

Translating

RESEARCH TO
IMPROVED CARE

78%

INCREASE IN
GRANT FUNDING

Advanced

TRAINING FOR
RHEUMATOLOGISTS

Three

NEW NIH
AWARDS

Top Ten

IN *U.S. NEWS &
WORLD REPORT*



Rheumatology

2015

YEAR IN REVIEW

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Dear Colleagues and Friends:

As a clinician and researcher, identifying which signposts of health and disease are the most critical to predict, diagnose, and treat autoimmune and rheumatic conditions is the ultimate aim.

Indeed, pulling out the most informative markers from a sea of information is a truly daunting task that requires a strong commitment to support fundamental research and innovation, build a world-class infrastructure, and facilitate multidisciplinary collaborations. Our vision for the division of Rheumatology is to step up to these challenges.

I'm proud to report that the division has harnessed our strengths in all three areas to extend our understanding of the molecular pathways that underlie autoimmunity and inflammation in patients of all ages. In 2015, we continued to gain ground in translating significant research findings into transformative clinical care.

Through local, regional, national, and international collaborations, our investigators are playing key roles in multiple efforts to improve prediction, early diagnosis, and treatment options. These efforts, in turn, are paying off in fundamental ways that have real-world benefits for the patients we serve. By carefully quantifying the risk of pregnancy among expectant mothers with lupus, for example, results from a large NIH-funded, multi-center study supported the conclusion that such pregnancies are safer than previously thought. Moreover, very specific risk factors were identified to predict which patients are more likely to experience adverse outcomes. These results will significantly improve the management of our many lupus patients who desire pregnancy.

With the considerable backing of the Stewart and Judith Colton Center for Autoimmunity, established in 2014, we're also building upon the Medical Center's solid foundation of microbiome research. Seminal work by researchers in the division, for instance, is beginning to tie certain species of the gut microbiome to the disease processes underlying rheumatoid arthritis, lupus, and psoriatic arthritis—a critical step toward more targeted approaches to disrupting major inflammatory and pathogenic pathways.

Thanks to the success of our SLE SAMPLE (Specimen and Matched Phenotype Linked Evaluation) Registry and Biorepository, we're attracting nationally recognized basic researchers to help translate fundamental observations in mouse models into more personalized therapeutic approaches for patients.

In collaboration with bariatric surgeons, we're revealing new insights into the association between weight loss and the alleviation of psoriasis, psoriatic arthritis, osteoarthritis, and gout-related pain—findings that could have a considerable impact on patients' quality of life. We're characterizing potential new biomarkers of knee osteoarthritis, and are developing new tools to detect skeletal fragility, predict fracture risk, and visualize the early stages of gout.

Finally, we've built upon the early success of the multi-center Accelerating Medicines Partnership in Rheumatoid Arthritis and Lupus Network to speed the development of skin-based biomarkers to assess the severity of lupus nephritis and to better predict therapeutic responses. In addition, we are discovering signatures in single renal cells that may reveal new pathways for treatment of lupus nephritis. We're also helping to create a national network to share clinical and research data to enhance the development of new approaches.

Forging a new trail is never easy, but I'm excited to see how a spirit of determination, resourcefulness, and collaboration has given us added momentum toward our lofty goal of fully integrated and personalized medicine.



JILL P. BUYON, MD

Lady Va and Sir Deryck Maughan
Professor of Rheumatology

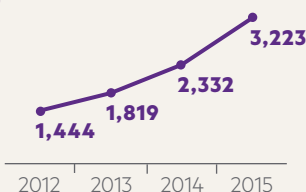
Director, Division of Rheumatology,
Department of Medicine

Rheumatology

CLINICAL VOLUME

3,223
patients

seen in an
average month



500+
psoriatic arthritis
patients

seen at the new, multidisciplinary
Psoriatic Arthritis Center which officially
opened its doors in 2015

Top Ten

for Rheumatology
in *U.S. News & World
Report's* 2015–16
Best Hospitals



FACULTY AND FELLOWS

100
faculty members

24 full time
76 part time

9
ACR Distinguished
Fellow Awards

in the past 14 years, including a
2015 award for Ashira Blazer, MD

2
new fellowship
positions

growing the program to a total of 8

RESEARCH AND FUNDING

78%
increase in total
active grant funding

compared to 2014

60
accepted ACR abstracts

including 35 posters, 11 oral
presentations, and one plenary session

59
publications

seven with an impact factor over 10

New NIH awards

JILL P. BUYON, MD

NIH R01 for her project on Preventive
Approach to Congenital Heart Block (PATCH)

ROBERT CLANCY, PHD

Leads a consortium on a NIH R01 with Mayo
Clinic to study Interferon Regulatory Factor 5 in
Human Lupus Pathogenesis

JOHANNES NOWATZKY, MD

Recipient of NIH K08 award for his project
on harnessing monoclonal Tregs for the
treatment of autoimmune uveitis

100
seminar attendees

at inaugural Psoriatic Arthritis CME course

**Other notable
grant funding**

SHAHLA ABDOLLAHI, PHD

Arthritis National Research Foundation
award to support her work on immunologic
mechanisms involving the microbiome in RA.

ASHIRA BLAZER, MD

Rheumatology Research Foundation
Development award to support her project on
Arterial Dysfunction Related to IFN in Carriers
of APOL1 (AFRICA)

SVETLANA KRASNOKUTSKY SAMUELS, MD

Rheumatology Research Foundation
Investigator Award to support her work on
colchicine for the treatment of osteoarthritis

ADAM MOR, MD, PHD

Rheumatology Research Foundation Disease
Targeted Innovative Research Grant for his
project on PD-1 in inflammatory arthritis

NYU Langone Medical Center



#1

overall patient safety
& quality for
three years in a row

AND

ambulatory care quality
& accountability

among leading academic medical centers
across the nation that were included in the
University HealthSystem Consortium 2015
Quality and Accountability Study

Top 15

in U.S. News & World Report



#12

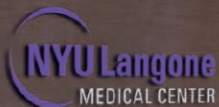
**BEST HOSPITALS
HONOR ROLL**



#14

**BEST MEDICAL
SCHOOLS FOR
RESEARCH**

and nationally ranked in 12 specialties,
including top 10 rankings in
Orthopedics (#5), Geriatrics (#6),
Neurology & Neurosurgery (#9),
Rheumatology (#9),
and Rehabilitation (#10)



**Tisch
Hospital**

The University Hospital
of NYU

BUILDING ON EXISTING PROGRAMS AND FORGING NEW GROUND



▲ Stephen Honig, MD

Defining Risk and Improving Care Among Osteoporosis Patients

NYU Langone continues to forge new ground in efforts to detect the risk of fragility fractures in osteoarthritis patients and to reduce subsequent fractures. In one recent study, Stephen Honig, MD, clinical associate professor and director of the Osteoporosis Center, teamed up with Gregory Chang, MD, associate professor of radiology, and other researchers. Together, the collaborators found that combining high-spatial-resolution 3T MR imaging of a patient's proximal femur with finite element analysis may hold promise as a supplemental tool for detecting skeletal fragility and assessing fracture risk.

For a second recent publication, divisional researchers teamed up with the Department of Orthopaedic Surgery to introduce the NYU Langone Osteoporosis Model of Care. The patient and physician education program aims to identify women at higher risk for recurrent fractures and to reduce those fracture rates. After implementing the program at NYU Langone, a team led by Dr. Honig and Amit Saxena, MD, assistant professor of medicine, found that it improved comprehensive fracture care. The researchers caution, however, that achieving long-term success in reducing subsequent fracture rates will require sustained efforts to enroll and educate a sufficient number of patients.

AMP: A New Foundation for Expanding Networks and Sharing Data

Building on NYU Langone's successful participation in the Accelerating Medicines Partnership in Rheumatoid Arthritis and Lupus (AMP RA/Lupus) consortium, divisional researchers are playing principal roles in expanding the multicenter collaboration.

Among the efforts, one is creating a national network that shares clinical and research data to support the goal of speeding the development of new treatments. "We're not just sharing data, we're sharing primary data, which really means that if we generate a sequencing file, we're putting it on a platform to let everyone look at it," says Robert Clancy, PhD, associate professor of medicine. The data will be vetted first by the AMP researchers and then uploaded via the NIH's Immunology Database and Analysis Portal (ImmPORT) to give other researchers unprecedented access.

Building on the NYU Langone-led discovery that skin biomarkers hold promise to reflect the intra-renal disease state of lupus, the division is also partnering with fellow AMP RA/Lupus awardee Betty Diamond, MD, at the Feinstein Institute for Medical Research in Manhasset, New York. The biomarker discovery goal of their joint proposal to the Immune Tolerance Network is to determine whether research can facilitate early disease identification and treatment, which is critical to renal survival. The effort could lead to a simpler design for clinical trials investigating immune status and treatment efficacy.

Such collaborations, Dr. Clancy says, could yield big dividends in identifying new therapies and understanding the molecular underpinnings of disease.

Recruiting Regulatory T Cells to Fight Behçet's-Linked Uveitis

Patients with Behçet's disease commonly suffer from painful uveitis. With the backing of a five-year, \$907,000 grant from the NIH's National Eye Institute, Johannes Nowatzky, MD, assistant professor of medicine, is developing a T cell-based immunotherapy to treat the eye inflammation.

