New Vice Chair of Medicine for Clinical Research

Dr. Kenneth Mahaffey, former Professor of Medicine at Duke University and Faculty Associate Director of the Duke Clinical Research Institute (DCRI), has joined the Stanford Department of Medicine as the Vice Chair of Medicine for Clinical Research as of Aug. 1, 2013.

Dr. Mahaffey received a B.S. in Chemistry from Stanford, followed by his M.D. from the University of Washington and residency training at the University of Arizona Health Sciences Center. He completed a cardiology fellowship at Duke, including research training in the Duke Databank for Cardiovascular Disease. After joining the faculty at Duke, he rose over the next 17 years to Professor of Medicine and a recognized international leader in the design and conduct of large-scale cardiovascular trials. At the DCRI, he led the development of the field of cardiovascular clinical event detection and adjudication as critical methods in the conduct of randomized clinical trials.

He has led efforts at the US FDA to standardize definitions for cardiac events in clinical trials while leading a multidisciplinary group at DCRI responsible for coordinating event detection and adjudication in clinical studies across multiple disciplines, including oncology, hepatology, renal disease, rheumatology and cardiovascular disease, among others. He has mentored and trained a large group of international clinical research fellows in research methods.

Over the last ten years, Dr. Mahaffey has served as Chair of one of the Duke IRBs while helping to lead efforts to streamline and improve the IRB application and review process. He maintains active research collaborations with dozens of clinical researchers across the US and the globe.

For more: http://tinyurl.com/kld7pqo.

Philip Sager, MD Joins CVI

The Stanford Cardiovascular Institute (CVI) is pleased to announce the appointment of Philip Sager, MD, FACC, FAHA, FHRs, as CVI Consulting Professor effective July 15, 2013. Dr. Sager performed his undergraduate work at MIT and attended medical school and completed his medical residency, cardiology, and electrophysiology fellowships at Yale University. After a long academic career at UCLA, where his research focused on physiologic and drug-induced alterations in cardiac repolarization and he was a tenured faculty member, he moved to the pharmaceutical industry.

He played key leadership roles in the development of Zetia,....
Duchenne Muscular Dystrophy Mystery Solved

BY KRISTA CONGER

Duchenne muscular dystrophy is a heart-breaking genetic disorder that usually kills patients in their teens and twenties. Although the most prominent symptom is a gradual, progressive muscle weakness, most patients die from respiratory or cardiac failure. It’s been difficult to determine exactly how the heart is damaged, though, since mice with the same genetic mutation display only mild symptoms.

This lack of an adequate animal model has hampered research on the disorder for decades. But now researchers from the laboratory of Helen Blau, PhD, the Donald E. and Delia B. Baxter Professor, have developed a new strain of mice that more-closely mimics the course of the disease in humans.

The investigators found that the reason humans suffer more serious symptoms than mice has to do with the length of the protective caps, called telomeres, on the ends of chromosomes: Mice have telomeres about 40 kilobases in length, while human telomeres range from around 5 to 15 kilobases (a kilobase is 1,000 nucleotides). When the investigators introduced a second mutation in the animals that reduced telomere length to more closely match that of humans, the animals began to display the typical symptoms of the disease, including progressive muscle weakness, enlarged hearts and significantly shortened life spans.

Humans with Duchenne muscular dystrophy (and mice serving as models for the condition) bear a mutation in the gene that encodes for the dystrophin protein, which supports and protects muscle fibers during contraction. When the researchers closely examined the heart muscle cells of the animals in the study, they found that their heart muscle cells accumulate damage due to oxidative stress as they maintained their rhythmic contractions in the absence of the dystrophin protein.

The researchers found that treating affected mice with antioxidants early in the course of their disease delayed the onset of heart failure and increased the animals’ life span.

Foteini Mourkioti (left) and Helen Blau (right)

Photo: Norbert von der Groeben

The study reveals an intriguing link between telomere length and cardiac function. But it’s real strength lies in the ability to advance our understanding of Duchenne muscular dystrophy and speed the development of new treatments.

