SPOTLIGHT ON RESEARCH
Grant Recipients

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From Bench to Bedside

These ten Arthritis Foundation grant recipients are investigating the causes of arthritis with the goals of advancing more effective therapies and personalized care for patients—and eventually finding a cure.

BY JO CAVALLO

The physical and financial ramifications of arthritis both on patients and society are enormous with costs from lost wages and productivity and direct medical spending rising to $128 billion each year. According to the American Pain Society, pain is the second leading cause of medically related work absenteeism, resulting in more than 50 million lost workdays each year.

Since its inception 64 years ago, the Arthritis Foundation has committed over $411 million to fund research investigating the causes of rheumatic diseases and finding more effective and safer treatments, with the ultimate goal of finding cures for these debilitating diseases.

In 2012, the Foundation awarded $1.6 million in Clinical to Research Transition Awards, Postdoctoral Fellowships and Innovative Research Grants to ten researchers from the Northeast region profiled on the following pages. The Foundation is proud to support the work these researchers are doing in such critical areas as investigating more effective interventions for osteoarthritis; strategies for personalized care for patients suffering from rheumatoid arthritis as well as other forms of arthritis; and methods for standardizing care for children with juvenile arthritis.

"These grants support both veteran and new leaders in rheumatology to help us achieve our goals of moving drugs from the laboratory into patients' hands and producing the next generation of clinicians who will use these drugs," says John Hardin, MD, Vice President for Research at the Arthritis Foundation.

"The Arthritis Foundation has a strong tradition of supporting research projects at early time points and helping bring them along to test their basic principles. Over the years, the Foundation has had a great impact on research in arthritis because it has been willing to take chances. In our current financial situation, the Foundation is providing funding that's keeping progress moving ahead that otherwise would be very slow," says Gregg J. Silverman, MD, Professor of Medicine and Pathology and Co-Director of the Musculoskeletal Center of Excellence at New York University School of Medicine, and one of the researchers profiled in this newsletter.

The research studies of the ten grant recipients featured on the following pages are categorized by disease type and include rheumatoid arthritis, osteoarthritis, juvenile arthritis, gout and scleroderma.

RHEUMATOID ARTHRITIS

Rheumatoid arthritis (RA) affects more than 1.5 million people in the U.S., according to the Centers for Disease Control and Prevention. RA is an autoimmune disease in which the body's own immune system mistakenly attacks healthy tissue.

Although RA can occur at any age, it is most commonly diagnosed in people ages 40 through 60 and is much more common in women than men. In addition to causing pain and inflammation in the joints, RA can also affect other organs in the body, including the skin.

These five Arthritis Foundation grant recipients are engaged in research to better understand the underlying causes of rheumatoid arthritis. ➤

SPOTLIGHT ON RESEARCH Grant Recipients

Jennifer Belasco, MD

Instructor in Clinical Investigation,
The Rockefeller University,
New York City, New York

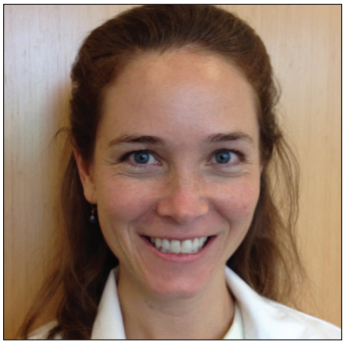
A rotation through pediatric rheumatology while completing her pediatric residency at St. Christopher's Hospital for Children in Philadelphia convinced Jennifer Belasco, MD, to pursue a fellowship in pediatric rheumatology at the Hospital for Special Surgery in New York City, launching her career in the field. "Pediatric rheumatology is often challenging and I thought that caring for children with rheumatic diseases would keep me on my toes and make for a very interesting career," says Dr. Belasco.

The recipient of a two-year, $60,000 per year Arthritis Foundation Clinical to Research Transition Award, Dr. Belasco is studying the *Characterization of Inflammatory Pathways in Psoriatic Arthritis* in adults with psoriatic arthritis (PsA), using patients with rheumatoid arthritis (RA) and osteoarthritis (OA) as control groups, and has plans to launch the same study in children with psoriatic arthritis.

