

From ToxPipe to FAIRkit

NIH-Built AI Chatbots Are Helping Scientists Sift Through the Data

BY PAIGE JARREAU, NIA; AND THE NIH CATALYST STAFF

ARTIFICIAL INTELLIGENCE (AI) tools are taking root across NIH, reshaping how researchers access information, analyze data, and advance biomedical discovery. From generative chatbots that streamline scientific queries to machine learning models that help harmonize massive datasets, AI is proving to be a powerful partner in tackling complex hypotheses in research topics spanning from toxicology to dementia and beyond. There are many, so let's chat about 'em!

Chatbot creation for NIH research

Speakers at a June 11 NIH Library event that featured members from the NIH Generative AI Community of Practice showcased a range of AI-driven chatbot initiatives under development across the agency. Speakers and topics at a roundtable discussion, archived on the NIH Library YouTube channel, included:

- “Generative AI Chatbots in the NIH Landscape: Foundations, Opportunities, and Considerations” by Alicia Lillich, NIH Library
- “Chatbot for the Intramural Research Program, or ChIRP,” by Steevenson Nelson, OD
- “ToxPipe: Chatbots and Retrieval-Augmented Generation on Toxicological Data Streams” by Trey Saddler, NIEHS

CONTINUED ON PAGE 8 ➡

Calling All Creators and Innovators to the BETA Center Makerspace

BY CHRISTOPHER WANJEK, THE NIH CATALYST



CREDIT: JASPER SHIDE, NINDS

Let us drill this into your head: the BETA Center's Makerspace is now open for business in Building 31. These 3D-printed skulls demonstrate the scaling capability at the Makerspace. The life-size skull is used as an example of encephalocele, a defect where the skull does not fully close during development. The Makerspace team additionally printed a life-size infant skull model with a defect from a medical scan to illustrate how it can support the workflow of solving a surgical problem.

LOOKING TO BUILD A CAMERA MOUNT, A MOUSE MAZE, OR A CUSTOM polymerase chain reaction rack—or just want an excuse to use a laser cutter? The NIH has you covered. NIBIB opened a new fabrication facility on the Bethesda campus housed in Building 13 and part of its Biomedical Engineering and Technology Acceleration (BETA) Center. The facility is called, simply enough, the BETA Center Makerspace,

CONTINUED ON PAGE 4 ➡

CONTENTS

FEATURES • [\[1, 4\]](#) Calling all Creators and Innovators [\[1, 8\]](#) From ToxPipe to FAIRkit [\[3\]](#) The Reservoir Within: How Nitrate Secures Our Nitric Oxide Supply [\[12\]](#) Sweetening the Pot While Empowering Others [\[13\]](#) **SNAPSHOTS:** Annual Margaret Pittman Lecture

DEPARTMENTS • [\[2\]](#) DDIR: Before the Curtain Rises [\[6\]](#) Catalytic Research [\[10\]](#) Colleagues: Recently Tenured [\[14\]](#) News You Can Use [\[15\]](#) The SIG Beat [\[16\]](#) Photographic Moment



Before the Curtain Rises

Readying the Public—and Ourselves—for Open Access

BY NINA F. SCHOR, DDIR

WE ARE EMBARKING ON A FUNDAMENTAL change in how scientific information is shared with the world. As of July 1, all NIH-funded research appearing in scientific journals must be made freely and publicly accessible as soon as it is published. On its face, this may sound like progress. But if we are not careful—if we do not prepare both the scientific community and the public for what this change means—we may inadvertently damage the trust we hope to build.

To be clear, we are about to release information to the world *before* the scientific community has had the opportunity to digest it. That is, before we have had time to argue, to question, to refine and resolve our interpretations; before we've been able to check each other's work or place it in a broader, coherent scientific framework; and crucially, before the public has been given any meaningful introduction to how science is actually conducted.

How does science work?

Much of the public thinks of science as a vehicle that delivers the truth, the whole truth, and nothing but the truth on a road that looks like a straight line. But no scientist worth their salt believes that. Still, too many of us hesitate to correct this misconception when we encounter it. In fact, some may unintentionally reinforce it. I mean, how come scientists are always arguing about what the truth actually is and the next generation of scientists always seems to prove the previous generation wrong?

At its very best, scientists use the tools they have to arrive at a best guess

proposal—a hypothesis—for how something works or what is going on and then use other tools to examine the experimental situation and a “normal” situation—the control—to test the hypothesis. It is not possible to definitively say that results prove that the hypothesis is right, because although the results may be consistent with the hypothesis, there may be other hypotheses that are also consistent with the results. In other words, science cannot unambiguously determine how something works. It can only unambiguously determine how it does not work.

Thus, negative (or null) results—the ones in which the experiment “fails”—are critically important because they eliminate one best guess from consideration and allow scientists to move on to testing an alternative best guess. Through this process, scientists develop models for how things really work that, in the best-case scenario, come increasingly close to the truth, with a capital “T”. The very best scientists challenge each model with other experiments that try to prove the model wrong. If they do not prove it wrong, they have not proved it right! They have just proved that their model is one possibility for how the Truth works. Other scientists then propose other models and test them, too. When more than one model is consistent with experimental results, often, teams of scientists work together to see whether one gives results that are closer to what is observed in nature than the other, or whether the consequences of the two different models converge at the same exact outcome.

Is it any wonder that, for example, how

we thought the virus that causes COVID-19 spread was different in 2020 than in 2023? With a public health emergency and not enough information to even make a best guess, some thought it best to hedge our bets and avoid every kind of contact with possible sources and spreaders of the virus. As, over the ensuing few years, we both learned more about how the virus spreads and developed methods to decrease likelihood of infection and prevent serious illness if we did get infected, public health advisors were able to zero in on what did or did not need to be done to keep us as safe as was possible. All the while, the virus itself was changing by a process we call mutation. In fact, the target continues to mutate as you read this! Some business we are all in, huh?

These uncertainties and challenges and misperceptions of those who are not scientists are what makes it exciting for us to do research, to teach, to mentor, and to write, both for other scientists and for the public. But they are also what makes it critically important that we take the time to listen and respond in appropriate language and with honesty, humility, and respect to what the public needs, thinks, and fears. Effective science in the public interest depends utterly on effective dialogue.

I hope that you and I will do everything we can to enter into this dialogue with neighbors, friends, family—anyone who has a question, a concern, or a need to talk. To neglect this dialogue as part of our civic duty as scientists and people is to endanger both science and those we and it serve. And as the new mandate of open access demands, we must start doing this now. ●

The Reservoir Within: How Nitrate Secures Our Nitric Oxide Supply

The Nitrate–Nitrite–NO Cycle

BY CHRISTOPHER WANJEK, *THE NIH CATALYST*

IN THE ANNALS OF METABOLIC biochemistry, some molecules grab all the glory. Nitric oxide (NO), a reactive gas with outsized influence on blood flow, neurotransmission, and immunity, earned “Molecule of the Year” honors in 1992 from *Science* magazine and was the subject of a 1998 Nobel Prize.

But what of nitrate, NO_3^- , its more stable cousin, long relegated to the role of inert byproduct and sometime dietary contaminant? Turns out that in mammals, nitrate may pool to serve as a key reservoir for NO production, buffering NO availability for cardiovascular functions and preventing oxidative damage through a cyclic metabolic pathway.

This theory is according to a review article published in April in the journal *Nutrients* (PMID: 40362853) by NIDDK scientists **Barbora Piknova**, **Ji Won Park**, and **Alan Schechter** of the NIDDK Molecular Medicine Branch. They propose that the nitrate–nitrite–NO cycle is a self-sustaining system that maintains tissue perfusion and blood flow under varying physiological conditions.

Additionally, they propose that organs such as skeletal muscle and skin are the locations of important nitrate reservoirs, ensuring NO bioavailability during external or internal stress, which broadens the

understanding of how NO homeostasis is regulated beyond the more classically studied enzymatic synthesis. In short, nitrate—once dismissed as a mere metabolic byproduct of NO breakdown—emerges as a vital ally in sustaining life’s most essential functions through its role in this dynamic, reversible cycle.