For more: http://tinyurl.com/mn-sy37s and http://tinyurl.com/knhwl9e.
Using 3D Printing to Model Organs

BY CAROLYN JOHNSON AND TIM DIDION (KGO-TV, ABC7)

Surgeons have made huge strides in treating the human heart. Now researchers at Stanford University are using a new technique that could soon make those surgeries even more precise, by starting with an exact replica of the patient’s heart.

“To be able to have a structure of the heart in front of us and be able to hold it in our hands and to be able to test the new devices that we’re developing is really a miracle,” says Dr. Paul Wang.

The key to creating these 3D models is a state of the art printer that looks something like a supersized microwave oven. Wang believes the combination of software and engineering that produces a nearly perfect copy of a patient’s heart could revolutionize medicine.

First, CT scans capture multiple views of the heart as a series of slices. And the next step after that is to take this data and select out what data we want to make our 3D model out of,” says research fellow Jeff Caves, Ph.D.

Caves says that engineers ultimately layer the CT images and their corresponding measurements onto sophisticated CAD software, similar to what architects use to create blueprints for buildings, slowly translating them into an accurate computer model to guide the 3D printer.

“All these angles represent surfaces and it’s full 3D at this point,” he explains.

Using reams of plastic, the 3D printer then reproduces the heart in a process that takes several hours. The results from the point of view of a cardiologist are stunning.

“It’s revolutionary,” says Wang. “One can leave the settings overnight and come back the next day and have a completed heart. You can see the exceptional detail, the structures that hold the valves in place.”

Wang says the models are so accurate that surgeons could potentially scale and fit devices ranging from catheters to coronary stents to the precise dimensions of an individual’s heart. The technology could allow doctors to test different surgical strategies in advance, before a patient ever enters the operating room.

There is also intense research underway across the country using 3D printing technology combined with living cells and other biological material. The goal is to someday construct functioning human organs. #

For more: http://tinyurl.com/q7kabh5 and ABC site: http://tinyurl.com/ocm3v8s.

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Reversing Pulmonary Hypertension

BY TRACIE WHITE

Mark Nicolls and his colleagues discovered that blocking a pathway that causes inflammation could reverse a deadly condition known as pulmonary hypertension in rats.

Pulmonary hypertension, a deadly form of high blood pressure that develops in the lungs, may be caused by an inflammation-producing molecular pathway that damages the inner lining of blood vessels, according to a new study by researchers at the Stanford University School of Medicine.

The results suggest that using medications to block this pathway could lead to the first-known cure for the disease, apart from lung transplantation. The new research could also lead to a better understanding of other diseases involving inflammation of blood vessels, such as coronary artery disease, said Mark Nicolls, MD, senior author of the study and division chief of pulmonary and critical care medicine at Stanford, as well as a staff physician at the Veterans Affairs Palo Alto Health Care System.

“We believe that targeting inflammation is an exciting approach to augment current treatments for pulmonary hypertension because it may reverse the underlying cause of the disease,” said Nicolls, who is also director of the Lung Immunology Program and an associate professor of Medicine.

PULMONARY continues on p. 4
PULMONARY from p. 3

medicine. “We believe this is going to be an approach that helps a large number of patients.”

Pulmonary hypertension, while rare, usually strikes young and middle-aged women, leaving them short of breath and often unable to complete simple daily tasks. The condition can be fatal. The risk is higher for people with certain autoimmune diseases (such as scleroderma or lupus), HIV, congenital heart disease or liver disease.

About 100,000 people in the United States and Europe have been diagnosed with pulmonary hypertension, but many others are believed to go undiagnosed because the main symptom, shortness of breath, is nonspecific. Until the 1990s, there were no treatments except lung transplantation, which has varying degrees of success.

What is known about the disease is that the narrowing of blood vessels in the lungs is caused by a mysterious proliferation of the smooth muscle cells that ring those vessels. As the vessel walls thicken, they become increasingly occluded, choking off blood flow.

The current treatment for patients with pulmonary hypertension is vasodilators, drugs that cause smooth muscle cells to relax, permitting more blood to flow through the diseased vessels. These drugs help to extend survival and relieve some symptoms, but they don’t provide a cure.