A form of chronic inflammation of the skin (psoriasis) and joints (arthritis), psoriasis is a common skin condition that affects two percent of the Caucasian population in the U.S., and includes symptoms such as thick patchy, raised, red areas of skin inflammation with flaky scaling. About 42 percent of patients with psoriasis will progress to arthritis in which the joints become painful and swollen; muscles or ligaments, especially in the heel and bottom of the foot, become tender; and tissues surrounding the eyes become red and painful. "What we don't know is how to identify which patients will go on to develop arthritis and catch the disease early so we can start therapy as soon as possible," says Dr. Belasco.

Dr. Belasco's study will investigate the connection between joint and skin involvement in PsA by comparing the skin and joint inflammatory pathways in PsA lesions with the synovial fluid in the joints of patients with RA and OA; establishing molecular and cellular pathways expressed in PsA synovium and compare them to RA and OA synovia; and expanding gene expression profile analysis in PsA skin lesions to determine which genes are activated and may be playing a role in the development of PsA.

"The first thing is to see what the connection is between the skin and joints in PsA. Understanding that may not only help us identify which psoriasis patients will go on to develop PsA but help find new targets for effective medications to prevent destructive arthritis as well. On our basic level, we just want to get a better understanding of what's going on with psoriatic arthritis and stop the disease from progressing," says Dr. Belasco.



Dana E. Orange, MD

Instructor of Clinical Research,
The Rockefeller University,
New York City, New York

A researcher in immunology while in medical school, Dana E. Orange, MD, decided to specialize in rheumatology after completing her residency in internal medicine because she was fascinated by the immune system and the complex nature of rheumatic diseases. "In a disease like cancer, you do a biopsy and the results put you on a certain track," says Dr. Orange. "But in rheumatology there is no one single test that will diagnose a disease like rheumatoid arthritis (RA). You have to have a really good conversation with patients to get a very thorough medical history before you can make a diagnosis and then some other symptom might show up down the road and you have to reconsider it. I like having to always stay on top of my game."

In May, Dr. Orange received a two-year, $60,000 per year Clinical to Research Transition Award for her study, *Using Dendritic Cells to Evaluate T-Cell Specificity in ACPA+RA*. The purpose of Dr. Orange's research is to determine which patients with RA have an abnormal B-cell antibody that is affecting the response of their T-cells.

B cells and T cells are types of white blood cells that help the body's immune system fight infection. Scientists know that B cells play several critical roles in the pathogenesis of RA and are the sources of anticitrullinated protein antibodies (ACPA), which contribute to disease activation in the joints. Surprisingly, the genetic marker that confers the highest risk for citrullinated antibodies in RA is a molecule that is important for activating T cell responses and T cells do not make antibodies. This had led to the hypothesis that patients with antibodies to citrullinated self-proteins may also have T cell responses to citrullinated protein.

Understanding T-cell responses to citrullinated proteins may help determine why RA develops and how best to treat it in patients with circulating anticitrullinated protein antibodies. Approximately 60 percent of RA patients have ACPA and incur greater bone erosion and increased disability.

In Dr. Orange's laboratory study, dendritic cells, immune cells that process antigen material and present it on the surface to other immune cells and function as antigen-presenting cells, are being used to illicit T-cell responses to provide a much more sensitive tool to test for abnormal T-cell activation in RA and may lead to the discovery of novel biomarkers for the disease. Understanding what antigens are driving T-cell responses in RA may also lead to methods to turn off those abnormal T cells and more effective therapies to treat RA.

> **"Right now, we don't have a clear algorithm to decide which drugs to use for individual patients."**

"Right now, we don't have a clear algorithm to decide which drugs to use for individual patients. We try the least toxic drugs and then we try stronger and stronger drugs but there's really no guideline to say in this patient we should try a TNF inhibitor or another type of biologic first. It's a random decision. I'm hoping that if we can identify patients with these abnormal T-cell responses, they will be much more likely to respond to a drug, such as, Orencia (abatacept) [an injectible manmade protein] that is thought to interfere with T-cell activation," says Dr. Orange.

Discovering novel biomarkers for RA can lead to personalized care for patients and provide more effective targeted therapies to not only manage the disease but potentially cure it as well.

Tanisha Jackson, PhD

Postdoctoral Fellow,
The Feinstein Institute for Medical Research,
North Shore-Long Island Jewish Health System,
Manhasset, New York

Understanding how the immune system is regulated is something that drives me and that's how I got interested in rheumatology," says Tanisha Jackson, PhD, whose research is currently focused on determining the role of the reactivity of B cells in the development of systemic lupus erythematosus (SLE or lupus). Lupus is a form of rheumatoid arthritis in which the immune system attacks the joint tissue, skin, blood, nervous system and internal organs, including the heart, lungs, kidneys and brain.

