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ASBMB TODAY

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

Biochemical innovations for rare diseases

ASBMB

Calling all science advocates

Get involved through the ASBMB advocacy action center.

ASBMB recognizes the impact of the current political climate on the scientific community. The society actively represents its members by engaging with policymakers, coalitions and other scientific societies to advocate for basic science research.

Stay engaged with our work through the action center:
asbmb.org/advocacy/action-center

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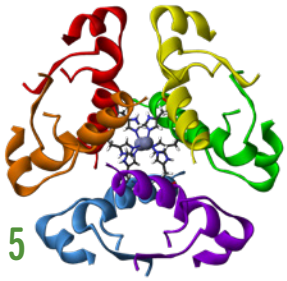

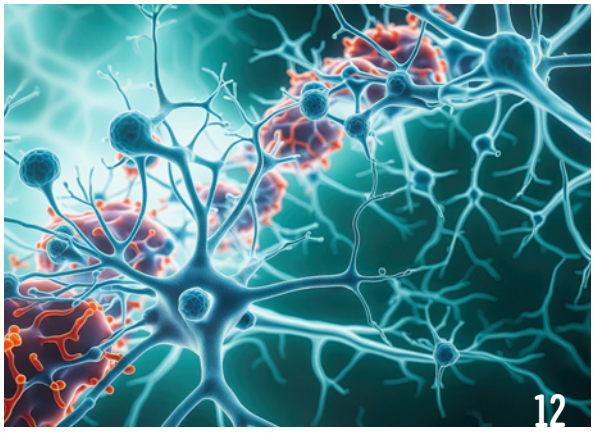

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ASBMBTODAY
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Supporting science through advocacy and community building

By Joan Conaway

JOAN CONAWAY

We are in the midst of one of the most challenging times in memory for American science. Like you, I have witnessed with great concern the tide of actions threatening the U.S. scientific enterprise, which for more than 80 years has been an essential American pillar of national and local economic strength, better treatments and cures for disease, stronger agriculture and much more.

Recent actions include slashing federal agency grants, budgets and staff as well as mandates that cut at the heart of scientific progress, transparency and workforces. Over the last five months, the American Society for Biochemistry and Molecular Biology and its allies have been working tirelessly to reach Congress and the administration to communicate the enormous harms and urging them to reverse the dismantling of the nation's research and innovation enterprise. In May, ASBMB and allied societies also filed a "friend of the court" brief in a major lawsuit, articulating our strong objection to the termination of the Maximizing Opportunities for Scientific and Academic Independent Careers, or MOSAIC, grants and affirming the tremendous value of diverse perspectives in science.

ASBMB updates its advocacy action center regularly with the latest news, actions and opportunities to mobilize, and I encourage you to visit often. It also highlights vital partners, including Research!America, the Coalition for the Life Sciences, the Federation of American Societies for Experimental Biology and the American Association for the Advancement of Science, ensuring we work as one with voices from basic scientists, industry, patient advocates and more. In May, ASBMB members also convened in Washington, D.C., for the society's annual Capitol Hill Day, where volunteers and staff visited 60 congressional offices to advocate for federal funding and grants supporting young scientists.

While visiting Capitol Hill is important, scientists need to think about what we can do regularly if we are to make a difference, and ASBMB is here to help. It is vital that policy makers hear directly from constituents and your institutions about how these federal actions and cuts negatively affect the nation and your communities.

Here's how you can act now and support these efforts:

- Write to your lawmakers and urge them to reject the proposed cuts and policy changes at U.S. science agencies and reaffirm their support for biomedical research.
- Spread the word on your social media platforms.
- Request a local congressional meeting and prepare for it.
- Share your story. You can do this through ASBMB's formal campaign site, and we will assemble them for future use. You can also share your story locally — with neighbors, friends and others who need to understand the impact of science in your local community.

ASBMB will continue to create opportunities to amplify our voices. Together, scientists can help convey the immeasurable value of basic research and ways that science serves us all.

In these times, I want to stress another vital resource: ASBMB's community. That powerful factor especially occurred to me as 2,800 of us gathered at ASBMB 2025 united by our passion for fundamental research. Memories of that meeting are a touchstone of hope and motivation. In particular, I think of Lawrence Tabak, winner of the 2025 Howard K. Schachman Public Service Award, who during the meeting shared his deep concern for the scientific enterprise. At the same time, he noted that science has faced adversity before, and we will reach a brighter day: "All hope is not lost. Whether you are a freshman in college, a professor emeritus or everyone in between, you have the power to do something proactively."

I am confident that our community is part of what will get us through these challenging times. ASBMB advocacy joins other key facets of our work — celebrating and sharing outstanding science, highlighting translational applications, providing collegial support and committing to support future generations. I hope you will continue to engage with ASBMB, and each other, for this kind of support. Through our scientific work and our personal interactions, we can be an inspiration to each other.

As I said in Chicago, I am deeply grateful to be in a community of determined and curious people. Together, we must take action and maintain hope individually and collectively. Both are essential as we strive to catalyze the infinite potential of molecular life sciences and ensure that the long arc of science bends toward progress.

Joan Conaway, president of ASBMB, gives opening remarks at ASBMB's 2025 annual meeting in Chicago.

Together, scientists can help convey the immeasurable value of basic research and ways that science serves us all.

Joan Conaway (jconaway@asbmb.org) 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.

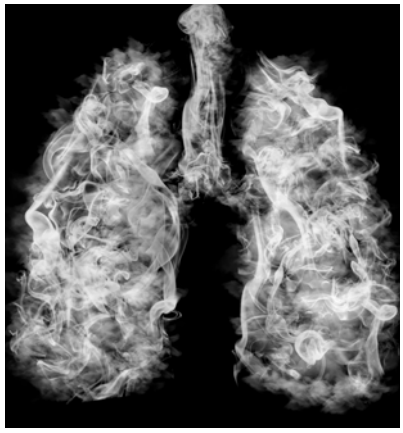
E-cigarettes drive irreversible lung damage via free radicals

By Andrea Lius

Thousands of substances in tobacco cigarettes can cause cancer. In the early 2000s, Chinese pharmacist Hon Lik invented electronic cigarettes, or e-cigarettes, which many believed were a safer alternative to tobacco cigarettes. E-cigarettes deliver nicotine, the substance in tobacco cigarettes, making them addictive, without many of the cancer-causing, or carcinogenic, substances found in traditional cigarettes. However, scientists have found that e-cigarettes may not be as safe as many once thought. For instance, e-cigarettes bring harmful side effects such as an increased risk of cardiovascular disease.

In a recent study published in the **Journal of Biological Chemistry**, an international group of researchers led by Jay L. Zweier of the Ohio State University found that nicotine exposure through the use of e-cigarettes, colloquially known as vaping, led to lung injury in mice. The researchers showed that aldehyde oxidase, or AOX, a critical enzyme in the process of nicotine metabolism, mediates this effect. In this study, Zweier and colleagues established a link between e-cigarette vapor exposure, nicotine metabolism and lung injury and explored the molecular basis for this connection.

"Inherently, they're all still nicotine delivery devices," Zweier said, referring to e-cigarettes. "So, we wanted to know if there is toxicity from nicotine itself."



Zweier's team previously showed that AOX generates superoxide, a reactive oxygen molecule that causes cell stress and damage. Furthermore, another group reported that nicotine iminium, or NICI, an intermediate product of nicotine metabolism, is a potent AOX substrate. In the current study, Zweier and his colleagues directly showed that NICI metabolism by AOX triggers high-level production of superoxide. They further sought to understand the connection between extended nicotine exposure, superoxide generation by AOX and lung injury.

To achieve this, the researchers designed a study in which they placed mice into chambers where they received whole-body nicotine exposure from e-cigarette vapor, at similar concentrations a person would receive while vaping, six hours per day for up to 16 weeks. The team then assessed the mice's organ damage using biochemical methods.

"Using a mouse model allowed us to predict the effects of chronic exposure in humans," Zweier said. "When we normalize the lifespans, about a year of exposure in mice corresponds to about three decades in humans."

Zweier and colleagues showed that exposure to nicotine from e-cigarette vapor increased the expression of the Aox1 gene in mice by more than six times the normal amount. Zweier and his colleagues found e-cigarette vapor also drove increased superoxide levels as well as molecular markers that indicate oxidative damage in the lung, such as carbonylated proteins, oxidized guanine species and nitrated tyrosine. They reversed these effects when they treated mice with raloxifene, an AOX inhibitor.

"E-cigarette juice can have a nicotine concentration as high as 150 millimolar," Zweier said. "And we saw that (lung) toxicity occurs at micromolar levels (1000 times less). It's just off the scale."

In the future, Zweier hopes to investigate the doses and duration of exposure to nicotine that will cause lung injury. This is important, he said, because the U.S. Food & Drug Administration is currently attempting to regulate e-cigarettes, and these devices are the most popular tobacco products among youth.

"We need to know what level of nicotine is tolerable, and what level is toxic," Zweier said. "Not only to the lung, but also to the whole body."

DOI: 10.1016/j.jbc.2024.107626

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

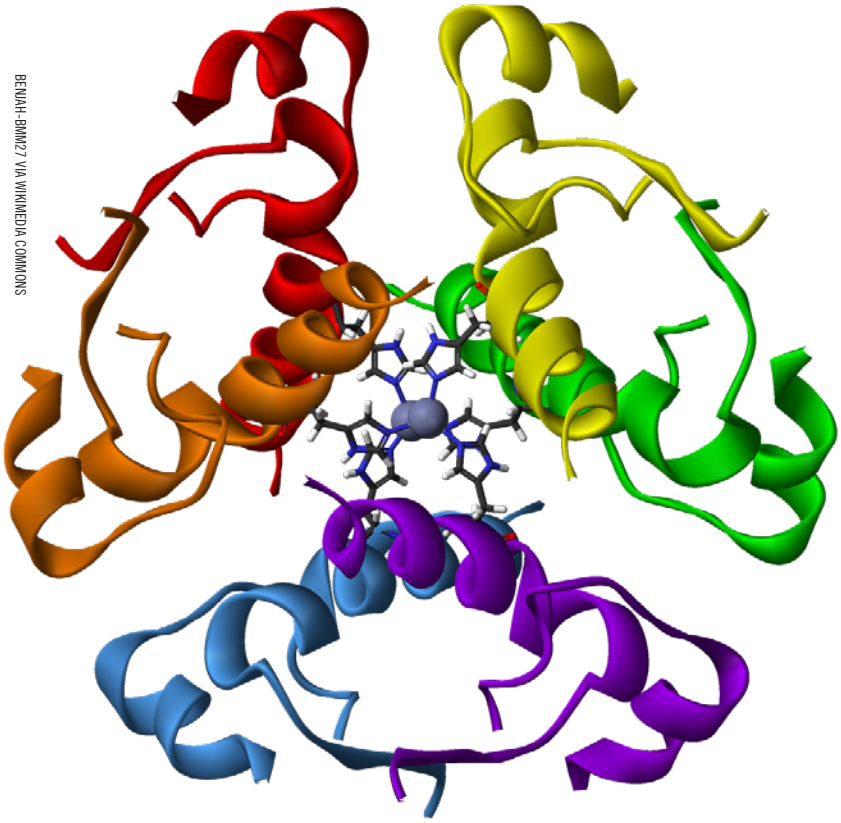
How transcription factor mutations shape diabetes risk. Elusive zebrafish enzyme in lipid secretion. Targeting toxins to treat whooping cough. Read about papers on these topics recently published in the *Journal of Biological Chemistry.*

By Isabel Casas and Emily Ulrich

How transcription factor mutations shape diabetes risk

Diabetes affects hundreds of millions of people worldwide. The disease features elevated blood glucose levels and disrupted fat and protein metabolism. The musculoaponeurotic fibrosarcoma, or MAF, family of transcription factors regulate various processes in tissue development, including hormone production in pancreatic islet cells. Within this family, MAFA and MAFB are essential for development and maturation of insulin- and glucagon-producing cells.

Previous research showed that MAFA and MAFB expression is affected in diabetes, both Type 1 and Type 2. In addition, a mutation in MAFA that prevents phosphorylation of a key serine residue causes monogenic diabetes. Mutation of the equivalent residue in MAFB leads to a pediatric multisystem disorder. In a recent **Journal of Biological Chemistry** article, Jeeyeon Cha, Xin Tong and Katie Coate from Vanderbilt University and collaborators in the U.S. examined how mutations in conserved DNA-binding domains of the MAF proteins impact their regulation of the insulin gene. The authors used targeted mutagenesis and artificial intelligence structure prediction using AlphaFold 2 for their analysis.



BENJAH-BMM27 VIA WIKIMEDIA COMMONS

A ribbon diagram of insulin as a hexamer stabilized by two zinc molecules with the coordinating histidine residues shown as sticks.

They found one MAFA variant, with a mutation in the conserved DNA-binding region, that exhibited normal activity. The equivalent mutation in MAFB did not retain normal activity. Therefore, the researchers searched for structural differences between the MAFA and MAFB proteins outside of the DNA-binding region that might also contribute to its activity. Their AlphaFold 2 models showed that the two proteins differed in the C-terminal domains. The researchers created chimeras by exchanging the two C-terminal domains of MAFA and MAFB, which changed how each protein regulated the insulin gene. These results help clarify differences between MAFA and MAFB, which of their domains affect activity and possible ways that they contribute to different disease states.

Future studies will focus on regions of MAFA and MAFB that may interact with other coregulators of the insulin gene.

DOI: 10.1016/j.jbc.2024.107938

To read more JBC news, scan the code.

Elusive zebrafish enzyme in lipid secretion

Lipids provide energy and structural components during vertebrate development. Lipoproteins aid in lipid transport throughout the body, and synthesized lipids are also stored in lipid droplets within the cell. Embryos of the model organism zebrafish receive nutrients, including lipids, from a maternally deposited yolk through extraembryonic tissue called the yolk syncytial layer, or YSL. Lipid nutrients are released through the production and secretion of lipoproteins rich in triacylglycerol, or TAG. Scientists want to understand which enzymes direct TAG production to lipoproteins for secretion versus lipid droplets for storage.

In a recent **Journal of Biological Chemistry** article, Meredith Wilson from Johns Hopkins University and U.S. and U.K. colleagues investigated the fate of TAG in zebrafish lacking certain TAG synthesis enzymes. They found that zebrafish lacking diacylglycerol acyltransferase-2, or Dgat2, can still produce TAG, but the TAG is channeled for YSL storage instead of secretion, as noted by the excessive accumulation of lipid droplets in the YSL that make it look opaque.

