8-2014

BenchMarks 2014, August 8

The Rockefeller University

SCIENCE FOR THE BENEFIT OF HUMANITY

BENCHMARKS

THE COMMUNITY NEWSLETTER OF THE ROCKEFELLER UNIVERSITY

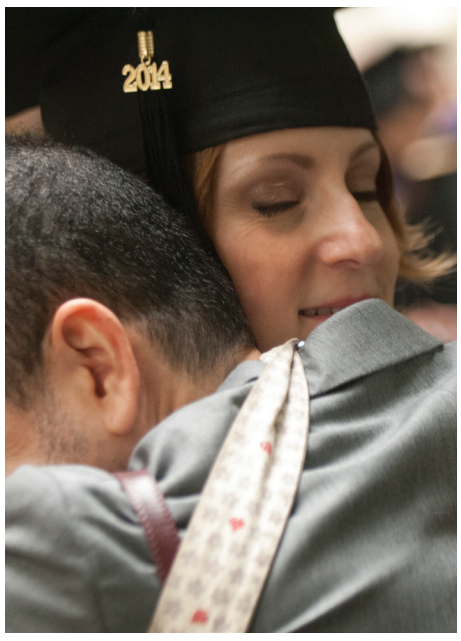
FRIDAY, AUGUST 8, 2014

The 2014 Convocation awarded 23 Ph.Ds to Rockefeller graduate fellows, bringing the total number of Rockefeller alumni to 1,150. The luncheon preceding the ceremony was held for the first time in the new, grandly restored Great Hall of Welch. Following tradition, faculty mentors joined their students in a procession across the campus, then presented each at a formal ceremony in Caspary Auditorium. Afterward, the campus community turned out for a reception in Weiss Café to celebrate the graduates.

Members of the class of 2014 — 14 men and 9 women — come from eight countries: Bulgaria, Canada, England, India, Singapore, Sweden, Taiwan and the United States. Of the graduates from the Ph.D. program, the majority will begin positions as postdoctoral fellows, while others have accepted positions in the biopharmaceutical industry and as consultants. Participants in the Tri-Institutional M.D.-Ph.D. Program will return to medical school to finish their medical degrees.

This annual Convocation issue of BenchMarks *salutes the university's class of 2014.*

To view more photos, visit www.rockefeller.edu/convocation.



CONVOCATION

FOR CONFERRING DEGREES · 2014

THURSDAY, THE TWELFTH OF JUNE

ALL PHOTOS: MARIO MORGADO PHOTOGRAPHY AND ZACH VEILLEUX

BENCHMARKS

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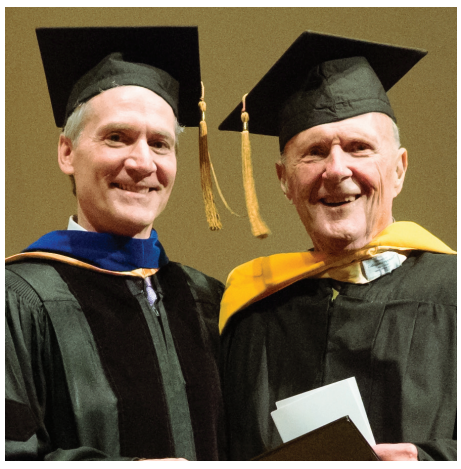
Honorary degrees awarded to Gurdon, Robertson and Yamanaka

by ZACH VEILLEUX

In addition to 23 students, three seasoned contributors to basic science — two Nobel Prize winners and a philanthropist — received degrees from Rockefeller this year. In a tradition dating back more than five decades, the university awarded honorary doctorate of science degrees to distinguished individuals who have made notable contributions in their fields. The recipients were: John Gurdon, distinguished group leader, Wellcome Trust/Cancer Research UK Gurdon Institute and the University of Cambridge; Julian Robertson, founder and trustee of the Robertson Foundation and chairman and chief executive officer of Tiger Management, L.L.C.; and Shinya Yamanaka, professor and director, Center for IPS Cell Research and Application at Kyoto University and senior investigator of the J. David Gladstone Institutes.



Third degree. John Gurdon (left), Julian Robertson (center, with President Tessier-Lavigne) and Shinya Yamanaka received honorary degrees this year.

Dr. Gurdon and Dr. Yamanaka are 2012 Nobel Prize laureates known for discoveries related to stem cells, and Mr. Robertson is an investor and philanthropist.

Dr. Gurdon, through iconic experiments with nuclear transplantation, demonstrated that a differentiated cell contains all the genetic information needed to guide the development of an organism, as well as how a mature cell could revert to an embryonic state. His work helped to launch the modern fields of stem cell biology and therapeutic tissue regeneration. Dr. Gurdon has a Ph.D. in zoology from Oxford University and joined Cambridge University in 1972, where he has remained since. He is also chairman of the Wellcome Trust and the Cancer Research Campaign Institute of Cancer and Developmental Biology, which was named the Gurdon Institute in his honor in 2004.

"Rockefeller is justly regarded as one of the world's most respected and successful universities by world standards," Dr. Gurdon said. "Recognition by Rockefeller is an extremely special and memorable event."

Mr. Robertson, an investor, environmentalist and philanthropist, is co-founder of Tiger Management, which he built from $8 million into one of the world's largest hedge funds, with capital of more than $23 billion. In 1996, with his late wife Josie, Mr. Robertson founded the Robertson Foundation to focus on high impact grants in education, the environment and medical research. Among the foundation's major initiatives are active support of New York City's public education reform, scholarships at Duke University and the University of North Carolina at Chapel Hill, combating global warming, and funding medical research at Memorial Sloan Kettering Cancer Center and Rockefeller. He has been a member of Rockefeller's Board of Trustees since 2001.

"The idea of any of us changing the world could be categorized as the ultimate conceit," Mr. Robertson said. "But in essence it is not about grand gestures but about simple, disciplined acts performed every day."

Dr. Yamanaka discovered that just four genes have the ability to re-program adult skin cells, allowing them to become induced pluripotent stem cells (iPS cells), a state similar to that of embryonic stem cells. Like embryonic stem cells, iPS cells have the capacity to differentiate into multiple cell types within the body, and they are a potential source of matched tissue for therapeutic transplantation. Dr. Yamanaka received his medical degree from Kobe University and his Ph.D. from Osaka City Graduate School. He has been on the faculty of Osaka City University Medical School, the Nara Institute of Science and Technology and the University of California, San Francisco.

"As the director of a newly established institute, I want to recruit outstanding scientists, I want to promote collaborations, I want to encourage our scientists to perform high-risk, high-reward projects, and I want to attract as many talented students as possible and help them to become brilliant scientists," said Dr. Yamanaka. "All of this is very difficult; however all of this was successfully done here at Rockefeller University. And that's why I am here today."

