ASBMB TODAY

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The science of **living longer, healthier lives**

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ASBMBTODAY

ABOUT THE COVER: The Timekeepers of Proteostasis by ASBMB member Megan Mitchem, University of North Carolina at Charlotte

Proteins shape the passage of time in this molecular hourglass. Just like snowflakes, each protein is highly unique. Each flake here is formed by radial projections of proteins that play a role in aging and neurodegeneration (or regeneration), including TDP-43, α-synuclein, Huntingtin, Hsp70, and Hsp90, reflecting the delicate balance between proteostasis and degeneration.

By Joan Conaway

120 Years of Discovery: ASBMB CONTINUES THE STORY OF SCIENCE

There is a room at the American Society for Biochemistry and Molecular Biology's headquarters lined from wall to wall with every print issue of the **Journal of Biological Chemistry** published since 1905. These volumes tell a remarkable story: enzymes once seen as mysterious "ferments" are now precision molecular machines, DNA has gone from an indecipherable code to something we can read and edit, and hand-drawn molecular sketches have become atomic-resolution structures. Laboratories, once illuminated by gaslight, now sit in state-of-the-art buildings with core facilities equipped with next-generation technologies. I marvel at how quickly we have advanced in a relatively short span of time when I see 120 years of discoveries arranged side by side on a shelf, each volume turning the page for the breakthroughs to follow.

That ever-evolving story continues today. ASBMB will be expanding its publishing portfolio with **Insights into Biochemistry and Molecular Biology**, a new open-access journal dedicated to emerging areas of research across the molecular life sciences. Like all ASBMB's journals, IBMB provides a trusted platform for rigorous, peer-reviewed science, but it also welcomes early-stage findings, new approaches and cross-disciplinary work that may point toward the next big leap in understanding.

Keeping this story going also requires science advocates. ASBMB works tirelessly to ensure that fundamental research is supported. As a member, you can contribute to this effort. You can apply for the Advocacy Training Program or the Art of Science Communication course to learn how to effectively share the importance of research. Stay up to date on policy developments and events through the advocacy Action Center, contribute to letter-writing campaigns, or use our advocacy toolkit to help amplify the voice of science. Your membership dues also directly support ASBMB's advocacy work — so if you haven't yet renewed for 2026, now is a great time to do so.

This issue of ASBMB Today underscores the essential role of fundamental research in advancing our understanding of the world and informing the development of future applications. In this issue you will find stories about NAD signaling and lifespan as well as genetic mutations that keep muscles young. You'll also read about Meng Wang's research on the connection between metabolism and aging, a topic she'll explore in her plenary talk at the 2026 ASBMB Annual Meeting during the "Racing the Clock: Molecular Mechanisms of Aging" deep dive session.

Lately, it has been a challenging time for science, but I am hopeful that the next generation of scientists will have the opportunity to continue contributing to discovery for both their own legacies and what they can offer to the world. I'm convinced that, despite the difficulties facing science today, we as scientists will continue to do what we do best — investigate, learn and discover through curiosity and perseverance. The story of science will keep moving forward — because discovery is what drives the next chapter.

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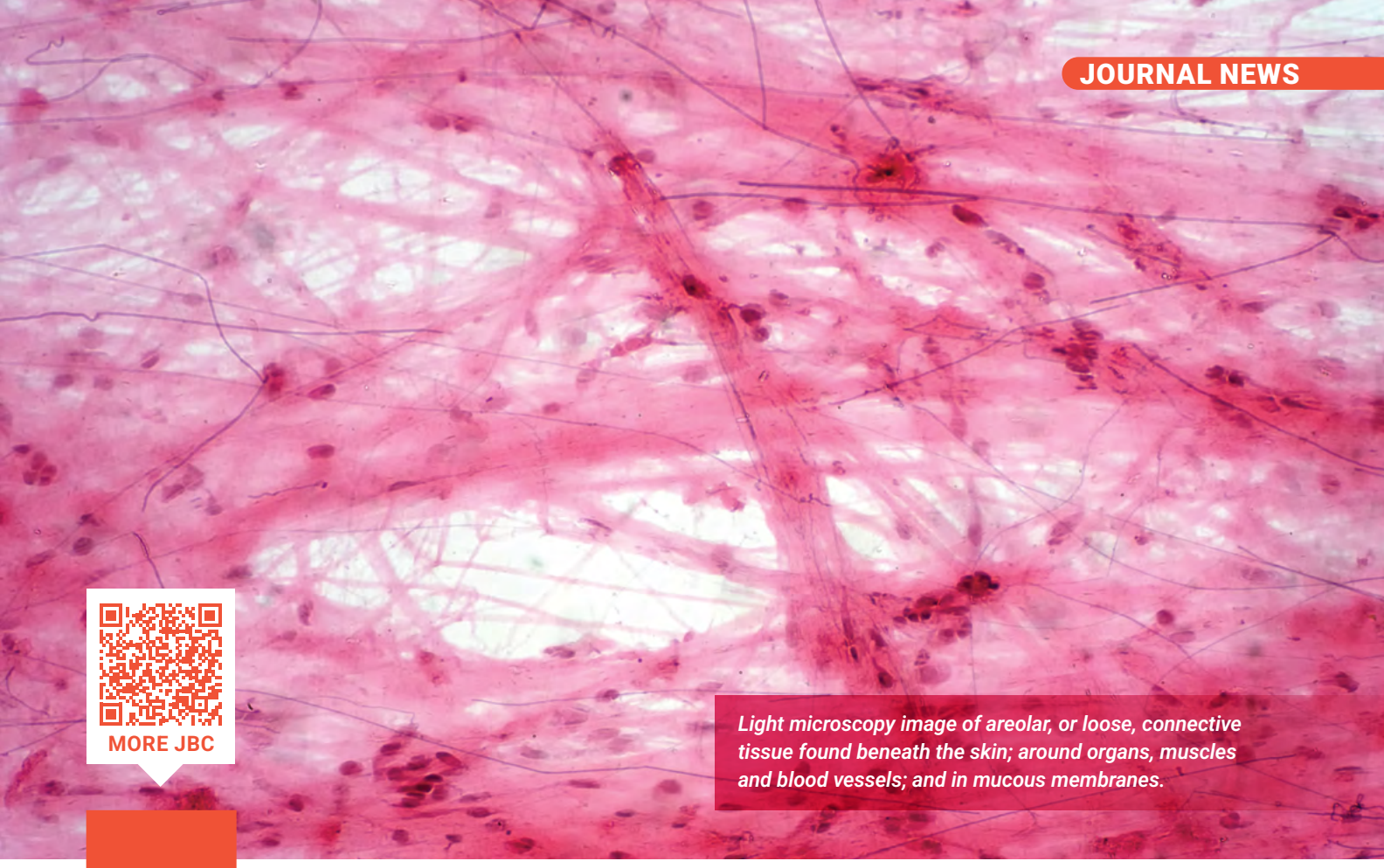


MORE JBC

Light microscopy image of areolar, or loose, connective tissue found beneath the skin; around organs, muscles and blood vessels; and in mucous membranes.

JBC

How sugars shape Marfan syndrome

By Elisabeth Marnik

Most people associate sugar glucose with sweet treats, but it also plays a critical role in protein function. In fact, new research from the Journal of Biological Chemistry suggests that variations in protein glucosylation may contribute to diseases like Marfan syndrome.

Marfan syndrome is an autosomal dominant disorder caused by mutations in the gene for fibrillin-1, a key extracellular matrix, or ECM, protein. These mutations weaken tissue integrity, leading to skeletal deformities, joint flexibility and cardiovascular issues such as aortic rupture, though the molecular mechanisms remain unclear.

A new study by Nicholas Kegley, a postdoctoral researcher, and Robert Haltiwanger, a professor at the University of Georgia, showed that Marfan syndrome–associated mutations in fibrillin-1 lead to unexpected changes in O-glucosylation, the addition of glucose to a specific amino acid, serine, in the protein.

"A lot of people think these sugar modifications are just decorations," Haltiwanger said. "But our lab is showing there is something more going on, they have relevant biological functions."

This research builds on earlier findings from the Haltiwanger lab showing that fibrillin-1 is heavily modified by two enzymes: POGLUT2 and POGLUT3. These enzymes attach single glucose molecules to a serine in short repeating motifs in fibrillin-1. They found that genetic deletion of both enzymes resulted in neonatal lethality and abnormal fibrillin-1 in the lungs. These findings highlight the enzymes' importance and spurred further investigation into their substrate specificity.

Initially, they thought that POGLUT2 and POGLUT3 required a strict amino acid motif to add glucose. However, they found that a less strict motif containing a serine is sufficient — significantly broadening the enzymes' potential target sequences.

To explore how Marfan mutations alter fibrillin-1 glycosylation, the team introduced known patient mutations into the protein and used mass spectrometry to analyze the sugar modifications.

"Typically, the wild-type version of the protein has the *O*-glucose monosaccharide, which is one sugar." Kegley said. "But some of the Marfan variants had extra sugars stacked up on top. This means you could have two or three sugars where you typically should have just one."

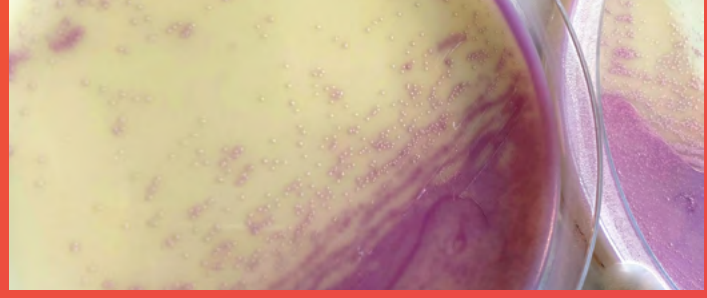
Haltiwanger added that while some mutations elongated the modifications, others reduced the glycosylation. Overall, this means these changes may influence how fibrillin-1 integrates into the ECM, increase its degradation by proteases, or binding to proteins in the ECM. One or all of these could be contributing to the symptoms patients experience.

Based on these findings, Haltiwanger estimates that nearly twice as many human proteins could be targets for POGLUT2 and POGLUT3 modification than previously thought.

Future work will explore how these sugar modifications influence fibrillin-1's incorporation into microfibrils, stability and interactions with other ECM proteins. Kegley said the research deepens understanding of Marfan syndrome and highlights glycobiology's broader role in health and disease.

"There's still a lot to learn about how significant these findings could be for the Marfan syndrome community and others affected by fibrillinopathies," Kegley said. "But it may eventually help guide the development of new treatments."

Elisabeth Adkins Marnik is the Director of Science Education & Outreach at the MDI Biological Laboratory in Bar Harbor, Maine. She is also an ASBMB Today volunteer contributor.



Pink colonies on an agar plate indicate growth of vancomycin-resistant enterococci.

Antibiotic sensor directly binds drug in resistant bacteria

By Emily Ulrich

Vancomycin-resistant enterococci bacteria, or VRE, cause serious hospital-acquired infections, prompting scientists to search for new ways to target these hard-to-treat pathogens. VRE detect vancomycin through a transmembrane histidine kinase, called VanS, which phosphorylates the transcription factor VanR. Once phosphorylated, VanR triggers the production of enzymes that shield the bacterial cell wall from vancomycin's effects. Ten genetic variants of this system exist, and disrupting it could restore vancomycin's effectiveness. However, scientists do not understand how VanS senses vancomycin. Lina Maciunas, Photis Rotsides and a team at Drexel University College of Medicine tackled this question in their recent **Journal of Biological Chemistry** article.

The team developed an assay to study type-B VanS in nanodiscs, which mimic the cell membrane environment for purified membrane proteins. VanS performs three functions: autophosphorylation, transferring the phosphate group to VanR and dephosphorylating VanR. Testing these functions with vancomycin, the authors found increased autophosphorylation and slightly decreased dephosphorylation, consistent with the antibiotic activating the resistance system.

They then used a modified vancomycin photoaffinity probe and detected direct binding of the VanS sensor domain in the nanodisc, as assessed by mass spectrometry. Isothermal titration calorimetry confirmed that this interaction is specific for vancomycin since VanS did not bind a similar antibiotic.

Future work will explore how other VanS variants interact with vancomycin. Detailed insight into this interaction could guide inhibitor design to block antibiotic resistance in severe infections.

Matrix metalloproteinase inhibitor reduces cancer invasion

By Emily Ulrich

Matrix metalloproteinase-9, or MMP-9, remodels the extracellular matrix, but its dysregulation contributes to many diseases, including metastatic cancer. Efforts to develop MMP-9 inhibitors to treat cancer have been hampered by small molecules' poor specificity and side effects from blocking other MMPs with similar catalytic domains. Tissue inhibitors of metalloproteinases, or TIMPs, block multiple MMPs, but tweaking their specificity is possible since some MMPs have unique domains that interact with larger protein inhibitors. Alireza Shoari at the Mayo Clinic and a team in the U.S. aimed to target the MMP-9 fibronectin domain for TIMP binding. They published their recent study in the **Journal of Biological Chemistry**.

Using yeast surface display, the team screened a large library in a directed evolution experiment to identify a TIMP-1 variant with enhanced binding to the MMP-9 catalytic and fibronectin domains. They selected the strongest binder, TIMP-1-C15, and confirmed its MMP-9 inhibition in enzyme assays. The authors demonstrated that TIMP-1-C15 inhibition decreases if the fibronectin domain is removed, providing evidence that the fibronectin domain heavily contributes to the interaction.

Flow cytometry and dose-dependent inhibition assays showed that TIMP-1-C15 selectively binds and inhibits MMP-9 over MMP-1, -2 and -3, even though MMP-2 contains a similar fibronectin domain. In cell invasion assays with triple-negative breast cancer cells that require MMP-9, TIMP-1-C15 reduced cell invasion at least as well, or slightly better, compared to unmodified TIMP-1.

Future structural studies will reveal how TIMP-1-C15 binds MMP-9's fibronectin domain over MMP-2's similar domain. In addition, animal model testing will help determine TIMP-1-C15's potential to treat metastatic cancer.

