University of Illinois at Urbana-Champaign
School of Molecular and Cellular Biology

393 Morrill Hall

505 South Goodwin Avenue

Urbana, IL 61801

mcb.illinois.edu

Nonprofit org.
U.S. postage
PAID
Champaign, IL
Permit no. 75

MCB

ILLINOIS
School of Molecular
& Cellular Biology
COLLEGE OF LIBERAL ARTS & SCIENCES

DEPARTMENTS OF

Biochemistry
Cell and Developmental Biology
Microbiology
Molecular and Integrative Physiology

ILLINOIS
College of Liberal Arts & Sciences

LETTER FROM THE DIRECTOR

Dr. Milan K. Bagchi, Director and Deborah Paul Endowed Professor of Molecular and Cellular Biology

Greetings to friends and alumni of the School of Molecular and Cellular Biology at Illinois. A change of guard in the School leadership took place last August as I became the new director. I would like to acknowledge the past leadership of Dr. Stephen Sligar, who shepherded the School through fiscal challenges and led it during a decade of notable scientific advancements.

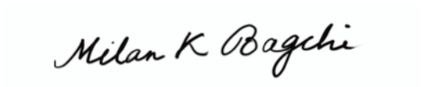
It is with great pride that I share this magazine with you. It highlights the inventive efforts of our faculty in deciphering cancer. In his Pulitzer prize-winning book *The Emperor of All Maladies*, Dr. Siddhartha Mukherjee calls cancer the most elemental and magisterial disease known to our species. In relentless battle with this malady, researchers keep inventing and reinventing, learning and unlearning strategies. Our MCB scientists are exploring new avenues to develop effective treatments for this complex disease. They are playing important research and leadership roles in the newly established Cancer Center at University of Illinois at Urbana-Champaign and contributing to the Center's goal of achieving national recognition and status.

This magazine also highlights the success of our research leaders in propelling bright minds and nurturing the careers of future scientists. Our graduates, with strong experience in research and innovation, are awarded postdoctoral fellowships in major laboratories that lead to a career in academia. They are also pursuing exciting employment opportunities in pharmaceutical industry or the government. Our undergraduates, equipped with fundamental, critical, and analytical skills imparted by MCB's curriculum, are moving on to opportunities for further education, most notably medical school and graduate education in frontier areas of modern biology. The stories included in this magazine are a sample of the valuable development of both graduate and undergraduate students in our research laboratories.

We are committed to furthering MCB's ability to attract and educate excellent undergraduate and graduate students, to support the continuing achievements of our faculty, and to promote MCB's contribution to the overall distinction of the University. A testimony to our strong commitment to undergraduate education is the creation of a new learning center space, for which an update is included. The nurturing environment of learning and discovery in the School of MCB helps build successful careers. Many of our alumni have made great contributions to their fields with the strong foundation of an MCB undergraduate degree.

Alumni really are the key to MCB's and the University's success in all areas. Your accomplishments, distinctions, and awards are a source of pride and a beacon for future scientists. The diversity of alumni career trajectories gives our students hope and a broader vision of scientific contributions. Alumni also make it possible to retain and honor excellent faculty and students by supporting their research and distinctions. On behalf of our faculty and students, I thank you for your support, and importantly, your continued engagement in our academic mission.

The research conducted in our school seeks the answers to fundamental questions about how organisms work at the molecular and systems level, how they evolve, and the resultant implications for life, health, and disease. Our science is foundational, translational, and transformative. I hope you will enjoy reading about the exciting work being accomplished by our faculty, students, and alumni. •



Milan K. Bagchi
Director
School of Molecular and Cellular Biology

TABLE OF CONTENTS

MCB is published by the School of Molecular and Cellular Biology

MCB Director
Milan K. Bagchi

Managing Editor
Steph Adams

Additional Editing
Judith Lateer

Development
Trent Reed

Photography
Steph Adams
L. Brian Stauffer
Thompson • McClellan

Design
Pat Mayer

MCB Communications Office
393 Morrill Hall, MC-119
505 South Goodwin Avenue
Urbana, IL 61801

phone | 217.333.2032

mcbcommunications@illinois.edu

mcb.illinois.edu

ILLINOIS
School of Molecular & Cellular Biology
COLLEGE OF LIBERAL ARTS & SCIENCES

Produced by the MCB Communications Office for the School of Molecular and Cellular Biology.
The University of Illinois is an equal opportunity, affirmative action institution.

Printed on recycled paper with soy-based ink. 12.038

MCB Faculty Help Lead the Cancer Center at Illinois in Collaborative, Interdisciplinary, and Translational Research

by Deb Aronson

Several MCB faculty members are part of an exciting new campus unit known as The Cancer Center at Illinois. The Center is an interdisciplinary collaborative hub with more than 100 faculty members from departments ranging from kinesiology, human development and nutrition to electrical and computer engineering, as well as graduate students and postdoctoral researchers pursuing "cancer-related research." The group has functioned as the Cancer Community at Illinois since 2011.

"Campus support is always helpful to have," said Benita Katzenellenbogen, Professor of Molecular and Integrative Physiology and of Cell and Development Biology, of the center's formal declaration. "It attracts outside money and it attracts additional colleagues. It allows for increased basic, translational, and clinical interactions."

Erik Nelson, assistant professor of molecular and integrative physiology, agrees.

"As a group, we are stronger than as individuals," he said. Nelson, who joined the faculty in 2014, focuses on breast and ovarian cancers. His most recent work found that a byproduct of cholesterol metabolism changes some immune cells and facilitates the growth of breast cancer.

Cancer research has been a major focus within MCB for decades. Faculty, including Katzenellenbogen, Nelson, Milan Bagchi, David Shapiro, David Kranz, K. Prasanth, Ed Roy and others, have long focused on understanding the biology of cancer and how to disarm it. However, cancer is a moving target. Often, once researchers figure out the mechanism of a given cancer and come up with ways to short circuit that mechanism, the tumor mutates or changes

> "As a group, we are stronger than as individuals."

in a way to evade the cure.

"Cancers change as a result of treatment," observes Katzenellenbogen. "That's why there is lots of interest in combination therapies. We want to extinguish cancer with minimal side effects and often combining two or more agents or approaches is best."

For this reason and many others, collaboration has been a byword of MCB's basic research mission for a long time. As advanced techniques, from high throughput screening and CRISPR to imaging modalities have become both more common and more specialized, the creation of the Cancer Center will further streamline and catalyze efforts and strengthen collaborations across campus, across the country, and even around the world.

Bioengineering faculty member and Cancer Center director Rohit Bhargava has been leading the efforts to organize, but many MCB faculty, including Katzenellenbogen, Roy, Bagchi, Nelson and Kranz have helped by serving in leadership and advisory capacities.

"MCB faculty are integral to the success of the Cancer Center by driving its scientific and education programs, as well as collaborations. I am grateful for their service on our steering committee and leadership in establishing the center," said Bhargava.

Under the auspices of the Cancer Center, faculty members have hosted seminar series, meetings and poster sessions to increase campus networking and connections with other places battling cancer, including the Mayo Clinic and Carle Hospital.

MCB faculty collaborate with one another, and they collaborate with imaging experts who have developed "spectroscopic expertise and highly discriminating tools," as well as others in nutrition, for example, such as Zaynep Madak-Erdogan, and with chemists at Illinois, like John Katzenellenbogen, said Benita Katzenellenbogen.

In addition to making campus-wide collaboration more effective and efficient, the Cancer Center represents one of many efforts to make MCB research even more translational than it has been in the past. One way that is happening is by partnering with Carle and Christie clinics, as well as medical centers elsewhere. Carle and Christie have supplied samples and have helped run trials for example, says Katzenellenbogen. The Cancer Center helps integrate and organize these kinds of undertakings with workshops, personnel, and student programs.

The next step for the center is to get National Cancer Institute (NCI) accreditation as a cancer center. As of 2017, there were 49 Comprehensive Cancer Centers, 13 Cancer Centers, and seven Basic Laboratory Cancer Centers. The University of Illinois is applying to be a basic laboratory cancer center. If it gets accredited, it would be the only such center in Illinois. Receiving the NCI-designation places cancer centers among the top four percent of the approximately 1,500 cancer centers in the United States, according to the NCI.

"This is a rigorous process, with different categories of designation," said Nelson, who says the campus group is going for a basic research center designation similar to the one that MIT has.

An external advisory board, comprised of internationally renowned experts, visited the campus recently to evaluate the program and were very impressed with the work being done.

> "With stronger science, we make better progress."

"They were very enthusiastic by what we've done so far," said Nelson. "Both with the science we are doing and the current level of collaborations and our productivity … We showed that we are already rolling on many cancer-related interdisciplinary collaborations and we showed that, while we have some great core facilities, the University of Illinois is positioned to create additional unique and cutting-edge facilities, including one for tumor engineering and phenotyping."

In addition to providing more translational opportunities, The Cancer Center provides special opportunities for students. One such initiative, C*Star (Scholar Translational and Applied Research initiative) scholarships for graduate students, pairs a student with a basic cancer research expert as a primary mentor, together with a clinician. The scholarship is jointly funded by Carle and the University.

This is one of several student opportunities offered through the Cancer Center. Another one, ResearcHStart, is targeted at high school students, who work full time with faculty mentors, including Nelson and MCB professor Kannanganattu Prasanth, who are established cancer researchers.

The new Cancer Center is, above all, a highly collaborative undertaking. And, because MCB faculty members have a long history of extensive and interdisciplinary collaboration, it is a perfect fit. Katzenellenbogen, for example, collaborates with numerous groups, not only on campus, with faculty in bioengineering, food science and nutrition, but also in Korea and France and across the United States.

In her view, interdisciplinary collaboration is, without a doubt, a key way to advance the science of cancer. There is no down side.

"With stronger science, we make better progress," she says. The Cancer Center is part of that progress. •

Benita Katzenellenbogen Lab

Hitting the (Moving) Target of Drug Resistant Cancers

by Deb Aronson

Benita Katzenellenbogen has delved into the causes and treatments for breast cancer and other hormone-dependent cancers for virtually her entire career. Her tireless work in the field has established her as a world-renowned expert.

Lately, Katzenellenbogen, Professor of Molecular and Integrative Physiology and of Cell and Developmental Biology, and her research group are "very much focused on targeted therapies."

Cancer treatment approaches have changed in the past decades. As researchers learn more about cancer biology, including identifying subtypes that respond to different treatments, they have figured out ways to target specific cancer cells with a more precise approach. And the more they understand about the mechanisms at work, the more targeted those treatments have become, meaning that they effectively treat the cancer with greatly reduced side effects.

Of course, targeted therapies are not always a panacea. Even with the exciting results of targeted treatments, some cancers figure out a way to make a comeback. Why and how do cancers fight back? Those cancers that become "treatment-resistant" currently present one of the major challenges in the field. Working to describe the biology by which cancers change in response to these targeted therapies, and looking at certain "aggressiveness factors" that block treatment has motivated Katzenellenbogen's lab for several years.

Of the ER positive cancers that metastasize or resist treatment, 30-40% of them have these mutated ERs. So, finding a way to inhibit them would be a major breakthrough.

Several effective treatments for hormone receptor (HR)-positive cancers (which comprise about 70% of initial breast cancers) block the action of estrogen, turning the estrogen receptor (ER) off and thus thwarting the cancer. In cases where this treatment stops working, it is often because the ER becomes "constitutively active," which means it no longer needs estrogen to turn on. This is one so-called aggressiveness factor, when the ER goes rogue, so to speak.

Katzenellenbogen's lab has been working to understand how it is that the ER becomes constitutively active and then why that makes the cancer more resistant to treatment. Her lab is collaborating with John Katzenellenbogen and his laboratory in the Department of Chemistry to develop anti-estrogen molecules that might be able to inhibit the activity of these mutated estrogen receptors.

