

Vol. 20 / No. 8 / September 2021

# ASBMB TODAY

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

**Personal  
chemistry**

**Proteomics  
tackles  
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# 2022 ASBMB Annual Meeting

## APRIL 2–5 | PHILADELPHIA

### Together once more

The ASBMB annual meeting offers unmatched opportunities to learn from top scientists, explore new research, expand your professional circle and make yourself known in the community.

All ASBMB meeting programming — scientific symposia, spotlight talks, poster presentations, hands-on workshops, teaching and mentoring sessions, networking events and more — is designed *for scientists by scientists*.

**Start thinking about what you'll present:** Early birds who submit abstracts by Oct. 15 will be guaranteed a decision within two weeks.

**Keep an eye out for travel award criteria:** The ASBMB awards more than \$275,000 in travel funding to help its members present their work at the meeting. Applicants must be first authors on abstracts.

[asbmb.org/annualmeeting](https://asbmb.org/annualmeeting)



The ASBMB annual meeting is held in conjunction with Experimental Biology.



## NEWS

2

### PRESIDENT'S MESSAGE

*One week, twice the thanks*

3

### MEMBER UPDATE

6

### IN MEMORIAM

8

### RETROSPECTIVE

*Noboru Sueoka (1929–2021)*

10

### SOCIETY NEWS

12

### NEWS

12 *Researchers find a cell surface decorated with sugar-coated RNAs*

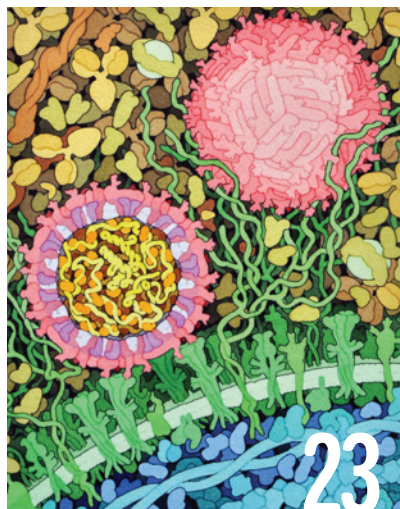
14 *An inhibitory modification in a membrane-binding protein*

16 *Journal of Biological Chemistry names new editor-in-chief*

19 *How a drug confuses SARS-CoV-2*

21 *Aggregates: Defect or protection?*

22 *From the journals*



23

## FEATURES

28

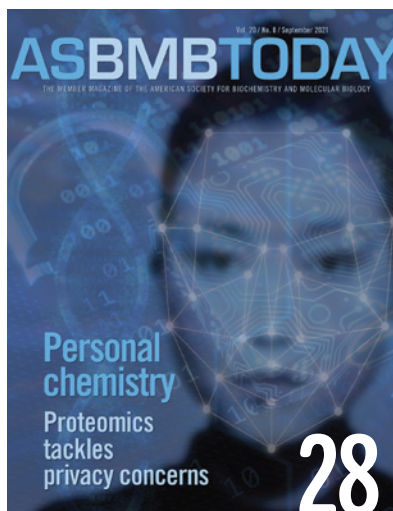
### PERSONAL CHEMISTRY: PROTEOMICS TACKLES PRIVACY CONCERNS

38

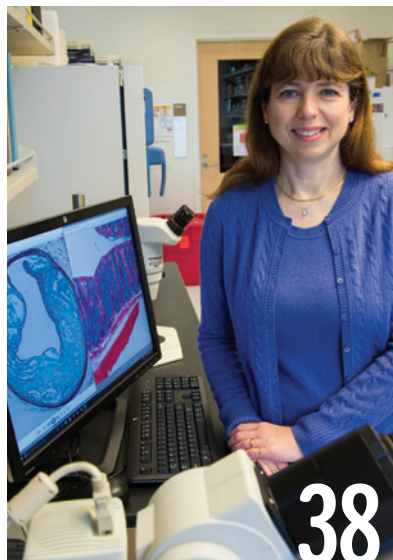
### BUILDING BRIDGES BETWEEN BASIC AND CLINICAL RESEARCH

42

### NAUTILUS FOUNDER UNSPIRALS A NEW APPROACH TO PROTEOMICS



28



38

## PERSPECTIVES

47

### HOW TO WRITE A KILLER ABSTRACT IN 10 SENTENCES

49

### US TRAVEL BANS: BIZARRE AND RUTHLESS

52

### SURVIVING AND (SOMETIMES) THRIVING AS A DEPARTMENT CHAIR

56

### GETTING THE SHOTS

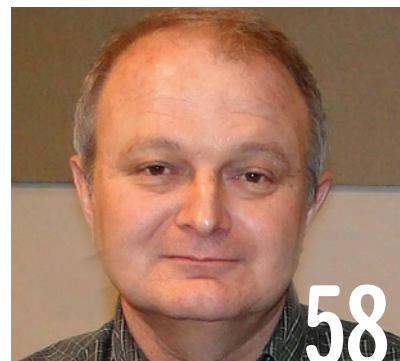
58

### FIVE QUESTIONS

*Paul Wright | A turbulent industry*



52



58

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## PRESIDENT'S MESSAGE

# One week, twice the thanks

*By Toni M. Antalis*

**W**e all have been through tough times over the past year, and COVID-19 has had a particularly disruptive effect on the research community, especially on our postdocs and graduate students. I'm taking this opportunity to ask you to join me in saying thank you to the dedicated individuals who have navigated many unexpected challenges and have stepped up to support the research community.

This month, Sept. 20-24, we celebrate both National Postdoc Appreciation Week and Peer Review Week.

Postdoc researchers are the lifeblood of our research and academic communities. Developing and nurturing their talent is critical to the generation of knowledge and innovation. During National Postdoc Appreciation Week, universities across the country will host a variety of activities and events to celebrate our postdocs and show appreciation for these dedicated individuals who make daily contributions to research and discovery.

We at the American Society for Biochemistry and Molecular Biology are thrilled to recognize the many talented and dedicated postdocs who contribute daily to the advancement of scientific research. As part of this celebration, we will host:

- Daily coffee breaks Sept. 20-23 at 1:30 p.m. EDT: Join us on Twit-

ter @ASBMB for science chat and the chance to win a cup of coffee on us!

- Wednesday, Sept. 22, at 2 p.m. EDT: Attend a webinar with Erica Gobrogge on building your personal brand, hosted by the Educational and Professional Development Committee.

- Friday, Sept. 24, at 2 p.m. EDT: #ASBMBLovesPostdocs Twitter chat about postdoc life — tell us about your good news, grumbles and best advice for those graduate students about to embark on their own postdoc journeys.

Also mark your calendars for Peer Review Week, an annual global event celebrating the central role peer review plays in maintaining the quality of scientific communication. This year's theme, "Identity in Peer Review," explores the identities that make up who we are as individuals, organizations and populations, and how diverse, equitable and inclusive peer-review processes best can be achieved.

As members of a nonprofit society dedicated to fostering science and the research community, we at the ASBMB appreciate the careful peer review by expert editorial board members of our journals, the Journal of Biological Chemistry, Molecular and Cellular Proteomics, and the Journal of Lipid Research. These

**CONTINUED ON PAGE 3**



## Johnson–Winters honored for essay in ASBMB Today

**Kayunta Johnson–Winters** received a silver EXCEL award in June from Association Media and Publishing in the category of editorial/opinion piece in a magazine for her



JOHNSON–WINTERS

essay “Being Black in the ivory tower,” published in the August 2020 issue of ASBMB Today.

A tenured associate professor in the department of chemistry and biochemistry at the

University of Texas–Arlington, Johnson–Winters is also a member of the ASBMB Minority Affairs Committee.

After police officers killed George Floyd in May 2020 sparking Black Lives Matter protests across the U.S., Johnson–Winters was moved to write about her young teenage son’s traumatic experience with gun violence and the judicial system as well as her own challenges as a Black professor on a university campus.

“While the circumstances around the article are tough, I am happy if my words were meaningful and impactful to our scientific and higher ed community,” Johnson–Winters said. “I thank the editorial staff of ASBMB Today for submitting the article, and I’m happy to share this

honor with them.”

The AMP EXCEL Awards, now in their 41st year, recognize excellence and leadership in media, publishing, marketing and communications for both nonprofit and for-profit associations. In previous years, ASBMB Today contributors honored for essays have included TL Jordan who won bronze for “What I wish people understood about being a trans scientist” in 2020, Byron Rubin who won bronze for “Up the creek without a sequence?” in 2019 and Jennifer Dubois who won gold for “Disappointed by cancer” in 2018.

## Dixon retires from UC San Diego

**Jack Dixon**, a distinguished professor of pharmacology, cellular and molecular medicine, and chemistry and biochemistry



DIXON

at the University of California, San Diego, former chief scientific officer at the Howard Hughes Medical Institute, and former president of the American Society

for Biochemistry and Molecular Biology, retired July 1.

In a 48-year research career that took him across the country, Dixon

focused on reversible phosphorylation, which controls signaling that’s important in many functions of the cell. His team discovered a protein tyrosine phosphatase in *Yersinia pestis*, the bacterium that caused black plague. The bacterium injects the phosphatase into host cells, blocking the immune response that depends on signaling through receptor tyrosine kinases.

Dixon also published the crystal structure of the tumor suppressor protein PTEN and demonstrated that it is a phospholipid phosphatase that acts on PIP3, which has implications for cancer. More recently, his group has investigated the role of a novel kinase family in biomineralization, the process by which teeth and bones are hardened.

Dixon earned his Ph.D. in chemistry at the University of California, Santa Barbara, and pursued post-doctoral research at UCSD. His first faculty appointment was in the biochemistry department at Purdue University, where he stayed for 19 years before joining the biological chemistry faculty at the University of Michigan.

In 2003, Dixon returned to UCSD to become associate vice chancellor of scientific affairs and a professor of pharmacology. In 2006, he became vice president and chief scientific officer at the Howard Hughes Medical Institute, where he spent seven years

## CONTINUED FROM PAGE 2

editorial boards are made up of scientists practicing in the research area of the manuscripts submitted, which is essential for ensuring that the published literature is kept to high standards of accuracy, rigor and reproducibility and that papers are

published based on the quality of their scientific research and contribution to advancing a field.

I extend my gratitude to these vital individuals, and I hope you all will take time to thank these important members of our research community.

**Toni M. Antalis** (TAntalis@som.umaryland.edu) is a professor of physiology at the University of Maryland School of Medicine, where she is also the associate director for training and education for the Greenebaum Cancer Center and the director of the graduate program in molecular medicine. She began her term as the ASBMB’s president in July 2020.



# MEMBER UPDATE

directing the investigator program and launching the institution's early career scientist program and collaboration awards. He returned to UCSD to focus on research in 2013.

Dixon served as the president of the ASBMB in 1996. Among his many honors are the ASBMB's William C. Rose Award in 2003 and the ASBMB–Merck Award in 2005. He is an elected member of the National Academy of Sciences, the Institute of Medicine and, as a foreign member, the Royal Society.

## Mississippi State recognizes Sparks

**Darrell Sparks**, an associate professor at Mississippi State University, has received Teacher of the Year and Excellence in Teaching awards from that university's college of agriculture and life sciences.

Sparks teaches in the biochemistry department and serves as its undergraduate coordinator, mentoring more than 500 students annually on top of a full teaching load.

"While the global pandemic exponentially changed how we approach teaching, our faculty and staff rose to the occasion to deliver a quality education to our students," interim dean of the agriculture and life sciences faculty Scott Willard said on presenting the awards.

In addition to teaching, Sparks also pursues service and research. He chaired a committee that earned American Society for Biochemistry and Molecular Biology accreditation for Mississippi State, and for the past few years he has collaborated on research into the gut microbiome of



SPARKS

giant pandas and red pandas. Because of pandas' aptitude for digesting woody, nutrient-poor bamboo, researchers hope that they might identify microbes that could be applied to generate biogas from cellulose.

## Conaway named vice provost at UT Southwestern

**Joan Conaway** has assumed the roles of vice provost and dean of basic research at the University of Texas Southwestern Medical Center in Dallas, effective July 1.

Before taking this appointment,



CONAWAY

Conaway was an investigator at the Stowers Institute for Medical Research in Kansas City, Missouri, where she studied the molecular mechanisms of RNA polymerase

II transcription in a lab she ran in collaboration with her husband, Ron Conaway.

Over the past 30 years, the Conaways' lab has studied molecular mechanisms by which transcription factors and regulatory protein complexes control multiple steps in transcription. Some of their earliest work defined and explored the roles of transcription initiation factors needed simply for RNA polymerase II to start at the right place to copy a gene into RNA. They also explored mechanisms that control the speed at which RNA polymerase elongates RNA as it copies a DNA template and revealed unexpected links between protein complexes that regulate initiation and elongation. Disruption of some of these steps in transcription can play a role in cancer and other diseases.

At UT Southwestern, Conaway

will not run a lab of her own; in an interview with a UT Southwestern publication, she said, "For a while, I've had a strong interest in trying to contribute not just to my own research program, but to building an environment that can be conducive to providing the very best research opportunities to colleagues." She plans to focus on building computational infrastructure and recruiting data scientists, and on increasing diversity in all levels of research.

Conaway earned her Ph.D. in cell biology at Stanford University School of Medicine and conducted postdoctoral research at the DNAX research institute of molecular and cellular biology in Palo Alto, California. She has served as the treasurer of the American Society for Biochemistry and Molecular Biology for the past two years.

Conaway is a former Howard Hughes Medical Institute associate investigator, a member of the National Academy of Sciences, and a member of the American Academy of Arts and Sciences. The Conaways jointly received the ASBMB – Amgen award in 1997.

## Spriggs to start lab at University of Michigan

**Chelsey Spriggs**, a postdoctoral researcher in the department of cell and developmental biology at the University of Michigan Medical School, has accepted a job as assistant professor in that department. She will begin her lab in 2022.

Spriggs, a virologist, is currently a postdoc in Billy Tsai's lab, studying polyomavirus trafficking. In her most recent publication, she described an unexpected link between viral escape from the secretory pathway into the cytosol and the disassembly of its capsid to let it invade the nucleus.



She is a member of the American Society for Biochemistry and Molecular Biology's first class of MOSAIC



SPRIGGS

scholars. MOSAIC, which stands for Maximizing Opportunities for Scientific and Academic Independent Careers, is a networking and career development program that supports a cohort of K99/R00 grant recipients as they transition from postdoctoral research into faculty positions.

Spriggs was a leader of the inaugural Black in Microbiology week; she and her co-organizers recently published an account of how that project launched the Black Microbiologists Association. She earned her Ph.D. at Northwestern University studying how human papillomavirus infection leads to tumorigenesis, and her bachelor's degree at Michigan State University.

## Emr receives Shaw Prize

**Scott Emr**, a professor of molecular biology and genetics at Cornell University and director of the Weill Institute, has received the 2021 Shaw Prize in Life Science and Medicine. He is being honored for discovering the endosomal sorting complexes required for transport, or ESCRT, pathway, using the awesome power of yeast genetics.

There are five ESCRT protein complexes, each of which enables membrane budding inward from the outer surface of the endosome to form intraluminal vesicles in what becomes the multivesicular body, whose contents are later degraded in the lysosome. Using yeast knockouts and loss-of-function mutants, Emr's

team has identified over a dozen ESCRT proteins and showed that they recognize information encoded in lipid phosphorylation, recognize and coordinate ubiquitin-bound cargo, and pinch and bend membranes into new shapes so that intraluminal vesicles can form. ESCRT-directed membrane bending is now recognized as a universal mechanism used by cells in other ways, such as repair of membrane damage, completion of cytokinesis and pruning of neuronal axons during brain development, and is also exploited by viruses, such as HIV, to bud and escape from host cells.

Emr earned his Ph.D. in molecular genetics from Harvard Medical School. Prior to joining the Cornell faculty, he held positions at the University of California, Berkeley, the California Institute of Technology and the University of California, San Diego School of Medicine, where he was also a Howard Hughes Medical Institute investigator. He is a member of the National Academy of Sciences, the American Academy of Arts and Sciences and the American Academy of Microbiology.



EMR

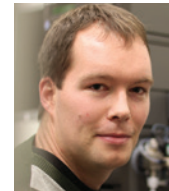
Hong Kong. Each consists of a \$1.2 million award and an award lecture. The prizes were established in 2002 by the late Run Run Shaw, an entertainment mogul and philanthropist.

## Cejka joins EMBO

The European Molecular Biology Organization, or EMBO, has

announced the election of 64 new life scientists to its ranks. One of the new members is **Petr Cejka**, a group leader at the Institute for Research in Biomedicine in Bellinzona, Switzerland, and a professor at the Università della Svizzera italiana.

In his lab, Cejka studies homologous recombination as a mechanism



CEJKA

for repairing double-stranded DNA breaks. Homologous repair pathways, which use the second, homologous copy of a damaged re-

gion to restore the broken section, are important for maintaining genome integrity and are involved in meiotic recombination and some types of genome editing. His team focuses on the earliest steps in the recombination pathway, when DNA around the break site is trimmed back to enable strand exchange proteins to bind. They also study proteins that separate Holliday junctions, hybrid DNA structures formed during recombination, back into separate DNA strands.

Cejka received the Ernst T. Jucker award for basic cancer research from the eponymous foundation in 2015 and the Friedrich Miescher Award for Swiss biochemists under 40 in 2017.

Before coming to work at the Institute for Research in Biomedicine in 2016, Cejka was a professor at the University of Zurich. He earned his Ph.D. at the University of Zurich, studying DNA mismatch repair, and conducted postdoctoral research in double-strand break repair at the University of California, Davis.

EMBO's goal is to promote research in life science and enable international exchange between scientists. The organization has over 1,800 members, who nominate and elect new members annually.

## John Turk



John Turk, a pioneer in mass spectrometry research, a faculty member for more than 40 years at Washington University School of Medicine in St. Louis and a member of the Journal of Biological Chemistry editorial board, died May 26 after a brief illness. He was 73.

Born Jan. 17, 1948, and raised in St. Louis, Turk earned both an M.D. and a Ph.D. at WUSTL before his internship at Billings Hospital at the University of Chicago, residency at Barnes Hospital at WUSTL and a fellowship at Vanderbilt University. For most of his time on the WUSTL faculty, he directed the department of medicine's National Institutes of Health-funded mass spectrometry core facility, supporting researchers who sought to analyze complex molecules associated with human diseases. He was also a clinician and taught toxicology to pathology residents.

In his research, Turk defined key mechanisms of phospholipid signaling that contribute to diabetes. Using tandem mass spectrometry, he determined molecular structures of complex lipids such as phosphatidylcholines. As one of the discoverers of iPLA2b, a phospholipase enzyme involved in insulin secretion and survival of pancreatic beta cells, Turk helped demonstrate that this enzyme is involved in cell proliferation, cell death, membrane biochemistry and medical conditions including infertility, metabolic syndrome, chronic inflammation and neurodegenerative disorders.

Turk was in his third term as a JBC editorial board member. He was an elected member of the American Society of Clinical Investigation and the Association of American Physicians.

Colleagues recall that he gave persimmon bread to faculty and staff every holiday season. Clay Semenkovich, a WUSTL professor, said in an obituary, "He was a great scientist, a devoted teacher and a compassionate mentor who would always stop whatever he was doing to help other scientists."

Turk is survived by his companion, Carol Thompson; ex-wife, Alice Turk; daughter, Amy Turk (Justin Prien); son, Andrew Turk; brother, Jim Turk; and three grandchildren.

## Shozo Yamamoto



Shozo Yamamoto, an emeritus professor at the University of Tokushima in Japan and a researcher focused on enzymology and the biochemistry of lipids involved in inflammation, died April 18 in Kyoto. He was 86.

Born May 12, 1933, in Osaka Prefecture, he earned an M.D. from Osaka University School of Medicine in 1960, then joined the Kyoto University lab of Osamu Hayaishi, who had discovered the first oxygenase in 1955. In the 1960s, while visiting Sweden, Yamamoto learned about prostaglandin research in Sune Bergström's lab at the Karolinska Institute. He then focused on steroid biosynthesis enzymes with Nobel laureate Konrad Bloch at Harvard University.

Back in Hayaishi's lab, he began to study enzymes involved in prostaglandin biosynthesis, contributing to the purification and characterization of cyclooxygenase 1. In a tribute in the Journal of Lipid Research, colleagues say that he also contributed to the Ono Pharmaceutical Company's research and development of commercial prostaglandins, many of which can be used today for a variety of medical purposes including inducing labor and treating hypertension.

In 1979, Yamamoto became a professor in the biochemistry department at Tokushima University School of Medicine; over the years, his group contributed to study of an endocannabinoid lipid known as anandamide and of inflammation-related transcriptional induction of the inducible enzyme cyclooxygenase 2. After retiring at age 65, he worked at Kyoto Women's University for six years.

Yamamoto served on the editorial board of the Federation of European Biochemical Societies Letters and was a member of the Japanese Biology-Chemical Society, Vitamin Society Japan, Japan Society of Clinical Chemistry and the New York Academy of Sciences.

He married Ikuko Tsubaki in 1963, and they had three children: Toshitaka, Yoritaka and Yukiko. In addition to being a researcher and educator, he loved classical music, travel and history, and he enjoyed playing the piano.

"We will never forget his great contribution to lipid biochemistry," his colleagues wrote in the JLR tribute, "and the irreplaceable time that we spent with him."



