone issues, demonstrate greater knowledge about metabolic bone issues, and show greater competence in assessing and communicating fracture risk after completion of the curriculum. Residents and students will report greater interest in rheumatology and metabolic bone disease after the sessions.



Michal Jennifer Cidon, MD
Lucile Packard Children's Hospital/Stanford University Medical Center

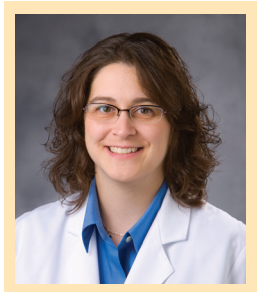
Web-based learning portfolios in pediatric rheumatology

The national deficit of pediatric rheumatologists compounded by recent changes in residency training significantly impacts residents' exposure to the field of pediatric rheumatology, as well as their preparation in managing rheumatic illness and chronic illness in children. In response to these training constraints, this project will develop a web-based construct called Web-Based Learning Portfolios (WBLP) that supports a longitudinal experiential curriculum through simulated problem-based learning in pediatric rheumatology. In order to create dynamic learning environments

where residents can self-reflect and individually authenticate their learning process, learning activities within the curriculum (e.g., discussion questions about particular topics, evidence based review and links to reference didactic lessons) will be developed to enhance content interaction as well as instructional interaction with educators and peers. Learning and teaching transparency within the WBLP will be developed by (1) linking the web-based construct's content to ACGME (Accreditation Council on Graduate Medical Education) competencies; and (2) strategically embedding formative assessments within it. WBLPs will be pilot-tested on pediatric interns (PL-1) in a multi-institutional, two-arm randomized phase control trial. Measures of resident learning outcome will involve (1) the Script Concordance Test for Pediatric Rheumatology (SCT-PR), a validated clinical reasoning tool; 2) the Pediatrics In-Training Examination, a knowledge acquisition and application test; and (3) a validated user survey assessing learner satisfaction with the WBLP construct. The effectiveness of a web-based learning tool will enable its widespread dissemination to enhance clinical instruction and problem solving in pediatric rheumatology within pediatric residency training.

EDUCATION and TRAINING

Clinician Scholar Educator Award *cont.*



Lisa G. Criscione-Schreiber, MD

Duke University

Development of ROSCE stations with follow-up modules

Objective Structured Clinical Examinations (OSCE) are especially useful for assessing complicated constructs such as the ability to formulate a differential diagnosis or how to counsel a patient. Since 2006, the Carolinas Fellows Collaborative (CFC) has annually administered a Rheumatology OSCE (ROSCE). One goal of our ROSCE is to identify areas in which individual trainees require further study and improvement. Although some trainees undertake self-study based on ROSCE performance, our data demonstrates repeated poor performance on some stations. Since trainees take the ROSCE in

two consecutive years, repeated poor performance shows that trainees do not uniformly adopt a program of study based on identified deficiencies.

Investigators hypothesize that a ROSCE can lead to measureable improvement in fellows' learning if identified deficiencies lead directly to a monitored educational program. As per adult learning theory, if the educational modules are directed toward problems identified in a real or plausible simulated clinical situation, knowledge gained will be in-depth and durable.

Specifically, investigators will (1) develop up to eight validated ROSCE stations; (2) create follow-up educational modules linked to performance on the ROSCE; and (3) disseminate the ROSCE stations and follow-up modules nationally.

This project will take advantage of an existing regional collaboration among four rheumatology fellowship programs to develop a validated educational product. The final product will be (1) an instructional manual containing all materials necessary for administering a ROSCE; (2) web-based educational modules accessible by trainees who have completed said ROSCE; and (3) train-the-trainer sessions to prepare program directors to administer a ROSCE and the follow-up modules. This product will allow rheumatology fellowships to incorporate the ROSCE into their training programs' assessment and teaching portfolios.



Rodney Tehrani, MD

Loyola University Medical Center

A dynamic, competency-based learning module

Learning management systems facilitate the delivering, tracking and management of training and education. The project will implement a medical learning management system for rheumatology that will facilitate the creation of 10 competency-based, computer-adaptive testing modules for medicine residents and rheumatology fellows. The difficulty of the modules will be developed for the level of the test taker.

The medicine residents will complete the modules as they rotate through rheumatology. The primary goal of the modules will be to enhance their educational experience and knowledge base. In addition, the modules also will serve as a tool for attracting more residents into a career in rheumatology by giving them an earlier exposure into our subspecialty.

The distinctive feature about the modules is that they will be dynamic and able to adapt and change questions and scenarios based on responses to previous questions. Their effectiveness can then be tracked temporally and used to pinpoint areas of weakness in the training program's curriculum. The modules also will provide another tool in the evaluation of the six competencies put forward by the Accreditation Council of Graduate Medical Education.

Web-based learning is an underutilized tool in medical training. It has the advantage of being efficient and easily accessible for training. It seems inevitable that web-based learning will become a critical and invaluable component of post graduate and subspecialty training in the near future.

EDUCATION and TRAINING

Amgen Pediatric Visiting Professorship

This program provides educational opportunities for medical students and residents in institutions where no established pediatric rheumatology expertise currently exists. Each visiting professor receives an honorarium and reimbursement for expenses. Funding is made possible through an endowment provided by Amgen.

Randy Q. Cron, MD, PhD
visited Jackson Memorial Hospital

Helen M. Emery, MD
visited Children's Hospital and Research Center at Oakland

Polly Ferguson, MD
visited St. Joseph's Hospital & Medical Center

Robert Fuhlbrigge, MD, PhD
visited University of Nevada School of Medicine

Esi Morgan DeWitt, MD, MSCE
visited Howard University School of Medicine

Barry L. Myones, MD
visited Tulane University School of Medicine

Kathleen M. O'Neil, MD
visited Sinai Hospital of Baltimore

C. Eglia Rabinovich, MD, MPH
visited Carolinas Medical Center

Deborah Rothman, MD, PhD
visited SUNY Upstate Medical University

Linda Wagner-Weiner, MD
visited St. John Hospital and Medical Center

Paula de Merieux Fellowship Training Award

This award ensures that a diverse and highly trained work force is available to provide clinical care to patients with rheumatic disease. It is funded for one year at \$25,000 to support the salary of a fellow who is an underrepresented minority or a woman. Funding is made possible in part by an endowment established by the Dr. Paula de Merieux estate.