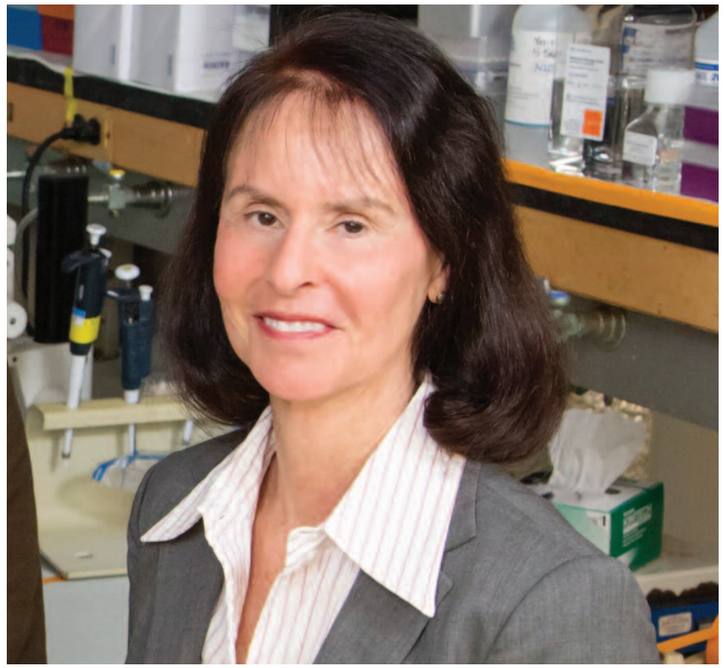
"Of the ER positive cancers that metastasize or resist treatment, 30-40% of them have these mutated ERs," said Katzenellenbogen. So, finding a way to inhibit them would be a major breakthrough.

In another recent finding that holds promise for some of the most difficult-to-treat breast cancers, Katzenellenbogen's lab has found an increase in the expression of the transcription factor FOXM1, another "aggressiveness factor."

In one recent collaboration with oncologists in Korea, Katzenellenbogen's lab observed an upregulation of FOXM1 in treatment-resistant cancers. Typically, FOXM1 is only found at significant levels during embryonic development. When it shows up in cancer, it makes the cancer much more robust and difficult to treat.

"High levels of FOXM1 are associated with less good patient outcomes," said Katzenellenbogen.

Katzenellenbogen is interested in understanding the biology of this transcription factor. FOXM1 appears to be upregulated, not only in some breast cancers but also in other cancers, including colon and prostate that also have become resistant to treatment, as well as some glioblastomas and gastric cancers.



Dr. Benita Katzenellenbogan, Swanlund Professor of Molecular and Integrative Physiology and Cell and Developmental Biology. *Photo by L.. Brian Stauffer*

"We are interested in understanding how FOXM1 works, in other words, what genes does it turn on, and how does that impact the invasiveness of the cells."

"We do know that many genes under FOXM1 regulation are genes associated with proliferation, invasiveness, and resistance to treatments," she added.

As with the constitutively active estrogen receptor project, Katzenellenbogen is collaborating with John Katzenellenbogen's laboratory to develop small molecule inhibitors that can effectively block FOXM1. For example, they are looking for compounds that might block FOXM1's interaction with DNA or in some other way block its activity. Some inhibitory molecules have been identified previously, but they are not active enough or are not selective enough, meaning they interfere with too many other cell activities, says Katzenellenbogen.

One finding that has Katzenellenbogen intrigued is that FOXM1 upregulation is present in triple-negative breast cancers. These are breast cancers that do not respond to hormonal therapies, including tamoxifen and aromatase inhibitors. If the action of FOXM1 can be blocked, that might represent a possible breakthrough in treatment for this especially challenging subgroup of cancer, which comprises about 15% of all breast cancers.

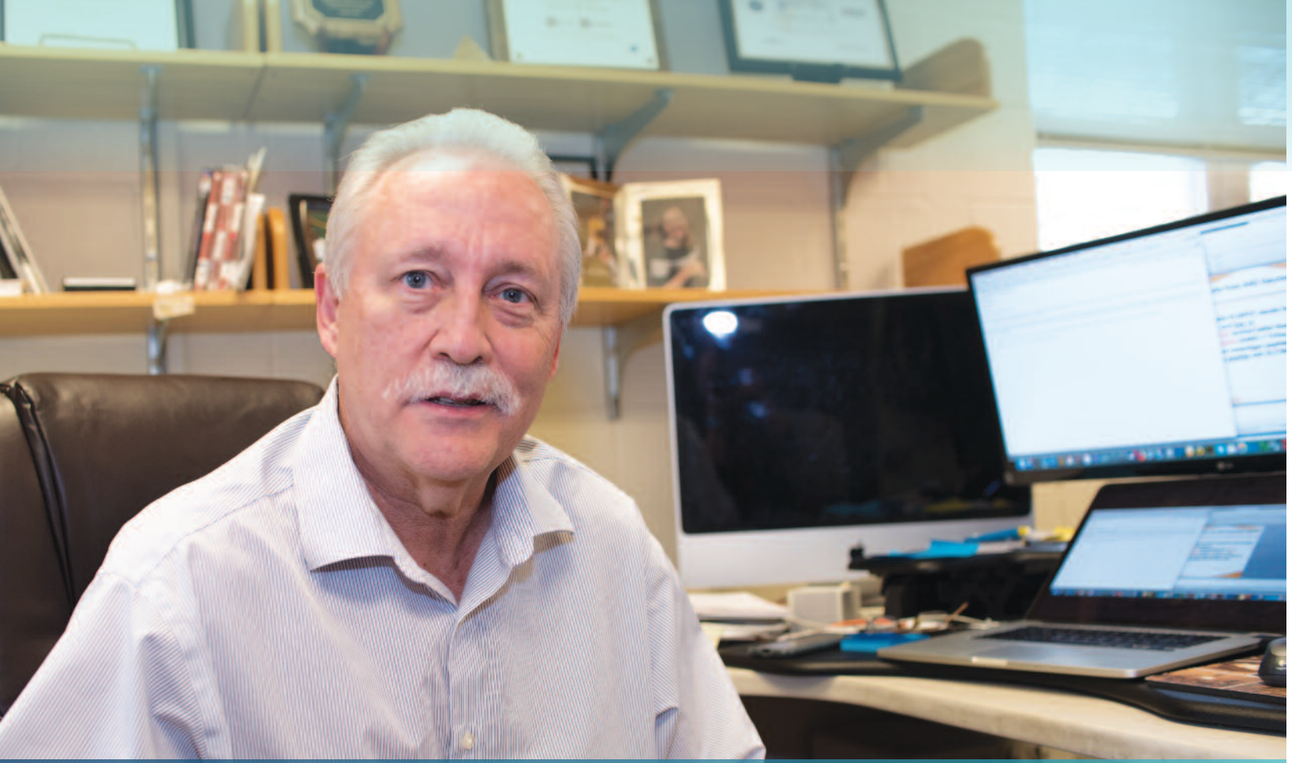
Katzenellenbogen rarely stops to consider how far the field has come, but when she does, it is to appreciate that cancer is no longer the immediate death sentence it once was.

"We have made great progress in trying to understand the main drivers in cancer subtypes and to identify the best targets for therapy," she said. "These days most cancers are considered diseases one survives; they are not always lethal. The numbers of people effectively treated even 20 years after diagnosis continues to increase. Through better sub-typing of cancers we know better the risk of recurrence so people are better monitored and even treated differently."

"These days most cancers are considered diseases one survives; they are not always lethal. The numbers of people effectively treated even 20 years after diagnosis continues to increase."

And that is progress worth celebrating. •

(Professor Katzenellenbogen gratefully acknowledges research support from the Breast Cancer Research Foundation, the Julius and Mary Landfield Cancer Research Fund, and the National Institutes of Health.)



Harnessing and Boosting T-cells to Fight Disease

David Kranz Lab

Dr. David Kranz, Phillip A. Sharp Professor of Biochemistry

by Brian Wallheimer

Last summer, the U.S. Food and Drug Administration approved the first gene therapy treatment for cancer, giving hope to patients who have not benefited from more traditional drug therapies. It's a treatment decades in the making, and some of the early stage work was done in the lab of David Kranz at the University of Illinois.

Kranz, the Phillip A. Sharp Professor of Biochemistry in the School of Molecular and Cellular Biology, has been involved in T-cell research since working as a postdoctoral researcher at the Massachusetts Institute of Technology, involved in early discoveries about how T-cells might be used to combat a host of diseases, including cancer. Prior to this, he trained as a graduate student at Illinois under Ed Voss, who at the time was the only immunologist on campus.

T-cells are involved in the body's immune response, destroying foreign or damaged cells and viruses. In some cases, T-cells don't recognize and attack like they're supposed to. Kranz has spent decades modifying and improving the receptors on T-cells to help them better target diseases and pathogens, improving the body's immune response.

"Immunotherapies are the biggest class of new therapies for cancer by far over anything else," he said.

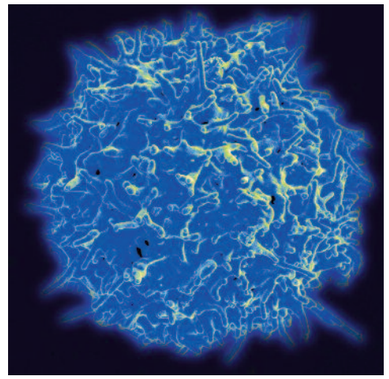
Kranz's early work at MIT included proof-of-concept work for using T-cells to fight diseases like cancer. He and colleagues now know that T-cells can be reprogrammed to fight cancer.

At Illinois, Kranz was involved in developing bi-specific antibodies. The proteins bind to T-cells on one end, and then attach to cancer cells on the other. The antibodies essentially act as guides for T-cells to find cancer cells and then do their natural jobs in destroying those cells.

"People constantly develop abnormal cells, and T-cells recognize and eliminate them. We never know it's even happening. But with cancer, those T-cells don't do their jobs," said Kranz. "We have been looking for ways to harness T-cells to act against cancer, and bi-specific antibodies are one way we've done that successfully."

There is currently one FDA-approved bi-specific antibody developed to treat lymphoma. Many others are in clinical trials.

Since the late 1990s, Kranz's lab has also been working with T-cells for recently approved gene therapy treatments. The technique, called CAR (chimeric antigen receptor) or TCR (T-cell receptor) T-cell therapy, involves removing a patient's T-cells, engineering the cells to express receptors so they will recognize cancer cells, and then putting them back into patients to attack those cancer cells.



Healthy Human T–Cell

"Now you've basically armed the patients with billions of T-cells that can recognize the cancer," Kranz said.

The Kranz lab started working on ways to modify T-cell receptors, some of the basic work that has led to today's cutting-edge treatments.

"We developed the methods that you could actually try to engineer and improve these receptors," Kranz said. "We've received 17 patents, which include methods of engineering receptors as well as some of the receptors themselves."

"We have been looking for ways to harness T-cells to act against cancer, and bi-specific antibodies are one way we've done that successfully."

Many of the patents revolve around a technique called "yeast display," a technique for engineering proteins using the cell walls of yeast to single out antibodies. The technique allowed researchers to create millions of mutated antibodies within days, and then use high-throughput screening to select the best candidates.

Kranz and K. Dane Wittrup (now at MIT), who had been a professor in chemical engineering at Illinois, founded a company called BioDisplay Technologies in 1998. The company used yeast display to develop better antibodies or other proteins such as T-cell receptors. The company was acquired by Abbott Laboratories in 2001.

In 2008, Kranz again founded a biotechnology company called ImmuVen. ImmuVen opened in EnterpriseWorks, initially focusing on developing a therapy for toxins produced by methicillin-resistant *Staphylococcus aureus* (MRSA), but later developing T-cell therapies for cancer. That company was acquired by a major pharmaceutical company at the end of 2014.

Today, Kranz and his lab continue to work with T-cells, CARs, and TCRs but they also are delving into a new research area that is looking at the development of vaccines against cancers. Vaccines are often thought of as a preventative measure against a disease, but the goal of the work is to use them as active therapies.

An overarching goal of these types of therapeutic approaches is to generate memory T-cells in cancer patients. Typically, these cells develop after an immune response to infectious agents, and they kick into action if the pathogen is reintroduced. Children who contract chicken pox, for example, have memory cells that play a role in keeping them from getting the illness again.

Kranz believes T-cells can do the same thing with cancer.

"We want to boost the patient's own T-cells in the body, activating enough of them to 'seek and destroy' the cancer and then convert some of the T-cells into long-lasting memory cells," Kranz said. "If you have memory cells and the cancer comes back, a patient would have the T-cells that can eliminate the cancer."