"I'm working primarily with human cells and trying to manipulate them in such a way that once they're re-infused into humans they can be used as therapy," Dr. Nowatzky says. His ex-vivo approach involves extracting a subset of immunosuppressive regulatory T cells from patients, massively expanding and modulating them in vitro, and then reintroducing them to fight the autoimmune-linked inflammation. He and his team have already isolated a functionally stable population of therapeutic cells and are testing the cells' safety and efficacy in a mouse model.

If the researchers can tailor the strategy to specific inflammation-promoting antigens, Dr. Nowatzky says the proof-of-principle work also could be translated to other autoimmune diseases. One major advantage of the cell-based immunotherapy approach, he adds, is the ability to exploit a biologically active cell's entire machinery. "If this works out," he says, "this therapy is probably more rapidly translatable than many of the molecular targets."

Assessing Long-Term Cardiac Neonatal Lupus Outcomes

There has been minimal substantive data on the long-term cardiac health of children born with neonatal lupus. In a new study, NYU Langone researchers are examining ECGs, heart size, left ventricular systolic function, and other factors to help identify divergent outcomes among affected children.

Among the findings so far: Left ventricular systolic function is abnormal in about 13 percent of patients and low normal in 9 percent. Intriguingly, most of these patients have grouped into two distinct age brackets: newborns and adults. Reduced function in the first group may be due to inflammation in utero that continues after birth. "The other peak, after age 20, might be associated with long-term pacemaker use, or something intrinsic about the disease that remains an issue as the patients age," says Amit Saxena, MD, assistant professor of medicine.

The study has revealed other findings as well. Dilated cardiomyopathy and lower heart rates in utero are associated with decreased heart functioning after birth.



▲ Johannes Nowatzky, MD

Probing the Microbial Underpinnings of Psoriatic Arthritis

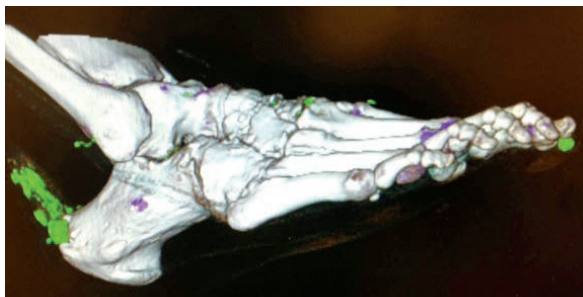
New research is shining a light on the functional relevance of the intestinal microbiome in patients with psoriatic arthritis. Shahla Abdollahi-Roodsaz, PhD, research assistant professor of medicine, and colleagues are collaborating with Sergei Koralov, PhD, assistant professor of pathology, to study the role of gut microbiota using a new "knock in" mouse model of psoriatic arthritis-like disease. The mouse model includes an overactive version of the STAT3 transcription factor, which drives the differentiation of T helper 17 cells and chronic inflammation. Together with Jose Scher, MD, assistant professor of medicine and director of NYU Langone's Psoriatic Arthritis Center, the team is examining the role of the microbiome in the resulting disease phenotype mediated by a hyperactive immune system.

"By probing the causal relationship between microbiota and disease pathogenesis and progression we hope to identify novel targets for therapeutic intervention," says Dr. Koralov. "In the future, this research may inform our choice of antibiotic or probiotic therapy as a means to regulate the hyperactive immune responses that characterize this inflammatory disease."

Supporting Patients Through Wellness Programs and Events

In addition to strong clinical care that involves the latest research-driven treatments, NYU Langone's Rheumatology Division also sought to support patients with numerous programs and events in 2015. These include:

- Monthly classes for osteoporosis patients in English and Spanish, which focus on managing symptoms and instruction on injection of Forteo
- Participation in community lupus events, including the Walk Along for Lupus
- Monthly Lupus Foundation of America support group meetings
- A community lecture for psoriatic arthritis patients



▲ Dual-energy CT illustrates urate deposition in a gout patient's foot. The green color indicates urate at the toe, in the midfoot, and along the Achilles tendon. (Purple indicates calcium deposition.)

Visualizing Gout and Addressing Its Comorbidities

In gout, an excess of serum urate can crystallize in joints and soft tissue structures, leading to acute and chronic inflammation. Tophi, particularly large and stone-like aggregates of urate, can permanently destroy joints. To prevent joints from being damaged, NYU Langone researchers have begun a public-private partnership that is employing dual-energy CT technology to highlight crystallized uric acid deposits in the foot joints of gout patients.

The scans, in which the deposits take on a bright green hue, are helping Michael Pillinger, MD, professor of medicine and of biochemistry and molecular pharmacology; Svetlana Krasnokutsky Samuels, MD, assistant professor of medicine; and their colleagues visualize the impact of various therapeutic options. "We're going to be looking at different gout treatment strategies for

lowering uric acid, how they affect even sub-clinical deposition of urate, and what might be better at lowering the uric acid burden," Dr. Krasnokutsky Samuels says.

Working with Soterios Gyftopoulos, MD, assistant professor of radiology, the group has already used dual-energy CT to show that the intravenous drug pegloticase can rapidly lower serum urate levels and reduce tissue urate deposition. Follow-up comparative effectiveness studies will gauge the drug's benefits against those of existing oral medications.

The study is being conducted under the auspices of NYU Langone's Crystal Diseases Study Group (CDSG), co-directed by Dr. Pillinger and Dr. Krasnokutsky Samuels. The group is also focusing on the causal relationships between gout and its comorbidities, and on whether proper gout management can reduce adverse events associated with these comorbidities.

In another line of research, the group has joined forces with Bruce Cronstein, MD, the Dr. Paul R. Esserman Professor of Medicine, as well as Dr. Stuart Katz, Helen L. and Martin S. Kimmel Professor of Advanced Cardiac Therapeutics, and other members of the Leon H. Charney Division of Cardiology, to form TRIAD (Translational Research in Inflammation and Atherosclerotic Disease). Supported by a \$1.2 million New York State Empire Clinical Investigator Award, the group's initial trans-disciplinary research suggests that the anti-inflammatory gout medication colchicine may significantly reduce the heart attack risk among gout patients. Two larger cohort studies are now underway to confirm these findings. With support from the Rheumatology Research Foundation, the group is also assessing whether colchicine might improve osteoarthritis symptoms and progression—another step toward boosting the overall quality of patient care.

NYU LANGONE MEDICAL CENTER NEWS

Groundbreaking Face Transplant Exemplifies Expertise and Multidisciplinary Collaboration

In August 2015, surgeons at NYU Langone Medical Center performed the most complex face transplant to date. The patient, former firefighter Patrick Hardison, had lost all of the skin around his entire face, scalp and neck, including his eyelids, ears, lips, and nose, while trapped in a burning building. Led by Eduardo Rodriguez, MD, DDS, the Helen L. Kimmel Professor of Reconstructive Plastic Surgery and chair of the Hansjörg Wyss Department of Plastic Surgery, the successful 26-hour operation—the first to include transplantation of eyelids capable of blinking as well as functional ears, among other milestones—involved more than 100 physicians, nurses, and technical and support staff. More than a dozen departments contributed to the planning and execution of the procedure, or to postoperative care.



▲ Svetlana Krasnokutsky Samuels, MD, and Michael Pillinger, MD

Fellowship Class

INCOMING FELLOWS

- **Benjamin Friedman**
Residency: Mount Sinai
- **Julie Nusbaum**
Residency: New York
Presbyterian Weill Cornell
- **Shudan Wang**
Residency: New York
Presbyterian Weill Cornell
- **Michael Toprover**
Residency: NYU School of Medicine

FIRST YEAR FELLOWS

- **Nicola Berman**
Residency: University of
Pennsylvania Hospital
- **Vinicius Domingues**
Residency: New York
Presbyterian Weill Cornell
- **Julia Manasson**
Residency: NYU School of Medicine
- **Anna Zazon**
Medical Fellowship: NYU School
of Medicine (Geriatrics);
Residency: Albert Einstein College of
Medicine, Montefiore Medical Center

SECOND YEAR FELLOWS

- **Adey Berhanu, Chief Fellow**
Residency: Loyola University Chicago
- **Aaron Garza Romero**
Residency: University of Miami,
Miller School of Medicine
- **Sabina Sandigursky**
Residency: Albert Einstein College of
Medicine, Montefiore Medical Center

