

## Rheumatology

**2017 YEAR IN REVIEW** 



# 

IN ijEêĒűdČǿêŰŰ ǿnĉŴÂŰĉČæenĉħ 50%

INCREASE IN NIH FUNDING 105 FACULTY MEMBERS

### Contents

- 1 MESSAGE FROM THE DIRECTOR
- 2 FACTS & FIGURES
- 4 2017 IN BRIEF

#### 10 2017 IN DEPTH

- **11** Building New Bridges to Overcome Autoimmunity
- **13** New Test May Point the Way Toward a Cure for Rheumatoid Arthritis
- 14 Interactions Between Platelets and Endothelial Cells May Contribute to SLE-Associated Cardiovascular Disease
- **16** Documenting and Addressing Patient Disparities in Lupus
- **18** Training Grant Boosts New Generation of Physician-Scientists

#### 20 ACADEMIC ACTIVITIES

#### 23 LEADERSHIP

 On the cover: cancer cells infiltrated by clonal, self-reactive T cells. Although these cells are not poised to suppress cancer, Adam Mor, MD, PhD, the Saul J.
Farber Assistant Professor of Medicine, and assistant professor of pathology, is studying these T cells in rheumatic diseases where their suppressive activities may prove highly advantageous.



#### MESSAGE FROM THE DIRECTOR

# Dear Colleagues and Friends:

The year 2017 was an unprecedented one of progress for NYU Langone Health's Division of Rheumatology. The growth and development of our research centers highlighted the benefits of forward-looking innovation. Groundbreaking research on disease risks, mechanisms, and interventions underscored the importance of clinical and basic science collaborations.

This past year, we welcomed Timothy B. Niewold, MD, as the new director of the Judith and Stewart Colton Center for Autoimmunity. We are encouraged by the center's maturation as both an innovative incubator for a range of promising pilot projects and a vital hub for networking and interdisciplinary partnerships. Our Psoriatic Arthritis Center continues its quest to cover new ground, and has now joined forces with the Department of Dermatology to launch an ambitious series of studies called Preventing Psoriatic Arthritis, or PreP. The project will follow psoriasis patients longitudinally to investigate the clinical and demographic factors that influence the development of psoriatic arthritis.

We saw many partnerships come to fruition and advance our understanding of disease risks, drivers, and potential targets. A collaboration with our cardiology colleagues, for instance, has revealed new evidence for an abnormal interaction between platelets and endothelial cells in lupus patients that could help explain a higher risk of accelerated cardiovascular disease. Through a separate collaboration, we uncovered evidence to suggest that an immune response to a gut-based commensal bacterium may drive or exacerbate lupus nephritis.

An international effort has helped us to link the perplexing pathogenesis of neonatal lupus and congenital heart block to a genetic variant that impairs the surveillance activity of natural killer cells and results in cardiac inflammation and scarring. Additionally, a new test that detects memory B cell-produced autoantibodies may help shift the clinical focus on rheumatoid arthritis toward the disease's true drivers and aid in the testing of new therapeutic agents that eliminate B cells or other autoimmunitylinked targets.

Other collaborations, including the multiyear Manhattan Lupus Surveillance Program, helped us to document and characterize significant disparities among racial and ethnic minorities. The population-based study found much higher lupus prevalence and aggressiveness among Hispanic and Asian residents than among their white counterparts, while confirming even higher rates among black individuals. Separate projects are illuminating the genetic foundations behind an increased prevalence of atherosclerotic cardiovascular disease among African American patients with SLE and a



JILL P. BUYON, MD

Jul P. Bugn

Lady Va and Sir Deryck Maughan Professor of Rheumatology

Director, Division of Rheumatology, Department of Medicine

lower-than-expected risk of renal flares and new-onset nephritis among pregnant women with lupus.

Finally, we have benefited from major new grants, including the Ruth L. Kirschstein National Research Service Award funded by the NIH, which will provide support not only for promising third-year rheumatology fellows to pursue academic careers but also for predoctoral MD/PhD candidates interested in the study of rheumatic diseases. A separate five-year, \$6.7 million award from the NIH will help us to determine why lupus develops in some individuals but not others, and what triggers a flare in those with established disease. Our new Translational Center of Molecular Profiling in Preclinical and Established Lupus, COMPEL, is launching three research arms, all aimed at resolving the underlying mechanisms of SLE's initiation and perpetuation.

We know that the path toward better treatments and cures remains steep for many autoimmune and rheumatic diseases. With this year's momentum pushing us onward, we embrace the opportunity to surmount additional challenges on our climb toward the ultimate goal of delivering personalized and effective patient care.

### **Division of Rheumatology**

#### CLINICAL CARE

### 3,500+

OUTPATIENT OFFICE VISITS monthly

### 14

FACULTY GROUP PRACTICE LOCATIONS in the NYC area

#### **RESEARCH AND FUNDING**

50%

INCREASE IN NIH FUNDING

### \$6.7 Million

**NIH GRANT** to back new research in the initiation and progression of lupus

### 48

ACCEPTED ACR ABSTRACTS including 10 oral presentations and 2 plenary sessions

7 New

#### FACULTY AND FELLOWS

6 NEW FULL-TIME FACULTY

105

FACULTY MEMBERS

### 165

**FELLOWSHIP APPLICANTS** 

T32

#### NIH TRAINING GRANT

educating the leaders of tomorrow in academic rheumatology

#### ACHIEVEMENTS & ACCOLADES

### **Top 10**

IN THE COUNTRY FOR RHEUMATOLOGY

in U.S. News & World Report's "Best Hospitals"



### One of the first

#### DIVISIONS OF RHEUMATOLOGY TO BE ESTABLISHED IN THE COUNTRY

Growing out of the Rheumatic Diseases Study Group (RDSG), founded in 1932, the division has contributed to six past presidents to the ACR and current president-elect, Paula Marchetta, MD.

Numbers represent FY17 (Sept 2016-Aug 2017) unless otherwise noted

### NYU Langone Health

View of NYU Langone Health's main Manhattan campus, including renderings of the new Science Building (left) and the Helen L. and Martin S. Kimmel Pavilion (right), both set to open in 2018. (Image credit: Ennead Architects)



#### BEST HOSPITALS USNews HONOR ROLL 2017-18



#### IN THE NATION

and nationally ranked in 12 specialties: Rehabilitation, Orthopedics, Rheumatology, Neurology & Neurosurgery, Geriatrics, Urology, Cardiology & Heart Surgery, Gastroenterology & GI Surgery, Diabetes & Endocrinology, Pulmonology, Cancer, and Nephrology



### #12

#### IN THE NATION BEST MEDICAL SCHOOLS FOR RESEARCH

and a leader in innovation in medical education, including accelerated pathways to the MD degree



### Leader

#### IN QUALITY CARE AND PATIENT SAFETY

For the past four years, NYU Langone has received top rankings for overall patient safety and quality of care from Vizient, Inc., formerly the University HealthSystem Consortium. In 2017, NYU Langone received two significant awards from Vizient the Bernard A. Birnbaum, MD, Quality Leadership Award and the Ambulatory Care Quality and Accountability Award for demonstrated excellence in delivering high-quality, patient-centered outpatient care.

### **5 Star Rating**

#### FROM CMS HOSPITAL COMPARE

NYU Langone Health is the only full-service hospital in New York State and one of 9 percent of hospitals nationwide to receive a five-star rating from the Centers for Medicare and Medicaid Services (CMS). The rating reflects overall safety, quality, and patient experience.

#### NIH Grant Drives New Research in Lupus Risk Factors and Flares

Backed by a major NIH grant, researchers are in pursuit of new insights to determine the mechanisms by which systemic lupus erythematosus (SLE) is initiated and perpetuated. The project will explore why some women who have anti-SSA/Ro antibodies never develop lupus while others do.

The program leverages unique patient resources, one a cohort of asymptomatic women presenting with a breakdown in B cell tolerance identified because of neonatal lupus (NL) in an offspring, and the other a robustly phenotyped cohort of established SLE patients spanning diverse racial backgrounds and with a high penetrance of serious illness. The five-year, \$6.7 million project combines the expertise of five investigators at NYU Langone Health, led by Jill P. Buyon, MD, the Lady Va and Sir Deryck Maughan Professor of Rheumatology, director of the Division of Rheumatology, and director of the Lupus Center. Through the newly launched Translational Center of Molecular Profiling in Preclinical and Established Lupus (COMPEL), the team hopes to discover the underpinnings of SLE's initiation and progression.

The program includes three main research arms. One, led by Robert M. Clancy, PhD, associate professor of medicine, will investigate associations between immunogenetics and the microbiome to determine why lupus emerges or remains dormant in at-risk women. Another arm, led by Gregg J. Silverman, MD, professor of medicine and pathology, will sequence gut microbiomes in established lupus patients and analyze the gut-associated B cell response to assess the root of disease flares and,



Boris Reizis, PhD; Jill P. Buyon, MD; Gregg J. Silverman, MD; Peter M. Izmirly, MD; and Robert M. Clancy, PhD

in particular, the involvement and progression of kidney injury. A third arm, led by Boris Reizis, PhD, professor of medicine and pathology, will investigate the fundamental enzyme DNASE1L3, which is essential for protection against SLE, and will explore how dysfunction of this enzyme may provide clues to the origins of disease and contribute to fluctuations in established disease.

The clinical core is directed by Peter M. Izmirly, MD, assistant professor of medicine. One of the clinical groups comprises women who have had a child born with congenital heart block or a skin rash and who have anti-SSA/Ro antibodies but were asymptomatic at the time their child was diagnosed. Dr. Buyon says researchers still know relatively little about the factors that contribute to the mothers' health outcomes. The new center's work, aided by the NYU Langone Research Registry for Neonatal Lupus—the world's largest data bank of its kind—will shed light on this question.

Dr. Silverman will lead the research technology core bringing advanced technology for molecular sequencing of autoantibodies and sorting of intestinal IgA-coated bacteria profiled by 16S rRNA microbiome surveys.

