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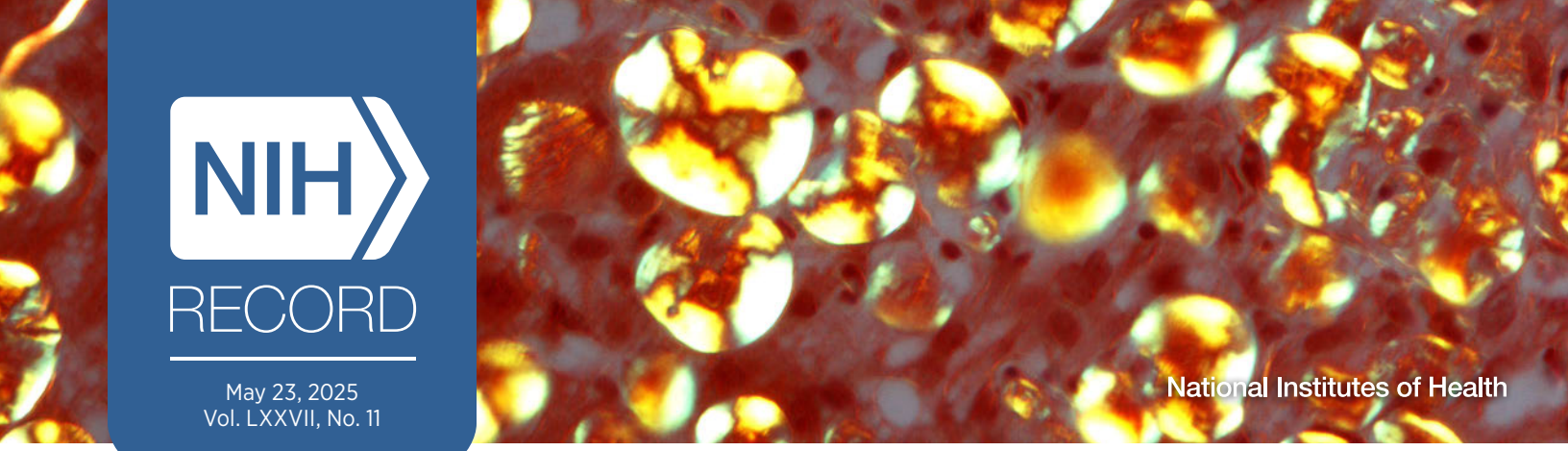

HEALTHY AGING

NIA Researchers Introduce the Healthspan

BY AMBER SNYDER

Can we live to 120? And do we want to?

Researchers from NIH's National Institute on Aging (NIA) pondered this topic in a recent Demystifying Medicine lecture. Dr. Luigi Ferrucci and Dr. Payel Sen approached these questions from two different angles: epidemiology for Ferrucci, and experimental biology for Sen.

Ferrucci, the scientific director at NIA, is a geriatrician and epidemiologist who studies the causal pathways leading to progressive physical and cognitive decline in older people. He contends that, rather than extending the lifespan, we should aim to increase the "healthspan," or the number of years lived in good health.

Dr. Luigi Ferrucci

"As the global population ages, we need to change our perspective on what we think about health and human care," Ferrucci said. In his days as a practicing gerontologist, he recognized that reducing time spent in disability and frailty was often more important to his patients than increasing overall life expectancy.

Aging is the strongest risk factor for all major age-related chronic diseases—like osteoarthritis, chronic heart disease and cancer—Ferrucci said, but he believes we are missing the window of opportunity for intervention.

Health care practitioners tend to intervene at the end of a person's disease-free life expectancy, when chronic diseases reach the clinical threshold and begin showing symptoms. This approach is expensive, he explained: the U.S. spends 97.5% of its

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NIH's longtime chief of staff retires. See p. 8.

ALSO THIS ISSUE

CATCHING UP

Chung Decodes Pediatric Type 2 Diabetes

BY AMBER SNYDER

Dr. Stephanie Chung

Why do some people get sick and others don't?

This question has fueled NIH researcher Dr. Stephanie Chung's interest in medicine since childhood. Her father was a cardiologist in Kingston, Jamaica where Chung grew up, and she and her sister would accompany him on his weekend hospital rounds.

"I did run away from medicine at first," she admitted, recalling being put off by the hardships of her father's career. But, when

SEE **CHUNG**, PAGE 5

NIH Launches Universal Vaccine Platform

BILLION PHOTOS/SHUTTERSTOCK

NIH is developing the next-generation, universal vaccine platform, Generation Gold Standard, using a beta-propiolactone (BPL)-inactivated, whole-virus platform.