AI-designed biomarker improves malaria diagnostics

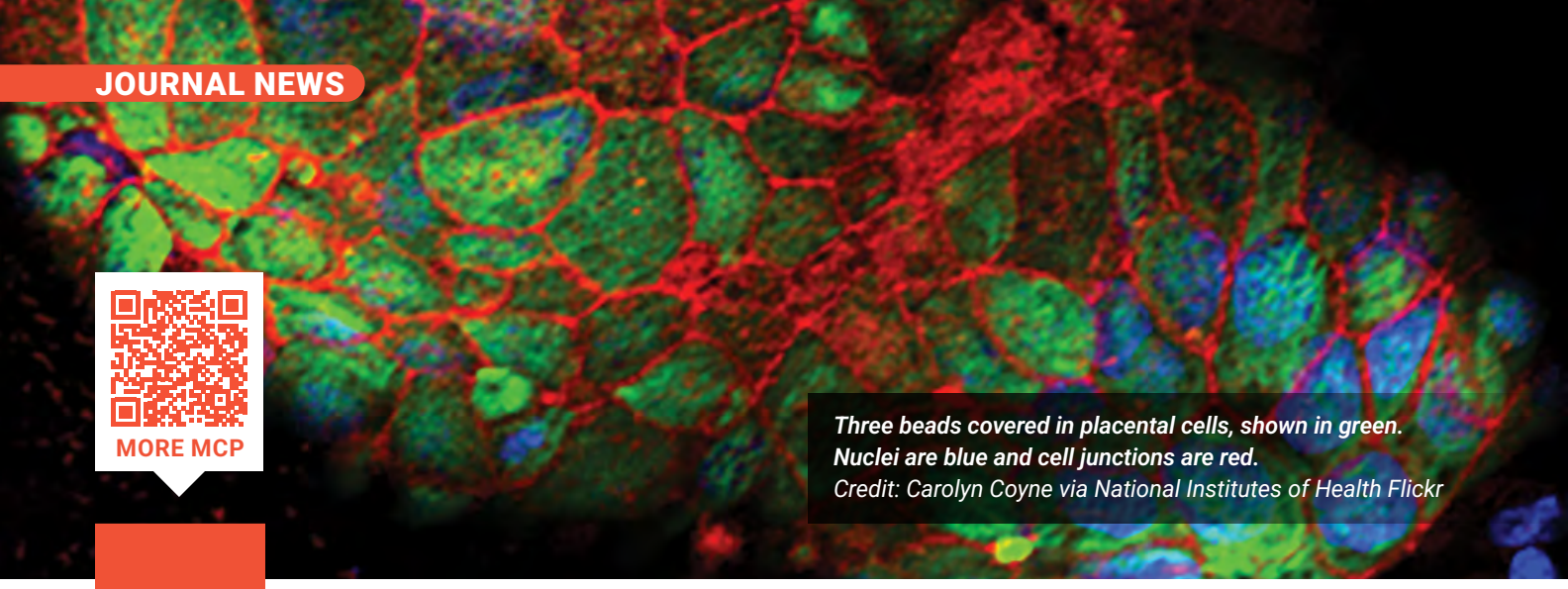
By Emily Ulrich

The malaria parasite *Plasmodium vivax* can persist in a dormant state, causing relapsed infections and ongoing transmission. To detect possible dormant infections, clinicians use a diagnostic test containing parasite proteins, such as reticulocyte-binding protein 2b, or PvRBP2b, that trigger a host antibody response. Of the biomarkers in this test, a response to PvRBP2b provides the strongest indication of a dormant infection, but PvRBP2b is difficult to produce and has low stability. Jaison D Sa at the Walter and Eliza Hall Institute of Medical Research and the University of Melbourne, Australia, and an international team recently reported stabilized PvRBP2b variants in the **Journal of Biological Chemistry**.

Because much of PvRBP2b's surface binds antibodies, the team had to preserve these sites while boosting stability to keep the protein viable for diagnostic tests. They determined that they needed to mutate residues in the protein core, a challenge to maintaining the overall protein structure. They used computational modeling and an artificial intelligence–based sequence generator to design three PvRBP2b variants. All three purified variants had higher yields and greater thermal stability than the nonmutated protein.

X-ray crystallography and biolayer interferometry, a technique measuring light reflection patterns to sense biomolecule interactions, confirmed that the variants retained the original overall structure and antibody-binding capabilities. Finally, in plasma assays using samples from individuals in malaria-endemic regions, the variants elicited antibody responses comparable to the original PvRBP2b protein. These variants could improve malaria diagnostic kits and may help solve protein stability issues in other diagnostic tests.

Emily Ulrich is ASBMB's former science editor.



MORE MCP

Three beads covered in placental cells, shown in green. Nuclei are blue and cell junctions are red.
Credit: Carolyn Coyne via National Institutes of Health Flickr

MCP

Mapping the placenta's hormone network

By Meric Ozturk

Pregnancy rewires a mother's physiology through complex hormonal signaling, with steroid hormones playing a central role. Produced in the adrenal glands, ovaries and especially the placenta, these hormones shape both maternal and fetal health. Mapping their pathways is key to understanding normal pregnancy and complications such as preterm birth or developmental disorders.

In a recent study published in **Molecular & Cellular Proteomics**, Christiane Albrecht, from the University of Bern, Rona Karahoda, from Charles University and colleagues profiled 51 steroids by analyzing maternal blood, newborn blood and placental tissue. Using liquid chromatography-mass spectrometry, or LC-MS, the researchers measured both classic steroid hormones and a newer class known as 11-oxygenated, or 11-oxy, steroids. The group has explored this complex hormonal landscape by studying samples from 37 healthy pregnancies.

"Steroid pathways are notoriously complex, but by applying a more advanced analytical technique, we were able to profile a much broader spectrum of hormones. This revealed compounds that had previously gone undetected in pregnancy," Karahoda said.

Although the placenta is not usually considered an androgen-producing organ, this study confirmed the presence of 11-oxy androgens and revealed that keto-steroids are also highly abundant. These findings suggest that the enzyme 11βHSD2 — best known for inactivating stress hormones — may play a broader role in placental steroid metabolism than previously appreciated.

"These results broaden our view of the enzyme's role in the placenta," Karahoda said. "They suggest that the placenta not only modulates glucocorticoids but also processes novel androgens and progesterones."

"Although we focused on healthy term pregnancies in this study, the next step would definitely be to look at conditions such as preeclampsia or fetal growth restriction to see how steroid metabolism, and particularly enzymes like 11βHSD2, are altered," Karahoda said.

Redefining placental steroidogenesis could shed light on fetal programming and complications in pregnancy and provide a more comprehensive understanding of how placental steroid pathways influence both maternal and offspring health across the lifespan.

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

Extracellular vesicles offer clues to cattle reproduction

By Samara Baksh

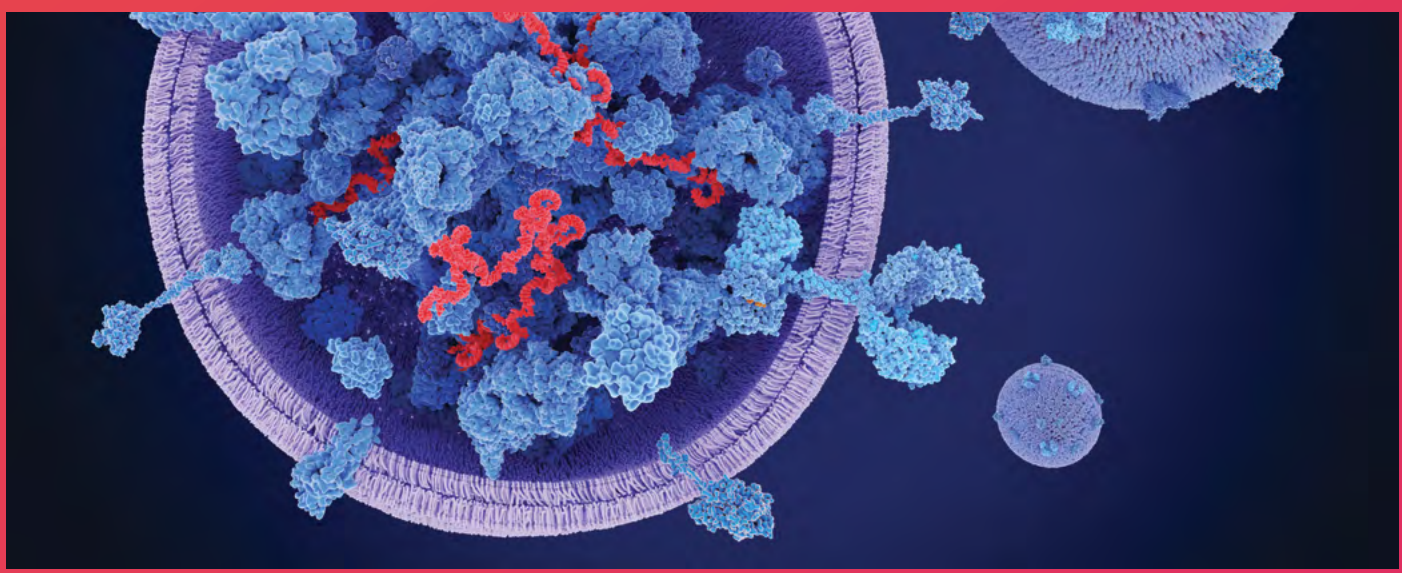
Improving bovine reproductive efficiency requires a better understanding of how the maternal reproductive tract interacts with the developing embryo. To study this complex communication, scientists are developing in vitro models that better mimic the oviduct's natural environment. Within this environment, extracellular vesicles, or EVs, regulate processes that support embryo health.

In a study conducted by Rosane Mazzarella from the National Institute for Agricultural and Food Research and Technology, published in **Molecular & Cellular Proteomics**, scientists examined the role of EVs in maternal–embryonic communication by comparing their protein cargo inside and outside of the maternal womb. Researchers collected EVs from the oviductal fluid of cyclic and pregnant heifers as well as from the conditioned media of oviductal explants removed from heifers and cultured in the lab, either with or without embryos.

About 78% of embryo-associated proteins overlapped between pregnant heifers and explants cultured with embryos, indicating that maternal-embryonic communication can be partially mimicked outside the body. However, label-free quantification revealed that EVs collected directly from the oviducts of pregnant animals and those produced by explants cultured with embryos displayed both qualitative and quantitative differences in their protein profiles. Notably, 49 EV proteins were unique to pregnant heifers. Among them, centromere protein E, which plays a role in cell division, is significant in identifying healthy, rapidly developing cow embryos and plays a key role in maintaining chromosome stability. Another protein, JAK3, helps relay signals from certain immune-related molecules and may promote embryonic cell survival and growth. These findings suggest that EVs from the natural reproductive environment may offer more robust support for embryo development.

Taken together, while laboratory systems replicate many features of maternal–embryonic signaling, EVs from pregnant heifers appear to deliver a more complete set of developmental cues, offering potential for enhancing artificial embryo culture systems via supplementation.

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



Extracellular vesicle

Protein modifications drive lung cancer resistance

By Vanshika Patel

Key protein modifications such as glycosylation and phosphorylation regulate a variety of cellular processes, such as cell-cell recognition, immune response and cell growth. Dysregulation of these tags is linked to human diseases, making them important to study disease progression and identify biomarkers and druggable targets using mass spectrometry. However, since these modifications are often low-abundance and dynamically modified, they need to be enriched first. Despite the progress in the development of individual enrichment strategies, scientists have yet to develop a highly sensitive and robust platform to focus on multiple modifications simultaneously.

Yu-Ju Chen's team from Taiwan published an article in **Molecular & Cellular Proteomics** introducing a streamlined enrichment strategy called Fe-ZIC-cHILIC, which captures both glycopeptides, or GPs, and phosphopeptides, or PPs, in a single step. This method uses a tandem tip that is packed with a special material combining iron ions with ZIC-cHILIC resin. In this setup, the sample flows through this tandem tip, minimizing protein loss and enriching GPs in the top tip while PPs are in the bottom tip. Using this strategy, they analyzed nonsmall cell lung cancer cells, or NSCLC cells with mutations in the *EGFR* gene, which is the first-line targeted therapy yet eventually drives drug resistance and leads to tumor recurrence. Within a single cell type, they identified 10,536 GPs and 11,329 PPs and found significant changes on the site-specific glycopeptides and phosphopeptides between drug-sensitive and drug-resistant cells. Notably, they observed changes in EGFR, ERBB2, MET and integrin family proteins, which are primary targets for cancer treatments.

This study highlights both the novelty of Fe-ZIC-cHILIC material in dual enrichment with high specificity to study protein modifications. Further investigations will assess the strategy in different sample types to determine how these modifications drive resistance and how therapies could be developed to improve outcomes.

How antigen-processing proteins shape immunity

By Vanshika Patel

In diseases such as cancer or infection, the body's process of presenting peptides through the antigen-processing and presentation machinery, or APPM, to immune cells is often altered. A subset of molecules called human leukocyte antigen class I, or HLA-I, presents these peptides to immune cells, constituting what is known as the immunopeptidome, which is critical for immune surveillance. However, scientists have yet to fully understand how individual components of the APPM influence the composition and diversity of the immunopeptidome.

Ilja Shapiro and a team of researchers based in Switzerland and the Netherlands published a study in **Molecular & Cellular Proteomics** where they knocked out 11 genes involved in the APPM in a cell line model to assess how these perturbations shape the immunopeptidomic landscape. They found that deleting the *CALR* gene had minor effects on reducing immunopeptidome diversity, while, as expected, deleting *B2M* led to a dramatic change in the immunopeptidome. More specifically, deleting genes such as *TAP1*, *TAP2*, or *IRF2* caused a significant change in the length preference, binding affinity, diversity and presentation capacity on HLA-I molecules. These results highlight the importance of the APPM in regulating immunity and may help explain how defects in antigen presentation reshape the immunopeptidome in diseases such as cancer. Future research can help develop predictive tools to investigate HLA-bound peptides when presentation defects arise in diseases.

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

MORE JLR

Chromosomes

JLR

Cholesterol as a novel biomarker for Fragile X syndrome

By Pearce Hyatt

Fragile X syndrome, or FXS, is the most common inherited cause of intellectual disability in the U.S. Each year, thousands of people are diagnosed with the condition. Many patients' families may be told their child will have the condition, with no real knowledge of what life will look like for their child.

While the cause of the disease is known — a mutation in the FMR1 gene — there is a wide range of symptoms among those with the trinucleotide repeat. Tests may catch the disease, but they do not give parents much guidance about what degree of learning disability to expect when raising their kids.

At the University of Sherbrooke in Quebec, Asma Laroui Artuela Çaku and Jean–Francois Lepage sought to identify better biomarkers of FXS function. Their work was published in the **Journal of Lipid Research**.

"We have access to the FXS clinic, which regroups patients from all around the province, so we can do a lot of associations between the lipid profile and the clinical phenotype," Çaku said.

The team discovered that levels of 24S-hydroxycholesterol, or 24-OHC — a downstream metabolite of brain cholesterol — decreased in patients with FXS compared with controls.

Cholesterol is synthesized in the brain by astrocytes, and excess amounts are converted to 24-OHC before being transported into peripheral circulation. Thus, a drop in 24-OHC in the blood offers a practical way to measure brain cholesterol metabolism in patients with FXS.

The researchers also found that 24-OHC levels were inversely correlated with several neurologic measures. Patients with lower 24-OHC showed slower motor evoked potentials, meaning their motor neurons fired less efficiently when stimulated by transcranial magnetic stimulation. On behavioral and cognitive questionnaires, patients with lower levels of 24-OHC reported greater difficulties with social communication as well as more symptoms of anxiety and depression.

Taken together, these results suggest that 24-OHC could serve as a useful biomarker to help clinicians give families a clearer picture of what to expect after an FXS diagnosis.

"We try to develop techniques that are not invasive," Çaku said. "In a simple blood collection, you can have information about the brain." These lipid profiles may help patients and their families better understand their neurologic development.

Laroui added that the findings also shed light on brain cholesterol biology more broadly.