Michael Pillinger, MD
New York University School of Medicine

Amgen Fellowship Training Award

This award helps to ensure that a highly trained work force is available to provide competent clinical care to those affected by rheumatic disease. It is funded for one year at \$25,000 to support the salary of one trainee during his or her clinical year of training. Funding is made possible through the financial support of Amgen.

Michael J. Battistone, MD
University of Utah

Bryce Anthony Binstadt, MD, PhD
University of Minnesota Amplatz Children's Hospital

Ernest Brahn, MD
University of California, Los Angeles

Richard D. Brasington, MD, FACP
Washington University School of Medicine

Calvin Brown Jr., MD
Northwestern University

Amy C. Cannella, MD
University of Nebraska Medical Center

Christopher Collins, MD
Washington Hospital Center

Lisa Criscione-Schreiber, MD
Duke University

Gregory C. Gardner, MD, FACP
University of Washington

Allan C. Gelber, MD, MPH, PhD
Johns Hopkins University School of Medicine

Samina Hayat, MD
Louisiana State University Health Sciences Center Shreveport

Jennifer Huggins, MD
Cincinnati Children's Hospital Medical Center

Laura B. Hughes, MD
University of Alabama at Birmingham

Beth L. Jonas, MD
University of North Carolina at Chapel Hill

Daniel A. Kietz, MD
University of Pittsburgh School of Medicine

Sharon Kolasinski, MD
University of Pennsylvania

Bonita S. Libman, MD
University of Vermont and Fletcher Allen Health Care, Inc.

Tzielan Lee, MD
Stanford University

Carlos J. Lozada, MD
University of Miami Miller School of Medicine

Deborah McCurdy, MD
University of California, Los Angeles

Terry L. Moore, MD
Saint Louis University

James T. Rosenbaum, MD
Oregon Health and Science University

C. Eglia Rabinovich, MD, MPH
Duke University Medical Center

David D. Sherry, MD
Children's Hospital of Philadelphia

Eric S. Sobel, MD, PhD
University of Florida

Virginia Steen, MD
Georgetown University

Robert Terkeltaub, MD
University of California, San Diego

Tammy Olsen Utset, MD, MPH
University of Chicago

Emily von Scheven, MD, MAS
University of California, San Francisco

Sterling G. West, MD
University of Colorado, Denver

EDUCATION and TRAINING

Training Program Development Award

The purpose of this award is to help ensure that a diverse and highly trained workforce is available to provide competent clinical care to those affected by rheumatic diseases. The Foundation will support two pilot awards for one year at \$50,000 each. These awards are designed to specifically address three objectives, listed below in priority order:

- 1. Creating a new fellow position, preferably one that was previously ACGME-accredited but was unfilled due to lack of sufficient fellow salary funding.*
- 2. Increasing the number of underrepresented minority* trainees.*
- 3. Providing a slot committed to trainees with a high likelihood of pursuing an academic rheumatology career.*

**For purposes of this pilot program, “underrepresented minority within rheumatology” shall mean black, Hispanic or Native American (American Indians, Alaska natives, and native Hawaiians).*

Stanley Ballou, MD
MetroHealth Medical Center

Anne M. Stevens, MD, PhD
Seattle Children's Hospital

“I continue to be committed to an academic research career in rheumatology. Funding from the Investigator Award allowed me to develop a research project and obtain a KO8 grant from NIAMS to further this goal. It is difficult to know whether I would have been able to stay in science without the support of this award.”

– **Julia F. Charles, MD, PhD**

Instructor, Brigham and Women's Hospital

RESEARCH AWARDS

Scientist Development Award

This award encourages physicians and health professionals without significant research experience to consider careers in biomedical or clinical research by supporting a structured research training program in arthritis and rheumatic disease. Recipients receive up to three years of funding for their research project.



Anna R. Broder, MD
Albert Einstein College of Medicine

Optimizing vitamin D replacement in lupus

Vitamin D is important in regulating calcium absorption and parathyroid hormone (PTH) secretion that are essential for bone health. The relationship of vitamin D with PTH and bone turnover markers has been studied extensively in post-menopausal women and in the general population. Surprisingly, this relationship has not been studied in lupus (SLE), even though vitamin D deficiency is highly prevalent in individuals with autoimmune diseases, and vitamin D supplementation is important for preventing osteoporosis and, possibly, improving outcomes in SLE.

The main objective of this project is to evaluate whether the established definitions for vitamin D deficiency and insufficiency apply to SLE patients. Current definitions of vitamin D deficiency and insufficiency are based on the observed relationship of vitamin D levels with PTH and bone turnover in the general population and post-menopausal women. These definitions may not apply to patients with autoimmune diseases, as current definitions do not account for the demographics, prednisone use and chronic inflammation specific for autoimmune diseases.

SLE patients may have different cutoffs for vitamin D deficiency and insufficiency compared with the general population. Therefore, data will be used from a well-characterized lupus cohort to evaluate whether bone mineral density, urine calcium excretion, and bone turnover markers correlate with established cutoffs for vitamin D deficiency and insufficiency, and to explore if different cutoffs should be identified. Furthermore, the investigator will evaluate whether SLE patients maintain adequate vitamin D levels over time, after the initial replacement. This project will help to optimize vitamin D replacement therapy in SLE. The information learned from this project will help design future studies exploring the effects of vitamin D in lupus and in other autoimmune diseases.



Carmelita Colbert, MD
Northwestern University

The relationship between sagittal plane knee motion during gait and outcome in knee osteoarthritis

Knee (OA) is a leading cause of disability. Few strategies to prevent knee OA-related physical function decline and disability exist, largely due to limited knowledge of factors leading to these outcomes.

Given the central role of the knee in weight-bearing activity, sagittal plane knee range of motion during gait may influence activity choices (e.g., what and how much) and self-efficacy, factors thought to be critical to function in knee OA. Joint level impairments may play a pivotal role in the events leading to a poor person-level outcome. Investigators will evaluate several sagittal plane knee motion parameters using quantitative gait analysis, a powerful, established tool which yields measurements during the most common locomotor activity humans perform.

