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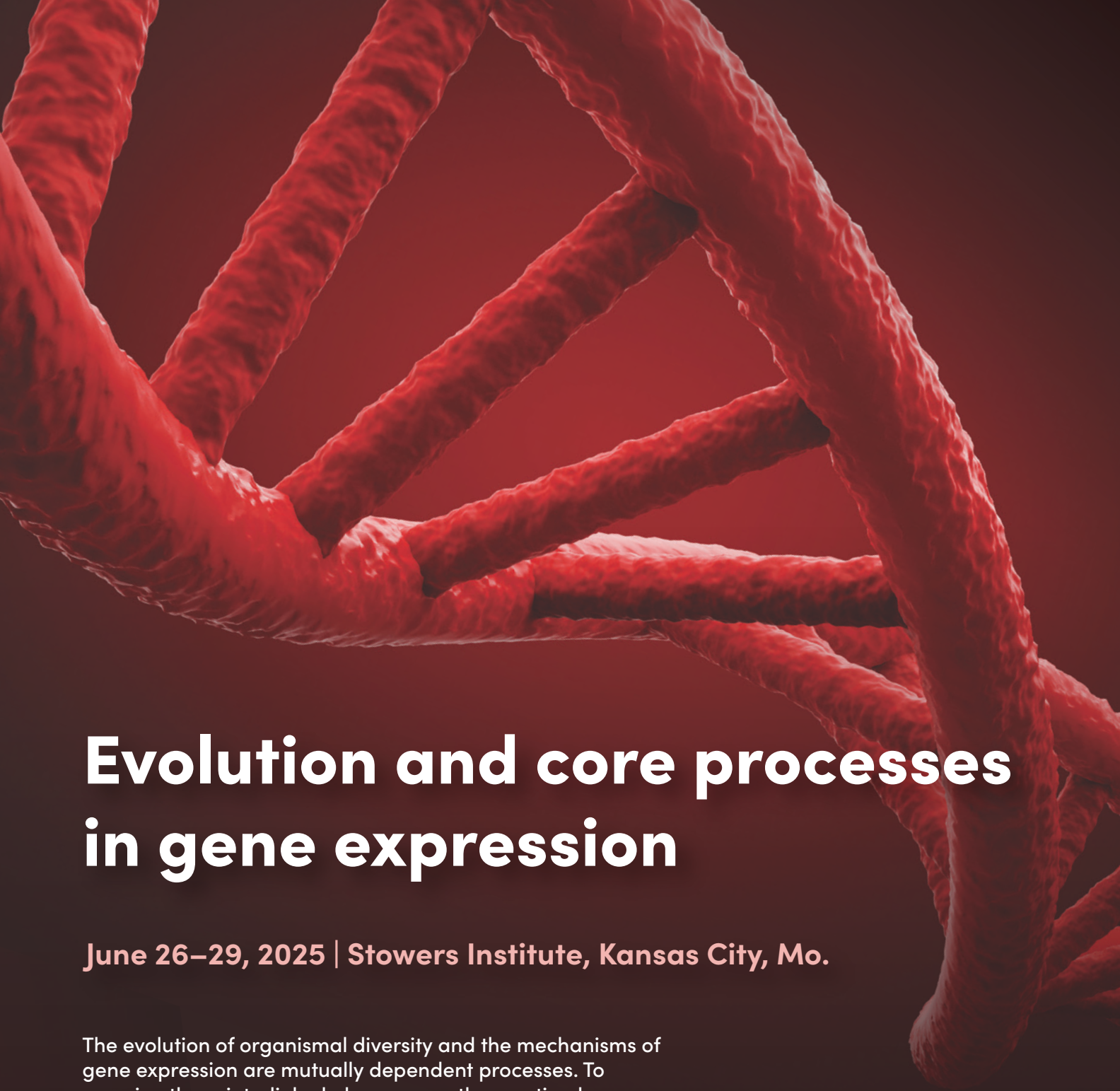
ASBMB TODAY

THE MEMBER MAGAZINE OF THE AMERICAN SOCIETY FOR BIOCHEMISTRY AND MOLECULAR BIOLOGY

Dynamic stem cells revolutionize regenerative medicine



Evolution and core processes in gene expression

June 26–29, 2025 | Stowers Institute, Kansas City, Mo.

The evolution of organismal diversity and the mechanisms of gene expression are mutually dependent processes. To examine these interlinked phenomena, the meeting has an interdisciplinary focus, ranging from the fundamental mechanisms of the cis-regulatory code to the phenotypic consequences in development and evolution.

The meeting will bring fresh perspectives from experts in different fields to obtain a deeper understanding of core life processes from a gene expression, developmental, and evolutionary perspective.

Scan the QR code to learn more, or discover other ASBMB conferences at **asbmb.org/meetings-events**.

asbmb.org/meetings-events/
gene-expression-2025

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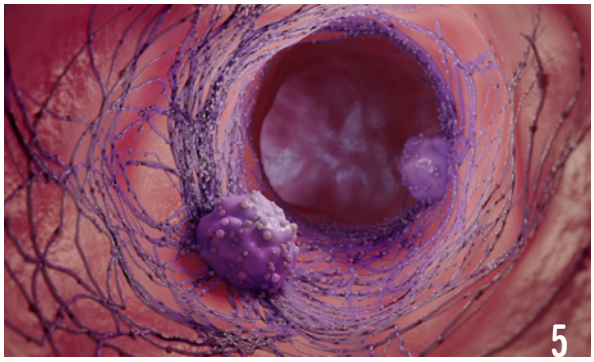

FEATURE

Front cover: Induced stem cell–derived neurons, courtesy of Frank Edenhofer

PERSPECTIVES

MEMBER NEWS

ASBMBTODAY
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A strategic plan for ASBMB

Ensuring a strong vision and setting priorities through challenging times

By Joan Conaway


JOAN CONAWAY

On a wide-open road, one might not need a GPS. A general direction — perhaps west — is enough to set course. But when route choices become more complex, twist or even abruptly close, directions are essential. This spring, the American Society of Biochemistry and Molecular Biology released a forward-looking strategic plan to serve as our GPS for the society's next 3–5 years. The long-term destination? A vibrant future serving the molecular life sciences.

I want to take this moment to share with you what the strategic plan means for the organization and initial steps ASBMB will take in the coming years to bring it to fruition. ASBMB faces very different terrain than past decades, and significant external changes face all of science. The plan is an essential road map for ASBMB to serve evolving organizational and member needs in the next five years and sets key priorities in these times. These include affirming our role in facilitating scientific excellence, sustaining our advocacy, inspiring and supporting our community and committing to efficiency and financial sustainability so we can serve the field and society for the long-term.

First, the new plan updates ASBMB's mission to anchor us in our larger purpose: discovery and community. Thus, it defines ASBMB's mission to build and empower a broad community of molecular life scientists to advance discovery.

Second, we set a bold vision: the society will catalyze the infinite potential of molecular life science. Simply but profoundly, the scientific knowledge we develop today fuels tomorrow's biotechnological and medical breakthroughs, and we affirmed that truth through our vision, as well as a core set of values that will guide all our work.

Third, we set five goals with tangible objectives, focused on what is possible in a 3–5-year period. There are many ways ASBMB could serve our community, and while we can do many things, we cannot do everything, nor can we do it all at once. ASBMB must be intentional about what it undertakes, how efficiently it does it and how fast. Also, as I shared in an article with ASBMB treasurer Russell DeBose–Boyd, while ASBMB has substantial reserves, it has a growing annual deficit that we must address.

Reflecting ASBMB's role as a mem-

ber society, the strategic planning process began with extensive volunteer and member input. We heard that members rely on ASBMB to drive scientific excellence, enable communication with external stakeholders and facilitate career development, in part through networking and mentorship.

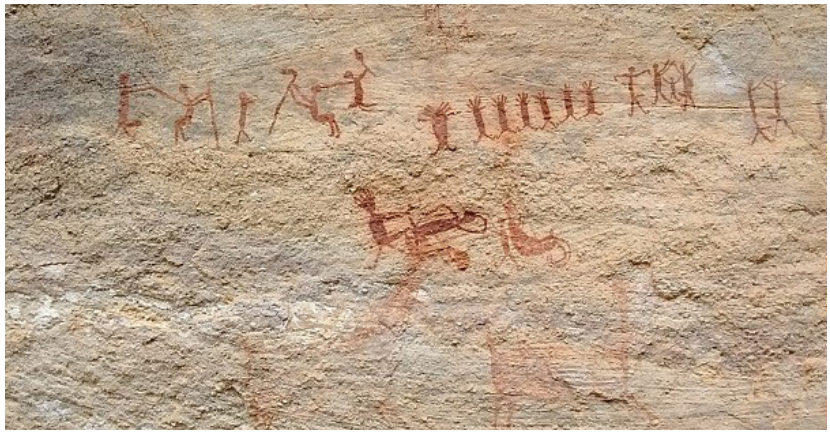
Looking ahead, Council will be asking ASBMB committees to develop implementation recommendations. Committees will evaluate how existing programs can be adapted to align with the plan and identify potential capacity by assessing program priorities. It is important to note that this plan is not intended to document every activity or initiative of ASBMB.

Council is as confident as ever about ASBMB's future and the society's role to advance molecular bioscience discoveries. ASBMB is in the process of planning for adaptation, innovation and growth that will ensure a strong foundation for ASBMB's second full century of impact. In the coming weeks and months, ASBMB will ask members to engage in propelling our science and community in myriad ways. I hope that when opportunity comes knocking for you with ASBMB — whether it be volunteering, engaging in advocacy efforts, publishing your research or attending meetings — you'll answer the door and help ASBMB catalyze the infinite potential of molecular life science.

Joan Conaway (Joan.Conaway@UTSouthwestern.edu) is a professor of molecular biology and the vice provost and dean of basic research at the University of Texas Southwestern Medical Center. She is ASBMB's president.

Liver enzyme holds key to adjusting to high-protein diets

By Andrea Lius



The Paleolithic diet mimics what human ancestors ate before the advent of agriculture. Doctors often prescribe this low-carbohydrate, high-protein diet to help manage weight and glucose levels.

A recent study by Pierre Maechler's group at the University of Geneva, published in the **Journal of Biological Chemistry**, investigated the role of the liver enzyme glutamate dehydrogenase, or GDH, in short-term adaptation to a high-protein diet.

GDH, encoded by the gene GLUD1, is important in amino acid metabolism and gluconeogenesis, a biochemical pathway in the liver that synthesizes glucose from noncarbohydrate precursors. When food is present, the intestine is the main supplier of glucose to the brain, and gluconeogenesis in the liver halts. However, when a constant supply of glucose is unavailable, the liver takes over this responsibility by means of gluconeogenesis, mainly by breaking down amino acids from a replenishable source: skeletal muscles.

In humans, Maechler explained, known GLUD1 mutations result in GDH gain of function and cause congenital hyperinsulinism/ hyperammonemia syndrome.

Hyperinsulinism causes hypoglycemia, a severe condition for newborns that may hinder neurodevelopment. As they age, these children are prone to epilepsy and possible mental disabilities. On the other hand, an abnormally high level of ammonia in the blood, hyperammonemia, can be life-threatening. Because GDH gain-of-function mutations result in hyperammonemia, Maechler's group expected that removing the enzyme would produce a low level of ammonia, or hypoammonemia — however, this was not the case.

"A surprising thing was when we knocked out GLUD1 in the (mouse) liver; instead of experiencing hypoammonemia, the animals experienced hyperammonemia," Maechler said. "Basically, there's this kind of bell-shaped effect of GDH function in terms of hyperammonemia."

Maechler and colleagues also showed that a high-protein diet, coupled with the absence of liver GDH, causes hyperammonemia in mice and, consequently, high ammonia in the blood and urine. This high level of ammonia made the blood more alkaline, and the mice had to significantly reduce their physical activity to maintain proper blood pH through compensatory slowed breathing.

"The mice can't handle fasting periods as well without GDH," Maechler said.

The researchers also found that while the expression of GDH is homogeneously distributed throughout the liver, the level of its activity is not. They monitored GDH enzymatic activity in the liver using a nitro blue tetrazolium assay and found that GDH is significantly more active in the area near the central vein than near the portal vein.

In future studies, Maechler plans to investigate GDH function in prediabetic patients, who often have fatty livers.

"We showed that you need robust GDH activity to maintain a high-protein diet," Maechler said.

"But when you have fatty liver, what does your GDH function look like? And can we still recommend a high-protein diet to these patients?"

DOI: 10.1016/j.jbc.2024.107473

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.

From the journals: JBC

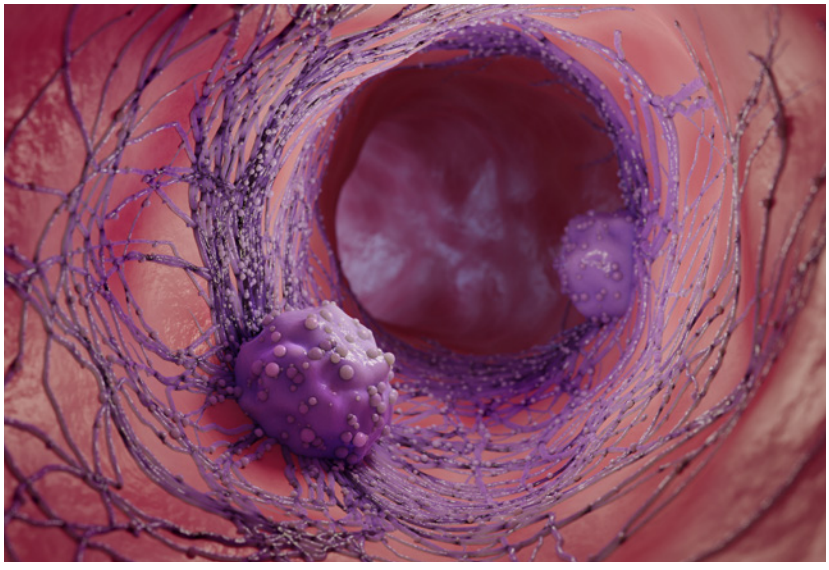
Linking modified cysteines to cell migration. Recognizing protein tags for degradation. Disrupting C. difficile toxin production. Read about papers on these topics recently published in the *Journal of Biological Chemistry*.

By Emily Ulrich

Linking modified cysteines to cell migration

Redox signaling includes the posttranslational modification S-glutathionylation, which results in an oxidized cysteine residue with glutathione attached via a disulfide bond. In cells, over 2,000 cysteine residues receive this modification, but scientists cannot easily assess which S-glutathionylation events connect to a given biological process. For example, researchers want to identify redox signaling proteins that drive cell migration, a process already known to involve S-glutathionylated proteins that regulate embryogenesis and maintain healthy tissues but can also lead to cancer metastasis under pathological conditions.

Dhanushika Kukulage from Drexel University and a team of researchers in the U.S. published a method in the **Journal of Biological Chemistry** to identify cell migration–related proteins with S-glutathionylated cysteines. Their platform uses proteomics, bioinformatics and biological screening. First, they used bioorthogonal chemistry and mass spectrometry to find proteins with the S-glutathionylation modification and compared this list with previously identified cell migration proteins. Next, they selected nine proteins based on activity classification for analysis in an oxidative stress–induced breast cancer cell migration assay. Expression of three of those proteins, the serine/threonine phosphatase PP2Cα, cytoskeletal reorganization protein ARHGEF7 and integrin signaling protein NISCH, increased oxidative stress and thus enhanced migration. The authors showed that this enhanced ability returned to baseline when they mutated the glutathionylated cysteine to serine, indicating a glutathionylation-dependent effect on migration.



An illustration of cancer cells. This study examined a breast cancer cell line to assess proteins involved in cell migration.

