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The science of learning

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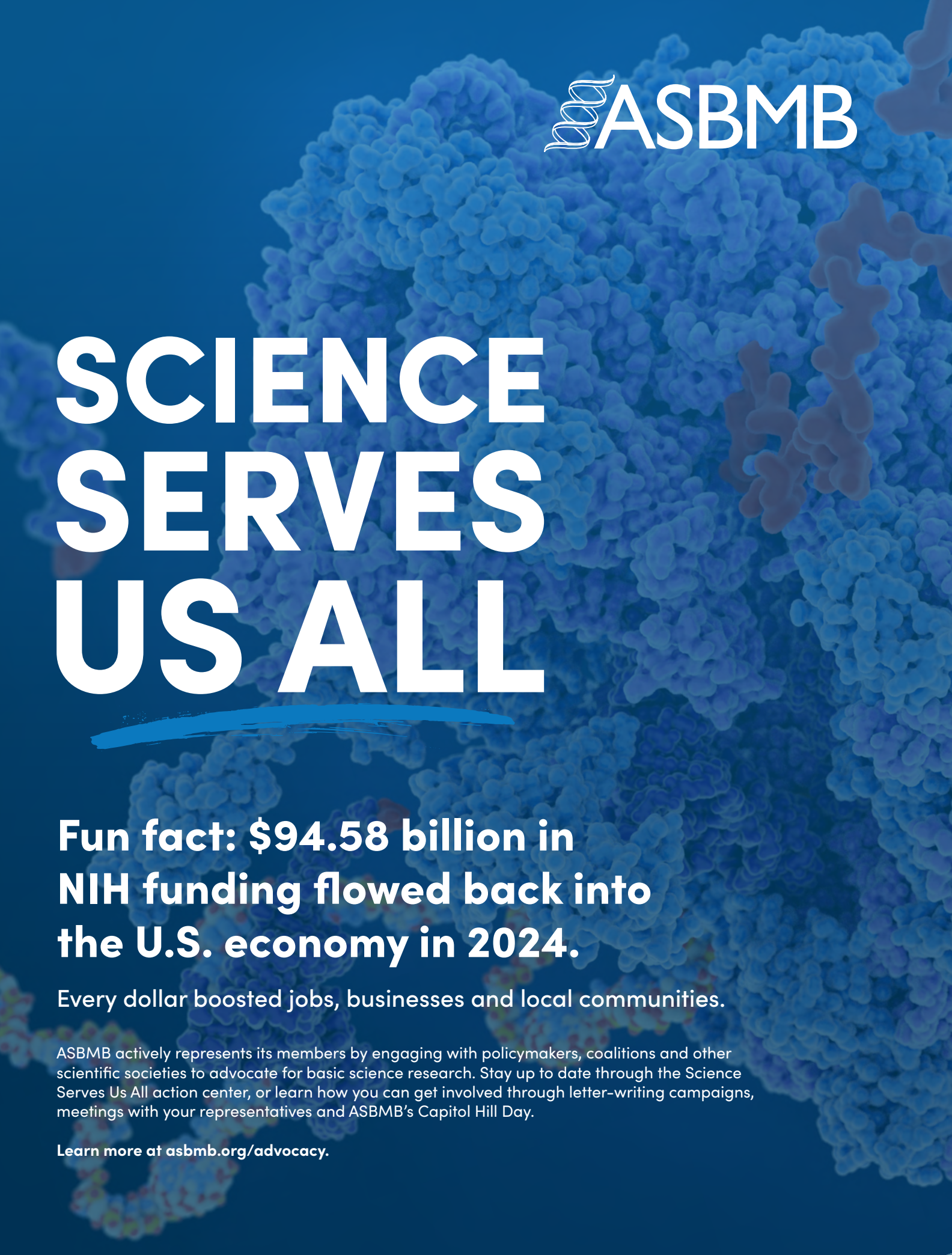
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Fun fact: $94.58 billion in NIH funding flowed back into the U.S. economy in 2024.
Every dollar boosted jobs, businesses and local communities.
ASBMB actively represents its members by engaging with policymakers, coalitions and other scientific societies to advocate for basic science research. Stay up to date through the Science Serves Us All action center, or learn how you can get involved through letter-writing campaigns, meetings with your representatives and ASBMB's Capitol Hill Day.
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ASBMBTODAY

ABOUT THE COVER: The chemical basis of memory occurs in neurons, shown here, with a calcium spark, calmodulin translation and CaMKII reinforcement. Credit: Leterrier, NeuroCyto Lab, via NIH Flickr

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By Joan Conaway

Reflecting on science, LEARNING & LEADERSHIP

As scientists, we use the scientific method every day to question, test and refine our understanding of the world. We form hypotheses, gather evidence and revise our conclusions as new data come to light. Discovery is built on curiosity and persistence, but also the humility to recognize that there's always more to learn.

Our community exemplifies this dedication to learning, and applying learning, at so many levels. Our members work across academia, industry, government and beyond, applying knowledge of biochemistry and molecular biology to understand the fundamental scientific questions that drive real-world impact.

Beyond our own research, part of our role as scientists is to enable and empower others to learn. Nearly all of us shape the next generation of leaders by serving as educators, guides and mentors at some point in our careers. Many in our community also take on the vital work of educating the next generation as a central part of their careers, striding across deeply connected worlds as scientist-educators. Effective STEM higher education cultivates critical thinking, resilience and the ability to evaluate evidence. It prepares future scientists to pursue scientific excellence with purpose and rigor. Recognizing this important work, this issue of ASBMB Today focuses on the science of learning and features reflections on teaching, innovative pedagogical practices, and the biochemistry behind learning. Rigorous science depends on strong higher education.

I would also like to take this opportunity to reflect on my time as president. In July, I will hand over the reins of the society to our president-elect, Ed Eisenstein, and step into my role as past president. My term has coincided with a time of tremendous progress at the American Society for Biochemistry and Molecular Biology alongside the unprecedented period for science and scientists.

Through it all, I have witnessed the extraordinary resilience of this community and reflect with pride on what we've achieved. We strengthened our advocacy efforts on Capitol Hill. We continued to publish rigorous, high-impact science in our journals and will also add Insights in Biochemistry and Molecular Biology to our family of journals. We supported early-career scientists, elevated member voices and reaffirmed our commitment to ensuring that biochemistry and molecular biology remain essential tools for discovery across disciplines. And we are building a pathway for a vibrant future to ASBMB with the society's strategic plan and emerging new programming.

I am deeply proud of how ASBMB members have come together to support one another, advance science and uphold the values that sustain the scientific enterprise. Thank you for the opportunity to serve this remarkable community. I am confident that under Ed's leadership, our society will continue to evolve, thrive and lead.

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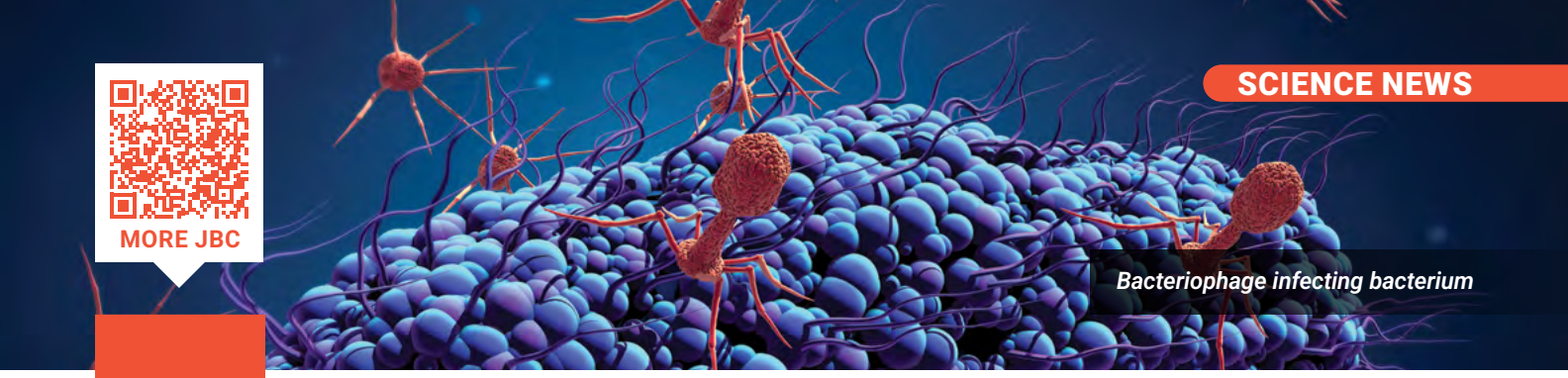
MORE JBC



Bacteriophage infecting bacterium

JBC

Bacteriophage protein could make queso fresco safer

By Anna Crysler

A slice of queso fresco might seem harmless, but in some instances, it can carry a deadly hitchhiker: *Listeria monocytogenes*. As Hispanic-style cheeses gain popularity in U.S. kitchens, scientists are working to ensure their safety. Traditional methods to curb *Listeria* have limits, so researchers are turning to an unexpected ally: viruses that hunt bacteria.

In the battle against foodborne illness, scientists are turning to an unlikely ally: bacteriophages, viruses that infect bacteria. A recent **Journal of Biological Chemistry** paper details the structure and function of PlyP100, an endolysin from bacteriophage P100.

Researchers from the labs of Michael Miller, professor of food microbiology at the University of Illinois Urbana–Champaign, and Pål Stenmark, professor of biochemistry at Stockholm University, combined their expertise in food safety and novel protein characterization to study PlyP100. Co-first authors Karson Bateman and Emma Scaletti Hutchinson said the P100 bacteriophage is already used in food production, so PlyP100 could be commercialized to prevent *Listeria* in queso fresco. PlyP100 is an endolysin, a protein bacteriophages use to break down bacterial cell walls.

"With Gram-positive bacteria, it is possible for endolysins to hydrolyze the surface exposed peptidoglycan when added exogenously which makes them an interesting tool for controlling unwanted, Gram-positive bacteria," Miller said.

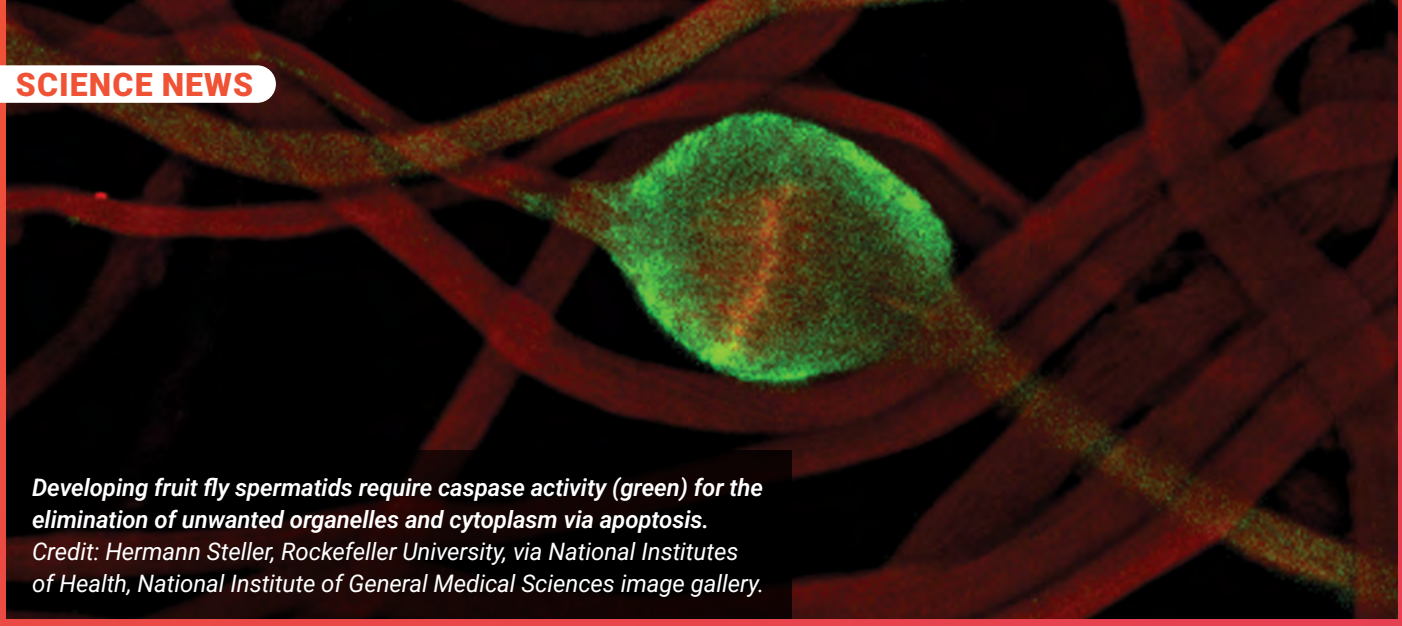
Few endolysins have been studied in detail, so identifying the best candidates for food safety remains trial and error, Bateman said. To move beyond trial and error, the team used AlphaFold to predict PlyP100's 3D structure, revealing three domains. Domain 1 proved key for hydrolytic activity, the team confirmed by testing mutated versions in lab models, including queso fresco. They found domain 3 binds *Listeria's* cell wall and discovered an unexpected role for domain 2.

"The second domain does not seem to have a discrete function, yet it is still necessary for full function of the endolysin," Scaletti said. "The importance of domain 2 was a bit of a surprise but this demonstrates why experimental validation is still needed." This structural insight could enable computer-based analysis of uncharacterized endolysins and expand their use. "We anticipate that work like this will enable analysis of uncharacterized endolysins identified in genomic sequence databases and facilitate greater application of these powerful proteins," Stenmark said.

Next, the team plans to produce PlyP100 in a GRAS, or a Generally Recognized as Safe, organism, such as yeast, to test conditions to keep it active in queso fresco and study synergy with other antimicrobials.

Food safety affects everyone, from farmers to families at the dinner table. By unlocking the secrets of PlyP100, researchers are moving beyond trial and error toward precision tools that could stop deadly outbreaks before they start. If successful, this work won't just protect queso fresco lovers — it could redefine how we safeguard the global food supply.

Anna Crysler is a Ph.D. student in bioengineering at the University of Pennsylvania and an ASBMB Today volunteer contributor.



Developing fruit fly spermatids require caspase activity (green) for the elimination of unwanted organelles and cytoplasm via apoptosis.
Credit: Hermann Steller, Rockefeller University, via National Institutes of Health, National Institute of General Medical Sciences image gallery.

How lipid metabolism shapes sperm development

By Meric Ozturk

Sperm cells are among the most specialized in the body, designed for a single purpose: fertilization. Each carries DNA and propels itself toward an egg using a whip-like tail called a flagellum. Inside the testes, sperm develop through several well-defined stages, acquiring the structures and molecules needed for fertilization.

One crucial part of this transformation is the production of seminolipids, specialized fats found in developing sperm that are essential for their formation and function. Seminolipids are synthesized by fatty acyl-CoA reductase, or FAR, enzymes. Mammals have two different FAR enzymes, FAR1 and FAR2.

Although both FAR1 and FAR2 are known to synthesize fatty alcohols, their specific roles in seminolipid production had remained unclear. To pinpoint which enzyme was responsible, Ayano Tamazawa and colleagues at Hokkaido University analyzed mice lacking *Far1* and *Far2* to clarify their roles in seminolipid production and spermatogenesis. They found that loss of *Far1* led to a dramatic decrease in seminolipids and impaired sperm development. The study was published in the **Journal of Biological Chemistry**.

Tamazawa said the findings show how the loss of seminolipids disrupts spermatogenesis, emphasizing the critical role of ether linkages in sperm development.

Seminolipids are categorized by the types of alkyl and acyl chains they contain, which differ in length and saturation. The most common seminolipid in the testis is O-C16:0/C16:0. Using liquid chromatography–tandem mass spectrometry, or LC–MS/MS, the researchers mapped the exact structure of these lipids.

