

CELL AND MOLECULAR BIOLOGY STUDENT NEWSLETTER

Volume 1, Issue 2
August 2016

LETTER FROM THE EDITORS

Dear CAMB students, faculty, and alumni:

We hope that you enjoyed the inaugural issue of the CAMB Student Newsletter this past May, and we are grateful for the positive feedback and encouragement that we have received for this project. In the second edition, we'd like to offer a special welcome to the 47 CAMB matriculates of 2016. Orientation begins with a BGS event on Monday, August 29th and concludes with a CAMB welcome barbeque on Friday, September 9th. The intervening two weeks will be packed with fun events and faculty talks, and we hope that you enjoy meeting new classmates, upperclassmen, and faculty.

For a brief introduction into some of the exciting research performed by CAMB students, be sure to check out the Research Spotlight section of this newsletter. There, you will find commentaries covering recent publications out of the CPM and CBG programs. You can also learn about ongoing efforts to understand and combat the current Zika virus outbreak in a special interest article covering research from several labs at and around Penn.

We also encourage you to start thinking about your post-graduate careers as soon as possible. To aid current CAMB students with career exploration and to celebrate the accomplishments of former CAMB students, the Where Are They Now section of the newsletter highlights the careers of recent alumni. In addition, those specifically interested in teaching can learn about the resources available at Penn in a special interest article written by a recent student fellow for Penn's Center for Teaching and Learning.

For additional articles, past publications, and information about our organization, be sure to visit our blog at cambnewsletter.wix.com/blog.

Current students, including our 2016 matrics, who are interested in contributing to the newsletter or blog should contact us at camb.studentnews@gmail.com.

Sincerely,
Kate Palozola and Neha Pancholi
Editors-in-chief

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RESEARCH SPOTLIGHT

When tumor cells fight back

Camille Syrett

The immune system possesses the essential ability to detect and destroy abnormal cells, such as cancer cells. Recently, the critical interplay between the immune system and cancer has gained much attention, as scientists now recognize that the immune system holds great therapeutic potential for use in the targeted destruction of tumors. However, tumor cells can also exploit certain populations of immune cells to boost their own growth and evade immune system-mediated destruction. Recent research by fifth year Cancer Biology student and Rustgi lab member Tatiana Karakasheva sheds light on the mechanisms underlying this complex contribution of immune cells to the promotion of tumor growth.

In the division of Gastroenterology, the lab of Dr. Anil Rustgi focuses on understanding the development and progression of human gastrointestinal

(GI) cancers. Previous studies have generated an interest in cancer immunology as the lab identified a striking expansion of tumor-promoting immune cells in upper GI cancers through creation of a novel genetic mouse model of esophageal squamous cell cancer (ESCC). These mice contain a GI-specific deletion of the cell junction protein p120-catenin (*p120*^{-/-}) and develop severe inflammation and esophageal tumors as a consequence of this mutation. Tatiana began studying the role of these remarkable tumor-promoting cells deemed myeloid-derived suppressor cells (MDSCs), in esophageal cancer. Interestingly, MDSCs are the only splenic immune population expanded in these tumor-bearing *p120*^{-/-} ESCC mice. Importantly, as their name hints, MDSCs have immunosuppressive capabilities and allow for the escape of tumor cells from immune surveillance. However, what drives expansion of MDSCs from normal

immature myeloid cells was previously undetermined.

In her recent publication, Tatiana first took a transcriptomics approach to identify candidate genes that contribute to the unknown tumor-promoting abilities of MDSCs in the *p120*^{-/-} ESCC mice. Compared to age-matched littermate controls, MDSCs from cancerous mice had significantly higher expression of the cell surface receptor CD38, among other proteins. CD38 was a promising candidate, as it is generally involved in immune cell processes such as myeloid differentiation and lymphoid proliferation. Few reports also associate CD38 cell surface expres-



Tatiana Karakasheva, CBG

sion with some immunosuppressive cell types like regulatory T and B cells. The elevated level of CD38 expression in tumor-bearing mice also correlated with an expansion of MDSCs compared to non-diseased animals, suggesting that CD38 may influence the increase in MDSC cell number.