The primary hypotheses deal with a risk factor at baseline and outcome evaluated over the subsequent two-year period. Investigators will examine the relationship of sagittal plane knee range of motion during gait and other gait characteristics to both function decline and to greater disability. Reduced knee range of motion during gait may contribute to these outcomes in knee OA by: leading to activity modification, reducing self-efficacy, and thereby decreasing strength and aerobic capacity; directly impeding task performance; contributing to joint damage and thereby to worse functioning. Investigators will explore potential mediating roles of self-efficacy, physical activity and weakness. Elucidation of the impact of reduced dynamic range of motion will aid formulation of rehabilitative prevention strategies to delay function limitation and disability progression.

RESEARCH AWARDS

Scientist Development Award cont.



Lisa Anne Davis, MD

University of Colorado, Denver

Pharmacoepidemiologic methods and genetic polymorphisms

The cornerstone drug for treatment of RA is methotrexate. However, methotrexate may be associated with multiple adverse drug events (ADEs), which are injuries resulting from the use of a drug. Polymorphisms (small changes in a gene, perhaps as small as one nucleotide different) in the absorption, distribution, metabolism and excretion (ADME) genes in the folic acid pathway may contribute to methotrexate ADEs. Unfortunately, correlation between methotrexate ADME polymorphisms and ADEs has been inconsistent—some studies show an association between a certain polymorphism and ADEs, while others do not. Application of novel pharmacoepidemiologic techniques may improve the statistical modeling of methotrexate ADME polymorphisms and methotrexate ADEs. Pharmacoepidemiologic techniques account not only for the presence of a drug, but additional information such as dosage of the drug, length of time on the drug, how the drug is administered, etc. Pharmacoepidemiologic techniques have not been commonly employed in gene-association studies.

It is hypothesized that the incorporation of pharmacoepidemiologic techniques will improve statistical modeling of methotrexate ADME polymorphisms and methotrexate-associated ADEs. Our aims for this project are: (1) to quantify the methotrexate-associated ADEs in patients with rheumatoid arthritis from the Veterans' Affairs Rheumatoid Arthritis (VARA) registry; (2) to create a "standard model" to predict methotrexate ADEs based on patients' ADME gene polymorphisms; and (3) to incorporate pharmacoepidemiological methods to improve the statistical model, using such techniques as medication exposure algorithms, adherence measures and time-to-event analysis.

Applying these novel pharmacoepidemiologic methods may refine statistical modeling methods and may have the potential to dramatically shift the paradigm for analyzing genetic polymorphism association studies, medication interaction studies and cumulative toxicity studies.



Angelica Gierut, MD

Northwestern University

P21: A novel suppressor of rheumatoid arthritis

The cyclin dependent kinase inhibitor, p21, has a role in suppression of inflammation and autoimmunity independent of its function as a cell-cycle regulator. The laboratory of Harris Perlman, PhD, has already established that p21 deficiency increases mortality from LPS-induced shock and augments experimental arthritis in mice. However, little information exists regarding the modulation of inflammation and arthritis by p21 specifically in macrophages. Macrophages are key effector cells in RA, and their synovial sublining number may be used as a biomarker for disease activity. This project will determine the effect of macrophage-specific p21 deficiency on experimental arthritis and macrophage differentiation. It also will investigate whether p21 may be used as a biomarker for disease activity in RA patients. An adoptive transfer model will be employed in which circulating monocytes are specifically deleted using a CD11b-diphtheria toxin receptor construct. The expectation is that both wild type (WT) and p21^{-/-} mice reconstituted with p21^{-/-} bone marrow will have the most severe arthritis, and depletion of circulating monocytes using diphtheria toxin will resolve disease. Secondly, the effect of p21 deficiency on macrophage differentiation using in vitro assays of WT and p21^{-/-} bone marrow-derived, and peritoneal macrophages will be tested. It is expected that p21^{-/-} macrophages will display less markers of alternative activation. Investigators also will pretreat WT and p21^{-/-} macrophages with a p21 peptidomimetic and observe the effects on macrophage differentiation. Finally, the use of p21 as a biomarker for disease activity in RA using immunohistochemical analysis of RA patient synovial samples before and after the initiation of therapy will be tested. It is expected that therapeutic response will correlate with levels of p21 in the tissues. Thus, this project will examine the relationship between p21 and macrophages in RA and experimental arthritis and aims to deliver new insights into RA pathogenesis.

RESEARCH AWARDS

Scientist Development Award cont.



Peter C. Grayson, MD
Boston University Medical Center

Nasal epithelial genomics in Wegener's granulomatosis

Nasal disease occurs in the majority of patients with Wegener's granulomatosis (WG) and is often a presenting symptom of the disease. Despite the burden of nasal disease in WG, the molecular mechanisms underlying upper respiratory disease in WG are poorly understood. Gene expression profiling is an emerging technology whereby thousands of genes can be measured simultaneously to create a global picture of cellular function. A few studies of gene expression profiling using circulating blood cells as a tissue source have been conducted in WG, but no study has examined whole-genome expression using nasal tissue. The investigators have pioneered noninvasive methods to collect and extract RNA from nasal epithelial cells and, using these techniques, have generated preliminary data which suggests that WG is associated with alterations in gene expression in nasal epithelial cells. The central hypothesis of the proposed research is that whole-genome gene expression profiling of nasal epithelial cells can be used to (1) differentiate WG from non-WG; and (2) identify subtypes of WG based on both clinical and molecular data. By comprehensively studying nasal epithelial cell gene expression patterns in WG, investigators hope to gain insight into the molecular mechanisms underlying WG and to discover novel biomarkers of disease and disease activity.



