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EDITOR’S NOTE

Well, to be honest

By Comfort Dorn

A couple of decades ago, I used to walk three miles every morning with two friends. The goal was fitness, but it was also social time. Some days we woke the neighbors with our hoots of laughter. But when my long-disintegrating marriage really started to fall apart, I got quiet and sad. Of course, my friends noticed. One cold dark morning we talked about it, and one of my friends told me that I should pretend to be happy. After a while, she reasoned, I’d start to believe myself and really be happy; in the meantime, other people wouldn’t feel uncomfortable around me. I tried to follow her advice, but I only felt worse. Not only was I failing at holding my family together, I couldn’t even fool myself into being cheerful.

With hindsight, I can now say that was awful advice. Pretending doesn’t solve much.

I thought about this when I first read several of the personal essays in this issue’s wellness section — what struck me most was their honesty. Before these writers figured out how to take care of themselves, they had to face up to what ailed them — to the ways they were broken. As they walked me (metaphorically) through their experiences of stress and pain, I realized that all of us need to see and be ourselves truthfully before we can find a way to heal and be well. I am grateful for these difficult shared journeys. I hope you will be too.

On a completely different note: It’s a new year. Sometimes that means changes, sometimes not. Here at the American Society for Biochemistry and Molecular Biology (and specifically at this magazine), 2020 means big changes to our online presence. We have a handsome new website, and ASBMB Today is moving to daily publishing.

What does that mean? Instead of throwing an entire issue of the magazine onto the website early in the month, we will post new articles and essays every day. We’re playing around with themes like “Member Monday” and “Fats Tuesday” (the “s” is not a typo — that’s the day for lipids). The magazine site will grow to include our public affairs department’s policy blog, weekly careers columns and monthly health observances. As a big bonus, the site also will be mobile friendly, so you can read ASBMB Today everywhere you go.

Be well — and may you thrive in the year to come.

Correction

A photo of carpenter ants on page 36 of the December issue was credited incorrectly. It was taken by Riley Graham.
Member update

ASBMB members elected as 2019 AAAS fellows

The American Association for the Advancement of Science has elected 441 of its members as fellows in recognition of their extraordinary achievements in advancing science. Of those honored, the 12 listed below are members of the American Society for Biochemistry and Molecular Biology. The fellows will be recognized at the 2020 AAAS annual meeting in Seattle in February.

Section on Agriculture, Food and Renewable Resources

Thomas Leustek, professor, department of plant biology, Rutgers State University of New Jersey

Section on Biological Sciences

Vernon B. Carruthers, professor of microbiology and immunology, University of Michigan Medical School

Julian J.-L. Chen, professor of biochemistry in the school of molecular sciences, Arizona State University

Caryn Elizabeth Outten, Guy F. Lipscomb professor of chemistry, department of chemistry and biochemistry, University of South Carolina

Nicola Partridge, professor, department of basic science and craniofacial biology and departments of biochemistry and molecular pharmacology and medicine, New York University

Section on Chemistry

Emily E. Scott, professor of medicinal chemistry and pharmacology, University of Michigan

Aleem Siddiqui, professor of medicine, University of California, San Diego, School of Health Sciences

Section on Dentistry and Oral Health Sciences

Kelly G. Ten Hagen, senior investigator, National Institute of Dental and Craniofacial Research, National Institutes of Health

Section on Medical Sciences

Nicholas O. Davidson, John E. and Adaline Simon professor of medicine and of developmental biology, Washington University School of Medicine in St. Louis and editor-in-chief of the Journal of Lipid Research.

Marsha A. Moses, Julia Dyckman Andrus professor at Harvard Medical School and director of the vascular biology program at Boston Children’s Hospital

Section on Pharmaceutical Sciences

Nicholas K. Tonks, Caryl Boies professor of cancer research, Cold Spring Harbor Laboratory
Nine members of the American Society for Biochemistry and Molecular Biology have been named to the inaugural class of fellows of the American Society for Pharmacology and Experimental Therapeutics. ASPET is a scientific society whose 5,000 members conduct basic and clinical pharmacological research and work for academia, government, large pharmaceutical companies, small biotech companies and nonprofit organizations. The fellows were selected for their efforts to advance pharmacology through their scientific achievements, mentorship and service to ASPET.

**William A. Catterall** is a professor and former chair of pharmacology at the University of Washington. He studies the voltage-gated sodium and calcium channel proteins, which initiate electrical and chemical signaling in excitable cells.

**Michael Gottesman** has been deputy director for intramural research at the National Institutes of Health since 1993. His laboratory identified the human gene that causes cancer cells to resist many anticancer drugs by pumping these drugs out of drug-resistant human cancers.

**F. Peter Guengerich** is the Tadashi Inagami professor of biochemistry at Vanderbilt University School of Medicine and a deputy editor of the Journal of Biological Chemistry. He studies the chemical and biological mechanisms by which molecules are processed.

**James Halpert** is a professor of pharmacology and toxicology and former dean of pharmacy at the University of Connecticut. He studies the structure and function of cytochromes P450 of the 2B and 3A subfamilies.

**Paul F. Hollenberg** is a professor emeritus of pharmacology at the University of Michigan Medical School. His research is related to the mechanisms of action of the cytochromes P450, mechanism-based inactivators, and the role of the human P450 isozymes in the metabolism of drugs, chemical carcinogens and other toxic agents.

**Paul A. Insel** is a distinguished professor of pharmacology and medicine at the University of California, San Diego, and co-director of the UCSD Medical Scientist Training Program. His research efforts focus on G protein–coupled receptors, heterotrimeric G proteins and G protein–regulated effectors.

**John S. Lazo** is a professor of pharmacology at the University of Virginia and associate director for basic research at the UVA Cancer Center. He studies the mechanism of action of small molecules and the fundamental biological role of protein tyrosine phosphatases.

**Robert J. Lefkowitz** is James B. Duke professor of medicine and professor of biochemistry and chemistry at the Duke University Medical Center and a Howard Hughes Medical Institute investigator. His recent research applies tools of structural biology to understand biased signaling at atomic-level resolution.

**Richard Neubig** is the chair of pharmacology and toxicology at Michigan State University. He has studied GPCR signaling for most of his career and currently explores G proteins in genetic epilepsies as well as academic drug discovery, particularly related to cancer, scleroderma and other fibrotic diseases.
Wand heads department at Texas A&M

Josh Wand has just completed his first semester as head of the department of biochemistry and biophysics at the College of Agriculture and Life Sciences at Texas A&M University. He was named to the position effective Aug. 1.

Wand was previously the Benjamin Rush professor of biochemistry and biophysics at the Perelman School of Medicine at the University of Pennsylvania. In addition to his department head duties at Texas A&M, Wand is teaching third-year biochemistry to undergraduates and a graduate course in biophysics.

His research looks at the intricacies of how to manipulate protein recognition of other proteins. Over the years, he has focused on the influence of the physical properties of proteins and their function, with an emphasis on how the internal structure of molecules fluctuates over time.

Wand is a fellow of the American Physical Society and the Biophysical Society. He earned his bachelor’s degree in biochemistry and master’s degree in chemistry from Carleton University in Ontario, Canada, and his doctoral degree in biophysics from the University of Pennsylvania.

Bermuda Principles to honor Steitz

Joan Steitz will be honored at the annual Bermuda Principles Impact Conference in February.

Steitz is a professor of molecular biophysics and biochemistry at the Yale School of Medicine. Her lab focuses on noncoding RNA–protein complexes, which are ubiquitous in eukaryotic cells.

In 2018, Steitz received the Lasker–Koshland Special Achievement Award in Medical Science from the Albert and Mary Lasker Foundation. She won the American Society for Biochemistry and Molecular Biology’s Herbert Tabor Research Award in 2015. Steitz is known as a generous mentor to young scientists and an ardent voice for inclusion in the scientific community.

The Bermuda Principles were drafted in 1996 to ensure all genetic data generated by experiment were put into the public domain within 24 hours and available to all for the advancement of science. The conference celebrates the effect of these principles on the pace and depth of scientific research. This year’s theme is transcriptomics. Steitz will deliver the ncRNAs keynote talk.

Lemons receives presidential award

Paula Lemons, an associate professor at the University of Georgia, was a 2019 recipient of the National Science Foundation’s Presidential Early Career Award for Scientists and Engineers.

The U.S. government’s highest honor for scientists and engineers in the early stages of their research careers who show exceptional leadership promise, the award acknowledges contributions to the advancement of science, technology, education and mathematics education and to community service as demonstrated through scientific leadership, public education and community outreach.

Lemons, who teaches undergraduates in the department of biochemistry and molecular biology, serves as director of the Scientists Engaged in Educational Research Center and as principal investigator of the NSF-sponsored Department and Leadership Teams for Action project.

Lemons studies how to support college biology instructors who use reformed teaching strategies shown to improve student outcomes. She also studies problem solving among undergraduate biology and biochemistry students. She has created a guide to writing good biology problems for teachers and an online problem-solving tutorial for students.

Education association honors Osheroff

Neil Osheroff received the International Association of Medical Science Educators’ 2019 Distinguished Career Award for Excellence in Teaching and Educational Scholarship, which recognizes an IAMSE member with a distinguished record of more than 10 years of educational scholarship, including educational research and dissemination of scholarly approaches to teaching and education.

Osheroff is a professor of biochemistry and medicine at the Vanderbilt University School of Medicine, where he holds the John G. Coniglio chair in biochemistry. He has been a course director since 1990, directs the School of Medicine Academy for Excellence in Education and chairs the master science teacher group. His lab specializes in the function and biology of DNA topoisomerase II, a ubiquitous enzyme that modulates the topological structure.
of DNA and plays a role in chromosome structure.

Osheroff earned his Ph.D. in biochemistry and molecular biology from Northwestern University and was a Helen Hay Whitney Foundation fellow at the Stanford University School of Medicine. He is a past president of the Association of Biochemistry Educators and now serves as the treasurer of the International Association of Medical Science Educators. He is a fellow of the American Association for the Advancement of Science.

IN MEMORIAM

Michael Rossmann

Purdue University structural biologist Michael G. Rossmann died May 14 at the age of 88.

Rossmann was born in Frankfurt, Germany, in 1930 and immigrated to England in 1939. There, he studied physics and mathematics at the University of London before receiving his Ph.D. in chemical crystallography from the University of Glasgow in 1956. After a postdoctoral fellowship at the University of Minnesota and a stint as a research assistant at the University of Cambridge, he joined the faculty at Purdue University in West Lafayette, Indiana, in 1964, where he would remain for more than five decades.

In 1985, he was the first scientist to map the structure of the virus that causes the common cold on the atomic level with X-ray crystallography. In subsequent decades, Rossmann continued to turn the towering microscopes he helped bring to Purdue on a variety of viruses, including Dengue virus, Zika virus and the giant Mimivirus.

A member of the National Academy of Sciences and the Royal Society of London, Rossmann continued his research career into his final years. He is survived by his three children, Martin, Alice and Heather, and four grandchildren; after his wife Audrey Pearson’s death in 2009, he remarried in 2018 and is survived by his second wife, Karen Bogan.

Salih Wakil

Salih Wakil, chairman emeritus of biochemistry at Baylor College of Medicine, died July 11 in Houston. He was 91.

Wakil’s fatty acid synthesis studies were very influential; his team discovered enzymes critical to metabolic pathways that govern the body’s ability to burn fat.

Born in Karbala, Iraq, in 1927 as the son of a shoemaker, Wakil won a scholarship to the American University of Beirut, where he earned a bachelor’s in chemistry in 1948. He completed his Ph.D. in biochemistry at the University of Washington.

Wakil was a research associate at the University of Wisconsin and a professor at Duke University before becoming chairman of Baylor’s biochemistry department in 1971. He held the position for 35 years. In 2010, he founded the discovery-stage biotechnology company FGH BioTech Inc.

His many honors include election to the National Academy of Sciences and the American Association for the Advancement of Sciences.

He is survived by his wife of 66 years, their four children and seven grandchildren.
Lina Obeid (1955 – 2019)

By Besim Ogretmen

Lina Obeid, a professor of medicine, dean of research and vice dean for scientific affairs at the Renaissance School of Medicine at Stony Brook University, died on Nov. 29. She was 64 years old.

Lina was a pioneer and leader in the sphingolipid field. She was also a personal and scientific mentor to many scientists, including myself. Lina opened her laboratory to many of us to teach us how to become independent investigators and behave as true researchers with open minds. She had great compassion and was a role model and mother figure to the whole field.

I have known Lina since 1999 when she and Yusuf Hannun, her husband, accepted me in their laboratories as a research assistant professor in the department of biochemistry and molecular biology at the Medical University of South Carolina. I was very lucky to have mentors like Lina and Yusuf, who gave me a chance to succeed in academia with great scientific and personal guidance. I could not have achieved anything that I have so far in my academic and research career without Lina's support, and she continued to help me at many levels, even just before she passed away. Her efforts to boost her mentees' careers and personal lives were exceptional, and this is one of the golden standards that we will carry forever in honor of Lina.

Lina's discovery that ceramide induces apoptosis was published in the journal Science in 1993. Since then, this field has exploded, and more than 4,000 manuscripts have been published in the ceramide-apoptosis-related research area (as reported in PubMed).

Lina was also a pioneer in the field of regulation of senescence by ceramide signaling, which provided the first clues for the link between lipid metabolism and aging. Her contributions to sphingosine 1-phosphate metabolism and signaling in inducing cancer growth and metastasis were instrumental in taking this field to the next level. I know first-hand that Lina was among the most brilliant scientists and made exceptional contributions to the field of bioactive lipids not only with strong science but also by providing novel and innovative ideas with pioneering discoveries.

Lina's research also opened doors for understanding the mechanisms of ceramide signaling for the regulation of cancer cell death, leading to clinical and preclinical trials. Her insights as a physician-scientist were critical for the development of many new ceramide-based drugs, such as cationic ceramides and acid ceramidase inhibitors, which are important regulators of inflammation and related diseases.

Her record of steady funding from various national sources for many years and her exceptional publication record also show that Lina was, without any doubt, a leader and pioneer in the field.

There are not enough words to describe how much we love Lina. Her contributions to the sphingolipid field are not only about exceptional research but also about her touching so many lives at a personal level with no expectations in return. She will be remembered for her bright personality and beautiful smile as she leaves behind a legacy that will last forever.

We miss Lina so much already. May she rest in peace.

Besim Ogretmen (ogretmen@musc.edu) is a professor and endowed chair of lipidomics and drug discovery, director of the Center of Biomedical Research Excellence in Lipidomics and Pathobiology and the Lipidomics Shared Resource, and program director of developmental cancer therapeutics at the Hollings Cancer Center at the Medical University of South Carolina.
What did you do last summer?
Undergraduate Research Award recipients describe their research

By Stephanie Paxson

It’s never too early to start thinking about summer plans. Each year, the American Society for Biochemistry and Molecular Biology awards up to 12 $1,000 grants to support summer research by ASBMB Student Chapter members.

We asked our 2019 Undergraduate Research Award recipients to summarize their summer research projects and what they plan to do in the future. Seven of the 11 2019 recipients shared their stories, which have been edited for length and clarity.

Virginia Blackwell
University of Texas at Dallas

In Sheena D’Arcy’s lab, I explored interactions between histone chaperone Nap1, the Taz2 domain of HAT p300 and linker histone H1. In particular, I investigated the possible formation of a ternary Taz2–Nap1–H1 complex.

My experience with full-time research taught me, more than anything, to have equal measures of patience and resilience. Some roadblocks take longer to resolve than others. Setting goals is a great exercise, but meeting goals is another matter entirely. Making progress toward those goals and resolving challenges along the way has been a satisfying experience and excellent opportunity for personal and professional growth.

I also had the good fortune to mentor a younger student while working on my project. This helped me learn that juggling priorities between bench work and other duties is a skill that takes practice to cultivate but is ultimately rewarding. I believe as much can be learned from sharing research with others as conducting the research itself. I am eager for more opportunities to mentor and educate in and out of lab.

I look forward to continuing with the characterization of members of the protein complex of interest. Continued work on this project includes identifying the key residues responsible for interactions between Nap1 and Taz2 and designing, cloning and purifying mutants to abolish binding.

Savannah Corradi
Bemidji State University

The goal of my research in Mark Wallert’s lab was to evaluate whether the combination of chemotherapeutic agent with an indirect NHE1 inhibitor could elicit a synergistic effect in ovarian cancer cells. This was previously shown through a reduction in proliferation rates. We did this work in a 3D environment, investigating the impact of these combination therapies in a spheroid assay for SKOV-3 and CAOV-3 cells. This new protocol provides a rapid and automated cell migration and cell invasion system in a highly reproducible standardized method. Combination therapies hold the potential to improve drug efficacy and patient experience. Specifically, this work could attenuate adverse side effects for her summer research project in Mark Wallert’s lab at Bemidji State University, Savannah Corradi tested a potential combination therapy in ovarian cancer cells.
Ryan Fink gained confidence in his techniques, especially handling RNA and performing next-generation sequencing, during his summer project in Martin Hicks’ lab at Monmouth University.

Danielle Jamison’s summer research project in Corina Maeder’s lab at Trinity University focused on the interaction between the splicing protein Dib1 and U5 snRNA.

associated with chemotherapy.

This project remains the highlight of my summer and academic career, and I will continue to work on it through my junior and senior years of college. I will broaden my focus to both the direct and indirect inhibitors of NHE1 and their pairing mechanisms in ovarian cancer. I will focus primarily on completing spheroid assay profiles and an intracellular pH assay and creating NHE1 knockout cell lines for both CAOV-3 and SKOV-3 cells.

