INSIDE STANFORD MEDICINE

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The new issue of *Stanford Medicine* magazine spotlights initiatives that add value to health care.

Page 5

New med students arrive from near and far

By Mandy Erickson

Wearing a red dress, a basket cap, abalone shell earrings and a mink pelt, Melissa Eidman strode up to the microphone during the School of Medicine's white coat ceremony and announced her home town: Weitchpec, California.

Weitchpec, in the state's far northwestern corner, is the home of her ancestors, the Yurok Tribe. Her accessories are traditional garb; the pelt is a family heirloom. Eidman grew up in Sacramento, but once she's a fully trained physician, she plans to return to the Yurok reservation to care for its residents.

"I'm excited and honored to be wearing it," Eidman said of the white coat, which had her name embroidered on it. The coat is new, but for Eidman, Stanford isn't: She transferred as an undergraduate after spending years at a community college. "It's been quite a long haul," she said.

Ninety medical students donned white coats at the Aug. 23 ceremony outside the Li Ka Shing Center for Learning and Knowledge. In a separate ceremony earlier that afternoon, 28 news students in the physician assistant program also put on white coats. The events marked the beginning of the students' respective programs.

All students also received stethoscopes, gifts from the Stanford Medicine Alumni Association.

STEVE FISCH

New medical students read the Stanford Affirmation on Aug. 23 at the white coat ceremony, held next to the Li Ka Shing Center for Learning and Knowledge. The affirmation includes the following sentences: "I pledge to devote my life to the service of humanity. The care of my patients will be my first consideration."

Diverse class

"By all accounts, you are among the most accomplished people on the planet," Dean Lloyd Minor, MD, told the new medical students at their ceremony. "When you receive your coat and stethoscope, you're showing your willingness to run toward crises and not away from them."

The medical students hail from New York; Houston; and nearby Redwood City, California, as well as from Kazakhstan, Venezuela and England, among other places. The class of 2023 is among the most diverse in Stanford's history, with 26% who identify as an ethnicity under-represented in medicine, 34% who were born outside the United States and 8% who are LGBTQ.

Many students said they would need to get used to wearing the white coat. "It feels really surreal," said Ibtihal Elfaki, a medical student from Sudan and Chicago. "It's something I've aspired to since high school."

"The idea of being a medical student has been abstract for a long time," said Samson Peter of San Mateo, California. "But this coat makes it all very tangible."

They also felt the burden of responsibility that the white coat carries — "an overwhelming sense of accountability," said Alexander

See CEREMONIES, page 4

Inflammation may trigger silent mutation, causing deadly lung disease, study shows

By Tracie White

Researchers at the School of Medicine have found that inflammation in the lungs of rats, triggered by something as simple as the flu, may wake up a silent genetic defect that causes sudden onset cases of pulmonary hypertension, a deadly form of high blood pressure in the lungs.

"It's a kind of one-two punch," said Amy Tian, PhD, senior research scientist in pulmonary and critical care. "Basically, the first hit is the mutation, and the second hit is inflammation in the arteries of the lungs. You can be healthy and carrying this mutation, and all of the sudden you get a bacterial or viral infection, and it leads to this terrible disease. "

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Tian is the lead author of the study, which was published Aug. 29 in *Circulation*. Mark Nicolls, MD, professor and chief of pulmonary and critical care medicine, is the senior author.

"This is important research for understanding how 'second hits' can render ordinarily silent genetic mutations deadly," Nicolls said. "It also further advances the the scientific understanding of the role of inflammation in pulmonary hypertension."

There is no known cause of pulmonary hypertension, a debilitating disease that causes difficulty breathing, fatigue and chest pain. It can leave patients too weakened to perform simple daily activities, such as climbing a flight of stairs. About 200,000 people a year are hospitalized with the disease in the United States, according to the Pulmonary Hypertension Association of America. The only available cure for severe forms of the disease is lung transplantation, but it has only a 30% survival rate.

See HYPERTENSION, page 6

Response to flu vaccine affected by gut bacteria, according to scientists

By Bruce Goldman

A new study in healthy adults suggests that antibiotics may reduce the effectiveness of the flu vaccine.

Bali Pulendran

The depletion of gut bacteria by antibiotics appears to leave the immune system less able to respond to new challenges, such as exposure to previously unencountered germs or vaccines, said Bali Pulendran, PhD, professor of pathology and of microbiology and immunology at the School of Medicine.

"To our knowledge, this is the first demonstration of the effects of broad-spectrum antibiotics on the immune response in humans — in this case, our response to vaccination — directly induced through the disturbance of our gut bacteria," he said.

The study was published Sept. 5 in *Cell*. Pulendran, who holds the Violetta

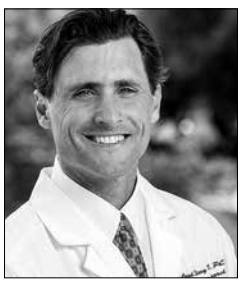
See BACTERIA, page 7

Researchers enhance neuron recovery in rats after blood flow stalls

By Jonathan Wosen

Researchers at the School of Medicine report in a new study that they found a way to help rats recover neurons in the brain's center of learning and memory. They accomplished the feat by blocking a molecule that controls how efficiently genetic instructions are used to build proteins.

If the approach described in the study could be applied to humans, it may one day help patients who've suffered a stroke, cardiac arrest or major blood loss and are thus at higher risk of memory loss.



Creed Stary

In the study, published online Aug. 19 in *eNeuro*, researchers induced extremely low blood pressure — as would happen when the heart stops beating — in rats. These rats lost neurons in a specific region of the hippocampus critical to learning and memory, but the researchers improved the animals' recovery of the cells by injecting a molecule that blocks a microRNA: a short molecule that tweaks gene activation by preventing the conversion of genetic blueprints into proteins. Interestingly, the scientists found that a microRNA blockade potentially causes astrocytes — cells that support neurons and make up 50% of cells in the brain — to turn into neurons.

The findings demonstrate that neurons, with some assistance from their astrocyte neighbors, recover in a region of the hippocampus not known to have a local stem cell population that can replenish lost neurons. Enhancing this recovery in humans could help those who've suffered a temporary loss of blood flow to the brain.

"There's currently no treatment to improve brain function in patients with heavy blood loss, cardiac arrest or stroke," said Creed Stary, MD, PhD, assistant professor of anesthesiology, perioperative and pain medicine. "This is the first study to show that the natural process of post-injury hippocampal recovery can be substantially improved with a pharmaceutical microRNA-based therapy."

Stary is the study's senior author. Lead authorship is shared by postdoctoral scholar Brian Griffiths, PhD, and senior research scientist Yi-Bing Ouyang, PhD.

Under (low blood) pressure

When fresh blood stops flowing through the brain, cellular waste piles up, and neurons starved of oxygen and glucose eventually die. This can occur when a person has a stroke, loses a significant amount of blood or suffers a cardiac arrest.

Amid the damage, levels of a microRNA known as miR-181a soar. In an earlier study, the researchers blocked miR-181a with a molecule designed to stick to and inactivate the microRNA. They found that blocking miR-181a before reducing the flow of blood to the brains of rats stopped neurons from dying.

"If you want to find a therapy for an injury, one approach is to look for disruptions that occur in cells and try to reverse them. The first step was asking, 'Is reversing the increase in this specific microRNA protective?'" Stary said.

But while the prior findings were encouraging, they didn't reflect how such an intervention would probably be used in a clinical setting; it's more likely that a patient would receive a microRNA blockade after an injury.

To test whether miR-181a blockade helped rats recover hippocampal neurons, the researchers decreased the rats' blood pressure dramatically by siphoning off much of their blood and reinfusing it 10 minutes later. Similar drops in blood pressure can occur in people during cardiac arrest, after a major loss of blood or during certain surgeries.

The blood pressure drop caused nearly 95% of neurons in a region of the hippocampus known as CA1 to die off. By around two months after the procedure, those neurons bounced back to nearly 50% of normal levels.

