2019 Stanford - Penn Cardiovascular Symposium

The Stanford-Penn Cardiovascular Symposium will occur November 4 - 5, 2019 in Paul Berg Hall at Stanford. Keynotes will be delivered by Peter Fitzgerald, MD, PhD, and Daniel Rader, MD. The event will feature talks from Stanford and Penn faculty, speed talks from CVI postdocs, graduate students, and instructors, and a poster session. For more information on those involved see page 2. To register: https://tinyurl.com/StanfordPenn.

Robert Harrington begins tenure as 2019-2020 American Heart Association President

CVI member Robert Harrington, MD, Arthur L. Bloomfield professor of medicine and chair of the Department of Medicine at Stanford University, is now the President of the American Heart Association. His tenure began on July 1, 2019. Dr. Harrington is a global leader in acute ischemic heart disease. He is also recognized for his abilities in building local, national and international collaborations for the efficient conduct of innovative clinical research and trying to better understand and improve upon the methodology of clinical research. Dr. Harrington also as a strong commitment to training and mentorship, and has served as the principal mentor for over 20 post-doctoral clinical research fellows focused on cardiovascular research. Congratulations Dr. Harrington!

CVI welcomes 2019 summer undergraduate interns

The Stanford Cardiovascular Institute hosted 19 undergraduate students from universities across the United States for a 10-week research experience this summer. The students performed individual research projects, had the opportunity to interact with Stanford faculty both formally and informally, and took a field trip to tour a local biotech company, all before presenting their research projects in a special seminar that featured Joseph Woo, MD, as a keynote speaker. Stay tuned for a call for applications for 2020!

Cardiovascular Institute faculty searches

CVI welcomes 2019 summer undergraduate interns

The Stanford Department of Surgery, Division of Vascular Surgery and the Cardiovascular Institute seek a faculty member in the University Tenure Line (UTL) at the Assistant or Associate Professor level. To apply: https://apply.interfolio.com/62922.

The Stanford Division of Cardiovascular Medicine and the Cardiovascular Institute seek an Interventional Cardiologist to join as an Assistant Professor in the Medical Center Line (MCL). To apply, submit your CV, a letter of interest, and 3 references to Dr. Randall Vagelos: http://facultyapplication.stanford.edu/
STANFORD - PENN
Cardiovascular Research Symposium
November 4-5, 2019 | Registration: https://tinyurl.com/StanfordPenn

PARTICIPANTS

Zoltan Arany, MD, PhD
Professor of Medicine, Perelman School of Medicine, UPenn

Elan Burton, MD
Clinical Assistant Professor, Adult Cardiothoracic Surgery, Stanford Medicine

Vinicio de Jesus Perez, MD
Associate Professor of Pulmonary and Critical Care Medicine, Stanford Medicine

William Hiesinger, MD
Assistant Professor of Adult Cardiothoracic Surgery, Stanford Medicine

Erik Ingelsson, PhD
Professor of Cardiovascular Medicine, Stanford Medicine

Dan Kelly, MD
Willard And Rhoda Ware Professor Of Diabetes and Metabolic Diseases, Perelman School of Medicine, UPenn

Bonnie Ky, MD, MSCE
Associate Professor Of Medicine, Perelman School of Medicine, UPenn

Alison Marsden, PhD
Associate Professor of Pediatric Cardiology and Bioengineering, Stanford Medicine

Daria Mochly-Rosen, PhD
George D. Smith Professor in Translational Medicine, Stanford Medicine

Saman Nazarian, MD, PhD
Associate Professor Of Medicine, Perelman School of Medicine, UPenn

Marlene Rubanovitch, MD
Dwight and Vera Dunlevie Professor in Pediatric Cardiology, Stanford Medicine

Elsie Ross, MD
Assistant Professor of Vascular Surgery and of Medicine, Stanford Medicine

Paul Wang, MD
Professor of Cardiovascular Medicine, Stanford Medicine

Sean Wu, MD, PhD
Associate Professor of Cardiovascular Medicine, Stanford Medicine

Shipra Arya, MD, SM, FACS
Associate Professor of Vascular Surgery, Stanford Medicine

Thomas Cappola, MD, ScM
Herbert C. Rorer Professor in Medical Sciences, Perelman School of Medicine, UPenn

Robert Harrington, MD
Arthur L. Bloomfield Professor of Medicine Chair, Department of Medicine, Stanford Medicine

Ngan Huang, PhD
Assistant Professor of Cardiothoracic Surgery, Stanford Medicine

Rajan Jain, MD
Assistant Professor Of Medicine, Perelman School of Medicine, UPenn

Kiran Khush, MD
Associate Professor of Cardiovascular Medicine, Stanford Medicine

Nicholas Leeper, MD
Associate Professor of Vascular Surgery and Cardiovascular Medicine, Stanford Medicine

Mark Mercola, PhD
Professor of Cardiovascular Medicine, Stanford Medicine

Edward E. Morrissey, PhD
Robinette Foundation Professor of Cardiovascular Medicine, Perelman School of Medicine, UPenn

Patricia Nguyen, MD
Assistant Professor of Cardiovascular Medicine, Palo Alto Veterans Affairs Health Care System

Daniel J. Rader, MD
Seymour Gray Professor of Molecular Medicine, Perelman School of Medicine, UPenn

Yasuhiro Shudo, MD
Clinical Assistant Professor of Adult Cardiothoracic Surgery, Stanford Medicine

Ron Witteles, MD
Associate Professor of Cardiovascular Medicine, Stanford Medicine

Paul Yock, MD
Martha Meier Weiland Professor Professor of Bioengineering, Stanford Medicine

Helen Blau, PhD
Donald E. and Delia B. Baxter Foundation Professor Director, Baxter Lab for Stem Cell Biology, Stanford Medicine

Scott Damrauer, MD
Assistant Professor of Surgery, Perelman School of Medicine, UPenn

Sarah Heilshorn, PhD
Associate Professor of Materials Science and Engineering, Stanford Medicine

Sharon Hunt, MD
Professor Emerita of Cardiovascular Medicine, Stanford Medicine

Ioannis Karakikes, PhD
Assistant Professor in the Department of Cardiothoracic Surgery, Stanford Medicine

Joshua Knowles, MD
Assistant Professor of Cardiovascular Medicine, Stanford Medicine

Ronglih Liao, PhD
Professor of Cardiovascular Medicine, Stanford Medicine

Lloyd Minor, MD
Carl and Elizabeth Naumann Professorship for the Dean of the Stanford School of Medicine

Kiran Musunuru, MD, PhD
Associate Professor Of Medicine, Perelman School of Medicine, UPenn

James Priest, MD
Assistant Professor of Pediatrics (Cardiology) at Stanford University Medical Center, Stanford Medicine

Kristy Red-Horse, PhD
Associate Professor of Biology, Stanford School of Medicine

Edda Spijkerkoetter, MD
Associate Professor of Pulmonary and Critical Care Medicine, Stanford Medicine

Joseph Wu, MD, PhD
Director, Stanford CVI Simon H. Stertzer MD, Professor of Medicine and of Radiology, Stanford Medicine
Stanford-led team receives $10 million award for myosin research by Bruce Goldman

Stanford scientists will direct a multidisciplinary, multi-institutional team focused on understanding in detail how tiny mutations in a protein, myosin, can cause the classic features of cardiomyopathy.