NO as a positive

The discovery of NO as a vital signaling molecule is itself an interesting irony. NO is a gas regarded historically as a toxic pollutant. Yet through the 1970s and 1980s, scientists began to understand how NO, despite being a short-lived free radical, is crucial for human physiology.

Among those behind these revelations were Robert Furchgott, Louis Ignarro, and Ferid Murad (the latter two trained at the NIH), who discovered that NO can be produced enzymatically by a family of NO synthase enzymes from L-arginine.

They demonstrated that NO activates soluble guanylate cyclase in target cells, leading to increased cyclic guanosine monophosphate concentrations and resulting in vasodilation. This discovery fundamentally changed the understanding of cardiovascular physiology. And for this find they shared the 1998 Nobel Prize in Physiology or Medicine.

Meanwhile, in the 1980s and 1990s, Schechter, who leads the NIDDK Molecular Biology and Genetics Section, was studying normal and sickle hemoglobin at a molecular level, including understanding the effects of hemoglobin interactions with various gases, knowledge that later helped inform how hemoglobin interacts with NO.

In particular, through the decade of the 2000s, Schechter and colleagues demonstrated that hemoglobin and red blood cells not only transport oxygen but also play an active role in regulating NO availability in the blood and tissues, thus modulating vascular tone and contributing to the broader understanding of NO biology.

Getting to NO

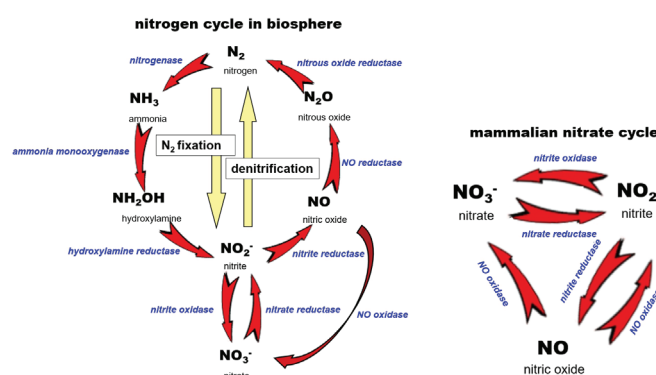
Nitrate is one of the primary sources of NO in the body, mostly obtained through consumption of vegetables such as beets and leafy greens. After ingestion, nitrate is absorbed into the bloodstream and concentrated in certain tissues. In the acidic environment of the stomach, through bacterial reduction in the oral cavity or by certain mammalian enzymes, nitrate is converted into nitrite (NO_2^-), which can then be further reduced to NO in tissues, especially under hypoxic or acidic conditions.

Nitrate seems like a terrible thing to just flush away, yet most of it does indeed wind up in urine and is excreted. How then does the body maintain an adequate supply of nitrate and other NO precursors for when short-lived NO is needed, such as during times of exertion?

Piknova has pondered this question for some time. In reviewing work by Lundberg et al. (PMID: 19915529) and others on the nitrate–nitrite–NO pathway, she began to wonder about the importance of her finding of large nitrate stores in certain tissues.

CONTINUED ON PAGE 5

CREDIT: PIKNOVA ET AL



A view of the overall pathways involving species in the biosphere is shown in panel a, with the cyclic nitrate–nitrite–NO pathway shown in panel b

CALLING ALL CREATORS

CONTINUED FROM PAGE 1

Officially launched in May, the Makerspace has already welcomed NIH scientists who have used the facility to fabricate specialized tools, including a jig to precisely position and stabilize mouse brains embedded in agarose gel, an acrylic positioning device designed to accurately center biological samples in Petri dishes for imaging, and some sort of squarish plastic thingamajig that fits onto another plastic thingamajig. (More on this below.)

“This is a space designed to make items and gadgets that can make your research projects run a whole lot easier,” said **Castle Kim**, manager of the BETA Center Makerspace.

Kim has many years of experience in maker spaces, most notably at Princeton University’s Keller Center, where he served as the maker-space educator and design-lab manager and worked with students, researchers, and entrepreneurs on a range of projects from simple to complex, and whimsical to lifesaving.

If you can dream it, you can probably print, cut, or polish it

The BETA Center Makerspace offers support across four broad areas: Laser cutting, 3D printing (including metal), electropolishing, and electronics. So, do you need a custom animal enclosure or containment shield? Cut it out of acrylic. Need biocompatible models for implant prototyping? Print it in resin. Need a surgical tool deburred before use in preclinical research or simulation? Print it in stainless steel and smooth the surfaces with a dry electropolisher.

Need to etch your boyfriend’s or girlfriend’s name stylistically onto an acrylic keychain for absolutely no research reason? Well, that’s certainly technically feasible, but let’s not push it.

Presented with all this high-tech equipment, **Manu Platt**, an NIBIB



Handiwork. A 3D-printed wrist-actuated prosthetic hand (front); and a 3D print of an individual’s hand with a second print of webbing (back) as a custom brace/cast design to demonstrate a perfect fit that can go, well, hand in glove.

CREDIT: JASPER SHIDE, NINDS

principal investigator and BETA Center director, started low and scored big. He used the Makerspace to print the aforementioned thingamajig, a custom-made set of frames to hold sheets of cellophane that his research group uses to dry polyacrylamide gels. The product he

had relied on for years was discontinued. He was faced with redesigning his experiment until inspiration hit and he realized he could 3D print that jaw.

“The only sheets we could find from other companies were much smaller and too small to fit within the old frames,” Platt said. “Since I had just had my orientation at the Makerspace, the possibility that they could just print us new frames that fit the size of the available cellophane surfaced—and ta-da!” The final, custom-made product is actually better than the original, he said.

Platt added that Makerspace has a natural home in the BETA Center, a collaborative hub that develops and applies engineering and physical science solutions to accelerate biomedical discovery and improve human health.

The Makerspace also works hand-in-glove with NIBIB’s Instrumentation Development and Engineering Application Solutions (IDEAS) lab, which develops novel instruments and methodologies for the intramural community. IDEAS can handle tasks

Searching for
community?
The BETA Center
hosts a very lively
“meet and greet”
each month.

Join the Listserv
email newsletter
BETACENTER-
ANNOUNCEMENTS to
learn more.

too complex or time-consuming for you to take on yourself at the Makerspace.

Not just for engineers

Don't worry if you don't know your PLA from your PETG. Makerspace staff is on hand to guide you through material selection, printer settings, and every step in between. The staff includes **Jake Brandt**, the Makerspace lab technician with a background in manufacturing and mechanical design.

"My job is to demystify the technology," Brandt said.

Fabrication isn't just about printing widgets. It is about solving problems faster, smarter, and cheaper. And it's about enabling innovation at any level in your research project.

For example, let's say you want to design a chamber for inhalation studies. Kim and Brandt can walk you through choosing the right materials, ensuring biocompatibility, verifying gas-tight seals, and minimizing print time. They can even help you design for sterilization



Not Pixar but it is a 'helping hands' soldering station with magnifying lamp.

CREDIT: JASPER SHIDE, NINDS

compatibility, be it by autoclave, chemicals, or gamma radiation.

Their consultation service encompasses

factors such as strength and durability, size, resolution and accuracy, sterilizability, biocompatibility, permeability, printability, and perhaps most important, time. Some large or high-resolution prints could take weeks to finish. The facility staff can walk you through project idea, to training, to fabrication.

The Makerspace is located in Building 13, room 3W31. (The best way to enter is via a door on the backside, near Service Road West; you can thank us later.)

Once trained, users can drop in to work on their projects during operating hours, 9:30 a.m. to 4:30 p.m. Monday through Friday.

The team offers introductory tours every other Friday. Take the tour to open your mind to what is possible. Contact BETACenterMakerspace@mail.nih.gov to get started.