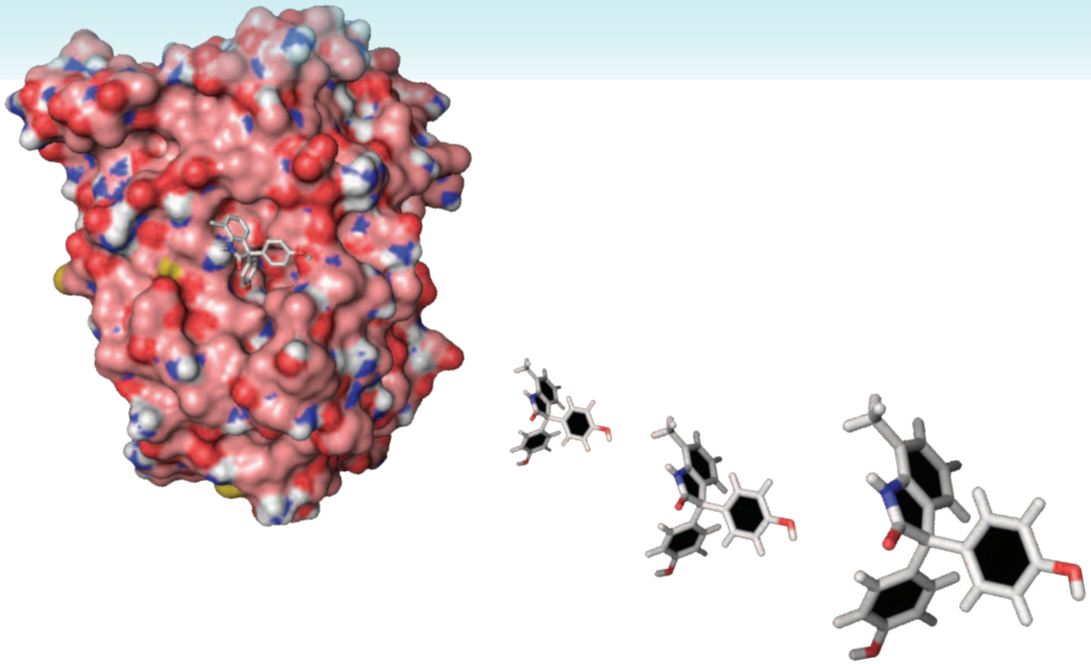
Kranz is working to identify the best cancer antigens to target with potential vaccines. He is now part of the Anticancer Discovery from Pets to People theme at Illinois' Carl R. Woese Institute for Genomic Biology.

Like many scientists whose work touches on cancer, Kranz wasn't originally focused on the disease. Instead, he was fascinated with immunology and the ability to use the body's natural defenses to treat or cure diseases and lessen or eliminate severe side effects such as those experienced by patients receiving chemotherapy.

"The promise of using immunology to fight cancer was clear in the 1980s. You have the potential to let the immune system go in and recognize only the cancer cells and not the healthy cells, eliminating those side-effects," said Kranz.

Over the years, Kranz studied basic molecular immunology and applied his research to autoimmune diseases but shifted focus to cancer because he saw the toll the disease can have on patients and families, including his own.

"As a disease, the need for cancer therapies is so significant that we decided to focus more on it," Kranz said. "In immunology, we just happened to be working on things that you could immediately see would have an impact on cancer." •



Left: Data-derived computer generated model of the binding of the preclinical anticancr drug, BHPI, to its binding site near one end of the estrogen receptor (ER). BHPI binds to ER at a different site than estrogen, tamoxifen and other antiestrogens (model by Mara Livezey). BHPI fits tightly into a binding cavity on the surface of ER. Unlike tamoxifen, BHPI kills breast cancer cells by acting through ER to induce lethal hyperactivation of a normally protective stress response pathway.

David Shapiro Lab

Employing the Help of the BHPI Molecule to Boost Anti-Cancer Drug Treatments

by Deb Aronson

For decades, MCB and the University of Illinois have been at the forefront of understanding the role of estrogen and the estrogen receptor (ER) in causing breast cancer. This is essential work: Breast cancer is the most commonly diagnosed cancer in women, affecting roughly one in eight women in the US alone.

MCB expertise began with the late Jack Gorski, co-discoverer of the estrogen receptor, the protein that estrogen must bind to in order to work. These days MCB faculty studying estrogen and ER include people like Milan Bagchi and Benita Katzenellenbogen, who have been here for a while, and others like Erik Nelson and Kannanganattu and Supriya Prasanth, who are relative new comers.

"MCB has been a major center for hormones and hormone action, especially as it relates to cancer, for many years," says David Shapiro, inaugural Eugene E. Howe Scholar in Biochemistry. Plus, "we work well together, exchange ideas and technologies, and generally get along well."

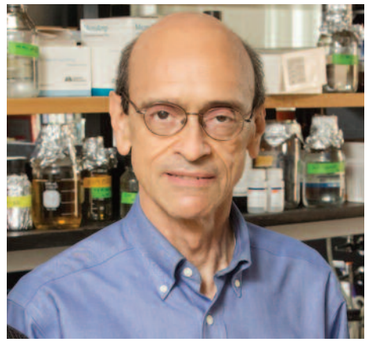
Enormous strides in fighting breast cancer have been made over the last few decades, with drugs that target estrogen production (aromatase inhibitors) and estrogen binding to ER, such as tamoxifen. Thanks in part to these drugs, breast cancer survival rates have improved.

However, although tumors often respond at first, cancer cells can mutate, become resistant and metastasize. Targeting these resistant tumors has become a high priority.

"This is the way of cancer," says Shapiro. "It only very rarely can be eradicated with a single drug. The normal thing in cancer is that typically the tumors become resistant so you come up with different sequential treatments."

Shapiro began his independent research career interested in the basic mechanism of how hormones work. As his work and the work of others began to illustrate how much hormones affected cancer cells, he shifted his focus to cancer. Like many others, Shapiro has lost close family members, including his mother, to cancer.

Cancer research is a crowded field. New tools are continually being developed and with these new tools, new understanding pushes the field forward. Shapiro has always been willing to seek out novel ways to look at and attack cancer. Recently that has involved screening tens of thousands of small molecules, not unlike looking for a needle in a haystack.



Dr. David J. Shapiro, Professor of Biochemistry. *Photo by L. Brian Stauffer*

"About six years ago, I realized that there were 200,000 papers on estrogen and in order to discover something fundamentally different we'd have to try a new approach," he says. With the help of colleagues experienced in using the high-throughput screening facility, Shapiro's group screened 150,000 small molecules.

"Instead of looking at small molecules whose actions we could explain with current knowledge, we'd actually study the small molecules whose actions we could not explain, with a view to identifying entirely new ways estrogen works," he says. "It turned out, happily for us, that the most effective molecule out of the 150,000 we screened worked in this exciting new way and led us to a normal pathway of estrogen action in cancer. That's taken over most of our work."

In other words, they found their needle. It is called BHPI.

In early experiments, BHPI appears to be highly effective, especially against drug-resistant cancers, precisely the cancers that are in Shapiro's cross hairs.

BHPI was the only effective compound from the screen that works through the unfolded protein response (UPR) pathway. The UPR is a protective pathway and is turned on by stress, such as an unfolded or misfolded protein. Cancer cells use this same pathway to protect themselves from being killed by toxic anticancer drugs. If the protective pathway becomes overwhelmed, i.e. more unfolded proteins than it can handle, it revs up to a very high level, triggering a self-destruct process in the cells. Shapiro's lab showed that estrogen bound to ER induces a moderate and protective activation of the UPR pathway in breast and ovarian cancer cells.

"BHPI hijacks this pathway and works through ER to throw the UPR pathway into overdrive, converting it from protective to lethal," says Shapiro.

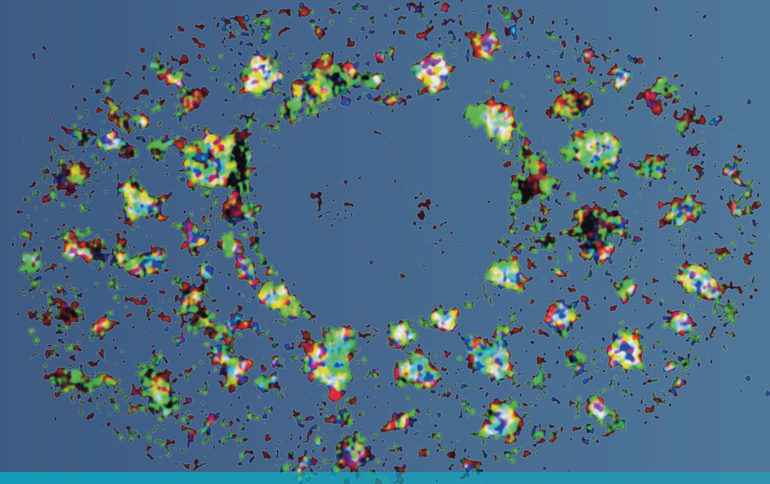
BHPI works even better in cancer cells resistant to anticancer drugs, because the UPR pathway is already turned on as part of the way cancer cells block the toxic action of anticancer drugs. This means that when BHPI turns the UPR up even more, those cells will tip over into the cell death pathway faster than in cells that are not under stress. Because the UPR is typically turned off in normal cells, when BHPI turns it on, it does not tip the UPR activation into the lethal range. This means healthy cells are not affected, says Shapiro.

BHPI also works by shutting down proteins that cells use to pump foreign compounds out of the cell. One way cancer cells become resistant to anticancer drugs is by using these pumps, called multidrug resistance proteins, to pump anticancer drugs out of the cells. Despite years of effort, drugs that directly block the action of the pumps have failed clinical trials. BHPI targets the multidrug resistance pump in a different way. One of BHPI's actions is to open up a compartment within the cell that stores calcium. When the cell becomes flooded with calcium, protein pumps snap into action, pumping the calcium back into the storage reservoir. But because the calcium continues to leak back into the body of the cell, the protein pumps burn through all the cells' energy. Since the multidrug resistance pump also needs energy to work, it basically runs out of gas, sputtering to a stop.

Shapiro's lab, in partnership with Nelson's lab, used a mouse ovarian cancer model that is highly resistant to other drugs. They showed that not only did BHPI starve the multidrug resistance pump, it enabled the original drug to work again, since it was no longer getting pumped out of the cancer cells.

Yet another way cancer outwits anticancer drugs is by mutating the ER. One-third of metastatic breast cancers have mutated estrogen receptors; these changes are associated with resistance to drugs. So in another project, Shapiro used CRISPR gene-editing technology to replace the normal ER with each of the two most common mutations found in metastatic cancer. His team also tagged the cancer cells with the gene from fireflies that enables them to light up. Using state-of-the-art imaging equipment, this allowed the researchers to see, or image, the tumors inside the mice. In a small study, they found that BHPI was exceptionally effective.

BHPI and its cancer fighting potential is easily one of the most exciting findings in Shapiro's long and successful career. The trick now is to continue down the road to clinical trials. Those efforts include collaborating with chemistry professor Paul Hergenrother, whose team is engineering new small molecules that might work even more effectively than BHPI. There are still many complicated and expensive steps ahead, but Shapiro is undaunted. Together with his colleagues, both in MCB and across campus, Shapiro will continue to match wits with cancer. •



Super resolution imaging of a human fibroblast nucleus reveals ordered assembly of oncogenic MALAT1 lncRNA (red), SR splicing protein (blue) and the pre-mRNA splicing U2 snRNA (green) in nuclear speckle sub-nuclear domains.

Rising to the Challenge

K.V. Prasanth Lab Discovers Cancer Connections with MALAT1

by Doug Peterson

Kannanganattu V. Prasanth was a graduate student in India a little over 20 years ago when he was told that no one was interested in long non-coding RNAs (lncRNAs). Some of the professors also lamented that there was little future for studying the function of the so-called "unimportant and junk RNAs."

But he wasn't discouraged.

"Maybe it was my young age, but I wanted to do something that was outside the box," said Prasanth, a University of Illinois MCB professor of cell and developmental biology. Today, not only is he still doing research on lncRNAs, but many other people have jumped on the bandwagon.

"The field of lncRNA is really hot," he said. "Everyone wants to study it," with more than 2,700 papers published on the topic in 2017 alone.

Prasanth's lab published several papers on a particular lncRNA called MALAT1, with significant findings showing an important link between cancer and MALAT1. They found that when MALAT1 is overexpressed in breast cells, the cells form tumors. Conversely, when it is depleted in breast cancer cells, the cells lose the properties necessary to produce tumors.

> MALAT1 is present in normal cells, operating under normal conditions, so cancer cells might be exploiting this to their own benefit.

Prasanth also gives credit to the lab of his wife and fellow MCB researcher, Supriya G. Prasanth, as they collaborate on this and other projects in both of their labs. Supriya's lab is pursuing research on cell organization that has cancer ramifications as well.

