

2017/18

ANNUAL



REPORT

*Arthritis, Rheumatology and Autoimmune Diseases*

**NOVEL ARTHRITIS RESEARCH  
IN TISSUE ENGINEERING  
AND STEM CELL BIOLOGY**

**GENETIC CLUES IN  
RHEUMATIC DISEASES**

**GROUNDBREAKING  
RESEARCH IN  
AUTOIMMUNITY**



★★★★

**CHARITY NAVIGATOR**

Four Star Charity

*A publication of the Arthritis National Research Foundation | [CureArthritis.org](http://CureArthritis.org)*



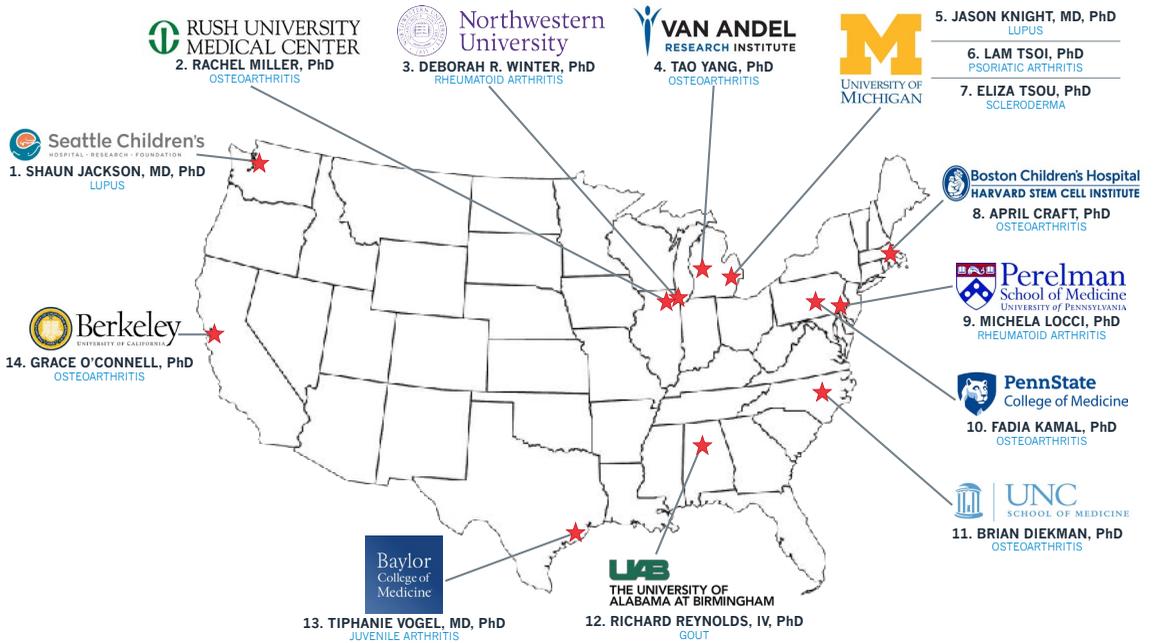
## INVESTING IN EXTRAORDINARY TALENT

2017-18 AWARDEES

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**\$17+ MILLION** IN RESEARCH FUNDING TO DATE  
**\$1.4 MILLION** IN GRANT AWARDS IN 2017  
**250** SCIENTISTS FUNDED  
**74** COMMITTED PARTNER INSTITUTIONS

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Throughout the year the #CureArthritis community hosts their own events to raise funds and awareness for research. We've highlighted a few below and encourage you to start your own project to help support this vital research!



SF Runs | #CureArthritis Edition



Comedy For A Cure



Blue Sky Cup | Skiing For A Cure



Rockin' Out Rheumatoid Concert



Mabel's Mission Fundraising Dinner

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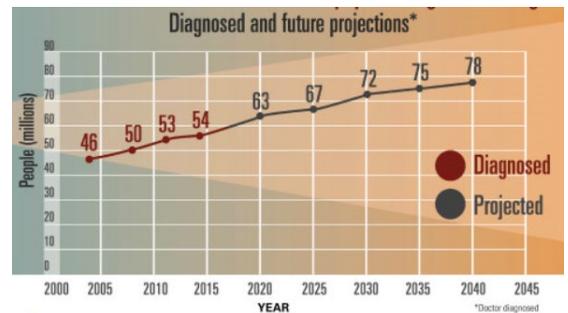




## A MESSAGE FROM OUR EXECUTIVE DIRECTOR

Dear valued Friend of ANRF,

The challenges presented by Arthritis and associated Autoimmune Diseases are formidable and diverse. According to the Centers for Disease Control and Preventions (CDC), 25.9 % of the adult population in the US (or 78.4 million Americans, Graph) will be diagnosed with Arthritis by 2040. Arthritis is also the top reason for medical discharge from service in the military, and the leading cause of disability among veterans and service members. In 2013 alone, the cost of arthritis-attributable medical expenditures and lost wages among US adults stood at an astonishing \$303.5 billion. Clearly, the heavy burden of this disease on our economy, national security and quality of life cannot be overstated.



At ANRF, we are mindful of these realities and cognizant of the size of this foe. Undaunted, we work tirelessly on behalf of millions of patients around the world to improve clinical outcomes through enabling innovation and advancing new discoveries. As one of those patients myself with a painful form of Arthritis, I admit that my commitment to this fight is deeply personal. Everyday, through the work of the Foundation, we seek to reduce knowledge gaps and identify effective therapies by collaborating with hundreds of trusted partners, scientists, supporters, patients and stakeholders - I invite you to join us in our plight that has a clear and defined purpose: Identifying new therapies and developing long-lasting cures.

This year in particular has been extraordinary for ANRF. Progress is palpable on numerous fronts. In this annual report, I summarize our strong financial position (pp 6) and share updates on our scientific progress (pp 7-21). Some key accomplishments are further highlighted below:

- **Bolstering Research Grants:** In 2018 we funded our largest cohort of research projects to date, and plan to continue that trend in years to come. This new cohort joins a multitude of 2017-funded projects (detailed in this report) and complements more than 250 completed interdisciplinary studies at 75 prestigious research centers across the United States. These projects are conducted by top young minds at the vanguard of innovation and technological advances.
- **Modernizing Infrastructure & Readiness:** We updated our grantmaking systems, research communications platforms and medical education channels for optimal impact. Through these changes, we will be able to perform our functions more effectively and remain competitive as a leading Medical Foundation. In addition, these changes ensure the highest standards in data security, privacy and encryption.
- **Creating Meaningful Programs:** We launched new research and education initiatives, including: i) An e-newsletter, *The ANRF Chronicle*, to help disseminate the latest credible news in Arthritis, Rheumatology and Autoimmune Diseases as well as Foundation news; ii) A Fact Sheet library as part of a larger plan to facilitate access to information about the disease. Future plans include research webinars, podcasts and other useful learning instruments; and iii) A scientific Symposium to be held in 2019. This conference will bring together annually leaders from across industry, academia, federal agencies, and biotech companies, to collaborate and tackle the most pressing issues that we confront in the field today. That is in addition to sponsoring travel grants to enable young investigators to share and publish their research findings at professional conferences.

We have ambitious plans for the Foundation, yet supporting all these projects without interruption is a costly proposition. To this end, the work we do has been made possible only because of your generosity. So in this season of giving, I hope that we continue to earn your trust as valued partners. I am very thankful to you for making us part of your journey.