THE RESERVOIR WITHIN

CONTINUED FROM PAGE 3

Drawing on multiple lines of evidence—such as biochemical measurements, tracer studies, and tissue analyses across various species, including humans—Piknova and her team reference multiple experimental studies that use advanced techniques such as chemiluminescence detection and isotope tracing to measure nitrate and nitrite concentrations in several mammalian tissues and fluids. Specifically, they cite measurements showing high baseline nitrate concentrations in certain tissues, such as skeletal muscle, which can increase significantly following dietary nitrate intake, raising the possibility that the stored nitrate in these tissues can be mobilized and shared among organs when needed.

Framing these tissues explicitly as

reservoirs with a central role in NO metabolism and systemic regulation, an example of the classical concept of homeostasis, is a relatively new and significant development in this field, Schechter said. And this expanded view opens new avenues for nutritional and therapeutic strategies aimed at supporting NO availability, especially in aging, cardiovascular disease, and metabolic disorders where NO pathways are impaired, he added.

"This reservoir concept reflects how the body maintains an overall constant internal environment, a concept that dates back to Claude Bernard's idea of the *milieu intérieur*, formulated in the 1840s," Schechter said. "This idea later evolved into the concepts of homeostasis and cybernetic control, which were central to physiology in the first half

of the 20th century but have been less emphasized in the past 60 years with the focus on reductionist studies in specific types of cells and organs."

Curiously, and perhaps not coincidentally, plants rely on a largely reverse cycle of nitrite-to-nitrate conversion, as seen for example in aquaponics, in which the ammonia in fish urine is converted by bacteria first to nitrite and then to nitrate, which is absorbed by the plants... which the fish then eat. In the reciprocal cycling of nitrogen, life's many biological forms become co-regulators of their shared environment, illustrating Gaia theory's premise that Earth's organisms and systems evolve together to sustain conditions favorable for life.

But will this get you to eat more beets? No or NO? ●

Intramural Research Briefs

Read About Scientific Advances and Discoveries by NIH Intramural Scientists

NIEHS, NCI, CC: SHORTER SLEEP LINKED TO DISRUPTIONS IN THE ORAL MICROBIOME



NIEHS, NCI, and CC researchers explored the association between sleep and the oral microbiome.

Your energy levels may not be the only thing suffering from lack of sleep—so may the vast microbial community co-existing in your body. According to a new NIH study, sleeping less than the recommended seven to eight hours a night is associated with reduced oral microbial diversity and distinct shifts in the types of bacteria present in the mouth.

Researchers at NIEHS and NCI compiled data from 1,139 older adults enrolled in the NIH-AARP Diet and Health Study. The team analyzed oral wash samples using 16S rRNA gene sequencing, a technique that can identify and compare bacteria from microbiome samples, to assess bacterial diversity and composition in relation to self-reported sleep duration.

The results suggest individuals who self-reported sleeping six hours or less per night had consistently lower microbial diversity in their mouths than those who slept the recommended seven to eight hours. These shorter sleepers also showed differences in certain families of bacteria. Specifically, they were found to have lower amounts of *Prevotella* and *Corynebacterium*—common commensal organisms in the mouth—and higher amounts of *Streptococcus* and *Rothia*. This finding persisted even after accounting for lifestyle factors such as smoking, neighborhood, and physical activity.

Although previous studies have focused on the gut microbiome, this study is the first of its kind to center on the role of the oral microbiome in sleep in healthy adults. It also uniquely accounted for the potential impact of lifestyle and neighborhood factors on associations with sleep and the oral microbiome.

The results of this study add to a growing body of evidence implicating the microbiome in the mediation of health consequences resulting from poor sleep. (NIH authors: K.R. Dalton, V.C. Chang, M. Lee, K. Maki, V. Purandare, X. Hua, Y. Wan, C.L. Dagnall, K. Jones, B.D. Hicks, A. Hutchinson, L.M. Liao, M.H. Gail, J. Shi, R. Sinha, C.C. Abnet, S.J. London, and E. Vogtmann, PMID: 40444264)

[BY ABIGAIL HOLDER]

CC, NINDS, NCI: HOPE FOR INTRACTABLE CANCER PAIN



Resiniferatoxin is a chemical analog to capsaicin, the active ingredient in peppers like the Carolina Reaper.

For many battling advanced cancer, pain remains an agonizing and persistent accompaniment, often defying even the most comprehensive medical strategies. Standard treatments, reliant on opioids and anti-inflammatory drugs, frequently fall short, leaving a significant gap in effective pain management. But what if a plant-derived compound could offer a new path to relief?

Enter resiniferatoxin (RTX), a novel nonopioid analgesic found naturally in resin spurge, a cactuslike plant native to Morocco.

RTX targets the transient receptor potential vanilloid 1 (TRPV1) ion channel, a key player in transmitting pain signals.

When administered intrathecally, that is, into the spinal fluid, RTX selectively interrupts pain signals by affecting specific neurons, offering pain relief without compromising motor function, touch, or vital central nervous system processes such as respiration.

(Irony warning: Resiniferatoxin is a chemical analog to capsaicin, the active ingredient in chili peppers...but 15,000 times as hot as the infamous Carolina Reaper pepper. You can burn yourself just by whispering its name.)

Knowledge of resiniferatoxin's analgesic properties dates back nearly three decades, and much work has been done in preclinical animal models and veterinary clinical pain studies examining its effectiveness.

Now, in a first-in-human phase 1 trial, a team led by investigators in the NIH Clinical Center has explored the safety and efficacy of intrathecal RTX in 19 patient volunteers with refractory cancer pain—the type that does not respond adequately to standard pain management strategies.

The findings offer a glimmer of hope. On average, these patients, with pain primarily in the abdomen or lower extremities, experienced a 38% reduction in “worst pain” intensity by the 15-day mark post-treatment, dropping from an average score of 8.4 to 5.2 on a 0–10 pain scale. Opioid consumption among participants decreased by a remarkable 57% at day 15, as well.

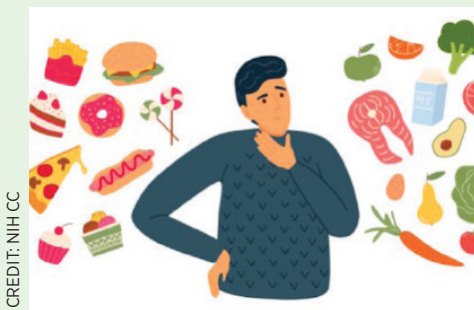
“The treatment was opioid sparing,” said Michael Iadarola, a senior research scientist in the CC Department of Perioperative Medicine and senior author on an interim study report published in *NEJM Evidence*. “The patients all voluntarily reduced their opioid consumption,” said Iadarola. “This increases

alertness and their ability to participate in the activities of daily living and interact socially with family and friends.”

All 19 patients experienced some treatment-emergent adverse events (AEs), with 37 serious AEs reported in 14 patients over the 188-day study period, most being consistent with the course of their advanced cancer. Expected AEs included a temporary loss of heat sensitivity in some areas and urinary retention. Nine deaths occurred during the study, but these were attributed to cancer progression rather than the RTX treatment.

This interim study suggests that intrathecal RTX could be a valuable, single-administration, opioid-sparing option for patients with severe, intractable cancer pain, offering a much-needed alternative in the ongoing fight against suffering. RTX is a versatile interventional treatment, and the Clinical Center has also just begun a clinical trial on a neuropathic pain condition called Morton’s neuroma in which RTX is injected next to the affected peripheral nerve. (NIH authors: A.J. Mannes, J.D. Heiss, A. Berger, C.C. Alewine, J.A. Butman, M.S. Hughes, N. Rabbee, C. Hayes, T.S. Williams, M.R. Sapio, and M.J. Iadarola, PMID: 40423401)

CC: THE HIDDEN PRICE OF ACCULTURATION AND ULTRAPROCESSED FOODS



CREDIT: NIH CC

Visual representation of decision making in food selection, between ultraprocessed foods and minimally processed, whole foods.