The authors concluded that zebrafish have multiple enzymes to ensure TAG production remains intact. Future studies will identify the enzyme that fails to properly channel TAG to lipoprotein formation for secretion in embryonic development.
DOI: 10.1016/j.jbc.2024.107973

Targeting toxins to treat whooping cough

Whooping cough is an infectious respiratory disease caused by the bacteria Bordetella pertussis. According to the U.S. Centers for Disease Control and Prevention, whooping cough cases are rising. While early antibiotic treatment can be effective, most diagnoses do not occur until after this therapeutic window has passed.

In a recent **Journal of Biological Chemistry** article, Stefanie Lietz from Ulm University, Germany, and an international team explored the human peptidome — the complete collection of peptides in the human body — for pertussis toxin, or PT, inhibitors using peptide libraries, fractionation and mass spectrometry. They identified the liver protein α1-antitrypsin, or α1AT, as a potent PT inhibitor. Additional cell culture and molecular modeling experiments indicated that α1AT likely binds to PT in solution and thus blocks the toxin from making contact with its known host glycoprotein cell surface interaction partner for endocytosis.

Patients with genetic α1AT deficiency receive synthetic α1AT in the clinic. Therefore, α1AT may be able to be repurposed to treat PT-mediated pertussis pathogenesis.
DOI: 10.1016/j.jbc.2024.107950

Isabel Casas is ASBMB's publications director.

Emily Ulrich is the ASBMB's science editor.

New mass spectrometry assay speeds up UTI diagnosis

By Jessica Desamero

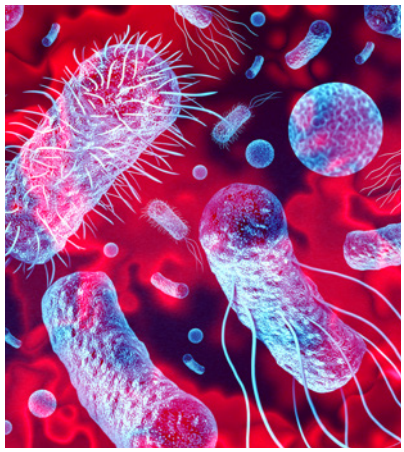
The urinary tract is a common site for bacterial infections that affect millions worldwide. UTIs are most often caused by E. coli and are treated with antibiotics.

To test for the bacterial cells in urine, doctors use a culture system to grow microorganisms. They then use an instrument called matrix-assisted laser desorption ionization time-of-flight, or MALDI–TOF, mass spectrometry to detect and identify bacterial species in the culture.

MALDI–TOF analysis is generally accurate, but because of the time needed to grow culture, it takes 24 to 48 hours to obtain results. This can delay treatment, prolong patient suffering and may contribute to increased occurrence of antimicrobial-resistant pathogens. Therefore, with bacterial infections such as UTIs, researchers want to develop quicker methods of detection.

In a recent study published in the journal **Molecular & Cellular Proteomics**, researchers at the Centre Hospitalier Universitaire de Quebec proposed a faster method to identify and quantify UTI infections in urine samples.

Clarisse Gotti, Florence Roux–Dalvai and their team used a technique called liquid chromatography–mass spectrometry, or LC–MS, to identify and quantify proteins. They suggested clinicians should use LC–MS for urine analysis, rather than MALDI–TOF because it is more efficient, specific and sensitive.



Most importantly, LC–MS can analyze urine samples directly, eliminating the need to grow bacterial cultures.

"There is, for now, a strong limitation in infection diagnosis in using bacterial cultures," Roux–Dalvai said. "We don't need a bacterial culture (for LC–MS)."

In a previously published study, the team established that LC–MS can identify bacterial species in urine by monitoring MS protein signatures, which can then be used for UTI diagnosis. Moreover, they combined LC–MS with machine learning algorithms to better differentiate unique peptides and distinguish UTI-causing species from other bacterial species. This allows for more accurate analysis.

A few downsides to this first method include a lack of robustness, high costs and relatively low throughput. In their recent paper, the researchers redesigned their initial strategy so that their method can be run routinely for potential clinical use.

"We demonstrated that the bacteria can not only be identified through this process, but they can also be quantified, which is important in the case of UTIs, because you need to know the level of infection to know if it requires an antibiotic therapy or not," Roux–Dalvai said.

In their redesign, they reduced the volume of urine required for analysis, which allows for faster sample preparation. They also switched from a more expensive, high-resolution detection mode and MS instrument to one that costs less. Their updated strategy reduced turnaround time while maintaining the quality of bacterial detection.

"For the moment, we can have results in about 10 to 15 minutes," Roux–Dalvai said.

In the future, they hope to further reduce the time needed to process urine samples and enrich the UTI peptide signature to make the bacteria easier to detect.

"We want to add information in the signature about whether the strains are resistant or not," Roux–Dalvai said.

After that, they aim to repurpose their method to detect other types of infections.

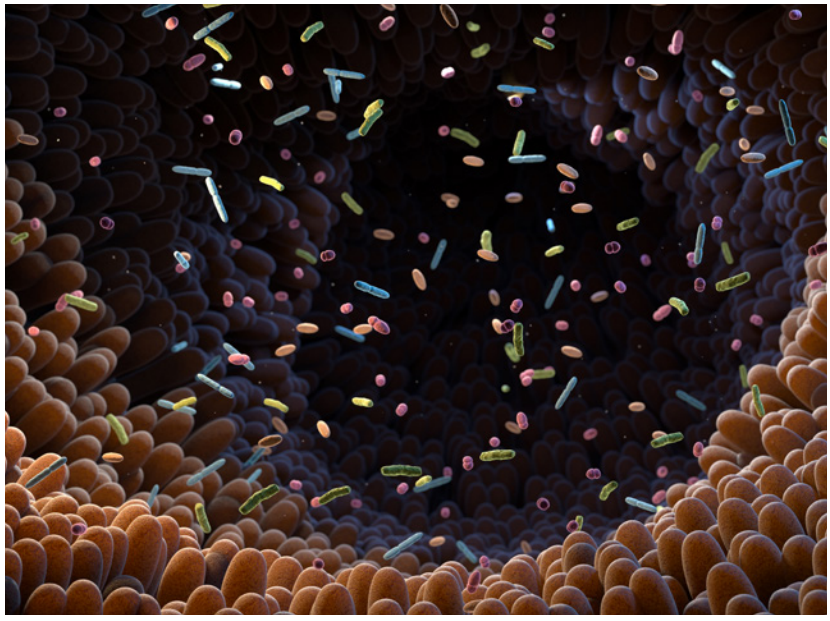
DOI: 10.1016/j.mcpro.2024.100832

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

From the journals: MCP

New mass spectrometry tool accurately identifies bacteria. Scientists identify pan-cancer biomarkers. New tool matches microbial and metabolic metaproteomic data. Read about papers on these topics recently published in *Molecular & Cellular Proteomics*.

By Ecem Arpaci



New mass spectrometry tool accurately identifies bacteria

Scientists identify bacterial species by analyzing their proteins using mass spectrometry, or MS. This technique first fragments proteins into smaller peptides using an enzyme called trypsin. These sequences can then be compared to references in databases. The largest MS-based studies on bacteria have focused on just a handful of species, so a team of researchers recently created a resource for mapping data onto a more diverse population of bacteria.

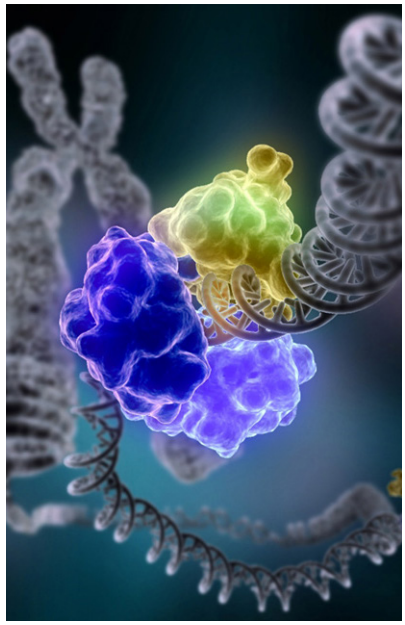
Miriam Abele, Armin Soleymaniniya and colleagues at the Technical University of Munich developed MS2Bac, a software system that enables bacterial identification from protein data. They published their resource in **Molecular & Cellular Proteomics**. MS2Bac maps tryptic peptides onto reference bacterial species or strains, achieving almost perfect accuracy for species identification. To develop this tool, the team first performed MS on the proteins from over 300 bacterial species to create a reference database. They also compared their identification method with other approaches, such as Fourier transform infrared spectroscopy, and found that MS2Bac was the most accurate.

MS2Bac can also identify specific proteins, including antibiotic resistance markers. It covers many hypothetical proteins, which are not well understood, providing a basis for further functional studies. This is the first study to incorporate single-cell organisms into the ProteomicsDB database, a proteomics resource for multiomics analyses. This tool will greatly help researchers and clinicians determine bacterial species from clinically and environmentally relevant samples.

DOI: 10.1016/j.mcpro.2025.100917

Scientists identify pan-cancer biomarkers

Genomics and transcriptomics have successfully identified many therapeutic targets for cancer. However, changes in protein abundances and their chemical modifications can also drive tumor progression. To consider this additional dimension, Guosheng Hu, Zao-zao Zheng, Yao-hui He, Du-chuang Wang and colleagues at Xiamen University analyzed RNA and protein data from thousands of patients with 13 cancer types. They published their findings in **Molecular & Cellular Proteomics**. Using bioinformatics tools, they identified upregulated and downregulated genes specific to each cancer type as well as genes common to most types analyzed.



TOM ELLENBERGER, WASHINGTON UNIVERSITY SCHOOL OF MEDICINE IN ST. LOUIS, VIA WIKIMEDIA COMMONS

DNA ligase encircles the double helix to repair a broken strand of DNA.

The team discovered that tissue-specific genes were downregulated at both RNA and protein levels in all cancer types, indicating a loss of tissue identity. They showed that many genes involved in messenger RNA splicing, inflammation, fatty acid metabolism and complement coagulation cascade, were dysregulated across several cancer types. The authors also found that ADH1B, the alcohol dehydrogenase that converts ethanol to acetaldehyde, was significantly downregulated in all cancer types. Conversely, the ribonucleotide regulatory subunit RRM2 was overexpressed. These proteins are examples of potential pan-cancer biomarkers, which can be used to discern cancer tissues from normal cells and potentially inform novel therapeutic strategies.

To read more MCP news, scan the code.

Effective cancer treatment also requires knowledge of the tumor's stage of progression. To identify biomarkers for each tumor stage, the team analyzed how the cancer proteome changed throughout tumor progression. They used these findings to build models for tumor stage classification of several cancer types based on these biomarkers. They further constructed prognostic risk stratification models for corresponding cancer types based on dysregulated genes. They found that these models, when combined with the tumor-node-metastasis classification system, predicted cancer patient prognosis more accurately than either approach individually.

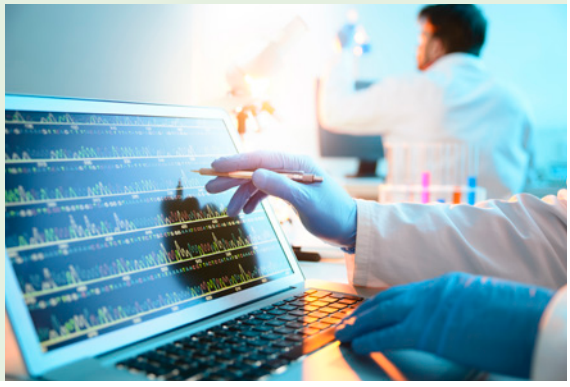
Protein-based approaches like these could be the key to better understanding cancer mechanisms and developing better treatments. Inhibitor drugs targeting RRM2 and other differentially expressed proteins identified in this study could be used to treat a range of cancer types and will be investigated further in future studies.
DOI: 10.1016/j.mcpro.2025.100919

New tool matches microbial and metabolic metaproteomic data

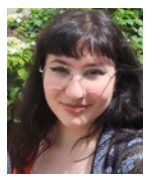
Metaproteomics is the study of all proteins expressed in entire communities, such as soil and gut microbiomes, and often requires bioinformatics approaches to analyze large quantities of data. However, until recently, none of these tools mapped protein or peptide data onto information about their biological functions.

To address this gap, in a recent study in **Molecular & Cellular Proteomics**, Tibo Vande Moortele of Ghent University and an international team of researchers created PathwayPilot. This bioinformatics resource combines proteomic data, including protein identification and abundances, with metabolic pathway data. Users can upload peptide or protein data, choose their desired pathways for analysis and export the results in various formats for further analysis. This makes it easier for researchers to identify the biochemical functions of proteins between organisms in a microbial community or between communities. The team tested PathwayPilot's accuracy by analyzing data from a study on lipid metabolism and found that nearly all results matched to a known pathway.

Researchers can use this tool to compare the metaproteomic effects of environmental factors such as temperature or determine the role of a microorganism in its community, with promising applications in medical microbiology and ecology. PathwayPilot currently can only be used to compare two groups, so the team plans to continue updating the resource.
DOI: 10.1016/j.mcpro.2025.100918



Ecem Arpaci is a biochemistry student at Imperial College London and a research intern at Radboud University Medical Center. She is an ASBMB Today volunteer contributor.

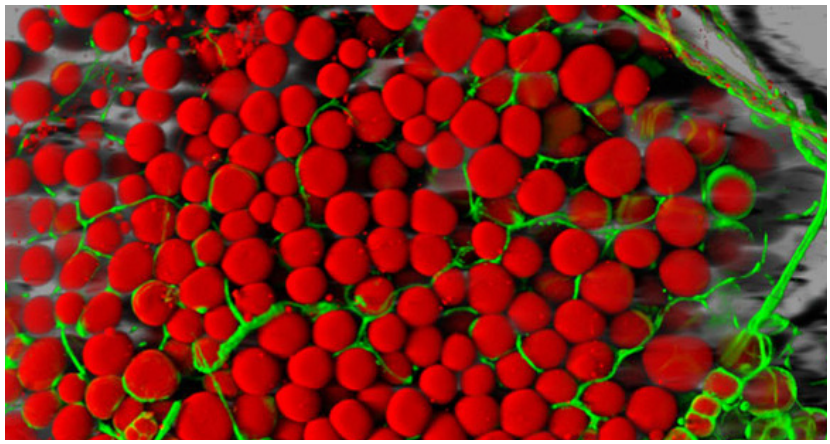


Butter, olive oil, coconut oil — what to choose?