With much gratitude,

Zaher Nahle, PhD, MPA

# FINANCIAL REPORT

## 2016, 2017 & 2018

AUDITED STATEMENTS OF PUBLIC  
FISCAL YEARS ENDING MARCH 31st

### REVENUE AND EXPENSES

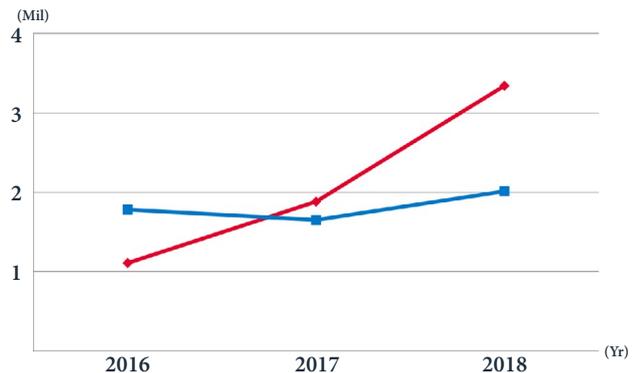
PUBLIC SUPPORT AND REVENUE	2016	2017	2018
Contributions and Bequests	1,212,531	877,844	2,564,271
Investment Income Net	508,816	383,106	378,310
Unrealized Gain (loss) on Investments	(614,508)	622,708	399,538
<b>TOTAL SUPPORT AND REVENUE</b>	<b>\$1,106,839</b>	<b>\$1,883,658</b>	<b>\$3,342,119</b>
<b>EXPENSES</b>			
Program Services			
Research	1,440,782	1,261,339	1,537,187
Education	224,973	243,640	236,428
Total Program Services	\$1,665,755	\$1,504,979	\$1,773,615
<b>SUPPORTING SERVICES</b>			
Management and General	80,039	107,364	115,912
Fund Development	37,027	37,554	120,016
Total Supporting Services	117,066	144,918	235,928
<b>TOTAL EXPENSES</b>	<b>\$1,782,821</b>	<b>\$1,649,897</b>	<b>\$2,009,543</b>
Change in Net Assets	(675,982)	233,761	1,332,576
Net Assets at Beginning of Year	8,651,710	7,975,728	8,209,489
<b>NET ASSETS AT END OF YEAR</b>	<b>7,975,728</b>	<b>8,209,489</b>	<b>9,542,065</b>

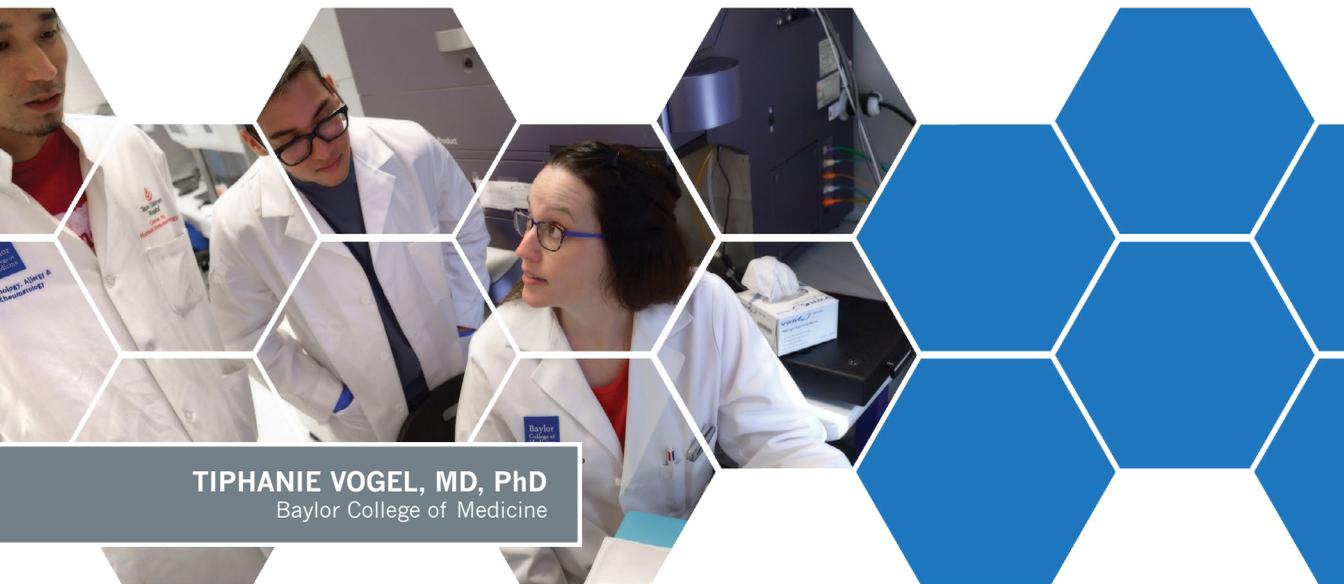
### BALANCE SHEET SUMMARY

Total Assets	\$8,030,832	\$8,267,105	\$9,586,356
Total Liabilities	\$55,104	\$57,616	\$44,291
<b>NET ASSETS AT END OF YEAR</b>	<b>\$7,975,728</b>	<b>\$8,209,489</b>	<b>\$9,542,065</b>

### A simple Revenue/Expenses Chart

- Total Revenue
- Total Expenses





**TIPHANIE VOGEL, MD, PhD**  
Baylor College of Medicine

## PEDIATRIC RHEUMATOLOGIST FINDS GENETIC CLUES IN JUVENILE ARTHRITIS

Early in her career, pediatric rheumatologist, Tiphonie Vogel, MD, PhD, cared for a seven-year old boy with a rare form of juvenile arthritis (JA). She discovered that her young patient had a specific genetic mutation that leads to damage in multiple organs and severely affects the joints.

Dr. Vogel, an Assistant Professor of Pediatrics at Baylor College of Medicine, played an instrumental role in a multinational collaboration that identified the brand-new disease caused by a mutation in a protein called STAT3.

Increased activity of STAT3 causes the immune system to become overactive, leading to inflammation, severe autoimmune disease and a syndrome called STAT3 gain-of-function (GOF). The harmful mutation affects a number of vital signaling and regulatory pathways throughout the body.

With funding from ANRF, Dr. Vogel is trying to pinpoint the region of the protein that spurs the overactivity in STAT3. “I believe that changes in a particular region of STAT3 are more likely to predispose children to JA as a complication,” she explains. “Targeting that area may decrease the overactivity of the STAT3 molecule, which may decrease the risk of autoimmunity.”

Dr. Vogel is working to understand the role of the STAT3 protein in JA. “STAT3 is a very diverse molecule that performs many important functions in the body,” Dr. Vogel explains. “When we switched my patient to a newer

“biologic” medication that blocks IL-6, we successfully treated his arthritis.”

The ultimate goal of Dr. Vogel’s is the discovery of a drug designed to block the specific region of the STAT3 molecule to help these kids.

“The average age of onset of STAT3 GOF syndrome is four and half,” says Dr. Vogel. “My thinking is that if we can show the role of STAT3 in juvenile arthritis it could lead to new therapeutic targets.”

Her approach is two-fold: How to target the cellular region where the STAT3 GOF mutation arises, and how to mimic the change in a mouse model. Since mice have an almost identical STAT3 protein, her research team takes the mutation from a patient and creates a “transgenic” mouse with the same mutation.

“We can examine the mutation at a level that’s not possible in people,” she explains. “Using a microscope, we can see exactly what overactive STAT3 does to the joint.”

### JUVENILE ARTHRITIS RESEARCH

**“I find my inspiration from taking care of patients,” says Dr. Vogel. “I work on understanding the causes and mechanisms of juvenile arthritis in my laboratory because I one day hope to be able to provide a treatment that resolves their disease.”**

## TISSUE ENGINEERING MAY CHANGE HOW WE TREAT OSTEOARTHRITIS

Re-growing cartilage that could simply be transplanted into weight-bearing joints like hips and knees is a new tissue engineering technique which may completely change how osteoarthritis (OA) is treated.

With funding from ANRF, Grace O’Connell, PhD, an Assistant Professor of Mechanical Engineering at the University of California, Berkeley, is investigating the mechanics of new cartilage formation.

When harvesting cartilage cells from patients to build new tissue, some cells are able to grow new healthy tissue, while others remain dormant. Dr. O’Connell’s research is aimed at improving early detection of which cells are most likely to produce strong cartilage.

“We wanted to identify early on which cells were likely to produce good tissue,” says Dr. O’Connell. She and her colleague, Dr. Lydia Sohn, also a professor in Mechanical Engineering at Berkeley, developed a novel screening technique to identify the best cell candidates for tissue production. Cells are pushed through a device that causes the cell to deform as they travel through a thin channel.