Environmental factors play a crucial role in shaping physiological changes and lifestyle, principally on dietary intake among foreign-born adults in the United States. In a recent

study, NIH Clinical Center scientists found that increased consumption of ultraprocessed foods (UPFs) occurred with greater acculturation.

From the National Health and Nutrition Examination Survey (NHANES, 2011–2018), researchers retrieved data from over 3,600 non-US-born adults age 19–70. Using an acculturation index based on primary language spoken and years spent in the United States, results demonstrated that for each unit increase in acculturation, there was a 3% rise in UPF consumption.

The standard American diet (SAD) is nearly half UPFs, which include industrially processed foods that are nutritionally unbalanced and deficient, high in sugar, salt, and unhealthy fats. In line with previous findings, UPFs are related to negative health outcomes such as increased risk of cardiovascular disease, obesity, all-cause mortality, type 2 diabetes, and cancer, as well as negative metabolomic changes.

As the United States grows more diverse, eating habits give way to convenience, and health risks rise from SAD. The authors emphasized the necessity for interventions that preserve and support healthy eating habits. (NIH authors: J.J. Barb, L. Yang, A. Ahmed, P.V. Medina, E.M. Valencia, A.E. Roberts, N. Farmer, and G.R. Wallen, PMID: 40443924)

[BY HÉCTOR CANCEL-ASENCIO, NINDS]

NIA, NINDS: UNTANGLING THE VIRAL LINK TO NEURODEGENERATION

Scientists have long sought to understand the connection between viral infections and brain health. Can common viruses, which can reside unnoticed within our bodies, contribute to the development of neurodegenerative diseases such as Alzheimer’s and other forms of dementia? A study published in *Science Advances* led by researchers at the NIA tapped into data from thousands of human subjects offers compelling new insights into this enigmatic area of research.

The investigation examined the neurocognitive and plasma proteomic profiles

of older adults in a community-based cohort from the Baltimore Longitudinal Study of Aging. Researchers focused on their antibody responses to four common coronaviruses and six herpesviruses with hopes of uncovering the molecular pathways linking the immune response to these viruses with brain aging and dementia risk.

The findings suggest an intriguing protective mechanism: Individuals with a more robust immune response to several common viruses, particularly the human coronavirus OC43, exhibited better neurocognitive outcomes. This level of immune robustness translated to lower odds of dementia, improved cognitive performance, and even a preservation of regional brain volumes.

How does the immune system mediate such a protective effect? The study points to a specific protein, IGDCC4 (immunoglobulin superfamily deleted in colorectal cancer subclass member 4), as a key player. Through advanced genetic analysis, the researchers demonstrated that IGDCC4 is associated with lower dementia risk over a 20-year period and causally linked to maintenance of medial temporal brain volume, crucial for memory.

This study, coordinated by NIA postdoctoral fellow Michael Duggan and NIA investigator Keenan Walker, has a strong international component that leverages data from diverse cohorts worldwide, including those from the United Kingdom, to validate its findings.

This new line of research illuminates a biological basis by which our immune interactions with viruses might influence brain health in later life. Identifying IGDCC4 as an important molecular mediator not only deepens our understanding of neurodegeneration but also opens new avenues for potential therapeutic strategies targeting this protein. (NIH authors: M.R. Duggan, S.M. Drouin, D. Zweibaum, Q. Tian, J. Candia, M. Bilgel, R.F. Gottesman, L. Ferrucci, S.M. Resnick, and K.A. Walker, PMID: 40446030) ●

- “CARDbiomedbench: Biomedical Benchmark of Chatbots, CARD.AI Arena, CARD.AI, FAIRkit” by Faraz Faghri, NIA
- “AI Chatbots: Opportunities and Considerations at NLM” by Dianne Babski, NLM
- “Using AI to Create a Travel Chatbot” by Fiona Vaughans, NCI

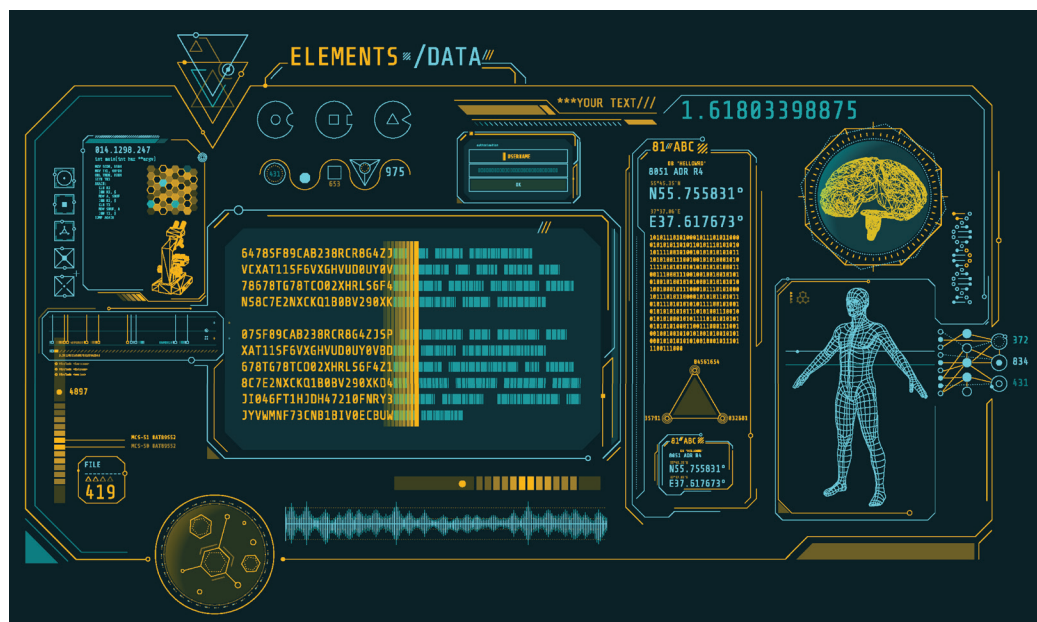
Lillich, an emerging technology specialist at the NIH Library, presented opportunities and ethical considerations in deploying generative AI chatbots within NIH’s information ecosystem. These tools, which use large language models (LLMs) to assist researchers, have the potential to improve literature discovery, scientific synthesis, and internal knowledge management, she noted.

Nelson shared ChIRP updates and reminded attendees that it was designed to respond to NIH intramural queries within a more secure environment than public LLMs offer.

ChIRP is being reimagined as a tool for all of NIH, encompassing both research and administrative tasks. The new and improved tool will be rolled out to the entire NIH community later this summer.

Saddler, a data scientist in the NIEHS Division of Translational Toxicology, highlighted ToxPipe, a chatbot-enabled platform that lets users explore toxicology databases through an intuitive interface powered by LibreChat. Saddler also demonstrated ChemBioTox, which uses autonomous AI agents to answer toxicology questions such as “What are the exposure levels of bisphenols?” Responses are generated through multistep reasoning. The functionality can be evaluated via open-source tools that allow scientists to rate accuracy and refine results.

Faghri from NIA’s Center for



NIH researchers are creating an array of AI-powered tools to address unique intramural biomedical research challenges.

Alzheimer’s and Related Dementias (CARD) presented several AI-driven platforms, including CARDBiomedBench, CARD.AI Arena, and FAIRkit.

These tools, which are described in more detail below, “are using AI to better describe diseases, predict disease progression, and identify new drug targets,” said Faghri, a computer scientist in the advanced analytics expert group at CARD. “AI-powered tools are helping us solve problems that weren’t solvable before.”

Advancing data harmonization

CARD’s advanced analytics expert group is applying AI to one of biomedical research’s greatest challenges—data harmonization. Different research groups collect different types of patient data, including genetic profiles, imaging, and environmental exposures. These datasets are often incompatible by default, which complicates, if not impedes, large-scale analyses.

