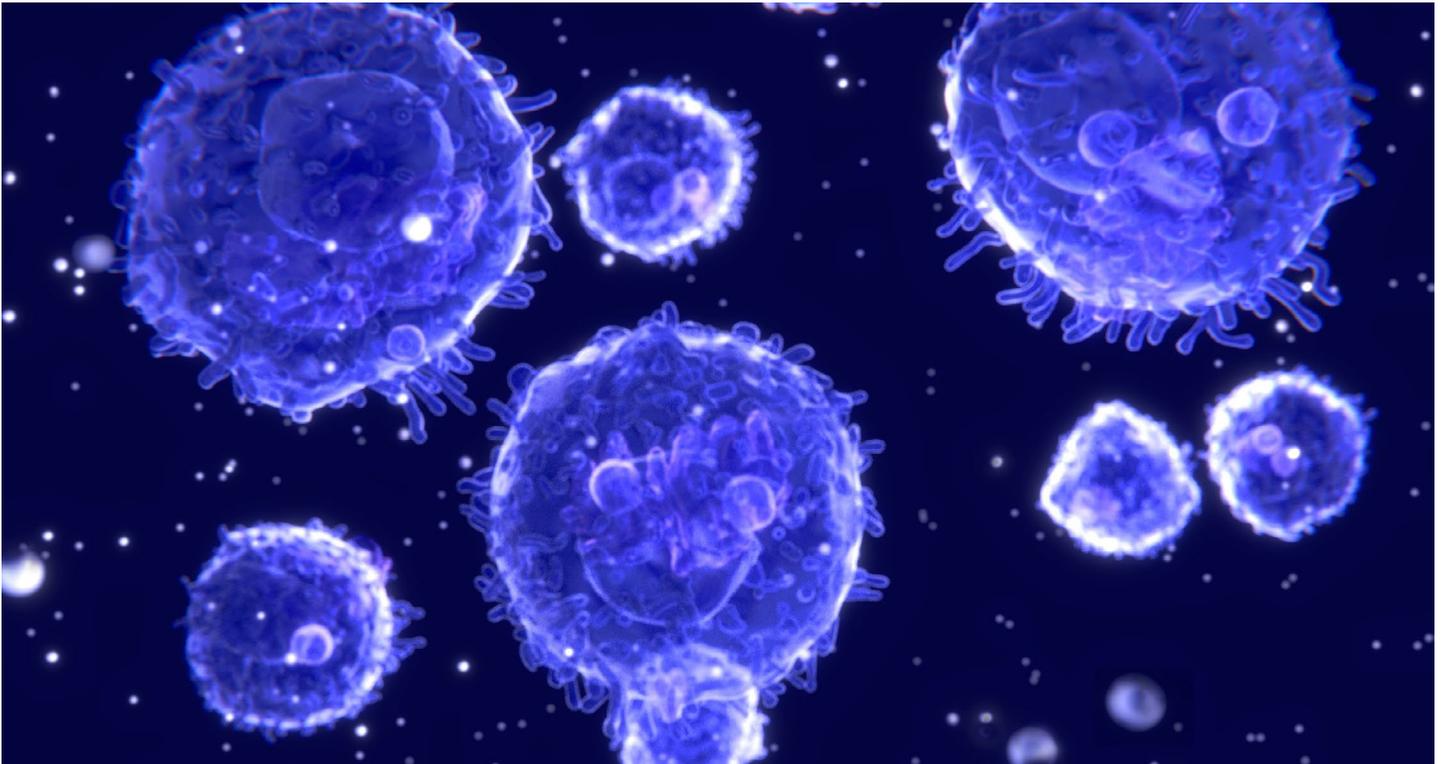


# Breakthroughs

Feinberg School of Medicine Research Office

May 2014



*Northwestern scientists found mutations in the enzyme EZH2 caused lymphocytes, white blood cells (shown here), to undergo uncontrolled growth leading to lymphoma.*

## Harnessing Chemistry to Halt Blood Cancers

**The body is a breeding ground for chemical reactions — and Jonathan Licht, MD, wants to harness those reactions to stop cancer in its tracks.**

“So much of life is about chemical reactions that are unlikely to spontaneously occur, and making them occur with enzymes. It is interesting to think about the mechanism of how enzymes work in the body, and how by inhibiting enzymes, we can potentially stop diseases such as cancer,” said [Licht](#), chief of the [Division of Hematology and Oncology](#) and Johanna Dobe Professor of Medicine.