Tatiana and colleagues further characterized MDSCs by CD38 expression (CD38^{high} and CD38^{low}) and hypothesized that the CD38^{high} cells would have the greatest potential to be immunosuppressive, given that CD38 is overexpressed in the suppressive MDSCs compared to normal myeloid precursor cells. Indeed, the CD38^{high} MDSCs suppressed T cell proliferation *in vitro* and increased tumor volume *in vivo* compared to their CD38^{low} counterparts. Additional transcriptomics experiments demonstrated that CD38^{high} and CD38^{low} MDSCs have different gene expression profiles, as around 500 genes are differentially expressed. These include genes important for immunosuppression, such as iNOS, and NF-κB activation, as well as genes required for the enzymatic activity of CD38. What then increases the expression of CD38 in MDSCs? To address this, Tatiana performed *ex vivo* cytokine differentiation arrays and determined that a mixture of tumor-derived pro-inflammatory

cytokines including IFN γ , TNF α , CXCL16, IL-6, and IGFBP3 increase the expression of CD38 in cultured MDSCs from tumor-bearing mice. Importantly, CXCL16, IL-6, and IGFBP3 were identified in this study for the first time as novel regulators of CD38 expression.

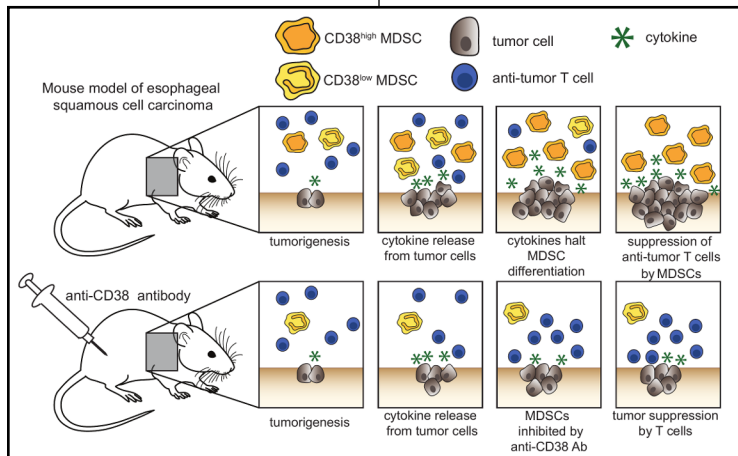
Because MDSCs with high levels of CD38 expression are immunosuppressive and have greater tumor promoting capacities than CD38^{low} MDSCs, the authors hypothesized that targeting CD38 may impair MDSC function, thus, reducing tumor load. To test this, Tatiana and colleagues cross-linked CD38 with monoclonal antibodies and observed that MDSCs from tumor-bearing mice had significantly impaired expansion and survival rates. Additionally, anti-CD38 antibody treatment decreased *in vivo* tumor growth. While most of these studies were performed in mouse models, peripheral blood from healthy donors and advanced-stage cancer patients was also analyzed for MDSC content. Intriguingly MDSC levels are elevated in cancer patients

compared to healthy donors, further demonstrating the potential for targeting CD38 and MDSCs as a new cancer therapy.

Tatiana's work presents a novel understanding of how immune cell subsets contribute to tumorigenesis. In this newly described mechanism, cancer progression and tumor growth cause pro-inflammatory signaling that induces the expansion of MDSC numbers and expression of immunosuppressive CD38. In the Rustgi lab, Tatiana is continuing her investigation of CD38 in MDSCs as a therapeutic strategy for human cancers. She says, "We are developing a genetic mouse model for suicide gene-based specific depletion of granulocytic or monocytic MDSCs. The work is still in its infancy, but if successful, we would be able to decipher

how the two subpopulations promote tumorigenesis." The ultimate goal of this work is to develop the use of CD38 in human clinical trials targeting solid tumors.

Karakasheva, T. A., Waldron, T. J., Eruslanov, E., Kim, S., Lee, J., O'Brien, S., Hicks, P. D., Basu, D., Singhal, S., Malavasi, F., and Rustgi, A. K. CD38-expressing myeloid-derived suppressor cells promote tumor growth in a murine model of esophageal cancer. *Cancer Research*, 2015; 75(19).



