

## Successful Stanford Drug Discovery Symposium 2021

April 19-20, 2021, was the first virtual Stanford Drug Discovery Symposium. As a virtual event, we were able to reach a far broader audience. There were over 5,400 registrants and over 7,400 individuals logging in to view the talks. Individuals were from the US, Europe, Asia, Australia, North and South America, and Africa, reflecting how our meeting has progressed and gained international recognition over the last five years.

The 45 Speakers, moderators, and panelists represented pharmaceutical and biotech companies, government policy makers, Nobel laureates and academic leaders, scientists, editors from major journals, venture capitalists, and angel investors. They presented their groundbreaking work and perspectives in the field of drug development and provided insights into how the COVID-19 pandemic adjusted how they worked.

The Symposium also honored Drs. Doug Lowy and John Schiller from the National Cancer Institute with the 2021 Lifetime Achievement Award for their work in HPV-associated cancers and the HPV vaccine. Read about more highlights on page 4.

Mark your calendars for next year's event - **SDDS 2022 will be held April 25-26!**

View recordings of the SDDS 2021: <https://med.stanford.edu/cvi/events/2021-drug-discovery-conference/event-recordings.html#day1>



## Faculty Position Opening: VA Palo Alto Health Care System and Stanford CVI

The VA Palo Alto Health Care System (VAPAHCS) and the Cardiovascular Institute at Stanford Medicine are seeking to recruit an outstanding academic cardiologist to join their full-time faculties. This appointment will be at the rank of Assistant Professor in the Medical Center Line at Stanford University School of Medicine and jointly supported by the Medical (Cardiology) and Radiology Services at VAPAHCS, Departments of Medicine (Cardiovascular Medicine) and Radiology at Stanford University, and the Stanford Cardiovascular Institute. A major objective of this recruitment is to identify a highly qualified individual with broad experience in clinical cardiology, strong expertise in basic or translational cardiovascular research, and the potential to stimulate and lead interdisciplinary collaborations among clinicians and scientists from the supporting services/departments, as well as the rest of VAPAHCS-Stanford research communities. The successful candidate will be expected to lead a prolific research group with a focus on structural heart disease, cardiovascular imaging, or stem cell biology. [Read the full announcement.](#)



## Faculty Position Opening: Cardio-Oncologist

The Division of Cardiovascular Medicine in the Department of Medicine and the Cardiovascular Institute at Stanford Medicine are seeking a board-certified Cardiologist with expertise in Cardio-Oncology to join the faculty as Assistant, Associate or Professor in the Medical Center Line or University Tenure Line. The predominant criterion for appointment in the University Tenure Line is a major commitment to research and teaching. The major criteria for appointment for faculty in the Medical Center Line shall be excellence in the overall mix of clinical care, clinical teaching, scholarly activity that advances clinical medicine, and institutional service appropriate to the programmatic needs the individual is expected to fulfill. The candidate should possess an MD or MD/PhD, be board certified in Cardiovascular Disease, and have current basic science/translational science research expertise in cardio-oncology. Additionally, we are seeking a candidate who is an outstanding clinician and is a nationally recognized or upcoming clinical and academic leader in the field. Applicants should submit a curriculum vitae, statement of research interests, and the names of three references (who will not be contacted without your permission) to search committee chair, Dr. Hannah Valentine at: <http://facultyapplication.stanford.edu/>.



# Standing Against Asian American Racism

Dear Colleagues and Friends,

We write to you to share our anger and frustration over the despicable violence that took place in Georgia on March 16th where 3 Asian/Asian American-owned business were targeted and 8 innocent lives were lost, including 6 Asian/Asian American women. This incident punctuated a year that has seen a nearly 150% increase in anti-Asian hate crimes across the country, including here in the Bay Area.

As members of a minority group that is expected to be quiet, polite, and obedient, we feel compelled to speak out and to let you know that many if not all Asian American and Pacific Islanders (AAPIs) in this country experience profound racism and discrimination. As children, we were kicked, punched, spat upon, called “Chink”, harassed with slanted eye gestures or told to “go back to your own country.” Yet we were led to believe this was just an expected part of being different.

As we became a part of the medical profession, the sense of alienation was reinforced by constantly hearing from our patients, our professors, our classmates, our colleagues: “You all look alike,” “you are too quiet,” “your English is pretty good,” reminders that we never fully belong. These near-universal experiences of racism by ourselves and our AAPI peers growing up in the US unfortunately persist to this day.

For all of AAPI students, staff, faculty, we are here for you and support you during this most difficult time. We understand your fears and your frustrations and want you to know that you have allies here at Stanford who will help you with what you need. For our non-Asian colleagues who are willing to take a stand against anti-Asian hate and violence, we ask that you do these three things: 1. Reach out to your friends in the AAPI community and let them know that you are thinking about them, 2. Call out racism and stop it anytime you see it happening to make it known that anti-Asian racism is not tolerated by anyone whether or not s/he is a member of the AAPI community, and 3. Contact your government officials and ask them what they are doing right now to support the AAPI community.

This past year of pandemic plus the scapegoating by the previous administration have led to an alarming rise in abhorrent acts of violence on top of an always-present current of racism against AAPIs. Now more than ever we need to show our support for one another and unite in our efforts against racism, sexism, and violence. Will you stand with your Asian American and Pacific Islander colleagues and friends?

Yours sincerely,

Joseph C. Wu, MD, PhD, Director, Cardiovascular Institute

Joy Y. Wu, MD, PhD, Vice Chair for Basic Science, Department of Medicine

Sean M. Wu, MD, PhD, Associate Professor of Medicine and, by courtesy, Pediatrics



Joseph C. Wu, MD, PhD



Joy Y. Wu, MD, PhD



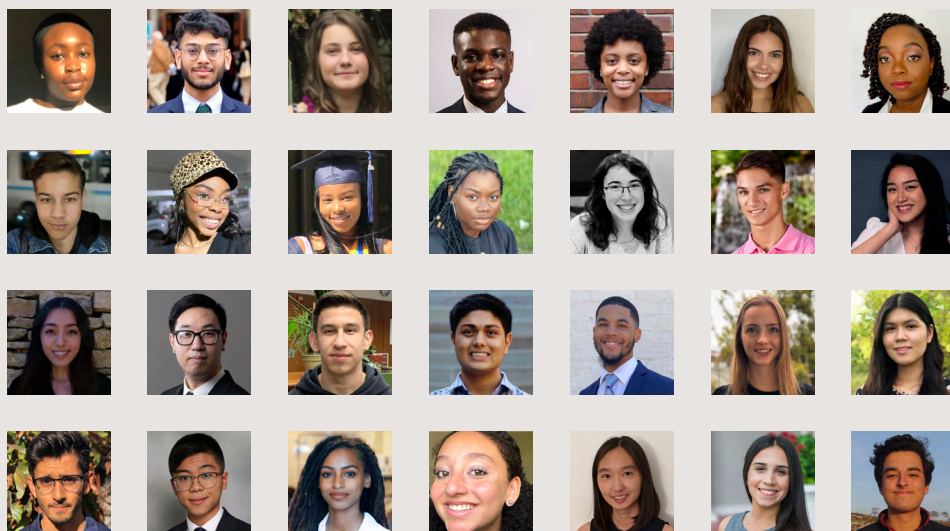
Sean M. Wu, MD, PhD

## Welcome 2021 Undergraduate Summer Researchers!

This June, 28 exceptional undergraduate students from across the globe began a virtual research program with Stanford CVI faculty research mentors. Support for this program includes two funding sources specifically devoted to promoting diversity in cardiovascular research and medicine (AHA SURE and NIH NHLBI R25).

### Final Symposium - August 9th

For more information about the program please [visit our website](#), check out the [public events calendar](#), or contact [cvi\\_outreach@stanford.edu](mailto:cvi_outreach@stanford.edu).





## Gambhir Symposium

Monday, July 19, 2021

8:30am - 4:00pm

Join us to celebrate  
Sam Gambhir's legacy, impact  
and scientific achievements!

### Welcome/Opening Remarks



**Lloyd Minor, MD**  
Stanford University



**Garry Gold, MD**  
Stanford University



**Joesph Wu, MD, PhD**  
Stanford University



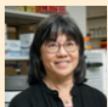
**Aruna Gambhir, MS, MBA**  
CellSight Technologies

### Keynote



**Joseph DeSimone, PhD**  
Stanford University

### Session 1: Molecular Imaging



**Anna Wu, PhD**  
City of Hope Medical Center



**Simon Cherry, PhD**  
University of California, Davis



**Michael Phelps, PhD**  
University of California, Los Angeles



**Katherine Ferrara, PhD**  
Stanford University

### Session 2: Cancer Early Detection



**Norman Sharpless, MD**  
National Cancer Institute



**Ralph Weissleder, MD, PhD**  
Harvard Medical School



**David Suhy, PhD**  
Earli



**Sangeeta Bhatia, MD, PhD**  
Howard Hughes Medical Institute

### Session 3: Precision Health



**Roderic Pettigrew, MD, PhD**  
Texas A&M University



**Michael Snyder, PhD**  
Stanford University



**Jessica Mega, MD, MPH**  
Verily, Google Health  
Stanford University



**Robert Califf, MD**  
Verily, Google Health  
Stanford University and Duke University

For More Information and Registration:  
<http://gambhir.stanford.edu>



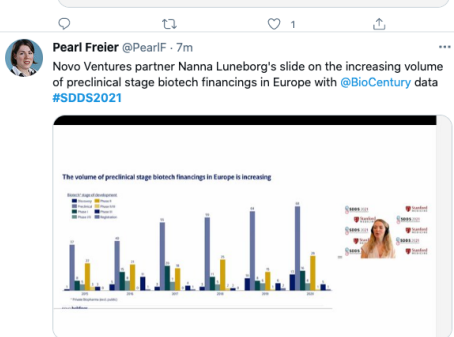
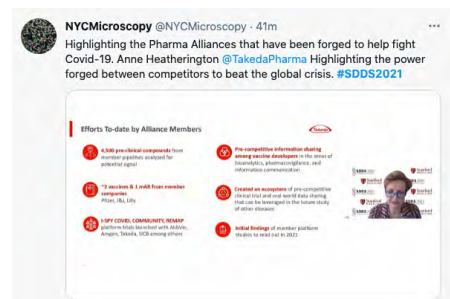
# Stanford Drug Discovery Symposium 2021 Highlights

During the Stanford Drug Discovery Symposium (SDDS) 2021, we heard about COVID-19 therapies, and the amazing work behind generating the Pfizer, Moderna, and Johnson & Johnson vaccines. We heard about treatments in cancer, heart disease, and rare orphan diseases, and about advances in discovery research. We also heard from a panel of editors from major scientific journals on how COVID-19 has influenced the scientific review process and how science news has been presented to the public. There was also the opportunity to hear about health care policy, and to learn more about how pharmaceutical, biotech, and start-up companies are pushing forward advancements in new technology and drug manufacturing. These exciting talks and panel discussions were enhanced by audience participation, both during panel sessions and via social media.