COMPEL utilizes the patient registries, interest in the microbiome, work on environmental factors, mouse models, and human clinical research. "When you integrate these multiple efforts, NYU Langone is uniquely well positioned to address the question of why people develop lupus," Dr. Buyon says. "We're approaching it in an entirely new way."

#### Collaborative Efforts at the Colton Center for Autoimmunity

The Judith and Stewart Colton Center for Autoimmunity at NYU Langone Health is forging ahead with a new director, new faculty, and a range of internally funded pilot projects. Director Timothy B. Niewold, MD, who arrived in 2017 from the Mayo Clinic, says the pilot projects and the center's growth as a networking hub are fostering cross-disciplinary collaborations, supporting diverse ideas, and increasing the chances of a breakthrough.

One funded project features a new method for creating artificial chromosomes and may allow researchers to better understand which portions of implicated genes drive autoimmune dysfunction. Another project is examining the degradation of extracellular DNA by DNase; knocking out the enzyme in a mouse model results in lupus-like disease, pointing to its potential role in prevention. A third effort, highlighted in a 2017 ACR plenary, has found evidence that the immune response to a single commensal bacterial strain in the gut may drive lupus nephritis.



"The Colton Center will become the link that helps connect the dots between autoimmune disease and mechanisms."

-Timothy B. Niewold, MD

#### Genetic Variant That Inhibits NK Cell Surveillance Linked to Congenital Heart Block

Deciphering the pathogenesis of neonatal lupus and permanent cardiac injury caused by maternal autoantibodies has posed a major challenge to rheumatologists, cardiologists, and obstetricians. Fetal exposure to maternal anti-SSA/Ro antibodies is necessary but not sufficient for the development of congenital heart block, or CHB, which has led researchers to seek out other contributing factors.

A new study published in *Arthritis & Rheumatology* suggests that CHB onset may be linked to the genetic variant of a major histocompatibility complex class I protein that impairs surveillance by natural killer (NK) cells, resulting in unchecked cardiac inflammation and scarring.

Based on a genetic comparison of affected and unaffected siblings, an international collaboration led by Robert M. Clancy, PhD, associate professor of medicine, discovered that the disease associates with alleles of the HLA-C gene. Children with CHB, but not their unaffected sibling with anti-SSA/Ro-exposed antibodies carry an Asn80Lys allele at the alpha1 helix of HLA-C molecules.

The study findings support a model in which this genetic variant, known as the C2 allele, binds with high affinity to an inhibitory receptor, called a killer cell immunoglobulinlike receptor (KIR), on the surface of NK cells. This excessive binding consequently impairs the ability of KIR to facilitate the killing function of NK cells, leaving this immune surveillance system unable to suppress inflammatory cells and thereby limit cardiac inflammation and injury.

Dr. Clancy hopes to extend the finding by studying potential links between dysfunctional NK cells and the pathogenesis of lupus nephritis, where autoantibodies may similarly link tissue injury to a defective disarming of inflammatory responses. He also plans to test whether such defects can be reset, enabling the "sentinels" to resume their normal role.

## Test May Lead to a Cure for Rheumatoid Arthritis

A new study shows that existing anti-inflammation treatments for rheumatoid arthritis do little to address the underlying immunological defect. NYU Langone Health investigators and their collaborators suggest that the clinical focus should shift to memory B cells, implicated as pathogenic drivers of RA, and the cells' secreted anti-citrullinated protein antibodies, or ACPAs, which can be useful biomarkers for diagnosis and prognosis.

Dr. Silverman's lab-developed test, which detects memory B cell-produced autoantibodies, indicated that clinical remission of RA after treatment with standard drugs had no effect on the circulating burden of ACPA-expressing B cells. The result suggests that the regimen didn't alter the underlying dysregulation of humoral immunity and may explain why stopping standard therapy often results in disease reactivation and a clinical flare.

For clinical trials, the researchers propose using their assay to help identify new therapeutic agents that may lead to a cure by eliminating B cells or other targets linked to autoimmunity.



## 35%

#### OF PSORIATIC ARTHRITIS CENTER PATIENTS HAVE

a first-degree relative with psoriasis but not arthritis. This familial access enables our pursuit of why some patients progress and others don't.

#### New Steps Toward Predicting, Preventing, and Treating Psoriatic Arthritis

Backed by an extensive and expanding patient database, biobank, and a growing roster of experts, several recent ambitious projects at NYU Langone's Psoriatic Arthritis Center are helping researchers reveal pathogenic mechanisms and refine therapeutic strategies.

Jose U. Scher, MD, assistant professor of medicine and director of the Psoriatic Arthritis Center, cites a series of studies under the umbrella of the Preventing Psoriatic Arthritis (PreP) cohort, one of the center's most intriguing and exciting new projects. One-third of patients with psoriasis will develop psoriatic arthritis (PsA), and researchers have identified several factors that enhance the likelihood. "We're now following people with psoriasis over time and looking at clinical, immunological, and environmental factors," says Dr. Scher, who serves as principal investigator of the collaborative effort with the Department of Dermatology.

About 35 percent of the center's psoriatic arthritis patients have a first-degree relative with psoriasis but not arthritis, a key observation that is allowing the center to ask why some patients progress and others don't. Access to the first-degree relatives enables the pursuit of the study. In addition, researchers will be characterizing joint and blood immune cells and microbes present in the gut and skin at the time of arthritis disease progression to track their changes and identify potential prevention targets.

Through a recent NIH grant, the center is also exploring whether microbes can trigger psoriasis or arthritis and how the microbiome's suite of drug-metabolizing enzymes can alter a patient's response to conventional therapy or predict a regimen's effectiveness. "We're trying to understand the relationship between microbes and disease pathogenesis," Dr. Scher says. "We're also trying to look at the question of whether or not microbes can metabolize certain therapeutic drugs and whether microbes that are only present in certain people can predict whether that particular drug is going to work."

### INFORMING CLINICAL TRIALS AND UNCOVERING COMORBIDITIES

Dr. Scher and Soumya M. Reddy, MD, assistant professor of medicine, are co-investigators on a separate NIH-funded study led by the multicenter Psoriatic Arthritis Research Collaborative, or PARC. "This grant will help us understand whether less conventional clinical data such as fatigue or certain patient-reported outcomes can be incorporated into clinical trials moving forward," Dr. Scher says.



🚹 Soumya M. Reddy, MD, and Jose U. Scher, MD

The project is likewise benefiting from the center's patient database, which has already helped NYU Langone researchers describe several significant comorbidities. One in three women seen at the center is taking medication for anxiety or depression, they've found, while the center's patient cohort also suggests a high prevalence of attention deficit disorder (ADD). Both observations may be highly relevant to adherence, given that half of all patients with inflammatory arthritis stop taking their prescribed biological therapy within the first six months. "If you control anxiety, depression, ADD, or other psychiatric comorbidities, are you more likely to see a benefit in psoriatic disease?" Dr. Scher asks. Could improving those comorbidities significantly aid adherence to the arthritis drugs? "These are hypotheses that we are deriving from the data that we collect," he says.

#### LENDING EXPERTISE

Dr. Scher and Dr. Reddy lent their own expertise to the panel that wrote the American College of Rheumatology's first-ever treatment guidelines for psoriatic arthritis, which was released at the end of 2017. In addition, Dr. Scher has been an active member of the FDA's Arthritis Advisory Committee, which advises the federal agency on new arthritis medications. Amid the field's recent therapeutic advances, Dr. Scher says, the advisory committee has helped to secure the approval of lower-cost biosimilar drugs for psoriasis and PsA and to expand the spectrum of available options for patients.

#### Platelet-Endothelial Cell Interactions May Contribute to SLE-Associated Cardiovascular Disease

Evidence of a critical interaction between platelets and endothelial cells that could drive cardiovascular disease pathogenesis in lupus patients is emerging from a collaborative effort between rheumatologists and cardiologists at NYU Langone Health.

Past research efforts documented accelerated atherosclerosis in Caucasian and minority lupus patients. The new study builds on those findings by revealing an abnormal platelet-mediated effect on endothelial gene pathways involved in cell activation. The research suggests that platelets from lupus patients release interleukin-1 beta, which causes an endothelial cell phenotype resembling atherosclerosis.

Because lupus patients can have chronic production of IL-1 beta from their platelets, the study suggests that the triggered endothelium could adopt atherosclerosis-like phenotypes. A follow-up search for factors that may activate platelets in SLE has identified a genetic variant of the Fc receptor that tightly binds infection-fighting immune complexes. The aggressive interaction, the researchers suggest, could activate platelets by delaying clearance of the immune complexes.

Read more on PAGE 14

#### Addressing Lupus Disparities in Minority Populations

New studies at NYU Langone Health are shedding light on which lupus patients have poorer outcomes, why that may be so, and how to improve care for underserved populations. One multiyear collaboration, the Manhattan Lupus Surveillance Program, recently documented higher disease prevalence and aggressiveness among Hispanic and Asian residents compared with their white counterparts and even higher rates among black individuals.

A second line of research, focused on variants of the apolipoprotein L1 (APOL1) gene, is helping researchers understand why some lupus patients may fare worse than others; a new study linked risk variants of the gene to an increased prevalence of atherosclerotic cardiovascular disease among African American SLE patients.

And in a third line of research in a multiethnic cohort of lupus patients, investigators dispelled the misconception that becoming pregnant can trigger lupus nephritis by finding that renal flares and new-onset nephritis are uncommon among pregnant patients with stable or mildly active SLE.



#### New Trials Seek to Expand Limited Treatment Options for Lupus

Over the past half-century, the FDA has approved only one new drug for lupus: belimumab in 2011. Within NYU Langone Health's large portfolio of lupus-targeting clinical trials, two major industry-sponsored efforts are on track for FDA approval. A third, an investigatorinitiated collaborative trial with the Department of Radiology, may provide some guidance on the relative risks of glucocorticoid-induced osteoporosis.