Cells are dynamic, squeezing, flexing and sticking as they are pushed through the device. “The screening device allows us to measure the stiffness of cells and surface expression of markers on the cell,” Dr. O’Connell adds. “We can see when cells start to deform and change their shape.”

The screening device is designed to predict which cell sources will result in sufficient tissue production.

Today, clinical trials at research centers around the country are taking patient cells, placing them in gel and growing them “in a dish,” but the device created by Dr. Sohn at Berkeley adds a crucial ability to measure the cell mechanics of these samples. Someday, scientists hope to harness the innate behavior of cells to repair cartilage lost to osteoarthritis.

### TISSUE ENGINEERING

**“Once we understand all the factors that influence new tissue production,” says Dr. O’Connell, “we can translate the findings to better tissue engineering solutions.”**

Dr. O’Connell is grateful to ANRF for funding the basic science that she believes will lead to new discoveries. “Professionally, the grant is a huge honor,” she says. “We are so excited to continue this promising research.”

“Osteoarthritis affects almost everyone as we age,” says Dr. O’Connell. “Right now, effective treatments are limited. People with painful OA, especially younger patients who are holding off on current surgical repair strategies, will greatly benefit from a biological repair strategy that can reduce or eliminate the need for total joint replacement.”



**GRACE O’CONNELL, PhD**  
University of California, Berkeley



**SHAUN JACKSON, MD, PhD**  
Seattle Children's Hospital

## HELPING MORE LUPUS PATIENTS TACI MAY HOLD THE KEY

Systemic Lupus Erythematosus (Lupus) hijacks the body's defenses, launching an attack on healthy tissues and organs. An estimated 1.5 million Americans suffer from the chronic autoimmune disease known as Lupus.

At Seattle Children's Research Institute, Assistant Professor of Pediatrics, Shaun Jackson, MD, PhD, is studying how and why the immune system goes awry. "Lupus is complex and manifests differently among individual patients," he says.

The severity of lupus can range from a mild skin rash or joint pain to life-threatening inflammation. B cells are a type of white blood cell that defend the body against infection. In healthy individuals, B cells produce targeted antibodies against disease-causing microbes.

Unfortunately, in certain susceptible individuals, faulty signaling in B cells can also result in the formation of disease-associated autoantibodies. Dr. Jackson's research focuses on understanding how B cells are activated to produce autoantibodies leading to the development of lupus.

Over the last decade, researchers have discovered that increased levels of a B cell survival factor called BAFF (also known as BlyS) are tied to the development of lupus in humans and animal models. The research led to the BAFF inhibitor Benlysta® (belimumab), which was the first new medication for lupus in more than 50 years.

"Unfortunately, Benlysta doesn't work for every lupus patient," says Dr. Jackson. BAFF promotes B cell survival and activation by binding to two different receptors on

the surface of B cells. Dr. Jackson's lab discovered that the B cell surface receptor, TACI, is required for BAFF to promote B cell autoantibody production.

"We found that removing the TACI receptor in mice with very high BAFF levels cured the disease in a mouse model of lupus," said Dr. Jackson.

"We then identified a specific subset of B cells that express high levels of TACI and appeared to spur the development of autoantibodies. We now believe that blocking BAFF signaling through TACI might be the key to preventing ongoing autoantibody production in a subset of lupus patients, particularly those with the highest serum BAFF levels."

"Lupus strikes genetically susceptible individuals," says Dr. Jackson. "Some patients respond well to treatment, while others don't. We predict that a reason for this is that distinct mechanisms may drive lupus in different individuals. Our hope is that improved understanding of how autoantibodies can be generated via different B cell activation pathways may allow for the development of new targeted therapies."

### LUPUS RESEARCH

**"I hope to continue to devote the majority of my time to research," says Dr. Jackson. "As a doctor who specializes in helping kids with kidney damage, new treatments can't come soon enough."**



**DEBORAH R. WINTER, PhD**  
Northwestern University

## THE INTRIGUING ROLE OF MACROPHAGES IN RHEUMATOID ARTHRITIS

Macrophages are immune cells that swallow bacteria, viruses or infectious germs. When the body comes under attack, these white blood cells engulf the attackers and eliminate the threat. With funding from ANRF, Deborah R. Winter, PhD, an Assistant Professor in the Division of Rheumatology at Northwestern University Feinberg School of Medicine, is studying the role of macrophages in chronic inflammatory diseases like rheumatoid arthritis (RA).

“Macrophages arise in the bone marrow as precursor cells called monocytes,” explains Dr. Winter. “When the immune system works normally, those circulating monocytes transform into macrophages that swallow the invaders. In RA, macrophages that are supposed to disappear can linger and inflame the joints.”

The immune system declines with age and scientists want to know whether the same processes that drive aging also drive RA. As people age, stem cells in the bone marrow often spur the overproduction of monocytes that lead to chronic inflammation.

“We found that monocytes are a problem even before they become macrophages,” says Dr. Winter. “In mice with RA-like disease, monocytes are predisposed to inflammation. These cells go straight to the joints via the bloodstream resulting in the painful joint swelling and stiffness of RA.”

Dr. Winter’s mouse models showed that while aging mice had a larger number of monocytes, in arthritic mice, monocytes were funneled directly to the joints. As research breakthroughs continue to bring new hope for

people with RA, scientists are looking into the genome in depth to understand why macrophages go awry in RA. In her lab, Dr. Winter is using the power of genomics to explore how macrophages promote chronic inflammatory diseases.

### RHEUMATOID ARTHRITIS RESEARCH

**“I believe that genomic approaches, such as mine, hold the key to fully understanding the development of RA and similar diseases,” she says.**

Dr. Winter is using her background in computational biology and immunology to decipher complex patterns of gene expression. Although macrophages are found throughout the body, they are programmed for specific functions.

Her findings suggest blocking the production of macrophages that boost joint inflammation could slow or prevent RA. “Currently available treatments decrease inflammatory substances throughout the entire body,” she says, “but these drugs also compromise the body’s ability to respond to infection. Instead of taking a hammer to the whole body, we want to find a way to hit the nail safely on the head.”

Thanks to ANRF, she was able to establish an independent lab. “This award was a big jump for me as an investigator,” says Dr. Winter. “Genomic approaches offer really exciting potential targets for individuals who are suffering from this devastating disease.”

## PINPOINTING THE DEVELOPMENT OF OSTEOARTHRITIS

Osteoarthritis (OA) is the leading cause of chronic disability in America. It is a painful inflammatory response in the joints, resulting in the breakdown of cartilage and damage to surrounding tissues. Without these important buffers, bones often become damaged, leading to agonizing pain, swelling, stiffness and loss of mobility.

With funding from ANRF, Tao Yang, PhD, an Assistant Professor and bone disease expert at the Van Andel Research Institute, is investigating the complex web of biochemical cues that regulate skeletal health, including the development of osteoarthritis.

### OSTEOARTHRITIS RESEARCH

**“We are laying a foundation for future research that will advance our efforts to develop new therapies for osteoarthritis,” says Dr. Yang.**

Dr. Yang’s lab studies mechanisms regulating skeletal physiology and disease development. “Given the prevalence of OA and the debilitating effects that it can have, new strategies for treating the disease are becoming increasingly urgent, particularly as current treatments are very limited,” he says.

Multiple factors contribute to the development of OA and the loss of cartilage, including aging, injury and a genetic predisposition. An additional culprit in the aging process is the exhaustion of stem cells that normally give rise to healthy bones. Researchers have also found a decrease in a vital enzyme in patients with OA and this specific mutation dramatically raises the risk of developing osteoarthritis.

“Our idea is that this pathway is key to maintaining the integrity of the joints and so we are looking for ways to enhance its activity or availability in order to delay the development of OA,” says Dr. Yang.

Thanks to his grants from ANRF, Dr. Yang’s lab is continuing to investigate these biochemical pathways using a unique model of the disease, which allows them to study OA in a controlled way. “We found that mice without this enzyme aged more quickly,” he says. “We are exploring whether we can protect against OA caused by aging or injury by increasing its activity in the cells of the joints.”