The researchers then tested the effects of a microRNA blockade by injecting the blocking molecule directly into the hippocampus of rats either two hours or seven days after the animals experienced a drop in blood pressure. These rats had significantly higher neuronal recovery than those injected with a control molecule that didn't target any known microRNAs. In earlier studies, the researchers showed they could deliver the blockade intravenously, making it well-suited for clinical use.

> "There's currently no treatment to improve brain function in patients with heavy blood loss, cardiac arrest or stroke."

Solving a puzzle

But the fact that there was *any* recovery was puzzling. The hippocampus is one of the few brain regions that harbors neural stem cells, which can form new neurons in adults, but not in the CA1 region the researchers were studying.

"If you don't have new neural stem cells and you don't have any evidence of cell division, then how are CA1 neurons being repopulated?" Stary said.

The researchers had one important clue: When CA1 neurons were at their nadir, specialized neuronal support cells known as astrocytes moved into the damaged region. Typically, astrocytes sit above and below the neuron-containing layer of the CA1 and support the metabolism and connectivity of their neuronal neighbors.

To figure out what the astrocytes were up to, the scientists tracked them with fluorescent molecules that labelled astrocytes green and neurons red. When they looked under the microscope, they found cells that glowed yellow — meaning the cells expressed both green and red markers. These yellow cells were found at higher levels in rats in which miR181a had been blocked.

The observation strongly implied that some of the astrocytes were beginning to turn into neurons. While the researchers are planning further experiments to confirm the finding, astrocytes have been shown to turn into neurons in other animal models of brain injury. Whether this phenomenon occurs in humans after loss of blood flow to the brain has not yet been fully established, but if verified, it could open a new realm of astrocyte-based gene therapies for survivors of cardiac arrest and stroke.

The researchers next plan to verify whether blocking miRNA-181a helps the rats recover their memory, learning and other cognitive abilities linked to the hippocampus. If so, the approach is one step closer to aiding recovery from brain injuries in which blood flow gets cut off.

"This paper shows that you can effectively augment the normal recovery the brain tries to do on its own by blocking this specific microRNA across injury models and across species, something of a holy grail for a gene therapy," Stary said. "It points toward blocking the microRNA being a protective agent itself, but also provides insight to identify new therapeutic gene targets, opening the possibility for combinatorial or adjuvant pharmaceutical therapies."

Stary is a member of Stanford Bio-X and the Wu Tsai Neurosciences Institute at Stanford.

Other Stanford co-authors of the study are research associates Lijun Xu, MD, and Xiaoyun Sun, MD; and Rona Giffard, MD, PhD, professor emerita of anesthesiology, perioperative and pain medicine.

This study was supported by the American Heart Association and the National Institutes of Health.

Stanford's Department of Anesthesiology, Perioperative and Pain Medicine also supported the work. ISM

Mildred Cho to co-lead new coordinating hub for biomedical ethics

By Patricia Hannon

The Stanford Center for Biomedical Ethics has been chosen by the National Human Genome Research Institute to help develop a coordinating hub for information on the ethical, legal and social implications of genetic research.

The new Center for ELSI Resources and Analysis is being funded with a $7.1 million award, the institute announced Aug. 14. ELSI is an abbreviation for the ethical, legal and social implications of genomics.

The effort will be led by Mildred Cho, PhD, associate director of the Stanford Biomedical Ethics Center, and Sandra Soo-Jin Lee, PhD, chief of the Division of Ethics and faculty in Columbia University's Department of Medical Humanities and Ethics. The Hastings Center, a bioethics research organization, and Harvard University, which houses the Personal Genetics Education Project, will collaborate on the new center.

Cho said the mission of the ELSI center is to build a community around conducting, sharing and using ELSI research, and to make that collection of material accessible and understandable to experts and the public.

Mildred Cho

Almost 30 years after the launch of the International Human Genome Project, which aimed to map the human genome, many ethical and legal concerns still surround genetic research, Cho noted. Yet there's no central repository where people grappling with such issues can easily find ethical or policy guidance, or published research, or connect with experts who can help guide them.

"I think it should be easier for people who are writing legislation that has to do with consumer genetic testing, or the release of data from Ancestry.com, to be able to find experts who are knowledgeable about the implications of data collection by genomic companies," said Cho, professor of pediatrics and of medicine.

Filling a void

The center is poised to fill that void with a new web portal for ELSI content that will be identified, collected and shared. Cho expects that content to come from such sources as scientific and academic research, news stories and commentary, scientific journals, laws and legal briefings.

Through such data collection, the center's leaders hope to gain a better understanding of gaps in knowledge and to communicate those gaps to researchers. They also hope to help influence scientific investigation by facilitating collaborations between researchers and by holding a separately funded biennial congress to bring them together.

But central to center's aim is to share ELSI knowledge with people who might not be aware it's available, and to tailor the information for specific stakeholders — policymakers, medical professionals, journalists, teachers, the general public and even scientists themselves. This is important, Cho said, because ELSI content draws from a number of disciplines, including philosophy, sociology, psychiatry, economics and politics.

"We're going to try to develop new methods for synthe- See ETHICS, page 3

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STANFORD MEDICINE

5 QUESTIONS

an occasional feature in which an expert answers five questions on a science or policy topic

On using 'real-world data' to inform clinical care

Electronic health records have paved the way for a new approach to biomedical research: the analysis of real-world data. The term "real-world data" comes from the idea that EHRs aren't just health histories of single individuals; they're a trove of information that can help guide clinical decisions, from diagnosis to treatment.

In 2016, the 21st Century Cures Act became federal law. It helped bring about a new focus on harnessing real-world data in medicine, as opposed to letting clinical trials be the sole guiding source. Today, doctors and scientists are increasingly turning toward algorithms trained on EHRs to guide diagnoses and determine treatment options for patients.

The problem is that EHRs can be quite messy, making data extraction complex, said Tina Hernandez-Boussard, MD, PhD, associate professor of medicine, of biomedical data science and of surgery at the School of Medicine. So she and a team of scientists set up a case study to determine how accurately algorithms trained on real-world data can predict the answer to medically relevant questions. In the case study, Hernandez-Boussard and a team of scientists posed a simple question: How well can an EHR-trained algorithm predict if a patient has a cardiovascular condition?

In a paper published Aug. 15 in the Journal of the American Medical Informatics Association*, Hernandez-Boussard, lead author of the study, shows that the answer is very well — and also not so well. It turns out the accuracy of the prediction depends on how the data is organized and gathered.*

Science writer Hanae Armitage spoke with Hernandez-Boussard about the value of real-world data, how it fits into the current clinical care landscape and what her study revealed about harnessing this immense dataset.

1 What's the most important contribution of real-world data to medical decision-making?

HERNANDEZ-BOUSSARD: It's really the generalizability that's key; pulling from real-world data provides information that's representative of the way most the population receives their care. Real-world data doesn't have strict inclusion or exclusion criteria, so it means that any clinical assertions made with that data can apply to a typical patient seeking routine care.

Very often "gold standard" clinical trials are looking at a particular drug for a particular disease, and patients can end up excluded if they have conflicting comorbidities, some of which can be quite common. So say, for example, there's a trial for a cancer drug; it might exclude a patient who has hypertension or diabetes. But in reality, the patients with hypertension and diabetes might be the ones who really benefit from the drug. And so looser inclusion and exclusion criteria enables us to see how this drug works, or doesn't work, in much broader populations and subpopulations of patients.

2 Your team set up a case study to evaluate real-word data in the context of cardiovascular medicine. What did the study investigate and what were the most significant takeaways?

HERNANDEZ-BOUSSARD: With our case study, we set out to see if and how real-world evidence could be used to guide clinical assertions, such as identifying a patient with a particular disease or guiding treatment options. It seems reasonable to expect that real-world data could help inform those clinical assertions, but this data largely comes from electronic health records and insurance claims. They're not intended to guide care for others; they're meant to capture information for billing and patient history purposes, and they're really complex and messy. So, any data we pull out of them has to be done with great caution. We wanted to assess how accurately EHR-based algorithms are when predicting a cardiovascular condition.