The National Institute of General Medical Sciences has awarded a 5-year, $10 million grant to a multidisciplinary, multi-institutional team of scientists led by School of Medicine researchers that will try to gain insights into a common cause of heart failure. The lead Stanford researchers are James Spudich, PhD, professor of biochemistry; Daniel Bernstein, MD, professor of pediatrics; and Sean Wu, MD, PhD, associate professor of cardiovascular medicine. Co-investigators are Alexander Dunn, PhD, associate professor of chemical engineering, and Kathleen Ruppel, MD, senior scientist in biochemistry.

The Stanford-led team — which also includes researchers from the University of California-Santa Barbara, the University of Washington and the Institut Curie in Paris — will seek a deep understanding of exactly how minute changes within genes, and the resulting alterations in the proteins for which those genes are recipes, can give rise to complex disease profiles.

The grant came about as the result of a national competition, with each institution limited to a single submitted application. This granting mechanism was initiated in 2017 with the express intent, according to the NIGMS, of supporting projects that “address complex and challenging biomedical problems.” Spudich and Bernstein’s proposed project was selected through an internal review process that took place at Stanford. It was one of only three such proposals the NIGMS selected this fiscal year for grants totaling an estimated $27 million over a five-year period.

The team will focus on a particular protein, myosin, which is responsible for cell contraction in numerous tissues and organs, notably including skeletal muscle and the heart. Mutations in myosin have been implicated in a variety of cardiomyopathies, irregularities in heart-muscle function that affect 1 in every 500 people and are major causes of heart failure and sudden death. Myosin mutations can also cause skeletal muscle diseases that lead to impaired motor function.


Using machine learning to predict heart failure

By Megan Mayerle, PhD

The human heart repeatedly goes through adaptive cycles of growth and remodeling in order to meet the body’s demand. However, in individuals with cardiovascular disease, chronic changes in the volume of blood the heart pumps or the pressure the heart experiences can lead to maladaptive growth and remodeling that compromises the heart’s function and increase an individual’s risk for ischemia, arrhythmia, and sudden death.

In order to prevent and treat heart failure, doctors need methods to help them anticipate and predict the rate and type of cardiac growth. Stanford Cardiovascular Institute member Ellen Kuhl, a professor in the Department of Mechanical Engineering, leads a group that recently published a machine-learning approach to solve this problem in Biomechanics and Modeling in Mechanobiology.

Together with their collaborators at the California Medical Innovations Institute and UC San Francisco, the scientists performed an 8-week long study of cardiac volume overload in six pigs and then combined hierarchical modeling, Bayesian inference, and Gaussian process regression to quantify the uncertainty in their experimental measurements. They then applied these data to a computational growth model and determined how well their experimental and computationally predicted myocyte measurements agreed and were further able to implicate the stretching of cells as key for myocyte remodeling.

Multiscale cardiac growth models such as the one employed by Kuhl and colleagues allow researchers understand how various subcellular-, cellular-, and organ-level changes can combine to contribute to heart failure and have the potential to provide a more holistic picture of the failing heart, helping clinicians best care for their patients.

Researchers discover gel reduces scar tissue after surgery in animals  by Mandy Erickson

Researchers at Stanford University have found that spraying a gel on the internal tissues of animals after cardiac surgery greatly reduces adhesions, fibrous bands that form between internal organs and tissues. Adhesions can cause serious, even fatal, complications. The gel, developed at Stanford to deliver medications, was far more effective than adhesion prevention materials currently on the market, the researchers said. A paper describing the research published in *Nature Biomedical Engineering*. Joseph Woo, MD and Eric Appel, PhD, are the senior authors.

Adhesions form after 95% of surgeries. Some are harmless, but after abdominal surgeries, they can twist or compress the intestines, causing life-threatening blockages. Gynecological surgery can also lead to adhesions that cause infertility. In cardiac re-operations, common for those born with heart defects, adhesions increase the risk of complications. Methods to prevent adhesions — including animal membranes, sheets of rubber and mineral oil — have existed for more than 100 years, but they have mostly failed. Current adhesion barriers approved by the Food and Drug Administration are are difficult to deploy and are considered ineffective.

The Stanford researchers had long pondered a solution to the adhesion problem. But one day, when they were working with lab rats to develop an injectable therapy to reduce tissue damage following a heart attack, Appel suggested spraying a polymer-nanoparticle hydrogel onto the hearts and surrounding tissue after surgery to see if it reduced the formation of adhesions. Weeks later, when they operated on the animals again, they saw that no adhesions had formed. The researchers then tested their compound in sheep, whose hearts are similar in size and shape to human hearts; they found similar results.


An unmet need for integrated mental health services for female cardiac patients  By Megan Mayerle, PhD

Coronary heart disease (CHD) is the leading cause of death of American women. Women with CHD are 2-3 times as likely to experience anxiety or depression as men, and women who experience depression or anxiety are also more likely to have higher CHD recurrence rates and be rehospitalized. Dr. Jennifer Tremmel recently published a study in the *American Journal of Cardiology* examining the mental well-being of women receiving treatment at a women's heart health clinic. Of the women surveyed, 38% scored in the moderate-to-severe range for at least 1 mental disorder, and 50% were experiencing insomnia. Women also experienced clinical depression and anxiety at significantly higher rates. Only about half of the women who reported psychological distress received mental health treatment, suggesting that there is an unmet need for integrated mental health services for female cardiac patients.


Inflammation triggers silent mutation to cause deadly lung disease  by Tracie White

Researchers at Stanford 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. Mark Nicolls, MD, is the senior author of the study, which was published in *Circulation*. “This is important research for understanding how ‘second hits’ can render ordinarily silent genetic mutations deadly,” Nicolls said.