This initiative represents a shift toward transparency, effectiveness and comprehensive preparedness, funding the NIH's in-house development of universal influenza and coronavirus vaccines, including candidates BPL-1357 and BPL-24910. These vaccines aim to provide broad-spectrum protection against multiple strains of pandemic-prone viruses such as

SEE **VACCINE**, PAGE 2

Vaccine

CONTINUED FROM PAGE 1

H5N1 avian influenza and coronaviruses.

The program realigns the operations of Biomedical Advanced Research and Development Authority (BARDA)—an agency within HHS—with its statutory mission to prepare for all influenza viral threats, not just those currently circulating.

"Generation Gold Standard is a paradigm shift," said NIH Director Dr. Jay Bhattacharya. "It extends vaccine protection beyond strain-specific limits and prepares for flu viral threats—not just today's, but tomorrow's as well—using traditional vaccine technology brought into the 21st century."

Generation Gold Standard, developed by NIH's National Institute of Allergy and Infectious Diseases (NIAID):

Recalibrates pandemic preparedness

Unlike traditional vaccines that target specific strains, BPL-inactivated whole-virus vaccines preserve the virus's structural integrity while eliminating infectivity. This approach induces robust B and T cell immune responses and offers long-lasting protection across diverse viral families. Moreover, the intranasal formulation of BPL-1357 is currently in Phase Ib and II/III trials and is designed to block virus transmission—an innovation absent from current flu and COVID-19 vaccines.

Embodies efficient, transparent research

The BPL platform is fully government-owned and NIH-developed. This approach ensures transparency, public accountability and freedom from commercial conflicts of interest.

Marks the future of vaccine development

In addition to influenza and coronavirus, the BPL platform is adaptable for future use against respiratory syncytial virus (RSV), metapneumovirus and parainfluenza. It also offers the unprecedented capability to protect against avian influenza without inducing antigenic drift—a major step forward in proactive pandemic prevention.

Clinical trials for universal influenza vaccines are scheduled to begin in 2026, with FDA approval targeted for 2029. The intranasal flu vaccine, now in advanced trials, is also on track for FDA review by 2029. R

Bhattacharya Visits Medical College of Wisconsin

(Above l), NIH Director Dr. Jay Bhattacharya (r) joins Medical College of Wisconsin (MCW) President and CEO Dr. John Raymond, Sr. for the MCW President's Speaker Series on May 2. At r, Bhattacharya shares NIH's vision at the quarterly series, which aims to advance diverse viewpoints of medical education and understanding of freedom of expression to engage in constructive exchanges of ideas and perspectives. **PHOTOS: MCW**

Early-Career Investigators to Speak on Disease Prevention

June 4-6

NIH's Office of Disease Prevention (ODP) will host a week of Early-Stage Investigator Lectures (ESIL) featuring the 2025 awardees, Drs. Nilay S. Shah, Julia Chen-Sankey and Michael Fang. Each will focus on a different prevention topic: cardiovascular disease, e-cigarette use and type 1 diabetes. Each lecture will take place via webinar, with Shah and Chen-Sankey presenting at 10:00 a.m. ET on June 4 and 5, respectively, and Fang presenting on June 6 at 2:00 p.m. ET.

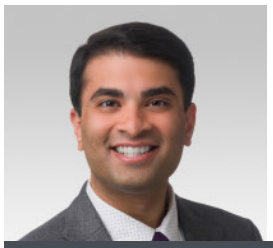
This lecture series recognizes early-career prevention scientists who are poised to become future leaders in prevention research.

On June 4, Shah—an assistant professor in the departments of cardiology, preventive medicine and medical social sciences at the Northwestern University Feinberg School of Medicine—will outline the science of cardiovascular disease prevention among Asian American populations.

Shah, who also is a general and preventive cardiologist in the Bluhm Cardiovascular Institute, will discuss the epidemiology of cardiovascular health across disaggregated Asian communities, characterizing upstream social and structural factors that influence cardiovascular disease risk in these groups. He'll also share evidence for adapted clinical and community-based prevention interventions that build on the unique characteristics of Asian populations.

On June 5, Chen-Sankey—an assistant professor in the department of health behavior, society and policy at the Rutgers School of Public Health—will share her research findings on young adults' responses to various e-cigarette marketing features, alongside their perceptions and intentions related to e-cigarette use shaped by these promotional elements. She also will discuss the broader implications of these findings, highlighting opportunities for developing impactful policies and public health interventions aimed at reducing ecigarette-related harms among young people.

Fang, presenting on June 6, will draw on emerging evidence from national health surveys and large electronic health record databases to describe the modern epidemiology of type 1 diabetes in the U.S. Fang— an assistant professor in epidemiology at Johns Hopkins Bloomberg School of Public Health and core faculty at the Welch Center for Prevention, Epidemiology and Clinical Research—will highlight areas of progress and emerging challenges in managing type 1 diabetes, with a focus on glycemic control, obesity and access to new diabetes technologies.



