2021 ANNUAL REPORT

ARTHRITIS NATIONAL RESEARCH FOUNDATION

ANRF

2020-21 Grant funded cohort progresses arthritis and related autoimmune disease research

ANRF welcomes new Chief Executive Officer

Researchers focus on COVID implications

★★★★
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ANRF MISSION

To provide initial research funding to brilliant, investigative scientists with new ideas to cure arthritis and related autoimmune diseases

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MESSAGE FROM THE CEO

ANRF Community,

It is with great pride and excitement to have been selected as ANRF's Chief Executive Officer, an organization with such a history of impact and purpose—and a clearly defined and important mission. I am already so impressed with the caliber of research, scientists, donors and partners who understand the value of funding early-career investigators, and are committed to finding better treatments and therapies, and eventually a cure, for arthritis and related autoimmune diseases.

As I write this, the Board of Directors and ANRF staff just concluded a strategic planning retreat where we brainstormed and envisioned the next five years—focused on increasing support to fund more research, keeping our scholars connected to collaborate and build their networks and thinking outside the box—even to consider venture projects.

Emily Boyd Stormoen, ANRF CEO

In addition to a productive planning session, it was great to meet in person. COVID-19 restraints had limited in person meetings, as it has taken its toll in many ways. For ANRF, the pandemic's impact had a ripple effect, like it did for many similar organizations:

- Current grant funded researchers determined alternate methods of research as labs and access to testing and tools were restricted. Research and results continued, with some grant timelines extended to align with access and accommodate schedules that were beyond the control of the scientists.

- Alumni scholars, and currently funded scientists, studied the impact—and possible impacts—of COVID and served as advisors or experts to their institutions and communities. Many published journal articles based on their research, and are continuing their work on the impact of COVID's long-term health implications.

- Fiscally, unlike the previous year, our investments performed well. Combined with contributions and bequests, we exceeded the $10M threshold for the first time, which is encouraging and makes it realistic to expect that we can provide 20 grants in the coming year—which is an ongoing goal of the organization.

The resilience of donors, researchers, partners and volunteer leadership is incredible to see and the commitment to ANRF hasn't wavered. In fact, it only has become stronger. I'm honored to now be a part of this community and look forward to working with the Board of Directors and the entire community to reach new levels of success and impact.

Sincerely,

Emily Boyd Stormoen

Emily Boyd Stormoen
CEO

FINANCIAL REPORT

2019,2020 & 2021
AUDITED STATEMENTS OF PUBLIC SUPPORT
FISCAL YEARS ENDING MARCH 31st

OPERATING STATEMENT

	2019	2020	2021
*Total Unrestricted Assets**	$8,530,041	$7,504,037	$9,828,5631
*Total Restricted Assets**	436,390	336,390	$326,104
Total Assets	$8,966,431	$7,840,427	$10,154,667
Total Liabilities	$37,323	$32,830	$78,521
NET ASSETS AT END OF YEAR	**$8,966,431**	**$7,840,427**	**$10,154,661**

REVENUE AND EXPENSES			
PUBLIC SUPPORT AND REVENUE	2019	2020	2021
Contributions and Bequests	1,391,531	1,799,375	1,446,537[A]
Investment Income Net	224,639	741,837	123,323
Unrealized Gain (loss) on Investments	142,447	(1,268,816)	2,924,126
TOTAL SUPPORT AND REVENUE	**$1,758,617**	**$1,272,396**	**$4,493,986**
EXPENSES			
Program Services			
Research	1,828,753	1,802,401	1,567,125
Education	250,165	308,666	254,691
Total Program Services	$2,078,918	$2,111,067	$1,821,816
SUPPORTING SERVICES			
Management and General	144,304	194,436	273,643
Fund Development	111,029	92,897	84,287
Total Supporting Services	255,333	287,333	357,930
TOTAL EXPENSES	**$2,334,251**	**$2,398,400**	**$2,179,746**
Change in Net Assets	(575,634)	(1,126,004)	2,314,240
Net Assets at Beginning of Year	9,542,065	8,966,431	7,840,427
NET ASSETS AT END OF YEAR	**$8,966,431**	**$7,840,427**	**$10,154,667**

*Beginning in 2019, new accounting regulations require reporting restricted and unrestricted assets as seen above.