## Brian Hartley



Brian Hartley, an honorary member of the American Society for Biochemistry and Molecular Biology since 1977 whose contributions to protein chemistry included the invention of new analytical methods and structural understanding of the properties of proteases, died May 3. He was 95.

Born April 16, 1926 in Lancashire County, England, Hartley attended Queens' College, Cambridge. He graduated with a degree in organic chemistry in 1947, then did two years of military service in Malta before earning a Ph.D. in biochemistry from the University of Leeds. He returned to Cambridge for postdoctoral studies and remained there in the Medical Research Council Laboratory of Molecular Biology, becoming a group leader in the protein chemistry division.

Hartley was named head of the biochemistry department at Imperial College London in 1974. He set up the Imperial Centre for Biotechnology in 1982 and was its first director. He was a founding board member of Biogen.

In addition to inventing new methods in analytical protein chemistry for identifying cysteine bridges, Hartley published the complete amino acid sequence of chymotrypsinogen-A in 1964, a time when no protein of comparable size had yet been sequenced. His interest then shifted to comparative evolutionary studies, and he developed genetic models to account for the evolutionary history of enzyme families and to produce ancestor trees. He showed that mammalian serine proteases, including the blood clotting cascade, had the same structures and mechanisms, indicating a common evolutionary origin.

Hartley was elected to the European Molecular Biology Organization and became a fellow of the Royal Society, both in 1971. A champion of young researchers, he supervised many successful Ph.D. students, among them the future Nobel laureate Gregory Winter. "He fizzed with ideas and would gesticulate ferociously with his pipe, expounding his latest brainwave or arguing science with César Milstein in the corridor," Winter recollected in a MRC LMB obituary. "... He advised me to get on with experiments and not waste time reading the latest papers, as 'if it's really important, someone will tell you.'"

## Gerhard Meissner



Gerhard Meissner, a professor at the University of North Carolina at Chapel Hill and a member of the American Society for Biochemistry and Molecular Biology for more than 40 years, died May 1. He was 84.

Born in Wilhelmshaven, Germany, on Jan. 26, 1937, Meissner received B.S. and M.S. degrees from the Free University of Berlin, then went on to earn a Ph.D. in physical chemistry from the Technical University of Berlin in 1965. He joined the UNC faculty in 1974 and was appointed professor of biochemistry and biophysics in the UNC School of Medicine in 1986.

Meissner's major research interests included determining the structure and function of ion channels and calcium signaling in cardiac and skeletal muscle. His lab used mutagenesis, Ca<sup>2+</sup> imaging and single-channel measurements to determine the molecular mechanisms underlying release channel/ryanodine receptor, or RyR1, function, with the goal of understanding the mechanisms of RyR1 channel ion conductance and selectivity, and gating by its multiple ligands, and how these processes are altered by mutations linked to muscle diseases such as central core disease and malignant hyperthermia.

Meissner was a Gosney fellow and Volkswagenstiftung fellow at the California Institute of Technology, a fellow of the Biophysical Society and an established investigator of the American Heart Association. He received continuous funding from the National Institutes of Health that included two NIH MERIT Awards from 1990 to 2000 and 2010 to 2021.

He is survived by his wife, Elizabeth M. Wilson, and sons, Eric G. Meissner and Geoffrey W. Meissner.

# Noboru Sueoka (1929–2021)

By Murray H. Brilliant & William G. Quinn

**N**oboru Sueoka died May 14. He was an early and active contributor to studies aimed at understanding the genetic code; the role of tRNA; development of a method of fractionating tRNA (together with his wife, Tamiko Kano–Sueoka); the variation in base composition and evolution of DNA sequences; and the mapping of bacteria genes. He made widely known contributions to our understanding of DNA replication. Indeed, he coined the term “origin of replication.” Using specific gene-transformation ratios and enlightened math, Sueoka showed that *Bacillus subtilis* cultures in exponential growth duplicate their chromosomes with replication forks traveling with the same velocity, all starting from the same chromosome origin. Using *Chlamydomonas*, he also found out that during meiosis two rounds of replication are all semiconservative.

Sueoka was born April 12, 1929, in Kyoto, Japan, the son of Masashin and Ayako (Nishida) Sueoka. By middle school, he had his mind set on becoming a geneticist, influenced by his uncle, a plant geneticist. In grade

**He made widely known contributions to our understanding of DNA replication. Indeed, he coined the term “origin of replication.”**



COURTESY OF MURRAY H. BRILLIANT

**Building on his continuing work on prokaryotic models, Sueoka aimed at understanding how the nervous system, particularly the mammalian brain, achieved its complex wiring and function.**

school, he often was lost in thought and was punished for not paying attention. This trait persisted for the rest of his life and enabled him to think outside of the box.

After receiving B.S. and M.S. degrees from Kyoto University, Sueoka earned his Ph.D. from the California Institute of Technology. As a postdoc at Harvard, with three advisors, James Watson, Paul Doty and Paul Levine, he discovered the correlation between guanine-cytosine content and cesium-chloride density and immediately used this finding to discover the first satellite DNA, the nearly pure adenine and thymine sequences from crab.

As a lab head for two years at the

University of Illinois and then for 10 years at Princeton, he carried out seminal studies on bacterial chromosome replication in the spore-forming bacteria *Bacillus subtilis*. This creature had two convenient properties. First, it could be genetically transformed with pure DNA — rare in those days. Better, its spores contained DNA without replication forks; when the spores germinated, the chromosomes replicated synchronously, starting from the same location on all chromosomes. Sueoka, with co-workers, verified this synchronous replication using ratios of gene-transformation efficacies (they doubled, in a fixed order, starting with *ade6* at the origin, as the chromosome fork passed the



loci). During rapid growth conditions, the chromosomes replicated, in his words, “dichotomously,” with each branch behind the first fork having daughter forks. Finally, his lab showed that the replication origin stayed permanently attached to the membrane throughout the cell cycle.

Building on his continuing work on prokaryotic models, Sueoka aimed at understanding how the nervous system, particularly the mammalian brain, achieved its complex wiring and function. His approach was twofold. First, it was cell-oriented; brain tumors were induced, and cells from those tumors were selected for neuronal and glial characteristics, one line of which, RT4, had stem cell properties and could differentiate to either, leading to the discovery of markers and cell-fate switch mechanisms. Second, he examined overall gene expression in these cell lines and in whole organs. This led to one of the first estimates of the number of genes in mammals and insight into the role of polyadenylation of messages.

Over his 50-year career, Sueoka was known for his unfailing kindness, his gentleness, his openness and his sincere dedication to his colleagues, postdocs, technicians and graduate and undergraduate students. He was an exceptional mentor. He and his wife, known as Tami, provided a pleasant laboratory environment where everyone felt safe and understood. He encouraged members of their team to work hard, succeed in their projects and, most importantly, be happy and enjoy life. He was always ready to talk to students about their projects and valued their input. He encouraged students to think independently and made them feel their ideas were

valued. One could ask him any question (or complain to him), and he would consider it seriously. He was a fundamentally curious person and had deep interest in understanding other cultures.

Sueoka shared his wisdom and observations of the human experience and what it meant to be a scientist. He distinguished between two approaches to science research: a riskier approach that aims to discover fundamental and novel processes, and a less risky path that aims to nail down the details of these previously discovered processes. He chose and valued the former approach.

One of the earliest Japanese scientists to study and have a career abroad, Sueoka’s scientific courage and personal independence helped transform Japanese biology from a feudal and technique-oriented discipline after World War II to its current position in the first rank of scientific cultures. He was not a pushy person; he did this by his pioneering example and by directly encouraging independence and initiative in others.

In addition to his seminal contributions to molecular biology, Sueoka will be remembered as an enthusiastic skier, mushroom hunter and trout fisherman, passing on his love for these activities to his students and postdocs. He will be missed by his many postdocs and graduate and undergraduate students. In particular, we both cherish our time in his lab. He will be especially missed by his wife and research partner, Tami, and by their daughter, Miki Sueoka.

Sueoka was an assistant professor in the microbiology department at the University of Illinois at Urbana-Champaign from 1960 to 1962. His career led him to Princeton University, where he was an associ-

**In addition to his seminal contributions to molecular biology, Sueoka will be remembered as an enthusiastic skier, mushroom hunter and trout fisherman, passing on his love for these activities to his students and postdocs. He will be missed by his many postdocs and graduate and undergraduate students.**

ate professor from 1962 to 1965, a professor from 1965 to 1969, and held an endowed professorship from 1969 to 1972, all in the biology department. He moved to the newly created molecular, cellular and developmental biology department at the University of Colorado, Boulder, where he was a professor from 1972 to 1997. He then served as an adjunct professor at the University of California, Irvine, from 1997 to 2002.

Sueoka was a member of the American Academy of Arts and Sciences, a fellow of the American Association for the Advancement of Science, and a Guggenheim fellow. He received a Waksman Award from the Theobald Smith Society and a fellowship from the Japanese Foundation for Promotion of Cancer Research.

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**Murray H. Brilliant** (mbrilliant@wisc.edu) is a senior scientist in the Waisman Center at the University of Wisconsin–Madison.

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**William G. Quinn** (cquinn@mit.edu) is an emeritus professor of neurobiology at the Massachusetts Institute of Technology.

## New science outreach and communication grant

The ASBMB Science Outreach and Communication Committee has established a new \$1,000 grant to support public-engagement activities by society members. These activities may be conducted in person or in a hybrid or fully virtual format. All members are eligible to apply. Applications will be accepted from Aug. 15 through Oct. 15. Learn more at [asbmb.org/soc-grant](http://asbmb.org/soc-grant).



## Organize a virtual event. We'll help!

You pick the scientific topic and speakers, and we'll manage the rest. We'll market the event to tens of thousands of contacts, handle registration and abstract collection, and present the digital event live to a remote audience. Propose an event at [asbmb.org/meetings-events/propose-event](http://asbmb.org/meetings-events/propose-event).

## Accreditation applications due Oct. 1

To date, more than 100 bachelor's degree programs in BMB and related disciplines across 38 states have received accreditation from the ASBMB. Accreditation provides a national, independent tool for evaluating program outcomes. By earning ASBMB accreditation, programs demonstrate a commitment to the highest standards of quality and innovation in BMB education. Applications for the next round of accreditation are due Oct. 1. Learn more at [asbmb.org/education/accreditation](http://asbmb.org/education/accreditation).

## New publications department intern

Heather Bisbee joined the publications team as an intern in June. Bisbee is a Ph.D. candidate at the University of Massachusetts,



Amherst, in Eric R. Strieter's lab. She studied English as an undergrad and is pursuing a career in scientific publishing. She'll be working on the Journal of Biological Chemistry's review articles. She can be reached at [hbisbee@asbmb.org](mailto:hbisbee@asbmb.org).

## Farewell and thanks, Lila Gierasch!

Lila Gierasch of the University of Massachusetts Amherst completed her five-year term as editor-in-chief of the Journal of Biological Chemistry on June 30. Under her leadership, the journal has enjoyed several major achievements, including the launch of JBC Reviews, improvements in the author experience, the move to open access and a jump in impact factor. In July, Alex Toker, associate director for the Cancer Research Institute at Beth Israel Deaconess Medical Center and professor of pathology at Harvard Medical School, was named the next editor-in-chief. (See article on page 18.)



## Fast-track your abstract

Hate waiting months to hear if your meeting abstract has been accepted?

The ASBMB's priority consideration program is for you! Submit your abstract for the 2022 ASBMB Annual Meeting in Philadelphia by Oct. 15, and you are guaranteed a decision within two weeks. Learn more about the 2022 ASBMB Annual Meeting at [asbmb.org/meetings-events/2022-annual-meeting](http://asbmb.org/meetings-events/2022-annual-meeting).

## Let us advertise your postdoc positions

Posting on the ASBMB job board is free for members. Please use this platform to get the word out about your open positions. Go to [careers.asbmb.org](http://careers.asbmb.org).

## RECENT ADVOCACY ACTIVITIES

**Weighing in on ARPA-H:** In response to President Joe Biden's proposal to create a new federal agency focused on significant health challenges, the ASBMB submitted comments in July to U.S. Reps. Diana DeGette, D-Colo., and Fred Upton, R-Mich. The society recommended (1) keeping funding for the Advanced Research Projects Agency for Health separate from funding for the National Institutes of Health and (2) requiring the agency to solicit feedback from the scientific community when it develops its strategic plan.

**Defending scientists with ties to China:** U.S. Reps. Jamie Raskin, D-Md., and Judy Chu, D-Calif., held a roundtable in June titled "Researching While Chinese American: Ethnic Profiling, Chinese American Scientists and a New American Brain Drain." The ASBMB submitted testimony outlining its concerns that the U.S. Department of Justice's targeting of scientists with ties to China has had a chilling effect on international collaboration.

Read these materials and others at [asbmb.org/advocacy/letters](http://asbmb.org/advocacy/letters).



### *New manager of diversity, equity and inclusion programs*

Ciarra Smith joined the society as manager of diversity, equity and inclusion programs in August. Smith previously was a postdoc in diversity, equity and inclusion at the University of Massachusetts Medical School, where she earned her Ph.D. in biomedical sciences. "I am very passionate about diversity, equity and inclusion, especially as it pertains to science," Smith said. "I am most excited about leading the charge of DEI initiatives within the ASBMB and working with the fantastic staff and members on providing an environment that is diverse, equitable and inclusive. Together we can make a positive impact within the ASBMB and the scientific community as a whole!"

## ASBMB journals' metrics improve

Clarivate released a 2021 update to its Journal Citation Reports in late June. All three ASBMB journals — Journal of Biological Chemistry, Journal of Lipid Research, and Molecular & Cellular Proteomics — saw significant gains in their metrics, including impact factor and immediacy index.

Impact factor	Citations index	Immediacy	
5.157 (↑21.68%)	~400,000	1.638	<b>JBC</b>   JOURNAL OF BIOLOGICAL CHEMISTRY
5.922 (↑32.1%)	>28,000	2.035	<b>JLR</b>   JOURNAL OF LIPID RESEARCH
5.911 (↑21.38%)	>20,000	2.068	<b>MCP</b>   MOLECULAR & CELLULAR PROTEOMICS



### New IT team member

James Chiang joined the society as a software developer on July 12. Chiang has a degree from the University of Maryland and previously worked at GEICO and a small housing consulting company. He has worked with various web technologies and on data-centric applications and data pipelines. He said he is excited to contribute and learn from his new position at the ASBMB. In his spare time, he enjoys playing basketball, listening to horror podcasts, and "having new food experiences."

### Call for ASBMB fellows nominations

The American Society for Biochemistry and Molecular Biology will begin accepting nominations for fellows in September. Selection as a fellow is an honor bestowed on our most distinguished members. Fellows are recognized for their meritorious efforts to advance the molecular life sciences through sustained outstanding accomplishments in areas such as scientific research, education, mentorship, commitment to diversity and service to the society and scientific community. The deadline for nominations is Nov. 12. Learn more at [asbmb.org/about/asbmb-fellows](http://asbmb.org/about/asbmb-fellows).



# Researchers find a cell surface decorated with sugar-coated RNAs

By Ankita Arora

It's been a great year for RNA biologists: First the Nobel Prize in chemistry 2020 was awarded to Emmanuelle Charpentier and Jennifer Doudna for the development of CRISPR–Cas9 as a technology for genome editing, and then mRNA vaccines came to our rescue against the COVID-19 pandemic.

In addition to these milestones, a recent paper in the journal *Cell* describes how RNA can be glycosylated and how these sugar-coated RNA molecules, or glycoRNAs, present themselves on a cell's surface, suggesting they play a role in immune signaling.

Carolyn Bertozzi, a professor of chemistry at Stanford University and the study's senior author, described the breakthrough: "All of a sudden, you have to rewrite all the textbooks,



Ryan Flynn, now an RNA biologist at Harvard University and Boston Children's Hospital, was a postdoc in Carolyn Bertozzi's lab when he identified glycoRNA on a cell's surface.

because we're dealing with a cell surface that has not just glycoproteins and glycolipids, but also glycoRNA."

Ryan Flynn, the lead author and an RNA biologist at Harvard University and Boston Children's Hospital, explained the discovery in simple terms. "If your body is made up of cells, and the cells need to communicate to each other, maybe before this work, the idea was that the cell surface has two hands," he said. "Our work says that now there are three hands, and we don't know what that third hand is doing. But there's a new hand, and it's out there."

## Bumpy, unexpected start

Flynn joined Bertozzi's lab as a postdoctoral researcher in 2017, wanting to expand his expertise from RNA to another biopolymer: glycans and carbohydrates. He had an interesting hypothesis regarding the possibility for cytosolic glycosylation of RNA with O-linked beta-N-acetylglucosamine, or O-GlcNAc. He thought this might work because the enzyme O-GlcNAc transferase, or OGT, responsible for glycosylation, also has an RNA-binding domain.

This is where Bertozzi's expertise in bio-orthogonal chemistry played an important role. Flynn was using bio-orthogonally functionalized sugars that could be fed to cells to look for sugars attached to RNAs. These functional sugars then could click to an affinity probe that allowed him to visualize, enrich and then separate out

the labeled molecules.

Flynn set out to look for RNAs that were labeled with the glycans used in that cytosolic glycosylation pathway. He did not find them.

"After nine months of rigorous experiments and controls after controls, I realized that I didn't know what I had found," Flynn said. "I asked myself, 'What do I know about all the experiments I did?' And I couldn't come up with any specific conclusions."

Going through the blots, however, he realized that the only consistency across every cell type tested was the presence of an RNA-sensitive single band that he observed on treatment with N-azidoacetylmannosamine, or ManNAz, a functionally labeled precursor for a group of glycans known for their role in glycosylating secretory and cell surface proteins and lipids.

"And that's where the big question mark and the head scratching started, because in a way ManNAz was a negative control," Bertozzi said. "There was no reason to believe that RNA could get modified with the kinds of sugars that get secreted and cell surface glycoproteins get labeled with, because there's no framework by which RNA accesses the secretory pathway."

When Flynn sequenced the RNAs, they all fell into the category of small noncoding RNAs, including types such as transfer, small nuclear and Y RNAs — all structured and highly conserved noncoding RNAs.

Researchers do not understand yet the cellular functions of Y RNAs, but they have been identified as autoantigens in a number of autoimmune diseases.

## GlycoRNAs, cell surface biology and immune signaling

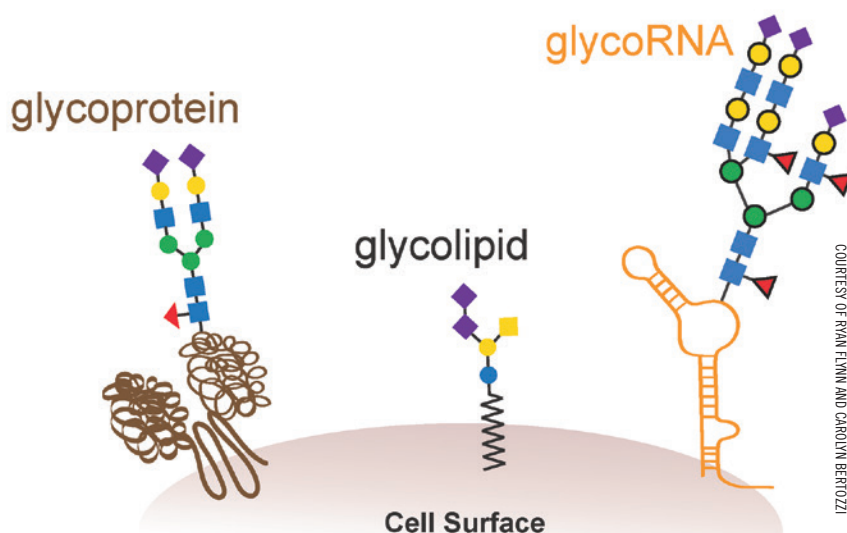
So where are these RNA present, and what is their function? The team found that the amount of glycoRNA decreases by more than 50% after an hour of incubation with a sialidase, suggesting that more than half of a cell's glycoRNA is present on its outer membrane.

"I think the position of things often either controls or infers their function," Flynn said. "And so, if it's on the cell surface, it really starts to help to fill out the black boxes, and you can begin to design new experiments."

He noticed that the glycan structures associated with these small RNAs are highly branched and are dominated with sialic acid sugars as capping groups on these branches. This triggered a connection; the Ber-



**Carolyn Bertozzi, a professor of chemistry at Stanford University and the study's senior author, said of the breakthrough, "All of a sudden, you have to rewrite all the textbooks."**



COURTESY OF RYAN FLYNN AND CAROLYN BERTOZZI

Researchers have long known that glycolipids and glycoproteins are present on the cell surface. Work in the Bertozzi lab revealed what Ryan Flynn describes as the "third hand": glycoRNA.

tozzi lab already was studying a family of sialoglycan binding receptors that are known to be immune modulatory — the Siglec family.

What if glycoRNAs could serve as biological ligands for members of the Siglec family? Flynn tested whether the binding of Siglecs' receptors to cells was blocked by or inhibited by treatment with RNase. While most of the Siglecs' receptor bindings were unaffected, the binding of two, Siglec-11 and Siglec-14, dropped considerably.

"It's not only that Ryan discovered that there's this new kind of molecule, but there's a whole new kind of pathway to produce this kind of molecule that is not understood," Bertozzi said. "And once again, we are humbled by how little we know about biology."

