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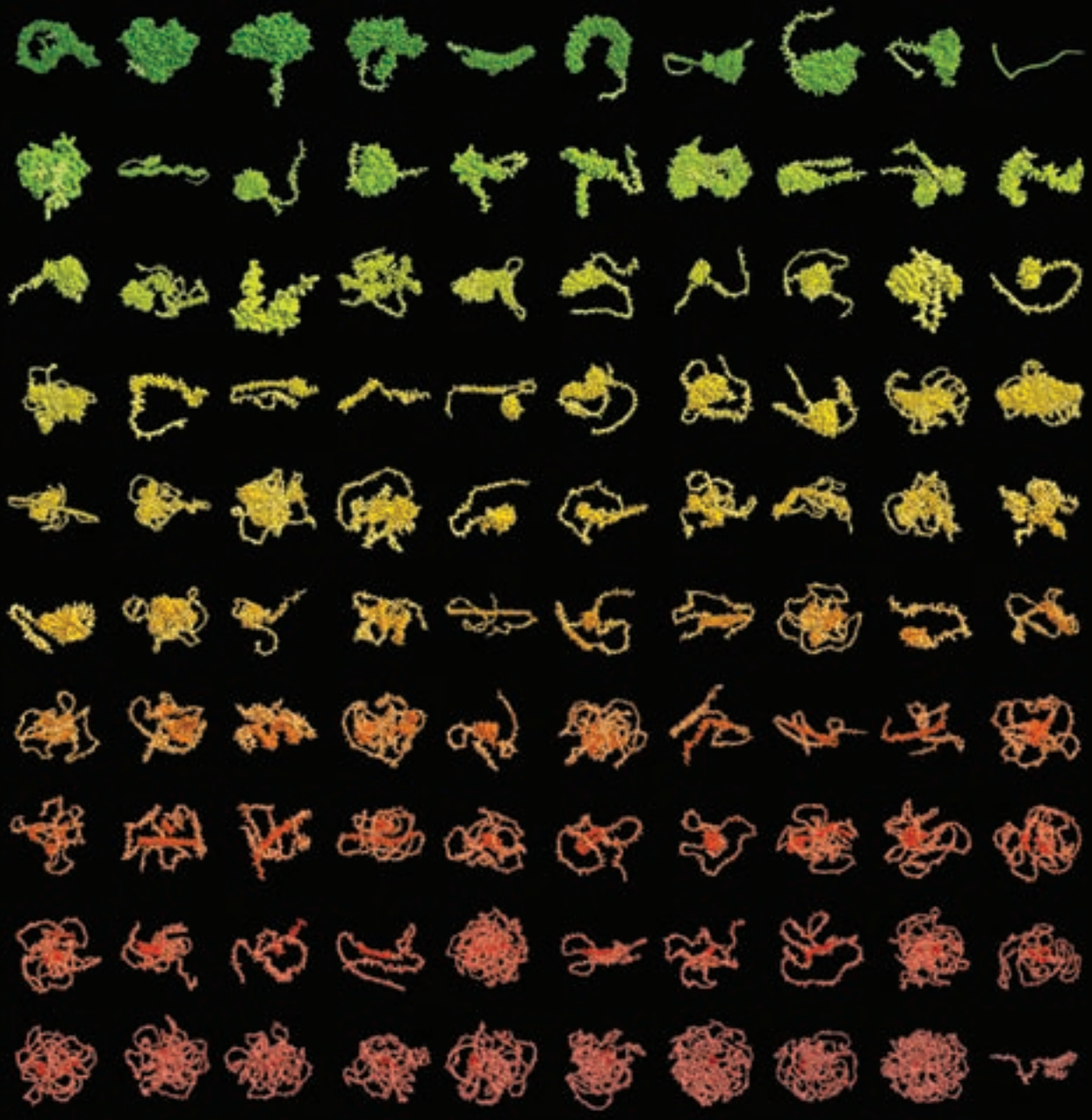
ASBMB TODAY

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

Using AI as a tool to advance molecular life science



SCIENCE, COMMUNITY, CONNECTION

Inside: your preview of the ASBMB annual meeting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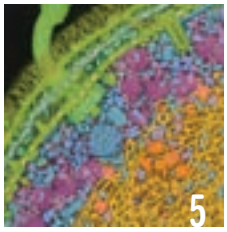
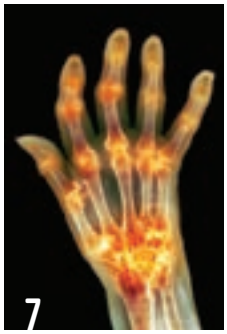
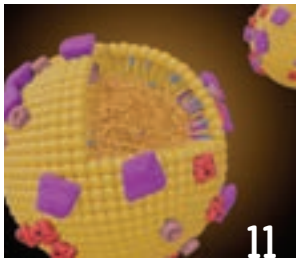






ABOUT THE COVER:

BioArt for Fall: From order to disorder

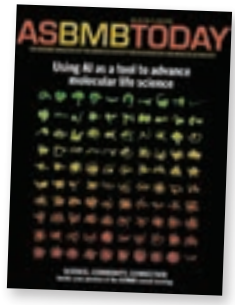
by ASBMB member, Soutick Saha, Wolfram Alpha LLC

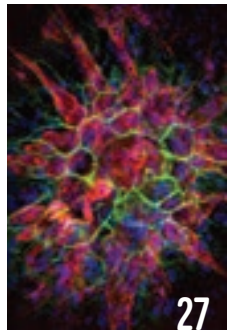
"Intrinsically disordered regions (IDRs) are ubiquitous in proteins. These are regions in proteins that lack a fixed or ordered three-dimensional structure. This image depicts different levels of disorder in 100 human proteins from the AlphaFold Protein Structure Database, with disordered regions highlighted in lighter shades. The hue varies from green to yellow to red, representing the increasing levels of disorder and also how leaves change color in fall, before they fall to the ground.

Each image was created using the Wolfram ResourceFunctions BioMoleculeIDRs and BioMoleculeIDRPlot3D, which allow for the computation and visualization of intrinsically disordered regions in protein structures."

ANNUAL MEETING

FEATURES





PERSPECTIVES

MEMBER NEWS

ASBMB TODAY
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Accelerating breakthroughs with artificial intelligence

By Joan Conaway

JOAN CONAWAY

Ever feel like discoveries in science and technology are coming faster than ever? That's the law of accelerating returns — the idea that every breakthrough fuels the next one, speeding progress along.

While debates around the specifics of this concept continue, especially within the computing world, I think the basic premise still captures what scientific research is all about: every one of us contributes to the foundation on which future discoveries will be built. The development and growth of artificial intelligence, or AI, is one obvious example, and the American Society for Biochemistry and Molecular Biology is actively exploring the implications and applications of AI, in many areas of our work.

This issue of ASBMB Today focuses on the opportunities and challenges presented by the rapid developments in AI in research, while the last edition and our recent conference for undergraduate educators highlighted its use in educational settings. In this issue, you'll find articles about how AI is advancing antimicrobial, ophthalmological, and transcription research. You'll also find essays from members sharing their experiences using AI in the classroom and for coding and clinical research.

This is such a transformative topic that the 2026 ASBMB annual meeting will feature an extended deep dive session on AI called "I, biochemist: Automation and AI in the lab." This session, organized by Polly Fordyce of Stanford University and Hua Su of Merck & Co., will showcase the transformative power of AI, machine learning and robotics in biochemistry, highlighting how computational advancements drive innovation from high-throughput experimentation to large-scale data analysis. I am especially looking forward to kicking off this deep dive session with a special plenary featuring Tanja Kortemme from the University of California, San Francisco.

Plan to attend ASBMB 2026 for all the great science and to share your research and connect with the molecular life sciences community. Be sure to renew your membership — members get discounts on abstract submission and registration and are eligible for travel awards.

Scientific progress doesn't just move forward. It accelerates, opening doors to possibilities that were unimaginable just a short time ago. Your experiments, insights and discoveries add to a growing body of knowledge.

So, read on to learn more about the groundbreaking discoveries AI is enabling. I look forward to continuing the conversation and hearing more about what you're making possible at the 2026 ASBMB annual meeting.

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

Scientific progress doesn't just move forward. It accelerates, opening doors to possibilities that were unimaginable just a short time ago.

Bacterial enzyme catalyzes body odor compound formation

By Courtney Chandler

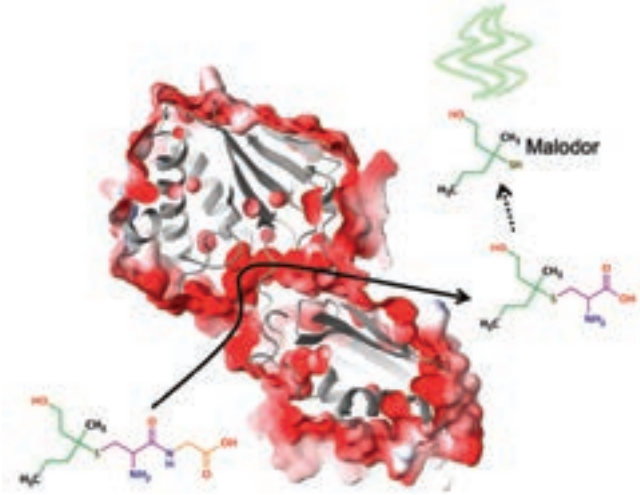
Body odor is universal to most mammals — the microbes inhabiting our skin turn odorless gland secretions into volatile chemicals with distinct scents. A skin-resident bacteria, *Staphylococcus hominis*, transforms sweat gland metabolites into sulfur-containing compounds, responsible for body odors such as onion-like smells, via a two-step process.

In a recent **Journal of Biological Chemistry** paper, researchers identified an *S. hominis* dipeptidase enzyme, called ShPepV, that catalyzes the first step in this chemical transformation. Reyme Herman, a graduate student in Gavin Thomas' lab in the department of biology at the University of York, is the first author on the paper.

Herman and colleagues from the University of York and Unilever previously reported on the second enzymatic step in the reaction, but the first eluded them.

Herman and colleagues initially tried to identify enzyme gene candidates computationally, but didn't find a clear target. The group then screened portions of *S. hominis* cellular extracts using an assay to detect the end-product of the reaction as a means to monitor enzymatic activity to try to identify the protein. Proteins from extracts that demonstrated enzymatic activity were separated on a gel and identified using peptide mass fingerprinting.

The research team found that the dipeptidase enzyme ShPepV was responsible. Herman said the discovery of this enzyme was surprising because ShPepV belongs to a class of enzymes not previously associated with this process.



The team monitored the enzyme kinetics of purified ShPepV to better understand its biochemistry. By removing and adding specific metals, researchers identified manganese as a metal cofactor. Based on models of the protein binding site, the team investigated the residues and structural features key to enzymatic activity with purified recombinant protein. They found that the enzyme demonstrated broad activity and had binding site properties — such as a large hydrophobic cavity — similar to other dipeptidase enzymes.

"The molecules that the bacteria processes to make (body odor) have a unique shape and requires special accommodation by bacterial proteins," Herman said. "We identified a similar structure feature in PepV that allows for the accommodation of this molecule."

Herman said the team plans to continue their work to uncover the roles other bacterial species and their enzymatic processes have in contributing to body odor.

Currently, the team is testing how other skin-associated species contribute to body odor. Like with *S. hominis*, Herman said the group hopes to eventually understand the molecular processes underpinning the roles of these other bacterial species in odor formation.

"This ancient process is intertwined with our behaviors, affecting our interactions with each other," Herman said. "(Our work will) contribute to our understanding of (body odor) formation, identifying enzymes that could be targeted for interventions of this process."

This research furthers the understanding of the interplay between bacterial enzymes and the production of body odor in humans.

DOI: 10.1016/J.JBC.2024.107928

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

From the journals: JBC

How bacteria fight back against promising antimicrobial peptide. Calcium channel linked to cancer drug resistance. Receptor antagonist reduces age-related bone loss in mice. Read about papers on these topics recently published in the *Journal of Biological Chemistry*.

By Emily Ulrich

How bacteria fight back against promising antimicrobial peptide

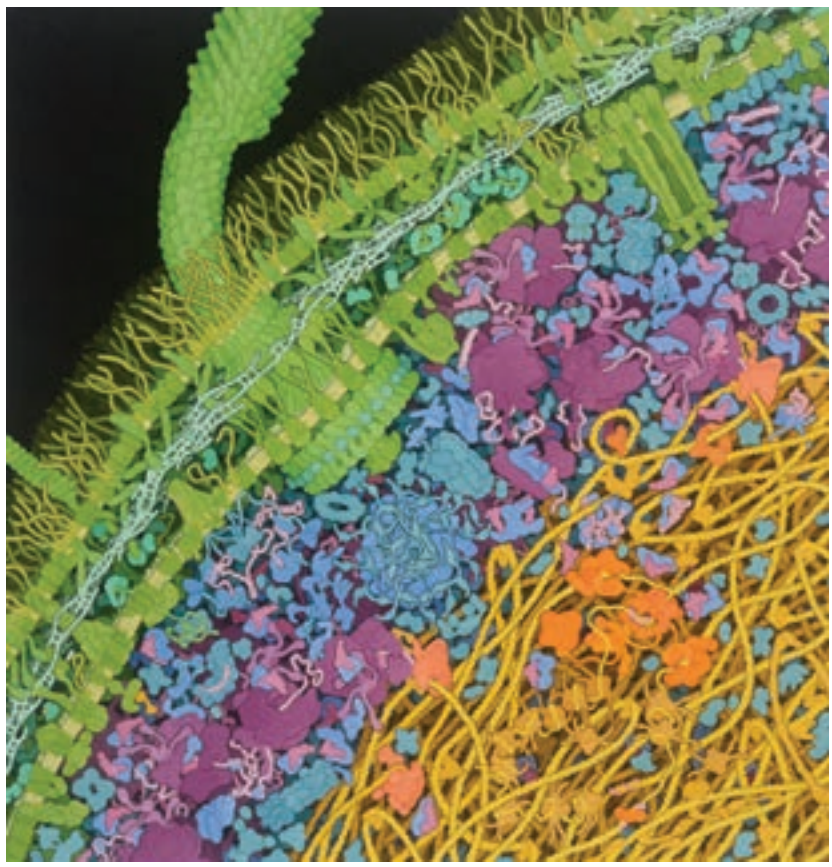
Antimicrobial peptides have potential in antibiotic drug development, including possible uses in combination with other antibiotics for infections that are difficult to treat. Scientists have shown that the peptide TAT-RasGAP317-326, originally developed as an anticancer compound, inhibits *E. coli* and *Staphylococcus aureus*, among other bacteria. The peptide contains residues 317-326 of the Ras GTPase-activating protein, or RasGAP, with an attached N-terminal cell-penetrating sequence from the HIV transactivator of transcription, or TAT, protein, and will be called TAT-RasGAP in this article for simplicity. Maria Georgieva at the University of Lausanne Hospital Center and a team in Switzerland performed a resistance selection experiment over 20 passages to obtain an *E. coli* strain resistant to TAT-RasGAP to identify mutations that could elucidate this peptide's mechanism of action. In a recent **Journal of Biological Chemistry** article, they showed that a mutation in BamA, an outer membrane protein critical for the insertion of other membrane proteins, helped block the peptide's antimicrobial activity.