Both bone and cartilage arise from particular stem cells or progenitor cells that also play important roles in skeletal growth and repair. They have found that a mechanism responsible for protein “tagging” and “de-tagging” can influence the functions and levels of aging-related proteins in skeletal progenitor cells. A disruption of such a mechanism in mice results in premature aging in the skeletal system.

“Using our animal model, we hope to find ways to enhance the effect of this important enzyme in the joints,” says Dr. Yang. “I continue to be inspired by my grandfather, whose OA caused him enormous pain and made him give up his favorite sports... I realized that OA, which is seemingly a disease of the joints, can cause a chain reaction that broadly affects a person’s life style and their overall health.”

Researchers in Dr. Yang’s lab are exploring ways to disrupt the degenerative process and improve skeletal health. Thanks to the discovery of this key enzyme, Dr. Yang’s research is bringing hope for a new OA treatment.



**TAO YANG, PhD**  
Van Andel Research Institute



**MICHELA LOCCI, PhD**  
University of Pennsylvania

## HOW DOES RHEUMATOID ARTHRITIS START? SPECIALIZED CELLS?

Our immune system has evolved to be on alert to protect the body. The immune system's adaptive mechanisms include disease-fighting T-cells and B-cells with "immunological memory." This memory allows the cells the capacity to thwart the same type of attack in the future.

In healthy people, B cells produce antibodies that circulate, seek out and destroy pathogens. Pathogens are infectious bacteria, viruses or harmful microorganisms in your body. The T-cells are crucial white blood cells that tailor the immune response to the invading pathogens.

### **RHEUMATOID ARTHRITIS RESEARCH**

**With funding from the ANRF, Michela Locci, PhD, at the University of Pennsylvania, is studying highly specialized cells (called T follicular helper (Tfh) cells) to understand how overproduction of these T-cells may fuel RA.**

"Tfh cells, defined by their particular function as B-cell helpers, are considered pivotal regulators of antibody responses," she explains. "They are highly specialized in helping the B-cells react to pathogens, and produce antibodies that can efficiently block pathogen infection."

Antibodies are strategic molecules, normally produced by our immune system to confer protection from infectious pathogens. When RA strikes, antibodies that are the

body's natural defense against infection and disease, instead turn on the body and attack self-tissue in the joints. As the autoantibodies accumulate in the joints they can amplify the swelling, stiffness and debilitating pain of rheumatoid arthritis.

The presence of autoantibodies has been linked to abnormally high levels of Tfh cells in RA patients. "It's consistent with the idea that Tfh cells are crucial for regulation of antibody responses," added Dr. Locci.

Dr. Locci has developed a high-throughput screen of molecules and identified the molecule, activin A, as an important player in the regulation of human Tfh cells. As a result, Dr. Locci is now investigating the association between activin A levels and Tfh cells in RA patients.

"We found that activin A is a potent stimulus in regulating multiple aspects of Tfh cell biology in healthy people."

"Tfh cells are crucial for regulation of antibody responses, but what happens when there is excessive Tfh production and does Activin A result in this excessive generation?"

Dr. Locci relates that the grant from the ANRF arrived at a very critical moment in her scientific career. "Personally, this award is invaluable," she says. "I'm especially grateful because it gives life to the possibility that my research might directly improve the health of sick people and reinforces my dedication to helping patients."

## STEM CELL RESEARCH TO REGENERATE CARTILAGE

Stem cell research discoveries are driving additional research into the molecular mechanisms of cartilage regeneration and repair.

### STEM CELL RESEARCH

**New stem cell driven therapies are desperately needed to heal or replace damaged cartilage in joints ravaged by osteoarthritis (OA).**

Articular cartilage develops when you're in the womb to provide a smooth gliding surface for joint movement. Once lost to OA, the smooth layer of cartilage no longer cushions the ends of the bones. Today, total joint replacement surgery is the only treatment available for this painful disease, but that is about to change thanks to stem cell research.

In the near future, scientists hope to treat damaged cartilage with grafts of newly grown cartilage tissues derived from stem cells. At Boston Children's Hospital, ANRF funded researcher, April Craft, PhD, is working to harness the power of stem cells to generate new articular cartilage. "The challenge with cartilage repair and regeneration is that the articular cartilage lining our joints forms prenatally and regeneration does not normally occur after birth."

Dr. Craft, an Assistant Professor at Harvard Medical School, is coaxing stem cells to grow into articular cartilage in a petri dish. Using pluripotent stem cells, cells

that can generate into any cell type, she has generated specialized cells called chondrocytes. Chondrocytes are the cells that make up cartilage tissues, and part of their job is to make proteins that help distribute the load and lubricate joints.

The goal of this stem cell research is to generate articular cartilage that can be successfully transplanted into a patient's knee or other joints. "The challenge with using pluripotent stem cells is to reliably and efficiently generate large numbers of articular chondrocytes and cartilage tissues," she explains.

The ability to create more effective implants for long-term cartilage repair will improve dramatically with increased knowledge of how these tissues develop normally in the body. "We were the first to demonstrate that human articular cartilage tissues generated in this manner are very similar to those found in our joints," says Dr. Craft. "Now we have an opportunity to learn more about cartilage development, and define characteristics of cells that have the greatest ability to regenerate this tissue."

Cartilage tissues grown in a lab setting offer a safe and non-invasive way to screen for new drugs that might protect against further damage.

"The field of regenerative medicine is exploding," says Dr. Craft. "With ANRF support, we are moving step by step to identify the most promising cells to grow 'cartilage in a dish' to achieve our goal of healing damaged joints."



**APRIL CRAFT, PhD**  
Boston Children's Hospital | Harvard Medical School

# ANRF | GIVING GUIDE

*Suggestions to Support our Foundation*

100K

## BIOMEDICAL RESEARCH GRANTS

Fund groundbreaking research in Arthritis, Rheumatology and Autoimmune Diseases conducted by talented MD and PhD professionals at prestigious institutions. This is critical for filling knowledge gaps, identifying new therapies and finding longlasting cures.

50K

## SCIENTIFIC SYMPOSIUMS

Convene top minds in scientific research and clinical care to collaborate and cross-pollinate. Information sharing is a key component in accelerating the discovery process and improving the quality of research and clinical care.

25K

## MEDICAL EDUCATION & AWARENESS

Disseminate the latest news in Arthritis, Rheumatology and Autoimmune Diseases through credible and FREE newsletters, webinars, fact sheets and other digital and print mediums. Access to accurate and concise information to stay up-to-date is needed in a constantly changing medical field.

10K

## RESEARCH TRAVEL GRANTS

Facilitate the direct access of young trainees and scientists with limited resources to participate in professional meetings and publish their work. This is indispensable for sharing ideas, receiving feedback and sustaining the pipeline of new talent entering the field of Arthritis and Rheumatology.

5K

## INFRASTRUCTURE & READINESS

Keep our grantmaking systems, research platforms and Medical Education channels up-to-date for optimal impact. This will enable us to perform our functions effectively and remain competitive as a leading Medical Foundation.

\$

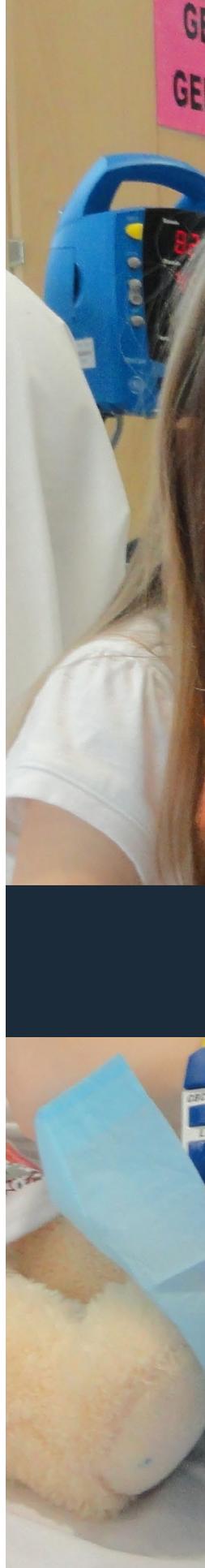
## EVERYTHING GIFT

Supports a combination of the abovementioned programs where funds are needed the most.

