A complete match

In his remarks before the reveal, Gesundheit noted some statistics that students would soon learn for themselves. “We have a complete match today,” Gesundheit said, meaning that all of the students had been matched to a residency program. “This only happens about once in a decade here at Stanford. So we’re very proud.” It was especially impressive, Gesundheit noted, because this year’s match had a lot of people going into competitive subspecialties.

A paper describing the work was published online March 14 in Circulation. Wu, who is the Simon H. Sterrett, MD, Professor, is the senior author. Former research fellow Tomoya Kitani, MD, now a clinical assistant professor at the Kyoto Prefecture University of Medicine in Japan, is the lead author. The investigators recruited study participants from among patients at Stanford Hospital who had experienced a stroke, Buckwalter said. In his remarks before the reveal, Gesundheit noted some statistics that students would soon learn for themselves. “We have a complete match today,” Gesundheit said, meaning that all of the students had been matched to a residency program. “This only happens about once in a decade here at Stanford. So we’re very proud.” It was especially impressive, Gesundheit noted, because this year’s match had a lot of people going into competitive subspecialties.

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Heart research shows abilities of wearables to detect atrial fibrillation

Researchers from the School of Medicine presented preliminary results of the Apple Heart Study, an unprecedented virtual study with over 400,000 enrolled participants, on March 16. The researchers reported that wearable technology can safely identify heart rate irregularities that subsequent testing confirmed to be atrial fibrillation, a leading cause of stroke and hospitalization in the United States.

The study was launched with sponsorship by Apple Inc. in November 2017 to determine whether a mobile app that uses data from a heart-rate pulse sensor on the Apple Watch can identify atrial fibrillation. The condition often remains hidden because many people don’t experience symptoms.

Key findings from the study include:

- **Overall**, only 0.5 percent of participants reported receiving regular pulse notifications, an important finding given concerns about potential over-notification.
- **Comparisons** between regular pulse-detection on Apple Watch and simultaneous electrocardiography patch recordings showed that the pulse detection algorithm (indicating a positive tachogram reading) has a 71 percent positive predictive value, which is comparable with that of in-person electrocardiogram readings. Among the participants, who received irregular pulse notifications, were found to be in atrial fibrillation at the time of the notification.
- **One-third** (34 percent) of the participants had an atrial fibrillation notification and followed up by using an ECG patch over a week later were found to have atrial fibrillation. Since atrial fibrillation is an intermittent condition, it’s not surprising for it to go undetected in subsequent ECG patch monitoring.
- **Fifty-seven** of those who received irregular pulse notifications sought medical attention.

The results of the Apple Heart Study highlight the potential role that innovative digital technology can play in creating more predictive and preventive health care, Lloyd Minor, MD, dean of the School of Medicine. "Atrial fibrillation is just the beginning, as this study opens the door to further research into wearable technologies and how they might be used to prevent disease before it strikes — a key goal of precision health.

For the study, each participant was required to have an Apple Watch (series 1, 2 or 3) and an iPhone. The most recent Apple Watch, which features a Holter-style ECG, wasn’t part of the study, as it was released after the study’s launch. The Apple Heart Study app intermittently checked the heart-rate pulse sensor for four features of an atrial fibrillation notification, with an irregular pulse was detected, the participant received a notification and was asked to schedule a telemedicine consultation with a doctor involved in the study through American Well. Participants were then sent ambulatory ECG patches through BioFelemetry, which recorded the electrical rhythm of their hearts for up to a week.

The Stanford principal investigators were Mintu Turiakh, MD, associate professor of cardiovascular medicine, and Marco Perez, MD, associate professor of cardiovascular medicine, and the study chair was Kenneth Mahaffey, MD, professor of cardiovascular medicine. "This study’s findings have the potential to help patients and clinicians understand how devices like the Apple Watch can play a role in detecting conditions such as atrial fibrillation, a deadly and often undiagnosed disease," said Turiakh. "The virtual design of this study provides a strong foundation upon which future research can be conducted to explore the health implications of wearable technology."