The authors traced the mutation that protects *E. coli* from TAT-RasGAP to a negatively charged loop in BamA that extends into the extracellular space. The mutation changes a residue from a negative to a neutral charge. The authors hypothesized that the positively charged TAT-RasGAP may interact with this negatively charged loop for cell entry, and a negative-to-neutral mutation could have developed in the resistant strain to block this electrostatic interaction. Modeling and molecular dynamics indicated that BamA's negatively charged loop likely interacts with the peptide.

However, further experiments showed that TAT-RasGAP does not produce the same changes as known BamA inhibitors based on bacterial morphology viewed by brightfield microscopy and outer membrane protein quantification, indicating that BamA is unlikely inhibited by TAT-RasGAP. Future experiments will help resolve the full mechanism of action for TAT-RasGAP and could lead to novel antibiotics.

DOI: 10.1016/j.jbc.2024.108018

To read more JBC news, scan the code.

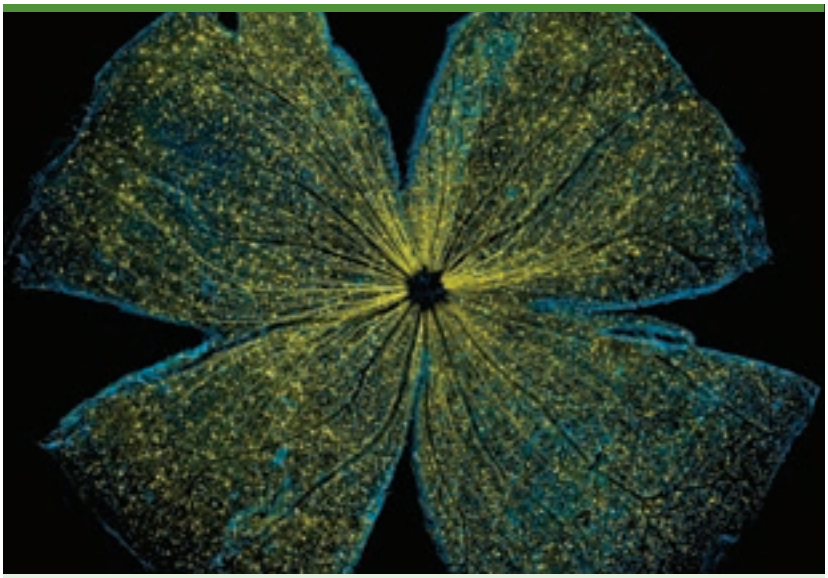


DAVID S. GOODSELL, RCSB PROTEIN DATA BANK, VIA WIKIMEDIA COMMONS

Illustration of a cross section of an *E. coli* cell. The cell wall is shown in green, the genome in yellow, DNA-binding proteins in tan and orange and ribosomes in purple.

Receptor antagonist reduces age-related bone loss in mice

Bone remodeling involves a regulation between osteoblasts for bone formation and osteoclasts for bone resorption, and this process shifts out of balance with age and with the development of osteoporosis. The complement system, activated as part of the innate immune response to infection, also functions in bone development.



KENYOUNG KIM, WONKYU JU AND MARK ELLISMAN, NATIONAL CENTER FOR MICROSCOPY AND IMAGING RESEARCH, UNIVERSITY OF CALIFORNIA, SAN DIEGO

Large-scale mosaic confocal microscopy image of a mouse retina.

The G protein-coupled receptor C3aR is expressed as a component of the complement system in bone marrow cells. Fangyu Li and Shun Cui at Huazhong University of Science and Technology investigated whether C3aR plays a role in age-related impacts on bone remodeling. They published their recent findings in the **Journal of Biological Chemistry**.

The researchers first noted that C3aR expression trends upward as mice age. In contrast, mice with C3aR knocked out showed an increase in bone mass compared to a control group of the same age. The C3aR knockout mice also exhibited higher expression of the osteogenic marker osteoprotegerin and lower expression of the osteoclast marker tartaric acid resistance phosphatase, suggesting a shift toward bone formation over resorption.

In addition, the authors tested a C3aR antagonist, called JR14a, in cells treated with D-galactose to mimic cell damage caused by aging and found that the antagonist restored cell viability. They applied JR14a to a mouse model, and their histologic staining showed an increase in osteoblasts and a decrease in osteoclasts, suggesting partial inhibition of bone loss.

Fluorescence labeling experiments performed in this study indicated that JR14a initiates YAP1/β-catenin signaling, a pathway known to promote osteoblast differentiation. Future studies will help determine the possibility of targeting C3aR for relieving age-related dysfunction in bone remodeling.

DOI: 10.1016/j.jbc.2025.108500

Calcium channel linked to cancer drug resistance

Chemotherapy tumor resistance develops after long-term regimens of the platinum-containing anticancer drug carboplatin. Scientists have observed an enlarged cell morphology and involvement of T-type calcium channels in resistant ovarian cancer cells. Sooyun Kim and researchers at Seoul National University wanted to find out if these characteristics also relate to carboplatin resistance seen in retinoblastoma, an aggressive childhood cancer. They published their findings in a recent **Journal of Biological Chemistry** article.

The authors generated a resistant population of retinoblastoma cells in the lab by exposing a retinoblastoma cell line to carboplatin. They observed a larger proportion of giant cells in the resistant subset compared to the original cell line. They also measured whole-cell electrical current of the resistant cells and determined that the giant cells had more sustained calcium currents compared to regularly sized resistant cells. Immunofluorescence staining and pharmacological inhibition experiments identified the Cav3.3 channel as the overexpressed calcium channel subtype that contributes to the sustained currents. The authors further showed that messenger RNA expression levels only for Cav3.3 increased after carboplatin exposure, while the levels for the other Cav3.1 and Cav3.2 subtypes slightly decreased in the resistant cells relative to the original retinoblastoma strain.

DOI: 10.1016/j.jbc.2024.107973

Emily Ulrich is the ASBMB's science editor.

RA patient blood reveals joint innerworkings

By Inayah Entzminger

Many people ignore lingering joint pain, but persistent swelling could signal rheumatoid arthritis — a chronic autoimmune disease that not only attacks joints but also harms the lungs and kidneys.

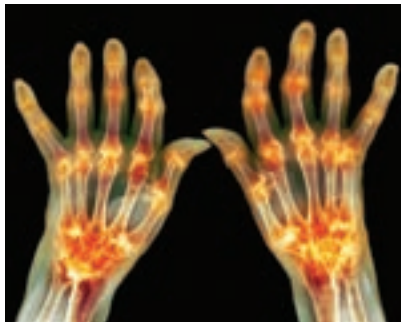
In a recent study published in the journal **Molecular & Cellular Proteomics**, researchers at Leiden University Medical Center and Utrecht University used proteomics to show that proteins, including antibodies and autoantibodies, pass from blood into joint fluid without bias in RA patients.

Eva Maria Stork, a postdoctoral fellow at LUMC, Sofia Kalaidopoulou Nteak, a Ph.D. candidate at UU and Danique van Rijswijck, a faculty researcher at UU, used multilayer proteome analysis to provide evidence that RA therapeutics introduced into blood enter the synovial compartment at predictable ratios.

While swelling after an injury typically resolves, in RA the immune system drives chronic inflammation, causing persistent pain and long-term mobility problems.

Therapeutic treatments for RA include systemic delivery of monoclonal antibodies, whose effectiveness depends on how well they are absorbed into the synovial fluid, or SF.

Their workflow catalogued the entire RA proteome and mapped the diversity of RA-specific IgG1 antibodies in blood and synovial fluid.



"Previous studies usually picked individual proteins and looked at their concentrations on both sides," Stork said. "We are now looking at a really high number of proteins and that gives us a more accurate way to conclude the correlation between (blood plasma and SF)."

LC–MS/MS analysis revealed 481 unique proteins across both plasma and SF in the RA patients.

Researchers determined that all detected plasma proteins, such as albumin and several plasma glycoproteins, were present in SF. Their abundance in plasma correlated with the abundance in SF.

About 33% of proteins detected in SF represented plasma proteins. Proteins that were more abundant or solely detected in SF included cartilage proteins, cell nuclei and cellular cytoskeleton proteins and proteins originating in neutrophils.

"From our findings, those histones and cartilage proteins that we see in the synovial fluid are not found back in the plasma," Kalaidopoulou said. "It really seems that (permeability) is one-way."

Analysis of patients that received IgG1-based mAb therapy showed IgG1 Fab molecules appeared in similar abundances in plasma and SF.

Researchers concluded that plasma proteins enter SF from blood plasma in an unbiased manner, regardless of size, charge, concentration or other biochemical properties.

These findings suggest that mAb therapy absorption could be determined via blood plasma analysis instead of invasive SF aspiration.

Stork believes that blood tests may become a patient-friendly method to determine therapeutic protein concentration in SF of RA patients in the future. Instead of obtaining SF through an invasive procedure, protein concentration could be determined through blood drawn for other medical checks.

LC–MS/MS for qualitative protein analysis is not possible in every hospital. However, Kalaidopoulou said that some hospitals are starting to use simple high-throughput liquid chromatographs for diagnostics.

"We are … at the direct interface of the hospital with the outpatient clinic," Stork said. "Patients come in every day and are then directly connected with the lab. That's what makes these studies feasible. We bring science to the needs of the patients."

DOI: 10.1016/j.mcpro.2024.100900

Inayah Entzminger is an ASBMB Today columnist.

From the journals: MCP

A look into the rice glycoproteome. Proteomic variation in heart tissues. Pesticide disrupts neuronal potentiation. Read about papers on these topics recently published in *Molecular & Cellular Proteomics*.

By Ecem Arpaci

SYARIFUL MSTH VIA WIKIMEDIA COMMONS

Oryza sativa in a rice field and surrounding natural scenery in Indonesia.

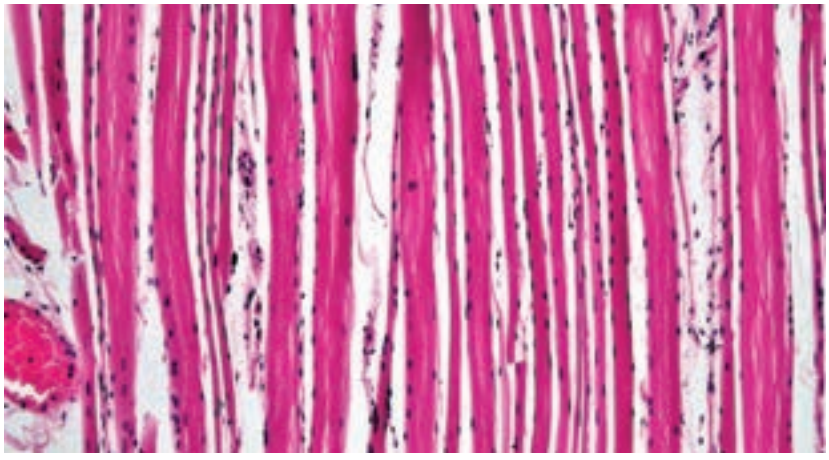
A look into the rice glycoproteome

Proteins undergo posttranslational modifications, such as N-glycosylation, during which a sugar chain is added to the amino acid asparagine at specific sites to form a glycoprotein. N-glycosylation aids cell-to-cell communication as well as pathogen interactions in animals, but scientists know little about this modification in plants.

To address this gap, Cong Lei, Xilong Li and Wenjia Li of Yazhouwan National Laboratory and a research team in China developed a metabolic glycan labeling approach, which they used to map N-glycans in rice. In their **Molecular & Cellular Proteomics** paper, the authors grew *Oryza sativa*, or rice, with a nutrient mixture containing N-azidoacetylgalactosamine, or GalNAz, an artificial glycan building block they could track over time. GalNAz has a unique chemical group to which the authors attached an affinity tag using click chemistry. This made it possible to study the glycoproteins with liquid chromatography–tandem mass spectrometry.

The team identified hundreds of rice-specific N-linked glycoproteins involved in essential biological processes such as plant growth, starch metabolism and protein processing. In addition, several identified proteins mapped to the endoplasmic reticulum–associated protein degradation, or ERAD, pathways, which maintain a balance between protein folding and degradation. The authors also found the core proteins of this pathway to be N-glycosylated in two human cell lines, suggesting that its regulation is conserved between species.

As changes in N-glycosylation have been linked to disrupted plant development, it is important to shed light on modified proteins and sites. This workflow can be used on other plants to expand our understanding of plant glycoproteomes for agricultural research and biotechnology.
DOI: 10.1016/j.mcpro.2024.100883



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Proteomic variation in heart tissues

Induced pluripotent stem cells, or iPSCs, can be used to model tissues and disorders, such as heart diseases. However, the human heart is com-

plex, with several regions and cell types. Therefore, analyzing single cardiac cells can be challenging.

Lizhuo Ai, Aleksandra Binek and Vladimir Zhemkov of the Cedars-Sinai Medical Center and a team in the U.S. used this approach to analyze heart cells throughout various stages of development. They published their findings in **Molecular & Cellular Proteomics**. The authors differentiated iPSCs into cardiomyocytes, the muscle cells of the heart, and analyzed their proteome at various time points using mass spectrometry.

The team detected proteomic differences between groups of iPSC-derived cardiomyocytes, or iCMs, at the end of their three-week differentiation period, suggesting that they had developed into multiple cell subtypes. Many differentially expressed proteins between two of these subtypes functioned in extracellular vesicles, called exosomes, which transport biomolecules such as DNA and proteins to other cells. They also compared iCMs to those isolated from adult human hearts, or aCMs. They found the two cell types shared many proteins involved in muscle function, but that aCMs had a higher proportion of mitochondrial proteins, suggesting that iCMs are metabolically immature.

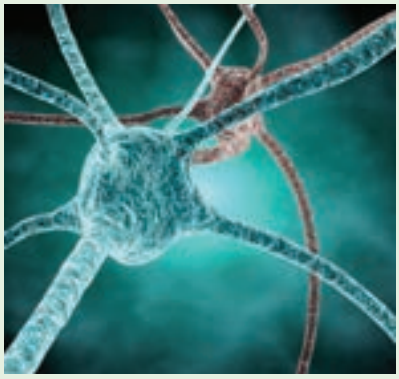
Furthermore, detailed analysis revealed that a few human adult heart cells expressed markers that traditionally identify heart and brain cells, which may represent a newly discovered cell type. These results could mean that heart cells are more diverse than scientists originally thought and could underlie some functions such as exosome formation. The ability of iCMs to recreate much of the cardiomyocyte proteome, combined with the diversity of tissues they produce, make iPSCs appealing candidates for studying and treating human heart diseases.