The disease primarily affects young women and while it can occur from infancy to old age, it usually strikes between the ages of 15 and 40 and is more prevalent in African-American and Asian women than Caucasian women. Because lupus is difficult to diagnose—and is often under diagnosed—prevalence rates vary from 322,000 to as high as 1,500,000, according to the Lupus Foundation of America. The causes of SLE are unknown but there appears to be both genetic and environmental components to development of the disease.

To support innovative research in SLE, the Arthritis Foundation has awarded a two-year, $50,000 per year Postdoctoral Fellowship grant to Dr. Jackson for her study, T*he Effect of CSK Expression on BCR Signaling and Autoantibody Production.* Csk (c-src kinase) is a molecule involved in regulating B-cell receptor signaling events.

"Previous work in our lab showed that mutations in the Csk gene both in mice and in humans has resulted in the production of autoantibodies and altered B-cell signaling but we don't know why. I want to further examine what role Csk may be playing in auto-reactivity in B cells," says Dr. Jackson.

Although there are several categories of drugs for the treat-

ment of lupus, there are not many agents FDA approved specifically for the disease. Learning how the increased expression of Csk leads to auto-reactivity in B cells may provide the clue to advancing the development of more effective therapies for SLE.

"My hope is that we will be able to figure out how the over-expression or decreased expression of Csk affects autoantibody production and apply this knowledge to target Csk expression in B cells as a therapeutic option for lupus," says Dr. Jackson. "Because there are so many genetic components to the development of SLE, just gaining an understanding of how Csk contributes to lupus-like autoantibody production during different stages of B-cell development, brings us one step closer to finding a cure for the disease."

Gregg J. Silverman, MD

Professor of Medicine and Pathology and
Co-Director of the Musculoskeletal Center of Excellence,
New York University School of Medicine,
New York City, New York

When Gregg J. Silverman, MD, was in medical school he was attracted to the science of immunology and how disorders of the immune system can sometimes result in devastating consequences. During his medical residency at the University of California, San Diego, Dr. Silverman learned more about the connection between immunology and rheumatology and decided to chose a career dedicated to providing clinical care and improving the understanding of the causes of rheumatic diseases, especially rheumatoid arthritis (RA).

In January, Dr. Silverman received a two-year, $100,000 per year, Innovative Research Grant from the Arthritis Foundation for his study, *Citrulline Epitope-Specific B Cells and RA Pathogenesis.* During any injury to the body, immune cells release an enzyme that chemically modifies local proteins to generate a nonstandard amino acid called citrulline. In some people, this injury repair process can induce a special type of autoantibody called an anti-citrulline protein antibody, or anti-CCP antibody. This autoantibody response can then lead to the joint inflammation and damage that is associated with RA.

"We're getting a much more detailed perspective on what are the key features commonly shared by RA patients and what features may discriminate patients likely to have more severe joint disease."

"Recognition of the amino changes that take place at the start of RA has been a tremendous advance to our understanding of what causes the disease. We're now better at understanding patients' prognosis since patients that have circulating antibodies to citrullinated proteins in their blood generally have a worse prognosis due to more severe disease than patients that do not have these antibodies," says Dr. Silverman. "There is also research suggesting that these patients are at greater risk of developing serious heart disease and stroke, which can even shorten their life expectancy."

While many cell types are involved in the development of RA, B lymphocytes, which make autoantibodies, are now believed to play a crucial role in the disease pathogenesis. One area of Dr. Silverman's research is to develop assays to help identify the autoantigen-specific B lymphocytes in RA and target them for therapy, while sparing the protective B lymphocytes that defend the body against infections.

"Right now, most of our therapies for RA are not very specific, so while they combat the disease they may also weaken patients' protective immune defenses. We're looking for a way to keep track of the 'bad' B lymphocytes in the blood, so we can better understand if therapy can preferentially target these disease-causing cells," says Dr. Silverman.

And his work is already producing results.

"We're getting a much more detailed perspective on what are the key features commonly shared by RA patients and what features may discriminate patients likely to have more severe joint disease. We're also very interested in the causes of heart disease in RA, because right now it's a great unknown," says Dr. Silverman. "Ultimately, we hope to develop strategies for personalized therapy to better choose which treatment agent is better matched to the pathways of an individual's own unique immune response."