Standardization of so many data points is impossible to achieve yet incredibly

necessary as biomedical research speeds toward a future of open access data and the application of machine learning.

The DIVER (Data Inventory and Verification Environment for Research) platform, which uses OpenAI’s GPT models to automate the creation of common data elements (CDEs), may help. Faghri’s team applied DIVER to 31 dementia-related datasets and achieved interoperability scores of up to 60% when combining data from the Alzheimer’s Disease Neuroimaging Initiative and the Global Parkinson’s Genetic Program (PMID: 39484274). By automating what is typically a labor-intensive and error-prone process, DIVER enabled them to merge datasets and perform cross-study comparisons, which is a critical step toward identifying early biomarkers and validating therapeutic targets.

Predicting missing data

Missing or incomplete data, especially in electronic health records (EHRs), is another persistent obstacle in biomedical research. Traditional data collection and analysis techniques often fall short in

capturing the complexity of health care information consistently.

The CARD team developed a machine learning framework called MUSE (Multimodal Unsupervised Embedding), which helps to predict missing values in patient data. MUSE uses graph neural networks to analyze the relationships among patient data across multiple data types such as brain scans, cognitive scores, and biomarkers. Rather than addressing each data gap in isolation, MUSE models the entire patient ecosystem to generate more accurate predictions.

The model improved predictions of Alzheimer's disease progression by more than 3% compared with standard approaches. "There's value in retaining data even from patients with large missing segments," Faghri said. "We're trying to figure out the broader structure of the data system and see where people with missing data might fit in. Graph neural networks help us connect the dots."

An AI web crawler

Babski, director of the NLM User Services and Collection Division, presented on NLM's efforts to pilot human-centered AI. Babski demonstrated the NLM Web Accessibility Assistant,

which was created by Dan Wendling to aid in making webpage content more accessible for users. The assistant identifies accessibility issues to help ensure Section 508 compliance and improve user accessibility. The assistant recommends fixes and provides code to make those webpage changes. To date, it has flagged over 67 unique error types across 9,000 website pages.

Babski and Nick Weber, acting director of CIT's Office of Scientific Computing Services, co-chair the NIH Generative AI Community of Practice group. Hundreds of NIHers attend the group's monthly meetings.

Will we see you there? ●

The NIH Office of Science Policy is currently seeking input on responsible development of generative AI tools using controlled-access human genomic data. NIH encourages staff and stakeholders to comment on best practices for mitigating data leakage while promoting innovation. Comments are due by July 16, 2025, and a roundtable discussion will follow on July 17. Visit our online version of this article to access the comment form: <https://irp.nih.gov/catalyst/33/4/from-toxpipe-to-fairkit>

ADDITIONAL RESOURCES SHARED AT THE EVENT

- GitHub: Learn more about NIH GitHub by emailing GitHub@nih.gov.
- CARD tools and benchmarks on GitHub:
- <https://github.com/NIEHS/ToxPipe>
- <https://github.com/NIH-CARD/CARDBiomedBench>
- GitHub Copilot clone: <https://continue.dev>
- CARD.AI Arena: <https://cardai-arena-809832168532.us-central1.run.app/>
- PubTator Central (NLM): extracts information from PubMed abstracts and articles to create annotations of biomedical concepts for use with AI
- Prompt engineering tips and tricks: <https://www.promptingguide.ai>
- Blog post by CARD's advanced analytics expert group: "Can GPT-4.5, Claude 3.7, and Gemini 2.0 Keep Up with Biomedical Research?"

SAVE THE DATE

2025 NIH Research Festival

• Sept. 9–12 •

Join us for this annual celebration of the innovative science of the Intramural Research Program featuring a dynamic program of lectures, poster sessions, workshops, and vendor exhibits.

NIH Bethesda Campus, Building 10



Colleagues: Recently Tenured

Meet your recently tenured colleagues: **Eli Boritz**, NIAID-VRC; **Maria Constanza Camargo**, NCI-DCEG; **Natasha Caplen**, NCI-CCR; **Peter Grayson**, NIAMS; **Katherine McJunkin**, NIDDK; **Richard Scheuermann**, NLM; and **Joshua Tan**, NIAID

ELI BORITZ, M.D. PH.D., NIAID

Senior Investigator, Virus Persistence and Dynamics Section

Education:

Williams College,
(B.S. in biology);
University of
Colorado Health
Sciences Center
(M.D., Ph.D.)



Training:

Internship and residency in Internal Medicine at Johns Hopkins Hospital, Baltimore (2006-2008); joined NIAID as an infectious disease fellow in 2008

Before coming to NIH: Internal medicine resident at Johns Hopkins Hospital

Came to NIH: In 2008 as an infectious disease fellow at NIAID

Outside interests: I became interested in conservation landscaping after receiving a 2019 "RainScapes" grant from Montgomery County, Maryland.

Website: <https://www.niaid.nih.gov/research/eli-boritz-md-phd-virus-persistence-and-dynamics-section>

Research interests: Though antiretroviral therapy in people with HIV can reduce viral load to undetectable concentrations, some CD4 cells still harbor HIV. It's thought that these cells cause viral rebound in patients who interrupt their antiretroviral therapy. The central question in my research involves these latent cells: What are the attributes of CD4 T cells that harbor HIV in the body, especially during antiretrovirals? These infected CD4 cells show no outward signs of infection, making them undetectable to the immune system, and they can live for decades. I set out to understand whether there was a way to distinguish these latent

HIV-infected CD4 cells from other CD4 cells despite their lack of viral expression. I partnered with engineers to build a microfluidic technology to describe these cells, using whole-transcriptome sequencing, to find that these CD4-infected T cells had different expression signatures. Specifically, these CD4 cells expressed higher levels of genes involved in regulating transcription of integrated viruses and translation of viral mRNA (PMID: 36599978).

COMPILED BY AMELIA MARVIT, NIAID

MARIA CONSTANZA CAMARGO, PH.D., NCI

Senior Investigator, Metabolic Epidemiology Branch

Education:

Universidad
Colegio Mayor de
Cundinamarca,
Bogotá,
Colombia (B.Sc.
in bacteriology);

School of Public Health, Cuernavaca,
Mexico (M.S. in epidemiology); University
of Illinois at Chicago (Ph.D. in public health,
epidemiology)

Training: Postdoctoral fellowship, Infections and Immunoepidemiology Branch, NCI-DCEG, NCI; Earl Stadtman Tenure-Track Investigator, Metabolic Epidemiology Branch

Before coming to NIH: Research associate II (epidemiologist), Louisiana State University Health Sciences Center, New Orleans, and research specialist senior (epidemiologist), Vanderbilt University Medical Center, Nashville

Came to NIH: In 2008 as a summer fellow during dissertation work at the NCI-DCEG.

Outside interests: Traveling; trying new restaurants; watching movies



Website: <https://irp.nih.gov/pi/constanza-camargo>

Research interests: I maintain a robust research program with a multidisciplinary team of international collaborators applying cutting-edge molecular epidemiology to the study of *Helicobacter pylori* and of premalignant and malignant gastric lesions. My portfolio combines studies on gastric cancer causation with projects that may have translational application for cancer screening, prevention, and treatment. Gastric cancer is a leading cause of cancer death worldwide. In the United States, this neoplasia affects multiple racial and ethnic groups and individuals of lower socioeconomic status. My descriptive studies on incidence trends have shown that gastric cancer is re-emerging, particularly affecting young non-Hispanic white individuals (PMID: 20442388; 29361173). Along with colleagues, I recently completed the *H. pylori* Genome Project, which established a large-scale biobank of clinical *H. pylori* strains for molecular studies addressing the role of bacterial diversity in the gastric cancer cascade. This publicly available worldwide collection of complete genomes with high-quality metadata will become a major asset for studies of *H. pylori* pathogenesis and disease outcomes (PMID: 38081806).