By Seema Nath

Olive, vegetable, canola, avocado, coconut, sunflower seed, almond and many other types of oil line the shelves of most grocery stores in the U.S. Deciding on the appropriate option can be daunting.

To help make the choice easier, Solomon A. Sowah, a research associate, and his team at the University of Cambridge analyzed how various types of dietary fats may impact the health of adults. They published their results in the **Journal of Lipid Research**.



DANIELA MALDE, NATIONAL HEART, LUNG AND BLOOD INSTITUTE/NIH

Murine adipocytes, shown in red, innervated by blood vessels, shown in green.

The health effects of saturated fats, or SFs, may be determined by their chain length and the number of carbon atoms, based on studies that examined the association between plasma fatty acids, or FAs, and the risk of cardiometabolic diseases. For example, plasma SFs with an odd number of carbon atoms are associated with lower risks of Type 2 diabetes and cardiovascular disease, or CVD, risk. Conversely, common SFs with an even number of carbon atoms show potential associations with higher risks of Type 2 diabetes and CVDs.

"We do know these associations exist, but what we are not sure about is how intakes of different dietary fats impact the concentrations of circulating FAs," Sowah said.

Different dietary fats contain varied amounts of individual FAs. Comparative studies on how common dietary fats affect blood levels of a variety of FAs are limited. In addition, existing observational studies are often confounded by inaccuracies in self-reported dietary intake.

The researchers used gas chromatography to measure plasma concentrations of 37 different phospholipid FAs in 96 adults before and after they consumed equal amounts of coconut oil, olive oil or butter for four weeks.

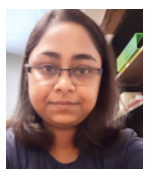
The researchers found that equal amounts of different fat sources altered plasma phospholipid FA concentrations in distinct ways. Coconut oil consumption doubled levels of medium-chain saturated FAs with 12 and 14 carbon atoms, while butter increased odd-chain saturated FAs containing 15 and 17 carbon atoms — an effect not seen with coconut or olive oil. Olive oil led to the greatest rise in oleic acid, a fat commonly found in olives, while coconut oil decreased it. Linoleic acid, which was highest in olive oil compared to coconut oil and butter, did not rise significantly in the olive oil group but did increase in those who consumed coconut oil, though the reason remains unclear.

Despite these changes in blood FA profiles, the study found no strong associations between changes in plasma FAs and shifts in metabolic markers. However, two subtle patterns emerged: higher plasma oleic acid levels were linked to lower high-density lipoprotein, or good cholesterol, and slightly elevated cholesterol markers, while increased trans fats from butter correlated with higher low-density lipoprotein, or bad cholesterol, and triglycerides.

These findings highlight how different dietary fats uniquely influence blood FAs profiles and potentially subsequent risk of cardiometabolic disease and suggest that choosing fats like olive oil, which are lower in saturated and trans fats, may be a heart-healthier option over time.

DOI: https://doi.org/10.1016/j.jlr.2024.100681

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



From the journals: JLR

Targeting Toxoplasma parasites and their protein accomplices. Scavenger protein receptor aids the transport of lipoproteins. Microglial lactic acid mediates neuroinflammation. Read about papers on these topics recently published in the *Journal of Lipid Research*.

By Oluwadamilola "Dami" Oke

Targeting Toxoplasma parasites and their protein accomplices

Toxoplasmosis is an infectious disease caused by the parasite Toxoplasma gondii and is transmitted via contaminated food or feces. The infection can cause a range of symptoms that may be mild or severe, resulting in blindness and brain infection. Current T. gondii therapeutics are not very effective, so scientists need to further investigate potential drug targets.

Sheena Dass and a team of researchers from the Université Grenoble Alpes, France, identified seven genes responsible for expressing enzymes of metabolic interest in these parasites. Their recent article in the **Journal of Lipid Research** characterizes one of these enzymes, T. gondii acyl-CoA synthetase 3, or TgACS3.

TgACS3 was found to be localized in the cytosol of the parasite and to upregulate its parasitic growth while increasing its chances of survival within its host. Gas chromatography–mass spectrometry was implemented to analyze the lipid content in the parasite, which revealed the role of TgACS3 in the uptake and utilization of its host fatty acids, generating the parasite phospholipid layer and maintaining the growth of new parasites.

This study is an important step towards achieving targeted therapeutic mechanisms in the treatment of Toxoplasmosis, as researchers can leverage the findings shared in a more rigorous analysis.
DOI: 10.1016/j.jlr.2024.100645.

Scavenger protein receptor aids the transport of lipoproteins

Lipoproteins are spherical molecules made up of fat and proteins that play a crucial role in transporting lipids, such as cholesterol and triglycerides, from the liver to other tissues in the body. Anton Potapenko of the University of Zurich, and a team in Switzerland recently published a study in the **Journal of Lipid Research** detailing structural characteristics of the scavenger receptor B1, or SCARB1, a protein that mediates lipid exchange between many cell types and facilitates uptake of high-density lipoproteins, or HDL, and low-density lipoproteins, or LDL, in some cell types. The gene encoding SCARB1 produces two major splice variants that share structural similarities but differ in their carboxy-terminal domains. Researchers wanted to understand if these splice variants play different roles in the cellular uptake of LDL and HDL by endothelial cells.

The researchers examined cultured endothelial cells to understand how expression of the two SCARB1 variants affected the binding, uptake and trafficking of lipoproteins. They found that variants 1 and 2 localized to the cell surface, and endosomes and lysosomes, respectively. Overexpression of variant 1 increased both

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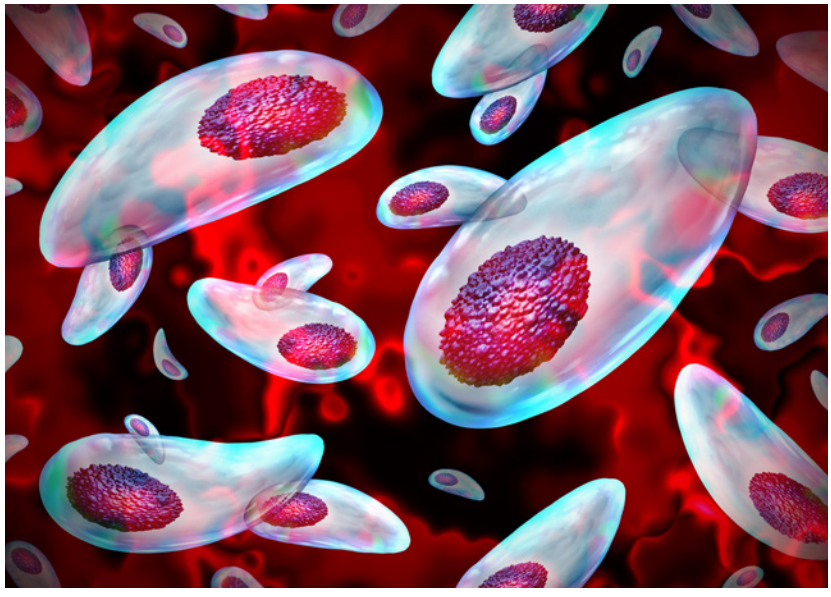

Illustration of Toxoplasma gondii parasites

Microglial lactic acid mediates neuroinflammation

By Vanshika Patel

Neuroinflammation of the central nervous system, or CNS, can cause neuronal death and neurodegenerative diseases. This activates microglia, which release proinflammatory, cytotoxic factors including lactate. Lipid droplets, or LDs, are organelles that store lipids and maintain lipid metabolism. Both lactate and LD accumulation are linked to neuroinflammatory conditions with known microglial–neuronal metabolic interactions. However, researchers do not know how microglia influence the aggregation of abnormal LDs.

Zhuoqing Lan and Shukai Lv at the Zhejiang University and their team in China published a study in the **Journal of Lipid Research** investigating abnormal LD aggregation in neurons using a neuroinflammatory mouse model stimulated with lipopolysaccharide, or LPS, a potent bacterial toxin triggering strong immune responses. RNA sequencing revealed that microglial-derived lactic acid, transported by monocarboxylate transporters, or MCTs, mediates LD aggregation in LPS-activated microglia. This correlated with increased expression of cell death and cholesterol genes in neurons. Neuroinflammation disrupts the bidirectional neuronal–microglial communication as microglia shift toward proinflammatory metabolic processes, releasing factors such as lactic acid.

Treatment with pexidartinib, a potent microglial inhibitor, significantly decreased LD aggregation and lactic acid levels in the neuroinflammatory mouse model. These results suggest targeting microglia-derived lactic acid as a therapeutic for diseases like Alzheimer's and Parkinson's. Future studies will explore the underlying mechanisms of genes related to LD aggregation.

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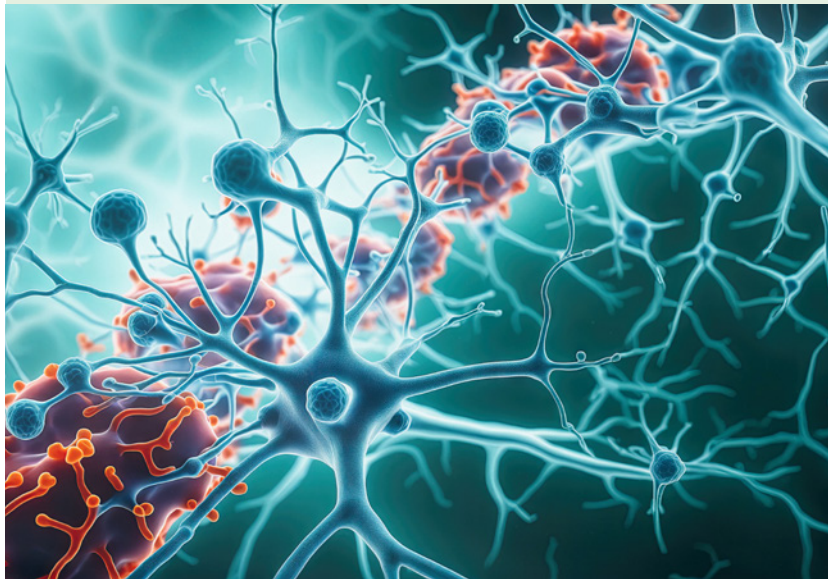

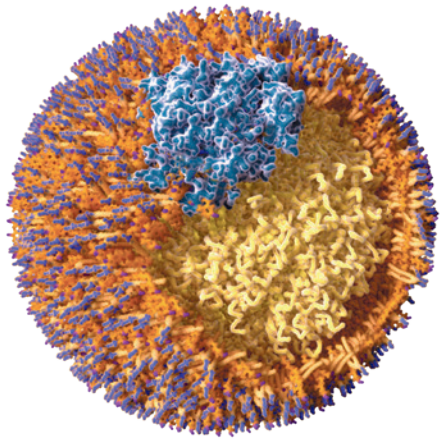

Low-density lipoprotein particle

HDL and LDL binding and uptake. However, overexpression of variant 2 also increased the uptake of either lipoprotein, but not via surface binding. Therefore, the researchers concluded that variant 2 facilitates lipoprotein uptake indirectly by regulatory and indirect mechanisms.

The study suggests that the two major splice variants of SCARB1 facilitate transendothelial transport of HDL and LDL by different mechanisms, either dependent or independent of the adapter proteins. Because of the limitations of overexpression, it will be important to examine how eliminating each SCARB1 splice variant affects cellular lipid metabolism and lipoprotein trafficking.

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Oluwadamilola "Dami" Oke is a Ph.D. candidate of biomedical engineering at the George Washington University and an ASBMB Today contributing writer.

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

ASBMB honors Lawrence Tabak with public service award

By Sarina Neote

The American Society for Biochemistry and Molecular Biology named Lawrence A. Tabak the 2025 Howard K. Schachman Public Service Award winner. This award recognizes an individual who best demonstrates dedication to public service in support of biomedical science. It is named after Schachman, who served as chair of ASBMB's Public Affairs Advisory Committee for more than ten years.

For 25 years, Tabak fulfilled many roles at the National Institutes of Health, including serving as institute director of the National Institute of Dental and Craniofacial Research from 2000 to 2010, principal deputy director from 2010 to 2025 and acting director from 2021 to 2023.

"Public service is so important for just about every facet of life in this country," Tabak said. "What has set this country apart from many others is the devotion and dedication of government resources, including its human capital, to supporting and enhancing scientific innovation, scientific growth and scientific development."

As a researcher and dentist, Tabak moved to the NIH from the University of Rochester School of Medicine and Dentistry in 2000, where his research focused on the structure, biosynthesis and function of glycoproteins.

"I've sort of lived in two worlds professionally," Tabak said. "I grew up in the dental research community and then made the jump from a school of dentistry to a school of medicine and became more of a biochemist and less a dentist."

He continued: "I've lived in these two worlds and my two core places were the International Association of Dental Research and ASBMB."

Tabak provided leadership for numerous trans-NIH activities, including the agency's effort to support team science, enhance rigor and reproducibility in research and improve the peer review process. But Tabak said the highlight of his career was the scientific talent with which he worked.

"I'm most proud of the accomplishments of all the people that I had the incredible pleasure to work with," he said. "Be it in the research lab ... (or) the administrative side, I've had an amazing cohort of people who I've worked with. Each of them has gone to bigger and better things, either within NIH or elsewhere."

Throughout Tabak's tenure at the NIH, he ran his own lab studying glycoprotein biosynthesis and function. Much of his work focused on mucin-type O-glycans. His discoveries have implications in numerous diseases, such as cancer, cystic fibrosis, inflammatory bowel disease and more.

Tabak emphasized that scientists have much work to do to convey the importance of basic science to the public.

LAWRENCE TABAK

"We have to continue to work hard at explaining how basic fundamental discovery is what ultimately drives applications," Tabak said. "We have to talk about it in terms that underscore and reinforce the notion that fundamental discovery is really what ultimately drives all the applied advances that we see around us."

Sarina Neote is ASBMB's director of public affairs.

