Hypothesis
SCHOOL OF GRADUATE AND POSTDOCTORAL STUDIES

our focus on RESEARCH.

ROSALIND FRANKLIN UNIVERSITY of MEDICINE AND SCIENCE
Hypothesis

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Dean’s Message

This edition of Hypothesis focuses on Research.

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Cover photo: A section of an eye from a mouse model of Batten disease. The lab is assessing the accumulation of potentially pathogenic lysosomal storage material in the retina. Nuclei are stained with DAPI (blue) for reference.
our focus on RESEARCH.
The mission statement of the School of Graduate and Postdoctoral Studies at RFU includes the phrase “…to advance knowledge through biomedical research.” Such a phrase in the mission statement seems straightforward and particularly appropriate for a school whose students discover new knowledge in partial fulfillment of their degree requirements. The congruities of the words and meaning of the phrase with our activities may appear to minimize the significance of those words that characterize our identity. Indeed, “to advance knowledge” is a core element of our purpose. It is partly why the school exists.

Given a national environment in which the purpose of higher education is under pressure to move toward practical, measurable and easily demonstrable outcomes, such as specific manual skills, the discovery of new knowledge as a purpose is not readily quantifiable. Research is often complex with many known and unknown variables, in spite of rigorous controls. It is unpredictable. In the popularly attributed words of Albert Einstein, “If we knew what we were doing, it would not be called research, would it?” The inability to accurately predict the outcome of an experiment, the inability to repeat an experimental result, the uncertainty of whether to progress down a potential rabbit hole of experiments are frustrating and anxiety-inducing aspects of research. Importantly, the long-term significances of an individual’s research and the knowledge revealed by it are also typically unquantifiable and unpredictable. Yet, the sense of discovery when results are confirmed and the realization that new knowledge has been revealed is exhilarating. Furthermore, it cannot be denied that collectively, biomedical research has vastly improved the human condition and every experiment contributes to that progress.

Research as a purpose is both a collective and personal undertaking. Together we collaborate, hypothesize, investigate and discuss our research ideas and results. Research is critically reviewed, substantiated and at times challenged through the community of biomedical investigators. However, scientific inquiry also addresses purpose in a personal, individual manner. People who base their life’s work in research have a need to understand “how.” How does memory exist from entangled neural connections? How does viral infection lead to cancer? These are the types of questions that ignite intellectual curiosity, that inspire students and their mentors to investigate, to discover. A purpose of the Graduate School is to foster this personal, intellectual development among our students who seek a career in research. This life’s work is a life of purpose. It is a life in discovery. 

Joseph X. DiMario, PhD
Dean, SGPS
Graduate student Isha Dey’s hypothesis: Regulating cellular lemur tyrosine kinase 2 (LMTK2) levels via protein kinase C (PKC) could provide a direction for creating targeted therapies that treat diseases such as cystic fibrosis.
Fifth-year graduate student Isha Dey is focusing her research on a basic level today, so she can build toward big things in the future.

“The work that I’m doing now is very basic – in order to do something big, you need some basic information to start. This protein that I’m working with, LMTK2, is involved in a variety of diseases, including prostate cancer, and the trafficking of a protein called CFTR, which is responsible for the development of cystic fibrosis (CF),” Isha said of her research.

Lemur tyrosine kinase 2 (LMTK2), a serine/threonine kinase, is a membrane-anchored protein with two transmembrane helices and a very long C-terminal tail. (This characteristic gives the protein its unique name, as its tail resembles that of a lemur.) The C-terminus contains the kinase domain, responsible for phosphorylating various substrates. Based on preliminary research conducted in the lab of Neil Bradbury, PhD, professor of physiology and biophysics and Isha’s mentor, the Bradbury Lab has proposed LMTK2 to be a novel therapeutic target for cystic fibrosis and prostate cancer, and that activating or deactivating the kinase could lead to an alternate path for a treatment for these devastating diseases. To date, there are no known activators or inhibitors of LMTK2, although the Bradbury Lab is working toward identifying such modulators.

“If we cannot modulate the activity of the protein, we can try and modulate the number of LMTK2 molecules inside the cell, which in turn would affect the overall activity of the protein,” Isha explained. “My project is to study the mechanism by which synthesis/ expression of LMTK2 is regulated inside the cell, so that we can regulate the amount of LMTK2 inside the cell.”