From l, Drs. Nilay Shah, Michael Fang, Julia Chen-Sankey

To register for each lecture, visit https://go.nih.gov/WYLfSir. The lectures will be recorded and available on ODP's website a few weeks after each session.

Investigator Studies How the Environment Affects Asthma

BY ELIZABETH WITHERSPOON

Asthma affects about 25 million people in the United States, including 4.7 million children and adolescents. Environmental exposures can worsen the coughing, wheezing, chest tightness and shortness of breath symptoms linked with asthma.

NIH research highlights the importance of taking each patient's unique environmental exposures into account when treating asthma. The lung disease occurs when the body's natural defenses against germs and sickness react strongly to environmental exposures like pollen or pollutants.

Dr. Stavros Garantziotis studies how the lung responds to environmental injury.

PHOTO: STEVE MCCAW / NIEHS

Dr. Stavros Garantziotis, a lung doctor and head of the Matrix Biology Research Group at NIH's National Institute of Environmental Health Sciences (NIEHS), recently discussed specific exposures driving asthma and how people's immune systems react differently.

"Personalized environmental medicine—which means evaluating each person's specific exposures and susceptibility factors and tailoring treatment specifically to their needs—I believe can help prevent or even reverse some lung diseases," said Garantziotis.

Where people live, work and play can affect asthma symptoms and severity. "Looking only for asthma symptoms and ignoring the exposures that may have caused these symptoms provides an incomplete picture," he noted. "It is like photoshopping the patient out of a photo; you lose a huge amount of information."

NIEHS research has started to move the needle so that doctors are more aware of the role of environmental exposures in asthma.

"Personally, I have been able to improve symptoms and even reduce the amount of medication my asthma patients need by thoroughly evaluating their exposure profiles and taking steps to address them. These are low-cost, low-tech, but high-impact solutions that can help patients tremendously."

Garantziotis has been studying a protein called TLR5, which he believes plays a central role in lung disease because it helps regulate immune responses to environmental injury.

For example, his team found that house dust in many homes of asthma patients activates TLR5 leading to an overactive immune response. This is probably caused by house dust bacteria shedding proteins that activate TLR5. Often, these bacteria are found in the excrement of house pests like cockroaches or mice. Cleaning up these sources of TLR5 activation is a simple and effective non-medication way to prevent asthma flare-ups caused by this pathway.

Garantziotis also shared advice he gives to empower his patients.

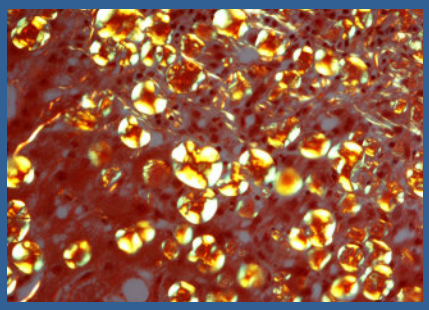
"I always talk to my patients about their home and work environments," he said. "I ask them to become detectives in their own asthma cases. Often, they start recognizing patterns of exposure and symptom associations. [Some] might feel sicker at home compared to when they are away or have more symptoms in the morning or at night. Such information helps them better manage their symptoms."

It's critical that patients learn how to manage their symptoms and take control of their asthma, he said. "After all, they visit my clinic only once every six months, but they are experiencing asthma 24/7." —adapted from NIEHS's *Environmental Factor*

For more information about NIH's asthma research, explore these resources:

https://www.niehs.nih.gov/health/topics/conditions/asthma

https://www.nhlbi.nih.gov/LMBBasthma



ON THE COVER: *Globules showing an apple green birefringence (Sirius red stain under polarized light)*

IMAGE: HALA MAKHLOUF / NCI

The NIH Record

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NIH National Institutes of Health
Turning Discovery Into Health

Dr. Payel Sen

Aging

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health care funds on people with some sort of disease, but spending money on patients already experiencing chronic disease(s) does little to expand their healthspan.

But what if we could detect and treat disease accumulation earlier in life? One way to conceptualize aging, Ferrucci said, is the "accumulation of macromolecule and cellular damage that eventually becomes pathology." By this, he means the buildup and deposition of abnormal proteins or other substances within cells, tissues or organs, which can lead to various diseases. If we could counter or slow this accumulation, then we could potentially reduce susceptibility to chronic diseases in older people.

The aging process tends to damage randomly different cellular structures. For example, DNA damage that remains unrepaired leads to mutations or transcription errors, and the burden of damage accumulated reaches a critical threshold that may lead to adverse cellular responses such as neoplastic transformation, cellular senescence or apoptosis. The accumulation of DNA damage also affects its functionality, causing degenerative diseases.