David Rockefeller Fellowship awarded to two neuroscientist third-years

by LESLIE CHURCH

Given annually, the David Rockefeller Fellowship is intended for an outstanding third-year student who demonstrates exceptional promise as a scientist and a leader. This year, for the first time, the award has been given to two recipients, a decision that is a testament to the high caliber of the university's student body, from which two equally strong candidates emerged: Raphael Cohn, of Vanessa Ruta's Laboratory of Neurophysiology and Behavior, and Alexander Nectow, of Jeffrey Friedman's Laboratory of Molecular Genetics.

The David Rockefeller Fellowship was established by alumni in 1995 as an expression of gratitude for Mr. Rockefeller's role in founding the university's graduate program and for his commitment to its success. Mr. Rockefeller has said that few honors have meant so much to him as the creation of this fellowship.

Mr. Cohn, who grew up in Canarsie, Brooklyn, had always had an interest in biology and the brain, but was skeptical of the possibility of achieving a complete understanding of such a complex system. It was a first-year graduate student seminar given by Dr. Ruta, a Rockefeller alumna herself who was awarded the fellowship in 2003, that changed his mind.

"I was amazed at her ability to use electrophysiology and imaging techniques to trace complete circuits in the fly brain," says Mr. Cohn. "It was the first time I really felt like we could actually begin to understand how the brain, as a whole, functions."

Mr. Cohn's thesis is centered on the mushroom body, a part of the olfactory processing pathway in the insect brain. The structure allows the fly to learn from experience — to connect a certain odor with a contextual clue such as an electric shock or a sugar reward. Mr. Cohn is using electrophysiology, functional imaging and other genetic tools to understand how neuromodulators like dopamine interact with synapses in the mushroom body to assign meaning to specific odors.

Mr. Cohn received his bachelor's degrees in computer science and engineering as well as cognitive science from the University of Pennsylvania, and earned a master of science in mathematics and the foundations of computer science from Oxford University. His passion for art, which led him to study as a Fulbright Scholar at the Bezalel Academy of Art and Design and the Technion Institute of Technology in Israel, also motivated him to organize weekly art classes at Rockefeller.

Fellowship. Raphael Cohn (left) and Alexander Nectow receive fellowship citations from Rockefeller alumnus Richard Bockman, professor of medicine at Weill Cornell Medical College.

Alexander Nectow also had a strong interest in neuroscience, and had intended to become an academic neurosurgeon before deciding that research — particularly the intersection of science and engineering — was his calling.

"Early on, I decided to split my time between questions that really fascinated me and technology developments to complement them," he says.

Mr. Nectow graduated with a bachelor's degree in engineering science and a master's in biomedical engineering from Tufts University. When he first visited Rockefeller as a prospective student, he found a place where everyone else was as excited about research as he was.

"I was blown away by the quality of the people here and the level of independence that I would be given," Mr. Nectow says. "There is so much incredible science going on in such a high concentration."

After joining Dr. Friedman's Laboratory of Molecular Genetics, Mr. Nectow was initially interested in the neural circuitry underlying susceptibility to neuropsychiatric disorders such as depression, but he soon realized that research was lacking on even the basic functions of those circuits. A new technology he has developed, called Retro-TRAP, has allowed him to profile neurons based on their patterns of projection — work that was published in *Cell* in June. He used the technique to identify molecular markers for the ventral tegmental area, a heavily dopaminergic structure in the midbrain.

Mr. Nectow also launched a journal club in the Friedman lab to discuss current topics and tools in neuroscience.

Mucida, Smogorzewska honored with teaching awards

Rockefeller University is best known for its innovative research. But the courses it offers, and the teachers who lead them, are no less impressive. Two such faculty members were honored at this year's Convocation luncheon with Distinguished Teaching Awards: Daniel Mucida, assistant professor and head of the Laboratory of Mucosal Immunology, and Agata Smogorzewska, assistant professor and head of the Laboratory of Genome Maintenance.

Dr. Mucida, who joined Rockefeller in 2010, is co-organizer of two courses. The immunobiology class is a two-hour weekly lecture and discussion featuring Rockefeller faculty and guest speakers discussing their research. Students write an original research proposal and review proposals from their peers. Experiment and Theory in Modern Biology introduces first year graduate students to the methods and principles behind current biological research.

Dr. Smogorzewska is a 2002 Rockefeller graduate and joined its faculty in 2009. The next year, she took the helm as organizer of Mammalian Genetics, a weekly, two-hour lecture and discussion that covers modern genetic tools including RNAi screening and genetic engineering using TALENS, human gene mapping, mouse genetics and human disease modeling, ethical issues in modern genetics and many other topics.

"Daniel and Agata's willingness to organize these rigorous courses is a real testament to their commitment to teaching and mentoring the next generation of young scientists," says Marc Tessier-Lavigne, the university's president. "Their commitment to teaching is an inspiration."

The 2014 Graduates

Below are the congratulatory tributes given to each of the 2014 graduates by their faculty mentors on June 12. Students in the Tri-Institutional M.D.-Ph.D. Program are denoted with an asterisk. Four students graduated in absentia: Rebecca K. Delker, Amanda Caroline Kohler, Suzannah J. Rihn and Johannes Fabian Scheid.

Jennifer Jeanne Bussell

presented by Leslie B. Vosshall

B.A., University of Chicago
Abdominal-B *Neurons Control* Drosophila *Virgin Female Receptivity*

I am pleased to present Jennifer Bussell to you today. Jennifer hails from South Carolina, where she graduated from the South Carolina Governor's School for Science and Mathematics on a full scholarship. She moved to Chicago to complete an A.B. with honors in Biological Sciences in 2004. At Chicago, Jen worked in Bruce Lahn's laboratory on the genetics of the PAR1 gene in humans and primates.

Before coming to Rockefeller, she took two years off from academic life and worked at a management consulting company in Boston. This experience challenged her with real-world problems and how to rock a business suit. While she came to Rockefeller fully intending to work in the area of biochemistry and gene regulation, her interests soon shifted to a fascination with innate behaviors. For her thesis, she set out on a completely independent project that sought to understand how a female chooses a mate. In her case, she focused on the fly *Drosophila melanogaster*. For the last 100 years, scientists have described almost every aspect of male fly sexual behavior, but largely ignored the female. Jen set out to correct this imbalance and carried out an extremely ambitious and far-ranging thesis project that identified an entirely new female behavior, pausing, which precedes copulation. She pinpointed a set of neurons that control this pausing behavior, and showed that females with these neurons inactivated are aloof and will reject the advances of interested males.