Regulating the amount of this particular protein could prove critical to developing a pathway for targeted therapies to treat diseases such as CF. LMTK2 is involved in the recycling of the cystic fibrosis transmembrane conductance regulator (CFTR), a chloride channel found on the plasma membrane of polarized epithelial cells that moves chloride ions out of the cell, allowing water to follow, and hydrating the lining of the airways and intestine. Defects in the recycling of CFTR make a significant contribution to the loss of CFTR from the cell surface, a key element in the pathology of the disease.

Isha has spent five years working in Dr. Bradbury’s Lab, where much has been discovered about LMTK2 expression and how it can be modulated. “We have discovered that LMTK2 expression in human cells can be modulated at the transcriptional level via protein kinase C (PKC) activation,” Isha said of the work conducted in the Bradbury Lab. “We then went on to identify the intracellular signaling cascade mediating this modulation. We are currently studying whether regulating cellular LMTK2 levels by PKC has a positive effect on the recycling of CFTR, so as to increase the number of CFTR channels on the plasma membrane. If it does, then our findings would provide a direction for targeting LMTK2 as a therapy for CF.”

Looking ahead, Isha would like to see her research move into the clinical realm. “As of now, I want to do something clinical, because I’ve worked a lot on the basic molecular biology part. I want to go into the clinical part next and see how the clinical applications work.”

Regulating the amount of LMTK2 could prove critical to developing a pathway for targeted therapies to treat diseases such as cystic fibrosis.
Rongxin Nie

facing a formidable OPPONENT.

Rongxin Nie’s hypothesis: A better understanding of how MATE transporters expel drugs out of cells will contribute to the development of novel drug design to overcome multidrug resistance.

Multidrug resistance poses a major threat to public health. It is estimated that at least 23,000 people die from drug-resistant bacterial infections each year in the United States. Graduate student Rongxin Nie is tackling this problem head on by researching ways to stop transporters from expelling drugs out of cells.

“I’ve been studying the structure and the function of MATE (multidrug and toxin extrusion) transporters by x-ray crystallography and other biochemical measures, to better understand the mechanism of this transport family and how they transport drugs out of the cells,” Rongxin said, describing his research. Specifically, “My work is to study the structure and function of the MATE transporter NorM from Neisseria gonorrhoeae (NorM-NG).”

Rongxin knows he’s facing a formidable opponent. “Bacteria are smart. They develop various mechanisms to fight against drugs; they can alter the targets, modify the targets to prevent the drugs entering the cells. One important mechanism is that they can express some transporter which can extrude the drug out of the cells, so this renders the bacteria resistant to these kinds of drugs. That’s why we want to understand the mechanism of this transporter [NorM] and, hopefully, find an inhibitor to stop the drug extrusion.”

The MATE transporter family is one of five multidrug transporter families. The MATE transporters consist of around 900 members. They contribute to multidrug resistance by extruding drugs across the cell membrane using either the Na+ or H+ gradient. The MATE transporters are conserved from bacteria to humans. One of the important properties of the MATE transporters is their substrate polyspecificity – they can extrude a wide range of structurally and chemically unrelated compounds. Since MATE transporters render bacterial pathogens and human cells resistant to antibiotics and anti-cancer drugs, respectively, reducing efficacies of these drugs, they are promising therapeutic targets for overcoming multidrug resistance in pathogens and cancer cells.
Min Lu, PhD, associate professor of biochemistry and molecular biology and Rongxin’s mentor, determined the crystal structures of the Na+-coupled MATE transporter NorM-NG in complexes with three distinct translocation substrates (ethidium, rhodamine 6G and tetraphenylphosphonium), as well as Cs+ (a Na+ congener), all captured in extracellular-facing and drug-bound states. Rongxin performed the functional study. Based on these structural and functional studies, they proposed that NorM-NG uses an indirect competition mechanism, in which Na+ triggers multidrug extrusion by inducing protein conformational changes, rather than directly competing for the substrate-binding residues.

Dr. Lu commends Rongxin for his dedication to his research, and considers him an asset to the lab. “Rongxin is very interested in getting into the field of structural biology and studying membrane proteins. He’s willing to spend his time and effort working in the lab, getting the experiments done, and he is willing to devote himself to his research.”