Because of the resiliency of our bodies, Ferrucci said, we can afford some biological damage without showing any outward (or "phenotypic") signs. Phenotypic aging is the third stage, and manifests as noticeable changes such as low muscle strength or changes in visual or hearing acuity. In the final stage, the aging process has progressed to functional challenges, during which individuals may experience problems with memory or mobility, among other impairments.

Not everyone ages at the same rate, and inherited and external factors also influence the aging process. "Investments in early life, particularly in utero, may help postpone age-related morbidity and mortality and extend the healthy lifespan," Ferrucci noted.

Sen's portion of the lecture focused on the effects of epigenetics on aging. Epigenetics is the study of how environmental factors can alter gene expression without changing the underlying DNA sequence. To highlight the difference, she referenced a study comparing the lifespans of twins.

"Genetics can only explain 25% of the lifespan variation in twins," she said. "The rest is epigenetics."

One way to learn more about aging is by studying organisms and species that are different from us. In comparative biology, which involves studying different organisms to understand the patterns of life at all levels, researchers have found that maximum lifespan is positively correlated to body mass. Larger species such as whales and elephants have the longest lifespans, while rodents trend in the opposite direction for both size and lifespan.

Humans are one exception, Sen noted, being one of the longest-lived mammals despite being much smaller than other mammals with similar lifespans. "That may be a sign that we've already pushed the limits of our longevity," she said.

Another famous exception is the naked mole rat, which can live up to 30 years. By studying them, researchers hope to gain insights into how these rodents acquired their longevity.

Aging and longevity studies on numerous categories of model organisms have revealed "repetitive molecular pathways" that emerge during aging. These mechanisms when they become dysfunctional are known as hallmarks of aging. Researchers have identified 12 hallmarks, which fall into three distinct categories: primary, antagonistic and integrative.

Primary hallmarks are the earliest, most causal changes—such as epigenetic alterations, Sen's area of research. Antagonistic hallmarks are cellular responses to primary changes, and integrative hallmarks are responses seen on a systemic level.

Cellular senescence—an antagonistic hallmark—creates "zombie cells," which release pro-inflammatory molecules and are also harder to destroy. Researchers created a molecule that initiated destruction of senescent cells in a mouse model, which improved both healthspan and lifespan for the animals.

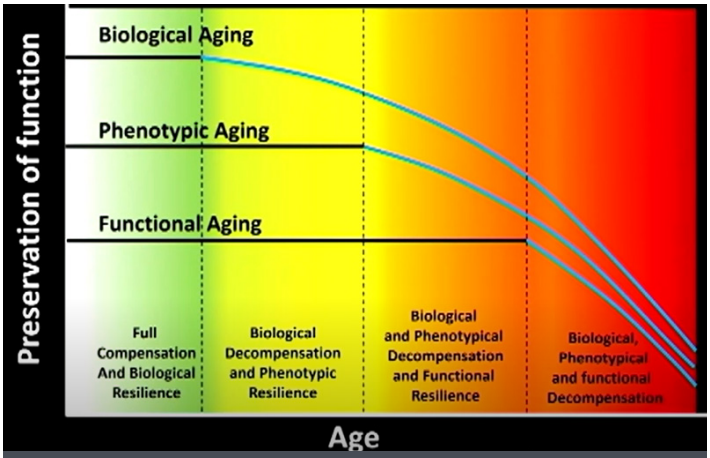
Chronic inflammation occurs at multiple levels throughout the human body. As an integrative hallmark, chronic inflammation can increase with age and even drive aging in other organs.

Are age-related molecular changes reversible? Researchers have identified a few promising interventions in animal models, but these are preliminary and will also be harder to translate into use for humans.

In the meantime, Ferrucci and Sen agree that healthy lifestyle choices such as eating well, prioritizing exercise and sleep, maintaining social ties and continuing to learn new things are the best ways to set yourself up for healthy aging.

"A healthy lifestyle is the key in the lock of creating successful, long-lived societies," Ferrucci said.

To view an archived version of the lecture, please visit https://go.nih.gov/0mJjkVz.



Window of opportunity: Ferrucci points out the compensatory period (the middle two columns) in the aging process where potential interventions might be effective.

Chung

CONTINUED FROM PAGE 1

she volunteered in the pediatric unit of her father's hospital after completing undergraduate studies, she felt drawn to the children there.

"I was struck by youth with obesity and diabetes and the relatively few treatment options available to them—at an early age and as they transition into adulthood," she explained.

