Human cord blood gives old mice an edge

By Bruce Goldman

Human umbilical cord blood can rejuvenate learning and memory in older mice, according to a study by researchers at the School of Medicine. The researchers identified a protein, abundant in human cord blood but decreasingly so with advancing age, that had the same effect when injected into the animals.

The findings could lead to new treatments for age-associated declines in mental ability. "Neuroscientists have ignored it and are still ignoring it, but to me it's remarkable that something in your blood can influence the way you think," said the study's senior author, Tony Wyss-Coray, PhD, professor of neurology and neurological sciences and a senior research career scientist at the Veterans Affairs Palo Alto Health Care System. The lead author is former postdoctoral scholar Joseph Castellano, PhD, who is now an instructor of neurology and neurological sciences.

The study was published online April 19 in Nature. In a widely discussed earlier study, Wyss-Coray’s lab showed that direct infusion of young mice’s plasma, the cell-free portion of blood, benefited old mice. Those benefits extended beyond biochemistry and physiology to actual performance on tests of memory and learning, the researchers found.

The new study marks the first demonstration that human plasma can aid older mice’s memory and learning, which both Wyss-Coray and Castellano said would seem to increase the likelihood that it could have a similar beneficial effect in people. It’s also a major step forward from a drug-development standpoint, they suggested, that a single protein appears largely capable of mimicking those benefits.

Age-associated changes in blood

Comparing blood plasma from 19- to 24-year-olds, 61- to 82-year-olds and umbilical cords, researchers identified age-associated changes in a number of proteins.

These changes, the investigators suspected, might affect a brain structure called the hippocampus, which in both mice and humans is critical for converting experiences into long-term memories. In particular, the hippocampus is essential for helping you remember spatial information, such as how to find your way back to the car you parked in a multilevel structure several hours ago, and information about autobiographical events, such as what you ate for breakfast.

For largely unknown reasons, the hippocampus is especially vulnerable to normal aging, said Wyss-Coray. “With advancing age, the hippocampus degenerates, loses nerve cells and shrinks,” he said. The capacity to learn and remember falters inロックstep. Hippocampal deterioration is also an early manifestation of Alzheimer’s disease.

To distinguish the effects of old, young and “young-est” human blood on hippocampal function, the researchers used immune-deficient laboratory mice that could be given repeated injections of human plasma without experiencing negative immune reactions. Experiments undertaken before injecting human plasma into the mice showed that, like their immune-compotent peers, these mice’s hippocampal activity, integrity and regenerative capacity dropped off in old age — indeed, a bit faster.

Old immune-deficient mice performed more poorly than younger ones on tests of memory and learning. One such test, the Barnes maze, employs a table, about 4 feet in diameter and 1.3 feet high, that had the same effect when injected into the mice. Old mice performed more poorly than younger ones on tests of memory and learning.

The researchers identified a protein, abundant in human umbilical cord blood that revitalized memory and learning ability in older mice.

Wearable sweat sensor holds promise for diagnosing cystic fibrosis, other diseases

By Jennie Dusheck

A wristband-type wearable sweat sensor could transform diagnostics and drug evaluation for cystic fibrosis, diabetes and other diseases.

The novel collects sweat, measures its molecular constituents and then electronically transmits the results for analysis and diagnostics, according to research led by researchers at the School of Medicine, in collaboration with UC-Berkeley. Unlike old-fashioned sweat collectors, the new device does not require patients to sit still for a long time while sweat accumulates in the collectors.

“This is a huge step forward,” said Carlos Milla, MD, associate professor of pediatrics at Stanford. The study was published online April 17 in the Proceedings of the National Academy of Sciences. Milla shares senior authorship with Ronald Davis, PhD, professor of biochemistry and of genetics at Stanford. Former Stanford postdoctoral scholar Sam Emmensnejad, PhD, is the lead author.

How does it work?

The two-part system of flexible sensors and microprocessors sticks to the skin, stimulates the sweat glands and then detects the presence of different molecules and ions based on their electrical signals. The more chloride in the sweat, for example, the more electrical voltage is generated at the sensor’s surface. The team used the wearable sweat sensor in separate studies to detect chloride ion levels — high levels are an indicator of cystic fibrosis — and to compare levels of glucose in sweat to that in blood. High blood glucose levels can indicate diabetes.

Genetic counseling could help prevent unnecessary double mastectomies

By Krista Conger

A recent survey of over 2,000 women newly diagnosed with breast cancer found that half of those who undergo bilateral mastectomy after genetic testing don’t actually have mutations known to confer increased risk of additional cancers, according to a study by researchers at the School of Medicine and four other U.S. medical centers.