J. Michelle Kahlenberg, MD, PhD
University of Michigan

The inflammasome in lupus-related vascular disease

Systemic lupus erythematosus (SLE) is an autoimmune disease with heterogeneous manifestations including severe organ damage and a greatly increased risk of cardiovascular disease. Interferon-alpha (IFN- alpha) is a cytokine that has been shown to have an important role in the development of lupus and lupus-related vascular dysfunction. The investigators have found that SLE patients have increased endothelial progenitor cell (EPC)/circulating angiogenic cell (CAC) apoptosis and impaired differentiation, which contributes to endothelial dysfunction and the development of cardiovascular disease. This EPC/CAC dysfunction is mediated by effects of IFN-alpha. The investigators have found that components of the molecular platform, the inflammasome, are regulated by IFN- alpha in EPC/CAC cultures. The goal of their research is to understand the downstream effects of IFN- alpha in SLE; specifically by examining its effects on inflammasome activity and cytokine production, which may lead to an increased risk of cardiovascular disease as well as modulation of disease activity. This will be explored by using both human in vitro and murine in vivo models in which inflammasome activity can be studied and modulated.

"Because I had this award to fund my research, my career has continued to progress. In July 2012, I was hired as an assistant professor in medicine at the University of California-San Francisco following a year as instructor. I have applied and successfully competed for K-level funding. I continue to pursue my research projects and be active in policy through the American College of Rheumatology Quality Measures Subcommittee work. This would not have been possible without the Scientist Development Award."

- **Gabriela Schmajuk, MD**

Assistant Professor, University of California, San Francisco

RESEARCH AWARDS

Scientist Development Award cont.



Alfred Hyoungju Kim, MD, PhD

Washington University School of Medicine

The role of B cell-derived cytokines in kidney disease

Numerous autoimmune and immune-mediated glomerular diseases are associated with proteinuria and glomerulopathy. B cell depletion therapies have been efficacious in the treatment many of these diseases. The most interesting disease where B cell depletion has demonstrated efficacy is minimal change disease (MCD). MCD is characterized by nephrotic-range proteinuria with podocyte foot process effacement. Notably lacking is the absence of glomerular inflammation, immune complexes, autoantibodies or complement deposition on the glomeruli. The investigator hypothesizes B cell

derived cytokines play a critical role in the induction of proteinuria, and that B cells mediate their pathogenic activity within the glomeruli following intraglomerular activation. To test this, the investigator developed a murine model of B cell induced proteinuria utilizing the B cell model antigen, hen egg lysozyme (HEL). Following delivery of HEL to the glomerular basement membrane, HEL-specific B cells are transferred into those mice. Proteinuria was induced with pathologic features mimicking minimal change disease.

This hypothesis will be tested with this model with the following specific aims: (1) to characterize this model of B cell-mediated proteinuria, and this will be tested with the following subaims—(a) to observe intraglomerular B cell activation utilizing intravital two-photon microscopy, (b) to target non-specific B cell activators to the glomeruli to potentiate B cell responses, and (c) Determine if defective genes important in podocyte function potentiate this phenotype; and (2) identifying the cytokine(s) responsible for proteinuria and foot process effacement

The investigator expects to find intraglomerular activation of antigen-specific B cells only in the presence of antigen embedded within the glomeruli, and this induces proteinuria through the effect of cytokines. Proteinuria and foot process effacement will be potentiated either by applying this model to mice with defects in slit diaphragm proteins or delivering other B cell activators to the glomeruli. Through this work, identification of cytokines responsible for the initiation of glomerulopathies can be made along with the development of targeted therapies.



Sang Taek Kim, MD

Yale University School of Medicine

Human follicular helper T cells and their clinical applications

CD4 T cells play a critical role in helping B cells produce antibodies in response to challenge with foreign antigens. This response classically occurs in germinal centers (GCs) located in B cell follicles of secondary lymphoid organs, a site of immunoglobulin (Ig) isotype switching and affinity maturation. GC formation occurs during a T dependent (TD) immune response in which specialized CD4 cells, termed T follicular helper (TFH) cells, localize to follicles and provide B cells with critical survival and differentiation signals, including CD40 ligand (CD40L, CD154), inducible costimulator (ICOS), IL-21 and IL-4. These survival and

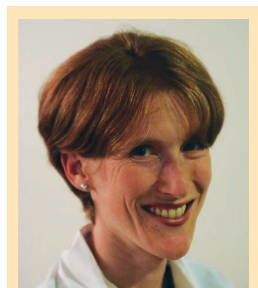
differential signals are essential for B cell selection with maturation into memory B cells and into long-lived plasma cells. Pathologic autoantibodies in human and murine lupus appear to largely arise in a similar manner.

Although CD4 T B-helper cells are critical for development of GCs and for full maturation of the extrafollicular (EF) response in systemic autoimmunity, they have been less clearly defined than other T cell effector subsets. Moreover, the genesis of human TFH cells and their role in promotion of autoimmunity are poorly understood, as is their potential as therapeutic targets in SLE.

This project will investigate human follicular helper T cells, with specific aims to fully characterize, in terms of phenotype and function, these cells in human tonsils, and to seek out and characterize their peripheral blood counterparts in normals and in patients with SLE. The investigator also hopes to determine their responsiveness to therapeutic interventions that, at least in mice, have an effect on their development and maintenance. Understanding the biology of human follicular helper T cells will help the investigators to enlighten pathogenesis of autoimmune disease better and develop therapeutic targets.

RESEARCH AWARDS

Scientist Development Award cont.



Melissa A. Lerman, MD, PhD

Children's Hospital of Philadelphia

Response of pediatric uveitis to TNF alpha inhibitors

Non-infectious eye inflammation (uveitis) occurs in many pediatric rheumatologic diseases, including juvenile idiopathic arthritis. It can lead to significant visual problems, including glaucoma, cataracts and blindness. Treatment options for uveitis include steroids or immunomodulators that suppress the immune system in specific ways. One of the newest classes of immunomodulators blocks tumor necrosis factor alpha (TNF-alpha), a chemical involved in amplifying immune responses. In addition to providing targeted immune blockade, therefore having fewer side effects, these newer agents may

be more effective therapeutics. Recent decreases in the percentage of children with uveitis who develop poor visual outcomes might be attributable to earlier detection of uveitis and/or the availability of newer treatments. While it is widely believed that TNF-alpha inhibitors have improved the long-term outcomes of pediatric uveitis, outcomes have been described only in single center studies. These studies suggest that TNF-alpha inhibitors are effective and that other treatments may vary in their effectiveness. Physicians are not able to predict which patients will respond to steroids or will require other immunomodulators, and studies have not examined which factors predict better responses to specific treatment options. As pediatric uveitis is a rare disease, it has been difficult to obtain enough patients for a study to provide statistically significant results. The investigators propose studying the largest, and only multicenter, group of patients with pediatric uveitis to date to more precisely estimate the outcomes of treatment with TNF-alpha inhibitors. The investigators will evaluate which factors increase the likelihood of achieving uveitis quiescence after treatment. The specific aims are (1) to estimate the probability of children achieving quiescence of uveitis within six months of treatment with TNF-alpha inhibitors; (2) to identify factors independently associated with achievement of uveitis quiescence in children treated with TNF-alpha inhibitors; and (3) to evaluate the risk of children with uveitis discontinuing TNF-alpha inhibitors because of side effects of therapy.