Chung attended medical school at the University of the West Indies and pursued a dual residency in internal medicine and pediatrics. She came to NIH as an assistant clinical investigator in 2013 and progressed to acting chief in the section on pediatric diabetes, obesity and metabolism in NIH's National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK). She also runs a joint research program with the Children's National Hospital in Washington, D.C.

Chung's research seeks to understand how the interaction of genes, social environment and lifestyles contribute to development outcome discrepancies in pediatric diabetes, obesity and heart disease. Her work is rooted in the belief that "improving health outcomes in diabetes and obesity means educating and caring for our youth and young adults, and empowering them and their families to lead healthy lifestyles."

Type 2 diabetes is a chronic disease occurring most commonly in middle-aged and older adults, but youth-onset type 2 diabetes (Y-T2D), once rare, is becoming increasingly prevalent. The disease progression in adolescents and young adults is faster than in older adults and young patients often require more medication sooner to maintain healthy blood sugar levels. Metformin, a common drug used to treat type 2 diabetes, is significantly less effective in younger patients. Additionally, people with Y-T2D may develop early evidence of heart disease within 5 years of Y-T2D diagnosis, suggesting a much faster progression than older adults experience.

Chung (r) with Dr. Kong Chen in one of NIH's metabolic chambers

Chung wants to improve techniques for identifying individuals at high risk for Y-T2D and heart disease and investigate why some adult treatments do not work well in younger patients. Her section conducts two groups of studies to further this goal: MIGHTY (metformin influences gut hormones in type 2 youth) and Young at Heart. MIGHTY focuses on improving current treatment regimens by understanding root causes, and Young at Heart follows Y-T2D patients and healthy control peers to define the causes and timing of developing heart disease.

"Our goal is to understand [disease progression], tailor medications and provide foundational data for larger trials," Chung summarized.

Chung's combination of research and medicine in the realm of Y-T2D earned her a Lasker Clinical Scholars award in 2020. The program supports a small number of exceptional early-career clinical researchers to promote their development to fully independent positions. She also pays it forward by mentoring post-baccalaureate students and clinical research fellows in her lab.

Chung encourages her mentees to approach and seek advice from many mentors, guidance she received from her own teachers.

"No one person is going to fit in your exact shoes, and that's a good thing," said Chung. "Success is born out of hard work [and learning from] people from all different backgrounds, experiences, abilities, and world views."

(From l to r) Members of the Section on Pediatric Diabetes, Obesity and Metabolism: Noemi Malandrino, Ila Kacker, Sophia Glaros, Natalie Macheret, Chung, Samson Cantor and Lilian Mabundo.

Medzhitov to Deliver Paul Lecture

Dr. Ruslan Medzhitov

Dr. Ruslan Medzhitov of Yale School of Medicine will deliver the annual William E. Paul Lecture as part of the NIH Director's Wednesday Afternoon Lecture Series (WALS) on Wednesday, May 21 at 2p.m. E.T. in Lipsett Amphitheater, Bldg. 10, and online at https://videocast.nih.gov/watch=55037.

This annual lecture was established in 2016 in honor of the late Dr. William E. Paul, who led the NIH immunology community. Medzhitov's talk is titled, "Immunology from First Principles."

Medzhitov, Sterling Professor of Immunobiology at Yale University and an HHMI investigator, is a renowned immunologist who has advanced the field of innate immunity, the body's first line of defense against infections. His research has clarified how toll-like receptors (TLRs) detect microbial threats and initiate immune and inflammatory responses through specific signaling pathways. For this work, he has earned major awards such as the Shaw Prize and membership in the National Academy of Sciences.

For more information or to request reasonable accommodation, email WALSoffice@od.nih.gov.
—Diana Gomez

NIH Study Reveals How Inflammation Makes Touch Painful

Study uncovers clues about how inflammation drives pain.

ADRIATICFOTO / SHUTTERSTOCK

Researchers at NIH have discovered clues about how our bodies turn sensations such as heat and touch into signals sent to the brain—and how these signals can be altered by inflammation to drive pain.

The research focuses on the nerve cells in the skin that help detect the location, intensity and emotional quality of touch, known as somatosensory neurons. By combining advanced imaging techniques with detailed molecular analysis, the researchers explored how heat and touch activate different types of receptor cells in mice.

The research revealed how different types of cells were "called into action" depending on whether the stimulus was innocuous, such as gentle warmth or touch, or noxious, meaning a stimulus strong enough to potentially cause damage to normal tissue. For example, heat and gentle touch were transmitted by entirely different types of cells.

Researchers know inflammation is linked to pain, but don't yet understand what is happening on the cellular and molecular levels. In their experiments, researchers injected prostaglandin E2 into the skin, a molecule that causes inflammation and drives pain. With the inflammatory response set into motion, researchers found that certain neurons used for signaling pain (nociceptors) became active and sensitized to heat for a long duration, demonstrating the cellular processes at play.