In general, we saw that people using this EHR data are mostly accessing something called "structured data," which are things like vitals and medical codes that denote different diseases. We tend to think of this data as more regularly curated, and therefore more accurate, but our case study showed us that the most rich information is actually in the clinical narrative text, or unstructured data — for example, the free text notes that a clinician takes during a patient visit.

So in our study we found that we were more accurately able to identify different aspects of the population — like diseases or procedures they'd had done — when using just the unstructured data. So for example, we wanted to see if we could identify the population of patients who had coronary artery disease. When we used structured data we could do so with 80% accuracy. But when we use the unstructured data, that jumped up to 95%.

Tina Hernandez-Boussard

When we think about how clinicians and patients interact, the clinician is an active listener and note taker, capturing much of their conversation in written narrative form. That's where they're likely able to provide the most details about the patient, as opposed to structured data, which fall more into the numbers and codes category. So we end up getting richer, more accurate information when we just analyze free text.

3 What were the limitations of using real-world data in your case study?

HERNANDEZ-BOUSSARD: I would say that the technology to actually run the algorithms that harness real-world data is, in many ways, a limiting factor. For this study, we used state-of-the-art artificial intelligence technologies. And to be able to harness real-world data effectively is a key part, but not everyone has access to it or the expertise to conduct that kind of analysis. The second limitation is actually having access to data on a large scale while being compliant with privacy laws. While we were able to conduct our study with the data set from Verantos, a collaborator on the study, data silos are a very real problem, and it's not always easy to gain access to such a large amount of data that encompasses different health care settings from different geographical locations. These diverse datasets are important to address the generalizability of the technologies.

4 Why is there an increasing trend toward using real-word evidence instead of, or in conjunction with, the clinical trial data to inform medical decisions?

HERNANDEZ-BOUSSARD: There are a handful of reasons. First, clinical trials are pricey — we're talking millions and millions of dollars — and they only encompass a very small portion of the population. Real-world data repurposes EHRs to guide clinical care at a fraction of the cost. The second big criticism of clinical trials is that they're often not broadly generalizable. The patients that are included in trials often do not fully represent the range of patients that could benefit from a new drug or therapy.

Third, these clinical trials are highly controlled. Patients come in for an appointment or treatment and in, say, exactly two weeks they must come back for a follow-up or their next dose of medication. But that's not really the way that patients receive care. In reality, patients aren't always able to stick to a regimented calendar. Maybe they're on vacation and need to wait three weeks until they schedule their next appointment.

5 How do you think real-world evidence can best fit into a clinical care context?

HERNANDEZ-BOUSSARD: Clinical trial data still provides the highest level of certainty for guiding clinical care. But if, for example, you run a clinical trial, and end up with a very low representation of a specific subpopulation, how would you know how well that drug works in that subpopulation? And that's where we think real-world data could fill in a gap.

We're not suggesting that real-world evidence should replace clinical trials by any means — clinical trials are still held as the gold standard. Instead, we see it as more of a hybrid situation, with patient care benefiting from both sides. ISM

Applications for Chan Zuckerberg Biohub fellowships open to residents, clinical fellows

The Chan Zuckerberg Biohub, a medical research organization that collaborates with Stanford, UC-Berkeley and UC-San Francisco, is offering two- to three-year research fellowships for medical residents and clinical fellows.

Biohub fellows will conduct research under a faculty mentor at one of the three campuses or at a Chan Zuckerberg Biohub lab in San Francisco. They will also learn about medical economics, grant writing, career development and ethics, as well as various biomedical and clinical research subjects.

The goal of the fellowship is to equip young doctors to incorporate research into their careers. The number of physician-scientists is in decline, largely because of financial pressures on academic medical centers.

"Despite their important role in biomedical discovery, fewer physicians are dedicating their professional lives to research," said David Cornfield, MD, the program's co-director and a professor of pediatric pulmonary medicine at the Stanford School of Medicine. "The CZ Biohub Physician-Scientist Fellowship Program aims to change that by providing medical doctors who have limited research experience with the opportunity to pursue a basic research path, gain expertise and cultivate their passion for advancing medical knowledge."

The program will award about six fellowships per year. Applicants must hold an MD degree but not a PhD, and be enrolled full-time in a medical residency or clinical fellowship at Stanford or UCSF. They will need to commit to at least two years at 75% time with CZ Biohub and spend at least 20% of their time seeing patients. The fellowship will begin July 1, 2020.

The Biohub is accepting applications until Oct. 1, 2019; awards will be announced in December. Fellows can expect to be paid a salary similar to the one they are receiving for their residency or current fellowship. Applicants can find more information and apply at https://www.czbiohub.org/psfp. ISM

Ethics

continued from page 2

sizing different kinds of knowledge that typically aren't combined," Cho said.

The center's collaborators bring a wealth of expertise to the project, with much of the team's work being conducted virtually, rather than at a single location. Lee, a former senior research scholar at Stanford's Center for Biomedical Ethics, and other Columbia faculty members who specialize in ethics, genetics and biomedical informatics will work at their campus to build the ELSI community. They also will partner with Columbia's Graduate School of Journalism to assess how journalists might use the portal, and what tools and information they would find useful in their reporting.

> "We have the opportunity to have our fingers on the pulse of ELSI research."

Other Stanford researchers will contribute to the project, including David Magnus, PhD, director of the Center for Biomedical Ethics and the Thomas A. Raffin Professor of Medicine and Biomedical Ethics. In addition, researchers from UC-San Diego and Case Western University will be involved.

Cho said a number of new people also will be brought in to work on the Stanford campus in various roles, including to build the web portal and develop the backbone of its technology, website and database.

"We have the opportunity to have our fingers on the pulse of ELSI research and understand where the field is going and where it needs to go," Cho said.

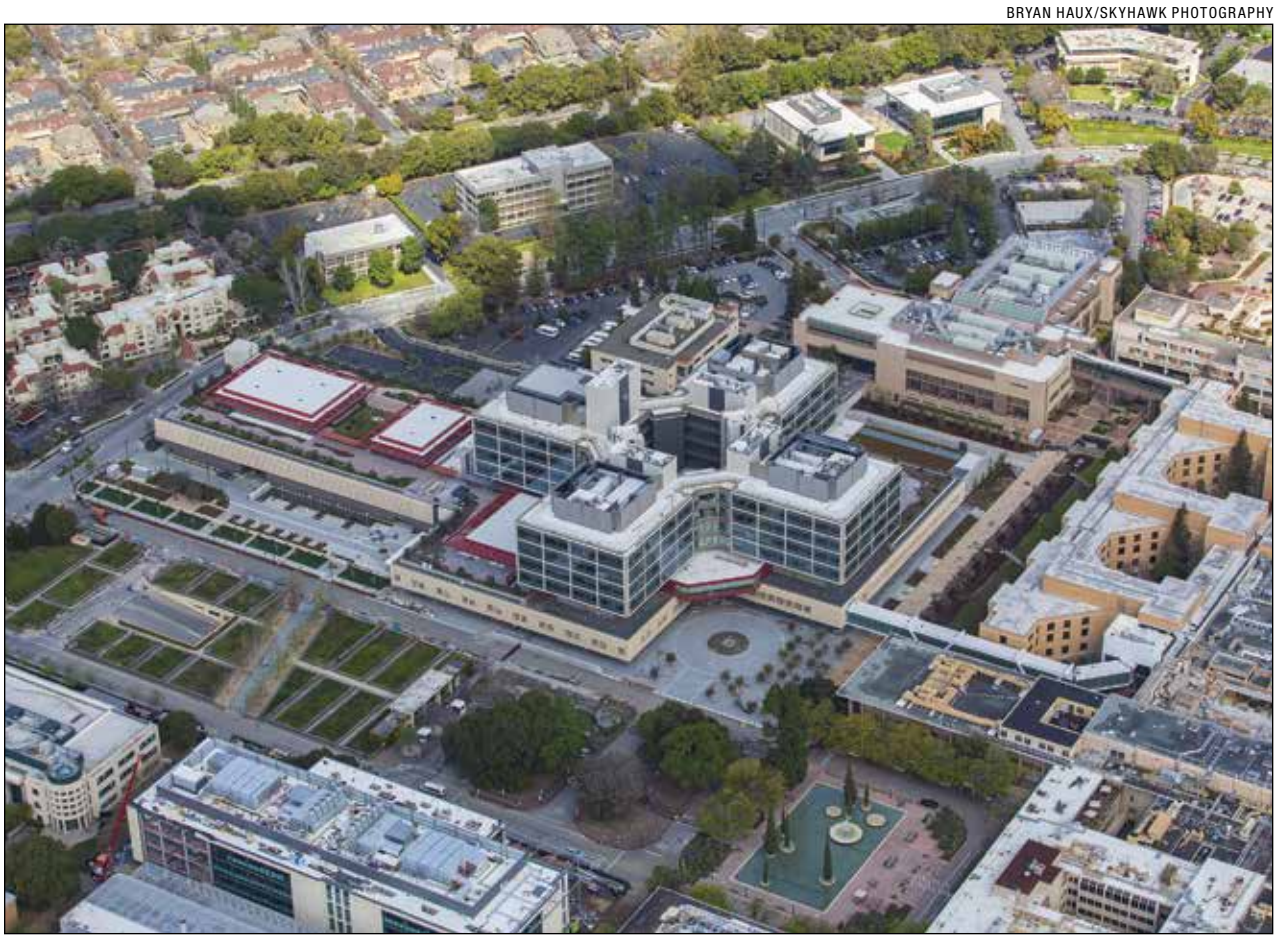
Cho also leads the Center for Integration of Research on Genetics and Ethics at Stanford. It was established in 2004 as one of several Human Genome Research Institute's Centers for Excellence in ELSI research and is based at Stanford's Center for Biomedical Ethics. ISM