Rebecca L. Manno, MD, MHS

Johns Hopkins University

Strength and exercise in GCA

Giant cell arteritis (GCA) is the most common form of vasculitis in persons 65 years and older in North America. Older individuals are particularly susceptible to the untoward effects of the acute and chronic inflammatory processes associated with GCA and the unintended consequences of glucocorticoid therapy. Aging is a major risk factor for accelerated decline of muscle mass and strength (sarcopenia), and this decline is even more pronounced in elderly with inflammatory disease. GCA treatment is limited almost exclusively to corticosteroids, which also contribute to loss of strength and sarcopenia.

We propose that GCA patients are at great risk for sarcopenia and muscle weakness given the synergistic effects of systemic inflammatory disease, corticosteroid treatment and advanced age; however, strength and body composition have not been characterized in GCA. Further, interventions that minimize or reverse this process in GCA patients are nonexistent. Resistance exercise is an intervention that can increase strength, improve quality of life and decrease inflammation, but it is unknown whether resistance exercise will have beneficial effects in elderly with GCA.

The objectives of this research are: (1) to define the scope of the problem of weakness and sarcopenia in GCA patients relative to the general population; and (2) to determine the effects of resistance exercise in elderly GCA patients.

Investigators will study a cohort of GCA patients and characterize their strength, functional capacity, and body composition and compare these measures to age-, gender-, race- and BMI-matched controls from the Baltimore Longitudinal Study of Aging. A subset of this GCA cohort will be enrolled in a 16-week resistance exercise program. Patients will participate in two supervised training sessions per week, performing five resistance exercises (latissimus dorsi pulldown, chest press, leg press, shoulder press, bicep curl) with slow and controlled cadence (taking five seconds to lift and lower resistance). Strength, body composition, functional capacity, fatigue and quality of life will be compared before and after the 16-week program and between those participating in the resistance exercise program and those who are not.

The results from this research will add to the understanding of the effects of GCA on strength, muscle and functioning and begin to establish whether resistance exercise is a safe and beneficial adjuvant approach to GCA treatment.

RESEARCH AWARDS

Scientist Development Award cont.



Jessica L. Maxwell, PT, DPT, OCS

Boston University

Limitations in participation following knee replacement

Knee replacements do not achieve successful outcomes in approximately 25 percent of persons, despite being the ultimate intervention for end-stage knee osteoarthritis. It is currently unknown whether these unsatisfied or functionally limited persons are limited at the participation level, where their home and community roles and abilities are negatively impacted and, if so, what factors are responsible.

This proposal will investigate the extent of and determine the risk factors associated with participation limitations following knee replacement in older adults. The central hypotheses are that greater than 20 percent of persons following knee replacement have participation limitations, and that both personal and physical factors are associated.

There are three specific aims: (1) to estimate and to describe the rates of participation at least 2 years following knee replacement; (2) to analyze the association between various patient factors and participation limitations. Personal (depression, coping), impairment level (range of motion, strength) and functional (performance-based and self-reported) exposure variables will be studied. Participation limitations will be measured using the Late-Life Disability Instrument (LLDI); and (3) to conduct qualitative analysis of the factors associated with participation limitation in older adults. Focus group discussions will explore limitations in activities both included and not included on the LLDI. Investigators will identify whether complex relationships between factors exist, or discover other potentially associated exposure variables that may not have been collected in our cohort studies.

Given the exploding rates of knee replacement and their often unsatisfying outcomes, the proposed study is timely and will offer new insights into the outcomes after knee replacement. These insights may change how patients undergoing knee replacement are treated.



Miriam A. Shelef, MD, PhD

University of Wisconsin-Madison

Citrullination in arthritis

RA is an autoimmune disease that causes permanent joint destruction and disability. Although many cell types are involved in joint destruction, synovial fibroblasts, which line the joint, play a key role by invading cartilage and bone. Relatively recently, it has been shown that people with rheumatoid arthritis develop anticitrullinated protein antibodies. These antibodies can precede disease, are associated with more erosive disease and are thought to contribute to disease pathology. The citrullinated proteins themselves often are thought to be pathologic, as well. It is true that some

citrullinated proteins are more arthritogenic than their noncitrullinated counterparts; however, the full role of citrullinated proteins in arthritis is unknown. Citrullination is the conversion of a protein's arginine residues to citrulline and is catalyzed by a family of peptidyl arginine deiminases (PADs). The inflamed joint in RA has more citrullinated proteins in addition to increased levels of PAD4 and PAD2. Interestingly, several proteins that are citrullinated in RA, such as fibronectin and collagen type II, are associated with the extracellular matrix.

Since extracellular matrix molecules are important for regulating cell behavior, investigators have begun to investigate the effects of citrullinated extracellular matrix on synovial fibroblasts. Investigators have found that synovial fibroblasts have impaired adhesion, spreading and integrin signaling on citrullinated fibronectin. Since fibronectin is thought to mediate the adhesion of synovial fibroblasts to the rheumatoid joint ultimately leading to joint destruction, impaired adhesion to citrullinated fibronectin could impede the development of arthritis. Thus, citrullination could have some protective functions.

Building on these data, this project aims to test the hypothesis that citrullination has protective functions in arthritis by thoroughly characterizing the behavior of synovial fibroblasts on citrullinated matrix proteins, as well as assessing the role of citrullination in arthritis. Investigators will evaluate synovial fibroblast invasion, focal adhesions, invadopodia, and gene expression in response to citrullinated fibronectin and collagen type II. They also will look at arthritis in mice which lack PAD4 as well as introduce excess PAD to inflamed joints. These studies promise to enhance understanding of rheumatoid arthritis and citrullination as well as potentially guide future drug development.