The study found that inflammation-related hypersensitivity to touch, known as tactile allodynia, was caused by the ongoing nociceptor activity induced by inflammation superimposed on the normal sensation of touch.

The research is part of a long-term collaboration between NIH groups. Together these labs conduct basic research focusing on how sensory input is detected and processed by the brain to evoke specific behaviors.

Genes Express Differently in Male and Female Placentas

The genes of male and female placentas have marked differences in how they are expressed, according to a recent study.

These differences involve the presence or absence of tags on DNA known as methyl groups, which switch genes on or off without changing their structure. Understanding these DNA methylation patterns may inform future research on the higher risk for pregnancy complications and later life health impacts.

The study identified 2,497 previously unreported DNA sites that had different methylation patterns for males and females. Of these and more than 2,500 sites that had also been identified by previous studies, 66.9% of large increases in methylation occurred in DNA from male placentas and 33.1% from female placentas. Increases in methylation in male placentas was linked with greater neonatal size whereas those in females was linked with greater placental size.

Some increases in methylation found in male placentas were located near the *CCDC6* gene. Lower activation of *CCDC6* has been linked with preterm birth in previous studies.

Higher methylation near the *FNDC5* gene was associated with lower expression of the gene in male placentas but not in female placentas. *FNDC5* is involved with the manufacture of irisin, which protects the placenta from damage. Lower irisin levels have been associated with the pregnancy-related high blood pressure disorder preeclampsia.

Variations in the genes *ATP5MG* and *FAM83A,* expressed in female placentas, have been associated with asthma, hay fever, eczema (dry, itchy, inflamed skin) and higher risk for breast cancer later in life.

New NIH findings may lead to insights into pregnancy complications and adult health.

ORAWAN PATTARAWIMONCHAI / SHUTTERSTOCK

Genetic factors influence the health differences between males and females, from before birth to later in life. Dysfunction of the placenta underlies many pregnancy complications and is thought to set the stage for male and female health differences that occur later in life. Variations in methylation patterns are thought to underlie many of these differences.

NIH Researchers Supercharge Ordinary Clinical Device to See Back of the Eye

NIH scientists have leveraged artificial intelligence (AI) to transform a device designed to see tissues in the back of the eye into one sharp enough to see individual cells. The technique provides imaging resolution that rivals the most advanced devices available. It's cheaper, faster and doesn't require specialized equipment or expertise.

The strategy has implications for early detection of disease and for the monitoring of treatment response by making what was once invisible now visible. The study report was published in *Communications Medicine.*

Imaging devices, known as ophthalmoscopes, are widely used to examine the light-sensing retina in the back of the eye. A scanning laser ophthalmoscope is standard in eye clinics, but its resolution can only detect structures at the tissue level—things such as lesions, blood vessels and the optic nerve head. Next-generation ophthalmoscopes enabled with adaptive optics provide greater diagnostic information, but adaptive optics-enabled imaging is still in the experimental phase.

Researchers developed a custom AI system to digitally enhance images of a layer of tissue beneath the light-sensing photoreceptors, known as the retina's pigmented epithelium (RPE). The first step was to teach the system to recognize image quality as poor, moderate or good. The researchers did this by feeding the system more than 1,400 images from different areas of the retina, obtained using adaptive-optics ophthalmoscopy.

Next, they fed the system corresponding images from the same retinal locations but obtained using standard ophthalmoscopy. An image sharpness test showed that AI improved clarity eightfold.

These techniques involve injection of a dye called indocyanine green (ICG) into the bloodstream to increase contrast of anatomical features. In the eye clinic, ICG is usually used to image the blood vessels of the eye.

The RPE cells' function is to nourish and support photoreceptors. A variety of blinding conditions first affect RPE cells, including age-related macular degeneration, vitelliform macular dystrophy and Stargardt disease. However, RPE cells cannot be easily imaged in the clinic. AI-enhanced ICG ophthalmoscopy puts RPE imaging within reach of the typical eye clinic.

NCI's Stine Retires

BY JENNIFER LOUKISSAS

Kathleen Stine, program analyst in the Radiation Epidemiology Branch (REB), retired after 41 years of federal service. Her invaluable expertise in government contracting, acquisitions and federal budgeting were essential in managing REB's many scientific contracts.

Kathleen Stine

In addition to conducting the business of scientific management for the branch, she also mentored others as they built expertise in these mission-critical fields. Stine helped to establish the initial contracts for studies of health effects after the Chernobyl nuclear power plant accident and second cancers after proton therapy in pediatric patients, as well as many other projects.