My summer experience is helping me build my scientific reputation and will help me reach my goal of a Ph.D. in biomedical science. I am vice president of the Bemidji State University ASBMB Student Chapter. As a Student Chapter member, I can present my research nationally at the annual meeting and locally in my community. My goal is to become a professor and have my own research lab to continue studying the role of NHE1 in a cancerous environment.

Ryan Fink
Monmouth University

The project “Secondary structure analysis by SHAPE-MaP of the EGFR pre-mRNA transcripts: Uncovering novel regions for RNA anti-sense targeted therapy” allowed me to learn and master the fundamentals of molecular biology. I began with design and implementation of a gene therapy targeting EGFR followed by RNA transcription and modifying the RNA to reveal structural secrets about itself. Primarily, I gained confidence in my techniques, especially handling RNA and performing next-generation sequencing, which I now can carry into graduate school research labs. I believe that the ability to design an innovative project is an essential skill at the graduate level.

Through working with my mentor Martin Hicks and developing this project, my skills now include reading through literature to discover new and niche techniques and being able to read through a protocol and implement it in a lab setting. The future of this project is to continue developing another skill set through bioinformatics.

Going forward, I will continue to analyze data through a command-line program, ShapeMapper2. Follow-up experiments will include varying conditions to further validate the secondary structure. I hope this will lead to new insight for our gene therapy and produce figures in an upcoming paper.

Danielle Jamison
Trinity University

My research project focused on the interaction between the crucial splicing protein Dib1 and U5 snRNA. I made mutations in the conserved loop 1 of U5 snRNA at residues proposed to be the location of binding interactions in an attempt to disrupt this hypothesized interaction. This interaction and subsequent disruption could provide critical insight into the role of Dib1. Elucidating the role of this protein...
is a major goal for Corina Maeder’s lab; we believe Dib1 may be acting as a linchpin to prevent premature splicing.

My project has expanded to consider also the broader interaction of Dib1 with spliceosomal components including the U5 snRNA. Due to its location in the heart of the precatalytic spliceosome, Dib1 can interact with numerous partners. Dib1 exhibits an intriguing property called autocleavage, which refers to its ability to cleave the last 13 amino acids from its own C-terminal tail. Dib1’s autocleavage activity then may be a regulatory mechanism, allowing the tail to interact with its neighbors but then depart when necessary so the spliceosome can transition to be catalytically activated.

Through my experience, I learned many new techniques and different ways to approach the original hypothesis. There is still room for more work on my project, specifically creating the remaining mutations and creating the necessary haploid single knockout yeast strain. I intend to continue working toward obtaining the necessary yeast strain and the last mutations while simultaneously running autocleavage assays with new conditions. I am excited to examine further the role of Dib1 in pre-mRNA splicing.

Meera Joshi
Wesleyan University

The Msh2–Msh6 protein complex is a member of the MutS family of proteins and is involved in post-replicative DNA mismatch repair. The protein diffuses along the DNA, specifically recognizing single base pair mismatches and small insertion/deletion loops. Recognition of a mismatch activates the recruiting of other proteins to repair the error.

Previous research in Ishita Mukerji’s lab and by others has shown that Msh2–Msh6 binds DNA Holliday junctions, a central intermediate in the DNA repair and recombination pathway, with high affinity.

This summer, the Msh2–Msh6 protein purification method was improved in order to ensure the purification of pure, nondegraded, active protein. Our investigation aimed to understand the Msh2–Msh6-junction binding interaction by studying nucleotide utilization upon binding. By determining the rate of ATP hydrolysis and ATP and ADP binding, we understood how ATP is utilized in binding to DNA junctions. In particular, we addressed whether protein conformational changes are correlated with the ADP–ATP hydrolysis cycle and whether junction binding is an important Msh2–Msh6 function in mismatch repair.

My summer research experience has taught me not only valuable scientific skills but also the mindset needed to be a good researcher and troubleshoot problems that occur during research. It is important to have a creative approach that considers multiple angles to tackle problems and find solutions.

Ryan McCool
University of San Diego

This project in Jessica Bell’s lab focused on the suppressor of IKK epsilon, or SIKE, a protein with unknown function in the dsRNA-activated TLR3 immune pathway. We did phosphomimetic studies based on replacing SIKE’s six phosphorylated serine residues with phosphomimetic glutamates, or S6E SIKE, to further our understanding of the previously noted SIKE–tubulin interaction. We used size exclusion chromatography to determine that S6E SIKE experiences a shift from primarily dimeric to primarily monomeric species.

While no blatant alterations to secondary structure have been observed through circular dichroism studies, S6E SIKE loses its ability to renature to its initial state, as determined by thermal denaturation/renaturation assays. Through modeling of the phosphomimetic SIKE on the wild-type SIKE model, these alterations to quaternary structure have been attributed to an increase in negative charge density near SIKE’s C terminus. This charge concentration also has suggested a possible site for the SIKE–tubulin interaction, specifically in the tubulin heterodimer’s region of positive charge density. Although initialized to determine...
association and dissociation constants of the SIKE–tubulin interaction, surface plasmon resonance, or SPR, studies were hindered by nonspecific binding of tubulin to the SPR chips. Future studies will attempt to reduce nonspecific binding, which currently may reduce our ability to detect SIKE–tubulin heterodimer interactions via SPR. Microtubule binding assays will test whether SIKE interacts with microtubules. Future studies will explore SIKE’s effect on tubulin de/polymerization and map the surface of the SIKE–tubulin interaction via limited proteolysis coupled to liquid chromatography with tandem mass spectrometry.

Matthew Paal
St. Olaf College

Alcoholic fatty liver disease, or AFLD, is characterized by an increase in lipid droplet accumulation in the liver after acute ingestion of alcohol. If left untreated, AFLD can progress to cirrhotic liver, ultimately requiring a liver transplant.

In Laura Listenberger’s lab, I examined how early changes to the structure of lipid droplets in AFLD could contribute to the progression of the disease. Specifically, I developed and utilized an in vitro assay to determine whether changes to the phospholipids at the surface of lipid droplets could impact protein binding. These experiments demonstrate that the relative levels of phosphatidylcholine and phosphatidylethanolamine (the two most prevalent classes of phospholipids) can impact association with perilipin 2, an abundant lipid droplet protein.

Through this experience, I developed a researcher’s mindset. Mainly, I learned how to approach a problem from a scientific viewpoint through in-depth literature research on both topics and the methodology of prior experiments.

The most important thing I gained from this project was learning how to troubleshoot and move on from past mistakes. Performing research is rarely a straightforward road but rather a gravel trail full of potholes that requires critical thinking and carefully thought-out experiments.

More recipients

Other 2019 Undergraduate Research Award recipients were Nana Aikins, working in Suzanne O’Handley’s lab at the Rochester Institute of Technology; Josiah Byler, working in Nik Tsotakos’ lab at Penn State; John Mullins, working in Tayo Odunuga’s lab at Stephen F. Austin State University; and Destiny Paige, working in Rajnish Singh’s lab at Kennesaw State University.

Destiny Paige received a 2019 Undergraduate Research Award for her work in Rajnish Singh’s lab at Kennesaw State University.
The ASBMB website has a new look. Visit asbmb.org
Do sperm offer a secret handshake?

Researchers discover endometrial receptor that can recognize surface molecule

By Laurel Oldach

Why does it take 200 million sperm to fertilize a single egg? One reason: When sperm arrive in the uterus, they are bombarded by the immune system. Perhaps, molecular anthropologist Pascal Gagneux says, many are needed so some will survive. On the other hand, the female may benefit by culling so many sperm.

“I’m a lonely zoologist in a medical school,” Gagneux said. “My elevator spiel is that all of life is one big compromise. (For an egg), being too easy to fertilize is bad; being too difficult to fertilize is also bad.”

Gagneux’s lab at the University of California, San Diego, has discovered the makings of something that might be compared to a secret handshake between sperm and the cells lining the uterus in mice and, perhaps, humans. Uterine cells, they report in the Journal of Biological Chemistry, express a receptor that recognizes a glycan molecule on the surface of sperm cells. This interaction might adjust the female’s immune response and help sperm make it through the leukocytic reaction.

The leukocytic reaction is not well understood. What we do know, Gagneux said, is that “after crossing the cervix, millions of sperm — a U.S. population worth of sperm — that arrive in the uterus are faced by a barrage of macrophages and neutrophils.”

This attack by the innate immune system kills most of the sperm cells in semen, winnowing hundreds of millions down to just a few hundred that enter the fallopian tubes. The defensive response may help prevent polyspermy, when an egg is fertilized by more than one sperm and cannot develop.

Sperm are coated in sialic acid–rich glycans, and the innate immune system uses sialic acid to differentiate human cells from invaders, so Gagneux and his lab expected that the glycan might interact with innate immune cells called neutrophils. But human neutrophils they tested were activated to a similar degree by sperm with and without sialic acid.

Meanwhile, the team noticed sialic acid–binding receptors called siglecs on endometrial cells. In solution, these endometrial receptors can bind to whole sperm. According to Gagneux, the binding interaction might help the sperm run the gauntlet of the leukocytic response — for example, by dampening the immune response. Alternatively, it may be a way for uterine cells to weed out faulty sperm. In the immune system, siglecs help cells to recognize sialic acid molecules as markers of the body’s own cells, and in that context they can turn inflammation either up or down.

“It’s somewhat embarrassing how little we can say about what this (interaction) means,” Gagneux said. To understand its physiological significance, researchers first must look for direct interaction between sperm and intact uterine tissue — this paper looked at only sperm interacting with purified proteins and isolated cells.

It’s humbling to work in such a poorly understood area, Gagneux said. Reproduction “is a very, very delicate tug-of-war at many levels. The fact that there is (also) this immune game going on is completely fascinating.”

DOI: 10.1074/jbc.RA119.008729

Laurel Oldach (loldach@asbmb.org) is a science writer for the ASBMB. Follow her on Twitter @LaurelOld.

An artistic rendering (not to scale) shows how the “secret handshake” between sperm and endometrial cells takes place. The surface of sperm is coated in glycans, branched structures that are rich in sialic acid. These can be recognized by sialic acid binding receptors, called siglecs, found on the surface of endometrial cells.
How to catch an HIF

Gregg Semenza’s lab and hypoxia-inducible factor 1

By Martin J. Spiering

Every animal cell needs molecular oxygen to survive and do its job. Because skin, tissues and organs block most oxygen diffusion directly from the air, molecular oxygen concentrations inside the body often are less than 5%, much lower than the 21% in Earth’s atmosphere.

As multicellular organisms evolved, this steep oxygen gradient necessitated the development biochemical mechanisms that monitor and control oxygen levels in the body.

A major player in sensing potentially harmful drops in cellular oxygen concentrations, or hypoxia, is the transcription factor hypoxia-inducible factor 1. HIF-1 and its close relative HIF-2 regulate many genes, including the erythropoietin, or EPO, gene, which encodes a hormone that stimulates production of red blood cells.

Catching HIF

Gregg Semenza and his lab at the Johns Hopkins University School of Medicine purified and biochemically characterized HIF-1 in the mid-1990s, a milestone in uncovering its pivotal role in oxygen sensing and part of the work that earned Semenza a 2019 Nobel Prize. The two Journal of Biological Chemistry papers reporting that early work are recognized as Classics.

“JBC was the first choice,” Semenza said of the decision where to publish the first paper, “to me, that was a classic JBC paper.”

A few years before identifying HIF-1, Semenza’s team had found that hypoxia induces the binding of a protein to an enhancer of the EPO gene. Although the researchers could pinpoint the DNA region that is bound by the hypoxia-induced protein, the protein’s identity was unknown, prompting Semenza to look for it.

Semenza’s team first had to cross some unexpectedly rough waters. “We used an expression cloning strategy,” he said. The researchers expressed human proteins from cDNAs in bacteriophages and used the HIF-binding oligonucleotide to find proteins binding to it. “We screened millions and millions of clones and got nothing.”

They changed tack, embarking on a biochemical purification that involved growing HeLa cells in large cultures, exposing them to hypoxia, preparing protein extracts from them and purifying HIF-1 via chromatography.

The authors could show that purified HIF-1 has two subunits: a larger one of 120 kilodaltons, called HIF-1 alpha, and a smaller one of 91 to 94 kilodaltons, called HIF-1 beta.

The purification yielded enough protein of the two HIF-1 subunits for amino acid microsequencing that gave short protein sequences the researchers could use to identify the full-length sequences of the HIF-1 subunits.

These sequences showed that both HIF-1 subunits belong to a group of transcription factors containing a motif required for dimerization and...
DNA binding and a domain for interactions with other proteins.

The HIF-1 beta sequence was identical to that of a previously identified protein, aryl hydrocarbon receptor nuclear translocator, known as ARNT, a subunit of the dioxin receptor, which senses dioxins and other toxic chemicals. However, the HIF-1 alpha sequence was unknown previously and only partially similar to single-minded homolog, a transcription factor in Drosophila.

The second Classics paper helped define the regions in the two HIF-1 subunits required for their dimerization, DNA binding and activation of transcription of hypoxia-induced genes.

What might look now like a series of straightforward steps of purifying and sequencing a protein was an exercise in continual improvisation.

“My lab was a molecular genetics laboratory,” Semenza said. “We didn’t even own a fraction collector.”

Luckily for Semenza, the lab of another Johns Hopkins researcher, Thomas Kelly, was just across the street.

“Tom was one of the first people to purify a protein based on its binding to DNA,” Semenza said. “We couldn’t have (isolated HIF-1) without the help from Tom Kelly’s lab.”

A ubiquitous factor

The biochemical characterization of HIF-1 threw open the doors to many additional studies from Semenza’s group and other labs.

These investigations showed that the specific HIF-1 binding site is ubiquitous in the human genome. This hinted at HIF-1’s role as the hub of metabolic regulation in response to oxygen.

“These genes are not induced by hypoxia in every cell type under every condition,” Semenza said. “They’re induced by hypoxia in some cell types under some conditions — the plasticity of the (hypoxic) response is remarkable.”

The HIF-1-regulated genes encode proteins in many metabolic processes, including glycolysis, angiogenesis and wound healing. They also are upregulated when people move from lower to higher altitudes, and they are hyperactive in some diseases, most notably cancer. HIF-mediated signaling is now a therapeutic target to combat cancer and manage disorders such as altitude sickness.

Specific prolyl-4-hydroxylase domain, or PHD, proteins regulate HIF-1’s activity. In the presence of normal oxygen concentration, the PHDs modify two conserved proline residues in HIF-1 alpha, leading to binding by the von Hippel–Lindau protein, which targets HIF-1 alpha for degradation.

“So (cells) make HIF1alpha, but it’s being degraded when oxygen is available,” Semenza said.

Limiting oxygen levels inhibits the PHD proteins, increasing the fraction of HIF-1 alpha that’s not hydroxylated. This stabilizes the HIF-1 protein, causing its accumulation and leading to the activation of its target genes.

“It is a really beautiful system,” Semenza said.

In addition to the 2019 Nobel Prize in physiology or medicine, which he shared with two other physician–scientists, William Kaelin Jr. and Peter Ratcliffe, Semenza received the Albert Lasker Award for Basic Medical Research in 2016 for his work on the role of HIF-1 in oxygen sensing.

DOI: 10.1074/jbc.270.3.1230
DOI: 10.1074/jbc.271.30.17771

Read more Journal of Biological Chemistry Classics at jbc.org
Taking the measure of glycans

By Laurel Oldach

When Lorna De Leoz invited labs to participate in her glycomics study, she hoped for 20 responses. Instead, she was deluged by emails from around the world.

De Leoz, then a research chemist at the National Institute for Standards and Technology was planning a study on how pharmaceutical and academic laboratories measure glycans, complex carbohydrate molecules that cells use to posttranslationally modify various proteins. The project, the subject of a recent paper in the journal Molecular & Cellular Proteomics, grew to include 76 participating labs.

The flood of responses, including from many well-known pharmaceutical companies, illustrates the industry’s appetite for a better understanding of protein glycosylation. A growing number of drugs are made from proteins — most often antibodies — and this is a big variable in manufacturing them.

Glycans make up just 3% of the weight of an antibody-based drug, but they have an outsized impact. A change in glycosylation from one batch to the next can alter a drug’s binding to its target or the likelihood that it will be attacked by the patient’s immune system. To make sure patients receive the safest and most effective medicine, researchers must be able to keep track of the glycosylation status of antibody drugs.

Stephen Stein, a fellow at NIST’s mass spectrometry data center, was the senior author of the study. “Glycosylation is one of the most important physical-chemical aspects of biology,” he said, “but one of the most difficult to analyze.”

Researchers have developed many methods to measure glycosylation, and their results can vary. That’s where the NIST comes in: The institute is dedicated to establishing shared measurements. To help glycomics scientists get clear about what they’re measuring, De Leoz sent each participating lab a sample of the same antibody, a failed drug candidate. It took almost a month to pack up samples and fill out dozens of customs forms.

The glycomics techniques among the participating labs were diverse. Some measurements, often used in manufacturing, are optimized to find the exact quantity of one or two glycan structures; others, more often used in academia, are better suited to showing the broad scope of glycan types that are present. “There was no one method that was clearly better than all the others,” Stein said. “They all have different advantages and disadvantages.”