"The performance and accuracy we observed in this study provide important information as we seek to understand the potential impact of wearable technology on the health system," said Perez. "Further research will help people make more informed health-care choices.

Researchers from the Lankenau Heart Institute, Jefferson Medical College, the University of Vermont and the University of Colorado School of Medicine, Cooper Medical School of Rowan University, StopABiP.org, the American Foundation of Atrial Fibrillation, and Duke University also contributed to the study. The Apple Heart Study was funded by Apple Inc.
Molecular data can predict cancer recurrence, scientists say

By Krista Conger

Molecular data obtained from breast cancer cells can be used to predict which patients are at a high risk for recurrence even after seemingly successful treatment, according to a new study jointly conducted by researchers at the School of Medicine and of genetics at Stanford and co-director of the Cancer Research Unit and professor of medicine and of genetics at Stanford. Curtis is also supported by the work of researchers at Harvard Medical School, who was not involved in the research. "This important scientific paper identifies molecular features that define the type of breast cancer recurrence. In the future, this type of genomic classification should help us separate patients that respond robustly to treatment from those that do not," said Harold Burstein, MD, PhD, co-founder of the Breast Care Program at Harvard Medical School, who was not involved in the research. "Our model uniquely accounts for the chronology of a patient's disease and is based on a genome-driven classification scheme that can inform personalized therapeutic approaches," Curtis said.

"We've shown that the molecular nature of a woman's breast cancer determines how their disease could progress, not just for the first five years, but also later, even if it comes back," Rueda said. "We hope that our research tool can be turned into a test doctors can easily use to guide treatment recommendations." The work is an example of Stanford Medicine’s focus on precision health, the goal of which is to anticipate and prevent disease in the healthy and precisely diagnose and treat disease in the ill. Curtis is a member of the Stanford Cancer Institute and Stanford Bio-X. Other Stanford co-authors of the study are instructors of medicine José Seoane, PhD, and Jennifer Caswell-Jin, MD, researchers from the British Columbia Lung Cancer Research Center; the Stanford Research Institute in Oncology and Hematology in Winnipeg, Canada; the University of Nottingham; King's College London; and the University of Valldolid in Spain also contributed to the work. The research was supported by Cancer Research UK, the Experimental Cancer Medicine Center, the National Institute of Health Research in the United Kingdom, the Breast Cancer Research Foundation, the American Association for Cancer Research and an NIH Director's Pioneer Award. A patent application has been filed on aspects of the research findings by Curtis, Caldas, Rueda and Seoane.

Curtis and her colleagues found that they could predict the course of the disease at different points during a patient's lifetime. The researchers were also able to identify a subgroup of women with triple-negative breast cancers — considered to be an aggressive and more difficult form of the disease to treat — who are unlikely to see their cancers recur after five years. Distinct patterns of metastasis