### A PROMISING NEW INTERFERON INHIBITOR

One phase II trial, a collaboration with London-based AstraZeneca and other academic institutions, is nearing the finish line of its investigation of the monoclonal antibody anifrolumab and its ability to treat non-renal manifestations of lupus. The randomized trial, which enrolled 360 subjects, is comparing anifrolumab to the current standard of care for lupus. "This is a particularly exciting clinical trial in terms of our lupus regimen," says Amit Saxena, MD, assistant professor of medicine.

Anifrolumab inhibits the binding of interferon protein to its receptor, and is widely viewed as a driving force in lupus. Pharmaceutical companies have been racing to develop multiple ways of blocking the downstream consequences of interferon activity. Dr. Saxena notes that anifrolumab's phase II data on treating rashes and joint pain have been particularly promising. The FDA recently gave the drug a fast-track designation to expedite the review process and facilitate its development. "Although we do not know which patients have received the active drug or placebo, we've seen impressive outcomes so far, and we're hoping to move this forward as the next drug to be approved for lupus," Dr. Saxena says. A related effort, also in phase II, is testing the same drug on renal manifestations of the disease.

#### REDUCED SIDE EFFECTS COULD RAISE REMISSION RATES FOR LUPUS NEPHRITIS

A second industry-sponsored clinical trial, backed by Victoria, British Columbiabased Aurinia Pharmaceuticals, Inc., is testing the experimental immunosuppressant voclosporin on lupus nephritis. The phase III trial is still enrolling patients, with a goal of 324 subjects in all at NYU Langone and other participating institutions.

Voclosporin belongs to a class of calcineurin inhibitors that includes older medications like tacrolimus and cyclosporine. Traditionally, the latter medications have been used as secondor third-line agents for kidney disease due to unfavorable side effects such as high blood pressure and kidney damage.

With standard-of-care interventions, lupus kidney disease remission rates have lagged, ranging from roughly 25 to 50 percent. Promising phase II trial data suggest that voclosporin compares favorably to standard of care in achieving remission, but with significantly reduced side effects. If it proves efficacious, the investigational drug could be an important add-on to help drive up remission rates.

#### Major Training Grant Bridges Basic and Clinical Research

The Ruth L. Kirschstein National Research Service Award, a T32 grant funded by the NIH, has provided a major new resource for promising predoctoral and postdoctoral trainees at NYU Langone. The division's program, which emphasizes customized skill sets, individualized training, and close mentoring, is helping awardees move toward their leadership goals in academic rheumatology.

Rheumatology fellow Julia Manasson, MD, is drawing upon the training grant for a skin microbiome study to determine why some patients with psoriasis go on to develop psoriatic arthritis while others never progress. Grant recipient and rheumatology fellow Sabina Sandigursky, MD, is studying SLAM-associated protein, or SAP, and its potential role in the function and signaling pathway of the cancer and inflammation-linked PD-1 protein. And MD-PhD student Elliot Philips is using the grant to investigate the functional repertoire of two PD-1 receptor ligands, knowledge that could aid new drug design for both cancer immunotherapy and autoimmune suppression.



#### BETTER SCANS MAY HELP REVEAL OSTEOPOROSIS RISK

Dr. Saxena cites an investigator-initiated collaboration with Gregory Chang, MD, associate professor of radiology, as yet another highlight within the clinical trial portfolio. "We're looking at the risk of osteoporosis in patients with inflammatory diseases, particularly lupus," Dr. Saxena says. Steroid medications can increase the risk for osteoporosis, and dual-energy X-ray absorptiometry, or DXA, scans are the current standard for diagnosing the condition.

Bone density scans can underestimate the risk, however, and the collaborators have hypothesized that patients' bone architecture might reveal more telling differences between osteoporosis induced by steroids and osteoporosis associated with other factors. Dr. Chang has developed a 3T MRI technique to reveal such differences, and he is testing his method on 218 enrollees in a trial done in conjunction with H. Michael Belmont, MD, professor of medicine, and other researchers at NYU Langone. The NIH-funded trial's three arms are comparing the bone structure of lupus patients on chronic steroids, of lupus patients not taking steroids, and of patients diagnosed with other autoimmune diseases. Through the trial, Dr. Saxena says, "we'll be able to better tell patients what their risks are for long-term steroid use." The research also could indicate whether the severity of inflammatory disease is associated with bone changes and could reveal other risk factors for osteoporosis.

## 218

#### ENROLLEES

in a new 3T MRI technique developed to reveal the differences between osteoporosis in lupus patients induced by steroids versus other factors. 2017 IN DEPTH

## Leading the Way in Clinical Care and Groundbreaking Research



### Building New Bridges to Overcome Autoimmunity

With a new director, new faculty, and a half-dozen internally funded pilot projects, the Judith and Stewart Colton Center for Autoimmunity is connecting a diverse group of researchers and aiding a promising range of collaborative efforts at NYU Langone Health.

Timothy B. Niewold, MD, the recently recruited director of the Colton Center, says the three-year-old center is unique in its mission: engaging in cross-disciplinary autoimmune disease research that bridges rheumatology, immunology, genetics, structural biology, neuroimmunology, and other medical specialties.

The Colton Center has made new strides toward that goal with an internal pilot project to foster collaborations and support for "outside the box" ideas, says Dr. Niewold, who arrived in 2017 from the Mayo Clinic. Providing seed money to many diverse ideas, he notes, increases the odds of a breakthrough. "If you're funding a large group of people, there will likely be natural synergies and ways that research can come together, even if it's not apparent when they start," he says.

#### UNCOVERING THE GENETIC ROOTS OF AUTOIMMUNE DISEASE

Dr. Niewold's lab has embarked on its own grant-funded pilot project with Jef D. Boeke, PhD, professor of biochemistry and molecular pharmacology and director of the Institute for Systems Genetics. Dr. Boeke's new method for creating artificial chromosomes, a system he calls the Genome Foundry, enables researchers to partially or completely rewrite chromosomes and recombine them into cell lines. Dr. Boeke's method may allow researchers to more easily express variants of still-mysterious autoimmune disease risk genes in relevant cell lines to better understand which gene portions may drive the disease-associated dysfunction. "That brings us closer to understanding this big question: How do the genes cause disease?" says Dr. Niewold.

In particular, the research may enable a more direct examination of human pathogenesis by allowing researchers to use human cell lines and work with the genome in its native structure, targeting specific genetic changes and observing the result.

#### **A GROWING BODY OF RESEARCH**

Dr. Niewold points to several other center pilot projects that could likewise deliver big dividends. One project, led by Boris Reizis, PhD, professor of medicine and pathology, and Timothy J. Cardozo, MD, PhD, associate professor of biochemistry and molecular pharmacology, is geared toward making a stable form of the enzyme DNASE1L3, which is being studied at NYU Langone's Translational Center of Molecular Profiling and Preclinical and Established Lupus, COMPEL, program. If successful, the project could lead toward a new type of treatment: DNase replacement therapy for deficient individuals and for patients with lupus who are found to have a dysfunction of this enzyme.



```
🚹 Boris Reizis, PhD
```

Separate investigations into autoimmune diseases' genetic and biological mechanisms include a related study by postdoctoral fellow Taro Iwamoto, PhD, a member of Dr. Niewold's research team; an investigation using blood samples to personalize treatments for rheumatoid arthritis patients by Theresa Wampler Muskardin, MD, assistant professor of medicine; research into the interferonproducing cells involved in lupus by cellular immunologist Mark Jensen, PhD, assistant professor of medicine; investigations into the genetic basis of lupus by bioinformatics expert Yogita Ghodke-Puranik, PhD, assistant professor of medicine; and mouse models of kidney disease created by Amrutesh S. Puranik, PhD, to support the lab's investigations of lupus nephritis.

#### A CONNECTIVE CENTER OF KNOWLEDGE

By hosting regular meetings and seminars where researchers can share their best ideas about autoimmune disease, Dr. Niewold notes, the Colton Center is becoming an important gathering and networking place for the scientific community. The center's design means that gastroenterologists studying inflammatory bowel disease and neurologists looking into multiple sclerosis can learn from each other in ways that inform and aid both efforts.

"In essence, the Colton Center will become the link that helps connect the dots between autoimmune disease mechanisms," Dr. Niewold adds.

#### LUPUS NEPHRITIS IS LINKED TO A BACTERIAL AGENT IN THE GUT

An interdisciplinary research team led by Gregg J. Silverman, MD, has now found evidence to suggest that the immune response to a single commensal bacterial strain in the human gut may be a primary driver for lupus nephritis. That association has held through four separate cohorts of lupus patients throughout the United States that were ethnically diverse and included Caucasian, Asian, African American, and Hispanic patient populations.

The collaborative research, funded in part as a Colton Center for Autoimmunity pilot project delving into the links between autoimmunity and the microbiome, was selected for a plenary session at the ACR meeting in 2017. By examining the gut microbiome of lupus patients, the team discovered a specific strain of a species within the *Lachnospiraceae* bacterial family, *Ruminococcus gnavus*, that makes a toxin that may contribute to immune complexmedia ted kidney disease in lupus nephritis. Compared with controls, the gut microbiome of systemic lupus erythematosus patients was found to host a fivefold greater representation of the implicated species. The bacterial abundance was even higher in patients with active lupus nephritis.

Timothy B. Niewold, MD, says the autoimmunity may be linked to autoantibody production against a bacterial component. "So this antibody could be a predictive marker of kidney disease," he says. Unraveling the underlying disease mechanism, Dr. Silverman says, could lead to more targeted and effective therapies. "We hypothesize that reducing or eliminating a single strain of this bacterium may have tremendous therapeutic potential," he says.

"We've developed a simple lab test to evaluate the human immune response and recognition of these bacteria that colonize the intestine but don't cause local intestinal damage." The research is moving rapidly toward refinement of a version of the test for use in the clinic, and toward the development of an animal model that would allow the team to test experimental therapies and further investigate the root causes of the disease.