Prior to beginning his graduate studies at RFU, Rongxin worked for the Institute of Biophysics at the Chinese Academy of Sciences in Beijing. “From that time, when I started working on protein structure with x-ray crystallography, I think I became very interested in this field.” Rongxin vividly remembers his first experiences working with x-ray crystallography. “When I saw that for the first time, it was very exciting, very interesting, how through the crystal you figure out what proteins look like and how they function.”

Rongxin has maintained that enthusiasm for his research throughout his time as a graduate student at RFU. Fortunately for the university, that dedication to discovery is staying right here. Rongxin successfully defended his doctoral dissertation in October, and began working as a research associate in Dr. Lu’s lab in November.

Bacteria are smart – they can modify targets to prevent therapeutic drugs from entering the cells. That’s why we want to understand the mechanism of this transporter [NorM] and, hopefully, find an inhibitor to stop the drug extrusion.
Soumyabrata Munshi

finding the LINK.

Soumya Munshi’s hypothesis: The basolateral amygdala (BLA) is a likely intermediary linking changes in immune system activity with conditions such as depression and anxiety.

Soumyabrata (Soumya) Munshi, a PhD candidate in neuroscience, is trying to answer three main questions with his research project: 1) How does chronic psychological stress affect parameters of the immune system?; 2) How does peripheral immune activation alter physiology in a brain region important in mood and emotion?; and 3) Does chronic stress alter activity of this brain region by effects on the immune system?

Working in the laboratory of his mentor, J. Amiel Rosenkranz, PhD, associate professor of cellular and molecular pharmacology, Soumya examines the changes in immune system activity in the peripheral
blood and within the brains of rats after exposure to a form of stress that models the impact of negative social interactions. The experimental rats are exposed to unfamiliar aggressive rats once per day for five consecutive days. Three days later, the experimental rats are examined to look for changes in their peripheral circulating T-cells and intracellular cytokine positivity, as well as changes in the serum inflammatory cytokines. These experiments will paint a holistic picture of the shift in peripheral immune function after repeated social stress.

In a separate set of experiments, Soumya examines the changes in the activity of neurons in the BLA using extracellular *in vivo* electrophysiology in rats, either after stress or after peripheral immune challenge, and compares those results with their corresponding control groups. These experiments will begin to uncover parallels between the effects of social stress and specific immune factors on BLA activity. This is important because changes in BLA activity have been linked to emotion, and pathological changes in BLA are linked to depression and anxiety. To evaluate if the changes in BLA function in stress are mediated by the recruitment of the immune system activity, Soumya will test whether the effects of social stress can be reduced by manipulations that target the immune system. Throughout these approaches, Soumya also measures parallel effects of stress and immune manipulation on behaviors that are sensitive to changes in BLA activity, such as anxiety-like behavior in an elevated plus maze and open field exploration. This work can lead to a new way to approach treatment of psychiatric symptoms that are triggered by stress.

Soumya recalls his clinical experiences with patients affected by co-morbid neuropsychiatric conditions including depression and anxiety disorders while working as an intern following his MBBS program in India. His subsequent exposure to basic science research while pursuing junior residency in India sparked his interest in neuroscience.

He decided to pursue additional training at the doctoral level in the United States, and during the final year of his junior residency in India, he started to explore ways he could achieve this dream. He was accepted into Rosalind Franklin University’s Interdisciplinary Graduate Program in Biomedical Sciences, and after a series of lab rotations and experiences, Soumya received the opportunity to pursue his doctoral research project under Dr. Rosenkranz. “I am extremely blessed and fortunate to have Dr. Rosenkranz as my advisor,” Soumya admits.

Dr. Rosenkranz is impressed by Soumya’s initiative. “Soumya came up with this project by himself. I don’t really work with the immune system, so he said, ‘OK, this is a bit outside what your lab does, but we can make it related,’ and together we figured out a link by which we can look at Soumya’s passion and connect that to the main interests of our lab (neurophysiology of emotion and memory). We work together to flesh out some aspects of the project, and I help him learn some of the approaches to study the neuroscience-related questions, but the hypothesis is his, and it’s a really impressive example of somebody branching out from what a lab already does.”
Graduate student Jen Chang’s hypothesis: By disrupting the protein that causes plaque buildup in the brain, scientists can lessen the symptoms of Alzheimer’s disease in patients with Down syndrome.