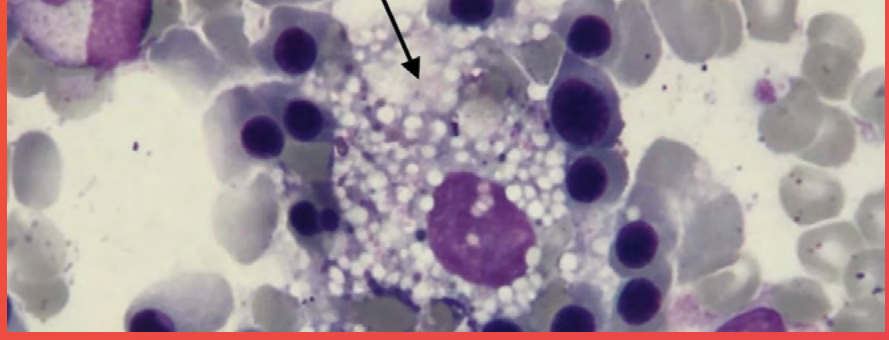
"There is a lot of research from the peripheral, but comparatively fewer studies on brain cholesterol," she said. "However, we found that in diseases where brain cholesterol is dysregulated, this imbalance can affect the phenotype. We are giving importance to research on brain cholesterol."

The team now hopes to uncover the source of the abnormal cholesterol metabolism in FXS.

"Maybe it's the enzyme that's dysregulated, or maybe the astrocytes cannot produce enough cholesterol," Laroui said.

Despite these unknowns, the researchers are optimistic that their work will deepen understanding of brain lipid metabolism and open the door to new tools for patient care.

Pearce Hyatt is a medical student at Wake Forest School of Medicine and an ASBMB Today volunteer contributor.



Foamy macrophage, often associated with atherosclerotic plaques, with several irregular vacuoles in the cytoplasm surrounded by erythroblasts (arrow). Credit: Perla Vicari via the American Society of Hematology Image Bank

ApoA1 reduces atherosclerotic plaques via cell death pathway

By Swarnali Roy

Atherosclerotic plaques form when cholesterol, fat and blood cells gradually build up in artery walls, narrowing them and reducing oxygen-rich blood flow from the heart to the body. The Centers for Disease Control and Prevention and the Cleveland Clinic report that one in five U.S. deaths is caused by heart disease, and half of adults ages 45–84 have atherosclerosis without knowing it. High low-density lipoprotein, or LDL, levels raise the risk of atherosclerosis; while apolipoprotein A1, or ApoA1, a major component of high-density lipoprotein, or HDL, protects against it.

In a recently published article in the **Journal of Lipid Research**, Alexander S. Qian and colleagues at McMaster University and Hamilton Health Sciences studied how ApoA1 modulates Bim, a cell-death mediator, in the development of atherosclerotic plaques and necrotic cores. Cholesterol buildup triggers endoplasmic reticulum, or ER, stress in macrophages, which increases Bim expression and leads to macrophage death.

In mice, overexpression of ApoA1 reduces plaque formation. The researchers engineered mice lacking low-density lipoprotein receptors, or LDLR, with or without ApoA1 and fed them a high-fat diet for 10 weeks. Mice lacking both LDLR and ApoA1 developed larger plaques, bigger necrotic cores and higher Bim expression. In bone marrow transplant studies, LDLR- and ApoA1-deficient mice that received Bim-deficient marrow showed reduced plaque and necrotic core size. They also had more circulating immune cells and lower cholesterol and triglyceride levels, regardless of ApoA1 status. The team plans to further study how ApoA1 lowers Bim protein levels in macrophages within plaques.

Swarnali Roy is a postdoctoral researcher at the National Institute of Diabetes and Digestive and Kidney Diseases, NIH and an ASBMB Today volunteer contributor.

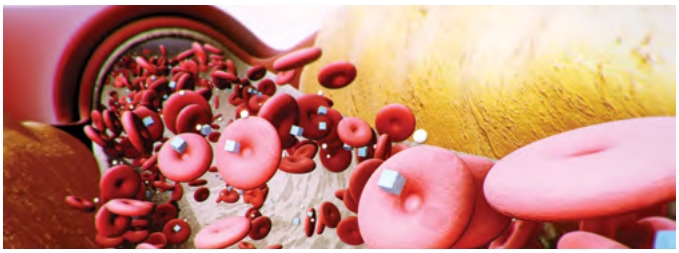
Lipid profiles reveal sex differences in type 2 diabetes

By Jessica Desamero

Type 2 diabetes, or T2D, is a metabolic disorder that affects global communities disproportionally. For example, South Asians face a particularly high risk. Changes in lipid species affect T2D, but scientists have not identified any reliable lipid biomarkers. Fatty acids, or FAs, are the major components of many lipids, and they can be potential lipid biomarkers. However, conflicting findings have left unclear how essential FAs relate to T2D development.

In a recent study in the **Journal of Lipid Research**, Madhusmita Rout and a team of researchers at the University of Oklahoma Health Sciences Center investigated the lipid profiles of individuals from a well-characterized cohort of Asian Indians. They found that in T2D, levels of two key cell membrane components — sphingomyelin and phosphatidylcholines — decreased, while free FAs and lysophosphatidylcholines, or LPCs, increased. After adjusting for age, sex and body mass index, or BMI, they saw significant increases in several essential FAs, such as the omega-6 FA arachidonic acid and the omega-3 FA docosahexaenoic acid. However, in obese individuals, most omega-3 and omega-6 FAs were reduced two- to six-fold. The team also observed sex- and age-related lipid differences. For example, one LPC type was elevated in men of all ages but rose in women only after menopause.

Ultimately, this study identified potentially useful lipid biomarkers that could possibly affect the development of T2D and obesity. Future directions include clarifying the relationship between omega FAs and T2D, as well as the role of essential FAs in human metabolic diseases.



Blood sugar

Sex and diet shape fat tissue lipid profiles in obesity

By Jessica Desamero

In obesity, adipose tissue expands and accumulates, driving chronic inflammation. Previous research showed that sex steroid hormones can influence adipose tissue distribution, accumulation and immune responses in men and women. Changes in lipid composition in the visceral or gonadal white adipose tissue, or GWAT, during obesity can drive immune cell accumulation and boost proinflammatory mediators. Prior studies revealed sex differences in GWAT lipid species in obese mice, but scientists still do not understand the exact role of sex hormones in lipid composition.

In a recent study in the **Journal of Lipid Research**, Mita Varghese and a team of researchers at the University of Michigan investigated the GWAT lipid profiles in obese mice and mice with their gonads surgically removed, or GX mice. In an untargeted lipidomics analysis where they comprehensively analyzed all lipids, they found sex differences in several lipid species, such as phospholipids and sphingolipids, which are important cell membrane components. Obese males had significantly more precursor fatty acids than females and GX mice. Targeted analysis revealed sex differences in polyunsaturated fatty acids, or PUFAs, with males showing a significantly higher omega-6 to omega-3 ratio. They also found diet-driven differences in oxylipins, inflammation-linked lipids, which were higher in both male and female obese mice than in lean mice.

This study suggests that sex hormone levels and diet equally induce inflammation and changes in lipid composition in obesity. Future studies include further confirming lipid profiles and understanding how sex differences arise in obesity.

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

FEATURE

The science of **living longer, healthier lives**

Exploring the link between lipids and longevity

By Courtney Chandler

On both sides of the family, Meng Wang's grandmothers lived long, healthy lives, well into old age — one to 100 years old, the other to 95.

"Seeing them always made me wonder, why can't everyone age like them?" she said.

That question guided her postdoctoral research at Harvard Medical School and Massachusetts General Hospital where she explored how metabolism influences lifespan using the nematode worm *Caenorhabditis elegans*. Wang investigated the link between metabolism and aging, focusing on how lipolysis — the breakdown of fats — affects lifespan.

Later, using multidisciplinary approaches over the course of a few years, her group at Baylor College of Medicine revealed that the enzyme LIPL-4 breaks down specific lipid molecules stored in the cell's recycling centers, the lysosomes. This process generates lipid messengers that travel to the nucleus, where they activate genes that enhance metabolism and extend lifespan. Her findings uncovered a novel molecular pathway linking fat metabolism to longevity.

"This was the beginning of my journey into aging research from a metabolic perspective," Wang said.

Now a senior group leader, Wang continues to dissect the molecular mechanisms of aging at the Howard Hughes Medical Institute Janelia Research Campus, focusing on how metabolic products, or metabolites, serve as signaling molecules to influence gene expression, inter-organ dialogue and microbiota-host interactions to promote longevity.

Wang's work on expanding the basic understanding of longevity and metabolic signals will also identify new avenues for interventions. By modifying metabolism or influencing its signaling pathways, aging could also be altered.

For Wang, aging research is about more than extending lifespan — it's about tackling age-related chronic diseases, including diabetes, heart disease, neurodegeneration and cancer. With demographics shifting towards an older population, these conditions

are expected to place growing pressure on society and healthcare systems. Wang believes that uncovering the biology of aging could provide critical insights for preventing or treating them.

"Aging is the single greatest risk factor for many chronic diseases," Wang said. "The idea is that if we could target aging itself, we might be able to combat multiple age-related diseases at once."

The fat–neuron connection

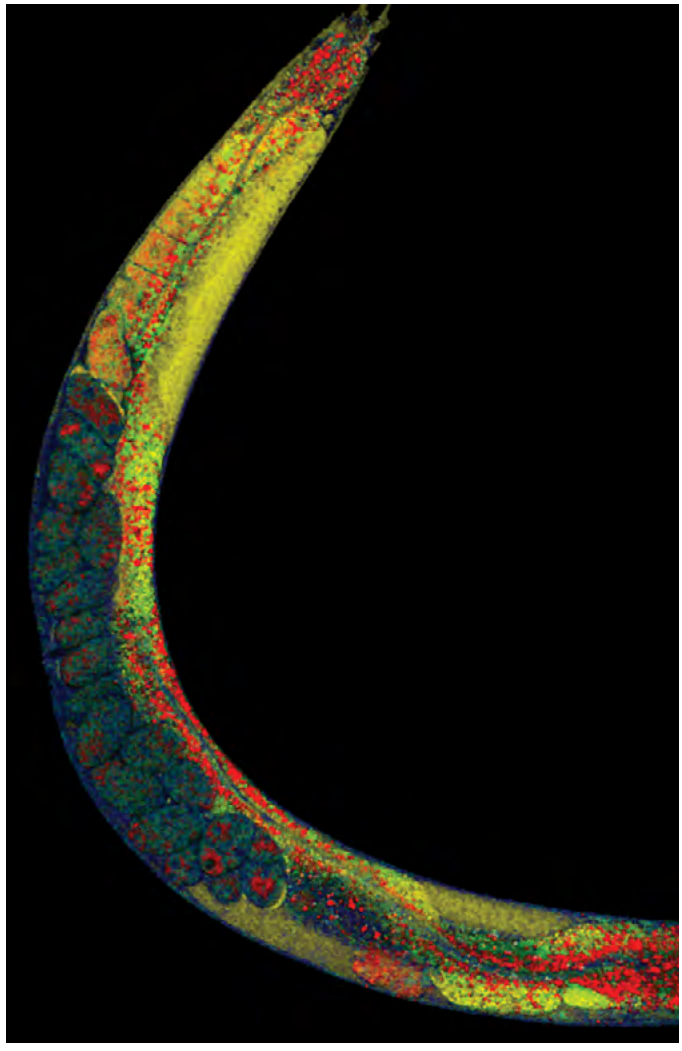
Building on the discovery of LIPL-4, Wang used an RNA interference screen to find genes involved in the lifespan extension seen in *C. elegans* engineered to overexpress this enzyme in intestinal fat cells. Her team identified *nlp-11*, a neuropeptide gene predominantly expressed in neurons, as a critical downstream effector mediating the whole-body longevity effect.

"The discovery of the link between *lipl-4* and *nlp-11* was a surprise to me," Wang said. "Previously, it was not known that lysosomes in one tissue could signal to another, but even more so, that lipid messengers derived from lysosomes could mediate such cross-tissue communication."

Lipids as molecular connectors

With LIPL-4 in fat cell lysosomes and NLP-11 in the neuronal nucleus serving as the bookends, Wang looked to identify the signaling intermediates in this pathway. Lipidomics showed that *lipl-4* overexpression increased production of lipids called polyunsaturated fatty acids, or PUFAs. Blocking PUFA synthesis eliminated the lifespan extension, identifying PUFAs as key messengers in the pathway.

Wang hypothesized that PUFAs are transported from fat cells to neurons by a lipid-binding protein, or LBP. By searching the *C. elegans* genome, her team identified LBP-3 as the key carrier. Knocking down *lbp-3* suppressed lifespan extension, while its overexpression increased longevity. She further showed that the receptor NHR-49 in neurons mediates PUFA signaling by activating *nlp-11*.



A stimulated Raman spectroscopy microscopy image of Caenorhabditis elegans reveals molecular vibrations of molecules, including lipids (red), allowing Meng Wang to visualize the worm's metabolic state in real time. Credit: Meng Wang

Her work established a fat-to-neuron signaling axis, where lysosomal lipolysis releases PUFAs that, carried by LBP-3, activate neuronal NHR-49 and neuropeptide signaling to promote lifespan extension.

"We find that specific metabolic products can actually serve as communication signals between different parts of the body," Wang said. "This communication helps to maintain physiological harmony."

Wang will present her work on aging and metabolism at the ASBMB 2026 Annual Meeting.

Melanie McReynolds and her former Ph.D. mentor, Wendy Hanna–Rose, examine a specimen using a light microscope in McReynolds's lab at Penn State in 2025. Credit: Dan Lesher

Fueling healthier aging, connecting metabolism stress and time

By Courtney Chandler

Melanie McReynolds first began asking questions about aging at home, not in the lab. Her parents had her later in life, and she grew up watching them age. Watching her parents grow older sparked her lifelong fascination with how our bodies change over time and what it means to age well.

"I was always intrigued by aging and how to age healthier because I saw my parents aging," she said. "I wanted to understand how to age healthier and avoid age-related diseases." Those early observations would fuel her quest to understand the molecular rhythms of aging — and how to make aging healthier.

At Alcorn State University, McReynolds' curiosity found direction. Selected as one of the school's top students, she traveled to Bangalore, India, and later attended her first Annual Biomedical Research Conference for Minoritized Scientists. "I realized there was a career in science," she said. "I've been hooked ever since."

Connecting metabolism and the aging process

McReynolds first explored the biology of aging in depth during her postdoctoral fellowship at Princeton University, where she began studying how metabolism influences the aging process in the lab of Joshua Rabinowitz. Around the same time, her research took on a deeply personal dimension: her mother was diagnosed with multiple myeloma, a cancer associated with older age.

"She was the healthiest senior citizen you ever met," McReynolds said. "Then after her diagnosis, her decline was rapid — she wasn't able to do anything."

Witnessing that transformation changed how McReynolds thought about her work. What had begun as an intellectual curiosity became an urgent mission to understand what happens as we grow older — and how to age healthier.

Aging is the leading risk factor for many of the most common diseases in developed countries, including cancer, diabetes, cardiovascular disease and neurodegenerative disorders. The impact of these conditions is expected to grow as populations live longer; by 2050, the number of Americans over age 65 is projected to increase by more than 40%.

"Older age is a time when we are supposed to retire, travel the world and truly benefit from the fruit of our labor," McReynolds said. "However, we have to worry about the darker side that comes with aging."

At Princeton, she began connecting the dots between metabolism and the physiological decline that comes with age. Her work sought to uncover how disruptions in metabolic pathways contribute to age-related diseases and whether manipulating those pathways could promote healthier aging.