1K

500

100





## LEAVE A LEGACY

*Make a planned gift or donation today to leave a lasting impact in the search to cure arthritis!*

- Give Online at [CureArthritis.org](http://CureArthritis.org)
- Leave a gift in your will
- Give through your workplace
- Donate through your Donor Advised Fund or IRA
- Donate your car
- Make monthly or annual gifts
- Give through the Combined Federal Campaign (CFC)
- Give us a call - 800.588.2873 or email us at [info@curearthritis.org](mailto:info@curearthritis.org)



**CHARITY NAVIGATOR**

Four Star Charity

*Use the enclosed envelope or go to*

**CUREARTHRTIS.ORG/DONATION**

## CAN EPIGENETICS SWITCH OFF GOUT?

Epigenetics, the study of changes in organisms caused by modification of gene expression, is shining light on the ability to switch off gout.

An acute attack of “gouty arthritis” comes on rapidly, triggering severe pain, warmth, swelling, redness and tenderness in the affected joint. High levels of uric acid in your bloodstream (called “hyperuricemia”) can cause urate crystals to build up in and around your joints. Scientists suspect that certain genes and changes in DNA may play a significant role in the development of gout.

Richard Reynolds, PhD, an Assistant Professor at the University of Alabama at Birmingham, is exploring genetic susceptibility to gout. With his ANRF funding, Dr. Reynolds is combing through large population data sets for biomarkers that may explain why gout frequently co-occurs with one or more additional inflammatory diseases or disorders.

8.3 million Americans suffer from gout, looking for specific causative genes is like looking for a needle in a haystack. However, he says that “rather than analyzing traits and genetic markers one by one, we have the statistical tools and computational methods to analyze all the genetic markers and traits at the same time.”

“We want to understand which genes and factors control gene expression affecting gout and hyperuricemia,” says Dr. Reynolds. “Our goal is to identify regions in our genome that contribute to the accumulation of uric acid crystals.”

### GOUT RESEARCH

**Dr. Reynolds theorizes that inflammatory arthritis is driven by a complex interplay between genetics and lifestyle. Meat, seafood and alcohol can cause a flare of gout, but family history is also a primary contributor to the disease.**

Researchers are now using engineered cells that can “turn off” segments of your DNA. Someday the hope is that DNA-guided medicine will help doctors tailor medications to each individual’s genetic markers.

However, these “control switches” that change the way genes are expressed can also be triggered by obesity and environmental factors. Although such “epigenetic” changes in gene activity don’t alter the genetic code, they can still be passed down through several generations.

One promising area of epigenetics is “DNA methylation.” DNA methylation silences specific genes by attaching methyl groups to specific bases that comprise sequences of DNA. “Genetics explains about 35% of variation in serum urate levels, and we estimate that DNA methylation can account for another 23%,” says Dr. Reynolds.

New approaches based on epigenetics may someday lead to novel treatment approaches for gout.



**RICHARD REYNOLDS, IV, PhD**  
University of Alabama at Birmingham



**BRIAN DIEKMAN, PhD**  
University of North Carolina at Chapel Hill

## THE IMPACT OF AGING IN OSTEOARTHRITIS

Aging is the primary risk factor for the development of osteoarthritis (OA). Every day, people suffer from grinding bone-on-bone pain, chronic stiffness, cartilage damage and the nagging aches of this degenerative joint disease.

Thanks to the co-funded Arthritis & Aging Research Grant, Brian Diekman, PhD, Assistant Professor at the University of North Carolina at Chapel Hill is examining biological aging, or “senescence.”

Senescence sparks a biological chain reaction that destroys articular cartilage. Cartilage lacks the capacity to regenerate and aging spurs destructive cellular changes that can lead to permanent joint damage. Scientists are racing to explore the biology and genetics of aging in an effort to turn back the clock on aging joints.

### OSTEOARTHRITIS & AGING

**“My lab is investigating changes that occur in cartilage during aging and osteoarthritis that predispose the tissue to break down.”**

“We are particularly interested in whether cartilage shows signs of senescence in response to chronic stress and if they produce molecules that drive age-related diseases.”

So far, it has been challenging to identify the underlying mechanisms that cause joints to fail with age and even harder to develop therapies to halt or reverse age-related changes. “As cells become senescent, they secrete

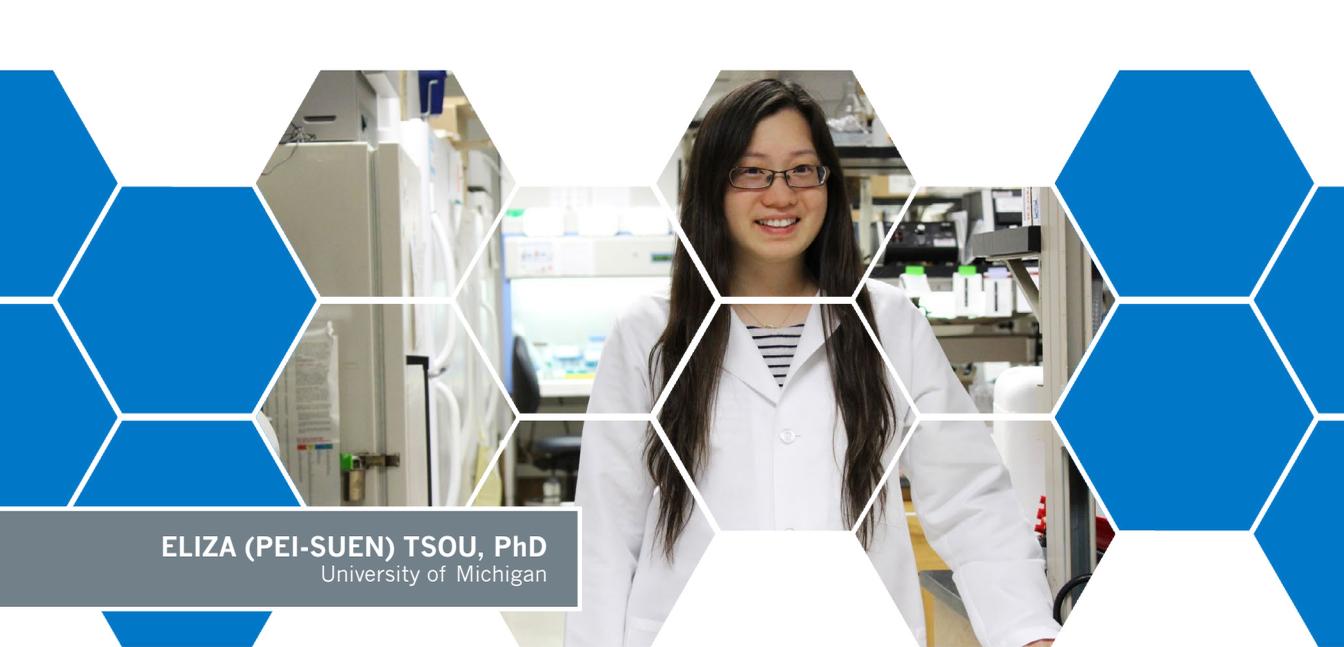
inflammatory factors that compromise cells and the tissues around them,” adds Dr. Diekman. “Since cartilage can’t regrow, attacking the senescent cells might allow the surrounding cells to thrive.”

“In our lab, we are developing tools to either limit the development of senescence or to specifically eliminate these cells from the joint to prevent them from affecting neighboring cells that might otherwise remain healthy.”

Dr. Diekman is researching ways to block senescence by finding precise ways to measure both the progression of the disease and the effectiveness of new treatments. He plans to target and test several molecules that might eventually be translated into new treatment options for osteoarthritis.

“The therapy would work by selectively removing senescent cartilage cells,” he explains. “If these senescent cells are part of what is driving the inflammatory component of OA, then eliminating these cells may allow the surrounding tissue to function more effectively.”