High-resolution lipidomics analysis of seminolipids and SGalDAGs (3-sulfogalactosyl-1-acyl-2-acylglycerols) showed that both lipid types have similar side chains composed of saturated acyl or alkyl groups. The main difference is that SGalDAGs have a 1-acyl group, whereas seminolipids have a 1-alkyl group.

“This finding is unique because SGalDAGs have never been characterized in detail in the testis,” Tamazawa said.

However, the precise mechanisms by which seminolipids contribute to spermatogenesis, and why the C16:0/C16:0 structure predominates, remain unknown.

The researchers suggest that understanding seminolipid function could inform new diagnostic or therapeutic strategies for male infertility, potentially leading to lipid supplements or biomarkers.

Meric Ozturk is a Ph.D. student in biochemistry at Iowa State University and an ASBMB Today volunteer contributor.

Gut microbes hijack cancer pathway in high-fat diets

By Andrea Lius and Jessica Desamero

Colorectal cancer, or CRC, ranks as the third leading cause of cancer-related deaths in men and the fourth in women in the U.S. as of 2024. Rates of CRC are also rising among young adults, increasing by about 2.4% each year from 2012 to 2021. Obesity is among the risk factors driving this trend.

In a recent study, a group of researchers in the U.S. found that in mice, a high-fat diet boosts ammonia-producing bacteria in the gut microbiome, which in turn promotes CRC. The researchers discovered that this effect is mediated by the transforming growth factor beta, or TGF-β, signaling pathway. The study, led by Lopa Mishra and Krishanu Bhowmick at the Feinstein Institutes for Medical Research, appeared in the **Journal of Biological Chemistry**.

"We started with a very profound clinical question," Mishra, co-director of the Institute of Bioelectric Medicine at the Feinstein Institutes and co-corresponding author of the study, said. "Colon cancer is on the rise in younger people, and we think the microbiome and diet play a big role in this trend."

In cancer, TGF-β signaling typically suppresses tumor growth in early stages but can promote it later on. For example, Mishra's team previously showed that disrupting TGF-β signaling caused mice to develop CRC and other gastrointestinal cancers. Other studies found that TGF-β deficiency did not trigger tumors in mice lacking a microbiome, and that microbiome-induced increases in ammonia levels can promote CRC. Ammonia produced by the gut microbiome likely helps tumors outcompete healthy tissue, Mishra explained, because normal cells cannot tolerate high ammonia levels. "We showed for the first time that such a major signaling pathway as TGF-β is targeted by a microbial metabolite," she said.

Bhowmick, a postdoctoral fellow and co-corresponding author of the study, added: "Before this, whether any particular signaling pathway was involved in mediating ammonia toxicity in CRC was not clearly known."

The team found that ammonia interacts with the N-terminus of βII-spectrin, or SPTBN1, a downstream component of the TGF-β pathway. Normally, SPTBN1 interacts with SMAD3, another downstream effector, in the nucleus to promote genes linked to tumor suppression. Ammonia disrupts this interaction, trapping SMAD3 at the cell membrane and in the cytoplasm. When researchers depleted SPTBN1 with small interfering RNAs, SMAD3 activation, nuclear localization and tumor suppression functions were restored.

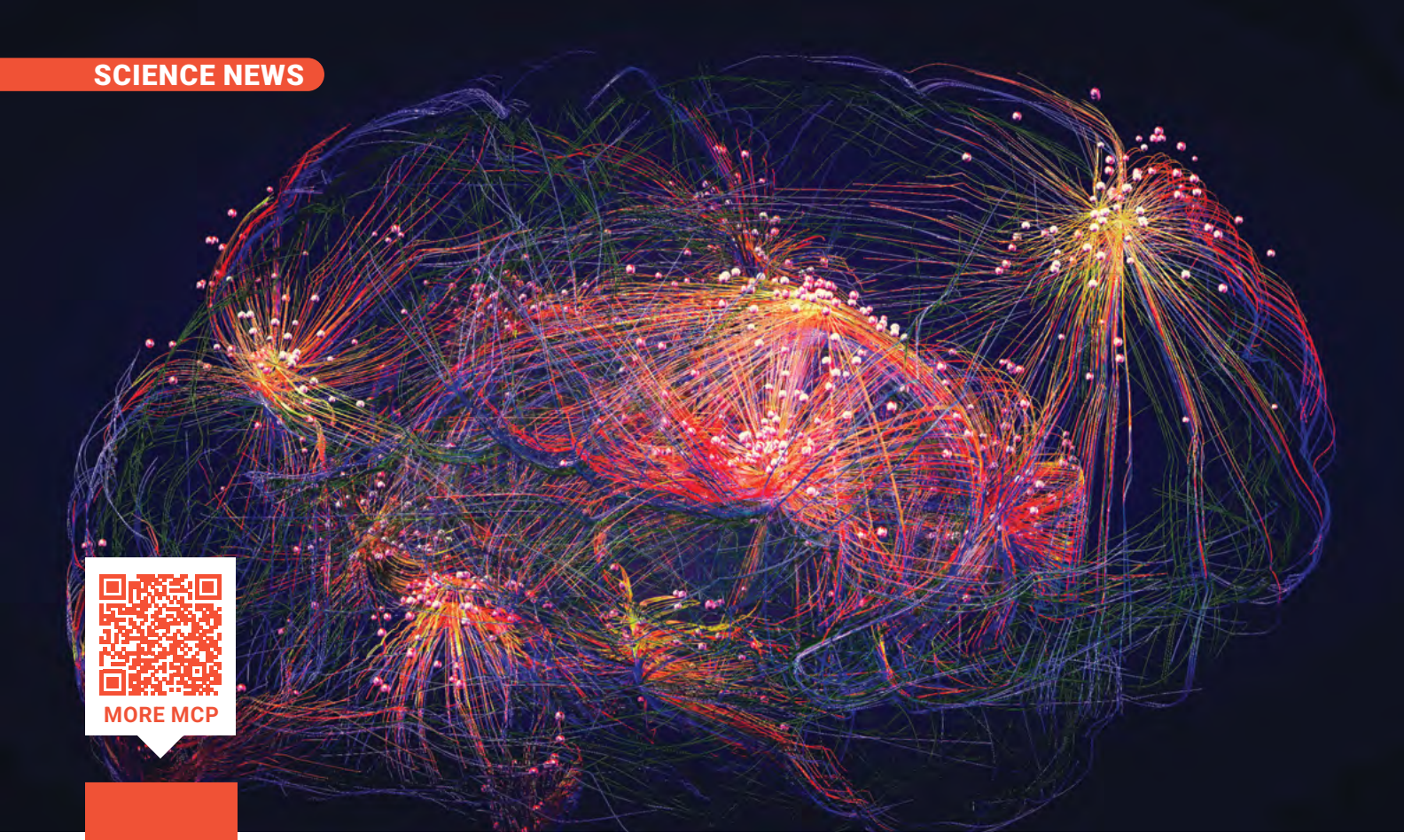
"This discovery was a surprise to us," Mishra said. "It's incredible to see that such a powerful pathway can be tamed. Gastrointestinal cancers are very difficult to treat, and these results showed us that spectrin inhibition can be very instrumental in the treatment of cancer, particularly colon cancers that are microbiome-driven."

Mice fed a high-fat diet — comparable to a fast-food-heavy human diet — and lacking proper TGF-β signaling showed significant microbiome changes. These mice had increased levels of two ammonia-producing bacteria: *Bacteroides ovatus* and *B. vulgatus*.

The team's findings reveal how microbial metabolites disrupt TGF-β signaling to promote CRC. They also suggest that inhibiting βII-spectrin could restore normal signaling and slow disease progression, making it a promising molecular target for CRC therapy.

Andrea Lius is a Ph.D. candidate in the Ong quantitative biology lab at the University of Washington. She is an ASBMB Today volunteer contributor.

Jessica Desamero is a graduate of the biochemistry Ph.D. program at the City University of New York Graduate Center and an ASBMB volunteer contributor.



MORE MCP

MCP

When oncogenes collide in brain development

By Manish Goyal

What happens when two critical proteins collaborate during neurodevelopment? A new Molecular & Cellular Proteomics study reveals insights that could transform how we understand childhood brain disorders.

The brain's intricate structure depends on precisely timed signals that guide cells to differentiate and migrate. When these signals go awry, the consequences can range from developmental disorders to tumor formation. Cortical development, which supports thinking, perception, language, movement and memory, is especially sensitive to these signals.

Wnt signaling and its downstream effector, CTNNB1, play a central role in this process, controlling the growth, development and migration of brain cells. Yet, when Wnt signaling becomes overactive, it can disrupt normal cortical development and, in some cases, lead to brain tumors in children. The study, headed by Jelena Navolić and Julia Neumann at University Medical Center Hamburg-Eppendorf in Germany, uncovered molecular mechanisms underlying specific brain malformations and tumors. The findings also point to potential therapeutic targets.

Building on this framework, the study centers on LIN28A, an oncoprotein, and CTNNB1, which encodes β-catenin, a core component of Wnt signaling. Previous work suggests that LIN28A and Wnt signaling have interconnected roles in brain development, but their precise functional link remained unclear. To address this gap, Navolić and colleagues investigated how coactivation of LIN28A and Wnt signaling affects cortical development, analyzing structural changes in the cerebral cortex of the mouse. They induced overexpression of *Lin28A* and stabilized the *Ctnnb1* gene in the same population of neural precursor cells, causing

accumulation of both LIN28A and CTNNB1 pathways. The team used nanosecond infrared laser technology to precisely sample tiny regions of mouse brain cortices, allowing them to map protein abundance at unprecedented resolution. They found that cortical layering was disrupted and neuron migration impaired in the embryonic brain. They also observed changes in the distribution of extracellular matrix, or ECM, receptors RPSA and ITGB1, along with reduced glycosylation of α-dystroglycan. ECM receptors anchor cells to their surroundings, while glycosylation allows α-dystroglycan to bind effectively to ECM proteins. These alterations weaken cell attachment and signaling, impairing neuron migration — an essential process for neurodevelopment and tissue repair. The defects resemble cobblestone lissencephaly type 2, a rare disorder marked by a bumpy brain surface and neurological deficits.

Overall, the study reveals a previously unrecognized role for LIN28A in maintaining the extracellular matrix during brain development, especially when combined with CTNNB1. Furthermore, the findings suggest that interactions between oncogenes can contribute to both tumor formation and developmental brain disorders. The work also highlights the power of nano-volume spatial proteomics to map protein distributions across tiny brain regions in remarkable detail. These insights may guide future research and therapeutic strategies for rare brain disorders and aggressive pediatric tumors linked to LIN28A and Wnt signaling.

Manish Goyal is a research assistant professor in the Department of Molecular and Cell Biology at Boston University and an ASBMB Today volunteer contributor.

3D rendered image of a scanning electron micrograph of budding yeast.

Method sharpens proteome-wide view of structural changes

By ASBMB Staff

Protein structure underlies function, and shifts in that structure can reveal changes in cell signaling, metabolism, stress responses and genetic variation.
But tracking such structural changes across the proteome remains a challenge. Luise Nagel of the University of Cologne and colleagues in Switzerland developed a method using limited proteolysis coupled with mass spectrometry, or LiP–MS, to distinguish true structural changes from confounding factors such as protein abundance. They published their findings in **Molecular & Cellular Proteomics**. LiP–MS works by probing protein structures with proteinase K, or PK, a protease whose digestion patterns shift when proteins change shape. The team validated their method using samples from budding yeast, fission yeast and human cerebrospinal fluid.

To isolate structural signals, they built a framework to remove unwanted variation (RUV) from LiP–MS data. The framework outperformed other approaches by separating structural changes from effects such as protein abundance, posttranslational modifications and alternative splicing. Beyond LiP–MS, the framework could apply to other peptide-centric structural proteomics methods, including fast photochemical oxidation of proteins and molecular painting.

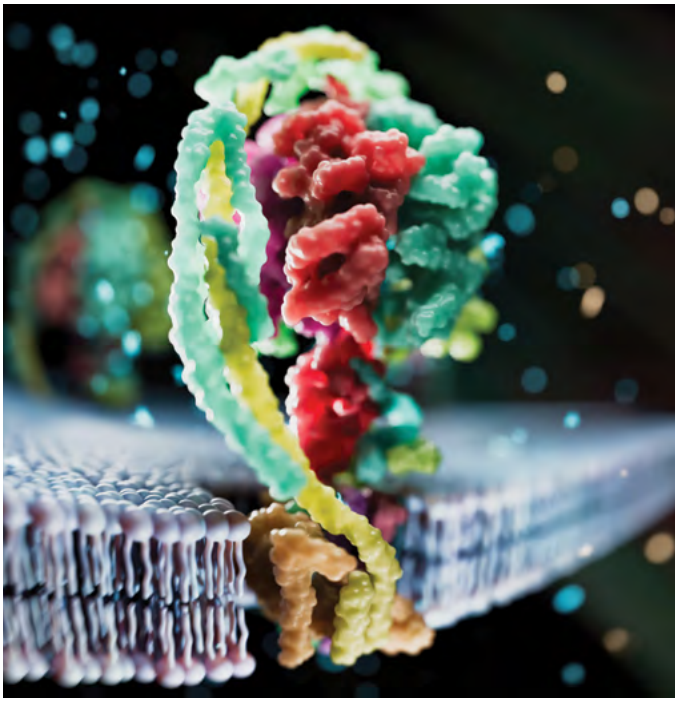


Illustration of the mitochondrial adenosine triphosphate synthase membrane protein.

Mass spec method captures proteins in native membranes

By ASBMB Staff

Cell function depends on how proteins and lipids are organized in native membranes, but studying those interactions has been difficult. Most approaches require extracting membrane proteins, or MPs, and stabilizing them in detergents that disrupt native interactions.

In a recent **Molecular & Cellular Proteomics** article, Wonhyeuk Jung and colleagues at Yale University and the University of Nebraska–Lincoln developed a protocol using supercharger-assisted prequadrupole activation and mass spectrometry. The approach combines collision-induced and electron-capture dissociation to fragment ions and detect proteins directly in intact membranes.

Applying this method to *E. coli* membranes, the team detected diverse protein complexes preserved in their natural state and demonstrated that the platform can track drug binding to antibiotic targets. They plan to refine the method by adding biochemical interventions to study any membrane protein directly in its native environment.

Laser-assisted cryoEM method preserves protein structure

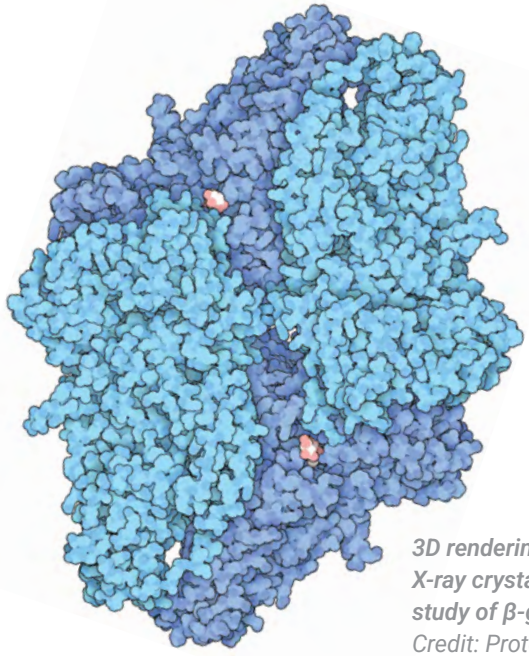
By ASBMB Staff

Combining mass spectrometry, or MS, and cryogenic electron microscopy, or cryoEM, has long promised high-resolution 3D reconstructions of proteins. Past attempts were limited by low resolution and protein compaction.