DOI: 10.1016/j.mcpro.2025.100910

To read more MCP news, scan the code.

Pesticide disrupts neuronal potentiation

The pesticide deltamethrin is widely considered a safer alternative to other chemicals, such as organophosphates due to being less toxic to mammals. However, previous studies in mice showed that deltamethrin exposure at early stages of development can lead to neuronal toxicity, but scientists do not understand the mechanism involved. Therefore, Leandra Koff and a team led by Fernanda Laezza from the Sealy Center of Environmental Health & Medicine at the University of Texas Medical Branch and colleagues in the U.S. investigated how deltamethrin induces neuronal toxicity and published their results in **Molecular & Cellular Proteomics**.



JULIENDM VIA FLICKR

The team focused on brain-derived extracellular vesicles, or BDEVs, structures that transport molecules, such as signaling proteins, between cells in the brain. Because changes in the BDEV proteome can be a sign of disease, the team used a mass spectrometry–based approach to compare the protein content of BDEVs in mice exposed to deltamethrin and controls. They found several differentially expressed proteins between the two groups. Some alterations were associated with neuronal structure, transport and long-term potentiation, which promotes synaptic connections and plays roles in learning and memory. These proteomic differences could be one explanation for neuronal toxicity due to impaired nutrient transport and growth.

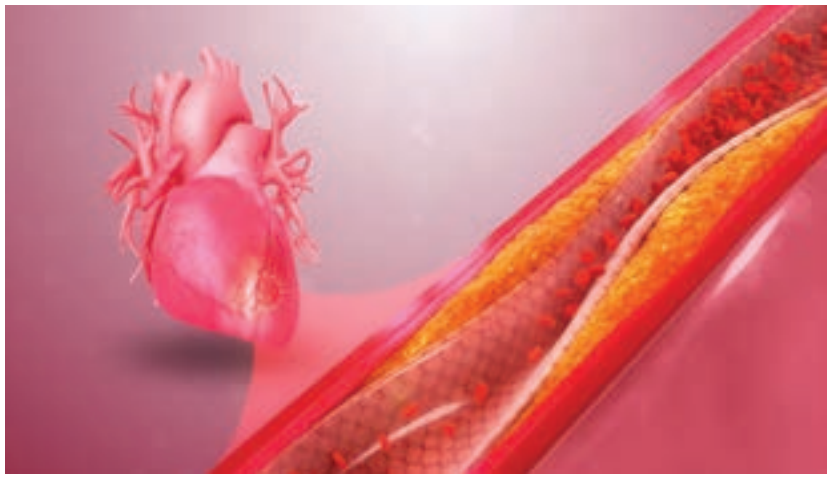
BDEV protein levels could be used as biomarkers to evaluate the risk of neurodevelopmental disorders. More research is needed to understand how these proteins disrupt neuronal function at later stages of development.

DOI: 10.1016/j.mcpro.2024.100902

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

Unexpected correlation between age and HDL-C levels

By Jessica Desamero



SCIENTIFIC ANIMATIONS

Coronary heart disease, or CHD, is a type of heart disease in which coronary arteries can't deliver enough oxygen-rich blood to the heart, causing chest pain, shortness of breath and potentially a heart attack. High-density lipoprotein cholesterol, or HDL-C, also known as "good" cholesterol, levels are a risk indicator of CHD. Naturally high HDL-C levels reduce the risk of CHD, while low HDL-C levels increase this risk.

In a recent **Journal of Lipid Research** study, researchers determined how various factors, such as age, physical activity and childbirth, predict HDL-C serum concentration in a large, diverse cohort of adults. This multicenter study is one of the few to analyze HDL-C over an extended period, over 30 years, rather than only one time point.

The Coronary Artery Risk Development in Young Adults study, or CARDIA, began in 1985 with a group of 5,115 Black and white men and women, aged 18–30 years, across multiple U.S. sites. James M. Shikany and his team led the coordinating center for the study at the University of Alabama at Birmingham. CARDIA examines the risk factors of CHD. Since then, Shikany has expanded the study to examine factors associated with HDL-C.

Participants received tests and responded to questionnaires every five years to measure these factors. Shikany and his team examined patterns in physical measurements, lifestyle factors, behavioral and psychological variables, medical and family history and blood concentrations of relevant substances. They then performed statistical regression analyses on the data. In their study, the team tested additional variables associated with HDL-C, including menopause status, hormone therapy and use of prescription drugs that lower cholesterol levels. Shikany said previous studies rarely examined these variables.

"I started noticing over the years that the HDL-C (serum levels) in CARDIA in men and women have been rising since year 15," Shikany said. "(T)hat just piqued my interest."

Shikany said this observation puzzled him because most participants showed higher weights, a characteristic usually associated with lower HDL-C levels.

The team observed that HDL-C levels positively correlated with age, alcohol intake, hormone therapy and birth control use and physical activity. Moreover, they found that individuals who had given birth had lower HDL-C levels than those who had not, with an even stronger correlation amongst those that birthed three or more children. Other factors associated with lower HDL-C concentrations include smoking and body mass index.

Future directions will explore additional factors that may impact HDL-C levels and examine details of patient HDL-C, such as subtypes and functionality.

CARDIA is ongoing, and its duration will soon reach 40 years. Shikany said he hopes that the study will continue for many more years, allowing researchers to determine additional factors that influence cholesterol levels.

DOI: https://doi.org/10.1016/j.jlr.2024.100717

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.

Omega-3 fats linked to healthy aging

By Naushin Raheema

To read more JLR news, scan the code.

The incidence of age-related obesity is rising within the global population along with the intake of high-fat foods. Age-related changes in body weight, fat distribution, insulin sensitivity and triglyceride metabolism have been linked to chronic metabolic conditions, including obesity, Type 2 diabetes and hyperlipidemia.

To combat this global health issue, researchers must understand how triglyceride metabolism changes over time.

The Brandon Davies Laboratory at the University of Iowa recently showed that a high-fat, Western diet reduces lifespan, while a diet high in polyunsaturated and omega-3 fatty acids increases cardiac triglyceride uptake and improves insulin sensitivity. The researchers published their work in the **Journal of Lipid Research**.

"We're interested in studying triglyceride metabolism, tracing the journey from the point where triglycerides are consumed, how they enter the bloodstream, and ultimately where they are stored or used for energy." Kathryn Spitler, lead author and a research associate at UI, said.

The members of the research team explored how different dietary fats influence metabolic health and whether these findings in mice could be relevant for human health.

While there is significant knowledge on triglyceride and fatty acid metabolism in humans, there is a lot to learn about how aging impacts this process. To close this gap in knowledge, they divided mice into three groups: a control group fed a standard diet, a group fed a high-fat diet rich in saturated fats and a group fed a high-fat diet rich in omega-3 fatty acids, commonly found in fish oil.

"Unlike saturated fats, omega-3 fats are often associated with potential health benefits." Spitler said. "By comparing these groups, we sought to understand whether the type of fat consumed influences metabolic health during aging."

In humans, after consuming a high-fat meal, triglyceride levels in the blood rise temporarily and then decrease as the body absorbs the fat. However, with age, the rate at which tissues absorb fat decreases, causing triglyceride levels to remain elevated in the blood for longer after a meal.

"When we eat fatty foods, our digestive tract packages them into these balls of fat called chylomicrons that will circulate throughout our bloodstream. These circulating chylomicrons provide triglycerides as a source for energy to our highly metabolic tissues or our bodies store triglycerides in adipose tissue," Spitler said.

They showed that as age increases, tissues absorb fat less efficiently, which can lead to harmful side effects such as increased risk of heart disease, stroke and pancreatitis.

The Davies team also found that mice fed a diet high in fat derived from fish oil, gained weight but demonstrated increased tolerance to insulin. These mice showed enhanced cardiac triglyceride uptake, suggesting more efficient fat processing. In contrast, the mice on the Western diet had impaired glucose tolerance and a lower ability to respond to insulin while having higher fat accumulation in non-adipose tissues, which can be linked to metabolic complications.

The researchers' future directions will explore how omega-3 fats alter cellular signaling involved in insulin tolerance and cardiac triglyceride uptake.

DOI: 10.1016/j.jlr.2024.100706

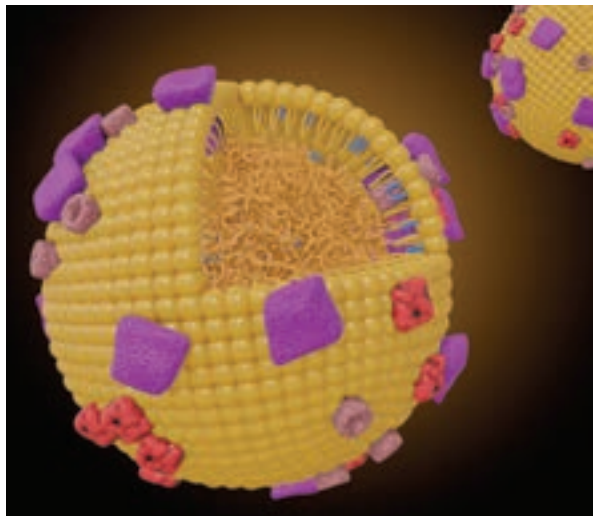
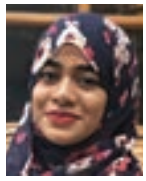


Illustration of chylomicrons, or ultra low-density lipoproteins

Naushin Raheema is a science communicator and writer. She writes articles on health, space, genetics and the environment. She writes poems and does art journaling in her free time.



Early lipid changes drive Zellweger spectrum disorder

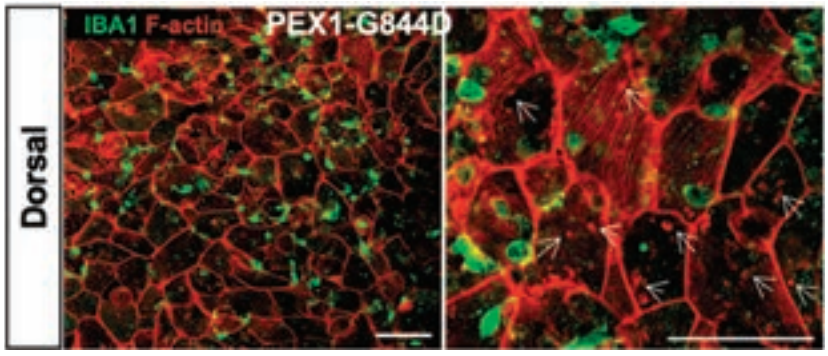
By Meric Ozturk

Zellweger spectrum disorder, or ZSD, is a rare genetic disorder that disrupts essential cellular processes in infancy and worsens over time, often leading to blindness and life-threatening complications. Individuals with mild ZSD typically live 30–40 years, though treatment is limited to symptom management. This disorder disrupts essential cellular processes by impairing peroxisomes — small organelles responsible for breaking down toxic substances and producing lipids, which are vital for brain and organ development.

In a recent **Journal of Lipid Research** study, Samy Omri, a research associate in Nancy Braverman's lab at McGill University Health Centre, and colleagues focused on mutations in the peroxisomal biogenesis factor 1, or *PEX1*, gene, the most common cause of ZSD. These mutations disrupt lipid biosynthesis and peroxisome assembly, leading to progressive retinal degeneration — a major cause of childhood blindness in affected individuals.

"Currently, there is no curative treatment, so supportive management aims to alleviate symptoms and improve patients' quality of life," Omri said. "This includes dietary modifications to address metabolic imbalances, supplementation with essential fatty acids to support neurological function."

To model ZSD-related retinal degeneration, the researchers used mice with the *Pex1-G844D* mutation, which mirrors the common human variant.



COURTESY OF SAMY OMRI

Confocal microscopy images of retinal pigment epithelium of mice engineered to have Zellweger spectrum disorder–like disease. Images are stained for a microglia/macrophage marker (green) and F-actin (red).

"The team has been deeply engaged in studying and treating peroxisomal disorders, and this project, investigating the influence of lipid metabolism on retinal health, naturally evolved from that work," Omri said. "My expertise in retinal physiopathology, along with my interest in early molecular changes preceding inflammation and tissue degeneration, aligns well with the lab's research focus."

The team focused on retinal pigment epithelium, or RPE, analyzing morphological, inflammatory and lipid changes in a mouse model at one, three and six months of age. They found that RPE degeneration could be detected by three months and worsened with age.

Using mass spectrometry imaging, generated by Pierre Chaurand's lab, the group identified 47 lipids in RPE that were altered before any visible structural degeneration in the retina. This approach allowed them to visualize the spatial lipid distribution in the retina, providing insight into early disease mechanisms.

"While previous work has largely focused on systemic disease characterization and functional recovery, our research shifts the focus to find new biomarkers for retinal degeneration and a deeper mechanistic understanding of ZSD pathology," Omri said.

Next, the team plans to validate the clinical relevance of these lipid signatures, a step toward developing future diagnostic tools or therapeutic targets.

They also discovered progressive subretinal macrophage accumulation in ZSD mice, revealing a previously unrecognized inflammatory pathway potentially driving retinal degeneration. Future studies will investigate these inflammatory signal pathways to identify anti-inflammatory therapies that could slow or prevent retinal degeneration.

DOI: 10.1016/j.jlr.2025.100771

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

Using AI as a tool to advance molecular life science

The following features explore how artificial intelligence is transforming research in gene regulation, medical care and antimicrobial discovery.