### What's next for glycoRNAs?

"It's funny how things flip," Bertozzi said. "Early in this project, I was skeptical and wary. I think Ryan was duly skeptical and wary but also intrigued and persistent. And we knew that for us to convince the world that we really had discovered a new molecule was going to be hard. But once

the paper got published, I've been really amazed at the response to it."

The ManNAz bio-orthogonal labeling method can't be applied easily to preserved human tissue samples or patient samples — a major limitation of the study. Scientists will need to develop other glycan labeling strategies to detect natural, unlabeled RNA–glycan conjugates.

Going back to his earlier analogy, Flynn said, "What we know is that the third hand might look like the first two hands but has different properties. Maybe it's a bigger hand, or it's stronger or weaker. But there's a third hand, and it's able to do something. And so then the question is why do the cells need it? How is it made? Where is it made? How many of the cells have three hands? And does that change in disease? And that's where we're now."

**Ankita Arora** ([ankita.arora@cuanschutz.edu](mailto:ankita.arora@cuanschutz.edu)) is a postdoctoral research fellow at the University of Colorado Anschutz Medical Campus. Follow her on Twitter: [@aroraankita](https://twitter.com/aroraankita).



# An inhibitory modification in a membrane-binding protein

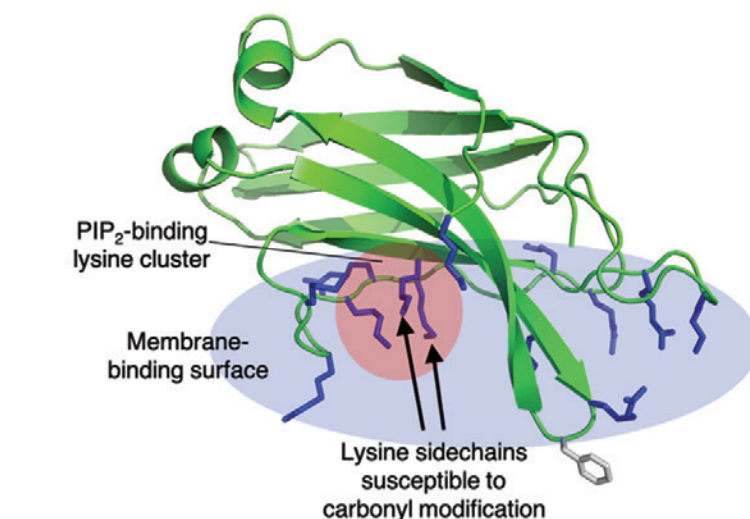
By Jefferson Knight, Colin T. Shearn & Cisloynny Beauchamp-Pérez

**P**roteins undergo nonenzymatic modification by reacting with various molecules. For example, cysteine residues react with the alkylating reagent iodoacetamide, and amino groups react with certain carbonyl compounds including succinimidy esters.

Nonspecific protein modifications in human tissues also are associated with a number of diseases; for example, most of the long-term consequences of diabetes arise from a damaging series of reactions involving blood glucose (an aldehyde) forming covalent bonds to amino groups on various proteins.

Physicians calculate average blood glucose concentrations in diabetic patients by measuring the levels of the glycosylated protein hemoglobin A1C, a modified form of the red blood cell protein with glucose attached to the N-terminal amino group of the beta-chain. Because such nonenzymatic reactions are typically irreversible, they result in damaged proteins that can lose function and must be removed via proteolytic degradation, a cell's normal process of breaking down proteins and recycling amino acids.

In a lab, when reactions occur on foreign proteins during bacterial expression, researchers need to design a purification strategy that removes the modified protein. Many modifications do not show up in sodium dodecyl sulfate polyacrylamide gel electrophoresis analysis and thus are



JEFFERSON KNIGHT

The C2A domain of granuphilin binds strongly to plasma membranes containing PIP<sub>2</sub> via a conserved lysine cluster (red circle) embedded in a large electropositive surface (blue oval) containing many basic residues (blue sticks). A single exposed hydrophobic residue (gray sticks) also contributes to membrane affinity.

easily overlooked.

In a recent study, we identified an endogenous protein modification occurring on a cluster of lysine residues that are central to lipid binding properties in the vesicle trafficking protein granuphilin, or Slp-4. This protein binds reversibly to plasma membranes via a conserved region called a C2 domain, which binds membranes independently of calcium via a large positively charged surface that interacts with negatively charged lipids. At the center of this surface is a cluster of lysines that has a high affinity for the plasma membrane lipid phosphatidylinositol-(4,5)-biphosphate, or PIP<sub>2</sub>. We also found that two of these conserved lysines are susceptible to modifica-

tion by the endogenous bacterial compound phosphogluconolactone, an intermediate in the pentose phosphate pathway. Not surprisingly, the modified protein binds much less strongly to PIP<sub>2</sub> lipids than the unmodified protein.

Researchers previously have reported phosphogluconoylation of bacterially expressed proteins but only as a modification on an N-terminal His-tag, never on an internal lysine sidechain. The modified protein manifested as an early-eluting peak during cation-exchange purification, and we identified the site of modification using mass spectrometry following trypsin digestion.

Researchers also already knew



that certain PIP<sub>2</sub>-binding C2 domains must be purified via cation exchange in order to obtain reproducible results. Our results suggest that the purpose of this step is not only to remove nucleic acid contaminants but also to separate out this endogenous bacterial modification, which can be a significant percentage of the total protein.

Why is this observation important to lipid biochemistry? Some of the most reactive carbonyl compounds in mammalian cells are aldehydes derived from polyunsaturated fatty acids, or PUFAs. These compounds are downstream products that arise from reaction of PUFAs with reactive oxygen species such as peroxide and superoxide,

which become more abundant during oxidative stress and inflammation. PUFAs are especially abundant as acyl chains in phosphoinositide lipids such as PIP<sub>2</sub>.

Therefore, our observation of reactivity in this PIP<sub>2</sub>-binding lysine cluster raises several questions: How reactive is this lysine cluster toward endogenous lipid aldehydes in mammalian cells? Does lysine modification affect membrane trafficking? Are there other proteins with lysine clusters that possess similar reactivity?

A serendipitous observation of protein modification during bacterial expression has opened the door to questions at the heart of protein chemistry and membrane biology.

**Jefferson Knight** (jefferson.knight@ucdenver.edu) is an associate professor and director of the biochemistry Bachelor of Science program in the chemistry department at the University of Colorado Denver.



**Colin T. Shearn** (colin.shearn@cuanschutz.edu) is a research assistant professor in the pediatrics department at the University of Colorado Anschutz Medical Campus.



**Cisloynny Beauchamp-Pérez** (cisloynny.beauchamperez@ucdenver.edu) is a Masters of Science student in Jefferson Knight's lab at the University of Colorado Denver.



## CALL FOR SUBMISSIONS



### Meeting Connections

**Have you made a connection, forged a collaboration, gleaned insight or had another meaningful experience at a scientific meeting? If so, tell us about it.**

We invite you to write about your own meeting connection in 300–500 words. We will publish the best stories in the March issue of ASBMB Today.

Email your submission to [asbmbtoday@asbmb.org](mailto:asbmbtoday@asbmb.org) with the subject line “Meeting connections.”

#### **Deadline: Dec. 1.**

And there will be prizes:

**FIRST PLACE:** Free ASBMB membership, free registration to the 2022 ASBMB annual meeting and a \$100 Amazon gift card

**SECOND PLACE:** Free registration to the 2022 ASBMB annual meeting and a \$50 Amazon gift card

**THIRD PLACE:** \$25 Amazon gift card

**ASBMBTODAY**

# Journal of Biological Chemistry names new editor-in-chief

*By Angela Hopp*

**T**he American Society for Biochemistry and Molecular Biology announced recently that Alex Toker, associate director for the Cancer Research Institute at Beth Israel Deaconess Medical Center and professor of pathology at Harvard Medical School, will be the next editor-in-chief of the Journal of Biological Chemistry, one of the society's three open-access, peer-reviewed journals. Toker's five-year term will begin Oct. 1.

Toker has a long relationship with the journal. He has been a deputy editor since 2020, an associate editor since 2013 and before that was an editorial board member. He also has served as chair of the editorial advisory board for ASBMB Today.

He is an expert in the signaling mechanisms that govern cancer progression. His lab specifically focuses on the PI3K signaling pathway in breast and other cancers and the mechanisms by which the protein kinase AKT promotes tumor cell survival and growth and the metabolic reprogramming of cancer cells.

"Alex has a compelling vision for the future of the JBC," said Toni Antalis of the University of Maryland School of Medicine, president of the ASBMB. "He was a standout candidate in our search, being a distinguished scientific leader with a strong commitment to the editorial processes and best practices that are the hallmark of the JBC. I believe the



**Alex Toker has served as an editorial board member, associate editor and deputy editor of the Journal of Biological Chemistry. His term as editor-in-chief begins Oct. 1.**

JBC will flourish in the open-access environment under his leadership and will continue to foster the dissemination of scientific advances that serve the scientific community."

Toker said he looks forward to continuing the move to open access for the JBC, exploring new approaches to manuscript submission and review, and embracing the open science movement, while maintaining the journal's commitment to data integrity and editorial board diversity and inclusion.

"I am so very excited to be taking the helm of the JBC," Toker said. "Over the years, I have experienced

the tremendous dedication of the JBC reviewing editors, the associate editors, the authors and our readership. The JBC is truly a journal that is for scientists run by scientists.

"I have become a fervent believer in open-access publishing and am delighted that during the past year the ASBMB and JBC leadership made the decision to move the society's three journals to full gold open access. For a journal with the long history, back content and sheer size of the JBC, this has been an enormous and complex undertaking, one we feel was not only important but essential for the JBC community."

Toker succeeds as editor-in-chief Lila Gierasch, a distinguished professor and former department head at the University of Massachusetts Amherst, whose five-year term ended June 30.

Antalis, who leads the society's governing council, said of Gierasch: "Lila took over the role of editor-in-chief in 2016 and has worked tirelessly to raise the profile and quality of the journal with the help and support of the JBC associate editors, the editorial board and the publications staff at the ASBMB. Under her leadership, the journal has enjoyed several major achievements, including the launch of JBC Reviews, improvements in the author experience, the move to open access, and a jump in impact factor."

Gierasch called Toker "an outstanding scholar and truly loyal to

JBC.” She said: “I am confident that the journal will continue to thrive under his leadership. We have worked closely together over the past few years, and I have witnessed Alex’s vision for scientific publishing, his adherence to rigorous peer review and his dedication to a society journal led by scientists and committed to fostering the work of scientists.”

Toker technically will take the reins from the journal’s interim editor, F. Peter Guengerich, a researcher at Vanderbilt University and longtime journal leader. Antalis expressed gratitude for Guengerich’s contributions both as deputy editor under Gierasch and as interim editor both now and during a previous editorship change. “We have been very fortunate to have Fred at the helm of the JBC during these periods of change,” she said. “Fred is an outstanding individual with an unwavering dedication to the journal and its mission.”

Toker earned his bachelor’s degree from King’s College at the University of London in 1987 and his Ph.D. from the National Institute for Medi-

cal Research in London in 1991. He conducted postdoctoral work with Lewis Cantley at Harvard.

In 1995, Toker published the first of many papers in JBC. “I recall with a deep sense of pride publishing in the JBC as a postdoctoral fellow, and thereafter as a principal investigator with my own lab. I have always considered the JBC the premier journal in biochemistry and cell and molecular biology,” he said. “I recall as a postdoc in the 1990s, my adviser would pass around the weekly JBC issue, with that unmistakable green cover, having scribbled the initials of each grad student or postdoc next to each paper in the table of contents. This was our signal to read that paper.”

He continued: “The world of publishing has changed dramatically in the ensuing 25 years, but the unwavering commitment to serve as a journal for scientists, run by scientists, has remained steadfast. I am deeply indebted to Lila Gierasch for her leadership as editor-in-chief the past five years and for the many initiatives she brought to the JBC. She will be a

hard act to follow.”

In 1997, Toker took a staff scientist position at the Boston Biomedical Research Institute. In 2000, he joined the faculty of Beth Israel Deaconess Medical Center and Harvard Medical School as an assistant professor. Today he is a full professor, chief of the Division of Signal Transduction, associate director for the Cancer Research Institute and Cancer Center at the Beth Israel Deaconess Medical Center, and a member of the Ludwig Center at Harvard.

Steve Miller, the ASBMB’s executive director, said, “I’m very much looking forward to working with Alex in this new role. I have every confidence that Alex will uphold JBC’s commitment to rigorous and constructive peer review — and take its service to authors and the greater life sciences community to new heights.”

**Angela Hopp** (ahopp@asbmb.org) is executive editor of ASBMB Today and communications director for the ASBMB. Follow her on Twitter @angelahopp.



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Monthly Twitter chats with @ASBMB

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# CALL FOR SUBMISSIONS

## The wellness issue — January 2022

As we creep out of our pandemic state, have you started a new practice to care for your body, mind or spirit that you intend to continue into the future? Do you have newfound appreciation for longtime healthy habits? Whatever you do for wellness, we want to read about it.

For information, email [asbmbtoday@asbmb.org](mailto:asbmbtoday@asbmb.org) or go to [asbmb.org/asbmbtoday](https://asbmb.org/asbmbtoday) and click **SUBMIT**.

**DEADLINE: OCT. 15**

**ASBMB**TODAY



 ASBMB

## ASBMB undergraduate program accreditation

**Deadline: Oct. 1**

Learn more at:  
[asbmb.org/education/accreditation](https://asbmb.org/education/accreditation)



# How a drug confuses SARS-CoV-2

By Gillian Rutherford

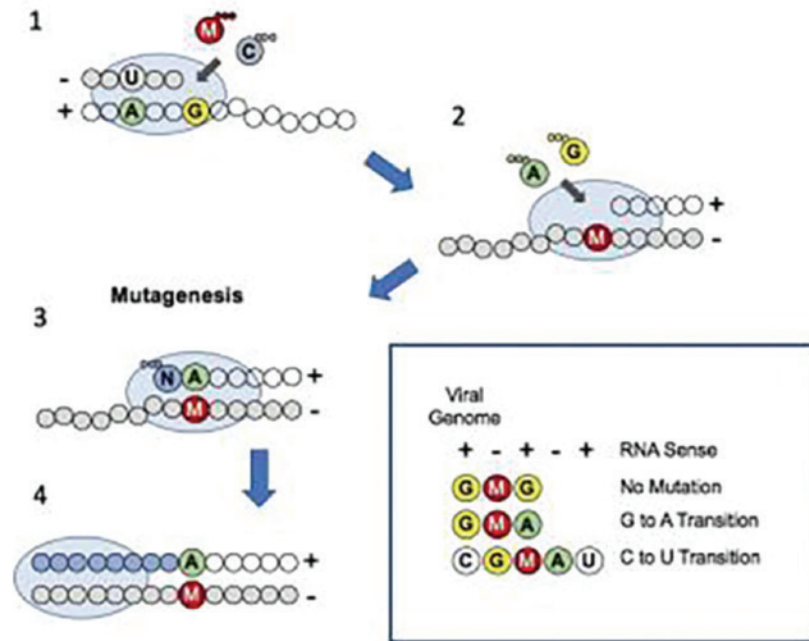
A University of Alberta virology lab has uncovered how an oral antiviral drug works to attack the SARS-CoV-2 virus. The findings were published recently in the *Journal of Biological Chemistry*.

The researchers demonstrated the underlying mechanism of action by which the antiviral drug molnupiravir changes the viral genome, a process known as excessive mutagenesis or error catastrophe.

Matthias Götte is a professor and chair of the medical microbiology and immunology department in the university's Faculty of Medicine & Dentistry and a member of the Li Ka Shing Institute of Virology. "The polymerase, which is the replication engine of the virus, mistakes molnupiravir molecules for the natural building blocks required for viral genome replication and mixes them in," Götte said. "It causes the polymerase to make sloppy copies — nonsense genomes that are useless and not viable."

In clinical trials for efficacy, molnupiravir eliminated SARS-CoV-2 infectivity in newly diagnosed patients after five days of treatment. The drug is taken as a pill, making it much easier to administer than other approved treatments such as remdesivir or monoclonal antibodies, which must be given intravenously."

"Our work to demonstrate that the effect of the drug is indeed mediated by the viral polymerase is reassuring, because if the drug somehow generates mistakes in the virus and you don't know how it happens, there



COURTESY OF MATTHIAS GÖTTE

This mutagenesis model of molnupiravir shows SARS-CoV-2 polymerase (oval)-mediated nucleotide incorporation into the RNA primer (grey circles)/template (white circles). Plus and minus signs indicate RNA sense. A, C, G and U refer to natural nucleotide bases, and M refers to the active forms of molnupiravir. Three small circles indicate the triphosphate form of nucleotides. (1) Molnupiravir competes with CTP for incorporation during synthesis of the negative RNA strand (copy of the genome). (2) When embedded in the template, molnupiravir base-pairs with either ATP or GTP. (3) Incorporation of ATP results in mutagenesis. (4) The mutation is fixed as RNA synthesis continues. The boxed information provides a summary of events that lead to drug-induced G to A and C to U transition mutations.

could be other mechanisms at work that could also harm the cell. Still, the safety of the drug for COVID-19 patients remains to be evaluated and monitored."

## The ongoing hunt for a weapon against pandemics

The active form of molnupiravir first was identified as a broad-spectrum antiviral at Emory University in Atlanta, Georgia. In 2003, it was developed as a treatment for chronic hepatitis C, but it was dropped due to possible side effects associated with long-term use. The drug then was

developed as an influenza antiviral, because the course of treatment for flu is much shorter. The focus of testing switched to SARS-CoV-2 after the COVID-19 pandemic emerged. The drug now is being developed in partnership by Merck and Ridgeback Biotherapeutics.

Merck has made deals with five generic drugmakers in India to make molnupiravir, and at least one of them has applied for approval to use it on an emergency basis, as at least 350,000 new infections are diagnosed in that country every day and vaccination levels are low.

Götte and his team previously uncovered the mechanisms of

action for remdesivir, a now-approved treatment that inhibits replication of the SARS-CoV-2 virus, and baloxavir, an influenza drug.

Next, they will test molnupiravir's mechanism of action against the polymerases of some of the other viruses the World Health Organization has identified as having high epidemic

potential.

"All are recognized as emerging pathogens where we need to develop countermeasures," Götte said. "We need to be prepared with broad-spectrum antivirals that can serve as a first line of defense.

"Even once vaccines are developed, we can't get them into all the arms

at once," he said. "To really fight outbreaks and epidemics, one tool is unlikely to be sufficient."

**Gillian Rutherford** (grutherf@alberta.ca) is a communications advisor for the University of Alberta.



## Upcoming ASBMB events and deadlines

SEPTEMBER

### SEPTEMBER

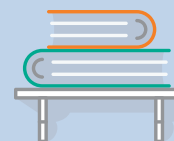
- 1 Cholesterol Education Month
- 1 Abstract deadline for *Serine proteases in pericellular proteolysis and signaling*
- 8 Blood Cancer Awareness Month
- 15 Hispanic Heritage Month
- 20–24 Postdoc Appreciation Week
- 20–24 Peer Review Week
- 29 World Heart Day
- 30 Early registration deadline for *Serine proteases in pericellular proteolysis and signaling*



OCTOBER

### OCTOBER

- 1 ASBMB accreditation program deadline
- 1 Student Chapters Outreach Grant deadline
- 1 Registration deadline for *Emerging roles of the nucleolus*
- 4 Breast Cancer Awareness Month
- 6–9 *Emerging roles of the nucleolus meeting*
- 10 World Mental Health Day
- 15 ASBMB annual meeting priority abstract deadline
- 15 ASBMB Outreach Grant deadline
- 16 World Food Day
- 12 Bone and Joint Health Action Week
- 18 Disability Employment Awareness Month
- 25 National Eczema Awareness Month
- 25–29 *Society for Advancing Chicanos/Hispanics & Native Americans in Science National Conference*
- 27 Registration deadline for *Serine proteases in pericellular proteolysis and signaling*
- 28–30 *Serine proteases in pericellular proteolysis and signaling meeting*



NOVEMBER

### NOVEMBER

- 8 National STEM Day
- 10–13 *Annual Biomedical Research Conference for Minority Students*
- 12 ASBMB fellows nomination deadline
- 18 LGBT in STEM Day
- 20 Student Chapters renewal deadline
- 27 Student Chapters Travel Award Deadline
- 30 ASBMB 2022 annual meeting abstract deadline





# Aggregates: Defect or protection?

By Lisa Nicole Learman

**P**rotein aggregates may spell trouble in the brain, but could they help protect our skin? In a recent study in the **Journal of Lipid Research**, researchers demonstrate that apolipoprotein E, or ApoE, a protein linked to Alzheimer's disease risk, can form aggregates with bacterial toxins to help clear infections.

ApoE binds to lipids to regulate their metabolism and transport.

Although the protein is also present in the intestines, lungs and skin, most ApoE studies focus on its role in the brain. This is because a particular variant of the protein is the greatest known genetic risk factor for late-onset sporadic Alzheimer's disease. Recently, however, Jitka Petrlova and researchers at the University of Lund found that ApoE made by skin cells can form large complexes, or aggregates, that help clear bacteria and toxins. This opens up a brand-new avenue for ApoE research as well as potential therapeutics.