COMPILED BY TAYLOR FARLEY, NIAID

NATASHA CAPLEN, PH.D., NCI-CCR

Senior Investigator, Functional Genetics Section, Genetics Branch

Education:

University of
Liverpool, England
(B.Sc. in genetics);
King's College
London, Faculty
of Medicine,
University of
London (Ph.D.)



Training: Postdoctoral fellow, St. Mary's Hospital, Imperial College, University of London (1991–1996)

Came to NIH: In 1996 as a visiting fellow, NHGRI

Outside interests: Mystery novels; yoga; traveling

Website: <https://irp.nih.gov/pi/natasha-caplen>

Research interests: My research background includes contributing to the early development of gene-therapy approaches to treat cystic fibrosis (PMID: 7584951). I conducted studies that demonstrated the presence of the RNA interference (RNAi) pathway in mammalian cells (PMID: 11481446), and my team's research has focused on the development of RNAi-based approaches to investigate the mechanistic basis of different cancer types and the response of cancer cells to approved and investigational drugs (e.g., PMID: 21834757). Our current research focuses on investigating the biology of tumors that depend on the expression of fusion oncogenes derived from genes encoding proteins with functions related to the binding of nucleic acids. The study of these tumor types enables investigation of the fundamental mechanisms that regulate gene expression and contributes to addressing a significant unmet clinical need because many fusion-driven cancers remain challenging to treat. One such tumor type is the aggressive bone and soft tissue malignancy Ewing sarcoma, which typically harbors the fusion oncogene EWSR1::FLI1 or EWSR1::ERG. My group is investigating the function of two heterogeneous ribonucleoproteins (hnRNPs) of importance to Ewing sarcoma biology: HNRNPH1 (PMID: 26776507; 31511320; 35639772) and EWSR1 (PMID: 38506112), as well as the mechanisms regulated by the transcriptional activity of EWSR1::FLI1/ERG fusion oncoproteins (PMID: 29873416; PMID: 38588446).

COMPILED BY ABIGAIL HOLDER, NIAAA

PETER GRAYSON, M.D., MSC, NIAMS

Senior Investigator, Vasculitis Translational Research Program

Education:

Brown University, Providence, Rhode Island (A.B. in history); Medical University of South Carolina, Charleston (M.D.); Brown University School of Public Health, Boston (M.Sc. in epidemiology)

Training: Internal Medicine Residency; Chief Medical Resident; and Rheumatology Fellowship, Boston Medical Center; Vasculitis Fellowship, Vasculitis Clinical Research Consortium, Rare Diseases Clinical Research Network

Before coming to NIH: Assistant professor of medicine, Boston University

Came to NIH: 2013 as a Lawrence Shulman Scholar in NIAMS

Outside interests: Musician in two NIH-related bands: Affordable Rock n Roll Act and Goldbug Revival (<https://www.goldbugrevival.com>)

Website: <https://irp.nih.gov/pi/peter-grayson>

Research interests: I am a rheumatologist who conducts clinical and translational research across many forms of systemic vasculitis. I founded the NIAMS Vasculitis Translational Research Program in 2013, and my group has clinically evaluated >1000 patients with different forms of vasculitis at the NIH Clinical Center. Work from my group has focused on biomarker discovery and development, advanced molecular imaging, molecular classification of disease, clinical trials, and genetics and genomics of vasculitis. My group co-discovered the VEXAS syndrome (PMID: 33108101). Translational work also has defined novel pathways of neutrophil-mediated inflammation in monogenic vasculitis and drug-induced vasculitis and has identified novel biomarkers of disease activity in



many forms of vasculitis that suggest novel therapeutic targets.

KATHERINE MCJUNKIN, PH.D., NIDDK

Senior Investigator, Section on Regulatory RNAs, Laboratory of Cellular and Developmental Biology

Education:

Princeton University (B.A. in biology); Watson School at Cold Spring Harbor Laboratory, New York (Ph.D. in biological sciences)

Training: Postdoctoral fellow, University of Massachusetts Medical School, Worcester (2011–2017, Victor Ambros Laboratory)

Came to NIH: In 2017 as a Stadtman tenure-track investigator

Outside interests: Baking sourdough; running

Website: <https://irp.nih.gov/pi/katherine-mcjunkin>

Research interests: I am interested in microRNAs, which are small noncoding RNAs that antagonize expression of mRNAs. So far, most of our work has used the model organism *Caenorhabditis elegans* because of its powerful genetic tools, especially the ability to use CRISPR technology to make genomic changes. We have devised new CRISPR-based methods to empirically determine biologically important microRNA-target mRNA pairs (PMID: 32820039). More recently, we have uncovered new aspects of how microRNAs are generated (PMID: 36711716; 40501874). We are intrigued by how microRNAs are targeted for decay, and we have investigated the role of microRNA decay in early development (PMID: 34586415; 35947946).

COMPILED BY JOHN CARLO COMBISTA, NIMH

RICHARD SCHEUERMANN, PH.D., NLM

Scientific Director

Education: MIT, Cambridge, Massachusetts (B.S. in life sciences); University of California, Berkeley



Sweetening the Pot While Empowering Others

Kelly Ten Hagen's Legacy of Scientific Discovery, Mentorship, and Advocacy Is on Tap at the Annual Anita B. Roberts Lecture

BY DAPHNE KNUDSEN-PALMER, NCI



Kelly Ten Hagen

FROM DECODING MOLECULAR SUGAR codes to shaping the next generation of scientists, **Kelly Ten Hagen**, associate scientific director at NIDCR, exemplifies the power of combining rigorous science with mentor-focused leadership. And given her longstanding leadership in the Women Scientists Advisors (WSA), it is no surprise that Ten Hagen has been received the honor of being the speaker this fall in the prestigious annual Anita B. Roberts Lecture Series—a named lecture that celebrates NIH women scientists.

A trailblazer in glycobiology

Ten Hagen is a senior investigator and chief of the Developmental Glycobiology Section. Her research has made fundamental discoveries towards understanding the roles of O-glycosylation. A post-translational modification involving the attachment of a sugar to the oxygen atom in the amino acid serine or threonine, the highly conserved process of O-glycosylation affects a wide variety of cell biological processes and diseases.

After completing her doctoral studies in DNA replication at Stanford University (Stanford, California), Ten Hagen jumped

straight into an assistant professorship at the University of Rochester (Rochester, New York). She began critical research on the biochemistry of one common type of protein glycosylation, the addition of N-acetylgalactosamine (GalNAc), to understand the relationship between these modified proteins and their transferases, GalNAc-Ts, for short. Ten Hagen was among the first in demonstrating that some of these transferases required glycopeptides as substrates, thus leading to the understanding that many GalNAc-Ts must be involved in densely modified targets.

Once at NIH, Ten Hagen expanded her research to ask what roles O-glycosylation might play in development. Key studies in the fruit fly (*Drosophila*) revealed that O-glycosylation is essential for viability, playing critical roles in secretion, cell adhesion, formation of the extracellular matrix, and in modulating furin cleavage.

After highlighting the importance of O-glycosylation in mammalian salivary gland development, oral microbiome stability, and chronic kidney disease, among others, Ten Hagen investigated the effect of O-glycosylation on SARS-CoV-2 transmissibility, in particular investigating how O-glycosylation may affect furin cleavage of the spike protein. The breadth of these discoveries shows Ten Hagen's tenacity and dedication to her field.