Since 2022, Melanie McReynolds has been studying nicotinamide adenine dinucleotide, or NAD⁺, and aging at Penn State University. Credit: Michelle Bixby

The power of NAD⁺

Now an assistant professor of biochemistry and molecular biology at Penn State University, McReynolds's work focuses on nicotinamide adenine dinucleotide, or NAD⁺, a molecule central to how cells communicate and convert food into energy.

"Many think of ATP as the main energy currency of the cell, but you need NAD⁺ to make ATP," McReynolds said. "NAD⁺ is the key molecule responsible for breaking down food into energy and is also needed for cells to be able to communicate with each other."

As we age, levels of NAD⁺ decline, affecting energy balance and cell repair. McReynolds has long been determined to understand this connection.

As a graduate student at Penn State University, McReynolds studied the synthesis and role of NAD⁺ in reproductive development. Yet even then, she was already thinking about the link between NAD⁺ and aging, said Wendy Hanna–Rose, her graduate adviser.

"(McReynolds) went and specifically got the training she needed to work on aging," Hanna–Rose said. "That's pretty impressive."

During her postdoctoral work, Rabinowitz's lab was developing techniques to trace metabolism in living systems. She initially thought she might have to leave NAD^+ behind, but discussions about aging kept resurfacing.

"Every time we talked, it was about aging this and aging that," McReynolds said.

They knew that no one had examined how NAD^+ metabolism shifts in older mice. That question — what happens to NAD^+ as we age — would define the next stage of her career.

NAD^+ and aging

Supported by a Howard Hughes Medical Institute Hanna Gray Fellowship, McReynolds used techniques including isotope tracing and mass spectrometry to track how fast NAD^+ was produced and consumed in young and old mice. Her findings revealed that while NAD^+ levels decline with age, production remains largely stable. This suggests that increased consumption drives depletion, and that targeting NAD^+-intensive pathways like stress and inflammation may be more effective at combating the consequences of aging than NAD^+ supplementation.

Now leading her own lab at Penn State, McReynolds uses the nematode *Caenorhabditis elegans* to capture metabolism in motion. Her lab combines high-resolution liquid chromatography/mass spectrometry, isotope tracing and multiomics approaches to map how NAD^+ and its precursors change as organisms age.

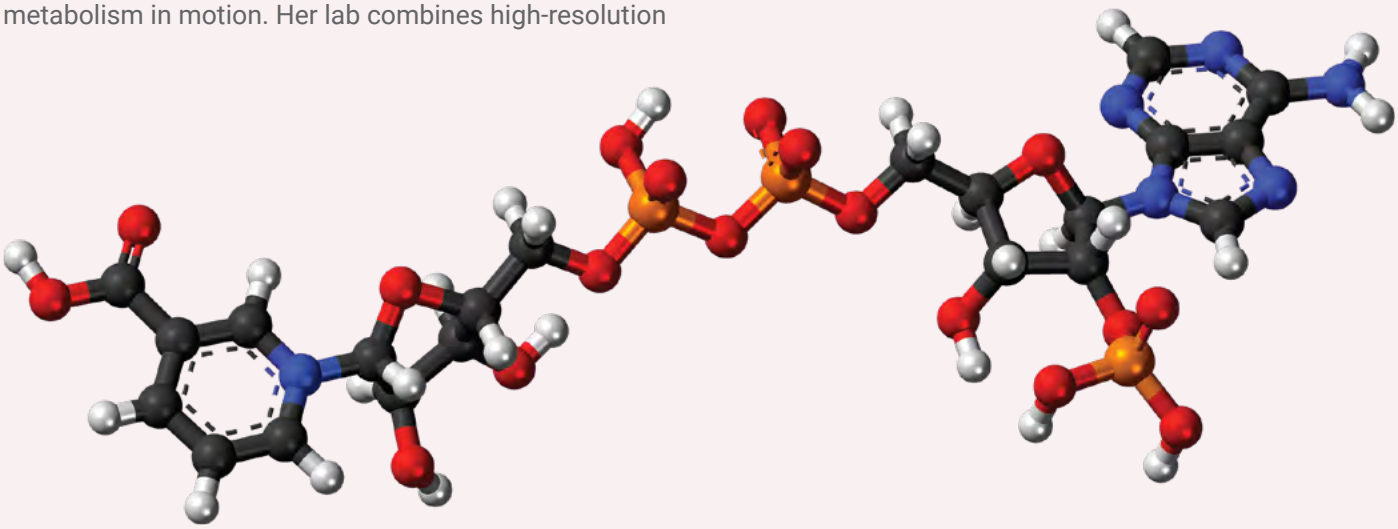
"We're using *C. elegans* to really understand cellular aging and stress over the lifespan," McReynolds said.

For this work, she was recently awarded the National Science Foundation Faculty Early Career Development Award, which recognizes early-career faculty who excel in both research and education.

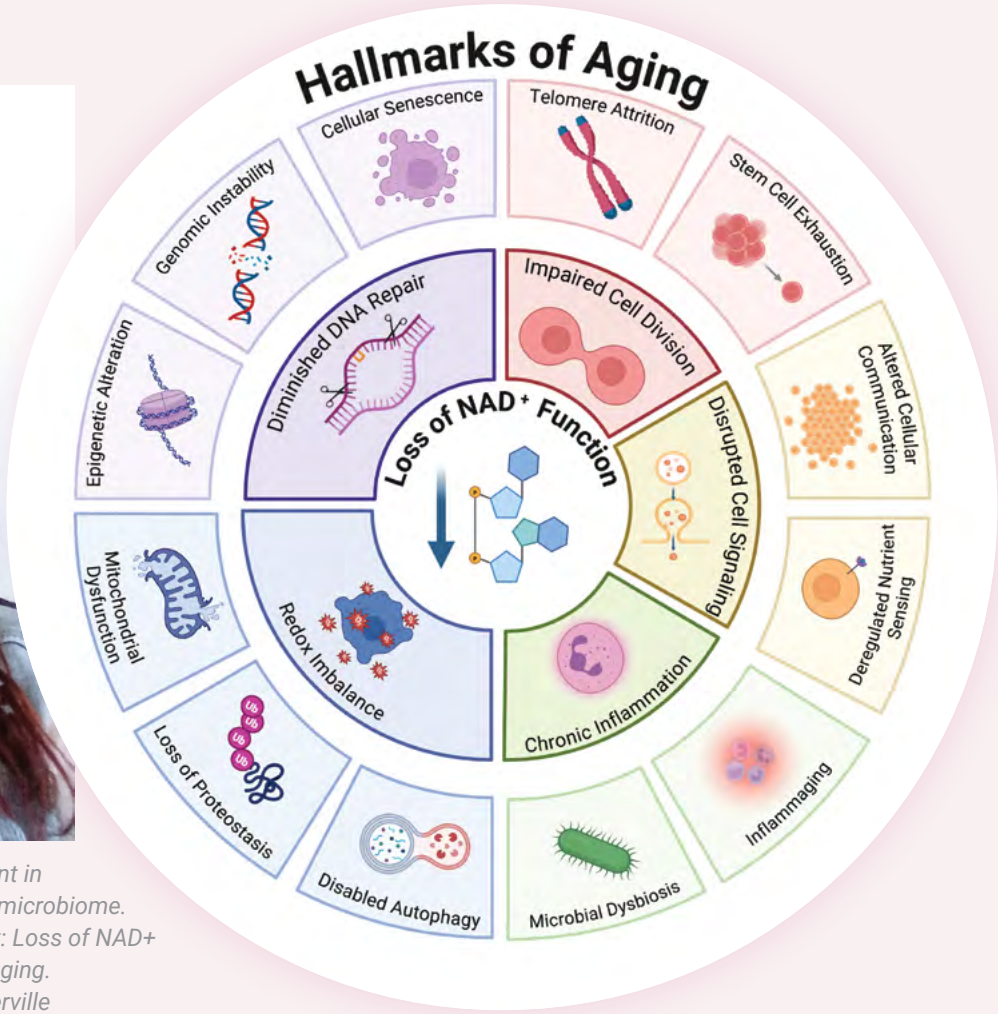
Mapping metabolism across the lifespan

In another line of research, McReynolds explores how energy metabolism intersects with the gut microbiome. In her postdoc, she and collaborators demonstrated that NAD^+ precursors move dynamically and bidirectionally between the host and the gut microbiome, revealing the microbiome's critical role in shaping systemic NAD^+ metabolism.

Victoria Baskerville, a graduate student in McReynolds' lab, is continuing this line of work to understand what happens when the gut microbiome is disrupted by stressors such as inflammation. "It is essential to know whether age-associated changes in the gut microbiome could contribute to the age-associated decline of NAD^+ across tissues," Baskerville said.



The small molecule NAD^+ is critical for metabolism and declines with age.



Above: Victoria Baskerville, a graduate student in McReynolds' lab studying NAD+ and the gut microbiome. Credit: Courtesy of Victoria Baskerville. Right: Loss of NAD+ function is one of the defining hallmarks of aging. Credit: Melanie McReynolds & Victoria Baskerville

Using multiomics techniques, Baskerville is mapping the NAD⁺ metabolome across tissues to track metabolism shifts during aging and identify potential sex-based differences. She said that most previous studies on the NAD⁺ metabolome have primarily relied on male model organisms, leaving sex-specific metabolic and longevity differences largely unaddressed — her research aims to fill this gap.

"Understanding sex-based differences in molecular aging will facilitate the development of tailored interventions that improve healthspan for all," Baskerville said.

Beyond the bench

McReynolds' passion for science extends well beyond her lab. Growing up in Mississippi, she had limited exposure to research opportunities, which motivates her commitment to mentorship and outreach.

"Once (McReynolds) came to my lab, there were other students who joined after her specifically because of her," Hanna–Rose said. "Her advocacy brought other people to my lab."

Baskerville echoed this sentiment. "I was inspired by the mission of promoting healthier aging," she said. "But I also joined because Dr. McReynolds is a passionate mentor who cares deeply about developing future scientists."

As part of her NSF Award, McReynolds is launching Science in Action, an annual outreach series for middle and high school students in her hometown of Louisville, Mississippi.

"My goal is to continue bringing STEM opportunities to middle and high school students in under-resourced areas, while also giving my undergraduate and graduate trainees the chance to lead experiments, serve on panels and mentor the next generation," she said. "It's my way of giving back while empowering my students."

A vision for healthier aging

Looking ahead, McReynolds sees the field of aging research shifting toward strategies that extend healthspan — the years of life spent in good health — rather than lifespan alone.

Her research already has a human impact. NAD^+-related supplements have surged in popularity in recent years, often marketed with ambitious promises of extended lifespan. However, McReynolds said the scientific evidence — especially around hyper-supplementation — remains limited, despite the industry's million-dollar scale.

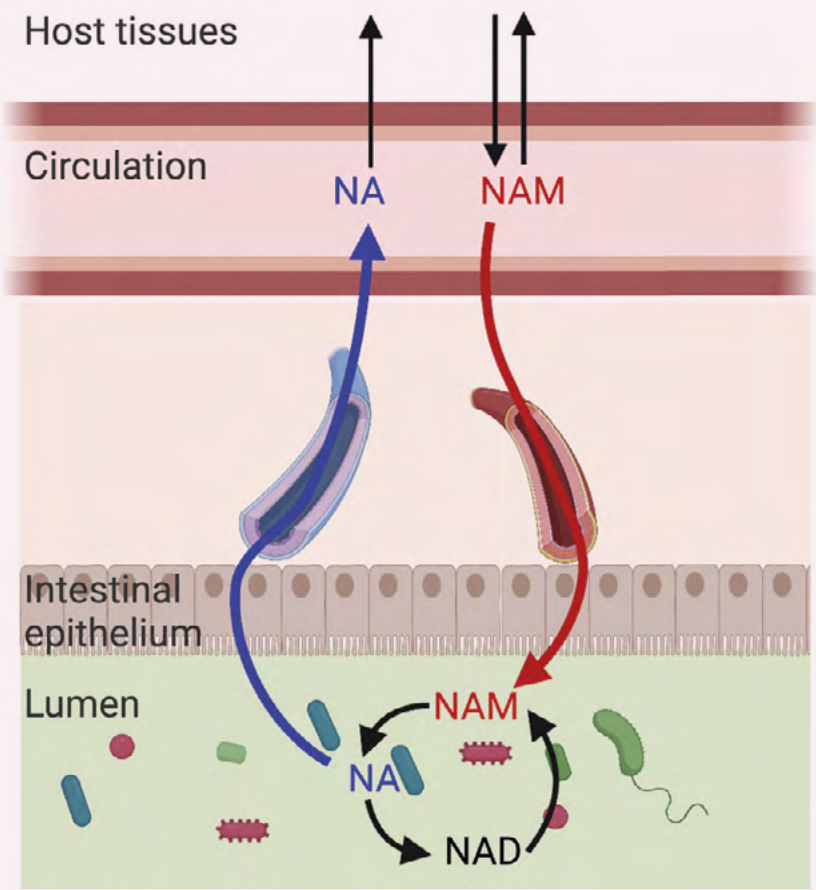
"It's fascinating because everyone's biology is different, and what benefits one person could be detrimental to another," McReynolds said. "Our goal is to develop safer and more effective ways to support healthy aging, without relying on the one-size-fits-all supplement approaches." She also sees a new era of convergence ahead, one that integrates genetics, metabolism, behavior and environment.

"By combining (multiple approaches and disciplines), we can begin to answer some of the biggest questions about what drives aging and age-related disease," she said.

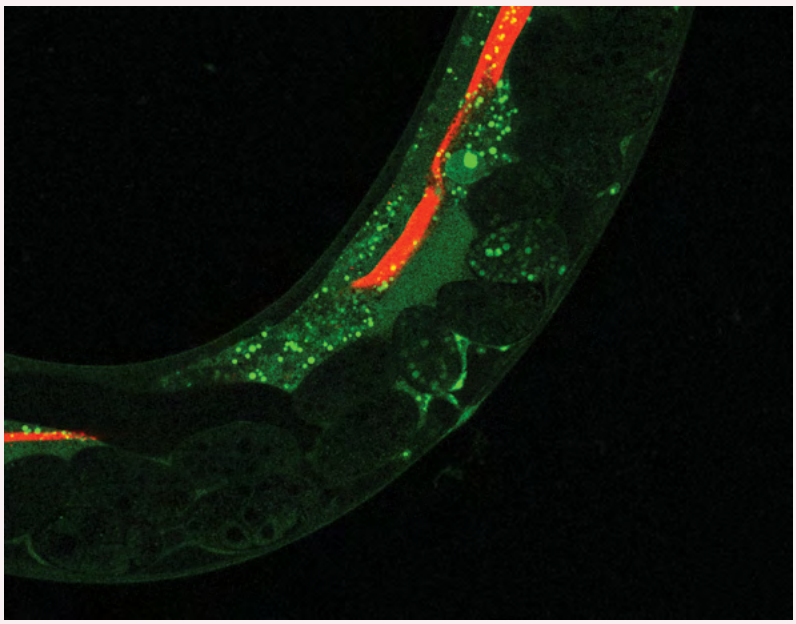
For McReynolds, pushing the boundaries of aging research also remains personal.

"Seeing my mother and how she transitioned really motivates me to continue this work," she said. "If we can age healthier, that's what I think matters and is what still pushes me."