[A] Major planned gifts (bequests over $100K) in the 2020-2021 fiscal year totaled $550,178.

2020-21 NEWS TO NOTE

THE ANRF BOARD OF DIRECTORS COMPLETED a thorough national search for a new Executive Director/Chief Executive Officer in early 2021 with the hire of Emily Boyd Stormoen. With 20+ years of progressive experience in nonprofit management, Stormoen brings deep understanding of organizational management, the nonprofit sector and a growth oriented approach, along with a success record of increasing revenue, fundraising and sponsorship funding.

TWELVE EARLY-CAREER SCIENTISTS WERE selected to receive $100,000 each in grant funding after demonstrating meritorious research projects with potential to lead to new treatments, improved therapies and a path to a cure for arthritis and related autoimmune diseases.

COVID-19 AND THE PANDEMIC CONTINUED ITS impact on nearly every part of life in the U.S. and around the globe. Most significantly was the loss of life. For some current and former ANRF physicians and scientists, researching its impact and potential long-term implications of COVID became an important part of their work and contribution to helping the world understanding COVID's impact.

- **P.J. Utz, Ph.D.,** a professor and Associate Dean for Medical Student Research at Stanford University, whose lab was already active in researching vaccine biology, both inducing protective immunity to pathogens and turning off immune responses in autoimmune diseases, began studying SARS-CoV-2 and testing the hypothesis the virus causes autoimmunity. Their work, still in progress, has been published in several journals and mediums, and Dr. Utz was selected to work on a National Institute of Health inititiative to track the recovery process following infection with SARS-CoV-2 and why some individuals have prolonged symptoms.
- **SAB Member Betty Diamond, M.D.,** from the Feinstein Institutes for Health, was selected as part of the leadership and Executive Committee of the same initiative for NIH.
- **Lauren Henderson, M.D., Ph.D.**, was a lead researcher/author on a published article in *Arthritis & Rheumatology*, focused on the correlation of outcomes in COVID-19 and the correlation with clinical and laboratory features of cytokine storm syndrome in Juvenile Idiopathic Arthritis patients. Other authors included SAB member **Peter Nigrovic, M.D.** and ANRF funded scholar **Pui Y. Lee, M.D., Ph.D.** All three are practicing physicians and researchers at Boston Children's Hospital/ Harvard Medical School.
- **Michelle Kahlenberg, M.D., Ph.D.,** a SAB member, along with fellow researchers at the University of Michigan, tracked COVID-19 infected patients, including those who also had Lupus (SLE), with results suggesting those with SLE may develop more severe COVID-19 infections, even compared to those with other autoimmune deficiencies. Their research appeared in Annals of the Rheumatic Diseases.
- **Jason Knight, M.D., Ph.D.**, and **Yu (Ray) Zuo, M.D.**, both of University of Michigan and working in the Knight Lab, produced a study, "Prothrombic autoantibodies in serum from patients hospitalized with COVID-19" which has appeared in more than 100 media outlets.
- **Michael A. Paley, M.D., Ph.D.,** Washington University, was a first author in a study focusing on COVID-19 Vaccines producing immune responses in patients with chronic inflammatory diseases.

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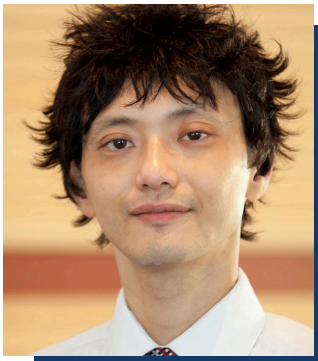
2020-21 GRANT RECIPIENTS

SARAH BAXTER, M.D., PH.D.
University of Washington
Research: Lupus
"Characterize the role of AIM2 in the autoimmune disease Lupus."

During her pediatric residency at Seattle Children's Hospital and her research and study at the University of Washington, Sarah Baxter, M.D., Ph.D., saw firsthand how a genetic diagnosis could drastically alter care and recommendations for treatment. A practicing rheumatologist, Dr. Baxter believes genomics can elucidate the diverse pathways leading to immune dysregulation and autoimmunity in those patients who have rheumatologic diseases. She hypothesizes children who develop lupus (cSLE) have genetic immune diseases. By identifying these genetic deficiencies, she is identifying biologic pathways crucial for effectively controlling lupus activity and preventing further damage.