Petrlova started studying ApoE as a postdoctoral fellow at the University of California, Davis, in John Voss's lab, which studies Alzheimer's disease. Alzheimer's researchers generally regard protein aggregation as a pathological process that should be stopped at all costs; uncontrolled aggregation can create massive sticky plaques on the brain that impair function. Researchers believe certain ApoE variants increase the risk of developing Alzheimer's by promoting formation of protein aggregates.

While studying ApoE structure, Petrlova noticed that parts of the protein looked like host-defense pep-

tides, small molecules made by cells in the immune system that can help neutralize bacterial toxins. Suddenly, she understood why skin cells would make ApoE.

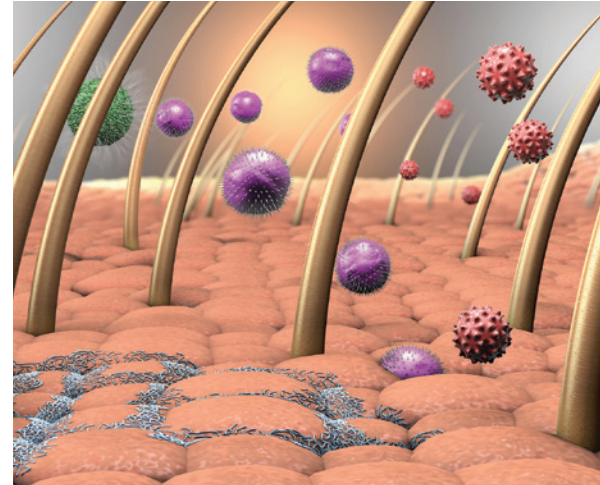
"It occurred to me how those two worlds could be connected," Petrlova said. "ApoE could promote aggregation on the skin as a simple mechanism to grab the toxins and neutralize them to prevent our body from overreacting to them and going into septic shock."

Now an associate professor of dermatology at the University of Lund, Petrlova continues to study whether ApoE's ability to promote aggregation might be beneficial in the skin. Her lab has shown that ApoE can bind and form complexes with lipopolysaccharide, a toxin on the surface of many bacteria, including *E. coli*, that can cause sepsis. These complexes help cells kill the bacteria and clear the infection. So ApoE — a protein linked to developing dementia, the villain of the Alzheimer's field — is actually a superhero on the skin.

Human bodies have made ApoE for millennia, and Petrlova believes that skin protection was the protein's original purpose. "A thousand years ago we weren't worried about getting dementia at age 70," she said. "Our life-span was not more than 20 years. It was more important to us to beat bacteria, to survive in our hostile environment.

"To me, neurodegenerative diseases are the negative unintended consequences of overactive aggregating proteins. The first aim of the body is to kill bacteria. This must be ApoE's true purpose."

Future studies will focus on figur-



Scientists at the University of Lund have discovered that, in skin cells, a protein linked to Alzheimer's disease can fight bacterial invaders, such as those illustrated here.

ing out how well genetic variants of ApoE clear bacteria relative to each other. Because the ApoE4 variant increases Alzheimer's risk by promoting aggregation, it likely will be better at clearing bacteria on the skin.

The findings could be translated readily to medical treatments. Petrlova envisions ApoE being developed as a topical antimicrobial dressing to help prevent infections at surgical incision sites and to combat immune system weakening that occurs with aging.

"Older people often get lesions that do not heal properly, and this can affect their lifestyle," she said. We need treatments to help people get back to their normal lives."

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## From the journals

By Clementine Adeyemi, Sarah May & Anand Rao

We offer summaries of research papers recently published in the **Journal of Biological Chemistry**, the **Journal of Lipid Research**, and **Molecular & Cellular Proteomics**.

### Antibody–receptor interactions alter ADCC

Monoclonal antibodies, or mAB, are a fast-growing class of biotherapeutic agents used for the treatment of many diseases, including cancer, autoimmune diseases, cardiovascular disease and, most recently, COVID-19. Binding of these antibodies to their receptors is a core component of the innate immune system, and understanding the interaction between antibodies and their Fc receptors — which interact with the tail, or Fc, region of antibodies — is essential for the engineering of effective mAB biotherapeutics. One such interaction that is important to consider when designing mAB therapies is antibody-dependent cellular cytotoxicity, or ADCC, a mechanism of cell-mediated immune defense whereby an immune effector cell lyses a target cell identified by bound antibodies. This effect can be either favorable or unfavorable, depending on the desired mechanism of action for the mAB being engineered; therefore, understanding how antibody–receptor interactions influence effector function is important for mAB design.

In recent work published in the **Journal of Biological Chemistry**, Yue Sun and colleagues at Genentech used hydroxy radical footprinting mass spectrometry to provide the first solution-phase evidence that an im-

munoglobulin G1 with an Fc region devoid of fucose sugar units requires fewer conformational changes for binding with its receptor. Then, using rational mutagenesis guided by molecular dynamics, the authors showed that fragment-antigen binding region–receptor interactions directly contribute to the modulation of ADCC.

DOI: [10.1016/j.jbc.2021.100826](https://doi.org/10.1016/j.jbc.2021.100826)

### What cholesterol does between cells

Two hundred years after Robert Hooke discovered cells, scientists got curious about the invisible barriers surrounding animal cells. Almost 50 years later, researchers described the dual nature of the fluid mosaic model of the cell membrane as both hydrophobic and hydrophilic due to its lipid and protein components. Chief among the lipids is cholesterol.

Cholesterol allows for a firm yet permeable cell membrane and is involved in steroid production. Now, a new study in the **Journal of Lipid Research** by Pawanthi Buwaneka and colleagues at the University of Illinois at Chicago has uncovered additional roles in cell signaling for cholesterol, specifically in the inner layer of the double-layered membrane.

Based on their previous work spotlighting interactions between cholesterol in this layer and intracellular proteins, the researchers' recent experiments using various cell types including fibroblasts and Leydig cells illustrate how these interactions precede cellular signaling. Using advanced imaging analysis, the authors

show how the level of cholesterol in the inner layer is tightly regulated to control intracellular signaling processes. This new role of cholesterol as a signal propagator could have implications for studying cell physiology.

DOI: [10.1016/j.jlr.2021.100084](https://doi.org/10.1016/j.jlr.2021.100084)

### How better storage improves transplant outcomes

The demand for kidney transplants exceeds the supply of available kidneys. Some donated kidneys go unused, however, due to the prolonged time between circulatory arrest and the start of cold storage. These kidney grafts often fail or are slow to function. Repairing such kidneys before transplant could greatly increase the available supply.

Kidneys donated after circulatory death are especially susceptible to injury due to low temperatures and lack of oxygen in conventional storage methods, such as static cold storage. An improved method, normothermic ex vivo kidney perfusion, or NEVKP, preserves kidneys at normal physiological temperature with nutrient and oxygen flow. NEVKP shows promise at improving kidney transplant function, but researchers do not know the molecular basis for this improvement yet.

In a new paper in the journal **Molecular & Cellular Proteomics**, Caitriona McEvoy and colleagues at the University of Toronto describe using pig models of donation after circulatory death to compare global protein expression in transplanted

kidneys after NEVKP and static cold storage. Using mass spectrometry and pathway analysis, they found that NEVKP increased expression of proteins linked to mitochondrial metabolism while it decreased markers of kidney injury. They propose that peroxisome proliferator-activated receptors, or PPARs, are master regulators of these effects. These findings

suggest therapeutic targets for improving kidney transplant outcomes.

DOI: 10.1016/j.mcpro.2021.100101

### Verifying the antiviral potential of A $\beta$

The role of amyloid-beta, or A $\beta$ , aggregates that form in the brains of Alzheimer's disease patients was in the

news after the Food and Drug Administration's recent controversial approval of the first Alzheimer's therapy. The therapy was based on the hypothesis that the pathological events that lead to Alzheimer's are triggered by accumulation of A $\beta$  peptides; however, researchers have offered alternative hypotheses about the disease's etiology. One such hypothesis suggests that A $\beta$

### Probing for anti-Zika natural products

Zika virus is a flavivirus related to yellow fever, dengue and West Nile. It emerged from obscurity in 2013 when it spread from Asia to the South Pacific and the Americas, reaching epidemic levels by 2016. While symptoms of Zika are generally mild, if a pregnant woman is infected, the virus can cross into the placenta, posing a risk to the developing fetus for microcephaly and other neurologic abnormalities. With no available vaccines or antiviral treatments, researchers are working to identify compounds that will address the need for Zika therapies.

Cyclohexadepsipeptides are natural products produced by a variety of organisms spanning the phylogenetic tree, including fungi, higher plants and cyanobacteria. The fungal cyclohexadepsipeptides destruxins, isaridins and isaridins — or DTXs, ISDs and ISRs, respectively — contain unusual nonproteinogenic amino acid-building blocks and perform a range of antiviral activities. Researchers have not yet identified the biosynthetic gene clusters for ISDs and ISRs or fully characterized the biosynthesis of a particular natural product nonproteinogenic residue, (3S)-methyl-l-proline residue.

In a paper published in the **Journal of Biological Chemistry**, Bochuan Yuan and colleagues at Peking University examined the extract of the marine-derived fungus *Beauveria felina* SX-6-22 and discovered 30 DTXs, ISDs and ISRs, including seven new compounds. Using anti-Zika virus assays, the authors showed that seven of the 30 compounds inhibited Zika virus RNA replication and nonstructural protein 5 production in Zika-infected A549 cells. The authors also sequenced the genome of *B. felina* SX-6-22; identified three biosynthetic gene clusters — *detx*, *isd* and *isr* — which are responsible for the biosynthesis of DTXs, ISDs and ISRs, respectively; and clarified the biosynthetic relationships among these cyclohexadepsipeptides. Finally, the authors defined the

entire biosynthesis of nonproteinogenic building block (3S)-methyl-l-proline.

Together, these findings identify compounds with anti-Zika properties and provide opportunities for biosynthetic pathway engineering to generate new anti-Zika cyclohexadepsipeptides.

DOI: 10.1016/j.jbc.2021.100822

— Anand Rao

This space-fill drawing shows the outside of one Zika virus particle and a cross section through another as it interacts with a cell.



DAVID S. GOODE/SEL/ROSB PROTEIN DATA BANK



## This oil check might be key to brain health

Chances are, you know someone affected by dementia — an umbrella of neurodegenerative conditions encompassing Alzheimer's, Parkinson's and other diseases that affect about 50 million people worldwide. Drugs



CHRISTIAN NEWMAN/UNSPLASH

**One in five Americans over age 65 is predicted to suffer from neurodegenerative diseases by 2030.**

developed to treat these conditions have been largely ineffective. However, a new study in the **Journal of Lipid Research** gives hope for possible therapeutics. Investigators Larry Spears and colleagues at Washington University in St. Louis link a lack of the lipid plasmalogen to the vascular abnormalities associated with these brain diseases. Plasmalogens are the most common type of phospholipid in the tissues of the nervous system and help protect the brain against oxidative stress, which is known to cause progressive neurodegenerative conditions. Plasmalogens are produced by the endothelial cells that make up the blood–brain barrier, vital for the protection of the brain. Hence, these lipids play two key roles in protecting the brain and keeping it running smoothly. The authors of this study genetically altered mice so their endothelial cells would have no PexRAP, an enzyme necessary for the synthesis of plasmalogen. Without PexRAP activity, circulating levels of plasmalogens decreased. This resulted in behavioral changes in the mutant mice and structural changes in their brains that are synonymous with neurodegeneration. The behavior changes included decreased physical activity, decreased attention to their environment and impaired spatial memory. Structurally, the number of neuroprotective glial cells increased, signaling a reaction by the nervous system as it sensed damage due to the lack of plasmalogens. In addition, the researchers saw a decrease in tyrosine hydroxylase activity, a consequence of neurodegeneration.

The authors concluded that plasmalogen decreases in the nervous system after vascular damage, leading to impaired brain health. Hence, checking on the levels of the brain's oil, plasmalogen, could serve as an indicator of brain health.

DOI: 10.1016/j.jlr.2021.100079

— Clementine Adeyemi

peptides possess antiviral effects and are produced by the central nervous system as a defense mechanism. The viral pathogens trigger a pathological cascade, presumably by seeding of A $\beta$  aggregates, which can entrap and neutralize CNS pathogens.

In a study in the **Journal of Biological Chemistry**, Olga Bocharova and colleagues at the University of Maryland School of Medicine explored the protective capacity of A $\beta$  against viral infection. The researchers infected young 5XFAD mice, which express human amyloid precursor protein and presenilin 1 transgenes with a total of five Alzheimer's-linked mutations, with one of two strains of herpes simplex virus 1, or HSV-1, at three different doses. They found that, contrary to previous work, the 5XFAD genotype failed to protect mice against HSV-1 infection. HSV-1 replication centers in the 5XFAD mice were partially excluded from the brain areas with high densities of A $\beta$  aggregates, but A $\beta$  aggregates themselves were free of HSV-1 viral particles, and the limited viral invasion to areas with a high density of A $\beta$  aggregates was attributed to phagocytic activity of reactive microglia.

While these findings challenge the antiviral role of A $\beta$ , further studies are needed to support or refute the viral etiology hypothesis of late-onset Alzheimer's.

DOI: 10.1016/j.jbc.2021.100845

## Deletion reveals more than meets the eye

Sphingolipids more commonly are known by their precursor, the popular skincare ingredient ceramides. However, these lipids play a more critical role in a host of physiological processes such as

## Drug discovery from deadly venom

The purple cone snail, *Conus purpurascens*, hunts fish and uses venom to immobilize its prey. Cone snail venom contains diverse toxic peptides, or conopeptides, that are biologically active, target-specific and valuable for drug discovery. One of the most powerful known painkillers, ziconotide, marketed as Prialt, was derived from a conopeptide.

Conopeptides vary among the more than 800 cone snail species and among members of the same species. Even a single cone snail specimen can produce unique conopeptide cocktails, called cabals, specialized for predation or defense. Cone snails also can hypermodify conopeptides at the post-translational step, increasing the diversity of the toxins and extending their range of biological targets. The extreme diversity of conopeptides provides a rich source of biologically active molecules for drug discovery.

In a recent study in the journal **Molecular & Cellular Proteomics**, Meghan Grandal and colleagues at the National Institute of Standards and Technology collected and analyzed the injected venom from 27 specimens of the purple cone snail. Using high-resolution mass spectrometry techniques, they discovered 543 unique conopeptides derived from 33 base peptide sequences and their toxiforms. A newly discovered conopeptide named PVIIIA has five sites of modifications and 33 toxiforms, illustrating the complexity and diversity of modifications to the base conopeptide that occur among various purple cone snail specimens. Building on previous studies, the researchers showed that the different snail specimens produce one of two unique venom cocktails. These two cocktails correspond to what are known as the “lightning

strike cabal” that rapidly induces paralysis of the snail’s prey and the “motor cabal” that acts more slowly to induce irreversible paralysis.

Knowing which conopeptides are co-expressed within a specific cocktail will give the researchers important clues as to the possible neural targets of newly identified conopeptides. This will be a critical step in developing new conopeptides into neural probes or therapeutics.

DOI: 10.1016/j.mcpro.2021.100100

— Sarah May

A purple cone snail uses its harpoon to pierce through a latex-covered tube, allowing a researcher to collect its venom.



ALEX HOLMIST

programmed cell death and inflammatory cascades, yet researchers know little about their regulation.

A recent study in the **Journal of Lipid Research** by Christopher Green and colleagues at Virginia Commonwealth University highlights the complexity of regulating sphingolipid biosynthesis. The researchers investigated the role of the different functional forms of mammalian

ORMDL, a protein that negatively regulates the activity of serine palmitoyltransferase complex, or SPT, the enzyme driving sphingolipid biosynthesis. Using a gene-editing tool, the authors developed stable, cancerous human lung cells with various functional forms of ORMDL to detect the unique roles of each form.

The results show how these vari-

ous functional forms of ORMDL uniquely affect sphingolipid metabolism, such as by determining the production of certain sphingolipid groups or by increasing the levels of certain ceramide species over others. These effects have broad implications for the critical body functions, such as cell growth and motility, that require sphingolipids.

DOI: 10.1016/j.jlr.2021.100082

## Mannose glycosylation linked to eye development

An unusual type of protein glycosylation, C-mannosylation, involves attaching a single mannose sugar to the amino acid tryptophan by a carbon-carbon bond. C-mannosylation, which regulates protein secretion, folding and function, occurs at a specific sequence of four amino acids that begins with the modified tryptophan. Even though about 18% of secreted or transmembrane proteins have this sequence, few studies have looked for the modification. Consequently, researchers know of few proteins that are C-mannosylated.

In a new study in the journal **Molecular & Cellular Proteomics**, Karsten Cirksena of the Institute of Clinical Biochemistry and a team of researchers in Germany found numerous proteins with altered secretion levels used mass spectrometry-based quantitative proteomics to profile cells lacking the C-mannosylation machinery. One of these potentially C-mannosylated proteins, a disintegrin and metalloprotease with thrombospondin motifs, or ADAMTS16, is essential during eye development and optic fissure closure. In Chinese hamster ovary cells and Japanese rice fish, the researchers demonstrated that ADAMTS16 can be C-mannosylated, that its secretion depends on C-mannosylation and that loss of a C-mannosylation enzyme causes a

developmental eye defect known as a partial coloboma — a gap in the eye tissue. Their findings suggest that C-mannosylation, an understudied protein modification, plays a critical role in eye development by regulating secretion of ADAMTS16.

DOI: [10.1016/j.mcpro.2021.100092](https://doi.org/10.1016/j.mcpro.2021.100092)

## An isoform-level gene atlas of macrophage activation

RNA sequencing, or RNA-Seq, routinely is used to measure changes to gene expression in response to cellular stimulus and perturbation. The technique provides wide coverage and high resolution of the transcriptome, but its need to fragment RNA molecules limits its ability to capture gene isoforms and their expression patterns. Thus, gene isoforms for follow-up studies may be selected based on annotation databases that are incomplete, not tissue specific, or lacking key information regarding expression levels; this results in lost time and resources when minority or nonexistent isoforms are selected.

In a study published in the **Journal of Biological Chemistry**, Apple Cortez Vollmers and colleagues at the University of California, Santa Cruz, used the long-read nanopore-based rolling circle amplification to concatemeric consensus method, which does not fragment RNA molecules, to generate an isoform-level transcriptome atlas of macrophage activation,

or IAMA, that identifies full-length isoforms in primary human monocyte-derived macrophages. The researchers characterized isoforms for most moderately to highly expressed genes in resting and activated macrophages, and they validated these isoforms by quantitative polymerase chain reaction.

The IAMA, which is freely available in a user-friendly data portal within the UCSC Genome Browser, is a resource for innate immune research that provides unprecedented isoform information for primary human macrophages.

DOI: [10.1016/j.jbc.2021.100784](https://doi.org/10.1016/j.jbc.2021.100784)

### Clementine Adeyemi

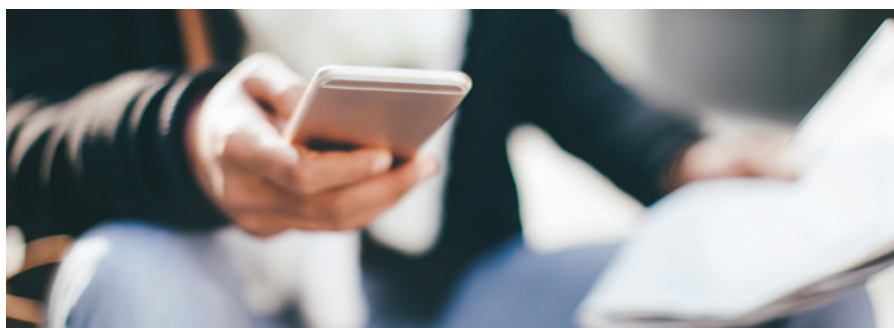
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ASBMB FELLOWS

# Call for nominations: 2022 ASBMB fellows

**Deadline for nominations: Nov. 12**

Selection as a fellow of the American Society for Biochemistry and Molecular Biology is an honor to be bestowed upon our most distinguished members. Fellows will be recognized for their meritorious efforts to advance the molecular life sciences through sustained outstanding accomplishments in areas such as scientific research, education, mentorship, commitment to diversity and service to the society and scientific community.

The ASBMB Fellows Program encourages nominations that reflect the breadth and diversity of the society's membership.

**[asbmb.org/fellows](https://asbmb.org/fellows)**





# Personal chemistry

# Proteomics tackles privacy concerns

By Laurel Oldach

Philipp Geyer was interested in learning how protein levels in the blood change when a person tries to lose weight. Several years ago, Geyer, then a postdoctoral fellow, and his colleagues in Matthias Mann's lab at the Max Planck Institute of Biochemistry began to analyze samples from a dieting study, hoping to identify biomarkers that could predict the outcome of dieting and other interventions. They found something a little different from what they were looking for.

While studying the plasma of 1,500 dieters tracked for 14 months, Geyer and his colleagues observed much more variation in protein levels across individuals than within any one person over time. Although some protein levels changed dramatically in response to dietary intervention, many others remained steady. "For example, alpha-two microglobulin," Geyer said. "It's tenfold different between people, but completely stable within a person."

As patterns emerged from the data, allowing them to pick out the same person at different time points, Geyer and his colleagues began to worry: Could these patterns one day be used to de-anonymize a study participant?

Around the same time, leaders of an international consortium of proteomics data repositories called ProteomeXchange were convening in Amsterdam to discuss ethical and legal issues in handling potentially identifiable biochemistry data.