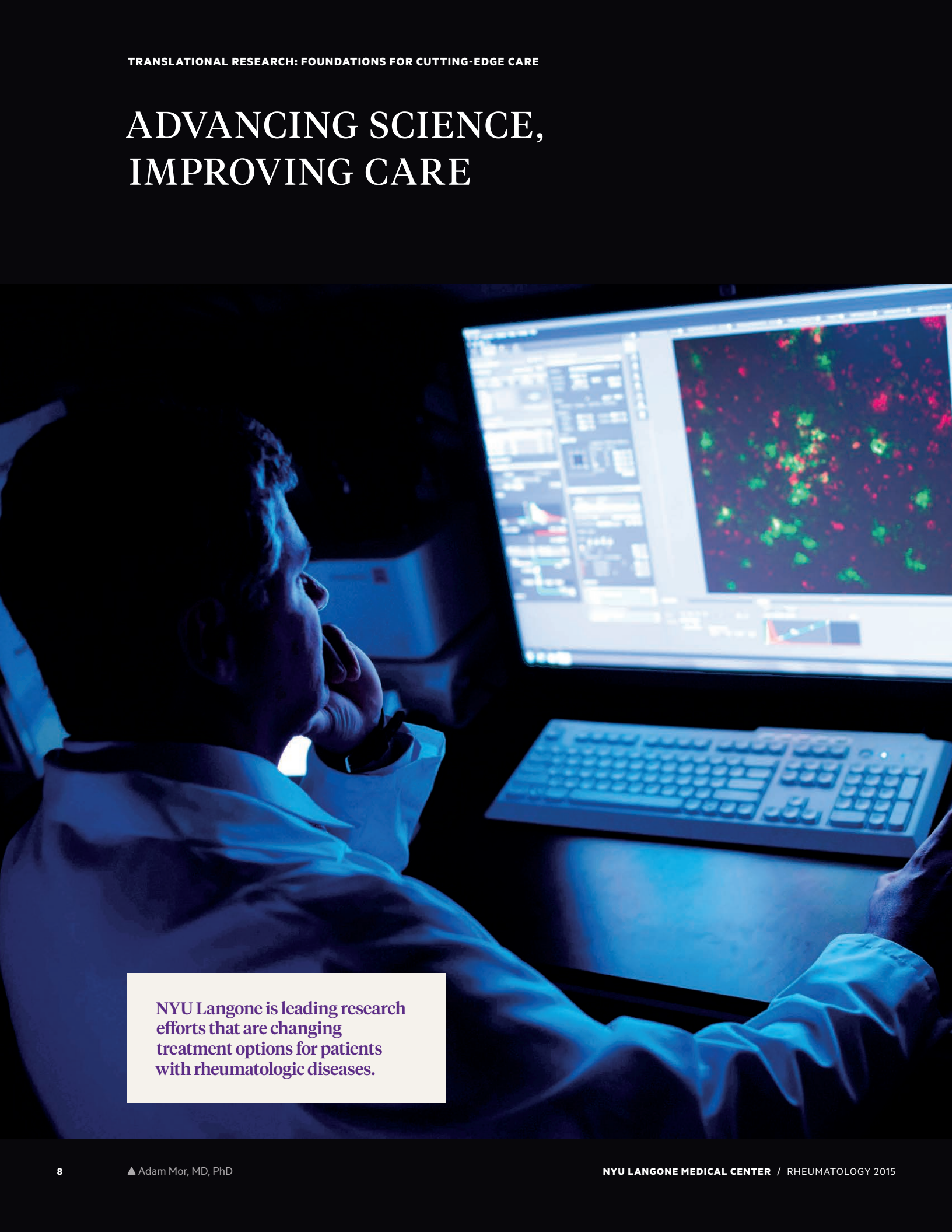
2016 CME Courses

- **7th Annual Pediatric Rheumatology:
An Update for Clinicians**
March 16
- **Seminar in Advanced Rheumatology**
March 17–19
- **Musculoskeletal Ultrasound for Rheumatologists:
Beginner and Intermediate Levels**
March 19–20

- **2nd Annual NYU Langone Advanced Seminar
in Psoriasis and Psoriatic Arthritis**
September 23
- **Boning Up on Osteoporosis**
October 26

For more information, visit nyulmc.org/cme

ADVANCING SCIENCE, IMPROVING CARE



NYU Langone is leading research efforts that are changing treatment options for patients with rheumatologic diseases.

Delivering Safer Pregnancies for Women with Lupus

PREGNANCY AND AUTOIMMUNITY

Expectant mothers with lupus face an array of potential adverse pregnancy outcomes, including fetal or neonatal death, delivery before 36 weeks, a small-for-gestational-age neonate, and preeclampsia. Mothers with anti-Ro antibodies also may give birth to children with cardiac and/or cutaneous disease referred to as neonatal lupus. Several recent studies by NYU Langone researchers, however, have identified multiple biomarkers that may help predict and manage the associated risks and improve outcomes.

Among the advances, Jill P. Buyon, MD, Lady Va and Sir Deryck Maughan Professor of Rheumatology, director of the Division of Rheumatology, and director of NYU Langone's Lupus Center, first authored a multi-center study concluding that pregnancy for women with lupus is safer than previously thought. The National Institutes of Health-funded study of 385 expectant mothers found that 81 percent delivered full-term, normal-weight, and healthy babies.

more than 4 in 5

pregnant women with lupus delivered full-term, normal-weight, and healthy babies in a recent study led by Dr. Buyon



▲ Jill P. Buyon, MD

The study, part of the Predictors of Pregnancy Outcome: Biomarkers in Antiphospholipid Antibody Syndrome and Systemic Lupus Erythematosus (PROMISSE) multi-center collaboration, under the directorship of Jane Salmon, MD, the Collette Kean Research Professor at Hospital for Special Surgery, also identified multiple risk factors for adverse outcomes. Beyond the presence of active disease and flare-ups during pregnancy, Dr. Buyon and her colleagues linked a positive lupus anticoagulant test, antihypertensive use, absence of a rise of complement 3, and low platelet count with adverse pregnancy outcomes.

For pregnant women with inactive or stable mild to moderate SLE and none of the identified risk factors, the study found infrequent flares and highly favorable outcomes. "Our new study is quite reassuring since in the majority of cases, both mother and baby can do well if lupus is under control at conception," Dr. Buyon says.

NEW MARKERS FOR CARDIAC NEONATAL LUPUS

Related studies are identifying new biomarkers that may help doctors assess the severity of the major cardiac manifestation of neonatal lupus, which includes congenital heart block and cardiomyopathy. Mothers with one newborn afflicted by the condition have a significantly higher risk of having another baby with the same condition.

Amit Saxena, MD, assistant professor of medicine, says complete heart block is irreversible, meaning that associated markers may not initially aid in prevention. "With biomarkers, we can try to gauge the severity of the disease in order to treat neonates and children more effectively, and try to find therapeutic targets that we might be able to use to prevent the disease in the future with novel drugs," he says.

Although exposure to maternal anti-Ro antibodies is required for heart block, only 2 percent of those exposed in utero ultimately get the condition. Dr. Saxena says the NYU Langone-led National Research Registry for Neonatal Lupus, which includes children with a myriad of manifestations of neonatal lupus such as cardiac disease, rash, liver abnormalities and cytopenias, their unaffected siblings, and their parents, has allowed the team to test whether other specific biomarkers might better predict disease onset and disease severity.

PREGNANCY AND AUTOIMMUNITY

The researchers found that levels of C-reactive protein, or CRP, in cord blood may provide one such marker. Patients with cardiac neonatal lupus had elevated levels of the protein, whereas their unaffected siblings did not. The finding showed for the first time that exposure to a maternal antibody can induce a fetal inflammatory response. Further efforts suggested that CRP is associated with both the development and severity of disease. “Now we have a biomarker that’s easily used because it’s so common, and we can track it in children to get a sense of how much inflammation is going on and how aggressively we may need to treat these patients after birth,” Dr. Saxena says.

The collaborators also found elevated levels of the N-terminal of the prohormone brain natriuretic peptide in affected children, suggesting its potential as a diagnostic during amniocentesis. Similarly, the research found a positive association between neonatal heart block and elevated levels of cardiac fibrosis markers, including matrix metalloproteinase-2, and the urokinase plasminogen activator, its receptor and plasminogen proteins. An assessment of vitamin D yielded less clear results, though the data hinted at a link between higher levels in pregnant mothers and ultimately less severe symptoms in their affected children. If that link is borne out, the researchers say, optimizing maternal vitamin D levels might aid the management of anti-Ro positive pregnancies.

The National Research Registry
for Neonatal Lupus, launched initially by the NIH
and maintained at NYU Langone, includes

503 families
with
627 affected children

▼ Amit Saxena, MD



Assessing Heart Block Severity and Prevention

NYU Langone researchers have created a severity score for cardiac neonatal lupus based on known risk factors for mortality, such as lower fetal heart rates and disease beyond the atrioventricular node. The score also makes use of recent work suggesting associations between severity and elevated levels of C-reactive protein and the N-terminal of the prohormone brain natriuretic peptide. In addition, Dr. Saxena and colleagues found that mothers treated with the drug hydroxychloroquine during pregnancy were less likely to give birth to a child with heart disease than those not taking the drug. The research suggested that treating mothers with fluorinated steroids during a cardiac neonatal lupus-affected pregnancy had no effect on the severity of the newborn's condition.

In collaboration with NYU Langone pediatric cardiologists and the Division of Maternal-Fetal Medicine, Dr. Buyon, Peter Izmirlly, MD, assistant professor of medicine, and other researchers are now assessing mothers of children with congenital heart block in a clinical trial to test whether hydroxychloroquine might help reduce the recurrence rate in subsequent pregnancies.