Because my laboratory actually does not work on this problem, Jen was completely on her own to gather reagents, design assays and surmount technical difficulties. Throughout this, Jen was resilient and persistent and had the epic patience to comb through many terabytes of videotapes of flies courting each other. This dedication paid off with an important paper that will publish this summer. Jen will go on to join Richard Axel's laboratory at Columbia for postdoctoral training later this year to continue her investigations in the mysteries of genes controlling innate behaviors. I join Jen's parents, friends, Logan and baby Sadie in congratulating her on this special day.

Rohit Chandwani

presented by Alexander Tarakhovsky

A.B., Harvard College
M.D., Yale University School of Medicine
Stochastic Activation of Enhancers in the Innate Immune Response by the Histone Demethylase JMJD2D

Rohit Chandwani joined my lab after completing his M.D. training. He was generally interested in epigenetics, apparently due to the lack of this term in his textbook for surgeons. After several fatal surgical attacks on epigenetics of the immune response, Rohit decided to dedicate his impressive enthusiasm and intellect to the problem of how cells' diversity can help us to establish an effective antiviral response. While infected cells, e.g. in the liver or lungs, may appear similar, they display quite a remarkable level of population diversity that enables specialization of cells during response. Rohit has identified a chromatin regulator that can fulfill the function of diversifier.

During his Ph.D., Rohit taught me a few lessons about having a surgeon do basic research in the lab. First, your life will not be the same to the extent that you may not recognize your lab anymore; second, the bills for one day of Rohit's experiments could rival the bills for treatment of members of the Middle East Royal Family for the entire duration of treatment; third, I do not hope to stay in touch — his agenda is too busy and he is with the next patient.

Overall, I am very proud of Rohit. I do not think that there are many doctors who are equally well-versed in the most sophisticated surgical manipulation and in chromatin biochemistry. I am very proud that Rohit is joining Memorial Sloan Kettering where he will certainly excel as a surgeon and researcher.

Chiung-Ying Chang

presented by Elaine Fuchs

B.S., M.S., National Taiwan University
Coordinating Stem Cell Behavior in the Hair Follicle

Chiung-Ying Chang received her bachelor and master of science degrees from National Taiwan University. She joined my laboratory in summer 2009, after completing her rotations and her first year in our graduate program. I was delighted that she chose my lab, as she is a careful experimentalist and is able to think through scientific problems, devise and perform experiments for testing her hypotheses and interpret her results with accuracy.

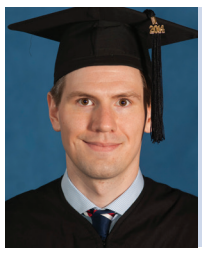
Chiung-Ying's graduate project centered on defining the role of transcription factor Nuclear Factor I B (NFIB) in mouse skin development. This transcription factor had initially surfaced as being more abundant in hair follicle stem cells than other cells of the skin. Little else was known about this factor, except that it is found in a number of tissues of our body.

Chiung-Ying used mouse genetics to engineer a null mutation specifically in the hair follicle stem cells of laboratory mice. At first she was disappointed in that the mice were viable and grew a seemingly normal hair coat. As the mice grew, however, their ears started to become black. For me this was exciting as I grew up watching the Mickey Mouse Club show. Chiung-Ying took wiser joy in sleuthing the underlying reason.

I won't elaborate on the details, which were published in *Nature*, but Chiung-Ying had inadvertently uncoupled the normal crosstalk that takes place in the hair follicle stem cell niche that allows the melanocyte stem cells and the hair follicle stem cells to become activated and differentiate at the same time, so that differentiating melanocytes can deliver melanin to differentiating hair cells so that your hairs are pigmented. NFIB turned out to repress a gene in hair follicle stem cells that encodes a secreted melanocyte differentiating factor. To get to the bottom of the story necessitated complex mouse genetics, cell and developmental biology approaches, in vivo chromatin immunoprecipitation and deep sequencing, RNA-sequencing and functional analyses.

What I particularly liked about Chiung-Ying's work is that many would have overlooked the phenotype and turned to another project (particularly if they did not grow up learning about Mickey Mouse). Chiung-Ying pays attention to details, she tackles problems head on and has the dedication and persistence to stick with an interesting scientific problem until she gets to the bottom of the story. This kind of strategy often pays off, but so very few have the motivation, patience and conviction to take this approach at such a young age.

Throughout her studies in my laboratory, Chiung-Ying showed a high level of enthusiasm and took a genuine interest in her science. It was Chiung-Ying's insight and observation that led to her exciting thesis, and she's developed into a fine and confident young scientist. She's now in the laboratory of Gerald Crabtree at Stanford, where she's learning the hard-core transcriptional biochemistry to round out her skills gained in my lab. She's back today to receive her diploma, and I look forward to watching her progress on the other side of the U.S.



Eric Fritz

presented by F. Nina Papavasiliou

A.B., Harvard College
Genome-wide Characterization of the Effects of Nucleic Acid Modifying Enzymes: Cytidine Deaminases and DNA Methylation

In biology, to show that something happens, what we call a positive result, is easy. To demonstrate with a high degree of accuracy that something does not happen, which we call a negative result, requires a type of meticulous and precise analysis that is uncommon to biologists.

Eric Fritz was trained as a chemist, and brought his meticulous and precise thinking to a very messy biological problem. This problem concerns B cells. These are the cells of the body that produce antibodies. But they don't just produce them, they also tweak them to fit bits and pieces that are foreign to the body, and tag them for destruction. This tweaking process requires the activity of an enzyme, which normally mutates the gene fragments from which antibodies are assembled, to produce families of mutant antibodies, some of which bind the foreign particles with very high affinity.

Intuitively, it makes sense that such a mutator would need to be particularly well regulated in the cell, or else it could attack other genes and cause all sorts of problems. Many labs have contrived artificial situations to show that, indeed, human intuition must be correct, because the presence of the unregulated mutator can be very, very bad news for the cell. But is it, really? Intuition aside, Eric Fritz relied on experimental data to demonstrate that under normal conditions, the only thing the mutator does is tweak antibody genes and that, at least in B cells, it has none of the terrible other activities ascribed to it. This body of negative data, of describing what a molecule does not do, was so compelling, that it was accepted in one of the top journals without a single revision — a rare occurrence, as I'm sure most of you know. Eric's work has set the bar high.

We'll miss Eric, his thoughtful precision but also his dry humor, as he moves on to the next phase of his career.

Paul William Furlow*

presented by Sohail Tavazoie

B.S., Michigan State University
M.S., Northwestern University
Mutations in a Mechanosensitive Channel Enable Intravascular Metastatic Cell Survival

Paul infuses a large dose of vitality into all that he does. This is most apparent during our annual lab karaoke outings, where he accomplishes two goals: the first is to teach us non-native English speakers a rich and new vocabulary. The second is to ensure a Michigan State style celebratory intoxication, which sadly makes us all forget the flavorful words we had learned.