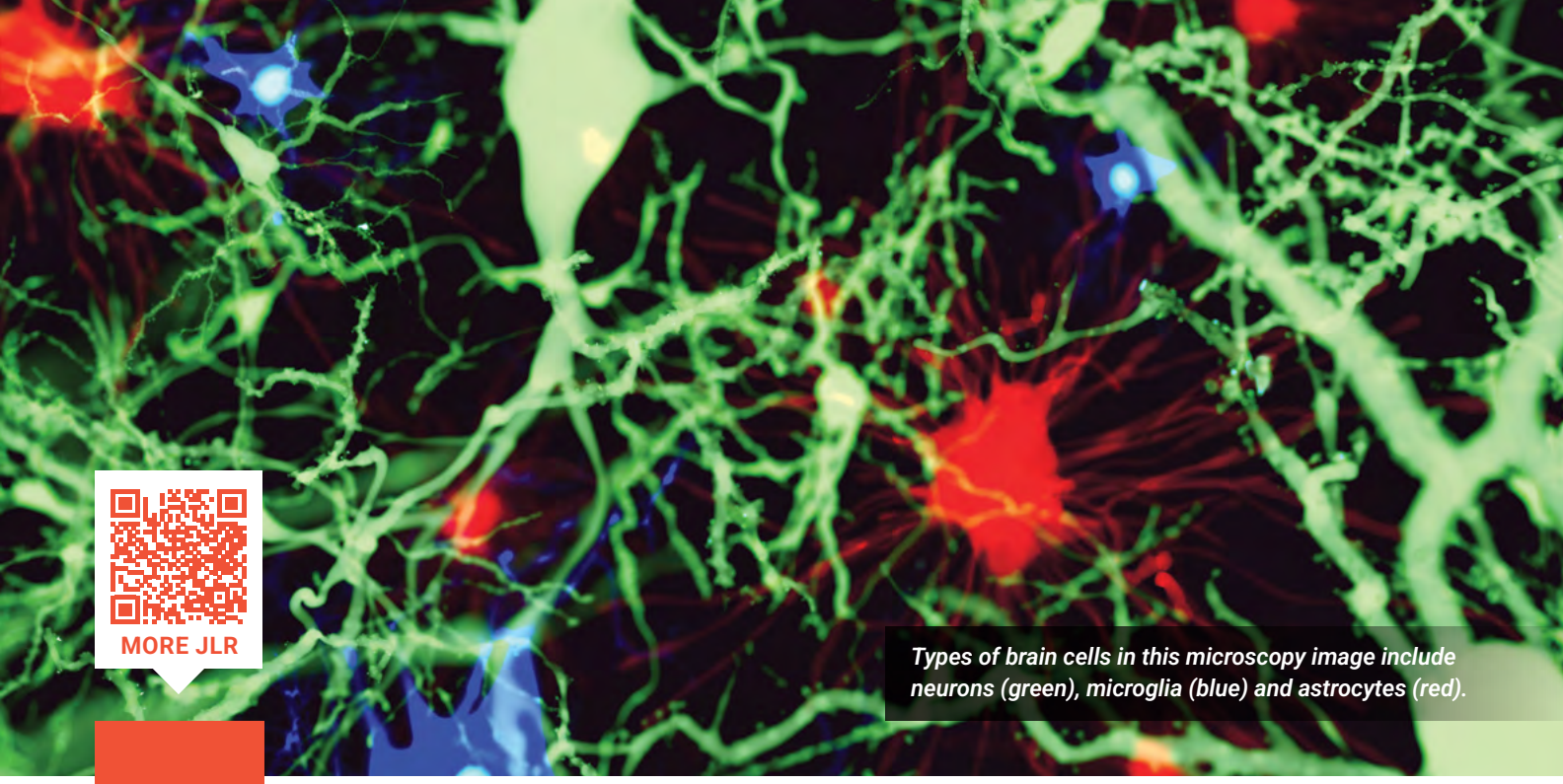
Keaton Mertz, Drew Jordahl and colleagues at the University of Wisconsin–Madison and the Morgridge Institute for Research developed a laser-assisted cryoEM method that overcomes these barriers, paving the way for broader MS–cryoEM integration. They published their findings in **Molecular & Cellular Proteomics**.

The method uses a laser built into transmission electron microscopy grids to liquefy ice particles. When the laser is turned off, proteins rehydrate and regain their native structure before refreezing, producing samples suitable for reconstruction.

Testing with β-galactosidase, the team showed that protein structure was restored without compaction. Results matched those from conventional plunge freezing but with fewer distortions. The researchers expect the technique will enable studies of more complex protein systems.



3D rendering from an X-ray crystallographic study of β-galactosidase. Credit: Protein Databank



MORE JLR

Types of brain cells in this microscopy image include neurons (green), microglia (blue) and astrocytes (red).

JLR

Mapping fentanyl's cellular footprint

By Caroline Junker

Emerging imaging tools are exposing ways fentanyl disrupts cells from within.

From its inception, fentanyl was designed for maximum potency. Synthesized as a painkiller, fentanyl's high lipophilicity allows it to easily cross the blood-brain barrier, which quickens its analgesic effect, but also heightens its addictive potential.

In a recent study in the **Journal of Lipid Research**, scientists used a new imaging approach to map fentanyl's effects on brain immune cells. Their findings could transform how addiction is assessed and lead to new diagnostic and therapeutic tools.

Supriya Mahajan, an associate professor of medicine at the State University of New York at Buffalo, and Rahul Das, a postdoc in Mahajan's lab, wanted to take a closer look at how brain immune cells respond to a sudden influx of fentanyl. They joined forces with photonics experts and fellow UB professors Paras N. Prasad, Andrey Kuzmin and Artem Pliss to form a multidisciplinary team. Using an emerging spectroscopic technique called Ramanomics, the team captured this response at unprecedented resolution, down to the level of a single organelle. The researchers wanted to follow fentanyl molecules into the lipid droplets and measure how the drug altered their composition.

Like Tupperware for fats, lipid droplets store lipids inside cells. But unlike containers, they are densely packed with lipids and active participants in cellular metabolism — regulating stress responses, inflammation and energy balance.

To observe fentanyl-induced changes in lipid droplets within astrocytes and microglia, two types of brain immune cells, the team used Raman microscopy to scan cultured cells with a laser beam. This approach revealed a cascade of subcellular changes triggered by high levels of fentanyl.

The team found that fentanyl caused a loss of carbon-carbon double bonds in the phospholipid membranes of lipid droplets in both astrocytes and microglia, a sign of reduced unsaturation. Reduced unsaturation makes

membranes more rigid and less fluid, disrupting permeability, protein function and signaling. Broadly, changes in phospholipid unsaturation in cell membranes are linked to a variety of neurological diseases.

They also detected changes in cholesterol, glycogen, phosphocholine and sphingomyelin levels. Many of these molecules are implicated in essential biochemical pathways, and certain changes in their concentrations can be linked to neuronal damage and cognitive decline.

This analysis is the first to reveal fentanyl's behavior within lipid droplets in such molecular detail. The findings add to growing knowledge of addiction, neurodegeneration and inflammation — and may inform future therapeutic initiatives and treatments. "Now, we are trying to understand the chemistry," said Das.

But in terms of Ramanomics, and of assembling these subcellular changes into a collection of addiction biomarkers, the researchers have their sights set on an even more ambitious goal: to identify susceptibility to and ultimately prevent addiction.

Das noted that there are wearable technological accessories that already track body metrics, like oxygen levels, noninvasively.

"Just imagine that kind of tech is applied to patients, or people who are given treatment, those who are nonaddicted and becoming addicted," Das said. "Those gadgets could help inform doctors."

The sensors that the team envisions could detect subtle biochemical changes that precede addiction or neurodegenerative symptoms.

Their work offers a glimpse into addiction's smallest mechanisms and into a future where early detection could save lives.

Caroline Junker is a writer and science communicator and an ASBMB Today volunteer contributor. She earned a bachelor's in neuroscience, with a minor in English, from Franklin and Marshall College in 2023.

Missing lipid shrinks heart and lowers exercise capacity

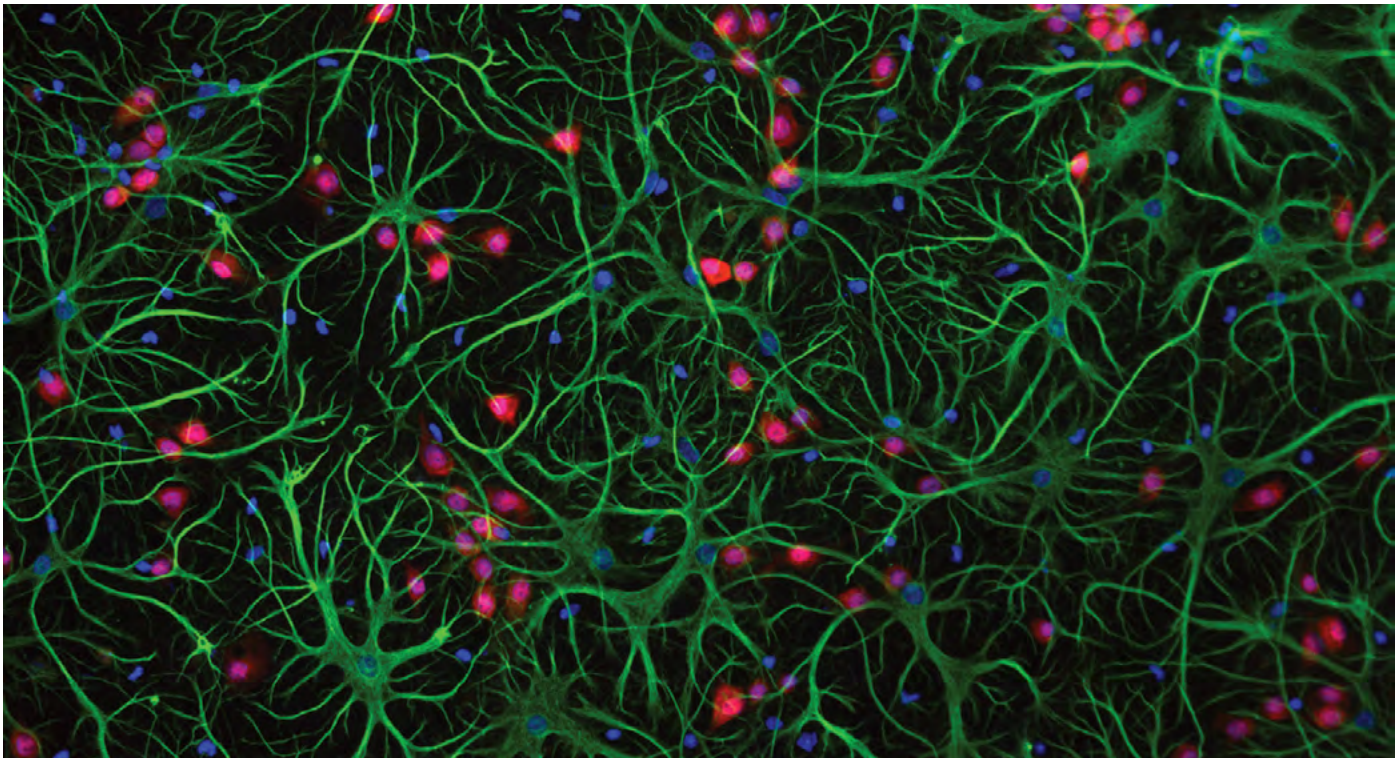
By Samara Baksh

Phospholipase A and acyltransferase 1, or PLAAT1, is an enzyme that catalyzes lipid-modifying reactions. Under laboratory conditions, PLAAT1 produces a lipid known as cardiolipin. Cardiolipin is essential for the inner mitochondrial membrane because it stabilizes respiratory complexes and ATP synthase to support energy metabolism; thereby, influencing diverse biological processes, including growth, cardiorespiratory function and exercise capacity. However, the impact of PLAAT1 loss in living organisms remains unknown. To address this, Ashkan Hashemi and colleagues from the University of Waterloo, in collaboration with a team of researchers in Scotland, generated mice lacking the PLAAT1 gene and compared them to mice expressing PLAAT1. They published their results in the **Journal of Lipid Research**.

The authors observed that PLAAT1-deficient mice had similar body weights to their normal counterparts, although males ate less. Their hearts were noticeably smaller — 14.2% smaller in males and 10.6% smaller in females — and cardiolipin content in the heart dropped by approximately one-third, primarily due to a loss of the linoleate-rich form of the lipid. Additionally, these hearts contained reduced amounts of succinate dehydrogenase complex flavoprotein subunit A, a mitochondrial protein vital for energy metabolism. PLAAT1 deficiency also diminished oxygen consumption, carbon dioxide production and total energy expenditure. While general activity levels remained similar, exercise capacity was impaired in both male and female mice.

Together, these findings highlight PLAAT1 as a critical regulator of cardiolipin composition, mitochondrial function and systemic energy metabolism, with direct consequences for cardiovascular performance and potential links to mitochondrial disease.

Samara Baksh is a graduate of the Master's in Biotechnology program at Johns Hopkins University. She works as a bench scientist and is an ASBMB Today volunteer contributor.



Cells of the brain, including astrocytes (green) and neurons (red). Blue represents cell nuclei.

Key regulator of cholesterol protects against Alzheimer's disease

By Jeyashree Alagarsamy

The mammalian brain contains more than 25% of the body's cholesterol and relies on cholesterol produced within the brain to support cognitive health. Dysregulated cholesterol metabolism has been linked to Alzheimer's disease, or AD, because cholesterol imbalance affects membrane organization in brain cells, promoting amyloid precursor protein cleavage and increasing amyloid-beta, or Aβ, production. In a recent study published in the **Journal of Lipid Research**, Arlette A. Kasongo and colleagues from the University of Ottawa investigated oxysterol-binding protein-related protein 6, or ORP6, and found that it is a critical regulator of brain cholesterol metabolism with protective effects against Alzheimer's-related neurodegeneration.

The researchers found that ORP6 is highly expressed in astrocytes and neurons of the hippocampus, a critical region for learning and memory. They generated mice lacking the *Osbpl6* gene, and the absence of ORP6 disrupted cholesterol metabolism and led to the accumulation of the cholesterol precursor desmosterol and toxic amyloid beta oligomers. Lipidomic, proteomic and correlational analyses revealed whole-body and brain lipid imbalances, brain atrophy, reduced synaptic proteins and behavioral deficits linked to early AD.

At the cellular level, ORP6 deficiency impaired the ability of astrocytes to export excess cholesterol and increased cholesterol esterification in astrocytes, resulting in decreased plasma membrane cholesterol and increased processing of the amyloid precursor protein into Aβ oligomers. ORP6 levels diminished in human AD brains and AD mouse models, especially in astrocytes, correlating with lipid droplet buildup. These results highlight ORP6 as a vital regulator of astrocyte cholesterol balance with potential protective effects against AD. Thus, enhancing ORP6 function could be a promising therapeutic approach for combating AD. Future research could investigate whether restoring ORP6 expression in AD models can normalize cholesterol metabolism and reduce Aβ burden.