In preliminary genetic analyses of children with lupus, where one parent also has lupus, Dr. Baxter identified a gene that might be causing their condition. This gene has not previously been documented to demonstrate a cause for the disease. Preliminary analyses showed the mutations present in a child with lupus cause the protein to act differently. Cells with mutant protein grow differently, and mutant protein monomers cannot form a bond with each other. As an ANRF scholar, Dr. Baxter's work is focused on mapping the genetic landscape of lupus and clinically translating findings to identify novel diagnostic and treatment options for patients.



CHARLES CHAN, PH.D.
Stanford University
Research: Osteoarthritis
"Investigating the role of resident stem cell populations in the regeneration of cartilage and in OA progression."

Evidenced by acceptance of his regenerative stem cell research in top-tier academic journals and with findings, results and interest in his work in publications including *Nature Medicine*, *Nature Biomedical*, *The New York Times* and *The Wall Street Journal*, Charles Chan's, Ph.D. second year as an ANRF funded scientist continued to progress.

An assistant professor at Stanford's Department of Surgery, Division of Plastic and Reconstructive Surgery, Dr. Chan works with a group of researchers who are determining ways to regenerate stem cells into cartilage that can be used to repair arthritic joints, finding this can even be done at the point when a patient is beginning to develop arthritis. This treatment could be a solution for those needing a long-term solution for their arthritis progression as there has been extensive research on mice demonstrating regenerated cartilage lasted for one fourth of their life span.

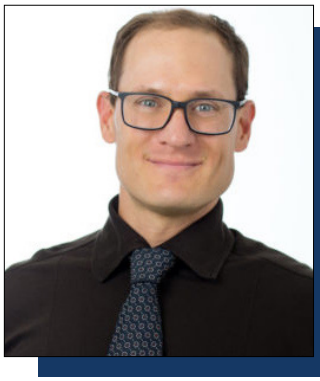
As told to the *Times*: "It is really a major advance in field of osteoarthritis," said Dr. Gerard Karsenty, a bone specialist at Columbia University who was not involved in the research. "When you demonstrate something in the mouse, I don't know of any example where it has not applied to humans."

CHRISTIAN JACOME-GALARZA, PH.D.
Brigham and Women's Hospital, Harvard Medical School
Research: Osteoclasts
"Investigate how such OCs form and how they lead to joint tissue destruction in arthritis."

An Instructor in Medicine and Orthopaedics at Brigham and Women's Hospital/Harvard Medical School, Christian Jacome-Galarza, Ph.D., research and studies led to the establishment of standard methods to isolate and study osteoclasts and their precursors, as well as a potential cell-based therapy to treat bone diseases.

In his ANRF grant funded work, Dr. Jacome-Galarza focused on the origins of synovial osteoclasts and macrophages in inflammatory arthritis, finding there are two distinct lineages of macrophages in synovial tissues a smaller proportion of synovial macrophages derived from bone marrow hematopoietic stem cell progenitors, while the majority derived from embryonic erythromyeloid progenitors.

His work is continuing beyond his grant funding as there continues to be more discoveries and research on osteoclasts, although he exceeded the goals intended for this timeline. Even in constrains of the COVID pandemic, he presented on more than ten occasions at local, national and global meetings and conferences throughout the time he was funded.



MICHAEL JURYNEC, PH.D.
University of Utah
Research: Osteoarthritis
"Hypothesizes that mutations in specific genes as found in OA susceptible families may underlie the enhanced joint inflammation that is a hallmark of OA progression."

Michael Jurynec, Ph.D., Assistant Professor at the University of Utah, has knowledge of the impact of osteoarthritis as it runs in his family, and he, too, is affected. His personal experience and interest in molecular biology and genetics led to a desire to better understand the disease, use his training and access the unique population genetic resources at the university to study OA affected families.

His ANRF grant funded study is focused on how mutations in genes underlie joint inflammation of OA progression.Since the study was initiated, families have been identified with gene variants that modify the NOD-RIPK2 pathwjointay. Functional analyses on variants are performed and tissue culture assays to perform high throughput screens to identify chemical modifiers of the NOD-RIPK2 pathway designed. Cumulative results allow Dr. Jurynec to determine which components of the pathway increase risk for developing OA and if they could be novel targets when developing treatments

A manuscript highlighting select findings was published in *Arthritis Rheumatol* and two additional manuscripts have been submitted. The Jurynec lab continues to focus on identifying pathways and mechanisms underlying OA susceptibility with the ultimate goal of developing therapeutics that prevent the onset or limit the progression of the disease.