In my lab, Paul studied the mechanisms by which cancer cells spread — or metastasize — to distal organs. To metastasize, cancer cells must leave the primary tumor site, enter the blood, travel to distant organs, and must squeeze and stretch through very tight blood vessels feeding into those organs. During this last step, the vast majority of cancer cells perish. However, rare cells are able to survive these traumatic forces and squeeze their way into organs where they form tumor colonies.

Until Paul's studies, we did not know how these rare cancer cells accomplish this feat. Paul found that cancer cells activate a channel upon mechanical deformation that allows them to survive. He further showed that patients whose tumors possessed more of this channel were more likely to have their cancers metastasize. Finally, Paul showed that he could use a small-molecule drug that blocks this channel to reduce the spread of breast cancer in mice.

Paul's work, for the first time, revealed a molecular mechanism by which cancer cells overcome this major physical barrier to metastatic spread posed by distal organs. To tackle this problem, Paul had to develop several techniques that did not previously exist in my lab since this was an entirely new area of study for us.

Paul has a number of unique traits that will ensure his continued success. He doesn't rest until he finds the answer to a question, and he is naturally drawn to challenges. His ability to engage, communicate with, and inspire us will continue to lead him into exciting uncharted territories at the interface of biology and disease. Paul, I'm proud of all you have done and I'm sure you'll bring the same energy and passion to your patients as you did to science. Just one bit of advice for those non-native English speaking patients: please use an interpreter.

Daniel B. Gilmer

presented by Vincent A. Fischetti

B.S., Howard University
Studies of a Novel Phage Lytic Enzyme, PlySs2

Bacteriophages, or phages for short, are viruses that infect bacteria. There are about 10 million phages per gram of soil or milliliter of water, so recent estimates predict that there are approximately 10^{31} phage on earth, making it the most prevalent biological entity on the planet.

When phages infect bacteria they replicate for about an hour, after which time they need to exit the bacteria to release their progeny phage to infect other bacteria. They exit by producing an enzyme called lysin that drills holes in the bacteria causing them to explode and die and, in doing so, release their progeny. Our lab was the first to successfully develop purified forms of these enzymes for therapeutic purposes. Adding purified lysin to a bacterium will punch a hole in the cell killing it instantly, much like popping a balloon with a pin.

Daniel was interested in identifying new lysins that would efficiently kill certain disease bacteria. He selected to work on a lysin that killed *Streptococcus suis*, an organism that causes serious infections in pigs but also could infect and sometimes kill pig workers and handlers. Daniel did a great job purifying and characterizing the enzyme called PlySs2, and when he checked to see what other organisms the enzyme was able to kill, he found to his surprise that it killed a wide range of disease bacteria that also caused serious human infections, particularly antibiotic resistant staphylococci called MRSA, responsible for deadly hospital infections, and *S. pyogenes*, responsible for strep throat and other serious infections like "flesh-eating disease." Daniel was able to show in a mouse model of blood infection that the lysin was able to kill both bacteria at the same time in mixed infections, saving the lives of these animals. The enzyme worked so well that it has been licensed by a biotech company which is developing it to treat MRSA infections in hospitals.

Not only was Daniel busy in the lab, he was also busy at home where he and his wife Chanel are proud parents of two children, Elijah and Kasja, who arrived during the last years of his Ph.D., during a time when Chanel was completing her master's degree.

In addition, Daniel has been very active in helping others whenever he can. As a Howard Hughes fellow, he was president of the Howard Hughes Medical Research Scholars Program, where he provided leadership to twelve research scholars. He also successfully mentored one or two summer students each year, and is the founder of MenBuild, a ministry that connects African American professionals with undergraduate students.

Daniel will now move from science to business where he will participate in an analyst training program at McKinsey and Company.

Claire Ellen Hamilton*

presented by F. Nina Papavasiliou

B.S., Yale University
Transcriptome-wide Characterization of APOBEC1-catalyzed RNA Editing Events in Macrophages

It is uncommon for a graduate student to work in a brand new area, especially in a branch of biology, such as immunology, where many feel we kind of know everything there is to know. But Claire Hamilton's thesis work involves just that.

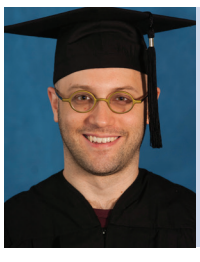
The transfer of genomic information from DNA to mRNA to protein is assumed to occur with high fidelity. However, this transfer can be specifically subverted by a variety of programmed RNA sequence alterations the most prominent of which are alterations that lead to base deamination, and the recoding of two DNA bases: base A (adenosine) as base I (inosine), or base C (cytosine) as base U (uridine).

Collectively these alterations are termed RNA editing and if you can remember two things about these types of events, you should remember, first, that these are stealthy alterations that do not leave a mark on the genome, and second, that they are extremely frequent and reproducibly robust in many immune cell types.

Claire's thesis work examined the functional relevance of an RNA editing enzyme called APOBEC1; in a paper that we are about to send out, she and her lab mates describe how this enzyme targets clusters of specific transcripts within immune cells, how this targeting results in differences in protein levels within individual cells, and how these differences produce functional diversity within immune cell populations. Claire's data predicts that RNA editing, by endowing each cell with slightly different informational content, stealthily plays a key role in the plasticity and adaptability of the immune repertoire.

It is also somewhat fitting that Claire is standing on the platform today along with one of the early pioneers in the field of APOBEC1 editing. Many of you will not know this but one of our honorary degree recipients started his career describing APOBEC1's role in promoting tumor progression in the liver. It took 20 years and the advent of new technologies to be able to take the next step but I think Dr. Yamanaka will be happy to know that his prescient hypothesis about how editing drives cancer progression was spot-on!

Claire is now back at the wards, finishing her dual degree. We already miss the unique energy she brought to the lab: intellectual but also physical — yoga challenge anyone? I am certain that this same energy will carry her far in her career from here on.



Evan Heller

presented by Elaine Fuchs

B.A., Columbia University
Forces Generated by Cell Intercalation Tow Epidermal Sheets in Mammalian Tissue Morphogenesis

Evan Heller contacted me shortly after he was accepted to Rockefeller's Ph.D. Program, and inquired about a possible rotation in my lab. He received his bachelor's degree from Columbia, where he worked in the laboratory of Mike Sheetz, whom I know very well and works on scientific problems that are similar to our own lab interests. From our early discussions about science, it was clear that Evan was going to do well in our graduate program here. In watching his progress throughout his Ph.D., my favorable impression only continued to strengthen. Like many of our students here at Rockefeller, Evan has a powerful intellect, and is extremely talented at the bench. Yes, Evan is hard-working too. He also has a very positive attitude and he is one of the best liked people I've ever had in my laboratory.