2021 Featured Speakers: **Hal Barron**, GlaxoSmithKline; **Michael Basson**, Nature Medicine; **Robert Califf**, Verily Life Sciences; **Andrea Carfi**, Moderna; **Carmen Chang**, New Enterprise Associates; **Stanley Crooke**, Ioanis Pharmaceuticals; **Jürgen Eckhardt**, Leaps by Bayer; **Levi Garraway**, Genentech/Roche; **Helene Gayle**, The Chicago Community Trust; **Anne Heatherington**, Takeda; **Thomas Hudson**, AbbVie; **Mir Imran**, Rani Therapeutics; **Kathrin Jansen**, Pfizer; **Nina Kjellson**, Canaan; **Peter Kim**, Stanford; **Brian Kobilka**, Stanford; **Roger Kornberg**, Stanford; **Douglas Lowy**, National Cancer Institute; **Nanna Luneborg**, Novo Holdings; **Fady Malik**, Cytokinetics; **Mathai Mammen**, Janssen Pharmaceutical; **Joan Mannick**, Life Biosciences; **Ken Mills**, REGENXBIO; **Michael Nedelman**, CNN; **Philip Pizzo**, Stanford; **Andrew Plump**, Takeda; **Camille Samuels**, Venrock; **Serge Saxonov**, 10X Genomics; **John Schiller**, National Cancer Institute; **Marcus Schindler**, Novo Nordisk; **Orla Smith**, Science Translation Medicine; **Young Sohn**, HARMAN; **Janet Woodcock**, FDA; **George Yancopoulos**, Regeneron; **Taiyin Yang**, Gilead; **Wendy Young**, Genentech; **Elias Zerhouni**, formerly with Sanofi, NIH, and former US Presidential Science Envoy.

Video recordings of talks and panel discussions are available on our website: <https://med.stanford.edu/cvi/events/2021-drug-discovery-conference/event-recordings.html#day1>

**We hope you can join us next April 25-26 for SDDS 2022.**



## Donate to the Stanford Cardiovascular Institute

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](mailto:joewu@stanford.edu) or **Cathy Hutton**, Senior Associate Director, Medical Center Development at [cathy.hutton@stanford.edu](mailto:cathy.hutton@stanford.edu).

For more: <http://med.stanford.edu/cvi/support-our-research.html> and <http://cvi.stanford.edu>



Joseph C. Wu,  
MD, PhD



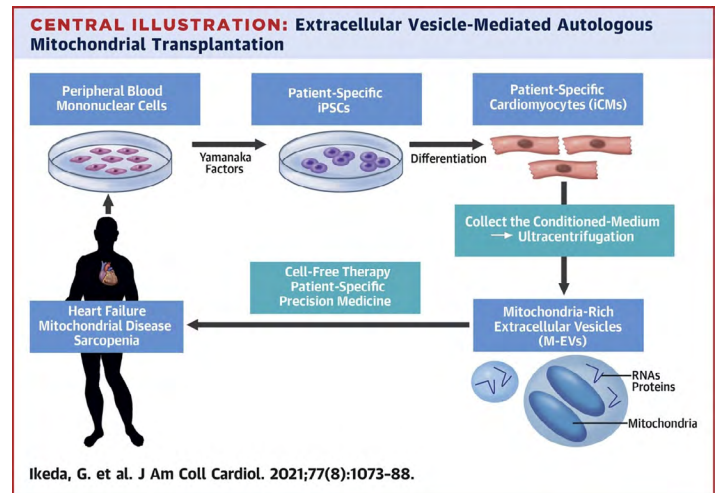
Cathy Hutton,  
MBA

The heart is an amazing muscle – able to pump about 2,000 gallons of blood through the body in one day. Two thousand gallons of blood that are necessary for the body to function. To achieve that fantastic feat, the heart must use energy, just as any of us must expend energy to lift and move heavy objects. In cardiomyocytes (cardiac muscle cells), most of the cell's energy is in the form of ATP produced by mitochondria.

When the heart is not able to pump as efficiently as needed, it results in heart failure. Importantly, heart failure is an imbalance between energy supply and demand. There is decreased ATP (energy) production while also increased energy demands from the failing heart, resulting in contractile abnormality and myocardial (heart muscle) dysfunction. As an energy imbalance is at the apparent root of heart failure, it is essential to develop a therapy that targets the intracellular energy supply directly, creating the potential for a curative therapy.

That critical need was recently addressed by a team of researchers led by first author Gentaro Ikeda, MD, PhD, and senior author Phillip Yang, MD, and published in the *Journal of the American College of Cardiology*. The team established a preclinical proof-of-concept that they could enhance cardiac function by transferring mitochondria and restoring myocardial energy production.

Their work relied on two important considerations: (1) Extracellular vesicles can transfer cargo to the recipient cells and mitochondria can exist inside the EVs, and (2) induced pluripotent stem cells (iPSCs) have tremendous therapeutic potential for cardiovascular disease treatment. Patient-specific iPSCs can be made into cardiomyocytes (iCMs) that produce EVs with functional mitochondria. Using iCMs, the team was able to show that transfer of mitochondria by EVs restored the energy needed by heart muscles cells to then also restore the normal contractility of injured heart muscle cells. Further, injection of EVs containing functional mitochondria improved cardiac function in mice after damage resulting from a heart attack. This critical study demonstrated the feasibility of using EVs to transfer mitochondria as a potentially curative treatment for heart failure.



Patient specific iPSCs can be generated and made into cardiomyocytes (iCMs). The resulting extracellular vesicles that contain fully functional mitochondria can be collected and used as a therapy to address the energy imbalance characteristic of heart failure.



Gentaro Ikeda, MD, PhD Phillip Yang, MD

## CVI Seed Grant Awards Fall 2021



**Eligibility:** CVI members who are Stanford faculty or instructor

Our goal is to ignite and support new ideas that will change how we diagnose and treat cardiovascular disease. To achieve this mission, the CVI is offering two calls for Seed Grant Proposals. We highly encourage proposals that establish new interdisciplinary collaborations.

Visit our website for any updates and informatoin:  
<https://med.stanford.edu/cvi/funding-opportunities/current-seed-grants.html>



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## Program Directors

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- Robert A. Harrington, MD, FACC
- David E. Kandzari, MD, FACC
- Alan C. Yeung, MD, FACC
- Vinod H. Thourani, MD, FACC
- Joseph C. Wu, MD, FACC
- Joseph I. Miller, III, MD, FACC
- Mani A. Vannan, MBBS, FACC

- ◆ Hot topics in cardiovascular medicine
- ◆ Conversation with global experts and legends
- ◆ Focused, expert reviews and updates
- ◆ Premium educational experience
- ◆ Exceptional value: 20 AMA PRA Category 1 Credits™

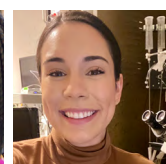
[Register Now](#)
[Download Agenda](#)
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## *How Does Research Experience Shape Your Future?* By Adrienne Mueller, PhD

Exposure to research can be transformative and two young scientists, who spent their pandemics working as research assistants in the lab of Dr. Joseph C. Wu, Director of the Stanford Cardiovascular Institute, explain how they have come to the decision to pursue higher degrees. Exposure to research has been valuable and inspiring in very different ways for both Jessica Malisa and Nicole Lopez, but they share passion to get at the root of hard questions. Jessica was motivated by directly seeing how patient care could be translated into scientific questions,



Jessica Malisa



Nicole Lopez

“Working in Joe’s lab was really great because I would see how a physician scientist would come to a question based on something that their patients were personally experiencing. Seeing that translational pipeline: from meeting the patient, to identifying a problem, to starting a study - was great for me.” Whereas Nicole has been motivated by the fundamental ‘whys,’ “I want to understand how the molecular mechanisms and cellular process – the processes that make us, that are us – go awry in disease and death. Studying science helps me make meaning out of life and why we’re here.” This Fall, Jessica will be starting her MD at the University of Southern California and Nicole will be starting her PhD at the University of California, San Diego.

But the decision to pursue advanced degrees wasn’t a foregone conclusion; both Jessica and Nicole struggled with doubts about their abilities and whether it was the right fit. When asked what advice they’d give to students trying to decide whether to pursue careers in science or medicine, Jessica and Nicole both urged students to overcome their personal demons. Academia and medicine will give both Jessica and Nicole the opportunity to support others in ways that are especially meaningful to them. For Nicole, “I want to have a position in science, where I can not only do science that is meaningful to me and helpful for others, but a position where I can mentor other students. Students are so impressionable, and I think they just need exposure.” And for Jessica, “I know that there’s a big gap in health care for minority communities and that causes a lot of medical distress. That’s part of the reason I want to go into medicine - to be a doctor for those people and those populations that are usually overlooked.” What’s next for Jessica and Nicole? Jessica is thrilled to start her MD, “Because the curriculum emphasizes not only being a physician and a scientist, but also just a model citizen that contributes to the world around you.” And Nicole is excited to start her PhD, “To understand disease and create new therapeutics - that’s the dream I hope I can spend my career pursuing.”

## CVI Trainee Mentorship Program



- First-hand advice on career and research goals
- 53 faculty mentors, spanning 16 Stanford departments
- Structured program makes mentorship easy and effective

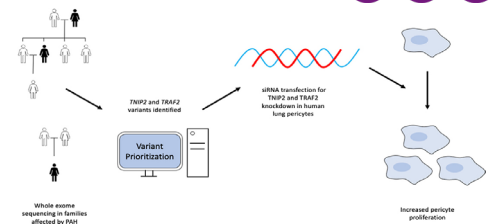
*Join the program!*

<https://med.stanford.edu/cvi/education/cvi-mentorship-program.html>

## Using Patient Genes to Further Understand Pulmonary Arterial Hypertension by Amanda Chase, PhD



Pulmonary arterial hypertension (PAH) is a rare, progressive disorder in which the blood vessels in the lung are narrowed or blocked, slowing blood flow through the lungs and making the heart work harder. The exact cause of PAH is unknown, although it is known that 15-20% are inherited due to a change or mutation in a gene. Of those, 20% are a result of an unknown gene mutation. Understanding what mutations contribute to PAH could significantly impact patient care.



A team from Stanford University, including CVI-affiliated senior author Vinicio de Jesus Perez, MD, and led by Dr. Jair Tenorio, from Spain, collaborated to identify two novel variants that cause PAH. Their findings were recently published in *Frontiers in Medicine*.

They used whole-exome sequencing (WES) on two unrelated families with PAH. Genes are the recipe for making proteins and are made up of parts that are used to make the protein (exons; game highlights) and other information. WES specifically reads the exons of most genes at once to find any gene changes, or variants. By comparing families with PAH, the researchers were able to identify two novel variants that contributed to PAH: TNIP2 and TRAF2. Both genes are involved with inflammation and immunity, and the team was able to show that the variants likely increased susceptibility to PAH by their ability to change immune responses and to drive abnormal cell growth in the vasculature of the lungs, ultimately leading to narrowing of the arteries. This is the first report to document a link between TNIP2 or TRAF2 loss of function and PAH in humans.