Mouse models of esophageal squamous cell carcinoma (ESCC) revealed that cytokine release from tumor cells halts the differentiation of myeloid-derived suppressor cells (MDSCs), resulting in increased levels of CD38^{high} cells. These cells suppress anti-tumor T cells, which results in uninhibited tumorigenesis. Treatment with an anti-CD38 antibody could limit the presence of CD38^{high} MDSCs, resulting in tumor suppression.

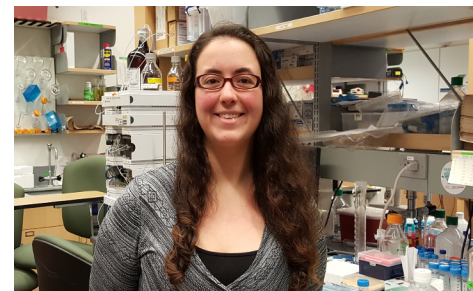
Don't read a mesenchymal stem cell by its gene expression levels

Hayley Hanby

One of the first successful tissue replacement therapies was artificial skin, developed by Drs. Ioannis Yannas and John Burke in 1979. Since then, tissue engineering has blossomed into a multidisciplinary field of biomaterials science, cell biology, and translational medicine. Advances in stem cell research, in particular, have greatly progressed the field of tissue engineering and regenerative medicine. Scientists have developed effective differentiation protocols to convert pluripotent or multipotent stem cells to their favorite cell type, but selecting superior subpopulations of lineage-committed cells for optimized performance of the engineered tissue is still an ongoing field of research. The lab of Arjun Raj takes a systems approach to biology in their work to quantify cellular functions. Allison Cote, a fourth-year CPM student in the lab, along with Claire Macleod of the Mauck Lab—a lab with expertise in cartilage differentiation—spearheaded a study that will force those in the stem cell field to think twice about correlating marker gene expression with phenotype.

Allison and colleagues utilized mesenchymal stem cells (MSCs), which can

undergo either osteogenic, adipogenic, or chondrogenic differentiation, to ask whether chondrogenic marker expression could predict chondrocyte potential. Chondrogenic differentiation is typically characterized by proteoglycan synthesis and matrix accumulation, along with a general increase in *aggrecan* gene expression over the first seven days. Single-molecule RNA fluorescence *in situ* hybridization (FISH) was used to visualize individual mRNA molecules within an MSC. The researchers observed that over an entire cell population, the canonical pattern of *aggrecan* expression held true. However, individual MSCs showed significant cell-to-cell variability in *aggrecan* copy number during chondrogenic differentiation. Given the inherent



Allison Cote, CPM

heterogeneity between MSCs, Allison and colleagues hypothesized that higher expression of *aggrecan* would yield chondrogenic cells more robustly. Interestingly, increased *aggrecan* mRNA was not predictive of high-performing cells—as indicated by aggrecan core protein, a central component of the extracellular matrix (low-performing cells lacked extracellular staining for aggrecan core protein). Even more remarkably, twelve hours after cell division, sister MSC cells showed vastly divergent levels of *aggrecan* and *GAPDH* expression, suggesting that the heterogeneity in marker expression is not heritable or, at least, not maintained when propagated. This is quite a surprising finding given the traditional notion of marker gene expression, but those in the field of single-cell biology would argue this is not unusual, long knowing that cellular heterogeneity is present in prokaryotes and eukaryotes.

Since expression of *aggrecan* ineffectually sorted MSC populations, high-throughput RNA sequencing was employed to find genes that are more predictive of cell fate. Strikingly, the analysis demonstrated no patterns of gene expression that correlated with matrix accumulation. The most predictive genes, *MMP13* and *aggrecan*, were only loosely correlative.