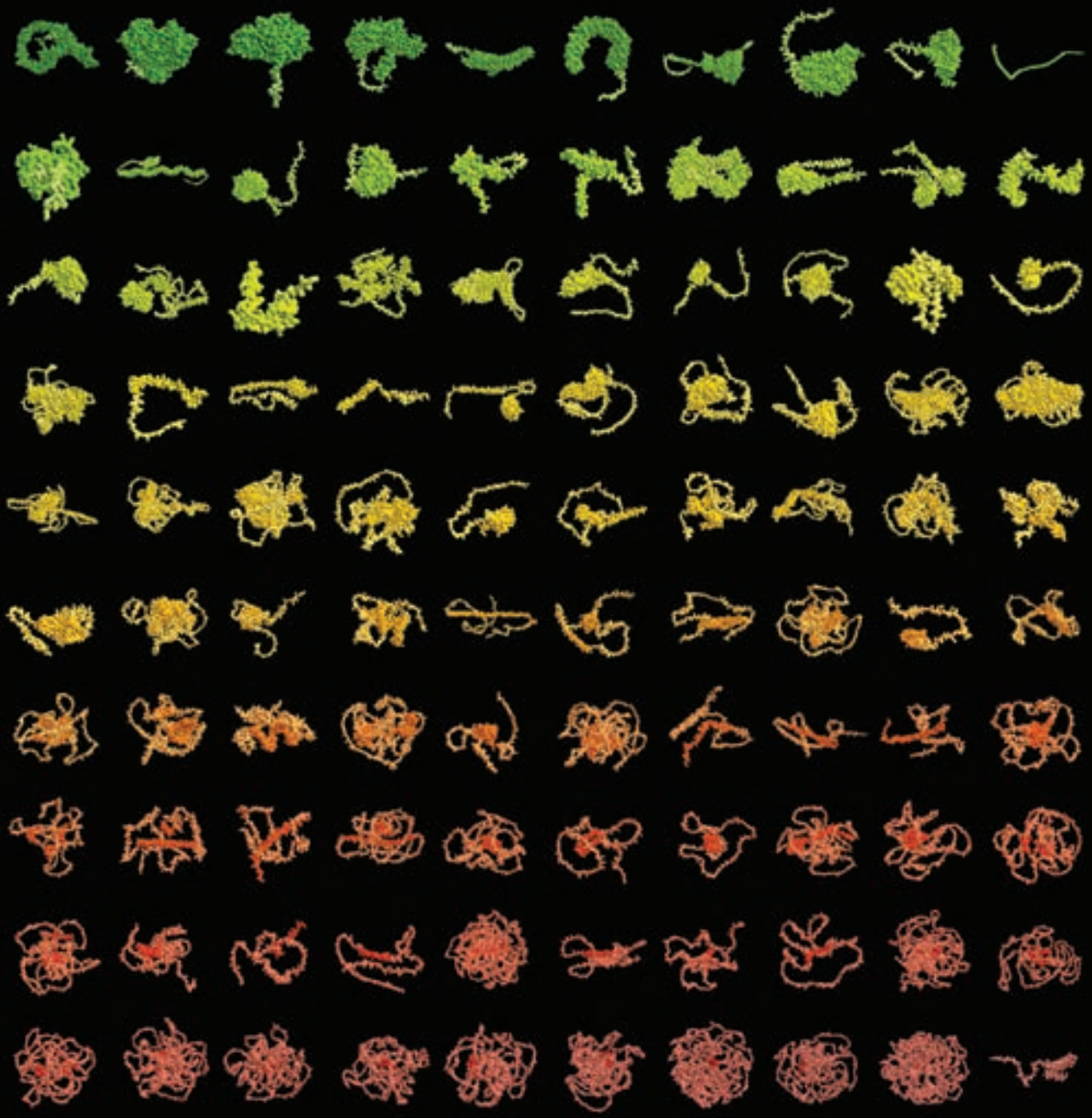
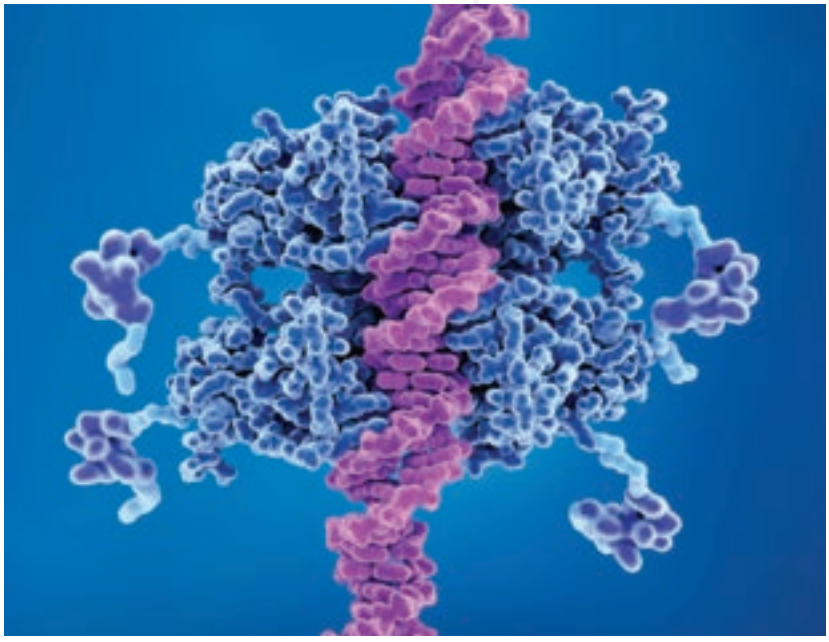


Illustration by ASBMB member, Soutick Saha, Wolfram Alpha LLC

AI unlocks the hidden grammar of gene regulation

By Courtney Chandler



The fruit fly *Drosophila* genome is like a 180-million letter book. Julia Zeitlinger, a professor at the Stowers Institute for Medical Research, is determined to read and dissect that book to understand how the sentences are built, and which words truly matter.

Zeitlinger studies how protein transcription factors, or TFs, bind to DNA to regulate gene expression, using fruit flies as her model. She likened TF binding sites, called motifs, to words in the genome's book. But, unlike words, motifs have no fixed location, precise definition or predictable arrangement. Their meaning depends on position and context. To decode them, Zeitlinger needed more than biology. She found her solution in what was at the time an emerging and unfamiliar field — artificial intelligence.

PHILIPPE NOGUERA

Julia Zeitlinger and bioinformatician Melanie Weilert discuss data in Zeitlinger's lab at the Stowers Institute.

Teaching AI to read the genome

An early adopter of using artificial intelligence, or AI, Zeitlinger began using it as an expert genome reader, allowing her to better define the linguistics of gene expression.

That innovative approach drew in bioinformatician Melanie Weilert, who, in 2017, was preparing to leave academia for industry because she struggled to connect with her work — until she joined the Zeitlinger lab.

"A lot of labs would pick a model system and work their way down to specific genes until they got to the crux of how a DNA sequence encodes what they're interested in," said Weilert, now a bioinformatician in Zeitlinger's lab. "The Zeitlinger lab works in the opposite direction. The goal is to take well-studied examples and discover the general rules that apply to the entire genome."

With 180 million letters to sift through, Zeitlinger needed tools to pinpoint which DNA base pairs might serve as TF binding sites. So, her lab developed chromatin immunoprecipitation-nexus, or ChIP-nexus, a method that maps TF genome binding sites down to the specific nucleotides.

This high-resolution method brought the entire genome under a magnifying glass. But, according to Zeitlinger, interpreting the data and rules of TF binding was daunting.

"We had this complex (ChIP-nexus) data we were trying to analyze," Zeitlinger said. "We sort of knew what TF binding trends we should find, but finding general rules that predict binding genome-wide was impossible. It's just too complex to

> **"The goal is to take well-studied examples and discover the general rules that apply to the entire genome."**
>
> **MELANIE WEILERT**

> **“Specialist AI tools excel at tasks that humans are not good at — they can find relationships between different pieces of experimental data and can computationally predict the experimental outcomes in seconds and at an incredible scale.”**
>
> **ŽIGA AVSEC**

dissect manually.”

That changed in 2015, when Zeitlinger heard Stanford University’s Anshul Kundaje describe how neural networks — machine learning models that can recognize, classify and predict patterns — could help unravel biological complexity.

Intrigued, Zeitlinger shared her ChIP-nexus data with Kundaje. A student in his lab,, Žiga Avsec, now a research scientist at Google DeepMind, took on the challenge of making sense of the large datasets. His work gave rise to BPNet.

BPNet is a neural network that discovers genome-wide links between DNA sequence patterns and TF binding profiles from sequence alone, acting like an advanced grammar decoder across the entire genome. Avsec called it a “specialized” AI tool that can move experiments into the virtual space in seconds and at an incredible scale.

“Specialist AI tools excel at tasks that humans are not good at — they can find relationships between different pieces of experimental data and can computationally predict the experimental outcomes in seconds and at an incredible scale,” he said. “This allows scientists to improve their intuition about how nature works and allows them to narrow down the hypotheses to test in the lab.”

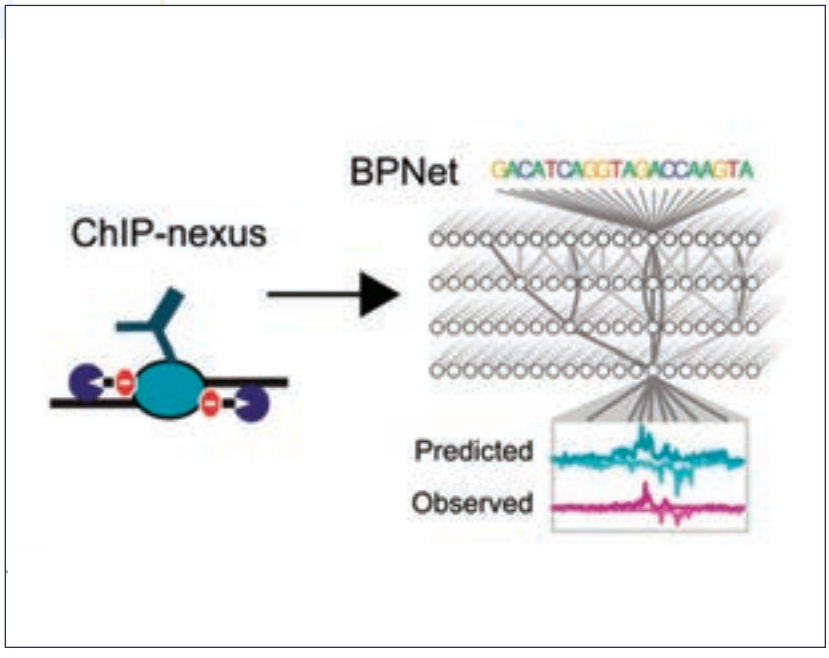
Zeitlinger said the precision and time savings from these tools are shaping both her research and the broader field, pushing science into new realms.

“We are going to get into a virtual biology world where we get knowledge at our fingertips and predict things really quickly,” Zeitlinger said. “And that’s super exciting.”

What AI revealed about chromatin and beyond

Zeitlinger quickly put BPNet to work. In a recent study, her team used it to predict how TFs interact while bound to DNA. Combining AI

COURTESY OF JULIA ZEITLINGER



Julia Zeitlinger of Stowers Institute developed a technique to capture transcription factor binding sites and a neural network artificial intelligence tool to analyze and predict these binding sites.

predictions and wet lab experiments, they showed that the program could forecast how one TF's binding influences another, offering a new view of the genome's syntax and how gene expression is controlled.

BPNet's abilities weren't limited to TFs. Soon, the team applied it to nucleosomes, structures of DNA wrapped around a protein core. These structures not only package DNA but also affect gene regulation by controlling which sequences are "wound" and which are accessible.

"They're not just packaging units, they have their own life," Zeitlinger said. "Understanding what's encoded in the nucleosome is not something we could easily address before, but with the (AI models) we can see things now that we were missing."

In one study, Weilert and colleagues combined experimental genomics with deep learning models to see how TFs unwind DNA during *Drosophila* embryo development. They identified several "sequence rules" that govern DNA accessibility and expression, better defining the multilayer coordination that influences development.

Weilert said AI has sped up discovery drastically. A decade ago, a Ph.D. student might spend years studying one motif. Now, a single experiment can reveal global patterns.

"It's not that they are reinventing science, but these tools provide us with the computational power to look at already existing data in completely different and powerful ways," she said. "(These models) are just really effective pattern finders — they offer a high-dimensional capacity to identify biological phenomena that previously scientists would have to nail down using a specific hypothesis."

Shaping how others use AI

Zeitlinger's embrace of AI wasn't always popular. Early on, some colleagues were skeptical. Over time, she adapted how she communicated its value and the way it intersected with her work, and the rise of general tools like ChatGPT made AI feel more familiar. Still, she remains cautious about its pitfalls.

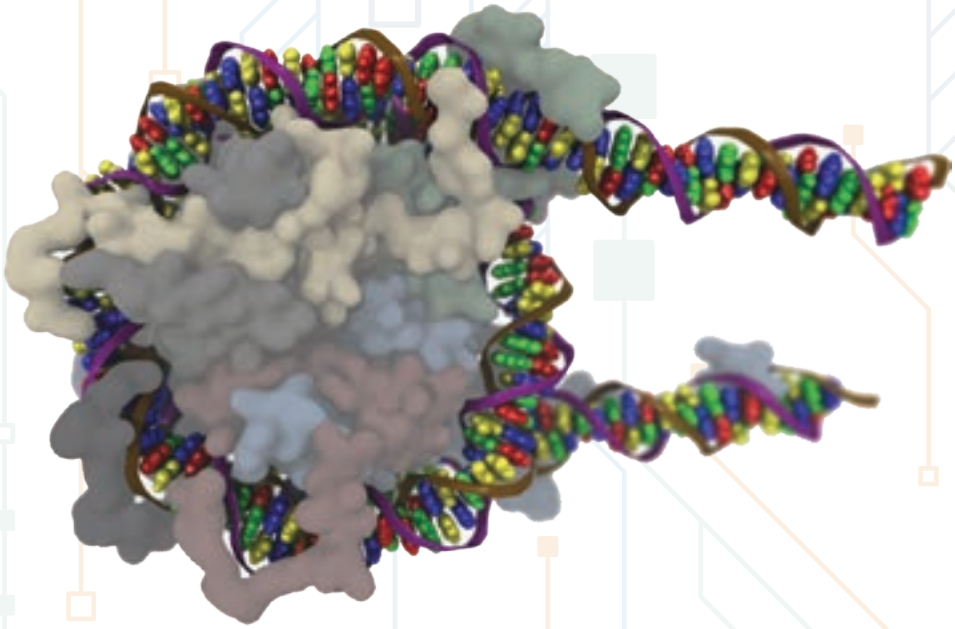
"Part of me is concerned that the science will get so complex that it will be harder to distinguish good science from bad science," she said. "It still feels a bit like the wild west."

To help integrate AI into biological research, Zeitlinger makes sure that the conferences she organizes, including the American Society for Biochemistry and Molecular Biology's evolution and core processes in gene expression conference, have speakers that apply advanced AI tools to the

> **"We are going to get into a virtual biology world where we get knowledge at our fingertips and predict things really quickly, and that's super exciting."**
>
> **JULIA ZEITLINGER**

Žiga Avsec, now a research scientist at Google's DeepMind, developed the AI tool BPNet with Julia Zeitlinger and Anshul Kundaje.

KATARINA VESELIĆ



Often referred to as DNA packaging units in a cell, nucleosomes, Julia Zeitlinger said, have a life of their own.

With AI in the toolkit, Zeitlinger hopes to predict the language rules behind healthy gene expression in humans — and uncover what goes wrong in diseases, such as cancer, neurological disorders and diabetes.

field. She also enjoys helping other labs navigate AI and was appointed at her institute to lead the Stowers Office of Scientific Leadership AI Initiative, which provides guidance and infrastructure support to researchers adopting AI tools in their work.