LAUREN HENDERSON, M.D., PH.D
Boston Children's Hospital & Harvard Medical School
Research: Juvenile Idiopathic Arthritis
"Identifying why some children outgrow JIA whereas others develop chronic disease."

A pediatric rheumatologist at Boston Children's Hospital and an Assistant Professor at Harvard Medical School, Lauren Henderson, M.D., Ph.D., cares for children with complex autoimmune conditions and conducts research of the loss of immunologic tolerance in pediatric rheumatologic disorders such as juvenile idiopathic arthritis (JIA).

Dr. Henderson's ANRF grant funded research is focused on investigating the ability of regulatory T cells to control inflammation in oligoarticular JIA. Through previous research, Dr. Henderson identified regulatory T cells in the synovial fluid which express inflammatory cytokines.

In addition, she is collaborating with Dr. Stephen Elledge who developed the breakthrough technology, TScan, that allows for rapid and unbiased discovery of the physiologic targets of any T cell. Using this technology, they are aiming to evaluate autoreactivity of regulatory T cells in oligoarticular JIA with the goal to better understand the mechanisms of oligoarticular JIA and identify novel treatments.

SUSAN MACLAUCHLAN, PH.D.
Brigham and Women's Hospital, Harvard Medical School
Research: Rheumatoid Arthritis
"Evaluating the impact of mutations in the gene TET2 in RA since her previous work demonstrated that such mutations result in accelerated CVD development."

Incidences of cardiovascular heart disease (CVD) are doubled in Rheumatoid Arthritis (RA) patients when compared to the general public, which is also true for those with diabetes. While a great deal of research continues to understand the frequency in diabetic patients, far less is being done to understand the link between CVD and RA. Susan MacLauchlan, Ph.D., Instructor of Medicine at Brigham and Women's Hospital, is working to change this in her research of why the cardiovascular system is so vulnerable.

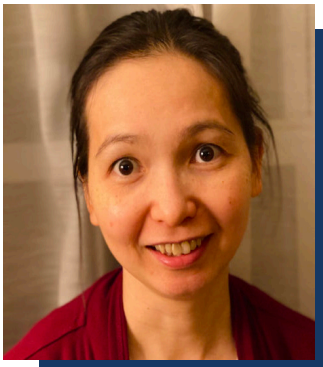
Great strides have been made to explain why heart disease happens , but somewhere between 20 to 50% of heart disease patients have none or one of the pre-existing conditions to predict for CVD. For those with RA, traditional risk factors do not explain the nearly double risk.

Recent studies determined acquiring a single mutation in the precursor cells, which populate a person's immune system, can be detected from blood samples if the mutation gives the cell a competitive advantage. This process identified as clonal hematopoiesis, often occurs in the aging population, which combined, they are more likely to develop cardiovascular disease. In her grant funded work, Dr. Maclauchlan seeks to answer if the process of clonal hematopoiesis occurring in RA patients can explain the increased frequency of CVD within this population.

RENUKA NAYAK, M.D., PH.D.
University of California, San Francisco
Research: Rheumatoid Arthritis
"Characterize the microbiomes and Methotrexate (MTX) metabolites of a number of RA patients to more fully understand how specific bacterial genes may influence the activity of MTX in RA patients."

The significance of the role gut biome (microorganisms including bacteria and fungi that live in the digestive tracts of humans) in overall health is becoming more obvious. As an ANRF scholar Renuka Nayak, M.D., Ph.D. is researching the increasingly more important role and addressing questions that include: 1) Do the microbiota residing in our gastrointestinal tract metabolize pharmacologic drugs and does this contribute to variation in clinical drug response? 2) Do the drugs we use to treat disease influence the human gut microbiota which then in turn has downstream consequences on the patient such as altered efficacy and toxicity of the drug?