David Spector, Prasanth's mentor from his postdoctoral years at Cold Spring Harbor Laboratory in New York, also published a paper in 2016 showing the connection between high levels of MALAT1 and breast cancer. Spector's lab focuses more on drug development to battle breast cancer, making small molecules to inhibit MALAT1, while Prasanth's lab, in contrast, concentrates on basic biology—How exactly does MALAT1 regulate the expression of genes in the genesis of tumors?

"MALAT1 is also present in normal cells, operating under normal conditions, so cancer cells might be exploiting this to their own benefit. That may be why cancer cells are addicted to high levels of MALAT1," said Prasanth, who has been an American Cancer Society Research Scholar since 2011.

His lab has shown that MALAT1 is found to enrich in nuclear speckles, a domain of the cell nucleus. He also discovered that it plays an important role in regulating the activity of a family of pre-mRNA splicing factors known as SR proteins. SR proteins themselves are oncogenic in nature—they have the potential to cause cancerous tumors. Prasanth believes that MALAT1 contributes to cancer progression by regulating the activity of several splicing factors, including SR proteins.

A human cell has about 2,500-3,000 copies of MALAT1 in its nucleus, but a cancer cell shows a two to threefold increase in the copy number of MALAT1, "and that completely changes the dynamics," he says. With so many copies at work, they control more SR proteins in the cell than normal, and the SR proteins, in turn, splice pre-mRNAs of genes that have the potential to cause cancer.

"It's like a puzzle," Prasanth said. "MALAT1 interacts with the SR protein here, and you have the splicing of oncogenic pre-mRNAs there. We're now trying to identify those oncogenic genes whose splicing is regulated by the MALAT1-SR protein axis."

MALAT1 seems to be involved in all subtypes of breast cancer, he adds, but their lab is focused on triple negative breast cancer (TNBC), which is one of the most aggressive forms of breast cancer. Presently, the only option to treat TNBC is through chemotherapy, but the disease becomes resistant to chemo over time, so treatments are limited right now. That's why research to understand the biology of molecules such as MALAT1 in TNBC is crucial in order to develop next-generation drugs.

> We're now trying to identify those oncogenic genes whose splicing is regulated by the MALAT1-SR protein axis.

LncRNA, such as MALAT1, is longer than 200 nucleotides, and it was once considered unimportant, Prasanth says, because biology was a protein-centric world 20 years ago. The central dogma of molecular biology says that genetic information flows from DNA to RNA to make proteins, which do all of the heavy lifting in cells. LncRNA upended this dogma because it does not code for proteins and yet it still performs numerous vital functions in a cell.

Back in 1995, researchers were aware of only a few lncRNAs, but today they have identified over 20,000, and they have pinpointed the function of several hundred of them.

Prasanth started his work on MALAT1 in 2005 while in Spector's lab at Cold Spring Harbor, but with his mentor's blessing he brought this research to the U of I when he and his wife came to Urbana as MCB professors in 2007. Today, Prasanth and Supriya have labs side-by-side in the Chemical and Life Sciences Building, and their students work together as one unit, even though each lab pursues its own unique projects. As Supriya and Prasanth continue to collaborate extensively on several ongoing research projects, they also challenge each other.

"That's the way science works," Prasanth poined out. "You always need to be challenged." •

Dr. K.V. Prasanth, Professor of Cell and Developmental Biology

Lin-Feng Chen Lab

Taking a Deep Look at Protein Structures that Contribute to Cancer

by Steph Adams

Thirteen years ago, when Lin-Feng Chen joined the Department of Biochemistry, he had his sights set on a specific protein that could both contribute to the growth of cancer and be a major factor in boosting the body's immune system.

"The protein in question was NF-kappaB (nuclear factor kappa-light-chain-enhancer of activated B cells), a kind of "master controller" of innate and adaptive immune response, and cell survival," explains Chen.

In healthy cells, it spends most of its life in the cell's cytoplasm, quietly awaiting orders to assist the immune system. In response to viral and bacterial infection, NF-kappaB moves into the nucleus and triggers the expression of many genes that help to fight against infection. After eliminating the pathogens, NF-kappaB returns to the cytoplasm, awaiting the next order. However, NF-kappaB can work against the body's best interests, too. Sustained nuclear NF-kappaB is associated with a variety of inflammatory diseases and cancers.

"These are exciting findings. This is very translational research."

Dr. Chen's postdoctoral work at the Gladstone Institute of Virology and Immunology at University of California at San Francisco focused on the post-translational regulation of NF-kappaB in immunity and cancer. What chemical modifications were needed to make NF-kappaB dormant, until needed, in healthy cells and run amok in diseased cells?

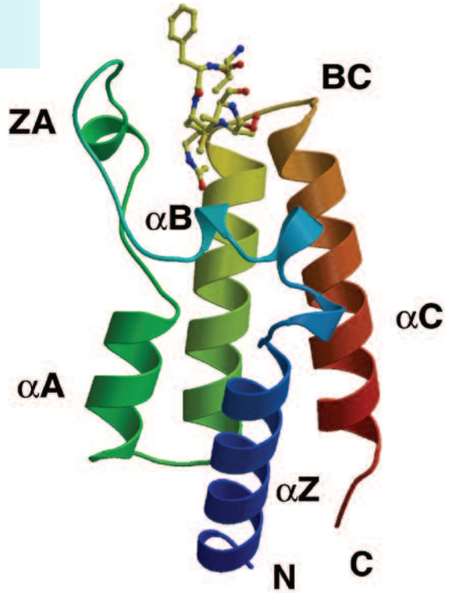
That work identified acetylation, a chemical tag that can be added to lysine, of NF-kappaB that dictates the transcriptional outcome of NF-kappaB. So the major question when Dr. Chen started his lab at the University of Illinois was to find out how the modification of lysines affected the activity of NF-kappaB.

At that time there were some clues that the acetylated lysine can control the protein's activity through a signaling partnership with a bromodomain, a pocket-like structure, that can accommodate acetylated lysine and change the properties of the proteins.

"We stared to look for bromodomains that might recognize the acetylated lysine of NF-kappaB and proteins that contain such a domain could be working with NF-kappaB," said Chen. In 2009, Chen's group identified bromodomain-containing protein 4 (BRD4) as a co-activator of NF-kappaB, specifically recognizing the acetylated lysine of NF-kappaB via its bromodomains.

This discovery launched a decade of work in Chen's lab focusing on BRD4's role in inflammation and cancer.

BRD4 belongs to a class of molecules that can recognize specific chemical tags on other proteins to spur the marked proteins to perform various tasks. Chemical tag "readers" such as BRD4 are important players in the field of epigenetics, which focuses on how specific genes are regulated.



Binding of bromodomain 2 of BRD4 to acetylated lysine of NF-kappaB

Dr. Lin-Feng Chen, Professor of Biochemistry

"In epigenetics, there are writers, there are readers, and there are erasers. BRD4 is the reader," Chen said. The writers and erasers add or remove tags to or from proteins, without changing the underlying sequence of the gene that codes for them. Drugs targeting epigenetic regulators have emerged as novel therapies in cancer treatment, and a number of small-molecule inhibitors against epigenetic regulators have already been developed for cancer therapy.

Exploring the role of BRD4 in cancer, Chen's group found that BRD4 prevents the degradation of nuclear NF-kappaB by binding to acetylated NF-kappaB, and contributing to the sustained presence of NF-kappaB in the nucleus of cancer cells. They also found that a small molecule, JQ1, which blocks the interaction between BRD4 and NF-kappaB, reduced proliferation of cancer cells and suppressed the ability of cancer cells to form tumors.

"These findings opened exciting possibilities for translational research. The interaction between BRD4 and NF-kapapB could be a target for therapies to stop the spread of cancer or inflammatory disease," said Chen.

Chen's lab recently discovered that there was a higher expression of BRD4 in gastric cancer cells and in gastric cancer patient samples. They identified an enzyme, PIN1, the expression of which was highly correlated to BRD4 in gastric cancer cells and regulated the tumor-promoting activity of BRD4 in gastric cancer cells. They also found that BRD4 promoted gastric cancer cell proliferation by preventing the cell senescence.

Chen's latest research is delving even deeper into the physiological role of BRD4 in cancer and inflammation. A recent paper in *Oncogene* demonstrated that removing BRD4 from specific mouse tissues compromised the mice's innate immunity to find against bacterial infection and tumor. With these tissue specific BRD4-deficient mice, they have tried to determine the contribution of BRD4 in inflammatory diseases and inflammation-associated cancer.

Various inhibitors targeting BRD4 are undergoing clinical trials for the treatment of cancer and inflammatory diseases. "Our studies will provide new insights for developing therapies targeting BRD4," said Chen. "This is very translational research."•

BROTHER-IN-LAW

Exploring RNA's Binding Role in Liver Disease and Cancer

Auinash Kalsotra Lab

by Brian Wallheimer

Auinash Kalsotra wasn't originally interested in studying cancer. In fact, he wasn't looking to study any particular disease at all.

Kalsotra wanted to know how the complex machinery of our bodies takes the codes embedded in our DNA and turns them into healthy organ tissues, specifically heart and liver. In other words, he wanted to know how things worked — not why they break or how to fix them.

"There's a molecular logic to how cells are put together, how something becomes a nerve or muscle cell," Kalsotra said. "Once these cells have features, how do they mature and give rise to a functioning adult tissue that carries out a particular function?"

But as often happens, scientists start on one path and eventually find themselves pulled onto others. In this case, Kalsotra's search for a deeper understanding of cellular mechanisms has led to significant findings that show linkages between liver diseases and cancer and may improve our understanding of the origin of these diseases.

Kalsotra's research has focused on RNA, the workhorse cousin of DNA, which makes up the genetic instructions for living things. RNA is often viewed as important for messaging and other functions, taking DNA's code and transporting it to and carrying out functions in cells. It's often taken a back seat to DNA in terms of significance.

In particular, Kalsotra is intrigued by the fact that one gene can give rise to multiple types of RNA based on the type of cell it will encode. A long stretch of RNA will be cut into pieces, called exons, which are spliced together to form messenger RNA (mRNA). The pieces spliced together, their order and other factors determine the type of function the RNA will carry out.

"That means one gene can give rise to multiple mRNAs, and those mRNAs are dependent on the cell type," Kalsotra said.

Kalsotra knew that RNA binding proteins played a role in the formation of mRNA, so his lab started knocking out these proteins from mouse livers to study their functions.

In liver cells, mice that lost certain RNA binding proteins started displaying signs of fatty liver disease. The condition is common among people who abuse alcohol, but also affects more than 100 million people in the U.S. and more than a quarter of people in the non-developed world as non-alcoholic fatty liver disease.

"One-third of people with non-alcoholic fatty liver disease get an aggressive form of the disease," Kalsotra said. "In addition to having fat in their livers, they're going to have a lot of inflammation, scarring and liver cell death."

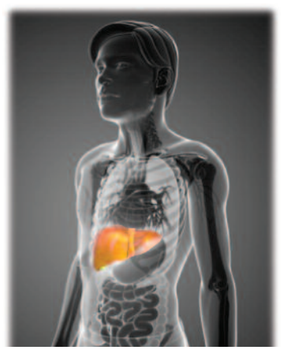
The mice eventually developed liver cancer. It got Kalsotra's lab looking for connections between genes and non-alcoholic fatty liver disease. Analysis revealed that the genes that ranked highest as likely contributing to the disease are associated with RNA splicing.

"Our goal in knocking out these genes was to understand how these cells mature and how RNA splicing plays a role in tissue development," Kalsotra said. "Instead, what we saw was a very specific disease that seems to match a human phenotype."

Kalsotra collaborated with the Carle Hospital to analyze biopsy samples of patients with non-alcoholic fatty liver disease. Each patient showed a striking decrease in RNA binding proteins.

The findings raise more questions, such as whether the loss of RNA binding proteins leads to the liver disease and cancer. It's possible that loss of RNA binding proteins could be a biomarker for fatty liver disease and cancer. Or maybe the opposite is true, that loss of the binding proteins is a result of the liver diseases.

Dr. Auinash Kalsotra, Professor of Biochemistry



The Kalsotra Lab has discovered that deletion of a splicing factor results in spontaneous development of nonalcoholic Steatohepatitis (NASH) in mice revealing an unexpected link between splicing deregulation and fat metabolism in the liver.

"Maybe one day, if we understand how normal cells work, we can understand what goes wrong in a cancer cell that makes it divide uncontrollably and not listen to the rest of the organ that is telling it to stop growing."