Currently there is no known cause for 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 US, 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.

Erik Ingelsson has been awarded an AHA Transformative Research Award for "Discovery and characterization of novel genes associated with risk for non-alcoholic fatty liver disease".

Michael Snyder was awarded the George Beadle Award from the Genetics Society of America.

Alison Marsden received an American Heart Association Transformation Project Award "Hemodynamic determinants of premature pulmonary valve dysfunction in children with Tetralogy of Fallot."

David Maron to lead the Stanford Prevention Research Center (SPRC) as Division Chief.

Deborah Ho was appointed as a Clinical Assistant Professor in Pediatric Cardiology.

Elsie Ross was promoted to Clinical Assistant Professor and received a K award "Using Artificial Intelligence To Enable Early Identification and Treatment Of Peripheral Artery Disease".

Sharon Paige was appointed Instructor in Pediatric Cardiology. She was also awarded a K08 "Patient-Specific Induced Pluripotent Stem Cells for Modeling Single Ventricle Congenital Heart Disease".

Tina Baykaner was awarded an NIH K23 for 'Personalizing Atrial Fibrillation Treatment'.

Jessica Brodt received an SCA/IARS Mid-Career Grant to support my research into Regional Anesthesia for Cardiothoracic Enhanced Recovery (the RACER study).

Kari Nadeau won the 2020 National Distinguished Scientist Award from the American Academy of Allergy, Asthma & Immunology.

Karina Nakayama will be joining the Department of Biomedical Engineering in the School of Medicine at Oregon Health and Science University as a tenure track Assistant Professor in January 2020.

Robert Wirka received Irvine H. Page Young Investigator Award at the Vascular Discovery Conference (ATVB).

Fatima Rodriguez was awarded a K01 for "SURPASS Statin Use and Risk Prediction of Atherosclerotic Cardiovascular Disease in Minority Subgroups". She also received an AHA Robert Wood Johnson Fdn Harold Amos Medical Scholars award.

Phillip Yang received an AHA Collaborative Science Award "Patient- and Disease-Specific Exosomes for Precision Therapeutics of Heart Failure".

Daria Mochly-Rosen was awarded the "Accelerate Australia Ecosystem leadership Award 2019" for supporting biomedical innovators globally.

Nicholas Leeper was promoted to Full Professor of Surgery (Vascular Surgery).

Irving Weissman and Johns Hopkins’ Bert Vogelstein will share the 2019 Albany Medical Center Prize in Medicine and Biomedical Research for discoveries in stem cell and cancer biology.

Mark Nicolls was awarded a $2M competitive renewal of our R01 entitled "A Critical Role for microvasculature in airway transplantation".

Joseph C. Wu received the British Society of Cardiovascular Research Bernard and Joan Marshall Distinguished Investigator Award.

Kari Nadeau won the 2020 National Distinguished Scientist Award from the American Academy of Allergy, Asthma & Immunology.

The Institute currently consists of over 240 faculty members representing physicians, surgeons, engineers, basic and clinical researchers. The Institute’s mission is integrating fundamental research across disciplines and applying technology to prevent and treat cardiovascular disease. To support cardiovascular research and education at CVI, please contact: Joseph C. Wu, MD, PhD. CVI Director at joewu@stanford.edu or Cathy Hutton, Senior Associate Director, Medical Center Development at cathy.hutton@stanford.edu. For more information: http://med.stanford.edu/cvi/support-our-research.html and http://cvi.stanford.edu
Dr. Ronald L. Dalman is president-elect of Society for Vascular Surgery

Dr. Ronald L. Dalman was elected president-elect of the Society for Vascular Surgery (SVS) at the organization’s annual meeting in National Harbor, MD, in June. Next year he will step up to president. Prior to joining the line of succession last year as vice president, Dr. Dalman served 3 years as program chair of the Vascular Annual Meeting and has served on the SVS Board of Directors, the Patient Safety Organization Governing Council, the Education and Research Councils, as well as the PAC Steering, Document Oversight, SVS Foundation Development, Membership, Publications and PSO EVAR Cost Committees.

A possible drug target for a deadly heart condition

A genetic mutation linked to dilated cardiomyopathy, a dangerous enlargement of the heart’s main pumping chamber, activates a biological pathway normally turned off in healthy adult hearts, according to a study led by Joseph C. Wu and colleagues at the Stanford University School of Medicine. The findings, which were published in Nature, suggest that existing drugs could one day be repurposed to treat dilated cardiomyopathy. More broadly, the study demonstrates how patient-derived heart cells can help scientists better study the heart and screen candidate drugs.

The researchers studied heart muscle cells grown from patients with a genetic mutation associated with dilated cardiomyopathy. Heart cells with a mutation in lamin, which forms part of the nuclear envelope, failed to beat properly — just like in patients with the disease. The scientists found that the defect was the result of a surge in the platelet-derived growth factor pathway. This pathway is important in the formation of blood vessels and normally only activates when the heart first forms or is under stress. Treating heart cells with existing drug inhibitors of the pathway restored regular, rhythmic beating.


Accurately assessing ASCVD risk in distinct Asian and Hispanic subgroups

Atherosclerotic cardiovascular disease (ASCVD) is the leading cause of death for men and women in the US. Though ASCVD is largely preventable, effective prevention requires an assessment of risk for selection of appropriate interventions. Clinicians often make use of a set of guidelines put out by the American Heart Association and the American College of Cardiology. The current version relies on Pooled Cohort Equations (PCEs), a statistical methodology to assess risk. However, the current PCE does not include Asian or Hispanics. Thus, it is possible that this method of assessing risk might not work properly for these, as well as other, racial and ethnic subgroups.

Using data from electronic health records, a team of scientists and clinicians led by Stanford Cardiovascular Institute members Doctors Fatima Rodriguez and Latha Palaniappan decided to explicitly test the accuracy of PCEs in predicting ASCVD risk in different Asian and Hispanic subgroups. The researchers’ study, which was recently published in the Journal of the American Heart Association, revealed that PCE generally overestimated risk in Hispanic and Asian patients. Furthermore, the degree of overestimation varied significantly within different Asian and Hispanic subgroups. Specifically, the researchers found that the PCE was the most accurate for Korean and Puerto Rican subgroups. In light of this information, the researchers suggest that clinicians use caution in interpreting risk assessments made using PCEs, since the degree to which the PCE overestimates risk can vary at the subpopulation level.