So why did it take Evan so many years to complete his dissertation? It wasn't from lack of productivity, as Evan's thesis research, published in *Developmental Cell*, was termed a tour de force by his reviewers, who had glowing remarks about his work and have predicted that the work will be a landmark in the field. Many have since praised this paper. Evan was also instrumental in his contributions to our whole-genome wide screens in mice for oncogenic regulators and tumor-suppressors of growth. And he was instrumental in our screens for self-renewal factors in stem cells. And he was instrumental in our studies on aging in skin. And in our studies on planar cell polarity in skin development. In fact, Evan was an invaluable contributor to six major papers published during this time. One insight surfaces when I tell you that these contributions were entirely at Evan's initiative, rooted in his broad curiosity and passion for science, not just his own but also those around him.

Another insight surfaces when I tell you that Evan's Ph.D. thesis was not on skin, stem cells or cancer, the mainstream topics of my laboratory. Rather, Evan chose to focus on the molecular mechanisms underlying how the eyelids form during embryonic development. In mammals, our eyes form before we develop eyelids. Prior to Evan's interest in the problem, no one understood how this happens. Is it localized proliferation at the border of the eye? Is it active migration? Is it a contractile mechanism, analogous to a purse string mechanism of closure? Evan showed that in fact the epithelium locally reshapes, expands and undergoes a collective movement over the eye. Evan showed that this process of cellular sheet movement is distinct from all the speculative models that were suggested at the time, and in fact different from all known mechanisms of cellular movements that were previously described, whether in wound-healing, or in fly or worm development. Evan showed that forces generated by cell intercalations at the border of the eye are leveraged to tow the surrounding tissue over the eye to form the eyelid.

To sleuth his way through the mechanism, Evan used live imaging of the process, laser ablation and quantitative analyses of tissue deformations, several other cell and biophysical strategies and some classy genetics. To choose a problem such as this one required fearlessness and passion; to solve the problem necessitated setting up the project from scratch, acquiring a diverse molecular toolbox of skills and crafting experimental strategies when no blueprint existed.

So in devoting this time to his thesis research, Evan has become a true scholar of biology. His depth of thinking is profound and his creativity and originality is exceptional. And all along the way, Evan has maintained his upbeat personality and honed his passion for science and collaboration. In my view, Evan is the perfect example of what our university strives for in developing the best and brightest minds in science.

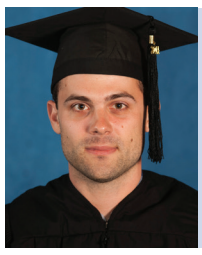
Jessica Sook Yuin Ho

presented by Alexander Tarakhovsky

B.S., University of Wisconsin–Madison
Chromatin Control of the Antiviral Response to Influenza

Jessica Ho joined the university as part of the A*STAR program from Singapore. She entered as a "star" and never ceased to be one. Jessica contributed to the identification of a novel mechanism of influenza pathogenesis. Viruses, such as influenza, attempt to conquer cells without killing them but to use them for the benefit of virus replication. There are many ways that viruses can do this. Jessica found an influenza protein, responsible for viral pathogenesis, which can mimic the histone proteins that are at the core of cell gene expression machinery — and by doing so, can attenuate cell efforts to kill the virus. This phenomenon of histone mimicry by viruses raised questions about the possibility of viral impact on cell differentiation.

Although from Singapore, Jessica reminds me of a character from "Russian Women" written by the nineteenth century Russian poet Nikolai Nekrasov. In this poem, Nekrasov said of Russian women that they can equally well stop a galloping horse or enter a burning house to rescue a child, all while maintaining unperturbed calm, dignity and beauty. There were lots of galloping horses and burning houses and even child-like personalities, but Jessica mastered it all.



Matthew Thomas Holt

presented by Tom Muir

B.S., Western Washington University
Identification of a Functional Hotspot on Ubiquitin Required for Stimulation of Methyltransferase Activity on Chromatin

Matt Holt hails from Seattle and, like many from that part of the world, was lured to New York by the promise of better coffee and a more diverse climate. Having majored jointly in biochemistry and chemistry, Matt found his way to my lab where he became interested in how a molecular "kiss of death" could be redeployed as an "embrace of life." More specifically, Matt became interested in how protein ubiquitylation (a modification more associated with destruction) helps positively regulate transcriptional outputs in the context of chromatin.

Previously my lab, working with colleagues here at Rockefeller, had shown that ubiquitylation of histone 2B directly stimulates key methyltransferase enzymes involved in transcription initiation and elongation. But how does ubiquitin do this? This is what drove Matt's thesis research. Matt's approach was to systematically tinker with the system in the hope of finding its essence — basically a structure-activity analysis on chromatin. Doing such a thing was but a pipe dream when Matt started — our approach to manufacturing the ubiquitylated chromatin was precise, but highly artisanal, and it would have taken decades to make all the molecules Matt needed to rigorously perform the analysis. Faster methods were needed.

Solving this synthetic chemistry problem was at the heart of Matt's thesis work, and solve it he did, by developing ingenious approaches that allowed him to systematically mutate the entire ubiquitin protein surface in the context of a chromatin template. From this analysis Matt concluded, quite shockingly I think, that only a tiny portion of the ubiquitin surface is needed to stimulate the methyltransferases — he discovered the key "functional hotspot" on the protein. Moreover, he found that a different hotspot on ubiquitin is needed for other aspects of ubiquitin activity on chromatin. Thus, we have come to think of ubiquitin as an information rich histone modification, able to orchestrate different outputs, probably simultaneously, from the same locale. All in all, Matt accomplished an impressive body of work during his time at Rockefeller. His methodical and careful approach to science has paid off handsomely.

Matt was equally systematic in his explorations of the New York nightlife. Indeed, he discovered several additional "hotspots" during his explorations of downtown Manhattan. These included the amazing Mars Bar in the East Village — certainly the greatest dive bar in the western hemisphere. The recent demolition of the bar was barely even noticed by the regulars, but was widely viewed as the end of an era in the city.

Matt's graduation sees the end of an era in my lab, as he was the last student in my group actually working at Rockefeller, with Tarun Kapoor's help. But as a chapter ends for me, a new one opens for him, and so I wish him all success in the future.

James Letts

presented by Sidney Strickland on behalf of Roderick MacKinnon

B.Sc., University of Victoria
Functional and Structural Studies of the Human Voltage-gated Proton Channel

I apologize, James, for not being here in person to celebrate this well deserved accomplishment. Congratulations on your much deserved doctoral degree! There are few good excuses for missing this occasion, but I know you will agree that my excuse is acceptable: I was finally granted time on the Titan Krios Electron Microscope at HHMI, so it is for the pursuit of science that we love.

James joined our laboratory in 2008. He undertook the extremely challenging problem — with full knowledge beforehand — of studying an ion channel called Hv, a voltage-dependent proton channel.

This channel is important, especially to the ability of white blood cells to combat bacterial infection. The channel is intrinsically unstable and thus had never been purified and reconstituted. An important question was still debated in the field: does Hv in the absence of other proteins really encode the channel?