Kalsotra also wants to know if and how the cancer profiles in mice match with humans, who tend to get cancer after fatty liver diseases. And whether there are mutations in genes from birth that lead to liver disease and cancer, or whether there are environmental triggers, such as diet, that are involved.

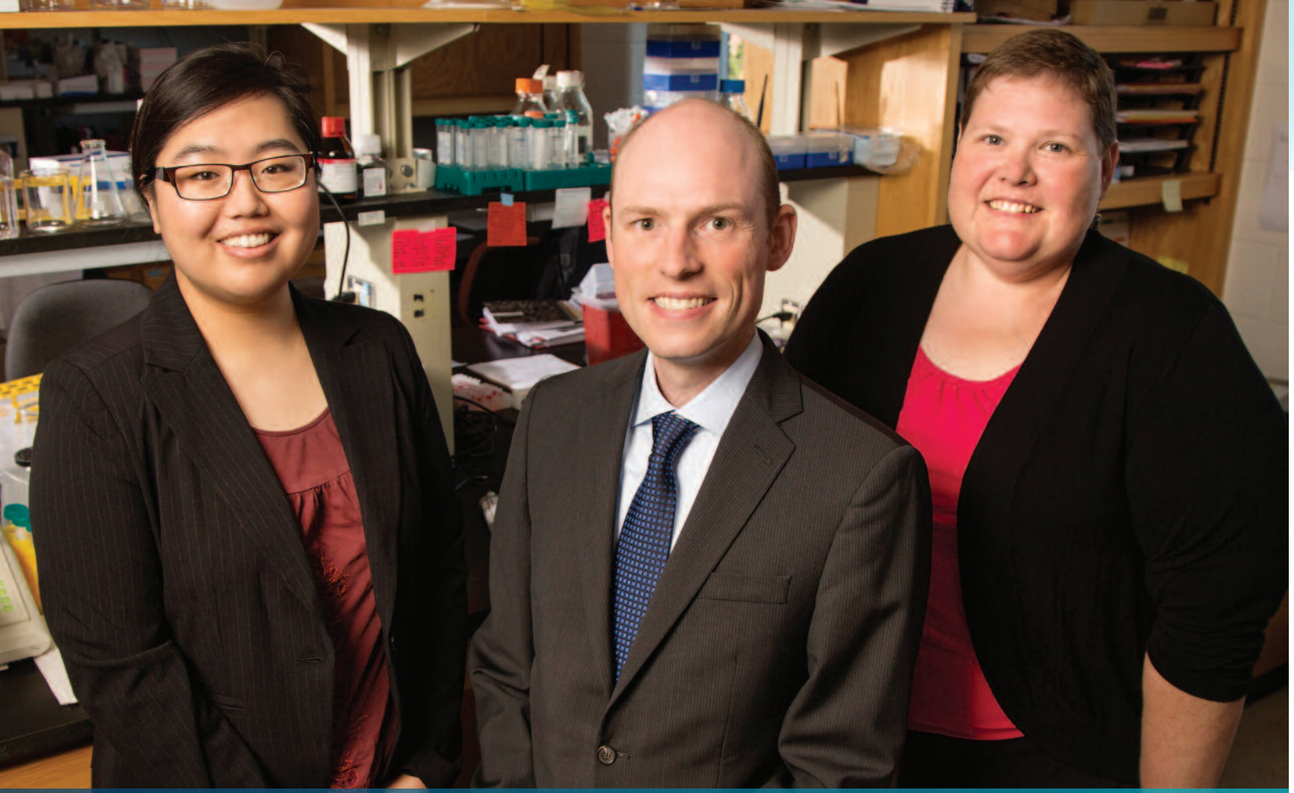
In addition, the Kalsotra Lab is looking at how the transition from the body making fetal cells to making adult cells might play a role in cancer formation. Fetal cells rapidly proliferate, much like cancer cells. But at some point, the body switches to making adult cells — a few years after birth in humans and a few weeks in mice.

That switch coincides with the creation of adult versions of mRNAs from the same genes. It's possible, then, that a malfunction in the production of these "adult mRNAs" could reinitiate cell proliferation, a hallmark of cancer.

"What we're seeing is that it may not only be at the level of mutation in a gene that controls cell proliferation or activating an oncogene that gives a cancer cell an advantage to grow, but also what kind of mRNAs you make," Kalsotra said. "If you impinge on that regulation, you now produce the wrong kinds of mRNAs that might not be good for that cell at that stage."

While Kalsotra wasn't aiming to study cancer, there is a sense of satisfaction knowing that his work might have an impact on the disease. His brother-in-law has advanced-stage esophageal cancer, and the work has become somewhat more personal. His overall goal is to understand the proper function of RNA splicing, but there is an incentive to further exploring the malfunctions as well.

"It kind of gives me extra motivation to see why normal cells all of the sudden become cancerous," he said. "Maybe one day, if we understand how normal cells work, we can understand what goes wrong in a cancer cell that makes it divide uncontrollably and not listen to the rest of the organ that is telling it to stop growing." •



Cholesterol Byproduct Hijacks Immune Cells, Lets Breast Cancer Spread

by Liz Ahlberg Touchstone, News Bureau

Postdoctoral researcher Amy Baek, professor Erik Nelson and breast cancer survivor Sarah Adams. *Photo by L. Brian Stauffer*

High cholesterol levels have been associated with breast cancer spreading to other sites in the body, but doctors and researchers don't know the cause for the link. A new study by University of Illinois researchers found that the culprit is a byproduct of cholesterol metabolism that acts on specific immune cells so that they facilitate the cancer's spread instead of stopping it.

The study, published in the journal *Nature Communications*, identifies new potential drug targets that could inhibit the creation or actions of the dangerous cholesterol byproduct, a molecule called 27HC.

"By inhibiting the enzyme that makes 27HC, we found a suppressor effect on breast cancer metastasis."

"Breast cancer impacts roughly 1 in 8 women. We've developed fairly good strategies for the initial treatment of the disease, but many women will experience metastatic breast cancer, when the breast cancer has spread to other organs, and at that point we really don't have effective therapies. We want to find what drives that process and whether we can target that with drugs," said Erik Nelson, a professor of molecular and integrative physiology who led the study.

Nelson's group fed mice with breast cancer tumors a diet high in cholesterol. The researchers confirmed that high levels of cholesterol increased tumor growth and metastasis, and that mice treated with cholesterol-lowering drugs called statins had less metastasis. Then they went further, specifically inhibiting the enzyme that makes 27HC during cholesterol metabolism.

"By inhibiting the enzyme that makes 27HC, we found a suppressor effect on breast cancer metastasis. This suggests that a drug treatment targeting this enzyme could be an effective therapeutic," said Amy Baek, a postdoctoral researcher at Illinois and the first author of the paper.

The researchers also saw unusual activity among specific immune cells—certain types of neutrophils and T-cells—at metastatic sites high in 27HC.

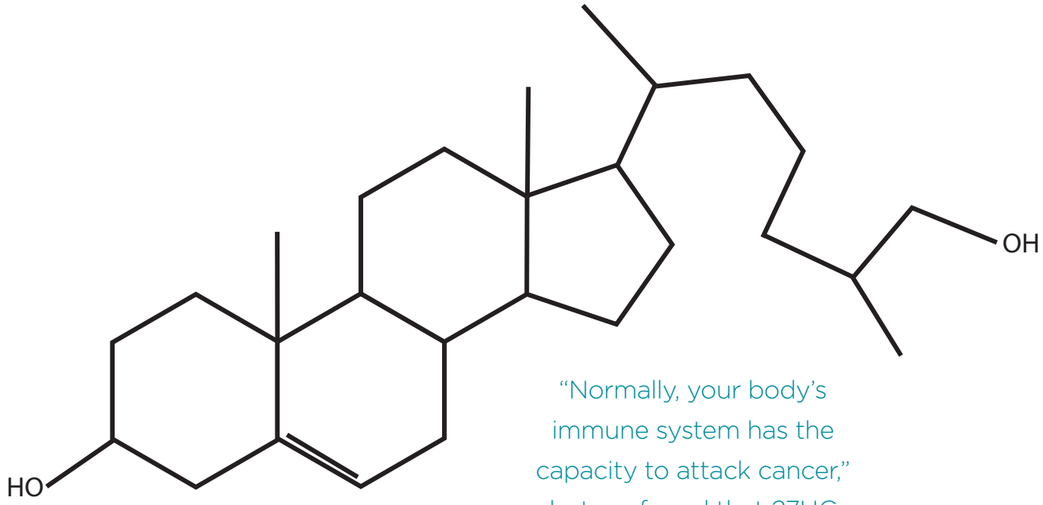
"Normally, your body's immune system has the capacity to attack cancer," Nelson said, "but we found that 27HC works on immune cells to fool them into thinking the cancer is fine. It's hijacking the immune system to help the cancer spread."

Because 27HC acts through the immune system, and not on the breast cancer itself, the researchers believe their findings have broad applicability for solid tumors. They performed experiments looking at colon cancer, lung cancer, melanoma and pancreatic cancer, and found that 27HC increased metastasis for all the tumor types, suggesting that a treatment targeting 27HC could be effective across multiple cancer types.

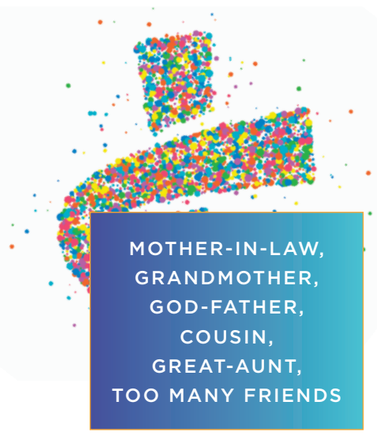
The researchers are working to further understand the pathway by which 27HC affects the immune cells. With clinical partners at Carle Foundation Hospital in Urbana, the team is working to establish whether 27HC has the same pathway in human patients as in mice.

"We hope to develop small-molecule drugs to inhibit 27HC," Nelson said. "In the meantime, there are good cholesterol-lowering drugs available on the market: statins. Cancer patients at risk for high cholesterol might want to talk to their doctors about it."

Nelson also is affiliated with the Cancer Center, the division of nutritional sciences, and the Carl R. Woese Institute for Genomic Biology at Illinois. The National Institutes of Health and the Susan G. Komen Foundation supported this work. •



"Normally, your body's immune system has the capacity to attack cancer," but we found that 27HC works on immune cells to fool them into thinking the cancer is fine."



Alumni Honors

Biology Alumnus Receives Illini Comeback Award

Robert Gaynes, a distinguished doctor known for his effective teaching and mentorship of medical students, has been chosen as a 2017 Illini Comeback Guest by the University of Illinois Alumni Association.

Robert P. Gaynes (BS, '75, biology-honors), professor of medicine and infectious diseases at Emory University, professor of epidemiology at the Rollins School of Public Health, and an attending physician at the Atlanta Veterans Medical Center, said that it's a thrill to be coming back to campus.

"There were a couple decisions I made as an undergraduate student at the University of Illinois that I reflect back on as being quite influential in my life," Gaynes said..

After receiving his degree from Illinois, Gaynes went on to receive his medical degree from the University of Chicago Pritzker School of Medicine. Since then, Gaynes has served as a consultant to the World Health Organization and the Joint Commission on Accreditation of Healthcare Organizations, earned induction into the Academy of Medical Educators for his teaching and mentorship of medical students, and hosted the Centers for Disease Control and Prevention's (CDC) Morbidity & Mortality Weekly Report podcast.

Gaynes said majoring in the Honors Biology Program gave him a solid academic background in the field, as well as an opportunity to work closely with others. As an undergraduate, Gaynes and 13 other students spent an entire three semesters taking courses together under the guidance of one professor.

"We all got to know each other quite well. We learned basic biology and learned critical thinking," Gaynes said. "It really was collaborative science. Medicine today is a team sport — you really work with a variety of allies and medical professionals. That collaborative approach is something I got out of Honors Biology." •

Microbiology Alumna Wins the 2018 Breakthrough Prize in Life Sciences

Joanne Chory (PhD Microbiology '84) is currently a plant biologist at the Salk Institute for Biological Sciences.

Chory, one of the world's preeminent plant biologists who is now leading the charge to combat global warming with plant-based solutions, has been awarded a 2018 Breakthrough Prize for her pioneering work deciphering how plants optimize their growth, development and cellular structure to transform sunlight into chemical energy.