How can analysis of *aggrecan* on a population, but not on a cell-to-cell basis, show a trend in expression? The answer lies in normalizing *aggrecan* copy number to housekeeping genes such as *GAPDH*. Previous studies in the Raj lab have demonstrated that expression of these housekeeping genes correlates with cell size, and that the chondrocyte spread – cell area and volume – increased with passage number. In examining gene expression of de-differentiating chondrocytes, Allison and colleagues noted higher *GAPDH* levels in these cells and only minor changes in *aggrecan* copy number, suggesting that the process of de-differentiation was primarily due to increased cell size. As has been reported recently, transcription is a stochastic process; this paper's findings bolster that idea and suggest that

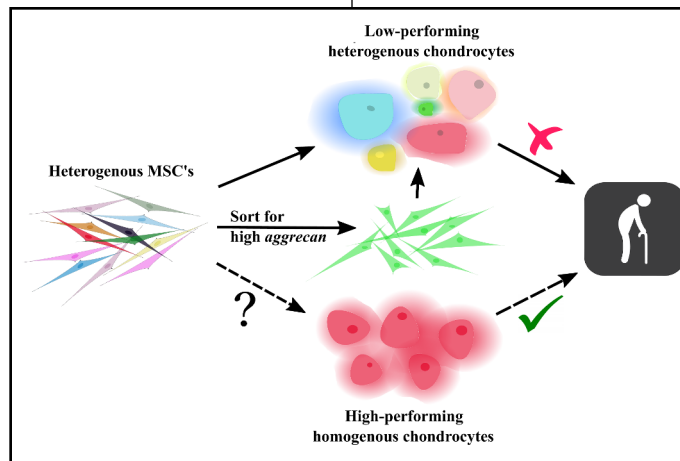
marker copy number fluctuates rapidly over a short timescale.

If gene expression profiles cannot predict chondrogenic differentiation, then what can? Other regulatory mechanisms, such as post-translational modifications, most likely dictate a chondrocyte's functional capacity – not every cell that translates aggrecan core protein will appropriately modify the protein for secretion. And once aggrecan is secreted, it associates with the extracellular matrix (through interactions with hyaluronic acid and collagen, among other molecules) so if there are abnormalities in that network, matrix accumulation may be inhibited. Allison remarks, “I think our paper highlights that successful differentiation of a set of stem cells really requires a functional readout, and gene expression of what would normally be considered ‘marker’ genes is often insufficient because successful differentiation requires the whole intracellular system to be in a differentiated state. I think it also highlights intrapopulation cell heterogeneity, a characteristic that is often explored in the fields of single-cell and stem cell biology, but less so in tissue engineering, particularly with mesenchymal stem cells. I think cell-to-cell variability is something that has caused difficulty in defining an

optimal differentiation routine for the massive quantities of cells needed for therapeutic applications.”

Currently, Allison is working on using expansion microscopy in combination with single molecule RNA FISH to examine the structure of RNA at regions where it is densely packed within the cell, such as transcription sites and mitochondria.

Cote, A.J., McLeod, C.M., Farrell, M.J., McClanahan, P.D., Dunagin, M.C., Raj, A., and Mauck, R.L. Single-cell differences in matrix gene expression do not predict matrix deposition. *Nature Communications*, 2016; 7.



An ongoing challenge in the field of tissue engineering is the use of MSC-derived chondrocytes to treat damaged cartilage in diseases such as osteoarthritis. MSCs cultured in vitro exist as a heterogeneous population, as defined by their gene and protein expression levels. These heterogeneous populations of MSCs differentiate to produce a heterogeneous population of chondrocytes that are low-performing and therefore not suitable for cell replacement therapies. Allison Cote et al. showed that obtaining a homogeneous population of MSCs by sorting for similar gene expression still resulted in a heterogeneous population of chondrocytes upon differentiation. The current challenge in the field is to generate a homogenous population of chondrocytes that can be suitable for therapeutic purposes.