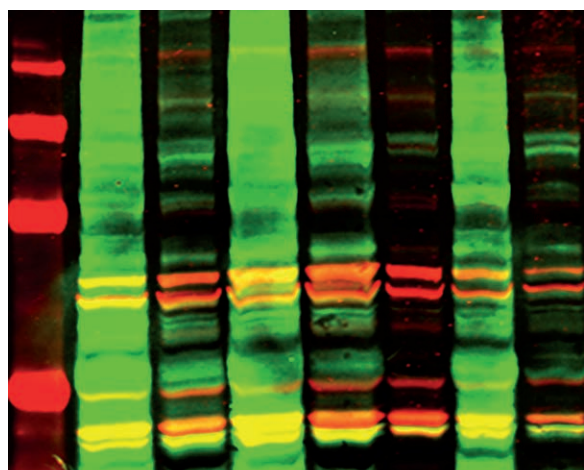
Unmasking the PD-1 Pathway to Inhibit Migration and Adhesion of Autoimmune T Cells

ARTHRITIS AND LUPUS

Most therapies for arthritis and lupus address inflammation by either inhibiting the functions or sharply reducing the number of lymphocytes. This approach may dampen autoimmunity, but a major side effect is an increased risk of infections and cancer. “You’re getting rid of the beneficial effect of those lymphocytes,” says Adam Mor, MD, PhD, assistant professor of medicine and pathology at NYU Langone.

Dr. Mor and colleagues have found a potential way around this catch-22 through an unusual lymphocyte co-receptor called Programmed Death-1, or PD-1. Unlike most other receptors, PD-1 inhibits lymphocyte activity when it binds its ligands. “If you think about it, this is the ideal way to treat inflammation without getting rid of the cell,” he says. “You basically control it: find a way to manipulate the receptor and you turn inflammation off. You’re using the body’s own system to treat autoimmunity.”

To control inflammation, however, Dr. Mor knew that researchers must first understand how PD-1 works at a molecular level. Previous studies had suggested that the inhibitory co-receptor PD-1 blocks T cell proliferation and the secretion of cytokines. For the first time, Dr. Mor and his team found that PD-1 also regulates cell adhesion and migration. Their research suggests that the co-receptor PD-1 works in part by inhibiting an enzyme called Rap1, which is crucial for T cell movement and adhesion.



▲ Western blot of phosphorylated proteins of T cells stimulated via the PD-1 receptor

EXPLORING INHIBITORY PATHWAY'S DOWNSTREAM DRUG TARGETS

Instead of targeting the receptor directly—a blunt-force approach that would be hard to balance—Dr. Mor’s lab is investigating the downstream events within the cell once PD-1 has been engaged. Clinicians could selectively disable downstream molecules to prevent certain types of adhesion or migration while retaining the T cells. One blocked pathway, for example, might restrain the inflammation-promoting T cells from migrating to a patient’s joints.

The team is using biochemical assays to quantify proteins in the cells under varying conditions and confocal laser microscopy to track the location of specific molecules in the cells before and after stimulation of the PD-1 receptor. So far, the approach has yielded multiple potential drug targets that are in the process of being patented. A recent expansion of the work has also begun to tie downstream events in migration and adhesion, along with the main agents of activation or deactivation, back to the PD-1 pathway.

In a related line of research, the group is asking whether researchers can manipulate a lupus patient’s T cells in the lab by changing the cellular function through PD-1 receptor manipulation. In collaboration with Jill P. Buyon, MD, the Lady Va and Sir Deryck Maughan Professor of Rheumatology and director of the Division of Rheumatology and the NYU Langone Lupus Center, Dr. Mor and colleagues are now seeking to validate their biochemical results in lymphocytes isolated from the blood of patients with lupus.

“For example, are the proteins that we found under the microscope really playing a role in cells isolated from patients with lupus? Then we try to inhibit those cells in a dish to see if it’s going to be an approach to treat patients with lupus,” Dr. Mor says. If successful, the research could pave the way for clinical trials of effective but gentler forms of autoimmune therapy that strike the right balance.

Confronting Health Disparities in SLE-Linked Renal and Cardiovascular Disease

LUPUS

While roughly nine in ten American lupus patients live a decade or more, survival rates in Ghana and much of sub-Saharan Africa remain unacceptably low. Ashira Blazer, MD, instructor of medicine, is taking on this stark disparity with groundbreaking field and lab research on the genetic underpinnings of divergent SLE outcomes. “I am specifically interested in health disparities in lupus, particularly why it is that people of African ancestry have worse outcomes than other people,” Dr. Blazer says.

An estimated 30 percent of African Americans possess a variant form of the APOL1 gene encoding apolipoprotein L1, a cytokine-mediated apoptosis factor. In its heterozygous form, the variant’s positive selection has been driven by its ability to confer resistance to African trypanosomiasis. In its homozygous form, however, the variant significantly increases the risk of renal and cardiovascular disease, and has been linked to a 4 percent lifetime chance of kidney failure.

Patients with SLE already have a higher risk of cardiovascular and renal disease, and the danger increases in patients of African ancestry. Some clues underlying this elevated risk burden have emerged from genetic analyses: Dr. Blazer and colleagues recently found that African American SLE patients who carry at least one APOL1 risk allele are at increased risk for hypertension before the onset of clinically significant renal disease. Research at the bench has linked chronic immune activation—a hallmark of SLE—to APOL1 overexpression.

An estimated
30 percent
of African Americans are heterozygous
for an *APOL1* risk allele, while

12 percent
are homozygous

Researchers have linked the
homozygous form to a

4 percent
lifetime chance of kidney failure
in these individuals

“Our hypothesis is that in the presence of the lupus inflammatory milieu, there is overexpression of the variant APOL1 gene. That increase in gene product would then lead to cardiovascular and renal damage,” she says. “Even in heterozygous patients with the variant, you might see a signal for increased risk.”

COLLABORATION WITH GHANA HOSPITAL TO AID GENETIC RESEARCH, IMPROVE DIAGNOSIS

To delve into the potential impacts of lupus-mediated gene overexpression in patients of African ancestry, her research is tapping into the translational efforts launched in both Ghana and New York. As part of NYU School of Medicine’s International Health Program, Dr. Blazer and study coordinator Janet Nwaukoni recently traveled to Accra, Ghana, to collaborate with Dr. Bgifa Dey, who runs a rheumatology clinic at the NYU Langone-affiliated Korle Bu Teaching Hospital.

Initially, the study is aiming to collect plasma serum, urine, and saliva from 100 patients to evaluate autoantibodies, cytokines, and extract DNA for APOL1 genotyping. “That way, we can make some clinical associations with the gene and then we can learn a lot more about lupus in Africa,” Dr. Blazer says. “Now it’s just sort of a black box, so it’s very exciting.”

The collaboration is also working to improve Ghana’s availability of antinuclear antibody testing, which is currently obtained only by shipping samples to South Africa at the patient’s own expense. Greater access to testing, she says, could lead to early diagnosis and improved patient outcomes.

The project has also made good use of innovative resources at NYU Langone. The division’s SAMPLE biorepository has helped Dr. Blazer and colleagues amass clinical samples from more than 100 lupus patients of African ancestry. In addition, a partnership with NYU Langone’s Shilpi S. Mehta-Lee, MD, assistant professor of obstetrics and gynecology, has helped the researchers develop a novel way to obtain endothelial cell samples. With the consent of African American parents, the collaborators are collecting the umbilical cords of newborns, from which they can extract endothelial cells.

“We are growing those endothelial cells in culture and subjecting them to an inflammatory milieu and at the same time genotyping the cells to determine if there are any differences across the APOL1 genotype,” Dr. Blazer says.

The culture method yields cells with none, one, or two risk variants, allowing the researchers to see how cells might respond to or be damaged by lupus-related stimuli. Already, the technique has shown that under the inflammatory conditions of lupus, APOL1 mRNA increases ninefold—yet another step toward deciphering and overcoming the potential contributors to SLE.

ASHIRA BLAZER, MD

Named a 2015 Distinguished Fellow Award recipient by the ACR, Dr. Blazer received a Scientist Development Award from the Rheumatology Research Foundation that helped fund her travel to Ghana to set up research infrastructure and obtain samples.



▲ Ashira Blazer, MD

A Translational Game-Changer: the SLE SAMPLE Biorepository

LUPUS

NYU Langone's SLE SAMPLE (Specimen And Matched Phenotype Linked Evaluation) Registry And Biorepository has been crucial in the search for the causes of lupus development. In fact, Boris Reizis, PhD, professor of medicine and of pathology, recently joined the NYU Langone faculty in 2015 in part because of the biorepository's existence.

Dr. Reizis plans to follow up on his 2014 study, published in the *Journal of Experimental Medicine*, which found that in two experimental mouse models, even a partial impairment of plasmacytoid dendritic cell (pDC) function lessened the severity of lupus symptoms. Researchers have long suspected that interferon-producing pDCs play a role in lupus, but until very recently there had been no direct link to a genetic cause. Dr. Reizis's studies are part of a growing body of evidence suggesting that the development of lupus may depend on the aberrant activation of pDCs.

"That paper is drawing quite a bit of attention because people can justify interest in the cell type as a potential therapeutic target," Dr. Reizis says. "We would be very interested to follow up on that in human patients to better characterize the number and the functionality of these cells."

The readily available biorepository of patient samples, he continues, has been an "absolutely amazing" bonus. "Having something like this already well established and on the ground, and all of the samples and all the clinical descriptions ready, it's just a gift."

50%

of patients in the SLE SAMPLE biorepository are diagnosed with lupus nephritis

AIMING FOR PERSONALIZED MEDICINE IN LUPUS

In addition, Dr. Reizis believes that the SLE SAMPLE biorepository could be critical in moving the field toward personalized medicine, a particular challenge given that lupus is highly variable. "Lupus is notorious for being heterogeneous," he says. "In fact, it's most likely multiple diseases masquerading as one."