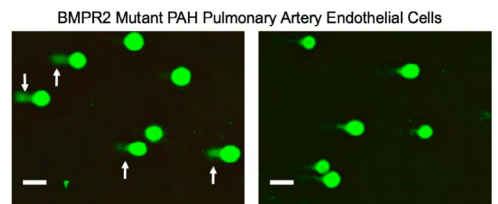


Vinicio de Jesus Perez, MD

## Reversing Genetic Damage in Pulmonary Arterial Hypertension By Adrienne Mueller, PhD



Our blood vessels' inner surface is lined by a single layer of specialized cells called endothelial cells, that have multiple functions including maintaining vascular contractility and permeability and preventing inflammatory cell invasions. When endothelial cells fail in their function, the effect on our system can lead to a host of disorders including a devastating disease of the lungs called pulmonary arterial hypertension (PAH). There are currently no existing treatments that reverse the narrowing of the large vessels or regenerate lost microvessels. To develop better treatments, a clearer understanding of the mechanisms underlying PAH is needed.



Failure of DNA repair can eventually lead to cell death and contributes to the loss and narrowing of blood vessels in PAH. It was not known what caused DNA damage or impaired the ability of cells to repair damaged DNA in PAH, but previous studies led the Rabinovitch lab to believe that this was directly related to a gene, BMPR2, that is mutant or deficient in PAH. One of the roles of BMPR2 is to activate a pair of DNA-repair proteins: PPAR $\gamma$  and p53. The question that had not been answered is what genes are controlled by these proteins that are critical for endothelial cells to repair damaged DNA, to regenerate lost vessels and reverse narrowing of larger arteries that can lead to PAH.

The comet tails reveal DNA damage in PAH endothelial cells that is reduced by Nutlin treatment in conjunction with induction of DNA repair genes owing to p53 transcriptional activity.



Jan Hennigs, MD



Marlene Rabinovitch, MD

First author Jan Hennigs, MD, and senior author Marlene Rabinovitch, MD, reported in *Circulation Research* that PPAR $\gamma$  and p53 join to form a complex in response to DNA damage that regulates a host of downstream genes influencing cell survival, regeneration, and DNA repair. They then deployed a pharmacological intervention, Nutlin, to increase p53 levels in a mouse without BMPR2 in endothelial cells, allowing the PPAR $\gamma$ -p53 complex to reform. Once the complex was restored DNA damage was repaired, microvessels were regenerated, and the narrowing of the larger arteries reversed. The investigators have therefore identified a novel strategy for reactivating a molecular genetic repair system that is otherwise not functioning in PAH.



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## PH AT STANFORD **PODCAST**

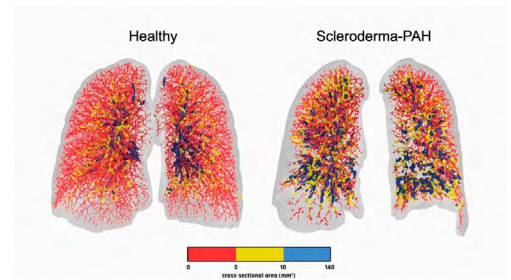
RESEARCH • EDUCATION • TREATMENT • PATIENT CARE

A new podcast series from the **Vera Moulton Wall Center for Pulmonary Vascular Disease at Stanford**, with the goal to eradicate pulmonary vascular disease by discovering fundamental causes, developing innovative therapies, disseminating crucial knowledge, and delivering transformative care.

[Listen >](#)

Scleroderma, from the Greek ‘hard skin,’ is a condition that afflicts approximately 100,000 people in the US and is characterized by a buildup of scar tissue in the skin and other organs. Scleroderma is an autoimmune disorder, meaning that the body’s immune system mistakenly identifies its own healthy tissue as a foreign substance and attacks it, causing tissue damage. One of the most serious and potentially fatal complications patients with scleroderma develop is a lung condition called pulmonary arterial hypertension (PAH). Current medications to treat scleroderma-associated PAH all act by dilating lung blood vessels to reduce blood pressure and there is currently no treatment that acts directly against the underlying autoimmune response.

In their recent *American Journal of Respiratory and Critical Care Medicine* article, first author Roham Zamanian, MD, and senior-author Mark Nicolls, MD, report the results of a 13-year clinical trial devoted to identifying an immunotherapy treatment to scleroderma-incited PAH. For their study, they chose to target B-cells, a type of immune cell that is thought to contribute to the autoimmune response in scleroderma. This trial is the first to evaluate the safety and efficacy of an entirely new, immunotherapy-based treatment using B-cell depletion. The investigators identified a trend in the data that suggested that the B-cell depleting drug rituximab reduces symptoms of PAH in patients. A secondary analysis then revealed a significant improvement in lung function caused by rituximab treatment. Future work is necessary to determine rituximab’s specific mechanism of action and to further investigate the potential of this promising drug for treating scleroderma-associated PAH.



Loss of distal lung vasculature in scleroderma-PAH, as visualized by blood vascular volume compartments on chest computed tomography. Image courtesy of Fluida.



Roham Zamanian, MD

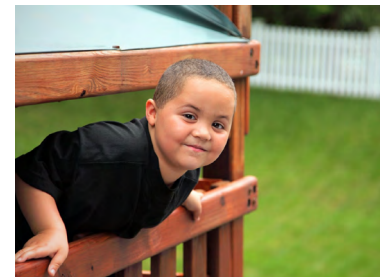


Mark Nicolls, MD

### *Study Helps Latino Children Manage Obesity Over Two Years* By Erin Digitale

A three-year intervention designed to reduce weight gain in overweight and obese Latino children generated improvements in body mass index and many other health measures during the trial’s initial two years, according to a study led by researchers at the Stanford University School of Medicine. The finding could help clinicians, health educators and policymakers in their approach to reducing childhood obesity in populations at risk for the condition.

A paper describing the research was published online April 29 in *The Lancet Diabetes & Endocrinology*. The study enrolled 241 Latino children and their families living in low-income neighborhoods in the Bay Area. All children were 7-11 years old and overweight or obese when the research began. The children were randomized to two groups. The treatment group participated in a multifaceted program that involved the children’s families, communities, and primary health care professionals. The control was designed to provide families with a beneficial treatment of their own through the entire study.



In the United States, children from low-income, nonwhite populations are more likely to be obese and suffer from related health problems than white children and children from higher socioeconomic tiers. Stuart Monk/Shutterstock.com.

When the study began, about three-quarters of participants were classified as obese, with a BMI above the 95th percentile for their age and sex. The children in the treatment group maintained significantly healthier BMIs than those in the control group in the first two years of the study. “The changes we saw are very positive, and good evidence that there’s a lot of promise for this community-based, multi-level model of intervention,” said Thomas Robinson, MD, professor of pediatrics and of medicine at Stanford, who led the study. “It motivates us: There’s more to learn, and more we can improve on.”



Thomas Robinson, MD



## CVI Proposal Development Resource

Questions about how to apply for a grant at Stanford? Steps for a fellowship, career development award, research, or collaborative grant? Templates to guide your proposal development?

Visit our new website to find answers to those questions and more.

<https://med.stanford.edu/cvi/funding-opportunities/research-development.html>,

or contact: [cvi\\_grants@stanford.edu](mailto:cvi_grants@stanford.edu)



Pulmonary arterial hypertension (PAH) is a severe and chronic disease, in which the blood vessels of the lung progressively narrow, and some become completely occluded. When this happens, PAH becomes life-threatening. Although the cause of PAH is poorly understood, we know that individuals with PAH have abnormal growth of the smooth muscle cells of the lung blood vessels - responsible for the narrowing and blockage of the vessels. Blood can only flow through the remaining vessels that have narrow openings, if the pressure is very high. Because the side of the heart pumping blood to the lungs must maintain an ever higher pressure, the heart muscle eventually weakens and fails.

Previous studies have shown that abnormal cell growth is associated with increased sugar metabolism – a means for our cells to produce energy. Suppressing sugar metabolism in lung smooth muscle cells can reduce cell proliferation by depriving the cells of the energy they need to divide. While it is known that cells that are rapidly dividing have intense energy demands, products of metabolism or metabolites can also drive changes in genes and proteins that may be necessary for growth. A study recently published in *Circulation* and led by first authors Dan Li, PhD, and Ning-Yi Shao, PhD, and by senior author Marlene Rabinovitch, MD, found a single enzyme that connected energy demands with the genes required for rapid growth of smooth muscle cells in PAH. Li and Shao et al identified the specific mechanism underlying lung blood vessel narrowing in PAH: excessive activity of the sugar-metabolizing protein ALDH1A3 leads to the activation of a master transcription factor that increases expression of genes required for excessive lung smooth muscle cell proliferation and produces the energy the cells need to divide. When they reduced the amount of ALDH1A3 in the smooth muscle cells, pulmonary hypertension was prevented because the vessels did not narrow with excessive smooth muscle cell growth. ALDH1A3 is therefore a potential drug target in PAH, if it can be delivered to the smooth muscle cells of lung circulation.



Dan Li, PhD

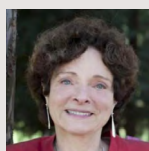


Ning-Yi Shao,  
PhD



Marlene  
Rabinovitch, MD

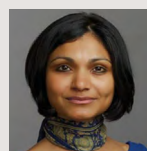
## Currently Recruiting for Postdocs



### Helen Blau Lab

Seeking creative, highly motivated, and team-oriented scientists to perform cutting-edge research on **aging and regenerative medicine**, including rejuvenation of multiple aged tissues. Successful candidates will work with a multidisciplinary, dynamic research team. Currently seeking both a Senior Research Scientist and a postdoctoral fellow. The senior scientist candidate will have a key role in directing a team of creative highly motivated researchers.

[Find out more!](#)



### Sushma Reddy Lab

Seeking a postdoctoral scientist with a background in **vascular biology and/or mitochondrial metabolism**. The overall goal of the Reddy Lab is to understand the cellular and molecular adaptation of the hypertrophied and failing right ventricle with a focus on angiogenesis, endothelial cell dysfunction, and endothelial cell metabolism. Candidates must have prior experience in cell and molecular biology, endothelial cell biology and genetic mouse models.

[Find out more!](#)



### Yasuhiro Shudo Lab

Seeking a postdoctoral researcher to **accelerate tissue engineering** studies. Competitive applicants will be interested in any of: stem cell biology, biochemistry, bioengineering, biomechanical engineering, cardiac surgery, computational biology, bioinformatics, or biostatistics. Candidates must hold a PhD/MD in a relevant research field, have strong laboratory, analytical, organizational, and communication skills; and be able to work independently, as well as part of a team.

[Find out more!](#)



### Joseph C. Wu Lab

Seeking a creative, motivated postdoctoral researcher to perform cutting-edge research on **epigenetics of heart disease using iPSCs and genome editing**. Candidate must hold a PhD and/or MD in a relevant field and have strong laboratory, analytical, organizational, and communication skills. Successful candidates will work independently in a dynamic research team composed of molecular and cell biologists, biochemists, and non-invasive imaging specialists.

[Find out more!](#)

## CVI's Dorothy Dee and Marjorie Helene Boring Trust Award

Applications due Sept. 15, 2021

Supporting Stanford medical students developing research solutions that impact how we treat and prevent cardiovascular diseases.

This award provides up to \$15,000 that can be applied to research-related supplies and expenses, educational costs and tuition, and travel to present research at a national conference. [Find out more!](#)



## *Stanford Health Care Recognized for Mitral Valve Repair* By Tracie White

Mia Cadua underwent surgery for mitral valve repair at Stanford Health Care, which was recently recognized for its excellent record with the procedure. Two years ago, when she was just 28, Mia Cadua discovered that she had a heart condition called mitral valve prolapse, which, if it worsened, could be fatal. Last fall, things did get worse. Cadua started experiencing shortness of breath and fatigue. She went back to her doctors in Fairfield. Heart-imaging tests showed that her defective mitral valve was failing. Facing heart surgery, Cadua had some serious decisions to make. One of the most significant was whether to repair the valve or replace it.