The NIST researchers used a statistical approach to combine measurements from disparate labs into a list of consensus estimates of the concentration of certain glycan compositions. The NIST will leave it up to the research community to figure out which measurement approach to use. MCP Editor-in-Chief Alma Burlingame said he hopes the paper will “point out to the biopharma industry and glycoanalytical community the urgent need to bring some methodological rigor to bear in the popular and important field of FDA-regulated antibodies.”

DOI: 10.1074/mcp.RA119.001677

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Junior associate editors organize virtual issues

The junior associate editors of the *Journal of Lipid Research* have organized four virtual issues highlighting cutting-edge research published by the journal. Check out all four issues at jlr.org.

**Lipoprotein (a)**

Many strides made, yet there is a long road ahead: In this virtual issue, the JLR highlights early-career researchers pushing our understanding of lipoprotein (a) pathophysiology forward. According to Gisette Reyes-Soffer of the Columbia University Irving Medical Center, a better grasp of lipoprotein (a) is necessary to improve risk evaluations and targeted therapies for cardiovascular disease and diabetes.

**Exploring the nuances and complexity of lipoprotein clearance**

This virtual issue, assembled by Brandon Davies of the University of Iowa Carver College of Medicine, highlights studies that advance our understanding of lipoprotein clearance and illustrate the breadth and diversity of lipoprotein-clearance research.

**Solving the enigma of the sphinx, one sphingolipid at a time**

Recent studies have advanced our understanding of sphingolipids and their biologic roles in health and disease. This virtual issue, organized by Rotonya Carr of the University of Pennsylvania Perelman School of Medicine, also highlights both established and new investigators in the field who undoubtedly are the future of sphingolipid research.

**Lipids in transcription and chromatin biology**

Ray Blind of the Vanderbilt University School of Medicine put together a virtual issue showcasing recent papers showing that lipids actively participate in epigenetic reprogramming, chromatin modifications and nucleosome structure, along with their more well-established roles in splicing, RNA export and transcriptional regulation.
Testing the lipoprotein–blood clot link

Blood clots affect 600,000 people in the United States each year. In a natural process called fibrinolysis, the enzyme responsible for keeping clots together is inactivated by a protease. High levels of the lipid-containing complex lipoprotein(a), or Lp(a), are linked to cardiovascular disease. Lp(a) may inhibit protease activation; therefore, lowering its levels could break up dangerous blood clots, though this hypothesized connection has not been confirmed in humans.

Michael Boffa and colleagues at the University of Western Ontario and the University of California, San Diego, write in the Journal of Lipid Research that their recent study shows substantially lowering elevated Lp(a) levels in patients does not affect fibrinolysis when compared to similar patients receiving a placebo. The researchers found that clotting factor levels and clot lysis times at several time points after administration were not significantly different between the two patient groups. These results suggest that Lp(a) may not have an appreciable role in inhibiting fibrinolysis; studies with more patients are needed.

DOI: 10.1194/jlr.P094763

Unlocking the role of vaults

Within many eukaryotic cells, small noncoding vault RNAs, or vtRNAs, associate with proteins to form large barrel-shaped cytoplasmic organelles known as vaults. Researchers have yet to uncover the function of vtRNAs, but now Nikolay Kolev and a team of researchers at Yale University and Bar-Ilan University in Israel provide novel insights by using a permeabilized cell system for the sleeping sickness-causing parasite Trypanosoma brucei. They identified the previously discovered and abundant noncoding TBsRNA-10 as a vtRNA and showed that downregulating it disrupts mRNA splicing. These results, published in the Journal of Biological Chemistry, implicate vaults in RNA metabolism and pave the way for studies to further elucidate their function.

DOI: 10.1074/jbc.RA119.008580

How deadly bacteria survive and multiply

Francisella tularensis is extremely infectious; just 10 of the bacteria can cause tularemia, a life-threatening infection. The Centers for Disease Control and Prevention has classified F. tularensis as a bioterrorism agent, and scientists now are getting a grasp on how this bacteria lives.

After being engulfed by white blood cells, F. tularensis is able to escape the phagosome inside the cell by using its type VI secretion system, or T6SS, to secrete its own proteins into the cell. This allows the bacteria to proliferate in the cytosol, causing the eventual death of the blood cell. Understanding this process can help us find ways to prevent it.

Jason Ziveri and colleagues at the Université Paris Descartes published a paper in the journal Molecular & Cellular Proteomics analyzing the change in the proteome and phosphoproteome during the assembly of a central component of the T6SS, the sheath. While the proteome remained unchanged, the team found one component of the sheath to be phosphorylated, the first evidence of phosphorylation in Francisella. More importantly, they found that when they mutated the phosphorylated residue to a non-phosphorylatable residue, the bacteria no longer could assemble the T6SS and escape the phagosome to grow in the cell, indicating that this phosphorylation event is essential to the bacteria’s survival.

DOI: 10.1074/mcp.RA119.001532

When proteins misfold, how do cells react?

Protein aggregation is a pathological hallmark of many devastating neurodegenerative diseases. While useful, current cell-culture models fail to capture proteasomal changes with high temporal resolution. Colin Gottlieb, Airlia Thompson and a team of researchers from Stanford University and Harvard Medical School addressed this gap using chemical biology approaches to observe an increase in ubiquitylation within minutes of protein misfolding. This work, published in the Journal of Biological Chemistry, suggests that protein unfolding is sufficient to induce a stress response. This discovery may lead to important new therapeutic targets for the treatment of neurological disease.

DOI: 10.1074/jbc.RA119.009654
Why CD23 from only some species can bind glycans

Sugars or carbohydrates, also known as glycans when found attached to proteins, are the underappreciated siblings of proteins, with which they often act in concert. Glycans play a critical role in cell–cell and cell–matrix interactions and the immune response. Despite their importance, glycobiology is still an emerging discipline, so glycans sometimes are referred to as the dark matter of the biological universe.

Lectins are proteins that bind to carbohydrates. One subtype is known as C-type lectin because its calcium ion dependency plays a crucial role in immune response to fungal pathogens and other microorganisms. Much remains to be learned about these interactions.

CD23 is a low-affinity immunoglobulin E receptor that also recognizes other ligands and is found in several cell types, including B lymphocytes. Its extracellular domain contains a C-type lectin–like domain that resembles a sugar-binding site, and previous studies reached conflicting conclusions on whether it can act as one.

In a paper published in the Journal of Biological Chemistry, Sabine A. F. Jégouzo and Hadar Feinberg, from Imperial College London and the Stanford University School of Medicine, respectively, and colleagues showed that CD23 acts as a glycan receptor in cows and mice.

The research team used solid-phase binding competition assays, glycoprotein blotting experiments and glycan array analysis to show that CD23’s lectin domain can bind several sugars (mannose, GlcNAc, glucose and fucose) alone or as part of a glycoprotein. They also provided a structural explanation of why this sugar-binding ability has been lost in humans and primates since their CD23 protein lost crucial amino acids required for sugar binding.

This research further elucidates CD23’s role as a cell-surface receptor and provides structural and functional evidence for the glycan binding of its C-type lectin–like domain. Because immunoglobulin E is an antibody isotope involved in allergy and resistance to parasites, these results are likely to influence future research into CD23’s role as a receptor for potentially pathogenic microorganisms.

DOI: 10.1074/jbc.RA119.010572

— Nathalie Gerassimov

An evolutionary overview of the sugar-binding ability of CD23 based on the conservation of the amino acid sequence of its C-type lectin–like domain. Green check marks on the right indicate likely sugar-binding ability based on the prediction from cow and mouse data. Red X’s indicated a loss of crucial sugar-binding amino acids.
How pathogens interact and exploit lipid rafts

Throughout a cell's plasma membrane are so-called lipid rafts. These small regions contain extra cholesterol and phospholipids and clusters of proteins involved in endocytosis and exocytosis for communication with the outside world. However, pathogens exploit lipid rafts to get through the membrane and into the cell. In a recent paper in the Journal of Lipid Research, Michael I. Bukrinsky and an international team reviewed this process from entry to exit.

Viruses, such as influenza A, make contact with specific receptors clustered in the lipid raft; the influenza virus rolls over many sites on the lipid raft until the precise host receptor protein is located. Other viruses gain entry by making pores in the membrane or hijacking the endocytic pathway to fuse virus with cell. To exit, the viruses can siphon a host pathway, where they are released outside the cell.

Although bacteria are larger than lipid rafts, they can bind host receptors and sometimes disrupt raft composition and gain entry. Other pathogens can benefit from lipid rafts without ever entering the cell; for example, by getting rid of cholesterol, the bacterial stomach bug Helicobacter pylori can bypass the immune response in intestinal macrophages. DOI: 10.1194/jlr.TR119000391

Salvage pathway not enough to make DNA

Deoxyribonucleoside triphosphates, or dNTPs — the building blocks of DNA — are formed in mammalian cells via either de novo synthesis or salvage of deoxyribonucleosides. To uncover to what degree cells can rely on the salvage pathway alone for DNA production, Phong Tran and a research team at Umeå University in Sweden developed a mouse model with a heart and skeletal muscle-specific deletion of ribonucleotide reductase — the enzyme that catalyzes the first step of DNA synthesis. These knockout mice exhibited aberrant production of DNA and proteins and underwent heart failure after the first postnatal week, which indicated that the salvage pathway on its own is inadequate in supporting DNA production. This work was published in the Journal of Biological Chemistry. DOI: 10.1074/jbc.RA119.009492

Golgi protein plays key role in virus maturation

Pathogenic phleboviruses can cause a range of diseases from mild to fatal. To find treatments for those infected, researchers must understand the virus lifecycle. One critical step that remains poorly understood is how these viruses bud and exit host cells. Zina M. Uckeley, Rebecca Moeller and a research team from Germany and Sweden published a paper in the journal Molecular & Cellular Proteomics on work that took a proteomics approach to elucidating this step.

The researchers examined the Uukuniemi virus of the phlebovirus family as it infected human cells. They performed a pull-down of the virus and used mass spectrometry to determine what host cell proteins came out with it. They found 39 cellular protein partners, picked the 12 candidates with the best likelihood of having a role in the virus lifecycle and knocked each down with siRNA. One gene, GBF1, decreased infection by 50% when knocked down, indicating that it plays an essential role. GBF1 resides in the Golgi and participates in the secretory pathway of cells, which viruses hijack. The researchers then examined other viruses that rely on the secretory pathway, finding that GBF1 plays a key role in the lifecycles of the Flaviviridae, Coronaviridae, Rhabdoviridae and Togaviridae families as well. GBF1 may be a promising antiviral target for many viruses that replicate in the cytoplasm of cells. DOI: 10.1074/mcp.RA119.001631

Where do FOGs come from?

O-glycosylation is a key protein modification in numerous essential cellular processes. Although free O-glycans, or FOGs, have been detected in the extracellular space, little is known about their origin. In research published in the Journal of Biological Chemistry, Hirayama Hiroto and team of researchers in Japan found that the yeast Saccharomyces cerevisiae, using mannose as a carbon source, produced FOGs similar to those attached to glycoproteins. They also deleted the general transcriptional repressor Cy8, which resulted in FOG accumulation and strong growth defects. These results revealed a novel mechanism of FOG removal from yeast and prompt the question of whether similar pathways exist in higher eukaryotes. DOI: 10.1074/jbc.RA119.009491

Blocking the SNARE-priming stage

Membrane fusion is required for vesicle trafficking and cellular homeostasis. Soluble NSF attachment protein receptor, or SNARE, proteins facilitate the fusion process and are highly conserved across species. N-ethylmaleimide sensitive factor,
Fatty livers and hormones

When too much fat is deposited in the liver, typically due to poor diet, a person can develop nonalcoholic fatty liver disease, or NAFLD. The hormonal system regulating the blood pressure renin–angiotensin–aldosterone system, or RAAS, may play a significant role in NAFLD presentation, although studies have yielded conflicting results. Some researchers have observed that blood pressure-lowering drugs such as beta blockers appear to affect weight gain associated with lipid accumulation, while others have not — or have found that blockade of a different pathway prevents weight gain.

Angiotensinogen, or AGT, an RAAS component primarily produced in the liver, is converted to the hormone angiotensin I by renin and angiotensin II by a different enzyme. Among its effects, angiotensin II increases blood pressure. This suggests that reducing AGT production could have an effect on NAFLD.

To study this question, Xin-Ran Tao and colleagues at the Zhejiang University School of Medicine engineered mice that did not produce AGT in the liver, placed some on a high-fat diet and investigated the effects. These mice gained less weight on the high-fat diet than mice with normal AGT. Also, fatty acid synthesis in the liver and liver steatosis, a precursor to NAFLD as fat accumulates, both were reduced in the non-AGT mice on a high-fat diet.

The researchers found that the pathway responsible for activating the transcription factor that controls fatty acid synthesis in the liver was reduced in the mice without AGT, linking AGT to fatty acid synthesis and suggesting why these mice had less weight gain on a high-fat diet.

This study, published in the Journal of Lipid Research, shows that hormonal systems may play a significant role in the lipid accumulation that occurs in NAFLD, and depletion of AGT may attenuate the effects of a high-fat diet.

DOI: 10.1194/jlr.M093252

— Dawn Hayward

or NSF, and its yeast counterpart Sec18 activate SNAREs, yet the lack of adequate reversible inhibitors has prevented a thorough understanding of molecular mechanism. In research published in the Journal of Biological Chemistry, Robert Sparks and Andres Arango of the University of Illinois at Urbana–Champaign and a team from several institutions used structure-based computational drug discovery to find a specific inhibitor of Sec18/NSF, providing a powerful new tool for deeper investigation of SNARE priming and membrane fusion.

DOI: 10.1074/jbc.RA119.008865

A STING defense against a parasite

Toxoplasma gondii is an obligate protozoan parasite that naturally infects all mammals, where it alters the host environment to establish chronic infection. In research published in the Journal of Biological Chemistry, Peiyan Wang and Siji Li of the Peking University Health Science Center and a team in China and the U.S. uncovered a new role for the T. gondii protein GRA15 in inducing an antiparasite response via the interferon stimulator STING. This parasite-driven host defense limits Toxoplasma replication while maintaining host survival, creating an ideal niche for the establishment of latency.

DOI: 10.1074/jbc.RA119.009172
A fast new way to ID bacteria in infections

When a patient with an infection needs treatment, doctors often take a sample of urine or blood to analyze and prescribe broad-spectrum antibiotics for a few days until the bacterial culture results come back from the lab. However, treating with broad-spectrum antibiotics is not very effective for some infections and has many unintended results, including contributing to antibiotic resistance. Using more species-specific antibiotics is the best route, because it lessens the number of bacteria unnecessarily exposed to antibiotics; however, current methods of determining what bacteria are present take a few days. Speeding this up would give the patient accurate, effective treatment more quickly.

Florence Roux-Dalvai and colleagues at the Université Laval Research Center in Quebec aim to address this problem by creating a new method to identify bacterial species in patient samples in under four hours. They recently published a paper on their work in the journal *Molecular & Cellular Proteomics*.

The researchers used liquid chromatography tandem mass spectrometry, or LC-MS/MS, of bacterial peptides and machine learning to identify peptide fingerprints of each bacterial strain. First, they grew pure bacterial cultures of 15 species commonly found in urinary tract infections and digested the proteins to create many short peptides. Then they analyzed the peptides by LC-MS/MS to create a library of thousands. Next, they inoculated healthy urine with bacteria and tested 190 samples to create lists of peptides, using the libraries created from the pure cultures as a reference. They then submitted those lists of peptides to the machine-learning program to determine peptide signatures for each strain. This resulted in a signature of between five and 26 peptides for each bacterial species. They then validated this algorithm by testing patient urine and successfully identifying the pathogens. This work holds real promise for improving the treatment of UTIs and many other infections.

DOI: 10.1074/mcp.TIR119.001559

— Elizabeth Stivison

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Cell membranes change shape during such functions as membrane trafficking, cell migration and cell division. Rearrangement of cytoskeletons and interactions of various proteins, including membrane-bending proteins, regulate these changes. When the composition and asymmetric distribution of lipids between the two leaflets of the membrane bilayer change, membranes are deformed, a requirement for processes such as immune response and nutrient uptake.

In mammalian cells, the phospholipids phosphatidylserine and phosphatidylethanolamine are abundant in the cytoplasmic leaflet of the bilayer, whereas phosphatidylcholine, or PC, and sphingomyelin are abundant in the exoplasmic leaflet. P4-ATPases, also called flippases, catalyze unidirectional flipping of lipid molecules from the exoplasmic to the cytoplasmic leaflets of the bilayer and are crucial for membrane trafficking, but we don’t know how they are coupled to the process of vesicle formation.

P4-ATPase–mediated phospholipid translocation might cause an imbalance of lipid mass between the leaflets of the bilayer, so our lab wanted to find out whether the flipping enzymes of the P4-ATPase family contribute to changes in the shape of cell membranes. PC is abundant in the outer leaflet, so we hypothesized that greater PC-flipping activity causes an increase in lipid mass in the inner leaflet of the plasma membrane and induces inward membrane curvature.

To test this hypothesis, we used BIN/Amphiphysin/Rvs, or BAR, domains, which can sense membrane curvature and induce tubular membrane structures via self-oligomerization. We examined plasma membrane-bending caused by BAR domains with a rapamycin-inducible dimerization system, using FK506 binding proteins, or FKBP, and FKBP–rapamycin binding domain, or FRB, proteins, that allow for acute recruitment of BAR domains to the plasma membrane. FRB was targeted to the plasma membrane by adding the N-terminal 11 amino acids of Lyn kinase, and FKBP was fused to the BAR domain.