Jen Chang, a fourth-year graduate student in the Department of Cell Biology and Anatomy (CBA), has spent much of her time in the lab doing the exact opposite of her lab mates. “While most of my lab uses ASOs [antisense oligonucleotides] to make a correct protein to help fix a disease, I’m doing the opposite where I want to disrupt a protein from causing a disease state,” Jen said.

Jen works in the lab of Michelle Hastings, PhD, associate professor of cell biology and anatomy, and is focused on using ASOs (short synthetic DNA molecules that can bind to messenger RNA [mRNA] and affect how a protein is translated from these mRNA molecules) as a therapy to correct inherited diseases.

“I’m specifically working on a therapy for Alzheimer’s disease (AD) in Down syndrome (DS) individuals,” Jen explained. “People with DS have a triplication of chromosome 21, which is the location of the amyloid precursor protein (APP) – the protein that creates amyloid beta 42 (Ab42). This protein, Ab42, causes the plaque buildup in the brains of people with AD. Because of this overexpression of APP, DS individuals have these plaques and other pathology of AD by the time they reach their mid-30s to 40s, which is much earlier than a karyotypically normal individual. My project aims to ameliorate the AD symptoms seen in DS by affecting how APP is processed into amyloid products, which includes Ab42.”

Her work in the lab is showing promise. “We have engineered an ASO that affects a significant part of the amyloid portion of the protein while leaving the majority of APP intact,” Jen said. “Applications of this work would aid greatly in preventive care for AD in persons with Down syndrome.”

Jen’s mentor, Dr. Hastings, praised Jen for the advances she has made in the lab. “When Jen started her training, she worked either with me or someone in the lab who was more experienced, to help her learn how to do the techniques, methods, protocols. Now, she’s to the point where she’s doing most of the bench work on her own and learning protocols and methods and able to troubleshoot if something goes wrong.”

Dr. Hastings and Jen meet on a weekly basis so that Jen can discuss her results with her mentor and talk about next steps. Jen’s mentor also encourages her to pursue...
professional development opportunities such as attending conferences and participating in workshops that help with writing skills for scientific papers. Jen contributed to a paper authored by one of her lab mates (CBA Research Associate Anthony Hinrich), “Therapeutic correction of ApoER2 splicing in Alzheimer’s disease mice using antisense oligonucleotides,” published in the April 2016 issue of EMBO Molecular Medicine.

When asked about any challenges she has encountered during her time as a graduate student at RFU, Jen spoke of learning to balance her time-intensive research with involvement in extracurricular activities, including co-chairing the 11th Annual All School Research Consortium in March 2016 and serving as the Graduate Student Coordinator for the university’s INSPIRE program (Influence Student Potential and Increase Representation in Education) this past summer. Jen also mentored an INSPIRE student in the Hastings Lab for 10 weeks over the summer, demonstrating laboratory techniques and nurturing the student’s interest in science. Jen reports that her INSPIRE student is now in her first year of college and “wants to major in chemistry and math and do all these really amazing things.”

“My project aims to ameliorate the Alzheimer’s disease symptoms seen in Down syndrome by affecting how APP is processed into amyloid products.

“Just watching her develop from this really shy girl to this really big go-getter, I got really excited about that,” Jen added. “I hope I get to experience more of that later on in my career.”
All School Research Consortium embodies the interprofessional educational philosophy at our university, where researchers and future healthcare professionals present their recent research discoveries and share ideas on how research addresses healthcare challenges to ensure better patient outcomes.

Scott Klappa, Clinical Psychology student, explains his methodology.
Physical Therapy student Nicolas Martin describes his project to a poster judge and the audience.

The poster sessions at ASRC provide an ideal platform for RFU trainees from across the university to present their work and practice their communication skills.

Dr. DiMario congratulates Physiology and Biophysics student Jiaju Wang on his selection for best SGPS student research talk.

Dr. James Carlson, College of Health Professions dean, shows his support and interest during a student poster presentation.
The annual “Art from the Benchtop” show, presented by the Graduate Student Association, brings the art and love of science out from the laboratory for everyone to see. In doing so, the images transition from analytic scientific assessment to emotive conceptual art.
The third annual “Art from the Benchtop” 2016 exhibition featured the scientific artwork of RFU postdocs and SGPS students.

Dehydrated western blot gels stained with Ponceau S and methylene blue create an intriguing visual landscape.

Immunofluorescent imaging reveals the beauty and complexity of neural networks.