Immune cells invade aging brains, disrupt new nerve cell formation

By Bruce Goldman

A study, published in *Nature*, led by Anne Brunet, PhD, has revealed that immune cells infiltrate the rare newborn nerve-cell nurseries of the aging brain.

There’s every reason to think those interlopers are up to no good. Experiments in a dish and in animals indicate they’re secreting a substance that chokes off new nerve cell production.

While most of the experiments in the study were carried out in mice, the central finding — the invasion, by immune cells called killer T cells, of neurogenic niches (specialized spots in the brain where new nerve cells, are generated) — was corroborated in tissue excised from autopsied human brains.

The findings could accelerate progress in hunting down the molecules in the body that promote the common deterioration of brain function in older individuals and in finding treatments that might stall or even reverse that deterioration. They also signify a crack in the wall of dogma that’s deemed the healthy brain impervious to invasion by the body’s immune cells, whose unbridled access to the organ could cause damage.


New ‘don’t eat me’ signal may provide basis for cancer therapies

By Christopher Vaughan

Researchers at the Stanford University School of Medicine have discovered a new signal that cancers seem to use to evade detection and destruction by the immune system.

The scientists have shown that blocking this signal in mice implanted with human cancers allows immune cells to attack the cancers.

Blocking other “don’t eat me” signals has become the basis for other possible anti-cancer therapies.

Normally, immune cells will detect cancer cells, then engulf and devour them. In recent years, researchers have discovered that proteins on the cell surface can tell macrophages not to eat and destroy them.

This can be useful to help normal cells keep the immune system from attacking them, but cancer cells use these “don’t eat me” signals to hide from the immune system.

A paper describing the research was published July 31 in *Nature*. Irving Weissman, MD, is the senior author.


Gene networks reveal transition from healthy to failing heart

By Hanae Armitage

Scientists investigating heart failure have been limited to studying diseased heart tissue in the lab — understandably, as people don’t tend to pluck out a healthy heart for the sake of research. But now, scientists with access to unusable, yet still healthy, donor hearts have been able to investigate the genomic pillars behind the transition from healthy hearts to heart failure.

In doing so, researchers at the Stanford University School of Medicine and their collaborators have created one of the first maps to reveal gene activity and connectivity as the heart shuts down.

Euan Ashley, MB ChB, DPhil, calls it a gene network. By delineating these gene networks, the group has discovered one gene in particular that seems to be at the center of the action. It appears to be highly connected in heart failure, meaning its activity is similar to that of many neighbors. What’s even more exciting, Ashley said, is that when the researchers disabled the function of this gene in mouse models of heart failure, the mice were protected and did not succumb to the cardiac condition.

“This study has a truly unique angle, which is that we had precious, healthy human tissue and we used it to tell us something new about how a disease manifests,” said Victoria Parikh, MD, clinical instructor of cardiovascular medicine. “And now someday we might even be able to translate that into a treatment.”

A paper providing details of the study was published June 24 in *Nature Communications*. Ashley is the senior author. Parikh shares lead authorship with Pablo Cordero, PhD, a former Stanford graduate student.

High blood pressure may prevent cognitive decline in a subset of older patients  By Megan Mayerle, PhD

High blood pressure has been associated with a wide variety of health issues including cardiovascular disease, cognitive decline, and mortality. However, the link between high blood pressure and cognitive decline is less clear in adults age 65 or older.

A team of researchers led by Dr. Michelle Odden had previously shown that the functional status of older adults, as measured by metrics such as grip strength or walking speed, impacted the association between blood pressure and health. High blood pressure was associated with worse health outcomes in higher functioning adults. Paradoxically however, low blood pressure was associated with worse outcomes in lower functioning adults.

In a study published in the *Journal of Hypertension*, researchers led by Dr. Odden set out to determine if there is a relationship between blood pressure, functional status, and cognitive decline and dementia in older adults. The scientists found that higher blood pressure was associated with improvements in cognitive function among persons with functional limitation. This association was attenuated or reversed among those without such limitations. Why this association occurs is unknown, however it may be explained by insufficient organ perfusion. In the case of dementia, the researchers suggested that low blood pressure may decrease the extent of blood perfusion to the brain, which could lead to hypoxia, which has been linked to an increased chance of cognitive decline and dementia. Current clinical guidelines recommend interventions to lower blood pressure in older adults; these findings challenge the universality of such guidelines.


Identity-shifting cells protect against rupture in atherosclerosis  By Hanae Armitage

Changing your identity to protect others might sound like something reserved for comic book vigilantes, but a study led by researchers at the Stanford University School of Medicine has found a select group of cells in artery walls do just that.

For these cells, the identity shift happens in a disease called atherosclerosis, which occurs when arteries get clogged by plaque, a buildup of fats, cholesterol and molecular particulate.

“We know that things like poor diet and lack of exercise contribute to atherosclerosis,” said Thomas Quertermous, MD, professor of cardiovascular medicine at Stanford. “But molecularly speaking, researchers still don’t know how the disease progresses or, conversely, is hindered.” This new work, he said, takes a big step toward addressing that question.

Plaque grows within the layers of tissue that form the artery, as opposed to inside the tube itself, causing the blood conduit to narrow. Too much plaque tears open the tissue, allowing the built-up gunk to flood the interior of the tube. That leads to a clot, which can cause artery blockage and often a heart attack.

In people with atherosclerosis, cells that make up the artery wall transform and invade the area containing the plaque, or lesion, and form something called a fibrous cap, which acts kind of like a lid to prevent the plaque from bursting into the artery. Now, Quertermous and his colleagues have characterized the identity of these transformed cells, giving key insights into something called plaque stability, which determines the likelihood of a plaque bursting. The more robust the fibrous cap, the more stable the plaque and the less likely it is to rupture.

The team has also pinpointed a gene that seems to be behind the cells’ transformation. What’s more, when they looked at population-wide genomic data, they saw that individuals who had more activity in this particular gene were at a decreased risk for heart attack.

“Logically, it makes sense — the more cells that help form the fibrous cap, the stronger the protection against plaque rupture and therefore the less risk of a heart attack,” said Quertermous, who is the William G. Irwin Professor in Cardiovascular Medicine. A paper describing the details of the study was published in *Nature Medicine*. Quertermous and Juyong Kim, MD, instructor of medicine, are the senior authors. The lead author is Robert Wirka, MD, instructor of cardiovascular medicine.