When one specific BAR domain, called N-BAR, is recruited to the plasma membrane, we know that it penetrates the lipid bilayer via its N-terminal amphipathic helix, causing a small inward curve to form. The protein senses this change in curvature, leading to the recruitment of more N-BAR domains, which oligomerize along a part of the membrane and trigger transformation of the membrane into a tube.

Delta-N-BAR and F-BAR domains sensed the change and bound to the plasma membrane. The proteins then oligomerized along the membrane and transformed that part of the membrane into an inwardly protruding tubule.

Our recent study shows that changes in the transbilayer lipid compositions induced by P4-ATPase can deform biological membranes. Increased inward plasma membrane bending by ATP10A expression enhances endocytosis.

The plasma membrane also changes shape during cell migration, cancer cell invasion, cell division, nutrient uptake and entry of pathogens into cells, so lipid-flipping activity may be involved in any or all of these processes.

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THE WELLNESS ISSUE

Life can shatter us, as several of the essays in this wellness section attest. But like practitioners of kintsugi, the Japanese art of mending fractured ceramics with gold, we can accept and celebrate our brokenness and the steps by which we make ourselves whole again.
The authors pass one another in the lab.
Nathan: “How ya doing?”
Dan: “Fine, thanks.”
Are we, though? When someone asks us how we are doing, we might think about our health and, sometimes, our wellness. But how often do we give an honest reply about our well-being?

Diagnosing our work culture

Until recently, when we discussed health and wellness, we were referring to our physical (and, more recently, mental) state of being and the conscious choices we made to live a healthy lifestyle. For example, health assessments primarily looked for the absence of disease symptoms; fever, cough, rash, edema or pain implied poor health.

Thanks to a new understanding of how the brain and stress hormones affect organ systems, we now know that physical health can influence mental health and that chronic mental and emotional stress can lead to disease. This understanding that health and wellness extend into our emotional and social health has given new meaning to the term well-being — a contentedness where physical, mental, emotional and social health coalesce.

Academia is beginning to pay serious attention to mental health. In part, this attention follows a 2018 Nature Biotechnology paper in which graduate students reported moderate to severe symptoms of anxiety (41% of respondents) and depression (39%). And mental health disorders are not limited to students: A 2006 study found that 20% of faculty at several medical schools had significant levels of depression. Such studies show that a variety of personal, professional, situational and environmental factors — including work–life balance issues — play a role in the development of these and other disorders.

Underrepresented and marginalized populations disproportionately bear interpersonal, professional and social/environmental stressors, making individuals in these groups particularly vulnerable to low well-being and consequent educational attrition. For example, female and transgender graduate students had the highest rate of anxiety and depression in the 2018 Nature Biotechnology study, and another 2018 study found that women of color leave science, technology, engineering and mathematics studies in higher education due to an unwelcoming environment that can include social isolation and microaggressions. Other studies point to child-rearing — disproportionately the work of women — as a major cause of attrition from STEM fields.

Institutional stressors and social structures in our disciplines and professional cultures lead to low well-being, which in turn disproportionately drives individuals from marginalized populations out of the upper echelons of STEM fields, perpetuating a work culture that is largely white, male and cisgender. This is more than undesirable: It is unethical. It robs marginalized individuals of opportunity and perpetuates inequity.

A call to action

Employees are making demands for policies that promote well-being, which can boost workplace productivity and attract talent. Millennials now expect jobs that permit well-being. A recent study found that attrition from the STEM pipeline beyond the bachelor’s level frequently reflects a mismatch between work objectives, personal values and social identity. To recruit the best talent and to construct a diverse workforce, the STEM community must understand and invest in well-being.

Well-being is not just good for the individual: It is also good for the productivity of an organization. So how can we establish well-being in our STEM work environment? An internet search will turn up myriad articles written by people sharing their work–life balance strategies. These are insufficient to cure a culture of poor well-being. First, most of these fortunate souls already have made it to the upper echelons of their professions. What about strategies for people who are at risk of leaving STEM due to an oppressive culture?
of low well-being? Second, these writers suggest that it is an individual’s responsibility to find well-being in a potentially toxic work culture rather than the responsibility of the collective to establish a culture that supports the well-being of individuals and, in turn, the organization.

We in the STEM community — and particularly in academia — have an image problem when it comes to well-being. Our flexible work schedules, which we rightfully claim as a blessing, also can be a curse: Fluid schedules make it difficult to keep track of work hours, which is doubly problematic when our salaries are the same whether we work 40 or 80 hours in a week. STEM researchers report feeling pressured to work far more than a 40-hour week, such that admitting to a 40-hour full-time job sounds like a guilty confession. As evidence, a 2013 study found that more than one-quarter of all papers sent to the journal Biological Conservation were submitted on weekends or on weekdays between 7 p.m. and 7 a.m.

Bragging about how much we work and judging others’ commitment to science by their work hours only exacerbates a lack of well-being. We need to change this culture. Some first steps:

- Don’t perpetuate the culture by joining labs and institutions whose culture and policies promote poor well-being; you can find supportive mentors doing excellent work in other places.
- Don’t just be an example of someone who successfully has managed a work–life balance; explicitly tell your trainees and employees that they do not have to sacrifice their families to start a career or vice versa.
- Seek opportunities to empathize. Rather than dismiss the lamentations of your co-workers and trainees, listen to their appeals for ways to balance their obligations as a STEM professional with their personal needs.
- Better still, assume the roles played by your trainees and co-workers so you can develop empathy for their competing needs. For example, fathers who take on child rearing report reduced productivity and professional mobility, just like the STEM researcher mothers who do a disproportionate share of child rearing. When we understand our peers’ need for well-being, we can find better solutions for both the individual and the organization.

Institutions now offer health and wellness programs, but these are insufficient and ineffective. This low return on investment, some say, is the result of these programs’ attention to the health of the whole person, their well-being. Rather than incentivize short-term fitness goals, we must build capacity for mental, emotional and social health within our institutional structures.
A meeting theme

In this spirit, we are developing a thematic track at the 2020 American Society for Biochemistry and Molecular Biology annual meeting that questions who we are and what we do. The first session — “Who we are” — challenges us to consider how we can better foster a culture of well-being. Voices from academia, industry and government ask: How do we prevent and overcome harassment in our STEM environments? What contributes to equity and retention of underrepresented students in STEM? How can we be better mentors to support the well-being, particularly the mental well-being, of our trainees? The second session — “What we do” — questions how we can go about dismantling institutional barriers that otherwise stymie equity and access. First, we ask how evidence-based practices can be used to make our classrooms a place where everyone can achieve their full potential. Then, two speakers challenge us to rethink the traditional delivery of curricula by demonstrating how stories can be used to elicit STEM content. In creating these sessions, we hope attendees will leave their preconceptions and traditions at the door. Instead, we want participants to reimagine how we teach by asking ourselves, “Whom do we bring along, and whom do we leave behind?”

Together, we hope we can develop a stronger culture of well-being within the STEM workforce, one in which mindfulness about equity and inclusivity removes the barriers that impede people in underrepresented groups from realizing their full potential. We invite you to share and learn with us as we build a more inclusive culture that moves beyond health and wellness and instead uses well-being as the benchmark.

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PULSE POINTS

Research can spark change. Scientific inquiry exposes health risks and leads to new treatments for disease. As you read this wellness section, track the Pulse Points highlighting studies on risks associated with e-cigarette use and permanent hair dye and chemical hair straighteners, efforts to understand aging in dogs and people, and a life-saving treatment for children with relapsed B-acute lymphoblastic leukemia. Links to information are at asbmb.org/asbmbtoday.

Do age research with your dog

With the help of tens of thousands of dogs and their owners, researchers are embarking on a study about how genes, lifestyle and environment influence aging. The Dog Aging Project, supported by a grant from the National Institute on Aging, is in the first phase of its project journey. Dog lovers based in the U.S. are invited to nominate their pets for the project. Pet owners will become citizen scientists by reporting back to researchers about their dogs’ health.

Researchers hope that dogs will provide insights about how to address challenges of aging for both pets and people. Learn more at www.dogagingproject.org.

Daniel Promislow, professor at the University of Washington and co-director of The Dog Aging Project, with his dog Frisbee.
THRIVING AS A MESSY OTHER IN ACADEMIA

By Kelly Chacón

The road to becoming a professor, particularly for people of color and those considered “Other” (LGBTQIA+, low socioeconomic status, first-generation immigrant), is rarely smooth. A quick skim of my Twitter feed shows me that the experience of being an aspiring academic while Other is alive with tears, laughter and outrage — and yet we persevere because we hope to arrive, at some point, at the dream job at a university or college we love, carrying out the research and teaching that fulfills us.

Thirteen years after starting at 23 with nothing but a GED and a vague dream, I became an assistant professor. I have a beautiful lab, great colleagues and amazing young minds to inspire. I have arrived. But what exactly does arrival mean? When do we truly feel we’ve made it? Why does everything I do not quite look like what my colleagues do? Why do we often feel so isolated and alone at work when everything is going so well?

My feeling of impostor syndrome is real and comes from a real place. As a long-closeted queer Latina who is the child of immigrants, is a first-generation college student and grew up broke, I find it intense and debilitating. Every day I am reminded that my journey and my worldviews are very different from my colleagues’. The only way to deal with such severe feelings of being an imposter is by watching our peers carefully, mimicking what we see and trying to produce work that is beyond what is asked so that no one will find out we’re faking.

For me, this watchful mimicry and perfectionism only worked for so long. A year into my dream tenure-track job, my alcohol intake increased to two, three and sometimes four drinks. Every. Single. Night. It is a dirty little secret that academics tend to abuse alcohol — for the social lubrication among strangers after a long conference session, for conviviality among institutional colleagues and as a so-called nightly treat in our pajamas during a 70-hour work week as a junior professor.

I began to unravel.

The biochemical effect of ethanol as a hormone — as a poison — is quite sinister. With the relief it provided, I could stop worrying at bedtime and drift to sleep. Yet it also disrupts sleep, and I woke in the middle of every night anxious and afraid of failing and of everything falling apart. During the workday, I was numb with withdrawal, anxious, depressed and tired; I thought longingly of my couch and a drink.

To be clear, I was doing my job and even thriving as viewed from the outside, but the negative cycle and pain continued in private. Under the hazy of alcohol abuse and undiagnosed anxiety, I began to question why I was fighting so hard to succeed at a job that was so difficult for me when others in my cohort seemed to find it much easier and even fun. I fantasized about walking away from the torment of unbelonging.

Fortunately, my college supports a junior leave and sabbatical, and I decided to take it a year earlier than most. I visited a university far away to do sabbatical research. Living in an adorable studio apartment, I nipped the alcohol abuse in the bud by setting limits, self-reflecting and getting therapy. The alcohol was a coping mechanism for my anxiety, and I began to take a prescribed low dose of medication along with psychotherapy to help me process what an astronomically impossible journey to success I had achieved.

This intervention revived my quality of life, and it saved my career. As I emerged from my endless cycle of self-doubt and anxiety, I also returned from sabbatical. I had cried in my therapist’s office the week before classes began, panicking about coming back to a job that I also knew I loved. She let me process these valid feelings, and I calmed down. Then she locked eyes with me and asked, “What is the worst thing that could happen if you made a real decision to start doing things your own way at work, Kelly?”

I was stopped in my tracks. In that emotionally charged moment, I accepted that I am different from most of my colleagues. I am a “diver-
sity hire.” I don’t naturally do things in a normative academic way, and if I wanted to thrive, I needed my institution to assimilate to my worldview rather than the other way around. As institutions improve their unbiased hiring practices (and my hire was potent evidence of this), they must adapt to a changing way of designing and doing academic work.

I decided to be a visible example of what it looks like to exist and work unapologetically as an Other in the sciences and in academia at large.

So much has happened in the years that followed that moment, but a few key changes have allowed me truly to blossom. My classroom now is centered around the practice of radical vulnerability: When I stopped overpreparing so as to appear invincible in my lectures, I began to make small mistakes in front of my students and to allow them to do the same. I share stories about who I am as a real, fallible person. I do this on purpose — my students need to see that professors are human so they can see that despite their own imperfections, they too could be in front of the class someday.

This extended to my interactions with colleagues — I became very open about how I grew up, about my cultural differences and my view on work–life balance as focused on la vida. Most significantly, I took a tip from another Latinx scientist who was overbooked with demands for service and student meetings, which often occurs for a professor who is an underrepresented minority. I cleared one entire weekday each semester to work uninterrupted off campus. My other workdays are long and completely booked, but off campus I am able to write grants, run self-care errands, and spend time on scientific and academic pursuits that make me a better profé when I am in my office.

Some people may look at these behaviors and see a person who is messy, lazy and not fit to be in the academy. But to my delight, I have become proud of my science, teaching and outlook, and my colleagues cheer me on as I fly.

I never imagined I would feel like I belong despite being different among my amazing peers. While I wish I had sought help sooner, the hard journey was absolutely worth this moment of true arrival.

Kelly Chacón prepares a protein sample to be analyzed by X-ray absorption spectroscopy at Beamline 4-3, a side station at the Stanford Synchrotron Radiation Lightsource in Menlo Park, California. She is using tender x-rays to probe sulfur oxidation state in sulfur homeostasis proteins.

Kelly Chacón is an assistant professor in the chemistry department at Reed College in Portland, Oregon, and a member of the ASBMB Today editorial advisory board. Follow her on Twitter @Kelly_N_Chacon.
HOLIDAYS MAY BREAK OUR RESOLVE, BUT NOT OUR MICROBIOMES

By John Arnst

If you’ve hit the potatoes, pies and thick turkey thighs a little too hard over the past six weeks, you’re not alone — least of all because trillions of bacteria in your gut were actively helping you pull as much energy as possible out of that pecan-crusted indulgence.

As you settle back into your nonholiday routine, though, you may wonder if your microbiome — arguably the hottest medical frontier of the past decade — has become a micro-PIE-ome, turning your recent cravings into habits and undermining your discipline for the foreseeable future.

The connection between what we eat and which bacteria wind up dominating our gut is well established, and people with high-fiber diets consistently have been found to have both a greater diversity of gut bacteria and a lower incidence of gastrointestinal inflammation than people consuming considerably less dietary fiber.

In a citizen project that sequenced the 16sRNA in stool samples from 248 volunteers, researchers from the Skolkovo Innovation Center in Russia and the University of Groningen in the Netherlands wrote in the journal Nutrients in 2018 that volunteers who self-reported switching to a diet that included more vegetables and fruit over the course of two weeks had increased levels of butyrate-producing Clostridiales bacteria and decreased levels of Bacteroidales and related bacteria associated with diets rich in saturated fats and animal protein.

Additionally, several research groups independently have found gut microbes to be correlated with disorders including depression, anxiety and Parkinson’s disease, a connection made plausible by signaling molecules transmitted along the gut–brain axis. Researchers have found that mouse microbiomes can be disrupted by low-fiber diets in as little as a week and that gut bacteria can direct cravings in amino acid-deficient fruit flies.

Despite these correlations and studies, Purna C. Kashyap, a physician who runs the Gut Microbiome Laboratory at the Mayo Clinic, says a few weeks of eating nonhabitual foods are unlikely to alter the composition of your gut bacteria significantly.

“Short-term diet changes usually don’t cause a lasting change in the (human) microbiome,” he said. “If you look at the majority of the human studies — the dietary interventions even for four or six weeks — if they’re small dietary interventions, they don’t change the microbiome as much. And almost always, when you revert back to your diet, your microbiome comes back to its original state.”

Kashyap compared the gut microbiome to an elastic waistband that can be stretched regularly but still retain its shape.

“The microbiome is actually pretty resilient … but, if you stretch it beyond a certain extent, then you can’t get its shape back. But it takes a lot of effort to get to that point; it doesn’t happen overnight,” he said.

“The only caveat to this is that each individual has its own microbiome, which has its own resiliency, which means that the degree to which it would get disrupted and the speed with which it would come back may vary from individual to individual.”

That resiliency depends on the initial state of a person’s gut microbes — subjects who have lower microbial diversity and fewer of the fiber-digesting bacteria generally associated with lower levels of inflammation are more susceptible to overall bacterial turnover than subjects with more robust microbiota.

As far as cravings are concerned, the signaling methods bacteria might employ to drive us to seek food they prefer aren’t well defined biochemically, though they were proposed in a 2014 review in Wiley’s Bioessays that opined on the evolutionary role that gut bacteria might play in their host’s eating behavior.

Additionally, in 2017, researchers from Monash University in Australia
and the Champalimaud Centre for the Unknown in Lisbon found that gut bacteria in Drosophila played a role in modulating food choice when flies were presented with diets deficient in selective amino acids. They described their work in the journal PLOS Biology.

“Ecologically and logically, if you think about this, it could make sense, because the bacteria drive a lot of the things we may be doing without realizing that they have to root for their survival,” Kashyap said. This can include increasing the levels of the hunger-related hormones glucagon-like peptide 1 and peptide YY.

“As you can imagine,” he said, “it’s a little bit more complex than Drosophila for us to be able to figure out what’s driving the food choices.”

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B eing chronically ill while studying or working in science, technology, engineering or mathematics can be a lonely experience.

So, not surprisingly, one of the first things Lauren Younger and I chat about in our first conversation is how glad we are to meet someone else in the same boat.

“As unfortunate as it is,” Lauren says early on, “it is nice to not be alone.”