A force for mentorship

The legacy of Ten Hagen's accomplishments reaches far beyond these key scientific findings. Along with having received many professional rewards and accolades, including the 2023 Rosalind Kornfeld Award for Lifetime Achievement in

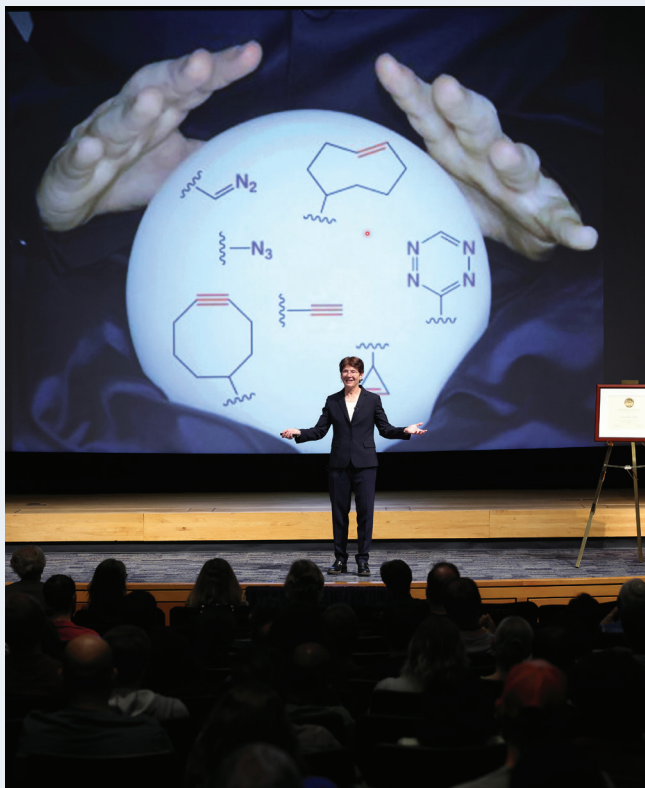
Glycobiology, her mentees praise her dedication to both scientific and professional growth through mentorship.

"She always nudged us to be the best versions of ourselves, through whatever self-doubt or experimental hiccup we were experiencing in the moment," said **Duy Tran**, scientific information officer at NEI. Tran is among many successful trainees that have praised the dedicated mentorship of Ten Hagen. These endorsements are plentiful.

Ten Hagen "exemplifies integrity, generosity, and intellectual curiosity," said Alisa Lee, who now works in Boston Children's Hospital Department of Dentistry (Boston) and is an instructor of developmental biology at Harvard University (Cambridge, Massachusetts). "What stood out most to me was her strength in leading with both clarity and compassion. Kelly is not only a brilliant collaborator and researcher, but also someone who empowers those around her to reach their full potential."

Similar sentiments were echoed by former postdoctoral researcher **Zulfeqhar Syed**, who is now director of the NHLBI Electron Microscopy Core. "She regularly took the time to offer thoughtful feedback and cared deeply about her lab members, not just as scientists but as individuals," said Syed. "She took pride in seeing us grow and move forward." ●

The 2025 Anita B. Roberts Lecture Series will feature two lectures this year. Ten Hagen's lecture will take place Sept. 10 at 11:30 a.m. in Lipsett Amphitheater as part of the annual NIH Research Festival. A second lecture featuring Cindy Dunbar, an NHLBI distinguished investigator, will follow in November.



Carolyn Bertozzi was a co-recipient of the 2022 Nobel Prize in Chemistry. Watch her delightful June 11 lecture on NIH VideoCast.



Kelly Ten Hagen, Carolyn Bertozzi, Jayanta Bhattacharya, and Amy Newman pose for a group photo before Bertozzi's lecture to a packed Masur Auditorium. Newman and Ten Hagen invited Bertozzi for the lecture.

The annual Margaret Pittman Lecture, part of the Wednesday Afternoon Lecture Series (WALS), featured a talk by Carolyn Bertozzi titled "Bio-orthogonal Chemistry: The Journey from Basic Science to Clinical Translation."

The annual Pittman lecture honors the legacy of Margaret Pittman, the first female laboratory chief at NIH.

PHOTOS BY CHIA-CHI CHARLIE CHANG

RECENTLY TENURED

CONTINUED FROM PAGE 11

Education cont.: (Ph.D. in molecular biology)

Before coming to NIH: La Jolla, California, campus director, J. Craig Venter Institute; adjunct professor, pathology, University of California at San Diego

Came to NIH: In September 2023

Outside interests: Travel (36 different countries so far); reading; planning outings with my wife; pickleball; cooking

Website: https://www.nlm.nih.gov/research/researchstaff/Scheuermann_Richard.html

Research interests: My research interests combine bioinformatics and computational biology. More recently, I have focused on the development of novel AI approaches for interpreting single-cell genomics data of the human immune and nervous systems. I developed biomedical ontologies and novel computational methods for gene expression, protein network, flow cytometry, and comparative genomics data analysis.

These informatics tools have been made available through public databases and analysis resources, including the Immunology Database and Analysis Portal, the Influenza Research Database, and the Virus Pathogen Resource.

COMPILED BY FELICITY FOX, NLM

JOSHUA TAN, PH.D. NIAID
Chief, Antibody Biology Unit
Education:

Monash University, Melbourne, Australia (B.S.); University of Oxford (Ph.D. in infection, immunology, translational medicine)

Training: Sir Henry Wellcome Postdoctoral Fellowship, University of Oxford

Came to NIH: In 2018 as a guest researcher, became an investigator in 2020 in the Laboratory of Immunogenetics, NIAID



Outside interests: Reading; traveling; guitar
Website: <https://www.niaid.nih.gov/research/joshua-tan-phd>

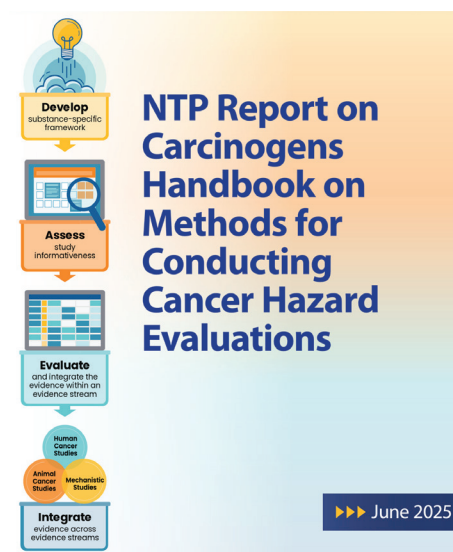
Research interests: I am an immunologist who studies the human antibody response to global infectious pathogens to understand how the human immune system responds to infection and to develop new monoclonal antibody therapeutics and vaccines. Recently, we identified protective antibodies that target the sporozoite stage (PMID: 39745947) and the blood stage (PMID: 39059381) of the deadly malaria parasite *Plasmodium falciparum*. We found potent antibodies that target a cryptic region of the dominant sporozoite coat protein that is not included in the current malaria vaccines, which reveals a new target site for prophylactic antibody discovery and immunogen design. Besides malaria, we also study human antibody responses to *Mycobacterium tuberculosis* and infectious viruses. ●

COMPILED BY TAYLOR FARLEY, NIAID

How Do We Come to Know What Causes Cancer?

Handbook Explains Rigorous and Transparent Processes Used to Assess Substances for Inclusion in the *Report on Carcinogens*

BY DOUGLAS MURPHY, NIEHS



The handbook, which captures the nuance and science of the systematic review process, was developed by the following team of experts: Ruth Lunn, Whitney Arroyave, Stanley Atwood, William Bisson, Andrew Ewens, Sanford Garner, Neela Guha, M. Elizabeth Hodgson, Gloria Jahnke, M. Elizabeth Marder, Alton Peters, Pamela Schwingl, Amy Wang, and Suril Mehta.

THE NATIONAL TOXICOLOGY Program (NTP) released a handbook detailing the steps and processes it follows to determine which chemicals or exposures may pose a cancer risk to humans.

The *Handbook on Methods for Conducting Cancer Hazard Evaluations* explains how NTP systematically reviews scientific research to identify substances for inclusion in the *Report on Carcinogens* (RoC). The RoC listing of substances known, or reasonably anticipated, to cause cancer guides policies and regulations across the nation and around the world.