The meeting's organizer, Juan Antonio Vizcaíno, runs a data repository from his lab at the European Molecular Biology Laboratory–European Bioinformatics Institute, known as EMBL-EBI, in the United Kingdom. Re-identification is "an issue that we have been aware of for years," he said, "but there needs to be a critical mass to start discussing it properly."

Sharing data is an important norm for the proteomics community. The benefits of openness are paid out in reproducibility, quality control and maximal use of each data set. Limiting access to data could impede scientific discoveries and their resulting therapies or even cures.

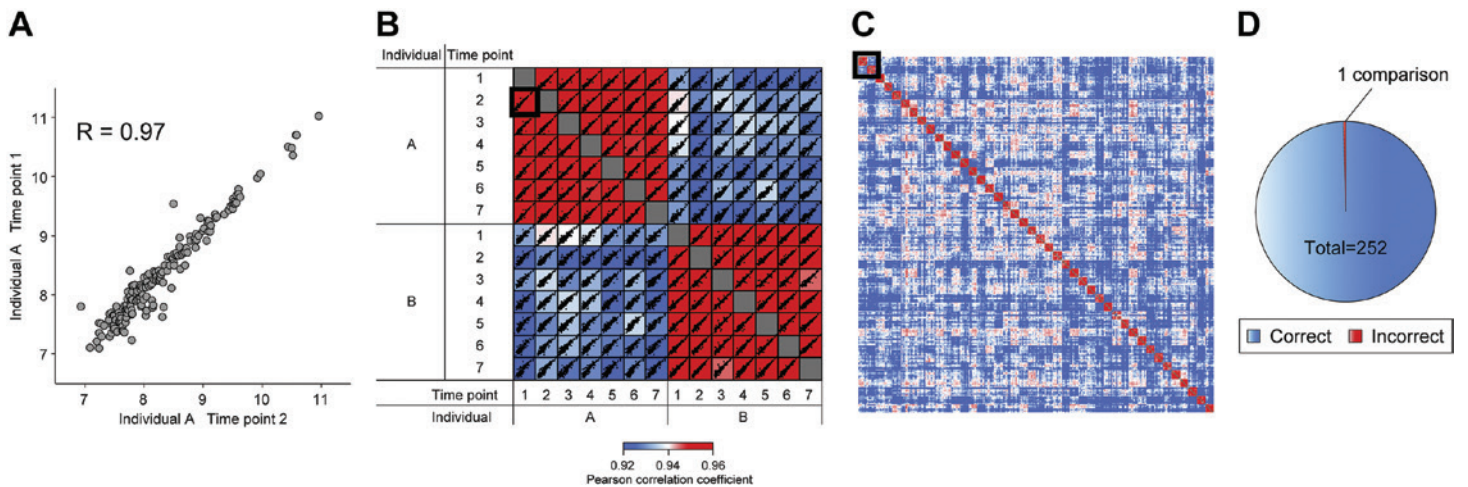
Still, even the most fervent defenders of open sharing agree that a communitywide discussion about access to proteomics data is worth having.

Earlier this year, the journal *Molecular & Cellular Proteomics*, which has played a significant role in creating a culture of openness, published three articles — two from the Mann lab and one from Vizcaíno and colleagues — on the topic.

ASBMB Today talked to the authors of those papers, the editor who

Many proteomics researchers agree that a communitywide discussion about access to proteomics data is worth having.





An analysis workflow shows how consistent an individual's protein abundance measurements between two time points (panel A); how that consistency can be used to match proteomes to a known individual over time (panel B); and how the team expanded the analysis to an entire cohort of participants (panel C).

oversaw them, legal and ethics experts, and other researchers about what is and isn't technically possible today, the risks and rewards of open proteomics data, and how to make scientific progress while still protecting people's privacy.

## What can a proteome reveal?

Whereas genome sequences are widely considered recognizable and linkable to an individual, most researchers so far have considered proteomes more anonymous.

Robert Gerszten, a physician-scientist at the Beth Israel Deaconess Medical Center, compared matching the pattern of protein levels in a clinical proteomics experiment to identifying a blurred photo. If a genome is like an image of an individual's face, a proteome, because of biological and technical factors, is more like a partially masked image at lower resolution. It takes longer and costs more to collect a proteome than a genome, and the depth of coverage — the number of times an experiment confirms that a specific gene or protein is present — is much lower in mass spectrometry-based proteomics. Due to technical factors in protein preparation and measurement, not all

proteins are equally likely to appear on a spectrum, and absence from the data doesn't mean that a protein is not present in the sample.

"If you take a population of thousands of people," Gerszten said, "you can clearly find person-specific signatures in that population (based on protein concentration). You couldn't have done that five years ago. ... But I don't think that the granularity is enough to figure out who that individual might have been in a huge population."

Stefani Thomas, whose lab at the University of Minnesota uses proteomics to investigate candidate biomarkers for cancer diagnosis and prognosis, said the Mann lab's diet study offers a thought-provoking proof of concept that protein level alone might be identifiable. However, she said, to determine traits such as an individual's age, gender or ethnicity using their proteome, researchers would need to know more about variability between and within those groups — similar to the research her lab does to differentiate between healthy and disease states.

Researchers don't know exactly how stable a level-based proteomic signature is over time. The Mann lab's study followed participants for over a year, which is brief compared



to a lifetime. And though weight loss left most parts of the proteomic fingerprint unchanged, the impacts of other physiological events are not yet established.

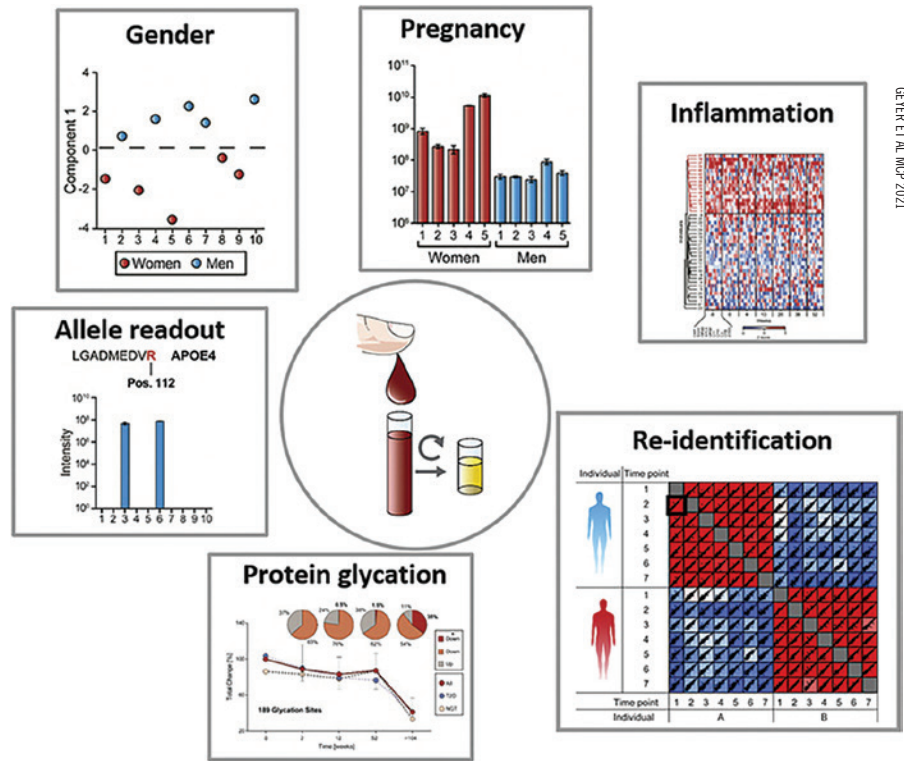
In addition to a single protein's presence, abundance and post-translational modification status, proteomic experiments increasingly can reveal something that doesn't change over a lifetime: genetic sequences, in the form of genetically variable peptides.

Proteomics yields sparser sequence information than DNA sequencing. The exome is smaller and subject to more selective pressure than the genome, and protein translation makes synonymous mutations invisible, so less person-to-person variation exists in protein sequences than in DNA or RNA. Still, when Geyer and colleagues went back over their data looking for genetic variants, they could indeed identify individual single-amino-acid variants in some spectra.

Geyer got to talking with Sebastian Porsdam Mann, Matthias Mann's son, who recently had finished a Ph.D. in bioethics, about how much of a risk this posed to participants — and whether the possibility of discovery outweighed the risk.

"You need to have high quality data, but in order to reduce identifiability risk, you need to do things to this data to filter out identifiable information. So there's always going to be a trade-off," Porsdam Mann said. In addition, there are times when being able to match a study participant to features of their proteome could be beneficial (see "Treatable discoveries" on page 32).

In the Mann lab, as in other labs handling clinical samples, specimens arrive stripped of participant name, date of birth and other identifying



GEYER ET AL. MCP 2021

information. Do the specimens themselves — or, more importantly, the data the lab uploads to international repositories — carry enough information that a motivated, proteomics-savvy adversary could trace them back from spectrum to person?

According to Thomas, a key question remains unresolved: "What is the minimum amount of data from a proteomic experiment that can be used to definitively identify an individual?"

Although the clinical proteomics community is just beginning to explore the question, forensic researchers have been trying to determine an answer for years.

## Forensic proteomics

Glendon Parker, an adjunct associate professor at the University of California, Davis, is one of a small number of scientists working to identify genetically variable peptides for forensic purposes; his lab focuses on hair. Inferring a genome, Parker

The graphical abstract of Geyer and Porsdam Mann's research article in MCP shows how they used data from population serum proteomics studies to determine potentially identifying features such as gender, pregnancy status and allele expression in a cohort.





## Treatable discoveries

Philipp Geyer, a researcher at the Technische Universität Berlin, had high cholesterol starting in childhood. As a postdoc, while setting up a workflow for a large clinical trial, he used a sample of his own blood as a quality control. By revealing the amino acid sequence of a peptide from apolipoprotein E, the proteomic data showed that Geyer had a variant of the gene linked to persistent high cholesterol levels.

After a lifetime of limiting chocolate and butter, pursuing sport and exercise regimens, and stubbornly declining pharmacological intervention, Geyer said, the discovery that his problem was genetic gave him a push. “I was convinced, okay, I can’t do anything with sports or nutrition, so I really have to take the drug.”

On a statin, Geyer’s cholesterol problem resolved. Monitoring his own plasma proteome, he watched his apolipoproteins drop. He shared this information with members of his family. “My dad and my brother actually went on statins because of this,” he said.

Geyer’s discovery is what an ethicist would describe as an incidental finding. It wasn’t the information he set out to find, but it gave an important insight into his health. And a simple intervention helped Geyer lower his risk of cardiovascular disease.

According to Sebastian Porsdam Mann, a bioethicist and son of PI Matthias Mann who worked with Geyer on a *Molecular & Cellular Proteomics* article about the ethics of clinical proteomics studies, this is part of why absolute anonymization of data may not always be in a study participant’s best interest. “In the extreme, you could literally anonymize it completely,” he said. “But then you could never follow up.”

Steven Carr, proteomics director at the Broad Institute, has experienced the frustration of being unable to follow up; while analyzing a lung cancer sample, he said, his lab stumbled across a clinically meaningful finding. He said that his team wanted to get in touch with the clinicians who collected the sample to tell them, “We don’t know who this person is, but by the way, they could have been — or should have been — treated with X because they have this particular set of characteristics in their proteome.” Because of privacy protections, he said, that kind of feedback was prohibited.

Some incidental findings are less actionable. For example, people who carry a different apolipoprotein E allele than the one Geyer discovered in his blood are up to 90% more likely to develop Alzheimer’s disease, but the connection is not well understood and someone who learns they have the allele can’t take any action but wait.

Medical ethicists see a line between actionable and nonactionable information; they say that in general, if information is actionable, patients ought to be informed — unless, fulfilling the principle of autonomy, they have stated they prefer not to be informed.

Certain incidental findings might convey information a study participant would prefer not to share with others. For example, physician–scientist Robert Gerszten mentioned that metabolomics studies designed to find heart disease biomarkers also might show what medications a person is taking or whether metabolites of illicit drugs are present.

said, depends on knowing whether each change to a peptide is genetic or is caused by environmental effects such as dyes or weathering. That means validating that each candidate genetically variable peptide matches a genotype before using it to infer genetic information — and also that it is distinguishable from the rest of the proteome.

Deon Anex, a chemist at Lawrence Livermore National Laboratory who works on a forensic proteomics project inspired by Parker’s research, frames the question: “Let’s say you have a unique peptide, and then you modify one of the amino acids. Is that still unique? Or is it now a common sequence somewhere else?”

In most cases, using the proteome to get a genotype is redundant to long-established methods based on DNA. However, in some environments, DNA falls to pieces but proteins survive. According to Anex, forensic proteomics is a good fallback for getting genetic information from hair or from brass ammunition cartridges that are inhospitable to DNA. His lab also is working on a project funded by the research agency of the Office of the U.S. Director of National Intelligence that aims to pull genetic information from the proteins left after bomb blasts. The project team has demonstrated that it can match proteins from partial fingerprints on objects to the volunteers who handled the objects. Now, it’s conducting field experiments with lab-made improvised explosive devices to determine whether those traces are still recognizable after an explosion.

Based on the variant peptides they detect in a sample, forensic researchers in Parker’s and Anex’s labs can determine a partial genotype. Using statistical methods from forensic science, which consider the number of variants detected and how common





each one is, researchers can calculate the odds of a false-positive match. Those odds would need to be on the order of one in 10 billion to identify someone uniquely in the world. Parker has an answer to the question Thomas posed; he believes that just a few hundred genetically variable peptide calls could be enough to identify some people uniquely.

But even when the odds of a false positive are very low — even with a genome that unambiguously belongs to a single person — it is difficult to trace a sequence back to the person who carries it in their body. Brad Malin is a professor of biomedical informatics at Vanderbilt University. “Uniqueness is insufficient to actually identify somebody,” he said. “You need to be able to link that data to some other resource to get back to their identity.”

He added later, “There have been illustrations that when an organization or an individual is sufficiently motivated, if all the stars align, they’ll be successful. But it requires a lot of effort.”

Back in the clinical proteomics community, some researchers say that the risk of a motivated, proteomics-savvy adversary making such an effort is dwarfed by the benefits of open data sharing. Some also ask whether, in a world where so much personal information already is collected and sold, the risks of adding proteomics data to the mix could possibly outweigh the advances in science and health that those data enable.

### What’s the harm?

Malin said a friend recently questioned him about why re-identifiability was a concern, asking, “What are the harms? What is it that we’re supposed to worry will go bump in

the night?”

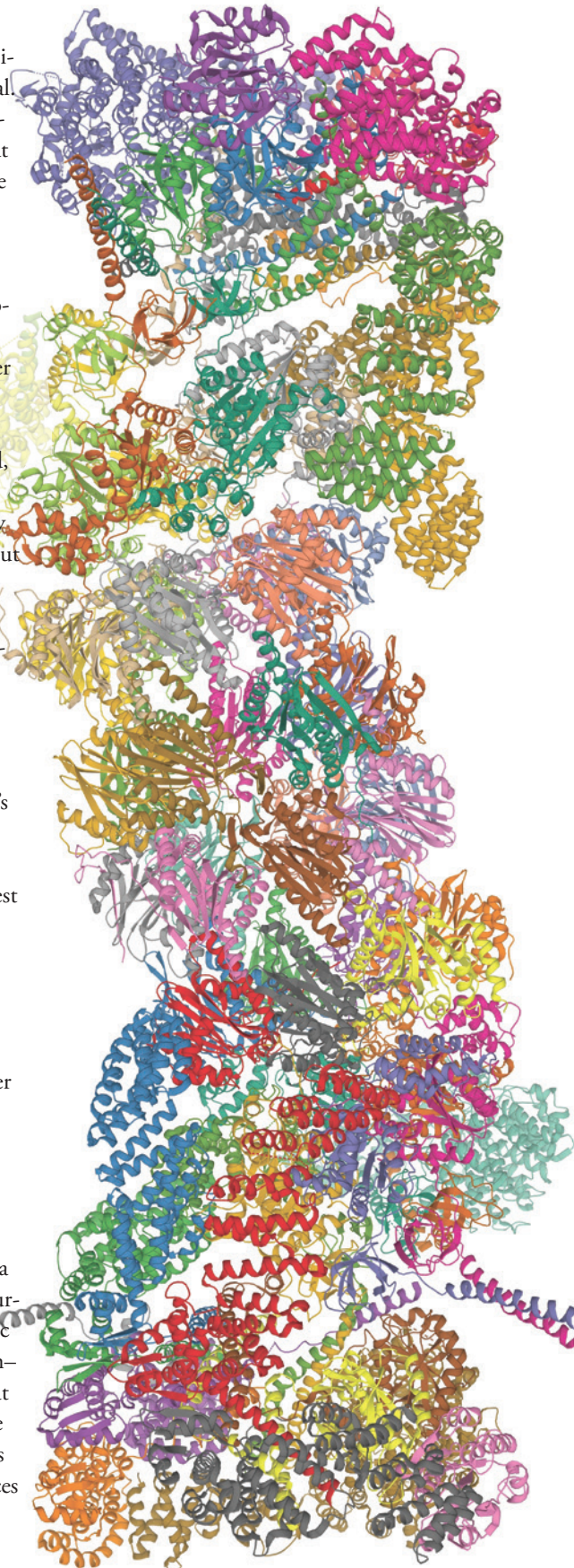
For one proteomics researcher, Michael Snyder, the question is personal. Snyder, a Stanford University professor, conducts multiomics studies that include himself as a study subject; he published his whole genome, thinly anonymized, years ago in the journal *Cell* and has followed it up with longitudinal transcriptomes, metabolomes and clinical assays.

Sporadically over the years, Snyder has heard from people who want to share analyses of his health. Some have had interesting insights, he said, but “some of it’s pretty loony.” Still, he doesn’t believe he has suffered any real harm from radical openness about his personal biochemistry. Nor is he aware of other study subjects who have suffered because their transcriptomic or proteomic data have been published.

“There’s a lot of RNA-Seq data out there,” he said. “I challenge you to show me one example where that’s been abused.”

He’s right; no examples of such abuse have been reported. The loudest bumps in the night come from privacy researchers sounding the alarm about data vulnerability and law enforcement agencies using genetic information from opt-in genealogy databases — not research data. Other possible attackers are so far a matter of conjecture.

Information in a proteome can give clues about a person’s health; American scholars have envisioned discrimination by health insurers as a concern, but it is now illegal for insurers to deny coverage based on genetic information. Gerszten, the physician-scientist at Beth Israel, suggested that marketers hypothetically could mine metabolomic or proteomic databases to find out about reproductive choices or disease status. There is no doubt that a market for this type of data





Juan Antonio Vizcaino and his team run the Proteomics Identification Database, or PRIDE, out of the European Molecular Biology Laboratory in Cambridge, UK.

exists; according to a cybersecurity firm's 2019 report, health records stolen from hospitals sell for about 50 times the value of a stolen credit card number. (Of course, health records are easier to interpret than a proteome, whose significance may not be known, and they generally include a patient's name or other identifiers.)

After revelations that social media and genealogy sites can gather and reveal more information than users thought they were sharing, societal conversations around privacy and data sharing have changed in the past decade. Snyder has noticed a similar uptick in caution among his study participants since he launched a longitudinal proteomic profiling project called iPOP in 2010.

"It did take this shift when people learned that Facebook and others were selling their data," Snyder said. However, he added that he sees participants on social media "posting some incredibly private stuff that's probably more harmful than proteomics data. Privacy is gone, whether you like it or not."

Malin, the bioinformatics expert, disagreed. "Google's not taking all of your search queries and throwing them online for everybody to see," he said. "Privacy is not dead. You've just shifted who you trust with information about yourself."

EMBL

From an ethical standpoint, Malin said, any research participant whose privacy is compromised has lost something of value, even if they suffer no further consequences. "Whether or not the individual was materially harmed ... simply the identification would be sufficient to claim that their privacy had been infringed upon, because they did not want that information revealed."

Lawsuits against hacked hospitals have argued the same thing. But researchers say that they, too, have something of value to lose: knowledge that could lead to biomedical progress. Broad Institute proteomics director Steven Carr, an MCP deputy editor, said, "At this point in time, adding unnecessary protections to the availability and use of proteomics data on human samples has the potential to do more harm than good."

## What do researchers stand to lose?

In the past year and a half, numerous large COVID-19 studies have illustrated the benefits of data sharing in proteomics. Data sets showing viral interaction with human proteins, linking detectable markers to patient outcomes and tracking immune responses over time have added to an internationally constructed picture of how the novel coronavirus works and have been mined by other researchers for further insights. And this is not a new phenomenon; according to Snyder, most of the annotation of the human proteome has depended on secondary analysis of publicly available data.

Since its launch in 2011, the ProteomeXchange, a consortium of repositories that make proteomic data freely available, has published more than 24,000 mass spectrometric data sets, about 45% of those from



ProteomeXchange is an international consortium of proteomics databases.



human or human-derived cell lines. Thousands of papers have reported new findings based on data in the six repositories that make up the exchange.

The field was not always so open. It took commitment from publishers, funders, data repositories and the Human Proteome Organization to require investigators to make their raw data freely available to colleagues. The editors of MCP, including Carr, take particular pride in the journal's early requirement that scientists publish raw data.

"There was a point where very few people submitted data to PRIDE or to other resources," Vizcaíno said. After a rapid change, "Now we are in a scenario that is basically the opposite."