We met over Twitter, as so many STEM students and early-career researchers do. This is how Lauren and I find ourselves, at 8 a.m. in Arizona and 10 a.m. in Massachusetts, chatting about our experiences as chronically ill college students.

Lauren and I have vastly different experiences, in terms of both illness and schools. I won’t get into details about our diagnoses, but she is an undergraduate at Arizona State University, which has an enrollment of almost 72,000 on a 664-acre campus, and I am an undergraduate at Lesley University, a liberal arts school with about 6,000 students where classes are small and my department is even smaller.

As we share pieces of our stories, many seem familiar. This is why I’m chatting with Lauren. I want to show that, regardless of the person or their illness, we can find through lines in our stories.

“I’m totally coming from the same place,” I say, for what must be the 10th time in 10 minutes as we chat about our similarly debilitating illnesses. We were both more able-bodied early in our college journeys, and we remember the health reserves we had the privilege of using during those first semesters.

This is not to say those semesters were easy. One of the first things able-bodied people should understand about their chronically ill peers, students and colleagues: Nothing is ever easy.

Lauren and I both remember the beginning of our time in college fondly for the energy we had and the coursework we were able to maintain, but that doesn’t mean we weren’t struggling. Our illnesses are constant and persistent, filling our days even when we’re able to push through.

By her second semester, Lauren says, she was spending “more time in the emergency room than class.” As a freshman, I remember thinking, “The ER can’t fix my illness; no one can.”

But the paths of our illnesses are not our only common ground. We chat about other parallels: She and I both are teaching and research assistants, both chemistry geeks, both huge fans of dogs (we realize this while gushing over a picture of Lauren’s Corgi–cattle dog mix, Gracie).

And this is the second piece of what I wish people knew about being chronically ill in STEM: We are still people.

We are still students and scientists, and we have merit outside of our illnesses.

Lauren and I discuss this topic as it relates to funding and grad school applications. She mentions that folks have suggested to her that a National Science Foundation grant would be an easy get “with what I’ve gone through.”

We both get angry at the implication. “Shouldn’t my merit and research get me funding, though?” Lauren asks.

Neither of us appreciate the suggestion that pity, rather than our accomplishments, should help us get ahead.

“I want to convince people that their pity is unfounded,” I say, and Lauren agrees. “The pity train,” as Lauren dubs it, is an incredibly discouraging lens through which to be viewed.

Though we are both chronically ill, we’re also incredibly passionate about the work we do. Lauren completed a NSF Research Experience for Undergraduates at the University of California, Davis, doing research in computational and theoretical chemistry. At Lesley, I do research in chemistry education. We are eager to talk about our work and the coursework that goes along with it.

We spend a few minutes chatting about our favorite chemistry subjects (organic for me, physical for Lauren), but that quickly turns into a conversation about inaccessible lab spaces
and course design.

“No one ever seems to have had experience with chronically ill people,” I say, and Lauren quickly agrees. We’ve both had wonderful experiences with individual courses and professors. Lauren mentions professors who offer extensions and waive required attendance, and I, for the thousandth time in two hours, say, “Same.” But neither of us has been in a science environment that puts forethought into accessibility.

This is the final piece of the puzzle (at least for this essay): Consider us. Remember that we exist before we remind you. Science, Lauren points out, is inaccessible by design because of a lack of thought. Neither of us has been put down by professors or staff for being disabled — though I note that we are both young white or white-passing women, and this may not be the case for people with marginalized identities in addition to disability — but we are always some of the first disabled people that our departments and their systems have worked with.

“They just literally have never come into contact with someone like me before,” Lauren says. “They don’t know what I need when it comes to help.”

We have to advocate for ourselves and for all disabled folks in STEM, the two of us agree. It’s a lot to put on our plates, on top of being students and teachers and researchers (and, and, and …).

In an ideal world, our able-bodied peers, colleagues, bosses and professors would lay some of the groundwork for our existence before we even show up. Lauren and I are not the first disabled scientists to bemoan our lack of access, and we certainly won’t be the last. It shouldn’t have to be a fight to exist in these spaces — we love what we do, and we just want to keep doing it.

If you have the chance, please advocate for the disabled scientists around you. One other thing Lauren and I have in common: We’d be grateful.

Katie Walsh is an undergraduate biology major and chemistry minor at Lesley University, where she currently works on chemistry education research. She can be found over at @khwalsh_ on Twitter.

Katie Walsh and Lauren Younger met on Twitter and bonded over their shared experience of chronic illness — and their love of dogs.
HOW I BARBECUED MYSELF WITH STRESS … AND RECOVERED

By Jill Bouchard

Graduate school for scientists is an apprenticeship. This means learning by doing, and it frequently means learning through failure. Grad students are new to the game but expected to make the next greatest scientific advance. They rarely have enough time to learn how to do something before actually doing it. As a result, when students get unexpected results, it’s unclear whether they made a mistake or the results are real. This constant uncertainty can cause students to worry and doubt their achievements continually.

To make matters worse, the culture in most labs is to work all the time, certainly more than the fair labor standard 40-hour week. Grad students are paid a meager salary that usually comes with a noncompete clause. Other professional programs don’t pay students, but they compensate with regimented program length and career prospects. The Ph.D. has flexibility in those respects, but the cost is ambiguity in how long it takes to finish and what nontraditional career one should take to be employable afterward. So Ph.D. students typically play a dangerous game of high-stakes, high-pressure roulette full of ambiguity and failure, making for a dangerous concoction of prolonged stress.

In high school and college, I learned to thrive under pressure by being self-critical and pushing hard. I excelled in my rigorous class schedule and landed promotions in my part-time jobs. But I let graduate school turn my formula for success into unhealthy self-doubt and self-loathing.

After all, scientists are skeptics; everything we know and learn is uncertain. For me this meant retroactively questioning whether I had done everything right and hating myself for every mistake. Being self-critical makes me a meticulous (and good) scientist; I try my best during experiments and check my math afterward, resulting in reliable work. But there’s a fine line between being your own best critic and having crippling self-doubt. I knew I was past the line when I drove through a nearly empty intersection one night and questioned whether I had just run a red light.

Constant anxiety, self-doubt and worry harmed both my mental and my physical health. I couldn’t eat a meal without hearing fireworks in my stomach and being in so much pain I almost couldn’t walk.

There’s serious science behind this; long-term stress has detrimental effects on many of our major organs. My stress in overdrive was turning up the heat on my internal organs and frying them with a steady influx of stress hormones.

The stress response is effective for the body’s flight-or-fight mechanism when it helps a person run fast (top half of infographic on page 35), pay attention and take care of immediate business. But when a body is always in fight-or-flight mode, stress hormones continually tell the heart to work harder, the immune system to be on the lookout, and the digestive system to wait or replenish quickly so as to be ready for the next battle. The neurotransmitters serotonin and dopamine also can contribute to barbecuing the body. Continued stress responses can have dire effects on the cardiovascular, immune and digestive systems (among others) and may cause permanent damage (bottom of infographic).

The key to rising above this abyss of stress is retraining the brain to handle stress in a more healthy way. I spent a lot of time in therapy retraining my brain. Below I share what worked for me. Everyone is different, and these five tips cannot replace finding a unique path through individualized therapy. Anyone faced with physical ailments due to stress should seek treatment for both body and mind.

Five tips to manage stress

Graduate school is hard enough. God forbid something happens in your personal life to push you off the cliff into anxiety and depression. Here are my five go-to strategies for climbing out of the pit of despair and not falling back in.

1) Ride the tide. After a group therapy class on how to manage anxiety, I walked away better equipped to
The bottom schematic shows the effect of stress hormones on the development of disease. "Unestablished factors" indicates that stress has been linked to the disease, but the biomolecular mechanism is still unknown. Plus signs (+) indicate that the effect listed is compounded with the effect listed above. IBS stands for irritable bowel syndrome. Inflammatory bowel disease, or IBD, is shown as Crohn’s disease and ulcerative colitis.

weather storms. It required a healthy dose of mindfulness and kindness, mostly toward myself.

I’m often stressed because I overthink my reactions (being mad that I feel mad about something or being frustrated with myself for making a mistake). In these situations, I try to practice willingness — giving myself the ability to notice my reactions from a distance (mindfulness) and giving myself permission to feel however I feel (kindness).

I use my critical side to observe and take notes while also allowing myself to make mistakes and have feelings (even negative ones). This is not a shoulda–coulda–woulda approach but rather a way to focus my critical nature on learning in the moment and taking everything in stride. It’s like when I tell a friend about something upsetting and she agrees she would be upset too. But I’m my own best friend.

I let myself feel whatever I’ve felt and let it pass in its own time — like riding a wave. I feel mad about an injustice or sad about the consequences of a mistake. This shifts the spotlight toward accepting the harsh truths of reality: Life’s not fair, bad things happen and I’m not perfect. As a result, I’m not fighting myself; I’m empathizing with myself, being more of a bystander to my own traumas. I can evaluate critically what triggers my feelings and how I respond to those triggers. This gives me the power to think of a better response the next time I’m faced with a similar trigger.

2) Accept that you always will have things to do.
My favorite self-help book is “Don’t Sweat the Small Stuff and It’s All Small Stuff” by Richard Carlson. I flip through its short chapters for inspiration. One of my favorites is Chapter 6: ‘Remind Yourself that When You Die, Your ‘In Basket’ Won’t be Empty.’ I’m a great list-maker, and this chapter reminds me that I never will finish a list. Or for every list I finish, I’ll make a couple more.

I still feel free to make lists, but I know it’s impossible to get it all done. I’ve learned to prioritize; sometimes this requires a hard look in the mirror to decide what I value most. As a result, I don’t beat myself up (as much) when my lower priorities don’t get done. And when I check something off my list, I try to enjoy it for a moment.

3) Stop multitasking. Most of my stress comes from trying to do too much with not enough time. I’m a normal person with limits to my average-sized brain, and I’m now comfortable with that (thanks to step 1). Instead I do things in bulk. When I have a lot of priorities, I pick the
thing that seems easiest, and then I do a bunch of it. If I need to respond to an email, I respond to other emails while I’m at it. Or if my brain is already trying to figure out how to do my next experiment, I let it. Then I keep the momentum going by planning an experiment to follow it. If I’m in the mood to get up and do something, I do the experiment, and then I make up some buffers ahead of time. For extremely time-intensive work such as molecular biology or protein purifications, I try to make multiples at one time. This method may slow me down initially, but once I finish the task at hand, I typically get results from two or more.

4) Learn when to say no. Healing takes time. Everyone knows this is true for the flu or a broken leg, but fewer people accept it for recovering from mental illness. Mental recovery is different for everyone and always a continual process. I’m an introvert, so I need to step back from whatever is causing me stress and breathe a bit. My mantra has become, “Under-promise and over-deliver.”

I have to gauge my own stress level, know the cost associated with a specific task (estimating time and multiplying by three is key), and weigh the costs and benefits. If I can’t estimate my stress or the timing for a task, I’m probably already overstressed and beginning to shut down. Then I know I need to say no. If I have a hard time saying no outright, I’ve perfected the lawyer version: “I’ll try to fit it in, but I’m not promising…” Even with someone who is unreceptive to hearing no, expressing reservations can open a dialogue about how to reduce my burden.

5) Channel a character. To train myself to react differently to stressful situations, I pretend to be a TV, movie or book character. I channel Phoebe from “Friends.” Phoebe sees crappy situations for what they are, calls them out and moves on without a beat. In one episode, she finds out her sister is a porn star using Phoebe’s name in the movies. Phoebe tries talking to her sister, but her sister refuses to change her behavior. So Phoebe goes to the porn company to change the address where they mail her paychecks. And she doesn’t look back.

I also like to channel Luna from the Harry Potter series. I love the moment Harry finds her walking barefoot as she tends some mythical creatures. She has suspicions about who stole all her shoes, but she seems unbothered and rises above the annoyance.

Jill Bouchard (jbouchar@alumni.nd.edu) completed her undergraduate degrees in chemistry and political science at the University of Montana and did graduate studies in biochemistry at the University of Notre Dame. As a postdoc at St. Jude Children’s Research Hospital in Tennessee, she studies biophysical properties of a tumor suppressor and correlates its in vitro behavior with cellular function and dysfunction. She is transitioning into a science communication career.

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PULSE POINTS

Study: E-cigarettes are a risk factor for respiratory disease

A study by researchers at the University of California, San Francisco, Center for Tobacco Control Research and Education concluded that “use of e-cigarettes is an independent risk factor for respiratory disease,” defined as chronic obstructive pulmonary disease, chronic bronchitis, emphysema or asthma. In the study, recently published in the American Journal of Preventive Medicine, researchers conducted a longitudinal analysis of the adult Population Assessment of Tobacco and Health, or PATH. The study followed adults who did not have any reported lung disease and reviewed their subsequent e-cigarette use for two years (baseline plus two years of follow-up), Jason Alvarez at UCSF explained in a press release. “Though current and former e-cigarette users were 1.3 times more likely to develop chronic lung disease, tobacco smokers increased their risk by a factor of 2.6, he said.” Researchers also found that people who followed the most common pattern of use, continuing to smoke combusted tobacco products while using e-cigarettes, had an even higher risk of developing a respiratory disease than those who used either product alone. (For more on e-cigarettes, turn to page 45.)
NIH: Permanent hair dye and straighteners may increase breast cancer risk

According to a press release from the National Institutes of Health, a study published in the International Journal of Cancer found that women who use permanent hair dye and chemical hair straighteners have an increased risk of developing breast cancer. The paper’s abstract states: “We observed a higher breast cancer risk associated with any straightener use and personal use of permanent dye, especially among black women.” The researchers reported that “permanent dye use was associated with 45% higher breast cancer risk in black women … and 7% higher risk in white women.” The study used data collected by the National Institute of Environmental Health Sciences’ Sister Study, which tracks the health of more than 50,000 women from the U.S. and Puerto Rico with a sister who had breast cancer.
WHEN THE GOING GETS TOUGH, I GO TO THE GYM

By Lauren Hudson

The first semester of my freshman year hit me much harder than I expected. When I got to the University of Kentucky, I threw myself into college life with everything I had. I was in class for four hours each day, doing six hours of homework, rushing from volunteer activities to shadowing experiences, attending meeting after meeting of clubs on campus. I told myself this packed schedule was the key to getting into my dream medical school. I didn't realize it was slowly eating away at my sanity, my optimism and my happiness.

One day, after months of this craziness, I came back to my dorm after my 10 a.m. class. I had just popped a piece of bread in the toaster, opened my laptop and pulled up my chemistry work when a reminder popped up for a volunteer opportunity — an opportunity I had signed up for months before and forgotten about. It started in just 15 minutes.

I hurriedly spread jam on my toast, then dropped it on the floor. As I looked at my ruined meal, something inside me snapped. I began to cry. Then I began to cry harder. And harder.

I skipped volunteering and lay face down on my bed for who knows how long, contemplating everything I was doing wrong. I got angry at myself for being homesick, for not working hard enough — and for crying.

Eventually, my roommate (and best friend) came in. She put her arm around me and let me talk. When my tears finally dried, she told me what I was afraid to admit to myself.

“Lauren, you’re working yourself to death.”

I knew right then it was time for a change. I needed something to ease my mind — something that I could do for myself every single day that would relieve some of the pressure. I soon found a hobby that allowed me to take my mind off my responsibilities while still satisfying my go-go-go attitude: exercise.

Settling on exercise as my stress reliever came naturally. I can hardly remember a time in my life when I haven’t been active. I grew up on the soccer field, always playing year-round. My parents’ idea of family bonding is an all-too-competitive doubles tennis match. My brother and I love to hike in the summer and snowboard in the winter.

I soon realized I am at my happiest when my body is moving. Everything else seems to fade away when I run, walk, shoot, hit or kick; the type of activity isn’t important as long as I’m active. So one October day, I swiped my student ID at the entrance to the Johnson Center gym on UK’s campus … and I was hooked.

For the first few months, I didn’t put too much thought into my exercise routine. After years of running laps around the field and up and down the bleachers during soccer conditioning, I’d formed a love–hate relationship with cardio. Roughly five days a week, I went to the gym and simply ran until I couldn’t run anymore.

While it was satisfying to see the mile counter on the treadmill tick up, I began to feel twinges of pain in my knees. I quickly realized just how hard such intense running was on my joints. I found myself at a crossroads. If I kept running like this, I might need knee surgery before my 30th birthday. But if I stopped going to the gym, I would slip back into the unhealthy stressed-out routine I’d worked so hard to overcome.

Soon after hitting on this dilemma, I began to develop a friendship with an acquaintance who also happened to be a personal trainer. He opened my eyes to the gym beyond my treadmill, teaching me to use the resistance machines I always had viewed as scary and confusing. He taught me how to isolate and work certain muscle groups by lifting weights while also protecting my knees and my back.

I soon grew to love weightlifting as much as I loved running. I developed a routine that was centered around weights but still allowed me to visit my favorite treadmill every now and then.

Mind–body benefits

Exercise has both physical and
mental health benefits. Raising the heart rate burns calories and can help with weight loss and maintenance. Regular activity can increase cardiovascular fitness and helps to reduce the risk of many chronic illnesses, such as diabetes; by working the muscles, exercise helps the body become more efficient at regulating the amount of sugar in the blood.

The physical effects of exercise are crucial to longevity, but I wasn’t trying to save myself from a heart attack by going to the gym. Instead, I found the greatest benefit of exercise in its effects on mental health and mood.