SPECIAL INTEREST

The Penn approach to tackling Zika virus

Lindsey Weed

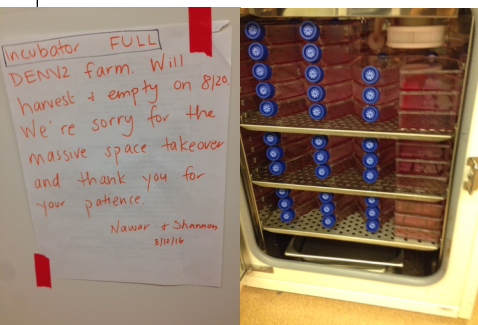
The first confirmed Zika virus (ZIKV) infections in Brazil were promulgated by the Pan American Health Organization in May 2015. By the start of 2016, the World Health Organization had declared the outbreak a Public Health Emergency of International Concern as evidence for a causal relationship between primary ZIKV infection during pregnancy and microcephaly amassed. Epidemiologic and pathogenic uncertainties hinder efforts to treat ZIKV. If a pregnant woman is exposed to ZIKV, how likely is she to contract the virus? What are the chances ZIKV will be transmitted to her fetus? Does sexual transmission of ZIKV pose a different risk of birth defects than mosquito-borne transmission? Fortunately, numerous labs at Penn are well poised to search for answers to some of these outstanding questions.

One such lab is that of Dr. Sara Cherry. Her group studies several mosquito-borne RNA viruses to identify cellular factors that impact infection as novel targets for therapeutics. ZIKV belongs to the Flaviviridae viral family along with dengue and West Nile virus, both of which Cherry's lab researches. “When it became clear that ZIKV was running rampant through South and Central America, we decided to include it in the experiments we were al-

ready doing. We had all the reagents to study it because we'd been studying very related viruses,” said Cherry. Additionally, her group screens small molecule libraries for their ability to block ZIKV infection in relevant cell types, including placental trophoblasts and blood- brain barrier endothelial cells. These compounds are FDA approved and could be rapidly repurposed to treat ZIKV. While these drugs may be useful to prevent Guillain-Barré syndrome, a nervous system disorder that can cause paralysis, and sexual transmission of ZIKV, it is unlikely that any of these drugs would be given to the truly negatively affected demographic: pregnant women. “ZIKV is a women's health issue as much as it is a global health issue,” mused Cherry. “The current recommendation for women of child-bearing age is to not get pregnant. Unfortunately, there are many women worldwide that aren't afforded the luxury of choosing when they become pregnant, especially in the countries affected by this current outbreak.”

If pregnant women were to be treated, it would most likely be with neutralizing antibodies. However, the similarity between ZIKV and dengue virus (DENV) heightens the risk of antibody-dependent enhancement (ADE).

(The extent of the similarity between the two viruses is reflected by the humoral immune system, where monoclonal antibodies (mAbs) generated from DENV-infected subjects exhibit cross-reactivity with ZIKV. The mAbs fail to neutralize the virus, instead potentially enhancing its infectivity. Observing ADE of ZIKV infection is worrisome because the mechanism is preferentially associated with the development of severe dengue hemorrhagic fever. Cross-reactive, non-neutralizing antibodies generated for one of four DENV serotypes facilitate viral uptake by macrophages and other Fc-receptor bearing cells upon secondary infection with an another serotype, increasing viremia and pathogenicity.



Shannon Barbour and Nawar Naseer, MVP, commandeered an incubator to grow stocks of dengue virus for experiments.

Rising second year Shannon Barbour screens hybridomas for mAbs that can differentiate between ZIKV, DENV, and other arboviruses. The results of her research could identify antibodies that can be safely administered to pregnant women infected with ZIKV without risk of cross-reactivity with DENV. Such antibodies could additionally be used to rapidly test pathogen

exposure and risk of severe disease development. Conversely, rotation student Nawar Naseer focuses on determining whether the repertoire of antibodies obtained upon ZIKV infection alone is different from that obtained with DENV pre-exposure, which would help identify antibodies that can neutralize both DENV and ZIKV.