Lipid-lowering therapies could help treat IBD

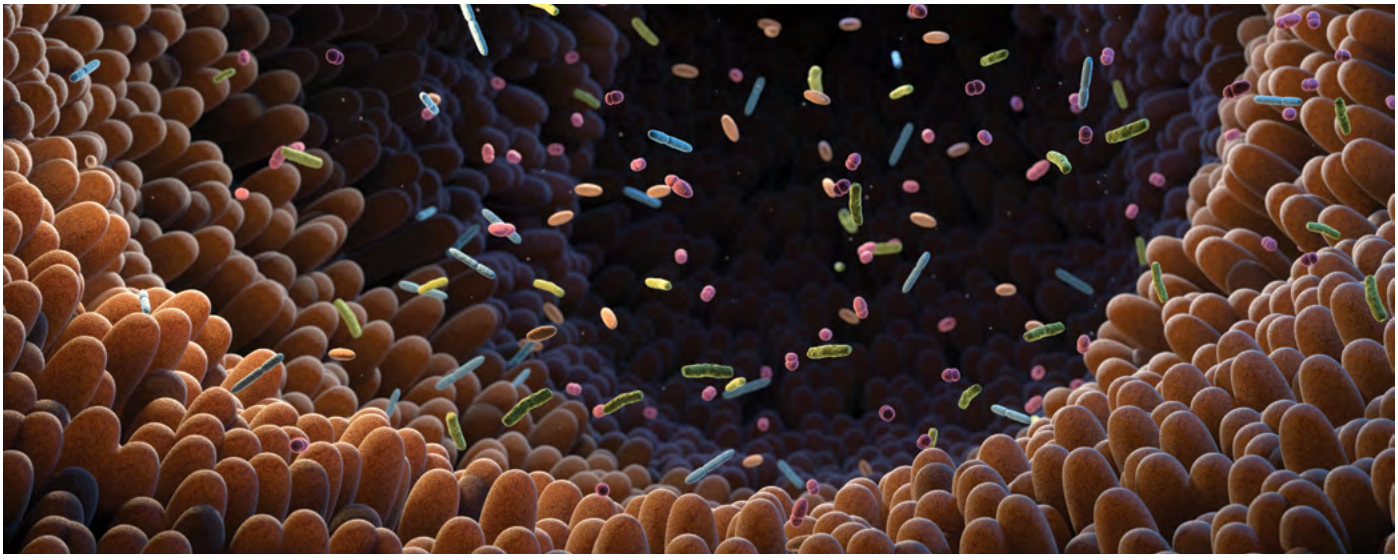
By Jeyashree Alagarsamy

Inflammatory bowel disease, or IBD, which includes Crohn's disease and ulcerative colitis, causes chronic gut inflammation and affects millions worldwide. While immune dysfunction and gut barrier defects are central to IBD, newer studies suggest lipid metabolism and the gut microbiome also play key roles. Dyslipidemia, abnormal levels of fats like cholesterol or triglycerides in the blood, has been linked to higher IBD risk. However, the effect of lipid-lowering drugs is less clear. A recent study in the **Journal of Lipid Research** by Xin Huang and colleagues at Shandong First Medical University explored how lipid-lowering drugs influence IBD through gut microbes and immune signaling.

Using drug-targeted Mendelian randomization, a genetic approach that uses naturally occurring variants to mimic the effects of specific drugs, the authors identified nine drug targets, including statins, proprotein convertase subtilisin kexin type 9 or PCSK9 inhibitors, 3-hydroxy-3-methylglutaryl–coenzyme A reductase or HMGCR, and triglyceride-lowering therapies. Blocking the lipoprotein regulator angiopoietin-like 3, or ANGPTL3, or the triglyceride regulator apolipoprotein C-III or APOC3 in humans raised IBD risk, while greater activity of lipoprotein lipase or LPL and low-density lipoprotein receptor, or LDLR, lowered risk. These results suggest IBD outcomes depend on the lipid pathway targeted.

Microbiome data showed that PCSK9 and APOC3 inhibition reduced diversity and beneficial gut bacteria such as *Bifidobacterium* and *Lactobacillus*, while HMGCR inhibition increased diversity and short-chain fatty acid producers. These microbial changes matched cytokine shifts, with higher IL-6 and TNF-α in PCSK9 and APOC3 groups, and lower levels in statin users. Overall, the study showed that lipid-lowering drugs can affect IBD not only through lipid changes but also by shaping the microbiome and immune pathways. These findings suggest that lipid-lowering therapies can influence IBD progression by reshaping gut microbial communities and inflammatory signaling, not just circulating lipid levels. Future research could explore microbiota-based strategies to mitigate the proinflammatory effects of certain lipid-targeting drugs in IBD.

Jeyashree Alagarsamy is a freelance scientific writer and experienced researcher with experience in atherosclerosis, lipid metabolism and neuroscience. She is also an ASBMB Today volunteer contributor.



Microbiome

ASBMB names 2026 fellows

By Jessica Desamero

The American Society for Biochemistry and Molecular Biology announced that it has named 16 members as 2026 fellows of the society. ASBMB fellows are members who have a history of exceptional and sustained service to the society, as well as a distinguished record of professional accomplishments that advance the molecular life sciences. Areas of accomplishment include basic and translational research, application and commercialization, education, outreach, increasing belonging and opportunity in science, mentorship, leadership and public service. The society will honor the 2026 fellows at the ASBMB annual meeting on March 7–10 in Maryland. Learn more about the 2026 fellows below.

Donald Becker

Donald Becker is the head of the biochemistry department at the University of Nebraska–Lincoln. His research examines proline metabolism and how it impacts stress response and the balance of oxidants and antioxidants within cells. These chemical changes can influence the growth of different organisms and are thought to be involved in many cellular processes that affect disease progression.

Becker served as an editorial board member of the Journal of Biological Chemistry. He is also a champion of several educational efforts, including ASBMB accreditation, certification and ensuring students can attend ASBMB's annual meeting.

Becker was nominated by ASBMB fellow Oleh Khalimonchuk.

Paul Black

Paul Black is a professor and chair emeritus of biochemistry at the University of Nebraska–Lincoln. His research addressing the mechanism of fatty acid transport led to multiple U.S. patents and therapeutic innovations for metabolic diseases.

His lab also pioneered and patented the use of algal photobioreactor systems to remove excess nitrogen-bound nitrate from groundwater sources. Black has served as a JBC editorial board member for two terms and continues to review manuscripts for both the JBC and the Journal of Lipid Research. He was also a judge for ASBMB's undergraduate poster competition. In 2017, the biochemistry club at the University of Nebraska–Lincoln won the ASBMB outstanding student chapter award. In 2018, he was named a fellow of the American Association for the Advancement of Science for his seminal work on fatty acid transport. In 2020, he won the ASBMB Award for Exemplary Contributions to Education.

Black was nominated by ASBMB fellow Peter Kennelly.

Rachell Booth

Rachell Booth is a professor of biochemistry at the University of the Incarnate Word. During the COVID-19 pandemic, she served on the ASBMB Student Chapters steering committee that provided ASBMB members with resources for pivoting to online teaching.

Booth served as the South-Central regional director of ASBMB Student Chapters for 10 years. She has contributed extensively to the society's educational mission, including judging the undergraduate poster competition at the ASBMB annual meetings, co-organizing the 2019 educational conference,

Transforming Undergraduate Education in the Molecular Life Sciences, and co-facilitating the Lab Management Workshops for early-career members. In 2021, she joined the Membership Committee.

Booth was nominated by ASBMB fellow Quinn Vega and ASBMB fellow Mary Huff.

Mary Dasso

Mary Dasso is the head of cell cycle regulation, associate scientific director for budget and administration and a senior investigator in the section on cell cycle regulation at the National Institutes of Health Eunice Kennedy Shriver National Institute of Child Health and Human Development, or NICHD. Her lab studies the components of nuclear transport machinery throughout the cell cycle. Dasso is known for discovering how the G protein Ran regulates mitotic spindle assembly during cell division, independent of its role in nuclear transport.

Dasso has won several national awards, including a 2020 National Institutes of Health Director's award and a 2021 NICHD Merit Award. In 2018, she was elected an AAAS fellow. Dasso has also been a spotlight session chair, workshop organizer and career speed networking panelist at the ASBMB annual meeting. She is currently a member of the Women in Biochemistry and Molecular Biology Committee.

Dasso was nominated by Juan Bonifacino.

Daniel Dries

Daniel Dries is an assistant professor of chemistry in the Schmid College of Science and Technology at Chapman University. His lab aims to advance STEM education research by assessing and reimagining effective teaching and learning strategies. He currently focuses on using the theories of motivation and identity to create more equitable learning environments.

Dries serves on the Accreditation and Exam Subcommittee and is a former regional co-director for the ASBMB Student Chapters Steering Committee. He was also on the program planning committee for the 2021 annual meeting. In addition, he has served as a judge for the ASBMB undergraduate poster competition and for the Promoting Research Opportunities for Latin American Biochemists program. In 2025, he co-organized the TUEMLS meeting, an event focused on rethinking teaching strategies to better support students in biochemistry and molecular biology. He is also chair of the Fellowships Committee for the International Union of Biochemistry and Molecular Biology.

Dries was nominated by ASBMB fellow Victoria Del Gaizo Moore and ASBMB fellow Joseph Provost.

Donald Elmore

Donald Elmore is a professor of chemistry and endowed chair in the health sciences in the chemistry department and the biochemistry program at Wellesley College. His lab studies cell membrane proteins and characterizes and designs histone-derived antimicrobial peptides. His lab also collaborates with colleagues to develop chemistry and biochemistry educational activities for high school and undergraduate students.

Elmore previously received the Wellesley Pinanski Prize for teaching and was named a Henry Dreyfus Teacher-Scholar. Elmore has served as a member of the WiBMB Committee and a coach for the Interactive Mentoring Activities for Grantsmanship Enhancement, or IMAGE, grant writing workshop. He also serves as an undergraduate poster competition judge and reviews journal articles and grant proposals for several domestic and international organizations.

Elmore was nominated by Louise Darling, ASBMB fellow Susan Baserga, ASBMB fellow Adele Wolfson, ASBMB fellow Squire Booker and ASBMB fellow Kayunta Johnson–Winters.

I. Robert Lehman

I. Robert Lehman is a professor emeritus at the Stanford School of Medicine. His work focuses on DNA replication and repair, and he has identified two enzymes that play critical roles in these processes. As a postdoctoral fellow, he collaborated closely with Arthur Kornberg to define DNA polymerase, which synthesizes DNA. As an independent investigator, he discovered DNA ligase, which stitches two separate DNA molecules together. Both enzymes are key elements in the cloning, amplification and sequencing of DNA molecules.

Lehman was the ASBMB president in 1997 and has served as a JBC associate editor and editorial board member. In 2008, Lehman won the Herbert Tabor Research Award for his work in DNA metabolism, track record in mentorship and service to the JBC. He has been a member of the National Academy of Sciences since 1977.

Lehman was nominated by ASBMB fellow Charles Samuel and ASBMB President Joan Conaway.

Mary Lipton

Lipton is a biochemist in the Environmental Molecular Sciences Division and the Environmental Molecular Sciences Laboratory at Pacific Northwest National Laboratory, or PNNL. She is known for using mass spectrometry–based omics analyses to characterize natural systems, including environmental microbes and microbial communities.

Lipton is a member of the ASBMB Public Affairs Advisory Committee. In 2023, 2024 and 2025 she visited Washington, D.C. for the society's annual Capitol Hill Day. She has won several PNNL awards, including the Emerging Health Innovators Performance award, Woman of Achievement award and Outstanding Performance award. She was elected an AAAS fellow in 2022.

Lipton was nominated by Ann West.

Lea Vacca Michel

Lea Vacca Michel is a professor of chemistry and materials science and the College of Science's Director of Access and Belonging at the Rochester Institute of Technology. Her lab studies the role of bacterial lipoproteins and crystallin proteins in bacterial infections and cataracts, respectively. Her group also studies bacterial extracellular vesicles as potential vaccine carriers and diagnostic biomarkers for bacterial sepsis.

Michel is currently the chair of the Maximizing Access Committee and has been an undergraduate poster judge at nine ASBMB annual meetings. She has also served as Director of the Rochester ACS Project SEED program and Chair of the Rochester ACS Women Chemists Committee. In 2022, she received the ASBMB Early-Career Leadership Award. In 2023, she received the ChemCUR Outstanding Mentor Award from the Chemistry Division and the Council on Undergraduate Research. In 2024, she was selected as a finalist for the Presidential Awards for Excellence in Science, Mathematics and Engineering Mentoring.

Michel was nominated by Sarah Bowman and ASBMB fellow Catherine Drennan.

Caryn Outten

Caryn Outten is a professor of chemistry and biochemistry at the University of South Carolina. Her research lies at the intersection of redox biology and

bioinorganic chemistry, with a focus on intracellular iron sensing and regulation, iron-sulfur cluster biogenesis and glutathione metabolism.

Outten previously served as a JBC editorial board member and an ASBMB Membership Committee member. As part of the Membership Committee, Outten helped establish the ASBMB Fellows program in 2020. She was also elected an AAAS fellow in 2019. Her awards and honors include the Presidential Early Career Award for Scientists and Engineers, the South Carolina Governor's Young Scientist Award for Excellence in Scientific Research and the South Carolina Chemist of the Year Award.

Outten was nominated by Joan Broderick and ASBMB fellow Dennis Dean.

Tanya Paull

Tanya Paull is a professor of molecular biosciences and chair in human health at the University of Texas at Austin. Her lab studies the DNA damage response in eukaryotic cells and examines the events that occur immediately after chromosomal double-strand breaks.

Paull previously served as a member of the Publications Committee and the Science Outreach and Communication Committee. She was also a speaker at the 2023 ASBMB annual meeting. From 2008 through 2019, she was an Investigator with the Howard Hughes Medical Institute, or HHMI.

Paull was nominated by ASBMB fellow Daniel Leahy.

Jared Rutter

Jared Rutter is a distinguished professor of biochemistry at the University of Utah. He is co-director of the Center for Metabolic Health at the University of Utah and co-leader of the Genomics, Epigenetics and Metabolism Program at Huntsman Cancer Institute. His lab studies how cells sense and detect energy needs for their growth and metabolism using a variety of experimental and computational approaches to define the molecules and interactions involved. His lab has developed a platform that integrates mass spectrometry with equilibrium dialysis to discover metabolite-protein interactions.

Rutter is a member of the ASBMB Meetings Committee and a past member of the ASBMB Council. He was also a session chair for the 2025 annual meeting and previously served as a mentor for the IMAGE workshop. In addition, he is a current Investigator of the Howard Hughes Medical Institute.

Rutter was nominated by ASBMB fellow Wesley Sundquist.

Alan Saltiel

Alan Saltiel is a distinguished professor of medicine and pharmacology at the University of California San Diego and director of the Institute for Diabetes and Metabolic Health. His research focuses on how cells use and store energy in response to hormones and nutrients in health and disease, including uncovering the molecular mechanisms of insulin action and links between obesity and diabetes. He has discovered and developed several drugs, including the first MEK inhibitors for cancer.

Saltiel is an editorial board member of the JBC. He has won several awards, including the Rosalyn Yalow

Research and Development Award from the American Diabetes Association, as well as the Goodman and Gilman award and Pharmacia award, both from the American Society for Pharmacology and Experimental Therapeutics. He is a member of the National Academy of Medicine, National Academy of Inventors and an AAAS fellow.

Saltiel was nominated by ASBMB fellow Alexandra Newton.

Ronald Wek

Ronald Wek is a professor of biochemistry at the Indiana University School of Medicine. His research focuses on understanding how cells cope with stress and how mechanisms of regulating protein synthesis and stress pathways contribute to disease progression.

Wek has been extensively involved with the ASBMB Journal of Biological Chemistry and has contributed manuscripts and served as a reviewer throughout his career. He joined the JBC editorial board in 2013, and he became an associate editor in 2016. He has also served on the ASBMB Public Affairs Advisory Committee and is currently a member of the ASBMB Today Advisory Board.

Wek was nominated by Scott Aoki.

Lance Wells

Lance Wells is a Georgia Research Alliance Distinguished Investigator and Associate Director of the Complex Carbohydrate Research Center at the University of Georgia. His lab studies the role of O-glycosylation in neurological and muscular disorders.

Wells previously served as an editorial board member of JBC and is an editorial board member of Molecular & Cellular Proteomics. He served as a 2022 co-organizer of the first ASBMB special meeting on O-GlcNAc and has been an interest group leader, theme organizer and chair for previous ASBMB and Experimental Biology meetings.

Wells was nominated by Adam Barb, Michael Tiemeyer, ASBMB fellow Gerald Hart and ASBMB fellow Robert Haltiwanger.

Michael Wolyniak

Michael Wolyniak is a professor of biology and the director of undergraduate research and creative activity at Hampden–Sydney College. He has received NSF grants for developing classes at the interface of biology and engineering as well as integrating CRISPR into the undergraduate classroom. He is a guest editor of an upcoming Biochemistry and Molecular Biology Education issue, which focuses on the current state of biochemistry and molecular biology education worldwide as well as the movements that have helped transform student learning. Wolyniak's research explores the biochemistry of malate dehydrogenase as a member of the Malate Dehydrogenase Course-Based Undergraduate Research Experiences Community.