Selectively targeting senescent cells in joints could transform the current landscape of OA treatment. “I am motivated by stories from arthritis patients who are hopeful knowing that people are working their hardest to develop new therapies,” he adds. “Launching a lab is an expensive and ambitious proposition. Funding from the ANRF gave me the opportunity to dream big and develop the innovative work that will hopefully lead to new cures for arthritis.”



**ELIZA (PEI-SUEN) TSOU, PhD**  
University of Michigan

## NEW UNDERSTANDING OF GENES MAY LEAD TO SCLERODERMA TREATMENT

Scleroderma stiffens connective tissue and decreases blood vessel formation throughout the body, scarring the skin and triggering potentially lethal damage to vital organs.

Researchers theorize that such “epigenetic” changes disrupt gene expression without altering the DNA sequence. Scientists suspect that the disease results from subtle flaws in how our cells read our genes.

With funding from ANRF, Eliza PS Tsou, PhD at the University of Michigan, is investigating how epigenetics influences both skin thickening and impaired blood vessel formation in scleroderma.

“New understanding of gene expression may completely change how we treat scleroderma,” says Dr. Tsou. “The high mortality rate and few therapeutic options reflect our lack of understanding of the underlying molecular mechanisms in scleroderma. Our goal is to understand the disease and identify targets for scleroderma treatment that could lead to a cure.”

Like other inflammatory disorders, there is no cure for scleroderma. The chronic autoimmune disease causes the body to attack the joints, skin and internal organs often with life-threatening consequences.

“What makes my project unique is that I am able to isolate two cell types from patient skin biopsies - cells from the blood vessels and others that cause the skin to thicken,” says Dr. Tsou. “From these cells we can examine

why they are sick, why they behave differently and identify the pathways or molecules causing them to act abnormally so we can find ways to intervene and make these sick cells normal again.”

Dr. Tsou and her lab can isolate and grow these blood vessel cells for research so they are tackling the key epigenetic players that stiffen skin and hinder blood vessel formation.

“Although the skin thickening cells are easy to isolate, the blood vessel cells are more difficult to purify,” says Dr. Tsou. Her research has shown that a substance, HDAC5, blocks blood vessel formation in scleroderma.

“When we decreased the expression of HDAC5 in scleroderma cells, those cells resumed normal function.”

### **SCLERODERMA RESEARCH**

**Using techniques to scan the genes in blood vessel cells and supercomputers to crunch the data, she discovered a protein, CYR61, is crucial for scleroderma blood vessel cell function and also slows skin thickening.**

“CYR61 may be beneficial for scleroderma through its anti-skin thickening and pro-blood vessel growth properties,” says Dr. Tsou. “Since CYR61 can tackle the blood vessel and skin thickening problems at the same time, it is a great candidate for a drug target.”

## OSTEOARTHRITIS TREATMENT MAY HALT OR REVERSE KNEE INJURIES

A sudden twist of the knee can tear the cartilage that cushions and stabilizes the joint. Knee injuries often predict the development of osteoarthritis (OA). Even when an injury is surgically repaired, damage to cartilage triggers a cascade of degenerative cellular changes.

“Physical insult or aging begins the process of OA,” says Fadia Kamal, PhD, an Assistant Professor at Penn State College of Medicine. “Once the process of degradation begins at one site of the articular cartilage, it doesn’t stop there,” says Dr. Kamal. “The destructive process spreads to the whole cartilage surface of the knee.”

Researchers have identified special cartilage cells that ensure a smooth gliding surface for the joints. Although all the cartilage in your body develops before you’re born, your cartilage is continuously being replenished and renewed. When you sustain knee injuries that tear your joint cartilage, cells at the injury site change and becoming bigger and with altered DNA profiles. Instead of continuously renewing and replenishing the cartilage, these unruly cells start eating up the cartilage around them.

“This process is a major contributor to disease, so it’s very attractive for drug development,” says Dr. Kamal. “No drugs are currently available that can slow down or stop cartilage degeneration in OA, but our data suggests that we have discovered drugs that can. We are now investigating a drug that not only stops the overgrowth of abnormal cells, but also restores their normal shape and function.”

With ANRF funding, Dr. Kamal is studying the role of proteins that activate key inflammatory signaling pathways. Her research involves cell surface receptors, G-protein-coupled receptors (GPCRs), that act like an inbox for messages that spark continuous cartilage degradation.

“These studies are game-changing in the field of OA,” says Dr. Kamal. “My hope is that the grant will enable me not only to discover new and effective treatments for OA, but also establish a major research line in my lab that will answer a lot of questions regarding the role of GPCR signaling in cartilage development and disease.”

One of the inhibitors researchers are studying is a repurposed FDA-approved drug, Paxil (paroxetine), which is currently used as an antidepressant.

### KNEE INJURY TREATMENT

**“We think that the drug also inhibits systemic inflammation,” says Dr. Kamal, “but we will answer this question with further studies.”**

As a pharmacist, Dr. Kamal saw people suffering with OA, including her own grandmother, who underwent two knee replacement surgeries. “Remembering the struggles of patients compels me to contribute to the development of new therapies. Someday, we hope to actually treat this disease and provide people with a better quality of life.”



**FADIA KAMAL, PhD**  
Penn State University

## DATA MINING UNEARTH'S GENETIC CLUES IN PSORIATIC ARTHRITIS

Psoriatic arthritis (PsA) is an aggressive and joint-destroying inflammatory disease that can lead to crippling joint pain, stiffness and swelling. Finding genetic clues in PsA has proven difficult, but ANRF Scholar, Lam “Alex” Tsoi, PhD, research assistant professor at the University of Michigan, has started finding those elusive genetic clues.

PsA is often preceded by psoriasis, which erupts as itchy, scaly patches on the skin. The same underlying inflammation that appears as red, flaky “plaques” on the skin can also progressively damage joints, cartilage and muscle tissue.

### PSORIATIC ARTHRITIS RESEARCH

**“Our goal is to assess who will be more likely to develop psoriatic arthritis before symptoms appear.”**

“Skin symptoms often develop first, and then joint pain follows. Once the joint pain occurs, it often signals that there has been significant joint damage.”

With funding from ANRF and our partner the National Psoriasis Foundation, Dr. Tsoi is studying both the genetic and environmental factors that play a role in the onset of PsA. His challenge is to develop an accurate predictive model to understand why and who is susceptible to PsA.

Using the power of data-driven approaches, called bioinformatics, Dr. Tsoi is exploring molecular mechanisms and susceptibility genes that promote inflammatory diseases.

The sequencing of the human genome offered researchers a window into the molecular genetics of every individual. Now, researchers are unscrambling the genetic mechanisms that trigger devastating inflammatory diseases.

By leveraging proprietary software analysis tools, big data and machine learning technologies, Dr. Tsoi is developing techniques to tailor personalized approaches to medicine.

“We are working to identify specific genetic signatures that will allow us to predict who will or will not develop psoriatic arthritis,” he explains. “Symptoms of psoriatic arthritis can change over time, getting better or worse without warning... Risk assessments would help clinicians offer personalized management to patients at a higher risk for developing the disease.”

Bioinformatics could offer a way to anticipate who will develop PsA, so doctors can follow up before symptoms appear. Most patients get an annual check up, but high-risk individuals could be followed more closely. Dr. Tsoi knows that any delay in diagnosis is harmful and he hopes that his research will lead to techniques that can help patients earlier, before the joint damage occurs.



**LAM (ALEX) TSOI, PhD**  
University of Michigan

**JASON KNIGHT, MD, PhD**  
University of Michigan



## LUPUS RESEARCH AIMS TO PREVENT HEART ATTACKS

Jason Knight, MD, PhD, an Assistant Professor of Rheumatology at the University of Michigan, is using his ANRF funding to drive his lupus research and investigate new potential treatments. “Lupus has a startling ability to damage any organ in the body—kidneys, lungs, liver, brain and blood vessels.”