Mia Cadua underwent surgery to repair her mitral valve at Stanford Health Care. Photo credit: Mia Cadua.

After interviewing numerous cardiologists and surgeons, Cadua decided to have the valve repaired at Stanford Health Care, a designated a Mitral Valve Repair Reference Center — one of only eight in the country, and the only one in the West. The designation, made by the American Heart Association and Mitral Foundation, recognizes best practices in repairing mitral valves. The designation was established to encourage more patients with severe mitral valve prolapse to get the valve repaired rather than replaced, according to a press release from the American Heart Association. Clinical guidelines, the release said, recommend repair over replacement whenever possible. But not all medical centers have surgeons capable of performing this advanced procedure.



Joseph Woo, MD

“I would say that at least one-quarter of the thousands of cases across the country of patients with severely defective mitral valve prolapse, which could technically be repaired, are either having the valve replaced or not operated on at all,” said Joseph Woo, MD, professor and chair of cardiothoracic surgery at Stanford Medicine and the Norman E. Shumway Professor. “Now, with these new designations, patients can be referred to a center like ours with the expertise to do these complex repairs.”

Cadua said she was initially afraid of having heart surgery. She prayed for a good doctor and a good hospital. On Jan. 20, she was rolled into the operating room at Stanford Hospital. Woo performed the surgery, which lasted three hours. “The surgeons told me before the operation that they would do their best to repair it, but there was a chance they might have to replace it,” she said. When she woke up from the anesthesia following the surgery, she learned the news. “They said, ‘You got what you wanted — you got the repair,’” she said. “I was so grateful. It was a miracle.”

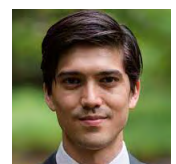
## *As Easy as ABC: From GWAS to Genes to Disease* By Adrienne Mueller, PhD

Since the completion of the human genome in the early 2000s, researchers have made big strides in their efforts to determine the relationship between genes and disease. Genome-wide association studies (GWAS) are a powerful approach that scans the genomes of many individuals to find common markers associated with a specific disease. The identified GWAS markers are often located in non-coding regions of the genome called “enhancers.” Enhancers are not genes themselves, but influence the expression of disease-related genes far away. Identifying which genes an enhancer influences is challenging in its own right, but it is made even more difficult by the fact that enhancers work differently in different cell types.



Image Credit: Zayna Sheikh, Broad Institute

Jesse Engreitz, PhD, newly appointed Assistant Professor in Genetics and the Basic Science and Engineering Initiative, as well as the Cardiovascular Institute at Stanford, recently led a project developing a model that overcomes these challenges. As reported in *Nature* and a *communication by the Broad Institute*, the ABC model connects over 5,000 genetic variants to nearly 2,250 genes across 72 traits and diseases - including heart disease and cancer. Dr. Engreitz states, “We now have the ability to look comprehensively across many cell types and, for the first time, make reasonably accurate predictions of what these non-coding enhancers and variants do.” The ABC model will accelerate our interpretation of GWAS markers and pave the way for a better understanding of how a person’s specific genome influences disease.



Jesse Engreitz, PhD



## Interested in a write-up of your publication?

Contact: [cvi\\_communications@stanford.edu](mailto:cvi_communications@stanford.edu)

Or visit:

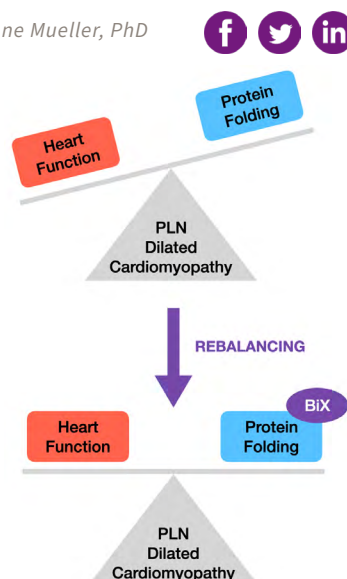
<https://med.stanford.edu/cvi/translational-research/memberpubs.html>

## Compensating for a Defective Gene: Potential Treatment for Heart Failure By Adrienne Mueller, PhD



Calcium plays a crucial role in our hearts. Our heartbeat is caused by changes in calcium in our heart muscle cells. Phospholamban is a tiny protein that plays an important role in regulating the amount of calcium and hence the force of heart contractions. When born with a genetic error in your phospholamban gene, your heart develops dilated cardiomyopathy, which is characterized by weakened heart muscle and eventual enlargement of the heart, arrhythmia and, too frequently, heart failure and death. The mechanism behind why this phospholamban mutation causes this unfortunate condition is unknown and there are no specific therapies.

Postdoctoral fellows Dries Feyen, PhD, and Isaac Perea-Gil, PhD, in the Mark Mercola and Ioannis Karakikes labs led a study, recently published in *Circulation*, that shed light on how the phospholamban mutation causes heart disease. In collaboration with researchers from the Netherlands and support from the Phospholamban (PLN) Foundation and the Fondation Leducq, the investigators harnessed stem cell technology to recreate the patients' heart muscle cells in the laboratory. The investigators then applied genome editing tools to correct the mutation. Comparing the corrected versus the patient heart cells, the defective contractility caused by the mutation was obvious. Moreover, they discovered a clue that might lead to a therapy: the mutant heart muscle cells had activated the cellular "unfolded protein response pathway". Thus, the investigators could use stem cell technology to reproduce this disorder in vitro and determine specific changes in gene expression in heart muscle cells with mutant phospholamban. "This research illustrates how iPSC disease modelling can facilitate discovery of mechanism-based therapeutics for heart disease", says senior author Karakikes.



Application of the small molecule "BIX" rebalances protein folding and restores heart function in phospholamban-associated dilated cardiomyopathy.

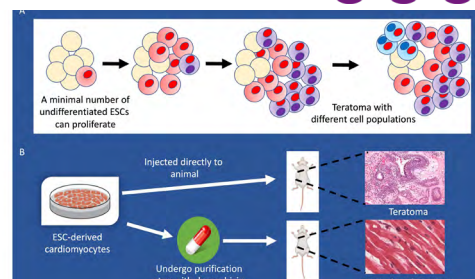
## Using a Cancer Drug to Advance Stem Cell Therapy By Amanda Chase, PhD



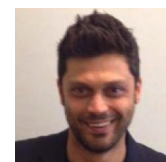
Despite the immense potential of stem cell therapy, there are still some important hurdles to consider. First, the clinical translation of pluripotent stem cell (PSC) derivatives is hindered by the tumorigenic risk from residual undifferentiated cells. Second, tumor growth can occur due to the cells continuing to multiply, a significant barrier to clinical trials using iPSC-CMs. Addressing these hurdles is critical for regenerative therapy to advance and move through clinical trials.

A team from Stanford Cardiovascular Institute (CVI), including first author Tony Chour and senior authors Evgenios Neofytou, MD, Senior Scientist at CVI, and Joseph Wu, MD, PhD, Director of CVI, established a novel method to decrease the chance of tumor formation from cardiac stem cell therapy, recently published in *JCI Insight*. In the study, researchers targeted the ability of the PSCs to continue to grow and multiply using a chemotherapeutic agent called Doxorubicin. Doxorubicin is an FDA-approved drug that has commonly been prescribed for patients with cancer, although can also be cardiotoxic.

Researchers found that a low-dose of doxorubicin is both an effective and safe means to remove the stem cells that could continue to divide while leaving the ESC-CMs that are terminally differentiated and would not contribute to tumor formation. This effective and feasible method to reduce the chances of stem cell therapy forming tumors provides a promise to advance stem cell therapies in cardiovascular clinical applications.



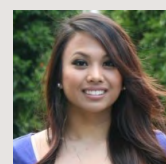
(A) An undifferentiated ESC can multiply and lead to tumor formation. (B) When ESC-CMs are injected into an animal, tumors can form from any remaining undifferentiated ESCs. When ESC-CMs are treated with a low-dose of doxorubicin before injection, no tumors form because all remaining undifferentiated ESCs are removed.



Evgenios Neofytou, MD

## CVI Staff Spotlight

**Francesca Mae G. Tongco, MS-HCA** has been with Stanford Cardiovascular Institute for just over two years. Depending on the time of year, her three main responsibilities currently include managing CVI's Frontiers in Cardiovascular Science events, providing administrative support to CVI core faculty, and developing the Cardiovascular Fellowship Alumni program. Little known fact - starting from when she was 6/7 years old and living the Bay Area, Francesca used to figure skate and wanted to be the next Michelle Kwan! Alas, she had to stop skating when she moved to Sacramento in junior high.



Francesca enjoys many things about working at CVI, but one thing in particular that she appreciates is the closeness between everyone working for the institute and the ability to learn so many new things from everyone's experiences and expertise. In exciting news, just last month Francesca completed her Masters in Health Care Administration. Congratulations, Francesca!







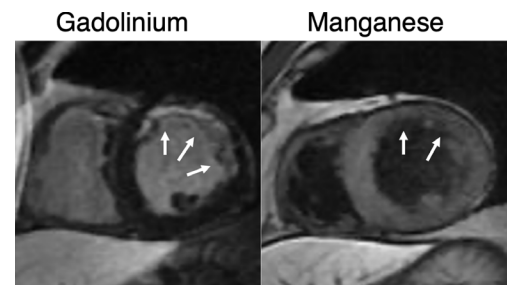
Diagram illustrating the location of a cystic tumor, lymph node, and bronchus in the right lung. The diagram shows the right lung with a cystic tumor (indicated by a black circle), a lymph node (indicated by a black dot), and a bronchus (indicated by an orange arrow). The text "Right" is written on the left side of the diagram.

[illegible]

Heart muscle cells are one of the few cell types in the body that do not regenerate. Coronary artery disease and heart attacks reduce blood flow to your heart, causing tissue death, or ischemic cardiomyopathy, which is the leading cause of death worldwide. In order to help treat or remove damaged heart tissue, the first thing you need to do is find it.

Clinicians and researchers visualize heart tissue using magnetic resonance imaging (MRI). In cardiac MRIs, contrast agents are used to distinguish between healthy and damaged heart tissue. Traditional contrast agents, such as gadolinium, indicate what parts of the heart are damaged by accumulating in damaged tissue. But, researchers have long-desired a more direct agent to measure heart cell viability: one that actually labels and distinguishes the healthy and injured cells.

Enter Manganese: a new candidate contrast agent that is similar in size and shape to calcium and is therefore able to enter only the live and injured heart cells. Moreover, because calcium is the main driver of heart electrical activity and contractions, the cell's uptake of manganese also provides a direct readout of heart health. Unfortunately, manganese's similarity to calcium, also means it may be cardiotoxic. Recently researchers have created a modified version of manganese that is less cardiotoxic, but still maintains the desirable calcium-like qualities. The big question is – how well does it work as a contrast agent?



Both gadolinium and manganese contrast agents detect heart tissue damage. Gadolinium detects fibrosis in myocardial infarction and manganese detects nonviable myocardium.