Instead the women had what are known as variants of uncertain significance, or VUS, that are often eventually found to be harmless. A bilateral mastectomy is a surgical procedure in which both of a woman’s breasts are removed after a diagnosis of cancer in one breast.

The finding highlights the need for genetic counselors to help both patients and physicians better understand the results of genetic testing intended to determine a woman’s risk for cancer recurrence or for developing a separate cancer in her ovaries or unaffected breast.

A wearable sensor that extracts sweat and analyzes its constituents could be a useful device for diagnosing and monitoring diseases. See SENSOR, page 6

Genetic counseling could help prevent unnecessary double mastectomies See BLOOD, page 7

See MASTECTOMY, page 6

See INSIDE STANFORD MEDICINE, page 5
Suppressing protein extends life of mice with ALS-like disease

By Jennie Dusheck

A study led by researchers at the School of Medicine has revealed a possible new therapeutic approach for amyotrophic lateral sclerosis, a progressive neurodegenerative disease.

The Stanford-led team performed a series of experiments showing that suppressing a certain protein in a mouse model of ALS, or Lou Gehrig’s disease, could markedly extend the animal’s life span. In one experiment, none of the untreated mice lived longer than 29 days, while some of the treated mice lived more than 400 days.

A paper describing the work was published online April 12 in Nature. The research was supported by the National Institutes of Health and the Robert Packard Center for ALS Research at Johns Hopkins University, the Glenn Foundation and the German Research Foundation.

Researchers from Stanford’s Department of Genetics also supported the work.

A different approach was needed. “Exactly how ALS works is still poorly understood, but knowing which genes are involved can point researchers toward processes inside cells that would be good targets for drugs,” said Becker. “One indicator of ALS, as well as other neurodegenerative diseases, is clumps of protein in the brain. In ALS, these clumps, or aggregates, are made up of a protein called TDP-43. Eliminating TDP-43, and therefore the TDP-43 aggregates, might seem like a good way to prevent the disease in mice that were not yet showing symptoms. Whether oligonucleotides or other protein-blocking treatments could work in humans is another question. ‘That’s a different story,’ said Becker, ‘but what was really astounding’ was that mice receiving the amount of ataxin 2 would protect a person from ALS.”

Suppressing protein extends life of mice with ALS-like disease

A preventive that worked in mice “We wanted to find out if we could protect these mice from the consequences of TDP-43 by lowering the amount of ataxin 2,” said Gitler. “Becker genetically engineered these ALS mice to have half the normal amount of ataxin 2, and also engineered TDP-43 to completely lack the protein. She found that with half the ataxin 2, the ALS-like mice survived longer. ‘But what was really astounding,’ said Becker, ‘was that when we completely removed ataxin 2, there was really an unprecedented survival gain—the mice lived hundreds and hundreds of days.’

Gitler’s team next tried something that could have a more direct therapeutic value: treating mice with a type of DNA-like drug, designed to block the production of ataxin 2. These so called ‘antisense oligonucleotides’ are strands of synthetic DNA that target a gene and block the expression of the protein that it encodes. Delivery of the antisense oligonucleotides to the nervous systems of some of the ALS mice enabled them to maintain their health much longer than the ALS mice treated with a placebo.

A similar antisense oligonucleotide was recently approved for safety trials in pediatric patients with spinal muscular atrophy, and other antisense oligonucleotides have passed safety trials—factors that Gitler said give him hope for a similar strategy for ALS.

Becker said the study showed that suppressing ataxin 2 delayed onset and slowed the progression of the ALS-like disease in mice that were not yet showing symptoms. Whether oligonucleotides or other protein-blocking treatments could reverse symptoms in mice that are already sick is another question. ‘That’s the next set of experiments that we are working on,’ she said. ‘Because TDP-43 clumping occurs in nearly all ALS cases, targeting ataxin 2 could be a broadly effective therapeutic strategy, she said.”

Researchers from St. Jude Children’s Research Hospital, Goethe University Frankfurt, the University of Utah, Howard Hughes Medical Institute and Ionis Pharmaceuticals also contributed to the study.

The research was supported by the National Institutes of Health, the National Science Foundation, the Robert Packard Center for ALS Research at Johns Hopkins University, the Glenn Foundation and the German Research Foundation.

Stanford’s Department of Genetics also supported the work.

Big Data in Biomedicine Conference set for May 24-25

The 2017 Big Data in Biomedicine Conference, set for May 24-25 at the School of Medicine, will explore success stories and opportunities for harnessing big data for both research and clinical care.