James settled this debate through expression, purification and reconstitution. He developed a nice mathematical model to interpret the proton fluxes. He then learned and applied X-ray crystallographic and NMR methods to characterize many aspects of Hv channel structure, though a complete native structure of the ion channel still eludes us. James' thesis work was technically of the highest quality and marked by cleverness and perseverance. He has now taken his mastered skills across the Atlantic to England, where he is studying in the laboratory of Leonid Sazanov.

James was a wonderful student to work with. We miss his enthusiasm for science, his critical comments in lab meeting, his "hey, let's have a movie night," and of course something will definitely be different at lab barbecues without a mad scientist brewing instant ice cream in a cloud of nitrogen vapor! Good luck, James. I look forward to great achievements in your scientific future.

Jeff Liesch

presented by Leslie B. Vosshall

B.S., University of Maryland–College Park
The Neuropeptide Regulation of Host-seeking Behavior in Aedes aegypti *Mosquitoes*

I am pleased to present Jeff Liesch to you today. Jeff came to Rockefeller with impressive research credentials, real-world experience in industry, and letters that raved about how smart, organized and effective he was. Indeed, Jeff is extremely smart, organized and effective, and in my lab he kick-started our mosquito research program at a time when he was the only person working on this creature.

He graduated from the University of Maryland–College Park with high honors. Jeff's thesis project in Caren Chang's plant laboratory established the roundworm *C. elegans* as a model system and won the award for best undergraduate thesis. After graduating from college, Jeff took a few years off to carry out atmospheric research on an NOAA research vessel in Maine and worked in industry as a media and web consultant. In my laboratory, Jeff was the first to figure out how to get mosquitoes to behave, by which I mean that he designed and built assays in which mosquitoes would tell us how attracted they are to a given human.

As chance would have it, Jeff is among the most attractive humans (to mosquitoes) ever to work in the lab. Jeff was on the hunt for the genes that cause a female mosquito to switch into a long-term state of ignoring humans after she takes a blood-meal. His thesis work identified a family of neuropeptide receptors that is important for modulating mosquito behavior: he systematically cloned these genes, matched receptors to the peptides that activate them, and made a mutation in one of these receptors.

Throughout his thesis work, Jeff impressed me with his precise thinking, incredible technical skills and collegiality. He tracked the detailed progress of every element of his thesis with project planning software — which was an impressive and unusual thing to see in an academic research laboratory. Jeff is also an amazingly nice person and was a pleasure to have as a colleague. Jeff recently transitioned to the private sector and is applying his smart, organized, and effective self to work as a senior consultant in health sciences at Navigant.

I join his family, friends, Josefina (his brand-new fiancée) and Lola (their dog) in congratulating Jeff on a fantastic performance as a scientist here.

María Maldonado

presented by Frederick R. Cross on behalf of Tarun Kapoor
member of the graduating class of 2013

B.A., M.Sci., University of Cambridge
Examining the Regulation of Cell Division by the Spindle Assembly Checkpoint

When we enter a different country we need valid passports to satisfy immigration officers and no contraband to keep customs officials happy. Similarly, our cells have molecular checkpoints to ensure that transitions between different states are only made when everything is in order. In particular, checkpoints ensure that our cells do not lose any of their DNA when they split into two daughters. This division process is repeated about 10,000 trillion times in our lifetimes and errors can lead to developmental defects and diseases.

María Maldonado devised a very clever solution to address the long-standing problem of determining which proteins detect errors during cell division and which correct errors. She engineered a checkpoint protein so that the checkpoint was blocked. In essence, she instructed a customs officer to not let anyone through the gate, but to allow all preceding steps to continue without interference.

Her powerful approach helped dissect the functions of many different proteins, including enzymes that are targets of new drugs being developed to treat cancer, at distinct steps of the cell division checkpoint. Her findings have uncovered fundamental cellular mechanisms and are likely to help improve anti-cancer therapeutics.

My lab members and I truly enjoyed working with such a creative and talented scientist. María currently holds the record for efficiency in completing thesis work in my laboratory. Not surprisingly, she has already completed another advanced degree in business.

Christina B. Marney

presented by Robert B. Darnell

B.Sc., University of East Anglia
RNA Deregulation in Metastatic Breast Cancer

Tina was a top honors, straight A student as an undergrad at the University of East Anglia, which turns out to be just beneath a place called the Twenty Acre Wood, likely where Tina got her Winnie the Pooh-like thoughtfulness and her questioning nature. For example, Tina is exceedingly smart and consistently polite when she doles out absolutely penetrating and sharp questions at our weekly lab meetings.

She has Pooh-like ambition, not for honey to eat, but for the honey of science, discovery. Tina has boldly climbed the tree of science projects in our lab, going out on branches ranging from comparing Nova regulation in human and chimp (relating to her days working in Charles Gilbert's laboratory), to Nova regulation of splicing in breast and ovarian cancer.

Perhaps also Pooh-like, Tina has always been creative and willing to buck dogma. She has not been afraid of changing her hair radically every year or two, and she was ambitious enough to make, by hand, a wonderful mermaid suit for a parade. In science, in the same way, Tina developed a brave and creative project to tackle the very hard question of Ago/miRNA in primary versus metastatic cancer. This work has potentially explosive implications, and was done with terrific support from the laboratory of Sohail Tavazoie.

This work then segued to the study of a new RNA binding protein, RBM47, which Tina found, together with her collaborators at MSKCC, is a suppressor of breast cancer progression. Tina did an enormous number of experiments, generating voluminous data using new, state-of-the-art technology. This led to a discovery exploring an entirely new frontier — mRNA regulation in metastatic versus primary cancer — and culminated in a first author paper published jointly with Joan Massagué at MSKCC.

Finally, Tina has been patient, has worked incredibly hard, through tough times and good times, always putting her deepest effort and commitment into the lab. She has truly been one of the most wonderful graduate students I've had the fun to work with, while developing the wisdom of a graduating Rockefeller Ph.D. student. She has not only developed the skill of listening to advice, but has also taught me the importance of listening to her wisdom.

As Pooh once said, and as Tina has taught our lab, if the person you are talking to doesn't appear to be listening, be patient. It may simply be that he has a small piece of fluff in his ear.

Jacob N. Oppenheim

presented by Marcelo O. Magnasco

A.B., Princeton University
Charting the Vasculome: High Resolution Maps of the Vasculature of Entire Organs

Richard Feynman said prophetically in 1959, as he heralded both nanotechnology and molecular biophysics, that "there's plenty of room at the bottom." In biology the last few decades have seen an explosion in our understanding of the bottom, meaning the molecular scale, and this revolution is here to stay. However attention is now also turning into how to build up from the smaller scales. We can now say that "there's plenty of room in the middle," meaning that there is still plenty to understand of the principles of mesoscopic organization of organs between the gross scale depicted in *Gray's Anatomy* and the finer structures of a pathologist's slide.