Yuko Tada, MD, Phillip Yang, MD PhD

Having previously demonstrated the utility of manganese-based contrast agents in pigs, the lab of Phillip Yang, MD, embarked on a pilot study, recently published in the *Journal of American College of Cardiology: Cardiovascular Imaging*, to compare manganese- and gadolinium-based contrast agents' ability to detect damaged heart tissue in human patients with ischemic cardiomyopathy. They found that the new manganese-based contrast agent worked – the investigators were able to distinguish damaged and healthy heart tissue in human patients. They also showed that use of this new agent had no severe adverse side effects. When they compared the readouts between the two methods, they found that the manganese-based method was more precise at determining heart cell viability, but it was still easier to discriminate damaged tissue from healthy tissue using the gadolinium-based method. The authors therefore suggest that, for now, a combined approach of gadolinium and manganese-based contrast agents will provide the most clear and sensitive information about a patient's damaged heart. This study has opened the door to the use of a new tool to directly measure heart muscle viability in humans, and future research with this method will doubtless provide valuable, clinically-relevant information for treating ischemic cardiomyopathy.

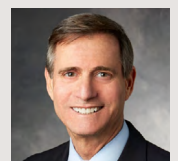
## ISCHEMIA Trial Receives Top Clinical Research Award

The *ISCHEMIA Trial*, a groundbreaking cardiovascular study coauthored by David Maron, MD, C.F. Rehnborg professor of medicine and Director of Preventive Cardiology, received the Clinical Research Forum's (CRF) prestigious Herbert Pardes Clinical Research Excellence Award in recognition of its high degree of innovation and impact on human disease.

Maron, who partnered with Judith Hochman, MD, professor and associate director of cardiology at the NYU Grossman School of Medicine, to lead the project, explains that the study was designed to determine whether adding invasive procedures to medical therapies improved outcomes for stable patients with coronary artery disease.

The first of its kind, the clinical trial revealed no difference in the likelihood of an adverse event, including heart attack and death, between those treated with surgery and those with medicine and lifestyle changes. The researchers say the findings underscore the importance of shared decision making between physicians and patients, as well as a decreased need for stenting in bypass surgery.

As noted in a *CRF announcement*, the research shows “potential to improve the management of millions of patients around the world.” The ISCHEMIA Trial was also named a *CFR 2021 Top 10 Clinical Research Achievement Award Finalist*. Maron and Hochman were honored during a virtual presentation on March 30th.



David Maron, MD

## Cardiovascular Clinical Trials at Stanford

Cardiovascular research at Stanford University is diverse and spans over 240 clinical research studies in the division of Cardiovascular Medicine alone. Stanford faculty physicians and scientists, many of whom are recognized internationally for their contributions to advancing science and knowledge of cardiac disease, conduct research aimed to treat patients suffering from a wide variety of cardiovascular issues. Cardiovascular researchers have made significant progress towards the understanding of coronary and vascular disease, endothelial function, cardiac mechanics and heart failure. There are opportunities for patients to participate in studies that may change cardiovascular care for millions of patients.

[Cardiovascular Medicine's Clinical Research Office](https://med.stanford.edu/cvmedicine/research/clinicaltrials.html) and the [Cardiovascular Institute's Clinical Trials Core](http://med.stanford.edu/cvi/translational-research/clinical-trials.html) support faculty with teams of talented Clinical Research Coordinators to move the trials and research forward in the most compliant and efficient way for the benefit of patients, and to ensure research goals are met even in the midst of COVID-19 pandemic. For more information, visit <https://med.stanford.edu/cvmedicine/research/clinicaltrials.html> and <http://med.stanford.edu/cvi/translational-research/clinical-trials.html>.

**Introduction to the Stanford Arrhythmia Service:** Linda K. Ottoboni, PhD, CNS, founded the Atrial Fibrillation Prevention and Lifestyle Management Program to help individuals reduce their cardiovascular risk. Research has shown that reducing cardiovascular risk improves atrial fibrillation outcomes. Dr. Ottoboni is also testing strategies to help patients manage the unpredictability of arrhythmias. In collaboration with Dr. Paul Wang, Dr. Sanjiv Narayan, Dr. Mintu Turakhia, and the other members of the Stanford Arrhythmia Service, Dr. Ottoboni is pursuing several research projects including: evaluating symptom management strategies that may improve patient quality of life; a multi-center clinical trial on whether bariatric surgeries improve patient outcomes; and an assessment of whether a digital health platform targeting a patient's psychometric profile can help modify behaviors to reduce cardiovascular risk factors and thereby improve access for underrepresented populations.

**Introduction to the Khush Research Team:** The Research Team of Kiran Khush, MD, including Helen Luikart RN, Research Manager, Kian Waddell ACRC, and Dave Morales ACRC, focuses on clinical research studies broadly related to the field of heart transplantation. They are leading the first prospective multi-center study of donor heart utilization in the United States, with a goal of developing risk models to guide donor heart utilization. They are currently collaborating with Interventional Cardiology colleagues to conduct a clinical trial to ameliorate chronic rejection after heart transplantation. They are also involved in several multi-center clinical trials of novel strategies for immunomodulation to prevent short- and long-term complications after heart transplantation.

**Introduction to the Cardiovascular Regeneration and Restoration Research Program:** During the last eight years, the Cardiovascular Regeneration and Restoration Program, led by Phillip Yang, MD (PI), David Lee, MD (Co-PI), Fouzia Khan CRC2, and Banu Rajaskeran ACRC, has conducted over 10 clinical trials. The NIH/NHLBI has funded over \$70M to the Cardiovascular Cell Therapy Research Network of seven leading US academic sites and completed TIME, LATE-TIME, FOCUS, PACE, SENECA, and CONCERT Trials to study acute myocardial infarction, heart failure, and peripheral vascular disease patients. Industry support completed MEMRI (FDA IND), DREAM, and CAPACITY trials to study heart failure patients. Currently, ACT, CardiAMP and DCM II trials are on-going or preparing to start.

## Black Americans are Harder Hit by Heart Disease

Black Americans disproportionately experience heart disease risk factors like high blood pressure, diabetes, and obesity.

Learn from Dr. Eldrin Lewis, cardiologist and chief of the Division of Cardiovascular Medicine at Stanford University School of Medicine, about how knowing your numbers can help protect your heart. One of the most important numbers is blood pressure.

High blood pressure is a silent problem because often people feel no symptoms when their blood pressure is elevated. High blood pressure, however, puts unnecessary strain on your heart and can cause damage. Talk to your doctor about making changes today that can positively impact the long-term condition of your heart. View the video at <https://youtu.be/JVhGBnGCYK4>



Eldrin Lewis, MD, MPH, explains how to help protect your heart.



## Courses in Cardiovascular Science and Medicine

### MED223 | Cardiovascular and Pulmonary Sciences Seminar



The purpose of this course is to familiarize students with the spectrum of basic, clinical and translational CVP research beyond their specific area of chosen investigation. After a Tuesday seminar, students will meet informally with the seminar speaker. Examples of thematic topics that will be covered include how genetics and developmental biology address mechanisms of congenital heart disease, the rationale for new drug development in atherosclerosis and cardiac protection, principles of biomechanics and computer technology in device and biomaterial development, ion channel physiology leading to anti-arrhythmic agents and the design of clinical trials, use of epidemiological studies, evidence based medicine, and design of new treatment or diagnostic algorithms. **Fall and Winter Quarter - Tuesdays and Thursdays, 12:30 - 1:20 pm | 2 credits**

Course Directors: Ngan Huang, PhD; Vinicio de Jesus Perez, MD; Edda Spiekerkoetter, MD; Ioannis Karakikes, PhD  
<https://med.stanford.edu/cvi/education/cvi-courses/med223.html>

### CTS 225 | Stem Cells in Cardiovascular Regenerative Medicine



This cardiovascular course focuses on the basic principles and translational applications of stem cells for treatment of cardiovascular diseases. Topics include the genetic modification of stem cells for precision medicine, as well as the science underlying how stem cells can be applied to regenerative medicine and drug development. Students will have the opportunity to develop their scientific reasoning and presentation skills as well as expand their professional portfolios through student-led journal club presentations and the development of a research proposal. After completion of this course, students should expect to get broad exposure to basic and translational applications of stem cell research to cardiovascular medicine, a key focus of many initiatives in both academia and the biotech industry. This course is open to graduate students, medical students, and upper-division undergraduates. **Spring Quarter - Tuesdays and Thursdays, 2:00 - 3:00 pm | 2 credits**

Course Director: Ngan Huang, PhD  
<https://med.stanford.edu/cvi/education/cvi-courses/cts225.html>

### MED 225 | Drug Development: From a Concept to the Clinic



CVI is launching a new course for the 2020-2021 academic year that is designed for medical students, trainees, basic scientists, clinicians, and clinician-scientists to provide an educational and practical perspective on the essential issues in drug development. Using a blend of seminars and dynamic workshops, the curriculum is focused on educating the audience on all stages of drug development and related research and business processes—from discovery and translational science and how to launch new projects to analyzing data, communication and interpretation of results of clinical trials, regulatory issues, and commercial considerations in product development. The emphasis will be on cardiovascular applications. Proposed seminar topics include How Drugs Are Discovered and Developed, Case Studies of the Challenges in Drug Development, Cardiac Safety, and the FDA Advisory Committee Process. **Spring and Fall Quarter - Tuesdays, 4:00 - 5:20 pm | 1 credit**

Course Directors: Peter DiBattiste, MD; Jonathan Fox, MD, PhD; Alexander Gold, MD; Jayakumar Rajadas, PhD; Philip Sager, MD  
<https://med.stanford.edu/cvi/education/cvi-courses/med225.html>

## Cardiovascular Medicine Fellowship Program

Our mission is to train future academic leaders in Cardiovascular Medicine through a tripartite commitment to clinical care, research, and education.

"The Cardiovascular Medicine Fellowship Program at Stanford University offers a rigorous but collegial training environment for individuals with an interest in developing an academic career. Intensive, individually tailored training in invasive and noninvasive clinical cardiology as well as in basic and/or clinical cardiovascular research prepares each fellow to pursue their career at the forefront of cardiology. *Come train with us!*" — Joshua Knowles, MD, PhD, Program Director

<https://med.stanford.edu/cvmedicine/education/gen-cardiology-fellowship.html>



# Recruitment for T32 Postdoctoral Training 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.

This program is directed by Joseph Wu, MD, PhD, John M. Pauly, PhD, and Koen Nieman MD, PhD.

**Currently accepting applications.**

<http://med.stanford.edu/cvi/education/cardiovascular-imaging-t32.html>

## Mechanisms and Innovations in Vascular 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.

This program is directed by Philip Tsao, PhD and Nick Leeper, MD.

**Currently accepting applications.**

<http://med.stanford.edu/cvi/education/mechanisms-and-innovations-t32.html>

## Research Training in Myocardial Biology T32 Training Grant

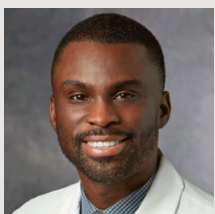
The multi-disciplinary Research Training Program in Myocardial Biology is funded by the National Institutes of Health to bring together post-doctoral fellows and faculty from six complementary areas – genetics and genomics, cellular signaling, molecular imaging, physiology and phenotyping, cardiac development 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 multi-disciplinary training.