RESEARCH AWARDS

Scientist Development Award cont.



Zejin Zhu, MD, PhD

University of California, Los Angeles

Tissue-resident DCs in eyes in rheumatic diseases

Ocular surface inflammation is common in rheumatic diseases and sometimes severe enough and tough to treat that eventually leads to blindness. Little is known about its pathogenesis. Cornea harbors dendritic cells (DC), including LCs that express Langerin (Lang), and Lang- DCs. Their contribution to the development of autoimmune ocular surface inflammation is unclear.

It is proposed that corneal LCs play a protective role against the development of ocular surface inflammation and they do so by migrating to eye-draining lymph nodes where they induce tolerance to ocular antigens. In this project, the hypothesis will be tested using MRL mice (MRL/MpJ-Fas^{lpr/lpr} and MRL/MpJ-Fas^{+/+}) that develop autoimmune-mediated inflammation in multiple organs including eyes. The investigator will first evaluate the phenotype and migration of corneal LCs, to test the hypothesis that the migration of corneal LCs is impaired in MRL mice, which contributes to the loss of immune tolerance and development of corneal inflammation. This idea will be tested using Lang-eGFP mice that express enhanced green fluorescence protein (eGFP) driven by Lang-promoter, which allows a clear detection of LC in tissues. In the second part of this project, the investigator will determine the role of LCs in the pathogenesis of ocular inflammation, to test the specific hypothesis that the ablation of LCs will accelerate ocular inflammation in MRL mice. This will be tested this using Lang-DTR.eGFP MRL mice where LC can be conditionally ablated by diphtheria toxin (DT) injections, as these mice express DT receptor (DTR) fused to eGFP driven by Lang promoter.

The proposal may open a new direction in the pathogenesis of ocular inflammation. Results obtained will form the basis for studies to investigate whether similar organ-specific DCs play immune homeostasis roles in organs such as joints.

Investigator Award

This award supports basic science, translational and clinical investigators engaged in research relevant to rheumatic diseases for the period between the completion of post-doctoral fellowship training and establishment as an independent investigator. Recipients receive funding for up to three years at \$125,000 per year.



Mara L. Becker, MD, MSCE

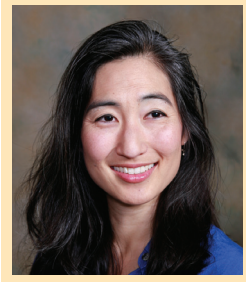
Children's Mercy Hospitals and Clinics

A signature for response to methotrexate in juvenile idiopathic arthritis

Although methotrexate (MTX) is the first-choice second-line agent used to treat juvenile idiopathic arthritis (JIA) worldwide, there is vast variability in response and toxicity to the drug that is unpredictable. Investigators hypothesize that differences in response to MTX reflect inter-individual variability in adaptation to pharmacologic folate deprivation which is dependent upon: (1) baseline folate status (supply vs. demand) and (2) the cellular adaptive response to MTX (dependent upon individual genotype and the clinical variables of age, sex and disease subtype). Investigators plan to test these hypotheses by prospectively studying polyarticular rheumatoid factor negative and extended oligoarticular JIA patients who are newly prescribed MTX and comparing intracellular folate isoform concentrations in addition to folate polyglutamate patterns in early responders vs. nonresponders. Responders to MTX (defined as achieving clinical remission and/or ACR ped70 improvement) are expected to have a distinct pharmacologic "signature of response" defined by lower concentrations of 5-methyltetrahydrofolate and 5,10-methylenetetrahydrofolate and higher proportions of long chain 5-methyltetrahydrofolate polyglutamates than nonresponders, suggesting a more effective "folate deprivation effect" by MTX. Investigators will then identify demographic and genetic variables that contribute to the differences in intracellular folate concentrations and polyglutamate patterns in responders and non-responders in this prospective cohort. It is expected that adaptation to cellular folate perturbation by MTX will depend on an individual's genotype in addition to age, sex and baseline folate status. This ability, or lack thereof, to adapt to perturbation may result in differences in MTX response. By identifying early predictors of response and toxicity to MTX it is hoped that the safe and effective dosing of MTX in children with JIA can be optimized.

RESEARCH AWARDS

Investigator Award cont.



Tamiko Katsumoto, MD

University of California, San Francisco

Protection of CD148^{-/-} mice from pulmonary fibrosis

Scleroderma is a rheumatic disease with limited treatment options that can have morbid consequences resulting from the pathologic fibrosis of skin and internal organs. Tyrosine phosphorylation, critical to the regulation of many cellular processes, is tightly regulated by the opposing actions of protein tyrosine kinases (PTKs) and protein tyrosine phosphatases (PTPs). Recent studies have demonstrated attenuation of experimental lung fibrosis and skin induced by bleomycin using the tyrosine kinase inhibitor (TKI) imatinib, and several clinical trials using TKIs in scleroderma are under way. The receptor-like protein tyrosine phosphatase (RPTP) CD148 is expressed on both

hematopoietic and non-hematopoietic cell types, and our lab has generated mice with a targeted deletion of the CD148 transmembrane domain (CD148KO) leading to loss of CD148 phosphatase activity. The investigators recently described a positive regulatory role for CD148 in B cell and macrophage immunoreceptor signaling, via dephosphorylation of the C-terminal negative regulatory tyrosine of Src family kinases (SFKs). As CD148 is highly expressed in lung cells and given the known relevance of tyrosine phosphorylation pathways in fibrogenesis, CD148KO mice were evaluated in the bleomycin model of pulmonary fibrosis. A marked reduction in mortality and in the development of pulmonary fibrosis was noted in CD148KO mice, with no differences in the early phase of bleomycin-induced acute lung injury. The central hypothesis is that lack of CD148 phosphatase activity protects against fibrosis as a consequence of SFK inhibition in lung fibroblasts and epithelial cells. The specific aims of this proposal are: (1) to identify the cell types responsible for attenuation of fibrosis, (2) to characterize the biochemical and functional effects of CD148 in signaling pathways and cell types relevant to fibrosis, and (3) to determine the consequences of CD148 inactivation on the signaling phenotype and gene expression profiles of specific lung cell populations before and after bleomycin induced pulmonary fibrosis.