Some studies suggest that mental health benefits from exercise are the result of what is known as “the distraction hypothesis,” which suggests that vigorous exercise provides a distraction from disorders such as anxiety or depression. Others state that it affects hormone levels by regulating the hypothalamic pituitary-adrenal axis. An ineffective HPA axis leads to heightened or reduced cortisol levels in the body, and voluntary exercise has been shown to regulate the release of this hormone.

I am motivated by seeing the numbers on the weight machines go up and up as I lift and by watching the miles add up as I run. This phenomenon also is backed by science. Self-efficacy is the belief in one’s ability to succeed, and a study has determined that it increases when coupled with self-set exercise goals. This same study observed decreased depressive levels in people after voluntary, consistent exercise.

Exercise is a constant in my life. It keeps me grounded no matter what else is going on. Sweating through my clothes and panting like a dog on the treadmill isn’t always easy to love, but I love the results in both my mind and body.

So whenever I find the semester getting tough, I get going — to the gym, that is.

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The five love languages are a way to think about how we communicate appreciation to those around us. Developed by Gary Chapman, a speaker and counselor who has outlined their uses in a series of books, they are:

1. Words of affirmation.
2. Quality time.
4. Receiving gifts.
5. Physical contact.

They have transformed marriages, helped adults communicate love to their children and students, and improved how people express thanks to co-workers. Don't believe me? Try asking Mr. Google, and you’ll find personal accounts extolling their authenticity. As for me, they saved my marriage and helped me become a better father and friend. In the past, I let work consume all my thoughts, time and energy. The love languages showed me how hurtful this could be to my wife and sons, who value my immediate presence. I now make it to every school performance and come home for dinner with my family. They notice and appreciate these efforts.

I believe the love languages have a place in all human relationships and should play a role in how we communicate as mentors and mentees. I’m not anything close to an expert, but I have both given and received mentorship in the sciences. I recently started my first faculty position, and as I make my transition from trainee to trainer, I want to provide examples of how the love languages might be used. My goal is to start a conversation about how they can play a positive role in the mentorship experience.

The connection between a mentor and mentee can be a multifaceted mix of our other personal and professional interactions. It resembles the relationship between a parent and child, teacher and student, and employer and employee and exists beyond age, gender and genetics. The mentor–mentee relationship therefore follows its own rules regarding communication.

As Mr. Chapman will tell you, we all express and receive the five languages in imperfect ratios. Some mean more to us than others. Speak our valued languages to us, and we feel gratitude. Starve us of these languages, and we feel malnourished. Academic science is built on the mentor–mentee relationship. The mentor and mentee therefore must use all of their skills in scientific observation to identify each other’s love language values to push the training experience forward.

Here are some examples from my experience.

Words of affirmation communicate empathy and caring. The mentor might support the mentee with celebratory compliments after achievements or encouraging words at stressful times. The mentee can say thank you after personal meetings or indicate respect by asking the mentor’s opinion on a topic or experiment. I am a father of three, and my spouse is also a working professional. Just before I started my new position, my postdoc mentor Judith Kimble told me she admired how I balance my scientific career with my family life. The thought of that comment supports me as I establish a new lab and home in Indiana.

Quality time is giving someone your complete attention. The mentor and mentee can share quality time during one-on-one meetings or when drafting manuscripts. When both make an effort to make the time productive, they show each other that their efforts are not wasted. Marvin Wickens, another mentor from my postdoc, insisted that we sit down and chat right before I left for my faculty job. The hourlong private talk was filled with reminiscing, personal commentary and professional advice. I left knowing that he believed I could succeed in this next phase of my career.

Acts of service demonstrate caring. Mentors may trust the mentee with responsibility, such as speaking at a conference on their behalf. Mentees may do a task requested by the mentor. Each might make a point of attending an event that is important
to the other. When I was in veterinary school, my research mentor, Thom- as North, attended all my summer research presentations. Many of them were cringeworthy, but he always re- marked on at least one highlight from my talk. And at that time, highlights were few and far between.

Receiving gifts can include group celebrations or sharing food. Gifts do not need to cost much to mean something. Mentees can write thank you notes to their mentors for recommendation letters. My Ph.D. mentor, Steve Harrison, bought me a Kit Kat candy bar after my first thesis committee meeting. I still buy him a Kit Kat whenever I see him.

Physical touch can be problematic in academic or professional settings. While many forms of touch may be unwanted or are unacceptable in the workplace, both mentors and mentees can work to make eye contact and exchange a handshake or a smile in the hallway. During my early days in graduate school, I worked closely with Ethan Settembre, a postdoc with Harrison, before he left for a job in industry. He came back for my thesis defense and shook my hand, an act that confirmed I had become a full-fledged scientist.

Every scientist has mentorship stories that are infused with the love languages. When I have stormy days, I look back on the events described above. I hope to share similar experiences with my mentees and future mentors in this next phase of my career.

Scott Aoki (staoki@iu.edu) grew up in Hawaii, earned a veterinary degree from the University of California, Davis, obtained a Ph.D. at Harvard University and did his postdoc work at the University of Wisconsin–Madison. He is an assistant professor of biochemistry and molecular biology at the Indiana University School of Medicine.

**Immunotherapy drug improves survival in children with relapsed B-ALL**

Acute lymphoblastic leukemia is the most common cancer diagnosed in children. At the American Society of Hematology annual meeting, researchers reported that blinatumomab, an immunotherapy drug, yielded such positive results during a clinical trial that it supports blinatumomab as a new standard of care in the course of treating children who are diagnosed with relapsed B-acute lymphoblastic leukemia, known as B-ALL. A press release from the ASH explains that during the clinical trial, which was led by the Children’s Oncology Group, part of the National Cancer Institute, pediatric patients who received two months of blinatumomab therapy following one month of standard chemotherapy treatment showed a significantly higher rate of disease-free survival and ability to proceed to a bone marrow transplant than those who received two additional months of chemotherapy.
As I approached age 58 in 2010, I realized I was entering a zone full of health dangers, including heart attacks, cancer, Alzheimer’s disease, diabetes, osteoporosis, obesity and depression. I read a lot of scientific literature, and I always look for tips for healthy living and good aging. I learned that one intervention helps with all of these diseases — exercise.

But how much exercise is optimal? I’d played tennis once a week for 40 years. Was that enough? My family doctor said no. Most health experts recommend a 30- to 60-minute daily routine, five days a week. I decided to take matters into my own hands and start exercising more frequently and more rigorously.

The treadmill regimen
I live in a 17th-floor condominium apartment with a large bedroom and windows all around. My building has a fully equipped exercise room. Exercising in common areas has advantages such as motivation from others, but the disadvantages can include wasted transit time and no privacy. I decided to buy my own treadmill. My wife predicted it soon would become an expensive trouser and shirt hanger.

I also bought a flat-screen tablet and high-quality speakers. I am a music lover. I have access to more than 200,000 songs (including a huge collection of Greek music), a Spotify subscription and unlimited YouTube access. I play the music through my speakers — I hate headphones when exercising.

My doctor suggested I start with 30 minutes of exercise per day, aiming to burn about 300 calories. I decided to more than double his suggestion, hoping for more benefit. I created a 70-minute program (including warmup and cool-down). My wife predicted that I would last a few days. How on earth could somebody my age be on a treadmill five days a week, 70 minutes at a time?

My regimen includes five minutes of warmup and 55 minutes of intense workout (fast walking, running and climbing on a slope), followed by about 10 minutes of slowing and cooling down. But this is not written in stone. I modify the program to fit my desire and mood, which vary daily. I am usually on the treadmill around 5:30 a.m. with the aim of finishing by 7, taking a shower and then walking to work. Following this protocol five days a week is more of a mental than a physical battle; early in the morning, my mind questions my ability. To overcome this obstacle, I’ve persuaded myself that this daily routine is not negotiable. That made everything easier.

To sustain the routine, I also have discovered that I must transform it from a punishment into a party.

The party and the data
The party starts with music transmitted from my phone or iPod to the speakers. Next, I turn on my tablet and watch videos of my liking, in most cases without audio. The combination of music and the visual effects creates a fantastic morning party atmosphere. When I sing, my poor wife shouts, “Shut up — it’s too early.”

Mondays, after two days off, are the most difficult. However, I have come up with a helpful strategy. Whatever I achieve on Monday in terms of time on treadmill, distance and calories burned I take as a baseline, even if the numbers are poor. I then am motivated to improve each day to have the best run of the week on Friday.

A motivating factor of this routine is my meticulous record-keeping of various parameters, including time spent on treadmill, distance covered and calories burned. I look forward to entering these data into Excel after my workout. I then can compare my current numbers to the previous day or any other day. Other tabs on the file summarize weekly output, monthly averages and yearly total. On Dec. 31, I compare the year’s numbers to the previous years’ to see where I stand.

Though they are not impressive in terms of athletic performance, I am proud of these numbers. I have walked or run 13,000 kilometers on
a treadmill (farther than the distance from Toronto to Beijing) and burned 1 million calories (equivalent to about 2,000 Big Macs). I’ve covered the equivalent of 300-plus marathons.

Benefits and a bed
I started exercising rigorously at age 58. Since then, I feel better and healthier, I am more energetic and confident, and I maintain my weight. But the health benefits might no longer be the major reason I exercise. My best time of day is sometimes my early party time. I’m not sure how long I’ll be able to do this, but I have no plans to stop. However, at 67, I’m not optimistic that my numbers will improve; my metrics show a definite decline, most likely due to aging. I am not worried; this is expected.

I have noticed no drawbacks with this activity except that my wife might be jealous of my treadmill. One day, she measured the distance between me and her in our king-size bedroom and the distance between me and the treadmill and found that the latter is shorter.

The proximity to my bed means the treadmill could function as an emergency cot if my wife ever kicks me off our bed. All I need is a good pillow, and I can start claiming much more than the 10 full days on a treadmill per year that my statistics show. I doubt that sleeping on a treadmill will evoke any health-related benefits. But you never know. This experiment has not been done yet, but it is clearly within my reach.

Eleftherios P. Diamandis (eleftherios.diamandis@sinahealthsystem.ca) is the chair in prostate cancer biomarkers and head of clinical biochemistry at Mount Sinai Hospital and University Health Network and professor in the department of laboratory medicine and pathobiology at the University of Toronto.

Eleftherios Diamandis and his treadmill in November 2019. He has been working out on the treadmill for almost a decade and keeps detailed records. See a sample at asbmb.org/asbmbtoday.
HOW VOLUNTEERING ENRICHED MY LIFE AS A PH.D. STUDENT

By Jessica Desamero

At a recent Saturday Science outreach event, we focused on teaching children what the brain is and how it works. At my assigned station, I presented several human skulls and a sheep skull to show what protects the brain and how skulls differ among species.

"Whoa, that skull is real?" "That's so cool!" "Can I touch it?" Most of the children were amazed to see real skulls up close and be able to touch them. I invited them to knock on the top of the skulls, and they were surprised at how hard and strong the skulls were. Some of them knocked softly on their own heads to compare.

Others wanted to know more. Sometimes I simply laid out the skulls and asked the children what they saw without giving them any information. They noticed that one skull was different from the others and pointed out some of these differences. Some had fun predicting what type of animal that one skull belonged to. Was it a deer? A horse? A fox? No, but they were all thoughtful guesses.

Seeing children so excited about science gave me a warm feeling. I've volunteered at 11 science outreach events so far, and I see this excitement every time. We might invite children to observe insects on a microscope or use markers, coffee filter strips and a cup of water to perform a simple version of a mixture separation experiment. The activities are designed to spark interest in various scientific fields, and judging by the reactions, it's working.

So why did I start science outreach in the first place? I did volunteer work in high school to fulfill community service requirements, but it was nothing special, just something I had to do. It wasn't until I started volunteering again in my fourth year of graduate school that I found its value.

At one point in that fourth year, I realized I was going to graduate in one or two years and I hadn't done much outside of research. I thought about where I could apply my expertise, and one possibility was teaching science to the public. I did numerous Google searches and eventually came across the term "science outreach." I did one last search for "science outreach organizations in NYC" and discovered BioBus and World Science Festival, the two organizations I now volunteer with. I recognized the name BioBus and remembered that a professor I knew from my school was a board member. This connection made me want to apply and try this organization out, and I haven't looked back since.

You might have other questions: With all the teaching and research for my Ph.D. program, how do I have time for this? And from my fellow introverts, why add another anxiety-inducing experience?

To answer the first question, BioBus sends out emails about their scheduled events and programs; if I would like to volunteer at an event, I reply with my interest. I cannot attend any weekday events, but several are scheduled on weekends, and I go to these. A long-term commitment is needed for a few of their programs, but, in general, I can participate as often as I like. Volunteering adds another task to my plate, but with time management and flexibility, it is doable.

To answer the second question, I am normally shy and quiet, so I was out of my comfort zone at my first volunteer event. Teaching college students is hard, but I had gotten used to it. Communicating to a younger audience, though? That's a whole other story. How would I get children interested in the science? How would I get parents to trust that what I'm saying will help their kids learn?

That first time was rough. I probably failed miserably at both of those objectives. Children came to my station and initially were amused at all the interesting objects in front of them. But after a few sentences of my flustered and monotone attempt at explaining concepts, they either stared at me in confusion or lost interest and asked their parents if they could move on. Parents couldn't hear me and asked me to speak up. From their faces, I could tell they doubted my ability to communicate.

Part of me wasn't sure I could do it again. But another part of me

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wanted to try, so I did. As I kept doing it, it got easier. I still get nervous, and I still mumble or speak too softly at times, but it’s not as bad as before. I’ve even gained a bit of confidence.

In surprising ways, volunteering has helped me continue in my graduate studies. As all current and former Ph.D. students know, grad school isn’t easy. From failed experiments to weak presentations, I sometimes doubt myself as a researcher. Science outreach boosts my spirits in these times of self-doubt. Every time I can answer a child’s question or educate a parent, I am reassured in my abilities, and I can say again, “Yes, I am a scientist.”

I’m glad I decided to volunteer. I feel fulfilled by helping people learn about a field I’m passionate about. And volunteering helps my overall mental well-being. After spending years immersed in my research, I find it refreshing to relearn other scientific topics and share this knowledge. Teaching these children reaffirms my love of science, and it reminds me of why I do all this in the first place.

It may not be for everyone, but for me, volunteering is indispensable.

Jessica Desamero
(jdesamero@gradcenter.cuny.edu) is a fifth-year graduate student in the City University of New York’s biochemistry Ph.D. program and volunteers with two science outreach organizations, BioBus and World Science Festival. Follow her on Twitter @JessicaDesamero.

A smiling balloon face welcomes children to a recent Saturday Science outreach event in New York where Jessica Desamero was a volunteer.

**PULSE POINTS**

Report: High school, middle school students most likely to use e-cigarettes

The 2019 National Youth Tobacco Survey, or NYTS, which is conducted annually by the U.S. Centers for Disease Control and Prevention and the U.S. Food and Drug Administration, showed that 5.3 million U.S. middle and high school students had used e-cigarettes in the past 30 days. According to the CDC, this is the sixth year in a row that e-cigarettes were the most commonly used tobacco product reported in the NYTS. One of the components measured by the NYTS is knowledge and attitudes regarding tobacco use. The 2019 survey found that students who reported trying e-cigarettes cited these as the three top reasons for use: “I was curious about them” (55.3%), ‘friend or family member used them’ (30.8%), and ‘they are available in flavors, such as mint, candy, fruit, or chocolate’ (22.4%).

![Bar chart](chart.png)

This bar chart depicts 2019 tobacco product use among high school students.
Craig Cameron, who long has worked to increase minority representation within professional societies, is now a Journal of Biological Chemistry associate editor.

By John Arnst

Craig Cameron hadn’t planned to become a biochemist. Then he found himself caring for people with AIDS. As an undergraduate at Howard University, Cameron volunteered at a hospital in the Washington, D.C., area during the height of the AIDS crisis, an experience that led him to graduate work with RNA viruses at Case Western Reserve University and a career investigating viral dynamics. At Pennsylvania State University, his lab used poliovirus and hepatitis C virus to examine nucleotide incorporation by the RNA-dependent RNA polymerase during viral replication, a mechanism essential for developing antiviral therapies. He recently relocated to the University of North Carolina at Chapel Hill to take a position as chair of the department of microbiology and immunology.

A member of the editorial board of the Journal of Biological Chemistry since 2003, Cameron was named a JBC associate editor in early 2018. He also served as chair of the Minority Affairs Committee at the American Society for Biochemistry and Molecular Biology from 2008 to 2011; before that, he was a committee member for more than a decade. Last summer, he was elected president of the American Society for Virology, a term he will begin in June.

Cameron spoke with John Arnst, an ASBMB Today science writer, about his work. The interview has been edited for clarity and length.

You recently moved from Penn State to UNC to chair the department of microbiology and immunology. What prompted the move?

I was at Penn State for 25 years — three years as a postdoc and 22 years on the faculty of the biochemistry and molecular biology department. I decided to move primarily because I was interested in doing more translational research. We’ve studied basic mechanisms of viruses for 20 plus years, but we have been recently more interested in trying to understand between-individual differences in the variability and outcomes of infection — you and I could both be in the same room with someone who has the common cold virus, and we won’t necessarily both get it. Some of that is related to our genetic differences. Here at UNC, there are people and research cores that enable looking at that more closely.

So you’re interested in looking at this translational work, how our genetics affect things like catching the common cold. How did you get started working on RNA viruses?