This program is directed by Daniel Bernstein, MD, Thomas Quertermous, MD, and Euan Ashley, MRCP, DPhil.

<http://med.stanford.edu/cvmedicine/education/timbs.html>

## Welcome CVI R38 StARR Resident-Investigators!

CVI's R38 Stimulating Access to Research in Residency (StARR) program is designed to train resident-investigators in cardiopulmonary research and to accelerate their development into independent clinician-investigators. The R38 StARR program is funded by the National Heart Lung and Blood Institute. CVI is excited to welcome its 2021 cohort of resident investigators, starting July 1st. [Find out more about the program.](#)



**Tomi Obafemi, MD**  
Resident in Cardiothoracic Surgery  
MD from University of Texas Medical Branch

Research Mentor: Anson Lee, MD  
Research Project: High resolution mapping of ventricular tachycardia using a Langendorff model.



**Krishna Martinez-Singh, MD, MPH**  
Resident in Vascular Surgery  
MD from Albany Medical College of Union University

Research Mentor: Purvesh Khatri, PhD  
Research Project: The use of FlowMet-R technology to predict wound healing in critical limb ischemia patients in a wound care center setting

## Grants, Awards, Appointments and Promotions

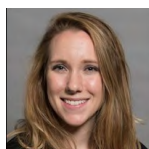


**Thomas Quertermous, MD**, William G. Irwin Professor in Cardiovascular Medicine, was awarded an NIH Research Project Grant (R01) for his project, "Identifying Tobacco-genetic Interactions Through Study of the Aryl Hydrocarbon Receptor Pathway."

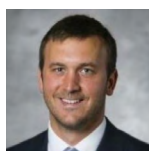
Dr. Quertermous was also awarded an NIH Research Project Grant (R01) for the project "PDGFD Regulates a Transcriptional Network to Modulate Smooth Muscle Cell Transition and Disease Risk."



**Joseph Wu, MD, PhD**, Simon H. Stertzer Professor and Professor of Radiology was awarded Honorary Lifetime Membership to the Society of Toxicology.



**Caitlin Bell, MD**, cardiology fellow in the labs of Drs. Nicholas Leeper and Irving Weissman, was awarded the Damon Runyon Cancer Research Foundation 2021 Physician-Scientist Training Award for her work towards understanding the connection between cardiovascular disease and cancer.



**Chad Weldy, MD, PhD**, fellow in cardiovascular medicine in the lab of Dr. Thomas Quertermous, was awarded an NIH Individual Postdoctoral Fellowship (F32) for his project, "A Transcriptional Network which Governs Smooth Muscle Transition is Mediated by Causal Coronary Artery Disease Gene PDGFD."



**Heyjun Park, PhD**, postdoctoral scientist in the lab of Dr. Michael Snyder, was an Emerging Leaders in Nutrition Science Finalist for her abstract, "Meal Timing-Based Dietary Patterns Are Associated With Glucose Regulation, Insulin Resistance, and Incretin Effect in Individuals at Risk for Type 2 Diabetes."



**Mario Funes Hernandez, MD** and **James Tooley, MD** will be joining Dr. Mintu Turakhia's AHA-sponsored Heart Health Technology Innovation Fellowship as fellows. They will be working building and then clinical

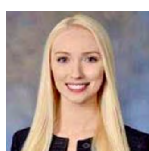
trialing technology-based remote management programs to optimize care of hypertension and heart failure in underserved and in complex comorbid populations.



**Adam Bush, PhD**, postdoctoral scientist in the lab of Dr. Shreyas Vasanawala, is starting a new tenure-track position as Assistant Professor in the Biomedical Engineering Department of the University of Texas at Austin in January 2022.



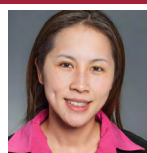
**Alice Popejoy, PhD**, postdoctoral scientist in the lab of Dr. Carlos Bustamante, is starting a new position as Assistant Professor in the Department of Public Health Sciences in the Division of Epidemiology, in the UC Davis School of Medicine.



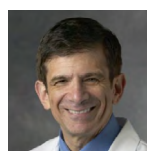
**Pauline Berens, MD**, will be joining the Vascular Surgery Integrated Residency Program this June. Dr. Berens received her medical degree from Baylor College of Medicine.



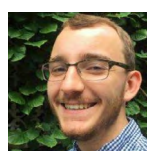
**John Cabot, MD**, will be joining the Vascular Surgery Integrated Residency Program this June. Dr. Cabot received his medical degree from the University of Texas Health Science Center at San Antonio.



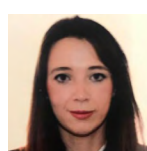
**Ngan Huang, PhD**, Assistant Professor of Cardiothoracic Surgery, was appointed as Courtesy Assistant Professor in Chemical Engineering. This appointment will foster interdisciplinary collaborations between the departments of Cardiothoracic Surgery and Chemical Engineering.



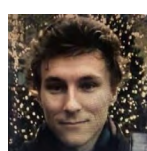
**Stanley Rockson, MD**, Allan and Tina Neill Professor of Lymphatic Research and Medicine, received the Nobility in Medical and Scientific Achievement Award at the Run/walk to Fight Lymphatic Diseases in April, 2021.



**Kevin Cyr**, a medical student in the lab of Dr. Paul Wang, received the 2021 Cardiac Electrophysiology Society and Heart Rhythm Society Young Investigator Award.



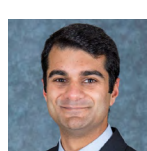
**Gema Mondejar Parreno, PhD**, a postdoc co-mentored by Drs. David Paik and Joseph Wu was awarded an AHA postdoctoral fellowship for her project "Elucidating Cardiomyocyte-Fibroblast Crosstalk Pathways in Atrial Fibrillation."



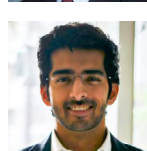
**Brian Wayda, MD, MPH**, postdoctoral fellow in the lab of Dr. Kiran Khush, was awarded second place in the Young Investigator Awards in Outcomes Research at the 2021 American College of Cardiology (ACC) Scientific Session.



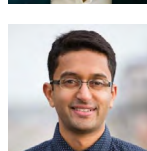
**Hao Zhang, PhD**, a postdoc in the lab of Dr. Joseph Wu, was awarded an AHA postdoctoral fellowship for his project "Elucidating the Role of Adenosine Receptor Antagonists in Cardiac Fibrosis Using iPSCs."



**Neil Kalwani, MD**, will be starting a new position as Clinical Scholar in the Division of Cardiovascular Medicine in July. He will be supported by an NIH T32 training grant through the Stanford-AHRQ Health Services Research Training Program.



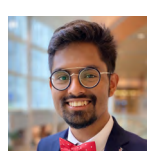
**Rohan Shad, MD**, a postdoc in the lab of Dr. William Hiesinger and co-mentored by Dr. Euan Ashley, was awarded an AHA postdoctoral fellowship for his project "Elucidating Receptor Specific Effects of Chemokine Mediated Myocardial Recovery."



**Mark Chandy, MD, PhD**, a postdoc in the lab of Dr. Joseph Wu, is starting a new position as Assistant Professor in the Division of Cardiology at the University of Western Ontario, Canada in July, 2021.



**Ruibin Feng, PhD**, will be joining Dr. Sanjiv's Narayan's Computational Arrhythmia Lab as a postdoctoral research fellow. His research interests lie at the intersection of machine learning, deep learning, medical imaging and bioinformatics, concentrating on computer-aided diagnosis and analysis for atrial fibrillation.



**Brototo Deb, MD**, will be joining Dr. Sanjiv Narayan's Computational Arrhythmia Lab as a postdoctoral fellow. He is interested in using machine-learning-based analytical approaches to gain novel, easily interpretable insights into the pathophysiological mechanisms of arrhythmias and translate that into clinical practice.



## Funding Opportunities

**For more information about funding opportunities or grant application support, please contact our Office of Research Development: [cvi\\_grants@stanford.edu](mailto:cvi_grants@stanford.edu).**

### JULY 2021

**NIH Mentored Research Scientist Development Award.** (Parent K01 – Independent Basic Experimental Studies with Humans Required). Resubmission Deadline: July 12, 2021. PA-20-191.

**Friedrich's Ataxia Research Alliance General Research Grant.** LOI Due: July 15, 2021. Application Deadline: September 15, 2021. FARA.

**Friedrich's Ataxia Research Alliance Postdoctoral Research Award.** LOI Due: July 15, 2021. Application Deadline: September 15, 2021 FARA.

**NIH/NHLBI R34 Planning Grant. Clinical Trial Pilot Studies.** (R34 Clinical Trial Optional). Resubmission Deadline: July 16, 2021. PAR-19-155.

**NIH R21 Exploratory/Developmental Research Grant. Improving Outcomes in Cancer Treatment-Related Cardiotoxicity** (R21 Clinical Trial Optional). Resubmission Deadline: July 16, 2021. PA-19-111.

**TRDRP Call for Applications.** Open July, 2021.

### AUGUST 2021

**Ruth L. Kirschstein National Research Service Award (NRSA) Individual Postdoctoral Fellowship.** (Parent F32) Deadline: August 8, 2021. PA-21-048.

**Ruth L. Kirschstein National Research Service Award (NRSA) Individual Predoctoral Fellowship to Promote Diversity in Health-Related Research.** (Parent F31 - Diversity) Deadline: August 8, 2021. PA-21-052.

**NIH Director's New Innovator Award Program** (DP2 Clinical Trial Optional) Deadline August 20, 2021. RFA-RM-21-016.

**NIH Support for Conferences and Scientific Meetings.** (Parent R13 – Clinical Trial Not Allowed), Deadline: August 12, 2021. PA-21-151.

### SEPTEMBER 2021

**NIH Directors Transformative Research Awards** (R01 Clinical Trial Optional) Office of Strategic Coordination (Common Fund) Deadline: September 01, 2021 (RFA-RM-21-017.)

**American Heart Association Predoctoral Fellowship.** Deadline: September 14, 2021.

**American Heart Association Postdoctoral Fellowship.** Deadline: September 15, 2021.

**The Thoracic Surgery Foundation Research Award.** Up to \$40,000/year for up to two years to support early-career cardiothoracic surgeon work. Deadline: September 15, 2021.

**The Thoracic Surgery Foundation STS Research Award.** Given to the highest-ranking TSF research application awarded by TSF based on merit. Deadline: September 15, 2021.

**The Thoracic Surgery Foundation Nina Starr Braunwald Research Award.** Up to \$40,000/year for 1-2 years to support the work of an early-career woman cardiac surgeon. Deadline: September 15, 2021.

**The Thoracic Surgery Foundation Resident Research Fellowship Award.** Up to \$30,000/year for 1-2 years supporting the research of a resident in cardiothoracic surgical training. Deadline: September 15, 2021.

**The Thoracic Surgery Foundation Nina Starr Braunwald Research Fellowship.** Up to \$30,000/year 1-2 years to support the research of a resident in cardiothoracic surgical training. Deadline: September 15, 2021.

**Friedrich's Ataxia Research Alliance General Research Grant.** LOI Due: July 15, 2021. Application Deadline: September 15, 2021. FARA.