"I see it as a responsibility to make sure our institute maximally benefits from AI in a larger sense, while also avoiding its dangers," Zeitlinger said. "My goal is to make sure that researchers are prepared for our changing landscape and maximally benefit from the new tools that become available."

Avsec also sees AI as being transformative for science in the years ahead.

"(AI) tools, both general and specialized, will allow scientists to tackle more ambitious research questions in the coming years and thereby accelerate science," he said.

Zeitlinger expects AI to keep shaping her own work. While *Drosophila* remains her model, she is also eyeing the human genome and how it governs development and function.

With AI in the toolkit, Zeitlinger hopes to predict the language rules behind healthy gene expression in humans — and uncover what goes wrong in diseases, such as cancer, neurological disorders and diabetes.

"I am very excited about the innovation that will come with using AI in biological research," Zeitlinger said. "Biology is so complex, and these tools are exactly what we need to put the pieces together."

The fruit fly *Drosophila melanogaster* is a model organism used by many researchers.

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

FEATURES CONTINUE ON P. 24

Shaping the future of science, side by side

A meeting for all in the molecular life sciences, sparking ideas and connections that last

By Allison Frick

This March, the American Society for Biochemistry and Molecular Biology annual meeting heads to the Gaylord National Harbor Resort and Convention Center, just outside Washington, D.C. By bringing together scientists with diverse backgrounds and experiences, the meeting aims to inspire new ideas and drive progress in molecular life sciences.

Co-chairs Sandra Gabelli, the global head of protein and structural chemistry at Merck, and Aaron Hoskins, a professor of biochemistry and chemistry at the University of Wisconsin–Madison, bring expertise from both industry and academia, spanning structural biology, drug discovery, RNA processing and single-molecule biophysics.

Gabelli and Hoskins spoke with ASBMB Today to share what's new in science, fresh in format and why the 2026 ASBMB annual meeting will offer more ways for scientists to make meaningful connections across sectors and disciplines.

Q: What is unique about the ASBMB annual meeting, especially for 2026?

Hoskins: It's a window into the entire field of biochemistry and molecular biology. Whether you're into nucleic acids, metabolism or artificial intelligence, you'll find something exciting. That breadth is what makes this meeting special.

This year's meeting features significantly more speakers and organizers from biotech and industry, nearly 25%, marking a major shift from previous years.

Gabelli: I'd add that this meeting is about building community. I started attending ASBMB as a grad student. It's not just about data — it's about shared experiences, mentorship and career journeys.

Whether you're into nucleic acids, metabolism or artificial intelligence, you'll find something exciting. That breadth is what makes this meeting special.

AARON HOSKINS

Q: Who should attend this meeting?

Gabelli: The ASBMB meeting is for everyone in the molecular life sciences — academic researchers, educators,

STORY CONTINUES ON P. 22

ASBMB

ANNUAL MEETING '26
NATIONAL HARBOR
D.C. METRO AREA | MARCH 7–10

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Submit your abstract by Nov. 17.

Keynote and deep dives

An extended look at a specific scientific topic, with a morning plenary followed by in-depth sessions throughout the day.

KEYNOTE SPEAKER

Wesley Sundquist

Professor and department of biochemistry co-chair, University of Utah

DEEP DIVE

Racing the clock: Molecular mechanisms of aging

PLENARY SPEAKER: Meng Wang, *Baylor College of Medicine*

ORGANIZERS: Rozalyn Anderson, *University of Wisconsin–Madison*
Daniel Jarosz, *Stanford University*

DEEP DIVE

Is anyone there? Information transfer in biology from proteins to organisms

PLENARY SPEAKER: Eric Betzig, *University of California, Berkeley*

ORGANIZERS: Philip Cole, *Brigham and Women's Hospital Harvard Medical School*
Graham Johnson, *Allen Institute for Cell Science*

DEEP DIVE

I, biochemist: Automation & AI in the lab

PLENARY SPEAKER: Tanja Kortemme, *University of California, San Francisco*

ORGANIZERS: Polly Fordyce, *Stanford University*
Hua Su, *Merck & Co.*

Tracks

Sessions on key scientific topics, held at the same time each day, so attendees can follow a focused path or explore across disciplines.

Enzymology today and tomorrow

ORGANIZERS: J. Martin Bollinger Jr., *Penn State University*
Michelle Arkin, *University of California, San Francisco*
Jin Zhang, *University of California, San Diego*

Lipids in health and disease

ORGANIZERS: Harini Sampath, *Rutgers University*
Hongyuan (Rob) Yang, *University of Texas Health Science Center at Houston*

Quantitative metabolism in health and disease

ORGANIZERS: Charles Burant, *University of Michigan*
Gary Patti, *Washington University in St. Louis*
Tania Reis, *University of Colorado, Anschutz Medical Campus*

Nucleic acid transactions

ORGANIZERS: Greg Lohman, *New England Biolabs*
Luisa Cochella, *Johns Hopkins University School of Medicine*
Mario Halic, *St. Jude's Children's Research Hospital*

Innovative approaches to educating across the continuum of career stage

ORGANIZERS: Shyretha Brown, *Building Bridges, Inc.*
Basudeb Bhattacharyya, *University of Wisconsin–La Crosse*
Jenna Roecklein–Canfield, *Simmons University*

Broadening engagement in BMB

ORGANIZERS: Alberto Rascón, *Arizona State University*
Oluwarotimi Folorunso, *McLean Hospital, Harvard Medical School*
Allison Augustus–Wallace, *Louisiana State University Health Sciences Center New Orleans*

Chemical biology for the greater good

ORGANIZERS: Fred Vaillancourt, *Remix Therapeutics Inc.*
Keriann Backus, *University of California, Los Angeles*
Matthew Disney, *The Herbert Wertheim UF Scripps Institute for Biomedical Innovation & Technology*

Peptide and RNA medicines

ORGANIZERS: Stefan Lutz, *Codexis Inc.*
Michelle Hastings, *University of Michigan Medical School*
Sujata Sharma, *Johnson & Johnson*

Observing a lot by just watching: Advances in biological imaging across multiple scales

ORGANIZERS: Mario Borgnia, *National Institute of Environmental Health Sciences*
Rinku Jain, *AbbVie Inc.*
Kevin Welsher, *Duke University*

Get inspired.

Scan the QR code above to find curated itineraries to maximize your ASBMB 2026 experience.

Dive deep into your area of research or explore beyond. Learn more and submit your abstract at **asbmb.org/annual-meeting.**

ASBMB 2025 photography by Matt Herp

Pull this poster out and hang it in your lab!

Aaron Hoskins is a professor of biochemistry and chemistry at the University of Wisconsin–Madison. His group studies eukaryotic RNA processing and single-molecule biophysics. They explore macromolecular machine assembly, regulation and interactions with other molecules such as proteins and nucleic acids.

Sandra Gabelli is the global head of protein and structural chemistry at Merck. She leads a team that uses structural biology to answer key biochemical questions: how targets are engaged, how mechanisms of action work, and how ligands interact with targets for drug discovery design. Her group expresses, purifies proteins and applies biochemical and biophysical techniques to reveal detailed molecular insights, including visualizing how drugs bind to their targets and the effect of drugs in tissue.

STORY CONTINUED FROM P. 19

industry scientists from startups to big pharma and those working in government, regulation or patent law. It's a chance to learn new, applicable methods you can bring back to your own lab while making meaningful connections across sectors. That mix of practical insight and community-building is what makes this meeting so valuable.

Hoskins: Anyone interested in biochemistry or molecular biology should attend, no matter what your career stage or path. Early-career scientists can meet potential mentors and future collaborators, while seasoned researchers can explore new directions or sharpen how they teach and communicate science. It's a rare opportunity to immerse yourself in cutting-edge research and join conversations that move the field forward.

Q: What keeps you coming back to ASBMB annual meetings?

Hoskins: The science and the people. The poster sessions are always a highlight. I advise the ASBMB undergrad chapter at my institution, and this meeting is the biggest event of the year for our students.

Gabelli: I keep coming back for the science and the community. While I was at Johns Hopkins, my lab benefited enormously from these meetings. Now in industry, I still find inspiration and fresh ideas — from new techniques to powerful conversations with peers.

Q: Tell me about this year's meeting themes.

Hoskins: Each day kicks off with a 'deep dive' session — one big topic, a keynote speaker and two breakout tracks. It's a focused way to start the day and sets the tone for broader exploration that follows in the afternoon.

This year's deep dive topics are aging, AI/robotics and biological/molecular communication. These aren't just trendy; they're relevant to researchers across disciplines and nonscientists as well.

Q: What are you most excited about for the 2026 D.C. location?

Hoskins: D.C. is an incredible host city, with world-class museums and cultural sites, most of them free. On the scientific side, it's a unique opportunity to engage with researchers who might not typically attend the ASBMB annual meeting, including those at the National Institutes of Health, National Science Foundation and others working in science policy, law and government. We're excited to draw in new voices and share the latest in biochemistry and molecular biology.

Gabelli: The D.C.–Baltimore region is rich with active research universities and a strong ASBMB presence. It's an ideal setting to connect across academia, industry and policy: sectors that shape the future of biomedical science. I lived there for 30 years, and I'm thrilled to return and experience everything the area has to offer.

Q: How can attendees contribute to the program?

Gabelli: One of the best ways to contribute is by submitting an abstract; many talks and posters will come directly from attendee

submissions. You can also get involved through mini-symposia and workshops. These member-driven sessions let you organize a session and shape the meeting program. It's a true grassroots effort that puts emerging science and fresh perspectives at the center of the conference.

Q: What advice do you have for attendees?

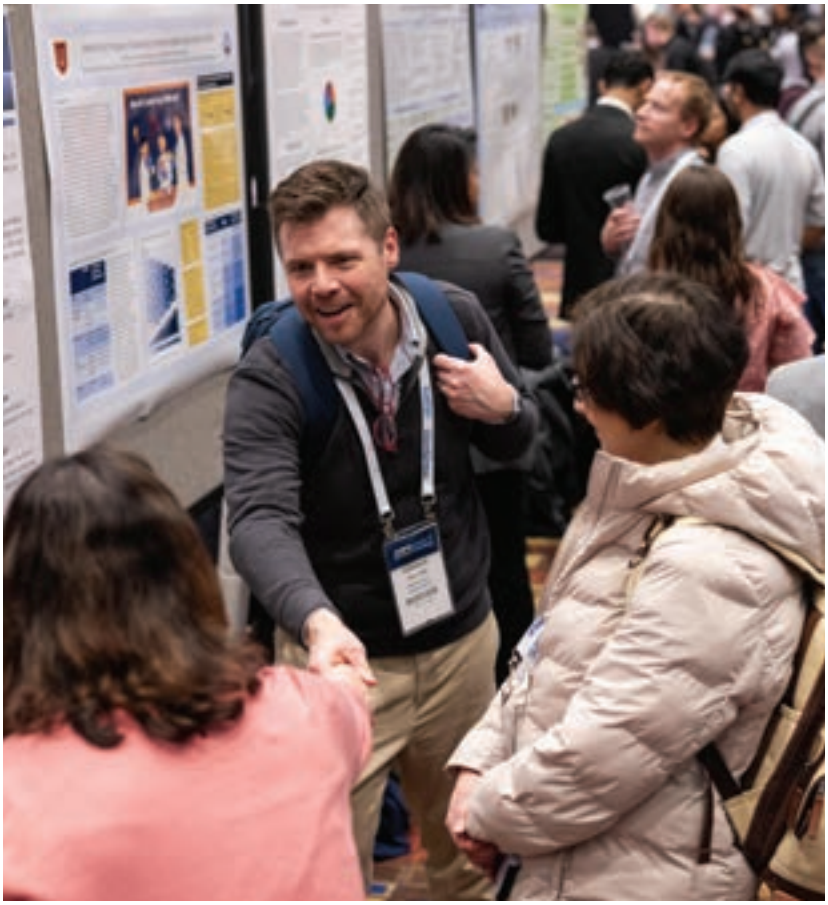
Hoskins: Attendees should take full advantage of the ASBMB meeting app — it's a great tool for planning your schedule so you don't miss the sessions and posters you're most excited about.

We're also introducing 'Speakers' Choice' stickers at the poster sessions. Invited speakers will tag standout posters, making it easy for attendees to spot must-see research as they explore the hall.

Q: Share an inspiring ASBMB meeting memory.

Hoskins: I remember one year when ASBMB was held jointly with the Experimental Biology meeting, one of my undergraduate students presented a poster while his mother, a nutritionist attending with another society, was there to watch. Seeing him explain his research to her, and how proud she was, was a truly joyful moment.

Gabelli: In the late 1990s, I attended an ASBMB annual meeting as a graduate student focused on solving protein structures with X-ray crystallography, often disconnected from functional studies. During one session, a speaker presented enzymatic data that perfectly complemented structural insights, showing how subtle changes in kinetics aligned with specific conformational shifts. That talk was a turning point; it opened my eyes to the power of integrating biochemical assays with structural data. I began incorporating enzymology into my research, which sparked a lasting shift toward an interdisciplinary mindset, connecting structure to function and static data to dynamic, biological understanding.



MATT HERP

At the 2025 ASBMB annual meeting in Chicago, Derrick Kolling, chair and professor of chemistry at Marshall University, greets a fellow attendee during afternoon activities in the exhibit hall, which included a poster session and networking opportunities.

This year's deep dive topics are aging, AI/robotics and biological/molecular communication.

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

Training AI to uncover novel antimicrobials

By Elizabeth Stivison

César de la Fuente

Antibiotic-resistant infections kill more than a million people each year, and the United Nations predicts that number could rise to 10 million annually by 2050. To address this crisis, scientists are racing to discover antibiotics that work in new ways.

"Traditionally you go around nature and try to purify compounds" César de la Fuente, an associate professor at the University of Pennsylvania said, describing how many antibiotics have been discovered. "It's a very physical process, you have to go on expeditions in nature, and you don't know what you'll find. It's like looking for a needle in a haystack."

If researchers could systematically scan vast numbers of proteins to find antimicrobial candidates, they would unlock a treasure trove of potential antibiotics. But manually, such a task would take decades.

"We decided to think of biology as an information source," de la Fuente said. Instead of explorers wandering the woods, custom-built artificial intelligence, or AI, models can mine the data.

Therefore, de la Fuente's lab began building AI models to comb proteomic data and identify antimicrobial compounds.

Antimicrobials across the tree of life

The lab first searched the human proteome. It may seem like an odd place to look, but all organisms need defenses against infection. Beyond the traditional immune system, organisms use other defenses — including "encrypted peptides," which de la Fuente's group identified and characterized.

These peptides are fragments of normal proteins that, once cleaved, act as antimicrobials and arise from a wide range of proteins, not just

WORLD HEALTH ORGANIZATION

World Health Organization Bacterial Priority Pathogens List, 2024

immune-related ones.