“We are particularly interested in enzymes called epigenetic regulators,” he says. “These are enzymes that chemically modify the combination of DNA and proteins present in the nucleus of the cell, collectively called chromatin. If DNA is the ‘hard drive,’ by analogy, epigenetic enzymes and their chemical

modifications of chromatin are the software that read the DNA instructions to the cell. In cancer these instructions become garbled, in part because the enzymes that modify chromatin malfunction.”

Licht, also associate director of clinical sciences research at [Robert H. Lurie Comprehensive Cancer Center](#), aims to develop inhibitors to these epigenetic enzymes and proteins to stop cancers such as leukemias and lymphomas from growing and spreading. He has built collaborations across a diverse array of departments, from chemistry to engineering, to better understand these regulatory proteins.

“We have the advantage of having a robust campus that can help support cross-disciplinary research, and the smaller size of laboratories creates a very collaborative, cooperative environment,” he says.

*(continued on page 2)*

Harnessing Chemistry to Halt Blood Cancers

(continued from cover page)

**Stopping Lymphoma**

A large multi-center collaboration led to a breakthrough in lymphoma treatment research this past year. Published in [Cancer Cell](#), scientists found mutations in the enzyme EZH2 caused lymphocytes, white blood cells, to undergo uncontrolled growth leading to lymphoma, cancer of the lymph nodes.

Licht’s group determined how mutated EZh2 alters the regulation in genes in a cancer cell model, and correlated findings with collaborators working in a mouse model of lymphoma with primary human tumor samples. The group learned that in healthy individuals, EZH2 works by allowing lymphocytes to divide without stopping, which leads to better antibodies. Here, the EZH2 promotion of growth is temporary—the lymphocytes eventually reach a state of maturation and rest. When the protein is mutated, the lymphocytes are locked in a continuous state of development, and the result is lymphoma.

Early studies show certain cases of lymphoma could be treated with inhibitors of EZH2.

“From this success we are trying to open a clinical trial of EZH2 inhibitor for the treatment of specific forms of lymphoma,” says Licht. “We are also trying to determine whether these EZH2 inhibitors will be useful in other forms of malignancy.”

**Targeting Multiple Myeloma**

Licht also studies the protein MMSET, which causes multiple myeloma—a cancer of plasma cells—when overexpressed.

To tackle this overexpression, he partners with [Neil Kelleher, PhD](#), professor in Medicine and Chemistry at the Weinberg College of Arts and Sciences and [Milan Mrksich, PhD](#), professor in Cell and Molecular Biology and Biomedical Engineering at McCormick School of Engineering. They have found that by depleting the cell of MMSET, cancer cells stop growing and become more sensitive to chemotherapy.

“The MMset gene not only is overexpressed, emerging data show that up to 20 percent of children with relapse of their lymphocytic leukemia have mutations of MMSET that make



*Jonathan Licht, MD, chief of the Division of Hematology and Oncology and Johanna Dobe Professor of Medicine, says enzymes and regulator proteins could hold the key to halting cancer.*

the protein hyperactive,” Licht says. Lymphocytic leukemia is a type of cancer in which the bone marrow makes too many lymphocytes, a type of white blood cells.

Licht is now investigating how mutations of MMSET might affect the structure and function of the genetic material of the cell, which allows cancer cells to become more resistant to chemotherapies.

**Finding Connections**

“It turns out a lot of the same genes we study in leukemia and lymphoma are also found in bladder cancer,” says Licht.

He partners with [Joshua Meeks, MD, PhD](#), assistant professor in Urology, to research a regulatory protein, UTX, found in both bladder cancer and multiple myeloma. Currently Licht and Meeks are comparing and contrasting both malignancies to determine if there is a common set of genes and pathways that are affected by a mutation of this protein.

“We’d like to find a unified theory, that in all the variety of these mutations there are common sets of genes that are mis-regulated,” says Licht. “I’d like to know whether or not these mutations converge on a common pathway or are going to be a cacophony of different pathways. Is each cancer different and will therapy have to be tailored to the individual, or can we find a common pathway that we can target for treatment?”

**Future Research**

In the near future, Licht hopes to create more accurate animal models of myelomas and acute leukemia and develop drugs to treat these diseases.

“In five years, I’d like to see more of these epigenetic protein inhibitors going into clinical trials,” he said. “I am interested in these enzymes and proteins because they are real medical problems, and relapsed acute leukemias are a huge dilemma. If a mutated enzyme might explain a quarter of how the mechanism behind the disease works, then we could make a dent in the treatment of those kids that aren’t saved by current chemotherapy.”