Grant Recipients SPOTLIGHT ON RESEARCH

Yu Qiao, PhD

Postdoctoral Research Fellow,
Hospital for Special Surgery,
New York City, New York

Although Yu Qiao, PhD, was interested in learning about autoimmune diseases as an undergraduate and graduate student, it wasn't until she received a postdoctoral fellowship in rheumatology at the Hospital for Special Surgery that she solidified her research career in the study of rheumatic diseases, with her greatest interest centered on unraveling the complexities of rheumatoid arthritis (RA).

Earlier this year, Dr. Qiao received a two-year, $50,000 per year, Postdoctoral Fellowship grant from the Arthritis Foundation for her study, Epigenetic Regulation of Inflammatory Gene Expression in Human Macrophages. Macrophages, white blood cells within tissues that are derived from peripheral monocytes, have an important role in the development of joint inflammation and destruction in RA and are key producers of inflammatory cytokines such as tumor necrosis factor-alfa (TNF-α) and IL-6. The goal of Dr. Qiao's study is to determine the mechanism involved in chronically turning on these cytokines in RA synovial macrophages and to develop targeted therapies to turn off the harmful gene expression.

Preliminary data of Dr. Qiao's study shows that interferon regulatory factor-1 (IRF-1) may be related to onset of RA. If her theory proves correct, it may be possible to develop small molecule therapies that target the gene.

"For drug design, you need a target first and if we can determine that target it will give us more treatment options. Disease mechanism in RA varies from person to person, so it's possible with this study to come up with some personalized strategies for individual patients," says Dr. Qiao.

Dr. Qiao is performing genome-wide analysis of IRF-1 to gain knowledge of inflammatory enhancers involved in human macrophages and the relationship between IRF-1 and these enhancers. The completion of her analysis will give researchers a better understanding of the genome-wide function of IRF-1. That understanding, says Dr. Qiao, could provide insight into RA pathogenesis and progression and potentially help with disease prevention and treatment.

"Hopefully, in the future we'll be able to figure out which patients are more prone to RA and have strategies to prevent it from developing," says Dr. Qiao.

OSTEOARTHRITIS

Osteoarthritis (OA) is the most common form of arthritis, affecting more than 27 million Americans and is the leading cause of disability in adults. To date, the Arthritis Foundation has awarded more than $150 million dollars in OA research.

Hui B. Sun, PhD

Associate Professor of Orthopedic Surgery and Radiation Oncology and Director of Orthopedic Research,
Albert Einstein College of Medicine,
Montefiore Medical Center, Bronx, New York

The devastating joint destruction and disability associated with osteoarthritis (OA) is what drives Hui B. Sun, PhD, to study more effective methods to prevent and treat the disease. The Arthritis Foundation recently awarded Dr. Sun a two-year, $100,000 per year, Innovative Research Grant for his study, *Maintaining Cartilage Integrity in OA by Mechanical Intervention.*

Although there is no cure—or even very effective pharmacological interventions—for OA, clinical trials show that moderate levels of exercise as an adjunct therapy in the treatment of the disease reduces pain, improves joint function and may also slow disease progression. However, why exercise is beneficial and what molecular mechanisms are involved in inducing these effects are not understood. Research from Dr. Sun's laboratory has identified a gene transcription factor called CITED2 that prevents cartilage loss in healthy joints by suppressing production of cartilage-degrading enzymes in articular cartilage cells (chondrocytes). Preliminary data from his work shows that moderate exercise in rats with inflammatory arthritis raise levels of CITED2 and prevent the loss of cartilage compared to rats not subjected to exercise.

The goal of Dr. Sun's research is to determine the role of moderate exercise and CITED2 in protecting the degradation of cartilage in joints. "We're trying to understand how mechanical loading, especially moderate loading such as exercise, can prevent, slow, stop or even reverse the OA process," says Dr. Sun. The study also aims to quantify the levels of exercise necessary to benefit individual patients.

"There is a common range of moderate loading that is beneficial to patients, so we have a general prescription but at the same time we want to personalize treatment for each patient by monitoring exercise or physical therapy intensity and checking his CITED2 molecular marker and saying come up or down to this molecular level to achieve maximum benefit. All drugs have side effects. Appropriate mechanical loading is noninvasive and has no negative side effects. If you do some exercises as we describe, the 'drug' is inside yourself," says Dr. Sun.