Carr and Snyder argue that limits on data sharing to accommodate privacy concerns would risk reversing the field's cultural shift. Without easy access to raw data, researchers would be unable to check one another's work for reproducibility or reanalyze data for follow-up studies. Based on how usage patterns differ between controlled-access and openly available transcriptomics data, Snyder is confident that research progress would slow if proteomics data were walled off.

Some proteomics data already are controlled because they are linked to other, more easily identifiable data such as clinical outcomes. Gerszten, whose lab conducts multiomics studies of heart disease, said that they keep all data from patients under what he called electronic lock and key. "The ability to say, 'these sets of markers track with individuals who had a better outcome' ... is actually very valuable information," Carr said. "It represents no harm to any individual. But it does represent a pathway to try to identify markers



## Laws and policies governing data sharing

The Bermuda Principles, established in 1996, supported rapid, public data sharing to advance the Human Genome Project. In the years since, concerns about credit for research and about participant identifiability have driven a shift to more cautious data sharing.

The European Union's General Data Protection Regulation took effect in 2018. Its protections on data collected in Europe and describing European citizens make exceptions for anonymized research data, but experts say that anonymization is not well defined in the law, making it hard to follow. In a Policy Forum article in the journal *Science*, legal scholars in the U.S. and Europe argued that GDPR protections impeded collaborative international research by preventing clinical data collected in Europe from being transferred to international collaborators. The barriers to transferring data, they wrote, "appear to be at odds with the generally research-friendly intent of the GDPR." According to Vanderbilt bioinformatics professor Brad Malin, until a court rules on anonymization requirements, the exact meaning of the GDPR for genetic and bioinformatics data will remain ambiguous.

In the United States, data sharing is governed by a federal policy, originally adopted in 1991 and since revised, known as the Common Rule. The policy, which applies to any research on human subjects carried out or funded by 15 federal departments, is forged by consensus among them. The rule exempts data "recorded such that subjects cannot be identified." As with the GDPR, the Common Rule is complex and does not spell out whether genomes are identifiable or can be anonymized. Health records, such as those produced in clinical trials, also are protected by the Health Insurance Portability and Accountability Act of 1996, or HIPAA; it, too, makes provision for sharing of de-identified data for research purposes.

that might be useful for diagnostic purposes."

Gilbert Omenn, a physician-scientist who directs the University of Michigan's center for computational medicine, said, "We want proteomic data not to just be about advancing tools of mass spectrometry and other methods; we want it to be linked to biomarker development and clinical diagnosis, and therefore we need to translate it to patients."

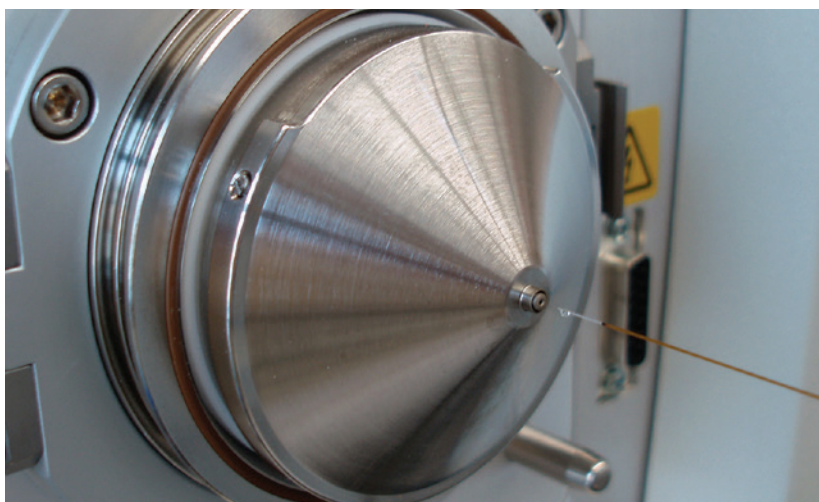
That goal is coming into view, he said. It is exciting for the field, but "I don't think it behooves us to try to claim that we're outside the boundaries of responsibility."

Malin believes that a major





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This photo shows detail of the electro-spray system on a mass spectrometer.

re-identification episode would do more harm to researchers than to their participants. “People are going to scream bloody murder that they will no longer trust scientists with their information,” he predicted. “That would be a big blow to biomedical research.”

## Legal requirements, technological solutions

Concerns about potential damage to the field motivated Vizcaíno to organize the Amsterdam meeting of ProteomeXchange leaders and other experts in 2019.

In a recent article in MCP inspired by that meeting, the researchers called for data access to become “as open as possible, as closed as necessary” to balance research transparency and data sharing with privacy concerns. The field needs further research into the likelihood and potential severity of data breaches, they concluded, but even in the absence of such research, it is important to begin to develop best practices for handling sensitive data.

Proteomics databases are designed to make data public and permanent. After a quality control check, PRIDE publishes all the data it receives,

sometimes waiting for a corresponding paper to be published. Here and there over the years, Vizcaíno said, “We have had a few cases that people who had originally submitted data to us have told us, ‘We have been told by our data officer ... that we shouldn’t do this.’”

University data officers are trying to follow complex rules. In many jurisdictions, consumer privacy laws address what identifiable data may be shared (see “Laws and policies governing data sharing”), and different authorities balance the risks and rewards of data sharing differently.

Omenn said there is “a lot of attention, a fair amount of angst, and, I think, rather high compliance” with legal requirements for identity protection.

Because policy changes slowly and tends to be more reactive than proactive in the face of technological advances, Malin expects that it would take a major breach of privacy affecting either millions of people or a powerful politician to force changes to privacy laws. When an American policy called the Common Rule was revised, a process that took six years, the agencies involved in the work decided against declaring genetic information identifiable.

“The implications of designating biological information as identifiable are quite breathtaking,” Malin said. “It would completely shift the way that research is performed in this country.”

Still, he said, “We’re somewhat at a crossroads ... it is possible that the United States is going to have its hand forced by the European Union.”

Vizcaíno, predicting that re-identification would become a concern for proteomics, has kept an eye on the DNA and RNA databases that his EMBL-EBI colleagues administer. Often, these administrators require



that researchers apply for access to sensitive data, providing only enough information to run analyses the labs describe ahead of time. Some databases layer in additional measures, such as suppression of highly identifiable sequences or scrambling of genotypes, to protect information that could identify an individual; meanwhile, in a sort of arms race, privacy researchers continue to report ways to breach those safeguards.

For now, PRIDE and other databases do not have the architecture in place to render raw proteomics data less identifiable. If Vizcaíno and his colleagues receive a request to delete a data set while it's still under review by editors and reviewers, they are able to honor that request.

Removing data sets after they are posted, however, may raise problems with the resulting publications. Carr said, "MCP will not accept papers where the data cannot be made public."

Vizcaíno hopes to build a database that, like databases for genomics, offers controlled access only to reviewers and researchers who explain why they need to see sensitive data, or researchers might be restricted to accessing data that corresponds directly to a research question. Approaches from genetics, he said, are practical, but an investment is needed to adapt them to mass spectrometry data formats. "We are at the very beginning of a very long road."

## Sightlines

Ongoing conversations about proteomic privacy are driven by what biomarker researcher Stefani Thomas called "an undercurrent of advances in technology" and a faster pace of proteomic data collection. That's good news for the field, she said, but to realize the goal of using the technology



in the clinic, questions about privacy and identifiability must be resolved. "I think it's exciting that people in the field are taking a step back and saying, 'Let's look at this from a broader perspective and make sure that what we're doing is ethical.'"

Porsdam Mann, the bioethicist, said, "If you look at the history of genomics research, but also other related fields, you'll find that responsible self-regulation early in the game is one of the wisest longer-term investments."

Carr said, "The way I view this is in clinical terms. ... I think we should be in a position of watchful waiting to see how confidence in potential identification of proteomics data goes."

Snyder argues that existing restrictions on data use are more than adequate. "Until somebody gets harmed, I'm not sure I'm so worried about it," he said. "Maybe when somebody gets harmed, it will all blow up and they'll say, 'Mike, you didn't foresee this very well.' And I'll say, 'Yeah — but we got a hell of a lot done in the meantime.'"

**Technical advances have enabled scientists to collect data on many clinical samples at once.**



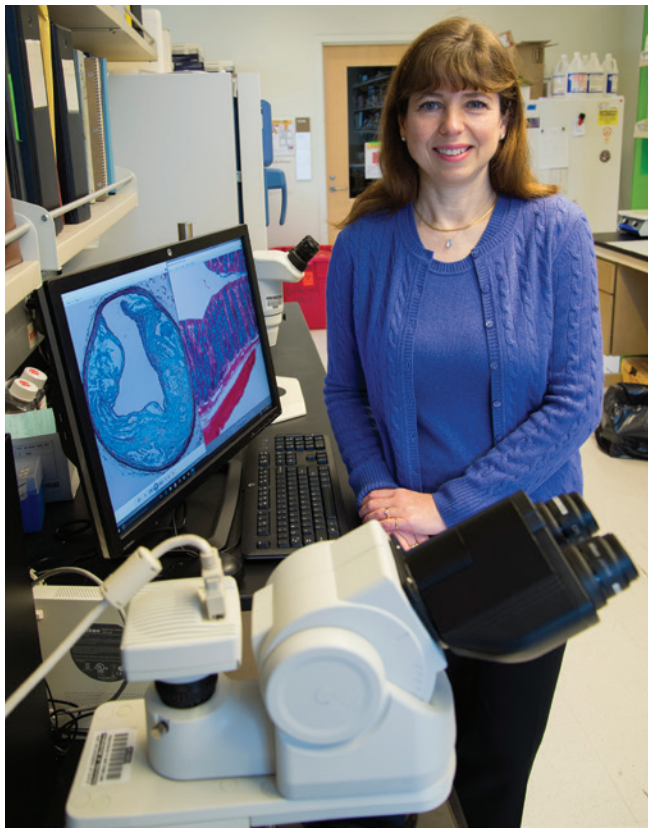
**Laurel Oldach** (loldach@asmb.org) is a science writer for the ASBMB. Follow her on Twitter @LaurelOld.



# Building bridges between basic and clinical research

JLR associate editor Karin Bornfeldt investigates how diabetes increases cardiovascular disease risk

By Laurel Oldach



COURTESY OF KARIN BORNFELDT

Bornfeldt in her lab at the University of Washington.

**K**arin Bornfeldt always knew she wanted to do research. She has fond childhood memories of examining fossils, stuffed and mounted birds, and formalin-preserved snakes and fish in her father's high school biology classroom in Sweden. Thanks to her parents' encouragement, Bornfeldt said, "I grew up having a very strong connection to the natural world and being very curious about how things work." She was drawn to medical research — although she might happily have studied ethology, plant physiology or other fields of biology.

As it happens, Bornfeldt became interested as a graduate student in how diabetes predisposes patients to heart disease. Atherosclerosis, a common complication of diabetes and the cause of heart attacks and strokes, arises when white blood cells squeeze in between an artery and its smooth-muscle sheath. Over time, these cells accumulate lipids and contribute to development of cholesterol-filled lesions that can rupture or fissure and block blood flow. However, it is not well understood why Type 1 diabetes, which arises when the immune system attacks the pancreatic beta cells, and Type 2 diabetes, which develops after insulin-secreting pancreatic beta-cells are overstressed, can accelerate atherosclerosis.

Bornfeldt's lab demonstrated that in diabetic mice, lipids are more important than glucose in accelerating atherosclerosis. She continues to probe the links between the two diseases as the associate director for research and leader of the diabetes complications research program at the University of Washington's Diabetes Institute. Since 2019, she also has served as an associate editor of the *Journal of Lipid Research*.

Bornfeldt recently discussed her work with *ASBMB Today*. This interview has been edited.



**Q. You study how diabetes accelerates atherosclerosis. How did you get interested in this area of research?**

I have worked in this area since I was a graduate student back in Sweden. I studied with a diabetologist there, a physician–scientist named Hans Arnqvist. I was his only graduate student at the time, and I got to know his clinical team and some of his patients. When we met, his pager would often go off for patients who had had heart attacks or strokes. Sometimes they were very young. Before my graduate studies, I had not heard of women having heart attacks in their 30s, and it made me realize that the connection between diabetes and early heart disease was something that we urgently needed to understand and find treatments for.

At that time there weren't any good animal models to study diabetes-accelerated atherosclerosis, the process that leads to cardiovascular disease. I thought better animal models would be needed in order to understand mechanisms and move the research area forward. As a graduate student, I learned a lot about diabetes, and I decided to pursue studies in atherosclerosis by moving to the University of Washington to train with Russell Ross, who was a leader in atherosclerosis research. I was also fortunate to study signal transduction in vascular cells with Ed Krebs.

After my postdoc period, I set up my own lab here at the University of Washington, and that's when I had the opportunity to really focus on cardiovascular complications of diabetes by generating a mouse model of that disease. So I moved from studying vascular tissue and cultured cells to animals; we generated a new mouse model that we still use a lot to study mechanisms whereby diabetes pro-

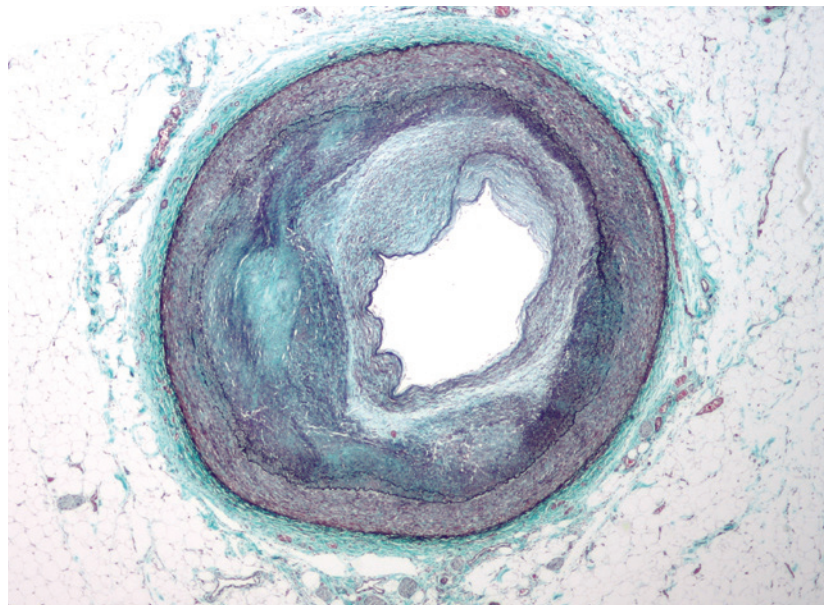
motes atherosclerosis. Those studies brought us to the understanding that diabetes affects several different stages of atherosclerotic lesions and that the mechanisms might be different for different stages of lesion progression.

More recently, my lab has taken another step toward human translational studies; we're combining data that we get from large human cardiovascular outcome studies with the mechanistic mouse models that we have in the lab. We're now basing our research on data from human studies to be sure that we're studying the most important drivers of cardiovascular disease risk in diabetes in humans. It's been a rewarding progression in my career, going from studying single cells in culture to animal models to translational and clinical studies.

**Q. How do you apply an insight from a large clinical trial to a mouse model?**

A good example is work we did recently on apolipoprotein C3 in collaboration with Janet Snell–Bergeon at the University of Colorado. We obtained baseline samples from humans with Type 1 diabetes involved in a study called CACTI. These subjects didn't have any cardiovascular disease when the plasma samples were collected, but they were followed over time so that we knew who developed a myocardial infarction later on. We used targeted mass spectrometry to identify proteins in those plasma samples that predicted who was going to have a cardiovascular event later in life. We identified apolipoprotein C3 as a risk factor, then silenced this protein in our diabetic mouse model to investigate if APOC3 is a causative factor or just a biomarker of increased atherosclerosis. It turned out to be a very strong causative factor: Animals

**“You might not find the answers to your most important research questions for many years, or decades even, but if you just have the determination to get the answer, you will often get there, and you will have advanced science in the process.”**



NEPHRON/MEDIA COMMONS

**A cross-section of a coronary artery shows thickening of the arterial wall and narrowing of the blood vessel, hallmarks of atherosclerosis. Bornfeldt's lab studies how lipid dysregulation in diabetes can contribute to the development of atherosclerotic lesions.**

without APOC3 were completely protected from atherosclerosis even when they were diabetic and had very high blood glucose levels. We think that APOC3 works by slowing the clearance of atherogenic remnant lipoprotein particles. We're now starting similar studies in people with Type 2 diabetes and also looking for other proteins that are involved in the pathway of remnant clearance.

**Q. You mentioned the mouse model that you developed. Do I understand right that it's a mouse with pancreatic  $\beta$ -cells that are inducibly attacked by its immune system?**

Yes, that's our main model of Type 1 diabetes. It's a really reliable model that accelerates atherosclerosis. We have also used other, more traditional models in the lab, like fat feeding to induce insulin resistance and a  $\beta$ -cell toxin called streptozotocin, but the T-cell mediated  $\beta$ -cell destruction model is our go-to. As with humans with

Type 1 diabetes, the diabetic mice need constant care — insulin injections, glucose checks and so on.

The main research area we're focusing on right now is centered on a group of lipoprotein particles called remnant lipoprotein particles. We think that those remnants can penetrate the artery wall and accelerate atherosclerosis and that they play a particularly important role in diabetes. And it looks like proteins that increase the accumulation of these remnants in the artery wall are very important in cardiovascular disease progression. How those remnants are related to inflammation and changes in immune cells is another area that we're very actively studying right now too.

**Q. Why are they called remnants?**

They are remnants of triglyceride-rich lipoproteins. There are two types of triglyceride-rich lipoproteins. One type is very large particles called chylomicrons that come from the gut following a meal. The other type is very low-density lipoproteins that come from the liver between meals. Both are larger particles than LDL, but they are acted upon by lipases that make them smaller and smaller by hydrolyzing the triglycerides carried in these particles — and then they are called remnants. These remnants can be cleared by the liver, or they can accumulate in the artery wall and promote atherosclerosis.

Actually, there aren't clear definitions of remnants or ways to measure them yet. As these particles become progressively smaller as they are hydrolyzed by lipases, they change in composition as well as size. We need better ways to measure them; that's another thing we're working on.

**Q. Tell me about your team. How many people are in your lab?**

Around 10 people. It's a good size and a really nice mix of collaborative people from different backgrounds and different parts of the world. The group includes undergraduate students, graduate students, postdocs and junior faculty, and sometimes MD fellows interested in cardiovascular complications of diabetes. And I'm lucky to have several amazing research scientists who often have their own projects in the lab and keep the wheels turning.

I've been very fortunate to have fantastic people in my lab. I couldn't do any research without this amazing group, and I'm hoping that I contribute to their excitement for science and to their careers.

**Q. Was there anything that you learned from your mentors along the way that you apply now as a mentor yourself?**

I've been extremely lucky to have such great mentors throughout my career. One thing that that Ross and Krebs taught me was to never let yourself be drawn down a path that you think might be right but your experiments show is not. If your experiments are not working, there is a reason, and that reason might be that your preconceived ideas are not correct. You should always keep an open mind and continue to ask questions. You might end up finding something unexpected. That's key in science, to keep an open mind and to not get stuck in a way of thinking.

Another important part of my personality that my mentors reinforced was to keep trying, never give up. You might not find the answers to your most important research questions for many years, or decades even, but if



you just have the determination to get the answer, you will often get there, and you will have advanced science in the process. We have come quite a long way in answering the question of how diabetes leads to an increased risk of cardiovascular disease. But of course, there are ever more questions to be answered. That is exciting to me.

**Q. What are your favorite things to do outside of the lab?**

I love spending time in nature, birdwatching and taking nature walks. Close to where we live, there is a beaver lodge, right next to the University of Washington campus. It's so peaceful to see these animals at sunset, only part of their heads showing above the water, chomping on water lilies. Sometimes, the osprey flies over. It's magical and rejuvenating. I often get new ideas related to the research in my lab during such times.

When she's not in the lab, Bornfeldt enjoys birdwatching.

**“It's so peaceful to see (beavers) at sunset, only part of their heads showing above the water, chomping on water lilies. Sometimes, the osprey flies over. It's magical and rejuvenating. I often get new ideas related to the research in my lab during such times.”**

**Laurel Oldach** (loldach@asbmb.org) is a science writer for the ASBMB. Follow her on Twitter @LaurelOld.



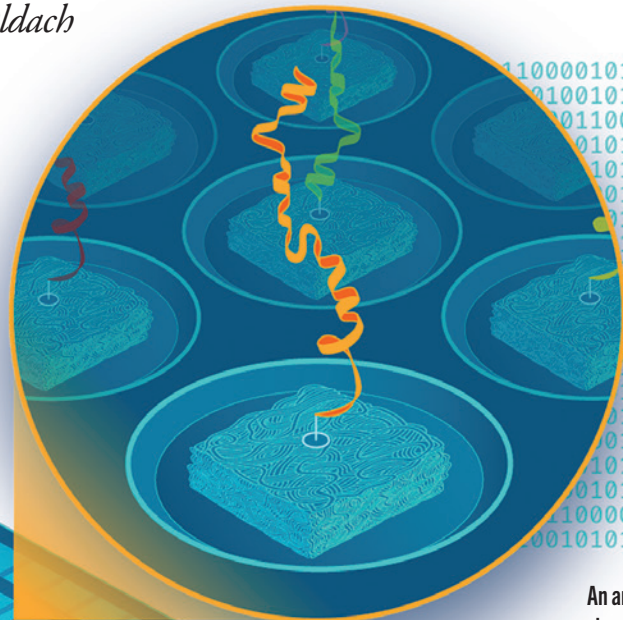


# Nautilus founder unspirals a new approach to proteomics

But Parag Mallick isn't leaving mass spectrometry behind

By Laurel Oldach

NAUTILUS BIOTECHNOLOGY



An artist's rendering of Nautilus Biotechnology's platform shows how individual proteins are adsorbed to a chip and repeatedly probed for short amino acid sequences.