As a rheumatologist, Dr. Nayak is frustrated with the trial-and-error approach, often necessary to be used to find a drug which the patient responds to. Currently, rheumatologists have no way of predicting individual patient response. As it takes between two to three months for most drugs to exert their full effect, patients who are ultimately deemed to be non-responders, suffer from continued pain and could potentially sustain permanent damage. Dr. Nayak is hopeful the data she generates will provide a foundation to create a predictive tool to help identify patients as non-responders, thus allowing them to start alternative therapies sooner.



TAM QUACH, PH.D.
The Feinstein Institute for Medical Research
Research: Autoimmune
"Investigating how TNF affects the generation of autoreactive B cells."

Treatments for inflammatory arthritis include TNF inhibitors or biologic anti-inflammatory medications. These lower immune responses so that they no longer occur at excessive levels. Unfortunately, there seems to be a link between the use of these medications and the development of a secondary autoimmune condition such as lupus.

Tam Quach, Ph.D., postulates that this happens because the medications alter the internal stable environment of certain immune cells, specifically B cells, which are responsible for fighting viruses and bacteria through the production of antibodies. Central tolerance, also known as negative selection, is the process of eliminating any B cells that are reactive to self so that an immune response is not launched against one's own cells. Destabilization of the internal environment of B cells could be responsible for autoreactive B cells escaping negative selection.

Dr. Quach is researching if TNF deficiency resulting from treatment with TNF inhibitors induces signal alterations in autoreactive B cells. This is intended to determine why there are occurrences in some patients and which are at risk for developing negative side.

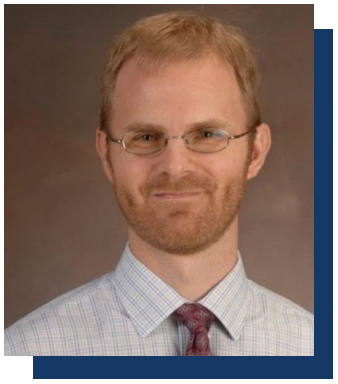
2020-21 GRANT RECIPIENTS

JOLIEN SUURMOND, PH.D.
The Feinstein Institute for Medical Research
Research: Lupus
"The genetics of antinuclear antibodies and the risk of lupus."

As a Ph.D. candidate at Leiden University in the Netherlands, Jolien Suurmond studied Rheumatoid Arthritis (RA) and was selected for postdoctoral position working with Dr. Betty Diamond at The Feinstein Institute for Medical Research, a leader in the rheumatology field and a member of the ANRF Scientific Advisory Board.

Working with Dr. Diamond, she shifted her focus to study to Systemic Lupus Erythematosus (SLE),and has already made discoveries including: the loss of IgG plasma cell tolerance checkpoint is the primary B cell abnormality in SLE. B cells can be ANA+ and distinct phenotypes of these ANA+ B cells and plasma cells, with different types arising from different pathways and resulting in different characteristics.

Categorizing SLE patients based on these different cells can target disease-specific pathways using precision medicine, decreasing the degree of immunosuppression in patient groups. If successful, treatments could target the elements of the system malfunctioning instead of targeting the system as whole. This could reduce side effects associated with blanket treatment approaches that include increased risk of infection and cancer. Dr. Suurmond is hopeful defining these differences in B cells in SLE will offer alternative therapeutic targets specific to distinct differentiation/activation pathways.

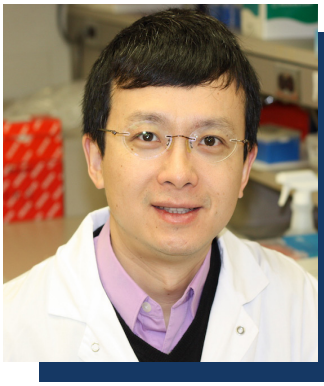


BRIAN SKAUG, M.D., PH.D.
The University of Texas Health Science Center at Houston
Research: Scleroderma
"Pathogenic effects of DNASSE1L3R206C polymorphism in systemic sclerosis."

In his second year of ANRF grant funding, Brian Skaug, M.D., Ph.D. continued to focus his research on the impact of genetic risk factors of an identified variant (DNASE1L3R206C) and how it contributes to systemic sclerosis (SSc) and scleroderma.