Dr. K. Michael Welch (right) takes a particular interest in Jiaju Wang’s creation.

The third annual “Art from the Benchtop” 2016 exhibition featured the scientific artwork of RFU postdocs and SGPS students.
Graduate students take a break from nourishing their minds by volunteering at Feed My Starving Children.

Taking a break at the National Postdoc Appreciation Week (NPAW) postdoc/mentor ice cream social.

Dr. Sahithi Pamarthy adds her name to the Microbiology graduates plaque after her doctoral defense with Dr. Alice Gilman-Sachs.
Graduate student Mira Repak is ready for a challenge at the GSA Game Night!

2016 SGPS doctoral recipients celebrate at Commencement.

Postdocs gather for the annual NPAW group photo.
Jaime Vantrease’s hypothesis: A better understanding of sex differences in the expression of anxiety disorders is the first step toward the development of sex-specific therapeutic approaches.
Although anxiety disorders are twice as likely to occur in women as in men, the disorders are treated similarly regardless of the sex of the patient. Studies that examine sex differences in the neurobiology related to these disorders are limited. Postdoctoral fellow Jaime Vantrease, PhD, is working to change that.

Jaime has spent nearly two years at RFU studying the physiology of the basolateral amygdala (BLA), the part of the brain responsible for mediating fear responses and processing emotion. The amygdala is hyperactive in patients with anxiety disorders, and is more active in females during specific affective tasks. To Jaime, this suggests that sex differences in BLA activity or output circuits may contribute to the sex differences observed in the pathophysiology of anxiety disorders.

“The BLA is an area that’s critically involved in anxiety and mood disorders, so our research is focused on understanding, specifically, the differences between males and females in order to pave the way for more therapeutic potential targets for men and women,” she explained.

To determine basal activity in BLA neurons, Jaime conducts single-unit extracellular electrophysiological recordings in vivo to record spontaneous BLA neuronal activity in naïve male and intact cycling naïve female rats. From these experiments, she has determined that, independent of estrous cycle stage, females have a significantly higher basal firing frequency in the BLA compared to males.

The activity of these neurons is regulated, in part, by small conductance calcium-activated potassium (SK) channels that inhibit neuron firing rates, Jaime explained. Since sex differences have been observed in the spontaneous firing frequency of BLA neurons, Jaime also measured relative expression of SK channel mRNA and protein levels in the BLA of naïve male and female rats via quantitative real-time polymerase chain reaction and Western blot analysis, respectively. She has observed that the relative mRNA levels of the SK2 channel isoform is comparable between sexes; however, the Western blot analysis revealed that SK2 channel protein expression is significantly reduced in intact female rats, irrespective of estrous cycle stage, compared to males.

Together, these data suggest to Jaime that sex differences in SK2 protein expression may contribute to the sexually divergent BLA neuron activity in naïve rats. Moreover, her studies have identified a potential therapeutic target for the treatment of anxiety-related disorders, an area Jaime hopes to pursue once she finishes her postdoctoral fellowship at RFU.

“I’m leaning toward pursuing a career in an industry setting. I like the idea of working with something a little more translational, something that has potential to reach the clinic. I still would want to be an independent scientist, doing bench work and collaborating with other scientists,” Jaime said of her future plans.

Collaboration has been key to Jaime’s success here at RFU from the beginning. For starters, she is co-mentored by J. Amiel Rosenkranz, PhD, associate professor of cellular and molecular pharmacology, and Janice Urban, PhD, professor and chair of physiology and biophysics.

“We are both her primary mentors,” explained Dr. Rosenkranz. “We have a cohesive plan so that Jaime can accomplish all the professional goals at the same time that we’re doing all the research.”

Dr. Urban agrees that Jaime has benefitted from the collaborative nature that infuses research at RFU. “Dr. Rosenkranz and I have been collaborating now for a few years, borne out of complementary expertise. That’s one of the strengths of various programs such as Jaime’s where she has co-mentors – that adds strength to the training of the individual.”
We think that the Cajal body functions to make the cell nucleus more efficient and more accurate.
Iain Sawyer’s hypothesis: The Cajal body makes cellular processes more accurate by affecting the fidelity of RNA processing through the regulation of spliceosome components.