Wolyniak serves as a member of the SOCC Committee and chair of the Outreach Subcommittee. He also advises the ASBMB Student Chapter at his institution. Wolyniak was a speaker for the 2023 and 2025 TUEMLS meetings. He is the current chair of the Biology Division of the Council on Undergraduate Research and a past president of the Virginia Academy of Science. In 2022, he received the Libby and Hiter Harris Excellence in Undergraduate Teaching Award from the Virginia Foundation for Independent Colleges.

Wolyniak was nominated by ASBMB fellow Jennifer Roecklein–Canfield and ASBMB fellow Kathleen Cornely.

Member update

Alrubaye wins research and teaching awards

Adnan Alrubaye has been awarded the University of Arkansas Faculty Gold Medal and the Educator Award from the North American Colleges and Teachers of Agriculture for his exceptional dedication to teaching and student mentorship beyond the classroom. The Faculty Gold Medal is the university's highest honor and is presented only once during a faculty member's career at the State and National Awards reception. Alrubaye was honored at the NACTA 2025 conference for receiving the Educator Award.

Alrubaye is an assistant professor of poultry science and the associate director of the graduate program in cell and molecular biology at the U of A. His research focuses on the etiology of bacterial chondronecrosis with osteomyelitis, or BCO, lameness in broiler chickens, a major animal welfare and economic challenge in the poultry industry. In addition to research, Alrubaye teaches one of the university's largest courses, general microbiology, which has an average enrollment of over 450 students.

"Both awards remind me how powerful good teaching can be in shaping futures," Alrubaye said in the U of A press release. "For me, teaching is not just about delivering knowledge — it's about inspiring curiosity, critical thinking and the confidence students need to lead in agriculture and science."

Throughout his career, Alrubaye has received numerous awards for teaching and research, including the U of A Imhoff Award for Outstanding Teaching and Student Mentorship, the Bumpers College Jack G. Justus Award for Teaching Excellence, the Hoyt H. Purvis Award for Service in International Education, the Imhoff Outstanding Research Publication Award and the Honors College Distinguished Faculty Teaching and Research Award.

ASBMB members receive ASM awards

The American Society for Microbiology has announced the recipients of its 2026 awards, including American Society for Biochemistry and Molecular Biology members Jennifer Doudna, Michael Ibba and Kim Orth.

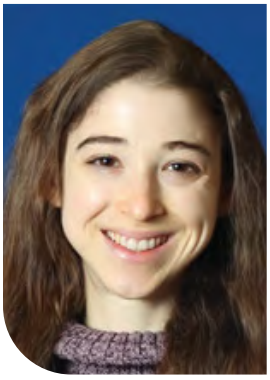
Jennifer Doudna, professor of biochemistry, biophysics and structural biology at the University of California, Berkeley, received the ASM Lifetime Achievement Award, which recognizes sustained contributions to the microbial sciences. She received the 2020 Nobel Prize in Chemistry with Emmanuelle Charpentier, a professor of the science of pathogens at the Max Planck Institute, for her work on CRISPR/Cas9 genome editing. Her lab and the Innovative Genomics Institute, which she founded, investigate the CRISPR bacterial adaptive immune system, ways to engineer novel CRISPR/Cas systems and how to apply CRISPR systems in microbiomes. Doudna is a member of the National Academy of Sciences, the American Academy of Arts and Sciences, the National Academy of Medicine, the National Academy of Inventors, the American Academy of Microbiology, the Royal Society and the Pontifical Academy of Sciences. She received the first American Society for Biochemistry and Molecular Biology Mildred Cohn Award in 2013.

Michael Ibba, executive vice president, provost, chief academic officer and professor at Chapman University, received the ASM Award for Graduate Education, which recognizes a scientist for outstanding achievements in graduate-level education. His lab investigates how cells ensure accurate translation of

the genetic code and how changes in translational control contribute to microbial pathogenesis and disease. Before moving to Chapman in 2020, he taught at Ohio State University for 19 years. He held several leadership roles there, including chair of the microbiology department and co-director of the graduate training program.

Kim Orth, an endowed chair and scholar in biomedical research at the University of Texas Southwestern Medical Center, received the ASM Award for Basic Research, which recognizes a scientist whose discoveries have been fundamental to advancing our understanding of the microbial world. Her lab investigates the pathogenesis of *Yersinia* and *Vibrio*, which are linked to the bubonic plague and food poisoning, and her lab uncovered two novel posttranslational modifications: YopJ Ser/Thr acetylation and VopS AMPylation. She is a Howard Hughes Medical Institute investigator and a member of the National Academy of Sciences and the American Academy of Microbiology. She has received numerous awards for her work, including the 2018 ASBMB–Merck Award and the 2012 ASBMB Young Investigator Award.

Bibel named assistant professor



Brianna Bibel began her position as assistant professor of chemistry and biochemistry at Loyola Marymount University in August 2025. Before joining the faculty at LMU, she taught biochemistry and chemistry and mentored undergraduate researchers as a visiting professor at Saint Mary's College of California.

Her lab investigates how microbes adapt and rewire their metabolism to clean up environmental pollutants in soil and water. Specifically, she investigates links between structure and function in the core metabolic enzyme malate dehydrogenase, or MDH, which is found in *Bacillus safensis* and *Bacillus subtilis* bacteria. In the future, she hopes to use her lab's in vitro findings to make in vivo changes and study effects. These findings could lead to the use of B. safensis and B. subtilis for environmental cleanup and sustainable biotechnology.

Bibel previously served on the American Society for Biochemistry and Molecular Biology Science Outreach and Communication Committee, where she helped plan and implement ASBMB's outreach activities, including a special symposium. In 2024, she helped create the ASBMB education special interest group for biochemistry and molecular biology educators, and she continues to moderate this online forum.

Bibel has also been deeply involved in STEM outreach and science communication. She is a Cohort Fellow of the Malate Dehydrogenase Course-based Undergraduate Research Experiences, or CUREs, community, where she leads students in their research on MDH. She also maintains a biochemistry blog, The Bumbling Biochemist, where she explains core biochemistry concepts and laboratory techniques, using accessible language and infographics.

Cotruvo named Blavatnik award finalist

For the second year in a row, Joseph Cotruvo Jr. was named as a finalist for the Blavatnik National Award for Young Scientists in the chemical sciences by the New York Academy of Sciences. The Blavatnik Family Foundation, founded by businessman and philanthropist Leonard Blavatnik, funds this award and other ventures that promote innovative scientific research, educational advances and cultural institutions. Cotruvo received a $15,000 prize and was recognized at a gala in October. Cotruvo, a professor of chemistry at Pennsylvania State

University, was recognized for his innovative protein engineering to selectively recover rare-earth elements, which are often found in smartphones, electric vehicles and wind turbines. His lab studies how bacteria utilize a family of lanthanide-binding proteins, which they use to develop biotechnologies that detect, extract and recycle rare-earth metals from technological waste.

Cotruvo recently won the 2026 American Society for Biochemistry and Molecular Biology Mildred Cohn Young Investigator Award. In 2025, Cotruvo received the Faculty Scholar Medal in Life and Health Sciences from Penn State and the Society of Biological Inorganic Chemistry Early Career Award. In addition, he was previously honored with the Eli Lilly Award in Biological Chemistry in 2024 for his work on rare-earth element coordination chemistry. Cotruvo has also won other early-career awards, including the 2022 Edward I. Stiefel Young Investigator Award, the 2020 Department of Energy Early Career Research Program Award and the Charles E. Kaufman Foundation New Investigator Award.

"Being named a finalist for the Blavatnik (National) Awards for a second year in a row is an incredible accomplishment," Kenneth L. Knappenberger Jr., professor and head of chemistry at Penn State, said in the Penn State press release. "It's a testament to Joey's creativity as a scientist and the important and impactful nature of his research."

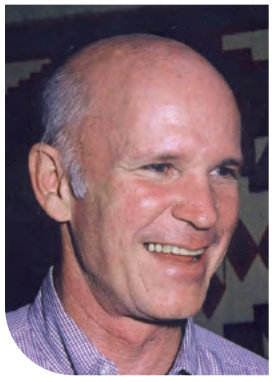
Jordahl named Gilliam Fellow

Eric Jordahl has been named a 2025 Gilliam Fellow by the Howard Hughes Medical Institute. He is one of thirty 2025 fellows. This award will provide Jordahl and his thesis advisor with three years of graduate funding. The program is named in honor of the late HHMI charter trustee, James H. Gilliam, Jr., who was an attorney, businessman and community leader. In addition to financial support, the awardees will have access to networking and professional development opportunities.

Jordahl is a graduate candidate at the University of California, San Diego. He performs research in the laboratory of Soyna Neal, an associate professor of cell and developmental biology at UCSD, studying the role of rhomboid protein rhbdl4 in pancreatic cancer. "By better understanding how RHBDL4 helps cancer cells to grow better and die less, I can identify new potential targets for anticancer therapeutics," Jordahl said in a UCSD press release.

He is the mentorship chair for UCSD's Biology Undergraduate and Master's Mentorship Program, which supports historically underserved students through mentorship, research and professional development opportunities. Jordahl received a National Science Foundation Graduate Research Fellowship in 2022. He obtained a B.S. in molecular biology and a B.A. in classics at the University of Pittsburgh.

McKnight wins Lasker Award



Steven McKnight was recently honored with the 2025 Albert Lasker Basic Medical Research Award, which he shared with Dirk Görlich of the Max Planck Institute in Göttingen, Germany. This award recognizes scientists who have made fundamental discoveries on the frontiers of basic biomedical research. Albert Lasker was an American businessman who pioneered modern advertising as an executive at Lord & Thomas. McKnight was presented with this research prize, which carries a $250,000 honorarium, at a gala ceremony in September.

McKnight is the distinguished chair in basic biomedical research at the University of Texas Southwestern Medical Center. His research spans several major themes, including transcription factors and gene regulation, oxygen sensing and low-complexity domains, or LCDs, for which he was recognized. LCDs are regions of proteins that do not form three-dimensional structures and contain low amino acid variability. As a result of McKnight's

discoveries, what were once deemed junk protein segments are now known to be active species that can help organize nuclear and cytoplasmic compartments.

McKnight is a member of the National Academy of Sciences, the National Academy of Medicine and the American Academy of Arts and Sciences. His awards and honors include the Welch Award in Chemistry, the Wiley Prize in Biomedical Sciences, the Monsanto Award from the National Academy of Sciences, the Eli Lilly Award from the American Society for Microbiology and the Newcomb Cleveland Award from the American Association for the Advancement of Science. McKnight served as president of the American Society for Biochemistry and Molecular Biology from 2014 to 2016 and is an ASBMB fellow.

"By chasing down the outrageously irrelevant observation, we came to realize how protein domains of low sequence complexity work," McKnight said in his acceptance remarks at the Lasker Awards ceremony in New York City, reflecting on his work investigating LCDs.

Mydy named Purdue assistant professor

Lisa Mydy has been appointed assistant professor of biochemistry at Purdue University, beginning in Fall 2025. She previously worked at the University of Michigan as a National Institutes of Health postdoctoral Ruth L. Kirschstein National Research Service Award fellow and senior research fellow in medicinal chemistry.

As part of Purdue's Chemical Biology and Biomolecular Structure and Biophysics training groups, Mydy's current work focuses on elucidating novel plant peptide cyclases for antimicrobials and other disease therapies. Her lab studies protein structure and function, enzyme mechanisms and plant natural product biosynthesis, working to characterize and engineer plant natural products for therapeutic and agricultural applications. She is also a member of Purdue's Center for Plant Biology.

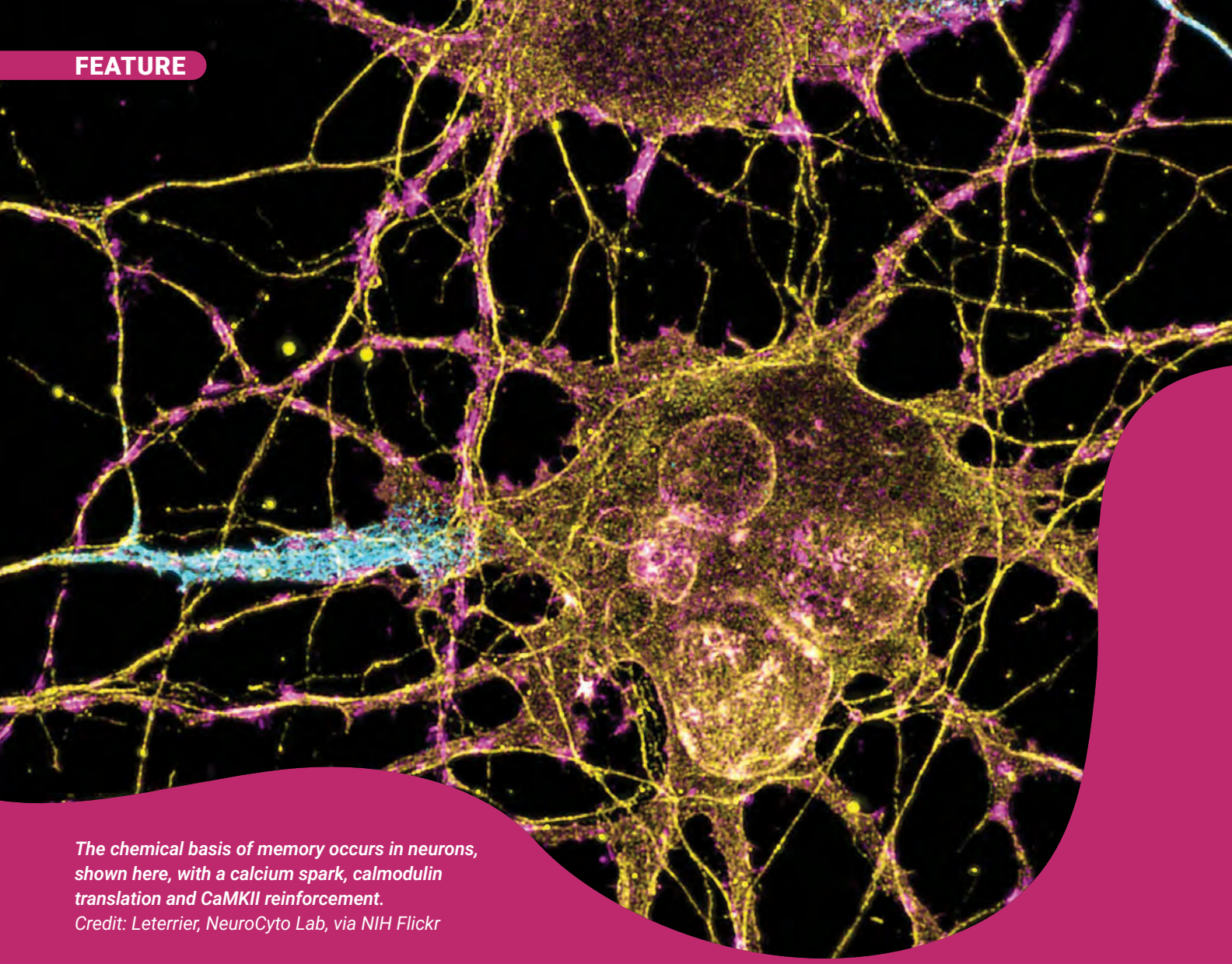
Mydy earned her bachelor's degree and Ph.D. in chemistry from the University of Wisconsin–Milwaukee. She subsequently completed postdoctoral training at the University at Buffalo, New York, and worked in industry as a senior scientist for Abbott. She was awarded the Journal of Biological Chemistry poster prize at the 2022 Enzyme Mechanisms Conference for her work in plant cyclic peptide biosynthesis.