### New Test May Point the Way Toward a Cure for Rheumatoid Arthritis

Existing treatments for rheumatoid arthritis (RA) may help relieve inflammation. However, the results of a new study published in *Arthritis & Rheumatology* reveal they do little to address the underlying immunological defect of self-attacking autoantibodies.

Researchers at NYU Langone Health and collaborators at the University of Pittsburgh and Dartmouth Medical School suggest that clinical trials for new RA drugs should shift course from standard anti-inflammation strategies to the elimination of B cell-produced antibodies.

#### WHAT'S THE TRUE DRIVER OF AUTOIMMUNITY?

Gregg J. Silverman, MD, the study's senior author, says RA symptoms generally flare whenever patients stop taking antiinflammation drugs such as methotrexate and tumor necrosis factor (TNF) inhibitors. "Every time a patient flares, there is more injury to the body and especially the joints," says Dr. Silverman. Current strategy, though, "does not go to the heart of the disease. It goes to a secondary downstream inflammatory response." In other words, measuring a drug's therapeutic success by clinical examination of RA-associated inflammation is overlooking the true driver of autoimmunity.

Dr. Silverman and colleagues focused on memory B cells, which secrete anticitrullinated protein antibodies, or ACPAs. Research has implicated autoreactive B cells as the pathogenic drivers of RA, and their secreted ACPAs can be useful diagnostic biomarkers and predictors of a poorer long-term prognosis. Even so, researchers haven't understood the immunobiological significance of persistent ACPA production at the cellular level.

After developing a sensitive enzymelinked immunosorbent assay (ELISA) and multiplex bead-based immunoassay to detect the presence of autoantibodies, the research team stimulated memory B cells in a cell culture system and analyzed the kinds of antibodies produced by the cells. In blood samples of RA patients with ACPAs, the team found high levels of ACPA-secreting memory B cells. Patients who lacked these autoantibodies and healthy volunteers, by contrast, had normal levels of these memory B cells.

In patients whose RA had been driven into remission by methotrexate or a TNF inhibitor, however, researchers found that the ACPA levels were still directly proportional to the recirculating memory B cells in the bloodstream; the patients' clinical remission had little or no effect on the circulating burden of these ACPAexpressing B cells. The measurement, then, suggested that neither drug regimen was impacting the underlying dysregulation of humoral immunity.

#### AN INDICATOR TO HELP DEVELOP NEW TREATMENTS

The study findings may explain why stopping standard RA therapy even after an apparent improvement in inflammation symptoms most often results in disease reactivation and a clinical flare. The findings conclude that a clinical disease activity score is not a reliable indicator of whether pathological recirculating B cell autoimmunity has been truly resolved.

"We now have a test that measures the autoimmunity and shows that current treatments don't address the autoimmunity that's the basis of the disease," Dr. Silverman says. "So we propose using this assay to help develop new therapies to hopefully cure the disease. This new assay is based on everything we know about the pathogenesis of the disease."

Backed by the new research, Dr. Silverman says his group's labdeveloped test could provide a far more sensitive gauge of investigational drugs targeting autoimmunity. "We need to develop a longer-term vision of how to improve the treatment of rheumatoid arthritis," he says. "Now that we can better measure the effects of medications," he adds, "this new tool should help to identify agents that target other molecules or cells that have therapeutic advantages that were previously not considered."



🚹 Gregg J. Silverman, MD

### Interactions Between Platelets and Endothelial Cells May Contribute to SLE-Associated Cardiovascular Disease

A collaborative research effort at NYU Langone Health has uncovered new evidence for a critical interaction between platelets and endothelial cells that could be an important accelerator in the pathogenesis of premature atherosclerotic disease in lupus patients.

Cardiologist and senior author Jeffrey S. Berger, MD, associate professor of medicine and surgery and rheumatology researcher, Robert M. Clancy, PhD, associate professor of medicine, say researchers have been trying to round up the major players that contribute to this critical lupus co-morbidity. A past collaboration with Harmony R. Reynolds, MD, associate professor of medicine and associate director of the Cardiovascular Clinical Research Center, confirmed the phenomenon of accelerated atherosclerosis in both Caucasian and minority populations of lupus patients. Importantly, lab measurements linked Dr. Reynolds's clinical assessment of cardiovascular disease to an endothelial disorder. The specific factors contributing to the lupus-atherosclerosis association remained unknown.

#### ILLUMINATING THE ROLE OF ACTIVATED PLATELETS

The new study, published in the journal Arteriosclerosis, Thrombosis, and Vascular Biology, highlights the poorly understood role of activated platelets in accelerating the disease process and clarifies how they associate with endothelial cells. In particular, an RNA microarray of normal human umbilical vein endothelial cells co-incubated with platelets from SLE patients revealed an abnormal plateletmediated effect on gene pathways involved in endothelial cell activation. By contrast, the co-incubation of platelets from healthy volunteers and human umbilical vein endothelial cells resulted in endothelial cells at a quiescent state similar to that of baseline endothelial cells.

### Collaboration

#### WITH NYU LANGONE CARDIOLOGISTS

helped uncover previously hidden platelet-endothelial links to cardiovascular disease in lupus patients.

"Moreover, we were able to identify that the platelets from lupus patients are releasing interleukin-1 beta, which is normally not present in healthy subjects," Dr. Clancy says. "So we identified not only a pathogenic axis of platelet and endothelial cells, but also the mediator that the platelets were releasing to cause a phenotype of the endothelial cells, which resembles the process in atherosclerosis." Because lupus patients can have chronic production of IL-1 beta from platelets in their blood vessels, the research suggests that the triggered endothelium could take on atherosclerosis-like phenotypes. Beyond the upregulation of proinflammatory surface markers, Dr. Clancy adds, endothelial cells activated by platelets in vitro begin processing lipids abnormally compared with untreated endothelial cells.

Study co-author Jill P. Buyon, MD, says the research project arose from the division's efforts to investigate co-morbidities in lupus patients and pursue partnerships with other medical disciplines. Dr. Berger, a cardiologist who specializes in platelet research, helped the team uncover the previously hidden platelet-endothelial links.

"That interdisciplinary vantage allowed us to think about how endothelial cells and platelets, which hadn't been well studied in lupus, might augment the risk of cardiovascular morbidities in SLE patients," Dr. Buyon says.

### A COLLABORATIVE SEARCH FOR SLE PLATELET ACTIVATORS

To better understand the underlying mechanism, a follow-up collaboration among the Berger, Buyon, and Clancy laboratories is asking what factors may be activating platelets in lupus. So far, the research has identified a key player, an Fc receptor on the platelet surface that binds immune complexes, a component of protective immunity. These immune complexes, normally only transiently produced and then cleared away, persist in elevated levels in lupus patients.

The team's emerging research is suggesting that a genetic variant of the Fc receptor, in which a histidine has been changed to an arginine, yields a highly reactive form of the receptor. The latter form, present in 40 percent of the U.S. population, interacts aggressively with immune complexes, potentially further delaying their clearance and activating platelets. "If we can get supportive data, it would have great implications in identifying risk for accelerated atherosclerosis because tests for this genetic condition are widely available," Dr. Clancy says.

The research is converging on an important clinical message, he adds: "If you are a lupus patient, and you have persistently circulating immune complexes, and you have the variant form of this protein, you are at higher risk." Dr. Clancy says the implications could extend far beyond atherosclerosis as well, given that blood vessel properties can influence blood flow to the kidneys and other important physiological functions. The continued collaboration, then, could help clinicians identify a wider pool of at-risk patients and enable earlier interventions. "An interdisciplinary vantage with cardiology allowed us to think about how endothelial cells and platelets might augment the risk of cardiovascular morbidities."

-Jill P. Buyon, MD

### Documenting and Addressing Patient Disparities in Lupus

New studies are helping to resolve long-standing questions of why some minority patients with lupus may experience poorer outcomes, and how researchers and doctors might begin to address these disparities and enhance care for underserved populations.

In one such study, the Manhattan Lupus Surveillance Program, Peter M. Izmirly, MD, assistant professor of medicine, and colleagues examined the incidence and prevalence of lupus in the borough of Manhattan, uncovering glaring racial and ethnic disparities in both lupus prevalence and aggressiveness. The population-based epidemiological study, published in *Arthritis & Rheumatology*, is the result of a multiyear collaboration with the New York City Department of Health and Mental Hygiene.

Combined with a sister study published by the California Lupus Surveillance Project, the research is among the first to provide substantive lupus estimates for Hispanic and Asian demographics. Paradoxically, these are two of the fastest-growing populations for lupus, but until this study, available data surrounding their risk were limited. The New York and California studies suggest that the prevalence and aggressiveness of lupus are significantly higher among Hispanic and Asian residents than among their white counterparts—though not as high as among black individuals.

"We found that about half the patients with lupus who are black, Asian, or Hispanic are likely to develop lupus kidney disease—a major driver of morbidity and mortality in lupus compared to just a quarter of white populations," notes Dr. Izmirly. In addition, across all demographics, the burden was found to be far higher in women than in men.

Study coauthor Jill P. Buyon, MD, notes that these findings both draw attention to

a disproportionate impact on Asians and Hispanics and underscore the even greater prevalence, incidence, and severity of the disease among black patients, particularly women. "The study proves without a shadow of a doubt that lupus is more severe in minorities," she says.

A critical take-home message from the research, adds Dr. Izmirly, is that frontline physicians should be on the lookout for symptoms such as joint pain, rashes, abnormal urinalysis, swelling in the legs, and unexplained hypertension within these demographics. "Often, by the time we've seen some of these patients the disease has already inflicted irreversible damage," he says. "With this research as context, we hope to tilt these time lines toward earlier diagnosis—and improved outcomes."

"With this new research as context, we hope to tilt time lines toward earlier diagnosis—and improved outcomes in minority patients."