“Current approved therapies for pulmonary hypertension have focused on dilating blood vessels without special attention being paid to the inflammation that is frequently seen around blood vessels,” Nicolls said. “We were interested in finding out how inflammation contributes to the disease.”

In laboratory experiments, researchers removed inflammation-producing cells called macrophages from the lung tissue of rats dying of pulmonary hypertension and put the cells in cultures with healthy rat endothelial cells.

“We were shocked to find that half of the endothelial cells were dead within 24 hours,” Nicolls said. “That totally surprised us.”

In subsequent lab experiments, researchers found that if they blocked the molecular pathway triggered by the macrophages, the endothelial cells didn’t die. Researchers conducted similar experiments in human tissue taken from the diseased lungs of patients. The results were similar. “The same pathways were at play in the human lungs,” Nicolls said.

In experiments in live rats with induced pulmonary hypertension, the disease was reversed when researchers blocked the inflammation-producing pathway.

“It was dramatic,” Nicolls said. “We could start the drug as late as three weeks when animals were breathing hard and walking slowly, and the disease reversed.”

For more: http://tinyurl.com/pnppnpsj.

Hormone Therapy: No Cognitive Effects

BY MICHELLE BRANDT

Some news affecting post-menopausal women who take hormone therapy: In a study involving 1,326 women who started conjugated equine estrogen-based hormone therapy when they were between 50 and 55 years old, the treatment was not associated with any sustained risk or benefit to brain health.

Much has been written about this topic, dating back about 10 years ago to the release of the Women’s Health Initiative Memory Study, which linked the same type of therapy to cognitive decline and dementia in older (65 years+) postmenopausal women. This study, however, involved younger women, whose cognitive function was assessed an average of 7 years after their participation in the trial was over.

“There was essentially no overall difference in cognitive function between women who had been prescribed an average of 7 years of hormone therapy compared to women who had been prescribed placebo,” the researchers report.

The research may be reassuring to women who take, or are consider taking, hormone therapy in their 50s. And the findings also, as co-author Marcia Stefanick, PhD, with the Stanford Prevention Research Center, points out, fail to “support the ‘window of opportunity’ hypothesis which suggests that women closer to menopause will benefit with respect to cognitive function as they age.”

For more: http://tinyurl.com/oayj4dp.
New Collagen Patch Aids Heart Repair

BY LOUIS BERGERON

When heart cells die from lack of blood flow during a heart attack, replacing those dead cells is vital to the heart muscle’s recovery. But muscle tissue in the adult human heart has a limited capacity to heal, which has spurred researchers to try to give the healing process a boost.

Researchers at the Stanford University School of Medicine and Lucile Packard Children’s Hospital have developed a patch composed of structurally modified collagen that can be grafted onto damaged heart tissue. Their studies in mice have demonstrated that the patch not only speeds generation of new cells and blood vessels in the damaged area, it also limits the degree of tissue damage resulting from the original trauma.

The key, according to Pilar Ruiz-Lozano, PhD, associate professor of pediatrics, is that the patch doesn’t seek to replace the dead heart-muscle cells. Instead, it replaces the epicardium, the outer layer of heart tissue, which is not muscle tissue, but which protects and supports the heart muscle, or myocardium.

“This synthetic tissue has the mechanical properties of the embryonic epicardium,” said Ruiz-Lozano, who is the senior author of a study that describes the researchers’ findings.

Embryonic epicardium is significantly more flexible than adult epicardium, but more rigid and structured than existing materials, making it more conducive to growth of new tissue. “We paid tremendous attention to the physical properties of the materials and how their elasticity could modify the function of the heart,” Ruiz-Lozano said.

The epicardium — or its artificial replacement — has to allow the cell migration and proliferation needed to rebuild damaged tissue, as well as be sufficiently permeable to allow nutrients and cellular waste to pass through the network of blood vessels that weaves through it. The mesh-like structure of collagen fibers in the patch has those attributes, serving to support and guide new growth. Because the patch is made of acellular collagen, meaning it contains no cells, recipient animals do not need to be immunosuppressed to avoid rejection. With time, the collagen gets absorbed into the organ.