With different genetic and environmental causes likely converging on a common collection of symptoms, he says, lupus demands more patient-specific approaches.

The first step is amassing a good database, records, and a biospecimen repository that offers easy access to antibodies, cells, and DNA for genotyping. "By looking specifically at the antibody profile and the key genetic polymorphisms in each patient, it could have a very immediate impact on how those patients are treated," Dr. Reizis says. "And ultimately, it could lead to better treatments."



▲ Boris Reizis, PhD

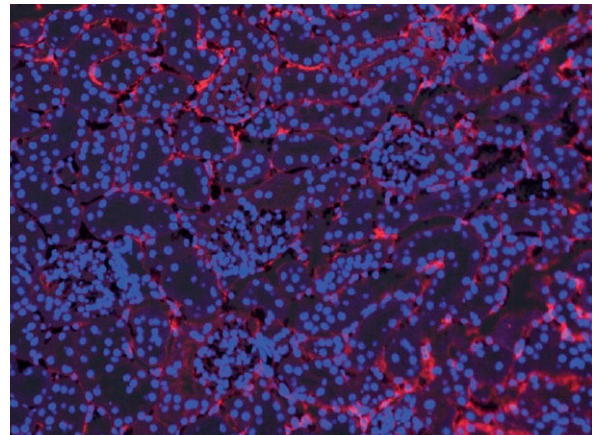
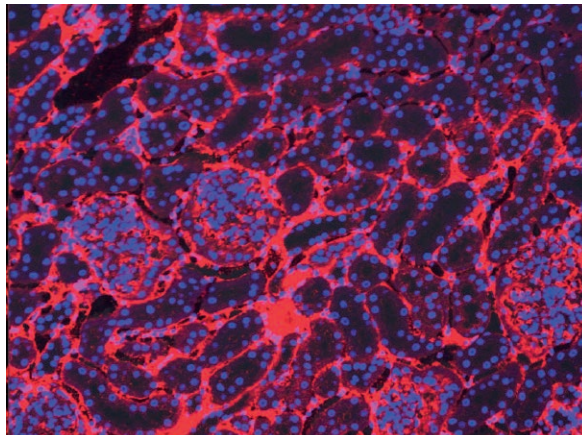
CAPITALIZING ON A CULTURE OF COLLABORATION

Dr. Reizis's lupus research has not just drawn on the strengths of the biorepository, however. It has also benefited from NYU Langone's strong culture of collaboration between researchers and clinicians. "A big draw in my coming to NYU Langone was that not only is its rheumatology department very research-savvy, but it also has a community that is eager to collaborate from the clinical side," he says. "I found it particularly appealing that people in rheumatology were speaking the language of science and were very keen on collaborating with more basic scientists."

Since Dr. Reizis joined NYU Langone, his lab has launched collaborations with several members of the

division, including Robert Clancy, PhD, associate professor of medicine, and Jill P. Buyon, MD, Lady Va and Sir Deryck Maughan Professor of Rheumatology, director of the Division of Rheumatology, and director of NYU Langone's Lupus Center. "It's a very interesting and useful experience to talk to people who see the disease firsthand. It certainly informs our research," he says.

For example, one recent project, a joint exploration of a genetic alteration that is associated with lupus, has required extensive genotyping to identify patients with the mutation. "We genotyped them and found some patients that would be of particular interest to us. We can immediately access their serum and plasma, which is exactly what we wanted to do," Dr. Reizis says.



Inhibitory Receptor May Curb Hyperactivity of Lupus-Associated Cells

Emerging evidence suggests that aberrant hyper-activation of plasmacytoid dendritic cells, or pDCs, may play an important role in lupus. That finding, reported by Dr. Reizis, begs several more questions, however. "What are the mechanisms that normally keep these cell types in check and prevent their activation? And second, what are the potential molecules to target if we want to deplete this cell type or functionally impair it?" he asks.

Follow-up research has shed new light on both questions with the recent finding by Dr. Reizis and collaborators that the PTPRS protein (receptor protein tyrosine phosphatase sigma) acts as an inhibitory receptor on human and murine pDCs—the first known receptor of its kind that works the same way in both species. "That just confirms that evolution invested heavily in this mechanism to keep the cells in check," he says. Continued research, he adds, is bolstering the inhibitory receptor's case as a prime therapeutic target.

▲ **Research by Dr. Reizis** and his NYU Langone collaborators has found that the deposition of autoantibodies (red, left panel) in the kidneys of lupus-prone mice is reduced when pDC function is impaired (right panel).

Zeroing in on the Contributors to Knee Pain

OSTEOARTHRITIS

In the ongoing battle against knee osteoarthritis, NYU Langone researchers are making headway in identifying potential treatment options and indicators of worsening severity.

In one major line of inquiry led by Jonathan Samuels, MD, assistant professor of medicine, researchers recruited patients preparing for bariatric surgery and asked whether they were experiencing knee pain. Of 536 consecutive patients considering the surgery with Christine Ren-Fielding, MD, professor of surgery and chief of the Division of Bariatric Surgery, or with fellow collaborator Manish Parikh, MD, at Columbia University Medical Center, the team identified 308 with knee pain.

Research has established a mechanical link between excess weight and potential for pain: four pounds of pressure push down on the knee for every pound of weight in the body. “But it can’t just be due to more pressure on the knee,” Dr. Samuels says. “There are inflammatory cascades coming out of the fat tissue that we think are irritating the joints and having quite an impact.” Obese patients, for example, also tend to have a higher incidence of hand osteoarthritis.

Among the 150 patients who ultimately completed bariatric surgery after enrolling in the study, Dr. Samuels and colleagues found a significant association: the more weight they lost, the less knee pain they felt. By tracking these patients over time, the researchers showed that the reduction in pain was sustained. The team also has approached the pain question from a slightly different angle by asking patients about the type and amount of medication and therapy needed to treat their knee pain. Again, they’re finding that patients who lose more weight require fewer treatments.

The combined findings, if validated in larger-scale studies, might bolster the case for bariatric surgery as an effective treatment option for some osteoarthritis patients—particularly those who are mildly obese—by sparing them from knee replacement surgery or steroid treatments. “That is the impact we are looking for: to change the way that we as musculoskeletal specialists are approaching patients with knee pain and obesity,” Dr. Samuels says. “We should be encouraging weight loss in many different ways, and some of the bariatric surgeries could make a significant impact.”



▲ Jose U. Scher, MD, and Jonathan Samuels, MD

New Osteoarthritis Trials

NYU Langone’s expertise in osteoarthritis will be put to good use in several new trials. In one, a multi-center team will compare outcomes in a cohort of severely obese patients who are candidates for knee replacement surgery but are first given the option of trying a gastric bypass or gastric sleeve operation.

Dr. Samuels says patients with a BMI over 40 often struggle with their recovery after knee replacement surgery, given the added difficulty of rehabilitating and exercising the joint. Researchers, then, are keen to determine whether patients who choose weight loss surgery can match or even exceed the outcomes associated with a knee replacement.

For the 150 patients who completed bariatric surgery, average knee pain and function improved significantly.

After six months, the percent of patients using over-the-counter analgesics dropped from

87.5% to 47.2%

visits to specialists fell from

55.7% to 24.7%

and patients needing prescribed treatments decreased from

40.3% to 12.4%

Bariatric Surgery May Also Alleviate Psoriasis and Psoriatic Arthritis Symptoms

Researchers increasingly suspect that an association between obesity and psoriasis and psoriatic arthritis (PsA) may be linked to tissue-driven systemic inflammation. In a recent study, NYU Langone investigators found that a majority of psoriasis and PsA patients reported significantly improved symptoms after undergoing bariatric surgery. The study also found postoperative improvement among patients with severe psoriasis, lending new support to a positive correlation between excess weight loss via surgery and symptom alleviation.

The collaborative research effort, led by Soumya Reddy, MD, assistant professor of medicine and dermatology, and Jose U. Scher, MD, assistant professor of medicine, Christine Ren-Fielding, MD, division chief of bariatric surgery, and Monica Sethi, MD, research fellow, retrospectively analyzed a database of 9,073 bariatric surgeries over a 12-year span. Among the 86 patients who were diagnosed preoperatively with psoriasis and who completed a follow-up survey, 55 percent reported improvement in their symptoms. Among the 21 patients who also had a PsA diagnosis, 62 percent reported improvement.

Beyond bolstering the potential benefits of weight loss surgery, the study also concluded that older age at diagnosis and severity of psoriasis could be used to identify those patients who are more likely to improve.

A POTENTIAL LINK TO URIC ACID LEVELS

Svetlana Krasnokutsky Samuels, MD, assistant professor of medicine, approaches knee pain another way. She studies the interplay of inflammation within and among patients with hyperuricemia, gout, and osteoarthritis. Although the contribution of uric acid to gout has been well established, she says, the chemical's potential links to osteoarthritis remain less clear.