This year’s meeting, titled “Big Data in Biomedicine: Transforming Lives Through Precision Health,” will focus on precision health in action, highlighting the Precision Medicine Initiative, the Chan Zuckerberg Initiative and other promising initiatives.

“We are living in a time of unprecedented complexity — and historic opportunities to use big data to improve health,” said McClellan. “Together, the School of Medicine and the Precocity of Medicine program will continue to develop and promote precision medicine programs that will improve health outcomes, reduce costs and empower patients.”

This year’s speakers will include Marc Tessier-Lavigne, PhD, president of Stanford; Stephen Quake, professor of bioengineering at Stanford; Jennifer Van Eyk, MD, professor of medicine at Cedars-Sinai Medical Center; Russ Altman, MD, PhD, professor of bioengineering, of genetics and of medicine at Stanford; Michelle Rohrer, PhD, senior vice president and global head of product development regulatory and policy at Genentech; Greg Moore, MD, PhD, vice president of Healthcare Google; Cori Bargmann, PhD, president of the Carnegie Institution for Science; and Nancy Brown, CEO of the American Heart Association.

To see the full list of speakers and register for the conference, visit http://big-data.stanford.edu.
Fibrosis reversed when ‘don’t eat me’ signal is blocked

By Krista Conger

Researchers at the School of Medicine have identified a pathway that, when mutated, drives fibrosis in many organs of the body.

The discovery unifies what have been considered somewhat disparate conditions, including scleroderma, idiopathic pulmonary fibrosis, liver cirrhosis, kidney fibrosis, and more, the researchers found. These diseases are often incurable and life-threatening.

Importantly, the researchers were able to reverse lung fibrosis in mice by administering an antibody called anti-CD47 now being tested as an anti-cancer treatment.

‘The variety of diseases caused by overproduction of fibroblasts has made finding a common root cause very challenging, in part because there has been no good animal model of these conditions,’ said Irving Weissman, MD, professor of pathology and of developmental biology. ‘Now we’ve shown that activating a single signaling pathway in mice causes fibrosis in nearly all tissues. Blocking the CD-47 signal, which protects cancer cells from the immune system, can also ameliorate these fibrotic diseases even in the most extreme cases.’

The researchers hope their findings will lead to the development of a reliable treatment of many types of fibrotic diseases. They are also planning to investigate whether the anti-CD47 antibody could be an effective treatment for people with fibrosis.

A study describing the research was published on line April 17 in the Proceedings of the National Academy of Sciences. Weissman, who directs Stanford’s Institute for Stem Cell and Regenerative Medicine and the Ludwig Center for Cancer Stem Cell Research and Medicine, is the senior author. Gerlinde Wernig, MD, assistant professor of medicine, is the lead author.

When injury response goes astray

Fibrosis occurs when the body’s normal response to injury goes astray. An overenthusiastic or inappropriate timed proliferation of cells called fibroblasts, which make up the connective tissue surrounding and supporting organs, can lead to many devastating diseases. Until now, it’s not been clear whether these diseases share a common biological pathway.

The researchers were building upon previous work from the Weissman lab. The discovery of the mechanisms of how other ‘dangerous’ controls fibroblasts from people with idiopathic pulmonary fibrosis, has shown that the diseased, c-Jun-expressing fibroblasts are surrounded by immune cells called macrophages. This is reminiscent of what’s often seen in human cancers.

Over the past eight years, researchers in Weissman’s laboratory have shown that many human cancers evade the immune system by expressing high levels of a protein called CD47 on their surfaces. Blocking this protein with an anti-CD47 antibody restores the ability of the macrophages to gobble the cancer and has proven to be a promising treatment in animal models of the disease. Anti-CD47 antibody is currently undergoing a phase-1 clinical trial in humans with advanced solid tumors.

‘Like in cancer, these fibroblasts are proliferating excessively beyond what should be their natural limit,’ Weissman said. ‘We wanted to see if they are also expressing the ‘don’t eat me’ signal on their surfaces to protect them from the immune system.

When Wernig treated mice with c-Jun-induced lung fibrosis with daily injections of anti-CD47 antibody, the animals exhibited significantly better lung function, lived longer than their peers and cleared the fibrosis.

The researchers plan to investigate whether any patients in the phase-1 trial of the anti-CD47 antibody also suffered from fibrotic conditions. ‘While we are eager to learn whether they experienced any relief as a result of participating in the trial. ‘We have a bit of key data unique in this study,’ Wernig said. ‘We identified a highly activated pathway that causes fibrosis in many tissues in mice, and we’ve shown that targeting the CD47 antibody reverses the fibrosis. We’re hopeful that this could be a potential treatment for people with many types of fibrotic conditions.