This past June marked an exciting time for Dr. Dave Weiner when the FDA approved the first human clinical trials for a ZIKV vaccine developed in part by his lab. The speed at which the DNA vaccine was developed is remarkable considering Weiner and his collaborators – Inovio Pharmaceuticals, GeneOne Life Science, and Dr. Gary Kobinger, the head of Special Pathogens at the Public Health Agency of Canada – only began their work last fall. “There weren’t any reagents or models when we started working on ZIKV,” recalled Weiner. “You learn a lot by focusing on new things because you’re forced to stretch your imagination. You have to apply things that you know about other pathogens, which is a great exercise for students and postdocs.” Although the nature of a protective immune response against ZIKV is unknown, both Weiner’s group and the Vaccine Center at the Wistar Institute are well experienced with indicators of consistent levels of immunity. Beginning with Dr. Stanley Plotkin’s rubella vaccine, the Wistar Institute has been developing life-saving vaccines for over half a century and Weiner’s novel synthetic DNA platform serves to advance this prestige. The trial participants are scheduled to receive the ZIKV vaccine within the next few weeks.

Penn fosters an environment conducive for cutting-edge research. This is particularly important when studying emerging infectious diseases like ZIKV, where time is a precious resource.

Teaching experience at Penn

Kelsey Speer

When I came to graduate school, I was excited to learn about both scientific research and science teaching. I specifically chose my thesis lab knowing that my mentors would support me in this pursuit. During my first three years of graduate school, I was a teaching assistant (TA) for a graduate-level survey course and participated in outreach programs. But then I felt stuck – there was so much more to being a successful instructor I felt I needed learn, but no clear way to continue to cultivate those skills.

If I’ve learned one thing in graduate school, it is this: whether you are perfecting a new technique in the laboratory or trying to figure out how to design your own course, it is always best to seek out expert help. Luckily, Penn is full of unbelievable resources of expert knowledge, particularly with regards to teaching. The first expert source I encountered was an extremely thoughtful professor who was very open about the difficulties he faced as a teacher. While I was a TA for his course, he taught me that being a good science teacher is similar to being a good experimentalist. A good science teacher is constantly thinking about, testing, and collecting feedback on new teaching methods to improve their courses. And, like bench work, failure is a very natural part of this process.

Another great expert resource I encountered was Penn’s Center for Teaching and Learning (CTL, <http://www.upenn.edu/ctl/>). Many members of its staff hold science Ph.D.s, and they provide excellent, FREE programs for all graduate students at Penn. Through CTL, you can earn a Teaching Certificate on your transcript, or take one of their awesome mini courses on teaching (yes, they exist). Even if you don’t think you are particularly interested in teaching, many of the skills discussed in these courses can be translated to other professions. For example, the experience of learning to organize a presentation “like a teacher” dramatically improved my own scientific presentations.

If you find yourself absolutely loving the experience of helping students

reach that “ah-ha” moment in their learning, CTL also offers a fantastic program for graduate students who are committed to a career in teaching. Each graduate group or department may nominate one student to apply for the Graduate Fellowship for Teaching Excellence. During the 2015-16 academic year, I had the privilege of representing CAMB as a CTL Fellow. As part of the program, I met regularly with other Fellows from departments across Penn’s campus (Education, History, English, Engineering, etc.), many of whom regularly taught entire courses during graduate school. The experience of discussing teaching topics with these “expert peers” helped me anticipate many of the obstacles I will face when teaching my first course.



Kelsey Speer, CPM

As a Fellow, I had the opportunity to design and implement my own “lessons” in a manner that went well beyond my experiences as a TA. I organized seven teaching workshops for BGS students during the 2015-16 academic year (many thanks to those of you who participated!). I also led one university-wide workshop each semester. During the planning of these workshops, I was forced to decide which topics were most important to my audience, what I wanted my audience to learn about these topics, and what activities I would use to help them successfully master this new knowledge. Having the opportunity to practice these skills before feeling the pressure of a real classroom situation was invaluable.

Penn is a community of experts. I encourage you take advantage of the opportunities available here, no matter what your passion is! If, like me, teaching is your passion, seek out mentors who think critically about their teaching methods. Ask them questions. Be their TA. Volunteer to give a guest lecture in one of their classes (particularly for undergraduate courses). In my experience, teaching is something that takes a lot of practice to get it right, so graduate school is a fantastic time to start!

The 2016-2017 CAMB CTL Fellow is Rebecca Rivard. Thanks to Rebecca, CAMB will have teaching-related workshops held throughout the coming academic year!