Nuñez receives Vallee Scholar Award

James Nuñez, assistant professor of molecular and cell biology at the University of California, Berkeley, has been named a 2025 Vallee Scholar by the Vallee Foundation. Established in 2013, the Vallee Scholar Awards program provides flexible, unrestricted funding to outstanding early-career investigators conducting basic biomedical research. According to the Vallee Foundation, Nuñez's work on CRISPR-based genome and epigenome editing, "exemplifies the program's mission to support bold, curiosity-driven science that pushes the boundaries of our understanding of human biology." He will receive $400,000 to support his research.

Nuñez's research focuses on understanding DNA methylation — the most abundant epigenetic modification in humans — and its roles in health, disease and aging. His team uses advanced CRISPR technologies and stem cell models of neuronal differentiation. They aim to uncover how unique DNA methylation patterns are established in neurons and how their misregulation contributes to human disease.

Nuñez has received many awards, including a Hanna Gray Fellowship from the Howard Hughes Medical Institute, the Pew Biomedical Scholars Award and the Alfred P. Sloan Research Fellowship.



The chemical basis of memory occurs in neurons, shown here, with a calcium spark, calmodulin translation and CaMKII reinforcement.
Credit: Leterrier, NeuroCyto Lab, via NIH Flickr

The molecular orchestra of *memory*

By Courtney Chandler

What makes you remember a favorite vacation — the smell of the ocean, the taste of new foods — but forget where you left your keys? Inside the brain, the answer comes down to a carefully timed chemical choreography. At its core, memory is biochemical. The brain's ability to receive, store and recall information relies on ions, proteins and signaling pathways acting in concert to convert electrical activity into chemical changes at synapses and within neurons.

When this choreography falters, the consequences can be profound. Memory disruption underlies disorders such as Alzheimer's disease, age-related cognitive decline and learning disabilities.

Among the key players in this molecular ensemble are calcium ions, known as calcium, the calcium-binding protein calmodulin, or CaM, and calcium and calmodulin-dependent kinase II, or CaMKII. Together, they form a signaling axis that drives the cellular changes required to form and maintain memories.

A.J. Robison

Neuroscientist A.J. Robison, professor in the department of physiology at Michigan State University, describes this signaling axis as fundamental to memory formation.

"We've known for more than 30 years that calmodulin functions as a calcium sensor and drives the activity of downstream signaling cascades, including CaMKII," Robison said. "This activity is essential for the cellular correlates of learning, like long-term potentiation and long-term depression, and for actual learning itself in mice and other models."

Yet this process is complex. Knowing the players is not the same as understanding the performance. Many questions remain about how these chemical changes produce lasting synaptic effects that support memories over days, years or even a lifetime.

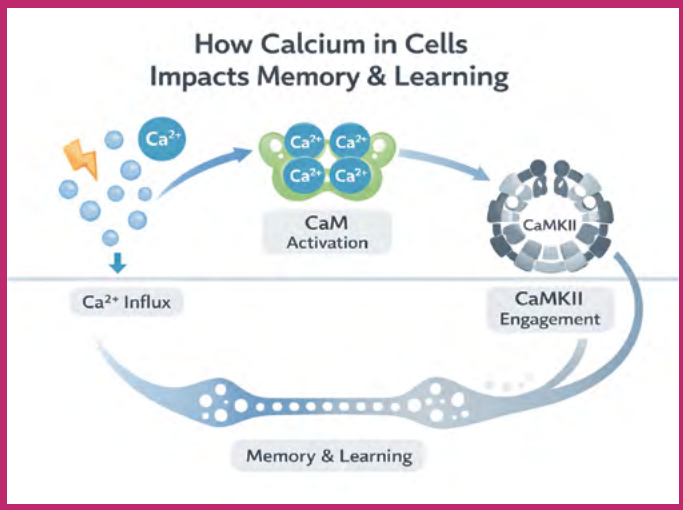
Understanding these mechanisms advances basic neuroscience and points to potential therapeutic targets for disorders that impair learning, cognition and memory. Recent research published in American Society for Biochemistry and Molecular Biology journals is helping to clarify this signaling network, revealing new layers of regulation — and unexpected opportunities for intervention.

Calcium: A chemical spark

Every memory begins with a surge. Sensory inputs trigger rapid electrical spikes, called action potentials, that race along neurons and cause neurotransmitter release. Activation of the neurotransmitter receptors causes calcium ions to flood into the cell, acting as a spark for downstream signaling.

Repeated calcium surges signal that something worth remembering may be happening. Recurrent rises in intracellular calcium trigger long-term potentiation, a sustained strengthening of synaptic connections widely regarded as a hallmark of memory formation. Over time, these connections embed patterns of activity that encode memories.

Chiho Sugimoto, a postdoctoral fellow at Vanderbilt University who earned her Ph.D. in Robison's lab, said this initial calcium spark is key to triggering the molecular cascade that underpins memory and learning. While calcium does not store memory itself, it sets the conditions that allow memories to form.

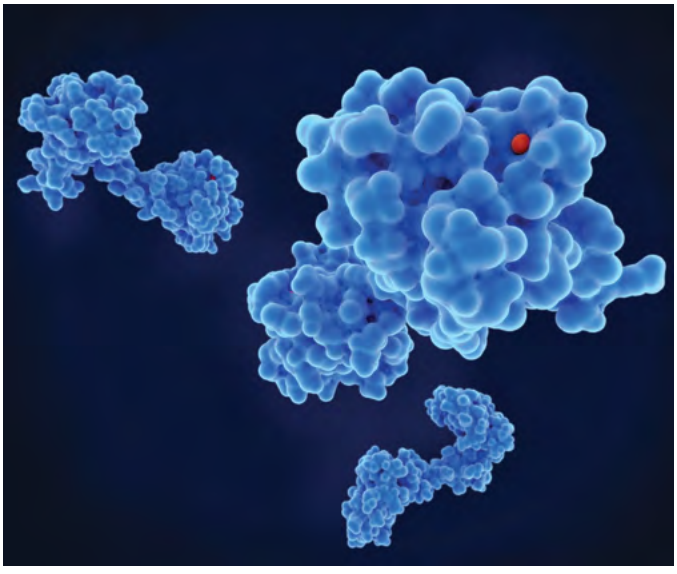


Calcium signaling links neuronal activity to memory formation and learning. Calcium (Ca^{2+}) influx triggers calmodulin (CaM) activation, which engages CaMKII to drive synaptic changes that support learning and long-term memory.
Credit: Courtney Chandler

Chiho Sugimoto

"Calcium is essential to how neurons communicate with each other by regulating the release of neurotransmitters and the strength of synapses, which are critical factors for learning and memory," she said. "Calcium can control the timing, location and magnitude of synaptic signals through pathways such as CaM–CaMKII, which can shape neuronal circuits, synaptic plasticity and gene expression, the key drivers of brain function."

A study published in the Journal of Biological Chemistry shows just how finely tuned that control can be. Using patch-clamp electrophysiology, a technique that measures electrical currents through specific ion channels, researchers studied voltage-gated ion channel activity in neurons. They focused on how retinoic acid, a vitamin A metabolite known to influence neural plasticity, modulates ion channel activity and synaptic function.



Once activated by calcium (red), the protein calmodulin (blue) interacts with downstream targets to influence synaptic plasticity, impacting learning and memory.

Researchers found that retinoic acid modulates neuronal firing by inhibiting potassium channels involved in repolarization and limiting calcium channel activation. By coordinating multiple ion channel targets, retinoic acid fine-tunes calcium entry into neurons, shaping neurotransmitter release and plasticity-related gene expression.

Vitamin A deficiency has been linked to cognitive impairments, highlighting how memory and nutrition may be connected. This work may help clarify how dietary or pharmacological interventions could support healthy brain function.

Calmodulin: The translator of calcium signals

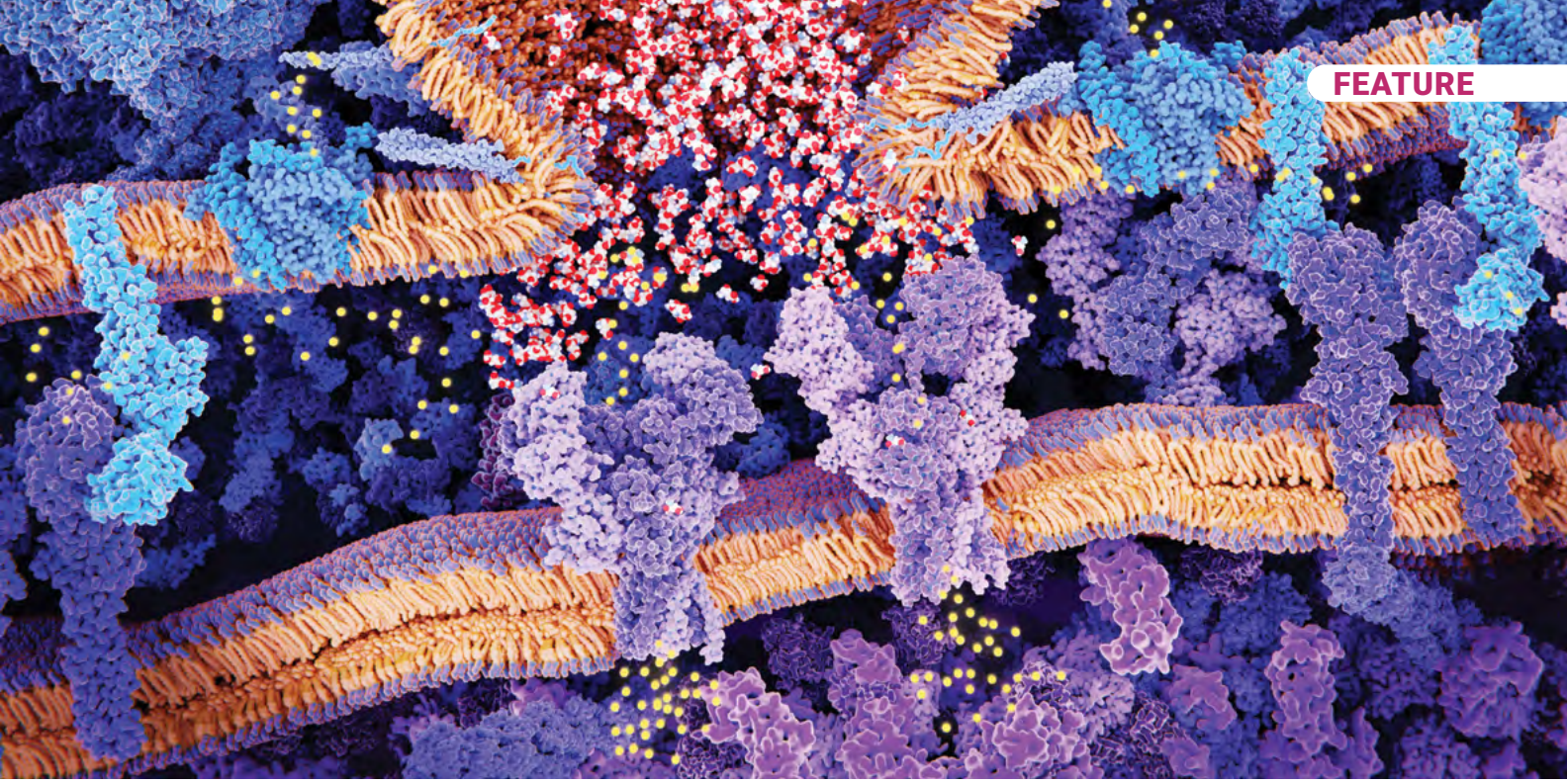
A calcium pulse is only meaningful if the cell can understand it. This translation falls to CaM, which functions as calcium's interpreter.

When calcium binds, CaM undergoes structural rearrangements that enable it to activate downstream enzymes, including CaMKII. In this way, CaM helps determine how neurons respond to activity patterns associated with learning, distinguishing background noise from signals strong enough to promote synaptic change.

CaM is not simply waiting for calcium to bind. CaM itself can also be chemically modified, and these posttranslational modifications can alter its activity and influence synaptic plasticity. Sugimoto said this layer of regulation may be critical for learning.

"CaM–CaMKII signaling is impacted by a variety of posttranslational modifications," she said. "The discovery (of new CaM modifications would) add critical detail to our model of calcium effects on neuronal activity and synaptic strength."

To this end, two complementary studies published in JBC highlighted the importance of a newly identified modification: acetylation. One study identified acetylation as a novel CaM modification and found that it is more prevalent during active learning. Mice engineered to carry a form of CaM that could not be acetylated showed reduced learning and weaker fear-related memory. A



The neurotransmitter glutamate is transported by synaptic vesicles to the presynaptic membrane. Calcium channels trigger the neurotransmitter release into the inter synaptic cleft. Glutamate binds to the NMDA (left) and AMPA receptors allowing calcium ions (yellow) to pass the postsynaptic membrane. This starts an action potential.

second study identified SRC3 as the enzyme responsible for CaM acetylation. When SRC3 was blocked in mice, the animals had difficulty strengthening synaptic connections and showed weaker fear-based learning.

Together, the studies suggest that CaM acetylation plays an important role in synaptic plasticity and learning, acting as a regulatory layer that links neural activity to memory formation. The findings suggest that targeting CaM modifications such as acetylation could offer new strategies for preserving or restoring learning and memory in disease. Sugimoto and Robison co-authored a commentary emphasizing the significance of these findings.

"What's novel about this is the discovery that calmodulin posttranslational modifications can drive differences in the activation of downstream pathways," Robison said. "These studies help model how modification of calmodulin at these residues can drive differences in learning."

Robison also emphasized the potential clinical implications of the findings.

"The SRC3 pathway could represent a (promising) approach to targeting CaM function in hippocampal neurons and may represent a novel pathway for therapeutic intervention in diseases of cognition, like Alzheimer's or cognitive disabilities," he said.

CaMKII: The molecular engine of memory

If calcium provides the spark and CaM interprets the signal, CaMKII does the heavy lifting. Once activated by calcium-bound CaM, CaMKII phosphorylates key synaptic proteins, reinforcing the connections that encode memory.

CaMKII has long been recognized as central to learning. However, precisely because of this central role, the enzyme has been viewed as an untouchable due to concerns that targeting it therapeutically could disrupt or erase established memories.

Recent studies published in JBC are beginning to address this uncertainty.

In one study, researchers temporarily inhibited CaMKII using the neuroprotective peptide tatCN19. Surprisingly, this short-term inhibition did not disrupt previously formed memories in mice, revealing unexpected stability in established memories.

This resilience is especially important when considering therapeutic applications. In pigs, temporary CaMKII inhibition reduced neuronal damage under physiological

stress, pointing to a neuroprotective effect that could translate to human conditions such as recovery from stroke or cardiac arrest.

To understand how this level of regulatory precision is achieved, a second study from the same lab examined phosphorylation of CaMKII at a specific threonine residue. The researchers found that this modification is essential for multiple forms of long-term synaptic plasticity, highlighting how tightly regulated CaMKII activity is.

Together, these findings highlight the importance of precise CaMKII regulation for learning while suggesting that the CaMKII pathway can be safely modulated without compromising stored information.

Sugimoto sees this regulatory balance as central to human health.