-Peter M. Izmirly, MD



🚹 Francis Appeadu-Mensah and Peter M. Izmirly, MD

#### RESOLVING THE GENETIC MECHANISM OF CARDIOVASCULAR DISEASE IN LUPUS PATIENTS

A second line of research, examining genetic variants, is beginning to help researchers understand why some lupus patients fare worse than others. The study, led by Ashira D. Blazer, MD, instructor of medicine, found that risk variants of the apolipoprotein L1 (APOL1) gene were associated with prevalent atherosclerotic cardiovascular disease in a cohort of 113 African American SLE patients.

Previous research had suggested that atherosclerosis is more predominant in African American lupus patients, who are at double the risk for cardiovascular disease relative to white patients, and that certain variants of the APOL1 gene are far more common in patients of African ancestry. The new study, published in the journal *PLOS ONE*, helps build the case that SLE may have a potentiating effect on APOL1-related cardiovascular phenotypes.

To get at the underlying mechanism, Dr. Blazer is now assessing whether and how APOL1 risk variants increase the vulnerability of human umbilical vein endothelial cells to injury. "The variant protein confers an increased risk of damage to endothelial cells when the cells are exposed to lupus-relevant inflammatory stimuli like interferons or autoantigens," she says.

The inflammatory insult of lupus activates the immune system, which in turn causes increased expression of the APOL1 gene, Dr. Blazer has found. Beyond a certain threshold, the immune factor becomes toxic. "Because lupus perpetually activates the immune system, a number of cell types including endothelial cells accumulate this toxic protein—with damaging effects," she says. The endothelial cell dysfunction includes decreased mitochondrial respiration and reduced angiogenesis, due in part to an impaired ability to complete autophagy. Paradoxically, some lupus patients who carry the APOL1 risk variant have lower inflammatory signals than other patients. The emerging picture, Dr. Blazer says, suggests that some of the damage accrual blamed on lupus, such as cardiovascular and kidney disease, may be more related to the APOL1 risk factors themselves. "Once we can characterize lupus in the setting of these genetic risk factors, we can better point to what's disease activity and what's not and where immunosuppression treatment should apply," she says.

In addition to the well-characterized lupus cohort at NYU Langone, Dr. Blazer is helping to build patient cohorts in Ghana and Nigeria to enable further characterization. "These new cohorts will help us to compare damage and disease and cytokine profiles, and tease out which lupus-associated manifestations are due to genetic versus environmental exposures," she notes. Identifying these clinical differences, Dr. Blazer adds, could provide insight into the cause and effect behind the disease, leading to more tailored treatment options.

#### BUILDING A PICTURE OF PREGNANCY'S TRUE RISKS IN MINORITY LUPUS PATIENTS

In a separate line of research at NYU Langone, investigators have helped assess the risk of renal flares during pregnancy in a multiethnic cohort of lupus patients in partial or complete remission after a previous diagnosis of lupus nephritis. The study, published in the *Clinical Journal of the American Society of Nephrology*, also analyzed the risk of new-onset nephritis in pregnant patients with stable or mildly active SLE.

Among the findings uncovered by Dr. Buyon, the lead study author: De novo kidney involvement in SLE, even in ethnic or racial minorities, is uncommon during pregnancy. This development could help clear up some patients' misconception that becoming pregnant could trigger lupus nephritis. "If you do not have kidney involvement as part of your lupus, just getting pregnant will not cause the kidneys to become a target," notes Dr. Buyon.

Additionally, the study found that past kidney disease and low C4 protein levels at the baseline measurement were independently associated with a higher risk of developing active lupus nephritis. However, researchers also concluded that the presence of antibodies to doublestranded DNA alone should not raise concerns in patients with past kidney disease, so long as they are in remission. "If you've had kidney disease in the past but the condition is inactive at the time of pregnancy, you can expect to do wellthough not quite as well as those who never had any kidney disease," says Dr. Buyon.

Of the 373 patients enrolled in the Predictors of Pregnancy Outcome: Biomarkers in Antiphospholipid Syndrome and Systemic Lupus Erythematosus Study, more than half were ethnic or racial minorities. Their inclusion means that the new risk assessment could help doctors provide better guidance before and during pregnancy to ethnically and racially diverse populations of lupus patients.

## 50%

#### **OF PATIENTS ENROLLED**

in a study to assess the risk of renal flares during pregnancy were ethnically or racially diverse.

# Training Grant Boosts New Generation of Physician-Scientists

The Ruth L. Kirschstein National Research Service Award, a highly competitive NIH training grant, is aiding a new generation of physician-scientists at NYU Langone Health.

The funding for the Division of Rheumatology, known as a T32 grant, is helping predoctoral and postdoctoral trainees on their quest for careers in academic medicine, be it at the bench or the bedside.

"This training grant is critical in helping to foster the development of the next leaders in rheumatology," says Jill P. Buyon, MD. Unlike standard clinical training programs, says Dr. Buyon, this program emphasizes the acquisition of customized skill sets, the opportunity for individualized research training experiences, and close mentoring and milestone expectations. The program is backed by highly qualified mentors spanning a broad range of expertise from basic immunology to public health. The division's program, Translational Basic and Clinical Research Training in Rheumatology, focuses on three main areas of investigation: lupus and diseases of systemic autoimmunity, inflammatory and autoimmune arthritis, and degenerative and metabolic joint and bone disease.



🚹 Elliot Philips, Sabina Sandigursky, MD, and Julia Manasson, MD

#### EXAMINING SKIN MICROBIOMES TO IDENTIFY THE ROOTS OF PSORIATIC ARTHRITIS

Julia Manasson, MD, a member of the first class of T32 trainees, is studying the skin microbiome of psoriatic arthritis patients. Dr. Manasson is in her third year as a rheumatology fellow and is working in the lab of Jose U. Scher, MD, assistant professor of medicine.

"We're looking at healthy individuals compared to patients who just have psoriasis of the skin and patients who have both psoriasis of the skin and psoriatic arthritis to see if there are any differences as you go along the disease spectrum," Dr. Manasson says. The overall goal, she adds, is to determine why some patients with psoriasis go on to develop psoriatic arthritis while others never progress. Although a myriad of factors is likely at work, the research aims to determine whether differences in the skin microbiome of patients might play a role.

While her research is still in its preliminary stages, Dr. Manasson says, the training grant has helped her learn how to conduct clinical analyses and use computational and statistical techniques to analyze her data, including a microbiome analytical tool called Quantitative Insights into Microbial Ecology, or QIIME.

Dr. Manasson's project also has benefited from Dr. Scher's large and well-organized sample repository from psoriatic arthritis patients, including both skin and stool samples. The training support, in turn, has helped her secure other grants to help launch her research portfolio and reinforce both sides of her physician-scientist aspirations.

#### RESOLVING THE REGULATION OF PD-1 IN RHEUMATOID ARTHRITIS

Training grant recipient Sabina Sandigursky, MD, is investigating T cell signaling as it relates to the PD-1 protein. Inhibitors of this receptor have demonstrated their effectiveness in achieving remission in multiple cancers.

"It could also be valuable in autoimmune disease if you potentially create an agonist drug," she says. Dr. Sandigursky is in her fourth year as a rheumatology fellow and working in the laboratory of Adam Mor, MD, PhD, the Saul J. Farber Assistant Professor of Medicine, and assistant professor of pathology.

For her project, she's studying the SLAM-associated protein, or SAP, which belongs to a family of cell surface receptors, and its potential role in the function and signaling pathway of PD-1. She's also looking at the way in which PD-1 is regulated in patients with inflammatory arthritis, particularly rheumatoid arthritis. Dr. Sandigursky hypothesizes that T cells in the synovial fluid of inflamed joints have elevated PD-1 levels while the levels are lower in cellular compartments with less inflammation, such as the peripheral blood. Ultimately, her research could yield new information about how to manipulate the signaling pathway to regulate PD-1 levels and activity.

#### ASSESSING STRUCTURAL AND FUNCTIONAL DIFFERENCES TO UNDERSTAND T CELL ACTIVATION

Elliot Philips, the third member of the T32 class, is an MD-PhD student who has embarked on a research project whose focus on cancer immunology could have implications for rheumatology and autoimmunity. He is investigating immune checkpoints and inhibitory receptors on T cells, which have been targeted for cancer immunotherapy. Researchers have suggested that the T cells' protein counterparts, stimulatory receptors, also could be targeted as part of therapies for autoimmune diseases. Philips is in the fourth year of his MD-PhD track and working in the lab of structural biologist Xiangpeng Kong, PhD, professor of biochemistry and molecular pharmacology.

In particular, Philips is studying PDL1 and PDL2, ligands for the PD-1 receptor. From a functional standpoint, he's investigating why the immune system's antigen presenting cells possess both PDL1 and PDL2 ligands. Once researchers understand the dynamic range of the ligands' functions, he says, that knowledge could be translated into better drug design for cancer immunotherapy on one side and autoimmune suppression on the other.

Too little immune activation can abet chronic infection and cancer, while too much can spur autoimmunity. "The goal for both therapies is to tip the balance back in the opposite direction," Philips says.

### Academic Activities

#### SELECTED PUBLICATIONS

Ainsworth HC, Marion MC, Bertero T, Brucato A, Cimaz R, Costedoat-Chalumeau N, Fredi M, Gaffney P, Kelly J, Levesque K, Maltret A, Morel N, Ramoni V, Ruffatti A, Langefeld CD, Buyon JP, Clancy RM. Association of natural killer cell ligand polymorphism HLA-C Asn80Lys with the development of anti-SSA/Ro-associated congenital heart block. *Arthritis & Rheumatology*. November 2017; 69(11): 2170–2174.

Azoulay-Alfaguter I, Strazza M, Peled M, Novak HK, Muller J, Dustin ML, Mor A. The tyrosine phosphatase SHP-1 promotes T cell adhesion by activating the adaptor protein CrkII in the immunological synapse. *Science Signaling*. August 8, 2017; 10(491): eaal2880.