Postdoctoral research fellow Iain Sawyer, PhD, has spent the past two and a half years at the National Cancer Institute (NCI) in Bethesda, MD, studying how the cell nucleus is organized. His specific interest is the Cajal body, a nuclear structure that forms at sites of small nuclear RNA (snRNA) gene activity, where it packages snRNAs into functional small nuclear RNP s (snRNP s). The mature snRNPs form a molecular structure known as the spliceosome and process protein-encoding mRNAs at other sites in the nucleus. Dr. Sawyer says his research indicates that the Cajal body is an exciting sub-nuclear hub that links genome organization to RNA splicing and is important for proper cellular function.

“What we’re really interested in is how the genome interacts with nuclear bodies. We think that the Cajal body functions to make the cell nucleus more efficient and more accurate, which is scary because it’s found in few normal cells but it’s frequent in cancers. We think the Cajal body brings specific target genes in close proximity to regulate their activity – and, of course, these target genes that it works on are involved in the processing of RNA.”

According to findings of a study conducted by Dr. Sawyer and colleagues, “Cajal Bodies are linked to Genome Conformation,” published in Nature Communications in March 2016, the depletion of Cajal bodies in cancer cells causes the global RNA processing step to become less accurate.

“We have described the genome-wide network of Cajal body-proximal genes using DNA sequencing and advanced microscopy techniques, including spectral imaging, to visualize five different genes plus a nuclear structure, and automated high-content microscopy to analyze thousands of cells overnight. We described how Cajal bodies frequently associate with many genes simultaneously that are often positioned on opposite ends of the same chromosome or even different chromosomes,” Dr. Sawyer explained.

“The RNA products of these Cajal body-proximal genes are greatly reduced when Cajal bodies are depleted from cancer cells. Intuitively, mRNA processing becomes less accurate, possibly due to spliceosome dysfunction linked to unavailability of components produced by the Cajal body.”

Dr. Sawyer became interested in molecular biology while finishing his graduate work at King’s College in London. “Part of my project was working on the position of a protein in the cell nucleus, so I knew I wanted to get into something to do with nuclear organization. I knew I wanted to move countries for my postdoc – I feel that as a scientist it’s important to try new things – and the advertisement that I came across stood out to me...two great labs working together, that of Dr. Mirek Dundr (assistant professor of cell biology and anatomy) at RFU and Dr. Gordon Hager (chief of the Laboratory of Receptor Biology and Gene Expression) here at the NCI. They’ve worked together for a number of years and their publication track record is phenomenal. So I came over to the NCI and interviewed with Gordon. They knew they wanted to position me here semi-permanently, so I started work two and a half years ago and it’s been pretty successful.”

Dr. Sawyer is grateful for the resources available to him by being based at the NCI. “There are microscopes here that would blow your mind, just in terms of what they can do. One of the experiments I perform regularly, I can acquire tens of thousands of cells automatically; if I were to do it by hand, it would probably take me a year. It’s incredible.”

Having his mentor, Dr. Dundr, hundreds of miles away at RFU has not been a problem as they speak almost daily. “The thing I appreciate about mine and Mirek’s relationship is he’s receptive to my ideas. We speak regularly, talking about what’s next. I think that’s what you have to do, when you have a person positioned in your lab elsewhere – keep an open channel and an open mind.”
Arunava Roy’s hypothesis: The Kaposi’s sarcoma-associated herpesvirus (KSHV) maintains its latency by using the IFI16 protein to suppress its lytic transcription.

Kaposi’s sarcoma-associated herpesvirus has evolved to utilize the innate immune sensor IFI16 to keep its lytic cycle transcription in dormancy.
Postdoctoral research associate Arunava Roy, PhD, finds the ever-evolving battle between virus and host fascinating to study. Through research of the Kaposi’s sarcoma-associated herpesvirus (KSHV), Dr. Roy and his colleagues are discovering that herpesviruses like KSHV have evolved mechanisms to utilize the first line of host cellular defense and turn them around to serve their own function.

“This virus is very interesting because it maintains a state of latency (dormancy) in human subjects for a very long time, before the host even knows it is infected with the virus. When there is immune deficiency in the body due to varying causes, one of the most prevalent causes is HIV infection, this virus can cause diseases such as Kaposi’s sarcoma,” Dr. Roy explained. “We work with the innate immune response against DNA viruses. These viruses elicit many kinds of immune responses and we primarily study two of them – one is the inflammasome pathway and the other is interferon response. My project involves a protein called IFI16, which is a very important protein for the inflammasome pathway.”