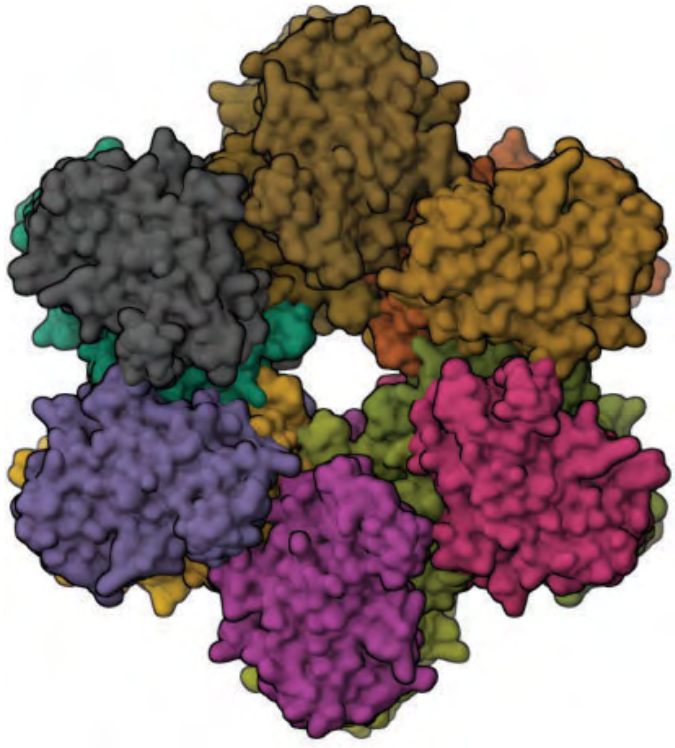
"CaM-CaMKII signaling is a central molecular mechanism for learning and memory that allows us to translate our experiences and environment into synaptic changes that encode long-term memories," she said. "Dysfunction in this signaling is implicated in diseases like mood disorders, schizophrenia, epilepsy and autism spectrum disorder, indicating that understanding how this signaling is regulated can provide us with novel therapeutic strategies."

The future of fine-tuning memory

The same chemistry that allows you to remember the details of a vacation also determines whether everyday moments are lost to distraction. Calcium sparks, CaM binding and CaMKII reinforcement together help determine what stays and what fades.

Because of the critical nature of this axis and the ubiquity of some of its components, developing therapeutics remains challenging. Among these players, CaM has been especially difficult to target because of its widespread presence in the body. However, recent research suggests that targeting specific regulatory nodes along the axis may be a promising alternative.

"CaM is ubiquitous and plays a role in so many cell types and functions that targeting it therapeutically would doubtless have myriad off-target effects," Robison said. "If we can target specific CaM modifications or target this CaMKII pathway in specific neurons in the brain through SRC3, maybe therapeutics won't have the detrimental effects that you might have by targeting calmodulin directly."



3D reconstruction of the human calcium/calmodulin-dependent protein kinase molecule. *Credit: Ozden, C., Abromson, N.L., Tomchick, D.R., Stratton, M.M. and Garman, S.C. 10.1038/s41467-025-63249-w.*

As researchers continue to untangle how calcium signals are interpreted, translated and reinforced in neurons, the picture of memory formation is becoming clearer. These insights bring the field closer to strategies that could improve and preserve learning and memory while protecting the brain, ensuring that the delicate chemical choreography underlying memory remains in step.

Courtney Chandler is a biochemist and microbiologist in Baltimore, Maryland, and a columnist for ASBMB Today.

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Patricia Becerra, ASBMB Membership Committee member and researcher at the National Eye Institute, National Institutes of Health, studies how protein structure governs function in biological pathways critical to retinal cell differentiation, survival and degeneration. Trained as a biochemist, she brings a molecular perspective to questions that drive discovery and mentorship.

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Designing *scientific careers*, in addition to experiments

By Elizabeth Stivison

Graduate students of microbe–host interactions and biochemistry work on their Career Architect plans at the Vanderbilt University Medical Center 2023.
Credit: Vanderbilt University

Students' reflections on the Career Architect course held in 2023.
Credit: Vanderbilt University

You can't live your dreams if you don't know what they are or how to reach them, yet universities often focus on rigorous science training for their PhD students and postdocs, neglecting intentional career planning. Many early-career scientists then face challenges as they come to the end of their training, with little preparation for how to build the next phase of their careers, or even how to start thinking about what they want.

In response, educators and institutions are developing more intentional, structured approaches to career preparation.

myIDP: Addressing the need

Cynthia Fuhrmann is an associate professor in the RNA Therapeutics Institute at the University of Massachusetts Chan Medical School. She has designed and implemented career preparation resources for two decades and conducts research on the pedagogy of career preparation.

Fuhrmann co-created myIDP, the Individual Development Plan for scientists, an online career planning tool hosted by the American Association for the Advancement of Science.

"University leadership is recognizing that we need to be more intentional about the career preparation of our trainees," Fuhrmann said. She said career preparation should help young scientists see themselves as whole professionals who can contribute to the scientific ecosystem in many ways, not only as professors at research-intensive institutions.

Fuhrmann began developing myIDP with her trainee experience in mind. "I saw that it would have been very helpful to have more structured ways to think about what we enjoy doing, or what's most rewarding or important to us; things that we might not really talk about as much," she said. "And then, crucially, to actually write down explicit goals."

myIDP is designed to guide users through this process. Participants first take stock of themselves by ranking over a hundred science-related skills and interests, along with life and work values.

Next, the tool synthesizes this information to generate a wide-ranging list of science-related careers that may align with each user's unique combination of skills and interests. Users are then encouraged to explore these possible paths and work with mentors to set specific, actionable goals.

They reassess their progress annually and adjust their plans as needed.

Career Architect: An alternative approach

Angela Zito

Angela Zito and Kate Stuart are assistant and associate directors, respectively, of the Biomedical Research Education and Training, or BRET, Office of Career Development at Vanderbilt University. They identified the same need for deeper introspective career preparation and pioneered a semester-long program called Career Architect to help early-career scientists design life plans.

Zito conceived of the course by combining elements of the book *Designing Your Life*, written by the founders of the Stanford Life Design Lab, where she'd taken a course, and combining them with the Clifton StrengthsFinder assessment, for which Stuart and Zito are certified coaches. "We melded the two together because it's all about finding yourself and what values you have and then applying that to where you want to go with your career and with your life," Zito said. "I saw it as a good steppingstone for grad students."

Their course first analyzes each participant's strengths, similar to the IDP, to help them understand themselves. The course then uses the Designing Your Life framework to guide self-discovery through activities such as graphing how rewarding daily tasks feel or identifying moments of flow.

Participants also write statements about what they believe the purpose of employment is and what constitutes a good life.

Kate Stuart

"A lot of people have said, 'Oh, I've never really sat down and actually thought about that,'" Stuart said. She said many people have ideas about the meaning of work and life in the back of their minds, but intentionally writing them down can be eye-opening, especially when classmates produce wildly different statements.

The course then walks participants through networking strategies, career exploration and actionable planning, all through discussion and brainstorming with fellow students. One encouraged strategy is "prototyping" career ideas through small, low-risk activities before committing to a major change.

Toward deeper career preparation

To effectively plan a career, a person needs to know who they are, what they are good at, what they value in life, and what kind of careers align with those qualities. It also requires identifying next steps to learn more, gain experience, and make meaningful progress toward long-term career and life goals.

myIDP and the Career Architect program both address all stages of this process, from thinking deeply about your life and where you get meaning and happiness, through how to put this new self-knowledge into

practice. This deep approach complements broad events such as career fairs, which can introduce students to potential career options.

Fuhrmann stressed that exposing students to career options alone is not enough. "Identifying one's career interests is a complex process. For many trainees, it means re-imagining themselves in a role that may be quite different from the academic roles they are used to or what they had originally anticipated. And then there is the question: how do I get there?"

Research shows that individuals who use strategies to set and pursue career goals are more successful by several measures than those who do not. While long-term follow-up data on the effect of IDP use is still in progress, in a 2014 study, 70 percent of postdocs using an IDP reported that the tool helped with their career planning.

As the benefits of holistic career preparation become clearer, these approaches are spreading. The Stanford Life Design Lab now reaches students and staff at institutions in nearly all 50 states and worldwide, and myIDP is used by more than 400,000 scientists.

Beyond optional career preparation

However, even the strongest resources have a common limitation: many trainees seek them out too late. "I wish people would start sooner," Stuart said. Fuhrmann echoed that concern, recalling graduate students who sought guidance only months before finishing their degrees. "They would say, 'I'm a couple of months away from graduating, and I'm just wondering, how do I learn more about these types of jobs?'" she said.

Recent Ph.D. graduates may already be familiar with one way this pitfall is being addressed: many programs now require trainees to complete an IDP. Major training grants expect trainees to complete an IDP, and many PhD programs require it regardless of funding.

However, the value of creating an IDP depends largely on the effort and thought invested. Rushing through to check a box, while better than nothing, limits its usefulness.

Fuhrmann addressed this issue, too, by piloting an IDP-centered career preparedness course required for all third-year Ph.D. students at UMass Chan, which integrates peer discussion and in-person guidance.

"There was pushback at first," Fuhrmann said. Students were initially reluctant to devote time to the course, but in end-of-term surveys, students wrote paragraphs about how helpful it actually was. It gave them confidence in their ability to build their career, and discussions with their peers were productive and made them feel less alone in their journeys.

Elizabeth Stivison is an ASBMB Today columnist and an assistant laboratory professor at Middlebury College.

Do you want to implement a course like Career Architect or the IDP at your institution?

Fuhrmann directs Professional Development Hub, pdIhub, an ASBMB-supported initiative filled with evidence-based usable modules that mentors and career offices can use to help teach about career prep.

The Life Design Lab offers courses for universities.

The Graduate Career Consortium also provides resources and a community for those in the career development field.

Getting students excited about INTRODUCTORY BIOLOGY

By John Peters

John Peters, an assistant professor of biology, works with undergraduate Nikki Heifler and postbaccalaureate student Ismar Alickovic in his lab at University of Richmond in 2025.
Credit: Courtesy of John Peters

Introductory cell and molecular biology is one of my favorite courses to teach. I love teaching early-career biologists and biochemists, but one challenge is the wide range of career interests they bring to the classroom. In an introductory biology classroom, future doctors sit next to future researchers, dentists, journalists, occupational therapists and more.

So, how do you capture the attention of such a diverse group of learners?

My strategy has been to lean into active learning and inclusive pedagogy, and I feel fortunate to have joined a teaching team in the biology department at the University of Richmond with a strong history of using evidence-based approaches. Building on this history, I largely flipped my introductory biology course, asking students to complete readings and study guides outside of class and reserving class time for hands-on learning.

Early in my career, active learning meant think-pair-share discussions or collaborative practice questions. I still use these strategies, but recently, case studies have become my most effective active learning tool. Science is done in a community, and case studies help students learn the same way. They are especially effective at pairing core biological concepts with real-world applications.

My most successful example explores the molecular mechanisms of opioids and naloxone. The learning objectives focus on protein structure and function, and cell signaling.

When I most recently taught this case study, I saw that students' learning extended well beyond the textbook. Working in groups of three or four, students analyzed molecular structures, discussed short passages and answered related questions.

The case study began with an overview of how endorphins are synthesized and trafficked through the endomembrane system. We then discussed G protein-coupled receptors and mu-opioid receptor structure and function.

Students explored agonists and antagonists by comparing endogenous and exogenous ligands and

examining how synthetic opioids structurally resemble β-endorphin. Finally, we examined the molecular mechanism of naloxone and how it saves lives.

This case study inevitably raises challenging topics.

The opioid epidemic has affected millions, and at least one student in any given classroom likely has some personal experience related to opioids. But students want to be challenged, especially when the topic feels relevant to their lives.

The classroom was loud in the best way, and student participation was high.

As I circulated through the classroom, one student shared their experience volunteering at clinics where overdoses were common.

At the end of the case study, I intentionally left time for students to share takeaways. This space allowed students to connect course material to their own lives The University of Richmond has a robust undergraduate emergency medical technician, or EMT, organization, and one EMT in my class described how she was trained to recognize an overdose and administer naloxone.

Another student asked about inhaled versus injected naloxone, sparking a discussion of why different drug delivery methods may be preferred.

Hearing additional conversations around the room made it clear that many students had more to share, and next time I teach this case study, I plan to dedicate more time to reflection.

How do you get students invested in agonism, signaling cascades and other core concepts in introductory biology? I learned that interest grows when students are given time in class to wrestle with those ideas in contexts that feel real. For me, case studies created that space and changed how students engaged with the material.

John Peters is an assistant professor of biology at the University of Richmond, where he started in 2024. The Peters lab studies the molecular mechanisms of AMPA receptor trafficking during long-term potentiation.

Backward design and beyond: LESSONS FROM A MOLECULAR GENETICS CLASSROOM

By Jeremy Hsu

I vividly remember the first exam I wrote as an instructor for our molecular genetics course. Students frantically wrote responses as the clock ticked down, their faces tense and expressions harried. Even after I warned that only a few minutes remained, every student stayed seated, many pages from finishing.

Afterward, students described feeling overwhelmed, citing both the exam's length and uncertainty about what to focus on while studying.

Later that day, I slumped in my office, acutely aware of how new I still was to teaching. Not a single student had completed the exam, and my desk was filled with pages of answers that recited nearly every fact students knew about gene expression.

Jeremy Hsu attends the 2024 National Association of Biology Teachers conference in Anaheim, California, with undergraduate and graduate students Grace Holick, Makaylee Dahms, Joelle Prate, Tammy Bui and Molly Niswender. Credit: Courtesy of Jeremy Hsu

That exam made it clear that I needed to rethink both how I taught the course and how I assessed learning. I responded by consulting mentors, colleagues and our teaching center, and by diving into the science education literature. Humbled, I began revising my teaching and assessments in deliberate ways.

First, I developed specific learning objectives, or SLOs, for each module: clear, measurable statements describing what students should know and be able to do. Students had previously shared that they were unsure what to study and felt unprepared for questions that required applying knowledge to new scenarios.

I began explicitly highlighting SLOs that emphasized higher-order skills before and after each unit, signaling the importance of critical thinking. I then redesigned my assessments to align directly with these objectives.

Second, I realized that students needed guidance in using the SLOs to direct their studying and reflect on their learning. I developed new study guides that showed students how to use the SLOs to plan and prioritize their studying.

Drawing from the literature, I also introduced metacognitive exam wrappers, guided assignments that prompt students to think critically about their learning and study strategies. I incorporated the SLOs in the wrapper to better help students reflect on their performance and mastery after each assessment.

Finally, writing the SLOs revealed just how many skills and concepts each unit contained, making it clear that assessing every objective on every exam overwhelmed students. Instead, I restructured the course to include more in-class activities and formative assessments aligned with the SLOs.

This application of backward design — starting with learning objectives and then designing assessments and curriculum accordingly — allowed me to better support students as they developed these skills.

In the decade since that first exam, these changes have transformed the course. Clearer objectives and reflective tools now help students from diverse backgrounds take ownership of their learning, leading to deeper engagement and stronger performance, and I have observed student interest, motivation and confidence increase.

The experience continues to remind me that effective teaching is not static but a continual process of learning, adapting and growing alongside my students.

Jeremy Hsu is an associate professor of biology and the assistant director for undergraduate research and creative activities at Chapman University. A biology education researcher, Jeremy is dedicated to promoting evidence-based teaching through his research, teaching and service.

When biochemistry STOPPED BEING SCARY

By Fatahiya Kashif

Teaching, for me, did not begin as an act of confidence. It began with a question: Why are so many students afraid of biochemistry?

Medical student Sharjeel from Fazaia Medical College, Air University, Islamabad, Pakistan, solves a puzzle that tests conceptual understanding of metabolic pathways, regulatory checkpoints, substrate flow and associated metabolic disorders in 2023.
Credit: Courtesy of Fatahiya Kashif

Early in my career, I noticed a troubling pattern. Students did not merely find biochemistry difficult; they avoided it. They described it as abstract and dry, overloaded with pathways and names detached from meaning. Many had already decided, before entering the classroom, that biochemistry was something to "survive," not to understand.

Entering academia as a woman in a resource-limited region, I was already familiar with barriers such as limited funds, institutional inertia and the quiet skepticism that often greets young female faculty. Watching students retreat from a subject that explains life at its most intimate level felt like a deeper loss.

If biochemistry is the language of living systems, why had it become a language of fear? At first, I followed the conventional path: slides, textbooks, diagrams and exams. The more I taught, the more I sensed that the problem was not students' ability but our mode of translation. We were presenting a three-dimensional, dynamic molecular world through flat images and rushed lectures, expecting wonder to emerge automatically. I wanted to change that.