**P**arag Mallick's latest project comes with an interesting challenge. "People keep trying to get me to say bad things about mass spectrometers," he said in a recent interview. "You're not going to get me to do it."

The goal of Mallick's company, Nautilus Biotechnology, sounds antithetical to the room-sized, half-million-dollar instruments. He intends to collect comparable data more cheaply using a benchtop instrument, proprietary reagents and an algorithm.

But Mallick, a Stanford University professor and founder and chief scientific officer of Nautilus, which recently went public, is the consummate mass spectrometry insider. Though his company's mission is to make proteomics more accessible — their slogan is "Anybody who wants a proteome gets a proteome" — he argues that the expanding field can accommodate both new approaches and established mass spec techniques.

Time will tell whether his generalist attitude can be balanced with a tech company's disruptive approach.

## The technology

Nautilus researchers plan to measure a proteome using a technique Mallick invented in 2016. It depends on repeated single-molecule imaging of a series of antibodies binding proteins fixed to a chip — Mallick compares it to a chessboard with 10 billion tiny squares.

Chip-based approaches to proteomics are not new; protein microarrays, in which proteins bind to antibodies adsorbed to known locations on a chip, have been in use for more than 20 years. The Nautilus approach is special because the protein sample, not the antibody probe, is fixed in place, with single proteins from the sample spaced apart by ligation to much larger polymer particles. More importantly, whereas a microarray is probed once, the proteomes Nautilus measures can be probed again and again using a line of antibodies developed in-house.

“People often do complain about their affinity reagents cross-reacting or not being specific enough,” Mallick said. “That was really the critical realization for us: to say, let’s take that concept to extremes as well, and build a class of reagents that are intentionally highly cross-reactive.”

The team developed hundreds of antibodies that bind to very short epitopes, as little as a few amino acids long, that are found in many proteins. They probe with one, wash it away, and probe with the next, collecting a series of images that shows whether the molecule at each location on the chip binds with antibody 1, antibody 17 or antibody 84.

“Each one of these touches, from each one of these probes, is just a tiny little nugget of information,” Mallick said.

Those nuggets feed a machine-learning algorithm that analyzes

terabytes of data per run, about 10 times as much information as is in a whole-genome sequencing run, and matches the characteristics of each spot to proteins in the proteome. The approach is particularly well suited to identifying proteoforms because of its single-molecule resolution. According to Mallick, although each binding event gives just a small amount of information about a protein, as few as 200 or 300 cycles might be enough to identify 95% of the human proteome, including specific proteoforms.

## The company

Though Mallick has invented a potentially revolutionary technology and founded a company that’s already attracting big-name venture capitalists, he comes off in interviews as careful, knowledgeable and a little self-effacing. He’s an amateur magician and juggler but hid the hobby for years, concerned that his colleagues might not take him seriously as a researcher if they knew he was performing sleight of hand in his spare time.

During a recent webinar sponsored by the Human Proteome Organization, Mallick discussed Nautilus with Mark Baker, a former HUPO president and past chair of its human proteome project.

That the technology behind Nautilus would nucleate a company was not a foregone conclusion, Mallick said in the webinar. When he first had what he called “a crazy brainstorm on a weekend,” he explored several options for making it a reality, including licensing the technology to a larger company or developing it within his university lab.

Before Nautilus, he was an academic, and he’s still a professor — albeit on partial leave — at Stanford. He trained in the lab of mass spectrometry and systems biol-

**Before Nautilus, he was an academic, and he’s still a professor — albeit on partial leave — at Stanford. He trained in the lab of mass spectrometry and systems biology pioneer Ruedi Aebersold, one of the most prominent researchers in the field.**



NAUTILUS BIOTECHNOLOGY

**Parag Mallick**

ogy pioneer Ruedi Aebersold, one of the most prominent researchers in the field. David Tabb, who was a graduate student at the same time in a friendly competitor's laboratory in Seattle, said that even then, Mallick was more focused on unsolved problems than on incremental process improvements.

Later, as professors, Tabb and Mallick, along with their students Matt Chambers and Darren Kessner, co-developed a software platform called ProteoWizard that could be used to analyze mass spec data no matter what type of instrument it came from. The two labs made the software openly available, and it has been cited almost 1,400 times.

Despite that open-science background, Tabb said, it's no great surprise that Mallick founded a company. "He's always been smart," Tabb said, adding that "Stanford, in general, has done a lot to incubate biotech businesses from its laboratories. ... I think he had a good idea and saw a way to pursue it."

When he was developing the idea

for the new technology, Mallick said, he concluded that solving problems in biochemistry, genomics, materials science, microscopy, machine learning and other fields would move fastest in a startup. "It can be a challenge to marshal that scale in academia," he said. "Even in the largest, most well-funded labs, you usually have a diversity of projects. We literally have every single person contributing to one goal."

Mallick reached out for advice to contacts in the business community, including Sujal Patel, an entrepreneur and investor. Patel became a cofounder and is now the company's chief executive officer.

"One of the lessons there is sometimes surprising things come from your network," Mallick told Mark Baker during the HUPO webinar.

## Will everyone get a proteome?

"Mass spectrometry is great for proteomics," Tabb said. "Is it always going to be the go-to technology for identifying proteins and protein differences? I think 100 years from now, we'll have very different answers to that question."

According to Baker, Nautilus is making a fair bid to become one of those future answers. In an email, he wrote, "Assuming Nautilus' platform can successfully quantify >95% of the human proteome (their vision) with a simple, fast and cheap sample prep and analysis, the concept could become as ubiquitous as sequencing a person's genome has become today."

Although he hopes the Nautilus platform will make proteomic experiments much more widely accessible, Mallick said that when it first rolls out, it will cost about the same per run as mass spectrometry.

Tabb, now a professor at Stellenbosch University in South Africa, said that while the Nautilus technology eventually may bring down the cost of acquiring a proteome, it is likely to remain out of reach for many researchers in the developing world. Instead, he said labs in poorer countries likely will have to find wealthier collaborators with access to instruments. That often can mean forfeiting senior authorship in order to get the experiments done. "That's the way things have unfolded with proteomics," he said. "That's the way things have unfolded with sequencing."

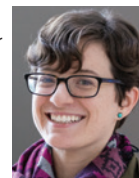
Whatever the cost of using a future system, it will be some time before Nautilus offers a product that any lab could buy. The company now is working exclusively with partners in the pharmaceutical industry, including Regeneron, on targeted assays that do not use its custom antibodies.

"It's very early days for Nautilus — they are only a recent spinout," Baker wrote, while also noting, "they are moving fast, they have the backing of major tried-and-true successful investors (and) they have launched on NASDAQ."

In the wake of its initial public offering on the stock market in June, which raised about \$200 million, the founders of Nautilus plan to double its research team and begin publishing on its approach to protein identification.

"It's a phenomenal opportunity to just go faster," Mallick said.

**Laurel Oldach** (loldach@asbmb.org) is a science writer for the ASBMB. Follow her on Twitter @LaurelOld.







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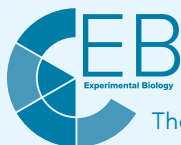
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# How to write a killer abstract in 10 sentences

*By Bill Sullivan*

The experiments were carefully designed. The data have been meticulously collected. The figures have been expertly prepared. The results are beautiful! All that stands between you and presenting your science at the 2022 American Society for Biochemistry and Molecular Biology Annual Meeting is writing an abstract. But where do you begin? How do you write an abstract that will get your science noticed?

Each day, we are bombarded with a deluge of information, each item vying for our attention. We rely on eye-catching headlines to draw us toward the content that might enrich our lives. In the scientific arena, we rely on abstracts to provide snapshots of studies that may be relevant to us. Abstracts are a crucial filter that helps us to gauge quickly which reports must go to the top of our to-read list.

Previously, I've written how to present a killer research seminar by pretending you are serving the audience a three-course meal at a mystery dinner theater. The appetizer introduces the scientific puzzle as a mystery. The main course is composed of the experiments that uncovered clues. The dessert course satisfies the audience by revealing how the clues addressed the mystery.

Below, I outline how this formula can be adapted to craft an irresistible abstract that will make people hungry to see more of your work.



## **Sentences 1–2. Set the stage.**

The beginning of a killer abstract must convey the scientific question that keeps you up at night and why. Use the first couple of sentences to describe succinctly the most salient features of the phenomenon you are investigating and, if applicable, how it is relevant to a medical or environmental problem. The stage you set should draw attention to a gap in our knowledge that you are attempting to fill.

## **Sentence 3. State the mystery.**

The next sentence is a clear and focused hypothesis framed as a question — this represents the mystery your study aims to solve. It should be an interesting and intuitive question derived from the background information in your introductory sentences. If the reader cannot guess your hypothesis after reading that introduction, you haven't set the stage well enough.





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## Sentences 4–8. Describe your detective work.

This section is the heart of the experimental effort you used to gather clues relating to the mystery. Explain the case as Sherlock would to Watson, concisely mentioning the rationale and techniques used to unearth the clues.

Like discriminating detectives, scientists demand precision. If you are vague, your poor reader is forced to wonder what you mean. For example, the statement “Transcript levels were greatly altered in the knockout” leaves people with questions: How many transcripts? How were they altered, and by how much? Instead, write, “We determined that 75% of transcripts increased twofold or more in the knockout.”

You do not want to disrupt a compelling story by going off on distracting tangents. You need not mention every experiment you completed, only those that provide the most important clues needed to answer the question.

## Sentence 9. Solving the mystery.

This is the sentence the audience has been waiting for, but if you’ve laid out the clues adequately, they should be able to deduce the conclusion themselves. This sentence should state clearly how your findings contribute to solving the mystery you posed in the introduction.

It is important to note that you don’t need to solve the mystery completely to make a compelling story. Negative data or replicative studies are vital to the scientific enterprise, providing critical clues for resolving biological questions.

## Sentence 10. So what?

The final sentence of your abstract should remind the reader why your findings are important — for example, in terms of providing new insights into behavior, mitochondrial function or gene expression. If applicable, mention how these findings may facilitate the development of new therapies for a disease.

In summary, a killer abstract tells a story by framing your scientific study as a mystery. As with all good stories, you’ll also want to devise an alluring or clever title to capture attention.

Remember: Conference organizers and participants have many abstracts on their plates. Be sympathetic to their plight and make yours enticing and easy to digest. You can keep it simple by avoiding technical jargon wherever possible, defining esoteric terms that must be used, and eliminating unnecessary words or phrases. Avoid the temptation to add extraneous information or research results that are superfluous to your story.

Finally, take advantage of the many resources available to assist writers. Be sure to use tools for checking spelling and grammar. Make certain that you include relevant keywords in your abstract; many people use them to scan the abstract book.

And don’t forget the most important resource of all — readers. Don’t submit an abstract before taking it on a few test spins by getting others inside and outside your field to read it. If they are not intrigued, you need to go back to the kitchen and cook up a better dish.

**Bill Sullivan** ([wjsulliv@iu.edu](mailto:wjsulliv@iu.edu)) is a professor at Indiana University School of Medicine and the author of several books. He is a member of the ASBMB Today editorial advisory board. Follow him on Twitter: [@wjsullivan](https://twitter.com/wjsullivan).



# US travel bans: Bizarre and ruthless

By Valerio Francioni

In March 2020, just a week after my arrival in the U.S. to start a postdoc position at my dream lab in Cambridge, Massachusetts, then-President Donald Trump temporarily banned noncitizens who had been physically present in the U.K. and the parts of the E.U. known as the Schengen area in the previous two weeks from entering the U.S. I perceived this ban as an inconvenient yet cautious act aimed at containing a public health emergency. At the time, SARS-CoV-2 was not well understood or controlled in several European countries, including Spain and my home country of Italy.

Almost 17 months later, the presidential proclamation(s) remain in effect and even have been expanded. They now include the 26 Schengen countries, the U.K., the Republic of Ireland, South Africa, Brazil, India, China and Iran. If I were to travel to any of these countries from the U.S., I would be unable to return. I have not seen my family in a year and a half.

When Joe Biden was elected president, my hopes for a visit home skyrocketed. After all, his agenda included strong international cooperation, welcoming policies and science-based decision making. Surely, the days of travel bans on Europe and European scientists were numbered.

I was therefore disconcerted that one of Biden's first executive actions was to reintroduce, on Jan. 27, the travel bans Trump had set to expire



Biden's reaction, in March 2020, to the travel ban imposed by Donald Trump on European countries.

two days before.

"OK," I thought, "nothing to worry about. January is still early. Cases are still rising, and vaccines are on their way but not quite there yet. Just hold on, and you will soon be able to see your family."

Time passed. Vaccinations gained momentum both in the U.S. and in Europe, leading Biden to declare in April that the U.S. would lift the U.K./E.U. travel ban by mid-May. I could not contain my excitement. I finally would be able to travel home.

Come May, however, there was no news about lifting restrictions on international travels.

June, no news. July, no news. No presidential declaration, no information on consulates' or embassies' websites. Nothing concrete in terms of policy changes. Nothing other

than speculation in a few newspaper articles.

Hope was turning into frustration. Eventually, pressure started to build: Journalists asked questions, airline and tourism companies lobbied the government, European diplomats got bothered about the lack of reciprocal immigration policies, and eventually the government was forced to provide some answers.

According to an April 30 presidential proclamation, "The national emergency caused by the COVID-19 outbreak in the United States continues to pose a grave threat to our health and security. ... It is the policy of my Administration to implement science-based public health measures, across all areas of the Federal Government, to prevent further spread of the disease."

## PERSPECTIVES

This was the justification to keep travel bans. Thereafter, government officials repeated the phrases “science-based,” “data-driven” and “evidence-based decision making” as if they were a mantra. Biden recently reiterated his intention to keep travel restrictions in place.

Remarkably, this science-based approach only applies to certain categories of visa.

For example, M-1 and F-1 visa holders (largely students), green card holders and U.S. citizens are exempt from the restrictions. We still have much to learn about COVID-19; however, virologists would agree that susceptibility to infection does not change according to the visa type you hold.

To circumvent the restrictions, a traveler must obtain a so-called National Interest Exception, or NIE, from a U.S. embassy or consular post after they are already abroad. The NIE is granted only to a few categories

of loosely defined essential workers. Consular posts can take up to 90 business days to reply to an applicant’s request, and approval is highly uncertain — the outcome depends on the consulate’s arbitrary discretion.

Some visa types are subject to further complicating restrictions; for example, under standard conditions, J-1 visa holders (mostly academics) are not allowed to leave U.S. soil for more than 30 consecutive days. The long time required to process NIE applications, combined with the fact that a request cannot be submitted until the applicant is outside the U.S., makes it impossible to apply without endangering one’s work and related immigrant status. The 90 days required to process NIE applications become even more grotesque if one considers that an average postdoc has between 15 and 20 days of vacation per year.

Aware of the absurdity of the situation, U.S. consulates and university

international offices recently started to suggest NIE applicants take what is now known as “the third-country approach,” spending two weeks in a country not affected by the ban before reentering the U.S. These countries include Turkey, Mexico and Russia, places where the pandemic is still raging and vaccination programs lag behind those in Europe.

Such an approach is not based on science.

Forcing European researchers to travel to a third country exposes us to heightened safety concerns and financial burdens. Ultimately, it likely increases the spread of COVID-19 by forcing people to move through infection hubs such as airports, hotels and restaurants in several countries with high infection rates.

I would expect a science-based approach to protecting public health to allow travel from countries that have higher vaccination rates and lower infection rates while banning travel from countries where rates are worse. As of late July, however, this was not the case. In banned countries such as the U.K., Italy, Spain and Germany, more than 60% of the population had received at least one dose of vaccine, compared to 46% in Turkey, 30% in Mexico and 23% in Russia (and 55% in the U.S.).

When it comes to daily infection rates, the situation is no different. Mexico, the country with the fewest new daily cases among the three cited above, has twice the infection rate of Italy, six times the infection rate of Germany — and a 20% lower rate than the U.S.

These numbers are hardly reconcilable with a data-driven approach to travel bans.

Keeping the new delta variant at bay is also not a viable justification for the ban; according to the Centers for Disease Control and Prevention, the

In this reply to an email inquiry from an NIE applicant, the U.S. embassy in Rome recommends traveling to a third country.

### Re: NIE request

Dear [REDACTED],

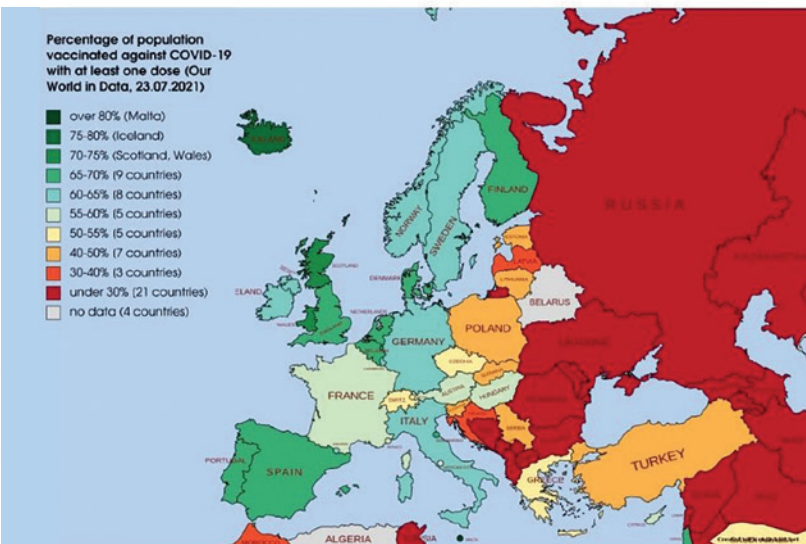
Please note that the current wait time is at least 60 business days. **If you have a valid ESTA or relevant visa, one option currently available is to spend at least 14 days in a country not listed in the Proclamations before attempting to enter the United States.** Please keep in mind that those transiting a country listed, even if one does not leave the airport, will be refused entry into the United States. Customs and Border Protection has jurisdiction over entry into the United States, not the U.S. Embassy. As such, we regret that we cannot provide further guidance on entry into the U.S., but this website might be useful: <https://help.cbp.gov>

Regards

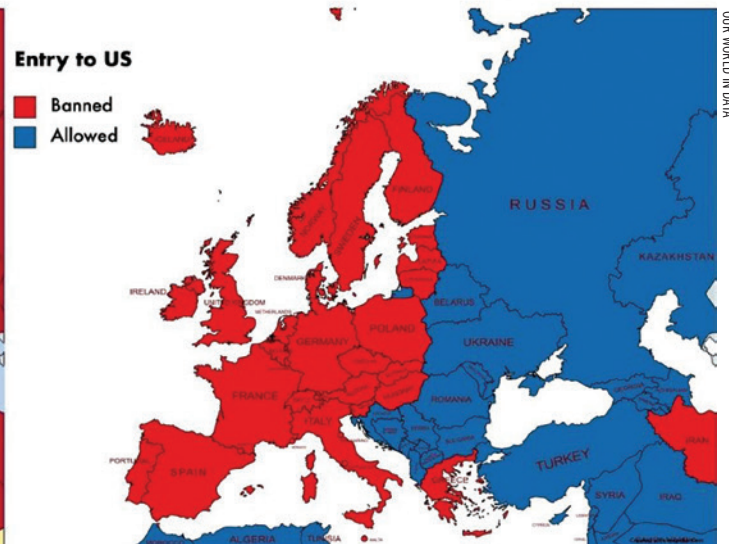
COURTESY OF VALERIO FRANCONI



## Vaccination Rate (at least 1 dose)



## US TRAVEL BAN (current, July '21)



These maps show the percentage of the populations in European countries that are vaccinated with at least one dose (left) and the countries that are impacted by the U.S. travel ban (right).

delta variant now accounts for 83% of new Covid-19 infections in the U.S. Considering the high efficacy of vaccines against symptomatic infections by delta, unvaccinated Americans are likely to pose a greater threat to public health than vaccinated Europeans traveling internationally.

The situation is bad, and the end is not in sight.

Even if President Biden revoked all travel restrictions tomorrow, a shortage of personnel and funds combined with the blockade of visa issuances to people who do not qualify for an NIE (but who otherwise legally would qualify for a visa) has created backlogs at U.S. consulates and embassies in banned countries that will take months to dissolve. This extends delays for people who need to receive or renew their visas. In June, the next available appointments for visa renewal at the U.S. embassy in Paris were almost a year away. This de facto

visa ban now faces a legal challenge.

My American colleagues now can take a selfie in front of the Basilica di Maria Maggiore in Rome, enjoy an aperitivo in Florence or marvel at the Sicilian coastline. Tourists from many countries can visit New York or Yellowstone National Park while I, and millions of other international visa holders, have been kept from visiting our families for almost a year and a half.