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



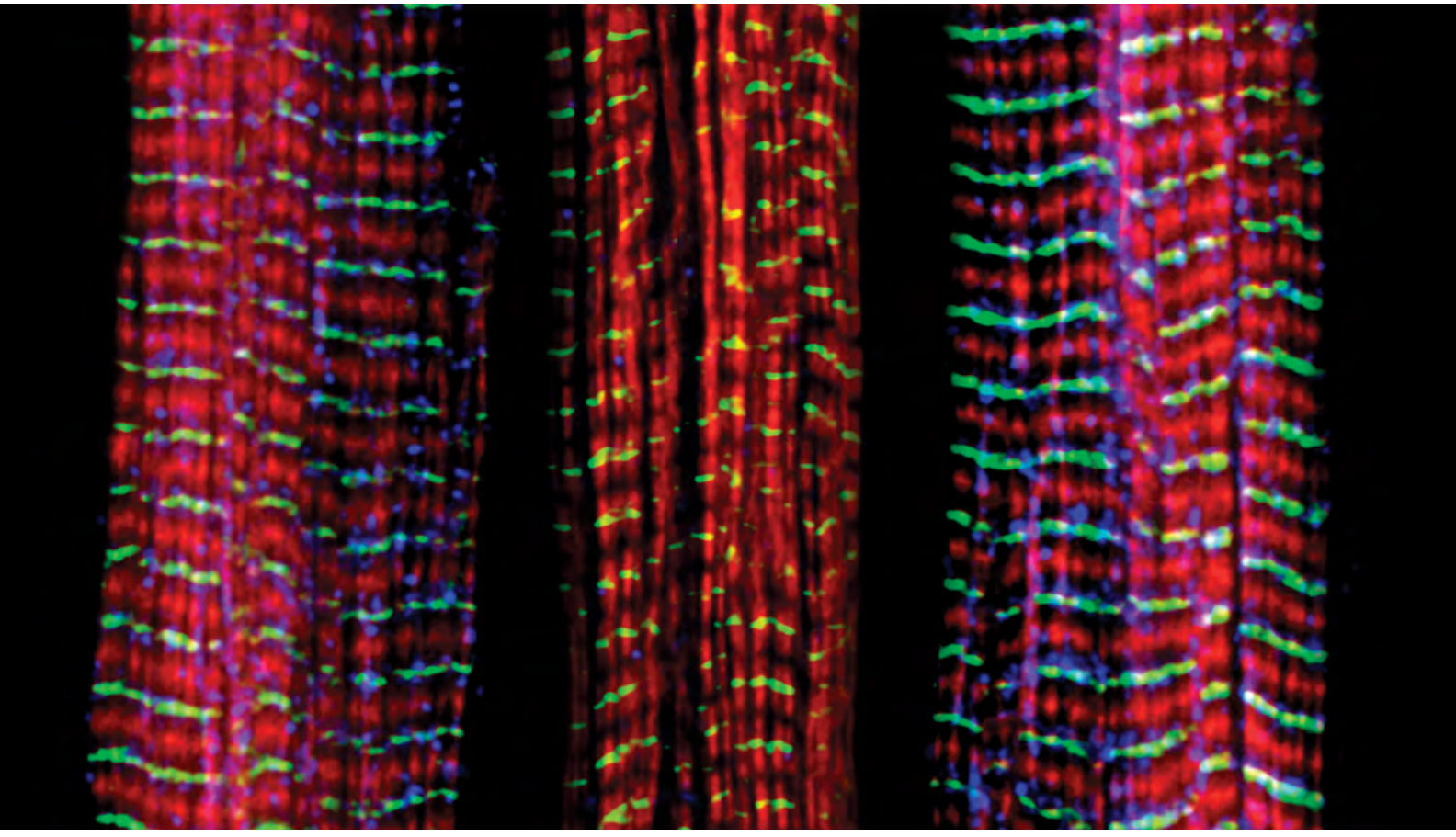
NAD^+ and its precursors, NA and NAM, cycle between the gut microbiome and circulatory system. Credit: Melanie McReynolds and Abrar Alsaadi



In this microscopy image of Caenorhabditis elegans*, green marks the animal's tissues and red marks the intestines. Credit: Abdul Kareem Alshaheeb and Mahmoud Yahia*

The science of staying strong

By Inayah Entzminger



Of the three muscle fibers shown here, the one on the right and the one on the left are normal. The middle fiber is deficient a large protein called nebulin (blue). Nebulin plays a number of roles in the structure and function of muscles, and its absence is associated with certain neuromuscular disorders. Credit: Christopher Pappas and Carol Gregorio, University of Arizona

Roughly 560 million years ago, the first striated muscular system appeared in the cnidarian ancestor of jellyfish and corals. *Haootia quadriformis*, which lived anchored to the sea floor, likely used its new muscle fibers to grasp food, an upgrade from the passive filtering of its sponge phylum.

Since then, muscles have powered astonishing feats: the white-throated needletail flies 105 mph, lions drag prey twice their weight, and crocodiles crush with 5,000 pounds of bite force.

However, that power has a price. Every contraction wears muscle down, and with age, repair lags behind decay.

"The ethos of aging is that entropy wins," Stuart Phillips, a professor of kinesiology at McMaster University in Hamilton, Ontario, said. "Eventually, the system just can't hold its integrity because of mutations, oxidative damage, telomere shortening, you name it."

Phillips has spent decades studying how skeletal muscle breaks down, regenerates and adapts, and how lifestyle, nutrition and molecular processes can slow that decline.

Scientists from other fields also contribute to the study of aging. By investigating how other animals use their muscles to work harder and longer than humans, evolutionary biologists determine what mechanisms to target for human muscle regeneration.

Stuart Phillips speaks at Cornell University in October 2025. Phillips presented data on dietary protein requirements, sharing information on changing protein needs across different ages. Credit: Ryan Issa

A broken leg and a new direction

In 1989, Phillips returned to McMaster for his senior year. A biochemistry major and rugby captain, he was ready for victory — until a broken leg benched him for months.

"I was devastated at the time," Phillips said. "And then I took a senior thesis and spent time in the lab instead, and all of a sudden I fell in love with science."

When his hip-to-ankle cast came off after 14 weeks, Phillips saw his leg had wasted away. His mentor explained how muscles rebuild, and Phillips was fascinated by their resilience. His undergraduate research turned into a doctoral project exploring protein intake and synthesis in athletes.

"For the first 15 years of my career, I was interested in what we could do in younger people," Phillips said. "And then maybe research became 'me-search' as I got older."

Today, his work focuses on slowing muscle loss in older adults. Drastic muscle reduction causes a condition known as sarcopenia, an age-related decline in muscle mass and function that reduces mobility, balance and independence.

The science and diagnosis of sarcopenia

Sarcopenia lacks clear genetic or biochemical markers, so clinicians rely on observation.

"You want to understand their baseline physical function," William McDonald, a board-certified geriatrician, said. "Where were they six months ago and five years ago, and when they were younger?"

Muscle size and strength decrease naturally over time, but patients often experience accelerated decline after events such as infection, hospitalization, or immobility. McDonald said home visits are key for assessing real-world function — how patients climb stairs, carry groceries, or use walkers.

"There are research-level metrics like muscle circumference," he said, "but they're hard to apply in a realistic clinical setting."

One practical test is the "get up and go" assessment, in which a patient rises from a chair without using their hands. "Because (the patient) has to get up in a short period of time, it's actually power that gets them out of a chair as opposed to strength," Phillips said. "Quickness is the outward manifestation of power."

William McDonald advises a patient in his office. McDonald diagnoses sarcopenia using a combination of house calls and practical testing. Photo credit: William McDonald

Molecular machinery of movement

Skeletal muscle is one of the body's most metabolically active tissues, making up 30 to 40% of total mass and functioning under voluntary control. Its contraction relies on adenosine triphosphate, or ATP, the molecule that powers every twitch and flex.

During exercise, ATP stores are rapidly depleted and must be regenerated by mitochondria. This process also produces reactive oxygen species, or ROS, unstable molecules that play dual roles: supporting signaling and inflammation at normal levels but causing oxidative stress and DNA damage when overproduced.

To counteract ROS, cells deploy a complex antioxidant defense composed of enzymes and nonenzymatic antioxidants. As people age, however, mitochondrial efficiency declines. ATP production falls, ROS accumulation increases and antioxidant defenses weaken. This imbalance contributes to muscle atrophy, slow recovery and reduced exercise capacity in older adults.

Molecular clues from evolution

The biochemical roots of muscular aging may trace back through evolution. Two key regulators, nuclear factor erythroid 2-related factor 2, or NRF2, and Kelch-like ECH-associated protein 1, or KEAP1, form a molecular switch that helps cells respond to oxidative stress.

At Vanderbilt University, Gianni Castiglione, an assistant professor of biological sciences, studies these proteins across species to understand how evolution fine-tuned muscle resilience.

In humans, NRF2 activity is tightly controlled by KEAP1, which contains cysteine residues sensitive to oxidation. Under normal conditions, KEAP1 binds NRF2 and sends it for degradation in the proteasome. When oxidative stress rises, KEAP1's cysteines oxidize, altering its structure and releasing NRF2 to the nucleus, where it triggers antioxidant gene expression.

"Antioxidants are Goldilocks compounds," Castiglione said. "Too much (antioxidant activity) is bad and can create reductive stress, which inhibits muscle signaling."

How other animals endure extreme muscle use

Gianni Castiglione

Horses, for instance, rely on sustained and powerful muscular output. Their muscles contain more mitochondria per centimeter cubed than humans, generating more ATP and more ROS. Yet. horses resist the oxidative damage that would debilitate humans. Castiglione's lab found that a single point mutation in KEAP1 lowers its ability to inhibit NRF2, leading to enhanced antioxidant production and better protection from oxidative stress.

"Whenever the cysteines get oxidized, it changes the conformation of KEAP1," Castiglione said. "It doesn't release NRF2, it just alters how it's bound." This "hinge and latch" model allows one part of KEAP1 to unbind while the rest stays connected, keeping NRF2 partially engaged. As a result, newly produced NRF2 can accumulate in the nucleus and strengthen antioxidant defenses.

Birds show similar adaptations. "NRF2 mutations in humans can lead to cancer," Castiglione said. "And yet birds have all of these mutations and they're thriving — not despite them, but because of them."

These comparative studies suggest that evolution has repurposed the same molecular machinery for vastly different ends: adaptation in animals versus disease in humans.

When adaptation turns harmful

In humans, overproduction of NRF2 can cause disease. Constant activation supports cancer cell survival, enabling tumors to handle high metabolic activity and ROS stress.

"The high metabolic activity of a cancer cell is analogous to the high metabolic activity of a horse or bird," Castiglione said. "You get disease or adaptation depending on the context."

NRF2 overactivity can also promote atherosclerosis, a chronic inflammatory disease marked by plaque buildup in arteries. Yet, despite the risks, controlled activation of NRF2 remains an attractive target for treating diseases caused by oxidative stress, from neurodegeneration to diabetes.

"A lot of (Food and Drug Administration)–approved drugs target NRF2 because for a lot of different diseases, a commonality is oxidative stress," Castiglione said.

Preventing decline

Balancing oxidative stress begins with lifestyle. Both Phillips and McDonald emphasize that regular exercise and proper nutrition remain the most effective ways to preserve muscle health.

"Resistance training is king, and good nutrition is queen," Phillips said. "Until we invent the anti-aging pill, we're left with lifestyle choices."

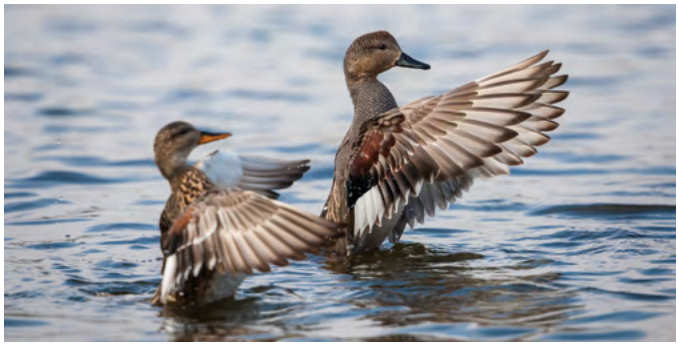
Sarcopenia is not inevitable. Resistance and strength training, combined with adequate protein and vitamin D intake, can slow muscle loss and preserve independence.

Evolutionary biology shows that species adjust ROS-regulating proteins based on their muscular demands, flying, sprinting or swimming. Humans cannot evolve that quickly, but exercise triggers epigenetic changes that enhance muscle function and antioxidant response even late in life.

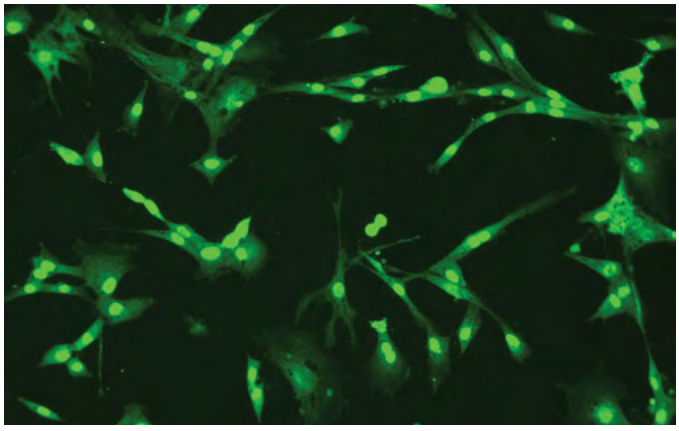
No amount of exercise will increase a human's lifespan. Instead, Phillips said, people should be focusing on increasing "healthspan."

McDonald agrees: "We should be realistic but optimistic. It may not be running a marathon again but getting out of the house to have lunch with friends."

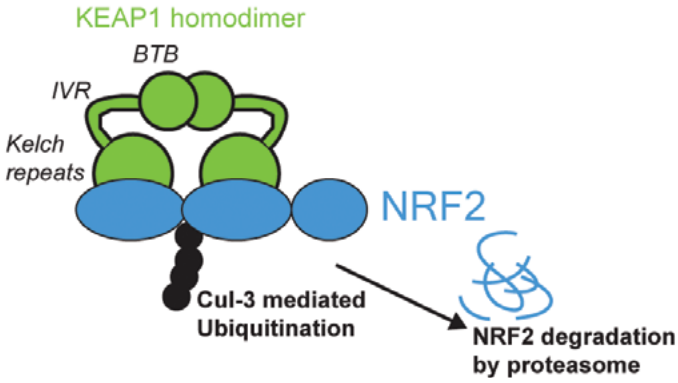
Inayah Entzminger is an ASBMB Today columnist.



Gianni Castiglione studies muscle aging in the gadwall duck, Mareca sterepera, which is common in Europe and North America.



Confocal microscopy showing the NRF2 transcription factor (green) in gadwall duck fibroblasts. These birds generate large amounts of reactive oxidative species, or ROS, during flight, and NRF2 resists cell damage from ROS accumulation. Photo credit: Gianni Castiglione



KEAP1 bound to NRF2. When the muscle is at rest, KEAP1 directs NRF2 to Cul-3 for ubiquitination and destruction. Oxidative stress alters the KEAP1 conformation, allowing still-bound NRF2 to promote gene transcription. Credit: Gianni Castiglione

From humble beginnings to unlocking lysosomal secrets

Monther Abu–Remaileh receives the ASBMB 2026 Walter A. Shaw Young Investigator Award in Lipid Research

By Anna Hu

Monther Abu–Remaileh

As a high school senior preparing for the Tawjihi, a high-stakes exam that would shape his college prospects, Monther Abu–Remaileh faced a major problem: his school was rarely open.