Building tissue scaffolds to repair damaged hearts

By Megan Mayerle, PhD

Over 700,000 people in the United States suffer from heart attacks each year. In order to treat these patients, scientists and clinicians have increasingly been looking to regenerative medicine and therapeutic cell delivery. However, clinical trials of therapeutic cell delivery have generally shown minimal to moderate benefit in improving the heart’s pumping capacity.

Tissue engineering has emerged as an alternative method to regenerate heart tissue and to encourage new blood vessels to form in the heart. A key component of engineered tissues is the extracellular matrix, a scaffold for cells that provides important cues to cells through properties such as spatial patterning, stiffness, and cell binding domains.

Blood vessel formation is critical to the survival of the engineered heart tissue. Stanford scientists led by Cardiovascular Institute member Dr. Ngan Huang have recently published a study in *Frontiers in Bioengineering and Biotechnology* looking at how the three-dimensional organization of engineered tissue scaffolds impacts how well blood vessel networks form in these tissues.

The researchers designed scaffolds with different levels of organization and seeded them with cardiomyocytes and endothelial cells differentiated from induced pluripotent stem cells and then compared how well the tissues functioned in a heart injury animal model. They found that different scaffold alignments facilitated different biological processes. For example, parallel-aligned scaffolds preferentially directed the formation of capillaries along the direction and plane of the scaffold microfibers. By contrast, endothelial cells survived better in randomly aligned scaffolds.

These findings are important because they suggest that scaffold topography plays a key role in modulating cellular survival, vascularization, and microvessel architecture.

Human iPSCs to model cardiac fibrosis

By Megan Mayerle

Interstitial fibrosis, which reduces tissue flexibility and increases the rate at which cardiovascular disease progresses to heart failure. Stopping fibrosis would decrease the incidence of heart failure significantly.

In a study published in *Circulation Research* postdoc Hao Zhang, Joseph Wu, and colleagues analyzed the single-cell transcriptomes of fibroblasts from mouse to identify tissue-specific signature genes, showed that cardiac fibroblasts are of epicardial lineage, and developed a protocol to make cardiac fibroblasts from induced pluripotent stem cells.

Stem-cell based advances such as this help doctors and scientists to gain a better understanding of the complexity of cardiac fibrosis, and to advance the development of more effective anti-fibrotic therapies.

RNA’s role in diagnosing rare diseases

By Hanae Armitage

An individual’s genetic makeup can reveal important and intimate details of his or her biology. Now, scientists are showing that RNA, the lesser-known cousin of DNA, can provide insights into rare diseases that DNA cannot.

Stephen Montgomery, PhD, is among scientists harnessing RNA to identify the cause of rare diseases. In a study in *Nature Medicine*, Montgomery and colleagues describe how RNA sequencing helps pin down rare diseases. While DNA can reveal certain mutations or gene abnormalities present from birth, RNA show what happens when those genes are turned on and how environment sways activation and expression of our genes.


iPSC Cardiomyocytes Model Diastolic Dysfunction in Hypertrophic Cardiomyopathy  

By Amanda Chase, PhD

Hypertrophic cardiomyopathy (HCM) is a common cardiovascular disease. It is characterized by abnormal thickening of the ventricular wall of the heart and increased risk of arrhythmia, sudden death, and heart failure. Many HCM patients display symptoms of diastolic dysfunction (DD), characterized by symptoms and signs of heart failure as a result of the left ventricle being unable to accept an adequate volume of blood. Without treatment, DD can progress to heart failure and significant morbidity and mortality. Despite the prevalence and severity of DD, the underlying cellular mechanisms are not well understood, which greatly hinders the development of specific and more effective treatments.

Human induced pluripotent stem cell (iPSC) technology has enabled patient-specific disease modeling of various cardiovascular disease. Researchers at the Stanford Cardiovascular Institute, led by Haodi Wu and Joseph Wu, generated iPSC-derived cardiomyocytes (iPSC-CM) models of DD to provide a novel patient-specific platform for understanding mechanisms of DD in HCM, recently published in *European Heart Journal*. Using various imaging techniques, they were able to reveal novel cellular mechanisms for DD, which can be developed into therapeutic targets in the future. 


Molecular Control of Blood Vessel Regeneration in Response to Injury  

By Megan Mayerle, PhD

Dysfunctional endothelial cells (ECs) are related to many cardiovascular diseases. Endothelial cells’ ability to properly regenerate is necessary to prevent progressive cardiovascular disease. Blockages can form in arteries and veins when ECs don’t properly regenerate into a thin monolayer lining the interior of the blood vessel. BMPR2 and Notch1 are required for angiogenesis, but little is known about how they coordinate EC metabolism, chromatin remodeling, and gene regulation to regulate EC proliferation and monolayer regeneration. Researchers led by Marlene Rabinovitch have published a paper in *Circulation Research* answering these questions. 

The team discovered that physical contact between smooth muscle cells and endothelial cells in blood vessels is required for BMPR2-mediated activation of Notch signaling. Notch signaling in turn leads to changes in EC metabolism, and gene expression that promote ECs’ ability to regenerate. The scientists also used a mouse model of pulmonary hypertension to study how Notch1 functions after injury. They found that the ECs of mutant mice who didn’t make enough BMPR2 didn’t have as much Notch1 signaling, were less proliferative, and that the linings of their blood vessels did not reform correctly after injury. Deleting Notch1 in the endothelial cells of transgenic mice led to worsening of pulmonary hypertension. Activating Notch1 in ECs might be a promising therapeutic strategy in vascular diseases like atherosclerosis and pulmonary arterial hypertension. 


Controlling Mitochondrial Dynamics to Help the Hearts of Huntington’s Disease Patients  

By Megan Mayerle, PhD

Huntington's disease (HD) is a deadly neurodegenerative disorder. While the central nervous system is most affected by HD, patients suffer from skeletal muscle atrophy, cardiovascular diseases, and heart failure. The heart and other muscles require a lot of energy and rely on mitochondria as a source. The cardiac cells of HD patients often contain dysfunctional mitochondria. In a paper recently published in the *Journal of Molecular and Cellular Cardiology*, Stanford researchers Dr. Daria Mochly-Rosen and colleagues set out to determine whether a hyperactive version of a protein called Drp1 underlies HD mitochondrial dysfunction.