Alexis R. Ogdie-Beatty, MD

University of Pennsylvania

Adverse cardiovascular events in psoriatic arthritis

Chronic systemic inflammation is associated with increased major adverse cardiovascular events. Based on this premise, this proposal investigates the relationship between major adverse cardiovascular events and psoriatic arthritis (PsA) using population-based studies. Research will focus on determining the risk of major adverse cardiovascular events among patients with PsA through the use of a longitudinal cohort study with prospectively gathered data from an existing medical records database in the United Kingdom, The Health Improvement Network (THIN). It is the central

hypothesis of this proposal that PsA is associated with increased major adverse cardiovascular events as compared to patients without PsA independent of traditional cardiovascular risk factors.

To test this hypothesis, two specific aims are proposed: (1) to determine the validity of THIN for the study of psoriatic arthritis by developing coding algorithms that will accurately identify patients with PsA; and (2) to define, among PsA patients, the risk of major adverse cardiovascular events including MI, ischemic stroke, cardiovascular mortality and the composite outcome adjusting for traditional cardiovascular risk factors. This will be the largest longitudinal study of PsA to date with more than 7,000 patients and will fill a gap in current knowledge on cardiovascular risk in PsA. Ultimately such data will be critical for defining the magnitude of cardiovascular risk in PsA and may establish an underpinning for further investigations to improve upon strategies for preventing adverse cardiovascular events in patients with PsA.

RESEARCH AWARDS

Investigator Award cont.



Daniel K. White, PT, ScD

Boston University, College of Health and Rehabilitation Sciences; Sargent College of California

Factors for change in habitual walking in knee OA

Knee osteoarthritis (OA) is the leading cause of limitations with walking compared with any other chronic disease in the U.S., leading to restrictions with community mobility and adverse health outcomes related to a sedentary lifestyle. While prior studies have examined changes in self-reported walking quantity or difficulty, or clinic-based walking speed, no study to date has examined change in objectively measured habitual walking experiences among persons with knee OA. Additionally, whether factors associated with self-reported walking activity or clinic-based walking speed are truly

associated with change in objectively measured habitual walking is not known. This is an important gap given that habitual walking is the most commonly performed physical activity and as little as 60 minutes of moderate intensity walking per week can substantially reduce the risk of adverse health outcomes related to a sedentary lifestyle.

This project measures habitual walking in subjects from the Multicenter Osteoarthritis Study (MOST) which includes persons with or at high risk of knee OA. In particular, habitual walking will be measured using an accelerometer-enabled monitor over two time points, representing an opportunity to obtain walking information objectively as opposed to by self-report. Investigators will define insufficient habitual walking as persons who do not walk more than 60 minutes over seven days at a moderate intensity. They will investigate knee-specific (e.g., knee pain and buckling) and person-specific (e.g., walking speed and psychosocial functioning) factors, and will evaluate the association of these factors with insufficient habitual walking at baseline and follow-up.

Investigators hypothesize that certain knee-specific characteristics such as intermittent pain and knee buckling and person-specific characteristics such as psychosocial functioning will be risk factors for insufficient habitual walking. Completion of this study will lead to decreasing the risk of adverse health outcomes in people with knee OA through understanding factors associated with habitual walking.

Bridge Funding Award

This award provides support to budding investigators as they revise outstanding applications for federal funding. Through the Bridge Funding Award, the Rheumatology Research Foundation and the Arthritis Foundation hope to ensure these young faculty members have the highest likelihood of achieving success in obtaining future NIH awards. Funding is made possible in part through the generous financial support of the Arthritis Foundation.

Christie M. Bartels, MD, MS

University of Wisconsin

Peter J. Kim, MD

University of California, Los Angeles

ANNUAL MEETING AWARDS

Student Achievement Award

This award recognizes medical and graduate students for significant work in the field of rheumatology by providing an opportunity for them to attend the ACR/ARHP Annual Meeting. Recipients will receive an award of \$750 and reimbursement for travel expenses to the annual meeting. Funding is made possible through the Abbott Endowment for Rheumatology Development.

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Elizabeth V. Arkema

Harvard School of Public Health

Marshall Davis, MPH

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Sofia de Achaval, MPH

University of Texas MD Cancer Center

Rachel A. Gordon

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Jan M. Hughes-Austin

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Rachel L. Morgan

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Arcadia University

Amy Phillips

Arcadia University

William M. Reichmann

Brigham & Women's Hospital

Tiffany N. Telarico

SUNY Upstate Medical University

Andrew A. Wilson

University of Mississippi School
of Medicine

Michelle S. Yau, MPH

University of Maryland

Medical and Pediatric Resident Research Award

This award is designed to motivate residents to pursue subspecialty training in rheumatology by providing the opportunity to experience rheumatology first-hand at the ACR/ARHP Annual Meeting. Recipients receive an award of \$750 and reimbursement of up to \$1,000 to cover travel expenses to the meeting. Funding is made possible through the Abbott Endowment for Rheumatology Development.

Medya Barbhaiya, MD

Weill Cornell Medical College

Pravitt R. Gourh, MD

University of Texas Medical School at Houston

Karim Masri, MD

University of Kansas School of Medicine Wichita

Amgen Pediatric Research Award

This award promotes scholarship in the field of pediatric rheumatology and recognizes outstanding abstracts with an award of \$1,000 and reimbursement of up to \$1,000 to cover travel expenses to the ACR/ARHP Annual Meeting. Funding is made possible through an endowment provided by Amgen, Inc.

Gina A. Montealegre Sanchez, MD

Rainbow Babies & Children's Hospital

MEMORIAL LECTURESHIPS

Memorial Lectureships

Endowed lectureships support rheumatology research and honor the lives of five outstanding physicians

Edmund L. Dubois, MD, Memorial Lectureship

Presented by: Mariana J. Kaplan, MD

Oscar S. Gluck, MD, Memorial Lectureship

Presented by: Nancy E. Lane, MD

Funding for the Gluck Lectureship is provided in part by support from Merck & Company, Inc. and Eli Lilly and Company.