PP2Cα expression showed a large surge in cell migration, so the researchers chose this protein for further study. They determined that glutathionylation disrupts PP2Cα interactions with the kinases JNK, ERK1/2 and MEK4, which increases their phosphorylation levels and may enhance cell migration–related signaling.

Future studies will clarify how S-glutathionylation of ARHGEF7 and NISCH increases migration under oxidative stress. Finally, the authors emphasized the versatility of their platform; with different biological screens, researchers can investigate additional cellular processes and identify the associated S-glutathionylated proteins active in redox signaling.

DOI: 10.1016/j.jbc.2024.107784

Recognizing protein tags for degradation

The E3 ubiquitin ligase MARCHF6 is part of the N-degron protein degradation pathway that maintains protein quality control. MARCHF6 recognizes proteins with an N-terminal acetylation tag called Ac/N degrons and ubiquitinates them to drive proteolysis. Previously, scientists determined

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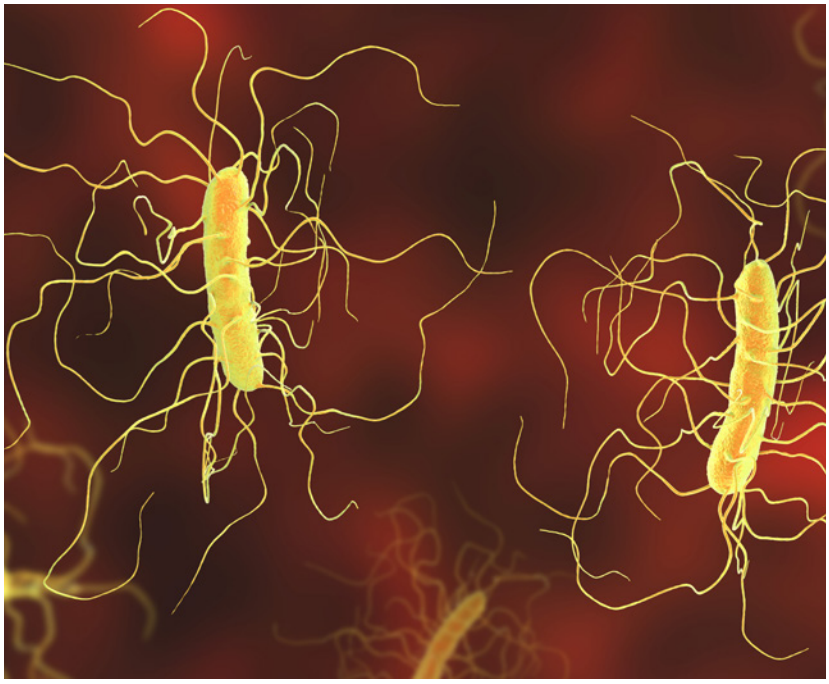


An illustration of Clostridioides difficile bacteria with peritricous flagella

that MARCHF6 suppresses the iron-dependent cell death pathway ferroptosis which is associated with numerous diseases, including cancer and neurodegeneration. Due to MARCHF6's complex structure of transmembrane helices and cytoplasmic domains, scientists do not know the structural details of MARCHF6's function.

Jihye Yang from Pohang University of Science and Technology and a group of researchers in South Korea examined the cytosolic domains of MARCHF6 and published their findings in the **Journal of Biological Chemistry**. The authors identified the region of MARCHF6 that recognizes acetylated N-terminal protein substrates and termed it the Ac/N domain because it distinguishes between acetylated and nonacetylated targets. The researchers also determined that MARCHF6-mediated degradation of the G-protein regulator RGS2 and the lipid droplet protein PLIN2 makes cells more susceptible to ferroptosis. They mutated the MARCHF6 Ac/N domain to determine that increased susceptibility to ferroptosis depends on MARCHF6's ability to recognize acetylated RGS2 and PLIN2.

Future work will focus on dissecting MARCHF6's substrate-dependent role in ferroptosis regulation. These findings could drive therapeutic developments for ferroptosis-related diseases, including cancer and cardiovascular diseases.
DOI: 10.1016/j.jbc.2024.107731

Disrupting C. difficile toxin production

Clostridioides difficile infects the colon and causes hundreds of thousands of infections annually in the U.S. Scientists want to find treatment options that target C. difficile's unique pathophysiology, such as the biosynthesis of the toxins TcdA and TcdB, to avoid killing beneficial bacteria that help block pathogen colonization. Ravi Marreddy from Texas A&M University and a team of researchers in the U.S. recently published their work in the **Journal of Biological Chemistry** on the discovery of plant-derived compounds that inhibit toxin production but do not abolish bacterial growth. They investigated plant-derived compounds and selected the compound enoxolone, a licorice metabolite, for further study due to its potency in stopping C. difficile toxin production.

The researchers used affinity-based proteomics and gene silencing to show that enoxolone halts toxin production by inhibiting adenine deaminase, which drives purine metabolism, and the adenosine triphosphate, or ATP, synthase, which is involved in energy production. The authors used surface plasmon resonance and isothermal titration calorimetry to show that enoxolone binds directly to the enzyme adenine deaminase. In addition, the authors used high-performance liquid chromatography to determine that enoxolone moderately reduced cellular ATP levels.

Future research will investigate how adenine deaminase and ATP synthase connect to toxin production and whether other enzymes in purine metabolism could become drug targets. However, in mice with C. difficile infection, treatment with enoxolone causes multiple side effects, so researchers will need to discover compounds related to enoxolone that mitigate these side effects to come up with possible treatments for C. difficile infections.
DOI: 10.1016/j.jbc.2024.107839

Emily Ulrich is the ASBMB's science editor.

A proteomic hunt for phosphosites in the aging brain

By Hallie Blevins

Proteins are cellular workhorses, executing important tasks based on their specific functions. However, misfolded, mutated, inactive or overactive proteins are often the culprits behind disorders, including neurodegenerative diseases. Researchers need a greater understanding of how aging drives protein dysfunction to develop strategies to slow or even reverse these diseases.

The proteomics field has advanced significantly with the evolution of mass spectrometry, which allows scientists to identify and analyze proteins on a molecular level.

Uma Kanta Aryal, a research associate professor at Purdue University, identifies and analyzes changes in protein phosphorylation patterns and sites, also called phosphosites, which are significant contributors to Parkinson's and Alzheimer's diseases. He and his colleagues published their recent work in **Molecular & Cellular Proteomics**.

Cells have mechanisms to activate and deactivate proteins, ensuring smooth cellular processes and preventing proteins from malfunctioning. Posttranslational modifications, or PTMs, are critical in this regulation, though they often go underrecognized.

"Many people focus on proteins going up and down in abundance," Aryal said. "But proteins are not only active when their expression goes up, it's more related to their PTMs."

Phosphorylation, a common PTM, is often implicated in disease. Protein hyperphosphorylation is responsible for aberrantly active proteins in several diseases. One example is tau, the protein that forms neurofibrillary tangles contributing to neuronal loss in Alzheimer's and Parkinson's diseases.

Aryal's recent study looked at proteins in the brains of young, middle-aged and old mice using a multi-enzyme digestion approach for analysis by liquid chromatography–tandem mass spectrometry. This method breaks down proteins into smaller peptide pieces to identify hidden alterations: the PTMs. By examining these fragments, Aryal can detect and locate the subtle changes.

"There is a precise reason for each residue being modified," he said. "That's where I got excited — it's not only the identification of those markers but characterizing them. Are they phosphorylated? If they are phosphorylated, where are they phosphorylated and what are the dynamics? How does this phosphorylation communicate with other PTMs?"

Aryal found increased abundance and activity of many kinases, enzymes that are responsible for phosphorylating other proteins, as well as changes in phosphorylation levels in proteins associated with neurodegeneration, including tau, NEFH and DPYSL2 in older mice.

Tau is widely studied due to its implication in Alzheimer's and Parkinson's diseases. Many phosphorylation

sites of tau have already been mapped; however, Aryal located two additional tau sites in older mice.

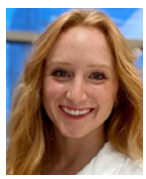
"At one of these sites, the phosphorylation level goes up, while at the other the phosphorylation goes down in old mice," Aryal said.

It's like an internal struggle within the cell where one part of the tau protein seems to drive the development of diseases such as Alzheimer's, and another part appears to counteract it.

Aryal is now researching the changes in lipid profiles in the aging mouse brain and plans to use mice that have been genetically altered to have Alzheimer's or Parkinson's in future proteomic studies to better identify the disease states.

DOI: 10.1016/j.mcpro.2024.100819

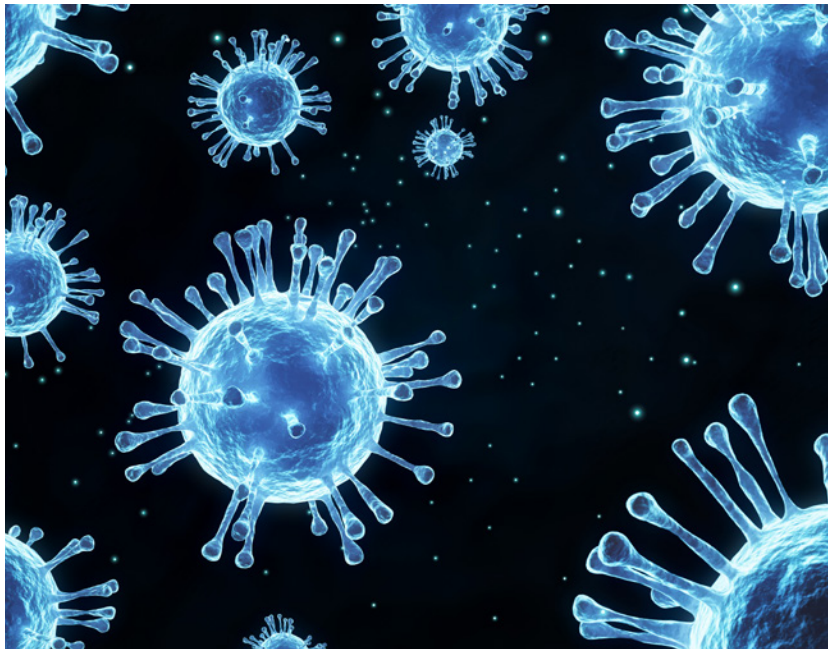
Hallie Blevins is a biochemist and a medical and science writer. She is an ASBMB Today volunteer contributor.



From the journals: MCP

Mapping brain changes from drug addiction. Rapid and precise SARS-CoV-2 detection using mass spec. Decoding plant osmotic stress response. Protein analysis of dopaminergic neurons. Read about papers on these topics recently published in the journal *Molecular & Cellular Proteomics*.

By Sneha Das



Mapping brain changes from drug addiction

Drug addiction poses a complex challenge with few effective treatments. Researchers previously identified changes in brain gene and protein expression in addicted individuals. But, they do not know how drug abuse affects sugar conjugate molecules like heparan sulfate, or HS, and chondroitin sulfate, or CS. HS and CS interact with growth factors and their receptors to modulate cell signaling pathways.

A recent study published in **Molecular & Cellular Proteomics** by Manveen Sethi of Boston University, Riccardo Maccioni of Scripps Institute and their team revealed how drugs like cocaine and methamphetamine change the brain, particularly sulfation patterns of HS and CS. The study examined two critical brain regions involved in addiction — the lateral hypothalamus and striatum — using advanced mass spectrometry–based proteomic and glycomic techniques in mouse models.

The authors found that repeated treatments with these drugs reduced CS4-O–sulfation and increased CS6-O–sulfation. When the authors restored alterations in CS4-O–sulfation, they eliminated anxiety in a cocaine withdrawal test in mice. These findings can lead to development of new therapeutics and treatments for substance use disorders in the future. *DOI: 10.1016/j.mcpro.2024.100803*

Rapid and precise SARS-CoV-2 detection using mass spec

The COVID-19 pandemic highlighted the need for diagnostic tests to rapidly and accurately detect SARS-CoV-2–infected patients to contain outbreaks. Real-time quantitative PCR, or RT-PCR, test kits that detect SARS-CoV-2 viral RNA are currently the gold standard. However, these tests are sequence-specific, making them difficult to adapt when mutated viral variants emerge. Therefore, scientists are exploring alternative diagnostic methods.

In a study published in **Molecular & Cellular Proteomics**, Nicholas Drouin from Leiden University and an international team of researchers developed a novel mass spectrometry, or MS, –based test to detect SARS-CoV-2. This approach identifies specific peptides from the nucleocapsid, the protein that packages the viral RNA. Using a highly selective multiple reaction monitoring-cubed, or MRM3, strategy, which eliminates the expensive and time-consuming immunopurification step, the authors showed that this assay provides better sensitivity than conventional methods.

The authors piloted the MS-based diagnostic test during the SARS-CoV-2 alpha variant outbreak. It detected the virus with 94.2% sensitivity and identified the variant type with 100% specificity. In a subsequent round of tests during the SARS-CoV-2 delta variant outbreak, it detected the virus with 93.1% sensitivity and 99.9% specificity. In addition, the test analyzed each sample in just two minutes, reaching a capacity of 400 samples per day. The test is also inexpensive, and costs 5 euros per sample, making it faster and

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more cost-effective than RT-PCR.

This study showed that MS-based tests can accurately detect respiratory viruses and monitor for the emergence of new viral variants, making it a critical diagnostic tool for future pandemic preparedness.
DOI:10.1016/j.mcpro.2024.100805

Decoding plant osmotic stress response

Osmotic stress occurs when changes in water balance cause cells to swell or shrink as water moves across the cell membrane. In plants, osmotic stress is caused by drought or high salt levels and can severely impair growth and crop yields. Previous studies showed that osmotic stress triggers a rapid calcium influx in plant cells, activating the kinase cascade consisting of Raf-like protein kinases, or RAFs, and sucrose nonfermenting-1-related protein kinase 2s, or SnRK2s. But, researchers do not completely understand the molecular interplay of calcium signaling and the RAF–SnRK2 kinase cascade.

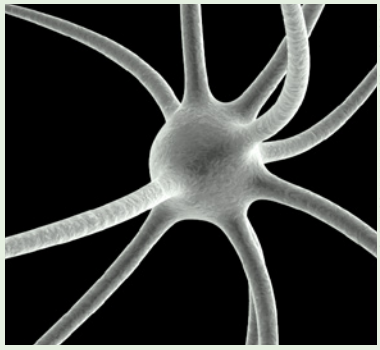
Tian Sang from the Southern University of Science and Technology and an international team of researchers uncovered new molecular insights into the osmotic stress response, in a recent study published in **Molecular & Cellular Proteomics**. The authors found that EGTA, a calcium chelator, mimics the effects of osmotic stress by activating the RAF–SnRK2 cascade. Using high-throughput data-independent acquisition–phosphoproteomics, they further discovered that EGTA activates other key signaling pathways, including mitogen-activated protein kinase cascades, calcium-dependent protein kinases and receptor-like protein kinases, capturing an accurate and detailed role of the plant phosphoproteome in osmotic stress.

These findings highlight how phosphorylation and calcium signaling regulate osmotic stress responses and offer pathways to improve crop resilience to environmental stressors.
DOI: 10.1016/j.mcpro.2024.100804

Sneha Das is a research development manager at the University of Illinois at Urbana–Champaign and an ASBMB Today volunteer contributing writer.