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mocarity globally from 1995 to 2015. They also found improvements in other health burdens in the countries with higher democracy. For example, found that true life expectancy in 2015 in China was lower than its expected life expectancy at birth in 1980 to 2000 and has only improved by 3.6 years in the past decade with increased government health spending. In Cuba, the degree to which its observed life expectancy exceeds the expected life expectancy has decreased from four-to-seven years higher than expected in 1970 to three-to-five years higher than expected in 2016. "It is widely accepted that democracy plays in child health and infectious diseases may not be generalizable to the diseases that disproportionately affect adults." Bolivia, said, are largely costly, to treat than most infectious diseases, and require more health care infrastructure and skilled medical personnel. The researchers hypothesize that democracy improves health care system in the long term. • When enforced through regular, free and fair elections, democracies should have a greater incentive than autocracies to provide health-promoting resources and services to a larger proportion of the population. • Democracies are more open to feedback from a broader range of interest groups, more protective of patient privacy, whether we can predictably improve the outcomes of these patient subgroups at high risk of recurrence with new therapies that target their specific genomic drivers. Curtis and her colleagues are currently planning clinical trials to do just that. They also developed a web-based research tool that may ultimately help clinicians more accurately predict an individual's risk of relapse and guide treatment decisions. "We've shown that the molecular nature of a woman's breast cancer determines how their disease could progress, not just for the first five years, but also later, even if it comes back," Rueda said. "We hope that our research tool can be turned into a test doctors can easily use to guide treatment recommendations." The work is an example of Stanford Medicine’s focus on precision health, the goal of which is to anticipate and prevent disease in the healthy and precisely diagnose and treat disease in the ill. Curtis is a member of the Stanford Cancer Institute and Stanford Bio-X. Other Stanford co-authors of the study are instructors of medicine José Seoane, PhD, and Jennifer Caswell-Jin, MD, researchers from the British Columbia Lung Cancer Research Center; the Stanford Research Institute in Oncology and Hematology in Winnipeg, Canada; the University of Nottingham; King's College London; and the University of Valldolid in Spain also contributed to the work. The research was supported by Cancer Research UK, the Experimental Cancer Medicine Center, the National Institute of Health Research in the United Kingdom, the Breast Cancer Research Foundation, the American Association for Cancer Research and an NIH Director's Pioneer Award. A patent application has been filed on aspects of the research findings by Curtis, Caldas, Rueda and Seoane.

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Mystery novel, dream spur key scientific insight into heart defect

By Bruce Goldman

Jim Spudich isn’t the kind of avid reader who devours a book in two days and moves immediately to the next on his nightstand. He just reads himself to sleep on evenings when he wants to retoast his brain from thinking about his work. Which is most of the time. “Creative research is not a job — it’s a full-time, almost childlike obsession driven by the curiosity that all kids have,” said Spudich, PhD, professor of biochemistry. “I see myself as a kid who’s never had to grow up.”

Yet even scientists have to sleep. Spudich, 77, ordinarily gets his fair share — close to eight hours a night. But when he needs some help winding down, a good murder mystery will do it, especially if it’s set in a mountain. “It’s not so unusual that we scientists go to bed and wonder what clue we’d been overlooking.”

One evening, in December 2014, Spudich’s wife, Anna, who knows his tastes well, slipped him some bedtime reading, a murder mystery. Spudich, 40 percent of all known HCM-inducing mutations are in cardiac myosin, making it an obvious candidate for intense scrutiny. But until about a dozen years ago, nobody could study the effects of HCM mutations, Spudich said, because the biotechnology for making significant amounts of it that would work didn’t exist yet.

Then one of his frequent collaborators at the University of Colorado figured out a way to pull it off. “We usually think of mutations as causing a protein not to work as well as the unmutated version does; they’re messing with what was a beautiful evolutionary design,” he said. “But HCM mutations are like what you’d get if you sawed the top off a mesa that provided an insight about what may be at the root of the disorder.”

JIM SPUDICH

Spudich dropped everything else he’d been doing and began systematically characterizing the effects of HCM-inducing mutations on the function of cardiac myosin molecules, asking: Why would so many diverse mutations be in cardiac myosin, making it an obvious candidate for intense scrutiny? But until about a dozen years ago, nobody could study the effects of HCM mutations, Spudich said, because the biotechnology for making significant amounts of it that would work didn’t exist yet.

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COURTESY OF JIM SPUDICH

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Top left) Jim Spudich studies how mutations in cardiac myosin could play a role in a deadly heart disorder. (Below left) An illustration of a myosin molecule in an “open” and “folded” position. (Above) An avid pilot, Spudich, shown here in the mid-1990s, has flown over many mesas. He has a dream about a mesa that provided an insight about what may be at the root of the disorder. (Top left) Jim Spudich studies how mutations in cardiac myosin could play a role in a deadly heart disorder. (Below left) An illustration of a myosin molecule in an “open” and “folded” position. (Above) An avid pilot, Spudich, shown here in the mid-1990s, has flown over many mesas. He has a dream about a mesa that provided an insight about what may be at the root of the disorder. (Top left) Jim Spudich studies how mutations in cardiac myosin could play a role in a deadly heart disorder. (Below left) An illustration of a myosin molecule in an “open” and “folded” position. (Above) An avid pilot, Spudich, shown here in the mid-1990s, has flown over many mesas. He has a dream about a mesa that provided an insight about what may be at the root of the disorder.