A lot happens in a year and a half. Among cases that made it to the news is that of the Bloomberg anchor Jonathan Ferro, who had to watch his father's funeral on his iPhone. But this is just the tip of the iceberg. The Instagram page Bring US Home is gathering stories of people affected by the travel bans, and some of them are distressingly sad. Couples have been separated since March 2020. Grandparents are unable to meet their newborn grandchildren. Adult children can't help their ailing parents.

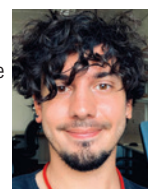
I was absent when my father had a tumor removed and my grandfather faced a life-threatening COVID-19 infection. I missed my brothers' birthdays when they turned 11 and

15, and then 12 and 16. I couldn't attend my own Ph.D. graduation ceremony. Fortunately, none of my stories ended tragically, but not everyone has been so lucky.

Such stories are not uncommon. Most international scholars have not been able to visit families or significant others for more than a year, with an indefinite wait ahead. This is affecting our mental health; many of us must choose between being present for the births, weddings and funerals of friends and family and the security of a job and immigration status in the country where we have established our lives.

A letter about the travel ban signed by more than a thousand scientists was submitted recently to the editorial board of the journal Nature. We hope it will help our voices be heard.

**Valerio Francioni** ([valeriof@mit.edu](mailto:valeriof@mit.edu)) is a postdoctoral scholar in the brain and cognitive sciences department at the Massachusetts Institute of Technology. He earned his Ph.D. from the University of Edinburgh. Follow him on Twitter: [@ValerioFrancio1](https://twitter.com/ValerioFrancio1).



# Surviving and (sometimes) thriving as a department chair

By Pam Mertz, Teaster Baird Jr. & Joseph Provost

About five years ago, we started what we call a “chair chat” network as an informal support group for chairs of biochemistry and chemistry departments at primarily undergraduate institutions. As faculty at public and private institutions of varied sizes, members of the group bring diverse perspectives to conversations about leadership. We have brainstormed solutions to address challenges in our departments and supported each other in the transition from faculty member to department chair.

Here we share some experiences and advice, which may help those who are already department chairs and those who may become chairs in the future.

## 1. How did you become chair?

**Pam Mertz:** The position is a three-year term at my college, rotating through tenured faculty members. We are a relatively small department with eight tenure-track or tenured faculty, so it was inevitable my turn would come. I put myself forward at a time when I thought I could do a lot to move some important department initiatives forward such as revising the curriculum and creating clear guidelines for faculty for promotion.

**Joseph Provost:** I was reluctant to become a chair but felt I should.

I have had a few leadership positions; in the Army, I was a chemical warfare officer and ended as a commander of a National Guard infantry headquarters company, and I was the CEO and COO of two small biotech companies. Thus, the call to service in academic leadership positions, including positions in the American Society for Biochemistry and Molecular Biology, the Council on Undergraduate Research, the American Chemical Society and Project Kaleidoscope, naturally fell into place. At both my last and current universities, my department needed a chair, and I felt a duty to fulfill that mission. I thought I could and should help the institution, even though the position took an incredible amount of time and competed with research, and personal goals often are pushed down the priority list.

**Teaster Baird:** For me, it was a combination of opportunity and a desire to try something new. I just had been promoted to full professor the semester before I became chair, and I was looking for new challenges, specifically ways I could have an impact on the greater university community. Being a chair wasn't on my planned career trajectory, but our chair was leaving in the middle of her term to take advantage of a new opportunity within the university. She encouraged me to consider taking over as

interim chair for the remainder of her term. I saw the “interim” tag as a way out in case I didn't like being chair.

## 2. What personal traits did you need to work on to succeed as chair?

**PM:** I had to learn how to communicate effectively with the provost and the president of my college. Specifically, I needed to be more aggressive in ways I might not have been as a faculty member. I needed this skill when negotiating for as much startup funding as possible for a new faculty member, speaking during department chair meetings on important issues for the faculty in my department, and negotiating with the provost on issues related to faculty course schedules or adjunct pay.

I also needed to think in advance about how to ask for support; I had to come up with solutions to problems and know what specific resources to ask for. I also sent routine emails, once or twice a year, to let the administration know what the faculty in my department had accomplished.

**JP:** Most people don't know that my inner voice says, “Let's get it done, let's quit discussing an issue to death.” I like to get things done. Early in my career, I didn't keep that inner voice inside, and I was a little too pushy, which wasn't always helpful in an academic setting. The dominant



voice has diminished some, and I have learned through experience that, especially in academia, people need a chance to share their voices. I am now happy to let a discussion get a little messy. I've worked on how to keep a conversation moving, to encourage other voices to be heard and to know when it is time to move to a decision.

**TB:** I made many adjustments, but I had to make the biggest change in two related traits. First, I had to learn to make my voice heard. I am not an outgoing and forward person by nature, so that requires effort. As chair, I represent the faculty, staff, and students in the department to the university decision-makers who control the flow of resources. I have to make sure they know and consider our department's needs and desires for resources and our positions on issues.

Second, I had to learn to navigate

various professional circles in the university. As a faculty member, I spent most of my time with other faculty within my department, so I knew that world pretty well. As chair, I communicate and interact with the greater university community, which doesn't always operate the way my department, or even my college, does. I find myself doing what I call "professional behavior translation" quite often.

### 3. How did your relationships with colleagues change when you became chair?

**PM:** I strove to be a fair chair and not play favorites with any faculty members in my department. I needed to handle a variety of conflicts and consider a number of different perspectives — students, faculty and

administrators, depending on the issue or conflict. This sometimes shifted my relationship with my department's faculty from friendly colleague to more of a manager role. Some relationships were definitely strained, but I believe that was temporary while challenging issues were being resolved.

**JP:** Sometimes I joke that the scariest thing a chair can hear is a knock on the door followed by "Do you have a minute?" From some of these impromptu conversations I learned many things about my department — things that are a chair's responsibility to lead and manage. That means making decisions that aren't going to make people your friends. Our chair chat group discovered that we learn many things about our colleagues. I try hard not to take comments from staff or faculty members personally, but it isn't easy. Having this group outside



the department and the university that I can talk things out with and being able to vent helps me keep good relationships with colleagues.

**TB:** Getting to know my colleagues more fully has been the biggest change in my relationships. As a faculty member, I could easily excuse myself from difficult or uncomfortable situations. As chair, that's not an option. In fact, those situations seem to seek me out. I have learned what people are passionate about, what they enjoy and do not enjoy doing, and how they respond to certain situations and ideas. I try to be fair and impartial and transparent. I don't think I've made any enemies, but I've been in a couple of tense situations.

## 4. What opportunities came your way outside of your department as a result of serving as chair?

**PM:** I became co-leader of a National Science Foundation-funded Council on Undergraduate Research Transformations project to scaffold research experiences into our curriculum. My department has been working on it for the past four years, and my role as chair was instrumental in helping to make this project a department priority so that we could make significant progress. For example, I set up meetings dedicated to project work and aimed to keep other department business out of these conversations, despite the presence of ongoing issues. I am also leading my department in the design of a new teaching lab space for biochemistry. I was department chair when planning for renovations

started, and I have continued to lead the project.

**JP:** As Pam describes, a number of internal opportunities opened up after I became a department chair. Also, I had never thought before about if or when I might consider another level of academic leadership. Prior to my latest chair position, I would get an occasional letter or email from a search firm, but now the queries are constant. I also get more requests from other universities asking for information and potential networking. I'll now often get a request to be an external reviewer or just to share information about departmental policies and practices. These are all great opportunities to expand my network of peers.

**TB:** Internal opportunities have also come my way because of my position as chair. I have served on several university-level committees that specifically asked for the involvement of department chairs. I have had the opportunity to meet and interact with influential people outside of my college, such as individuals in upper administration, and learn how the university operates and what it takes to make real and lasting change at the university level. For example, I had the opportunity to serve on a committee to advance diversity, equity and inclusion in faculty hiring because I was a department chair.

## 5. How do you attempt to maintain work-life balance?

**PM:** This is an ongoing endeavor. I am working to set better boundaries with regard to my work and personal life, making sure I spend quality time with my husband and son. I like to

hike and garden, and I am an amateur potter. Working with clay and being creative allows me to set aside work issues and other concerns.

**JP:** I have always liked to do a lot of things. However, I am horrible at the whole work-life-balance thing. Sometimes I am not even sure that to be successful, the balance is balanced. Over a larger time scale there has to be some sort of balance but rarely on a day-to-day or even week-to-week scale. However, helping is my very supportive wife and that my three children are grown up. This is good, as I spend way too much time on chair duties. When I started my current position, I kept track of and graphed the amount of time I spent on chairing, teaching, research and miscellaneous professional duties over a semester. That graph was depressing, but it helped me realize that a 70-hour week was unsustainable. My summers focus on chair work, and research drops to about 45 to 50 hours a week. It's still not where I want it to be and a work in progress. I now try to play hockey and spend more time on my stained glass and playing drums and guitar. Those activities come in bursts, often not frequent enough.

**TB:** Learning to set boundaries is absolutely essential. I find it helpful to recognize—and remind myself—that not every situation is an emergency that demands 100% of my attention at that very moment. Some things can actually wait. I also try to make sure my time with my family is committed time and not an afterthought. I limit my screen time, and I won't answer calls or check emails on my phone if I'm spending time with them. I also play the piano and dabble in photography for my own personal escapes.

## 6. What advice would you give to a new department chair?

**PM:** Expect the unexpected and learn to go with the flow. The pandemic illustrated the many ways department chairs have to be prepared to respond to crises and adapt. Don't assume you can schedule out your week in advance; you never know what concerns or requests will show up at your department chair door. These requests can come from students, faculty, or your dean or provost.

Have a vision for ways you can work with your faculty to improve the department. During my time as chair, we scaffolded research experiences throughout our curriculum to provide support for all our students, and we developed detailed guidelines for faculty scholarship, teaching and service expectations.

**JP:** I've tried hard to keep my research group of eight to 12 undergraduates going, write grants and publish. It hasn't been easy and has been much slower than I had hoped. But it's important to keep going. Find a way to not give up on research; find a way to focus on the most important aspects of research and keep swinging at the low-hanging fruit. It is entirely too easy to put going into the lab off until later. I've done that too many times.

Know the difference between leadership and management. A good chair needs to be both a leader and a manager. Making decisions and setting a path with the department is leadership; managing resources and setting schedules are management. Finding a way to involve faculty in



achieving a goal but keep things focused is leadership (something I still need help with).

**TB:** A new chair needs to understand and accept that most people will not fully understand what you do. I didn't fully appreciate the job(s) of the chair until I became one, and that's one reason I was so happy to be invited to join the chair chats. Not only were Pam and Joe trusted friends, they were also chairs and could relate to my experiences and give specific and relevant advice. Sometimes, we'd just have venting sessions.

That leads to my second piece of advice: Get a support group. Talking with Pam and Joe has helped me successfully navigate some sticky situations and know that many of my experiences were not unique.

**Pam Mertz** ([psmertz@smcm.edu](mailto:psmertz@smcm.edu)) is a professor of biochemistry at St. Mary's College of Maryland. She is the chair of the ASBMB Student Chapters Steering Committee and previously served for many years as Student Chapters director for the Southwest region.



**Teaster Baird Jr.** ([tbaird@sfsu.edu](mailto:tbaird@sfsu.edu)) is a professor and chair in the chemistry and biochemistry department at San Francisco State University. He has served on the ASBMB Student Chapters Steering Committee and the Public Outreach (now Science Outreach and Communication) Committee. He is a 2021 ASBMB fellow.



**Joseph Provost** ([josephprovost@sandiego.edu](mailto:josephprovost@sandiego.edu)) is a professor and chair of the chemistry and biochemistry department at the University of San Diego. He served as chair of the ASBMB Student Chapters Committee for five years as well as on the Educational and Professional Development Committee and the Membership Committee.



# Getting the shots

A real-life science communication story

By *Hannah Alexander*

I recently took a Lyft ride in Chicago. The driver was a woman in her late 50s. We chatted during the entire ride. I learned that she is a mother of three sons and a grandmother.

She had been driving throughout the entire pandemic, and, because she had to spend time in close proximity to so many people, I assumed she had been vaccinated for COVID-19 as soon as she possibly could. She had not. She said her three adult sons were not vaccinated either.

She told me she was still hesitant and fearful. I told her the data show that vaccination is safe, that getting the disease is a lot more dangerous and has much worse consequences than the vaccine. I told her that my work used to be immunization related, so I know that it is the right thing to do. And so on and so on.

She kept saying, “You make good points,” but she also said that she’s not sure what happens when a person gets the vaccine. I said nothing bad happens; at most, you might feel bad for a day or two, just like when little babies get vaccinated.

She: “What? They give it to babies too?”

Me: “No, not yet. I meant when babies get vaccinated against childhood diseases.”

She: “I’m not sure what you mean.”



Me: “You know, when you take babies to the doctor for the two-, four- and six-month visit, and they get vaccinated?”

She: “Oh! You mean when they get shots.”

And just like that it all became clear and incredibly sad to me. I realized two things: First, she does not understand the word vaccination (let alone immunization). She knows “shots.” But the people who try to convince her to get vaccinated do not use the word she knows (including me, throughout our entire conversation, even though I have 13 years of experience in practicing science communication). So having a story on the news that uses words such as efficacy, variants, mutation and virulence does not help much.

It might be making things worse. Second — and more disconcerting — she clearly does not understand what happens when babies “get shots.”

Where do we go from here? I am not sure. We scientists think that the vaccine and its mechanism of action have been explained a million times to everyone. But, at least in this case, it’s been explained in a language some people do not understand.

So here’s my message.

**To science communication and outreach practitioners:** Our message can never be too simple when we reach out to the lay public. We need to avoid using professional terms (yes, even vaccination and immunization fall into this category). We need to be aware of the public’s frustration at seemingly contradicting messages



— wear a mask, don't wear a mask; the vaccine will protect you, the vaccine won't protect you 100% — and patiently explain that this virus is new to the landscape, that scientists are figuring it out as we go along, and, as such, with new information, guidelines may change. We need to realize that, yes, for some the resistance to vaccination is out of spite or politically motivated, but for many it is out of real fear, primarily as the result of a lack of understanding and lack of correct information.

**To pediatricians and family doctors:** Please take a minute to explain what is happening when you vaccinate — “give shots to” — babies and small children.

**To high school science teachers:** Please tackle the issue of immunization in your science classes; seek ways to explain in simple terms what the process is and what the ramifications are. You have a unique conduit into people's homes.

**Most importantly, to our country's leaders who are battling COVID 19 — the government, the Centers for Disease Control and Prevention, the National Institutes of Health, the medical personnel, and the scientific community at large:** Please simplify your message. Explain in a few very simple words what happens when a person gets vaccinated and what happens when a person doesn't, and please call it “getting shots.”

**Hannah Alexander** (alexanderh@missouri.edu) is a research associate professor emerita of biological sciences at the University of Missouri. She served six years on the ASBMB Science Outreach and Communication Committee and recently mentored her 14th group in the ASBMB's Art of Science Communication course. Follow her on Twitter: @HABOCOMO.



## CALL FOR SUBMISSIONS



### DISABILITY EMPLOYMENT AWARENESS

#### October is National Disability Employment Awareness Month.

To mark this observance, ASBMB Today welcomes essays, interviews, opinion pieces and other articles relating to disabilities and doing science.

We encourage submissions from people with disabilities, employers/managers, researchers, allies and others who wish to share personal experiences, inclusive practices, lessons learned and advice, recent disability employment research and perspectives.

**Email submissions to [asbmbtoday@asbmb.org](mailto:asbmbtoday@asbmb.org) with the subject line “Disability Employment” or use the Submit link at [asbmb.org/asbmb-today](https://asbmb.org/asbmb-today).**

**Deadline: Dec. 31.**

(While the Department of Labor observance is in October, we will publish these submissions from October onward until we run out.)

**ASBMBTODAY**

## A turbulent industry: 5 questions with Paul Wright

By Laurel Oldach

After finding his first job at an American Society for Biochemistry and Molecular Biology meeting in the 1980s, Paul Wright spent his career in the pharmaceutical industry. Now retired, Wright serves on the ASBMB's Industry Advisory Committee. This interview has been condensed and edited.

### 1 What did you work on?

Quite a few things. I was with Sanofi for 28 years; when I started, it was called Merrell Dow Pharmaceuticals in Cincinnati. They were acquired, and then they were acquired and then they were acquired, until finally it became Sanofi. Later, Sanofi spun off their site here in Tucson to Icagen.

As the owners changed, I would find a position within the company that fit my background. I ended up working in different therapeutic areas: parasite biology, molecular pathology, metabolic diseases and oncology.

I've worked at the very early end of drug discovery, using combinatorial chemistry and high-throughput screening to identify lead compounds. I've led project teams carrying compounds into preclinical research and I worked on the clinical project team for one drug candidate.

### 2 After all those changes, do you feel there's an optimal company size?

I really liked a small to midsize company, big enough that they could fund their projects but not so big that there was a gap between the



Paul Wright

#### CURRENT POSITION

Retired

#### CAREER PATH

Ph.D., Purdue University

Postdoctoral research: University of Iowa and University of California, Davis

First job outside of academia: Associate scientist, Merrell Dow Pharmaceuticals

#### FAVORITE MOLECULE OR PROTEIN

"I'm split between the cyclin-dependent kinases and VEGF (vascular endothelial growth factor)."

people running the company and the research divisions. In a large company, you may have less opportunity to communicate directly with senior leadership.

### 3 Is there a project that you're proudest to have worked on?

Doing compound discovery for angiogenesis early in my career was very rewarding. At that point I was still able to devote my time more purely to research. Later, I went from being a lab scientist to a project leader, then a department director.

Those were rewarding, but I feel best about the work I did right at the interface between research and project leadership.

### 4 What's most important to know about leading scientists?

For me, the challenge was to keep good people involved at a high level in their projects, while realizing that in two or three years, it could be quite different. It's hard to say, "If you accomplish this, it's going to have this final outcome for the company." You have to train people to realize that things will change every few years, and they're going to have to be ready to carry their skill set into a different area.

### 5 Advice for aspiring industry researchers?

An internship or a postdoctoral stint is a great starting point. It's an opportunity to see how a company works, how you might fit in and whether you would like to fit in.

If you've developed a research interest, see if you can find a company that's looking for someone in that area. If not, be flexible to get a start. See if you can find a good working group in a nice environment. Can you bring your skills, your talents into that and have an impact?

Laurel Oldach (@oldach@asbmb.org) is a science writer for the ASBMB. Follow her on Twitter @LaurelOld.





# Planning a scientific conference?

The ASBMB is here to help.

The ASBMB provides a variety of opportunities for its members to bring people together, both virtually and in person, to share their research, make connections and cultivate the scientific community. From webinars, to networking get-togethers, to multi-day conferences, the ASBMB will help you to bring your event to fruition.



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Recently added member content includes the following titles:

- Mentoring from both sides: How to find, be and utilize a great mentor
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- Inclusive teaching: Supporting undergrads and grads in in-person and remote classrooms and labs
- Workshop and networking for inclusive practices and inclusive course content
- Improving visual literacy using AR and LEGO® bricks in biology classrooms
- Science policy and advocacy for early-career researchers

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# CLASSIFIEDS

## Faculty Position in Microbiome Biochemistry

Duke University School of Medicine

The Department  
of Biochemistry  
and the Duke



Microbiome Center at the Duke University School of Medicine in Durham NC invite applications for a tenure-track faculty position. The most appropriate candidates will be those whose activities complement and integrate research programs within the Department of Biochemistry and Duke Microbiome Center and should have demonstrable expertise in the biochemistry, chemical biology, or biology of the microbiome.

<https://careers.asbmb.org/job/faculty-position-in-microbiome-biochemistry/58005571/>

## Assistant Investigator

The Stowers Institute for Medical Research

The Stowers Institute for  
Medical Research invites  
innovative early career



scientists in the Life Sciences to apply for an Assistant Investigator position in 2022. Research programs of interest include, but are not limited to biochemistry, biophysics, genetics, neuroscience, cell biology, genomics, epigenetics, evolution, quantitative biology, and synthetic biology. Our interests run the gamut of experimental organisms and approaches.

<https://careers.asbmb.org/job/assistant-investigator/57918791/>

## BioCAT User Support Specialist

Illinois Institute of Technology

BioCAT is seeking  
an experienced  
biochemist to



**ILLINOIS TECH**

provide user support for our solution small angle X-ray scattering (SAXS). You will have primary responsibility for supporting both in-person and remote/mail-in SAXS users, and maintaining the state-of-the-art sample preparation and sample delivery and characterization equipment. You will also have the opportunity to collaborate scientifically with users if desired.

<https://careers.asbmb.org/job/biocat-user-support-specialist/57907362/>

## Chair, Department of Biochemistry & Biophysics

Weill Cornell Medicine

Weill Cornell Medicine,  
the medical school  
of Cornell University,



**Weill Cornell  
Medicine**

is searching for the next Chair of the Department of Biochemistry & Biophysics. Reporting to the Stephen and Suzanne Weiss Dean of Weill Cornell Medicine, the department chair is a member of the senior research leadership team and works collaboratively with the central administration, fellow chairs, center and institute directors, and faculty members.

<https://careers.asbmb.org/job/chair-department-of-biochemistry-biophysics/58064004/>

To see a full list of jobs, please visit [careers.asbmb.org](https://careers.asbmb.org)

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