Protein analysis of dopaminergic neurons

By Vanshika Patel



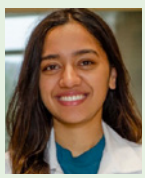
NICOLAS P. ROUGIER VIA WIKIMEDIA COMMONS

Dopaminergic neurons regulate fundamental physiological processes such as motivation, reward mechanisms, motor control and mood. They are the primary cell type affected in Parkinson's disease, or PD. However, studying dopaminergic neurons can pose challenges due to their heterogeneity and presence in deep brain regions. In addition, most of the protein dynamic studies of dopaminergic neurons have been performed in animal cells, not in cultured human dopaminergic neurons, or hDaNs. Scientists still do not understand these mechanisms in human cells.

In a recent article published in **Molecular & Cellular Proteomics**, Claudia Cavarischia–Rega, Karan Sharma and a research team at the University of Tübingen, Germany, analyzed protein composition, turnover and spatial regulation in untreated, healthy human induced pluripotent stem cells, or iPSC, –derived hDaNs. They performed mass spectrometry and a differential dynamic labeling technique to analyze the human iPSC-derived hDaNs.

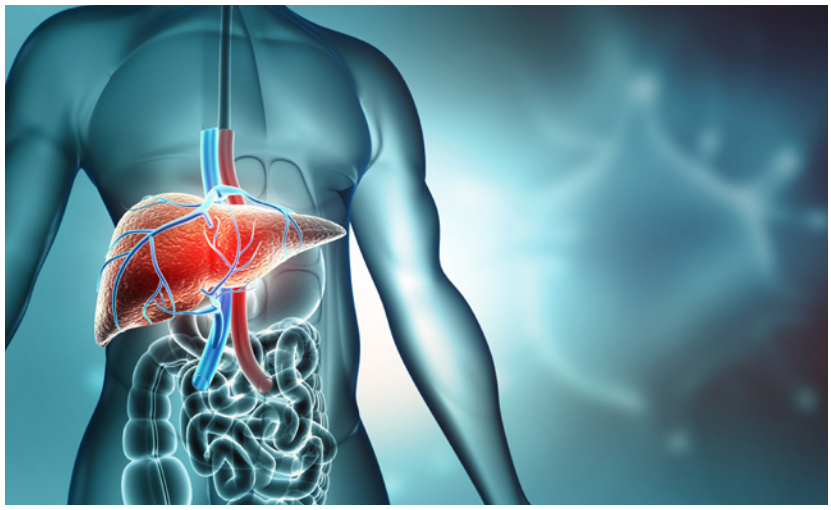
The authors produced the first protein turnover dataset for iPSC-derived hDaNs and identified novel axonal markers. In addition, they detected translocation of proteins between neuronal structures, the axon and soma, and identified proteins involved in axon guidance, synaptic vesicle trafficking and mitochondrial homeostasis, which promote neuronal health. They also detected a significant over-representation of PD-related proteins. These findings set a foundation for PD research and other neurodegenerative diseases in exploring the dynamic proteome of dopaminergic neurons.
DOI: 10.1016/j.mcpro.2024.100838

Vanshika Patel is a Ph.D. candidate in the pharmaceutical sciences department at the University of Maryland, Baltimore. She is an ASBMB Today volunteer contributor.



Predicting fatty liver disease from a tiny blood sample

By Christopher Radka



In the U.S., 40% of adults are obese. Obesity is the primary risk factor linked to metabolic dysfunction–associated steatotic liver disease, or MASLD, a condition characterized by chronic fat accumulation in the liver. More than 40% of U.S. adults have some form of MASLD.

Now, more lean and normal-weight people are developing liver disease, reinforcing the need for more accurate indicators.

Oswald Quehenberger is a professor in the School of Medicine at the University of California, San Diego.

"Liver biopsy is the gold standard method to determine which people are at risk of developing or already have liver disease," Quehenberger said. "However, the procedure is invasive, and it's impossible to subject everyone at risk to liver biopsies. There's a desperate clinical need for a biomarker for this disease."

Quehenberger and colleagues analyzed over 300 patient samples from a study on nonalcoholic steatohepatitis to find such a biomarker. They reported their work in the **Journal of Lipid Research.**

After performing a comprehensive lipid screen comparing diseased samples with healthy controls, they focused on eicosanoids, which are oxygenated metabolites of unsaturated fatty acids such as arachidonic acid. This analysis resulted in 12 eicosanoids that accurately predicted fatty liver disease.

Other laboratories have searched for, and found, biomarkers for MASLD, he said. "But during the validation process, they didn't hold up. This has been a problem all along, especially with fatty liver disease. Here in this study, we were able to verify and validate our initial data with a validation cohort that was independently collected."

About two decades ago, coauthor Edward A. Dennis of UC San Diego organized the LIPID MAPS Consortium to categorize lipids, establish a universal nomenclature and develop methods for their accurate measurement. After coauthor Arun J. Sanyal of Virginia Commonwealth University gave a talk on fatty liver disease at a LIPID MAPS meeting, he collaborated with Dennis and Quehenberger to search for noninvasive biomarkers for MASLD.

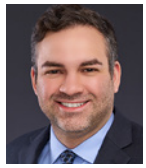
Sanyal secured samples from a biorepository for this study. The team developed a method to analyze thousands of metabolites in every sample. They couldn't do this manually, so Quehenberger worked with researchers at the University of Graz, Austria, to develop a software algorithm that could identify and annotate the metabolites detected by mass spectrometry.

Only a subclass of lipids was used for this paper, but the entire plasma lipidome was measured. The contribution of other lipids to MASLD is part of an ongoing investigation. Quehenberger can analyze a sample in less than six minutes.

"It is a fairly easy transition now to put this into a clinical laboratory," he said. "It's very specific, cheap, and most important of all, it's noninvasive. It's 50 microliters of blood. That's all it takes."

DOI: 10.1016/j.jlr.2024.100647

Christopher D. Radka is an assistant professor in the microbiology, immunology and molecular genetics department at the University of Kentucky and an ASBMB Today volunteer contributor.



From the journals: JLR

Belly fat and liver disease crosstalk. Fixation method to quantify brain metabolites. Stopping heart diseases in schizophrenic patients. Liver gene silencing mitigates atherosclerosis. Read about papers on these topics recently published in the *Journal of Lipid Research.*

By Seema Nath

Belly fat and liver disease crosstalk

Nonalcoholic fatty liver disease, or NAFLD, correlates with obesity as well as belly and blood fat deposition. Patients with NAFLD often progress from simple body fat deposition to free fatty acid, or FFA, accumulation in the liver, a condition called nonalcoholic steatohepatitis, or NASH. This fat imbalance disrupts the very low-density lipoprotein–triglyceride, or VLDL–TG, equilibrium in the abdomen, viscera, or organ cavity, and liver cells. In addition, obese individuals uptake more visceral and hepatic FFAs than healthy individuals. However, scientists do not understand whether obese individuals with NAFLD show different visceral and hepatic FFA and VLDL–TG levels compared to obese individuals with NASH.

Jeyanthini Risikesan from Aarhus University Hospital, Denmark, and an international team quantified FFA and VLDL–TG levels in the blood of obese patients with NAFLD or NASH. They published their work in the **Journal of Lipid Research**. The authors used a palmitate uptake assay to show that patients with NASH and NAFLD showed similar VLDL–TG uptake, FFA levels and abdominal fat metabolism. The authors administered radiolabeled compounds to the patients to study the VLDL–TG kinetics in plasma and carbon dioxide in breath. The results suggested that the visceral balance of FFAs and VLDL–TGs do not differ between obese men with NASH and NAFLD under fasting and high insulin conditions.

As obese people with NASH and NAFLD had no significant differences in abdominal and liver fat uptake and release rate, treatments with other targets, such as inflammation, should be considered as alternative treatments for NASH. *DOI: 10.1016/j.jlr.2024.100580*

Fixation method to quantify brain metabolites

Prostanoids, or PGs, are bioactive lipids in the brain that regulate processes like inflammation, pain, fever, sleep and blood vessel development. Therefore, accurate PG quantification is necessary. High-temperature focused microwave irradiation, or MW, is the preferred method for brain tissue fixation that does not alter PG levels. However, this expensive method can lead to high variability in results, tissue loss and technical difficulties with small samples.

Recently, Derek Besch from the University of North Dakota and colleagues described a cost-effective, simplified PG quantification method in the **Journal of Lipid Research**. They quantified PG and arachidonic acid levels in murine brain tissue samples without fixation and samples fixed via MW or saline water boiling by ultra-high-pressure liquid chromatography–mass spectrometry. Their results showed that boiling the brain samples in saline water neither alters the morphology nor PG levels compared to other fixation methods.

This inexpensive method could allow for more detailed brain tis-

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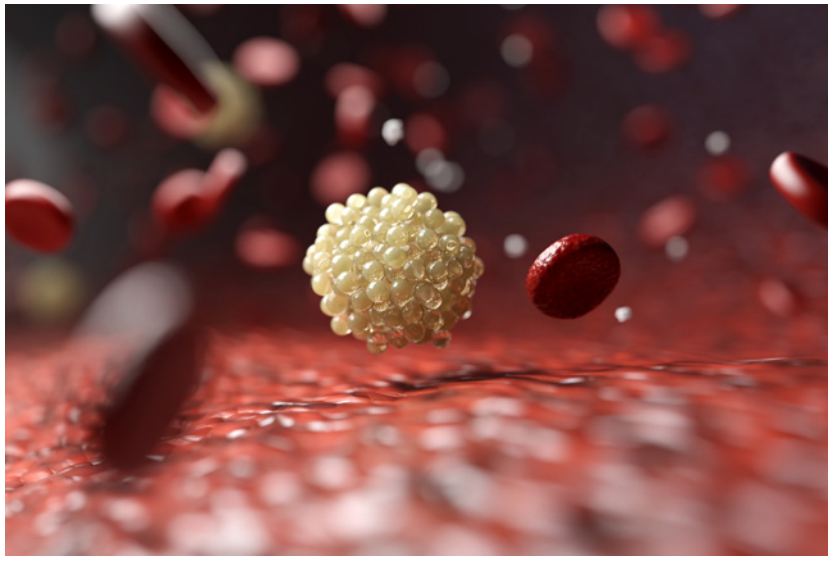


Illustration of omega 3 fatty acid molecules amid flowing red blood cells in arteries.

sue study of other labile metabolites and help in understanding complex neurological processes that may be responsible for different diseases.
DOI: 10.1016/j.jlr.2024.100583

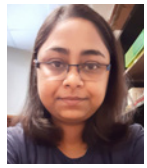
Stopping heart diseases in schizophrenic patients

Schizophrenic, or SZ, patients often suffer from cardiovascular diseases and high-blood triglyceride, or TG, levels. However, this association may be due to unhealthy lifestyles, excessive dietary intake or medication use. Scientists do not understand how cholesterol, TG, TG-rich lipoprotein, or TRL, apolipoprotein, or apo, and angiopoietin-like protein, or ANGPTL, are connected in SZ patients.

Recently, Jeffrey Wang and colleagues from the University of Sydney, compared lipid and lipid-associated protein levels in SZ and healthy individuals. They published their work in the **Journal of Lipid Research**. The authors found higher levels of TG, TG-rich lipoproteins, remnant cholesterol and high-density lipoproteins in SZ patients. The authors hypothesized that the elevated lipid levels may be due to the combination of increased production, reduced lipolysis and impaired TRL clearance. The group found significantly elevated levels of apoCII and apoCIII, both of which are key regulators of TRL uptake and removal, in whole plasma. In addition, they showed that medications did not influence SZ patient lipid or lipid-associated protein levels.

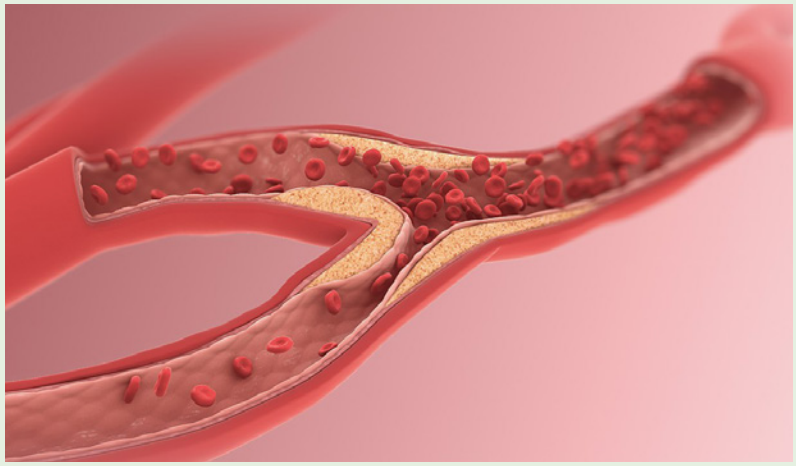
Novel molecules targeting apoCIII may be potential therapeutics for SZ patients with elevated TG and TRL levels.
DOI: 10.1016/j.jlr.2024.100577

Seema Nath is a postdoctoral research fellow at the University of Texas Health Science Center at San Antonio. She is an ASBMB Today volunteer contributor.



Liver gene silencing mitigates atherosclerosis

By Andrea Lius

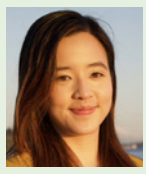


High cholesterol levels contribute to the onset of cardiovascular diseases such as atherosclerotic cardiovascular disease, or ASCVD. Statins, common cholesterol-lowering drugs to treat ASCVD, inhibit HMG-CoA reductase, or HMGCR, a key enzyme in the cholesterol biosynthesis pathway. In addition, the phosphorylation of the enzyme ubiquitin-specific peptidase 20, or USP20, increases cholesterol biosynthesis by blocking HMGCR degradation.

In a recent study in the **Journal of Lipid Research**, Yi Ding and Qiu–Bing Chen and colleagues from Wuhan University silenced the Usp20 gene in the mouse liver using short interfering RNAs, or siRNAs, in lipid nanoparticles, or LNPs. Using ligands that bind target cell receptors, LNPs can be delivered to specific cells, such as liver hepatocytes.

The researchers introduced LNP-packaged Usp20 siRNA to normal mice and observed reductions in USP20 and consequently, HMGCR, protein levels in the liver but not in other organs. Furthermore, when these mice ate a high-fat diet, they had lower body weights compared to untreated controls. The authors also tested the effect of Usp20 siRNA LNP treatment on mice that lacked the low-density lipoprotein receptor, or LDLR. These mice are a widely accepted model for atherosclerosis, as LDLRs are needed to clear LDL, often known as "bad" cholesterol, from the blood. Among the mice that lacked LDLR, those treated with Usp20 siRNA LNPs had reduced aortic atherosclerotic plaques and lower levels of serum cholesterol and triglycerides. This method of silencing Usp20 may offer a promising alternative for patients who have developed statin resistance.
DOI: 10.1016/j.jlr.2024.100626

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.