Basavaraj A, Segal L, Samuels J, Feintuch J, Alter K, Moffson D, Scott A, Addrizzo-Harris D, Liu M, Kamelhar D. Effects of chest physical therapy in patients with non-tuberculous mycobacteria. *International Journal of Respiratory & Pulmonary Medicine*. January 21, 2017; 4: 065.

Berube LT, Kiely M, Yazici Y, Woolf K. Diet quality of individuals with rheumatoid arthritis using the Healthy Eating Index (HEI)-2010. *Nutrition & Health*. March 1, 2017; 23(1): 17–24.

Blazer AD, Clancy RM. ApoL1 and the immune response of patients with systemic lupus erythematosus. *Current Rheumatology Reports*. March 1, 2017; 19(3): 13.

Blazer A, Wang B, Simpson D, Kirchhoff T, Heffron S, Clancy RM, Heguy A, Ray K, Snuderl M, Buyon JP. Apolipoprotein L1 risk variants associate with prevalent atherosclerotic disease in African American systemic lupus erythematosus patients. *PLOS ONE*. August 29, 2017; 12: e0182483– e0182483.

Broder A, Mowrey WB, Izmirly P, Costenbader KH. Validation of systemic lupus erythematosus diagnosis as the primary cause of renal failure in the US Renal Data System. *Arthritis Care & Research*. April 1, 2017; 69 (4): 599–604.

Broder A, Mowrey WB, Khan HN, Jovanovic B, Londono-Jimenez A, Izmirly P, Putterman C. Tubulointerstitial damage predicts end stage renal disease in lupus nephritis with preserved to moderately impaired renal function: a retrospective cohort study. *Seminars in Arthritis & Rheumatism*. July 13, 2017; 10.1016/j.semarthrit.2017.07.007.

Brucato A, Clancy R, Buyon JP, Meroni M. Neonatal lupus syndromes: pathogenesis and clinical features. In: Atzeni F, Dorea A, Nurmohamed M, Pauletto P (Eds). *The Heart in Systemic Autoimmune Diseases, Volume 14, Second Edition.* March 2, 2017: 63–87. Elsevier Science.

Buyon JP, Kim MY, Guerra MM, Lu S, Reeves E, Petri M, Laskin CA, Lockshin MD, Sammaritano LR, Branch DW, Porter TF, Sawitzke A, Merrill JT, Stephenson MD, Cohn E, Salmon JE. Kidney outcomes and risk factors for nephritis (flare/de novo) in a multiethnic cohort of pregnant patients with lupus. *Clinical Journal of the American Society of Nephrology*. June 7, 2017; 12(6): 940–946.

Caminer AC, Haberman R, Scher JU. Human microbiome, infections, and rheumatic disease. *Clinical Rheumatology*. December 2017; 36(12): 2645–2653.

Choi J-H, Wallach AI, Rosales D, Margiewicz SE, Belmont HM, Lucchinetti CF, Minen MT. Clinical reasoning: a 50-year-old woman with SLE and a tumefactive lesion. *Neurology*. September 2017; 89(12): e140–e145. Choi MY, Clarke AE, St. Pierre Y, Hanly JG, Urowitz MB, Romero-Diaz J, Gordon C, Bae S-C, Bernatsky S, Wallace DJ, Merrill JT, Isenberg DA, Rahman A, Ginzler EM, Petri M, Bruce IN, Dooley MA, Fortin P, Gladman DD, Sanchez-Guerrero J, Steinsson K, Ramsey-Goldman R, Khamashta MA, Aranow C, Alarcon GS, Manzi S, Nived O, Zoma AA, van Vollenhoven RF, Ramos-Casals M, Ruiz-Irastorza G, Lim SS, Kalunian KC, Inanc M, Kamen DL, Peschken CA, Jacobsen S, Askanase A, Buyon JP, Mahler M, Fritzler MJ. The prevalence and determinants of anti-DFS70 autoantibodies in an international inception cohort of systemic lupus erythematosus patients. *Lupus*. September 2017; 26(10): 1051–1059.

Clancy RM, Markham AJ, Jackson T, Rasmussen SE, Blumenberg M, Buyon JP. Cardiac fibroblast transcriptome analyses support a role for interferogenic, profibrotic and inflammatory genes in anti-SSA/Ro-associated congenital heart block. *American Journal of Physiology—Heart & Circulatory Physiology*. June 16, 2017; 313(3): H631-H640.

Costedoat-Chalumeau N, Houssiau F, Izmirly P, Le Guern V, Navarra S, Jolly M, Ruiz-Irastorza G, Baron G, Hachulla E, Agmon-Levin N, Shoenfeld Y, Dall'Ara F, Buyon JP, Deligny C, Cervera R, Lazaro E, Bezanahary H, Leroux G, Morel N, Viallard J-F, Pineau C, Galicier L, Van Vollenhoven R, Tincani A, Nguyen H, Gondran G, Zahr N, Pouchot J, Piette J-C, Petri M, Isenberg D. A prospective international study on adherence to treatment in 305 patients with flaring SLE: assessment by drug levels and by self-administered questionnaires. *Clinical Pharmacology & Therapeutics*. November 9, 2017; doi: 10.1002/cpt.885.

Curtis JR, Herrem C, Ndlovu MN, O'Brien C, Yazici Y. A somatization comorbidity phenotype impacts response to therapy in rheumatoid arthritis: post-hoc results from the certolizumab pegol phase 4 PREDICT trial. Arthritis Research & Therapy. September 29, 2017; 19(1): 215.

El Bannoudi H, Anquetil C, Braunstein M; Pond SLK, Silverman GJ. Unbiased RACE-based massive parallel surveys of human IgA antibody repertoires. Methods in Molecular Biology. In: Kaveri S, Bayry J (Eds). *Natural Antibodies. Methods in Molecular Biology, Vol. 1643.* Humana Press, July 1, 2017: 45–73.

El-Haddad C, Castrejon I, Gibson KA, Yazici Y, Bergman MJ, Pincus T. MDHAQ/RAPID3 scores in patients with osteoarthritis are similar to or higher than in patients with rheumatoid arthritis: a cross-sectional study from current routine rheumatology care at four sites. *RMD Open.* July 18, 2017; 3(1): e000391.

Erkan D, Unlu O, Sciascia S, Belmont HM, Branch DW, Cuadrado MJ, Gonzalez E, Knight JS, Uthman I, Willis R, Zhang Z, Wahl D, Zuily S, Tektonidou MG. Hydroxychloroquine in the primary thrombosis prophylaxis of antiphospholipid antibody positive patients without systemic autoimmune disease. *Lupus*. August 1, 2017; doi: 10.1177/0961203317724219.

Friedman D, Lovig L, Halushka M, Clancy RM, Izmirly PM, Buyon JP. No histologic evidence of foetal cardiotoxicity following exposure to maternal hydroxychloroquine. *Clinical & Experimental Rheumatology*. September–October 2017; 35(5): 857–859.

Fruchter R, Ahmad M, Pillinger M, Galeano C, Cronstein BN, Gold-von Simson G. Teaching targeted drug discovery and development to healthcare professionals. *Clinical & Translational Science*. November 7, 2017; doi: 10.1111/ cts.12523. Furie R, Khamashta M, Merrill JT, Werth VP, Kalunian K, Brohawn P, Illei GG, Drappa J, Wang L, Yoo S. Anifrolumab, an anti-interferon-alpha receptor monoclonal antibody, in moderate-to-severe systemic lupus erythematosus. *Arthritis & Rheumatology*. February 2017; 69(2): 376–386.

Garcia Ferrer HR, Azan A, Iraheta I, Von Feldt J, Espinoza LR, Manasson J, Scher JU, Garcia Kutzbach A, Ogdie A. Potential risk factors for reactive arthritis and persistence of symptoms at 2 years: a case-control study with longitudinal follow-up. *Clinical Rheumatology*. November 14, 2017; doi: 10.1007/s10067-017-3911-3.

Gronwall C, Amara K, Hardt U, Krishnamurthy A, Steen J, Engstrom M, Sun M, Ytterberg AJ, Zubarev RA, Scheel-Toellner D, Greenberg JD, Klareskog L, Catrina AI, Malmstrom V, Silverman GJ. Autoreactivity to malondialdehyde-modifications in rheumatoid arthritis is linked to disease activity and synovial pathogenesis. *Journal of Autoimmunity*. November 2017; 84: 29–45.

Gronwall C, Hardt U, Gustafsson JT, Elvin K, Jensen-Urstad K, Kvarnstrom M, Grosso G, Ronnelid J, Padykov L, Gunnarsson I, Silverman GJ, Svenungsson E. Depressed serum IgM levels in SLE are restricted to defined subgroups. *Clinical Immunology*. October 2017; 183: 304–315.

Harbi S, Park H, Gregory M, Lopez P, Chiriboga L, Mignatti P. Arrested development: infantile hemangioma and the stem cell teratogenic hypothesis. *Lymphatic Research & Biology*. June 1, 2017; 15(2): 153–165.

Harrold LR, Etzel CJ, Gibofsky A, Kremer JM, Pillinger MH, Saag KG, Schlesinger N, Terkeltaub R, Cox V, Greenberg JD. Sex differences in gout characteristics: tailoring care for women and men. *BMC Musculoskeletal Disorders*. March 14, 2017; 18: e108.

Hatemi G, Meara A, Ozguler Y, Direskeneli H, Mahr A, Easley E, Gurcan M, Davis T, Gul A, Yazici Y, Zottenberg K, Esatoglu SN, Erer B, Kamali S, Yazici H, Cronholm PF, Merkel PA. Developing a core set of outcome measures for Behcet Disease: report from OMERACT 2016. *Journal of Rheumatology*. November 2017; 44(11): 1750–1753.

Hatemi G, Yazici Y. Apremilast for the treatment of Behcet's syndrome. *Expert Opinion on Orphan Drugs*. July 24, 2017; 5(8): e677–e681.