MARCH 25, 2019 INSIDE STANFORD MEDICINE
Modified immune cells issue alert when cancer detected

By Hanae Armitage

Immune cells imbued with the power to detect and reveal tumors could be a new method of diagnosing cancer, according to a study from the School of Medicine.

The research, performed in mice, involved modifying a specific type of immune cell to patrol the body for cancer and send a signal through blood or urine when they found trouble.

"In the early cancer diagnostics for years, but this time, we came at it from another angle," said Sanjay "Sam" Gambhir, MD, PhD, professor and Integrated Diagnostics Center at Stanford for Cancer Medicine.

"We said, if nature doesn't give you sufficient signal that cancer is present, can we force the body to make one? In this case, can we force immune cells to emit detectable markers if cancer exists somewhere in the body?"

The question prompted the engineerings of a sort of immune cell turned-informant, which Gambhir said is possibly the first in-animal example of a phenomenon called "immunodiagnosis." Like immunotherapy, immunodiagnostic repurposes the body's own cells to perform histochemical testing for the presence of disease or damaged cells.

Currently, the technique can detect tumors as small as 4 millimeters in diameter or 0.03 millimeters in length -- which outperforms some of the most advanced early tumor detection methods currently available, said Gambhir, who is also the director of the Precision Health and Integrated Diagnostics Center at Stanford for Cancer Medicine.

He said the immune cell signals are flagging a specific class of malfunctioning cells that other methods are not limited to them. The technique also could be tailored to detect more than just cancer, and could potentially flag other disorders or malfunctions, such as diabetes and autoimmune disorders, such as multiple sclerosis or cancer, and could potentially flag other disorders that are not limited to them. The technique also could be tailored to detect more than just cancer, and could potentially flag other disorders or malfunctions, such as diabetes and autoimmune disorders.

The technique then became one of determining which marker mammalian for detecting a possible tumor environment.

And that's where Gambhir’s team came up with a solution.

The team played to the macrophage's advantage.

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"The molecular marker is called Gaussia luciferase, and under certain chemical circumstances, it glows," said Gambhir. "So the idea is, we pick a gene that turns on when a macrophage senses a tumor cell, we link that gene's promoter to Gaussia luciferase and finally, we integrate it into the macrophages.

These re-engineered immune cells can roam the body in their usual man- ner, only this time, if they find a dam-aged cell, they tell you. Gambhir and his team tested the refined macrophages in mice and were able to detect mouse models of breast cancer from a vial of blood or urine when the tumor was just 4 millimeters in diameter.

At this nascent stage of the research, the team is noting the diagnostic outperformed other cancer detection methods: Because the tumor is so small, it's not just producing enough circulating DNA to reliably show up in blood; and a PET scan, which is a standard, image-based cancer detection method, is only sensitive enough to find tumors 8 millimeters in diameter.

To see if macropathogens have staked a claim to a portion of cells, he and one of the lab's other members, and a special molecule to the blood or urine sample that reacts to the presence of Gaussia luciferase, a fluorescent fluid.

Fluorescing fluid means the modified macropathogen has activated its tumor-associating role.

Refining the diagnosis

Although a fluorescing via likely reveals the presence of cancer, it's not a definitive diagnosis at this point in the development of the technique, Gambhir said. Macropathogens activate certain sets of genes that can correspond to a handful of off-kilter cells, such as a tumor or a skin cancer, he said. Once the macropathogen's signal, Gambhir said, doctors would use one of several tests to deter-mine whether it's reacting to a cancerous target.