"Surely this is not the only place we can find these peptides," de la Fuente thought after searching the human proteome. He was right. His team has now searched across the tree of life — eukaryotes, prokaryotes, archaea and even extinct animals such as the woolly mammoth.

Every proteome they've searched contained antimicrobials, offering a wealth of molecules with potential to save lives where current drugs fail.

When AI meets the bench

The models rank thousands of potential antimicrobial peptides, but human expertise is needed to choose which to test.

"We have a human–machine meeting," de la Fuente said. Half the lab builds and runs AI models, the other half tests candidates at the bench.

The team focusing on the model meets with the biochemists to decide which candidates to test. For example, a scientist might note that a promising compound is too

hydrophobic and likely to aggregate, making it unsuitable.

Each half relies on the other: without AI, discovery would take years; without wet-lab testing, no one would know if candidates actually work.

Once hits are selected, the team synthesizes them, gives them names (such as "mammuthusin" from mammoths), and tests them biochemically and in mouse models.

Anna Crysler, a Ph.D. student, has been characterizing an antimicrobial peptide from an ancient zebra, mutating residues to see how structure affects function.

The team tests the candidates against the World Health Organization's 11 high-priority bacterial strains, known as ESKAPEE, which includes *Staphylococcus aureus, Klebsiella pneumoniae, Acinetobacter baumannii, Pseudomonas aeruginosa* and more.

It takes more than a combination of expertise to get good results though. "César is a natural leader" Crysler said. "He does a great job promoting collaboration between the two halves of the lab."

Crysler described lab meetings where all members are encouraged to ask questions and understand what each other is working on. "I couldn't imagine a different environment to do this type of work," Crysler said.

The collaborative method works. In a recent study of archaea proteomes, the model ranked more than 12,000 peptides. After human prioritization, the lab synthesized 80 — and 93% showed activity against ESKAPEE pathogens.

Long road to rapid prediction

Sorting through vast proteomic data might seem magical, but de la Fuente's AI models are built on hard work and massive datasets. The lab merged its own antimicrobial peptide data with large public repositories, then spent years refining the system.

It takes years for models to accumulate enough data to work well. And the only real way to tell if they are working, is to painstakingly test what they predict and be ready for negative data.

"Negative outcomes are valuable because they go back into the model and refine the next predictions," Marcelo Der Torossian Torres, a research associate in the lab explained. "Synthesizing peptides and testing them across different conditions simply takes time, but that data is exactly what makes the models stronger in the end."

Even so, it can be challenging in the moment. After finding one model was not working well after 2.5 years of training, "I thought it was possible I was off by an order of magnitude," de la Fuente said. Maybe instead of a few years it would take a decade, he wondered. However, the team carried on and in just an additional 1 year, for 3.5 years total, the model began to work and predict new active compounds from proteomic data.

Designing their own deep-learning model lets the team focus on specific needs, avoiding compounds that mimic existing antibiotics, for example, or seeking antimicrobials that target only one strain, preserving the microbiome and reducing side effects.

Speed is another advantage: the models can analyze astronomical amounts of data.

"We've saved many years of human research time by doing this," de la Fuente said. "Probably thousands of Ph.D. students working for six years each." That makes the 3.5-year development seem short by comparison.

Anna Crysler

Marcelo Der Torossian Torres

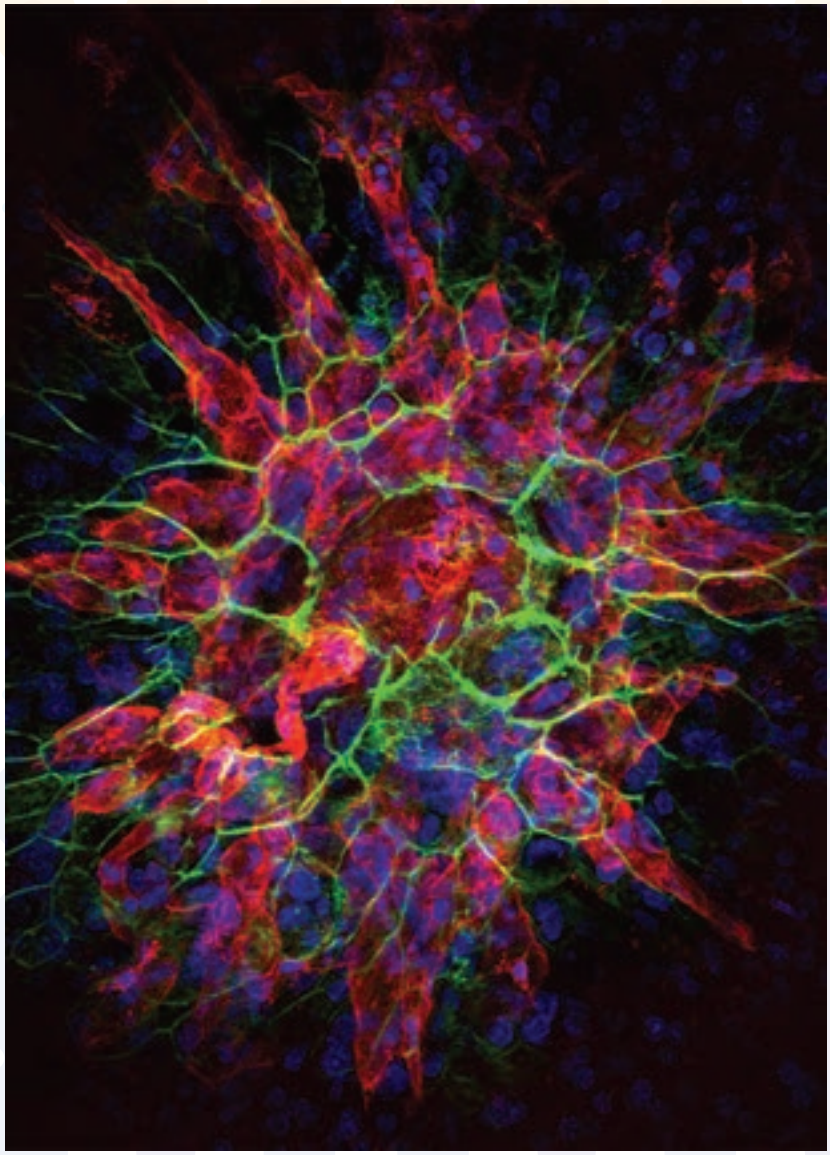
For Crysler, Torres and others in the lab, the payoff is clear: the models are not just predicting antimicrobials, they're delivering molecules that work where existing drugs fail.

With a high success rate against WHO's deadliest pathogens, their work offers real hope against the antibiotic resistance crisis that already claims millions of lives each year and threatens to claim millions more.

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

A biological camera: How AI is transforming retinal imaging

By Inayah Entzminger



SONIA SAMTANI ET AL., NATIONAL EYE INSTITUTE, NATIONAL INSTITUTES OF HEALTH, VIA NIH FLICKR

Confocal microscope image of an eye with choroidal neovascularization, which can occur in patients with age-related macular degeneration and diabetic retinopathy. Endothelial cells are shown in red, F-actin of stress fibers is shown in green and cell nuclei are shown in blue.

Five years ago, a 35-year-old convenience store manager learned he had diabetes. At first, he kept his blood sugar in check, took medications on schedule and saw an eye doctor every year. But, after a difficult divorce, he stopped his medications, relied on fast food and soon his vision began to blur.

At his next eye exam, the doctor found small retinal hemorrhages, a sign of diabetic retinopathy, or DR, in both eyes. Caught early, DR can often be slowed with laser treatment. Once complications set in, patients may face invasive surgery.

Caused by long-term high blood sugar that damages retinal vessels, DR is the leading cause of blindness worldwide. Yet, up to 90% of cases are preventable with regular screening

Michael Balas

and early treatment, according to The Lancet.

Artificial intelligence, or AI, is giving ophthalmologists new ways to detect eye disease earlier. Before patients notice blurry vision, AI can reveal cellular changes in the retina, allowing treatment to begin sooner and vision loss to be prevented altogether.

Some AI tools are already in clinics, but research continues to push the field forward. Advances in imaging and diagnosis could extend vision care even to people without access to eye specialists.

A sharper view of disease

The eye is a biological camera, capturing not only the world around us but also early signs of systemic disease. Now, AI is giving the camera an upgrade, making it a sharper, faster, more accessible diagnostic tool that can reveal disease before symptoms appear.

Most eye exams include fundus photography, a snapshot of the retina, optic nerve and vessels. These images form the baseline of modern vision care, and with AI, they become even more powerful. Algorithms can now analyze the photos to provide earlier, clearer answers, often with quicker, more comfortable scans.

By spotting microscopic retinal changes, AI can flag DR long before symptoms, speeding treatment and improving outcomes. Beyond diabetes, AI scans are also showing early signs of cardiovascular, hypertensive and even neurological disease.

Equity in eye care

As populations age and systemic diseases rise, early diagnosis and long-term monitoring are more critical than ever.

According to Michael Balas, resident ophthalmologist in the department of ophthalmology and vision sciences at the University of Toronto, fundus images reveal more than vision. They reflect systemic health.

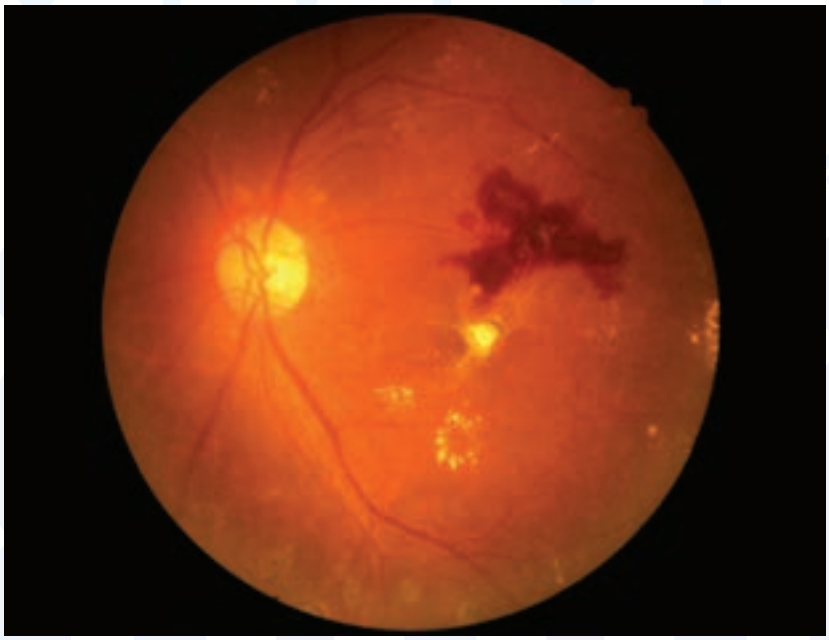
Yet the most advanced cameras remain out of reach in rural and under-resourced areas, and travelling to specialists can be difficult. In addition, low-quality images from non-specialist clinics can delay diagnoses and worsen outcomes.

Teleophthalmology helps bridge the gap: clinicians send images for remote interpretation, and specialists return diagnoses and treatment plans.

"It allows us to extend our reach far beyond the physical walls of our clinics," Balas said.

By integrating AI into everyday clinics, even older devices can generate diagnostic-quality scans, a step toward earlier, more equitable care.

Had such AI-powered screening been available at his primary-care



Fundus photograph of a diabetic retina exhibiting hallmark hemorrhages and microvascular damage characteristic of diabetic retinopathy, emphasizing the critical need for early detection and intervention.

office, the manager from the story's opening might have been flagged months earlier, sparing him invasive surgery.

Sharpening the picture

Fluids inside the eye distort light, adding noise to optical images. To correct this, researchers use adaptive optics, or AO, a technology originally developed in the 1950s to sharpen telescope views of distant stars.

An AO-assisted retinal imager uses a deformable mirror to cancel distortions, producing corrected images captured by a camera. Paired with AI, AO reduces scan times from hours to minutes while preserving cellular detail.

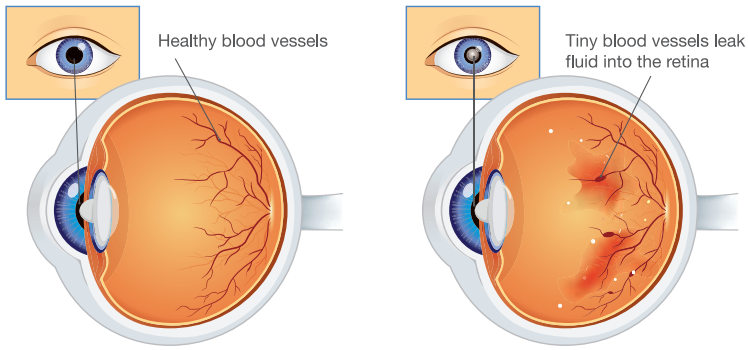
Researchers at the National Eye Institute, or NEI, at the National Institutes of Health developed an AI method that extracts more data from fewer low-quality AO images, cutting analysis time from days to hours.

"So many people think about AI as a tool that you apply after the images (are collected)," Johnny Tam, an NEI senior investigator, said. "We're thinking about AI as part of the whole imaging procedure."

Tam and Joanne Li, a biomedical engineer at NEI's Clinical and Translational Imaging Section, built a system that recovers features of the retinal pigment epithelium, or RPE, from poor-quality scans. RPE cells nourish photoreceptors, which support vision and are disrupted in many eye diseases. Seeing cell-level changes early can change the course of care.

"Medical imaging is a whole is a tissue-level technology," Tam said. "The ability to go in and look at individual cells is a game-changer."

The NEI team also developed P-GAN, an AI model that sharpens AO optical coherence tomography, which images 3D retinal structures at the cellular level. Instead of collecting hundreds of 3D images to reduce noise, P-GAN cleans them computationally, even improving results from low-cost cameras.



NORMAL EYE

EYE WITH RETINOPATHY

"It can supercharge an ordinary device and make it even more helpful to both the scientists and clinicians," Li said. "It's not about replacing. It's about assisting."

Tam hopes similar tools will one day deliver real-time insights during routine exams.