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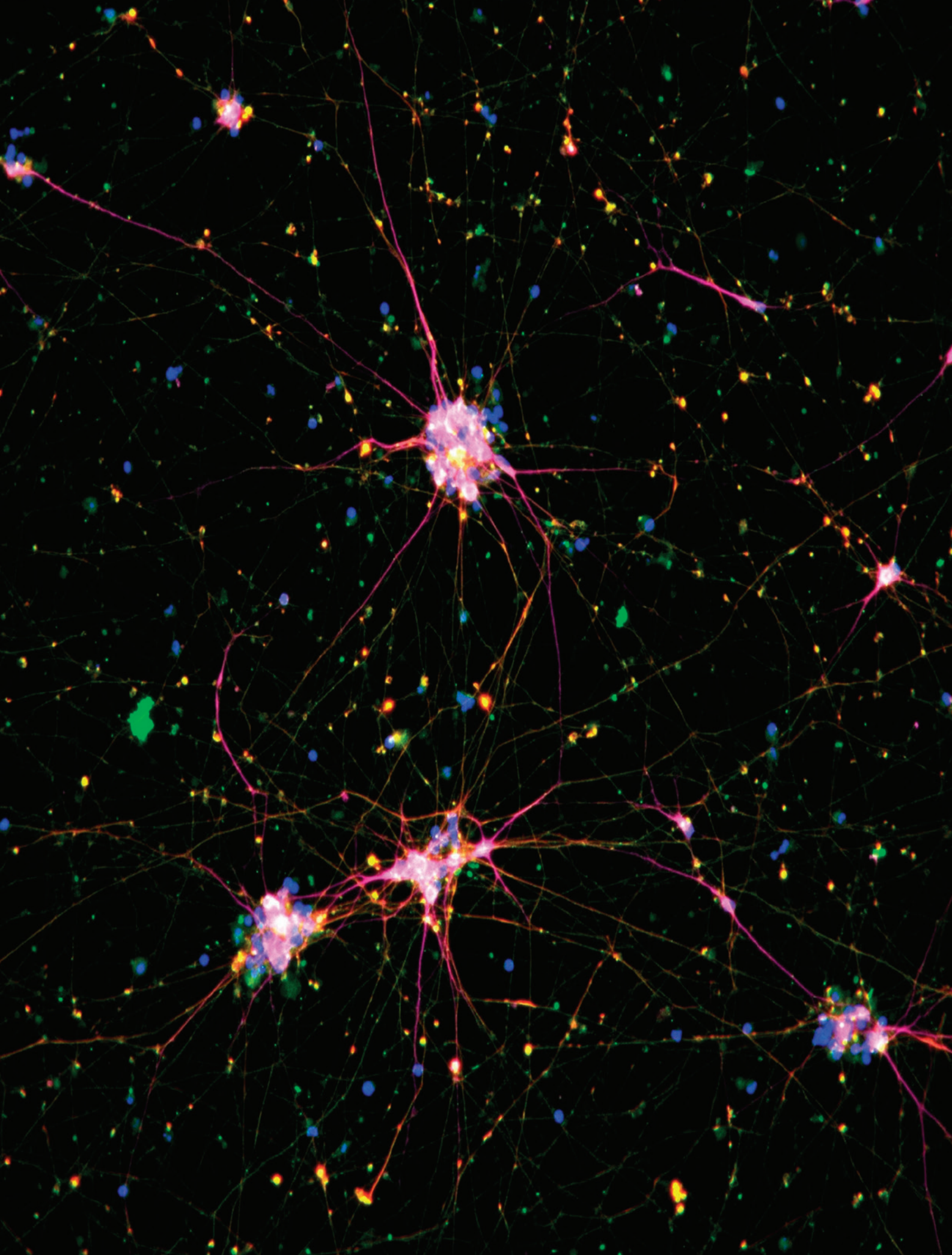
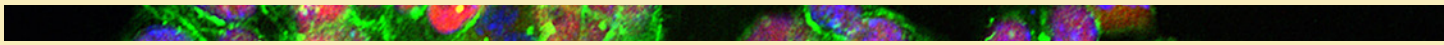


Dynamic stem cells revolutionize regenerative medicine



Personalized induced pluripotent stem cell applications address ethical conundrums and technical issues

By Marissa Locke Rottinghaus

Regenerative medicine is rewriting the future of health care, with stem cells leading the charge as researchers report that their experimental induced pluripotent stem cell, or iPSC, therapeutics could restore vision and cure Type 1 diabetes.

Unlike embryonic stem cells, iPSCs are reprogrammed from adult cells to a pluripotent state from which they can differentiate into myriad cell types such as neurons, cardiomyocytes, insulin-producing pancreatic cells and more. According to experts, iPSCs have the potential to repair or replace damaged tissues and treat a range of diseases considered incurable, including Parkinson's, heart failure, spinal cord injuries and diabetes.

Constantinos Chronis

Constantinos Chronis, an assistant professor of biochemistry and molecular genetics at the University of Illinois Chicago, said iPSCs made from a patient's own cells minimize the risk of immune rejection as well as other issues with traditional stem cell therapies.

A game-changer

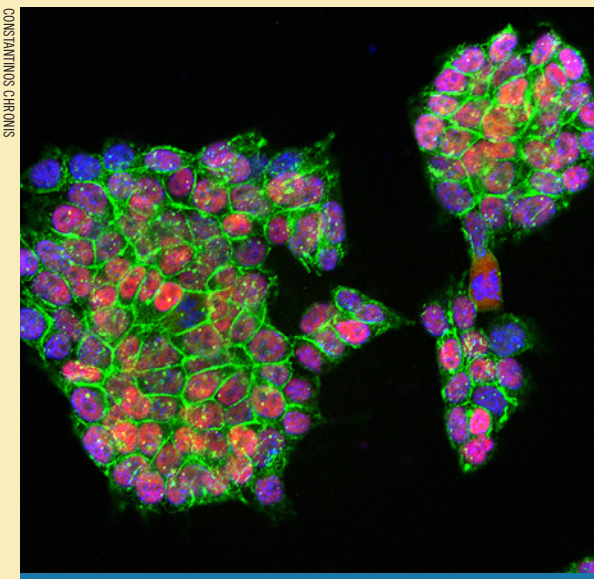
Traditional stem cell research and therapies primarily relied on embryonic stem cells, or ESCs, derived from the inner cell mass of blastocysts in the early stages of embryonic development. Scientists began studying ESCs in the early 1980s. In 1998, U.S. scientists isolated the first human ESC, and controversy soon followed because human ESC research requires the destruction of human embryos.

"I think the U.S. is still very torn over embryonic stem cell use," Anne Zimmerman, a bioethicist at Columbia University, said. "(It's) reasonable to say adult cells will be able to provide some of the stem cell therapies."

ESC research laid the groundwork for modern stem cell science, including the development of iPSCs in 2006 by Shinya Yamanaka.

When researchers introduce specific transcription factors, known as the

CONSTANTINOS CHRONIS



Microscopy image of mouse embryonic stem cell colonies labeled for E-cadherin, a structural protein, (green); nanog, a stem cell transcription factor, (pink); and cell nuclei (blue).

FRANK EDENHOFER

COURTESY OF ELIZABETH GODWIN

Elizabeth Godwin, a 41-year-old mother with Type 1 diabetes, said she is interested in any therapy that could restore her body's ability to produce insulin. Godwin said a stem cell therapeutic would allow her to shift her financial resources away from medical care and toward her family.

Yamanaka factors — OCT4, SOX2, KLF4 and c-MYC — into an adult somatic cell, such as a skin fibroblast, the result is an iPSC. This technique, using either chemical or virally encoded instructions, resets the cell's identity and fate.

The clinical cutting-edge

In October 2024, researchers in China reported that they successfully restored glycemic control in a patient with Type 1 diabetes using islets derived from chemically induced iPSCs.

The team reprogrammed mesenchymal stromal cells from the patient's adipose tissue into iPSCs, which were then converted to insulin-producing pancreatic beta cell islets using growth factors and cytokines that promote pancreatic cell differentiation. After differentiation, the researchers transplanted these synthetic islets under the patient's abdominal muscles. One year after the transplant, the patient needed no insulin injections to maintain glycemic control.

Frank Edenhofer, head of genomics, stem cell biology and regenerative medicine as well as the Molecular Biology Institute at the University of Innsbruck, Austria, said such an autologous approach, deriving beta cells from a patient's own tissue, minimizes the risks of immune rejection. However, he said, this method will likely be "too costly to produce in a personalized manner" for a large number of patients in hospitals with standard equipment.

Frank Edenhofer

In addition, the initial study's small sample size — one patient — limits any broad conclusions about safety and reproducibility. According to Chronis and Edenhofer, future research must address scalability and long-term consequences of transplantation.

Chronis pointed out that the scientists transplanted the iPSC-derived beta cells outside of their natural niche, the pancreas. Unfamiliar surroundings could negatively impact their physiological function, interactions with neighboring cells and potentially lead to tumor formation.

"This study leaves open questions about the nurturing environment," Chronis said. "Cells don't just grow in the air, but they interact with other cells. They are in specific niches that support their function."

Two weeks after the Type 1 diabetes study was published, researchers in Japan reported they used iPSCs to treat limbal stem cell deficiency. In this rare eye condition, corneal stem cells are unable to regenerate and repair the eye's surface. The team in Japan differentiated human iPSCs to corneal epithelial cells and engineered them into cell sheets suitable for transplantation. Over two years, the patients' vision sharpened, and their corneas became less cloudy.

This trial involved four patients who received the iPSC-derived corneal epithelial cells from a third-party cell line, so the transplanted cells did not genetically match the patients' cells. Therefore, some patients had to be treated with immunosuppressive drugs to mitigate the risk of autoimmunity.

"This study uses an allogenic approach," Edenhofer said. "You can make a big batch of cells which can then be distributed to clinical centers applying this therapy to numerous patients. That is relatively cost-efficient."

A similar early clinical trial for limbal stem cell deficiency is ongo-

ing at UCLA Health.

Despite their limitations, Edenhofer and Chronis agreed these studies represent important advances in the field.

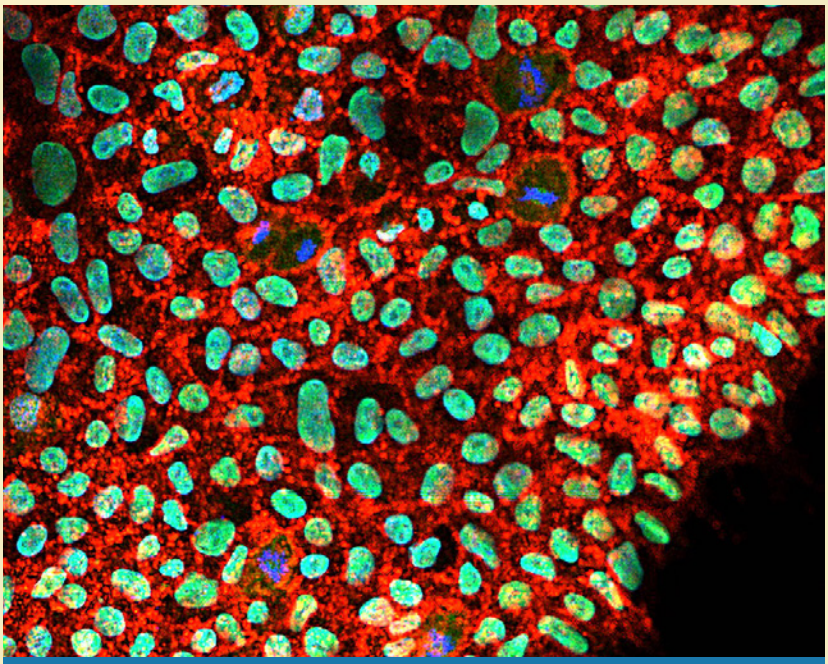
"(These studies) are fantastic," Chronis said, "in the sense that we're now talking about doing something that we envisioned maybe a decade ago or five years ago, in real patients and looking at the outcome of these cells in real time, while continuously monitoring for adverse effects."

Elizabeth Godwin, a 41-year-old adult woman with Type 1 diabetes, said she is interested in any therapy that could restore her body's ability to produce insulin.

"Even with the intensive blood sugar management that I practice … I can't mimic the same results as naturally produced insulin with the synthetic version I have to take," Godwin said. "It takes longer to start working, takes longer to stop working and is more unpredictable, which requires me to put in a lot of mental and practical effort to keep myself in a safe range."

Godwin also said that a stem cell therapeutic would likely allow her to shift financial resources and mental energy to engaging with family and travelling. But she would also be wary of potential side effects, such as cancer, and whether the stem cells would remain effective for the rest of her life.

"It can feel like I am living minute-to-minute at the mercy of the insulin, which provides my only choice to stay alive," Godwin said. "I would love to see treatment options advance beyond the incremental improvements to the current approach, which has been the standard of care for Type 1 diabetes since the first injection of insulin in the 1920s."



CONSTANTINOS CHRONIS

Microscopy image of human induced pluripotent stem cell colony labeled for CD24, a membrane protein, (red); OCT4, a stem cell transcription factor, (green); and cell nuclei (blue).

Understanding what makes iPSCs tick

Because researchers can transform stem cells into a variety of cell types, Chronis underscored the need to distinguish between stem cell–derived cell behavioral and molecular characteristics.

"Being able to produce a functional cell is the goal," Chronis said. "But, having a real molecular identity that very accurately and closely recapitulates the (bodily) in vivo counterpart is equally important."

The stem cell field is advancing far more rapidly than many anticipated, he added, but it lacks strong foundational knowledge.

"Different labs have proposed multiple transcription factor combinations, and even chemicals, for generating these reprogrammed (cells)," Chronis said. "(It) then becomes very difficult to have a standardized set of practices that everybody can work

on and try to improve to increase the protocol fidelity."

Chronis and his team are working to create a roadmap of mammalian stem cell development that scientists can use as a guide to create an iPSC that closely mimics the early stages of human ESC development.

"Our current research focuses on understanding how the Yamanaka factors dismantle somatic cell identities," Chronis said.

They recently converted skin fibroblasts into nonskin endodermal tissue, a type of tissue that develops into many of the body's internal organs, using a combination of the Yamanaka factors and other signals. Using this technology, researchers could bypass iPSC generation altogether, which could reduce production time, lower manufacturing costs and potentially enhance safety, Chronis said.

"The full extent of fibroblast plasticity remains to be seen," Chronis said. "But this presents an exciting new avenue for cell replacement therapy."

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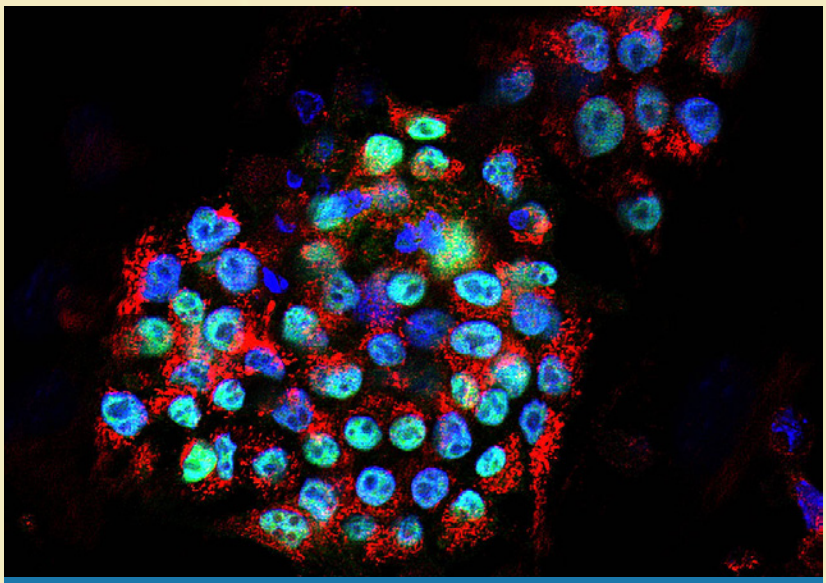


Microscopy image of human induced pluripotent stem cell colonies labeled with CD130, a membrane protein, (red); OCT4, a stem cell transcription factor, (green); and cell nuclei (blue).

Stem cells in the brain

According to Edenhofer, stem cells of the nervous system represent an ideal cellular source for cell replacement therapies because they can generate the major cell types of the nervous system including neurons, astrocytes and oligodendrocytes. This plasticity could allow them to integrate into existing neural circuits to restore lost functions in patients with neurodegenerative diseases.

However, access to and utility of patient-derived neural stem cells is limited. These naturally occurring cells are difficult to harvest because they are found in the inner regions of the brain, and they have limited proliferative potential due to their genetic programming.