Born and raised in East Jerusalem, Abu–Remaileh's final year of high school coincided with the second Palestinian uprising. Teachers were delayed for hours at military checkpoints and classes were frequently canceled, he recalled.

The dedication of a few mentors kept his academic passion alive, especially one physics teacher, Yousef Alhroush. During that senior year, Alhroush would walk through mountainous regions from southern Palestine to East Jerusalem to avoid the checkpoints, then sleep in his classroom all week to help his students prepare.

Now mentoring his own trainees, Abu–Remaileh said he feels indebted to his former teachers.

"Seeing all of that, even 25 years after, and probably 100 years after, it's stuck in my brain," he said. "This is what they did for you; you need to help your students as well."

Their support helped him earn a scholarship to the Jordan University of Science and Technology and two graduate degrees at the Hebrew University of Jerusalem. From there, he joined Massachusetts Institute of Technology's Whitehead Institute as a postdoc and now leads a lab at Stanford University advancing the field of lysosomal lipid biochemistry.

For this work, he will receive the American Society for Biochemistry and Molecular Biology 2026 Walter A. Shaw Young Investigator Award in Lipid Research.

His lab focuses on how lysosomal dysfunction contributes to neurodegenerative disease. They began by studying conditions with known genetic causes but unclear biological mechanisms, systematically knocking out genes to see how they affected the lysosome. The team used mass spectrometry to measure changes in lipids, metabolites and proteins and found striking shifts in lysosomal lipids linking altered lipid metabolism to neurodegeneration.

One of the lab's key discoveries centers on the ceroid lipofuscinosis neuronal 5 gene, or *CLN5*. Loss of this gene causes a severe childhood neurodegenerative disorder, and variants in *CLN5* also increase Alzheimer's risk.

The lab found that *CLN5* produces a phospholipid, bis(monoacylglycero)phosphate or BMP, which is essential for lysosome metabolism. This finding reframes the lysosome as not only a recycling center but also as a site of molecular creation.

The team went on to identify phospholipase A2 group XV, or PLA2G15, an enzyme that degrades BMP. Knocking it out increased BMP levels and rescued severe neurodegeneration in a mouse model. The work, recently published in Nature, positions PLA2G15 as a promising drug target for lysosome-related diseases.

Abu–Remaileh will present his work on how dysfunction in lysosomal lipid catabolism drives neurodegenerative diseases at the ASBMB 2026 Annual Meeting.

As a teenager, Abu–Remaileh lived through political upheaval and in an East Jerusalem refugee camp for several years. Now, he mentors students who don't have all the privileges of their peers.

"It's important to diversify our faculty… because we have different perspectives," he said. "We see different types of people, and that these people also have potential."

Anna Hu is a research assistant at the Harvard School of Public Health and an ASBMB Today volunteer contributor.

Mentorship as immortality

Suzanne Barbour receives the ASBMB Sustained Leadership Award

By Inayah Entzminger

Suzanne Barbour

Suzanne Barbour found her first science mentor in her grandfather, a lab technician. As a child, she stood beside him in his chemistry lab and watched him work, increasing her desire to become a scientist like him. "If I hadn't had my grandfather, I wouldn't be talking to you today," Barbour said.

As an undergraduate at Rutgers University, Barbour said she often felt like "a number more than a name." Yet, a few professors stood out, providing mentorship and shaping her education in a way that made a difference.

"I want to make sure that there are people to support the next generation," Barbour said. "The right thing to do was to go into academia. And I never looked back."

Today, Barbour is a professor of cell biology, dean of the Graduate School and vice provost for graduate education at Duke University. She was also recently elected the American Society for Biochemistry and Molecular Biology secretary.

For her record of leadership and mentoring in academia, government and society, she will receive the 2026 ASBMB Sustained Leadership Award.

Barbour also served as a mentor for the National Institutes of Health Maximizing Opportunities for Scientific and Academic Independent Careers, or MOSAIC, program, launched in 2019 to support early-career scientists from underrepresented backgrounds. She helped participants navigate the realities of running a lab, including managing personnel and resolving conflicts.

Although the MOSAIC program ended in 2025, Barbour remains positive about its impact.

"It did what it was supposed to do," she said. "The folks who came through our (iteration) of MOSAIC, they know each other, they're networked together, they'll be colleagues and peers for life. And nobody can take that away from them."

Having served in many ASBMB leadership roles, Barbour said deeper involvement in the society gives members the opportunity to help people, advance science and sustain the organization.

"When we train our students and postdocs, it's a kind of immortality," Barbour said. "It's a legacy. And ASBMB gives you the opportunity to expand that legacy."

Takita Felder Sumter and Joseph Provost wrote in support of her nomination that Barbour has "a sustained record of not only mentoring women and students of color, but (also) providing a visible and motivating presence as an impressive female scientist."

At the 2026 ASBMB Annual Meeting, Barbour will speak about the second half of her career after closing her laboratory in 2013. Instead of mentoring a small group of students, she wanted to extend her leadership to graduate cohorts and early-career investigators.

After closing her lab, Barbour was program director in the division of molecular and cellular biosciences at the National Science Foundation, where she guided applicants through creating successful grant proposals.

"Despite the fact that I'm not at the bench anymore, I think I have even more influence on developing people's careers now," Barbour said.

Inayah Entzminger is an ASBMB Today columnist.

Redefining excellence to drive equity and innovation

Donita Brady receives the ASBMB Ruth Kirschstein Award for Maximizing Access in Science

By Jessica Desamero

Donita Brady

As an undergraduate, Donita Brady had limited access to hands-on research and little sense of where a chemistry degree might take her. As she advanced in research, she sometimes struggled with a lack of representation and shared identity in science. Now, Brady helps expand access for scholars from underrepresented backgrounds. For her work expanding access to science, Brady has been named the recipient of the 2026 ASBMB Ruth Kirschstein Maximizing Access in Science Award.

At the University of Pennsylvania, Brady said she takes pride in the school's pathway programs she helped develop. These initiatives expose students to research early and provide an opportunity for advanced consideration into Penn's biomedical Ph.D. programs.

She also emphasizes Penn's holistic reviews for applicants to its summer, postdoctoral and Ph.D. programs. To redefine recruitment, Brady developed and implemented a rubric-based review system, an evidence-driven approach to selection. The rubric has made Penn's recruiting practices more equitable and has been key in sustaining strong matriculation rates among underrepresented scholars.

"We are redefining the future of diversity, equity, and inclusion through the lens of excellence," Brady said. "To realize this vision, we must reconsider how we define excellence itself and pursue it in ways that advance the frontiers of research and medicine."

For the past decade, Brady has led the Office of Research Trainee Affairs, serving as both assistant dean for research training and faculty director. She also serves as associate director of professional opportunities and engagement at the Abramson Cancer Center and vice chair for strategic access and community enrichment in the department of cancer biology. She has mentored countless scholars and consistently fosters a culture grounded in equity, empowerment and belonging.

Now a presidential associate professor of cancer biology, Brady's research focuses on the role of metal micronutrients in cancer cell growth. Her innovative work positions her as a scientific leader, but what sets her apart is her dedication to dismantling systemic barriers that limit participation in research careers.

In their nomination letter, Penn colleagues George M. Burslem and Kristen W. Lynch wrote, "Dr. Brady is a trailblazing scientist and an extraordinary advocate for inclusion and equity whose contributions have transformed the scientific landscape — not only through her groundbreaking research into the roles of copper signaling in cancer biology, but also through her deeply rooted and sustained commitment to mentoring and empowering historically excluded groups in science."

Brady served as co-chair of the 2025 ASBMB Annual Meeting and will speak at the 2026 Annual Meeting. She will discuss programs and strategies that reimagine admissions, mentoring and recruitment to strengthen the scientific training pipeline and build resilient pathways for future scientists.

"By fostering persistence and redefining excellence, we can build a research ecosystem that drives both equity and innovation," she said. "And we need to continue to define excellence such that it's broad enough to capture anyone who finds themselves without equal opportunity to achieve their dreams."

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

Defining a 'crucial gatekeeper' of lipid metabolism

George Carman will receive the Herbert Tabor Research Award

By Courtney Chandler

George Carman

George Carman, board of governors professor at Rutgers University and founding director of the Rutgers Center for Lipid Research, first met Herbert Tabor at an ASBMB meeting in 1992. When Carman became an associate editor with the **Journal of Biological Chemistry**, or JBC, he began working closely with Tabor during Tabor's tenure as editor-in-chief of JBC, frequently receiving mentorship.

Carman has been awarded the ASBMB's Herbert Tabor Research Award for his outstanding accomplishments and contributions to the scientific community — a full-circle moment honoring his mentor.

"This respect from one of science's most famous figures was deeply meaningful," he said of his time working with Tabor. "I am both humbled and profoundly gratified to receive this prestigious recognition."

Carman's research focuses on phospholipids — fat molecules with a hydrophilic head and hydrophobic tail that form the foundation of cellular membranes and play key roles in signaling and metabolism. Using the yeast *Saccharomyces cerevisiae*, which shares genetic similarities with humans, his work has revealed how these lipids are synthesized, regulated and linked to disease.

As essential components of membranes and active players in cell signaling and metabolism, lipids must be tightly regulated — Carman's research focuses on uncovering the molecular mechanisms behind this balance.

"The imbalance of lipid metabolism is a central driver for major diseases, including obesity, diabetes and heart disease," Carman said.

Carman identified and characterized the enzyme phosphatidic acid phosphatase, or PAP, a metabolic gatekeeper, which directs lipid building blocks either toward membrane synthesis or fat storage. His work shows that loss of PAP causes excess membrane production, seen in cancer, while excess PAP drives fat accumulation linked to conditions such as obesity, diabetes and heart disease.

Carman also helped uncover the biochemical functions of human lipin proteins, identifying them as PAPs. His yeast studies have offered critical insights into their regulation, enzymatic activity and potential roles in disease.

"The ultimate goal of our work is to understand how to 'fine-tune' this enzyme's activity," Carman said. "By deciphering its genetic and molecular control, we aim to provide the foundational knowledge needed to develop new strategies for controlling lipid metabolism and combating these pervasive, lipid-based human diseases."

In his nomination letter, Edward Dennis of the University of California, San Diego, emphasized how Carman's "seminal contributions to biochemistry and molecular biology" have "defined the details of phospholipid synthesis and numerous important signaling events in yeast." Dennis also noted Carman's "rigor and enthusiasm" as well as "outstanding devotion" as an associate editor of JBC and the **Journal of Lipid Research**.

Carman will give a talk at the 2026 ASBMB Annual Meeting focusing on the function and regulation of PAP, and its potential as a therapeutic target for combating fungal infections.

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

Mining microbes for rare earth solutions

Joseph Cotruvo, Jr., receives the ASBMB Mildred Cohn Young Investigator Award

By Elizabeth Stivison

Joseph Cotruvo, Jr.

Joseph Cotruvo, Jr., is seeking to understand how cells handle metals. But along the way, he patented revolutionary techniques for purifying and detecting rare earth elements, even helping U.S. national security. The inventions stem from his discovery of lanmodulin, a protein that binds particular rare earth elements with remarkable specificity.

Rare earth elements are elements 21, 39 and the lanthanides, numbers 57–71, which have the same +3 charge and very similar sizes. The similarity makes isolation difficult, which in turn is a hurdle for society as specific rare earth elements are used in batteries, electric motors, magnets and other technology. The U.S. depends heavily on imports, so domestic purification could ease both economic and national security concerns.

Cotruvo "has made great strides toward implementing lanmodulin-based constructs for rare earth element extractions and separations, (and) as fluorescent sensors for rare earths," wrote J. Martin Bollinger, a Penn State professor who nominated him. "The value of Cotruvo's work is profound."

Cotruvo's discoveries also hold promise for environmental cleanup. "The Mount Everest of lanthanide remediation is selectivity," wrote Harvard University professor Daniel Nocera in his letter of support. Cotruvo's work on lanmodulin's selective binding may be solving this issue, too.

Cotruvo's fascination with chemistry was inspired by his father, a chemist at the Environmental Protection Agency. As an undergraduate, Cotruvo became intrigued by bioinorganic chemistry — how metals interact with biological systems — and worked with Ed Stiefel, who studied using bioinorganic chemistry and bacteria to clean up oil spills.

"The combination of that work and seeing how my dad used chemistry at the EPA showed me that chemistry can be used to solve some really important problems," Cotruvo said.

He went on to conduct graduate and postdoctoral research. "The unifying feature," he said, "was selectivity of binding of metals to proteins, and the question of 'how does an organism manage the metals?'"

When starting his own lab seeking to study this question, he was intrigued by *Methylobacterium extorquens*, a bacterium that uses specific lanthanides. "I didn't know anything about lanthanides or the bacteria," he said, but he could tell it was a rich biochemical problem. "There are 17 rare earths, but the organism could only use a subset. Why and how? What mechanisms exist to select between them?"

During his purification of a lanthanide-dependent enzyme, Cotruvo noticed another protein that came out along with it. It contained EF-hand motifs, structural features common in calcium-binding proteins like calmodulin. He found that this protein, which he named lanmodulin, bound specific lanthanides.

"No one had seen a protein so selective for lanthanides," Cotruvo said. He showed that selectivity comes from disordered regions of the protein folding into more stable conformations when bound to the correct metal ion.

In her letter of support, Northwestern University professor Amy Rosenzweig wrote of Cotruvo, "His work epitomizes the combination of innovation, creativity and impact that should be recognized by the Mildred Cohn Young Investigator Award."

Cotruvo will discuss his discovery of lanmodulin and its industrial applications at the 2026 ASBMB Annual Meeting.

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

Defining JNKs: Targets for drug discovery

Roger Davis receives the Bert and Natalie Vallee Award in Biomedical Science

By Vanshika Patel

Roger Davis

"If you are going to be successful as a scientist, you want to have fun while you do it," Roger Davis said, reflecting on the curiosity and enthusiasm that have fueled his four decades in research. After earning his Ph.D. at the University of Cambridge, Davis moved to the United States and has spent the past 40 years as a professor at the University of Massachusetts Chan Medical School.

A pioneer in defining Jun N-terminal kinase, or JNK, Davis is being honored with the American Society for Biochemistry and Molecular Biology Bert and Natalie Vallee Award in Biomedical Science, recognizing his discovery of this signaling protein and the decades of insight that followed.

In his letter of support, Michael P. Czech, also a professor at UMass Chan, wrote, "The discovery of JNK by Davis has had enormous impact, leading to him being the most highly cited scientist in all fields subsequent to the discovery… Honoring the discovery of JNKs is deserving… and will be well received by basic biomedical scientists across the globe."

Davis's lab is best known for studying stress signaling. "We came into this from basic biochemistry and protein phosphorylation, identifying sites of phosphorylation and mutating them to understand function," he said. A strange proline-directed phosphorylation site with two prolines caught their attention, leading to further work exploring MAP kinases and eventually the initial cloning of the Jun kinases, followed by work on p38 MAP kinases.