Jacob Oppenheim attacked this problem ferociously, even though, having been trained mostly as a theorist, he was initially ill-equipped to do so. His direct interest was in vasculature, and he concentrated on an organ whose relatively simple structure still remains mysterious in the middle: the liver. Jacob attacked, single-handedly, the construction of an automated cryosectioning imager capable of imaging the 3D structure of liver vasculature at the scale of hundreds of gigavoxels. It was soon obvious this was not going to be a smooth ride, but he persevered. There was obstacle after obstacle, and the thesis committee insisted he carry out in parallel a Plan B. Which he did, to great success and four published papers. But he still pined for those hepatic fjords, and even though his committee felt he could graduate with plan B, he stubbornly asked for permission to continue attempting to finish a thesis on vascular liver structure.

Like a neurotic one-man orchestra, Jacob still persevered, and even as time was running out he succeeded to produce a hundred-gigavoxel 3D image of liver. He then wrote the algorithms to analyze these enormous stacks of photographs and convert them into the first-ever complete digital map of the vasculature of a rodent liver, a feat no smaller than the first, but requiring a completely different set of skills. Jacob prevailed twice. Jacob's work is illuminating, and it will continue to illuminate how the little pieces of vasculature are assembled together into structures at various scales of length in order to construct a highly optimized organ.

Nora Pencheva

presented by Sohail Tavazoie

B.A., Kenyon College
Identification of a MicroRNA Network that Regulates Melanoma Metastasis and Angiogenesis by Targeting ApoE

Nora hails from a tiny town in central Bulgaria. There are two things that she absolutely loves: her nightlife and her science. She approaches both with utmost passion and ferocity.

In my lab, Nora studied how melanoma spreads, or metastasizes, to distant organs. She found that melanoma cells that metastasize possess high levels of three specific small RNAs. These small RNAs block a gene called ApoE. Nora found that ApoE is a potent inhibitor of metastasis formation. By blocking ApoE, these small RNAs remove a key barrier that prevents their metastatic spread. ApoE works by preventing melanoma cells from migrating through tissue as well as blocking their ability to form blood vessels.

To see if ApoE's activity could be harnessed for therapeutic benefit, Nora treated mice with a small-molecule compound that increases ApoE. Remarkably, she found that this treatment reduced metastasis formation in mice and increased their survival. These findings are exciting because they suggest that metastatic spread of melanoma in humans might also be prevented by small-molecule drugs. Nora will continue to contribute to our understanding of cancer biology as a postdoctoral fellow in René Bernards's lab in Holland.

Nora not only has great intuition for science, she is also extremely rigorous in her work. She has been a tremendous role model and teacher for many in my lab. Nora, I'm very proud of all you have accomplished. Keep up the great work. We'll miss you dearly.

K. Rashid Rumah*

presented by Vincent A. Fischetti

B.S., Stanford University
The Origin of Multiple Sclerosis Revisited: The Case for a Soluble Toxin

Multiple sclerosis is a devastating neurological disease that attacks people in the prime of their lives. Though intensive research has gone on for decades, the cause of MS remains unknown. Rashid Rumah had been interested in this problem even before he entered the M.D.-Ph.D. program. Based on histopathological findings, Rashid had the idea that the brain lesions seen in MS patients appeared to be caused by a toxin — but what toxin?

Toxins can easily enter a host from intestinal bacteria that have been acquired from the environment. So he searched stool samples of MS patients to identify such a toxin-producing organism, but with little success, until, as luck would have it, he received a sample from a patient that was very early in the disease process. He was successful in isolating the organism — *Clostridium perfringens* type B — that produces ETX, a neurotoxin. *C. perfringens* type B are usually found in the intestines of ruminant animals like sheep and cows and can cause neurologic symptoms in these animals.

Armed with this new information, he went on to discover that ten times more MS patients had antibodies to ETX toxin than normal controls, strongly suggesting that they were exposed to the toxin. He also found that normal controls carried *C. perfringens* type A in their intestines, which prevented colonization of type B organisms, while MS patients were less likely to carry this blocking organism. All his data pointed to ETX as the candidate toxin responsible for the MS lesions. He then purified the ETX toxin and used it in a mouse model and showed that it caused neurological symptoms in these animals.

Though very promising, these preliminary data lay the groundwork for a direction to try to understand the etiology of this mysterious disease.

When not in the lab working with dangerous toxins, Rashid relaxes at local karaoke bars where he sings a few of his favorite songs. Rashid is now back at Weill Cornell Medical College next door, completing his clinical duties.

Neel Shah

presented by Tom Muir

B.S., New York University
Split Inteins: From Mechanistic Studies to Novel Protein Engineering Technologies

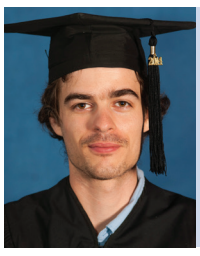
Neel Shah joined the Rockefeller graduate program in the fall of 2008 following undergraduate studies at NYU where he graduated with top honors in chemistry. Neel acquired a taste for two things during his years in Washington Square: firstly, protein chemistry, which ultimately led him to my lab, and secondly, insanely hot kebabs which ultimately led me to Prilosec.

Neel was an outstanding student, incredibly productive and an intellectual leader in the group. Although having no prior exposure to the world of the illusionist, Neel nonetheless became fascinated by the art of the escape artist. His "Harry Houdini" was not a man in a straightjacket but a protein called an intein precariously lodged in another protein. Like Houdini, inteins are consummate escape artists, being able to vanish from their immediate surroundings, in this case a protein host. Neel was driven to understand their secrets.

Now my lab has worked with inteins for years and we thought we knew all their tricks. Neel thought otherwise. Using a combination of informatics and cell based screens, Neel discovered a family of inteins that were not only split in half — two separate pieces — but that came together and then "escaped" in a matter of a few seconds; as far as I know, Houdini never pulled that off! The identification of these new ultrafast split inteins — so called because they escape several orders of magnitude faster than anything we had seen before — has been something of a watershed for my lab and beyond, enabling experiments that were impossible even a few short years ago when I was still in New York.

Following this initial discovery, Neel went on make many important contributions to the functional analysis and applications of his ultrafast split inteins — there have been lots of papers. My favorite relates to his work on the initial association of the intein fragments. Remarkably, the two polypeptides become topologically entwined in the final folded complex, forming a knot. Using a battery of methods, Neel figured out how they do this, elucidating the graceful molecular dance that allows the largely disordered intein fragments to capture each other and then, through a kind of adagio movement, fold into the final knot. Unlike Houdini, split inteins actually tie themselves up before escaping.