Stine started her NIH career in the Clinical Center as a medical laboratory technologist in 1984. She worked as an administrative officer for NCI's Division of Cancer Epidemiology and Genetics (DCEG) and the Office of Administrative Management from 1987 until 1999. This experience allowed her to flourish as an exceptionally proactive program analyst when she moved to REB in 1999 as she understood how the science was executed from multiple perspectives.

Through these different positions, she developed deep institutional knowledge of REB and the workings of NCI and NIH. Branch leadership relied on Stine for her wise counsel as she always examined the larger picture in decision-making and anticipated—and therefore avoided—challenges. Her keen judgment on matters related to fiscal management and execution of research contracts honed from decades of experience have been a boon to the Division and will be sorely missed.

NCI Senior Investigator Retires

Dr. Hormuzd Katki, senior investigator in the Biostatistics Branch (BB), accepted early retirement in April.

Katki is internationally recognized for his work in developing and applying quantitative methods to identify and answer the most pressing questions in cancer epidemiology. He translated these findings into tools for the prevention and early detection of cancer in individuals and populations, particularly for cancers of the cervix and lung, and developed metrics for evaluating risk models and biomarkers.

New cervical cancer screening guidelines were based on critical investigations he led to calculate cervical cancer risks over time for 1.4 million women at Kaiser Permanente Northern California, facilitating an evolution of the guidelines from test result-based to risk-based management and ensuring "equal management of women at equal risk of cancer."

Despite the recommendation of the U.S. Preventive Services Task Force for use of low dose CT (LDCT) to screen for lung cancer in current and former smokers, adoption is still limited, partly due to the inefficiency of screening which results in false positives. Katki validated several individual risk assessment tools, in collaboration with others in BB, to make screening more efficient by selecting ever-smokers at highest risk who would receive greatest benefit. Today, these tools provide the computational engine for a prediction-based online lung screening tool and the clinical decision support intervention in the EPIC electronic health record system.

Dr. Hormuzd Katki

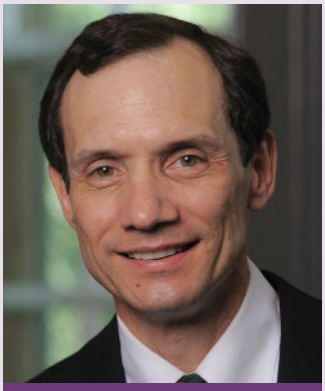
Katki evaluated all aspects of potential biomarkers for clinical trials and created the mean risk stratification to better compare test results across populations with different disease prevalence, including cervical screening and risk models to identify who in a family carries a variation in BRCA1/2.

Katki mentored both statisticians and epidemiologists. He received a B.S. in math from the University of Chicago, an M.S. in statistics from Carnegie-Mellon University and a Ph.D. in biostatistics from Johns Hopkins University, where he received the Margaret Merrell Award for research by a biostatistics doctoral student.

He joined NCI in 1999, became a principal investigator a decade later, was appointed senior investigator upon receiving NIH scientific tenure in 2015, and became an American Statistical Association fellow in 2025.

NIAID Team Honored for First FDA-Approved Drug for Ultra-Rare Genetic Disease

BY DYLAN DROBISH



Dr. Michael Lenard

NIH's National Institute of Allergy and Infectious Diseases (NIAID) recently received a 2025 Impact Award from the Federal Laboratory Consortium (FLC). The award honors laboratories whose technology transfer efforts have made a tangible, lasting impact on the populace or marketplace.

The award honors NIAID researchers who, in 2016, were the first to identify CHAPLE disease—a rare genetic disorder leading to deficiency in the complement protein CD55. In 2024, coordinated technology transfer efforts between the public and private sectors have resulted in FDA approval of the first treatment option for the disease, Pozelimab (Veopoz®) by Regeneron.

Dr. Michael Lenardo, former chief of molecular development at NIAID's Laboratory for Immune System Biology and lead inventor on the new technology, noted that most individuals diagnosed with CHAPLE disease are children who face severely debilitating symptoms and life-threatening complications.

"I saw firsthand the transformational clinical improvement that pozelimab achieves in those [living with] CHAPLE," he said. "The approval of pozelimab is a milestone to celebrate."

Following initial discovery of the disease by Lenardo's lab in 2017, multidisciplinary experts at the Technology Transfer and Intellectual Property Office (TTIPO) worked with the research team to file a patent application covering methods for the diagnosis and treatment of CHAPLE. TTIPO also worked with the team to navigate the next crucial phase of development—a 2019 collaboration with Marmara University to investigate the off-label use of eculizumab in 16 people with CHAPLE.

Although CHAPLE disease affects fewer than 100 known people worldwide, this success story serves as an inspiration—and model—for future initiatives aimed at tackling rare and complex health challenges through meticulous research and strategic partnerships.