Initially, scientists thought JNK was oncogenic, but Davis's work revealed that it can also act as a tumor suppressor, depending on the tumor microenvironment.

"It is exciting that there may be a way to use the role of JNK in the tumor microenvironment to develop therapies against cancer," Davis said.

In other diseases, JNK signaling affects communication among organs such as the brain, liver, and fat tissue, influencing satiety, metabolism and inflammation in type 2 diabetes.

"There's this intricate crosstalk," Davis said. "And we're just beginning to figure out how JNK fits into it."

Davis and his team showed that JNK is not a single protein but a family encoded by three genes, diversified through a process called alternative splicing. In some tissues, this can produce up to 10 isoforms.

At the 2026 ASBMB Annual Meeting, Davis will present his work showing that swapping exon 7a or 7b changes the shape of JNK's docking site, determining which proteins it interacts with and ultimately altering the signaling output.

"We have really good mouse models to study where we can control the splicing genetically and force the kinase in specific cell types to include only exon 7a or 7b," Davis said. "If you can more precisely target the spliced isoform that's mediating the biology you're studying, I think we may have more selective and better drugs."

Despite his many awards and honors, Davis is quick to credit his trainees. "Very little of the work was done by me," he said. "It's the students and postdocs. They did the science, and many have gone on to lead labs of their own."

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

Mapping proteins, one side chain at a time

Roland L. Dunbrack Jr. receives the ASBMB DeLano Award for Computational Biosciences

By Jessica Desamero

Roland L. Dunbrack Jr.

As a college student, Roland L. Dunbrack Jr. began his research career at the lab bench, but it was the numbers — not the pipettes — that captured his imagination.

He loved how computation delivered results fast and how debugging a stubborn code felt like solving a puzzle. That curiosity became a calling. Over the next several decades, Dunbrack reshaped how scientists model protein structure.

As a Ph.D. student, Dunbrack made a breakthrough in understanding how proteins fold. While fitting molecular dynamics parameters for the amino acid proline and developing an algorithm for protein structure prediction, he discovered that a protein's backbone conformation, especially its dihedral angles, determines the positioning of its side chains.

"It turned out there was a very clear pattern to it, that as you changed each of the backbone dihedrals, the populations of the three chi1 rotamers of each amino acid changed in a similar way," Dunbrack said.

He went on to build the first backbone-dependent rotamer library, a reference that predicts likely side-chain positions based on backbone geometry.

"This work is effectively a Ramachandran map for protein side chains, making it fundamental to understanding protein conformations," Helen M. Berman wrote in her nomination letter. "And (it) is included in some textbooks on protein structure and computational biology."

As a postdoctoral fellow, Dunbrack refined the library using Bayesian statistics, boosting its accuracy and utility. Since then, labs worldwide have relied on it to predict side-chain placement, analyze protein folds and design new proteins. "All the way up through the beginning of AlphaFold, the rotamer library played a big part in a lot of protein structure prediction and protein design programs that people developed," he said.

Among those users was David Baker's lab, later recognized with the Nobel Prize in chemistry.

"I think it was pretty key to the first design of a de novo design of a protein," Dunbrack said.

For his pioneering computational tools that have advanced structural biology, Dunbrack has received the 2026 ASBMB DeLano Award for Computational Biosciences.

Currently, Dunbrack is a professor at Fox Chase Cancer Center, where he leads the molecular modeling facility. His team builds structural models that help researchers visualize how proteins function and how they can be targeted in disease.

Recently, his group has focused on analyzing more than 10,000 kinase structures in the Protein Data Bank and using AlphaFold to model all 493 human kinase domains, work that could reveal how these key drug targets shift between active and inactive states to guide cancer therapy development.

"We provide an analysis of proteins that are targets for drug development, and we hope that what we provide helps people either in industry or academia to develop drugs," Dunbrack said.

At the 2026 ASBMB Annual Meeting, Dunbrack will discuss structural bioinformatics in the AlphaFold era, his rotamer library's impact, his team's work on cancer-related proteins, and how experimental data refine predicted models. He will also speak about his advocacy for the LGBTQIA+ community.

"(I want) to make sure young queer people know there are experienced scientists who are out in our jobs and still have our careers," he said. "We're still successful and respected."

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

Creating change in biochemistry education

Pamela Mertz receives the ASBMB William C. Rose Award for Exemplary Contributions to Education

By Inayah Entzminger

Pamela Mertz

When Pamela Mertz walks into any room — her classroom at St. Mary's College of Maryland or an American Society for Biochemistry and Molecular Biology meeting focused on education — she carries her enthusiasm for science with her. Mertz traces that passion back to her 11th-grade chemistry teacher, Mr. Nolt, who inspired her to become a first-generation college student — and the only scientist in her family.

The American Society for Biochemistry and Molecular Biology has named Mertz the 2026 ASBMB William C. Rose Award for Exemplary Contributions to Education recipient, recognizing her dedication to teaching biochemistry and molecular biology.

A longtime mentor to undergraduate scientists, Mertz has served 12 years on the ASBMB Student Chapters Committee, including the past five as chair.

Mertz contributed to several National Science Foundation education-focused programs, including service as an associate director for BioMolViz, culminating in an open educational resource on teaching biomolecular visualization.

"I always liked school; I always liked learning. I appreciated all the amazing teachers I had, and teaching was something I was drawn to," Mertz said.

Early in her faculty career, Mertz developed an interest in pedagogy. At the 2011 ASBMB Transforming Undergraduate Education in Molecular Life Sciences, or TUEMLS, meeting, she said she "found her people."

TUEMLS is a biennial meeting dedicated to improving how professors teach biochemistry and molecular biology. Mertz helped organize the 2023 TUEMLS meeting at Suffolk University and served as co-principal investigator on a National Science Foundation grant to expand access to the conference.

"The passion and connections with other (educators) helped me think about my teaching," Mertz said.

For the past 25 years, Mertz has been a professor of chemistry and biochemistry at St. Mary's College of Maryland, where she also chairs the health sciences advisory committee. She led the effort to earn ASBMB accreditation in 2016, ensuring her program met national standards for excellence in biochemistry and molecular biology education.

"It's important for institutions like mine that are small and public," Mertz said. "To be able to tell prospective students and their parents that you have this nationally recognized program, it's not insignificant." To Mertz, teamwork is key in creating the best outcomes.

At the 2026 ASBMB Annual Meeting, Mertz will discuss how professional communities can drive transformation in science education and research.

"Pam is not simply a participant, but a leader who helps prepare assignments, facilitates workshops and develops laboratories," John Tansey of Otterbein University wrote in her nomination letter. "I have found her to be a creative and committed leader in the biochemistry education community."

Inayah Entzminger is an ASBMB Today columnist.

Redefining lipid biology from droplets to ferroptosis

James Olzmann receives the Avanti Award in Lipids

By Elisabeth Marnik

James Olzmann

When most people think of lipids, they think of fat. James Olzmann thinks of life itself — how cells store energy, maintain balance and decide when to die. His discoveries have reshaped how scientists understand lipid biology, revealing how these molecules drive health, disease and survival.

Impressive. Innovative. A dedicated mentor and lifter of others. These are the words colleagues use to describe James Olzmann, the 2026 recipient of the Avanti Award in Lipids from the American Society for Biochemistry and Molecular Biology.

Olzmann, a professor at the University of California, Berkeley, has helped redefine how researchers think about lipids. His work has advanced knowledge of lipid droplets, lipid metabolism and ferroptosis — a regulated form of cell death defined by the accumulation of toxic lipid peroxides.

In his nomination letter, Jeremy Thorner, distinguished professor emeritus at UC Berkeley, wrote that Olzmann has an "unerring ability to identify important questions and to have the courage and creativity to develop new interdisciplinary strategies to tackle these questions."

Early in Olzmann's career, lipid droplets were seen as little more than blobs in the cell. His work helped reveal them as dynamic organelles with critical roles in lipid and energy balance. To determine which proteins localize to these droplets, Olzmann developed a proximity labeling proteomics approach — a technique that uses enzymes to tag proteins near specific cellular structures.

He next built a functional genomics platform to identify genes that alter lipid storage within droplets. The resulting datasets became field standards, shared openly through databases such as DropletProteome.org and CRISPRLipid.org.

Olzmann's discoveries also extend beyond lipid droplet biology. Recently, his lab discovered ferroptosis suppressor protein 1, or FSP1, which protects cells from oxidative lipid damage and ferroptosis. The group later showed that FSP1 helps cancer cells resist death and identified potential inhibitors that could sensitize tumors to ferroptosis, opening new possibilities for cancer therapy.

Beyond his scientific impact, Olzmann is celebrated as a mentor, collaborator and advocate for equity and inclusion. At UC Berkeley, he serves as equity chair in his department, and he co-led a Bridges to the Doctorate program that creates research pathways for students from historically excluded backgrounds.

"(Olzmann) gave me wonderful practical and professional advice that I still use to this day and pass on to junior (principal investigators) I now mentor at UTSW," W. Mike Henne, an associate professor at the University of Texas Southwestern Medical Center, wrote in his letter of support. "This is one of (Olzmann's) many strengths — he is absolutely committed to mentoring others and helping others succeed."

In a letter of support, Ron Kopito, professor of biology at Stanford University, wrote that Olzmann "is a terrific example of a scientific leader dedicated to advancing cell biology and building a diverse and inclusive scientific community."

At the 2026 ASBMB Annual Meeting, Olzmann will present his work on how cells maintain lipid quality control, the interconnected processes that prevent, detect and repair lipid damage to preserve cellular function and viability.

Elisabeth Adkins Marnik is the Director of Science Education & Outreach at the MDI Biological Laboratory in Bar Harbor, Maine, and an ASBMB Today volunteer contributor.

Decoding how bacteria flip host's molecular switches

Kim Orth receives the Earl and Thressa Stadtman Distinguished Scientists Award

By Courtney Chandler

Kim Orth

Kim Orth's lab studies bacterial pathogens that act as "alien invaders," taking over a host by flipping molecular switches. "My job is to figure out what the switches are and how the bacteria flip them so that I can understand how the host system has been manipulated," Orth said. For her work, Orth has won the American Society for Biochemistry and Molecular Biology's 2026 Earl and Thressa Stadtman Distinguished Scientist Award, which recognizes distinguished scientists who have made outstanding achievements in basic research.

Orth, a professor at the University of Texas Southwestern Medical Center and an investigator at the Howard Hughes Medical Institute, studies the molecular effect of bacterial virulence factors on host cells — revealing insights into pathogenesis and eukaryotic biology.

As a postdoc, Orth investigated signal transduction not in bacteria but in eukaryotic cells — until she hit a roadblock because existing technology couldn't capture the detail she needed. She pivoted to studying *Yersinia pseudotuberculosis*, a relative of the plague-causing bacterium *Y. pestis*. When she discovered that the secreted protein YopJ modified host proteins to enhance bacterial survival, she was hooked.

"I saw this new world of how bacteria were impinging on the signal transduction I had been studying and thought it was really cool," she said.

Orth has since shown how bacterial effectors manipulate eukaryotic proteins through posttranslational modifications. Her groundbreaking work on *Vibrio parahaemolyticus*, a bacterium that causes gastroenteritis, revealed that the protein VopS hijacks human cells by attaching the metabolite adenosine monophosphate, or AMP, to Rho guanosine triphosphatase proteins — a process her lab termed AMPylation. This disrupts binding of partner proteins that control cell shape and signaling, dampening the immune response. This work identified AMPylation as a posttranslational modification that bacterial effectors use to target eukaryotic proteins.

Orth then explored AMPylation in eukaryotes, showing in *Drosophila* that a Fic-domain enzyme homologous to VopS adds AMP to inactivate the ER chaperone protein BiP under normal conditions, and removes AMP during ER stress to boost protein folding. Extending this research to mice, her team found that loss of Fic, and thus proper AMPylation, heightened pancreatic stress responses. Recent findings linked Fic mutations to diseases including neonatal diabetes, and Orth's Fic-mutant mouse mirrored aspects of this condition. Together, Orth's work shows that AMPylation is a fundamental, conserved regulatory mechanism across evolution.

In her nomination letter, Margaret Phillips of the University of Texas Southwestern Medical Center wrote that Orth's "pioneering biochemical research has reshaped our understanding of microbial pathogenesis and key aspects of eukaryotic cell biology."

Orth said she was shocked and elated to receive this award, as her research builds on Stadtman's 1960s discovery of proteins modified with AMP in *E. coli*.

"What my lab has studied for the past 15 years aligns with what (they) discovered," she said. "It's the most incredible honor to receive the Stadtman award."

Orth will present her collective work on AMPylation at the 2026 ASBMB Annual Meeting.

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

Chemistry meets biology to thwart parasites

Margaret A. Phillips receives the Alice and C. C. Wang Award in Molecular Parasitology

By Jay Thakkar

Meg Phillips

Growing up, Margaret (Meg) Phillips was inspired by her father, a radiation oncologist and academic physician whose passion for research sparked her own curiosity about science. "He was an academic… interested in research, so growing up that was something that inspired me (toward) science and research," Phillips said.

Today, Phillips is a professor of biochemistry at the University of Texas Southwestern Medical Center, where she focuses her research on the biochemistry and drug discovery aspect of parasitic protozoa. For her work, she will receive the Alice and C. C. Wang Award in Molecular Parasitology.

As an undergraduate at the University of California, Davis, Phillips discovered her fascination with the chemistry–biology interface.

"When I was in school it was only the beginning of the molecular biology revolution, but I really liked enzymology," Phillips said. "I liked to understand how an enzyme catalyzed a reaction, so that was partly the chemist in me."

After earning her bachelor's degree in biochemistry, she worked for a small diagnostic company developing kits to measure drug levels in patients. That experience strengthened her interest in how biochemical processes could be targeted for therapeutic benefit. She then pursued a Ph.D. at the University of California, San Francisco, joining C. C. Wang's lab.

"At the time many biochemistry departments were very molecular biology focused," Phillips said. "But I really wanted to study proteins so, that's what drove me to pharmaceutical chemistry because it was more mechanistic, more chemistry."

Wang's mentorship left a lasting impact on Phillips. "He was a really great scientist and an outstanding mentor," she said. That influence came full circle when Phillips received the 2024 American Society for Biochemistry and Molecular Biology Herbert Tabor Research Award, which honors her dedication to research and mentorship.

"I cannot think of a more deserving candidate than Meg Phillips for this award," Vernon Carruthers wrote in Phillips' nomination letter. "C. C. Wang had an enormous impact on Meg, not only when she was a Ph.D. student in his lab, but also throughout her career as a caring mentor and supporter."

Phillips' lab focuses on understanding the metabolism of parasites such as *Trypanosoma brucei*, which causes African sleeping sickness, and *Plasmodium falciparum*, which causes malaria. Her work on malaria has focused on DHODH, a key enzyme in pyrimidine synthesis, where she has exploited DHODH in a target-based drug discovery approach to identify and optimize molecules that inhibit this enzyme for the treatment of malaria.

At the 2026 ASBMB Annual Meeting, Phillips will present her group's latest findings on inhibitors of dihydroorotate dehydrogenase, or DHODH.