(The role of IFI16 in the innate immune response was first identified by Nagaraj Kerur, PhD, when he was a graduate student at RFU in the lab of Dr. Roy’s mentor, Bala Chandran, PhD, professor and chair of microbiology and immunology.)

Dr. Roy described what he and his colleagues have discovered about the role of IFI16 in transcription regulation of KSHV in their paper, “Nuclear innate immune DNA sensor IFI16 is degraded during lytic reactivation of Kaposi’s sarcoma-associated herpesvirus (KSHV): Role of IFI16 in maintenance of KSHV latency,” published in the Journal of Virology in July 2016.

“Our paper revealed that KSHV has evolved to utilize the innate immune sensor IFI16 to keep its lytic cycle transcription in dormancy,” Dr. Roy said. “We demonstrated that IFI16 binds to the lytic gene promoters and acts as a transcriptional repressor, thereby helping to maintain its latency. We also discovered that during the late stage of lytic replication, KSHV selectively degrades IFI16, thus relieving itself of this transcriptional repression. This is the first report to demonstrate the role of IFI16 in latency maintenance of a herpesvirus, and further understanding of this phenomenon can lead to the development of strategies to eliminate latent infection.”

Dr. Roy is currently working on a similar type of project with the Epstein-Barr virus (EBV), while mentoring a graduate student in his department. Together, they are co-authoring a paper detailing the findings of their research.

Mentorship is pervasive throughout Dr. Chandran’s lab, and comes from the top down, according to Dr. Roy. “Dr. Chandran is very involved with the six postdocs working in his lab. We meet with him weekly or even daily if we have something interesting to share. Most of us design our own experiments and then Dr. Chandran makes suggestions for how to mold the project in a way so that it can become more interesting. He helps us to think independently, and also encourages us to think out of the box.”

Dr. Roy appreciates the collaborative environment he’s discovered at RFU. “It’s very motivating – everyone around you is performing well, which drives you also to put in that extra effort so that together you can do something really good. Sharing knowledge, that’s how the scientific community works.”

Hypothesis
Throughout the year, graduate students and postdocs attend a variety of research-focused conferences, seminars and poster sessions, as well as workshops on topics such as preparing and presenting a scientific paper. These events, considered vital for professional development, provide students and postdocs with opportunities to network, share their research and connect with the larger scientific community.
The Career Enhancement and Development Program for Postdoctoral Fellows sponsored multiple events and workshops this year:

Dominik Duelli, PhD, RFU adjunct assistant professor of cell biology and anatomy and principal R&D scientist at AbbVie, and Thomas Macek, PharmD, PhD, scientific director of clinical sciences CNS at Takeda Development Center Americas, both presented seminars on working in industry.

Heather Snyder, PhD, senior director, medical & scientific relations for the Alzheimer’s Association, presented a seminar on working in non-profit organizations.

Rekha Hanu, PhD, JD, director, intellectual property at Akorn Pharmaceuticals, presented a workshop in careers in pharmaceutical industry/patent law.

Bethany Brookshire, PhD, Scicurious blogger and science education writer at Science News and Eureka! Lab, presented a lunch-and-learn seminar on science writing and blogging.

Joan Lakoski, PhD, and Robert Milner, PhD, delivered a half-day, in-depth discussion of the process for developing successful NIH grant applications, including a mock study section by NIH study section participants at RFU.

SGPS celebrated the seventh annual National Postdoc Appreciation Week, Sept. 19–23, 2016, with a mentor/trainee ice cream social and a screening of “The PhD Movie 2 – Still in Grad School” (from the creator of “Piled Higher and Deeper” web comic).
Every day, our graduate students and postdoctoral fellows commit themselves to scientific discovery and academic excellence. And every year, many of these hard-working individuals are recognized by this university and the larger scientific community for their efforts. We are proud to share with you several of this year’s honorees.
Lisa Monteggia, PhD ’99, was featured in the fall 2016 issue of Rosalind Franklin University’s Helix magazine. The article followed her “nontraditional path to academic biomedical research” and highlighted the importance of “high-quality mentorship” in a trainee’s professional development. Dr. Monteggia received her PhD in neuroscience under the mentorship of Dr. Marina Wolf.