Therefore, Edenhofer wanted to use easy-to-reach, patient-derived cells to generate neural stem cells for replacement therapy. To meet this need, his team created a protocol to produce what they call induced neural stem cells, or iNSCs.

Edenhofer's team generates iNSCs directly from patient dermal fibroblasts or blood cells. First, they infect the cells with a nonintegrating virus, containing a modified version of the Yamanaka factors — OCT4, KLF4, SOX2 and c-MYC. After a short incubation, the team transfers the dedifferentiating cells to a neuroinduction media containing growth and survival factors that promote proliferation and neural differentiation.

Though iNSCs are artificially constructed, Edenhofer said, they do not differ transcriptionally from their counterparts derived either from pluripotent stem cells or primary tissue.

"Pragmatically, it's much more cost efficient and faster to generate induced neural stem cells, because if you want to go via iPSCs to neural stem cells, we have to first generate

the iPSCs," Edenhofer said. "That means lots of cell culture time, lots of resources, costs, labor and the risk of undesired cellular changes during prolonged culture time. After that, you still have to redifferentiate those iPSCs into neural cells. We make a shortcut there, which allows us to do it four times faster."

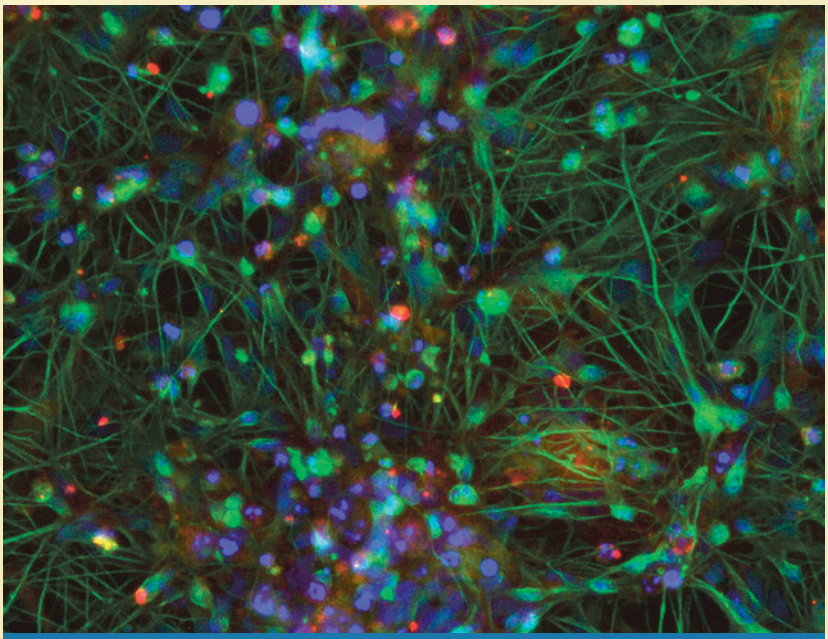
Neuroregeneration

Edenhofer's lab showed that iNSCs could reduce neuroinflammation and restore motor control in mice that have symptoms of multiple sclerosis, or MS, and spinal cord injury. The cells did this by reducing levels of the proinflammatory metabolite succinate in the cerebrospinal fluid after the researchers transplanted them into the mouse via intracerebroventricular injection, which bypasses the blood–brain barrier.

"In the long run, we must change the microenvironment from anti-regeneration to pro-regeneration," Edenhofer said. "When you transplant our induced neural stem cells at the site of injury, they have an anti-inflammatory effect, meaning they recognize the proinflammatory molecules, like extracellular succinate, and suck it up like a sponge. They also activate anti-inflammatory genes to induce remyelination and neuronal rewiring."

To use this method in humans without risking immune rejection, Edenhofer said, the iNSCs must be derived from a patient's own fibroblasts, and a person's illness is likely to affect those cells.

Therefore, his team recently performed a study in mice to compare dermal fibroblast–derived iNSCs from human healthy donors to those from patients with progressive MS. They found that iNSCs from MS patient fibroblasts had increased glucose-dependent fatty acid and cholesterol synthesis, which led to lipid droplet accumulation. This accumulation caused the iNSCs to upregulate inflammatory signaling and take on a senescent-like phenotype, which promoted neurotoxicity in mature neurons.



FRANK EDENHOFER

Microscopy image of neural stem progenitor cells differentiated into neurons and astroglia labeled with beta 3-tubulin, a structural protein, (green) and cell nuclei (blue).

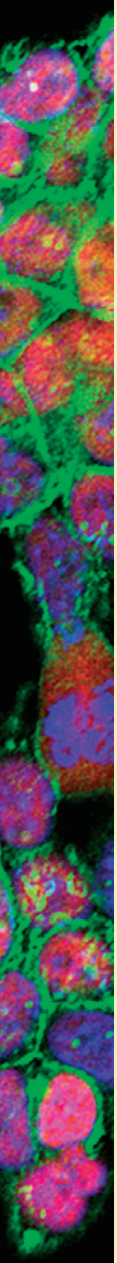
To combat this, the researchers administered the HMGCR inhibitor simvastatin to the MS patient–derived iNSCs, which reversed the hyperinflammatory phenotype; thus, reducing potential neurotoxicity.

Overcoming challenges to translation

Among the most significant challenges when working with iPSCs, are the task of efficient generation of the cells and mitigating the risk of tumors forming after they are transplanted.

Reprogramming adult cells into iPSCs is inefficient. Only a small fraction of treated cells reach the pluripotent state; studies show that fewer

> **"We must change the microenvironment from anti-regeneration to pro-regeneration. When you transplant our induced neural stem cells at the site of injury, they have an anti-inflammatory effect, meaning they recognize the proinflammatory molecules, like extracellular succinate, and suck it up like a sponge."**
>
> **FRANK EDENHOFER**



> **“There have to be clear guidelines and clear regulations, and there are always concerns of how things are going to be used. It’s better to have regulations in the open than to put pressure to stop research in one field and drive researchers to take their work somewhere else, outside the U.S.”**
>
> **CONSTANTINOS CHRONIS**

than 1% of treated cells successfully become iPSCs, depending on the methods used. The type of donor cell, the specific combination of reprogramming factors and the delivery system can all influence outcomes.

“Cells are problematic, in particular stem cells,” Edenhofer said. “Our novel technologies nowadays, single-cell omics, allow us to see that in apparently homologous populations, we still see a lot of heterogeneity that might be due to failed differentiation, survival or integration after transplantation.”

Undifferentiated iPSCs or incompletely differentiated cells can cause tumors because they retain the ability to proliferate indefinitely, Edenhofer said. To address this, scientists are exploring emerging technologies, such as CRISPR-based editing and molecular “suicide switches” that trigger the destruction of aberrant cells to prevent tumor formation.

Yet, another challenge is the large volume of cells needed for stem cell therapeutics. Unlike hematopoietic bone marrow transplants used to treat leukemia, nonhematopoietic stem cell therapeutics require hundreds of millions of cells.

“Scaling up will be an issue,” Edenhofer said. “We know from the very early days of stem cell transplantations, relatively few hematopoietic stem cells are sufficient to repopulate the complete blood system in a recipient mouse. But that is not true at all for all stem cells. For example, in the brain, as one can imagine, you cannot repopulate the nervous system with just a few neural stem cells.”

As of early March 2025, the U.S. Federal Drug Administration had only approved one nonhematopoietic stem cell therapeutic: Ryoncil, an allogeneic bone marrow–derived mesenchymal stem cell therapy for steroid-refractory acute graft versus

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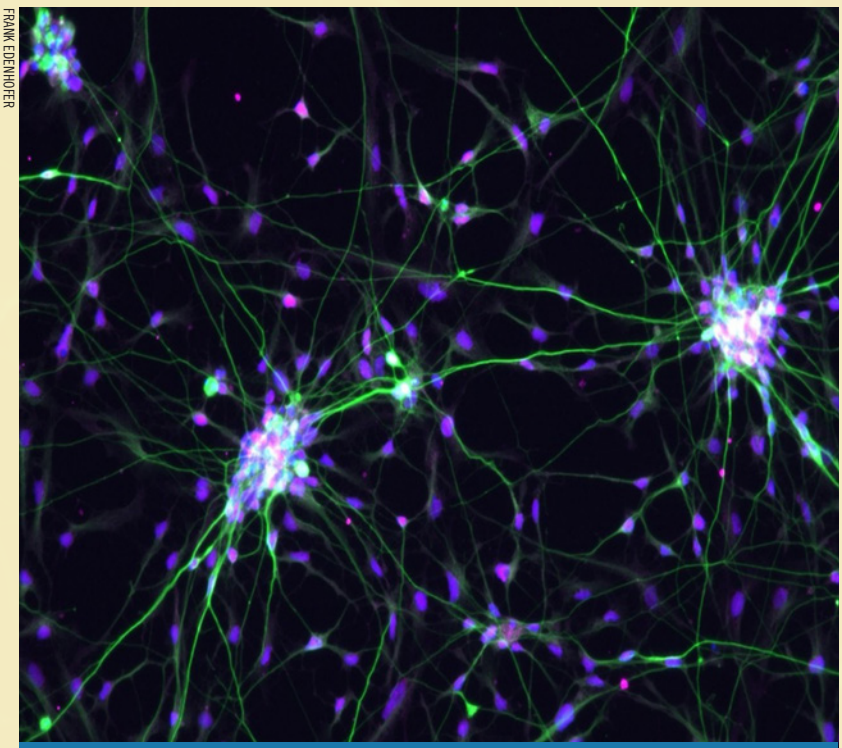
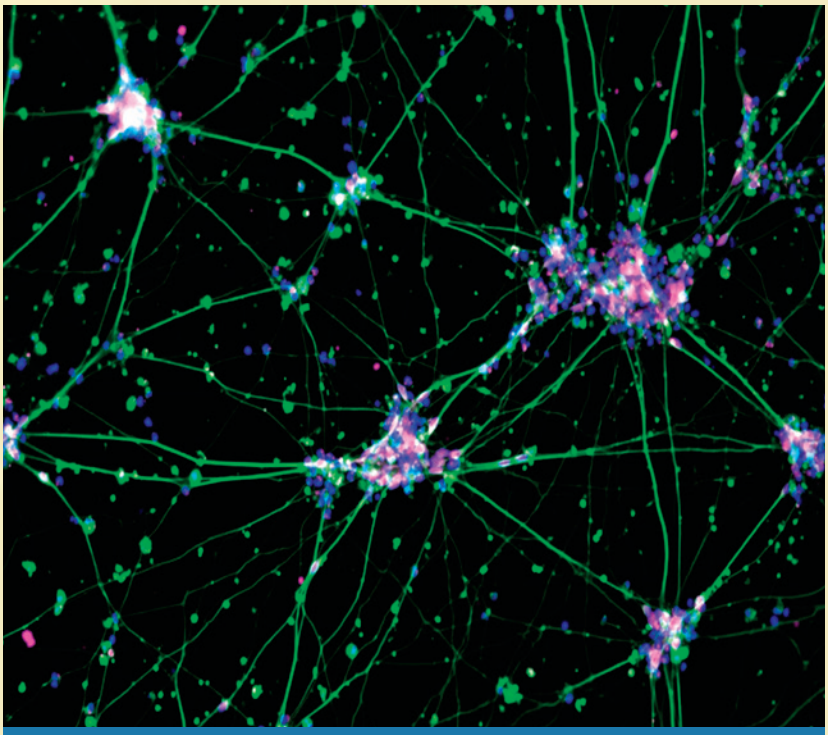


Microscopy image of induced pluripotent stem cells differentiated into neurons labeled with beta 3-tubulin, a structural protein, (green) and cell nuclei (blue).

host disease in pediatric patients two months of age and older.

First discovered in the 1970s, mesenchymal stem cells are multipotent cells known for their ability to modulate immune responses and promote tissue repair. These anti-inflammatory properties make mesenchymal stem cells valuable for treating diseases where the immune system is overactive, such as graft versus host disease. However, Ryoncil uses mesenchymal stem cells directly from a donor and does not involve iPSC technology.

A recent study identified 115 clinical trials with regulatory approval, testing 83 human pluripotent stem cell products. Most of these trials target eye diseases, central nervous system disorders and cancer.



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Microscopy image of induced pluripotent stem cells differentiated into neurons labeled with beta 3-tubulin, a structural protein, (green) and cell nuclei (blue).

Confronting ethical conundrums

The National Institutes of Health requires graduate students in NIH-funded research labs in the U.S. to complete a bioethics course. However, this course rarely touches on stem cell research, focusing instead on interpersonal relationships, research misconduct, data management and research using human subjects. This lack of education often leaves scientists wary of stem cell research, Edenhofer said.

Chronis stressed the importance of continued collaboration and discussion among scientists, ethicists and regulators to drive the stem cell field to meet patient medical needs.

"This research is not just being done for the sake of it, but rather to benefit humanity and to try and cure incurable diseases and bring new therapies to patients," Chronis said. "The field is certainly much more mature than it was five years ago."

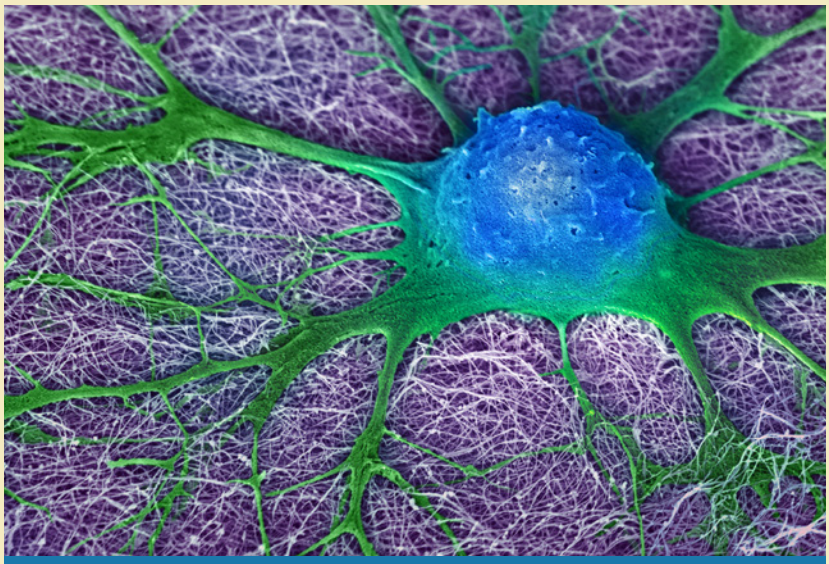
Chronis and other researchers say that ethical concerns and outdated regulations could hamper research to advance stem cell therapeutics in the U.S. while other countries forge ahead.

"There have to be clear guidelines and clear regulations, and there are always concerns of how things are going to be used," Chronis said. "It's better to have regulations in the open than to put pressure to stop research in one field and drive researchers to take their work somewhere else, outside the U.S."

Furthermore, critics of cellular therapies assert that they are so expensive most people could never afford them.

In March 2025, Mesoblast announced that a complete course of Ryoncil will cost $1.55 million. Edenhofer does not think the potential cost is a reason to halt research and development.

"If you go back to the first days of hematopoietic stem cell transplantation, they were not affordable at all,



MARK MCCLENDON, ZAIDA ALVAREZ PINTO AND SAMUEL I. STUPP, NORTHWESTERN UNIVERSITY, VIA NIH FLICKR.

Microscopy image of a mouse neural stem cell (blue and green) in a lab dish, atop a special gel containing a mat of synthetic nanofibers (purple). The cell is growing and sending out spindly appendages, called axons (green), in an attempt to establish connections with nearby nerve cells.

and very few people thought they would work out," Edenhofer said. "Today, not everybody worldwide can expect to get such a treatment, but many transplantations have been carried out. So, it's definitely a success story. Why not think that new stem cell treatments can also develop this way?"