New adult hospital to host community open house Sept. 14-15

The new Stanford Hospital will officially open its doors to patients in the fall, but on Sept. 14 and 15, local residents can get a sneak peek as part of a two-day community open house featuring a multimedia experience and a health- and wellness-inspired street fair with food trucks and family activities.

"We are thrilled that our vision of expanding our world-class academic medical center has come to fruition," said David Entwistle, president and CEO of Stanford Health Care. "The new Stanford Hospital is first and foremost our community's hospital."

BRYAN HAUX/SKYHAWK PHOTOGRAPHY



Local residents can get a sneak peek of the new Stanford Hospital at a community open house scheduled for Sept. 14-15.

Open house visitors will be able to take part in a 50-minute multimedia experience of the new facility. They'll have the opportunity to learn about the robots that deliver supplies throughout the building, the technology-rich patient rooms and operating suites, the safety features, original art and rooftop gardens. Experts also will be on hand to answer questions about the building's design and construction and the advanced patient care technologies.

"The community open house events will be a once-in-a-lifetime opportunity for the general public to go behind the scenes of the new hospital before it opens," said Maggie Pringle Grauer, chair of the Stanford Medicine Community Council. "We are creating an experiential tour that will give visitors a firsthand look at the delivery of modern medicine."

In the promenade between the original Stanford Hospital and the new building, there will be a street fair with interactive activities for all ages. Booths will feature demonstrations of the latest telehealth and virtual reality tools used in surgery and patient care. There also will be health and nutrition advice, a larger-than-life game of Operation, Stanford's therapy dogs, face painting, a treasure hunt, balloons, live music and a teddy bear triage area, where kids can care for the stuffed animals and take them home.

Visitors will also have the chance to view the Stanford VOICES digital mural project, a mosaic of more than 4,000 drawings completed by patients, staff and community members.

The community open house will run 9 a.m.-5 p.m. Sept. 14 and 9 a.m.-3 p.m. Sept. 15. It's free and open to the public, but pre-registration is required. Sign up at www.stanfordhealthcares.com. Free parking is available at the Stock Farm Parking Garage, with shuttle access to the hospital entrance. **ISM**

Ceremonies

continued from page 1

Noonan, a student in the physician assistant program.

Speakers at both ceremonies acknowledged the students' competitiveness, but cautioned that they must set that tendency aside if they are to learn and grow during their time at Stanford.

"Now is the time to work together and help each other out," Zachary Stone, a third-year PA student, said at the physician assistant ceremony.

Kimberly DeBruler, a third-year medical student, warned the new MD students that they would fail along the way. She also encouraged them to reach out to students who appear to be struggling. "Check on each other. Text that person who didn't make it to the party, or who stopped coming to class," she said.

looking forward to the years ahead.

"I'm eager to get back to being with patients," said physician assistant student Alex Topmiller, who worked at a dermatology clinic before arriving at Stanford. "I can't wait for the clinics." **ISM**

STEVE FISCH

Compassion and science

Faculty speakers reminded the students that caring for patients calls for compassion as well as scientific knowledge. Abraham Verghese, MD, professor of medicine and a bestselling author, told the physician assistant students that he learned the difference between healing and curing from his own physician assistant when he was practicing in El Paso, Texas. When the physician assistant took him to the home of a young man dying of AIDS, he asked, "What are we doing here?" She responded, "We're not here to do anything. We're just here to be with the patient."

He realized that their visit signaled to the patient that his caregivers would not abandon him. In the days before there was an effective treatment for AIDS, he said, "We could heal when we could not cure."

Arturo Molina, MD, president of the Stanford Medicine Alumni Association, described the relationship between physician and patient as "sacred." He told the medical students that they would need to practice empathy.

"You will see patients who have had their dreams shattered by illness," he said. "They will remind you not to take anything for granted."

At the receptions after the ceremonies, as the new students started feeling more comfortable in their new garb, they said they were

(Clockwise from top left) Dean Lloyd Minor addresses the new medical students. First-year medical students with Iris Gibbs (fourth from right), associate dean of MD admissions. New students in the physician assistant program participated in a white coat ceremony earlier in the day on Aug. 23.

Stem cell researcher Irving Weissman awarded Albany Prize

By Krista Conger

Irving Weissman, MD, director of Stanford's Institute for Stem Cell Biology and Regenerative Medicine, will receive the 2019 Albany Medical Center Prize in Medicine and Biomedical Research for his pioneering work in stem cell and cancer biology, including the identification of blood-forming stem cells and their role in blood cancers, as well as the discovery of a "don't eat me" signal on the surface of many cancer cells that protects them from being eliminated by the immune system.

Weissman is a professor of pathology and of developmental biology at the Stanford School of Medicine and is the director of the Ludwig Center for Cancer Stem Cell Research at Stanford. He will share the $500,000 prize with Bert Vogelstein, MD, who is the Clayton Professor of Oncology and Pathology at Johns Hopkins University's Sidney Kimmel Cancer Center and the director of the Lustgarten Foundation Pancreatic Cancer Research Laboratory at Johns Hopkins.

Vogelstein is known for discovering that a protein called p53 functions as a tumor suppressor and that its inactivation is critical to the development of many human cancers. He was also the first to demonstrate in colorectal cancer that disease progression is a multistep process resulting from the sequential accumulation of mutations in specific cancer-associated genes. Together, Weissman and Vogelstein transformed the understanding of cancer biology, cancer genomics and disease initiation and progression, paving the way for earlier diagnosis and more effective treatments for a wide range of diseases including leukemia, non-Hodgkin lymphoma and severe combined immunodeficiency (also known as "bubble boy" disease).

The two will be presented with the prize at a Sept. 25 ceremony in Albany, New York.

"Dr. Weissman's groundbreaking work in advancing our understanding of blood-forming stem cells and cancer has transformed many aspects of modern medicine," said Lloyd Minor, MD, dean of the Stanford School of Medicine. "The discovery of the 'don't eat me' signal on cancer cells promises to lead to novel clinical applications that will improve human health. We congratulate Dr. Weissman on this well-deserved recognition."

Adult stem cells are unique in that they can both self-renew and make progenitor cells that give rise to all the specific cell types in a particular tissue of the body. In 1988, Weissman was the first to identify and isolate in mice the hematopoietic, or blood-forming, stem cells that form all the cells of the blood and immune system. In 1992, he and his group found the human blood-forming cells. He and his group have since painstakingly traced the cellular steps leading from a stem cell to each of the many types of mature blood and immune cells in humans, and identified those that go awry in many blood diseases and cancers.