I was an undergraduate right in the thick of the AIDS epidemic, at a time before HIV had been discovered. I was contemplating going to medical school, doing volunteer work in local hospitals. No matter what you were doing, you saw individuals suffering from complication of AIDS-related disease. Before I
graduated, it was clear that HIV was a retrovirus, so I decided I wanted to study retroviruses.

We now have so many therapeutics for HIV. One reason we were able to move so rapidly was that a lot of people were studying the avian retroviruses — for example, Rous sarcoma virus. (Author’s note: RSV, which causes sarcomas in chickens, was the first oncovirus to be discovered.) At Case Western, people were studying these avian tumor viruses either because they were interested in the oncogenes or because they were interested in basic molecular details of replication of retroviruses. There weren’t a lot of centers for HIV at the time.

My thesis project was related to discovery of mutations that might occur in the retroviral genome in response to treatment with inhibitors, antiretroviral agents. The question was: Will resistance develop? So we devised a strategy to isolate mutants and make predictions about mutation that would cause resistance. And in doing so, we had a biochemical component and a biological component, and it was that biochemical component that led me to do a postdoc at Penn State studying enzymes.

You ended up staying at that institution for two decades. What was the environment like?

It was great. I went to Howard University as an undergrad, and while there were sports, there’s nothing like Division I sports. I had never been in a culture where you have tailgating and all the enthusiasm where, come football season, the whole weekend is devoted to the team.

The first Penn State game I went to, I felt like I was going into some temple where you could feel the energy and wonderful thoughts from everybody. A lot of life revolves around that in terms of the social aspects of being in Happy Valley. (Author’s note: This nickname for the Penn State area is derived from the fact that the area was not hard hit by the Great Depression.)

“I was an undergraduate right in the thick of the AIDS epidemic, at a time before HIV had been discovered.”

From a scientific point of view, it was also great. I worked for Steve Benkovic, who is now in his 80s, still running a lab, having started companies. He’s one of the most intense, energetic scientists I know. And he established an environment where there were maybe 15, 20 postdocs at one time. It was like a mini department.

Everyone did something different, came from a different
environment. But for the most part, everyone was friendly. So I made a lot of lifelong friends and collaborators. Through journal clubs and events like that, I came to know a lot of the faculty, including the chair of the department, which provided an opportunity for me to interview there for a position.

That sounds like a phenomenal environment. When you were younger, did you know you wanted to be a scientist?

I did have chemistry sets when I was growing up, but I really wanted to be a teacher. Any opportunity I got to put a group of my cousins together and teach them something, I would.

The transition to science came from an interest in medicine. I had an aunt who had cancer when I was in junior high school. Because she was in some clinical trials, I became more aware of the process of drug development and discovery. So, at Howard, I majored in chemistry, since I was told that a chemistry major with a math minor would stand out in the pool of applicants for medical school.

At what stage, between Case and your years at Penn, did you become involved with JBC and the ASBMB?

I came from an environment that had a really rich history in terms of the ASBMB and JBC. Richard Hanson, a former president of the ASBMB, was at Case Western in the department of biochemistry, and Harland Wood was very active in the ASBMB. (Author’s note: Hanson, who also served as a JBC associate editor, died in 2014; Wood died in 1991.) We all published in JBC, and every year for the ASBMB meeting, the whole department essentially shut down because everyone went.

And you chaired the Minority Affairs Committee a little while back.

I was a member of the MAC for many, many years before I became chair. A lot of that was Judy Bond, who was at Penn State College of Medicine in Hershey. (Author’s note: Judith S. Bond was president of the ASBMB from 2004 to 2006 and served as a JBC associate editor.) I knew her because we served in some joint graduate programs together, and one of her students was co-mentored by my postdoctoral mentor. Once I joined the faculty at Penn State, she was among the first to get me involved in committees, including an ad hoc membership committee.
Now that it’s been some time since you served as chair, what were some of your proudest accomplishments from your time on the MAC?

I think the biggest contribution that I made was to bring people on the committee that have been sustaining members of the MAC, including Squire, Sonia, Takita and Regina. All of these people came in under my leadership and stayed on and developed different, synergistic activities within the ASBMB that had a clear impact on the way diversity initiatives were embraced. (Author’s note: Squire J. Booker succeeded Cameron as chair of the MAC, which now is chaired by Sonia C. Flores; Takita Felder Sumter is a voting member of the ASBMB’s Governance Council; Regina Stevens-Truss is a former member of the MAC who chaired the ASBMB HOPES project.)

Professional development continues to be a very important aspect of the MAC’s mission. One example is the IMAGE grant writing workshop that was initiated under Squire Booker’s leadership, which has had a huge impact on the success rate of participating faculty.

The ASBMB has been a leader in minority affairs. I am president-elect of the American Society for Virology, and we had a council meeting over the summer where we were able to arrange a call with Barbara (Author’s note: Barbara Gordon is the executive director of the ASBMB) and Sonia and Squire so that they could share with us the successes of the ASBMB so we could start bringing those initiatives to the ASV.

And you continue to be involved with the ASBMB in new ways. How has your role at JBC been going, especially in the wake of the move?

At Penn State, I would teach a fall course of general biochemistry to 150 students. When you’re trying to deploy such a course and be innovative in your teaching, with both the pedagogical approaches and the technology in the classroom, it takes a lot of time. By taking that off my plate, I have a lot more time than ever before.

I look at my inbox in the morning, and if there’s a JBC paper to be assigned, I’ll usually do it while I’m having my coffee. Then, if reviews have returned, I’ll deal with it after dinner while I’m having a glass of wine. I wake up to it and go to bed with it, but as long as you stay on top of it, it’s not overwhelming, right?

That sounds like a good way to handle it. What do you like to do when you’re not reviewing papers or working in the lab?

I enjoy traveling, but one of the things that I can do locally and that I’ve been doing for many years now is cooking. I’m a biochemist; I love to cook things in the lab. And in the kitchen, you really have to keep a good notebook and weigh everything out.

So I try to develop good recipes for meals that I’ve had that have left a positive impact or impression on me.

What’s your favorite meal that you were able to re-create in the last year or so?

There’s this mushroom bisque that I had a couple years ago, but it took a long time for me to get the right combination of mushrooms because it wasn’t clear which mushrooms were used. I tried a lot of different things, like mushroom soup plus cream or water to make it denser. But eventually I got it.

That sounds delicious. One last question — do you have any advice or words of wisdom for young scientists?

I try to tell my students they first need to make sure that what they’re doing is exciting to them. Just because you enter a lab doesn’t mean you need to stay in the lab, right? You need to follow your interest. In my lab, I would say most of the students stay, but being an honors adviser at Penn State, I have seen a lot of circumstances where people try to make the research fit their personality, and it doesn’t always work.

Just having students engaged in the process of discovery resonates with them. And it’s also exciting for their mentors. I always match undergraduate students with either graduate students or postdocs, and it’s exciting for me to watch the graduate students and postdocs light up from the excitement and the joy of the undergraduates that they’re mentoring when they learn something new or see something for the first time.

John Arnst (jarnst@asbmb.org) is ASBMB Today’s science writer. Follow him on Twitter @arnstjohn.
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Renew your membership today at asbmb.org/membership.
Workshops, networking events and more
A preview of offerings at the 2020 ASBMB Annual Meeting

With three months to go, the committees, staff and members of the American Society for Biochemistry and Molecular Biology are putting the finishing touches on the programming and activities at the 2020 annual meeting April 4–7 in San Diego.

Just in case you need a nudge to hit that Jan. 30 deadline for last-chance scientific and outreach activity abstracts or the Feb. 5 early registration deadline, here’s a preview of the great stuff we have on tap.

More details are available at asbmb.org/meeting2020.

A workshop sampler

Mentoring from both sides: How to find, be and utilize a great mentor | 6 p.m. April 5

Mentoring should not be a scary or imposing concept — it’s really just about getting and giving advice, support and encouragement for ongoing learning. Anyone at any stage can be both mentored and mentoring at the same time. In fact, one of the most effective, supportive mentoring models involves peer mentoring groups. I am proud to be seeding mentoring groups all over the country, and I am happy to advise any groups of postdocs, grad students or young faculty who want to get a group started.

At every mentoring program I have participated in, invariably, the mentors tell me they get even more out of it than the mentees. So join me for the workshop “Mentoring from both sides: How to find, be and utilize a great mentor” to hone your skills and learn best practices. Make it your goal for the year to find someone to mentor.

My workshop is intended for trainees and faculty ready to learn practical tactics in identifying mentors, asking for mentoring support and taking the best advantage of mentoring relationships — from both sides.

— Joanne Kamens

Emerging technologies in the glycosciences | 6 p.m. April 5

Sugars, carbohydrates, saccharides and glycoconjugates, collectively known as glycans, permeate every kingdom of life, where they play critical structural and functional roles.

In the workshop “Emerging technologies in the glycosciences,” our goal is to promote the study of glycans, enabling participants to develop a more comprehensive understanding of the roles that glycans play in physiology and disease. Join us at the ASBMB annual meeting on the
evening of April 5 to meet colleagues, learn about innovative solutions to glycoscience problems, interact with vendors and participate in roundtable discussions focused on addressing glycoscience challenges.

This workshop is ideal for experts, researchers new to the field and trainees. Presenters include Richard Drake, Kamil Godula, Catherine Grimes, Matt Pratt, Ajit Varki, Lance Wells and Natasha Zachara. Topics covered include approaches for glycoprotein engineering, glycomic and glycoproteomic approaches, the detection and analysis of sialic acids, modulation and detection of O-GlcNAc, tools for studying the bacterial cell wall, synthetic glycoconjugates for fine-tuning cell fate and educational opportunities.

— Natasha Zachara & Catherine Grimes

Engaging the next generation of biochemistry students | 5:45 p.m. April 6

Does it frustrate you that your students take away so little from your biochemistry class? Do you want to promote depth of understanding but feel that you just have no time to innovate?

We did too. We got frustrated. Then we got funded. We used the funding to make a comprehensive suite of modules that are demonstrated to improve educational outcomes in two core undergraduate biochemistry concepts. The first concept focuses on helping students build accurate mental models of 3D macromolecular structures from 2D images. The second concept centers on systems and dynamics — how metabolism works not only in discrete modules (e.g., glycolysis, citric acid cycle and electron transport chain) but also as part of larger and dynamic interconnected systems within cells and organisms.

We did it for all instructors, especially those who (like us) are busy and may not have time to try to incorporate the latest and greatest teaching approaches in their classrooms. So our modules come with everything: interactive activities that can be used in the classroom, as homework, or in the lab; lecture slides; clicker questions; and assessment questions. We also suggest best practices and even an assessment to tell how effective the modules are in your classroom.

You can do this. We can help. Let us help you at this year’s ASBMB meeting. You’ll do two of the modules, and you’ll see data for the effectiveness of all eight of them. We’ll also feed you.

— Rebecca Roston, Tomas Helikar & Brian Couch

Education and professional development

Reimagining STEM: Who we are and what we do | 9:30 a.m. April 5 and 9:15 a.m. April 6

We lose the potential for new ideas and new perspectives when our training caters to a particular type of person. Diversifying our science, technology, engineering and math workforce demands that we rightfully challenge the norms for how we structure our classrooms, our instructional laboratories and our organizational cultures. The ASBMB Education and Professional Development Committee is sponsoring two sessions at the annual meeting on these matters.

In the first session, “Who we are: Creating a culture of wellness in science,” we will challenge the biochemistry and molecular biology community to examine who we are. Who’s missing? Where do they go, and why? What barriers do we erect in our educational and training spaces that discourage talented individuals and impel them to pursue a career elsewhere? How do these issues impact self-care? We will ask attendees to confront their own shortcomings and consider new ways to make a career in
STEM accessible to all.

In our second session, “What we do: Choosing pedagogy over content,” we will challenge the biochemistry and molecular biology community to examine what we do in the classroom. Why do we teach and train in the ways that we do? Do we have evidence to justify our choices? Are there better ways that reach a broader swath of students? Do our educational and training spaces merely allow students to survive, or are we building capacity for students from a variety of backgrounds to thrive?

Advances in science and technology require collaboration and creativity. When we build structures that establish a diverse and inclusive workforce, we deepen the creative pool, fostering innovation and progress. Please join us for discussions that will challenge and inspire us to best serve tomorrow’s leaders.

— Daniel Dries & Nathan Vanderford

Women in BMB

Leadership awards and networking dinner | Evening of April 5

The ASBMB Women in Biochemistry and Molecular Biology Committee, or WiBMB, formed at the 2019 annual meeting, will sponsor the women’s networking event at the 2020 meeting in San Diego. The ASBMB Council has approved two awards to be presented at this event.

These annual ASBMB Leadership Awards will recognize individuals with a strong commitment to advancing the careers of women in biochemistry and molecular biology along with demonstrated excellence in research, discovery and/or service. The Early-Career Leadership Award will honor an assistant professor, associate professor or equivalent and with no more than 10 years of experience since receiving a Ph.D. and/or M.D. The Mid-Career Leadership Award will recognize those at the full-professor or senior-scientist level.

Both nominees and nominators must be members. Self-nominations are encouraged. Submit your nomination at asbmb.org/awards/leadership. The deadline is Feb. 1.

Sharon Milgram, director of the Office of Intramural Training and Education at the National Institutes of Health, will speak about becoming a resilient scientist at the networking dinner.

— Susan Baserga
Publications

Data stewardship discussion, early-career investigators and award winners | Various times and dates

Research outputs are becoming increasingly complex. Ensuring that your research data will be usable in a meaningful way when you write a proposal or paper requires good data stewardship practices. Good data stewardship isn’t just about saving your data; it’s also about archiving it carefully so that others can use it in the future. It also applies to protocols and data analysis. How do you navigate all these topics and develop good habits to ensure that your data is useful in the future? How do you also handle these issues when research is more often than not a collaborative process? Kaoru Sakabe, the data integrity manager for the ASBMB, and Catherine Goodman, the scientific editor for the Journal of Biological Chemistry, will lead a panel discussion with researchers on how they tackle these important concerns.

Each ASBMB journal also will sponsor talks at the annual meeting. The Journal of Biological Chemistry will present five winners of the JBC Herbert Tabor Early Career Investigator Award, which honors first authors of papers published in the JBC during the past year. The Journal of Lipid Research will host talks by its four junior associate editors, early-career investigators who have distinguished themselves in the field of lipid research, on the theme of lipid diversity and disease. Molecular & Cellular Proteomics will host talks by three researchers on exciting biological insights revealed by proteomics.

Finally, two ASBMB award winners also are associated with the Journal of Lipid Research. Jean Schaffer, winner of the Avanti Award in Lipids, is an associate editor and Ed Dennis, winner of the Bert and Natalie Vallee Award, is a former editor-in-chief. Put their award lectures on your to-do list.

— Kaoru Sakabe & Comfort Dorn

Science outreach and communication

Poster session, hands-on workshops and flash talks | Various times and dates

Are you interested in sharing your science with students, community members, policymakers or even potential funders? Whether you’re just starting out or a seasoned professional, the ASBMB’s Science Outreach and Communication Committee will offer several workshops and activities at the annual meeting that can help you find inspiration, hone your skills, and connect with others engaged in science outreach and communication.

Learn more about science outreach initiatives run by ASBMB members from around the globe at the science outreach poster session during the opening reception on April 4. Abstract submission for this session is free and open until Jan. 30. Submit an abstract at asbmb.org/meeting2020/abstracts.

If you’d like to develop your own outreach activity, participate in a hands-on workshop, “Transforming research into outreach,” on April 6.

Do you want to focus on your communication skills? Learn how to engage your audience at “Storytelling and the art of giving a great presentation” on April 5.

Want to share your science but find that you’re short on time? Graduate students and postdoctoral travel awardees can learn how to construct an elevator pitch on April 4. Or you might consider participating in the ASBMB Student Flash Talk Science Communication Competition on April 6. Ten finalists will have just three minutes and one slide to convey the essence of their research to a nonexpert audience.

— Nicole Woitowich
Minority affairs

Sessions on the rules of life and workplace harassment, plus a networking reception | Various times and dates

The ASBMB Minority Affairs Committee’s mission is to increase cultural diversity in the fields of biochemistry and molecular biology. This year the committee will present the session “Understanding the rules of life,” with speakers who use computational, modeling, biochemistry and molecular biology approaches to capture dynamic data, analyze changes over time, and make predictions about responses and behaviors.

You’re also invited to take part in the special session titled “Best practices for preventing/managing incidences of harassment in the workplace.”

Finally, join the MAC at the ASBMB welcome reception to network with other members and learn about programs within the society. MAC programs include the annual Interactive Mentoring Activities for Grantsmanship Enhancement grant writing workshop and the Marion B. Sewer Distinguished Undergraduate Scholarship. MAC travel awardees will present their research in a poster session during the reception. Come mix and mingle with fellow scientists interested in supporting diversity and inclusion in BMB.

—Stephanie Paxson

Public affairs and science advocacy

Annual town hall | 12:15 p.m. April 5

Policies developed and enacted in Washington, D.C., have an impact on how your science is funded, how your grants are reviewed and how reliable the future workforce of the research community will be.

Join us for the ASBMB Public Affairs Advisory Committee annual advocacy town hall to hear from policy experts about critical funding and policy discussions that will directly impact your lab and the wider research community.

Public Affairs Director Benjamin Corb and Terri Kinzy, chair of the PAAC and vice president for research at Western Michigan University, will discuss the committee’s activities over the past year and how the society represents its members’ needs to policymakers on Capitol Hill and in the White House and within the federal agencies that fund your science.