The researchers differentiated induced pluripotent stem cells containing a disease-linked version of the Huntington protein to cardiomyocytes. Joshi and colleagues were able to show that P110, a synthetic peptide that selectively inhibits Drp1’s interaction with another mitochondrial protein, Fis1, mitochondrial function in cells. The researchers also showed that P110 has similar mitochondrial benefits in a mouse model of Huntington’s disease. Joshi and colleagues hope that their studies with P110 will help pave the way for the development of a therapeutic that improves cardiac and muscular function for HD patients. 

Congratulations to the 2019 Dorothy Dee & Marjorie Helene Boring Trust Awardees

Cardiovascular disease remains the leading cause of death in the world. The iHeart Research award is meant to support Stanford medical students excited about research solutions that impact how we treat and prevent cardiovascular diseases.

**Nicolas Quach**  
MS2 / Cardiothoracic Surgery  
“Engineering a Supra-therapeutic C-X-C Chemokine Receptor Type 4 (CXCR4) Agonist to Prevent Ischemic Heart Failure”  
Mentor: William Hiesinger, MD

**Saad Syed**  
MS3 / Pediatric Cardiology  
“A Non-invasive Signature of Myocardial Signaling in Children with Single Ventricle Heart Failure”  
Mentor: Sushma Reddy, MD

**Ting Hsuan Wu**  
MS2 / Pulmonary and Critical Care Medicine  
“Single-cell Analysis of Inflammation-induced Pulmonary Hypertension in Bmpr2 Dysfunction”  
Mentors: Mark Nicolls, MD and Peter Kao, MD, PhD

**Recruitment for T32 Fellowships**

**Multi-Disciplinary Training Program in Cardiovascular Imaging T32 Training Grant**  
The Multi-Disciplinary Training Program in Cardiovascular Imaging at Stanford is funded by the National Institute of Biomedical Imaging and Bioengineering of the National Institutes of Health. With the impact of cardiovascular disease on U.S. and world health, and the rapid advances in imaging technologies and cardiovascular biology, it is critical that fellows be provided a broad, multi-disciplinary, and collaborative training program to foster their ability to translate CV imaging research into clinical applications. The program is designed to train the next generation of CV imaging investigators by exposing them to three complementary areas—clinical, engineering, and molecular imaging.  
http://med.stanford.edu/cvi/education/cardiovascular-imaging-t32.html

**Mechanisms and Innovations in Cardiovascular Disease T32 Training Grant**  
This program provides training in the following areas of vascular medicine and research: Vascular Reactivity and Thrombosis, Vascular Regeneration and Development, Metabolic or Lifestyle Influences on Vascular Outcomes, Proteomic Markers & Genetic Determinants of Vascular Disease, Gender and Ethnicity Differences in Vascular Disease, and Vascular Bioengineering. Twenty-nine faculty mentors from eighteen different departments within the School of Medicine and the University provide a variety of angles from which to address fundamental questions about vascular disease.  
http://med.stanford.edu/cvi/education/mechanisms-and-innovations-t32.html

**Research Training in Myocardial Biology T32 Training Grant - 2 Openings**  
The Multi-Disciplinary Research Training Program in Myocardial Biology is funded by the National Institutes of Health to bring together post-doctoral fellows and faculty from six complementary areas – genetics and genomics, cellular signaling, molecular imaging, physiology and phenotyping, cardiac development and regeneration and outcomes research and population science. Although many possible divisions exist in the spectrum of cardiovascular investigators, one of the most discrete is the division between those researchers interested in blood vessels and those primarily interested in the biology of the heart muscle itself. Myocardial biologists at Stanford are found in diverse departments and divisions within the wider Stanford community and this provides a natural vehicle for multidisciplinary training.  
http://med.stanford.edu/cvmedicine/education/timbs.html
Frontiers in Cardiovascular Sciences Seminar Lineup

Join us Tuesday afternoons to hear the latest in Cardiovascular and Pulmonary Research. Exact times and locations available at https://med.stanford.edu/cvi/mission/frontiers-in-cv-science.html

We are excited to report the official accreditation of The Stanford Hereditary Hemorrhagic Telangiectasia (HHT) Program as a Center of Excellence by the Cure HHT Foundation! Edda Spiekerkoetter will be the center director, and David Stevenson from LPCH Medical Genetics will be the associate director.

The center will be administered and supported by the adult PCCM pulmonary vascular disease program. Over two years ago, Edda and David began the process of creating a multidisciplinary program which includes collaboration with medical genetics, interventional radiology, neurosurgery, ENT surgery, and support from hospital administration. In accrediting the program as a center of excellence, the Cure Foundation cited not only excellence in clinical care but specifically the existing research expertise of Stanford University as a major strength.

MED223 | Cardiopulmonary Research & Medicine

The focus of MED223 is to fine-tune critical thinking skills by analyzing original publications and understanding the current complexities of the cardiovascular system.

MED223 is part of the Scholarly Concentration: Cardiovascular-Pulmonary Sciences.

For more information, contact MED223 Directors: Ngan Huang, PhD, Ioannis Karakikes, PhD, Edda Spiekerkoetter, MD, and Vinicio de Jesus Perez, MD. MED223 website: https://med.stanford.edu/cvi/education/cvi-courses/med223.html
October 2019

**Burroughs Wellcome Fund Career Awards for Medical Scientists.** For physician scientists to bridge postdoctoral/fellowship and faculty. Deadline: Oct 1, 2019.

**NIH NHLBI Early Phase Clinical Trials for Therapeutics and/or Diagnostics (R33 Clinical Trial Req).** Deadline: Oct 2, 2019. PAR-18-684

**NIH National Heart, Lung, and Blood Institute. Pediatric Cardiac Genomics Consortium (U01 - Clinical Trial Not Allowed).** Application Receipt Date(s): Oct 2, 2019. RFA-HL-20-015

**NIH National Heart, Lung, and Blood Institute. Cardiovascular Developmental Biology Data Resource Center (U01- Clinical Trial Not Allowed).** Application Receipt Date(s): Oct 2, 2019. RFA-HL-20-017

**NIH Research Project Grant (Parent R01 Clinical Trial Not Allowed).** New application Deadline: Oct 5, 2019. PA-19-056

**NIH Research Project Grant (Parent R01 Clinical Trial Req).** New application Deadline: Oct 5, 2019. PA-19-055

**NIH Dissemination and Implementation Research in Health (R01 Clinical Trial Opt).** Deadline: Oct 5, 2019. PAR-19-274

**NIH Improving Outcomes in Cancer Treatment-Related Cardiotoxicity (R01 Clinical Trial Opt).** Deadline: Oct 5, 2019. PA-19-112