The prize will be presented at a Sept. 25 ceremony in Albany, New York.

Weissman also identified a molecule called CD47 that exists on the surface of nearly every human cancer cell and protects them from attack by immune cells called macrophages. An antibody targeting CD47, which the researchers have termed a "don't eat me" signal, is in clinical trials in people with several types of blood and solid cancers. Overexpression of CD47 is also implicated in fibrotic diseases such as scleroderma and surgical adhesions. Recently, Weissman identified additional "don't eat me" signals, each of which is expressed by particular types of cancers.

NORBERT VON DER GROEBEN

Stanford's Irving Weissman will share the 2019 Albany Medical Center Prize in Medicine and Biomedical Research with Bert Vogelstein of Johns Hopkins University for discoveries in stem cell and cancer biology.

"I'm especially honored to share this award with Bert Vogelstein, whose work I have followed for many years and greatly admire," said Weissman, who is the Virginia and D.K. Ludwig Professor for Clinical Investigation in Cancer Research. "Inspired by his earlier work on colon cancer, we were able to show that nearly all stepwise mutations that lead to the development of leukemia and blood diseases, such as myelodysplastic syndrome, occur in blood-forming stem cells, apparently hitchhiking in these self-renewing cells to form disease clones. It's a fantastic feeling to join the group of highly accomplished past recipients of the Albany Prize."

The Albany Prize is funded by a $50 million gift from New York City philanthropist Morris Silverman. It has been awarded since 2001 to encourage and recognize extraordinary and sustained contributions to improving health care and promoting biomedical research with translational benefits for better patient care. In 2015, Karl Deisseroth, MD, PhD, Stanford professor of bioengineering and of psychiatry and behavioral sciences, received the award. ISM

Stanford Medicine spotlights efforts that add value to health care

By Patricia Hannon

Eugene Celis can name many family members who died before the age of 60 from heart disease or complications from diabetes, and that worried him. But Humanwide, a yearlong pilot project at Stanford that integrated detailed health data about him with team-based care, helped Celis see his way toward a different future.

The project is emblematic of the initiatives explored in the latest issue of *Stanford Medicine* magazine, which features ways that Stanford Medicine's focus on value is addressing the needs of patients and clinicians.

In a letter introducing the issue, Lloyd Minor, MD, dean of the Stanford School of Medicine, writes that shifting the focus of health care to detect disease earlier, strengthen patient-provider relationships and deploy the latest health technology enhances value in medicine and demonstrates the power and promise of precision health.

"Personalized medicine means we can modify care down to an individual's genome," he writes. "And a better understanding of the social determinants of health — everything from our behavior to our environment — means we have the opportunity and duty to treat each person individually."

The stories in this issue touch on projects that are aimed at doing just that:

• In the Humanwide project, clinicians and patients worked together — using information from genetic screenings, at-home digital monitoring devices and detailed wellness assessments — to better prevent, predict and cure disease.

• The Stanford Clinical Informatics Consult service harnesses a trove of health data from millions of anonymous patient records to provide the ultimate consult when a physician is stumped by a patient's condition, symptoms or treatment options.

• Initiatives to battle physician burnout at Stanford are aimed at increasing professional fulfillment for doctors by improving their work experiences and building efficiency to promote better teamwork and work-life integration.

• A new emergency protocol at Stanford Hospital drastically cuts down on the time it takes to give stroke patients a lifesaving treatment that can mean the difference between walking and not walking, and between living alone and relying on caregivers.

• When the director of a key outpatient clinic at Lucile Packard Children's Hospital Stanford set out to determine whether it was possible to increase patient capacity without compromising care, she turned to Stanford mathematicians for help.

• Biobanks aren't new, but a 2017 high-tech revamp of Stanford's biobanking approach is bridging the gap between the latest biomedical research and patient care.

• Delivering bad news to patients and their families is about the toughest part of a physician's job, but a new virtual reality training program gives them tools that can help.

Also in this issue, read about how the vision of a mesa in a scientist's dream changed the course of research into hypertrophic cardiomyopathy, a deadly heart condition. And learn about how scientists see promise in a study showing that use of the hormone vasopressin helps children with autism improve their social skills.

Print copies of the magazine, which is also online at http://stanmed.stanford.edu, are being sent to subscribers. Others can request a copy at (650) 723-6911 or by sending an email to medmag@stanford.edu. ISM

■ OBITUARY

James Trudell, who helped make anesthetics safer, dies at 77

By Tracie White

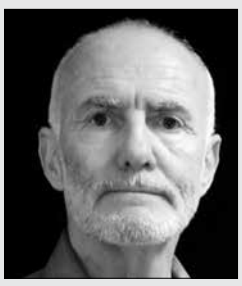
James Trudell, PhD, a Stanford Medicine chemist who spent 50 years hunting down molecular clues to help make anesthetic drugs safer for patients, died July 29 at his home in Woodside, California, with his wife by his side. He was 77.

The cause was complications from acute myeloid leukemia.

Trudell was a professor of anesthesiology, perioperative and pain medicine at the School of Medicine, where he could be seen for most of his career bicycling to his lab in the Grant Building. His many decades of research advanced the understanding of how anesthetics work to limit pain and contributed to the ongoing pursuit to make newer, better anesthetics with fewer dangerous side effects.

"He was very obviously fascinated with understanding, as a chemist, how molecules work," said Ron Pearl, MD, professor and chair of anesthesiology, perioperative and pain medicine and the Dr. Richard K. and Erika N. Richards Professor, who worked with Trudell since 1985. "How exactly do they interact? How can we make them do things we want them to do and not do things we don't want them to? It was important to him that his basic research would make people's lives better."

Trudell's fascination with chemistry began early in life when his parents gave him a chemistry kit at the age of 9. His passion for the work never waned, continuing until the day he died, with a laptop computer and molecular modeling research papers still open on his desk, said his wife, WeiQi Lin, MD, PhD.



James Trudell

"For 50 years, Dr. Trudell's innovative research played a major role in advancing the field of anesthetics and improving patient safety," said Lloyd Minor, MD, dean of the School of Medicine. "More than just an exceptional scientist, he also strengthened the Stanford Medicine community by serving as a caring mentor and thoughtful collaborator to so many."

Native of Michigan

Trudell was born in Iron Mountain, Michigan, in 1941. He graduated magna cum laude from the University of Michigan before going on to earn a PhD in organic chemistry at Stanford in 1969. While a student at Stanford, he studied under Carl Djerassi, PhD, a chemistry professor at the time, who later became known as the father of the birth control pill. They remained lifelong friends. Before starting graduate school, Trudell served two years on the aircraft carrier USS Randolph as a photo and electronics officer during the Vietnam War.

> "It was important to him that his basic research would make people's lives better."

He joined the Anesthesiology Department at Stanford in 1969, just four years after it was created. He initially studied the molecular mechanisms that made halothane, one of the first modern-day general anesthetics, toxic to the liver. At the time, there were only 12 faculty members in the department; today there are 200, Pearl said. Trudell met Lin, his future wife, when she came to work as a postdoctoral scholar in in his lab.

Lin describes her husband as the consummate scientist, for whom chemistry was more than just a job.

"He was a true scientist at heart," Lin said. "He followed his research wherever it took him. He was never discouraged by any failure. He would just take a break and go to one of his many hobbies, and come right back to his work. "

Trudell was also an accomplished athlete who enjoyed a variety of activities, from rock climbing, paragliding and running marathons in his earlier years to bicycling and sailing throughout his life.

"We expected him to live to 100," said longtime friend and Stanford colleague Edward Bertaccini, MD, professor of anesthesiology, perioperative and pain medicine. "This guy was in the best shape all of his life. He was windsurfing up until 15 years ago and always riding his bike."