ASBMB undergraduate education programs foster tomorrow's scientific minds

By Allison Frick

Learning together and sharing experiences on teaching practices is an important benefit of ASBMB membership for scientist–educators.

Developing the next generation of scientists is woven into the fabric of the American Society for Biochemistry and Molecular Biology's mission to build and empower a broad community of molecular life scientists to advance discovery. The society offers multiple programs that help prepare tomorrow's scientists by supporting both scientist–educators and undergraduate institutional departments. They also form valuable forums that enable scientist–educators to engage one another on best practices for training, education and professional development.

Opportunities to gather and explore

Learning together and sharing experiences on teaching practices is an important benefit of ASBMB membership for scientist–educators. ASBMB's annual meeting supports all of their important responsibilities: as researchers, they can dig deep into scientific discoveries; as educators, they can also attend workshops and presentations on educational research. The meeting also encourages the next generation of scientists through the Undergraduate Poster Competition. Periodic webinars and virtual events help interested members connect year-round.

The Transforming Education in the Molecular Life Sciences meeting, or TUEMLS, is a signature forum to dive deep into education and training. It occurs biannually during the summer and provides opportunities for educators to learn and share engaging approaches that support students.

"Suddenly I found this community of faculty who were practitioners of biochemistry education," Betsy Martinez–Vaz, TUEMLS organizer and professor of biology at Hamline University, said. "And it really opened my eyes to the different ways in which I could make my courses better, the different ways in which I could serve all the students in my classroom much better."

Dan Dries, a fellow TUEMLS organizer and assistant professor of chemistry and chemistry education research at Chapman University, said science pedagogy standards are moving toward national competency-based instruction and alternative grading practices.

The 2025 National Academies report "Transforming Undergraduate STEM Education" highlights competency-based education as a personalized, self-directed approach grounded in students' interests and experiences. It also contrasts traditional grading, which often lacks reflection on learning objectives, with alternative methods that promote self-assessment and better communication. These al-

MATT HERP

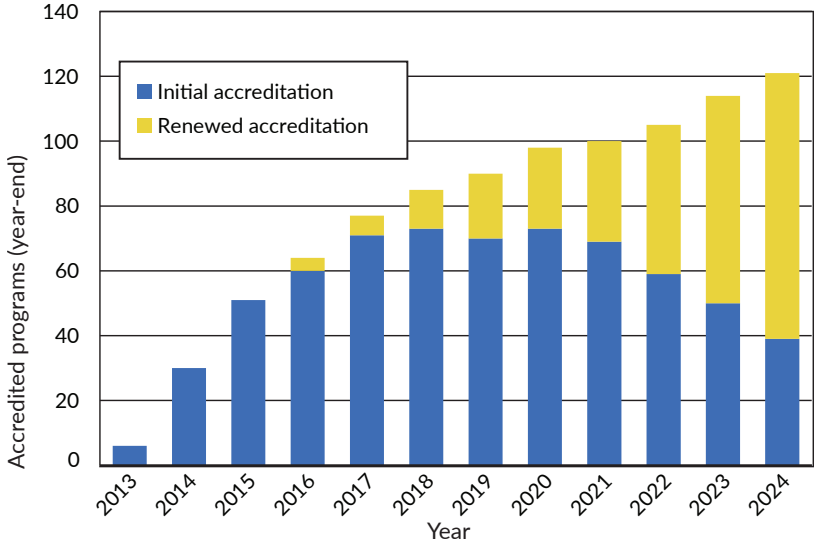
Undergraduates Bhumika Balani and Nitya Punjal of Nova Southeastern University examine a 3D protein model at ASBMB's 2025 annual meeting in Chicago.

ternatives — like specifications-based and standards-based grading — can enhance feedback and help students develop a growth mindset.

"There's also been a consistent push for more active learning and the implementation of course-based undergraduate research experiences," Dries said. "So, we thought it would be a great opportunity to have a space (TUEMLS) where we could talk about, more holistically, what a contemporary life sciences education could, and frankly, maybe must be."

Martinez–Vaz said she focuses on culturally responsive pedagogy, as well as concepts, themes and tools from a variety of practitioners.

Much like Martinez–Vaz's own teaching practices, the National Academies report recommends a comprehensive, collaborative teaching approach and offers a common framework for academic dialogue. It outlines seven principles for equitable and effective teaching, emphasizing that courses should be student-centered. The report also highlights culturally responsive teaching, which values and leverages the diverse backgrounds, experiences and goals students bring to the classroom.



Shown is the growth of ASBMB accredited programs over time. Bars represent the total number of programs actively accredited at the end of the specified year, divided into programs covered by an initial accreditation (blue) and programs covered by a subsequent renewal (yellow).

Recognizing program excellence

Since 2013, ASBMB has recognized excellence in undergraduate programs in BMB and related fields through an accreditation program that provides a national, independent, outcomes-based evaluation mechanism. Starting with six approved programs, today's ASBMB's accredited programs total 121 at 115 institutions. Programs can be fully or partially accredited, and the accreditation is renewed on a regular basis.

Hampden–Sydney College's biochemistry and molecular biology program earned a full seven-year ASBMB accreditation in 2023. According to Michael Wolyniak, a professor of biology and director of undergraduate research at H-SC, he and his colleagues sought out this honor to confirm and signify their program's excellence.

"ASBMB accreditation was crucial for the successful introduction of a biochemistry and molecular biology major at my institution," Wolyniak said. "The accreditation showed my colleagues across the institution that the program we developed has been vetted and approved by world-class experts in biochemistry and molecular biology education. The accreditation also shows prospective students that they will receive a sound biochemistry and molecular biology education."

Earlier this year, Nathan Vanderford, an associate professor at the University of Kentucky College of Medicine and ASBMB's Education and Professional Development Committee chair, and John Tansey, a professor of chemistry at Otterbein University and ASBMB's Education Subcommittee chair, worked with the ASBMB Council to streamline the accreditation application and renewal process.

MATT HERP

Nathan Henderson, a recent graduate of Purdue University, discusses his research with Mona Al–Mugotir, an instructor at the University of Nebraska–Lincoln and ASBMB Membership Committee member, at ASBMB 2025.

According to Vanderford, "Our goals were to maintain standards while also creating greater capacity to sustainably administer the program."

Tansey added, "The changes have many benefits, including maintaining quality, rigor and intentions of the program; reducing burden for applicants, reviewers and staff; and creating some space to improve or grow the program."

The accreditation process includes a National Institutes of Health–style review where the primary and secondary reviewers present the applicants' evaluation for additional discussion and final decision by the full group of participating reviewers. Volunteers are welcome year-round to assist with reviews.

ASBMB also offers:

- **ASBMB exam:** An assessment of core competencies in biochemistry and molecular biology at both foundational and advanced levels. Volunteer scorers are accepted year-round.
- **Undergraduate student chapters:** A national community of undergraduate students and faculty members that promotes research, education and outreach.
- **ASBMB's Education and Professional Development Committee:** A group of volunteer leaders who develop and oversee programs that promote effective molecular life sciences curricula and educational practices and provide career resources. Applications are accepted annually during the call for applications to committee appointments.

To get involved and support the professional education programming at ASBMB, scan the code.

Allison Frick is ASBMB's lead social media and multimedia manager.

AI can be an asset, ASBMB educators say

Since ChatGPT's initial release in 2022, educators have been navigating the growing use of artificial intelligence in classrooms, including embracing its strengths while navigating challenges. During a 2024 webinar entitled, "Breaking the mold: Exploring AI tools and alternative assessments in BMB education," educators shared how they use AI to save time, increase accessibility and prepare students for a changing world. This offering is part of a larger collection of teaching and education resources provided by ASBMB.

"Generative AI has great potential as an interactive tutor."

— Ning Sui, assistant professor of molecular and structural biochemistry North Carolina State University

Sui shared how AI can interactively explain concepts to students while maintaining a positive tone and being "much more patient than a tutor, and probably (herself)."

"AI can increase accessibility for neurodivergent students."

— Christin Monroe, assistant professor of chemistry, Landmark College

Monroe works with neurodivergent students, many of whom have executive function challenges. When they struggled to choose their final assignment topic, Monroe suggested some AI prompts to help guide them.

"AI can help students' conceptual understanding."

— Didem Vardar-Ulu, senior lecturer of chemistry, Boston University

Vardar-Ulu gives her students assignments that reflect on their "conversations" with AI. She said that this encouraged them to think deeply about the inputs needed to prompt a specific and reliable response.

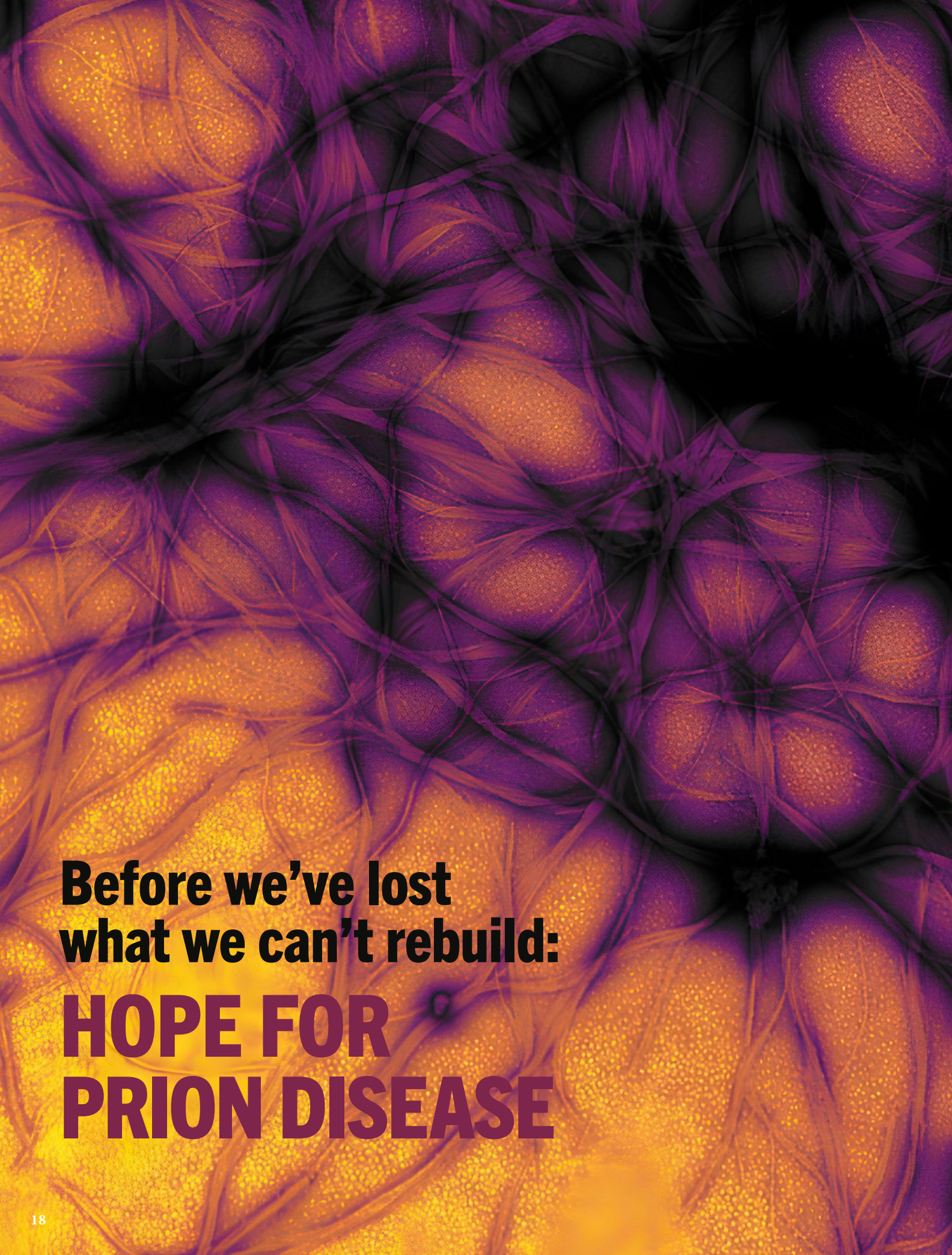
"We need to inform students about the capabilities, limitations and professional standards of generative AI."

— Emily Ruff, associate professor of biochemistry, Winona State University

Ruff shared that many of her students enjoy learning about what AI can and can't do, and how professionals take advantage of it at work. She encourages educators to teach students how to use AI, as dismissing this "powerful tool" may hinder students' professional development.

MATT HERP

Chinenyenwa Nora Okolie, recent graduate of Eastern Illinois University, participates in the Undergraduate Poster Competition at ASBMB's 2025 annual meeting in Chicago.

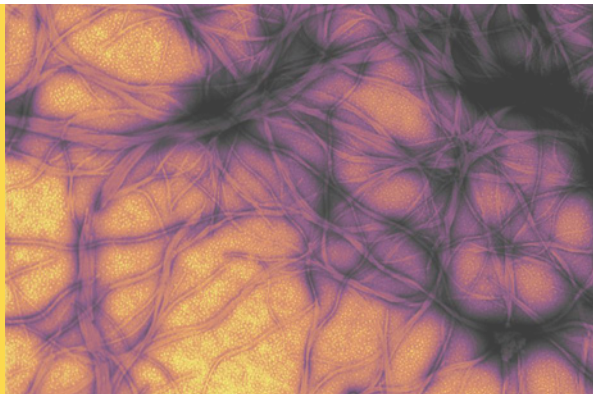


Before we've lost what we can't rebuild:

HOPE FOR PRION DISEASE

"If you bring a loved one to a physician, will they give you prescriptions? Yes, but none of them are aimed at slowing (prion) disease."

ERIC MINIKEL



By Elizabeth Stivison

Eric Minikel and Sonia Vallabh, a husband-and-wife team, describe themselves as "on a lifelong quest to prevent prion disease." They run a lab at the Broad Institute, studying this rare, aggressive and fatal neurodegenerative disease that affects 300 people in the U.S. annually. All together, they do research, raise funds, collaborate with pharma companies, educate patients, physicians and genetic counselors and maintain a patient registry.

Their motivation comes from their own experiences. Vallabh carries a genetic predisposition to prion disease and could develop symptoms at any time, and currently, there is no preventative, treatment or cure. To raise awareness and advance research, they founded the Prion Alliance.