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# HIV Research Workshop Unites Scientists and Inspiration

Leading HIV/AIDS scientists from around the world and Chicago-land gathered recently at Northwestern Medicine's HIV Cure Research Workshop with same goal in mind: to work towards finding a cure for HIV/AIDS.

The unique workshop brought a number of international scientists and clinicians together to share their work and collaborate on feasible HIV cure research and prevention efforts, as well as develop solutions to the growing HIV epidemic among young people.

Among the list of presenters was special guest speaker Timothy Ray Brown, also known as "the Berlin Patient," the first person to be cured of HIV infection. Seven years ago, Brown underwent a risky stem cell bone marrow transplant that replaced his immune system with one from a donor with an extremely rare genetic variant resistant to infection by HIV. Since the procedure, Brown has tested negative and has been off all medicines for HIV.

"I am the first person in the world cured of HIV, but I know in my heart I will not be the last," Brown said to the room of scientist. "Without your dedication and hard work, there would be no hope for an AIDS-free world."

Throughout the day, many presenters made a point to highlight the impact Brown's cure is making on current and future research.

"It's very important to emphasize that just a few years ago, we were not able to use the word 'cure.' If it weren't for Mr. Brown this would not be possible," said [Richard D'Aquila, MD](#), professor of [Infectious Diseases](#) at Northwestern University Feinberg School of Medicine and organizer of the event.

Many attendees also expressed their gratitude to Brown for his



The workshop brought together a coalition of scientists and supporters to share research in hopes of an HIV cure. From left: Timothy Heinrich, MD, Brian Mustanski, PhD, Tom Hope, PhD, Robert Murphy, MD, Timothy Ray Brown - also known as "The Berlin Patient," Deborah Persaud, MD, Richard D'Aquila, MD, and Luis Montaner, DSV, MSc, PhD. [More photos](#).

willingness to undergo the high-risk procedure.

"When the paper on the Berlin Patient came out in 2007, that was proof-of-concept that you can take someone living with HIV infection and eradicate that," said Timothy Heinrich, MD, Harvard Medical School. "That was really my inspiration to continue on in my own academic training. (Brown) was an inspiration to everyone I was training with at the time; it was huge to our field and really energized us."

"When I was first cured of HIV, I thought people were only interested in my story for scientific reasons, but have since realized that my story provides people with hope," Brown said. "I strongly believe that together we can turn hope into action that will then someday lead to a cure."

## New Director of Galter to Strengthen Library Informatics



[Kristi Holmes, PhD](#), a bioinformaticist with a background in research impact and genomic medicine, recently joined Feinberg as director of the [Galter Health Sciences Library](#) and associate professor of Preventive Medicine-[Health and Biomedical Informatics](#).

"I'm delighted to have this opportunity to join Galter at such an exciting time for biomedical libraries," Holmes said. "Now, more than ever, libraries are uniquely

poised to provide resources, technology and services to support scientific discovery and clinical care. Feinberg is advancing medicine and research in some remarkable ways and I can't

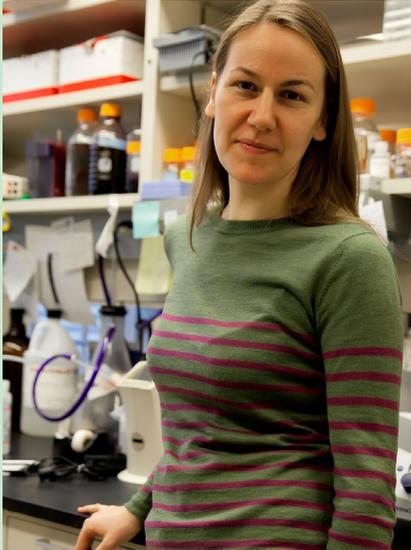
imagine a more exciting place to be."

Holmes earned her PhD at Iowa State University before joining the Bernard Becker Medical Library at Washington University in St. Louis, where she has worked as a bioinformaticist since 2007.

"After carefully surveying where Galter is positioned, one of the things that became apparent is our need to enhance bioinformatics capability while maintaining the traditional resources of a health sciences library. Kristi will work to ensure we're providing 21st century resources to our scientists and physicians," said Donald Lloyd-Jones, MD, ScM, senior associate dean for Clinical and Translational Research, chair of Preventive Medicine, and director of the Northwestern University Clinical and Translational Sciences Institute (NUCATS). [Read more](#).