JUVENILE ARTHRITIS

Juvenile arthritis (JA) is a general term for all types of arthritis that occur in children, including juvenile idiopathic arthritis (JIA), lupus and localized scleroderma. JA is one of the most common diseases found in children, affecting about 300,000 children and adolescents in the U.S.

These two Arthritis Foundation grant recipients are conducting studies to standardize and develop new tools for better outcomes in JA diseases.

Yukiko Kimura, MD

Chief, Pediatric Rheumatology,
Joseph M. Sanzari Children's Hospital,
Hackensack University Medical Center,
Hackensack, New Jersey;
Chair, JIA Research Committee of the Childhood Arthritis & Rheumatology Research Alliance

Yukiko Kimura, MD, decided on a career in pediatric rheumatology after witnessing the physical disability of juvenile arthritis (JA) patients during her pediatric residency over 20 years ago. Although treatments for JA have greatly improved over the last two decades, halting disease progression and improving the quality of lives for many children, physicians still often cannot predict which treatments are most effective and safe for individual patients.

To better enable physicians to choose optimal care for JA patients, the Arthritis Foundation has awarded Dr. Kimura a two-year, $100,000 per year, Innovative Research Grant for her study, *Implementing CARRA Standardized Treatment Plans for JIA*. This pilot study will implement four standardized consensus treatment plans (CTPs) in 30 newly diagnosed systemic juvenile idiopathic arthritis (sJIA) patients at 15 Childhood Arthritis & Rheumatology Research Alliance (CARRA) registry sites.

The pilot study will develop strategies that allow widespread use of these CTPs, as well as decision-making tools to aid physicians. Information gathered from this study will also help to propel the launch of a larger comparison effectiveness study of CTPs for sJIA at additional CARRA sites.

"We did a survey of pediatric rheumatologists in the CARRA network and in systemic JIA in particular there is a wide variation in the way these kids are treated," says Dr. Kimura. "There are very effective new treatments for systemic JIA patients but they have not yet been adopted by the majority of practitioners, at least at the onset of disease. We hope to see whether using these treatments from the very beginning when these kids first get sick might actually change the course of their disease, especially when compared to the treatments we have traditionally used."

The ultimate goal, says Dr. Kimura, is to be able to identify which patients will benefit most from specific treatment regimens or to be able to personalize their care.

Grant Recipients SPOTLIGHT ON RESEARCH

Suzanne C. Li, MD, PhD

Assistant Professor of Pediatrics,
Joseph M. Sanzari Children's Hospital,
Hackensack University Medical Center,
Hackensack, New Jersey

Suzanne C. Li, MD, PhD, was drawn to pediatric rheumatology because she saw firsthand the devastating effects of arthritis on young children and the limited effective therapies to treat them. Her current area of research is focused on the most common form of scleroderma found in children called juvenile localized scleroderma (jLS). Juvenile localized scleroderma is a type of autoimmune disorder that causes a thickening and hardening of the skin and can also affect underlying tissues, such as muscle and bone. The disorder often causes major musculoskeletal problems for children, including inflammatory arthritis, limited joint mobility and inhibits growth of affected limbs, in many cases, leaving children permanently disfigured and disabled.

In January, Dr. Li was the recipient of a two-year, $100,000 per year, Innovative Research Grant from the Arthritis Foundation for her study, *Developing Tools for Localized Scleroderma Comparative Effectiveness Studies.* Data on effective treatments for jLS is limited partly because there are no agreed-upon sensitive standardized assessment tools to evaluate treatment response. Dr. Li's study is monitoring 50 jLS patients treated with one of three current consensus treatment plans (CTPs): methotrexate alone, methotrexate with oral prednisone and methotrexate with IV methylprednisolone, using recently developed standardized clinical assessment tools.

The jLS patients will be treated for one year by pediatric rheumatologists and dermatologists from nine Childhood Arthritis & Rheumatology Research Alliance (CARRA) network sites in the U.S. and one site in Canada and the information entered into the CARRAnet registry. (For information about the trial, visit carragroup.org/content_dsp.do?pc=LS-CompEffTools.) Dr. Li's study will allow the assessment tools to be refined to improve their accuracy and to evaluate how well the different CTPs are tolerated and help future studies of these CTPs and other treatments to identify the best therapies for stopping disease progression. This study also includes collecting blood samples from the jLS patients so future studies can look for disease biomarkers to improve treatment management.

"In the existing CARRA registry, 38 percent of the children with jLS have some type of significant problem. Thirty-eight percent is high and that's for current patients, so we're not doing such a good job of halting the disease," says Dr. Li.