Joanne Li

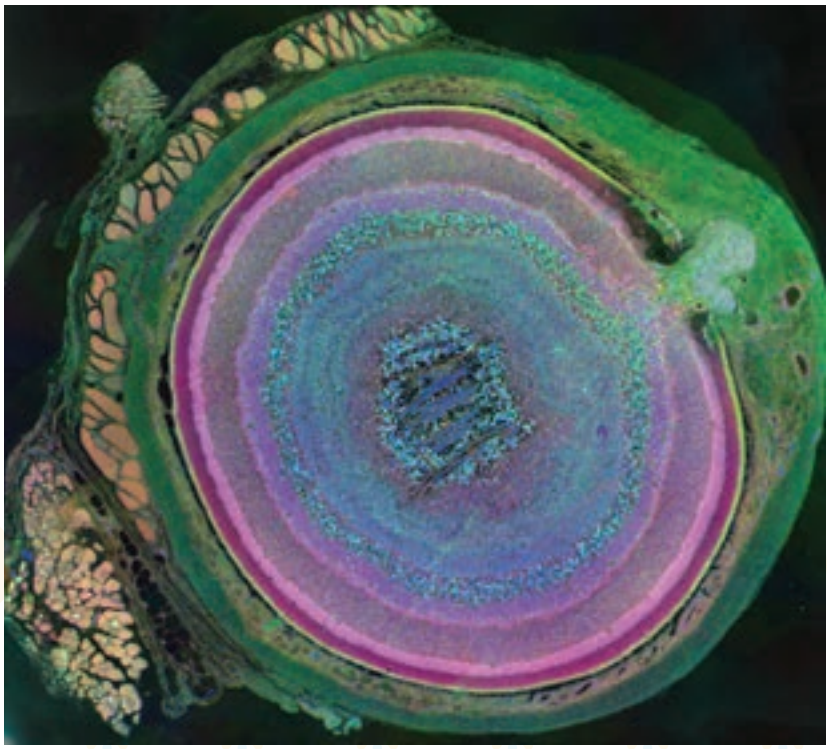
Johnny Tam

Speeding up the scan

The Tam lab used AI to collect fewer images with high-quality results. However, to see some features of the retina, many images at different angles of light refraction are necessary.

At Duke University, researchers developed an open-source AI system that separates many angles of captured light into individual images, boosting the speed and accuracy of retinal scans.

"The difference between this system and a system developed on mice is that we can put a mouse in front of an imager for five hours," Sina Farsiu, a professor of biomedical engineering and director of the Vision and Image Processing Laboratory at Duke, said. "A patient in their 50s or 60s wouldn't be able to sit in front of a bright light for a long time."



NATIONAL CENTER FOR MICROSCOPY AND IMAGING RESEARCH VIA NIH FLICKR

Laser scanning microscope image of a healthy mouse retina in the optic fiber layer. This layer is responsible for relaying information from the retina to the brain and was fluorescently stained to reveal the distribution of glial cells (green); DNA and RNA in the cell bodies of the retinal ganglion neurons (orange); and their optic nerve fibers (red); and actin in endothelial cells surrounding a prominent branching blood vessel (blue).

Trained on thousands of retinal scans, diagnostic AIs learn patterns of health and injury and flag who needs urgent referral.

To overcome that challenge, Farsiu's team combines machine learning and medical imaging into a technique called deep compressed AO scanning light ophthalmoscopy, or DCAOSLO. The technique combines adaptive optics with machine learning, using multiplexed light signals to capture single-cell detail in a fraction of the time, cutting acquisition by nearly 100-fold.

Adaptive optics sharpens images to the single-cell level, while AI reconstructs missing detail, much like wiping condensation from a lens. By combining the two, DCAOSLO delivers high-resolution images in minutes instead of hours, with less strain on patients.

The result: faster scans, earlier detection and greater comfort for patients.

Farsiu hopes that DCAOSLO will reshape screening workflows, noting that open-source AI is already "leveling the playing field" by squeezing more performance from affordable cameras.

"You can detect diseases at a much earlier stage, so your treatment will hopefully have a higher efficacy," Farsiu said.

AI in the clinic

Trained on thousands of retinal scans, diagnostic AIs learn patterns of health and injury and flag who needs urgent referral.

A clinician takes retinal pictures with whatever camera is available; then AI analyzes the images against large databases, spotting subtle leaks, vessel damage or cell loss that the human eye might miss.

One clear example is LumineticsCore, the first AI software approved by the U.S. Food and Drug Administration to detect and diagnose more than mild DR. In primary-care pilots, trained staff captured two images per eye. From just four retinal images per patient, the AI correctly identified nearly all patients with and without DR, in seconds.

The payoff: even if a primary-care provider isn't comfortable reading retinas, the AI tells them who to refer, shifting detection earlier and expanding access.

For patients like the 35-year-old store manager diagnosed with DR, such tools could mean earlier referral and treatment, before vision loss becomes irreversible.

Beyond diabetes

Ophthalmologists are uncovering deeper links between eye structure and systemic disease.

"Linking structural biomarkers with genetic phenotypes is transforming how we stratify and manage patients,"

Jay Chhablani, director of clinical research at the University of Pittsburgh Medical Center and a professor of ophthalmology, said.

Chhablani studies the choroid, the eye's vascular layer, where early signs of diabetes, hypertension and cardiovascular disease often appear in small vessels and pericytes before they show in larger vessels near the heart.

Early, accurate diagnosis of progressive conditions such as age-related macular degeneration and inherited retinal diseases can prevent vision loss. Connecting imaging biomarkers, like geographic atrophy or photoreceptor loss, to specific diseases speeds diagnosis and guides treatment.

"A key challenge is translating complex imaging data into clinically actionable insights," Chhablani said.

By tracking AI-derived biomarkers over time, clinicians could predict disease progression or treatment responses even outside the clinic. He envisions home-based imaging and remote monitoring extending care to underserved populations.

Standardized AI tools can help by improving reproducibility and clinician confidence and supporting clinical trials that track response to gene and cell-based therapies.

"Ultimately, we aim to integrate imaging, genetics and function into a single, personalized disease model," Chhablani said.

Keeping the patient in focus

Loss of vision reshapes quality of life. Blindness progresses on a spectrum — from seeing normally, to seeing differently, to losing sight altogether.

Halting that progression requires early detection and continual monitoring. When patients receive sight-preserving treatments, regular exams confirm they're working.

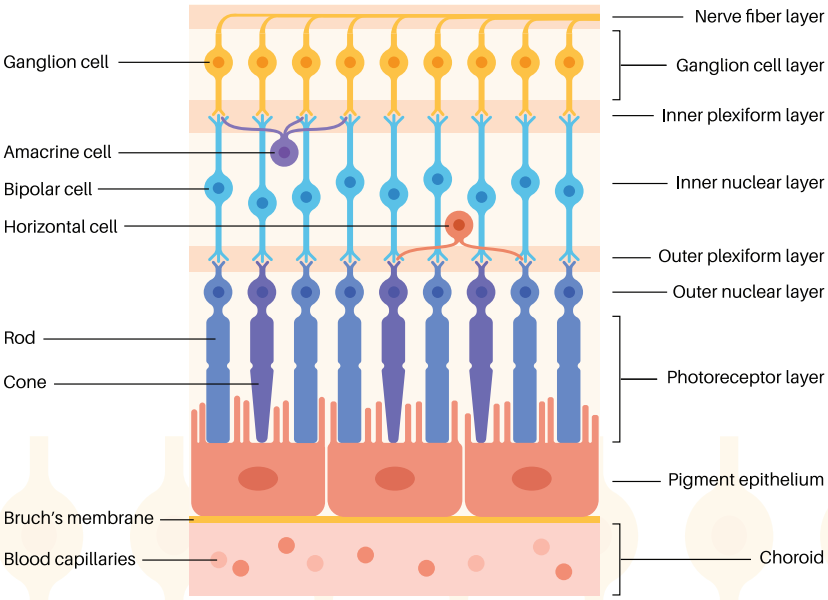


Diagram showing various cell types that make up the retina

Whether AI clarifies low-quality images for remote review or flags patients for referral, the technology keeps researchers, clinicians and patients connected. Behind it all, Balas said, "the human element is still the optical nerve that keeps everything connected."

The eye has always been a kind of camera, reflecting both vision and health. With AI, that camera is becoming sharper, faster and more accessible. For the millions worldwide at risk of diabetic retinopathy, including store clerks and CEOs, the promise of AI is not just sharper images but clearer futures.

"In ophthalmology, the future is brilliant," Balas said. "But the present, where pixels meet people, is what gets me out of bed."

Jay Chhablani

Inayah Entzminger is an ASBMB Today columnist.

Teaching AI to listen

How natural language processing is reshaping clinical genomics

By Meghana Devineni

Our models combine classic machine learning methods ... with domain-specific rules and genetic lexicons.

When I entered the world of clinical genomics, I expected long days parsing through journal articles, tidying spreadsheets of patient data and running Python scripts late into the night. I was prepared to code, not to listen.

That changed when I began working with clinical notes — messy, personal and inconsistent footprints left by physicians. Buried in them were powerful clues: a family history of breast cancer, a *BRCA1* mutation, a hesitant mention of a diagnosis. These fragments shaped diagnosis, care and discovery.

The problem? These clues are easy to miss. Manually reviewing thousands of records is slow, subjective and error-prone. That's where artificial intelligence, or AI, specifically natural language processing, or NLP, began to change my work.

As a bioinformatics graduate student at Michigan Medicine, I build hybrid NLP systems that bridge structure and storytelling — algorithms trained to "read" closely, knowing that "positive" doesn't mean hopeful and that "no history" could be a red flag.

Our models combine classic machine learning methods, like conditional random fields, with domain-specific rules and genetic lexicons. The rules, based on medical knowledge, prevent errors like flagging "no family history of breast cancer" as a risk. The lexicons act as specialized dictionaries of gene names, mutations and terms that help the system extract key variants, inheritance patterns and clinical details from messy notes.

The result: faster, more consistent identification of patients who may benefit from genetic testing or clini-

cal trial enrollment. For example, if a note states, "Mother diagnosed with breast cancer at 42; patient reports positive *BRCA1* mutation," our system flags this as a high-risk case and alerts clinicians to recommend genetic counselling or appropriate clinical trials. This cuts delays, reduces subjectivity and helps ensure fewer patients slip through the cracks.

What surprised me most wasn't the power of these tools, but how they've challenged my assumptions. What does "accuracy" mean in medicine? What errors are acceptable? In clinical NLP, a mislabel isn't minor — it means someone gets missed. If the system misreads "no history of colon cancer" as "history of colon cancer," a patient might face unnecessary testing, or worse, real risks could be overlooked. In genetic medicine, such errors can delay diagnosis and treatment with serious consequences.

There's still a long road ahead. I'm exploring how to move beyond extraction toward interpretation. Can we build models that not only find genetic risks but summarize them meaningfully? Could AI reveal disparities in access to genetic counseling or care?

AI hasn't replaced my work — it's reshaped it. It's made me more careful, collaborative and curious. In a field where the data is deeply human, the greatest thing AI has taught me is how to listen.

What surprised me most wasn't the power of these tools, but how they've challenged my assumptions.

Meghana Devineni is a bioinformatics graduate student at the University of Michigan, where she develops computational tools to analyze genetic and clinical data.

How AlphaFold transformed my classroom into a lab

By Sarmistha Ghosh

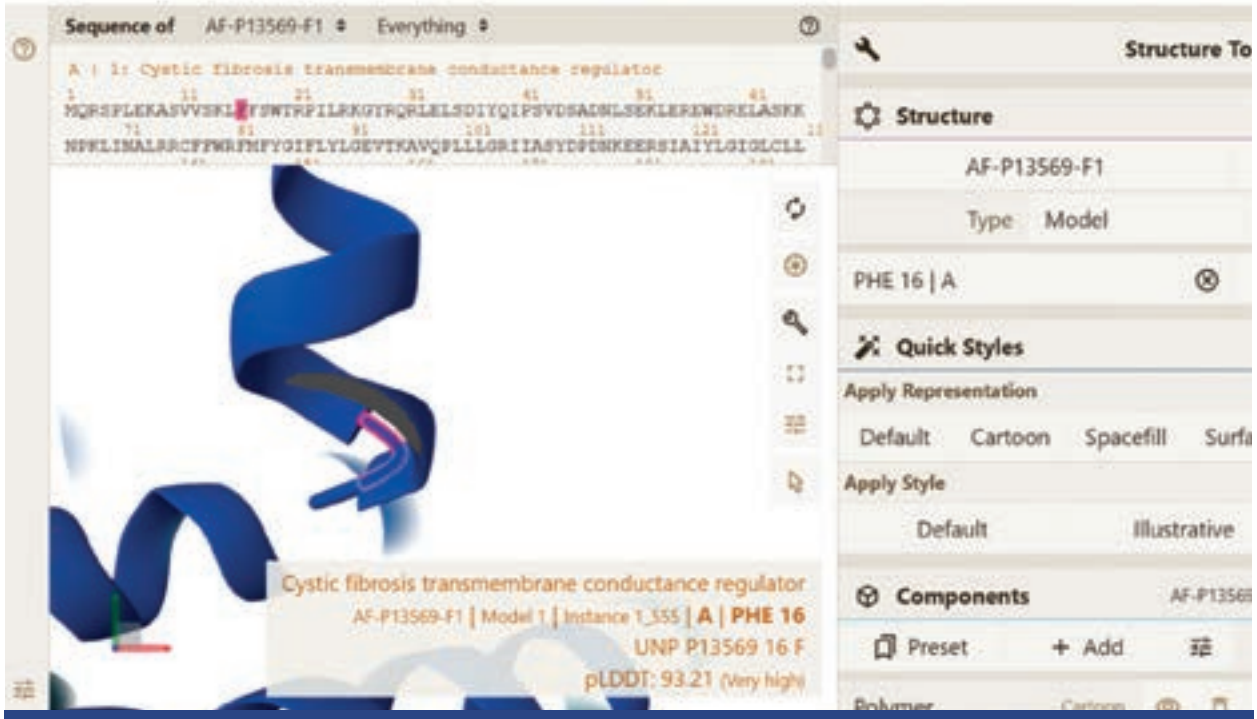
I first heard about AlphaFold, not in a lab, but in my high school Advanced Placement biology classroom. Colorful pipe cleaners, beads and half-finished 3D kits cluttered the desks, remnants of our best attempt to mimic the complexity of protein folding.

South Mecklenburg High School Advanced Placement biology students, Reese Briggs, Ella Canty, Katherine LiBrizzi and Kennady Benton, examine microcentrifuge tubes in a blue-light transilluminator with their teacher, Sarmistha Ghosh, in the Kirill Afonin lab at the University of North Carolina at Charlotte.

My students grasped that protein shape dictates function, but the real, atomic-level structures felt out of reach. Then I discovered AlphaFold's free online database. With a few clicks, we could enter a protein sequence, like insulin or the cystic fibrosis protein CFTR, and view a highly accurate 3D structure within minutes. Watching my students spin these models on screen felt like stepping into the future of science education.

For decades, protein folding research meant years of work using costly X-ray crystallography or cryogenic-electron microscopy. AlphaFold changed that. Trained on millions of known structures, it predicts a protein's shape from sequence alone — letting my students explore complex structures with nothing more than an internet connection.

Our first "aha" moment came during our cystic fibrosis unit. Students compared the normal cystic fibrosis transmembrane conductance regulator, or CFTR, structure to the ΔF508 mutant, the most common disease-causing variant. They saw how a single missing amino acid disrupted folding, and their eyes lit up as they were watching, in 3D, how a tiny molecu-



Structural comparison of normal CFTR and ΔF508 mutant as predicted by AlphaFold2.

lar error causes devastating disease.