Hoffman RW, Merrill JT, Alarcon-Riquelme MME, Petri M, Dow ER, Nantz E, Nisenbaum LK, Schroeder KM, Komocsar WJ, Perumal NB, Linnik MD, Airey DC, Liu Y, Rocha GV, Higgs RE. Gene expression and pharmacodynamic changes in 1,760 systemic lupus erythematosus patients from two phase III trials of BAFF blockade with Tabalumab. *Arthritis & Rheumatology*. March 2017; 69(3): 643–654.

Igel TF, Krasnokutsky S, Pillinger MH. Recent advances in understanding and managing gout. *F1000Research*. March 10, 2017; 6: e247.

Inbar A-A, Strazza M, Peled M, Novak HK, Muller J, Dustin ML, Mor A. The tyrosine phosphatase SHP-1 promotes T cell adhesion by activating the adaptor protein CrkII in the immunological synapse. *Science Signaling*. August 8, 2017; 10(491): eaal288.

Ince D, Swearingen C, Yazici Y. Finger and wrist pain in children using game consoles and laptops: younger children and longer time are associated with increased pain. *Bulletin of the Hospital for Joint Diseases*. June 2017; 75(2): 101–103.

Isaac S, Scher JU, Djukovic A, Jimenez N, Littman DR, Abramson SB, Pamer EG, Ubeda C. Short- and long-term effects of oral vancomycin on the human intestinal microbiota. Journal of Antimicrobial Chemotherapy. January 2017; 72(1): 128-136.

Izmirly PM, Halushka MK, Rosenberg AZ, Whelton S, Rais-Bahrami K, Nath DS, Parton H, Clancy RM, Rasmussen S, Saxena A, Buyon JP. Clinical and pathologic implications of extending the spectrum of maternal autoantibodies reactive with ribonucleoproteins associated with cutaneous and now cardiac neonatal lupus from SSA/Ro and SSB/La to U1RNP. *Autoimmunity Reviews*. September 2017; 16(9): 980–983.

Izmirly P, Saxena A, Buyon JP. Progress in the pathogenesis and treatment of cardiac manifestations of neonatal lupus. *Current Opinion in Rheumatology*. September 2017; 29(5): 467-472.

Izmirly PM, Wan I, Sahl S, Buyon JP, Belmont HM, Salmon JE, Askanase A, Bathon JM, Geraldino-Pardilla L, Ali Y, Ginzler EM, Putterman C, Gordon C, Helmick CG, Parton H. The incidence and prevalence of systemic lupus erythematosus in New York County (Manhattan), New York: The Manhattan Lupus Surveillance Program. *Arthritis & Rheumatology*. October 2017; 69(10): 2006–2017.

Kaplowitz ET, Ferguson S, Guerra M, Laskin CA, Buyon JP, Petri M, Lockshin MD, Sammaritano LR, Branch DW, Merrill JT, Katz P, Salmon JE. Socioeconomic status contributes to racial/ethnic disparities in adverse pregnancy outcomes among women with systemic lupus erythematosus. *Arthritis Care & Research*. May 8, 2017; doi: 10.1002/acr.23263.

Kerr GS, Swearingen C, Mikuls TR, Yazici Y. Use of biologic therapy in racial minorities with rheumatoid arthritis from 2 US health care systems. *Journal of Clinical Rheumatology*. January 2017; 23(1): 12–18.

Kim M, Merrill J, Kalunian K, Hahn B, Roach A, Izmirly P. Brief report: longitudinal patterns of response to standard of care therapy for systemic lupus erythematosus: implications for clinical trial design. *Arthritis & Rheumatology*. April 2017; 69(4): 785–790.

Kissin EY, Pillinger MH. The sound and the fury: musculoskeletal ultrasound in the diagnosis of gout. *Arthritis & Rheumatology*. February 2017; 69(2): 249–252.

Krasnokutsky S, Oshinsky C, Attur M, Ma S, Zhou H, Zheng F, Chen M, Patel J, Samuels J, Pike VC, Regatte R, Bencardino J, Rybak L, Abramson S, Pillinger MH. Serum urate levels predict joint space narrowing in non-gout patients with medial knee osteoarthritis. *Arthritis & Rheumatology.* June 2017; 69(6): 1213–1220.

Lane NE, Corr M, Baer N, Yazici Y. Wnt signaling in osteoarthritis: a 2017 update. *Current Treatment Options in Rheumatology*. June 2017; 3(2): 1–11.

Leccese P, Yazici Y, Olivieri I. Behcet's syndrome in nonendemic regions. *Current Opinion in Rheumatology*. January 2017; 29(1): 12–16.

Madelin G, Xia D, Brown R, Babb J, Chang G, Krasnokutsky S, Regatte RR. Longitudinal study of sodium MRI of articular cartilage in patients with knee osteoarthritis: initial experience with 16-month follow-up. *European Radiology*. January 2018; 28(1): 133–142.

Makarem N, Bandera EV, Lin Y, Jacques PF, Hayes RB, Parekh N. Carbohydrate nutrition and risk of adiposityrelated cancers: results from the Framingham Offspring cohort (1991-2013). *British Journal of Nutrition*. June 2017; 117(11): 1603–1614.

Manasson J, Shen N, Garcia Ferrer HR, Ubeda C, Iraheta I, Heguy A, Von Feldt JM, Espinoza LR, Kutzbach AG, Segal LN, Ogdie A, Clemente JC, Scher JU. Gut microbiota perturbations in reactive arthritis and post-infectious spondyloarthritis. *Arthritis & Rheumatology*. October 26, 2017; doi: 10.1002/art.40359.

Modjinou DV, Krasnokutsky S, Gyftopoulos S, Pike VC, Karis E, Keenan RT, Lee K, Crittenden DB, Samuels J, Pillinger MH. Comparison of dual-energy CT, ultrasound and surface measurement for assessing tophus dissolution during rapid urate debulking. *Clinical Rheumatology*. September 2017; 36(9): 2101–2107.

Nhek S, Clancy R, Lee KA, Allen NM, Barrett TJ, Marcantoni E, Nwaukoni J, Rasmussen S, Rubin M, Newman JD, Buyon JP, Berger JS. Activated platelets induce endothelial cell activation via an interleukin-1beta pathway in systemic lupus erythematosus. *Arteriosclerosis, Thrombosis, & Vascular Biology.* April 2017; 37(4): 707-716.

Nikiphorou E, Galloway J, Riel P, Yazici Y, Haugeberg G, Ostor A, Gogus F, Kauppi M, Sokka T. The spectrum of early rheumatoid arthritis practice across the globe: results from a multinational cross sectional survey. *Clinical & Experimental Rheumatology*. May-June 2017; 35(3): 477-483.

Okhovat J-P, Ogdie A, Reddy SM, Rosen CF, Scher JU, Merola JF. Psoriasis and Psoriatic Arthritis Clinics Multicenter Advancement Network Consortium (PPACMAN) Survey: benefits and challenges of combined rheumatology-dermatology clinics. *Journal of Rheumatology*. May 2017; 44(5): 693–694.

Pelzek AJ, Gronwall C, Rosenthal P, Greenberg JD, McGeachy M, Moreland L, Rigby WFC, Silverman GJ. Persistence of disease-associated anti-citrullinated protein antibody-expressing memory B cells in rheumatoid arthritis in clinical remission. *Arthritis & Rheumatology*. June 2017; 69(6): 1176–1186.

Reed JH, Gorny MK, Li L, Cardozo T, Buyon JP, Clancy RM. Ro52 autoantibodies arise from self-reactive progenitors in a mother of a child with neonatal lupus. *Journal of Autoimmunity*. May 2017; 79: 99–104.

Rogier R, Ederveen THA, Boekhorst J, Wopereis H, Scher JU, Manasson J, Frambach SJCM, Knol J, Garssen J, van der Kraan PM, Koenders MI, van den Berg WB, van Hijum SAFT, Abdollahi-Roodsaz S. Aberrant intestinal microbiota due to IL-1 receptor antagonist deficiency promotes IL-17- and TLR4-dependent arthritis. *Microbiome*. June 23, 2017; 5(1): 63.

Rogier R, Evans-Marin H, Manasson J, van der Kraan PM, Walgreen B, Helsen MM, van den Bersselaar LA, van de Loo FA, van Lent PL, Abramson SB, van den Berg WB, Koenders MJ, Scher JU, Abdollahi-Roodsaz S. Alteration of the intestinal microbiome characterizes preclinical inflammatory arthritis in mice and its modulation attenuates established arthritis. *Scientific Reports*. November 15, 2017; 7: doi:10.1038/s41598-017-15802-x.

Sandigursky S, Silverman GJ, Mor A. Targeting the programmed cell death-1 pathway in rheumatoid arthritis. *Autoimmunity Reviews*. August 2017; 16(8): 767-773.

Shah N, Sandigursky S, Mor A. The potential role of inhibitory receptors in the treatment of psoriasis. *Bulletin of the Hospital for Joint Diseases*. May 2017; 75(3): 155–163.

Shefler I, Salamon P, Levi-Schaffer F, Mor A, Hershko AY, Mekori YA. MicroRNA-4443 regulates mast cell activation by T cell-derived microvesicles. *Journal of Allergy & Clinical Immunology*. August 16, 2017; doi: 10.1016/j.jaci.2017.06.045.

Slobodnick A, Krasnokutsky S, Lehmann RA, Keenan RT, Quach J, Francois F, Pillinger MH. Decreased colorectal atypia among a cohort of gout patients. *Scandinavian Journal of Rheumatology*. June 26, 2017; doi: 10.1080/03009742.2017.1293728.

Soleymani T, Reddy SM, Cohen JM, Neimann AL. Early recognition and treatment heralds optimal outcomes: the benefits of combined rheumatology-dermatology clinics and integrative care of psoriasis and psoriatic arthritis patients. *Current Rheumatology Reports*. November 20, 2017; 20(1): 1.