# Faculty Profile: Eva Gottwein, PhD

## Assistant Professor in Microbiology/Immunology



When [Eva Gottwein, PhD](#), assistant professor of [Microbiology-Immunology](#), was searching for a postdoctoral position, microRNAs, tiny bits of genetic material, began to emerge as a large class of regulatory RNAs and the first viral microRNAs were discovered. Interested in this field, Gottwein began a new line of work in the lab of Bryan Cullen, PhD, at Duke University studying microRNAs encoded by Kaposi's Sarcoma-associated Herpesvirus (KSHV).

"When I began this work, not a single target of the KSHV microRNAs was known," said Gottwein. "At the conclusion of my postdoctoral work, we had identified thousands of viral microRNA binding sites at nucleotide resolution, which allowed me to initiate the functional work that we are pursuing now."

## Q&A

### What are your research interests?

My broad research interest is on how viruses manipulate the cellular environment to their advantage. We focus on how virally encoded microRNAs alter cellular gene expression. We have shown that the microRNAs encoded by the oncogenic human herpesvirus KSHV likely regulate hundreds of human messenger RNAs, by binding to thousands of sites in the human transcriptome. Our current projects take advantage of this information to identify and understand roles these microRNAs play in viral oncogenesis. A secondary interest is to use virally encoded microRNAs as models to study the fundamental rules of target recognition by microRNAs. I enjoy the inherently multidisciplinary nature of our work, which touches on virology, genomics, molecular biology, cancer biology, and cell biology.

### What is the ultimate goal of your research?

Over the next few years, we hope to gain a detailed understanding of the KSHV microRNA targetome, i.e. to identify the messenger RNAs regulated by these viral microRNAs in the different cell types this virus infects. We plan to use this information to achieve a basic understanding of the most important functions of the KSHV microRNAs. Our data suggest that there are several unexpected cellular processes these viral microRNAs interfere with and we hope that our current work will open up new frontiers in the study of virus-host interactions and viral oncogenesis.

### How does your research advance medical science and knowledge?

As the causative agent of Kaposi's Sarcoma and B cell lymphomas, KSHV remains an important human pathogen, and our work contributes to a better understanding of KSHV pathogenesis. Gaining knowledge of the basic mechanisms of microRNA target recognition is essential for the study of the rapidly emerging roles of cellular or viral microRNAs in many different diseases, including cancer.

### How is your research funded?

My lab is funded by a grant from the American Cancer Society, Illinois Division, and by an R01 grant from the National Cancer Institute (NCI). In the NCI-funded work, we study how and why some viral microRNAs mimic the function of cellular microRNAs.

### Who makes up your research team?

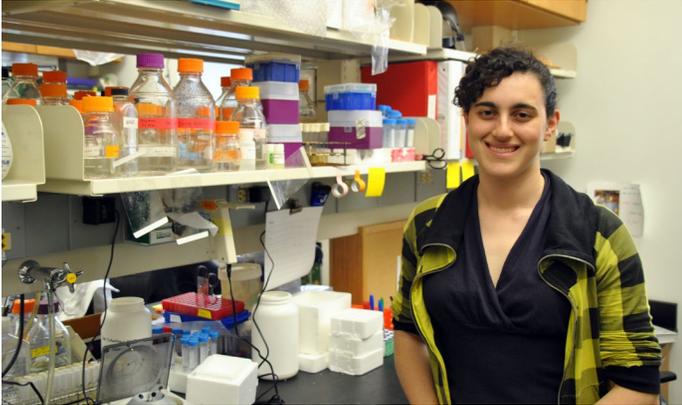
I work with two postdocs, Mark Manzano, PhD, and Eleonora Forte, PhD. When I have time, I still do experiments myself. Mark and Eleonora pursue both independent and collaborative projects. I'm involved in all projects and enjoy establishing new techniques and projects in the lab.

### What types of collaborations are you engaged in across campus (and beyond)?

We have benefitted in many ways from the collaborative atmosphere at Feinberg and have had help from several labs who have shared reagents or expertise. For example, the lab of [Greg Smith, PhD](#), associate professor of Microbiology-Immunology, helped us to establish bacmid recombineering technology in our lab. We are also working with the [Next Generation Sequencing Core](#) at Northwestern University. Outside of Feinberg, we have collaborated with Dr. Nikolaus Rajewsky's lab at the Max Delbrück Center for Molecular Medicine in Berlin, Germany, on work that uses the KSHV microRNAs as tools to better understand microRNA target recognition.