The prestigious award, founded in 2013 by Silicon Valley luminaries Sergey Brin and Anne Wojcicki, Mark Zuckerberg and Priscilla Chan, and Yuri and Julia Milner, honors top achievements in life sciences, physics and mathematics.

Chory, a professor and director of the Plant Molecular and Cellular Biology Laboratory at the Salk Institute for Biological Studies, received the prize, which included a $3 million award, on December 3 at a televised event at the NASA Ames Research Center in Mountain View, California.

"By celebrating science and recognizing its importance to our world, the visionary founders of the Breakthrough Prize are having a significant impact on promoting life-changing discovery and encouraging bright young minds to bring their talents to these exciting fields," says Chory, who is also a Howard Hughes Medical Institute Investigator and holder of the Howard H. and Maryam R. Newman Chair in Plant Biology. "I'm truly honored to receive this award, humbled to be in such distinguished company and tremendously gratified that the study of plants, which is essential to developing everything from better agricultural practices to mitigating global warming, has been put in the spotlight with this award."

Chory joined the faculty of the Salk Institute in 1988 as one of the first plant biologists at the Institute. In 2003, she was named Scientific American's Research Leader in Agriculture, and in 2016 she made Thomson Reuter's list of the World's Most Influential Scientific Minds. She is a member of the U.S. National Academy of Sciences, the German National Academy of Sciences (Leopoldina), the American Philosophical Society, the American Academy of Arts and Sciences, and is a fellow of the American Association for the Advancement of Science. She also is a foreign member of the Royal Society of London and a foreign affiliate of the French Academy of Science.

Dr. Chory did her PhD research in the lab of Samuel Kaplan, former Chair of Microbiology and Director of the School of Life Sciences. •

Alumnus Thomas Cycyota Receives American Association of Tissue Banks Award

Cycyota received the Jeanne C. Mowe Distinguished Service Award, which recognizes an individual who has made a significant contribution in tissue banking or transplantation, whether in research, education, or laboratory improvement, or who has served the Association or the field of tissue banking.

Biology alumnus ('80), Thomas Cycyota is the President and CEO of AlloSource, one of the nation's largest providers of cartilage, bone, skin, soft-tissue and cellular allografts for use in surgical procedures and wound care to advance patient healing.

Under his leadership, the organization has grown into a leading provider of allografts for use in spine, sports medicine, foot and ankle, orthopedic, reconstructive, trauma and wound care applications. AlloSource continues to identify new opportunities to create innovative products that advance patient healing.

In 2015 Cycyota was also awarded the LAS Alumni Humanitarian Award. He considers the donation of tissue to be a "sacred gift." "Everybody understands organ donation because a heart of kidney saves somebody's life," he said. "But with tissue donation, a donor who has passed away can affect hundreds of people." •

Faculty Honors

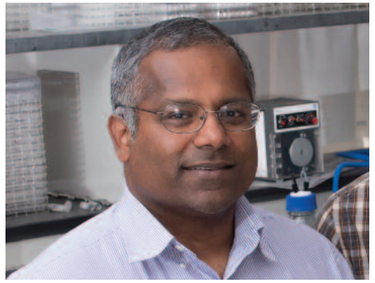
Emad Tajkhorshid Named the J. Woodland Hastings Endowed Chair in Biochemistry

Emad Tajkhorshid is a professor of biochemistry, biophysics and quantitative biology, pharmacology, and bioengineering. He is also interim head of the Department of Biochemistry and is the director of the NIH Biotechnology Center for Macromolecular Modeling and Bioinformatics..He was chosen for the award by a committee of his senior colleagues who hold endowed positions. He is a member of the Beckman Institute for Advanced Science and Technology.

This endowed chair is named for the late John Woodland "Woody" Hastings (1927-2014), and set up by donors George and Tamara Mitchell. Hastings was a decorated scholar who served as a faculty member at Illinois from 1957-66 and was best known for his innovation in the field of bioluminescence. He was also a founder in the field of circadian biology, focusing on the biological cycles of plants, animals, fungi, and other living things.

Tajkhorshid has authored over 180 research articles, including over 17,500 citations in high-profile journals such as Nature, Science, eLife, and PNAS. He has delivered nearly 150 invited lectures at international meetings, universities, and research institutes. He serves on the editorial boards of multiple journals, including Biophysical Journal, Journal of Biological Chemistry, and PLoS Computational Biology.

"Most importantly, I would like to acknowledge my students, post-docs and colleagues," Tajkhorshid said. "They are the reason I'm standing here, presenting what they have been doing for many, many years; I really appreciate and enjoy working with them."



Satish Nair Named I.C. Gunsalus Professor in the College of Liberal Arts & Sciences

Satish Nair, a professor of biochemistry in the School of Molecular and Cellular Biology and director of the Center for Biophysics and Quantitative Biology, is a leader in studying how bacteria can make antibiotics and other medicinally relevant molecules.

The professorship is named in honor of the late I.C. Gunsalus, who joined the University of Illinois as a professor of microbiology in 1950. In 1955, he became head of the biochemistry division in the Department of Chemistry. He taught and researched at Illinois for 32 years.

Nair's lab conducts research that allows for certain drugs to be produced in test tubes and facilitates production of drug derivatives that do not exist in nature. This research will help develop new classes of therapeutics for the treatment of infections from drug-resistant bacteria. Nair has served on various review panels around the world, and his laboratory has trained more than 50 undergraduate students, almost all of whom have gone on to advanced careers in science.

"This professorship means a lot to me and one of the things it provides is independence for work that is not yet funded," Satish said. "My lab is very interested in expanding what we do, and moving on to doing new things and moving on to new areas of science is not something you'd get money for except for this endowed appointment."

Llano Becomes Professorial Scholar

Dan Llano, associate professor in molecular and integrative physiology and member of Beckman's Neurotechnology of Memory and Cognition Group, was recently appointed the Benjamin R. and Elinor W. Bullock and Edwin E. and Jeanne Bullock Goldberg Professorial Scholar in the Department of Molecular and Integrative Physiology in the School of Molecular and Cellular Biology.

Dr. Edwin Goldberg, M.D., and Dr. Jeanne Bullock Goldberg, M.D., established the professorship to enhance the opportunity for University of Illinois faculty scholars to conduct research that is novel, translational, and sustainable.

Dr. Llano is a leader in his field investigating the neuronal circuits in the midbrain, thalamus, and cortex that participate in the processing of auditory information. He has developed a well-respected research program designed to understand cellular and network mechanisms of auditory processing and alterations associated with pathology of this system. His laboratory has made several innovative advances to study the complex interaction between key areas of auditory function. In addition, he has used computational approaches to construct predictive models that can be used to better understand normal and pathological functions of the auditory circuitry. Using these tools, Dr. Llano has made significant contributions to the field through fundamental new discoveries regarding the structural and functional organization of auditory feedback pathways and through the generation of novel hypotheses regarding the underlying information processing principles. His studies also established the auditory cortex as a primary site of pathology during both normal aging and in the setting of tinnitus (ringing in the ear). •

Team Discovers How Bacteria Exploit a Chink in the Body's Armor

by Steph Adams

Scientists have discovered how a unique bacterial enzyme can blunt the body's key weapons in its fight against infection.

Researchers at the University of Illinois at Urbana-Champaign and Newcastle University in the U.K. are investigating how infectious microbes can survive attacks by the body's immune system. By better understanding the bacteria's defenses, new strategies can be developed to cure infections that are currently resistant to treatments, the researchers said.

The study, reported in the journal PLOS Pathogens, focused on the bacterium *Staphylococcus aureus*, which is found on approximately half of the population. While it usually safely coexists with healthy individuals, *S. aureus* has the ability to infect nearly the entire body; in its most pathogenic form, the bacterium is the so-called "superbug" methicillin-resistant *S. aureus,* or MRSA.

The human body uses a diverse array of weapons to fight off bacteria like *S. aureus*. "Our immune system is very effective and prevents the majority of microbes we encounter from causing infections," said U. of I. microbiology professor Thomas Kehl-Fie, who led the study with Kevin Waldron, of Newcastle University. "But pathogens such as *S. aureus* have developed ways to subvert the immune response."

S. aureus can overcome one of the body's key defenses, nutritional immunity, which prevents bacteria from obtaining critical nutrients. It starves *S. aureus* of manganese, a metal needed by the bacterial enzyme superoxide dismutase, or SOD. This enzyme functions as a shield, minimizing the damage from another weapon in the body's arsenal, the oxidative burst. Together, the two host weapons usually function as a one-two punch, with nutritional immunity weakening the bacteria's shields, enabling the oxidative burst to kill the bacterium.

S. aureus is particularly adept at causing devastating infections. Differing from other closely related species, *S. aureus* possesses two SOD enzymes. The team discovered that the second SOD enhances the ability of *S. aureus* to resist nutritional immunity and cause disease.

"This realization was both exciting and perplexing, as both SODs were thought to utilize manganese and therefore should be inactivated by manganese starvation," Kehl-Fie said.

The most prevalent family of SODs, to which both of the *S. aureus* enzymes belong, has long been thought to come in two varieties: those that are dependent on manganese for function and those that use iron.

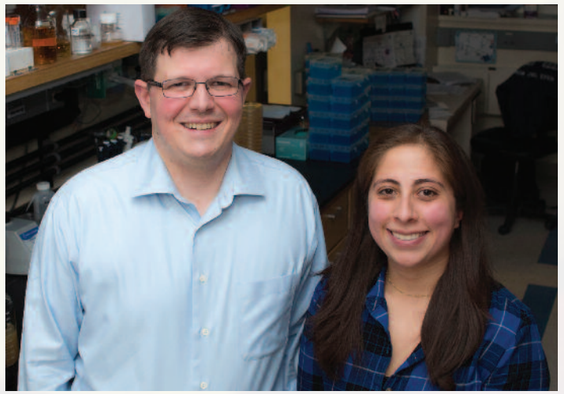
In light of their findings, the team tested whether the second staphylococcal SOD was dependent on iron. To their surprise, they discovered that the enzyme was able to use either metal. While the existence of these cambialistic SODs, capable of using both iron and manganese, was proposed decades ago, the existence of this type of enzyme was largely dismissed as a quirk of chemistry, unimportant in real biological systems. The team's findings dispel this notion, demonstrating that cambialistic SODs critically contribute to infection.

"We found that, when starved of manganese by the body, *S. aureus* activated the cambialistic SOD with iron instead of manganese, ensuring its critical bacterial defensive barrier was maintained," said Yuritzi Garcia, senior graduate student and a lead author on the study. "By learning how *S. aureus's* defenses work we are able to better understand the inner workings of this human pathogen," she said.

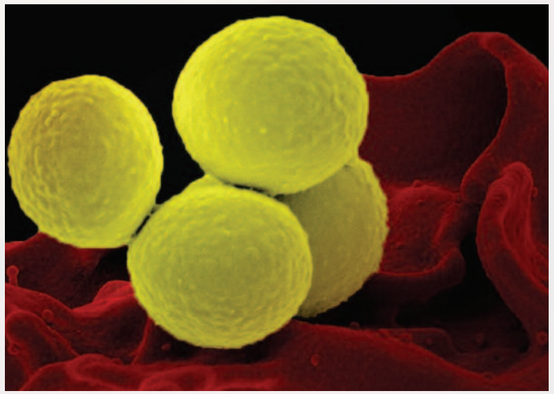
"The cambialistic SOD plays a key role in this bacterium's ability to evade the immune defense," Waldron said. "Importantly, we suspect similar enzymes may be present in other pathogenic bacteria. Therefore, it could be possible to target this system with drugs for future antibacterial therapies."

The emergence and spread of antibiotic-resistant bacteria, such as MRSA, make such infections increasingly difficult, if not impossible, to treat.