In addition to helping heart tissue regenerate, the patch could be used as a delivery system for getting medications or stem cells into a patient, Ruiz-Lozano said.

Daniel Bernstein, MD, professor of pediatric cardiology and a co-author of the paper, said the potential of the patch as a delivery system could make it useful in treating children with heart problems.

“For pediatric patients with congenital heart disease, or who have heart damage from a viral infection or other heart injury, we could use this to introduce growth factors directly to the heart in a way that would persist for a long period of time,” he said.

Ruiz-Lozano and her colleagues are already at work on studies to explore the use of the patch as a delivery system, along with conducting studies of how the patch will perform in larger animals. ✹

For more: http://tinyurl.com/mk4swff.

Link to full article in Biomaterials:

U.S. News & World Reports Ranks SHC Highly

BY JAMES LARKIN

Stanford Hospital & Clinics has been ranked one of the best hospitals for 2013-14 by U.S. News & World Report in 13 out of 16 medical specialties. The magazine also ranked Stanford one of the top two hospitals in the state of California and the best in the metro area.

The hospital also earned national rankings in three additional specialties over last year. Stanford was ranked among the nation’s very best hospitals in: cancer; cardiology/heart surgery; ear, nose & throat; gastroenterology; geriatrics; gynecology; nephrology; neurology/neurosurgery; orthopaedics; psychiatry; pulmonology; rheumatology; and urology. ✹

For more: http://tinyurl.com/naptew3.
Familial Hypercholesterolemia Summit

BY SARA WYKES

An international summit this month focuses on an inherited but highly underdiagnosed condition that causes premature cardiovascular disease, and will feature two prominent Stanford medicine community members.

Stanford instructor Josh Knowles, MD, PhD, is the chief medical officer of the Familial Hypercholesterolemia Foundation, the group hosting the summit and the first patient advocacy organization to represent people with the condition. Knowles is also an attending physician with the Familial Hypercholesterolemia Clinic at the Stanford Center for Inherited Cardiovascular Disease.

Ted Tussing, director of the Stanford Hospital Corporate Partners program, serves on the FH Foundation’s board of directors. He was diagnosed with FH at age 13, and both his sons also have it.

“Dr. Knowles’ leadership and involvement with the FH Foundation reflects not only Stanford’s strengths but also the future of medical care—leveraging the best synthesis of basic science discovery, big data, care delivery and wellness strategies,” Tussing said.

While cholesterol is a natural and necessary part of the body’s chemistry, genetic mutations can alter how it is eliminated from the bloodstream. FH affects cholesterol processing from birth, affecting the cardiovascular system so pervasively that over the course of a lifetime the toll on arteries means that men with FH have a 50 percent chance of having a heart attack by age 50. Women with FH have a 30 percent chance of heart attack by age 60.

Though the condition is common and affects over 600,000 in the U.S., the condition is diagnosed in less than 10 percent of those who suffer from it. The need for heightened awareness is clear: FH accounts for 20 percent of heart attacks in people less than 45 years old.

FH is a genetic condition: If just one parent has the condition, children have a 50 percent chance of inheriting it. The familial nature and the serious health consequences of FH have placed it on the Centers for Disease Control and Prevention’s Tier 1 list of conditions for which screening is recommended, in particular at an early age for children of adults diagnosed with FH.

The Familial Hypercholesterolemia Summit is an effort to bring together scientists, clinicians, public health officials, interested companies, and individuals with FH to find innovative solutions. The Summit will be held on Sept. 18-19 in Annapolis, Maryland. “The main goal will be to develop actionable recommendations that will embed FH in a strong public health movement,” Knowles said. ♦


PAD Stem-Cell Study Seeks Participants

BY TRACIE WHITE

The Stanford University School of Medicine is recruiting people with pain from peripheral arterial disease (PAD) for a clinical trial to determine whether injections of adult stem cells into leg muscles help treat the condition.

Stanford is one of seven sites participating in the randomized study, which will evaluate leg blood flow and symptoms of PAD in two groups of patients who suffer leg pain while exercising as a result of the condition: those treated with their own stem cells taken from bone marrow and those treated with placebo. All patients will be followed for one year. The phase-1/phase-2 study has begun recruitment and is expected to enroll 10 patients at Stanford.