# Student Profile: Celeste Mallama

## Driskill Graduate Program in Life Sciences



Celeste Mallama, a fourth-year PhD/MPH student in the [Driskill Graduate Training Program](#), studies the interaction between *Legionella* and the innate immune system in the laboratory of [Nicholas Cianciotto, PhD](#), professor of [Microbiology-Immunology](#). She also serves as one of the editors-in-chief for a new student-run journal called the *Northwestern Public Health Review*.

Mallama received her undergraduate degree in biological-sciences from Wellesley College in Massachusetts. After spending four years studying a variety of academic disciplines, she knew science was her strong point. With a little encouragement from a college professor, she decided to pursue a graduate degree. She chose to study at Northwestern because of the program's unique focus on basic science research and the application of the research to issues in public health.

## Q&A

### What is your hometown?

I grew up in Bowie, Maryland.

### What are your research interests?

I work in Dr. Cianciotto's lab on *Legionella pneumophila*, a gram-negative bacterium and the etiologic agent of Legionnaires' disease, a form of pneumonia. My project focuses on the type II secretion system of *Legionella* and how it interacts with the host's innate immune system during infection. This secretion system exports proteins that allow the bacterium to successfully infect the host and dampen the host immune response. I'm interested in learning more about how this dampening occurs.

### What exciting projects are you working on?

I work on the board of an exciting new student-run journal called the *Northwestern Public Health Review* ([NPHR](#)). Twice

annually we publish an academic journal of public health research, reflections, histories, and essays written by faculty and students from Northwestern and other public health institutions. We published our first edition in fall of 2013, and our second edition will be coming out in the spring of 2014. The NPHR has provided me with a wonderful opportunity to not only work on my science writing, but also to meet other students who are interested in public health and to interact and connect with faculty members and public health specialists at Northwestern and beyond who are working on fascinating and innovative public health projects. We also have a public health [blog](#) that we encourage students and faculty to contribute to.

### Why did you choose Northwestern?

Being a part of Feinberg has afforded me a lot of amazing opportunities. One of my favorites was the opportunity to rotate through the Illinois Department of Public Health (IDPH) for my MPH field experience. I shadowed the lab staff at the IDPH for a month through the different departments (newborn screening, water testing, STD screening, influenza tracking, etc.) and learned about the services provided by the state. It was the first time I had seen the concrete link between science and public health in action, and it was eye opening. I think most of us tend to take for granted the preventive public health measures that the state provides for us and it was a wonderful experience to take a look behind the scenes.

### What are your plans for after graduation?

The great thing about graduate school is that you have time to figure out what interests you and cultivate those interests. I know that when I graduate, I'd like to pursue a position that incorporates my interest in public health, science, and science writing. There are a lot of amazing fellowship opportunities such as the epidemic intelligence service through the Centers for Disease Control where the fellows are at the forefront of public health surveillance and epidemiology, or the American Association for the Advancement of Science policy fellowship where fellows are informing decisions on science and technology policy and implementation.

### What do you do in your free time?

I love music, so in my free time I volunteer for a local independent radio station and I write for a Canadian music blog, reviewing albums and live shows. One of my lab mates got me interested in running, so during the summer I'm out on the lakefront running, and I finished my first marathon last November. I also set a goal to see all 50 states, so I travel whenever possible.

# Research in the News

## **USA Today April 30**

Even routine housework may help stave off disability  
Dorothy Dunlop's research was featured.

## **Wall Street Journal April 28**

Early births for pregnant women on antidepressants  
more common  
William Grobman was quoted.

## **US News & World Report April 28**

Training programs protect young athletes from ACL tears  
Cynthia LaBella's research was featured.

## **Huffington Post April 23**

Can you catch the same cold twice?  
Mark Huffman was quoted.

## **ABC News (National) April 22**

How to sleep like you're on vacation  
Kelly Glazer Baron was quoted.

## **FOX News (National) April 22**

3 real-life success stories from high-tech diets  
Bonnie Spring was quoted.

## **The Washington Post April 16**

Even casually smoking marijuana can change your brain,  
study says  
Hans Breiter's research was featured.

■ This research was also featured on CNN, ABC News, NBC News, FOX News, in *The Wall Street Journal*, *Los Angeles Times*, *Chicago Sun-Times*, *TIME Magazine*, *Shape Magazine*, Yahoo! News, and more.