This has prompted leading health organizations, such as the Centers for Disease Control and Prevention and the World Health Organization, to issue an urgent call for new approaches to combat the threat of antibiotic resistance. •



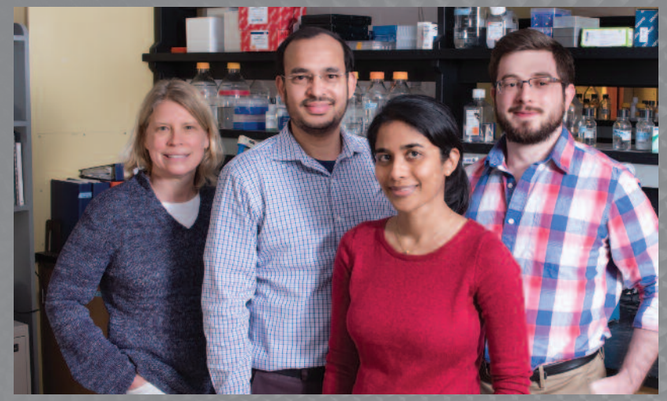
Dr. Thomas Kehl-Fie, Assistant Professor of Microbiology and Yuritzi Garcia, graduate student



Staphylococcus aureus, in yellow, interacts with a human white blood cell. *Photo courtesy the National Institute of Allergy and Infectious Disease*

RNA-Binding Protein, Mov10, is Key to Both Survival and Brain Function

by Serina Taluja



Dr. Stephanie Ceman (cell and developmental biology), Dr. Auinash Kalsotra (biochemistry), Dr. Geena Skariah (Ceman lab), and Joseph Siemetz (Kalsotra lab)

A study led by Dr. Geena Skariah, a recent Neuroscience graduate of the Ceman lab in Cell and Developmental Biology, and current postdoctoral researcher at the University of Michigan, revealed the importance of the protein Mov10 (Moloney leukemia virus 10) in neurological development in animals. The findings were published in BMC Biology.

The study began with an idea from fellow graduate student in the Ceman lab, Miri Kim. "Why don't you look at the brain over time and see what happens with the protein Mov10?" she asked.

"It was a novel question because Mov10 has been characterized only in cell lines as an RNA helicase and as a regulator of retrotransposons," said Dr. Stephanie Ceman. "No one was really looking at its effect on the brain."

Skariah was intrigued. She walked over to the lab of Dr. Auinash Kalsotra, a biochemist who, along with Dr. Ceman, focuses on RNA biology. Skariah knew that the Kalsotra lab was working on cytosolic poly(A)-binding protein 1 (PABPC1) in the livers and hearts of mice and asked if they would be willing to share resources.

"We both share the interest in how RNA brings things together and makes the cells and tissues in our bodies work," said Dr. Kalsotra.

From the resources available in the Kalsotra lab, Skariah was able to test post-mortem brains of mice at different points in their development and measure the level of Mov10.

"We saw that Mov10 goes up during development and goes back down in the adult," said Skariah. "That was my first piece of real data."

No one had ever looked at Mov10's role in the development of the brain, and this was a completely new finding. "That finding started off this whole project," said Skariah.

Mov10 was thought to be unimportant to brain function because there are usually low levels of it found in adult brain.

"I didn't think they were going to see anything. I didn't say not to do it, but I did say it was not going to work," admitted Dr. Ceman, with a laugh.

But Skariah was able to convince Dr. Ceman with the impact of her first results.

"That's great!" Ceman said. "Now test it in different sexes and with different strains."

"Every single day. Over and over and over!" quipped Dr. Kalsotra.

The initial finding opened the door to a cascade of questions. Why is this protein going up in early development? What is it is doing in the brain?

"What's fun about this," said Ceman, "it's the biggest discovery project I have been part of in a long time." Every time they did an experiment in this project, they had no idea what they were going to find.

Further experiments showed that reduced levels of the protein resulted in behavioral changes that suggested neurological impairment. They were seeing several defects in the cells, but where were they coming from? To address this question, they moved to a neuroblastoma cell line (Neuro2A), on which they could use CRISPR-Cas9 to remove both copies of Mov10. Then they examined the effect of Mov10 loss on total RNA levels and on the ability to grow a neurite, Ceman explained.

"They sequenced the cellular RNA millions of times at the Carver Biotech Center on campus and obtained a huge amount of data showing a lot of differences in the RNA levels," said Kalsotra. "We all started to talk about what to make of it and delve into the data."

The Ceman and Kalsotra labs have an RNA journal club that meets every week, and through this club, Skariah learned that Joseph Seimetz, in the Kalsotra lab, could help with the bioinformatics on this project.

"There is a list of gene candidates, and you can't test every one because there are thousands of them," said Skariah. "Bioinformatics is so powerful because you can pick important ones and remove them from the cell."

"The Ceman lab basically did all the work," said Seimetz. "It was perfect timing. We had started to build our own bioinformatics pipelines for analyzing "Big Data," so we combined it with our work."

"Joe developed the graphics that were so important for the paper," said Skariah. "We could move from a point of taking out genes to a point where we see a phenotype, we remove it and see how the genes change and how they function."

There is a lot more research that can come from this data. Their next step is to see what happens when Mov10 is removed from the brain. The lab is continuing to look at how the reduction of Mov10 changes the neurons.

The findings were published in *BMC Biology*, and the paper has piqued significant interest, with nearly 5000 views in the last six months.

Seimetz's work organizing the sizable amounts of data collected during the project is now shared in a public database that allows scientists around the world to work with and use the data.

"It's like our data keep on giving," Dr. Ceman said. •

Exploring the Effect of Liver Disease on the Heart

Dr. Sayeepriyadarshini Anakk and Bhoomika Mathur

by Sayantani Sarkar and Dr. Sayee Anakk

Infants and children with liver diseases are more likely to succumb to heart failure than to liver failure, and in fact, the heart's health often improves after a liver transplant. New research from the Annak lab, published in the journal *Hepatology*, demonstrates that excess bile acids in the liver cause metabolic change and stress in the heart. "That there is a connection between liver and heart disease has been known for hundreds of years," said Dr. Sayee Anakk, assistant professor of Molecular and Integrative Physiology, whose lab studies the regulation of metabolic signaling in the liver. "High levels of bile acids in diseased livers is associated with consequent cardiac dysfunction, but we do not know the mechanisms responsible for that connection."

Using mouse models that resembled the clinical cholestasis, or very high levels of bile acids in the liver, Anakk, with graduate student and first author Bhoomika Mathur, observed the effect of cholestasis on a key indicator of heart health.

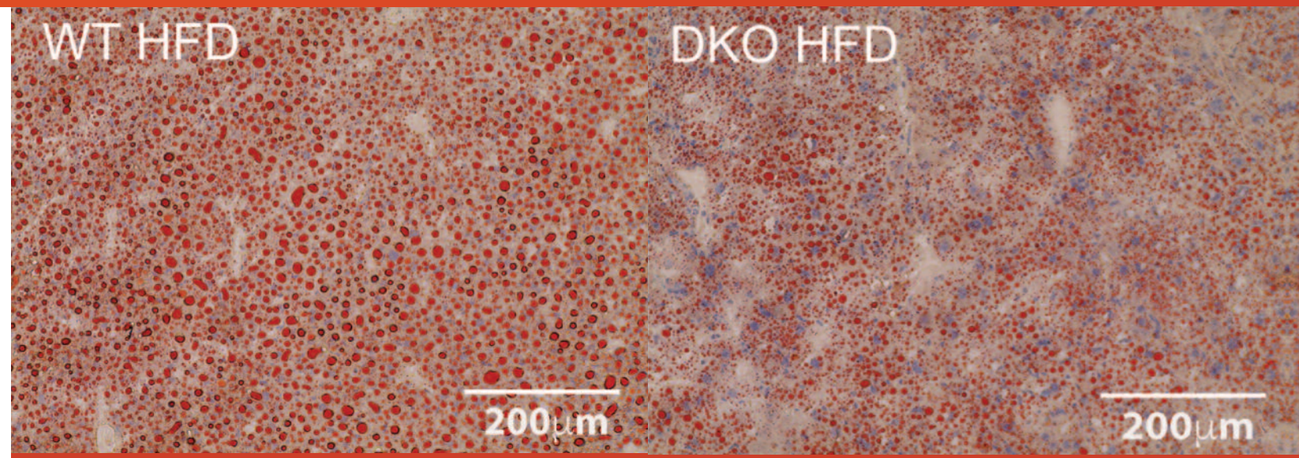
"Healthy hearts metabolize fat as its main energy source to pump blood," said Mathur. "Whereas, a stressed heart exposed to high levels of bile acids, switches to glucose as its energy source, and this switch in fuel metabolism is seen during heart disease."

The researchers also discovered that increased bile acids suppressed a master regulator of mitochondrial function, Pgc1α, that is central for regulating fat breakdown. Thus, suppression of this key gene can result in reprogramming the substrate preference and heart failure. The researchers named this Bile acid mediated effect on the heart: "Cholecardia."

"The discovery that excess bile acid affects heart metabolism was a big step forward, since this could allow us to identify potential treatment options," said Anakk.

Based on these findings, Ms. Mathur tested whether reducing bile acid levels would be sufficient to reduce the heart defect. They treated the mouse model with Cholestyramine, an FDA approved drug to treat high cholesterol that can remove bile acids from the blood, and found that the use of Cholestyramine protects against heart failure.

The team, with Dr. Moreshwar Desai, a pediatrician at Texas Children's Hospital and first author with Bhoomika Mathur, undertook this study. "The curative potential is exciting, but we have many more questions for bile acid's role in cardiac malfunctioning," said Anakk. •



Uncovering a Way to Fight Fatty Liver Disease

Oludemilade Akinrotimi, now a medical student at Loyola University Stritch School of Medicine

Ryan Riessen, now a medical student at Michigan State University's College of Osteopathic Medicine

In a second paper, the Anakk lab explored the mechanism by which the liver can be protected from developing fatty liver disease. This work was also published in *Hepatology* and led by two former undergraduates of the MCB program, Oludemilade Akinrotimi (Demi) and Ryan Riessen along with another MCB alumnus, Phil Vanduyne. Using the same experimental mouse model with double knockout (DKO) of Farnesoid X Receptor (Fxr) and Small Heterodimer Partner (Shp), they found that these mice are lean and resistant to diet induced obesity.

"To learn why the mice are lean we examined their caloric intake, activity and basal metabolism," said Riessen. DKO mice ate the same amount of food as the mice, which became obese but they were more active and burnt more fat. "Therefore we asked why are they more active?" said Akinrotimi. The answer was in skeletal muscle of these mice, which had more type I, or slow twitch fibers, with high oxidation capacity. "These mice were like marathon runners!" said Riessen.

Since the Anakk lab is focused on understanding liver diseases, they examined why the DKO mouse model did not develop fatty liver disease despite eating high fat containing food. It is typically thought that the receptor named Fxr (Farnesoid X Receptor) is crucial to control the amount of fat in the liver. Fxr functions by recruiting another receptor, Shp, to do its bidding. So when they found that really it was Shp that was important to causing fatty liver it was very surprising. "I didn't believe it," said Anakk. "I told them to do the experiment over and over again." The study showed that Shp has a role of its own. The paper was highlighted in an editorial by Dr. Richard Green from Northwestern, who suggests that "Targeting Shp may be efficacious for drug development to treat fatty liver disease."

"Research is really exciting and takes a lot of hard work," said Anakk. "But what makes this journey truly stimulating are the individuals who make it happen. I have been very fortunate to have wonderful undergraduate and graduate students in my laboratory who have made my job easy, and it is to them I owe my success." •

MCB GRADUATES

The lists are organized first by degree, then by major. Students earning the certificate in Microbiology are noted with *. Students earning the certificate in Neuroscience are noted with Ψ.