Participants are encouraged to ask questions about public policy and share their stories and perspectives. Let us know how the ASBMB’s policy efforts best can represent you and your needs.

In addition, as we did last year, the Public Affairs Advisory Committee will schedule opportunities for ASBMB members to meet with program officers from the National Institutes of Health and other federal agencies. Members will have a chance to ask questions about grant writing and learn about these agencies’ funding opportunities.

—Benjamin Corb

Student Chapters

Workshop on starting a chapter and networking reception | 6 p.m. and 7 p.m. April 5

The ASBMB Student Chapters program is devoted to building a community of undergraduate students and faculty members through networking, science outreach and career-development opportunities. The Student Chapters Steering Committee will host two networking events at the ASBMB annual meeting for our members and those interested in joining the program.

The first event, a workshop titled “Organizing a successful ASBMB Student Chapter,” is part of the ASBMB
undergraduate programming. Members of Student Chapters and the regional directors will share advice and ideas for organizing chapter activities, getting involved with the society and hosting outreach events. Student and faculty members of the program and those interested in starting a chapter are encouraged to attend.

The second event is the ASBMB Student Chapter advisers’ networking reception. Chapter advisers will have an opportunity to network with the regional directors and other advisers. Faculty members who are interested in starting a Student Chapter at their institution are welcome to attend as well.

— Stephanie Paxson

Career Central
One-on-one coaching, CV reviews, mock interviews and more | Various times and dates

The five host societies at the Experimental Biology conference are teaming up once again to offer a variety of career services for attendees. Visit experimentalbiology.org to sign up for one-on-one career advice. You can work with a coach on your poster presentation, revising your CV and much more. Also check out the bulletin board with job postings, quick presentations on career topics and deep-dive workshops.
What I wish people knew about being a mother of young children while having a career in academia

By Laura Rowe

I got pregnant with twins during the last part of my postdoctoral training. The pregnancy was planned, but the two-for-one special was a shock and wreaked havoc on my detailed financial and career plans.

I had planned to work right up until I gave birth and then not work for at least six months afterward. My due date was in December, and a new academic year didn't start until August … see how perfectly we timed it? Then we would put the several-month-old baby into daycare, and I would continue in another postdoc or a tenure-track faculty position while my soon-to-be husband finished graduate school.

It all seemed great on paper, but then life gave our plans the finger. Everything I read indicated that twin pregnancies are high-risk and I'd increase my chance of a full-term pregnancy and healthy babies if I didn't work full-time, or perhaps at all, during my third trimester. So working till I gave birth was out.

My search for an academic position that year was a disaster. I went to a two-day on-site interview at a good R1 university less than a month after having twins via C-section. During my presentation, several professors asked me basic questions about the rationale of my Ph.D. work. I drew a complete blank. I had barely slept in three weeks, and since I still hadn't recovered from my C-section, I winced in pain every time I stood up, sat down, tried to walk fast or coughed. Because they had me scheduled 8 a.m. to 8 p.m. both days without any significant breaks, I couldn't pump, so my breasts felt like they were going to explode and I was leaking continuously throughout my interview. I had scheduled all my on-site interviews for academic jobs for the following year within six weeks of giving birth. Needless to say, I didn’t get offered any positions.

For more than two years, I worked either not at all or part-time as an adjunct professor at colleges and universities that were either very close to my partner while he finished graduate school or very close to my parents. I was uncomfortable leaving our babies in the care of someone who wasn’t family. My reluctance to leave them for 40-plus hours a week was a personal, emotional reaction and a result of their temperament. If I was away for more than three or four hours, one or both would scream inconsolably until I returned. Many women are comfortable going back to work full-time six weeks after their children are born. I wasn’t, and I didn’t know that until after I actually had children.

When the twins were almost two, I started a full-time, tenure-track position at a predominantly undergraduate institution in the Midwest. A year later, we found out I was pregnant again, but with only one baby this time. Thus, I was pretenure with three children under the age of five.

Fast forward four years for the Hollywood ending: I was approved for tenure, I am still happily married to the same man, and all three of our children are alive and seem pretty happy and well adjusted.

Sounds pretty good, right? Well, here are the parts I didn’t mention — that I wish someone had told me and others understood — in convenient bullet-point form.

The bad and ugly …

- The two years after having twins, even only working part-time, were the hardest of my life so far. Nothing else comes close. I suspect this is true for many women with single babies too.
- Until the twins started kindergarten, I had zero time for social life, hobbies or personal relaxation. Every moment of every day, I was doing things I had to do for other people or my job. That takes a toll.
- I chose to work at a predominantly undergraduate institution partly because I believed it would give me adequate time to raise a family. I did not believe I could achieve tenure at an R1 institution unless we let someone else do the majority of raising our children. I may have been wrong, but that is what I believed.
Our house is rarely clean, I am not a PTA member, and I severely limit our children’s extracurricular activities because I do not have the time or energy to be their evening and weekend chauffeur.

**The good-ish …**

During the academic year, I never work more than 45 hours per week, some of this from home, and during breaks I generally work less than 25 hours per week. I take six weeks completely off, four in the summer and two at Christmas time. These might seem like lazy work hours to many, but I hope it is a beacon of hope to women who want to have children and have a career in academia.

To limit my work hours, I must be extremely efficient. I do no social media or water-cooler chitchat at work, I severely limit meetings, and I close my office door for a couple of hours every morning. This is my most productive time, obviously.

If I cannot bring my children, then I say no to (almost) all evening and weekend meetings and events. My kind and understanding colleagues accepted this choice and still approved me for tenure. Not all colleagues will be this progressive, but maybe if they understood what it is like to be a mother with young children in academia, they could be?

Were the sacrifices worth it? For me, yes. Our children have secure attachments to my husband and me that hopefully will serve them well in their futures. I have a career I enjoy. I get to teach great students and do research that I love. I don’t do as much research as I’d like, and I will likely never be a leader in my field, but this is an acceptable balance for me.

A lot of female students ask me, “Can I have a family and have a career in academia in science? Can I realistically raise kids while being a professor?” Here’s what I want to tell them:

“Yes, you can, but it will be difficult when your children are young. You must decide how much time you want to spend with your family and come to terms with the reality of your biological clock and the reality of what it will take to achieve tenure. Something will have to give, because there are only so many hours in a day. You can’t be the very best in your job and the very best mother. You will have to choose what to let go of.”

For those of you who are like the young women who wander into my office to ask me such questions, I will end with more bullet points (I love the efficiency of bullet points). Here are some ways to make it easier, though still not easy, to have an academic career while raising young children:

- Find the right partner — someone who fully supports your career and is willing to be a true and equal partner when it comes to raising little ones and keeping a household. Sheryl Sandberg is correct on this, and I am extremely lucky in this respect.
- Try to live close to some extended family. I have been able to attend conferences once or twice a year only because my mom is

Laura Rowe and her family. She works at a predominantly undergraduate institution in part because she believed it would give her adequate time to raise her three children.
just a five-hour drive away. Our geographical location was not an accident; I wish we lived even closer.

- Accept that your house will not be clean for a few years, and that is OK. Better yet, hire a housekeeper (if you can afford it).
- Decide how many hours a week you will dedicate to your job and enforce it. An academic career will take as much time as you give it; it’s as easy to work 60 hours a week at a small college as it is at a big university. Say no to everything that puts you over your time allotment. Accept the consequences of your decision without guilt or shame.
- Learn to say no in a diplomatic way, and do so as often as possible. This is perhaps the most important skill to master if you want a meaningful life outside of work.
- Check and respond to email only once a day. Limit meetings as much as possible. Accept that you may have no social life or free time until your youngest starts school.
- Learn to love coffee.

In less than a year, our youngest will start kindergarten and our twins will enter third grade. This, combined with the relaxation I’ve begun to feel since being approved for tenure, leads me to believe that my future life will not need to be quite as streamlined as the past seven years. Who knows, maybe I’ll be able to focus on doing more research and publish twice a year instead of once every couple of years. Or maybe I’ll just have time to go out occasionally for drinks with friends. One can dream.

Laura Rowe (laura.rowe@valpo.edu) is an associate professor in the department of chemistry at Valparaiso University, where she teaches biochemistry, analytical chemistry and various introductory chemistry courses while leading a small research group of undergraduates.
VIRTUAL ISSUE

Cancer

jbc.org/site/vi/cancer/

2020 ASBMB–Deuel Conference on Lipids
Fat in Liver and Beyond

MARCH 3 – 6, Hotel del Coronado, Coronado, Calif.
Learn more, register and submit your abstract at
www.asbmb.org/deuelconference/
Feb. 4: Abstract submission deadline
For most of my life, I rarely stopped to think about what people did at night. As a kid with conservative parents, I was bound by nightly curfews. I assumed all families were at home together and everyone slept at night. Now I know that’s not the case. While others are sleeping, I work as an emergency medical technician at a local Level IV* trauma center.

During the day, I go from class to class, a senior in the department of chemistry and biochemistry at Stephen F. Austin State University. After class, I go to the lab. I’m a full-time student in a difficult area of study, juggling the responsibilities of academics and research. I get notification after notification from my calendar. All my friends say, “That’s a busy person.”

Many people do not know that my day begins again at night. My days and nights endlessly run together. It’s a ride that never stops going around and around, but I love it.

Before I landed my job in the emergency department, I was living in a large city attending graduate school for cellular and molecular biology. As I found my interests slowly veering away from biology and into chemistry, I decided to leave my program and return home to Nacogdoches. I was an undergraduate again, and I needed employment.

My two sisters work at the local hospital, and they were kind enough to drop my name to the director of the emergency department. When he called me for an interview, I reported to his office holding a CV of academics and research; my only related experience was as a medical scribe. After the interview, he called and said the job was mine, but I would need to earn some certifications and take a six-week orientation.

I’ve been doing the job for a year and a half now.

A shift for me begins at 1900 hours (7 p.m.). As the sun sinks and the day fades, I put on a clean pair of scrubs and my name badge and then head to the hospital. I walk through the ambulance bay doors, get a report from the day shift on what’s left for me to pick up and begin my duties.

As a tech, I assist the nurses and physicians with patient care. This typically entails drawing blood for lab work, performing electrocardiograms, hooking up patients to the monitors and cleaning rooms. Other duties include transferring patients, paging doctors and getting patients admitted to the hospital.

Personally, I love doing orthopedic splints, and the ER crew gives me every opportunity to place splints on patients who have broken a bone or two. To me, it is an art form: the correct splint placement for particular fractures mixed in with wrapping elastic bandages so they lie completely flush with one another, producing a beautiful splint.

In a shift, we see anywhere from 18 to 40 patients. A regular shift lasts a full 12 hours, but on slower nights I might get to leave early, around 4 a.m. I started as a full-time employee working what’s known as a 5:2 rotation. I’d work five days straight, have two days off, work two days, and then have five days off. I worked that rotation for a year, essentially getting a crash training. I soon realized that I could not sustain a full-time work schedule as well as school, staying awake sometimes for 30-plus hours. I now work a manageable three shifts a week.

Unlike most hospital departments, in the emergency room we never know what might roll through the doors. Most ERs have a red phone (we call it the bat phone) where we get reports from incoming ambulances. The piercing ring of the bat phone evokes simultaneous dread and curiosity. It could be a simple headache or a foreign body stuck in a sphincter. Once, it was someone who drank too much at the local bar. The individual’s ethanol did not settle well, and upon arrival they reenacted a scene from “The Exorcist,” painting the trauma bay with whiskey-soaked emesis.

Television medical dramas paint a fairly accurate portrait of the emergency department, though in rural areas, specialists and surgeons don’t freely walk into the ER to see patients. It takes something akin to an act of Congress to get the specialist to call you back after certain hours. But the ignorance about specific medical procedures on TV is sometimes cringe-worthy. I recently watched a scene where a patient had a hemotorax (accumulation of blood in the chest cavity) requiring a chest tube, but instead they placed an endotracheal tube in the pleural space. Endotracheal tubes are used in intubations to secure airways.
We have some unspoken superstitions in the ER. The No.1 rule is to never speak the words “quiet” or “slow.” These words are a hex, sending atmospheric signals out to people that they are sick enough to come to the emergency department. And full moons genuinely do bring out the crazies.

The work can be stressful. When the power doors slide open at the start of my shift, they sometimes reveal a scene like a New York City rush hour, with patients filling every bed and spilling out into the hall. The beeping and dinging of IV pumps and telemetry monitors tell me that Bed 2 has low blood pressure and that Bed 6 has finished their bolus of normal saline. As I see the numbers climb on the electronic board that tracks how many patients we’re treating and how many are in the waiting room, my stress level rises.

This work can be both scary and heartbreaking — the scary cases more often than not have heart-breaking results. A person comes in with wrenching abdominal pain and vomiting only to learn they have metastatic disease. Or someone comes in complaining of feeling unwell and suddenly goes into cardiac arrest because they have developed irreversible sepsis.

The emergency department is unpredictable, and therefore so is my night. I might walk out laughing at the end of my shift, or I may leave defeated after losing a patient. When the sun comes up, my work day is over. At home, I take a short nap before heading back to class. I try not to let the events of the night before show on my face. I fight to stay alert. I listen to lectures about symmetry and group theory in inorganic chemistry or the mechanisms of second order nucleophilic substitution reactions in organic chemistry, and the night shift fades into day.

* This designation, ranging from I to V, indicates that a hospital can provide advanced trauma life support. The level is based on the hospital’s capabilities and specialties and how much of the time these doctors are available. As a level IV trauma center, our specialties include general surgery, orthopedic surgery and cardiothoracic surgery, but our more critical patients are transferred to a Level I or Level II trauma center with 24-hour coverage of all medical specialties.

Justin Lovett preps for an incoming trauma in the emergency department at a hospital in Texas.
You're invited to join the Lipid Research Division of the ASBMB

The Lipid Research Division was born from a grassroots discussion of broad concerns shared by all lipid research scientists. These included issues such as increased national and international visibility for lipid research and increased funding for lipid research. We invite you to join this community, contribute to the ongoing discussions and help support the lipid community.

www.asbmb.org/lipidcorner
The Department of Chemistry & Biochemistry in the College of Arts and Sciences at The Ohio State University seeks to fill a tenure-track faculty position at the assistant professor level in the general area of biochemistry, broadly defined. The new faculty member will join an interdisciplinary community on the main campus in Columbus, Ohio. Our partners and outstanding facilities, including state-of-the-art cryo-electron microscopy, NMR spectroscopy, mass spectrometry and systems biology, offer numerous opportunities for collaboration and advancement of research programs.

The Ohio State University:
Assistant Professor

The Department of Chemistry and Biochemistry at the University of Arizona is seeking applicants for a full-time Career Track Lecturer to teach foundational and advanced biochemistry topics to undergraduate students with science majors, many of whom are interested in the health professions and/or graduate school. Additional courses in the Chemistry & Biochemistry Department curriculum may also be taught by them as the need arises. In addition to teaching, they will contribute to the department through service and outreach. This is an academic year (9-month) position at 1.0 FTE and is located in Tucson.

University of Arizona
Dept. of Chemistry & Biochemistry:
Lecturer (Career-Track)

Laboratoire d’Information Genomique et Structurale (IGS): Postdoctoral position in Structural Biology of Giant Viruses

A funded postdoctoral position (A*MIDEX) is available in the new team of Elsa Garcin (Professor at AMU starting in January 2020, Chaire d’Excellence A*MIDEX; Adjunct Associate Professor at UMBC) in the Laboratoire d’Information Génomique et Structurale (IGS), a CNRS/AMU laboratory located on the Luminy Campus of Aix-Marseille University. The IGS is a dynamic lab led by Chantal Abergel and Jean-Michel Claverie, world-renowned experts in giant virus research. Giant virus discovery was revolutionary for the field of virology, as they revived old debates about the concept of virus, their position in the tree of life, their biology, and the role they may have played in the emergence of life on earth. Characterizing new metabolic pathways in these viruses may therefore provide new clues about chemical and biological processes at the origin of life.

Laboratoire d’Information Genomique et Structurale (IGS): Postdoctoral position in Structural Biology of Giant Viruses

The Department of Chemistry at Tennessee Tech University invites applications for a Lecturer of General & Nursing Chemistry and General Chemistry Laboratory Coordination position. This is a 3-year, nine-month, non-tenure-track position at the rank of Lecturer to begin August 1, 2020, and is renewable based on satisfactory annual reviews, the needs of the department, and future funding. Minimum qualifications: A Ph.D. degree from an accredited institution in Chemistry or STEM Education with appropriate background in related areas. Candidates must have a demonstrated potential to excel as an educator and ability to teach both non-STEM chemistry and associated laboratory sections, as well as nursing chemistry. Preferred qualifications: Prior teaching experience beyond that of a teaching assistant (lecture and laboratory).

Tennessee Tech University, Department of Chemistry: Lecturer, General & Nursing Chemistry and General Chemistry Laboratory Coordination

To see full descriptions of these jobs and more, visit careers.asbmb.org
The ASBMB Leadership Awards recognize individuals with a strong commitment to advancing the careers of women in biochemistry and molecular biology along with demonstrated excellence in research, discovery and/or service.

The awards are being inaugurated in 2020 and will be given annually by the ASBMB Women in Biochemistry and Molecular Biology Committee.

The deadline for nominations for these awards is Jan. 31. Details are at asbmb.org/awards/leadership.