As alluded to, Neel has left quite an amazingly legacy in my group with many new projects and even a start-up company emerging from his thesis work. But, he also has left another, altogether darker, or at least enduring, legacy. As some of you know, I left Rockefeller a few years ago. What you certainly don't know is the real reason why, which brings me back to those caustic kebabs. In the end, my GI track could no longer handle the increasingly frequent Shah-led group trips to Mamoun's Kebab House in the West Village. I simply had to put some distance between myself and Mamoun's. Neel is now out at UC Berkeley where he is a Damon Runyon postdoc in John Kuriyan's lab — apparently he has a thing for ex-Rockefeller scientists.



Frej Tulin

presented by Frederick R. Cross

M.S., KTH Royal Institute of Technology
Exploration of Cell Cycle-specific Essential Gene Functions in the Microbial Plant Chlamydomonas reinhardtii

My laboratory has worked in the budding yeast model system for some years. Many labs have devoted a lot of effort over the years to understand this one organism as well as possible, resulting in a large amount of information as well as very advanced techniques. Although this approach could seem to reflect an undue interest in budding yeast entirely disproportionate to its importance aside from making beer, people stuck to it because it provided huge insights into animal cell biology, as a result of evolutionary conservation of basic functions. Microbes are generally much faster and easier to work with than are animals, so this strategy turned out to be very effective.

Despite these advantages, Frej Tulin wanted to work in a different organism, the green alga *Chlamydomonas*. His reason was that while budding yeast is a surprisingly good model for animal cells, it is much less good for the hugely important plant kingdom — a consequence of early evolutionary divergence. Frej reasoned that experiments in the microbial alga could yield insights into fundamental plant cell biology, much as yeast had for animals.

In principle this was a good idea, but there were two critical problems. First, next to none of the advanced methods available for budding yeast worked in *Chlamydomonas*; second, even the methods that did work were completely unfamiliar to me or to anyone in the lab, or indeed in the university. So the first thing Frej did was to spend a month in the lab of Susan Dutcher at Washington University, to learn the basics of dealing with *Chlamydomonas*. He returned knowing enough at least to get started.

The project he wanted to do was to identify and analyze most or all of the genes involved in control of the cell division cycle. This task was accomplished in the yeast system only over decades of work by a great number of researchers. So the proposal was to develop new methods for *Chlamydomonas* in a lab that had never worked on the organism, and to carry out a project that had taken great resources and thousands of researchers 20 years or so. This proposal was a bit less crazy than it sounds, because of recent advances in robotics for microbiology, and new DNA sequencing technologies that can greatly speed gene identification. Still, it was a very tall order. It would be necessary to collect and analyze huge numbers of mutants, while at the same time inventing new methods for this collection and analysis — a really challenging lift-yourself-by-your-bootstraps problem.

Ultimately, the project succeeded remarkably well. The list of genes is well on the way to being truly comprehensive, there is clear functional information as to the role played in the cell cycle by more than 50 genes, and, most important, there is clear conservation of much of what Frej has learned about in higher plants — justifying the fundamental assumption of the project. Thus, genes known for years to exist in higher plants, with no clearly assigned role in the cell cycle or perhaps anything else, now for the first time can be analyzed functionally in a rapid experimental system.

I'd like to take this opportunity to reinforce comments made by the graduate student speakers earlier — the strong commitment of the Rockefeller program to allowing students to work independently on subjects of interest, without undue worrying about financial support, was essential in allowing Frej to carry out this project. This is a unique aspect of the program, and we are grateful to all the people who make this possible.

Frej now plans to move on to work directly in higher plants, focusing on issues of development and chromosome organization that are not accessible from the alga system.

Siddarth Venkatesh

presented by Paul Bieniasz

B.Tech., University of Madras
Ph.D., Auburn University
Mechanism and Evolutionary Origins of HIV-1 Virion Entrapment by Tetherin

For his thesis project Siddarth Venkatesh worked on a protein, called tetherin, that is expressed in response to viral infection. Tetherin is a coiled-coil cell surface protein that traps nascent enveloped virus particles as they bud through the membranes of infected cells, preventing them from going on to infect other cells.

Sid worked on two problems: First, how precisely does tetherin work? By designing biologically active but cleavable and tagged derivatives of tetherin, Sid showed in molecular detail exactly how tetherin ensnares virions.

The second question that Sid asked was: From where did tetherin arise? It has a nearly unique configuration and no clear homology to any other protein. Working with another student, Daniel Blanco-Melo, Sid built a strong case that tetherin arose via duplication of a neighboring, similarly configured but more ancient gene, *PV1*, that encodes a component of caveolae. Most compellingly, Sid showed that a small change to PV1 could transform it, functionally, into tetherin.

Sid was determined, dedicated and talented but also generous, kind and always cheerful. His skills and deep commitment to his science were most obvious in the extraordinary quality of his work. His passion for science manifested itself in another way too; he is the only student I have known who was so moved by what he was doing in the lab that he composed poetry about it:

My name is tetherin, and my fortunes are on an upward spiral,
For I am an effector of intrinsic immunity; indeed, I am antiviral.
So when a virus is sensed, and the time is ripe,
I am induced by interferon in almost any cell type.
My amino acid sequence can be wild-type or chimeric,
But in order to be potent, I have to be dimeric.
With an anchor in the cell, and another in the virion,
Axially configured, I trap them by the million.
I have a predilection that is not entirely insane,
I'd rather insert my GPI anchor into the viral membrane
This facilitates endocytosis of the rogues that I hold,
Or, alternatively, I function as a signaling scaffold.
All in all, our struggles always live up to their billing,
My life in genetic conflict — endlessly fulfilling!

Today Sid is receiving his second Ph.D. (his first was from Auburn University in chemical engineering). He has just (finally) moved on to postdoctoral work on the microbiome with Jeff Gordon at Washington University in St. Louis. We wish him the very best in his new endeavors.

Coming soon, to The David Rockefeller Graduate Program

As the graduating class of 2014 moves on to the next stages of life and career, the Rockefeller community welcomes the incoming group of graduate fellows. There were 744 applications received this year, and after careful consideration by the admissions committee, 77 applicants were offered admission to the university. Twenty-five students will enroll — 14 men and 11 women from 8 countries: Bulgaria, China, India, Italy, Mexico, Norway, Turkey and the United States. Their alma maters include: Amherst College, Bard College, Bilkent University, Brown University, Columbia University, Cornell University, Indian Institute of Technology–Delhi, Ithaca College, Johns Hopkins University, Northeastern University, Occidental College, Purdue University, SUNY Geneseo, Stony Brook University, University of Bologna, University of California–Berkeley, University of California–Santa Barbara, National Autonomous University of Mexico, University of Oxford, University of Pittsburgh, University of Wisconsin–Madison and Wesleyan University.