Together with Steven Abramson, MD, Frederick H. King Professor of Internal Medicine, and chair of the Department of Medicine, and Michael Pillinger, MD, professor of medicine and director of the Rheumatology Fellowship Training Program, Dr. Krasnokutsky Samuels is assessing whether uric acid levels associate with knee osteoarthritis in a non-obese patient population. "We were curious about those who don't have gout but do have osteoarthritis: is the uric acid biologically doing anything and could it potentially be associated with any difference in severity or progression of the arthritis?" she says.

In an initial retrospective analysis of 80 patients with knee osteoarthritis, the collaborators found that higher uric acid levels may be associated with increased rates of joint space narrowing. "So the progressors seemed to have higher uric acid levels than the non-progressors," Dr. Krasnokutsky Samuels says. Because so little is known about the role of uric acid in osteoarthritis, she says, establishing an association with disease progression could have significant implications. "Should our positive results be validated, that would certainly lead to biologic studies to investigate the mechanisms," she says.

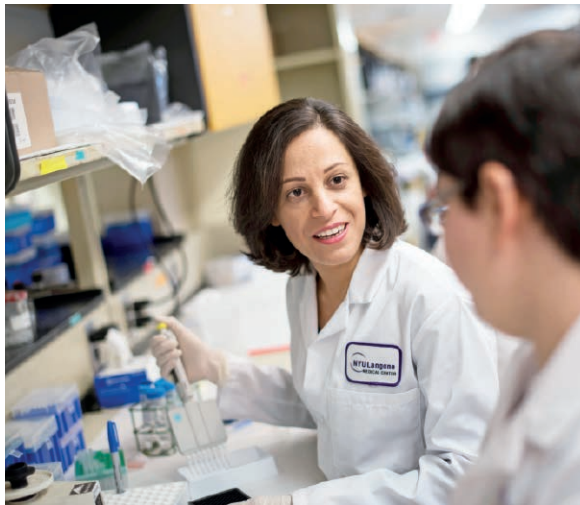
In addition, the research could open a new line of investigation into whether urate lowering in patients with early osteoarthritis might help reduce or prevent progression. Given the sizeable number of patients with the condition, she says, "Even findings that might affect a portion of people with osteoarthritis could have a very large impact."

Uncovering Agents of Autoimmunity within the Intestinal Microbiome

RHEUMATOID ARTHRITIS AND LUPUS

Interest is surging in the human microbiome as an arbiter of health and disease. Researchers at NYU Langone Medical Center are playing a key role in tying our collection of gut microbes to a range of autoimmune diseases. Work by Shahla Abdollahi-Roodsaz, PhD, research assistant professor of medicine, previously showed that arthritis is suppressed in germ-free mice, suggesting that a constituent of the gut microbiota may help trigger the disease. Studies led by her chief collaborator, Jose U. Scher, MD, assistant professor of medicine, also found an association between rheumatoid arthritis and an overabundance of the gram-negative bacterium *Prevotella copri*.

▼ Shahla Abdollahi-Roodsaz, PhD



NYU Langone's Rodent Genetic Engineering Core

This group and its gnotobiotic mouse facility are aiding investigations into the potential links between autoimmunity and specific bacterial species. In germ-free mice genetically predisposed to SLE, psoriatic arthritis, or rheumatoid arthritis, researchers are determining whether bacterial patterns in the microbiome of human patients can be replicated and deconstructed in the animals.

Although scientists haven't been able to determine whether proliferation of the commensal microbe is a cause or a consequence of the disease, Dr. Abdollahi-Roodsaz is finding intriguing new clues in a mouse model in which the IL-1 receptor antagonist has been knocked out. "Mice that lack this IL-1 receptor antagonist spontaneously develop several autoimmune diseases, one of which resembles rheumatoid arthritis," she says. "And when we keep these mice under germ-free conditions, they don't develop severe disease, so they're protected."

In collaboration with Radboud University Nijmegen Medical Centre in the Netherlands, where she holds a dual appointment, Dr. Abdollahi-Roodsaz has found that one or more gram-negative bacteria are driving the disease. "We're not sure whether it's *Prevotella* or not, but we know that it's skin from a gram-negative bacteria, and that these bacteria act through an innate immune receptor called Toll-like receptor 4," she says.

By using selective antibiotic treatments, the researchers found that only elimination of gram-negative bacteria could suppress the disease. They also crossed IL-1 receptor antagonist-deficient mice with Toll-like receptor 4-deficient mice and found that those double knockout mice were protected from the arthritis. The "very exciting" results, Dr. Abdollahi-Roodsaz says, point to at least one pathway through which intestinal microbiota can trigger joint inflammation.

In collaboration with the Division of Gastroenterology, the researchers are also collecting biopsies of human intestinal mucosa and stimulating the donated tissue *in vitro* with intestinal microbes derived from rheumatoid arthritis patients or from healthy volunteers. The collaborators are then determining what kind of immune response might be triggered by arthritis-associated microbiota, and by *Prevotella copri* in particular.

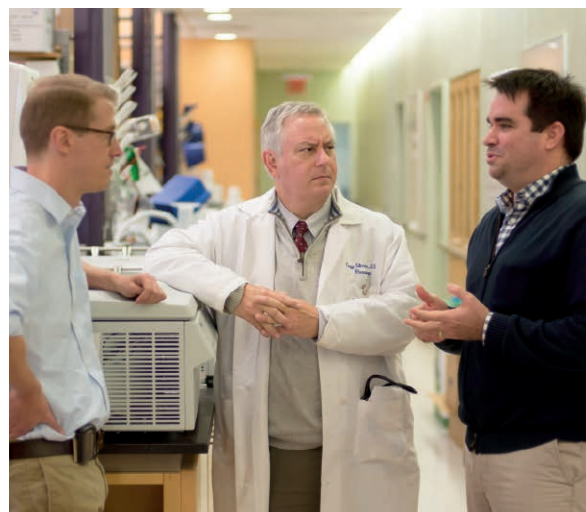
SPOTTING SIGNIFICANT PATTERNS IN LUPUS

Thanks in large part to an ambitious research effort led by Gregg J. Silverman, MD, professor of medicine and pathology, connections are also emerging between the intestinal microbiome and systemic lupus erythematosus. For the project, Dr. Silverman's research group has teamed up with Adriana Heguy, PhD, professor of pathology and director of the Genome Technology Center, and with Jill P. Buyon, MD, the Lady Va and Sir Deryck Maughan Professor of Rheumatology and director of the Division of Rheumatology and of NYU Langone's Lupus Center. The joint studies have been supported in part by the Colton Center for Autoimmunity.

To determine whether specific gut microbes might associate with disease activity, the team performed community profiling of 16S bacterial rRNA genes using next-generation sequencing of stool samples from 83 well-characterized adult SLE patients and 16 healthy controls. "It's been a daunting task, because our lupus patients are very heterogeneous—they are so ethnically and genetically diverse," Dr. Silverman says. "At first glance, I thought it was a worthy challenge but I was also concerned it might prove to be impossible."

Instead, the collaborative effort has begun to reveal important and distinctive differences, including a significant increase in Proteobacteria and a decrease in Firmicutes among the bacteria in SLE patients, when compared to healthy volunteers. "It's astonishing and, in fact, I'm having a hard time getting sleep because each new finding evokes the next question and the next question, and now there are many things lining up," Dr. Silverman says.

One hypothesis suggests that inflammatory processes of this disease may increase the body's oxidative metabolism, resulting in a decrease among pro-homeostatic and protective anaerobes such as Firmicutes. As those microbes become less frequent, other bacteria including the Proteobacteria may fill the vacuum, potentially



Dr. Silverman and colleagues are beginning to identify specific pathobionts that may be opportunistic agents of immune dysregulation and disease. **Pathobionts**, he says, refer to the subset of bacteria that in healthy people are part of an essential balance for robust immune defenses and metabolism. In genetically predisposed individuals, however, they can become the drivers of this relentless and inflammatory autoimmune disease process.

bringing along some types of bacteria that promote breaches of immunologic tolerance and loss of regulation by T cells. This sequence could then trigger an increase in organ-damaging autoantibodies.

Although many details await resolution, Dr. Silverman says, the emerging patterns may lead to "more elegant, targeted approaches" that tweak the microbiome and thereby interfere with major inflammatory and pathogenic pathways.

A Novel Hunt for Skin-Based Biomarkers of Lupus Nephritis Severity

LUPUS NEPHRITIS

Doctors have faced the same frustrating scenario for years: After delivering a therapy to two patients with lupus nephritis, they often observe vastly different outcomes. Now, NYU Langone laboratory and clinical researchers have partnered to find out why the responses diverge. Specifically, they are trying to develop skin-based biomarkers that may more clearly assess disease severity and better predict therapeutic responses.

This approach is based in groundbreaking research performed in the 1990s by professor of medicine H. Michael Belmont, MD. Dr. Belmont tested a series of candidate markers from the biopsies of patients' non-lesional, non-sun-exposed skin and found that up-regulation of adhesion molecules in endothelial cells predicted lupus severity. Robert Clancy, PhD, associate professor of medicine, says the "exciting finding" was among the first to identify biomarkers within such biopsies that reflected a patient's disease activity.

To expand that breakthrough, Dr. Clancy, Dr. Belmont, and Peter Izmirly, MD, assistant professor of medicine, are teaming up with Jill P. Buyon, MD, professor of medicine and director of the Division of Rheumatology and of NYU Langone's Lupus Center, to launch an "agnostic biomarker discovery" effort in both adult and pediatric patients. "We're trying to identify cell types and pathways that are associated with lupus nephritis," Dr. Clancy says. "We feel that there are some biomarkers in the skin that are reflecting what is occurring in the diseased kidneys of the patient."