Study of drug metabolism

Trudell's early work involved studying how drugs metabolize within the human body. After figuring out how the drug halothane causes liver failure, he examined the same problem in other anesthetics. Along the way, he discovered that anesthetics work through specific protein interactions rather than interactions with lipids, the fatty molecules in tissues, as was believed at the time.

His early work eventually led him to what was a new approach 30 years ago: the use of molecular modeling to study receptor sites, the binding structures on the surface of cells involved in how drugs cause unconsciousness. He began collaborating with Bertaccini in the early 1990s, using this research to help create new anesthetics with fewer side effects, including liver toxicity and lowering blood pressure.

"Our research, the molecular mechanism of how anesthesia works, is a small niche in the world, but he played a key role," Bertaccini said.

At times, during their long collaboration, the two scientists would delve into the metaphysical questions surrounding their search to understand how people become unconscious to begin with — what scientists refer to as "the human off switch," Bertaccini said. "It's a metaphysical question, this idea that all human beings have the same 'off switch.' What happens to the human soul when you are anesthetized? These drugs are used thousands of times a day throughout the world. People wake up and they are still there. We know it works; we just don't know how it works."

He continued: "Jim probably knows the answer now and is laughing at us."

Trudell's dogged pursuit toward solving these mysteries continued throughout his illness. He worked from his laptop while at the hospital undergoing chemotherapy and a bone marrow transplant, Bertaccini said.

"His contributions continued to flow as we just had spoken and set up calculations during the week before he died," Bertaccini said.

Trudell co-authored more than 170 papers. He was a member of Stanford Bio-X and the Wu Tsai Neurosciences Institute at Stanford.

In addition to Lin, Trudell is survived by his brother Ronald Trudell and his sister Cher Trudell, both of Michigan.

Per his wishes, Trudell's ashes were scattered outside of the Golden Gate Bridge, on the San Francisco Bay, on an outgoing tide. Donations in his memory may be made to the Department of Anesthesiology, Perioperative and Pain Medicine at Stanford. ISM

Hypertension

continued from page 1

Pulmonary hypertension occurs when the arteries that transport blood from the heart to the lungs mysteriously thicken and become increasingly clogged, thereby weakening the heart, which has to pump extra hard to get blood to flow through the body. After diagnosis, most patients face a prognosis of just a few years of life before they die of heart failure. Some patients are born with the disease, but often it strikes in later life.

Treatment is limited to vasodilators, drugs that cause the smooth muscle cells of the diseased blood vessels in the lungs to relax, permitting more blood to flow through. These drugs help to extend survival and relieve some symptoms, but they are not a cure. Thus, scientists have been searching for other therapies.

Past research has shown that the majority of patients with the inherited form of pulmonary hypertension, which is also the most lethal, carry a mutation in the gene BMPR2. Whether the mutation plays a role in causing the disease has been unclear. Surprisingly, 80% of people with the mutation don't get the disease and remain perfectly healthy, Nicolls said.

> "It's a kind of one-two punch."

A hypothesis

Based on previous research into inflammation in the lungs, the Stanford researchers hypothesized that an inflammation-producing pathway may provide the second "hit" that triggers the mutation to cause the disease in certain patients. To test the theory, the researchers developed a rat model with a mutation in the BMPR2 gene. They followed the rats for a year, and found that the animals remained healthy. Yet when the rats were injected with a virus carrying the 5LO enzyme that triggered temporary lung inflammation, they developed pulmonary hypertension.

"At first, the rats with this mutation were healthy, running around the cage," Tian said. "Then we administered the virus into their lungs, which stimulated the production of inflammation in the vessels of the lung, and they got really sick."

The lung inflammation caused by the virus usually lasts only a few weeks and, in humans, can also be caused by environmental triggers, such as a severe flu or bacterial infection or even hiking to high altitudes. However, in the genetically susceptible rats, the virus led to permanent inflammation, damaging the lung vessels and causing a lethal form of pulmonary hypertension.

"Asthma, a bad flu, temporary types of lung injury from bacterial or virus infections — all can be 5LO-mediated," Tian said. "This type of inflammation normally has a pretty short life span. But in these rats, even after the injected virus died, the damage to the endothelial cells in the lining of the blood vessels continued. The cells become the bad player, and they continued to proliferate the inflammation."

STEVE FISCH

Mark Nicolls is the senior author of a study that found that a simple viral infection in the lungs of rats can become a lethal form of pulmonary hypertension if a common mutation is present.

These results indicate that limiting potential environmental causes of lung inflammation in patients with a genetic risk for pulmonary hypertension may help prevent the development of the disease, the study said.

Other Stanford co-authors of the study are Xinguo Jiang, MD, PhD, project leader; Yon Sung, MD, clinical assistant professor of pulmonary and critical care medicine; medical student Ting-Hsuan Wu; Peter Kao, MD, PhD, associate professor of pulmonary and critical care medicine; research scientists Aiqin Cao, PhD, and Lingli Wang, MD; research assistant Patrick Zhang; former postdoctoral scholar James Chappell, PhD; Shravani Pasupneti, MD, instructor of medicine; research assistants Petra Dahms, Allen Tu, Eric Shuffle and Yesl Kim; biostatician Peter Maguire; Hassan Chaib, PhD, director of laboratory operations; Roham Zamanian, MD, associate professor of medicine; Michael Snyder, MD, professor and chair of genetics; and Marlene Rabinovitch, MD, professor of pediatric cardiology.

Researchers at the University of Michigan, Virginia Commonwealth University, the Université Paris-Sud and the Université Paris-Saclay also contributed to the study.

This work was funded by the National Institutes of Health and the Vera Moulton Wall Center.

Nicolls and Tian are co-inventors on a patent titled "Treatment of Pulmonary Hypertension with Leukotriene Inhibitors."

Nicolls is a member of the Stanford Cardiovascular Institute.

The Stanford Department of Medicine also supported the work. ISM

OBITUARY

Stanley Schrier, founding member of hematology division, dies

By Tracie White

Stanley Schrier, MD, a founding member of the Division of Hematology at the Stanford University School of Medicine whose research advanced the field of red blood cell biology, died Aug. 16. He was 90 years old.

Schrier continued to treat patients and conduct research until just two months before his death, said Ravi Majeti, MD, PhD, professor of medicine and chief of the hematology division.

Stanley Schrier

"He had a full clinic up until July," Majeti said. "He was beyond a legend. It's so sad to think he's gone."

Schrier was known not only for his research skills, but as a compassionate, caring physician to thousands of patients during his 60 years at Stanford and for his talent for teaching and training physicians and scientists.

"Dr. Schrier's contributions to the field of hematology and to Stanford Medicine are immeasurable," said Lloyd Minor, MD, dean of the School of Medicine. "Not only was he instrumental in developing Stanford Medicine's hematology division, but he also served as a trusted physician, innovative researcher, and committed mentor. We will miss Dr. Schrier's unique and contagious passion that inspired so many."

Schrier joined Stanford as an instructor in 1959, the same year the school moved from San Francisco to the Palo Alto campus. He became a professor of medicine in 1972, then an active emeritus professor in 1999.

"Stan Schrier was a remarkable figure in the Department of Medicine," said Robert Harrington, MD, chair of the department, which houses the hematology division. "He was considered a 'doctor's doctor,' as well as an exceptional investigator, educator, mentor and leader."

Early appetite for science

Schrier grew up in the Bronx, New York, where as a child his mother recognized his love for science and encouraged him to attend the Bronx High School of Science, where he was admitted in 1943, said his wife, Barbara Klein.

"His parents were immigrants from Hungary," Klein said. "They were poor but rich in spirit. His mother saw she had a really bright son."

As an undergraduate, he transferred from New York University to the University of Colorado-Boulder after spending a summer with relatives in Denver and falling in love with the mountains, she said.

Schrier graduated from Johns Hopkins School of Medicine in 1954, then attended the University of Michigan and the University of Chicago for additional training. He served in the military as a member of the Commissioned Corps of the U.S. Public Health Service, conducting research into treatments for Korean vivax malaria. The malaria project sparked Schrier's interest in studying blood, his wife said.