## **TIME Magazine April 14**

Young dads are at risk for postpartum depression  
Craig Garfield's research was featured.

■ This research was also featured in *USA Today*, *US News & World Report*, on FOX News, and more.

## **NPR April 7**

Good day sunshine: Could morning light help keep us lean?  
Phyllis Zee's research was featured.

■ Zee's research was also featured in the *Washington Post*, *Huffington Post*, *Web MD*, *Men's Health*, *Shape*, *Prevention*, on CBS News, NBC News, FOX News, and more.

## **NBC News (National) April 7**

Viagra may boost risk of deadly skin cancer, study finds  
June Robinson's work was included.

[More media coverage](#) available online.

Northwestern University

# NUCATS

Clinical and Translational Sciences Institute

## NUCATS Corner

### New Venture Services

Do you lack the time or expertise to accelerate or commercialize your great idea? NUCATS New Venture services can help you identify resources to advance your idea through the proof-of-concept stage. We can link you to many appropriate resources including: how to protect your intellectual property and provide legal advice, [research study support](#), networks for partnerships in the private sector and academia, and sources of [pilot funding](#) opportunities. In addition, if you're interested in starting a company, we can help you create a business model and provide you with counsel and development support.

Contact the NUCATS [preclinical navigator](#) and arrange a consultation appointment to get advice on how to make your ideas come to life.

## Core Facility Open House

The [Cancer Informatics Core](#) addresses the increasing demand for bioinformatics and clinical trial informatics from investigators at the Robert H. Lurie Comprehensive Cancer Center.

Analysis of big data from cancer genomics, proteomics, and clinical trials requires smart data analysts—those with good computer programming skills and data-mining expertise. Without the right skills, researchers will end up finding patterns that mean nothing and missing those that are gold. The core provides local expertise to develop and maintain state of the art computational approaches for translating the big data into clinically actionable patterns, and contribute to transforming basic science into practical benefits for cancer patients.

Faculty, staff, and students are welcome to learn more about the Cancer Informatics Core on Wednesday, May 14, from noon to 1 p.m. An [open house](#) will be held in the Robert H. Lurie Research Building's Hughes Auditorium, at 303 E. Superior Street on the Chicago campus.

# Sponsored Research



**PI: Ramana Davuluri, PhD**  
**Professor of Preventive Medicine**  
**and Biomedical Informatics**

**Sponsor: National Library of Medicine**

**Title: "Informatics Platform for**  
**Mammalian Gene Regulation at**  
**Isoform-level"**

In recent years, the notion of "one gene makes one protein that functions in one signaling pathway" in mammalian cells has been shown to be overly simplistic. Recent evidence suggests that more than 50 percent of human genes produce multiple protein isoforms, through alternative splicing and alternative usage of transcription initiation and/or termination. Notably, the disruption of many of these genes is implicated in cancer and several neuropsychiatric disorders.

For majority of human genes the resulting multiple protein isoforms are functionally different and can participate in different signaling pathways. However, nearly a decade since the completion of the human genome draft sequence, we still assume "gene" as the basic functional unit in a cell.

Davuluri's team argues that the isoform-level gene products, "transcript variants" and "protein isoforms," are the basic functional units in a mammalian cell, and accordingly, the informatics resources for managing and analyzing gene regulation data in mammalian cells should adopt "gene isoform centric" rather than "gene centric" approaches. They propose to build an informatics platform for understanding gene regulation at isoform-level by developing statically rigorous bioinformatics resources for processing Next-Generation Sequencing (NGS) data.

Recently, computational approaches that combine seemingly disparate experimental data have been successful in developing concise gene regulation models and transcriptional modules. Davuluri plans to extend these methodologies to perform integrative analysis of multiple high-throughput data sets currently generated across different laboratories, including Wistar, into computational models to predict different transcriptional isoforms of mammalian genes and protein-DNA interactions at isoform level.

They will apply innovative statistical modeling approaches that combine state-of-the-art meta-classification algorithms, such as Naïve Bayes Tree, Bagging, and LogitBoost, with Random Forest feature selection to classify different types of target promoters with good classification accuracy and reduced instability, in order to predict gene promoters and infer the protein-DNA interactions from ChIP-seq data. The computational models and the derived information will be integrated into a novel database,

which will serve as an in silico platform for transcriptional regulation studies. This will be completed by pursuing the following aims: 1) develop statistically rigorous novel algorithms and bioinformatics pipelines to identify the orthologous promoters, corresponding transcript variants, and protein isoforms that are conserved between human and mouse; and 2) develop novel algorithms and informatics pipelines for integrative analysis of NGS datasets to estimate the activity and expression of both known and novel promoters and their transcript variants, in various tissues, developmental stages, and disease conditions.