The number of stem cell transplants in the U.S. has grown exponentially over time. In the 1980s, only about 1,500 patients received this therapy compared to almost 23,000 in 2022.

Looking ahead

In 2018, the NIH spent $31 million on research using fetal tissue. In 2019, President Donald Trump signed regulations prohibiting NIH scientists from conducting such research. During the first Trump administration, policy shifts favored research on adult stem cells and induced pluripotent stem cells as less controversial alternatives.

Robert F. Kennedy Jr., the Secretary of the U.S. Department of Health and Human Services, was asked about stem cell research during his confirmation hearing by Sen. Maria Cantwell, D-Wash, and said he would "protect stem cell research."

The field has made significant progress using organoids, 3D self-organizing cell cultures that mimic organ structure and function, in the past decade. According to Edenhofer, the ability to grow full-sized organs may not be far off.

"We are now at the stage where the scientists can produce or generate … early stages of human embryos in situ without implantation into the uterus," Edenhofer said "But what does this mean for us in the future?"

Indeed, a laboratory at Columbia University is creating lifelike, 3D skin gloves using foreskin-derived neonatal fibroblasts and keratinocytes to treat burn victims.

Bioethicist Zimmerman is unsure about the ethical implications of reproducing complex organs.

Anne Zimmerman

"I just don't think, as a country, that we are ready for (scientists) to grow a brain in a lab," Zimmerman said.

However, Chronis and Edenhofer underscored the need to keep pushing the field forward while balancing ethical concerns.

"The stem cell field is very young," Chronis said. "And there is a fantastic future for it."

Marissa Locke Rottinghaus (mlocke@asbmb.org) is the editorial content manager for ASBMB.

Survival tools for a neurodivergent brain in academia

By Andrea Lius

Working in academia is hard, and being neurodivergent makes it harder. Here are a few tools that have helped me as a Ph.D. student with attention-deficit/hyperactivity disorder, or ADHD.

COURTESY OF ANDREA LIUS

Andrea Lius (left) and her colleagues Kacey Rosenthal (middle) and Maryanne Kihiu (right) enjoy DiscoverBMB 2023 in Seattle.

Notes, notes, everywhere

Stick notes on your walls, laptop, desk, bench, biosafety hood and any other equipment you're going to use for the next week — although I'd try to avoid putting sticky notes on your lab mates. I highly encourage color-coding your notes. You can do it based on day, experiment, importance or anything, really. Whatever floats your note.

When it comes to your lab notebook, it's better to have multiple copies of the same entry than zero. This allows you to check everything two, three, four times (and a half, because that final time, you got distracted and thought about cats). Your principal investigator may never understand why it took you more than five minutes to "consult your notes," but that's much less important than actually messing up and not knowing that you ever did. Just make sure to check that you're using the right version of a protocol before you start an experiment and/or send it to a collaborator.

Make sure that you have a "meta" note so you can remember, or at least try to trace, when and where you made each lab notebook entry (e.g., on paper, your phone or the computer), what's it about, where else you may find it and whether you've actually used it for something. The last two may not seem that important, but they help give you some peace of mind, kind of like an "it's ok if you accidentally lost or deleted it" flag — because trust me, it happens all the time.

Calendars, alarms and reminders

Yes, you'll need all of them, each in physical and digital forms. For a physical wall calendar, I personally recommend Space Cats. With pictures of cats (photoshopped into foods, floating in space), you're just that much more likely to look at your calendar, update it and check it to make sure you don't walk into a seminar 45 minutes late, which, to be fair, is still better than completely forgetting to show up.

Don't forget to check that all your digital alarms are off before you go to seminars. If you forgot and your alarm accidentally rang during a talk, do not panic and hit snooze because it'll go off again in nine minutes. If you do hit snooze, absolutely do not panic and hit snooze again. This is all, of course, totally hypothetical and has never happened to anyone, ever. Especially not me.

Finally, friends and colleagues

For me, it's preferable and logical to rely on friends who don't also have, or think they may have, ADHD. Don't get me wrong, they're great. Just like you're great. It just might be helpful not to be stuck in a constant loop of reminding each other of things, when at least half of those reminders are actually meant for yourself.

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.

Quieting the static: Building inclusive STEM classrooms

By Elisabeth Marnik

Landmark College is one of the only accredited colleges in the U.S. designed for neurodivergent students, including those with ADHD, or attention-deficit/hyperactivity disorder, autism or executive function challenges. Christin Monroe is an assistant professor of chemistry at Landmark and is a valuable source of expertise for those seeking to make science and classrooms more inclusive.

This interview has been edited for clarity.

Q: How did you find yourself at Landmark College?

Monroe: While earning a Ph.D. in chemistry from Princeton University, I realized that lab work would never get me up in the morning. Instead, science outreach helped get me through the ups and downs of research. I ended up leaving before my dissertation was submitted and worked for the New Hampshire Upward Bound program for two and a half years (a program for high school students from low-income households that are potential first generation college students). This experience taught me so much, such as the fact that students deserve to be given grace and the benefit of the doubt.

This experience was overall positive, and I had supportive bosses who encouraged me to return to chemistry, so I took their advice. I became an adjunct, finished my Ph.D. and started applying for faculty positions. The one at Landmark popped up, and it felt like a good fit. I applied and got the job.

Q: How do you structure your classes?

Monroe: At Landmark, we recognize that many of our students face executive function challenges (difficulty starting or completing tasks, poor time management, being easily distracted and more). To address this, we build our classes to minimize the impact of these challenges on their learning and overall grades. My goal is to create an environment where, if the student has the ability, they are likely to succeed and progress with their degree.

I can't tell my students how they learn best. Instead, I give them a variety of ways to engage with the content. For example, I assign videos

LANDMARK COLLEGE

Christin Monroe, an assistant professor of chemistry at Landmark College, (right) explains DNA base pairing to a student Rose Heathcliff (left) in her chemistry lab.

as homework; these videos are made with Edpuzzle and include embedded questions for them to complete. This helps lower the activation energy — students can easily find the video and don't also need to find a separate worksheet.

In class, we review the video content and then engage in active learning activities. Each classroom has a pile of small whiteboards. I circle the room during activities and use them to draw and explain things to students, and students use them as well. We have small class sizes — typically between 12 and 16 students, which makes it easier to give students this one-on-one attention.

Q: How do you assess your student's learning?

Monroe: Many of my students struggle with perfectionism, where getting stuck on one problem can prevent them from moving forward. I've seen students working in front of me and then never turning the assignment in because they would rather risk getting a zero than face perceived judgment for getting something wrong. To combat this, I use an online platform (MacMillian Achieve) that gives instructors the option to allow unlimited attempts on questions while tracking their grades as they go.

This system helps students know how they're doing without being penalized for getting stuck. The immediate grade feedback helps them develop a better sense of when to spend more time on a topic or when they are OK to stop. These are important skills for my students, because many have been in environments where they haven't been successful academically, so they haven't yet developed this kind of self-assessment.

To help them further, I use three deadlines. If an assignment isn't turned in within two weeks a placeholder zero is entered, but they can still submit the work. Then I have a mid-semester and end-of-semester cutoff. Students can also choose to revise their work for a better grade.

When I review final grades, I still see a decent bell curve. Not all students earn A's, but they all have the same access to succeed.

Q: How do you build positive relationships with your students?

Monroe: Other faculty at Landmark have taught me to model my imperfections and lean into them. If I am having a bad day, I am honest about that. Jumping right into learning chemistry can be hard for my students, especially if they have something on their minds. This can prevent full participation. I give them space to share, and I acknowledge moments of distractibility as they arise.

For example, one time during lab a bobcat ran up the driveway. One of my students was really into animal science, and he was excited to see the bobcat. Soon, other students went to the windows too. Instead of trying to force them to focus we took a bobcat break. Afterward, we were able to refocus on the chemistry topic.

I also build in catch-up days when there is no new material. This is a day for students to catch up on work and receive extra help. During these sessions, I often pull students into my office one-on-one to check in with them and see how everything is going. I do it for all students, whether they have an A or an F.

Q: What should people know about neurodivergent students in STEM?

Monroe: The difference between neurodiversity and neurodivergence is important. Neurodiversity refers to the whole bell curve. Neurodivergence refers to the edges of the bell curve, those who have skills and abilities above and/or below the average. Someone who is neurotypical would

be in the center of the curve. At Landmark, we often discuss how the world is designed for the "average" person, which means if you are not an average person, then the world wasn't built for you. I've consistently found that students have the underlying skills needed to succeed. It is like static on a TV — there's still a signal behind it, but it's blocked. It is similar for these students; it is sometimes hard to see their skills through the static. Accommodations or adaptations that help remove the static, allowing students to show their abilities and ensure everyone has equal access to education.

There is also a thought that neurodivergence isn't a form of disability. I know that there are a lot of stigmas about disability, but that needs to change. If you don't acknowledge the barriers and challenges these students face, then you may trigger masking (a defensive behavior where a person conceals their natural personality or behavior). I've had students who mask so well that I don't see the challenges they're facing until things fall apart. One of my colleagues always says, "A blind man is not disabled in the cave, and a blind man is your best friend in a cave." Similarly, a neurodivergent person may appear neurotypical in certain environments, either because of masking or because the environment hasn't presented specific challenges. This does not mean they do not need or deserve support.

At the same time, neurodivergent people problem-solve differently, which offers a valuable perspective in STEM. So, we need to get away from looking at those with disabilities as less than, but as those with differences we can support so they can tap into their potential.

Finally, there are still stereotypes about neurodivergence, such as the misconception that those who are autistic lack feelings or gratitude. This is not true. In fact, many of the students I work with feel deeply. They just express these feelings differently. A colleague once pointed out that while autistic students might not say thank you, they will often show engagement and gratitude in other ways. For example, some students have made my lab their comfort zone, even when they don't have class. They'll walk into the building in the morning, drop their stuff and hang out in the space. This is how they show engagement and gratitude. You can reframe your perception of engagement to allow you to see the many ways a student may show they're engaged and grateful.

Q: What resources do you suggest for more information?

Monroe: One of the books we read on campus was Grading for Equity which helps you think differently about how we measure learning. Landmark also offers publicly available courses, webinars and training workshops. These resources helped me understand the concept of executive function and cognitive load, two foundational concepts that influence how many learning differences manifest. This knowledge helps shape the way I teach and interact with students.

I also use the Birkman survey with my students as part of a National Science Foundation grant on which I'm the principal investigator. The Birkman survey can help provide students with language they can use to reframe neurodivergent traits that might be stereotypically bad into positives.

Finally, prioritize professional development tied to these topics, such as learning what accommodations are and more about neurodivergence.

Elisabeth Marnik is the science education and outreach coordinator at the MDI Biological Laboratory in Bar Harbor, Maine, and a volunteer contributor for ASBMB Today.

Hidden strengths of an autistic scientist

By Taylor Stolberg

It shouldn't take you that long just to think of an answer, the faculty judge said.

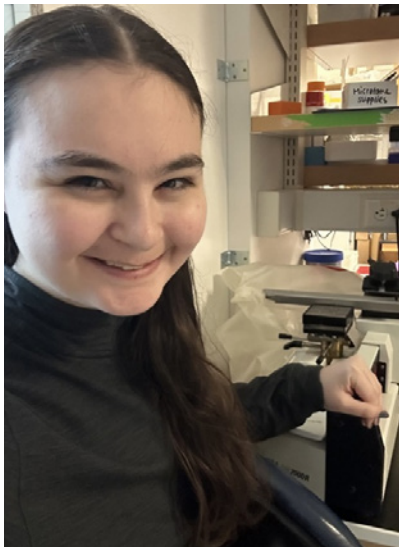
I was at a loss for words. I was sweating through my cardigan, and I was parched. All I could think about was my autism and whether I was talking normally during my first poster presentation at an undergraduate research symposium.

Did I come across as coherent , enthusiastic or bored when talking about my work? Most of all, did I deserve to be here, or was I just masquerading as a research student?

Since beginning my journey in science, comments like this one have plagued my career. Some professors hurled microaggressions at me in core classes and lab presentations. Others made assumptions about my abilities. One professor even stopped mid-lecture to berate me for not making eye contact while taking notes.

As an autistic rising scientist, small nuances of communication, such as interpreting nonverbal social signals and translating my thoughts into spoken word, are challenging for me. Therefore, it often takes me longer to formulate a response to a question, because I'm trying to emulate normal social conventions and focus on the conversation.

Do you remember how many times you spoke to someone today, such as your lab coworkers or the grocery store cashier? Now imagine you planned — in advance — what you'd say to everyone. During those interactions I constantly interpret facial expressions, force myself to maintain eye contact and strive to ensure a reciprocal exchange of ideas.



COURTESY OF TAYLOR STOLBERG

Taylor Stolberg is postbaccalaureate researcher at the University of Michigan Medical School studying the genetic disorder spinocerebellar ataxia type 3.

Scientific communication is a critical skill for researchers. Because every day social interactions do not come naturally for me, I often feel like an inadequate researcher and communicator. I internalize comments and microaggressions. These leave me feeling like an imposter, questioning whether I can attain a career in science.

Neurodivergence is a spectrum. Even though I struggle with some common tasks, I also bring unique skills to the table, like discerning patterns and making connections between seemingly unrelated ideas.

In my postbaccalaureate research lab, I noticed one of our immunofluorescence experiments produced high background. My lab members thought outdated antibodies caused the problem; however, the issue persisted despite testing two new antibodies.

After further reading, I predicted the antibody was not working due to an improper blocking technique. After using a different method, I eliminated the abnormally high background. My nonlinear thinking allowed us to resolve this problem quickly.

As a neurodivergent scientist, my experiences taught me to diversify my research explanations to ensure the material is accessible to broad audiences. For example, I embed speech-to-text software in my presentations to accommodate hearing impaired individuals and/or those who learn best by reading.

Being a neurodivergent scientist makes me a stronger scientist and more inclusive communicator. Scientists are trained to embrace out-of-the-box thinking and celebrate new ideas. However, many higher education institutions still put up social barriers for those who think and communicate differently. Although I have worked to overcome many of these barriers personally, they still permeate scientific circles. To advance scientific endeavors, we need to make science accessible for everyone, including those who process information differently than the majority.

Taylor Stolberg is postbaccalaureate researcher at the University of Michigan Medical School. She investigates the molecular mechanisms that drive the genetic disorder spinocerebellar ataxia type 3.

Embrace your neurodivergence and flourish in college

By Elizabeth Stivison

Embracing neurodiversity means a few things: On one hand, it means making classes accessible so that any student who is willing to put in the effort, can flourish. A few years ago, I wrote a pair of articles about what professors can do to make their labs more inclusive to neurodiverse students. On the other hand, it means making sure you, as a student, get your own access needs met.

College may be the first time you've had to navigate bureaucratic systems to get assistance, navigate healthcare to get diagnoses, or it may be the first time you've considered you might be neurodivergent, as many conditions don't fully manifest until early adulthood.

You can get assistance in many ways if you know where to look: formally, through the disability office in your school, or informally through study and time management resources.

Let's start with formal accommodations.

College accommodations are not the same as those in high school

In college, accommodations for students with disabilities fall under the Americans with Disabilities Act or Section 504 of the Rehabilitation Act. In both cases, the laws put the onus on the student to make sure their needs are met.

This situation is different from K–12 education, where the Individuals with Disabilities Education Act puts the onus on the school and the teachers.

This means you must be your own advocate in college. So, you have to know what you need and how to get it.

Another difference between high school and college is what accommodations can do. In K–12 education, it is possible to modify the curriculum for a student. Conversely, college curriculums cannot be modified. Only access to the material can be modified.