Doctor of Philosophy

Biochemistry
Zehua Bao
Jonathan Chekan, Fall 2016
Alexander Cioffi
Joshua Gajsiewicz, Fall 2016
Michael Gregory, Fall 2016
Daniel Harris, Jr., Fall 2016
Gus Lawrence
Paween Mahinthichaichan, Fall 2016
Kiruthika Selvadurai, Fall 2016
Nitesh Shashikanth, Summer 2016
Shannon Walsh
Xin Ye, Fall 2016
Xiaobin Zheng, Fall 2016

Biophysics & Quantitative Biology
Javier Baylon Cardiel, Summer 2016
Janish Desai, Fall 2016
Tyler Harpole, Summer 2016
Seyfullah Kotil, Fall 2016
Bo Liu, Summer 2016
Stuart Rose
Kai Wen Teng, Fall 2016
Erdal Toprak, Fall 2007
Kevin Whitley
Charles Wilson

Cell and Developmental Biology
Yu Chen, Summer 2016
Xiang Deng, Fall 2016
Paul Hamilton, Summer 2016
Anika Jain
James Kemp
Ruiqi Liao
Ambika Nadkarni, Summer 2016
Christina Rosenberger, Summer 2016
Amir Saberi, Summer 2016
Younguk Sun, Summer 2016
Rachel Waldemer-Streyer, Summer 2016
Yating Wang, Summer 2017
Hui-Chia Yu-Kemp, Fall 2016
Xinying Zong

Microbiology
Maksym Bobrovskyy
Ariana Bravo-Cruz
Whitney England, Fall 2016
He Fu
Nicholas Hess
Crystal Hopp, Fall 2016
Song Jiang, Summer 2016
David Krause, Summer 2016
Madeline López Muñoz
Kenneth Ringwald, Fall 2016
Margaret Wetzel, Fall 2016

Molecular and Integrative Physiology
Congcong Chen, Fall 2016
Kirsten Eckstrum
Itamar Livnat
Janelle Mapes
Daniel Ryerson

Neuroscience
Aadeel Akhtar, Fall 2016
Amogh Belagodu
Alexandra Brooks
Petra Majdak, Fall 2016
George Nikolaidis, Summer 2016
Matthew Petrucci, Summer 2016
Jenessa Seymour
Bernard Slater, Fall 2016
Benjamin Zimmerman, Fall 2016

Master of Science

Biochemistry
Gulishana Adilijiang
Aaron Primel, Fall 2016
Carissa Klanseck

Biophysics & Quantitativel Biology
Sreeradha Biswas, Fall 2016

Undergraduate Degrees—Bachelor of Sciences

Academic Distinction
Abdul-Rahman Abdel-Reheem, MCB
Nainika Roy, MCB
Kimberly Sam, MCB Honors Conc.
Diane Schnitkey, MCB Honors Conc.
Madeline Steiner, MCB
Benjamin Wang, MCB

Distinction, Research
Ali Aijaz, MCB
Humza Ashraf, Biochemistry
Katrina Dovalovsky, MCB, Fall 2016
Brandon Jones, MCB
Amish Khan, MCB Honors Conc., Fall 2016
Giyeong Kim, Biochemistry
Xinyi Li, Biochemistry
Katie Liang, MCB
Alaa Mansour, MCB*
Andrew McClintock, Biochemistry
Omar Shennib, Summer 2017
Edward Wang, Biochemistry
Alexander Willis, Biochemistry*
Adam Wylder, Biochemistry

Molecular and Cellular Biology Honors Concentration
Abdul Bhuiya
Nicole Blum
Macey Coppinger
Allison Hollatz
Nhan Huynh, Fall 2016
Eun Ji Jeong
Amish Khan, Fall 2016
John Krapf
Udit Nangia
Kimberly Sam
Diane Schnitkey
Ross Skelly*
Pha Thaprawat*
Andy Wu*

Biochemistry, Specialized Curriculum
Humza Ashraf
Jesse Barr
Ivan Chau
Yiling Ding, Fall 2016
Zhuoli Huang
Giyeong Kim
Philip Kim
Anastasia Kondyukh
Nickolaus Lammer
Hugo Lee
Xinyi Li
Hayoon Lim
Jiayi Luo
Anita Mathew*
Andrew McClintock
Richard Park
Zoe Petros
Emma Schuster
Malvika Singh
Aleksas Valaitis
Edward Wang
Alexander Willis*
Adam Wylder

Molecular and Cellular Biology
Alyssa Abay
Abdul-Rahman Abdel-Reheem
Malini Achar
Farjad Adamjee, Fall 2016
Ariana Adelman Ψ
Anusha Adkoli
Hajira Ahmed, Fall 2016
Hamza Ahmed
Arjun Ahuja
Ali Aijaz
Abdul-Quddus Akinlusi
Waleed Aldadah
Irfan Ali
Lauren Andersen
John Anderson, Summer 2017
Gabriel Arce, Fall 2016
Josephine Awah-Kanneh, Fall 2016Ψ
Kayla Bachar
Jessica Bachman, Fall 2016
Eun Seol Baek
Samiat Balogun
Megan Bartimoccia
Hannah Behle
Vanessa Belknap
John Belz
Franco Benyamin, Fall 2016
Rohit Bhonagiri
James Biondo
Samuel Boctor
Paul Bogdan
Kristopher Bosi
Diamond Brown
Paul Brown
Saloni Buch
Paul Camacho Ψ
Stefani Canadzic
Corinne Cannavale Ψ
Alejandro Cardona
Arturo Chaidez
Cecilia Chan, Fall 2016*
Audra Chaves
Marialicia Chavez
John Cheline, Fall 2016
Larry Chen, Fall 2016
Max Chen Ψ
Rachel Chinchilla
Maham Choudry
Grace Clayton, Fall 2016
Adam Cohen
Joshua Cohrs
Haven Comeaux
Kyle Coots
Azariel Coss
Elizabeth Cullum
Nicole Curtis
Ryan Cyriac Ψ
Farnaz Dadrass
Paroma De, Summer 2017
Zachary Dean
Matthew Del Pino
Senem Demir, Summer 2017
Taylor Desanto, Fall 2016
Samantha DiCaro
Nathaniel Dimaano, Summer 2017
Chuhong Ding
Patrick Doah
Heidi Doden*
Alexander Dolinar, Fall 2016
Vinisha Doshi
Katrina Dovalovsky, Fall 2016
Kevin Drent, Fall 2016
Kevin Duffin, Fall 2016
Clarence Dukes
Kevin Dwyer
Neil Edat
Samantha Eichner
Cortney Eiers, Fall 2016
Ehitare Emuze
Jessica Endres
Jonathan Eng
Adam Esmail
Tristan Estep
Lauren Feld, Fall 2016
Mary Kate Feldner
Ryan Fell
Mary Figura
Alejandra Flores
Sandra Folarin
Zachary Foust*
Dulce Frausto
Sadie Freedman, Fall 2016Ψ
Rachel Full
Lauren Gabra, Summer 2017
Hannah Gaddam
Tea Garibovic
Christopher Gavigan
Palak Genge, Summer 2017
Stephanie George
Michael Giuliano
Mahima Goel
Lolita Golemi, Fall 2016
Jose Gonzalez, Summer 2017
Aleksandra Gorski
Jeffrey Gougis, Jr.
Daniel Gryzlo Ψ
Qingrou Gu, Summer 2017
Jovanny Guadarrama-Aniceto*
Ellen Guo
Juhi Gupta Ψ
Armaan Haleem
Mohammed Hamad, Fall 2016
Owen Haupt
Matthew Heffernan
Brittany Hermosillo
Octavio Herrera
Hui Ting Iris Hou
Samantha Hunt
Mark Huston
Helena Igbo
Abdalah Ismail
Teodora Ivanova
Jacek Iwanicki
Chitra Iyer, Summer 2017Ψ
Stephen Jan
Adrianna Jelen Ψ
Kun Jing, Fall 2016
Varsha John Ψ
Erin Johnson, Fall 2016
Brandon Jones
Linyang Ju, Fall 2016
Ayub Julany
Christian Kalfas
Constadina Kalouris Ψ
Wei-Chun Kao
Andriy Kapeniak
Sarah Kaschke
John Keene
Elizabeth Kehl, Fall 2016
Patrick Kelly
Shivani Khakhkhar
Nadia Khan, Fall 2016
Sana Khan
Akash Khurana
Connie Kim
Kyung Il Kim, Summer 2017
Lois Kim
Sally Kim*
Sang Min Kim
Se Yi Kim
Tae Gyoon Kim
Marit Kirklys
David Koo
Jessica Kosapatti
Rahul Koshy*
Vladimir Koveshnikov
Susmitha Kowligy Ψ
Emily Kroeger
Jordan Kryza
Benjamin Lamar
Maureen Lawless, Summer 2017
Frank Leuzzi
Junyu Li
Rao Li
Katie Liang
Justin Lien Ψ
Bailey Link
Qingyuan Liu
Di'Reon Lowry, Summer 2017
Lauren Lundquist, Fall 2016
Scott Lyons
Jad Maamari
Jared Madden
Jennifer Main
Theodore Manahan
Gio Manguerra, Summer 2017Ψ
Alaa Mansour*
Hanna Matseshe
Kyle McClatchey
Janayah McClellan
Jasmine McDowell
Matthew McKenna*
Yuhao Mei
Elan Melman-Volchenko
Daniel Melnikov
Gibran Mendez, Fall 2016
Stephanie Menezes
Molly Mevis*Ψ
Maria Mihailescu
Jeremy Mikrut
Michelle Miller, Fall 2016
Katherine Mitchell
Tina Moazezi
Mitchell Mohr
Allison Monie
Rachel Moore*
Grace Morrissey
Samuel Mounce
Robert Mowery, Fall 2016
Angelica Mulica, Fall 2016
Vamsikrishna Naidu
Kimberly Nalley
Sara Napoli
Christopher Nardone
Emilee Nawa
Yusuf Nekzad
Michael Nemsick
Elizabeth Nettleton
Joseph Neumann
Heather Newman
Gabriele Noreikaite, Fall 2016
Margaret O'Brien
Quincy Ogbogu
Christine Oksas, Fall 2016Ψ
Amy Ou
Diana Pacyga Ψ
Brandon Pagni
Weilun Pang
Tara Park
Yoonho Park
Stephanie Pascual
Akash Patel
Dhruv Patel
Dil PatelΨ
Jay Patel
Payal Patel
Priya Patel
Ravi Patel
Reema Patel
Shivaliben Patel, Fall 2016
Shivam Patel Ψ
Thomas Pickett, Fall 2016
Porsha Pilate
Nathan Pinsky
Kovas Polikaitis, Fall 2016
Renee Pond
Sarah Rahman
Neha Rai
Jeffrey Rajkumar
Adin Rand
Alyssa Reitz
Denise Reynish
Kurt Reynolds
Matthew Rhee
David Rhode
Andrea Rice
Noah Risner
Jordyn Robare, Fall 2016
Aleetra Roberts
Megan Robin
Abraham Rodriguez
Alexander Rodriguez, Fall 2016
Daniel Rodriguez, Fall 2016
Samuel Romo
Evi Rondo, Fall 2016
Anita Rong, Summer 2017
Kathryn Ross
David Rossi
Nainika Roy
Daisy Salgado
Sidra Salman
Donel Samuel
Diana Sarfo
Ryan Schaab
Chelsea Schultz, Summer 2017
Minjoo Seo, Fall 2016
Alexandria Shadid, Fall 2016
Shane Shafi, Fall 2016
Amy Shah
Binoy Shah, Fall 2016Ψ
Vaibhav Sharma
Toral Shastri
Christine Shen
Omar Shennib, Summer 2017
Jinrong Shi
Mackenzie Shimmin
Julia Sidor
Matthew Siegel
Matthew Simanonis Ψ
Meghan Smith
Lacey Solomon
Madeline Steiner
Mallory Stimac
Nathaniel Stringer
Mayu Sugikawa, Fall 2016
Zarin Sultana
Cindy Sun
Kalee Sweeney, Fall 2016
Shahbaz Syed
Steven Szymanski
Sabrina Tahseen
Daniel Tan
Lawrence Tan
Claire Tang
Bridget Tesher
Zachary Thomason
Christopher Thuruthiyil
David Tian*
Thomas Topalis Ψ
Elizabeth Tran, Fall 2016
Michael Trevino Ψ
Tiffany Tsoi, Summer 2017Ψ
Joshua Valentin
Christian Vanhooser
Pravin Vence
Okker Verhagen Metman
Lauren Walling
Benjamin Wang
Ashley Wehrheim
Kaitlyn Wehrheim
Hailey Weidner, Fall 2016
William Weissinger
Theresa Welle Ψ
Kevin Wells
Jaleel Williams
Alexander Willis*
Julia Witowska
Julia Witvliet
Kui Wu
Charley Xi
Xuling Xu
Yiran Xu, Fall 2016
Jason Yeh
Daniel Yoakum
Ivanna Yu Ψ
Neil Yu
John Zahour
Chenqi Zhu

MCB Learning Center for Teaching and Advising

- Learning Center with computer stations and tables for student group work and TA or Faculty office hours
- Six new active learning iFlex classrooms
- New MCB Advising Center
- Seminar Rooms for Career Services Events
- New space for limited-distraction exams
- Meeting spaces available for student use
- Space for Writer's Workshop Personnel

Coming Soon in Fall 2018

Support the MCB Family

Members of the Graduate Student Association, the "McBees"

Give online:
mcb.illinois.edu/giving

With over 15,000 alumni, we're proud of our graduate family and want to keep in close contact. Our future is dependent on the generosity of our graduates, and we welcome your contributions to the school and departments, each of which offers a unique and excellent mission.

Your gifts support general and specific programs in research, teaching, training, and public service, at the school or departmental level.

Please visit mcb.illinois.edu/giving. You can also contact Assistant Director of Development Trent Reed, reed15@illinois.edu, (217) 300-4462, to discuss making a donation. Every gift helps maintain the excellence of MCB.