Renske Erion

Iryna Shakhmantsir

Crafting a non-academic career path for a typical CAMB graduate student can be a complex balancing act of juggling never-ending hours of lab work, hobbies, and a personal life. Meeting recent CAMB alumni who have successfully navigated the real-life labyrinths to secure non-academic jobs can thus be refreshing and inspiring. Renske Erion is a recent alumna of the Cancer Biology subgroup who completed her Ph.D. in Amita Sehgal's lab, where she studied mechanisms of circadian rhythm regulation. She is currently a project director for the Emerging Leaders in Science and Society (ELISS) Program in Seattle, Washington.

The ELISS program is a service-oriented experiential-learning organization that allows graduate students from the selected partner campuses to tackle real-world problems, Renske enthusiastically explains. For instance, 2016 ELISS fellows are responsible for devising better solutions for the future of safe, sustainable, and affordable drinking water in the United States. The ELISS fellows participate in a dialogue between the stakeholders within the community and the scientists, and then use scientific data to inform the policy. Importantly, by attracting Ph.D. students from diverse disciplines, from health law and educational policy to mathematics and mechanical engineering, the ELISS program aims to create a powerful workforce that is not myopically attached to any narrow field of study, but can instead successfully operate in a larger societal context.

As the program director, Renske coordinates the daily operations of the ELISS program and strategizes future developments of this educational endeavor. "This entails communications with the public about the ELISS program as well as internal communications with our fellows and advisors to keep the operations running smoothly," Renske elaborates. She produces the written and graphic content for the media, official program reports, and grant proposals. One can learn more about the ELISS program by visiting the website that Renske designed. Though she never considered herself much of a creative person, this recent venture into the sphere of web design has uncovered her previously dormant artistic skills and has been highly rewarding. As part of a small team, Renske has plenty of room to put herself

into positions of responsibility and create visible impact. Renske's experiences speak to the potential of "alternative" careers not only to promote professional development, but also to foster continuous personal growth.

As early as her third year in graduate school, Renske began exploring a range of career options, gravitating towards science policy, STEM education, and not-for-profit career opportunities. Reflecting on the experiences that paved the way for landing her first job, Renske underscores the value of volunteering. Only with luck can you stumble upon an employer who might be willing to hire you without all the necessary qualifications. That same employer, however, might be more open to placing you into a temporary volunteering position. While such a volunteering gig might not add to your bank account balance, it can certainly boost your résumé and convince your dream employer of your motivation to pursue the career path of your choice.

Renske strongly encourages all CAMB students to take initiative to step outside of the lab and try new things, join professional communities, and seek networking opportunities. Renske has found that contacting people via LinkedIn to get advice on transitioning out of academia can be extremely effective. "If you don't ask, they are not going to come find you," she rightfully points out. The process of scientific discovery is central to the goals of STEM graduate education. The idea of *self-discovery*, on the other hand, is still a bit far-fetched within the academic circles. Renske has very simple yet powerful advice: "Whatever you want to do, it should be something that provides the most meaning to you."



Renske Erion, CBG

Jessica Bryant

Siddharth Kishore

There is a noticeable trend in recent times of science graduates pursuing alternative careers in science, to an extent where one might question ascribing the term "alternative" to these career paths. It was thus a rejuvenating experience to chat with Jessica Bryant, who followed her love for scientific research by pursuing the "traditional" career path of a post-doctoral researcher in academia. Jessica was a former GGR student in Dr. Shelley Berger's lab. She graduated with her Ph.D. in 2014, after which she began a post-doctoral fellowship at the Pasteur Institute in Paris, France.

Jessica has harbored dreams of having her own lab ever since she worked as an undergraduate researcher at the University of Georgia. During her first year at Penn, she was inspired by a talk given by Dr. Shelley Berger to enter the field of epigenetics. She went on to join Dr. Berger's lab and studied the epigenetic mechanisms governing gametogenesis in

yeast and mice. Looking back at her years as a Ph.D. student, she says, "While the challenges and stresses involved in grad school can weigh you down, at the end of six years I still held on to my dreams of having my own lab." This led her to an academic post-doctoral position, a decision she recommends to scientists who still have a passion for science at the end of their graduate studies. She does acknowledge the element of luck involved in getting a faculty position, where the supply of life science graduates is greater than the demand. "It is always important to keep your options open and think about contingencies," she explains. She highlights the importance of the various career talks organized by the CAMB program in keeping her informed about other career options, as she now notices the dearth of these resources at the Pasteur Institute.