He added that the engineering trick done with Gaussia luciferase is modular and can be added to any detectable reporter, such as a PET scan reporter or tracer. That way, a tumor-activated macrophage can act like a fluorescent PET scan, making it easy for doctors to see cancer cells.

"In the immediate future, Gambhir said that he plans to continue to test the detection method in other types of can-cers and animal models, likewise refining the method to home in on tumors only, rather than cells with other types of damage.

Dream

continued from page 4

It was 5:30 a.m. Aflame with inspiration, Spudich jumped out of bed and beelined to his computer, a new hypothesis for his group of researchers rapidly forming.

Protein molecules are born as linear sequences of chemical building blocks called amino acids, but that one-dimensionality doesn't last long. Almost as soon as a protein forms, it 'folds up' with a solution.

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Protein molecules are born as linear sequences of chemical building blocks called amino acids, but that one-dimensionality doesn't last long. Almost as soon as a protein forms, it 'folds up' with a solution.

What Spudich and others had been overlooking was the head so it can't grab the actin filament.

Looking at the folded cardiac-myosin molecule on the computer screen, Spudich realized that by weakening the overall attraction between a myosin molecule's head and tail, HCM-inducing mutations were freeing up myosin heads to grab onto a neighboring actin filament, increasing the number of myosin heads actually available to do their work and put more heads in play. A 2018 Proceedings of the National Academy of Sciences paper co-authored by Spudich suggested that a drug called mavacamten, now in phase-3 clinical trials for HCM, may be successful in reversing the hypercontractility induced by a wide range of HCM-inducing mutations on the myosin molecule.

All patients in two earlier trials of mavacamten showed significant improvement. These trials have been sponsored by a South San Francisco-based biotechnology company, MyoKardia Inc., that Spudich co-founded in 1998 to speed the translation of his findings from the lab to the clinic in the search to treat HCM. Mavacamten was the fruit of this discovery effort.

"As the drug is pushing available heads into the unavailable state — the opposite of what I believe most of these mutations are doing," Spudich said.

"The principle, this approach might apply to HCM-causing mutations.'"
Stroke continued from page 1

at two weeks, one month, three months and one year after their strokes. Using mass cytometry, the researchers assessed the properties and activation states of the 10 million or so individual immune cells obtained in each blood draw.

Measuring cognitive ability

At days 3, 30, 90 and 365 after their strokes, participants also took the Montreal Cognitive Assessment, or MoCA. The test measures several aspects of cognition, including spatial knowledge, memory and ability to calculate. It is designed to detect cognitive deficiencies. In particular, the scientists compared each participant’s MoCA performance on day 90 with the participant’s performance on day 365. “Nearly one-third of the participants’ MoCA scores showed declines between three months and one year out from their stroke,” Buckwalter said. Each participant’s mass cytometry results and MoCA scores were fed into an algorithm developed in Aghaeepour’s laboratory. This algorithm was specifically designed for its ability to reduce massively complicated collections of highly correlated data to meaningful data sets.

The investigators identified three distinct phases — recognizable at post-stroke days 2, 5 and 90 — through which the immune system progresses before returning to normal about a year after a stroke. Each phase is marked by its own unique set of deviations in relative numbers and activation levels of a small number of immune cell types in the blood.

It was the first, or acute, phase, peaking just two days after the stroke, that grabbed the researchers’ attention. The more exaggerated deviations among the hallmark of immune cells identified as defining that phase, the more likely a patient was to suffer a drop in performance on the mental test between three months and one year out from the event, the study found.

The correlation, which was big enough to achieve statistical significance in this small study, was independent of age, sex, body mass index, location of the stroke or type of treatment given when the participants initially reached the hospital. Remarkably, cognitive loss was also unrelated to the initial size of the stroke.

“We didn’t know what to expect,” Aghaeepour said. “This was an exploratory study looking to understand how cells function after a stroke. We had no idea we would find a correlation with long-term cognitive outcomes. It was an exciting accident.”