PAD occurs when arteries in the arms and legs become narrowed by plaque. About 1 million to 3 million Americans suffer from a form of the disease that causes pain while exercising because muscles are not getting enough oxygenated blood.

For this study, stem cells will be taken from the bone marrow of all participants. Those randomized to the treatment group will have their stem cells injected into the diseased area of the calf and lower thigh muscles to see if it improves blood flow or walking time, or both. (The control group will receive a placebo injection.) Bone marrow contains special stem cells that may promote blood vessel growth, prevent cell death and transform themselves into new tissues.

The study is funded by the NHLBI of the National Institutes of Health (grant UM1HL12026). The principal investigator for the Stanford portion of the trial is Phillip Yang, MD, associate professor of cardiovascular medicine. To enroll contact Fouzia Khan at (650) 736-1410 or fouziak@stanford.edu. ♦

CVI NewsBeats

Atul Butte, MD, PhD, Chief of Systems Medicine and Associate Professor of Pediatrics and of Genetics, was recognized by The White House as an open science Champion of Change. He was among 13 entrepreneurs, academics and researchers honored June 20 at the White House. For more: http://tinyurl.com/k59jv84.

Andrew Connolly, MD, PhD, Associate Professor of Pathology, received the Kaiser Foundation Award for Excellence in Preclinical Teaching, whose recipients are chosen by students in preclinical medicine. For more: http://tinyurl.com/k4zdzov.

Ngan Huang, PhD, was appointed Assistant Professor of Cardiothoracic Surgery, effective May 1. Her lab aims to understand the chemical and mechanical interactions between extracellular matrix proteins and pluripotent stem cells that regulate vascular and myogenic differentiation.

Julia Ransohoff, CVI-affiliated Stanford medical student, and Bruno Huber, PhD, have shown that a dual agent approach of CTL4Ig and anti-LFA1 is more effective than prednisone and cyclosporine A for preventing rejection of human ESC-derived endothelial cells in animal model of myocardial infarction. For more: http://tinyurl.com/mdhu39a and http://tinyurl.com/m6nv7zt.

Nazish Sayed, MD, PhD, Former CVI T32 fellow (July 2010 - June 2013) has been hired as a Scientist in the Dept. of Cardiovascular Sciences at Methodist Hospital Research Institute, Houston, TX as of July 1. When at Stanford his Mentors included John Cooke, MD, PhD; Edward Mocarski, PhD; and Karla Kierkegaard, PhD.

Sonja Schrepfer, MD, PhD, received the Leducq Transatlantic Career Development Award for her work investigating mechanisms of immune tolerance, which might someday be used to avoid the unwanted immune reactions provoked by the use of embryonic stem cells as a treatment for heart failure. For more: http://tinyurl.com/kq9lkhn.

Michael Snyder, PhD, Director of the Stanford Center for Genomics and Personalized Medicine, was featured in a Palo Alto Weekly cover story about research that could translate genetic discoveries into widespread personalized medicine. For more: http://tinyurl.com/lathjua; http://tinyurl.com/l8l8lxp; and http://tinyurl.com/knqrzj3.

Sean Wu, MD, PhD, has been named an Endowed Faculty Scholar of the Child Health Research Institute and the Lucile Packard Foundation for Children’s Health at Stanford. The newest class of Endowed Faculty Scholars are appointed to 5-year terms, beginning Sept. 1. For more: http://tinyurl.com/k89o2po.
CVI NewsBeats

Tim Assimes, MD, PhD (left) and Philip Tsao, PhD (right) received one of only three NHLBI contracts awarded in response to a Broad Agency Announcement for their project, “Integrative genomics and risk of CHD and related phenotype in the Women’s Health Initiative (WHI).” Other PIs include Devin Absher (Hudson Alpha Institute of Biotechnology) and Steve Horvath (UCLA). The study will apply genomic technologies on over 2000 WHI participants to identify circulating miRNA and DNA methylation patterns associated with coronary heart disease. Detailed study objectives and more can be found at: http://tinyurl.com/m9lzkom.