**NIH Research Supplements to Promote Diversity in Health-Related Research (Supp).** Deadline: see announcement. PA-18-906

**NIH Improving Patient Adherence to Treatment and Prevention Regimens to Promote Health (R01 Clinical Trial Op).** Deadline: Oct 5, 2019. PA-18-722

**NIH The Mechanistic Role of the Microbiome in the Pathobiology of Heart, Lung, Blood, and Sleep Diseases (R01 - Clinical Trial Not Allowed).** Deadline: Oct 5, 2019. PA-18-784

**NIH Addressing Health Disparities in NIDDK Diseases (R01 Clinical Trial Not Allowed).** Deadline: Oct 5, 2019. PA-18-412


**NIH Mentored Career Development Award to Promote Faculty Diversity in Biomedical Research (K01 Independent Clinical Trial Required).** Deadline: October 10, 2019. RFA-HL-19-025

**NIH Mentored Career Development Award to Promote Faculty Diversity in Biomedical Research (K01 Independent Clinical Trial Not Allowed).** Deadline: Oct 10, 2019. RFA-HL-19-026

**AHA Established Investigator Award.** To support mid-career investigators (typically at associate professor level). Required Letter of Intent Deadline: Oct 10, 2019. Full Application: Jan 15, 2020

**NIH Single-Site Investigator-Initiated Clinical Trials (R61/R33 Clinical Trial Required).** R61/R33 Exploratory/Developmental Phase Award. Deadline: Oct 11, 2019. PAR-19-328

**NIH Mentored Research Scientist Development Award (Parent K01 - Independent Clinical Trial Not Allowed).** New application deadline: Oct 12, 2019. PA-19-126

**NIH Mentored Research Scientist Development Award (Parent K01 - Independent Clinical Trial Required).** New app deadline: Oct 12, 2019. PA-19-127

**NIH Pathway to Independence Award (Parent K99/R00 - Independent Clinical Trial Req).** Deadline: October 12, 2019. PA-19-128

**NIH Pathway to Independence Award (Parent K99/R00 Indep Basic Exp Studies with Humans Req).** Deadline: Oct 12, 2019. PA-19-090

**NIH NIDDK Mentored Research Scientist Development Award (K01 - Clinical Trial Req).** Deadline: Oct 12, 2019. PAR-18-418

**NIH NIDDK Mentored Research Scientist Development Award (K01 No Clinical Trials).** Deadline: Oct 12, 2019. PAR-18-419

**AHA Career Development Award.** Healthcare and academic professionals, in first professional appointment. Deadline: Oct 15, 2019

**NIH Improving Patient Adherence to Treatment and Prevention Regimens to Promote Health (R21 Clinical Trial Opt).** Deadline: Oct 16, 2019. PA-18-723

**NIH Pilot and Feasibility Therapeutic Clinical Trials in Diabetes, and Endocrine and Metabolic Diseases (R21 Clinical Trial Req).** Deadline: Oct 16, 2019. PA-18-405

**NIH New Research Directions that Advance the NHLBI Strategic Vision Normal Biology (R21 - Clinical Trial Not Allowed).** Deadline: Oct 16, 2019. PA-19-049

**NIH Small Grants for New Investigators to Promote Diversity in Health-Related Research (R21 Clinical Trial Optional).** Deadline: October 16, 2019. PAR-19-222

**AHA Innovative Project Award.** Funds ideas that may introduce new paradigms, challenge current paradigms, look at existing problems from new perspectives, or exhibit other uniquely creative qualities. Letter of Intent: Oct 22, 2019. Full Application: Jan 18, 2020

November 2019

**NIH Research Project Grant (Parent R01 Clinical Trial Not Allowed).** Renewal, Revision, Resubmission Deadline: Nov 5, 2019. PA-19-056

**NIH Research Project Grant (Parent R01 Clinical Trial Required).** Renewal, Revision, Resubmission Deadline: Nov 5, 2019. PA-19-055


**NIH Mentored Clinical Scientist Research Career Development Award (Parent K08 Independent Clinical Trial Not Allowed).** Renewal, Revision, Resubmission Deadline: Nov 12, 2019 (Standard K deadlines). PA-19-117

**NIH Mentored Clinical Scientist Research Career Development Award (Parent K08 Independent Clinical Trial Required).** Renewal, Revision, Resubmission Deadline: Nov 12, 2019 (Standard K deadlines). PA-19-116

**NIH Mentored Patient-Oriented Research Career Development Award (Parent K23 Independent Clinical Trial Required).** Renewal, Revision, Resubmission Deadline: Nov 12, 2019 (Standard K deadlines). PA-19-118

**NIH Mentored Patient-Oriented Research Career Development Award (Parent K23 - Independent Clinical Trial Not Allowed).** Renewal, Revision, Resubmission Deadline: Nov 12, 2019 (Standard K deadlines). PA-19-119

**NIH Mentored Research Scientist Development Award (Parent K01 - Independent Clinical Trial Not Allowed).** Renewal, Revision, Resubmission Deadline: Nov 12, 2019. PA-19-126

**NIH Mentored Research Scientist Development Award (Parent K01 - Independent Clinical Trial Required).** Renewal, Revision, Resubmission Deadline: Nov 12, 2019. PA-19-127

**NIH Pathway to Independence Award (Parent K99/R00 - Indep Clinical Trial Req).** Renewal, Revision, Resubmission Deadline: Nov 12, 2019. PA-19-128

**NIH Pathway to Independence Award (Parent K99/R00 Indep Basic Exp Studies with Humans Req).** Renewal, Revision, Resubmission Deadline: Nov 12, 2019. PA-19-090

December 2019

**Ruth L. Kirschstein National Research Service Award (NRSA) Individual Postdoctoral Fellowship (Parent F32) Postdocs.** New application deadline: Dec 8, 2019. PA-19-188
OCTOBER 2019

Echo in Congenital Heart Disease: Special Emphasis on Adult Congenital Heart Disease: Including Uses of Multimodality Imaging. October 2-5, 2019. Hyatt Regency Coconut Point, Bonita Springs, FL.

American College of Cardiology Core Curriculum for the Cardiovascular Clinician. October 2-5, 2019. Washington, DC.

American College of Cardiology Heart Valve Summit: Medical, Surgical and Interventional Decision Making. October 3-5, 2019. Chicago, IL.


Cases in Echocardiography, Cardiac CT, and MRI. October 23-26, 2019. The Meritage Resort, Napa, CA.