**Friedrich's Ataxia Research Alliance Postdoctoral Research Award.**

LOI Due: July 15, 2021. Application Deadline: September 15, 2021 FARA.

### OCTOBER 2021

**NIH Research Project Grant.** (Parent R01 Clinical Trial Not Allowed). New application Deadline: October 5, 2021. PA-20-185.

**NIH Research Project Grant.** (Parent R01 Clinical Trial Required). New application Deadline: October 5, 2021. PA-20-183.

**NIH Research Project Grant.** (Parent R01 – Clinical Trial Not Allowed). New Application Deadline: October 5, 2021. PA-20-185.

**NIH Research Project Grant.** (Parent R01 – Clinical Trial Required). New Application Deadline: October 5, 2021 PA-20-183.

**NIH Research Project Grant.** (Parent R01 – Basic Experimental Studies with Humans Required). New Application Deadline: October 5, 2021. PA-20-184.

**NIH - R01 Improving Outcomes in Cancer Treatment Related Cardiotoxicity.** Deadline: October 5, 2021. PA-19-112.

**NIH - R01 The Mechanistic Role of the Microbiome in the Pathobiology of Heart, Lung, Blood, and Sleep Diseases.** Deadline: October 5, 2021. PA-18-784.

**Patient-Centered Outcomes Research Institute.** Broad PCORI Funding Announcements 2021. Letter of Intent Deadline: October 5, 2021. Application Deadline: January 11, 2022.

**NIH Pathway to Independence Award.** (Parent K99/R00 - Independent Clinical Trial Required). Deadline: October 12, 2021. PA-20-187.

**NIH Pathway to Independence Award.** (Parent K99/R00 Independent Clinical Trial Not Allowed). Deadline: October 12, 2021. PA-20-188.

**NIH Pathway to Independence Award.** (Parent K99/R00 Independent Basic Experimental Studies with Humans Required). Deadline: October 12, 2021. PA-20-189.

**NIH K08 - Mentored Clinical Scientist Research Career Development Award.** (Parent K08 Independent Clinical Trial Not Allowed). Deadline: October 12, 2021. PA-20-203.

**NIH K18 - Career Enhancement Award.** (K18 Basic Experimental Studies with Humans Required. Deadline: October 12, 2021. PAR-20-226.

**NIH K24 – Midcareer Investigator Award in Patient-Oriented Research.** (Parent K24 Independent Clinical Trial Not Allowed) Deadline: October 12, 2021. PA-20-186.

**NIH K24 – Midcareer Investigator Award in Patient-Oriented Research.** (Parent K24 Independent Basic Experimental Studies with Humans Required). Deadline: October 12, 2021. PA-20-192.

**NIH K24 – Midcareer Investigator Award in Patient-Oriented Research.** (Parent K24 Independent Clinical Trial Required). Deadline: October 12, 2021. PA-20-193.

**NIH Mentored Research Scientist Development Award.** (Parent K01 - Independent Clinical Trial Not Allowed). New Application Deadline: October 12, 2021. PA-20-190.

**NIH Mentored Research Scientist Development Award.** (Parent K01 - Independent Clinical Trial Required). New Application Deadline: October 12, 2021. PA-20-176.

**NIH Mentored Research Scientist Development Award.** (Parent K01 – Independent Basic Experimental Studies with Humans Required). New Application Deadline: October 12, 2021. PA-20-191.

**NIH R21 - Improving Outcomes in Cancer Treatment-Related Cardiotoxicity.** Deadline: October 16, 2021. PA-19-111.

## National and Global Cardiovascular Conferences

Please note: some events may be canceled or postponed due to COVID-19. Please check directly with event organizers.

### JUNE 2021

**Education Symposia- Noninvasive Vascular Imaging 2021.** June 10-12, 2021. Hybrid

**World Stem Cell Summit 2021.** June 14-18, 2021. Virtual

**European Society of Cardiology- EuroHeartCare: ACNAP Congress 2021.** June 18-19, 2021. Virtual

**American Society of Echocardiography- ASE 2021.** June 18-20, 2021. Virtual

**Keystone Symposia- Precision Oncology: Translating Discovery to the Clinic.** June 21-24, 2021. Virtual

**Keystone Symposia- Innovative Vaccine Approaches.** June 28-30, 2021. Virtual

**Heart Rhythm 2021- Kick Off.** June 30, 2021. Hybrid

**European Society of Cardiology- Heart Failure 2021.** June 29-July 1, 2021. Virtual

### JULY 2021

**San Diego Heart Failure Symposium.** July 9-10, 2021. Virtual

**Complex Interventional Cardiovascular Therapy.** July 16-17, 2021. Chicago, IL

**Barth Syndrome International Science, Medical, and Family Conference.** July 19-24, 2021. Clearwater, FL

**Heart Rhythm 2021.** July 28-30, 2021. Hybrid

**Cardiovascular Digital Innovations 2021.** July 29-31, 2021. Virtual

### AUGUST 2021

**2021 UCSF Vascular Symposium.** August 1-3, 2021. Napa, CA

**VESS Spring Meeting.** August 18, 2021. San Diego, CA

**BCVS Scientific Sessions 2021.** August 23-25, 2021. Virtual

**European Society of Cardiology- ESC Congress 2021: The Digital Experience.** August 27-30, 2021. Virtual

### SEPTEMBER 2021

**HFSA Annual Scientific Meeting 2021.** September 10-13, 2021. Hybrid

**Mayo Clinic- Internal Medicine review for Nurse Practitioners, Physician Assistants & Primary Care Physicians.** September 16-17, 2021. Virtual

**UCLA Heart Failure Symposium 2021: State of the Art Updates & Therapies for Advanced Heart Failure.** September 18, 2021. Hybrid

**Columbia Medicine- 9th Annual Challenges in Hypertension.** September 24, 2021. Virtual

**Mayo Clinic- Innovations in Structural Heart Interventions.** September 25-26, 2021. Virtual

**AHA Hypertension Scientific Sessions 2021.** September 27-29, 2021. Virtual

### OCTOBER 2021

**Mayo Clinic- Echo in Congenital Heart Disease.** October 1-3, 2021. Hybrid

**Vascular Interventional Advances- VIVA2021.** October 5-7, 2021. Las Vegas, NV

**2021 SCAI SHOCK Virtual Conference.** October 7-8, 2021. Virtual

**Mayo Clinic- The genetics of Heart Variation.** October 7-9, 2021. Hybrid

**Mayo Clinic- Artificial Intelligence in Cardiology.** October 14-16, 2021. Hybrid

**16th Annual Cardiometabolic Health Congress.** October 14-17, 2021. National Harbor, MD

**Mayo Clinic- Coronary Artery Disease.** October 15-17, 2021. Hybrid

**Mayo Clinic- Cases in echo, Cardiac CT, and MRI.** October 20-23, 2021. Hybrid

**Mayo Clinic- Innovations in Atrial Fibrillation Management.** October 23, 2021. Hybrid

### SAVE THE DATE

**CATCH - Caring for Adults and Teens with Congenital Heart Disease.** February 17-19, 2022. Honolulu, HI

**SDDS - Stanford Drug Discovery Symposium.** April 25-26, 2022. Stanford, CA

**Save the Date!**

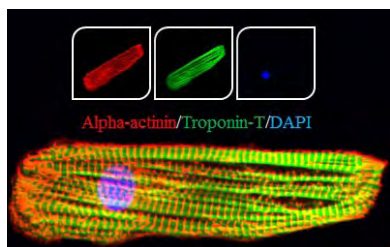
## CVI Early Career Symposium

Join Stanford CVI early career scientists for a day of cardiovascular and pulmonary science, professional development, networking and fun!

**November 3rd, 2021**



### Stanford CVI Human iPSC Biobank Service



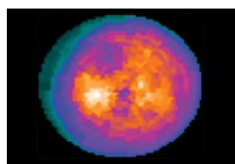
Normal and patient-derived reprogrammed cardiomyocytes are a tremendous resource for researchers and physicians here at Stanford and around the country. Understanding the disease process directly at the population level and observing these cells as surrogates under a myriad of conditions has the potential 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, CVI 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 the 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.

### 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, 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 a full spectrum of support to CVI members and their clinical trials. The coordinator 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

**Contact: Ed Finn**, Clinical Trials Manager, efinn@stanford.edu



### Cardiovascular Pharmacology (ADD-ReB)

The Advanced Drug Delivery & Regenerative Biomaterials (ADD-ReB) 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. 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. Between March 1st and May 31st, Stanford Cardiovascular Institute members published **573** original manuscripts and reviews, further contributing to our understanding of cardiovascular biology and disease. Here, we highlight selected manuscripts by our members.**

## March

**Cell type-selective secretome profiling in vivo.** Wei W, Riley NM, Yang AC, Kim JT, Terrell SM, Li VL, Garcia-Contreras M, Bertozzi CR, Long JZ. *Nat Chem Biol.* 2021 Mar;17(3):326-334. doi: 10.1038/s41589-020-00698-y. Epub 2020 Nov 16. PMID: 33199915

**Safety and Efficacy of B-Cell Depletion with Rituximab for the Treatment of Systemic Sclerosis Associated Pulmonary Arterial Hypertension: A Multi-center, Double-blind, Randomized, Placebo-controlled Trial.** Zamanian RT, Badesch D, Chung L, Domsic RT, Medsger T, Pinckney A, Keyes-Elstein L, D'Aveta C, Spychala M, White RJ, Hassoun PM, Torres F, Sweatt AJ, Molitor JA, Khanna D, Maecker H, Welch B, Goldmuntz E, Nicolls MR; NIH ASC01 Study Group. *Am J Respir Crit Care Med.* 2021 Mar 2. doi: 10.1164/rccm.202009-3481OC. PMID: 33651671

**Global analysis of shared T cell specificities in human non-small cell lung cancer enables HLA inference and antigen discovery.** Chiou SH, Tseng D, Reuben A, Mallajosyula V, Molina IS, Conley S, Wilhelmy J, McSween AM, Yang X, Nishimiya D, Sinha R, Nabet BY, Wang C, Shrager JB, Berry MF, Backhus L, Lui NS, Wakelee HA, Neal JW, Padda SK, Berry GJ, Delaidelli A, Sorensen PH, Sotillo E, Tran P, Benson JA, Richards R, Labanieh L, Klysz DD, Louis DM, Feldman SA, Diehn M, Weissman IL, Zhang J, Wistuba II, Futreal PA, Heymach JV, Garcia KC, Mackall CL, Davis MM. *Immunity.* 2021 Mar 9;54(3):586-602.e8. doi: 10.1016/j.immuni.2021.02.014. PMID: 33691136

**Transforming Growth Factor-induced Protein Promotes NF- $\kappa$ B-mediated Angiogenesis during Postnatal Lung Development.** Liu M, Iosef C, Rao S, Domingo-Gonzalez R, Fu S, Snider P, Conway SJ, Umbach GS, Heilshorn SC, Dewi RE, Dahl MJ, Null DM, Albertine KH, Alvira CM. *Am J Respir Cell Mol Biol.* 2021 Mar;64(3):318-330. doi: 10.1165/rcmb.2020-0153OC. PMID: 33264084