Inaugural CVI Postdoctoral Travel Award Recipients

Ivan Carcamo-Oribe (Josh Knowles Lab)
Abstract: Modeling Insulin Resistance Through iPSC Technology; Meeting: International Society for Stem Cell Research

Gadryn Higgs & Alexandre Ribeiro (Beth Pruitt Lab)
Abstract: Inducing Variations in the Shortening of Single Cardiomyocytes with Localized Mechanical Stimulation; Meeting: International Conference on Microtechnologies in Medicine and Biology

Clint L. Miller (Thomas Quertermous Lab)
Abstract: Disease-relevant pathways modulate a cis-regulatory element at the TCF21 coronary heart disease locus; Meeting: Gordon Research Conference on Human Genetics and Genomics

Nils P. Nickel (Marlene Rabinovitch Lab)
Abstract: The Elastase Inhibitor Elafin Restores Endothelial Cell Homoeostasis in Pulmonary Arterial Hypertension And attenuates Vascular Remodeling In The Sugen/Hypoxia Rat Model; Meeting: American Thoracic Society International Conference

Stephen Pan (Euan Ashley Lab)
Abstract: Cardiac Structural and Sarcomere Genes Associated with Cardiomyopathy Exhibit Marked Intolerance of Genetic Variation; Meeting: American Heart Association Scientific Sessions

Chirag J. Patel (John PA Ioannidis Lab)
Abstract: Environment-wide Association Studies to Connect Multiple Personal Exposures to Health; Meeting: International Society for Exposure Sciences

Julia D. Ransohoff (Joseph Wu Lab)
Abstract: Blockade of Costimulatory Molecule Signaling Promotes Survival of Human Embryonic Stem Cell-Derived Endothelial Cells by Inducing T Cell Immunoglobulin 3 Uregulation and Improves Cardiac Function; Meeting: International Society for Stem Cell Research

Vahid Serpooshan (Pilar Ruiz-Lozano Lab)
Abstract: MicroRNA-24 Controls Macrophage Trafficking in Murine Abdominal Aortic Aneurysm via Chi3l1; Meeting: Arteriosclerosis, Thrombosis, and Vascular Biology Scientific Sessions

Joshua M. Spin (Philip Tsao Lab)
Abstract: MicroRNA-24 Controls Macrophage Trafficking in Murine Abdominal Aortic Aneurysm via Chi3l1; Meeting: Arteriosclerosis, Thrombosis, and Vascular Biology Scientific Sessions

Ke Yuan (Vinicio de Jesus Perez Lab)
Abstract: Impaired Pulmonary Angiogenesis in IPAH Is Linked To Abnormal Pericyte Function And Reduced Endothelial-Pericyte Interactions; Meeting: American Thoracic Society International Conference

Now Recruiting:
Assistant/Associate/Full Professor Dept. of Medicine
Stanford University

Application Deadline: Thursday, October 03, 2013

The Stanford Cardiovascular Institute and the Division of Cardiovascular Medicine seek to jointly recruit a basic science researcher with an interest in the fundamental molecular and cellular basis of cardiovascular disease.

The applicant will have M.D., Ph.D., or M.D. /Ph.D degrees and will be appointed in the Medical Center Line or University Tenure Line. Faculty rank will be determined by the qualifications and experience of the successful candidate. Preference will be given to researchers who are able to apply new and innovative approaches to the investigation of the risk for cardiovascular disease, including the use of novel animal models and the use of modern human and molecular genetics approaches. The ideal candidate will have completed rigorous training or have acquired applied experience in one or more related areas of translational research such as epidemiology, clinical trials, physiology, cellular and molecular biology and human genetics. The applicant will be expected to develop an independently funded research effort focused in this area, and to help with teaching activities in the Division and the Department of Medicine. Appropriate resources will be made available to help the applicant develop their research program. It is highly desirable that the applicant have an MD degree, be trained in cardiology and spend some time contributing to the patient care mission of the Division, but exceptional PhD applicants will also be considered. Stanford University is an equal opportunity employer and is committed to increasing the diversity of its faculty. It welcomes nominations of and applications from women and members of minority groups, as well as others who would bring additional dimensions to the university’s research, teaching and clinical missions. Rank and salary will be commensurate with qualifications and experience.