Strazza M, Azoulay-Alfaguter I, Peled M, Smrcka AV, Skolnik EY, Srivastava S, Mor A. PLCepsilon1 regulates SDF-1alphainduced lymphocyte adhesion and migration to sites of inflammation. *Proceedings of the National Academy of Sciences of the United States of America*. March 7, 2017; 114(10): 2693–2698.

Strazza M, Mor A. Consider the chemokines: a review of the interplay between chemokines and T cell subset function. *Discovery Medicine*. August 2017; 24(130):31–39.

Thottam GE, Krasnokutsky S, Pillinger MH. Gout and metabolic syndrome: a tangled web. *Current Rheumatology Reports.* August 26, 2017; 19(10): 60.

Tocheva AS, Mor A. Checkpoint inhibitors: applications for autoimmunity. *Current Allergy & Asthma Reports*. September 27, 2017; 17(10): 72.

Yazici H, Yazici Y. An update on vasculitides—lumps and constructs. *Current Opinion in Rheumatology*. January 2017; 29(1): 1–3.

Zuily S, Domingues V, Suty-Selton C, Eschwege V, Bertoletti L, Chaouat A, Chabot F, Regnault V, Horn EM, Erkan D, Wahl D. Antiphospholipid antibodies can identify lupus patients at risk of pulmonary hypertension: a systematic review and meta-analysis. *Autoimmunity Reviews*. June 2017; 16(6): 576–586.

### Academic Activities

#### **AWARDS AND HONORS**

**Stephen Honig, MD,** was promoted to Associate Professor of Medicine.

Julia Manasson, MD, research fellow, Department of Medicine, received the 2017 American College of Rheumatology Distinguished Fellow Award. **Adam Mor, MD, PhD,** was appointed the Saul J. Farber Assistant Professor of Medicine.

#### GRANTS

Mukundan G. Attur, associate professor of medicine, received a grant from Roche for "The Role of Vascular Adhesion Protein-1 (VAP-1) in Inflammation."

Ashira Blazer, MD, instructor of medicine, received the NIH KL2 grant, "ESCALATE: Elevated SLE Cytokines, Autophagy Levels, and APOL1 Transcription in Endothelium."

Jill P. Buyon, MD, Lady Va and Sir Deryck Maughan Professor of Rheumatology, Department of Medicine, director of the Division of Rheumatology, and director of the Lupus Center, received the NIH T32 grant, "Translational Basic and Clinical Research Training in Rheumatology."

Jill P. Buyon, MD; Peter M. Izmirly, MD, assistant professor of medicine; **Robert Clancy, PhD**, associate professor of medicine; **Gregg Silverman, MD**, professor of medicine and pathology; and **Boris Reizis, PhD**, professor of medicine and pathology, received the NIH P50 grant "Translational Center of Molecular Profiling in Preclinical and Established Lupus (COMPEL)."

**Jill P. Buyon, MD; Robert Clancy, PhD;** and **Jeffrey S. Berger, MD,** associate professor of medicine and surgery, received the NIH R21 grant, "FcγRIIA, Platelet Activity, and Vasculopathy in Systemic Lupus Erythematosus." Jill P. Buyon, MD; Robert Clancy, PhD; and Carl D. Langefeld, PhD, section head and professor of biostatistical sciences at Wake Forest School of Medicine, received the NIH R21 grant, "Genetics and the Microbiome in Anti-Ro Preclinical and Established Autoimmunity."

**Gregory Chang, MD,** associate professor of radiology and section chief of musculoskeletal imaging: **Amit Saxena, MD,** assistant professor of medicine; and **H. Michael Belmont, MD,** professor of medicine, medical director at NYU Langone Orthopedic Hospital, director of infusion services at NYU Langone Orthopedic Hospital, and associate director of clinical affairs, rheumatology, received the NIH RO1 grant, "Translation of Hip Microarchitectural Assessment Technology to the Clinic to Diagnose Glucocorticoid-Induced Osteoporosis."

Julia Manasson, MD, research fellow, Department of Medicine, received a pilot award from the Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA), "The Effect of Biologic Therapies on the Gut Microbial and Fungal Composition in Psoriatic Arthritis." She also received the National Psoriasis Foundation Early Career Grant, "The Role of the Cutaneous Microbiota in Psoriasis and Psoriatic Arthritis."

**Paolo G. Mignatti, MD,** associate professor of medicine, received an NIH R21 grant for "The Role of MT1-MMP Proteolytic Activity in Osteogenesis." Adam Mor, MD, PhD, Saul J. Farber Assistant Professor of Medicine and assistant professor of pathology, received a grant from NTB Pharma, "Targeting novel checkpoint inhibitors for cancer immunotherapies."

**Sabina Sandigursky, MD**, research fellow, received the NYU School of Medicine Physician Scientist Training Program Grant.

Jose U. Scher, MD, assistant professor of medicine, director of the Arthritis Clinic at NYU Langone Orthopedic Hospital, director of the Microbiome Center for Rheumatology and Autoimmunity-MiCRA, and director of the Psoriatic Arthritis Center, received the NIH R03 grant, "Human Gut Microbiome as Modulator of Drug Response in Inflammatory Arthritis." He also received the NIH R01 sub-award, "Refining Outcome Measurement in Psoriatic Arthritis: Preparation for Pragmatic Trials," with Soumya M. Reddy, MD, assistant professor of medicine, and Alexis R. Ogdie-Beatty, MD, MSCE, assistant professor of medicine at the Hospital of the University of Pennsylvania, director of the Penn Psoriatic Arthritis Clinic, and senior scholar, Penn Center for Clinical Epidemiology and Biostatistics. He also received a grant for the CHOICE substudy/ industry "Effects of IL-17 Blockade on Gut Microbiota.'

### Leadership

#### **DIVISION OF RHEUMATOLOGY**

#### Jill P. Buyon, MD

Lady Va and Sir Deryck Maughan Professor of Rheumatology Director, Division of Rheumatology Director, Lupus Center jill.buyon@nyumc.org

#### H. Michael Belmont, MD

Professor of Medicine Medical Director, NYU Langone Orthopedic Hospital Associate Director, Clinical Affairs, Division of Rheumatology michael.belmont@nyumc.org

#### James P. Darrow

Administrator, Division of Rheumatology james.darrow@nyumc.org

#### Timothy B. Niewold, MD

Director, Judith and Stewart Colton Center for Autoimmunity timothy.niewold@nyumc.org

#### Michael H. Pillinger, MD

Professor of Medicine and Biochemistry and Molecular Pharmacology *Program Director, Rheumatology Fellowship Program* michael.pillinger@nyumc.org

#### Ann Rupel

Grants Manager, Division of Rheumatology ann.rupel@nyumc.org

#### Ranit Shriky

Administrative Director, Seligman Center for Advanced Therapeutics Director of Clinical Trials, Division of Rheumatology ranit.shriky@nyumc.org

#### **DEPARTMENT OF MEDICINE**

#### Steven B. Abramson, MD

Frederick H. King Professor of Internal Medicine Chair, Department of Medicine Senior Vice President and Vice Dean for Education, Faculty, and Academic Affairs

#### MUSCULOSKELETAL

#### David A. Dibner, MPH, FACHE Senior Vice President, Operations and Musculoskeletal Strategic Area, NYU Langone Orthopedic Hospital

### Leadership

#### **NEW YORK UNIVERSITY**

William R. Berkley Chair, Board of Trustees Andrew Hamilton, PhD President

#### **NYU LANGONE**

Kenneth G. Langone Chair, Board of Trustees

Robert I. Grossman, MD Saul J. Farber Dean and Chief Executive Officer

Steven B. Abramson, MD Senior Vice President and Vice Dean for Education, Faculty, and Academic Affairs

Dafna Bar-Sagi, PhD Senior Vice President and Vice Dean for Science, Chief Scientific Officer

Andrew W. Brotman, MD Senior Vice President and Vice Dean for Clinical Affairs and Strategy,

Chief Clinical Officer

Michael T. Burke Senior Vice President and Vice Dean, Corporate Chief Financial Officer

Richard Donoghue Senior Vice President for Strategy, Planning, and Business Development

Annette Johnson, JD, PhD Senior Vice President and Vice Dean, General Counsel

**Grace Y. Ko** Senior Vice President for Development and Alumni Affairs

Kathy Lewis Senior Vice President for Communications and Marketing Joseph Lhota Senior Vice President and Vice Dean, Chief of Staff

Vicki Match Suna, AIA Senior Vice President and Vice Dean for Real Estate Development and Facilities

Nader Mherabi Senior Vice President and Vice Dean, Chief Information Officer

Robert A. Press, MD, PhD Senior Vice President and Vice Dean, Chief of Hospital Operations

#### Nancy Sanchez

Senior Vice President and Vice Dean for Human Resources and Organizational Development and Learning

#### **NYU LANGONE BY THE NUMBERS\***

**1,519** Beds

**98** Operating Rooms

**3,633** Physicians

5.087

Original

Papers

Research

**5,104** Nurses

549,707 Square Feet of Research Space

;

**516** MD Candidates

172,072

Emergency

**Room Visits** 

\$359M

Funding

NIH

MD/PhD s Candidates

> **\$364M** Total Grant Revenue

68,884

Discharges

Patient

85

**4,500,000** Outpatient Faculty Practice Visits

263

Candidates

PhD

**418** Postdoctoral Fellows

9,654

Births

1,327

Residents and Fellows

\*Numbers represent FY17 (Sept 2016–Aug 2017) and include NYU Langone Hospital—Brooklyn

Produced by: Office of Communications and Marketing, NYU Langone Health Writer: Bryn Nelson Creative Direction: Ideas On Purpose, www.ideasonpurpose.com Design: Craig Williams, www.thecraigwilliams.com Primary Photographer: Karsten Moran Printing: Allied Printing Services, Inc.



NYU LANGONE HEALTH 550 First Avenue, New York, NY 10016 NYULANGONE.ORG