Along with the clinical research team led by Lenardo, the dedicated specialists at TTIPO managing the technology portfolio will be formally honored at a special ceremony during the 2025 FLC National Meeting in May.

NIH Chief of Staff Burklow Retires

BY DANA TALESNIK

In April, NIH Chief of Staff John Burklow retired after 39 years of federal service, all of those years at NIH. He has been a role model and mentor to countless NIH'ers over the years, sought for his expert guidance, renowned for his institutional memory and admired for his savvy, wit and compassion.

John Burklow in his office

Burklow began his NIH career as a communications intern on an unbearably hot day in July 1986.

"It was hotter than blazes that summer and I was living in a basement apartment in Kensington," he recounted. On that first day, while driving home in his '76 Mustang, his car died and the engine caught fire. "I took that as a good omen," he quipped.

After more than a decade working on health communications at NIH's National Cancer Institute, Burklow moved to NIH's Office of the Director in 1999. Three years later, Dr. Elias Zerhouni was appointed NIH director and chose Burklow to head NIH communications.

"Working with John during my tenure as NIH director has been a hallmark of my time at NIH and professional life altogether," said former NIH Director Dr. Elias Zerhouni. "His sense of humor, loyalty and dedication for NIH and its mission, and his always thoughtful and sensitive approach to the many projects and challenges we worked on together, are unforgettable. He became one of my most trusted partners through thick and thin. I always relied on his insights."

While in the OD, Burklow worked under seven different U.S. presidents and nine NIH directors. In his early days, he helped navigate NIH leadership through stem cell policy, a conflicts-of-interest controversy and, following 9/11, biodefense, to name a few.

From l, Burklow, former NIH Director Dr. Elias Zerhouni and Calvin Jackson, former NIH deputy associate director for public affairs, in front of Bldg. 1

"John is one of the truly irreplaceable people who have made NIH great over the years," said Dr. Michael Gottesman, former deputy director for intramural research. "His calm and diplomatic management amid some turbulence was always appreciated."

Another colleague, Evelyn Castro-Rubio, who works in the Immediate Office of the Director, said, "John has been an incredible gift to NIH. Whether offering guidance, solving a challenge or simply lending an ear, he has been a steady source of wisdom and strength."

In 2009, Burklow recalled hitting the ground running from day one when Dr. Francis Collins became NIH director and the pace barely slowed over the next 12 years.

Burklow recounted a particularly fast-paced series of events that kept him on his toes in 2014. In March that year, he was the point person when the Dalai Lama visited and, that same month, the Porter Neuroscience Research Center opened.

That summer, Burklow found himself coordinating with multiple federal agencies after several vials of smallpox were inadvertently found in a cold storage room on the Bethesda campus. Beyond containment, that incident led to NIH checking and compiling a detailed inventory of every fridge, freezer and cold room on campus. Soon after, as Ebola erupted in West Africa, NIH found itself amidst a media frenzy when its Clinical Center admitted and treated a nurse who had contracted the deadly, contagious virus.

Burklow (l) with former NIH Director Dr. Francis Collins, whom he worked with for 12 years, and Collins's wife Diane Baker

PHOTO: CHIA-CHI CHARLIE CHANG

"John has been a model of wisdom, grace and good humor to all who have known him," said Dr. Richard Hodes, director of NIH's National Institute on Aging. "The trust he earned by repeatedly demonstrating strength of character allowed him to facilitate productive conversations across NIH, all to the good of this wonderful agency."

Burklow was honored with more than 20 NIH Director's Awards and received the coveted Presidential Rank Award in 2012. Throughout his tenure, he coordinated ten presidential visits to NIH. He was especially proud of being the first person to greet President George W. Bush during the president's first visit to NIH. Burklow's colleagues have long marveled at his ability to work across the aisle.

"I liked the challenge of giving honest responses in such a way as not to alienate the White House or get into too much trouble," Burklow said. "I've always been mission driven and it's bipartisan here. NIH's mission has been one of the few points we can all agree on despite being a divided society."

Burklow is still finalizing his post-retirement plans but said he hopes to work outside government but inside the NIH ecosystem.

"It's been a wonderful ride," Burklow reflected. "I've had the opportunity to work with brilliant, dedicated people who strive to make a positive difference in the world. I can't ask for more than that."

Burklow (c) with wife Debra and son Thomas at a recent gathering at NIH PHOTO: CHIA-CHI CHARLIE CHANG

Burklow credits his NCI boss and mentor Paul Van Nevel for his long career at NIH.

"I'll never forget his advice on how to spin a story," Burklow said. Van Nevel had told him, "When you're being run out of town, act like you're leading the parade."