“The first step to cancer prevention is knowing what causes cancer,” said **Ruth Lunn**, who leads the *Report on Carcinogens* Group in the Division of Translational Toxicology (DTT) at NIEHS. “This handbook details the rigorous and

scientifically supported methods we use to determine whether a substance is a cancer hazard and ensure consistency in our evaluations.”

Transparency and confidence

The handbook serves multiple audiences. Scientific researchers use it as a reference when evaluating the rigor of cancer hazard assessments provided in RoC monographs—the reports used to recommend substances for inclusion in the RoC. Industries and chemical producers turn to the handbook to understand what processes were used to designate their products as potential carcinogens. Other experts in the scientific community rely on the tools and methods set forth in the handbook to conduct their own cancer hazards assessments.

The largest beneficiaries of the handbook are members of the public who seek transparency and confidence in the process to determine whether substances or environmental exposures may cause cancer.

“People don’t want us just to say, ‘this causes cancer.’ They want to see that our assessments meet the most rigorous standards,” said **Suril Mehta**, an epidemiologist in the RoC Group who conducts cancer hazard evaluations. “The handbook guides that rigor and also communicates it broadly.”

Incorporating scientific advances

The new handbook communicates advances in the systematic review process. Notably, there is an extensive discussion of innovative methods used to appraise the evidence from mechanistic studies—those that examine how a substance causes changes at the molecular and cellular levels that can lead to cancer. The handbook also discusses

new features of the RoC monographs that present the conclusions of assessments. These enhancements include the following.

- Improved methods to incorporate evidence from different types of scientific research on cancer, not only from mechanistic studies but also from epidemiology inquiries and animal research
- Incorporation of systematic evidence maps—visual tools that allow users to scope and explore information on the studies evaluating the substance
- Enhanced tools and processes for assessing the informational value of individual studies and for judging whether errors in design, conduct, or analysis of a study create a bias that casts doubt on the conclusions
- Revised reporting methods for each evaluation that better describe how scientists concluded the likelihood a substance could cause cancer

Up-to-date and evolving

Publication of this new information does not signal a sudden shift in the way researchers conduct assessments. Instead, it clearly documents how the methods for drawing conclusions have evolved and improved since the handbook was last published in 2015.

“It builds on concepts that we have learned over the years and incorporated into our assessments,” Mehta said. “It adds structures and processes that we adopted since writing the previous handbook and fills in information that was missing.”

Release of the latest handbook also does not preclude further development and refinement of assessment strategies. “We are always trying to incorporate the best practices and most rigorous methods,” Lunn said. ●

The SIG Beat

News From and About the Scientific Interest Groups

NEW SIG: Rare Endocrine Tumors Scientific Interest Group

The NIH-Rare Endocrine Tumors Scientific Interest Group (NIH-RET SIG) stands as a pioneering initiative within the NIH dedicated to the field of endocrine oncology. The group emphasizes understanding rare endocrine and neuroendocrine tumors, which pose significant clinical and research challenges due to their complexity and often under-researched malignancies.

The NIH-RET SIG aims to foster the sharing of knowledge, resources, and expertise by promoting cross-institutional and cross-disciplinary partnerships—both within the NIH (intramural) and with external academic and clinical institutions.

This SIG facilitates interdisciplinary collaboration among basic scientists and clinical researchers by cultivating a supportive community in which researchers can exchange ideas, discuss challenges, and collaborate on solutions to longstanding questions in the field, and by promoting innovative research efforts that deepen our understanding of the biological, genetic, and pathophysiological

mechanisms underlying rare endocrine tumors. These efforts are critical for advancing scientific knowledge as well as developing improved diagnostic tools and therapeutic strategies.

Webpage: <https://oir.nih.gov/sigs/rare-endocrine-tumors-scientific-interest-group>

Contact: Myriem Boufraquech (myriem.boufraquech@nih.gov) or Jaydira Del Rivero (jaydira.delrivero@nih.gov)

NEW SIG: Sarcoma Interest Group

The Sarcoma Interest Group (SAIG) fosters collaboration among NIH and extramural researchers to advance sarcoma research. To facilitate knowledge exchange, SAIG will host monthly online seminars and workshops, providing a dedicated platform for discussion and innovation. Given that sarcomas are systemic diseases arising from various tissue and organ types, SAIG will engage researchers across multiple NIH institutes. Initially, all meetings will be virtual.

Through its seminar series, SAIG aims to keep NIH researchers informed about the latest sarcoma research; strengthen intramural-extramural collaborations; support the development of novel therapeutic strategies; and attract highly qualified trainees to NIH laboratories.

Annually, SAIG will feature a presentation from the Fellows Award for Research Excellence (FARE) recipient, showcasing their research contributions. Looking ahead, SAIG envisions hosting a biannual in-person workshop at NIH, featuring keynote speakers from outside institutions. These initiatives will enhance mentorship, foster career development,

further strengthen the sarcoma research community, and contribute to NIH's mission of advancing human health.

Webpage: <https://oir.nih.gov/sigs/sarcoma-interest-group>

Contact: Jing Huang (huangj3@mail.nih.gov)

NEW SIG: Vet Med Interest Group

The NIH Vet Med Interest Group strives to connect anyone curious about veterinary medicine and animal care. The Vet Med Interest Group organizes volunteer opportunities in the area, fosters connections between fellows and faculty, and provides networking opportunities for NIH members across all institutes.

Additionally, the interest group will organize guest speakers from both within NIH and the broader veterinary community to share their career paths, schooling, research, and advice with trainees who may be interested in pursuing veterinary medicine.

This group strengthens the intramural research program by hosting discussions across institutes, hosting career development talks, and supporting translational and comparative research efforts. The SIG meets monthly to provide information and support to members. Participation in this group does not require active involvement in veterinary medicine, and all NIH members, regardless of career goals, are encouraged to attend events and talks.

Webpage: <https://oir.nih.gov/sigs/vet-med-interest-group>

Contact: Kate Silver (kate.silver@nih.gov) or Hana Petersen (hana.petersen@nih.gov)




Check out the full list of scientific interest groups at <https://oir.nih.gov/sigs>.

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We also welcome “letters to the editor” and other commentary for publication consideration, as well as your reactions to any content on the *Catalyst* pages.

**READ LONGER ARTICLES AND
VIEW MULTIMEDIA ONLINE AT**
<https://irp.nih.gov/catalyst/33/4>

PHOTOGRAPHIC MOMENT



CREDIT: NIH CLINICAL CENTER

THE 2025 NIH GRADUATE MEDICAL EDUCATION GRADUATES: ON JUNE 6, THE Clinical Center hosted the second annual graduation ceremony celebrating the latest crop of clinicians. The ceremony was held in Masur Auditorium with a packed room full of graduates, mentors, family members, and colleagues. Visit the *NIH Catalyst* online for a full list of graduates and the programs in which they studied. ●

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Address correspondence to:
Building 1, Room 160, NIH
1 Center Drive
Bethesda, MD 20892

phone: 301-201-1106
email: catalyst@nih.gov

Read the *NIH Catalyst* online:
<https://irp.nih.gov/catalyst>

PUBLISHER
NINA F. SCHOR
Deputy Director for Intramural Research, OD

EDITORS
JANICE LEE
Clinical Director, NIDCR
Deputy Director for Intramural Clinical Research
RICHARD S. CHADWICK
Scientist Emeritus, NIDCD
CHRISTOPHER WANJEK
Director of Communications, OIR

MANAGING EDITOR
JENNIFER L. HARKER

COPY EDITOR
SHAUNA ROBERTS

CONTRIBUTING WRITERS
JOHN CARLO COMBISTA, TAYLOR
FARLEY, FELICITY FOX, ABIGAIL
HOLDER, AMELIA MARVIT, DOUGLAS
MURPHY, DAPHNE KNUDSEN-PALMER

RESEARCH BRIEFS
HÉCTOR CANCEL-ASENCIO,
ABIGAIL HOLDER

PHOTOGRAPHERS/ILLUSTRATORS
CHIA-CHI CHARLIE CHANG,
JASPER SHIDE

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