That mishappen protein disrupts salt and water balance in the lung leading to thick mucus, chronic infections and breathing difficulties. For many, it was the first time they could visually connect a DNA mutation to the molecular mechanics of illness.

AlphaFold became more than a research tool; it was a bridge letting students see the intersection of genetics, chemistry and health. Their questions grew bolder: Could artificial intelligence, or AI, predict how a CRISPR/Cas9 edit alters folding? Could it guide drug design? Suddenly, our classroom felt less like a lecture hall and more like a research lab.

AlphaFold changed my view of AI. No longer a black box in distant computational labs, it became part of my teaching toolkit. It put real research in my students' hands.

AI didn't replace the bench; it gave it meaning. It showed my students why proteins fold, why misfolding matters and how modern tools can tackle age-old questions. Now they see AI not as hype, but as a tool that can help improve lives when used wisely.

The challenge is ensuring it enhances, not eclipses, the curiosity and creativity that make us human. AlphaFold showed me that AI can spark a lifelong love for science in the next generation.

AI didn't replace the bench; it gave it meaning.

Sarmistha Ghosh is a high school educator at Hawthorne Academy of Health Sciences in Charlotte, North Carolina.

AI in the lab: The power of smarter questions

By Miguel Minaya

A couple of years ago, artificial intelligence felt like a futuristic buzzword, intriguing but not yet part of my research reality. That changed in late 2022, when ChatGPT-3.5 was released, and I thought, "Why not give it a try?"

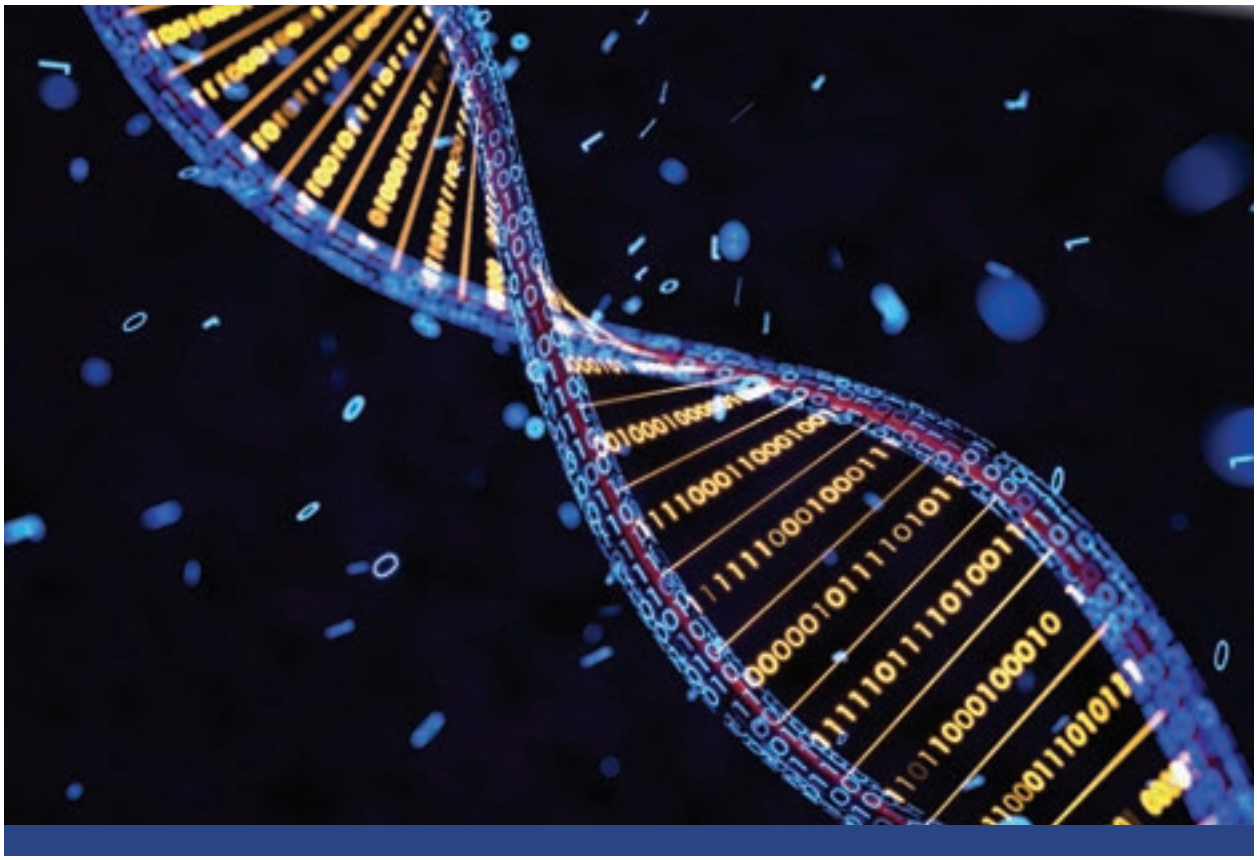
The first time I used artificial intelligence, or AI, was at a conference. The speaker mentioned an intriguing paper, and mid-talk, I used an AI tool to query the PDF directly. To my surprise, it pulled up exactly what I needed. Since then, I've hit my stride with these tools, and I'm still amazed by just how much they can do.

To be clear, AI isn't magic. It can't replace critical thinking, deep reading or lab work. But used thoughtfully, it can make a real difference.

Today, dozens of AI tools are available, many with free accounts, and each excels in different areas. Some, like Writefull or ChatGPT, excel at fine-tuning writing by adjusting tone, tightening sentences and improving clarity. Others, like AskYourPDF or ChatPDF, can dissect dense scientific papers, pulling out key elements like the main hypothesis, sample size or even the specific statistical methods used.

I often test the same prompt across platforms just to compare interpretations. The variation is surprising, and, honestly, a little fun. Sometimes I even ask AI to merge the results into one tidy summary.

For technical tasks, AI is a serious time-saver. Need to fix a command-line error or learn a new Windows trick? It's like having a patient,



on-call tutor.

Not long ago, I needed a free C++ code editor and compiler, a search that once took over half an hour of sifting through forums and reviews. Instead, I asked ChatGPT, and within seconds, it returned a curated list of options with pros and cons. That kind of time saved adds up quickly.

In my research, I study gene expression patterns to better understand brain diseases like Alzheimer's. Mapping gene functions once meant hours of cross-referencing databases and papers. Now, with tools like Perplexity AI or Elicit, I can get reliable, peer-reviewed summaries in seconds. And yes — I always double-check the sources. That's key.

Of course, AI isn't perfect. It can sound confident while being completely wrong. It reflects the biases in its training data and, if unchecked, can introduce errors into your work. That's why I always verify outputs and stick to platforms that cite sources. I constantly remind myself that AI should assist, not replace, the scientific method.

Ultimately, AI's value hinges on asking the right questions. Clear, well-crafted prompts yield the best answers. But it's our critical thinking, deep reading and domain expertise that keep results grounded. With curiosity and a healthy dose of skepticism, AI becomes a powerful collaborator that amplifies rather than replaces human insight.

I always verify outputs and stick to platforms that cite sources ... AI should assist, not replace, the scientific method.

Miguel Minaya is an assistant professor at Washington University in St. Louis. His research integrates stem cell biology, functional genomics and bioinformatics to uncover the molecular mechanisms driving tauopathies such as Alzheimer's disease and frontotemporal dementia.

MEMBER NEWS

Maria Ahmed has been named a 2025 Barry Goldwater Scholar. The recipients of these scholarships are second and third-year undergraduates from across the U.S. Ahmed is one of 441 awardees. Ahmed is an undergraduate at the University of Rochester majoring in biochemistry. Her career goals are to earn an M.D./Ph.D., conduct translational research in biomedical science and mentor the next generation of physician–scientists.


AHMED

Itay Budin, Sarah Cohen, Ileana Cristea and **Christopher Obara** are among 14 researchers selected as Allen Distinguished Investigators by the Paul G. Allen Family Foundation.

Budin is an assistant professor of chemistry and biochemistry at the University of California, San Diego. His lab uses biophysical, engineering and chemical approaches to understand the inner workings of cell membranes and their lipid building blocks. He won the ASBMB Walter A. Shaw Young Investigator Award and the Journal of Biological Chemistry/Herbert Tabor Young Investigator Award.


BUDIN

Cohen is an associate professor of cell biology and physiology at the University of North Carolina at Chapel Hill School of Medicine. Her lab studies how lipids move within and between cells.


COHEN

Cristea, a professor of molecular biology at Princeton University, investigates the intersection of virology and proteomics. Cristea is the editor-in-chief of the American Society for Biochemistry and Molecular Biology journal Molecular & Cellular Proteomics.


CRISTEA

Obara is an assistant professor of pharmacology, chemistry & biochemistry also at the University of California San Diego. His lab focuses on understanding the complex signaling and communication that happens between organelles inside of single human cells in response to their environment.


OBARA

Bonnie L. Bassler is one of 14 scientists to receive the 2025 National Medal of Science. She was recognized for her research on the molecular mechanisms that bacteria use for intercellular communication. Bassler is a Howard Hughes Medical Institute Investigator and professor and chair of the department of molecular biology at Princeton University. Her lab focuses on quorum sensing, a process that allows bacteria to communicate using chemical signaling.


BASSLER

Steven J. Fliesler received recent awards from the University at Buffalo and the American Oil Chemists' Society, or AOCS, for his outstanding contributions to research and service and for upholding high standards of academic integrity. Fliesler received the Faculty/Staff Distinction in Academic Integrity Award for his accomplishments in promoting academic integrity. In addition, he was named the AOCS 2025 Schroepfer Medal recipient and earned the UB Stockton Kimball Award. Fliesler is a professor of ophthalmology and biochemistry at the Jacobs School of Medicine and Biomedical Sciences of the University at Buffalo. His lab described the role of the lipid intermediate pathway in glycoprotein synthesis in the human retina as well as the importance of protein glycosylation for normal retinal photoreceptor cell differentiation.


FLIESLER

Claude Gagna was recognized by the Long Island Business News as a 2025 Health Care Hero in the innovation category. Gagna is a professor of biological and chemical sciences at New York Institute of Technology. He is also an adjunct professor at the Rutgers New Jersey Medical School. His lab develops DNA and RNA technologies to study gene function, complex DNA structures, cell death and spatial genomics as well as novel diagnostics.


GAGNA

Wesley Sundquist received the American Association for the Advancement of Science Mani L. Bhaumik Breakthrough of the Year Award for his role in finding that HIV's cone-shaped capsid was a target for treatment. Sundquist is the chair and a distinguished professor of biochemistry at the University of Utah. His lab continues to study the cellular, molecular and structural biology of retroviruses, including HIV, and the roles of the ESCRT pathway in cell division. Sundquist has previously received the American Society for Biochemistry and Molecular Biology–Amgen Award. In 2021, he was named to the first class of ASBMB fellows. He has served on the ASBMB Council and previously chaired the Public Affairs Advisory Committee.


SUNDQUIST

Mark Hargrove has received the Iowa State University College of Liberal Arts and Sciences Award for Early Achievement in Departmental Leadership. Hargrove is a professor and chair of biochemistry, biophysics and molecular biology. His lab studies protein structure and function.


HARGROVE

Qing Yu received a research award from the American Society for Mass Spectroscopy. Yu is an assistant professor of biochemistry and molecular biotechnology at the University of Massachusetts Chan Medical School. His research focuses on understanding the proteotype, the complete interactive set of proteins expressed by an organism or in a cell.


YU

Ralph G. Yount

Ralph G. Yount, professor emeritus of chemistry and biochemistry at Washington State University, or WSU, died in June at the age of 92. He was a member of the American Society for Biochemistry and Molecular Biology for 58 years.

Born in 1933, Yount received his Ph.D. in biochemistry from Iowa State University and did his postdoctoral work at Brookhaven, where he was trained as one of the first chemical biologists. He was a member of the faculty of Washington State University for 44 years. His work focused on the way muscles contract, specifically the relationship between muscle protein myosin and adenosine triphosphate, or ATP. His pioneering work on the creation of an ATP analog was critical to research that eventually led to two Nobel prizes.

"Ralph was an icon at WSU, building its biochemistry department and serving as chair of chemistry multiple times," James Wells, a former doctoral student of Yount's and now a professor of pharmaceutical science at the University of California, San Francisco, said. "Ralph did so much for me and was my inspiration for science."

He received a National Institutes of Health MERIT award in 1986. In 2001, he was the first recipient of WSU's Eminent Faculty Award, the university's highest honor for excellence over an extended time. In 2003, he was among the first three faculty members named to the newly established rank of Regents Professor. He was also the first Edward R. Meyer Distinguished Professor of Biochemistry and Chemistry. In 2020, the university presented Yount with an honorary doctorate, and the Ralph G. Yount Distinguished Professorship in Sciences was established in his name.

Yount was a past president of both the Biophysical Society and the Federation of American Societies for Experimental Biology. He was also a long-time editor of the Journal of Biological Chemistry.

He is survived by two daughters.

William S. Sly

William S. Sly, professor emeritus of biochemistry and molecular biology at Saint Louis University School of Medicine, died Saturday, May 31, 2025, at the age of 92. He served on the American Society for Biochemistry and Molecular Biology Council in 2005 and 2006 and was an ASBMB member for 35 years.

Born on October 19, 1932, in East St. Louis, Illinois, Sly earned his undergraduate degree from Saint Louis University in 1953 and his M.D. from the School of Medicine in 1957. He trained in internal medicine at Washington University in St. Louis and conducted research at the National Institute of Health laboratories in Bethesda, Maryland; Madison, Wisconsin; and Paris. He served as head of the division of medical genetics at Wash U for 20 years before returning to his alma mater to become a professor and chairman of the department of biochemistry and molecular biology. Sly retired from SLU in 2014 and was named an emeritus professor.

Sly is best known for work on mucopolysaccharidosis type VII MPS-VII, a rare genetic disorder characterized by bone defects, developmental delays, intellectual disabilities and premature death. Sly identified the first patient with this disorder and helped develop the first lifesaving Federal Drug Administration–approved enzyme replacement treatment. In recognition of his contributions to the understanding of this condition, it is now referred to as Sly syndrome.

Sly won numerous awards during his career, including the Association of American Medical Colleges' Award for Distinguished Research in the Biomedical Sciences; Passano Award; Coriell Medal; Peter H. Raven Lifetime Achievement Award from the Academy of Science of Saint Louis; Distinguished Scientist Award from the Clinical Ligand Assay Society; and the Life for MPS award from the International MPS Network. He was also a member of the National Academy of Sciences.

Sly is survived by his wife of 64 years, Peggy Sly, seven children, 26 grandchildren and seven great-grandchildren.

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