Stuart Richer, OD, PhD ’96, was interviewed for an article in AOA Focus magazine titled “6 Nutrition Questions You Should Be Asking Patients.” Dr. Richer completed his PhD in physiology and biophysics at RFU under the mentorship of Dr. Richard Rose and is now a clinical associate professor of family medicine at the Chicago Medical School.

Nagaraj Kerur, DVM, PhD ’11, visited RFU in February 2016 as an invited speaker to discuss the innate immune mechanisms of age-related macular degeneration. Dr. Kerur received his PhD in microbiology and immunology under the mentorship of Dr. Bala Chandran, and is now an assistant professor of ophthalmology and visual sciences at the University of Kentucky.

Sayan Chakraborty, PhD ’12, presented a special seminar entitled “Elucidating an Oncogenic Role of Agrin in Liver Cancer” at RFU in April 2016. Dr. Chakraborty, who completed his postdoctoral training under the mentorship of Dr. Bala Chandran, is now a senior postdoctoral researcher at the Institute of Molecular and Cell Biology at A*STAR in Singapore.

In May 2016, the Illinois Board of Higher Education approved a proposal submitted by SGPS to begin offering a PharmD/PhD program. This new degree offering is designed to educate and train future clinician-scientists toward careers that unify biomedical research and clinical practice for the wellness of the population. The proposal and corresponding academic processes were developed via collaboration between SGPS and RFU’s College of Pharmacy. This new combined degree leverages the particular strengths of both colleges as well as the collaborative and interprofessional nature of RFU.

SGPS initiated a major review and planned revision of its core year Interdisciplinary Graduate Program in Biomedical Sciences (IGPBS) curriculum in 2016. This revised curriculum, designed to ensure that SGPS is keeping pace with the rapidly changing body of knowledge in basic sciences, advances in bench and computer technologies, and education of future biomedical researchers in this changing landscape, also incorporates additional career-centric competencies such as scientific writing.

Project Dreams, a community service project led by Kalpit Shah, PhD ’16, was featured in the Lake County News-Sun on May 18, 2016. The Project Dreams initiative brings military veterans to RFU to learn research lab skills. Dr. Shah developed the project as part of the Rosalind Franklin Fellowship Program. The initiative continues in its second year under the guidance of physiology and biophysics student Jiaju Wang, with the help of cell biology and anatomy student Jessica Centa.

RFU hosted 92 girls ages nine to 14, from schools in Waukegan, North Chicago, Wadsworth and Zion, during the annual Empowerment Day for Girls Conference held in April 2016. Aimed at inspiring middle school girls to enter STEM fields, the event featured a keynote address by SGPS alumna Nicole Woltowich, PhD ’16, founder of Women in Scientific Discovery or Medicine (WISDOM), a mentoring group for undergraduate women who are interested in but underrepresented in STEM and healthcare-related fields.
Publications & Presentations

JOURNAL PUBLICATIONS


ABSTRACTS, REVIEW ARTICLES AND BOOK CHAPTERS


MEETINGS AND POSTERS


Chandrasekharan JA. November 2016. “KSHV infection alters inflammatory microenvironment.” CAVA Symposium, Loyola University, Chicago, IL.

Christian DT, Tseng KY, Wolf ME. November 2016. Extended-access cocaine self-administration leads to increased GluN3-containing NMDA receptor function in the rat nucleus accumbens. Society for Neuroscience Annual Meeting, San Diego, CA.


Mandal T, Shin S, Aluvila S, Choe JY, Cheng EH, Oh KJ. October 2016. BAK assembles into homooligomers to form the mitochondrial apoptotic pore. Young Investigators Meeting, Chicago, IL.


Sawyer IA. December 2016. “Characterizing the relationship between nuclear bodies and the genome.” National Cancer Institute Symposium on Chromosome Biology: Nuclear Structure, Genome Integrity and Cancer, Bethesda, MD.

Stefanik MT, Wolf ME. November 2016. Incubation of cocaine craving and the regulation of eIF2α in the nucleus accumbens. San Diego, CA.


INVITED TALKS

Goswami, S. July 2016. Invited speaker at the Launch Party of the inaugural season of the ACS Real Men Wear Pink (RMWP) campaign, Chicago, IL.


SCIENCE, FOR ME, GIVES A PARTIAL EXPLANATION FOR LIFE. IN SO FAR AS IT GOES, IT IS BASED ON FACT, EXPERIENCE AND EXPERIMENT.

DR. ROSALIND FRANKLIN
1920–1958
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