A Rheumatologist, Dr. Skaug provides care and consultation at the University of Texas Health Center at Houston, the Texas Medical Center and at Lyndon Baines Johnson Hospital. To date, research findings solidified his hypothesis that the variant is a major determinant for digestion of DNA and the variant is a risk factor for SSc. Previous research identified dysfunction of a gene, DNASE1L3, as a risk factor for developing scleroderma. Dysfunction of this gene appears to be a risk factor for multiple autoimmune diseases including systemic sclerosis, lupus, vasculitis and rheumatoid arthritis, suggesting that DNASE1L3 is important for maintaining proper functioning of the immune system, which Dr. Skaug's work continued to support.

With his two-year funding, he established new understandings of the mechanism underlying the excess immune cell activity observed in systemic sclerosis, scleroderma and other autoimmune diseases, developed an imaging tool for future research, and developed data that can serve as a resource for future research. His submission to present and discuss his research at the 7th Systemic Sclerosis World Congress was accepted, providing the opportunity to share his work globally.

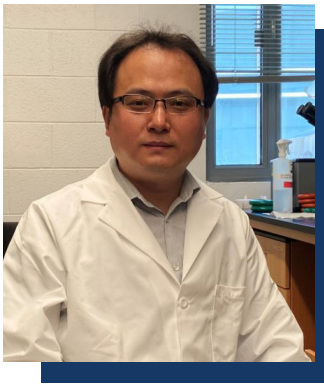


HU ZENG, PH.D.
The Mayo Clinic
Research: Rheumatoid Arthritis
"Analyze ICI patient samples to determine the mechanisms that lead to the development of this form of arthritis following ICI therapy."

Hu Zeng, Ph.D. started his career as a biochemist before studying genetics as part of his graduate program, doing research in labs in China and Japan and at St. Jude Children's Research Hospital in Memphis. In 2017 he joined the Division of Rheumatology at Mayo Clinic to research the immunological basis of rheumatic diseases in his own laboratory, where he continues to work.

A histology of autoimmunity, ranging from mild to severe, in his own family drew Dr. Zeng to this research. Growing up in an impoverished city, he saw the overburdened medical facilities struggle to diagnose and treat autoimmune conditions, in desperation residents turned to a variety of "magic treatments" offered by unqualified individuals. Dr Zeng is trying to improve the way in which these conditions are diagnosed and treated and has made huge inroads into achieving these objectives.

His research focuses on lymphocytes, mainly composed of T cells and B cells, and the major blood cells that find and destroy invading pathogens. Dr. Zeng's lab is investigating how lymphocyte function is controlled by various metabolic processes in the context of normal development and autoimmune diseases.



CHENG-HAI ZHANG, PH.D.
Harvard Medical School
Research: Osteoarthritis
"Investigating the role of gene Creb5 in lubricin expression during the development of osteoarthritis."

Chenghai Zhang completed his Ph.D. in Biology at Nanjing University, China. During this time, he made significant contributions regarding the regulation of airway smooth muscle pathophysiology. Now an instructor in Biological Chemistry and Molecular Pharmacology at Harvard Medical School, he has identified a master transcription factor, Creb5, that regulates the formation of the synovial joints, specifically the articular chondrogenic lineage, and initiates/maintains expression in articular chondrocytes.

As an ANRF scholar, Dr. Zhang is studying the mechanism of how Creb5 regulates synovial joints formation during the embryonic stage, the role of Creb5 in regulating the formation, growth and degradation of articular cartilage during aging and in osteoarthritis conditions, and finally to test whether exogenous expression of Creb5 can protect articular cartilage degradation during the progression of aging/osteoarthritis or restore articular cartilage in surgery induced osteoarthritis.

The long-term goal of his research is to understand the developmental mechanism of synovial joint/cartilage formation and to provide the basis to develop novel therapies to slow down or prevent the progression of human osteoarthritis.

ARTHRITIS NATIONAL RESEARCH FOUNDATION
19200 Von Karman Avenue Suite 350
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HOW DONATIONS FUND LIFE-CHANGING RESEARCH

Supports innovative ideas and treatments to improve the quality of life for those living with arthritis and related autoimmune diseases.

Research scientists perform groundbreaking work to understand causes and find new targets for treatments.

Allows early-career scientists to establish and build sustainable careers while focusing on their research funded by ANRF.

Continues to support all ANRF funded scientists, current and previous, throughout their careers by providing resources and a network of peers to collaborate and share their research.

58.5 million

adults live with arthritis in the U.S.

that is ONE in FOUR adults.