GOUT

Gout is a form of arthritis that occurs when uric acid builds up in the blood and causes joint inflammation and pain, especially in the feet, ankles, knees, hands, wrists and elbows. The disorder is the most common type of inflammatory arthritis and affects as many as 8.3 million people in the U.S., according to a government health survey, and is on the rise due to increasing rates of obesity and high blood pressure. Patients with chronic gout are also at increased risk of developing cardiovascular disease.

Daria B. Crittenden, MD

Chief Fellow, Division of Rheumatology,
New York University School of Medicine,
New York City, New York

I choose rheumatology as my medical specialty because it's a field where you have to consider all the organs in the body and it's also where a lot of innovation research is happening, and that's very interesting to me," says Daria B. Crittenden, MD. During her residency at New York University School of Medicine, Dr. Crittenden investigated the prevalence of comorbidities in gout patients and the limita-

tions of treatment and also conducted a study comparing rates of myocardial infarction (MI) in gout patients taking the medication colchicine versus those patients not treated with the medication.

This spring, the Arthritis Foundation gave a two-year, $60,000 per year, Clinical to Research Transition Award to Dr. Crittenden to continue that work in her new study, *Effects of Colchicine on Cardiovascular Disease in Patients With Gout.* The retrospective study is examining the electronic medical records of gout patients to determine whether colchicine has cardioprotective effects.

"We have biological reasons to believe that colchicine, which is an anti-inflammatory agent that's been used for a very long time to treat gout, could be cardioprotective and our goal with this study is to gather evidence for or against that hypothesis," says Dr. Crittenden.

Because so many gout patients have at least four comorbidities, including hypertension, diabetes and coronary artery disease, and as many as seven, treating the disorder is difficult and more effective therapies are needed to better manage the disease.

"There's been a growing interest in atherosclerosis (hardening of the arteries) as an inflammatory process so there is a lot of overlap with rheumatology because we treat inflammatory diseases. As we learn more about the mechanisms of inflammation and start to see the overlap with other illnesses, we can treat patients more effectively. This is a very exciting time in rheumatology," says Dr. Crittenden.

SCLERODERMA

Scleroderma (also known as systemic sclerosis) is one of the autoimmune rheumatic diseases and causes a thickening and hardening of the skin and damage to internal organs, including the heart, lungs and kidneys. The disease is most common in women. There is no cure for scleroderma.

Elana J. Bernstein, MD

Rheumatolgy Fellow,
Hospital for Special Surgery,
New York City, New York

Elana J. Bernstein, MD, got hooked on the study of rheumatic diseases during her course in musculoskeletal pathophysiology in her second year in medical school and has devoted her career to the study and treatment of arthritis ever since. Scleroderma, a relatively rare, incurable disease, is a major focus of Dr. Bernstein's research. In May, the Arthritis Foundation gave Dr. Bernstein a two-year, $60,000 per year, Clinical to Research Transition Award for her study, *A Submaximal Stress Test to Identify Pulmonary Hypertension in Scleroderma.*

Pulmonary hypertension, an increase in pressure in the artery that connects the heart to the lungs, is common in patients with scleroderma. Because screening tests such as standard echocardiograms are not accurate in picking up the problem and the only definitive test for diagnosis, a right heart catheterization, which involves the passage of a thin flexible tube into the right side of the heart, can result in cardiac complications, more accurate diagnostic tools are needed for pulmonary hypertension in patients with scleroderma.

The goal of Dr. Bernstein's study is to determine whether a noninvasive submaximal heart and pulmonary evaluation test called SHAPE can accurately distinguish scleroderma patients with pulmonary hypertension from scleroderma patients without pulmonary hypertension. The test consists of a 5.5-inch high step that patients step up and down on for three minutes at their own pace as they breathe into a mouth piece that measures changes in exhaled carbon dioxide levels and breathing efficiency.

"People with scleroderma tend to have multiple organ systems involved, including the musculoskeletal and cardiopulmonary systems. If we can diagnose pulmonary hypertension earlier in the course of their disease and start treatment earlier, I'm hoping that there will be better outcomes in terms of morbidity and mortality," says Dr. Bernstein.

Eligibility criteria to participate in Dr. Bernstein's study include a diagnosis of scleroderma and a right heart catheterization procedure within the last 24 months. For additional information, contact Dr. Bernstein at bernsteine@hss.edu.

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