As a post-doc at the Pasteur Institute, Jessica researches the epigenetic mechanisms that underlie the transcriptional control of virulence gene expression in different species of *Plasmodia*, the parasites that cause malaria. While the quality of the science was an important factor, she does not deny the fact that having the opportunity to experience a different culture played an important role in her decision to move to France. Although she had studied French in college, her French was rusty at best after six years of graduate school. "The first six months were difficult," she says, "especially while



Jessica Bryant, GGR

dealing with the bureaucracy involved in getting the necessary paper work and being at a considerable distance from friends and family.” However, the initial transition period gave way to a plethora of great experiences, both professionally and socially. Her lab, like many others at big institutes, is fairly international and this gives her a chance to communicate in English as well as in French. She cherishes this multicultural environment, both inside and outside of the lab. “The work culture is very different as compared to working in science in the U.S.” she explains. “You are allowed two months of vacation a year and while you may not publish three or four *Nature* papers a year, it’s good to slow down a bit and take a breather while, nonetheless, staying productive and motivated.” Maybe it is this change of pace and work culture that have helped her maintain her love and passion for science.

Reflecting on her time at Penn, Jessica highlights the time she spent as a teaching assistant for an epigenetics course, one of her favorite learning experiences at Penn. “If there’s one thing CAMB could change, it would be to

encourage students who show a genuine interest in teaching, to pursue more [teaching assistant] opportunities, and work with PIs and professors to make these positions more accessible,” she asserts.

Jessica concludes our conversation with some advice for current graduate students. “Learn bioinformatics!” she says emphatically. “In the current world of scientific research, most questions involve the use of sequencing and having a basic knowledge of bioinformatics will benefit you and shield you from relying heavily on bioinformaticians throughout your career.” Jessica’s story provides hope and encouragement to current graduate students that are struggling with the stresses of graduate school and questioning their love for science. Sometimes, all it takes is a change of scenery and work culture to reinvigorate your passion.

To learn about funding opportunities in France and more about Jessica’s experience, visit the CAMB Student Newsletter blog at <http://cambnewsletter.wix.com/blog>.

CAMB ORIENTATION SCHEDULE

Scavenger Hunt: Thursday September 1st, 4:00pm-5:30pm: Penn Campus

Harvest Happy Hour: Thursday September 1st, 6:00pm-8:00pm: Harvest Seasonal Grill

Welcome BBQ: Friday September 9th, 4:00pm-6:00pm: Biopond

WELCOMING NEW-CAMB-ERS:

Incoming First Year Ph.D. Students

CBG

Jonuelle Acosta
Jason Godfrey
Grant Grothusen
Sierra McDonald
Joshua Parris
Laura Ritenour
Osvaldo Rivera
Clarissa Rous
Tiffany Tsang

CPM

Olivia Farrelly
Olivia Harding
Daneille Minichino
Jessica Phan
Shahadat Reza
Katelyn Sweeney
Jaye Weinert

DB

Amaris Castanon
Elizabeth Howell
Derek Liberti
Mailyn Nishiguchi
Christopher Pai
Alexander Salomon
Isabel Sierra
Kelly Sullivan
Aoi Wakabayashi

GGR

Desiree Alexander
Zhendong Cao
Ayano Kondo
Jennifer Luppino
Alexis Oguh
Ana Petracovici
Stephanie Sansbury
Tammy Ying

GTV

Sangya Agarwal
Maxwell Chappell
McKensie Collins
Richard Martino
Erin Reagan
Lucas Van Gorder

MVP

Nathan Krump
Natasha Lopes Fischer
Tomaz Manzoni
Nawar Naseer
Prioty Sarwar
Elisha Segrist

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