I tried to access educational tools, molecular models, kits and manipulatives, anything that could bring molecules into students' hands. Financial constraints were real. Institutional support was limited, and innovation was often discouraged.

I was advised to "focus on finishing the syllabus," "maintain decorum" and "avoid experimentation."

When I finally managed to acquire a small set of physical models, they were later stolen. It felt symbolic. Every time I tried to build something new, something seemed to pull it back into absence.

Gradually, I began building my own models and discovered a capacity for creative design I did not know I had. Cardboard, clay, magnets, beads, flexible wires and pipe cleaners. Using whatever I could find, I built molecular models students could hold and assemble.

I created pathway puzzles, hormone-signaling Rube Goldberg machines, metabolic board games and immune receptor kits. Not as "extras," but as central teaching tools. If biochemistry is about interactions, flows and transformations, students should be able to touch them. That decision changed everything.

My classroom slowly transformed into a workshop. Students stopped asking, "Will this be on the exam?" and started asking, "Can we try this again?" They delved deeper into pathways, simulated disease models and built receptors. They failed, adjusted, laughed and tried again. I was no longer the only one explaining biochemistry; the room itself became part of the explanation.

What surprised me most was not just improved understanding, but a shift in emotional state. Fear softened into curiosity, silence into debate and memorization into ownership. Word spread.

As an external examiner, I began carrying these games and models to medical colleges across Pakistan. The first reaction was almost always the same: nervous smiles, stiff posture and the expectation of judgment.

But, when I invited students to assemble, compete, diagnose and design, the room changed. Hands rose, laughter broke out and groups formed. I watched students who believed they "couldn't do biochemistry" suddenly teach it to each other.

Those students taught me something essential: engagement is not decoration. It is cognition. What began as a solitary effort became a collaborative one. Through sustained feedback and classroom interaction, the models evolved into tools co-created with my students.

Looking back, my teaching did not evolve because I discovered a better technique. It evolved because I refused to accept a myth: that biochemistry is inherently boring and hard. It is neither. It is visual, mechanical, logical and alive. When we design learning environments that honor those qualities, students do not run from biochemistry. They run toward it.

Every time I watch that happen, I am reminded that teaching is not the transfer of knowledge. It is the continuous redesign of the conditions under which wonder becomes possible.

Fatahiya Kashif is a biochemistry professor at Federal Medical College in Islamabad, Pakistan.

A handmade magnetic translation puzzle constructed from foaming clay and magnetic materials by Fatahiya Kashif, allowing medical students to physically assemble messenger RNA, ribosomes, transfer RNAs and amino acids to understand the dynamics of protein synthesis. *Credit: Courtesy of Fatahiya Kashif*

In memoriam: Alan G. Goodridge

Alan G. Goodridge, a longtime associate editor of the Journal of Biological Chemistry, died Aug. 8, 2025, at age 88. He had been an American Society for Biochemistry and Molecular Biology member since 1971.

Born April 2, 1937, in Peabody, Massachusetts, Goodridge developed a passion for birdwatching that shaped his scientific path. He received a B.S. in biology from Tufts University and a Ph.D. in zoology from the University of Michigan. During his Ph.D., Goodridge studied how migrating birds fuel their wing muscles and identified a critical role for fatty acid biosynthesis in maintaining energy balance. After his Ph.D., he completed postdoctoral research in biochemistry at Harvard Medical School.

Goodridge's academic career included being an assistant professor of physiology at the University of Kansas Medical Center, working in the Banting and Best Medical Research Department at the University of Toronto, being a professor of pharmacology and biochemistry at Case Western Reserve University, and becoming chair of the department of biochemistry at the University of Iowa.

Goodridge became best known for his work in lipid metabolism. He was part of the first generation of biochemists to use the techniques of molecular biology to investigate the synthesis of lipogenic enzymes in response to dietary and hormonal changes. His laboratory was the first to clone cDNAs for malic enzyme and fatty acid synthase, key enzymes in the synthesis of fatty acids. Most notably, he characterized the transcriptional regulation of these enzymes by thyroid hormone via the thyroid hormone receptor. This work highlighted the importance of DNA-binding proteins in regulating intermediary metabolism and identified these proteins as potential targets for drugs to treat obesity and hyperlipidemia, a condition of excess fat in the bloodstream.

In 1996, Goodridge switched from academic to administrative roles. He began as dean of the College of Biological Sciences at the Ohio State University, followed by an appointment as Provost of the University of Toledo, Ohio, and later, as provost and acting president of Alfaisal University in Riyadh, Saudi Arabia. Goodridge was instrumental in establishing four colleges at Alfaisal: Life Sciences, Medicine, Engineering and Business.

Throughout his career, Goodridge trained numerous graduate students and postdoctoral fellows. Through his mentorship, many went on to productive careers at universities and in academic research. "He was always a stickler for precision in scientific writing and public speaking, a process few of his mentees will forget," Lisa Salati, a former postdoctoral fellow in the laboratory and current professor emeritus at West Virginia University, said.

Goodridge is survived by his two sons, three grandchildren and his wife.

In memoriam: Stuart A. Kornfeld

Stuart Arthur Kornfeld, a physician–scientist and educator, died Aug. 17, 2025, in St. Louis at 88 from complications of Parkinson's disease. Kornfeld was the David C. and Betty Farrell Distinguished Professor Emeritus at Washington University School of Medicine. A pioneer in glycobiology, his discoveries transformed cell biology and medicine, and his mentorship shaped generations of physician–scientists. Kornfeld was a member of the American Society for Biochemistry and Molecular Biology for more than 50 years and was awarded the society's highest honor, the Herb Tabor Award, in 2012.

He was born October 4, 1936, in St. Louis to Max and Ruth Kornfeld. His father, a dentist and faculty member at Wash U, nurtured his interest in science. As a student, Kornfeld excelled academically and athletically, captaining his high school basketball and baseball teams. He earned

a bachelor's degree from Dartmouth in 1958 and a medical degree from Washington University in 1962. After completing his residency at the Barnes Jewish Hospital and training at the National Institutes of Health, he joined the Washington University faculty in 1966.

Kornfeld made groundbreaking contributions to the study of glycoproteins, molecules made of sugars attached to proteins that play crucial roles in cell function. His research revealed how sugar chains are built and modified and identified the mechanism by which the mannose-6-phosphate signal that directs enzymes to lysosomes is specifically added to lysosomal enzymes. These discoveries clarified how proteins fold, move within cells and reach their destinations. They also advanced the diagnosis and treatment of lysosomal storage diseases. His work influenced many fields, including genetics, metabolism, immunology, microbiology and oncology.

Much of his research was done with his wife, Rosalind Hauk Kornfeld, a fellow scientist and professor at Wash U. Together they helped lead the field of glycobiology, publishing numerous foundational papers, including one cited more than 4,500 times. Rosalind died in 2007 after 48 years of marriage.

Over his career, Kornfeld published more than 250 papers and held continuous NIH funding for five decades. In 2018, he co-founded M6P Therapeutics, a biotechnology company developing enzyme replacement therapies for lysosomal storage diseases such as Tay–Sachs, Gaucher, Pompe and mucopolysaccharidosis.

In addition to his research, Kornfeld was dedicated to training physician–scientists. He also co-directed the Division of Hematology–Oncology from 1976 to 1992 and the Division of Hematology from 1993 to 2009. He was elected to the National Academy of Sciences, the National Academy of Medicine and the American Academy of Arts and Sciences. His honors included the Passano Award, the Kober Medal, the E.B. Wilson Medal and Washington University's Distinguished Faculty Award.

He is survived by his daughter Katherine Kornfeld; his son, Kerry Kornfeld; six grandchildren; and two great-grandchildren. He was preceded in death by his daughter Carolyn Kornfeld Lesorogol.

In memoriam: David Baltimore

David Baltimore, Nobel laureate and president emeritus at the California Institute of Technology, died Sept. 6, 2025, at the age of 87. Baltimore was a world-renowned researcher, educator and scientific advocate who made significant contributions to molecular biology and genetics. He was a member of the American Society for Biochemistry and Molecular Biology for over 50 years.

Born in New York City in 1938, Baltimore earned his Ph.D. from Rockefeller University in 1962. He conducted postdoctoral and early career work on viral replication and enzymology at the Massachusetts Institute of Technology, Albert Einstein College of Medicine and the Salk Institute for Biological Studies. While at Salk, he met a fellow scientist, Alice Huang, whom he married in 1968.

In 1968, Baltimore joined MIT as an associate professor of microbiology, where he and Huang studied an RNA-dependent polymerase in a livestock virus. They expanded this work to tumor-causing RNA viruses that infect mice and chickens, leading to the ground-breaking discovery of reverse transcriptase. This discovery challenged the central dogma of molecular biology and led to the classification of retroviruses, which use RNA to make viral DNA and includes human immunodeficiency virus, or HIV.

For this discovery, Baltimore was awarded the Nobel Prize for physiology or medicine in 1975 along with Howard Temin and Renato Dulbecco.

Before receiving the Nobel Prize, Baltimore was widely recognized for his contributions to biomedical research. He received the Gustave Stern Award in Virology in 1970, Eli Lilly and Company Award in Microbiology and Immunology in 1971, and was elected to the National Academy of Sciences and American Academy of Arts and Sciences in 1974.

As director of the Whitehead Institute for Biomedical Research, Baltimore continued pioneering research, including discovering the immune-regulating transcription factor NF-κB with Ranjan Sen. His lab also produced key findings, such as George Daley's work on the fusion protein BCR–ABL, which results from the combination of the BCR and ABL genes. Daley and Baltimore demonstrated that this protein was a driver of the cancer chronic myeloid leukemia, which helped pave the way for the anticancer drug imatinib. Baltimore's lab also discovered and characterized the genes RAG1 and RAG2, which are crucial for development of the adaptive immune system.

In 1997, Baltimore was appointed president of the California Institute of Technology. He stepped down in 2005 but continued his research until 2019 as president emeritus and a distinguished professor of biology. At the time, his lab studied the immune system and developed viral vectors to enhance the anticancer immune response. His lab also continued to focus on basic genetic concepts such as homologous recombination. A postdoctoral fellow in his lab, Matthew Porteus was the first to demonstrate the application of precise gene editing in human cells.

He received many honors recognizing his contributions to science. In 1999, President Bill Clinton awarded Baltimore the National Medal of Science. His contributions to biomedical research were also recognized internationally — he received the Canada Gairdner International Award in 1974 and was elected as a foreign member of the Royal Society in the United Kingdom in 1987. In 2019, Caltech founded a biochemistry and molecular biophysics graduate fellowship program in honor of Baltimore. His received the Laske–Koshland Special Achievement Award in Medical Science 2021 in recognition of his lifetime of achievements.

Baltimore is survived by his wife, daughter and granddaughter.

In memoriam: Michael J. Chamberlin

Michael J. Chamberlin, an editorial board member of the Journal of Biological Chemistry and a pioneer in the study of transcription, died on Nov. 1, 2025, at age 88. He was an American Society for Biochemistry and Molecular Biology member for nearly 60 years. He was also an elected member of the National Academy of Sciences and the American Academy of Arts & Sciences.

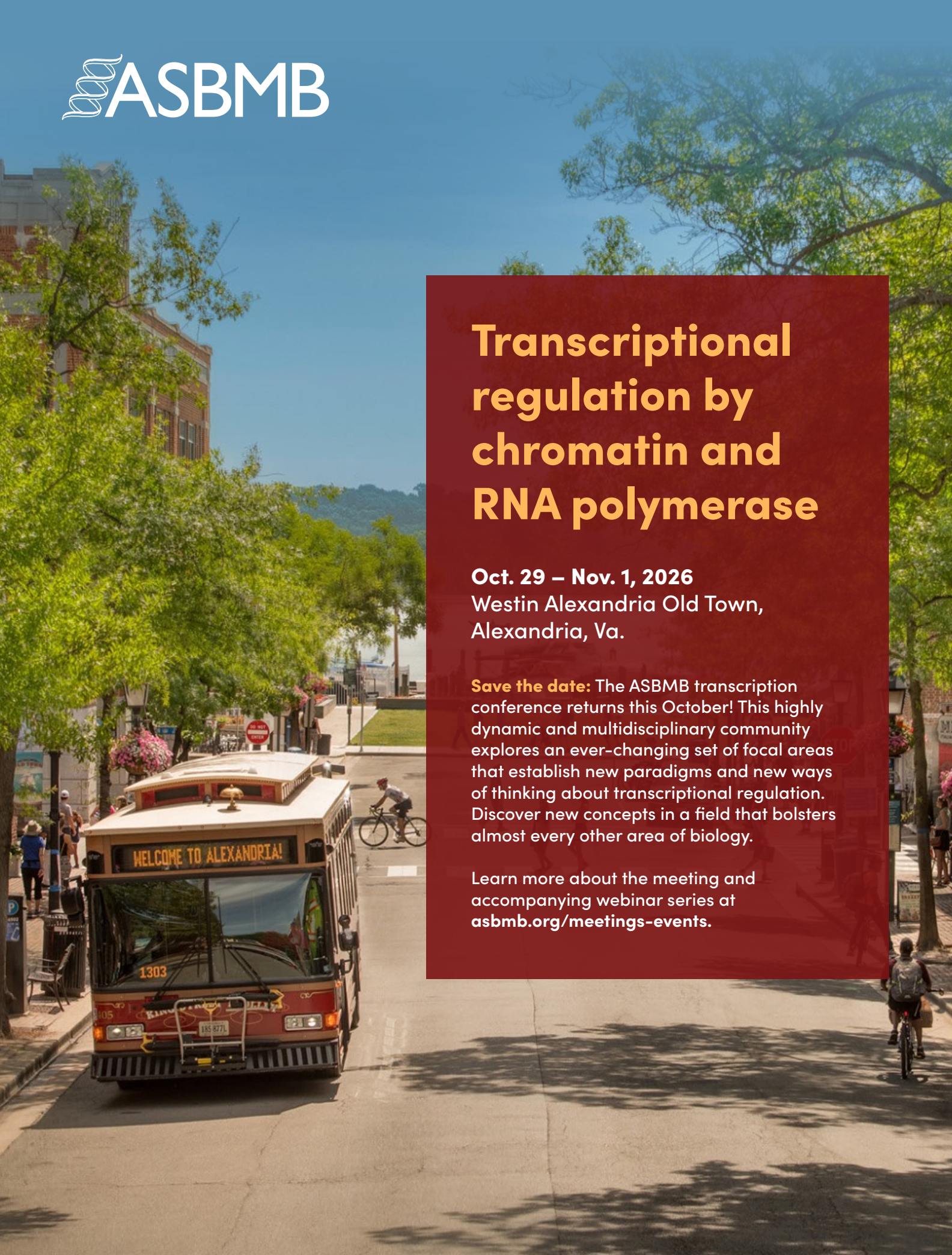
Born June 7, 1937, in Chicago, Chamberlin received a B.S. in chemistry from Harvard University and a Ph.D. in biochemistry from Stanford University. After earning his Ph.D., Chamberlin joined the University of California, Berkeley, as a professor of biochemistry and molecular biology.

Chamberlin advanced the study of RNA polymerases and transcription regulation. In graduate school, Chamberlin worked with Nobel prize–winning biochemist Paul Berg and was the first to isolate RNA polymerase from E. coli.

Chamberlin discovered that RNA polymerases regulate gene activity, a function that was unknown at the time. He showed that transcription begins when RNA polymerase binds to DNA and locates a specific promoter. In 1974, he published three JBC papers that led to a model for RNA chain initiation by RNA polymerase.

In 2021, the Stanford Medicine Alumni Association honored Chamberlin with the Arthur Kornberg and Paul Berg Lifetime Achievement Award in Biomedical Sciences.

Chamberlin is survived by his wife, Caroline Kane, a professor in residence emerita at UC Berkeley, and his brothers Peter, Steve and Tom.



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