Paul Klemperer, MD, Memorial Lectureship

Presented by: Roland W. Moskowitz, MD

Marshall J. Schiff, MD, Memorial Lectureship

Presented by: Kurt P. Spindler, MD

Memorial Lectureship, In Honor of Dr. Lawrence E. Shulman

Presented by: Sergio A. Jimenez, MD

Funding for the Memorial Lectureship is provided in part by friends and colleagues of Dr. Lawrence E. Shulman.

ONGOING SUPPORT

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Erika H. Noss, MD, PhD

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Brian T. Walitt, MD, MPH

Brigham and Women's Hospital-Research

University of Utah School of Medicine

Brigham and Women's Hospital

University of Michigan Medical Center

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Jessica Berman, MD

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David Sherry, MD

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Hospital for Special Surgery

Washington Hospital Center

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Disease Targeted Research Grants

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Jeffrey R. Curtis, MD, MPH, MS

Sonye K. Danoff, MD, PhD

Kevin Deane, MD

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Patricia Katz, PhD

Larry W. Moreland, MD

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Nancy J. Olsen, MD

University of Miami

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Ted R. Mikuls, MD, MSPH

Daniel L. Mueller, MD

Columbia University

University of Nebraska
Medical Center

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The Rheumatology Research Foundation Corporate Roundtable: A Dynamic Collaboration Investing in the Future of Rheumatology.

The Corporate Roundtable brings together the Foundation and pharmaceutical industry leaders in a dynamic partnership. As a direct result of the exemplary support of Corporate Roundtable donors, the Foundation continues to increase funding for research and training.

Participation in the Corporate Roundtable is a direct investment toward helping to ensure that well-trained and qualified rheumatologists are entering the field, while also making advancements in patient care. The Foundation thanks the Corporate Roundtable donors, including current Industry Roundtable members, for their exemplary support.

2011-2012 Corporate Roundtable Donors

Leadership



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Journey to Cure

THE CAMPAIGN TO
ADVANCE PATIENT CARE AND ACCELERATE DISCOVERIES

Journey to Cure is the Rheumatology Research Foundation's \$60 million multiyear campaign, which seeks to further invest in efforts to advance patient care and accelerate discoveries in rheumatic disease research. The Foundation's mission objectives are as follows:

Advancing patient care

- Recruit and train future rheumatologists and rheumatology educators
- Develop future researchers and foster the best novel research ideas in each niche of rheumatology

Accelerating discoveries

- Advance research leading to cures in the most serious of the rheumatic diseases—rheumatoid arthritis—and other conditions where inflammatory arthritis is a major pathology, including the spondyloarthropathies

Journey to Cure funds an extensive peer-reviewed grants program, spanning a rheumatologist's career, to ensure a pipeline of qualified rheumatology care providers and inspire the targeted research that can advance treatment and find cures to rheumatic disease.

The Foundation is the largest private funding source of rheumatology training and research programs in the United States and has provided more than \$100 million of research support over its 27-year history. For five consecutive years, the foundation has received a 4-star rating—the highest available—from Charity Navigator, and more than 90 cents of every dollar raised is directly invested in career development research grants and rheumatology training. No other organization is better positioned to address upcoming challenges to the rheumatology community than the Rheumatology Research Foundation.

Funds raised through the *Journey to Cure* campaign will contribute to the development of the rheumatology work force so that it can meet upcoming, unprecedented demands for patient care, cultivation of the next generation of researchers dedicated to rheumatic disease and support of targeted inflammatory arthritis research.

FINANCIALS



ORGANIZATIONAL EFFICIENCY

Statements of Financial Position

June 30, 2012 and 2011

Assets	2012	2011
Cash and cash equivalents	\$ 14,136,005	\$ 9,352,249
Investments	35,945,546	37,342,599
Contributions and grants receivable, net	17,946,163	12,084,373
Prepaid expenses and other assets	25,492	231,776
Property and equipment, less accumulated depreciation of \$45,226 and \$36,037 in 2012 and 2011, respectively	68,851	77,977
Total assets	\$ 68,122,057	\$ 59,088,974
Liabilities		
Accounts payable	\$ 312,124	\$ 86,226
Net Assets:		
Donor restricted:		
Temporarily restricted	34,999,458	24,759,258
Permanently restricted	2,305,795	2,305,795
Other:		
Designated by board for education and research awards	22,831,651	19,955,738
Unrestricted	7,673,029	11,981,957
Total net assets	67,809,933	59,002,748
Total liabilities and net assets	\$ 68,122,057	\$ 59,088,974

ORGANIZATIONAL EFFICIENCY

Statements of Activities

For the years ended June 30, 2012 and 2011

Changes in unrestricted net assets	2012	2011
Revenue:		
Gifts and grants	\$ 1,159,674	\$ 1,056,816
Investment and interest income	510,964	407,207
Net realized and unrealized gains (losses) on investments	(840,207)	4,322,864
Miscellaneous income	57,523	59
Net assets released from restriction	6,889,007	9,920,349
Net assets transferred to temporarily restricted	-	(172,375)
Contribution from American College of Rheumatology	-	5,000,000
Total unrestricted revenues	7,776,961	20,534,920
Expenses:		
Program services - research and education	8,031,395	10,683,667
Support services:		
Administrative	293,128	343,386
Fundraising	885,453	664,290
Total support services	1,178,581	1,007,676
Total expenses	9,209,976	11,691,343
Change in unrestricted net assets	(1,433,015)	8,843,577
Changes in temporarily restricted net assets:		
Gifts and grants	17,199,854	7,946,268
Investment and interest income	193,225	162,017
Net realized and unrealized gains (losses) on investments	(263,872)	1,101,555
Net assets released from restriction	(6,889,007)	(9,920,349)
Transfer from unrestricted net assets	-	172,375
Change in temporarily restricted net assets	10,240,200	(538,134)
Change in net assets	8,807,185	8,305,443
NET ASSETS AT BEGINNING OF YEAR	59,002,748	50,697,305
NET ASSETS AT END OF YEAR	\$ 67,809,933	\$ 59,002,748

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Rheumatology Research Foundation

Advancing Treatment | Finding Cures

Rheumatology Research Foundation

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