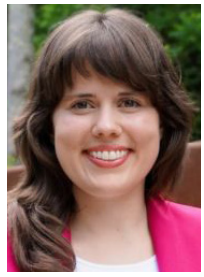
For example, a professor probably can't tell you that you don't have to take a certain test or read a certain text. What they can do is make the test and the reading more accessible to you. They may allow you to use assistive devices, have extended time or sit in a quiet room for the test. For the reading, a professor may make a text more accessible by providing an audio recording or making sure it's usable by screen readers.

How to receive accommodations

Each school's protocol is a little different, so take the time to find out how your institution works. The steps to accommodations might involve emailing the disability services office, setting up an appointment, filling out an online form or all of the above. Common things students may have to provide include documentation of a medical diagnosis, a neuropsychological report, school records and a self-report of past accommodations you received.

This process typically requires a meeting where you'll be asked to suggest the accommodations you need, like extended time on assignments, a reduced distraction environment for tests, notetaking assistance, sensory breaks, recordings of lectures or priority for classes at certain times of the day. Afterward, you'll receive an official letter of accommodation for your professors.

Mariel Pfeifer, an assistant professor at the University of Mississippi, who studies the experience of neurodivergent students in STEM, recommends that students register with the disability office early on, so they have all your documentation, even if you aren't sure you want to use accommodations. Stephen Podowitz–Thomas, an assistant professor at Thomas Jefferson University, who studies the experiences of neurodivergent undergraduates in STEM, agreed.



PFEIFER

PODOWITZ-THOMAS

Both shared that once you've registered with the office, it is easier to gain accommodations, should you need them, even if you want to have the experience of not using accommodations first to test out what you really need. Waiting until an

emergency or crisis will make it much harder to get the paperwork done and get the accommodations fast.

"Accommodations are not retroactive," Pfeifer explained. You can't ask to take a test again with different accommodations, it can only apply to the future, so being able to get them when you need them is important.

Learn about yourself

Podowitz–Thomas said that it is key for students to use college to learn about their needs because this knowledge will help you after college too.

He emphasized the importance of metacognition, or understanding what you are thinking and experiencing.

"It's hard to say, 'Why is this challenging?'" he said, but deeply reflecting can teach you what you need. "There can be a lot of emotions facing challenges and facing failure but being able to process and learn what you need, and how to translate that to self-advocacy" can be transformational, he said.

Podowitz–Thomas also noted that even though it is essential for students to learn about themselves and self-advocate; advocacy doesn't get faculty off the hook for doing their part to make classes as accessible as possible. It's a two-way street, he said.

Other support

There are additional ways to set yourself up for success in addition to the disability office.

As you learn about your needs, you learn how to help yourself, Podowitz–Thomas said.

For example, you may learn that planning ahead really helps you. If you get assigned a group project, you can take your self-knowledge and ask your teammates to reply to scheduling emails promptly.

You can also talk to your professors, if you feel comfortable, to come up with strategies together.

Some ideas to make a classroom more accessible include:

- Being told ahead of time if Zoom meetings or classes will have breakout rooms or discussions
- Moving your seat away from loud equipment
- Allowing a longer time for verbal responses
- Being allowed to walk around

Not everything can be accommodated in a large, dynamic classroom. However, educators usually want their students to learn, and most are willing to do whatever they can to help students.

Outside the classroom, most colleges have centers for learning, usually based in the library. These are treasure troves of study skills and study help. While not specifically targeted to neurodivergent students, they are valuable free resources.

You can also look into one-on-one peer mentoring programs.

While it takes real effort and courage to learn about and advocate for yourself, it's effort well spent. A bit of time can help make sure you get the most out of college and set yourself up for a fulfilling career.

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

ASBMB names 2025 fellows

By Marissa Locke Rottinghaus

The American Society for Biochemistry and Molecular Biology named 24 members as 2025 fellows of the society.

Designation as a fellow recognizes commitment to ASBMB through a history of exceptional and sustained service to the society as well as accomplishments in research, education, mentorship, diversity and inclusion, advocacy and service to the scientific community.

The fellows were selected by the ASBMB Membership Committee, including Rick Page, associate vice president for research and innovation at Miami University and the Membership Committee chair, as well as Chi Fung Lee, assistant professor of cardiovascular biology research at the Oklahoma Medical Research Foundation and the Membership Committee fellows task force chair.

"We are glad to welcome the 24 new ASBMB fellows in the 2025 class," Lee said. "They have shown remarkable commitment to ASBMB through their sustained service as well as impactful accomplishments in their professions of research, education, advocacy and service to the scientific community. We are honored to have these colleagues to represent ASBMB. We look forward to their continued contribution as role models and mentors to inspire members of ASBMB."

This is the fifth year that ASBMB has named fellows. The society will recognize the 2025 class at its annual meeting, April 12–15, in Chicago.

Learn more about the 2025 fellows in the pages ahead.

Ann Aguanno

Ann Aguanno is a professor of medical genetics and biochemistry and the chair of molecular sciences at the Medical University of the Americas in Nevis, West Indies. She is also a professor emerita at Marymount Manhattan College. Aguanno previously studied the role of cyclin-dependent kinases in neurodegenerative diseases and insulin regulation as well as effective methods for training undergraduate biology researchers. She currently focuses on best pedagogical practices in medical education. She is a former ASBMB Educational and Professional Development Committee member and the former chair of ASBMB Student Chapters Committee.

Jeremy Berg

Jeremy Berg is the associate senior vice chancellor for science strategy and planning at the University of Pittsburgh School of Medicine. His lab explores how zinc-containing proteins bind to DNA or RNA and regulate gene activity. Berg's service awards and honors include the ASBMB Howard K. Schachman Public Service Award. He served as the president of ASBMB from 2012 to 2014.

Judith Bond

Judith Bond is an adjunct professor of biochemistry and biophysics at the University of North Carolina at Chapel Hill. Her lab focuses on proteolysis and metalloproteases known as meprins. In 1988, she received Virginia's Outstanding Scientist Award. Bond is a former president of the Federation of American Societies for Experimental Biology and served as ASBMB president from 2004 to 2006. She also served on the Membership Committee as chair of the ASBMB fellows program subcommittee and as an associate editor for the Journal of Biological Chemistry.

Karin Bornfeldt

Karin Bornfeldt is a professor at the University of Washington. Her lab focuses on understanding the mechanisms of diabetes-accelerated atherosclerosis so that cardiovascular complications of diabetes can be treated or prevented. She has served on the editorial board of the Journal of Biological Chemistry and is an associate editor of the Journal of Lipid Research.

Victoria Del Gaizo Moore

Victoria Del Gaizo Moore is an associate professor of chemistry at Elon University. Her research focuses on the role of mitochondria in disease and apoptosis. She is also interested in undergraduate teaching and learning in biochemistry and molecular biology, with a focus on diversity, equity and inclusion. Del Gaizo Moore is a former member of the ASBMB Education and Professional Development Committee as well as the exam and accreditation subcommittee, where she has worked to refine the national accreditation exam. She also served as the cochair for education and professional development programming at the 2019 and 2022 ASBMB annual meetings.

Edward Eisenstein

Edward Eisenstein is a fellow at the University of Maryland Institute of Bioscience and Biotechnology Research, an associate professor at the university's Fischell Department of Bioengineering and associate director of the Agricultural Biotechnology Center. His team engineers poplar trees with new traits for use as improved feedstocks for the bioeconomy. He has served on the ASBMB Outreach Committee and Membership Committee and on the editorial board of the Journal of Biological Chemistry and has written articles for ASBMB Today. He is a member of the ASBMB Council.

2025 FELLOWS

Martin Gellert

Martin Gellert is a distinguished investigator at the National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases. His research explores the genetic rearrangement mechanisms of the immunoglobulin and T-cell receptor genes, which are essential for lymphoid cell development. In 1985, he won the ASBMB–Merck Award. Gellert served as president of ASBMB in 1993.

Gordon Hammes

Gordon Hammes is a distinguished professor emeritus of biochemistry at Duke University. His research focuses on understanding enzyme dynamics, conformational changes and reaction intermediates using biophysical methods, such as fast-reaction kinetics and fluorescence resonance energy transfer. He previously won the ASBMB William C. Rose Award.

Joseph Jez

Joseph Jez is a professor of biology at Washington University in St. Louis. His lab studies how environmental changes remodel biochemical pathways in plants at the molecular, cellular and organism levels with the aim of engineering these systems to address agricultural and environmental problems. He has served on the ASBMB Public Affairs Advisory Committee and is an associate editor of the Journal of Biological Chemistry.

Kayunta Johnson–Winters

Kayunta Johnson–Winters is an associate professor of chemistry and biochemistry at the University of Texas at Arlington. Her research interests are on F420 cofactor dependent enzymes, using steady state and pre–steady state kinetic methods. In 2021, Johnson–Winters won a Silver EXCEL Award from Association Media & Publishing for her ASBMB Today essay, "Being Black in the ivory tower." She served on the ASBMB Maximizing Access Committee and is now a member of the ASBMB Council.

Oleh Khalimonchuk

Oleh Khalimonchuk is a professor of biochemistry and the director of the Nebraska Redox Biology Center at the University of Nebraska–Lincoln. His lab uses yeast, mammalian cell and roundworm models to study the molecular bases of mitochondrial function and dysfunction as they relate to human disease and aging. He is a member of the ASBMB Meetings Committee.

Judith Klinman

Judith Klinman is a professor of chemistry at the University of California, Berkeley. Her research explores fundamental aspects of enzyme mechanism, demonstrating the roles of hydrogen tunneling and protein scaffold energy transfer in enzyme action. She discovered a class of protein-embedded, quinone redox cofactors and elucidated the pathways that produce these structures as well as the antioxidant pyrroloquinoline quinone. She won the ASBMB–Merck Award in 2007 and the Mildred Cohn Award in Biological Chemistry in 2015.

Moshe Levi

Moshe Levi is the Chief Science Officer for Research and Development at Georgetown University Medical Center and is a professor of biochemistry and molecular and cellular biology. His research explores the role of nuclear hormone receptors and transcription factors in complications of obesity, diabetes and aging, mineral metabolism regulation and

molecular imaging of lipids, inflammation, oxidative stress, metabolism and fibrosis. He serves on the editorial board of the Journal of Biological Chemistry.

Steven McKnight

Steven McKnight is the distinguished chair in basic biomedical research at the University of Texas Southwestern Medical Center. His research uses biochemical, genetic and molecular biological approaches to study how genes are switched on and off in mammalian cells. McKnight was president of ASBMB from 2014 to 2016.

Karin Musier–Forsyth

Karin Musier–Forsyth is an Ohio Eminent Scholar at the Ohio State University. Her lab investigates the RNAs and proteins involved in retroviral replication and translation fidelity mechanisms. She is an associate editor for the Journal of Biological Chemistry.

Himadri Pakrasi

Himadri Pakrasi is a professor of biology at Washington University in St. Louis. His research uses systems and synthetic biology to explore bioenergy production in cyanobacteria. Pakrasi has served on the ASBMB Public Affairs Advisory Committee.

Gregory Petsko

Gregory Petsko is a professor of neurology at Brigham & Women's Hospital and Harvard Medical School. His research focuses on finding treatments for neurodegenerative diseases such as Alzheimer's, Parkinson's and amyotrophic lateral sclerosis. Petsko was ASBMB president from 2008 to 2010.

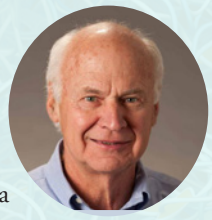
Suzanne Pfeffer

Suzanne Pfeffer is a professor of biochemistry at Stanford University School of Medicine. Her research focuses on understanding the molecular basis of inherited Parkinson's disease, with a focus on LRRK2 kinase and Rab guanosine triphosphatase phosphorylation. The Pfeffer lab also studies cholesterol transport and how mutations in this pathway cause Niemann–Pick C disease. Pfeffer served as president of ASBMB from 2010 to 2012.

Joseph Provost

Joseph Provost is a professor and chair of the chemistry and biochemistry department at the University of San Diego. His research focuses on the role of transport proteins in cell motility and tumor progression. He served as chair of the ASBMB Student Chapters Committee for five years as well as on the Educational and Professional Development Committee and the Membership Committee. He won the 2022 ASBMB Award for Exemplary Contributions to Education. In addition, Provost has authored many articles in ASBMB Today.

Charles Samuel



Charles Samuel is a research professor and a distinguished professor emeritus of molecular, cellular and developmental biology at the University of California, Santa Barbara. His lab studies the role of interferon-inducible double-stranded RNA–dependent enzymes during viral infection, with focus on the protein kinase R and the RNA adenosine deaminase 1. He is a former associate editor of the Journal of Biological Chemistry.

2025 FELLOWS

Ann Stock

Ann Stock is a distinguished professor at Rutgers University–Robert Wood Johnson Medical School. Her research focuses on bacterial signal transduction and the molecular mechanisms that allow bacteria to elicit adaptive responses to changes in their environments, such as the gut. Stock was president of ASBMB from 2022 to 2024 and served on the Council, Finance Committee, Education and Professional Development Committee and the society's accreditation application review subcommittee.

Quinn Vega

Quinn Vega is a professor of biology at Montclair State University. His research explores cellular signal transduction and the mechanisms by which cells respond to external environmental and biochemical clues by activating specific molecular signals and activating transcription of specific genes. He has served on the ASBMB Membership Committee and the Education and Professional Development Committee and was previously the chair of the ASBMB Student Chapters Committee.

Hao Wu

Hao Wu is a professor of structural biology, biological chemistry and molecular pharmacology at Harvard Medical School. Her lab uses cryogenic electron microscopy and other biophysical methods to understand molecular complexes involved in innate immunity, including signalosomes and pore-forming complexes such as gasdermin D. She received the ASBMB Bert and Natalie Vallee Award in Biomedical Science in 2024.

Yan Jessie Zhang

Yan Jessie Zhang is a professor of biochemistry at the University of Texas at Austin. Her lab studies the molecular mechanisms of the enzymes that govern the post-translational modification states of eukaryotic RNA polymerase II and their implication in transcription. Zhang is a member of the Meetings Committee and has served as a theme organizer for the ASBMB annual meeting multiple times.

Benjamin A. Garcia received the Award for Outstanding Achievements in Mass Spectrometry from the Eastern Analytical Symposium. Garcia is a professor and the head of biochemistry and molecular biophysics at the Washington University School of Medicine in St. Louis. His lab uses quantitative mass spectrometry–based proteomics to characterize modified proteins and proteomes, especially those involved in epigenetic mechanisms. He serves on the editorial board of Molecular & Cellular Proteomics. He received the 2025 ASBMB Ruth Kirschstein Diversity in Science Award.


GARCIA

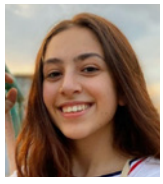
Alice H. Lichtenstein has been named a distinguished professor of nutrition science and policy by Tufts University. The honor recognizes senior professors who have made exceptional contributions to their disciplines, their students and the university. Lichtenstein is a senior scientist and the director of the Cardiovascular Nutrition Team at the Jean Mayer U.S. Department of Agriculture Human Nutrition Research Center on Aging. Her research focuses on the intersection of diet and cardiometabolic health. She is an associate editor of the Journal of Lipid Research.


LICHTENSTEIN

Shubhik DebBurman received the 2024 Biology Division Faculty Mentor Award (Advanced Career) from the Council on Undergraduate Research. This award honors biology mentors for their sustained efforts in supervising undergraduate research students. DebBurman is a professor of biology; biochemistry and molecular biology; and neuroscience at Lake Forest College. His lab studies the protein folding issues linked with neurodegenerative disease pathology mechanisms in yeast systems.