Over the last decade, Vallabh and Minikel collaborated with the biotech company Ionis Pharmaceuticals to develop a potential prion disease treatment and preventative. In December 2024, Ionis announced that an early clinical trial of ION717, the product of their collaboration, was fully enrolled.

When proteins turn against the brain

Prion disease is a family of conditions including Creutzfeldt–Jakob disease, sometimes referred to as "mad cow" disease, as well as fatal familial insomnia and Gerstmann–Sträussler–Scheinker disease.

These diseases are united by the misfolding of prion protein, or PrP, in the brain. Normally folded, this neuronal cell surface glycoprotein may play a role in cell adhesion, signaling, stress protection and more. Once PrP misfolds, it induces surrounding PrP misfolding, triggering a cascade of misfolded protein that spreads and causes widespread cell death.

However, the exact mechanism of this harm is unknown. PrP may twist and damage the membrane's structure as it misfolds or get internalized by the cell, triggering the unfolded protein response and disrupting synaptic transmission. Misfolded PrP also aggregates, forming fibrils which may also induce neurotoxicity.

Prion disease is a family of conditions including Creutzfeldt–Jakob disease, sometimes referred to as "mad cow" disease, as well as fatal familial insomnia and Gerstmann–Sträussler–Scheinker disease.

NIH / IMAGE POINT FR / BSIP

3D rendering of prion proteins.

> “If you’ve seen someone you love go through this, you know how much is lost every single day.”
>
> SONIA VALLABH

In each of the prion diseases, PrP misfolds slightly differently. However, all forms lead to severe dementia and ultimately death.

On average, prion diseases induce death within five months of the first symptom. Due to the rarity of the disease and symptom overlap with other neurodegenerative diseases, the diagnostic odyssey can be so long that a patient may be diagnosed with only weeks left to live or until after autopsy.

Vallabh’s mother died in 2011 after a mysterious and rapid decline, which was identified as prion disease only during her autopsy.

“If you’ve seen someone you love go through this, you know how much is lost every single day,” Vallabh said. “To me, this is the biggest issue in neurodegeneration; we are losing things we have no idea how to rebuild. It’s particularly extreme in prion disease. It moves so fast.”

The charge

Watching her mother’s illness and the subsequent discovery that Vallabh carries a gene predisposing her to prion disease, upended Vallabh’s and Minikel’s lives.

They left their careers in law and engineering, obtained Ph.D.s and reinvented themselves as prion disease researchers and advocates.

“We are coming at this with an unusual amount of specificity and focus,” Vallabh said. “We don’t have (a lot of) time. We have the mandate and the opportunity to be the people who lead the charge.”

Targeting the molecular cause

About 85% of prion disease cases occur sporadically and are “totally random bad luck,” Minikel said. “For decades people searched for some link, maybe exposure, geography, family.” But it truly seems stochastic, developing in the brain for unknown reasons, he said.

The other 15% of cases are genetic, like Vallabh’s, and are caused by genetic alterations, such as missense mutations or insertions, in the PrP gene, PRNP, that make it more likely to misfold. The disease trigger is unknown; however, some scientists speculate that symptoms may be set off by oxidative stress or inflammation.

In addition, prion disease can be contracted from an external source such as contaminated meat or surgical instruments. However, less than 1% of reported cases develop via this route.

While normal PrP may have a beneficial role in the brain, like helping maintain myelination, the disease seems to be caused by the accumulation of misfolded protein, not the loss of normal PrP. From a therapeutic perspective, Minikel said, this means lowering the levels of PrP protein in the brain could help patients.

“If you know what the disease is that you’re up against, the best thing you can do is go after the root molecular cause,” Minikel said. “People have tried to chase downstream things like neuroinflammation, but you’re chasing after a train that’s already left the station.”

Scientists have known for decades that animals lacking Prnp are fully protected from infectious prion disease. However, deleting a gene, which can be difficult in humans, isn’t the only way to eliminate the pathogenic protein.

Instead, Minikel and Vallabh’s ION717 aims to lower PrP levels by reducing its messenger RNA, or mRNA, and preventing protein translation.

Thwarting the prion protein

Vallabh and Minikel first connected with Ionis in 2014 while pursuing their Ph.D.s, beginning a collaboration to develop a treatment for prion disease.

Around that time, Ionis discovered that antisense oligonucleotides, or ASOs — short strands of DNA, RNA or analogs that bind to target genes to modulate expression — could potentially treat prion disease. They found that ASOs could reach the brain when delivered at high doses via cerebrospinal fluid, or CSF, injection every few months, leading the company to explore ASOs for a range of neurological conditions.

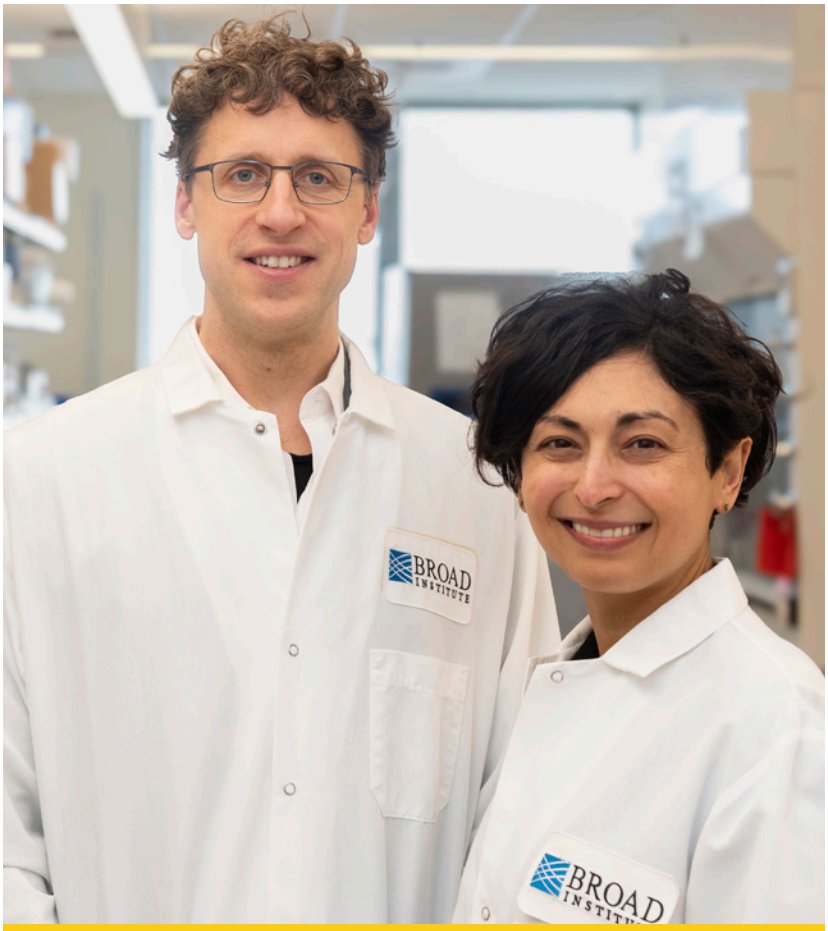
The collaboration brought together Ionis' clinical trial capabilities and the couple's expertise in prion biology, launching early discussions about what it would take to develop a PrP-lowering therapy.

"The neuroscientist (at Ionis) gave us a checklist: a humanized mouse model, biomarkers (to monitor patients), a patient registry," Vallabh said. "That became our to-do list for the next five years."

In collaboration with scientists at Ionis, the National Institutes of Health, or NIH, and others, Vallabh and Minikel published a series of papers evaluating PrP knockdown by ASO as a therapeutic strategy for prion disease.

To evaluate ASOs for this disease, Ionis scientists screened hundreds of DNA-like ASO candidates in cell culture to identify those that bind PRNP mRNA and trigger RNase H–mediated cleavage, thereby lowering PrP levels. They passed top hits to Vallabh and Minikel for disease studies.

In mice, ASO injections reduced both PrP mRNA and protein in the brain without obvious side effects. Most importantly, ASO-mediated PrP lowering extended survival in mice infected with mouse-adapted prions. Treating early and regularly tripled the lifespan of mice compared with untreated controls. Even when administered after symptom onset, ASOs provided some benefit.



COURTESY OF THE BROAD INSTITUTE

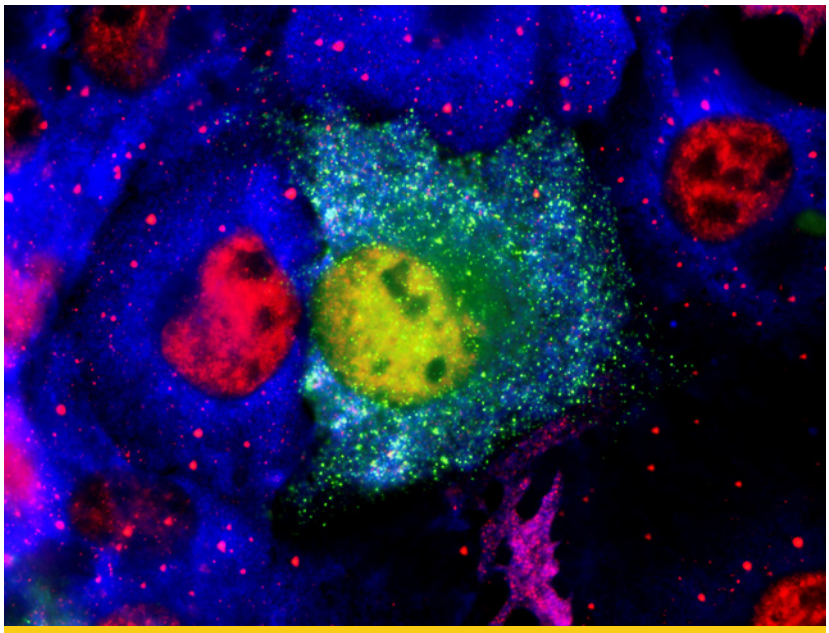
Eric Minikel and Sonia Vallabh pose for a photo in their lab at the Broad Institute.

These mouse experiments were the first time Vallabh and Minikel had seen live animals succumb to prion disease.

"Even though they're mice and we're people, it was still spooky how much the disease reminded us of (Vallabh's) mom's illness," Minkel said.

Vallabh said seeing the treated

The collaboration brought together Ionis' clinical trial capabilities and the couple's expertise in prion biology, launching early discussions about what it would take to develop a PrP-lowering therapy.



NANCY KEDERSHA VIA ALAMY

Confocal microscopy image of prion-like aggregating protein in a cell.

mice continue to live normally made her think, "I want to be those mice."

After Vallabh and Minikel's initial preclinical studies, Ionis continued optimizing the ASO strategy to target human PRNP, eventually selecting the ASO formulation ION717 to move into clinical trials.

Inside the clinical trial

Now that the ION717 trial is ongoing, no new patients can be recruited. Vallabh said she is happy it is underway, but "(i)t's agonizing to be so much further down the development arc but still be telling people as of right now, there's nothing we can do (to help them)."

During the trial, 56 prion disease patients will spend the first 30 weeks in a double-blind, placebo-controlled phase; afterward, all participants will receive the ASO for 70 weeks, followed by 32 weeks of monitoring. Although the primary goal is to assess safety rather than efficacy, scientists will track disease progression and biomarkers, such as PrP levels in cerebrospinal fluid.

"A lot of outcomes are possible from this trial." Vallabh said. "It would be positive to see any movement in the right direction. Markers (like PrP levels) coming down or rising at a slower rate even if (the changes) don't rise to clinical benefit, could still tell us we are going in the right direction."

If the treatment is safe and appears to slow the disease course, the next steps are easy: move on to the next trial phase. Otherwise, Vallabh and Minikel will go back to the drawing board.

"Lowering PrP could be the right idea, and yet the trial could still fail for any number of reasons," Minkel said. "Drug too toxic, not potent enough, dosed too high or too low or not frequently enough, not enough drug in the deepest brain regions, or patients already too advanced."

Vallabh said it is difficult to derive answers from neurodegeneration trials because they don't know when a patient reaches the point of no return, or a milestone after which treatment will not be effective.

"It breaks my heart that in neurodegeneration we're seeing so much money invested and still we can get to the end of a clinical trial and maybe not have learned one way or the other, because you can always argue that you treated too late," she said. "It's something the field more broadly needs to get its head around."

Because treatments work best when begun early, Vallabh and Minikel hope raising prion disease awareness will lead to earlier diagnoses. An accurate test exists — it measures a patient's cerebrospinal fluid for prion-induced misfolding — but because prion disease is so rare, many doctors don't consider it.

"Just in the last decade there's been an explosion of ways to target DNA and RNA," Minikel said. The couple is working on other ways to knock down PrP levels, including base editing, splice site alteration and epigenetic approaches.

"Whatever happens to this exact drug in this exact trial," Minkel said, "some kind of first-in-human test like this of a PrP-lowering drug was always going to be the first step towards eventually developing a meaningful therapy. In that sense, whatever the outcome, it will be a step forward."

Vallabh added: "Having something in (clinical trials) is not the finish line, it's the starting line."

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

Using 'nature's mistakes' as a window into Lafora disease

By Courtney Chandler

As a teen, Anissa Merriam was a talented artist and diligent student. When she started struggling with her studies and dropping basic items like her pencil, she told her parents something was wrong. Eventually, at age 18, she was diagnosed with Lafora disease, or LD, a rare neurodegenerative childhood dementia and epilepsy.

Now, at age 26, Anissa doesn't draw anymore and receives full-time care from her family and nurses.

"Cognitively, she is like a little girl," Anissa's mother Jenifer Merriam said in a Washington Post article. "This is a disease where we watch them go backward."

LD is a genetic neurodegenerative disease that typically manifests in adolescence. It occurs when the body cannot properly process the energy storage molecule glycogen due to mutations in the phosphatase EPM2A or E3 ubiquitin ligase EPM2B/NHLRC1. Patients with LD suffer from seizures, cognitive decline and loss of muscle coordination that worsen as the disease progresses. These symptoms are due to the buildup of insoluble glycogen aggregates that form in the brain, skin, heart, liver and other peripheral tissues.

LD is part of a larger family of diseases called glycogen storage diseases, or GSDs, which collectively affect 1 in 20,000 to 43,000 newborns annually.

For LD patients, including Anissa, there is no effective treatment or cure — care focuses around managing symptoms and providing support. From the time of their first seizure, most patients diagnosed with LD typically die within 10 years, or by age 30.