The predominant criterion for appointment in the University Tenure Line is a major commitment to research and teaching. The major criterion 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 need the individual is expected to fulfill.

Applicants should send a copy of their curriculum vitae, a brief letter outlining their interests, and three reference letters either electronically to marisha.smith@stanford.edu or via regular mail to:

Tom Quertermous, MD
Search Committee Chair, c/o Marisha Smith
1070 Arastradero Rd, Suite 220
Palo Alto, CA 94304-5850

Stanford University is an equal opportunity employer and is committed to increasing the diversity of its faculty. It welcomes nominations off and applications from women and members of minority groups, as well as others who would bring additional dimensions to the University's research, teaching, and clinical missions.
June: 129 publications


*Meta-analyses of hydroxyethyl starch for volume resuscitation.* Ioannidis JP. *JAMA*; 309(21):2209


Genetic measurement of memory B-cell recall using antibody repertoire sequencing. Vollmers C, Sit RV, Weinstein JA, Dekker CL, Quake SR. *Proc Natl Acad Sci USA*; 2013 Jul 29 [Epub ahead of print]


Mendelian Randomisation Studies Do Not Support a Causal Role for Reduced Circulating Adiponectin Levels in Insulin Resistance and Type 2 Diabetes. Yaghootkar H, Lamine C, Scott RA, [+30 authors], Assimes TL, [+4 authors], Knowles JW, [+4 authors], Quertermous T, [+33 authors], Fryault TM. *Diabetes*; 2013 Jul 8 [Epub ahead of print]


US studies may overestimate effect sizes in softer research. Fanelli D, Ioannidis JP. Proc Natl Acad Sci USA; 2013 Aug 26 [Epub ahead of print].


Computation of Coding Variants in African Americans: Better Performance using Data from the Exome Sequencing Project. Duan Q, Liu EY, Auer PL, [+ 21 authors], Assimes TL, [+ 9 authors], Li Y. Bioinformatics; 2013 Aug 16 [Epub ahead of print].


Upcoming Grants

**SEPTEMBER**

*Foundation Leduq*
Transatlantic Networks of Excellence
$6 million over 5 years
LOI: September 15, 2013

*American College of Cardiology*

**ACCF/Merck Research Fellowships in Cardiovascular Disease and Cardiometabolic Disorders**
$70,000/year
September 23, 2013

**ACCF/William F. Keating, Esq. Endowment Career Development Award**
$70,000/year
September 23, 2013

**ISCTR-ACCF Cardiovascular Translational Research Scholarship**
$60,000/year
September 23, 2013

**OCTOBER**

**ACCF Young Investigator Awards Competition**
$2,000 + $1,500 travel costs
October 4, 2013

**NIH Director’s New Innovator Award**

**NOVEMBER**

**AP Giannini Family Foundation Medical Fellowship Program**
$136,000 over 3 years
November 4, 2013

**Stanford Prevention Research Center SPARC Research Fellowship Program in Cardiovascular Disease Prevention**
November 15, 2013

**DECEMBER**

**American Federation for Aging Research Ellison Medical Foundation / AFAR Postdoctoral Fellows in Aging Research Program**
$47,114 to $55,670 for 1 year
LOI: December 16, 2013

Postdoctoral & Pre-doctoral Awards

**SEPTEMBER**

*American Society of Transplantation*

**AST Basic Science and Clinical Science Fellowship Grants**
$40,000/year for up to 2 years
Open for applications: September 9, 2013

**Katherine McCormick Advanced Postdoctoral Scholar Fellowships**
$35,000/year
September 16, 2013

**National Institutes of Health Director’s Early Independence Award**
$250,000/year for 5 years
Internal Deadline September 30, 2013

**OCTOBER**

**The Helena Anna Henzl-Gabor Young Women in Science Postdoctoral Scholars Travel Fellowship**
$2,000 per awardee
Open for application: October 1, 2013