Predictive power of acute phase

Intermediate- and late-phase analyses added something to the acute phase’s predictive power. The investigators hypothesized that if one doesn’t recover, whatever immune cells are disrupting a patient’s cognitive apparatus are acting within the brain itself, so their activity might not get picked up in screenings of blood circulating peripherally.

“A lot more needs to be done to differentiate the people who will go on to have post-stroke dementia from those who won’t,” Buckwalter said. But with the key cellular players predictive of long-term dementia at least tentatively identified, the job gets much simpler.

“Being able to detect something in the bloodstream would be much easier than doing a brain biopsy,” Gaudilliere said. “Mass cytometry lets you measure the entire haystack in order to find the needle. Once you have the needle, you don’t need the haystack anymore. You can focus on just a few features of a few cell types,” opening the door to clinical applications.

A study Gaudilliere conducted in 2014 with other Stanford researchers, including Martin Angot, MD, professor of anesthesiology, perioperative and pain medicine, and a co-author of the new study, used mass cytometry to detect whether differences in immune signatures of patients undergoing hip-replacement surgery, an intensive and traumatic procedure, could serve as telltale indicators of a patient’s ability to recover fully and quickly from the operation.

Gaudilliere said there were “provocative similarities” in the post-event immune trajectories of participants in the surgical and stroke trials. “In both studies, we found similar patterns among participants in the later stages of either signaling molecules in certain immune cell types,” he said. “It suggests a shared immune response between surgical and neurological injury may be implicated in the neurocognitive dysfunction seen after stroke or major traumatic injury.”

Buckwalter and study co-author Maarten Lansberg, MD, PhD, associate professor neurology and neurological sciences, are now running a scaled-up trial to try to validate the results of the small study. Buckwalter is a member of Stanford’s Wu Tsai Neuroscience Institute and of Stanford Bio-X. Gaudilliere is a member of Stanford’s Maternal & Child Health Research Institute. Aghaeepour is a member of Bio-X.

Other Stanford co-authors are former clinical research assistant Kacey Berry; former visiting medical resident Maxim Beneyto, MD; Dyani Gaudilliere, DMD, MPH, clinical assistant professor of plastic & reconstructive surgery; research scientist Edward Gavio, PhD; research data analyst Anthony Culos; postdoctoral scholar Mohammad Ghaemi, PhD; former visiting medical resident Benjamin Choisy, MD; former visiting researcher Karim Djebali, DMD; former medical student researcher Jacob Einhaus; visiting student researcher Basile Bertrand; research assistant Athena Tanida; postdoctoral scholars Natalie Stanley, PhD, and Ram Fallahzadeh, PhD; Quinten Baca, MD, PhD, clinical instructor of anesthesiology, perioperative and pain medicine; former research assistant and lab manager Lisa Quach; clinical research coordinator Elizabeth Osho; and neuroscientist Lauren Drop, PhD.

The work was funded by a Big Ideas in Neuroscience grant from the Wu Tsai Neurosciences Institute at Stanford and by the National Institutes of Health. Stanford’s departments of Anesthesiology, Perioperative and Pain Medicine, of Neurosurgery and of Neurology and Neurological Sciences also supported the work.

March 25, 2019 inside stanford medicine

(Brige Gaudilliere)

(Marion Buckwalter)

Nina Aghaeepour

(Above) Madeline Grade, who matched in emergency medicine at UC-San Francisco, with Dean Lloyd Minor. (Right) Associate Dean of MD Admissions Iris Gibbs, center, on Match Day with medical students who had learned where they would be spending their residencies.
Cancer continued from page 1

author.

Between 15 and 20 percent of breast cancer patients currently in the HER2 clinical trials, for which the most effective treatment is the chemotherapy drug trastuzumab, sold under the brand name Herceptin. But trastuzumab also causes more heart problems than other biologic agents and can lead to heart failure by interfering with the heart’s ability to pump blood, according to an analysis of more than 3,000 women enrolled in the trials. Heart problems are likely to get worse over time, as cancer cells grow and divide, making it more difficult for the heart to pump blood.