Matt Gentry, professor and chair of biochemistry and molecular biology at the University of Florida, has been researching LD for over 20 years. He knows first-hand how impactful rare disease research can be to the patients and families affected by these diseases — he has been collaborating with the patient community since his time as a postdoctoral fellow in the

CHELSEA'S HOPE

Anissa Merriam, who suffers from Lafora disease, enjoys a bike ride outdoors on a utility trike, which offers extra stability for those with motility challenges.

BONG RIOLA, CHELSEA'S HOPE

Matt Gentry, a professor and chair of biochemistry and molecular biology at the University of Florida, chairs a session on "Treating Lafora Disease" during the 2024 Lafora Disease Science Symposium in San Diego.

BONG RIOLA, CHELSEA'S HOPE

Kit Donohue, scientific and executive director at Chelsea's Hope, speaks to the audience during the 2024 Lafora Disease Science Symposium in San Diego.

lab of former American Society for Biochemistry and Molecular Biology President Jack Dixon at the University of California, San Diego.

"The LD community got me hooked on studying the glycogen storage diseases and Lafora disease in particular," Gentry said. The community now hosts annual workshops and supports both patient advocacy and research.

Gentry's work has been bolstered by this community throughout his career. He has maintained close connections with the LD advocacy group Chelsea's Hope. The group — which was founded in honor of Chelsea Gerber, who was diagnosed with LD at 15 and succumbed to the disease at age 26 — helps connect families and promotes LD research. Kit Donohue, a former graduate student in Gentry's lab and now scientific and executive director for Chelsea's Hope, said patients and their families are important partners in rare disease research.

"If you empower the patient community and (work hand in hand with them), they can really be huge supporters of the science, and they see the direct benefit to their community," Donohue said.

By collaborating with the rare disease community, Gentry has contributed to the biochemical understanding of LD and developed three potential therapeutics for LD-related conditions. Yet, as with all rare diseases, many hurdles and unknowns remain, especially for patients like Anissa and their families hoping for a cure.

Common themes, shared treatments

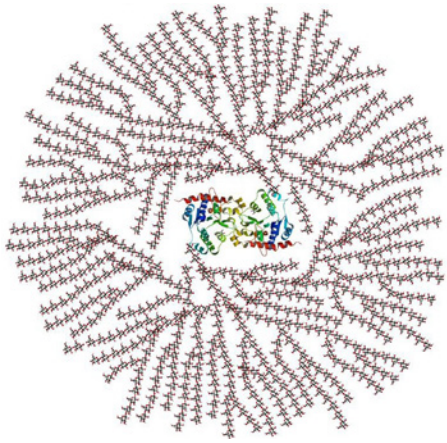
Since starting his lab in 2014, Gentry's research has expanded from LD to also include other GSDs, leading to a better picture of the disease biochemistry.

"We rolled out this idea that there are about six different diseases that we could (call) neurological GSDs," Gentry said. "For five of the six, the centerpiece is the idea that glycogen needs to be downregulated or degraded in order to treat patients."

These include LD, adult polyglucosan body disease, Cori's disease (also known as glycogen storage disease type III), Pompe disease and RBCK1-deficiency.

Since the grouping of these diseases, Gentry said GSD researchers are focused on finding therapeutics that prevent or degrade the disease-causing glycogen aggregates, sometimes known as Lafora bodies. Because five of these diseases share a common mechanism, a single drug could be effective. In addition to having more patient impact, this approach helps address some of the challenges associated with developing rare disease therapeutics, including cost.

Gentry said rare diseases often have complex biology and limited patient populations, making it difficult to identify and test new



MIKAEL HÄGGSTRÖM VIA WIKIMEDIA COMMONS

Shown is a crystal structure ribbon diagram of glycogen, an extensively branched glucose polymer with the protein glycogenin at its core. During Lafora disease, glycogen forms insoluble aggregates known as Lafora bodies.

therapeutics. According to the U.S. Government Accountability Office, almost one in 10 people in the U.S. have a rare disease but only about 5% of rare diseases have treatments approved by the U.S. Food & Drug Administration.

"LD is so ultrarare that no company wants to take a treatment forward," Gentry said. "This idea that one drug could now be used for five different diseases is a game changer in terms of the finances that companies are willing to look at."

So far, Gentry and others have developed multiple therapeutics to treat neurological GSDs that are currently in preclinical and clinical development. Gentry and colleagues are targeting GSDs from three different biochemical angles: degrading existing glycogen aggregates using an antibody-enzyme fusion, downregulating glycogen synthesis using an antisense oligonucleotide, and inhibiting the glycogen synthase enzyme using a small molecule drug.

Two of these therapeutics have already undergone successful early clinical trials. The antisense oligonucleotide, called ION283, targets the messenger RNA of a key enzyme in the glycogen synthesis pathway, glycogen synthase 1. Studies with this therapeutic conducted by Donohue during her time in Gentry's lab showed promise in an animal model and a safety study with LD patients is underway.

Gentry has also collaborated with Valerion Therapeutics to develop an antibody-based system capable of delivering therapeutics directly into cells. This system takes advantage of an anti-DNA antibody, called 3E10, that is taken into cells through a nucleoside transporter. By fusing an enzyme capable of degrading glycogen to this antibody, this therapeutic can effectively reduce glycogen accumulation. One version of this therapy has been investigated in early clinical trials for Pompe disease. Gentry's lab studies a similar antibody-enzyme fusion and, in a mouse model of LD, observed a significant reduction of Lafora bodies in the brain and other tissues.

Lafora disease is a rare, fatal and progressive brain disorder that causes epilepsy, childhood dementia and intellectual decline.

Donohue said the collaboration between the patients and scientists has been a key driver of this therapeutic development. In fact, the patient community is funding the safety study for the antisense oligonucleotide.

"You need patients, scientists and clinicians collaborating on understanding what's going to be not only the best from a basic science perspective, but what will be the best therapy for the patient," Donohue said.

From rare to common

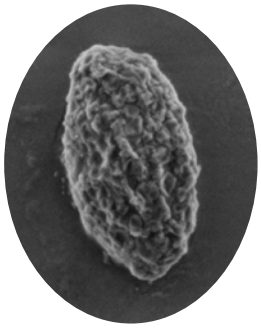
Beyond therapeutics, Gentry said that studying rare diseases provides insight into more common diseases.

"Rare diseases are nature's mistakes so they can give us insight into how

biology works," he said. "The work we did on LD directly drove our understanding of a new mechanism in lung cancer and that's not something I would have necessarily expected."

In a recent publication, Gentry and colleagues observed that glycogen metabolism is a key driver of lung adenocarcinoma, the most common lung cancer in the U.S. The team used spatial analysis of human lung adenocarcinoma samples and observed that glycogen accumulation was associated with more aggressive tumors and poorer survival. Using genetic models and dietary intervention, Gentry and colleagues also observed that increased glycogen levels were correlated with tumor progression.

A multiplexed spatial imaging technique developed by Ramon Sun at the University of Florida, based on mass spectrometry imaging further demonstrated the link between glycogen levels and metabolites essential for tumor growth, supporting the idea that glycogen accumulation drives cancer progression in lung adenocarcinoma. This biochemical understanding and link between glycogen accumulation and cancer progression could open the door for new treatments.



M. KATHRYN BREWER, UNIVERSITY OF FLORIDA

Scanning electron micrograph of a Lafora body, an insoluble glygogen aggregate, that can form in the brain, heart, skin and other peripheral tissues of patients with Lafora disease.

Although his work has expanded beyond LD, Gentry said he still credits the success of rare disease research and the insights provided into more common diseases, to a key group — the patients and their families.

CHELSEA'S HOPE

Emine Malaj, who suffers from Lafora disease, poses for a photo outdoors.

Patients as partners

Gentry maintains close contact with patients suffering from the diseases he researches — he is a scientific advisor for advocacy groups for adult polyglucan body disease and GLUT1 deficiency syndrome and Chelsea's Hope. Both Gentry and Donohue have seen the active role patients and their families can play in advancing rare disease research.

"Through an advocacy organization, (the patients) become part of the conversation," Donohue said. Researchers also benefit from these connections. "It gives them a better concrete vision of what their research is going towards and potential applications for their research that maybe they hadn't considered before," Donohue said.

Fatos Malaj, whose daughter Emine Malaj was diagnosed with LD after she started suffering from seizures at age 10, reached out to Chelsea's Hope after receiving Emine's diagnosis. He said Chelsea's Hope has become their "big family" that offers unlimited support, and he has even participated in the annual research symposiums.

"My participation in the (2023 Lafora Science) Symposium was a turning point for me," Fatos said. "There I met family members with the same problems and doctors that I had never imagined meeting, even in my wildest dreams. My best experiences with the researchers were participating in both symposiums."

Emine and Anissa's families have helped advocate and fundraise for the safety trials for the LD therapeutics in development. Without any current treatment options, the LD therapeutics in development serve as a beacon of hope for these patients and their families.

When Merriam learned about the ongoing LD research, "I just sat and cried," she said in a Washington Post article. "I was given a death sentence for my daughter 10 years ago and sent home. They gave me hope that I will not have to bury my daughter."

Although there is still plenty of work ahead, the patients, their families and the researchers can rely on the tight-knit collaborative community they have built to continue to push towards new insights and treatments.

"I think the rare disease community offers a unique avenue to help innovate in the world of science," Donohue said. "We know the current development pipeline doesn't work for us. But, if we can fix the system for us, it will benefit the entire community because you will have all of the nuts and bolts lined up to create treatments for everyone."

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

DEFEATING DELETIONS AND DUPLICATIONS

Promising therapeutics for rare chromosome 15 neurodevelopmental disorders

By Lydia Smith

Angelman, Prader–Willi, and Dup15q syndromes are all distinct neurodevelopmental disorders caused by changes within the specific q11 to q13 region of chromosome 15, known as 15q.

Researchers are making significant strides in understanding the molecular mechanisms behind 15q disorders and developing targeted therapies.

A small alteration on chromosome 15 can drastically affect development, influencing communication, motor skills, sleep and more. For families of children like Coral, who suffers from Dup15q syndrome, daily life involves navigating challenges such as seizures, communication barriers and the need for constant care.

Yet, amidst these challenges, researchers are making significant strides in understanding the molecular mechanisms behind 15q disorders and developing targeted therapies. Innovative approaches, including antisense oligonucleotide treatments, could one day be used to treat underlying genetic causes of conditions like Dup15q syndrome, offering the potential to transform patient care and improve quality of life for affected individuals and their families.

15q neurodevelopmental disorders comprise three distinct conditions: Angelman syndrome, Dup15q syndrome and Prader–Willi syndrome. Each is unique, yet all are caused by a change or mutation in the 15th chromosome. No cures have been identified for 15q disorders and treatment typically focuses on managing symptoms.

The National Library of Medicine estimates that Angelman syndrome affects one in every 20,000 people and Prader–Willi syndrome affects one in every 10,000 to 30,000. Dup15q has an unknown frequency, and it is estimated that it could be as high as one in every 5,000 people.

COURTESY OF THE BLOG, OUR SUPER DUPER CORAL

Coral, who suffers Dup15q syndrome, from plays with a xylophone outdoors in 2023. Coral and her family participate in research studies with the Dup15q Alliance, a patient advocacy organization for patients with at least one extra chromosome 15 region, 15q11.2 – 13.1.

Prader–Willi syndrome

According to the Mayo Clinic, Prader–Willi syndrome, or PWS, is caused by a genetic mutation of undefined mechanisms. Current research and the National Institute of Child Health and Human Development suggest that this mutation causes an inability to express paternal genes. The critical region for PWS is on chromosome 15(q11–13). The disorder occurs when the paternal genes are not expressed due to a deletion, uniparental disomy (inheriting only maternal copies) or a defect in the imprinting center, a region that regulates parent-specific gene expression.

Typically, symptom presentation begins around two years of age, including feelings of persistent insatiable hunger, or hyperphagia, poor responsiveness, underdevelopment and decreased muscle tone, or hypotonia. Hyperphagia often leads to obesity, which means that individuals with the syndrome also maintain a higher risk of experiencing obesity-related complications such as Type 2 diabetes, high blood pressure, elevated cholesterol and heart disease. Additionally, decreased hormone pro-

duction due to underdevelopment may lead to complications including sterility and osteoporosis.

Several therapeutic avenues have recently emerged, reporting promising results for PWS patients:

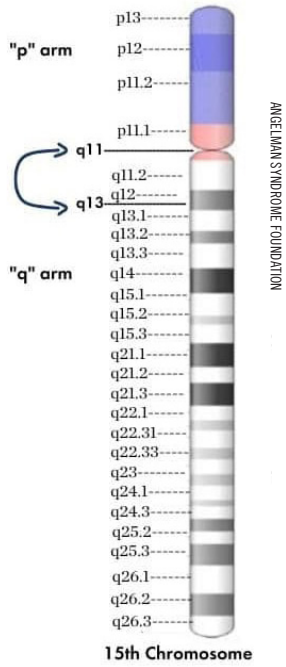
The drug, Vykat XR, produced by Soleno Therapeutics was recently approved by the U.S. Federal Drug Administration for PWS patients. It treats hyperphagia in patients aged four and older by activating potassium channels.

Harmony Biosciences recently received an Orphan Drug designation for Pitolisant, a therapeutic designed to treat excessive daytime sleepiness and behavioral disturbances. It works by increasing the histamine activity in the brain. This designation provides incentives for further testing, such as clinical trials testing the safety and efficacy of the drug in PWS patients.

Researchers at Duke University recently developed an epigenetic therapy using small molecule inhibitors to prevent H3K9 methylation in PWS patients, which enables expression of silenced maternal chromosomes to compensate for paternal deletion.

Angelman syndrome

Angelman syndrome, or AS, is often caused by abnormalities related to the ubiquitin ligase UBE3A gene on chromosome 15q. According to the Angelman Syndrome Foundation, this condition commonly occurs due to activation of only the maternal copy of the gene, with either a missing or defective paternal gene. However, AS can also be caused by the inheritance of two paternal genes. Symptoms of AS, including difficulty walking, lack of speech and seizures, typically



Since Angelman, Prader-Willi and Dup15q syndromes are caused by changes in the 15th chromosome, there are symptoms that all three syndromes share.

first appear around six to 12 months of age.