### LUPUS RESEARCH

**Autoimmune disorders like lupus and rheumatoid arthritis overwhelmingly afflict women, especially during the childbearing years. “Women are 10 times more likely than men to develop lupus,” says Dr. Knight.**

Currently, immune-suppressing drugs are used to prevent sudden disease flares and symptoms. But Immune-system suppressing treatments are ‘nuclear options’ because they disrupt the entire immune system.

Thanks to modern medications and kidney transplants, the odds of dying from lupus related kidney disease have decreased sharply. However, as patients live longer, cardiovascular diseases such as heart attacks, strokes, and blood clots are now the new leading cause of death from lupus.

“When someone develops blood clots, we prescribe potent blood thinners,” says Dr. Knight. “These drugs must be taken for life, but pose dangerous bleeding risks.

Treating an autoimmune disease with blood thinners is inherently dissatisfying to the patient and the physician.”

Researchers are creating targeted therapies to treat, and ultimately prevent, life-threatening cardiovascular complications in lupus patients.

Neutrophils, the most abundant type of disease-fighting white blood cells, attach to the walls of the blood vessels to block assaults on the immune system. Neutrophils extrude their sticky insides into spider web-like structures, perfect for capturing invaders. Although these neutrophil extracellular traps (NETs) help stave off microbial infections, they also serve as excellent ‘scaffolding’ for the assembly of blood clots.

“We hypothesize that the over-exuberant release of NETs is why lupus patients form blood clots,” explains Dr. Knight.

Gene-profiling experiments have revealed that molecules responsible for “adhesion” are highly expressed on the surface of neutrophils from patients with lupus. These molecules function like Velcro, making the neutrophils stickier and more likely to adhere to the blood vessel wall.

With funding from ANRF, Dr. Knight’s laboratory is pioneering gene techniques and cutting-edge microscopy. “We believe the NET-releasing weapon is not fully activated until a neutrophil sticks to the blood vessel wall,” he says. “If we can block those interactions, we can prevent clotting, which in turn may prevent heart attacks.”

## UNDERSTANDING THE BIOMECHANICS OF OSTEOARTHRITIS PAIN

When osteoarthritis (OA) strikes, pain flashes to sensory receptors, unleashing tiny electrical pulses that race up the spinal cord to the brain. Neurons, or specialized nerve cells, release chemical messengers that deliver pain signals.

ANRF Scholar, Rachel Miller, PhD, Assistant Professor at Rush University Medical Center, is seeking answers to puzzling questions about OA by investigating how mechanical stimuli trigger OA pain. The debilitating pain of osteoarthritis is a leading cause of disability worldwide and to date, there are no targeted therapies.

Designing treatments for OA are difficult because researchers don't know which cells sense mechanical stimuli and lead to pain. Dr. Miller is looking at neurons & cartilage cells that sense and respond to mechanical stimuli.

“Despite the fact that pain is the major symptom of OA, the sources of pain associated with OA are still understudied,” explains Dr. Miller. “We don't even know precisely where the nerves that sense pain signals and other types of touch signals are located within the joint.”

Dr. Miller is exploring pain mechanisms and she hopes to pinpoint how sensory neurons misfire to cause pain. “Everybody has sensory neurons that respond if you touch a hot stove,” she says.

### OSTEOARTHRITIS PAIN

**“When you are in chronic pain, just walking around is painful. We want to understand the complex process that occurs when cartilage degrades and signals the nerves to feel pain.”**

Dr. Miller developed an imaging technique that allows her team to visualize what happens when researchers apply a pinch or a twist to the knee. “We can watch the neurons fire in real time,” she explains. “The images show that a particular type of neuron responds in OA.”

The goal is to find ways to directly target pain pathways. “I hope to discover how cells within the knee joint sense and respond to mechanical loading,” says Dr. Miller, “Understanding how cells respond to mechanical load normally, and how it changes in OA, may lead to the development of new drugs to treat osteoarthritis.”

Dr. Miller says that studying OA is personal because a lot of her friends and family members have suffered from the disease. “Receiving a grant from the ANRF at this early stage in my career is very important, it establishes me as an independent researcher and will help others in pain.”



**RACHEL MILLER, PhD**  
Rush University Medical Center



## A MESSAGE FROM THE PRESIDENT OF THE BOARD OF DIRECTORS

Dear Partner,

In 2018, I was privileged to be elected President of the Board of Directors of ANRF. One of our core functions at the Foundation is to select for funding, through a highly competitive review process, research proposals submitted by some of the brightest minds in the fields of Arthritis, Rheumatology and Autoimmune Diseases. This support enables awardees to conduct innovative studies that push the boundaries of science and medicine through new discoveries and groundbreaking results. Importantly, our grantees are invariably young investigators at the formative stages in their careers. As such, ANRF support is very important to their productivity - it accelerates not only their professional growth but also shapes their outlook as future leaders in their fields.

The quality of our research program is matched by an unwavering commitment for transparency and good governance at ANRF. On our board, we are extremely fortunate to have consummate professionals from various disciplines volunteering their time and expertise in the service of the Foundation. Collectively, our Board oversight - performed through frequent board meetings as well as specialized committee functions - ensures timely and accurate reporting as well as excellent fiduciary management throughout the organization. Recent work by various committees includes revising the bylaws, updating our investment policy and institutionalizing a series of system-wide changes. These changes have been designed to give us the flexibility needed to execute and expand our functions effectively, while simultaneously performing our duties with the highest ethical and transparency standards. To date, we are thrilled to have maintained a four star charity navigator rating for 10 years in a row.

This year was also a year of conspicuous gains and losses at the Foundation with new beginnings and difficult goodbyes. We welcomed Dr. Nahle as the new Executive Director, an outstanding leader who is taking the Foundation methodically to new heights. Sadly, we also endured the sudden departure of Dr. Morrie Granger, a giant who left an indelible mark on ANRF and transformed the Foundation to what it is today. Morrie (pp 24) is credited, among other things, with assembling a world-class Scientific Advisory Board (SAB) that continues to provide great depth to our work as well as the skill to select the most meritorious research grants every year.

Going forward, we are committed to using all the different instruments and tools at our disposal to further the growth of our Foundation. We will fill severe knowledge gaps, invest in innovative ideas, partner with patients for solutions, facilitate meaningful collaborations, create reliable sources with trusted research information, participate in and lead conferences and think tanks, advance medical education, spread much needed awareness about the disease, and work constructively with stakeholders and partner organizations towards better understanding Arthritis and associated Rheumatic and Autoimmune Diseases, joining forces whenever possible.

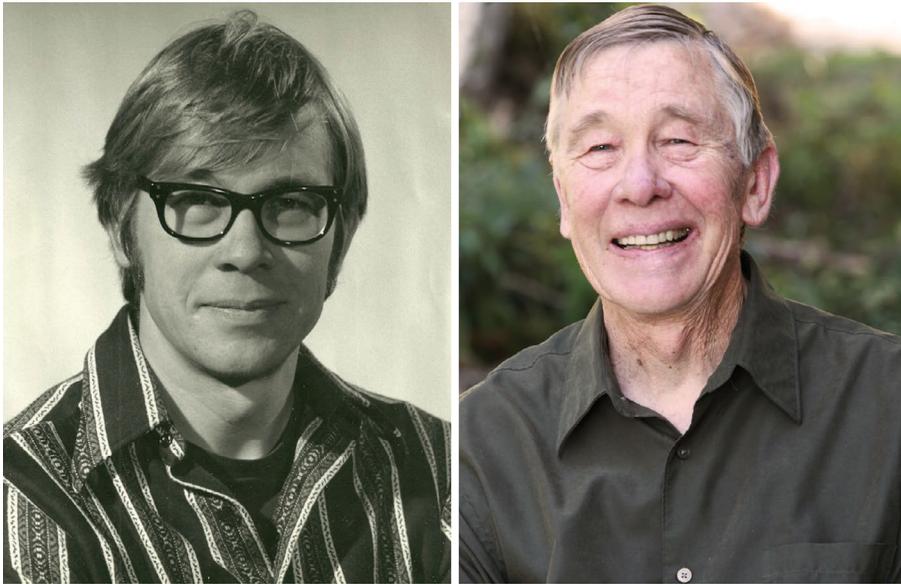
In this holiday season, I wish the best to you and to your family. I hope that we can continue to count on your generosity and support in this unprecedented phase of growth at ANRF.