The novel bioinformatics methods developed by this project will help in silico discovery and research for accelerating the linkage of phenotypic and genomic information, at gene-isoform level.



**PI: Edward Thorp, PhD**  
**Assistant Professor of Pathology**

**Sponsor: National Heart, Lung, and Blood Institute**

**Project: Efferocytosis Directed Inflammation Resolution and Repair in the Hypoxic Heart**

Heart failure after heart attack is an escalating cause of morbidity and mortality. Pharmacological advances and effective acute care have reduced mortality, and Northwestern Memorial Hospital and its Bluhm Cardiovascular Institute boast one of the lowest heart attack mortality rates in the United States. A flip side is that residual risk of heart failure after heart attack is now increasing. Thus, this National Institutes of Health (NIH)-funded project seeks to understand and develop new approaches to preserve heart function, specifically by harnessing the body's own inflammatory cellular and humoral defense system.

The extent of tissue damage in the acute phase of heart attack is a critical determinant of subsequent adverse cardiac remodeling that leads to impaired pumping capacity. As such, an important goal is to minimize early injury to salvage precious non-regenerative cardiac cells. To this extent, it may surprise some that the body's natural inflammatory response after heart attack is both helpful and harmful. Such adverse effects are partly explained by collateral damage when blood supply is suddenly returned to the myocardium after a period of ischemia, or lack of oxygen. Also important, the inflammatory response in the heart has not been optimized by evolutionary pressure, as heart attacks are typically a disease of aging and hyperlipidemia. This proposal seeks to selectively target the beneficial aspects of inflammatory wound healing while sup-

*(continued on page 8)*

## Sponsored Research

(continued from page 7)

pressing maladaptive or other clinical side effects.

In this context, Thorp's studies will focus on an inflammatory cell called the phagocyte and its interactions with cardiac muscle and stem cells. Phagocyte interactions also encompass "efferocytosis." Efferocytosis is the phagocytic removal and metabolism of dying cells, which is naturally escalated during tissue injury. More importantly, efferocytosis is also the first step in a signaling cascade driven by lipid mediators to suppress chronic inflammation. In a recent publication from Thorp's lab, the team provided the first evidence that efferocytosis benefits cardiac function. In humans (in collaboration with surgeons here at Northwestern and at Columbia University), their data also show that efferocytosis is naturally inefficient in the heart and therefore a therapeutic target, including during secondary atherosclerosis.

Beyond physiology and cell biology, the molecular factors that govern phagocyte interactions with muscle and stem cells are unknown. Also unclear is how phagocyte function and inflammation is regulated under low oxygen (i.e., hypoxia). Thorpe and his team are actively examining these unknowns through unbiased screening. Separately and in collaboration, they also study how hypoxia inducible factors regulate phagocyte function.

In sum, these studies will uncover the mechanistic basis and preclinical potential of phagocyte signaling pathways after muscle injury and during hypoxia.

## Welcome New Faculty



**Sangeeta Bhorade, MD**, joins as associate professor of Medicine-Pulmonary and director of the Northwestern Medicine Lung Transplant Program.

She most recently was associate professor of Medicine and medical director of the Lung Transplant Program at the University of Chicago. She received

her medical degree from the University of Chicago Pritzker School of Medicine, where she also completed her internship in medicine, as well as her residency and fellowship in pulmonary/critical care.

Bhorade is interested in better understanding why lung rejection occurs after lung transplantation in certain patients, most likely due to secondary to factors including type of immunosuppression, genetics, and infectious causes on the immune system. She currently studies the effect of lung microbiota on the immune system in lung transplant recipients.

## Funding

### Strategic Alliances for Medications Development to Treat Substance Use Disorders (R01)

[More information](#)

**Sponsor:** United States Department of Health and Human Services, National Institutes of Health, National Institute on Drug Abuse

**Submission deadline: July 28**

**Upper Amount: \$6 million**

**Synopsis:** This opportunity supports research that advances compounds towards FDA approval by leveraging NIDA funds with the strengths and resources of outside organizations, such as for-profit and not-for-profit entities, including academic institutions, pharmaceutical and biotechnology companies, private and public foundations, and small businesses. In comparison with traditional grant-funded research, strategic alliances will increase the pace at which medications to treat substance use disorders move through the drug development process. Both the term and budget of the grant are consistent with the objective of accelerating the pace of medications development compared to traditional grant funding. Project aims can range from development of a new molecular entity to the expansion of an existing medication's clinical indication(s). It is hoped that support for these collaborations will accelerate the rate of medications development for substance use disorders.