When he arrived at Stanford, he was one of just four members of the division of hematology. In 1968, he became division chief, a position he held for 27 years, mentoring generations of hematologists.

"He was larger than life, a maestro of hematology," said Ranjana Advani, MD, professor of medicine and the Saul A. Rosenberg, MD, Professor in Lymphoma, who worked with Schrier for 30 years, first as a fellow and later a colleague. "He shaped my career from day one and continued to be a mentor till the very end. He was like a father figure. He's going to live in my heart forever."

Schrier's research focused on the study of the biology of red blood cells. In 1982, he began a more than 20-year study of thalassemia, a hereditary blood disease widespread in Mediterranean, African and Asian countries and among the most common genetic diseases in the world.

He conducted research projects in Israel, Italy and Thailand — countries with high rates of thalassemia — and was involved with the American Society of Hematology's volunteer outreach programs in Uganda, Peru and Cambodia. He also served as president of the American Society of Hematology in 2004 and helped initiate the organization's doctor volunteer programs to improve care for various hematological disorders in developing countries. He was editor of UpToDate, an online medical information service for physicians, and he had research grants to study anemia in elderly people when he died.

Sharing the patient's journey

It was his passion for his work that spilled over into his teaching style and drew many into the field of hematology, said Jason Gotlib, MD, professor of medicine at Stanford.

"He made me fall in love with hematology as a medical student," Gotlib said. "He was able to translate blood smears at the microscope into what patients were experiencing at the bedside. He had a boyish sense of wonder that never faded."

He also knew how to entertain his students, using his wit and storytelling skills to teach both a love of research and how to show compassion for patients, Gotlib said.

"He liked to sit down with his patients, not stand, and maintain eye contact, maybe hold their hands," Gotlib said. "He taught us it's OK to shed a tear as long as you show you're going to be there with your patient through their journey. It's hard for me to believe that future fellows won't have the benefit of interacting with Stan. However, we can sustain his legacy by nurturing what he imbued in all of us — excellence in patient care, teaching and scholarship."

Schrier received numerous teaching awards, including the Albion Walter Hewlett Award from the School of Medicine and the Walter J. Gores Award from Stanford University.

In his spare time, he enjoyed making wine with his son, David, and traveling with his first wife, Peggy Schrier, who died in 2001, and their children, and later with Klein and their grandchildren. He rode his bicycle to work at Stanford until he was in his 80s.

In addition to Klein, he is survived by his two daughters, Rachel Schrier of San Diego, and Leslie Schrier of Foster City, California; his son, David Schrier of San Carlos, California; and two grandchildren, Andres and Emilia.

Private graveside services were held Aug. 19 in Colma. A memorial event is being planned. ISM

Bacteria

continued from page 1

L. Horton Professorship, is the senior author. Lead authorship is shared by Stanford postdoctoral scholars Thomas Hagan, PhD, and Mario Cortese, PhD; and Nadine Rouphael, MD, PhD, associate professor of medicine and infectious disease at Emory University.

Inspired by a mouse study

The idea that the trillions of bacteria inhabiting the human gut play a role in our health is far from new, but it hasn't been rigorously proved. Hard data in humans has been sparse, with causal evidence coming mainly from studies in mice.

The new study was inspired by a mouse study that Pulendran and his colleagues conducted in 2011. Those investigators found that mice raised from birth to have germ-free intestinal tracts failed to mount as strong an immune response to vaccination as their normal counterparts. So did mice given antibiotics or bioengineered to lack an immune sensor for flagellin, the chief protein constituent of the threadlike flipper that bacteria use for swimming around.

"The question was, does this have any relevance to humans?" Pulendran said. To try to answer this question, he and his associates conducted a study involving 22 adults ages 18 to 45: During the 2014-15 flu season, 11 took broad-spectrum antibiotics over five days and got a flu vaccine on day four; 11 others took no antibiotics but got the flu vaccine on day four, as well.

The antibiotics lowered the gut-bacterial population by 10,000-fold. The resulting loss of overall diversity was detectable for up to one year after the antibiotics were taken. Still, 30 days after vaccination, vaccine-induced increases in antibodies capable of preventing influenza infection were comparable among the two groups.

But the participants in this experiment tended to have pretty high levels of those antibodies to begin with, suggesting they'd already had some exposure to the flu strains represented in the current or prior seasons' vaccines.

To see if low counts of gut bacteria might pose a greater obstacle to the immune system's ability to respond to previously unseen elements in a vaccine — such as new viral strains represented in the seasonal flu vaccine — than to those the immune system remembered seeing before, Pulendran's team recruited another 11 similarly aged participants for the 2015-16 season. But this time, they selected only individuals whose low level of flu antibodies indicated low prior exposure to the virus or to the vaccine itself. None of the new recruits had gotten flu vaccinations for at least the past three years. Five individuals got broad-spectrum antibiotics, as in the previous year. The other six served as controls. All 11 got vaccinated.

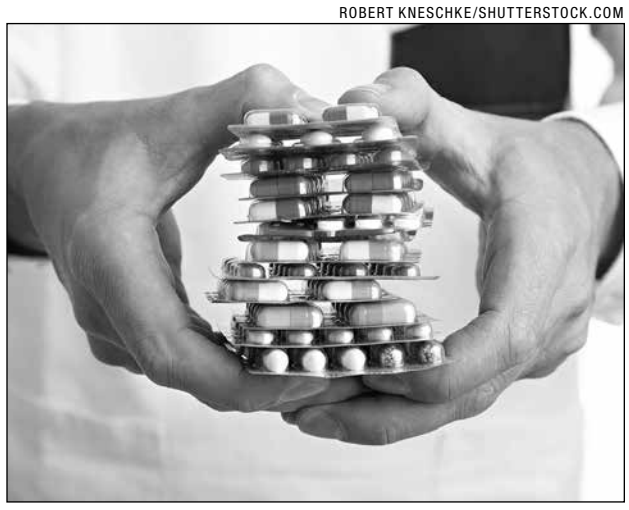
Again, gut-bacteria counts in individuals who received antibiotics plummeted, as in the previous year. But this time there was a big change in levels of an antibody subtype most responsible for countering the influenza virus: This subtype failed to burgeon in the blood in response to the vaccine. Notably, the deficit in this antibody subtype correlated strongly with post-antibiotics decreases in total gut bacteria as well as in flagellin, the bacterial protein, in volunteers' stool samples — a proxy for microbial abundance in the gut.

Signs of inflammation

The recipients of the antibiotics exhibited many signs of systemic inflammation — the same immunological signature Pulendran has observed among people ages 65 and older after an influenza vaccination in a prior study. The degree to which antibiotics recipients' immune systems exhibited this bodywide, aging-associated systemic inflammation mirrored the extent of depletion, in participants' blood, of a series of metabolites whose generation requires gut-bacterial assistance. These metabolites, called secondary bile acids, are known to dial down inflammatory processes in the immune system. Intestinal bacteria fashion them from primary bile acids initially produced in the liver. Levels of one important secondary bile acid, lithocholic acid, plunged by 1,000-fold in the bloodstreams of antibiotic recipients, and was inversely correlated with the amount of inflammation.

"The study indicates that when it comes to responding to vaccination against a previously encountered infectious pathogen, our immune systems are remarkably resilient even in the face of the most severe depletion of our intestinal bacteria," Pulendran said. "But they seem to lose this resilience when confronted with a vaccine containing new pathogenic elements of which they have little or no prior memory."

ROBERT KNESCHKE/SHUTTERSTOCK.COM



Decimating levels of intestinal bacteria with antibiotics reduced the immune system's responsiveness to a seasonal influenza vaccination, a study found.

The findings, Pulendran said, imply that when next season's flu strain comes along, you want your gut-resident microbes to be in full bloom in order for your immune system to rise to the occasion. Pulendran offered some advice. "Get your annual flu shot," he said. "The greater your inventory of immune memory to influenza strains bearing any resemblance to the one that's coming over the hill, the more likely you'll be able to deal with it, even if your gut microbes are in short supply."

Pulendran is a member of the Stanford Institute for Immunity, Transplantation and Infection and a faculty fellow of Stanford ChEM-H.