If the effort succeeds, skin-based markers that faithfully reflect the activation of endothelial cells, inflammatory cells, and resident renal cells might eventually replace more invasive and riskier renal biopsies and advance patient management strategies.

Specifically, the team is using a single-cell RNAseq technique to measure the entire transcriptome of each cell type within a non-lesional, non-sun-exposed skin biopsy and identify transcriptional signals of significance. The unbiased and collaborative approach draws on the expertise of multiple centers and has attracted both industry support and significant funding from the NIH.

EARLY SIGNS OF SUCCESS IN ENDOTHELIAL CELL ASSOCIATION STUDIES

The effort is part of the Accelerating Medicines Partnership in Rheumatoid Arthritis and Lupus (AMP RA/Lupus) Network, a public-private partnership among the NIH, biopharmaceutical companies, and nonprofit organizations. In 2014, the National Institute of Arthritis and Musculoskeletal and Skin Diseases (NIAMS) awarded Dr. Buyon and research partners at the Albert Einstein College of Medicine and Rockefeller University a prestigious AMP RA/Lupus grant, which is helping to fund the new endeavor.

In an initial demonstration of the technique's potential, the group recently presented their results from a pilot study comparing skin from a healthy control to five patients with varying severity of lupus nephritis. The RNAseq, performed on cell suspensions prepared from punch biopsies, indicated that the endothelial cells from patients with less advanced disease had significantly lower expression of NFkB-related genes than those with more advanced disease. In addition, IFN α -responsive genes were strongly represented in all five patients. The healthy control's skin biopsy, by contrast, showed neither NFkB nor IFN α -responsive genes.

The collaborators are continuing to perform association studies to determine whether these and other candidate skin-derived biomarkers track the invasiveness and aggressiveness of proliferative nephritis and could serve as less invasive proxies. In addition to looking at cell types and pathways that might predict disease, the researchers are also working to identify new therapeutic targets. "I have sat across from afflicted families, and I am deeply moved by that experience," Dr. Clancy says. "We have such a strong connection to these families that we would do everything in our power to deliver an advance to them."

The METRO Consortium

The division's lupus nephritis agnostic biomarker discovery project is tapping the wealth of clinical information contained within the Multi-Ethnic Translational Research Optimization (METRO) Lupus Consortium. This new consortium, part of the broader AMP umbrella, is pairing the expertise of scientists and clinicians to improve the care of NYU Langone's multiethnic and multiracial patient population.

The METRO pool includes substantial numbers of Black, Hispanic, Asian, and White patients from clinic populations at the NYU Langone's Hospital for Joint Diseases, Bellevue Hospital Center, and NYU Langone's Center for Musculoskeletal Care. Collectively, this rich dataset will help researchers develop, standardize, and validate cutting-edge technologies to identify key signaling pathways in patients' renal and skin tissues, cells, and urine. The ethnically and racially diverse patient population also may reveal possible pathophysiologic changes that increase the risk of progression to end-stage renal disease in minority populations with nephritis.



▲ Robert Clancy, PhD, and H. Michael Belmont, MD

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AWARDS & RECOGNITION

Ashira Blazer, MD, instructor of medicine, was named Distinguished Fellow by ACR (American College of Rheumatology). NYU Langone's fellows have now received this honor nine times in the past 13 years.

Paula Marchetta, MD, clinical associate professor of medicine, was named incoming treasurer for the ACR.

Purvi Parikh, MD, clinical instructor of medicine and of pediatrics, was appointed National Spokesperson for the Allergy and Asthma Network and was appointed to the American College of Allergy, Asthma, and Immunology (ACAAI) Board of Directors for Advocacy Council.

Mark Philips, MD, professor of medicine, delivered the Presidential Lecture at Memorial Sloan Kettering Cancer Center.

Michael Pillinger, MD, professor of medicine and biochemistry and molecular pharmacology, was named to the ACR Committee on Nominations and Appointments, appointed chair of the ACR Curriculum Task Force for Basic and Clinical Science Online Education, and chair of ACR's Curriculum Task Force for Online Clinical Education.

Gregg Silverman, MD, professor of medicine and pathology, was named Kroc + Visiting Professor, Washington University School of Medicine.

Gerald Weissmann, MD, professor emeritus of medicine, was named Marine Biological Laboratory (MBL) "Legend" on his 85th birthday.

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ACR ABSTRACTS ACCEPTED FOR ORAL PRESENTATION

IL-1 Receptor Antagonist Maintains Intestinal Microbial Homeostasis to Prevent Overt Toll-like Receptor 4-Dependent Intestinal Th17 Differentiation and Autoimmune Arthritis

Session: Innate Immunity and Rheumatic Disease

Rebecca Rogier, Tom Ederveen, Jos Boekhorst, Harm Wopereis, Johan Garssen, Peter van der Kraan, Sacha van Hijum, Marije Koenders, Wim van den Berg and Shahla Abdollahi-Roodsaz

Single-Cell RNA Sequencing of Human Podocytes, Endothelial Cells, and Tubular Cells Identifies Markers and Gene Profiles Differentiating Class IV and Class V Renal Disease in Lupus Nephritis

Session: Systemic Lupus Erythematosus—Human Etiology and Pathogenesis: Genetics, Gene Expression, and Epigenetics
Evan Der, Kemal Akat, Robert Clancy, Beatrice Goilav, Anna R. Broder, H. Michael Belmont, Peter M. Izmirly, Nicole Jordan, Tao Wang, James Pullman, Daniel Schwartz, Ming Wu, Thomas Tuschl, Jill P. Buyon, Chaim Putterman

Complement Activation Predicts Adverse Pregnancy Outcome in Patients with SLE and/or aPL Antibodies

Session: Plenary Session II: Discovery 2015
Jane E. Salmon, Mimi Kim, Marta Guerra, Elianna Kaplowitz, Carl Laskin, Michelle Petri, Ware D. Branch, Michael Lockshin, Lisa R. Sammaritano, Joan T. Merrill, Mary D. Stephenson, Munther Khamashta, Alan M. Peaceman, Anne Lynch, Jill P. Buyon

Netrin-1 and Its Receptors Unc5b and DCC May Be Useful Targets for Preventing Multiple Myeloma Bone Lesions

Session: Biology and Pathology of Bone and Joint: Bone Remodeling
Aranzazu Mediero, Tuere Wilder, Bruce Cronstein

HLA Associations in Mothers of Children with Cardiac Manifestations of Neonatal Lupus

Session: Genetics, Genomics, and Proteomics
Hannah Ainsworth, Carl D. Langefeld, Miranda Marion, Nathalie Costedoat-Chalumeau, Antonio Brucato, Jill Buyon, Robert Clancy

Long-Term Outcomes in Cardiac Neonatal Lupus and Associated Risk Factors for Morbidity

Session: Pediatric Rheumatology—Clinical and Therapeutic Aspects II: Pediatric Systemic Lupus Erythematosus
Amit Saxena, Peter M. Izmirly, Sara Sahl, Deborah Friedman, Jill P. Buyon

Economic Evaluation of Lupus Nephritis in an International Inception Cohort: Comparing the Hospitalization, Medication, Dialysis, and Procedure Costs of Those with and without Nephritis

Session: Systemic Lupus Erythematosus—Clinical Aspects and Treatment I: Epidemiology and Prognosis
Megan Barber, John G. Hanly, Aidan O’Keeffe, Li Su, Murray Urowitz, Yvan St. Pierre, Juanita Romero-Diaz, C. Gordon, Sang-Cheol Bae, Sasha Bernatsky, Daniel J. Wallace, Joan T. Merrill, David A. Isenberg, Anisur Rahman, Ellen M. Ginzler, Paul R. Fortin, Dafna D. Gladman, Jorge Sanchez-Guerrero, Michelle Petri, Ian N. Bruce, Mary Anne Dooley, Rosalind Ramsey-Goldman, Cynthia Aranow, Graciela S. Alarcon, W. Winn Chatham, Kristján Steinsson, Ola Nived, Gunnar K. Sturfelt, Susan Manzi, Munther Khamashta, Ronald F. van Vollenhoven, Asad Zoma, Manel Ramos-Casals, Guillermo Ruiz-Irastorza, S. Sam Lim, Thomas Stoll, Murat Inanc, Kenneth C. Kalunian, Diane L. Kamen, Peter Maddison, Christine A. Peschken, Søren Jacobsen, Anca Askanase, Jill P. Buyon, Chris Theriault, Kara Thompson, Vernon Farewell, Ann E. Clarke

Differing Perspectives Between Doctor, Nurse and Patient Views on Professionalism and Empathy: An Inter-Professional 360-degree Rheumatology Objective Structured Clinical Examination

Session: Education
Jessica Berman, Juliet Aizer, Meng Zhang, Anne R. Bass, Irene Blanco, Anne Davidson, Theodore R. Fields, Jane Kang, Leslie Kerr, Deana M. Lazaro, Stephen A. Paget and Michael H. Pillinger

Adenosine A2A Receptor, but Not A2B Receptor, Deletion Leads to Development of Osteoarthritis (OA) in Mice and Administration of a Liposomal Suspension of Adenosine Prevents/Treats Osteoarthritis in Rats

Session: Biology and Pathology of Bone and Joint: Osteoarthritis Pathogenesis
Carmen Corciulo, Matin Lendhey, Austin Ramme, Tuere Wilder, Oran Kennedy, Bruce Cronstein

Development of Lupus Nephritis: Preclinical Evaluation of Patients Who Subsequently Develop Systemic Lupus Erythematosus Demonstrate Elevation of Select Soluble Mediators Prior to and at Disease Classification in Patients with Nephritis

Session: Systemic Lupus Erythematosus—Human Etiology and Pathogenesis: Modulators of Disease
Melissa E. Munroe, Jourdan R. Anderson, Julie M. Robertson, Timothy B. Niewold, George C. Tsokos, Michael P. Keith, Joan T. Merrill, Jill P. Buyon, John B. Harley, Judith A. James

Does Dysbiosis within the Intestinal Microbiome Contribute to SLE Pathogenesis?