**Efficacy of a Centralized, Blended Electronic and Human Intervention to Improve Direct Oral Anticoagulant Adherence: Smartphones to improve rivaroxaban ADHERence in Atrial Fibrillation (SmartADHERE) A Randomized Clinical Trial: SmartADHERE rivaroxaban adherence trial.** Turakhia M, Sundaram V, Smith SN, Ding V, Michael Ho P, Kowey PR, Piccini JP, Foody J, Birmingham M, Ianus J, Rajmane A, Mahaffey KW; smartADHERE Investigators. *Am Heart J.* 2021 Mar 4:S0002-8703(21)00065-X. doi: 10.1016/j.ahj.2021.02.023. PMID: 33676886

**Aortic Dissection and Other Acute Aortic Syndromes: Diagnostic Imaging Findings from Acute to Chronic Longitudinal Progression.** Murillo H, Molvin L, Chin AS, Fleischmann D. *Radiographics.* 2021 Mar-Apr;41(2):425-446. doi: 10.1148/rg.2021200138. PMID: 33646901

**Mitochondria-Rich Extracellular Vesicles From Autologous Stem Cell-Derived Cardiomyocytes Restore Energetics of Ischemic Myocardium.** Ikeda G, Santoso MR, Tada Y, Li AM, Vaskova E, Jung JH, O'Brien C, Egan E, Ye J, Yang PC. *J Am Coll Cardiol.* 2021 Mar 2;77(8):1073-1088. doi: 10.1016/j.jacc.2020.12.060. PMID: 33632482

**County-Level Factors Associated With Cardiovascular Mortality by Race/Ethnicity.** Zuma BZ, Parizo JT, Valencia A, Spencer-Bonilla G, Blum MR, Scheinker D, Rodriguez F. *J Am Heart Assoc.* 2021 Mar 3:e018835. doi: 10.1161/JAHA.120.018835. PMID: 33653083

**Precision Public Health Matters: An International Assessment of Communication, Preparedness, and Coordination for Successful COVID-19 Responses.** Sales C, Kim Y, Kim G, Lin B, Palaniappan L. *Am J Public Health.* 2021 Mar;111(3):392-394. doi: 10.2105/AJPH.2020.306129. PMID: 33566659

**The Effects of Canagliflozin on Heart Failure and Cardiovascular Death by Baseline Participant Characteristics: Analysis of the CREDENCE Trial.** Arnett C, Li JW, Cannon CP, de Zeeuw D, Neuen BL, Heerspink HJL, Charytan DM, Agarwal A, Huffman MD, Figtree GA, Bakris G, Chang TI, Feng K, Rosenthal N, Zinman B, Jardine MJ, Perkovic V, Neal B, Mahaffey KW. *Diabetes Obes Metab.* 2021 Mar 26. doi: 10.1111/dom.14386. PMID: 33769679

**Deep learning-based intravascular ultrasound segmentation for the assessment of coronary artery disease.** Nishi T, Yamashita R, Imura S, Tateishi K, Kitahara H, Kobayashi Y, Yock PG, Fitzgerald PJ, Honda Y. *Int J Cardiol.* 2021 Mar 16:S0167-5273(21)00477-0. doi: 10.1016/j.ijcard.2021.03.020. PMID: 33741429

**miR-106a-363 cluster in extracellular vesicles promotes endogenous myocardial repair via Notch3 pathway in ischemic heart injury.** Jung JH, Ikeda G, Tada Y, von Bornstädt D, Santoso MR, Wahlquist C, Rhee S, Jeon YJ, Yu AC, O'Brien CG, Red-Horse K, Appel EA, Mercola M, Woo J, Yang PC. *Basic Res Cardiol.* 2021 Mar 19;116(1):19. doi: 10.1007/s00395-021-00858-8. PMID: 33742276

**Fast variable density Poisson-disc sample generation with directional variation for compressed sensing in MRI.** Dwork N, Baron CA, Johnson EMI, O'Connor D, Pauly JM, Larson PEZ. *Magn Reson Imaging.* 2021 Apr;77:186-193. doi: 10.1016/j.mri.2020.11.012. PMID: 33232767

**Aortic growth and development of partial false lumen thrombosis are associated with late adverse events in type B aortic dissection.** Higashigaito K, Sailer AM, van Kuijk SMJ, Willemink MJ, Hahn LD, Hastie TJ, Miller DC, Fischbein MP, Fleischmann D. *J Thorac Cardiovasc Surg.* 2021 Apr;161(4):1184-1190.e2. doi: 10.1016/j.jtcvs.2019.10.074. PMID: 31839226

**ALDH1A3 Coordinates Metabolism with Gene Regulation in Pulmonary Arterial Hypertension.** Li D, Shao NY, Moonen JR, Zhao Z, Shi M, Otsuki S, Wang L, Nguyen T, Yan E, Marciano DP, Contrepolis K, Li CG, Wu JC, Snyder MP, Rabinovitch M. *Circulation.* 2021 Mar 25. doi: 10.1161/CIRCULATIONAHA.120.048845. PMID: 33764154

**Comparison of the investigational device exemption and post-approval trials of the Melody transcatheter pulmonary valve.** Kreutzer J, Armstrong AK, Rome JJ, Zellers TM, Balzer DT, Zampi JD, Cabalka AK, Javois AJ, Turner DR, Gray RG, Moore JW, Weng S, Jones TK, Khan DM, Vincent JA, Hellenbrand WE, Cheatham JP, Bergersen LJ, McElhinney DB. *Catheter Cardiovasc Interv.* 2021 Mar 29. doi: 10.1002/ccd.29657. PMID: 33780150

**On the impact of vessel wall stiffness on quantitative flow dynamics in a synthetic model of the thoracic aorta.** Zimmermann J, Loecher M, Kolawole FO, Bäuml K, Gifford K, Dual SA, Levenston M, Marsden AL, Ennis DB. *Sci Rep.* 2021 Mar 23;11(1):6703. doi: 10.1038/s41598-021-86174-6. PMID: 33758315

**Peripheral Oxygen Extraction and Exercise Limitation in Asymptomatic Patients with Diabetes Mellitus.** Kobayashi Y, Christle JW, Contrepolis K, Nishi T, Moneghetti K, Cauwenberghs N, Myers J, Kuznetsova T, Palaniappan L, Haddad F. *Am J Cardiol.* 2021 Mar 20:S0002-9149(21)00252-6. doi: 10.1016/j.amjcard.2021.03.011. PMID: 33757787

**Activity data from wearables as an indicator of functional capacity in patients with cardiovascular disease.** Rens N, Gandhi N, Mak J, Paul J, Bent D, Liu S, Savage D, Nielsen-Bowles H, Triggs D, Ata G, Talgo J, Gutierrez S, Aalami O. *PLoS One.* 2021 Mar 24;16(3):e0247834. doi: 10.1371/journal.pone.0247834. PMID: 33760846

**Large-scale labeling and assessment of sex bias in publicly available expression data.** Flynn E, Chang A, Altman RB. *BMC Bioinformatics.* 2021 Mar 30;22(1):168. doi: 10.1186/s12859-021-04070-2. PMID: 33784977

**Establishing a Data Science Unit in an Academic Medical Center: An Illustrative Model.** Desai M, Boulos M, Pomann GM, Steinberg GK, Longo FM, Leonard M, Montine T, Blomkalns AL, Harrington RA. *Acad Med.* 2021 Mar 23. doi: 10.1097/ACM.0000000000004079. PMID: 33769342

**Analysis of deep complex-valued convolutional neural networks for MRI reconstruction and phase-focused applications.** Cole E, Cheng J, Pauly J, Vasanawala S. *Magn Reson Med.* 2021 Mar 16. doi: 10.1002/mrm.28733. PMID: 33724507

**Gene replacement of  $\alpha$ -globin with  $\beta$ -globin restores hemoglobin balance in  $\beta$ -thalassemia-derived hematopoietic stem and progenitor cells.** Cromer MK, Camarena J, Martin RM, Lesch BJ, Vakulskas CA, Bode NM, Kurgan G, Collingwood MA, Rettig GR, Behlke MA, Lemgart VT, Zhang Y, Goyal A, Zhao F,

Ponce E, Sfrifa W, Bak RO, Uchida N, Majeti R, Sheehan VA, Tisdale JF, Dever DP, Porteus MH. *Nat Med*. 2021 Mar 18. doi: 10.1038/s41591-021-01284-y. PMID: 33737751

**Avoiding Catastrophe: Understanding Free Light Chain Testing in the Evaluation of ATTR Amyloidosis.** Witteles RM, Liedtke M. *Circ Heart Fail*. 2021 Mar 19;CIRCHEARTFAILURE120008225. doi: 10.1161/CIRCHEARTFAILURE.120.008225. PMID: 33736459

## April

**Targeted proteomics of right heart adaptation to pulmonary arterial hypertension.** Amsallem M, Sweatt AJ, Arthur Ataam J, Guihaire J, Lecerf F, Lambert M, Ghigna MR, Ali MK, Mao Y, Fadel E, Rabinovitch M, de Jesus Perez V, Spiekerkoetter E, Mercier O, Haddad F, Zamanian RT. *Eur Respir J*. 2021 Apr 8;57(4):2002428. doi: 10.1183/13993003.02428-2020. PMID: 33334941

**Effects of Exercise Training on Vascular Markers of Disease Progression in Patients with Small Abdominal Aortic Aneurysms.** Niebauer S, Niebauer J, Dalman R, Myers J. *Am J Med*. 2021 Apr;134(4):535-541. doi: 10.1016/j.amjmed.2020.07.029. PMID: 32835687

**Aortic growth and development of partial false lumen thrombosis are associated with late adverse events in type B aortic dissection.** Higashigaito K, Sailer AM, van Kuijk SMJ, Willeminck MJ, Hahn LD, Hastie TJ, Miller DC, Fischbein MP, Fleischmann D. *J Thorac Cardiovasc Surg*. 2021 Apr;161(4):1184-1190.e2. doi: 10.1016/j.jtcvs.2019.10.074. PMID: 31839226

**A Novel Aortic Regurgitation Model from Cusp Prolapse with Hemodynamic Validation Using an Ex Vivo Left Heart Simulator.** Zhu Y, Imbrie-Moore AM, Paulsen MJ, Priomprintr B, Park MH, Wang H, Lucian HJ, Farry JM, Woo YJ. *J Cardiovasc Transl Res*. 2021 Apr;14(2):283-289. doi: 10.1007/s12265-020-10038-z. PMID: 32495264

**US National Trends in Vascular Surgical Practice During the COVID-19 Pandemic.** Ho VT, Eberhard AV, Asch SM, Leeper NJ, Fukaya E, Arya S, Ross EG. *JAMA Surg*. 2021 Apr 15:e211708. doi: 10.1001/jamasurg.2021.1708. PMID: 33856428

**Statin Use in Older Adults with Stable Atherosclerotic Cardiovascular Disease.** Spencer-Bonilla G, Chung S, Sarraju A, Heidenreich P, Palaniappan L, Rodriguez F. *J Am Geriatr Soc*. 2021 Apr;69(4):979-985. doi: 10.1111/jgs.16975. PMID: 33410499

**Unique Complications and Failure Modes of Iliac Branch Devices.** Stern JR, Tran K, Li M, Lee JT. *Ann Vasc Surg*. 2021 Apr 6:S0890-5096(21)00246-6. doi: 10.1016/j.avsg.2021.03.008. PMID: 33836229

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