Pediatric Palliative Care West Coast Summit: It's About the Journey. Monday, October 28, 2019 7:30 AM - Tuesday, October 29, 2019 12:45 P. Preservation Hall, Oakland, CA.

NOVEMBER 2019

How to Become a Cardiovascular Investigator. November 1, 2019 - November 2, 2019.

Coronary Artery Disease: Case-Based Learning 2019. November 1-3, 2019. Four Seasons, Las Vegas, NV.


Core Topics in Point-of-Care Ultrasound Course. Saturday, November 9, 2019 8:00 AM - Sunday, November 10, 2019 5:00 PM. Li Ka Shing Center for Learning and Knowledge (LKSC), Stanford, CA.

Echo Best Practice | Echocardiography in Patient Care and in Clinical Trials: When to Use Multi-Modality Imaging. November 10-12, 2019. Fairmont Scottsdale Princess, Scottsdale, AZ.


DECEMBER 2019


8th Annual Heart Rhythm & ECG Course: A Case-Based Approach - General Session. Scottsdale, AZ US. December 5, 2019 to December 8, 2019.


Echo on Marco Island: Case-Based Approach. December 16-19, 2019. Marco Island Marriott Beach & Golf, Marco Island, FL.
Clinical Biomarker & Phenotyping Core Lab (BPCL)

BPCL provides quantitative assessment of clinical cardiovascular phenotypes for translational research and clinical trials. These cardiovascular phenotypes include evaluating cardiac structure and function, measuring carotid intimal thickness and arterial stiffness, and testing endothelial function and cardiopulmonary exercise testing.

In collaboration with the Human Immune Monitoring Center at Stanford and members of the Cardiovascular Institute, we also offer central blood processing and banking capabilities. In addition, we develop new biomarker platforms and imaging modalities.

Contact: Francois Haddad, MD / fhaddad@stanford.edu

CVI Clinical Trials Core

The CVI Clinical Trials Core provides full spectrum of support to CVI members and their clinical trials. The coordinators has extensive clinical research experience in both industry and academia. The team provides services and support to principal investigators and sponsors, including:

- Consultation
- Study start-up management, including IRB applications, budget development
- Subject recruitment, site visits, and follow-ups (AE reporting and queries)
- Data management
- Regulatory compliance and documentation
- Closeout
- Closeout

Contact: Ed Finn, Clinical Trials Manager at efin@stanford.edu

Stanford CVI Human iPSC Biobank Service

Normal and patient-derived reprogrammed cardiomyocytes are a tremendous resource for researchers and physicians here at Stanford and around the country. Understanding the disease process directly at the population level and observing these cells as surrogates under a myriad conditions has the potential to be a game-changer for cardiovascular medical research.

To facilitate research in a dish that allows screening of new compounds or characterization of human disease phenotypes using cardiomyocytes, the Institute created a service by which de-identified peripheral blood mononuclear cell (PBMC) samples from selected patients can be sent to Stanford CVI for reprogramming free of cost.

SCVI biobank is supported in part by National Heart, Lung and Blood Institute (NHLBI) and the Stanford Cardiovascular Institute (CVI).

Contact: Joseph Wu, MD, PhD / joewu@stanford.edu
or Biobank manager, Yan Zhuge, PhD / yanzhuge@stanford.edu with any questions.

Cardiovascular Pharmacology (BioADD)

The Cardiovascular Pharmacology/Biomaterials and Advanced Drug Delivery (BioADD) Laboratory is a cutting edge research facility that specializes in the creation of biomaterials and drug delivery agents. The lab lends its expertise toward designing and analyzing biomaterials, developing drug delivery devices and formulations, pharmacokinetic and pharmacodynamic studies, and developing smart materials for biomedical applications. The CVI Cardiovascular Pharmacology also offers trainings and lectures.

Contact: Jayakumar Rajadas, PhD jayraja@stanford.edu

3DQ Imaging Laboratory

Stanford’s 3DQ Imaging Laboratory develops new approaches to exploration, analysis and quantitative assessments of diagnostic images that result in new and/or more cost-effective diagnostic approaches, and new techniques for the design and monitoring of therapy. The lab processes over 1,200 clinical cases to deliver relevant visualization and analysis of medical imaging data at Stanford. The lab is co-directed by Dominik Fleischmann, MD, Roland Bammer, PhD and Sandy Napel, PhD. Contact: Dominik Fleischmann, MD / d.fleischmann@stanford.edu


Communication is at the heart of scientific advancement and innovation. This quarter, the Stanford Cardiovascular Institute members published over 350 original manuscripts and reviews, further contributing to our understanding of cardiovascular biology and disease. Here, we highlight selected manuscripts by our members.


Leadership

Joseph C. Wu, MD, PhD
Director, Stanford Cardiovascular Institute
Simon H. Stertz, MD, Professor of Medicine and Radiology

Robert A. Harrington, MD
Arthur L. Bloomfield Professor of Medicine
Chair, Dept. of Medicine

Ronald L. Dalman, MD
Walter C. and Elsa R. Chidester Professor of Surgery
Chief, Division of Vascular Surgery

Stephen J. Roth, MD, MPH
Professor and Chief, Pediatric Cardiology
Director, Children’s Heart Center

Dominik Fleischmann, MD
Professor, Dept. of Radiology
Chief, Cardiovascular Imaging

Michael Snyder, PhD
Professor and Chair, Dept. of Genetics
Director, Stanford Center for Genomics and Personalized Medicine

Kenneth Mahaffey, MD
Professor, Dept. of Medicine
Vice Chair of Medicine for Clinical Research

Y. Joseph Woo, MD
Norman E. Shumway Professor in Cardiothoracic Surgery
Chair, Dept. of Cardiothoracic Surgery

Mark Nicolls, MD
The Stanford Professor of Pulmonary and Critical Care Medicine, Dept. of Medicine, Chief, Pulmonary and Critical Care Medicine

Alan Yeung, MD
Li Ka Shing Professor of Medicine
Co-Chief (Clinical), Division of Cardiovascular Medicine

Tom Quertermous, MD
William G. Irwin Professor of Medicine
Co-Chief (Research), Division of Cardiovascular Medicine

Paul Yock, MD
Martha Meier Weiland Professor, Bioengineering and Medicine; and Professor, by courtesy, of Mechanical Engineering, Director, Byers Center for Biodesign

Marlene Rabinovitch, MD
Dwight and Vera Dunlevie Professor in Pediatric Cardiology