Innovative transplant surgeon Oscar Salvatierra dies at 83

By Erin Digita

Oscar Salvatierra Jr., MD, professor emeritus of surgery and pediatrics at the Stanford University School of Medicine and a leader in the effort to enact national legislation regulating organ donation, died March 16 at his home in Menlo Park, California. He was 83. The cause was complications from Parkinson’s disease, according to his wife, Charlotte Salvatierra.

A pediatric kidney transplant surgeon, Salvatierra was the physician most involved in the policy and passage of the National Organ Transplant Act of 1984, which established a national policy of using the fair and equitable allocation of donor organs to patients across the country.

The law, on which Salvatierra collaborated with then-Congressman Al Gore, also banned buying and selling donor organs. It has served as a model for laws regulating organ transplantation around the world.

It saddens me to hear of the passing of a friend and former colleague Dr. Oscar Salvatierra,” said Gore, who went on to serve as vice president to President George H. W. Bush. “Oscar’s tireless dedication to the development of the National Organ Transplant Act helped revolutionize the medical field and human development.”

Salvatierra was also a beloved clinician and prominent scientist whose research on metformin had fewer cardiac side effects, they hope to treat Type 2 diabetes. As the researchers report in the journal Diabetes, the work could help patients live longer and contracted more vigorously with AMPK activators.

Plan for retrospective study

The researchers plan to follow these findings with a retrospective study of patients who were taking metformin for diabetes while they were receiving trastuzumab. They noted that patients on metformin had fewer cardiac side effects, they hope to conduct a trial to see if giving patients metformin with trastuzumab might reduce heart problems.

Wu said testing drugs on cells in a lab can vastly reduce the time needed to bring drugs to patients, as well as reduce the cost. “This significantly will cut the cost of drug development, providing more and better drugs to patients,” he said.

The work is an example of Stanford Medicine’s focus on precision health, the goal of which is to anticipate and prevent disease in the healthy and precisely diagnose and treat disease in the ill.
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helped many other physicians develop their careers, often serving as a mentor and role model.

“When I was just beginning my surgical training, I happened to see his name listed from UCSF as their division of abdominal transplantation at Stanford. And he was a very deliberate and skillful surgeon.”

Children who need kidney transplants are quite ill, and Salvatierra became known for his ability to connect with young patients and help calm their parents’ fears.

During their years of collaboration, Esquivel learned aspects of neural circuit functioning, in particular the roles of synaptic plasticity in spinal cord circuitry, as well as the effects of pain, acute stress and drug addiction on synaptic plasticity in the brain.

“His research uses computational science to identify biomedical phenotypes with the aim of improving the mechanistic understanding of psychiatric disorders and treatment of neuropsychiatric disorders.”

Laura Roberts, MD, the Katherine Drexler McCormick and Stanley McCormick Memorial Professor and chair of psychiatry and behavioral sciences, is the 2019 recipient of the annual lifetime service award from the American Association of Directors of Psychiatric Residency Training. The award recognizes a member who has provided significant service to the association, had an impact on psychiatric residency education nationally, demonstrated excellence in psychiatric residency education, and is committed to empowering and educating the next generation of psychiatrists.

Laura Roberts

Salvatierra retired from his clinical responsibilities in 2006, becoming associate dean for medical students at the School of Medicine, a position he held until 2015. He received many major awards during his career.

Salvatierra is survived by his wife of 25 years, Pam; son Mark Salvatierra of San Jose, Calif.; daughter Lisa Rudloff of Centerport, New York; four grandchildren; five siblings; and many nieces and nephews.

A memorial service was held last week. In lieu of flowers, the family requests donations to the Dr. Oscar Salvatierra Emergency Fund, Tax ID #11-19484-100/112/T, which provides emergency funds for medical students who experience financial crises. Donations can be directed to Development Services, Stanford University, PO Box 24666, Stanford, CA, 94309-4666.