### Chronic Inflammation and Age-Related Disease (R01)

[More information](#)

**Sponsor:** United States Department of Health and Human Services, National Institutes of Health

**Submission deadline: June 5**

**Upper Amount: \$2.5 Million**

**Synopsis:** There is a critical need to establish the knowledge base that will allow a better understanding of the complex interplay between inflammation and age-related diseases. Applications submitted should aim to clarify the molecular and cellular basis for the increase in circulating inflammatory factors with aging, and/or shed light on the cause-effect relationship between inflammation and disease, using pre-clinical (animal or cellular based) models.

[View more funding opportunities](#)

# High Impact Factor Research

## March 2014

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## Help Feinberg Track Journals

The Feinberg Research Office regularly tracks research published by Feinberg investigators. The citations are used on web pages, in newsletters and social media, for internal reporting, and more. To more accurately track these journals, the Research Office asks that Feinberg investigators use the following institution name in the address field when publishing in peer-reviewed journals: "Northwestern University Feinberg School of Medicine."

# Calendar

Friday, May 16

## Lurie Cancer Center Grand Rounds

“Translating preclinical discoveries to more effective treatments for children with cancer,” presented by Malcolm Smith, MD, PhD, National Cancer Institute.

**Time:** 8 to 9 a.m.

**Location:** Lurie Research Center — Gray Room  
303 E. Superior St. (Chicago campus)

**Contact:** [cancer@northwestern.edu](mailto:cancer@northwestern.edu)  
[More information](#)

Tuesday, May 20

## Lectures in Life Sciences

“Fibrosis associated with chronic liver disease,” presented by Scott Friedman, MD, Mt. Sinai Alcoholic Liver Disease Research Center.

**Time:** 8 to 9 a.m.

**Location:** Lurie Research Center — Hughes  
303 E. Superior St. (Chicago campus)

**Contact:** [wamuller@northwestern.edu](mailto:wamuller@northwestern.edu)  
[More information](#)

Wednesday, May 21

## Silverstein Lecture Series

“Cancer stem cells: Are we targeting the right cells?” presented by Max S. Wicha, MD, University of Michigan Comprehensive Cancer Center.

**Time:** 6 to 7 p.m.

**Location:** Lurie Research Center — Hughes  
303 E. Superior St. (Chicago campus)

**Contact:** [michelle.mohney@northwestern.edu](mailto:michelle.mohney@northwestern.edu)  
[More information](#)

### [More Events](#)

Event organizers are encouraged to submit calendar items on [Plan-It Purple](#) for consideration. Please contact the [Research Office](#) with further questions.

## NIH News

In April, the National Institutes of Health (NIH) issued the following announcements:

- [New NIH resubmission policies](#)
- NIH updating [grant closeout policies and procedures](#) to align with Health and Human Services requirements
- NIH [HeLa genome sequence data submission and access policy](#) announced
- [Changes to the financial conflict of interest module](#) for submission of reports to NIH beginning April 25

[NIH is joining the National Science Foundation](#) (NSF) to teach university-based researchers to become entrepreneurs. The program, known as the Innovation Corps, or I-Corps, has been adopted by more than 100 universities since it was established by NSF in 2011. The I-Corps learning model consists of three-person teams, often a faculty researcher and students, who together receive online and in-person instruction on the basics of entering the commercial marketplace.

Story Landis, PhD, director of the NIH National Institute of Neurological Disorders and Stroke (NINDS) [posted an analysis](#) indicating that from 1997 to 2012, NINDS expenditures on applied research increased from 13 to 29 percent while the proportion of basic research declined from 87 to 71 percent. Moreover, expenditures devoted to fundamental basic rather than disease-focused research declined from 52 to 27 percent. Landis writes, “We are concerned by the possibility that many investigators falsely believe that NINDS is no longer interested in supporting research into the normal function of the brain and nervous system, and that their chances of obtaining NIH funding are better if they propose disease-focused basic or applied studies, rather than fundamental basic science research.”

Sally Rockey, PhD, NIH deputy director for external research, highlighted the Institutes’ [programs targeting new scientists](#).

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