Her group's structure-based design has improved compound potency from the micromolar to subnanomolar range, a crucial step toward clinical development. One early success was DSM265, a promising DHODH inhibitor for malaria that advanced to clinical trials. Although testing was later halted due to toxicity, the experience paved the way for ongoing work in her lab.

"Science is a very important path to take," Phillips said. "It has the potential to contribute so much to society and make a better world for everybody."

Jay Thakkar is a researcher who specializes in computer-aided drug design and discovery, and an ASBMB Today volunteer contributor.

Finding my way back to science AND FORWARD INTO EDUCATIONAL RESEARCH

By Christin Monroe

Assistant professor Christin Monroe performs a chemistry experiment showing the decomposition of potassium chlorate while undergraduates Rebecca Lynch and Michael Vittum observe at Landmark College in 2023. Credit: Courtesy of Christin Monroe

Yet another failed attempt at protein expression.

I stared at the results of a gel that once again showed no sign of my target protein. About three years into graduate school, it felt like nothing in my experiments was working. To combat the growing frustration, I began seeking opportunities outside the lab, and that's when I discovered science outreach — something that actually worked and allowed me to share my passion for science with others. Those experiences sparked a new sense of purpose and ultimately set me on an entirely different path.

I began volunteering for every outreach event I could — more than a dozen in one year — and even designed and ran a month-long summer program for high school students. Those experiences opened new doors. Through networking and mentorship, I secured a teaching fellowship focused on inclusive instruction, a science communication fellowship, and a role as a career services ambassador for graduate students. At the time, I didn't see the thread tying all this together: that I was building a foundation in mentoring, communication and education. I just knew I was energized by it.

Balancing these interests with my research was challenging. Self-doubt crept in often; was I losing focus, or redefining success? A mentor once advised me to think about skill-building rather than simply chasing what I found fun. That perspective helped me reframe my choices and focus on transferable skills that aligned with my values.

While I was "all but dissertation," or ABD, — meaning I had completed all my Ph.D. requirements except for the dissertation — I accepted a position as an educational counselor and math/science expert for Upward Bound. The role allowed me to merge my love of science with mentoring and teaching first-generation college students — and, unexpectedly, it set the stage for my future career. Encouraged by colleagues to make use of my chemistry background, I began adjunct teaching at the college where I worked. That combination of advising and teaching experience uniquely positioned me for my current role as a teaching professor at a college serving neurodivergent students.

Looking back, my path into academia was far from traditional, but it was exactly what I needed. Over time, I've developed a passion for researching and addressing barriers in education — work that lets me apply my scientific training to improve access, inclusion and belonging in STEM. These interests have guided my teaching and continue to shape my next professional direction.

Now, as I prepare to begin a new role as an educational research associate at the University of Illinois Urbana-Champaign Center for Innovation in Teaching & Learning, I find myself coming full circle — combining my passion for research with my love of teaching and learning. This next chapter feels like the natural extension of every step that came before it, a space to continue exploring, collaborating and creating programs that make education more inclusive for all learners.

Christin B. Monroe is an assistant professor of chemistry and an Access to Innovative Education in Science, Technology, Engineering and Mathematics co–primary investigator at Landmark College.

The tortoise wins: HOW SLOWING DOWN SAVED MY PH.D.

By Amy Bounds

Just as I was becoming a Ph.D. candidate, my body forced me to shift my mindset from a speedy hare to a slow and steady tortoise.

Burning out as the hare

Early in graduate school, I prided myself on keeping up with the whirlwind pace. I pushed through late nights and early mornings, convinced that speed and productivity were the only paths to success. The evening after I passed my candidacy exam, I should have been celebrating months of hard work, but instead, I felt a tightness in my back and chest. I brushed it off as fatigue, but weeks later, the discomfort persisted. After multiple doctor visits, chest X-rays and blood tests, I was told nothing was wrong.

Months dragged on, and the pain continued. I developed jaw pain known as temporomandibular joint, or TMJ, disorder and recurring stomach issues. I stopped seeing friends, abandoned hobbies and struggled to work at the bench.

Finally, one doctor asked if I was often stressed. I laughed and told them I was a biochemistry Ph.D. candidate. Stress was my life. That conversation revealed the culprit: stress-induced irritable bowel syndrome, or IBS.

I felt defeated, like I couldn't cut it as a scientist. Graduate students were "supposed" to be stressed, but my body was screaming at me to slow down. So, I did — and the outcome surprised me.

Becoming the tortoise

With the support of my principal investigator, I began to prioritize a more balanced workflow in the lab. Mondays became planning days. I outlined experiments, broke tasks into daily chunks and shifted experiments when days became full. Delaying experiments was one of the hardest changes. I had a habit of trying to do everything immediately, but waiting a week rarely mattered in the long run.

Knowing that deadlines triggered my IBS flare-ups, I prepared early instead of pushing myself to the brink. I broke big deadlines into smaller, manageable steps, such as writing a first draft, storyboarding a slide deck or drafting bullet points for a paper.

Slowly, my mindset shifted. I stopped overbooking my schedule. Instead, I used incubation times to read papers, maintain my lab notebook and take walks when my focus lagged. I even carved out time for therapy each week.

After a year of retraining my habits, I am more productive than ever. I make fewer mistakes and generate more reliable data. Time I once used for redoes or extra experiments is devoted to planning, reading and writing. I prioritize my weekends and evenings. I play soccer again and even train for triathlons. I have energy again — for myself, my friends, my family and my thesis.

I still experience flare-ups, but now I know how to manage them. I've learned that to do well, you must first be well.

As Aesop's fable of the tortoise and the hare reminds us, "the race is not always to the swift." In graduate school, I've learned that the race is won by those who slow down enough to prioritize their well-being.

Amy Bounds is a biochemistry Ph.D. candidate in the Hoppins lab at the University of Washington. Amy studies how mitochondria fuse to form dynamic networks in our cells. She is an ASBMB Today volunteer contributor.

Amy Bounds poses at the finish line of the 2023 Seafair triathlon in Seattle, Washington. Since 2023, she has raced every summer and plans to run her first half Ironman in 2026. Credit: Courtesy of Amy Bounds

Attie named honorary professor

Alan Attie has been named the Henry and Annrita Lardy Professor of Biochemistry by the Wisconsin Alumni Research Foundation, or WARF. He is one of 10 who received this honor in 2025. This award includes $100,000 in research funding and recognizes faculty who have made major contributions to the advancement of knowledge through their research endeavors and their teaching and service activities.

Attie is a professor of biochemistry at UW–Madison. His lab investigates the genetic and biochemical mechanisms underlying metabolic diseases, including β-cell function, insulin secretion, lipid metabolism and gene–diet interactions. Attie is a fellow of the American Association for the Advancement of Science and a former Shaw Scholar. He has also received an Established Investigator Award from the American Heart Association. Attie serves as an associate editor for the Journal of Lipid Research.

Cadichon honored for academic achievement

Melodie Cadichon is the inaugural recipient of the Dr. Henry Teoh Award for Outstanding Collegiate Science and Technology Entry Program Graduating Senior, which recognizes exceptional achievement, leadership and promise in a student. Teoh, founder and former director of the CSTEP, created this award to support outstanding seniors. She is being honored for enhancing connections between CSTEP and the American Society for Biochemistry and Molecular Biology Student Chapter at the State University of New York at Old Westbury. Cadichon will receive a $1,000 honorarium.

Cadichon, a biochemistry undergraduate at SUNY Old Westbury, is president of the ASBMB Student Chapter at SUNY Old Westbury. She conducts research with Youngjoo Kim, an associate professor of chemistry and physics, on epidermal growth factor receptor as a potential cancer drug target. Cadichon presented her research at the 2025 ASBMB annual meeting. In fall 2025, she will pursue an advanced degree in biochemistry and molecular biology at Penn State University.

Castiglione and Ingolia win Keck Foundation grants

Gianni Castiglione, with several colleagues at Vanderbilt University, and Nicholas Ingolia received at least $1 million from the W.M. Keck Foundation to fund their research. The foundation supports projects that are distinctive and novel in their approach, as well as high-risk with the potential for transformative impact.

Castiglione is an assistant professor of biological sciences, ophthalmology and visual sciences at VU and a member of Vanderbilt's Evolutionary Studies Initiative. His lab explores new directions in aging research by collaborating with experts from a wide range of disciplines across the Vanderbilt campus. Castiglione's grant will help further research in reverse-engineering the life span of birds. He hopes to uncover biological mechanisms behind exceptional longevity that could one day help safely extend the lives of humans.

Ingolia is a professor of molecular and cell biology at the University of California, Berkeley. His lab studies how cells control the translation and stability of messenger RNA in the cytosol, and how this regulation fulfills important biological functions. Ingolia's grant will be used toward the study of systematic testing of sequence–function relationships in intrinsically disordered proteins.

Cedeño–Rosario and Kaweesa win research award

Luis Cedeño–Rosario of the University of Utah Spencer Fox Eccles School of Medicine and Elizabeth Kaweesa of the University of Illinois Chicago received the Dr. Eddie

Mendez Scholar Award from the Fred Hutch Cancer Center. The award honors outstanding early-career scientists studying cancer, infectious disease and basic science and is named after Méndez, who was a physician–scientist at Fred Hutch.

Cedeño–Rosario is a postdoctoral researcher in the Jared Rutter lab at the University of Utah. His work explores how cancer cells alter their internal wiring to support unchecked growth and resist treatment, uncovering how shifts in metabolism can give tumors a survival advantage. In 2020, he won the Tony Quinn Inclusive Excellence Award, which honors a scientist who is committed to mentoring students and increasing the participation of underrepresented students in science. Cedeño–Rosario earned his Ph.D. at the University of Toledo.

Kaweesa is a postdoctoral research associate in the Joanna E. Burdette lab, where she is exploring how natural compounds might be used to treat high-grade serous ovarian cancer, an aggressive malignancy often diagnosed in its later stages. Kaweesa was recently featured in an ASBMB Today article. She is a National Institutes of Health Institutional Research and Academic Career Development Award recipient and previously received an NIH Maximizing Opportunities for Scientific and Academic Independent Careers award. Kaweesa earned her Ph.D. at the University of Florida.

Doudna wins Priestley Medal

Jennifer Doudna has been awarded the prestigious 2026 Priestley Medal for her groundbreaking work on RNA molecules with enzymatic functions by the American Chemical Society. This medal is the highest honor given by ACS and recognizes distinguished scientists in chemistry. Specifically, ACS cited her work on ribozyme function, Dicer, double-stranded RNA processing and CRISPR–Cas9 gene editing. Doudna will receive a $20,000 research grant.

Doudna is a faculty scientist at the Lawrence Berkeley National Laboratory, founder of the Innovative Genomics Institute, a professor at UC Berkeley and an investigator with the Howard Hughes Medical Institute. Her lab investigates RNA as it forms a variety of complex globular structures, some of which function like enzymes or form functional complexes with proteins.

Previous awards include the 2025 National Medal of Technology and Innovation, presented by former President Joe Biden in January for her work on CRISPR–Cas9 gene editing, as well as the 2020 Nobel Prize in Chemistry alongside Emmanuelle Charpentier. She is a member of the National Academy of Sciences, the National Academy of Medicine, the National Academy of Inventors and the American Academy of Arts and Sciences. Doudna is also a Foreign Member of the Royal Society and has received numerous other honors, including the Breakthrough Prize in Life Sciences, the Japan Prize, the Kavli Prize, the LUI Che Woo Welfare Betterment Prize and the Wolf Prize in Medicine. She will formally accept the award and deliver remarks at the ACS Spring 2026 conference.

Simcox and Gisriel receive mentoring award

Judith Simcox and Christopher Gisriel received the Distinguished Faculty and Staff Postdoc Mentoring Award from the University of Wisconsin–Madison Postdoctoral Association. Given annually, the award recognizes faculty and staff who contribute their time, knowledge, energy and enthusiasm to mentoring postdocs in their labs. The awardees were honored at the association's Celebration of Postdoc Excellence in May.

Simcox is an associate professor of biochemistry at UW–Madison. The Simcox lab studies plasma lipids, which largely serve as signaling molecules to regulate cardiovascular disease and Type 2 diabetes. She and her team are striving to identify novel lipids and determine how their production is regulated and how they function in metabolic disease. Simcox was named an Emerging Investigator by the University of Illinois

Chicago in 2020 and a member of the Howard Hughes Medical Institute's inaugural class of Freeman Hrabowski Scholars in 2023. She won the American Society for Biochemistry and Molecular Biology 2024 Walter A. Shaw Young Investigator Award in Lipid Research.

Gisriel is an assistant professor of biochemistry at UW–Madison. Gisriel's lab studies the conversion of light energy into chemical energy in photosynthesis and the role of macromolecules called photosystems. Using structural biology techniques, the team is investigating the molecular mechanisms, diversity and evolution of the photosystems involved in oxygenic photosynthesis. Gisriel received a National Institutes of Health Pathway to Independence Award and a Wisconsin Space Grant Consortium's Early-Stage Investigator Award.

Subramanian receives electron microscopy honor

Ramaswamy Subramanian has been awarded the 2025 Professor N. N. Dasgupta Memorial Lecture Award by the Electron Microscope Society of India. Dasgupta was a pioneering biophysicist who helped construct Asia's first electron microscope. This award is the highest honor given by EMSI and recognizes Subramanian's contributions to advancing electron microscopy and global access to cutting-edge scientific tools. His vision and advocacy helped position India as an early leader in cryogenic electron microscopy, establishing one of the country's first centers and laying the groundwork for a nationwide network of advanced imaging facilities.

Subramanian is a professor of biological sciences and biomedical engineering at Purdue University. His lab studies how certain bacteria evade the immune system by stealing and displaying sugar molecules that mimic human cells, leading to an industry collaboration to understand the molecular basis of promising drug candidates in clinical trials.

Truttmann recognized for cell stress research

Matthias C. Truttmann, assistant professor of molecular and integrative physiology at the University of Michigan, has received the 2025 Ferruccio Ritossa Research Scholar Award from the Cell Stress Society International (CSSI). This award recognizes early- to mid-career scientists whose work exemplifies the pioneering spirit of Ferruccio Ritossa, who discovered the heat shock response in the early 1960s — a fundamental mechanism by which cells adapt to stress. Truttmann was honored and delivered remarks at the CSSI annual meeting in October.

Truttmann's research focuses on the regulation of protein quality control mechanisms, particularly the role of heat shock protein 70 (Hsp70) family chaperones. His lab investigates how posttranslational modifications of Hsp70 proteins influence their function in maintaining proteostasis, with implications for aging and age-related diseases, including neurodegenerative and cardiovascular disorders. Using a multidisciplinary approach that includes molecular biology, genetics, neuroscience, and biochemistry, Truttmann's team aims to uncover and exploit novel therapeutic targets for these conditions.

He earned his Ph.D. in infection biology from the Biozentrum University of Basel, Switzerland, and completed postdoctoral work at the Whitehead Institute for Biomedical Research, MIT, and Boston Children's Hospital. He has received many awards, including the U-M Innovative Multidisciplinary Research Pilot Award and the U-M Drug Discovery Award, which both recognized his innovative work in proteostasis and stress biology.

According to the CSSI announcement, Truttmann's work "advances our understanding of Hsp70 chaperone regulation and its role in proteostasis," continuing Ritossa's legacy by linking molecular chaperone biology to human health.

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