Session: Systemic Lupus Erythematosus: Human Etiology and Pathogenesis: Modulators of Disease
Gregg J. Silverman, Lelise Getu, Haitao Niu, Hanane El Bannoudi, Adriana Heguy, Alexander Alekseyenko, Jill P. Buyon, Doua Azzouz

A Novel Severity Score Based on Cardiac Neonatal Lupus Manifestations Serves As a Predictor and Outcome Measure of Morbidity in Anti-Ro Exposed Fetuses

Session: Reproductive Issues in Rheumatic Disorders: Basic and Clinical Aspects
Amit Saxena, Peter M. Izmirly, Sung Won Han, Paraskevi Briassouli, Tania Rivera, Mark Halushka, Hua Zhong, Deborah Friedman, Robert Clancy, Jill P. Buyon

Platelet Activation and Endothelial Reactivity in Pathogenesis of Tissue Inflammation/Injury in Systemic Lupus Erythematosus

Session: 2015 Rheumatology Research Foundation Edmond L. Dubois, MD Memorial Lectureship
Robert Clancy, Sokha Nhek, Jonathan Newman, Janet Nwaukoni, Sara Rasmussen, Jill P. Buyon, Maya Rubin, Kristen Lee, Jeffrey Berger

Clinical Characteristics and Disease Outcomes in Psoriatic Arthritis Patients by Extent of Body Surface Area Affected by Plaque Psoriasis: Results from Corrona Registry

Session: Spondylarthropathies and Psoriatic Arthritis—Clinical Aspects and Treatment: Psoriatic Arthritis—Comorbidities
Philip J. Mease, Chitra Karki, Carol J. Etzel, Arthur Kavanaugh, Christopher T. Ritchlin, Wendi Malley, Vivian Herrera, Jacqueline B. Palmer, Jeffrey D. Greenberg

A Cluster Randomized Trial of a Behavioral Intervention to Incorporate a Treat to Target Approach in the Clinical Care of Rheumatoid Arthritis Patients in the U.S.

Session: Rheumatoid Arthritis—Clinical Aspects V: Goal = Remission
Leslie Harrold, George W. Reed, J. Timothy Harrington, Christine J. Barr, Katherine C. Saunders, Allan Gibofsky, Eric M. Ruderman, Tmirah Haselkorn, Jeffrey D. Greenberg, Ani John, and Joel M. Kremer

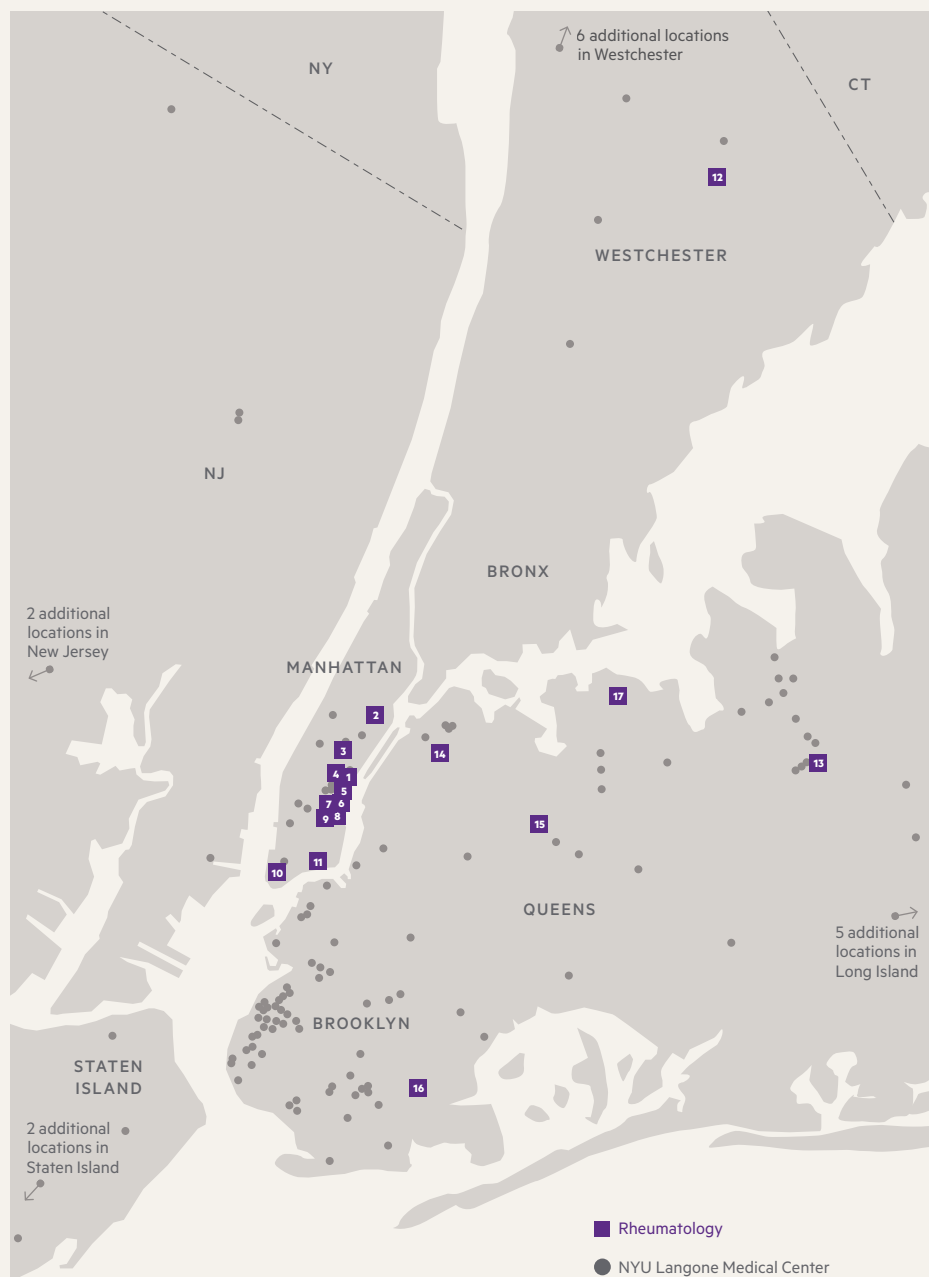
Netrin-1 and Its Receptor Unc5b Are Novel Targets for the Treatment of Inflammatory Arthritis

Session: Rheumatoid Arthritis Animal Models II
Aranzazu Mediero, Tuere Wilder, Bruce Cronstein

Locations

As of December 2015

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Center for
Musculoskeletal Care
333 East 38th Street
New York, NY
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Joan H. Tisch Center
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207 East 84th Street
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- 3**
Preston Robert Tisch Center
for Men's Health
555 Madison Avenue
New York, NY
- 4**
Ambulatory Care Center
240 East 38th Street
New York, NY
- 5**
NYU Langone
Medical Center
550 1st Avenue
New York, NY
- 6**
Bellevue Hospital Center
462 1st Avenue
New York, NY
- 7**
Ambulatory Care Center
324 East 23rd Street
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jill.buyon@nyumc.org

H. Michael Belmont, MD

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Medical Director, NYU Langone Hospital
for Joint Diseases
Associate Director, Clinical Affairs,
Division of Rheumatology
michael.belmont@nyumc.org

James P. Darrow

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

Michael H. Pillinger, MD

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

Ann Rupel

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

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of Rheumatology
ranit.shriky@nyumc.org

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By the Numbers*

NYU LANGONE MEDICAL CENTER

*Numbers represent FY15 (Sept 2014–Aug 2015)

1,069

Total Number of Beds

1,469

Full-Time Faculty

611

MD Candidates

3,800

Publications

77

Operating Rooms

1,392

Part-Time Faculty

79

MD/PhD Candidates

550,000

Square Feet of Research Space

38,554

Patient Discharges

2,627

Voluntary Faculty

272

PhD Candidates

\$178,000,000

NIH Funding

1,216,428

Hospital-Based Outpatient Visits

128

Endowed Professorships

400

Postdoctoral Fellows

\$295,000,000

Total Grant Funding

5,766

Births

2,740

Physicians

1,063

Residents and Fellows

2,900,000

Faculty Group Practice
Office Visits

3,465

Registered and Advanced
Practice Nurses

730

Allied Health Professionals

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