DEBBURMAN

Anush Margaryan received a Projects for Peace grant of $10,000 to teach science, technology, engineering and mathematics to refugees in Armenia. With the funds, she hosted a camp where 75 displaced students performed hands-on biology, chemistry, physics, robotics and engineering experiments. Margaryan grew up in a small village in Avshar, Armenia. Now, she is a senior Richmond Scholar at the University of Richmond, studying biochemistry and molecular biology and pursuing a minor in data science.


MARGARYAN

Andrew Santiago–Frangos has joined the University of Pennsylvania School of Arts and Sciences as the M. Jane Williams and Valerie Vargo Presidential Assistant Professor of Biology. The professorship honors UPenn chemistry graduates Williams and Vargo. Santiago–Frangos' lab studies how bacterial and archaeal CRISPR adaptive immune systems make DNA-based memories of past phage infections as well as how CRISPR-generated nucleotide messengers prompt an immune response. He's a scholar in the ASBMB Maximizing Opportunities for Scientific and Academic Independent Careers, or MOSAIC, program.


SANTIAGO–FRANGOS

Scott Emr and **Wesley Sundquist** won the 2024 Louisa Gross Horwitz Prize from Columbia University for discovering the endosomal sorting complexes required for transport, or ESCRT, pathway. The prize was established by Columbia graduate S. Gross Horwitz to honor his mother. ESCRTs are sets of proteins that enable vesicles to bud out from the cytoplasm. They are required for formation of vesicles within endosomes, some types of viral envelope budding and release as well as the final steps of cell division.

Emr is a professor emeritus of molecular biology and genetics at Cornell University. The Emr lab studies the regulation of cell signaling pathways by phosphoinositide kinases, vesicle-mediated transport reactions and selective ubiquitin modifications. He won the ASBMB Avanti Award in Lipids in 2007 and the ASBMB Lifetime Achievement Award in 2022.


EMR

Sundquist is the chair and a distinguished professor of biochemistry at the University of Utah. The Sundquist lab studies the cellular, molecular and


SUNDQUIST

structural biology of retroviruses, particularly HIV, and the roles of the ESCRT pathway in cell division.

Adam Ohm and **Madeline Szoo** have been awarded scholarships by the Fellowship Board of Tau Beta Pi, an engineering honor society. Awardees are selected based on their academic work, campus leadership and service as well as promise of future contributions to the engineering profession. They receive a cash prize of at least $1,000.

Ohm is an undergraduate in chemical engineering at Tennessee Technological University. He received the Badiru Scholarship, which is named for Adedeji B. Badiru, professor and dean emeritus of the Graduate School of Engineering and Management at the U.S. Air Force Institute of Technology.


OHM

Szoo is an undergraduate in chemical engineering at Northeastern University. She received a Stabile Scholarship, which honors Vincent A. Stabile, an engineer and philanthropist. Szoo studies the effects of anticancer drugs on the extracellular matrix of gliomas.


SZOO

César de la Fuente received the American Society of Microbiology's Award for Early Career Basic Research recognizing an early-career investigator with distinguished basic research achievements in the microbial sciences. de la Fuente is a presidential associate professor of chemistry at the University of Pennsylvania Perelman School of Medicine. His lab develops computational methods to mine the world's biological information to identify new antimicrobial compounds, and he has pioneered the emerging field of artificial intelligence–driven antibiotic discovery.


DE LA FUENTE

Tanja Mittag and **Enrique M. De La Cruz** have been named fellows by the Biophysical Society. This honor goes to the society's distinguished members who have demonstrated excellence in science and contributed to the expansion of the field.

Mittag is a member of the department of structural biology at St. Jude Children's Research Hospital. Her lab explores the role of phase separation in functional compartmentalization and how dysregulation of this process can lead to cancer and neurodegenerative diseases.


MITTAG

De La Cruz is a professor of molecular biophysics and biochemistry, and he also serves as the head of Branford College, one of Yale's undergraduate residential colleges. His lab uses biology, chemistry, physics and engineering techniques to study molecular motor proteins and the cytoskeleton. De La Cruz is a Journal of Biological Chemistry associate editor and a 2021 ASBMB fellow.


DE LA CRUZ

Adnan Alrubaye received the Poultry Science Association Early Achievement Award recognizing the achievements of PSA members in the early stages of their careers in poultry academia. Alrubaye is an assistant professor of poultry science and associate director of the cell and molecular biology graduate program at the University of Arkansas. His lab investigates the cause of and ways to mitigate bacterial chondronecrosis with osteomyelitis in broiler chickens.


ALRUBAYE

Anindya Dutta has received the Rous–Whipple Award from the American Society for Investigative Pathology, which recognizes a senior scientist who has advanced the understanding of disease and made meaningful contributions via teaching, mentorship and leadership in the pathology field. Dutta is a professor and chair of genetics at the University of Alabama at Birmingham. His lab investigates genomic instability in cancer cells and noncoding RNAs in differentiation and cancer. Dutta discovered extrachromosomal circles of DNA in normal and cancer cells and demonstrated that the circles from cancers are released into the blood and can be exploited as a biomarker.


DUTTA

Todd Strochlic has been appointed assistant dean of curricular integration at the Drexel University College of Medicine. He will oversee and manage the medical education curriculum. Strochlic is an associate professor of

biochemistry and molecular biology at Drexel. He also codirects several courses and serves on the medical school admissions committee. His lab studies protein kinase signaling using Drosophila as a model.


STROCHLIC

Vishal Gohil recently received the 2024 Ivano Bertini Award at the 13th International Copper Conference in Sorrento, Italy. The award recognizes a mid-career or senior investigator whose basic science research has uncovered fundamental aspects of copper biology. Gohil's award-winning research led to the first approved use of a copper-transporting drug in a child with Menkes disease by the Spanish Agency of Medicines and Health Products. Gohil is a professor of biochemistry and biophysics at Texas A&M University. His lab investigates the biochemical and genetic basis of mitochondrial dysfunctions in rare genetic disorders.


GOHIL

Pablo Sobrado has been named the Richard K. Vitek/FCR Endowed Chair of Biochemistry in the chemistry department at Missouri S&T. The position was established by Richard K. Vitek, a 1958 S&T chemistry graduate, and his wife, Marilyn Vitek. Prior to this appointment, Sobrado was a professor of biochemistry at Virginia Tech. Sobrado's lab studies natural product biosynthesis, plant metabolism and defense and xenobiotic resistance in the fields of biochemistry and drug discovery. Sobrado is a member of the Journal of Biological Chemistry editorial board.


SOBRADO

Lynne Maquat won the Dr. Paul Janssen Award for Biomedical Research for her fundamental discoveries about RNA decay in the context of human diseases. She shares the award with Alexander Varshavsky. In addition, Maquat received the Albany Medical Center Prize in Medicine and Biomedical Research for her research on RNA mechanisms that contribute to a wide range of diseases, including spinal muscular atrophy, cancers and autoimmune disorders. Maquat is a professor of biochemistry and biophysics at the University of Rochester School of Medicine and Dentistry. Her lab studies problems with splicing and other steps in messenger RNA production and maturation.


MAQUAT

Joseph Heitman and **Hao Wu** have been named members of the National Academy of Medicine.

Wu is a professor of structural biology, biological chemistry and molecular pharmacology at Harvard Medical School. Her lab uses cryogenic electron microscopy and other biophysical methods to understand molecular complexes involved in innate immunity, including signalosomes and pore-forming complexes like gasdermin D. She won the ASBMB Bert and Natalie Vallee Award in Biomedical Science in 2024.


WU

Heitman is a professor and chair of molecular genetics and microbiology at the Duke University School of Medicine. His contributions include the discovery of TOR and FKBP12 as targets of the immunosuppressive natural product rapamycin and definition of nutrient sensing pathways in the model yeast Saccharomyces cerevisiae. He received the ASBMB AMGEN Award.


HEITMAN

Lena Pernas has been named a Packard Fellow by the David and Lucile Packard Foundation. She will receive $875,000 over five years to pursue her research. Pernas is an assistant professor of microbiology, immunology and molecular genetics at UCLA. Her research focuses on how organellar function and metabolism are rewired during infection to counteract pathogens such as Toxoplasma.


PERNAS

Shelby Sliger and **Tara Young** have been named Astronaut Scholars by the Astronaut Scholarship Foundation, which was founded by the Mercury 7 astronauts. They are two of 71 awardees in their junior and senior year of college studying science, technology, engineering or mathematics with the intent to pur-

sue research or advance their field.

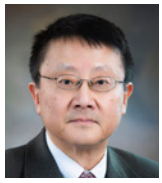
Sliger is an undergraduate biochemistry major at Purdue University. She conducts research on epigenetics and chromatin remodeling in plants with Joe Ogas.


SLIGER

Young is an undergraduate biochemistry major at the University of Washington. She conducts research on the role of role of a novel DNA-binding protein in DNA replication with Monica Guo, an assistant professor.


YOUNG

Patrick Sung received the 2024 Basser Global Prize from the Basser Center for BRCA at Penn Medicine. This honor recognizes a leading scientist who has advanced BRCA1 and BRCA2-related research, providing $100,000 in unrestricted support of the winner's research efforts, a Basser sculpture and a $10,000 honorarium. Sung is director of the Greehey Children's Cancer Research Institute, associate dean of research and a professor of biochemistry and structural biology at the University of Texas Health Science Center at San Antonio. His lab studies DNA damage repair and homologous recombination as a mechanism for repairing double-strand DNA breaks. He has been an associate editor of the Journal of Biological Chemistry since 2014.


SUNG

Bruce Ames

Bruce Ames, an emeritus professor at the University of California, Berkeley, and a senior scientist at Children's Hospital Oakland Research Institute, died Oct. 5, 2024. He was a former Journal of Biological Chemistry editorial board member and an American Society for Biochemistry and Molecular Biology member for 53 years.

In the 1970s, Ames invented a cheap and easy way to assess mutagenicity that helped identify many environmental and industrial carcinogens; it became known as the Ames test.

Ames was born Dec. 16, 1928, in New York City to Maurice and Dorothy (Andres) Ames. Ames earned an undergraduate degree in chemistry and biochemistry at Cornell University before heading to the California Institute of Technology for graduate studies. Ames did postdoctoral work on enzymology at the National Institutes of Health and stayed on as an independent investigator.

There, he discovered histidine pathway enzymes and developed Salmonella mutants unable to produce histidine to test for chemical carcinogens. When these mutants are exposed to a suspected mutagen, only those that mutate to restore histidine production grow on histidine-free media, which allowed Ames to quantify relative mutagenesis.

In 1967, Ames moved to the University of California, Berkeley. There, he further developed his eponymous test. These efforts led the test to be widely adopted in industrial and regulatory settings. In addition, Ames studied the regulation of the histidine operon by transfer RNAs. In early 2000, Ames moved his lab to the Children's Hospital of Oakland Research Institute.

Ames received the National Medal of Science in 1998, was an elected member of the National Academy of Sciences and the American Academy of Arts and Sciences and an American Association for the Advancement of Science fellow.

Ames is survived by his wife, Giovanna Ferro–Luzzi Ames; their children, Sofia and Matteo Ames; and two grandchildren.

— Comfort Dorn

Margaret Lee Fonda

Margaret Lee Fonda, a pioneer in biochemistry education and a member of the American Society for Biochemistry and Molecular Biology since 1972, died July 25, 2024, in Louisville, Kentucky.

Born July 13, 1942, in Cleveland, to Albert and Jean Loweth Fonda, she grew up in Alexandria, Virginia, and attended Salem College in North Carolina. She then transferred to the University of Delaware where she received her B.S. in chemistry in 1965. At the University of Tennessee, she earned her Ph.D. in biochemistry in 1968.

Fonda pursued postdoctoral study at Iowa State University where she worked with biochemist David E. Metzler, studying interactions of pyridoxalphosphate analogs with aspartate aminotransferase. She also developed computer methods to generate the spectra of enzyme–inhibitor and enzyme–substrate complexes and study various physiochemical parameters.

Fonda started her independent research career as a lecturer in the biochemistry department at the University of Louisville School of Medicine. According to a news obituary, she was one of few women teaching at the time and was also younger than many of her students, who were mostly male.

In her lab, Fonda initially aimed to find activities of different decarboxylase and aminotransferase enzymes in mouse brains. Later, she extended her research to explore the effect of modifications of vitamin B6 phosphatase and pyridoxal phosphatase present in human erythrocytes. She published more than 39 papers in peer-reviewed journals and had over 965 citations.

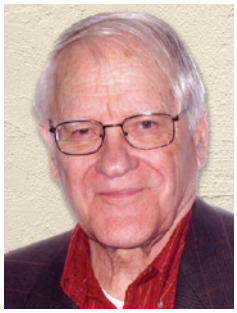
In 1976, Fonda married George Herbener, a colleague at the medical school, and they retired together in 1995. Fonda loved to travel around the world and was a skilled photographer.

She is survived by a sister, her five stepchildren and extended family.

— Swarnali Roy

IN MEMORIAM

Arnis Kuksis



Arnis Kuksis, a professor emeritus at the University of Toronto who studied the complex mechanisms dictating lipid metabolism, died Sept. 2, 2024. He was 96 and had been a member of the American Society for Biochemistry and Molecular Biology for 42 years.

Born Dec. 3, 1927, in Valka, Latvia, Kuksis earned bachelor's and master's degrees from Iowa State University and a Ph.D. in biochemistry from Queen's University in Kingston, Ontario. He remained in Ontario for his postdoctoral studies at the Royal Military College, then returned to Queen's to join J.M.R. Beveridge's lab as a research associate in lipid biochemistry.

After he was promoted to assistant professor of biochemistry at Queen's, Kuksis' research focused on the analysis and characterization of triglycerides and phospholipids. He pioneered the use of high-temperature gas chromatography to resolve molecular species of triglycerides from natural fats and oils, a technique that became a standard in lipid research. His work extended to steryl esters and glycerophospholipids, enhancing the understanding of lipid structures and their metabolic pathways.

Kuksis authored numerous publications that had a significant impact on the field of lipid biochemistry. His research on the metabolism of molecular species of diacylglycerophospholipids and the lipid analysis of glycoinositol phospholipid membrane anchors are among some of his most cited works.

He is survived by his children Anda, Davis and Inga; seven grandchildren; and six great-grandchildren.

— Meg Taylor

Horst Schulz

Horst Schulz, a professor emeritus at City College of New York and at the City University of New York Graduate Center in Manhattan, died Oct. 15, 2024, at the age of 88. His work concentrated on understanding mitochondrial fatty acid metabolism. He was a member of the American Society for Biochemistry and Molecular Biology since 1971.

Schulz was born Sept. 16, 1936, in Berlin. He obtained his master's and doctoral degrees from the Technical University of Berlin, where he worked with Ferdinand Bohlmann on quinolizidine natural products.

Schulz became a research associate at Weill Cornell Medical College, where he worked with Vincent du Vigneaud on penicillamine-derived antagonists of oxytocin. After three more years at the Technical University of Berlin, he joined the Duke University Medical Center in 1968 to work with Salih J. Wakil, where he published on the mechanisms of fatty acid biosynthesis.

In 1970, Schulz began his tenure at City College and at the CUNY Graduate Center. He retired as full professor and executive officer of the CUNY Graduate Center's Biochemistry Doctoral Program in 2007.

Schulz's research interests concentrated on fatty acid metabolism, on which he published 130 of his 140 papers. His published work has been cited over 5,000 times.

Schulz mentored 33 doctoral students. The Horst Schulz Award was created in his honor and is awarded annually to the CUNY biochemistry doctoral student with the best first-author paper.

Horst Schulz leaves behind his wife Barbara; his children, Kurt, Karina and Nadja; his brother, Knut; and four grandchildren.

— Manfred Philipp

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