Sincerely,

A handwritten signature in black ink, appearing to read 'Debra Sampson'.

Debra Sampson  
Board President

# *In Dedication and Loving Memory of*



## *Gale "Morrie" Granger, PhD*

1937-2018

*By Zaher Nahle*

Morrie Granger, whose groundbreaking discoveries shaped modern therapeutic modalities in Arthritis, Immunology and Cancer, left us earlier this year at the age of 81.

Morrie's commitment to raising the next generation of thinkers was unparalleled, and his selfless dedication to ANRF remains our inspiration and will for generations to come. Whether you knew Morrie for ten minutes or ten years, you came away energized, even touched by the generosity of his spirit and the kindness of his words.

Remarkably, Morrie himself was an early recipient of funding from ANRF and the Foundation's first ANRF scholar, a distinction he embraced with pride and humility. Such deep connection to ANRF fueled his lifelong commitment to raising the next generations of leaders through the work of the Foundation.

As we celebrate Morrie's life, we reflect on his many accomplishments including the discovery of Lymphotoxin (later known as TNF) in his lab and early in his productive career. TNF is regarded as one of the most critical regulators in immune signaling. It also belongs to a class of key therapeutic targets that modulate the immune and the inflammatory response in myriads of complex maladies from Arthritis, to cancer to autoimmune diseases.

On behalf of ANRF, I wish to express my deepest condolences to anyone who was transformed by Morrie's legacy and call to action. One of my proudest moments was the day when I was entrusted by Morrie to help guide ANRF through its next phase of growth and development as its Executive Director.

Morrie invited us to embrace a life of purpose - like the one he lived. We shall emulate!

## WHO IS OUR NEW EXECUTIVE DIRECTOR? DR. ZAHER NAHLE LEADS ANRF



Dr. Zaher Nahle took the helm of The Arthritis National Research Foundation in early April 2018.

“We were determined to find a uniquely qualified individual who is simultaneously an inspirational leader, a proven executive, and a compassionate soul” read a statement from the search committee “We found all that and more in Dr. Nahle – We cannot be more thrilled to welcome him to our fold to lead the new era of growth and development. ANRF is poised to play an even more dynamic role in the disease space and this development is a tremendous step forward.”

Dr. Nahle is an award-winning scientist with interdisciplinary training in biomedical research and public administration. He has a history of securing prestigious funding in healthcare and patient-oriented research. He holds an MPA from Harvard University, where he also completed a fellowship in Public Policy and Management at the John F. Kennedy School of Government; and a PhD in Physiology and Biophysics from Stony Brook University/Cold Spring Harbor Laboratory (New York).

“I am very humbled to be the one entrusted by this truly committed board of directors, working alongside an all-star Scientific Advisory Board, a dedicated group of colleagues, and our wonderful volunteers,” Dr. Nahle said. “This is an incredible opportunity to serve patients through finding solutions to a complex, multifactorial disease. I live with inflammatory (Gout) Arthritis myself, as does my father. My mother, too, had a knee replacement surgery as a result of her severe Osteoarthritis, so I know the challenges firsthand. For the longest time, I have been impressed by the work of ANRF – The Foundation supports the most promising ideas from the brightest MD and PhD investigators at formative stages in their careers. Going forward, we

remain laser-focused on that mission as we expand our reach and bolster our impact in the disease space. **I truly believe that our contributions at ANRF will markedly accelerate the discovery process and challenge the status quo.**”

Prior to joining ANRF, Dr. Nahle was the Chief Scientific Officer at a Los Angeles-based nonprofit organization. In that capacity, he oversaw a comprehensive investment program with medical centers and industry partners to increase biomedical research in the fields of Women’s Health, Autoimmunity, Bioenergetics and Pain management. He also directed a robust scientific portfolio including peer-review grant programs in basic, translational and epidemiology research, as well as specialized biobanking services and Electronic Health Record and patient registry platforms.

Dr. Nahle is a frequent speaker at international conferences and served on specialized committees and panels at federal agencies, including the NIH and the CDC. Earlier in his career, he was on the faculty of leading universities, such as Weill Cornell Medicine and Vanderbilt University and developed numerous medical education and community engagement initiatives in various disciplines. Dr. Nahle’s research work is featured in prestigious journals like *Science* and *Nature*. During his academic career, he received numerous awards including ones from the American Heart Association (AHA), the US Department of Defense (DoD), the American Cancer Society (pay if), the Robert Wood Johnson Foundation (RWJF) - Genetic Alliance (GA). He was an Edward Mason fellow at Harvard University. While at Harvard, he co-founded a nonprofit organization focused on the disability agenda in Africa.

The late Gale A. Granger, PhD (Page 24), whose groundbreaking work shaped therapeutic modalities in Arthritis and cancer research, put forward that, “Dr. Nahle’s recruitment will help propel the Foundation to new heights and make ANRF the key driver of scientific innovation in Arthritis and associated diseases.” The Hon. Sally Anne Sheridan, who is on the ANRF board and served as the Mayor of the City of Irvine agrees: “We were very impressed with Dr. Nahle’s vision and background and have great confidence in his ability to lead our Foundation at this critical point in its growth.”

*Dr. Nahle and his wife, Irina Yarovaya (pictured), live in Long Beach, California. He is a Harley Davidson enthusiast and together they enjoy attending theater and ballet productions.*

## ALL SMILES AT THE ANRF SOCIAL

The Foundation's annual event at the American College of Rheumatology (ACR) conference, ANRF Social, was held in Chicago on Monday October 22nd.

Guests in attendance had the opportunity to meet key opinion leaders in Arthritis and Rheumatology research in a relaxed, friendly atmosphere. A unique feature of this year's event was a one-on-one chat with the ANRF leadership, Executive Director, Dr. Zaher Nahle, Board President, Debra Sampson and Vice-President, Sally Anne Sheridan.

This complimentary "meet and greet" reception celebrated the cutting-edge science of our grant recipients and the continued support of our donors and patient community.

We were pleased to host many of our scientists and supporters as you can see from the photos of the many who were in attendance.



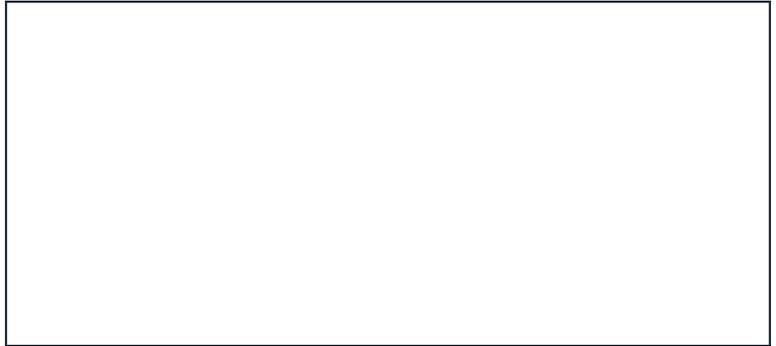


**Thank you to all who were in attendance:**

I. Adamopoulos | J. Ashouri-Sinha | D. Austin | N. Bottini | T. Caskey Brown | I. Chepelev | D. Constein | F. Fitzgerald  
 D. Guglielmo | S. Jackson | M. Jeffries | A. Jonsson | M. Kahlenberg | G. Kallioliias | E. Koliopoulos | I. Koliopoulos  
 M. Leach | G. Li | H. Makinda | R. Miller | A. Montgomery | Z. Nahle | T. Niewold | P. Nigrovic | E. Noss | D. Orange  
 A. Paine | D. Sampson | P. Sapao | A. Sawala | D. Schwartz | M. Shelef | S. Sheridan | S. Stanford | M. Teves  
 K. Van Raemdonck | T. Vogel | T. Wampler Muskardin | M. Waterfield | L. Wellscendt | T. Westrich-Roberston  
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