A few avenues have recently emerged that may lead to AS treatments:

In 2024, Ionis Pharmaceuticals reported positive results for early clinical trials of a drug designed to unsilence the paternal UBE3A allele; thus, compensating for loss of function in the maternal copy of the gene in AS patients. After treatment, 65% of patients exhibited both cognitive improvements as well as enhanced fine motor skills.

The Foundation for Angelman Syndrome launched two biotech companies, MavriX Bio and CourageAS Bio, to advance therapeutic development for AS. MavriX focuses on developing the first Adenovirus gene replacement therapy for AS, while CourageAS aims to develop a nonviral gene-editing therapeutic for patients.

Dup15q syndrome

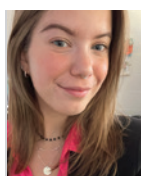
The National Institutes of Health describes Dup15q syndrome, also referred to as maternal 15q duplication syndrome, as a condition caused by the presence of at least one extra chromosome 15 region (15q11.2-q13.1). Dup15q typically only occurs when the duplicate copy is maternally inherited.

Common characteristics of Dup15q syndrome include hypotonia, intellectual disabilities, autism spectrum disorder and epilepsy. Currently, treatment options for the condition are limited to the treatment of specific symptoms and surveillance, as well as genetic and prenatal testing to monitor development throughout pregnancy.

The 15q chromosome contains several regions known as segmental duplications, which have a higher susceptibility to rearrangement and thus mutation. Several genes of interest that may contribute to disease progression have been observed in this region, including the ubiquitin ligases UBE3A and HERC2 as well as neurotransmitter receptor components GABRB3, GABRA5 and GABRG3.

In 2023, Children's Hospital Los Angeles launched Quindecim, a clinical trial focused on evaluating the safety and efficacy of Basmisanil, a drug that modulates neurotransmitter receptor activity, which can be overactive in Dup15q syndrome.

Lydia Smith is a Ph.D. student at the University of Utah and an ASBMB Today volunteer contributor.



Hope for a cure hangs on research

By Elisabeth Marnik

"Wait for science to catch up," a physician told Hailey Adkisson after failing to diagnose or effectively treat Adkisson's daughter Juniper, who suffers from undiagnosed epilepsy. Statements like these are common in the rare disease world that Adkisson's family navigates.

Adkisson's reality has become a nightmare amidst the cuts and dismantling of scientific research happening in the U.S. For example, the president's proposed budget for fiscal year 2026 slashes National Insitutes of Health and National Science Foundation funding by 40% and 55%, respectively.

"If rare disease research slows down or stops," Adkisson said, "people — including children (my family) know(s) directly — will die. It's not a question of if; it's when. This includes Juniper. Her seizures are getting more severe as she gets older. Seizures lead to more seizures. We need treatment options before it is too late. We need people to care."

In the U.S., a disease is considered rare if it affects fewer than 200,000 people, but many impact

COURTESY OF HAILEY ADKISSON

Hailey Adkisson and daughter Juniper, age 4, pose with the book Adkisson wrote to explain epilepsy to children. Find her on Instagram: @growing_juniper.

far fewer. With over 10,000 rare diseases, there are nearly 30 million Americans, mostly children, impacted. Most of these conditions are life-threatening, genetic and lack approved treatment.

For families like Adkisson's, research is their only hope. Adkisson became part of the rare disease community after her daughter, Juniper, began having seizures at six months old. Until then, Adkisson said Juniper seemed healthy. But once the seizures began, they never stopped.

Juniper's doctors have tried more than a dozen antiseizure medications, and she has undergone two brain surgeries. One was a hemispherectomy to remove one side of Juniper's brain in the hopes of stopping her seizures. It didn't work. She now lives with a device implanted in her chest to help regulate brain activity.

In search of answers, Juniper and her family underwent genetic testing multiple times, which yielded no known deleterious mutations. Eventually, they reached out to the Undiagnosed Diseases Network, or UDN, a federal research initiative that studies cases like Juniper's. They are currently conducting studies using some of her brain tissue. According to a UDN's 2024 report, they have helped diagnose 750 patients and defined 75 new conditions.

However, Juniper remains undiagnosed, making it difficult to find treatments or assess whether relatives are at risk of a similar disorder. Continued research through the UDN is their only path forward, but the federally funded program could be affected by proposed budget cuts.

Those who have rare and complex health conditions often feel alone, especially now. Adkisson has taken her family's experience and launched an organization, Simply Complex Stories. They publish illustrated books to teach children about complex medical conditions, like epilepsy.

COURTESY OF LAUREN HUGHES

Lauren Hughes is a mother to a rare disease patient, a pediatrician and a social media educator. Find her on Instagram: @bloomdpc.

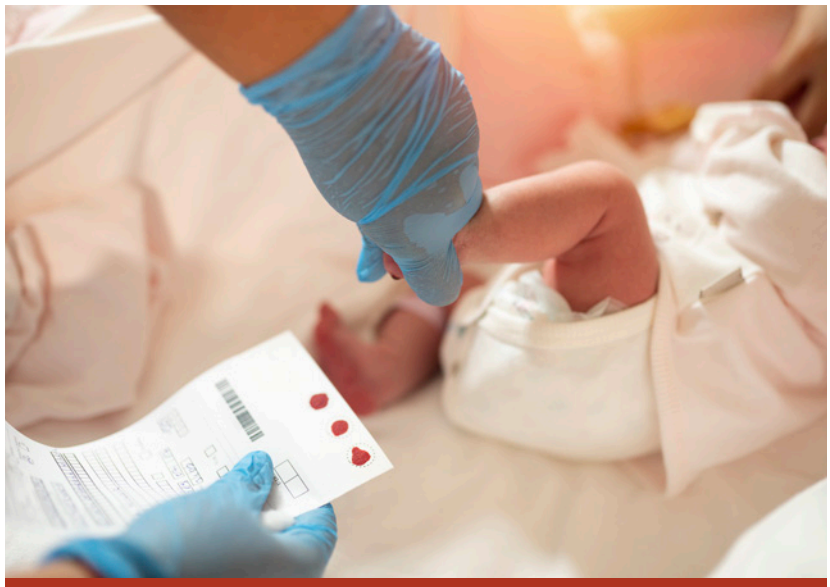
Life-saving power of newborn screening

Lauren Hughes, a pediatrician and mother to a rare disease patient, said research saved her son's life.

He lives with medium-chain acyl-CoA dehydrogenase deficiency, or MCAD, a rare metabolic disorder that prevents the body from breaking down medium-chain fatty acids. MCAD affects about 1 in 20,000 children in the U.S.

Doctors identified MCAD in Hughes' son via newborn screening, an optional medical initiative that tests newborns for about 38 genetic conditions, enabling early detection and treatment. This program would not exist without federally funded science that helped identify the mutations responsible for these

> **"Without newborn screening and the advisory committee that expanded the testing to include my son's condition, I wouldn't have a living and healthy five-year-old boy."**
>
> LAUREN HUGHES



Newborn genetic screening is a series of laboratory tests that screen for genetic diseases. The test is usually performed within 48 hours of birth by taking a small blood sample from the baby's heel and sending it to a lab.

Unlike many rare diseases, MCAD can be managed. However, early diagnosis is key to patient survival.

conditions.

The Trump administration recently terminated the advisory committee responsible for recommending updates to the newborn screening program, meaning rare genetic diseases discovered in the future could go undetected. Hughes said she finds this action greatly concerning.

"If (my son) had been born even 25 years in the past, he wouldn't have survived," Hughes said. "Without newborn screening and the advisory committee that expanded the testing to include my son's condition, I wouldn't have a living and healthy five-year-old boy."

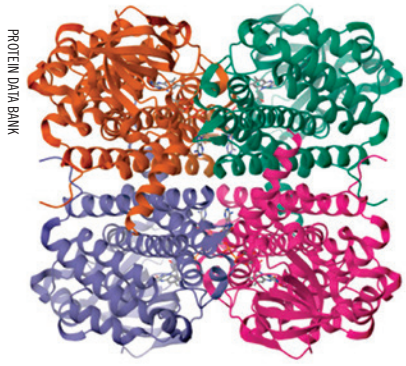
Unlike many rare diseases, MCAD can be managed. However, early diagnosis is key to patient survival. Those with MCAD cannot go without food for long periods; if they do, they develop dangerously low blood sugar levels, causing them to become comatose and potentially die.

According to Hughes, receiving her son's diagnosis early allowed her and her family to adapt and keep him alive.

"When he was a newborn, I had to wake and nurse him every two hours, around the clock," she said. "(I)t was scary, stressful and exhausting, but it enabled him to survive."

Hughes said she still lives with daily fear because each overnight requires careful planning, and even the common cold could become a health crisis for her son.

"Even if a rare disease isn't always



PROTEIN DATA BANK

Shown is a crystal structure ribbon diagram medium-chain acyl-CoA dehydrogenase. Lauren Hughes' son suffers from a rare disease caused by a deficiency in this enzyme.

COURTESY OF HAILEY ADKISSON

Juniper Adkisson, age 4, poses for a photo on Rare Disease Day wearing her "rare" shirt. Juniper is one of the millions of people with an undiagnosed rare disease in the U.S.

fatal, it can still be life altering," Hughes said. "These kids matter. These families matter. Their lives can be improved by continued research."

Rare disease research benefits all

The Trump administration's proposed cuts to biomedical and public health research might seem like distant policy decisions to some and a professional crisis to scientists — but for families affected by rare disease, it could mean the difference between life and death.

"Our daughter is in the rarest of rare categories," Adkisson said. "There is so little funding already for undiagnosed rare disease."

Rare disease research benefits all of us, even if we aren't part of the rare disease community. It has led to breakthroughs in our understanding of genetics, cell biology, cancer and more.

Despite this mutual benefit, many rare disease families feel invisible. Adkisson said conditions like epilepsy receive less funding than other neurological disorders. For example, in fiscal year 2023, the NIH budget totaled approximately $47.7 billion. Of this, $245 million funded epilepsy research, while Alzheimer's and dementia research received over $4 billion.

This is why parents like Adkisson and Hughes speak out on social media about the dismantling of scientific research happening in the U.S.

From both stories we can learn an important lesson: Science isn't just a job or new knowledge. It's a lifeline to patients who have little hope. When the government cuts rare disease research funding, it tells families across the country, like Adkisson and Hughes', that they don't matter. Science can't catch up to help these rare disease patients and families if we don't fund it.

> **"Our daughter is in the rarest of rare categories. There is so little funding already for undiagnosed rare disease."**
>
> HAILEY ADKISSON

Elisabeth Adkins Marnik is the Director of Science Education & Outreach at the MDI Biological Laboratory, the Chief Scientific Officer of Those Nerdy Girls and an ASBMB Today volunteer contributor.

The 2025 Annual Meeting of the American Society for Biochemistry and Molecular Biology was held at the McCormick Place Convention Center in Chicago. Nearly 2800 scientists — established scientists, trainees, educators, and industry leaders — attended the four-day conference, organized by co-chairs Donita Brady of the University of Pennsylvania Perelman School of Medicine and Dave Pagliarini of Washington University School of Medicine in St. Louis.

ABOVE: Tran Dang, a graduate student at the University of Georgia, presents a poster on her methyltransferase research.

LEFT: Benjamin Garcia, winner of the ASBMB Ruth Kirschstein Diversity in Science Award, gives an award lecture on his academic journey.

BELOW: The exhibit hall inside McCormick Place Convention Center was filled with posters, exhibitors and activities.

BELOW LEFT: Benjamin Sabari, an assistant professor at the University of Texas Southwestern Medical Center, asks the presenter a question at a scientific symposium.

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RIGHT: Ann West, chair of the ASBMB Public Affairs Advisory Committee, and Ann Stock, ASBMB past president, enjoy the interactive exhibits at the Griffin Museum of Science and Industry.

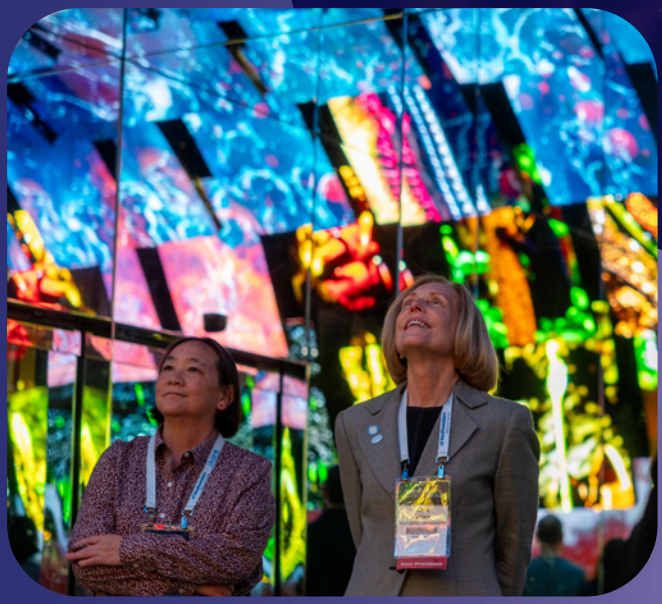
MIDDLE RIGHT: Jamare McMurtry, an undergraduate at the University of Wisconsin–Parkside, presents a poster on his research.

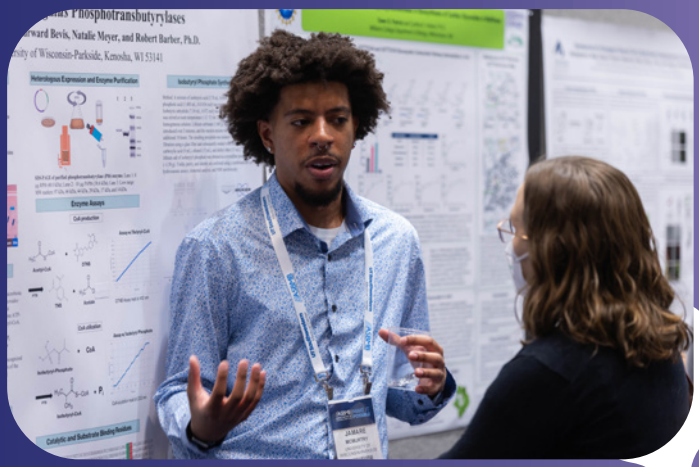
BOTTOM RIGHT: Tigist Tamir, an assistant professor of biochemistry and biophysics at the University of North Carolina at Chapel Hill, enjoys the interactive Science Storms exhibit at the Griffin Museum of Science and Industry.