**Life Sciences Research Foundation Postdoctoral Fellowship Program**
$50,000 to $60,000 for 2 years
October 1, 2013

**Cystic Fibrosis Foundation Clinical Fellowships 1st & 2nd Year**
$47,600-$49,250 for 2 years
October 2, 2013

**National Institutes of Health K Career Development Awards**
October 12, 2013

**CVI Postdoctoral Fellow Travel Awards**
$750 per awardee
October 15, 2013

**Thoracic Surgery Foundation For Research and Education Nina Staff Braunwald Research Fellowship**
$30,000/year up to 2 years
October 15, 2013

**NOVEMBER**

**MDA Research Grants & Development Grants**
$60,000/year
LOI: December 15, 2013

**DECEMBER**

“Cardiovascular Disease” is now on the left hand navigation table in the Research Management Group’s (RMG) Funding Information Resource webpage. This webpage provides links to recent announcements, internal Stanford funding opportunities, NIH, NSF, foundations, postdoctoral fellowships, graduate student funding opportunities, as well as to a searchable funding database. Visit this great resource at [http://med.stanford.edu/rmg/funding/](http://med.stanford.edu/rmg/funding/).

To be added to funding opportunity email distribution lists, please contact Jeanne Heschele at RMG at jheschele@stanford.edu.
CVI Frontiers Seminars

12 noon to 1 p.m. Tuesdays
Li Ka Shing Center
Stanford School of Medicine
291 Campus Drive, Stanford

8/20/2013
Michael S. Lauer, MD, NIH/NHLBI
-- Director, Division of Cardiovascular Sciences; National Heart, Lung, and Blood Institute

9/24/2013
Aaron Gitler, PhD, Associate Professor
Dept. of Genetics, Stanford

10/1/2013
Gerald Dorn, MD,
Philip & Sima K Needleman Professor of Medicine, Washington University in St. Louis, MO

10/8/2013
Neil Ingels, PhD, Consulting Professor
Cardiothoracic Surgery - Adult Cardiac Surgery, Stanford

10/22/2013
Alan Daugherty, PhD, Editor, ATVB/
Senior Assoc. Dean of Research, University of Kentucky

10/29/2013
Philip Sager, MD, CVI Consulting Professor, Stanford

11/5/2013
Mark E. Anderson, MD, PhD, Professor of Medicine and Physiology Head,
Department of Internal Medicine Director, University of Iowa Cardiovascular Research Center

11/12/2013
Bernard J. Gersh, MB, ChB, Dphil,
Professor of Medicine, Cardiovascular Diseases, Mayo Clinic

11/26/2013
Luiz Berardinni, MD, Senior Vice President of Cardiovascular Therapeutics
at Gilead Sciences, Inc.

Upcoming Meetings

SEPTEMBER

The Familial Hypercholesterolemia Summit: Awareness to Action
September 19, 2013
Annapolis, MD

Western Vascular Society
September 21-24, 2013
Jasper, AB, Canada

Heart Failure Society of America
17th Annual Scientific Meeting
September 22-25, 2013
Orlando, FL

NHLBI Symposium on Cardiovascular Regenerative Medicine
September 25-26, 2013
Bethesda, MD

OCTOBER

Women at the Heart of Leadership: 2013 Women in Medicine and Science Conference
October 12, 2013
Stanford, CA

Vascular Biology 2013 – North American Vascular Biology Organization (NAVBO)
October 20-24, 2013
Hyannis, MA

NOVEMBER

AHA Scientific Sessions 2013
November 16-20, 2013
Dallas, TX

Vascular Endovascular Issues Techniques Horizons (VEITH)
November 19-23, 2013
New York, NY

SAVE THE DATE
SEPTEMBER 12 & 13 2013
Register Here

CVI Annual Retreat
Li Ka Shing Center for Learning & Knowledge
291 Campus Drive, Stanford, CA 94305

Keynote Speaker:
Shaun R. Coughlin, MD, PhD
Director, Cardiovascular Research Institute, UCSF

Photo: Linda A. Cicero, Stanford News Service

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**Cardiovascular Institute Leadership**

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