Other investigators at Emory University, as well as researchers at the Ragon Institute, the University of Chicago, Georgia State University and the Food and Drug Administration contributed to the work.

The work was funded by the National Institutes of Health, the Soffer Endowment and the Violetta Horton Endowment.

Stanford's departments of Pathology and of Microbiology and Immunology also supported the work. ISM

Melissa Bondy named chair of Epidemiology and Population Health Dept.

Melissa Bondy, PhD, has been appointed chair of the Department of Epidemiology and Population Health, formerly known as the Department of Health Research and Policy.

The appointment takes effect Oct. 1.

Bondy, who comes to Stanford from Baylor College of Medicine, also will serve in the newly created role of associate director for population sciences at the Stanford Cancer Institute. In that role, she will spearhead the research enterprise of the institute's population sciences program, which is designed to reduce the burden of cancer and improve outcomes for patients with cancer.

"Dr. Bondy is a renowned cancer epidemiologist whose leadership will elevate Stanford Medicine's multidisciplinary efforts to improve the health of individuals and populations through the study of the distribution, determinants and control of illness and impairment," said Lloyd Minor, MD, dean of the School of Medicine. "We're thrilled that she has joined our faculty. Her career has exemplified the core tenets of precision health."

Bondy earned a PhD in epidemiology at the University of Texas School of Public Health. Before coming to Stanford, she spent nearly two decades at Baylor College of Medicine. In 2001, she became director of the Childhood Cancer Epidemiology and Prevention Center, a joint center of Baylor College of Medicine, MD Anderson Cancer Center and Texas Children's Hospital. Ten years later, she took on a new role as the associate director of Cancer Prevention and Population Sciences at Baylor College of Medicine's Dan L. Duncan Cancer Center, where she focused much of her research on understanding heredity patterns and genetic susceptibilities in brain and breast cancer.


Melissa Bondy

"I'm excited to continue my work at Stanford, and to further develop and expand this new department," Bondy said. "Part of that will be building an outstanding educational program and increasing the number of doctoral and postdoctoral students that we bring in."

Bondy serves on the National Cancer Institute's board of scientific advisers and is a member of the external advisory board for several NCI-designated cancer centers. Last year, she received the visiting scholar award from the NCI Division of Cancer Epidemiology and Genetics.

"My goal will be to try to find ways to genuinely influence and improve the way we evaluate and inform health at a population level," Bondy said. "To do that, we will harness integrative epidemiology, with a focus on genomics and digital approaches to monitor population health. I also look forward to collaborating not only with those in the department, but with researchers from across the School of Medicine and around the campus." ISM

OF NOTE

reports on significant honors and awards for faculty, staff and students


Christopher Almond


Manual Amieva


Leah Backhus


Alistair Boettiger


Leslie Mateo

CHRISTOPHER ALMOND, MD, was promoted to professor of pediatrics, effective May 1. He directs clinical research in the Pediatric Advanced Care Therapies Program at Lucile Packard Children's Hospital Stanford. His primary research interests are in multicenter clinical trials and learning health networks in pediatric heart failure, ventricular assist device support and cardiac transplantation.

MANUEL AMIEVA, MD, PhD, was promoted to professor of pediatrics and of microbiology and immunology, effective July 1. He specializes in treating pediatric infectious diseases. His research focuses on understanding how bacteria colonize our skin and gastrointestinal epithelium, and how that can lead to infection and disease.

LEAH BACKHUS, MD, associate professor of cardiothoracic surgery, received the Dr. Dwight C. McGoon Award from the Thoracic Surgery Residents Association for her commitment to teaching and mentoring. She also received the Levi Watkins Innovation and Leadership Development Scholarship from the Thoracic Surgery Foundation. The Watkins award includes $5,000 to cover travel expenses incurred for the purpose of learning new thoracic surgery techniques.

ALISTAIR BOETTIGER, PhD, assistant professor of developmental biology, and **LESLIE MATEO**, a graduate student in developmental biology, were awarded a Gilliam Fellowship for Advanced Study. They will receive $50,000 a year for up to three years to promote inclusivity in laboratories and increase diversity in the life sciences.

CATHERINE CURTIN, MD, was promoted to professor of plastic and reconstructive surgery, effective July 1. She specializes in peripheral nerve surgery and upper limb reconstruction. Her research focuses on reducing pain and improving function for people with peripheral nerve injury, and improving upper limb function for people with spinal cord injury.

LISA GIOCOMO, PhD, assistant professor of neurobiology, received a scholars award from the Vallee Foundation. The $300,000 grant is for basic biomedical research. Giocomo researches the neural mechanisms of cognition, specifically navigation.

JEREMY HEIT, MD, PhD, was appointed assistant professor of radiology, effective June 1. His research uses neuroimaging techniques to study diseases affecting the blood vessels and blood supply to the brain, with the goal of developing new minimally invasive treatments for these diseases.

MARIE HOLLENHORST, MD, PhD, clinical instructor of pathology, was awarded an early-career scientist grant from the National Blood Foundation. The two-year, $75,000 grant will support her research to understand how the sugar molecule CMP-Neu5Ac is made and how it regulates platelet number and function, with the goal of illuminating fundamental aspects of platelet biology and developing effective therapies for bleeding and clotting disorders.

K.C. HUANG, PhD, was promoted to professor of bioengineering and of microbiology and immunology, effective June 1. His research investigates the physical mechanisms of cell growth and the ecological principles underlying microbial community assembly and function.

ELIZABETH KIDD, MD, was promoted to associate professor of radiation oncology, effective Aug. 1. Her clinical focus is treating gynecologic cancer and performing brachytherapy. Her research aims to optimize and individualize treatment for gynecologic cancers to improve disease control and quality of life for patients.

SHIVAANI KUMMAR, MD, professor of oncology and radiology, was awarded the 2019 David R. Gandara Lectureship on Developmental Therapeutics from the UC-Davis Comprehensive Cancer Center. The award is for visionary leadership in developmental therapeutics for cancer.

ROBBIE MAJZNER, MD, was appointed assistant professor of pediatrics, effective June 1. His research focuses on optimizing chimeric antigen receptor T cell therapies to treat neuroblastoma, sarcomas and brain tumors, particularly for pediatric cancer patients.

PHILIP PIZZO, MD, the David and Susan Heckerman Professor and professor of microbiology and immunology, received the New York Academy of Medicine's John Stearns Medal for Distinguished Contributions in Clinical Practice. The award is for extraordinary, career-long contributions to the clinical practice of medicine.

ELSIE ROSS, MD, assistant professor of surgery, was awarded a K01 grant from the National Institutes of Health. The five-year, $162,000-per-year grant will support her study of artificial intelligence to enable early identification and treatment of peripheral artery disease.

BIRGITT SCHUELE, MD, associate professor of pathology, was awarded a $100,000 grant from the Neukermans Family Fund. The grant supports her research into neuronal microcircuits in Parkinson's disease.

SHARON SHA, MD, associate professor of neurology and neurological sciences, was appointed to the Alzheimer's Prevention and Preparedness Task Force for the State of California. The task force presents recommendations to the governor on how local communities, private organizations, businesses, government and families can prepare for the rise in the number of cases of Alzheimer's disease.

KATHERINE TRAVIS, PhD, was appointed assistant professor (research) of pediatrics, effective July 1. Her research uses advanced neuroimaging techniques to examine how white matter structures of the brain contribute to reading and language skills in children, with the goal of identifying and developing interventions for children who are at greatest risk of learning difficulties. ISM


Catherine Curtin


Lisa Giocomo


Jeremy Heit


Marie Hollenhorst


K.C. Huang


Elizabeth Kidd

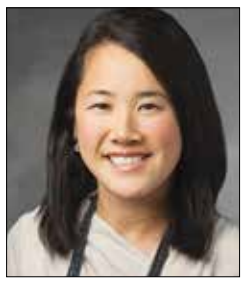

Shivaani Kummar


Robbie Majzner


Philip Pizzo


Elsie Ross


Birgitt Schuele


Sharon Sha


Katherine Travis