BOTTOM LEFT: Alex Toker, editor-in-chief of the Journal of Biological Chemistry, addresses a crowd at the JBC Editorial Board Meeting.

BELOW: Mireille J. Aleman, an instructional associate professor at the University of Florida, speaks with meeting attendees at the Career and Education Fair.

Photos by Matt Herp





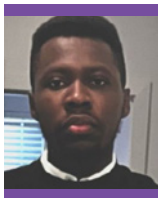
Bashir Ali, Omolara Falade and **Olalekan Usman** have been selected to participate in the Scientist Mentoring & Diversity Program for Biotechnology, or SMDP. The program pairs ethnically diverse students and early career researchers with industry mentors in the medical technology, biotechnology and consumer healthcare industries for one year.


ALI

Ali is a graduate student at the University of California, Santa Barbara. He performs research in assistant professor Brooke Gardner's laboratory on the function of substrate-binding domains of the enzyme peroxisomal biogenesis factor 1 and factor 6 in peroxisome biogenesis. In 2020, he won a National Science Foundation Graduate Research Fellowship.


FALADE

Falade is a graduate student at Texas A&M University pursuing her Doctor of Pharmacy degree. She is also an AbbVie EMERGE Program Scholar and completed an internship with the Federal Drug Administration in 2024.


USMAN

Usman is a graduate student at Florida State University. He is studying the mechanobiology of cancer progression with assistant professor Jerome Irianto at FSU. In 2023, Usman completed an internship with Eli Lilly and Company.

Lu "Lucy" Bai has been named the Verne M. Willaman Professor of Biochemistry and Molecular Biology at Penn State University. The appointment is awarded by the Penn State Office of the President and honors Bai's research contributions, teaching and service to the university. Willaman was a Penn State alumnus and businessman who presided over Ortho Pharmaceutical Corporation.


BAI

Bai's research focuses on understanding how gene expression is regulated by chromatin. Her lab is currently working to identify and characterize pioneer factors, specialized proteins that can bind to densely packed regions of chromatin and make them accessible to the cellular machinery required for gene expression. She has received many awards, including being named a Suzanne and Bob Wright Fellow of the Damon Runyon Cancer Research Foundation in 2008 and receiving a Women and Science Fellowship in 2007.

"I am deeply honored to receive this award and grateful for the confidence that the college and the department have placed in me and my research," Bai said. "This recognition is not solely mine. It belongs to my entire research group whose hard work and dedication make this possible. I am especially grateful to our graduate students, who are the driving force behind the lab, continually pushing the boundaries of our chromatin research."

Sandhya Visweswariah has been named the president-elect of the International Union of Biochemistry and Molecular Biology. IUBMB's mission is to promote research and education in biochemistry and molecular biology throughout the world. The organization gives particular attention to promoting opportunities for trainees in areas where biomolecular sciences are actively developing.


VISWESWARIAH

Visweswariah is an honorary professor in the department of developmental biology and genetics at the Indian Institute of Science. Her lab studies signal transduction mediated by cyclic nucleotides and their receptors, including receptor guanylyl cyclases, phosphodiesterases and novel nucleotide cyclases in bacteria. In addition to her research, she works to remove roadblocks for women in science in India. In 2019, she won the Indian Institute of Science's Rustom Choksi Award for Excellence in Science and Engineering. Visweswariah is a fellow of the World Academy of Sciences, the Indian National Science Academy

and the Indian Academy of Sciences. She has mentored more than 30 Ph.D. students and trained several postdoctoral fellows and research assistants in her laboratory.

IUBMB posted their congratulations on social media: "Congratulations to Professor Sandhya Visweswariah … for her election to president-elect of IUBMB at the 26th Ordinary General Assembly. Welcome to the leadership team!"

Paul Thompson has been named the Endowed Chair in Biochemistry and Molecular Biotechnology II by the University of Massachusetts Board of Trustees. Thompson is one of 12 joint endowed chairs at UMass Chan and UMass Memorial Health.


THOMPSON

Thompson is a professor of biochemistry and molecular biotechnology at UMass Chan. His lab develops chemoproteomic tools for biomarker discovery and chemical probes to target disease-modifying enzymes. Thompson is a fellow of the Royal Society of Chemistry and recently received a BRIDGE Innovation and Business Development award from UMass Chan to develop a PAD2 inhibitor for the treatment of inflammatory diseases. In addition, he has founded several biotech companies, including Danger Bio and Padlock Therapeutics, which was acquired by Bristol Myers Squibb.

"With the approval of these endowed chairs by the University of Massachusetts Board of Trustees, we are further strengthening our deep relationship with the University and the Medical School as we together forge a clear path in what has become a challenging health care environment," Eric Dickson, president and CEO of UMass Memorial Health, said. "This investment demonstrates our shared values and commitment to our faculty and to the future of our organizations."

Nikea Pittman, Chelsey Spriggs and **César de la Fuente** are among the scientists receiving 2025 awards from the Microbiology Society. They will be honored and give prize lectures at the MicroSoc's annual conference in Liverpool, England, this spring. Each recipient will receive an honorarium of 1,000 euros.

Pittman is a teaching assistant professor of biochemistry and biophysics at the University of North Carolina at Chapel Hill. Her lab collects evidence to identify the best practices in STEM higher education. Pittman has won many honors including the Diversity Award from the UNC Office of the Provost and a Burroughs Wellcome Fund Postdoctoral Diversity Enrichment Program award. She completed her Ph.D. at the University of Florida and a postdoctoral fellowship at UNC. Pittman won the MicroSoc's Equality, Diversity and Inclusion Prize collectively with other Black Microbiologists Association board members; she is the BMA's secretary.


PITTMAN

Spriggs is a research assistant professor in the Life Sciences Institute at the University of Michigan. Her lab explores the mechanisms of oncogenic virus entry. She was an inaugural scholar in the American Society for Biochemistry and Molecular Biology Maximizing Opportunities for Scientific and Academic Independent Careers, or MOSAIC, program. Spriggs is also a Burroughs Wellcome Fund Postdoctoral Enrichment Program fellow and a recipient of the U-M Office of Health Equity and Inclusion Faculty Diversity Fund Award. Spriggs earned her Ph.D. at Northwestern University studying how human papillomavirus infection leads to tumorigenesis and completed a postdoctoral fellowship at the U-M. She won the MicroSoc's Equality, Diversity and Inclusion Prize collectively with other Black Microbiologists Association board members. She is a BMA co-founder and works to ensure the needs of the membership are met, according to a press release.


SPRIGGS

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 informa-

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tion to identify new antimicrobial compounds, and he has pioneered the emerging field of artificial intelligence–driven antibiotic discovery. de la Fuente has won many awards including the Miklós Bodanszky Award, the Early Career Basic Research award from the American Society of Microbiology and the Princess of Girona Prize. He is also a member of the Royal Academy of Pharmacology, a National Academy of Medicine Emerging Leaders in Health and Medicine Scholar and an American Institute for Medical and Biological Engineering fellow. de la Fuente won the MicroSoc's Fleming Prize, which honors an early career researcher who has achieved an outstanding research record. This prize is named for Alexander Fleming, founder and the first president of the MicroSoc.


DE LA FUENTE

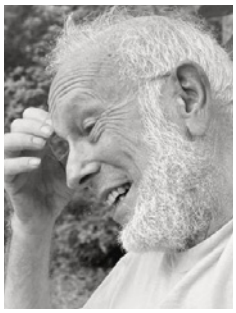
Squire Booker of Penn State University has been appointed the Richard Perry University Professor in the School of Arts and Sciences and a Penn Integrates Knowledge professor. Perry established this professorship in 2005 and is the president and CEO of Perry Capital, a private investment management firm that he cofounded. The Penn Integrates Knowledge professorships are awarded to faculty who hold appointments in two or more schools at Penn and draw on their breadth of knowledge to collaborate with colleagues.


BOOKER

Booker is a professor of chemistry, biochemistry and molecular biology and a Howard Hughes Medical Institute investigator. His lab studies biosynthetic enzymes that use S-adenosylmethionine and iron–sulfur clusters as radical catalysts.

A member of the American Society for Biochemistry and Molecular Biology for more than two decades, Booker has led the ASBMB Maximizing Access Committee and served on the Nominating Committee, Meetings Committee, Finance Committee and Program Planning Committee. He established the ASBMB Interactive Mentoring Activities for Grantsmanship Enhancement, or IMAGE, workshop and was a mentor for the Maximizing Opportunities for Scientific and Academic Independent Careers, or MOSAIC, program. In 2022, he received both the ASBMB Ruth Kirschstein Diversity in Science Award and the ASBMB–Merck Award. Booker has also received the Percy L. Julian Award from the National Organization for the Professional Advancement of Black Chemists and Chemical Engineers and the Hans Neurath Award from the Protein Society. He is a member of the National Academy of Sciences and the American Academy of Arts and Sciences.

Norman D. Meadow



Norman D. Meadow, a professor of biochemistry at Johns Hopkins University, died July 23, 2024, at the age of 87 in Maryland. He was a member of the American Society for Biochemistry and Molecular Biology for 34 years.

Born in May 1937, Meadow obtained his Ph.D. from the University of Pennsylvania before becoming a faculty member at Johns Hopkins University, where he served for three decades performing protein phosphorylation and bacterial signal transduction research.

Meadow's scientific contributions significantly advanced the field's understanding of the bacterial phosphotransferase system, PTS. His work with Saul Roseman led to the isolation and characterization of the glucose-specific bacterial phosphocarrier protein IIIGlc and its role in sugar transport. He also contributed to understanding glucose transport kinetics within the PTS framework and pioneered a technique to characterize kinase–substrate interactions.

He is survived by his wife, Karen; three children, Max, Paul and David; and six grandchildren. He was predeceased by his parents, Mary and Paul, and brother, Harold.

— Seema Nath

Donald A. Bryant

Donald A. Bryant, professor emeritus of biochemistry and molecular biology at Penn State University, died August 28, 2024, at the age of 74. He was a member of the American Society for Biochemistry and Molecular Biology for over 35 years.

Bryant was born on March 12, 1950, to Roger Bryant Jr. and Wanda Partin Bryant in Henry County, Kentucky, and spent his early life on a dairy farm. He graduated as valedictorian of Oldham County High School in 1968. He then attended the Massachusetts Institute of Technology, earning a bachelor's degree in chemistry and biology with honors in 1972. Bryant's passion for bioenergetics led him to UCLA, where he obtained his Ph.D. in molecular biology in 1977.

Following his doctoral studies, Bryant pursued postdoctoral research at the Institut Pasteur in Paris and later at Cornell University. In 1981, he joined the faculty at Penn State University, where he dedicated over four decades to advancing the field of microbial physiology. In 1992, he was appointed the Ernest C. Pollard Professor of Biotechnology, a position he held until his retirement in 2022.

Bryant's research focused on chlorophototrophic bacteria, particularly cyanobacteria and green sulfur bacteria. His work provided significant insights into photosynthesis, including the discovery of how cyanobacteria adapt to far-red light by remodeling their photosynthetic apparatus. This finding has potential applications in enhancing crop productivity by expanding the usable light spectrum for photosynthesis.

Beyond his scientific endeavors, Bryant was an avid birdwatcher and photographer. He traveled extensively, capturing high-quality images of raptors and other bird species. He actively participated in the Stone Mountain Hawk Watch and supported various environmental organizations, including the Cornell Lab of Ornithology and Hawk Mountain Sanctuary.

Bryant is survived by mother, Wanda; brother, Larry; sister-in-law, Catherine; and nephews, Seth and Jordan.

— Meg Taylor

IN MEMORIAM

Jeffrey C. Cameron

Jeffrey C. Cameron, an associate professor of biochemistry at the University of Colorado Boulder, died September 25 at the age of 43 in Colorado. He made seminal contributions to understanding microbe cellular processes.

Born on February 16, 1981, in La Grange, Illinois, Cameron studied plant science at Montana State University and Washington University, where he obtained his Ph.D. and completed his postdoctoral training.

In 2015, he opened his lab at CU Boulder, which focused on understanding the physiology, cell biology and biochemistry of the photosynthetic microbe, cyanobacteria. His research on cellular signaling and metabolic regulation in cyanobacteria aimed to harness their carbon-fixing mechanisms to combat climate change, leading to innovations like bacteria-grown biomaterials that reduce emissions from traditional cement production. In 2016, Cameron cofounded the biotech company Prometheus Materials, which uses microalgae to create zero-carbon bio-cement and bio-concrete.

Cameron was also a mentor and educator. Many of his students and colleagues remember him for his brilliance as well as his kindness. He was deeply committed to fostering a collaborative research environment and was known for his generosity in sharing knowledge.

Outside of the lab, Cameron enjoyed cooking, hiking and mushroom hunting.

He is survived by his wife, Jennifer Michelle Cameron, and four children, Noelle Rose, Jasper Carlyle, Evette Blue and Margot Love. He is also survived by his parents, three siblings and three grandparents.

— Seema Nath

Daniel N. Hebert

Daniel Hebert, a faculty member of the biochemistry and molecular biology department at the University of Massachusetts Amherst for 27 years died December 8, 2024. He was 62.

Hebert was born on May 6th, 1962, in Corning, New York, to Normand and Therese Hebert. He earned his Bachelor of Science in chemistry from the University of New Hampshire. Hebert then earned a Ph.D. at the University of Massachusetts Medical School working in the lab of Anthony Carruthers.

Hebert pursued postdoctoral work with Ari Helenius at the Yale School of Medicine, where he demonstrated the potential of working at the nexus of cell biology and biochemistry and began his career focus on folding and quality control in the endoplasmic reticulum, or ER. In 1997, he became a faculty member at the University of Massachusetts Amherst. During his time at UMass Amherst, he mentored and trained many future researchers in his laboratory and promoted an inclusive, supportive research environment, according to a UMass obituary.

Hebert's research focused on understanding the steps involved in cellular secretory protein maturation and degradation. He showed that carbohydrate modifications act as signals to facilitate proper folding and quality control of several proteins, specifically those secreted from the cells via the ER. His work has implications for diseases such as diabetes, lung disease, liver cirrhosis and neurodegenerative disorders.

Outside of the lab, Hebert loved learning, books and sports.

He is survived by his wife of 33 years, Leah (Kelley) Hebert; their son, Dylan; and daughter, Shannon. Hebert's family and colleagues established a graduate scholarship award in his memory.

— Jay Thakkar

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