Wernig also tested inhibitors of other genes activated by c-Jun in the abnormal fibroblastic cells, and inhibitors of two pathways also reduced the fibrotic lesions.

“This study shows once again how basic science investigations in one field can lead to advances in what appeared to be unrelated diseases,” Weissman said. “Here, our discovery of the discovery of the mechanisms of how other ‘dangerous’ cells in fibrosis escape removal by the body’s scavenger cells. It shows how important it is to develop appropri- ate animal models of human diseases and then to use those models to identify disease-specific pathways that can be targeted.”

Weissman is the Virginia & D.K. Ludwig Professor for Clinical Investigation in Cancer Research. He is a member of Stanford’s Bio-X, Cardiovascular Institute and Cancer Institute.

Other Stanford co-authors are postdoc- toral scholars Shih-Yu Chen, MD, PhD, and Liu Chu. Wei, a graduate student Camille Van Neste; graduate stu- dent Jonathan Tsai; professor of pathology Neetaja Kambham; MD, professor of pa- thology John F. Gnarra; MD; John Cui, MD, PhD; professor of pathology Yaso Naruki- nam, MD, PhD; and professor of microbi- ology and immunology Garry Nolan, PhD.

The research was supported by the Na- tional Institutes of Health; the Department of Defense; the Food and Drug Administration; the Gates Foundation; the Virginia and D.K. Ludwig Fund for Cancer Research; the Stanford Cancer Institute; the Stanford Physical Sciences and Engineering for Immunity, Transplantation and Infection; Northrop- Grumman Corporation; Novartis; Pfizer; and Juno Therapeutics.

Wernig is the founder of Forty Seven Inc., which is exploring ways to use immunotherapy like the anti- CD47 antibody to fight cancer. A patent titled “Antifo- bic activity of anti-CD47 blockade” has been filed by the researchers.

Stanford’s Department of Pathology also supported the work.

At annual lab swap, one researcher’s trash is another’s treasure

By Jackie Flynn

Armed with spare pipettes, vials, centrifuges and chemicals, more than 150 people from across campus carried or shipped unneeded lab supplies to the lawn next to the Li Ka Shing Center for Learning and Knowledge on April 12 for the Office of Sustain- ability’s third annual lab swap.

Throughout the day, scientists rep- resenting more than 100 labs sifted through the boxes and bins of lab equipment for unused or reusable ma- terials worth thousands of dollars. The list of materials that would otherwise probably get thrown away don’t end up in a landfill. Not just that, they get reused. They have a second life,” said Rashmi Sahai, the assessments program manager at the Office of Sustainability, who helped organize the swap.

Among the items for taking were laboratory supplies such as ink car- tridges, biohazard disposables, biohazard disposal kits, pro- tein-extraction chambers, freezer racks, textbooks, hot plates, blenders and office supplies. A central figure of the pur- euse also emerged, floppy disks, a Microsoft Windows 95 starter kit, filter paper estimated by the donor to be from the 1960s and cassette tapes from The Who and Steely Dan.

“We can give these items to some- one who can use them, or we can just let them deteriorate,” said Rebecca Agin, a life science technician and intern in immunology, was an intern in research with his love for the natural world.

“A lot of the items we use to con- duct research are disposable, single-use items made out of some material that’s recyclable, some of that isn’t. Seeing bags and bags of this pile up every day, you wonder, was laconic?” Cus- ros said.

The event falls under the Office of Sustainability’s Cardinal Green Labs program, which supports labs’ reduc- tion of waste, energy and water use across campus. The program offers services including free installation of energy-saving equipment timers and low-flow faucets, a biannual campus cleanup event and rebates for energy efficient low-temperature freezers.

When Cusinos envisioned the lab swap program, which had already taken shape for small labs, he noted that there was already shared use equipment within lab networks, but there wasn’t a centralized place for people not on campus. “We knew that there was a demand. We

The Office of Sustainability’s third annual lab swap, held April 12, was an opportunity for Stanford laboratories to get rid of unneeded equipment and reagents and also find stuff they need.

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By Erin Digitale

By the time she sees them, psychologist Anya Griffin’s young patients have been in pain for months to years. In 2015, concerned that standardized questionnaires and 1- to 10-pain scales didn’t give a personal- ized view of their struggles, Griffin, PhD, decided to try something different.

Then newly hired as clinical director of the Pediatric Rehabilitation Program at Stanford Children’s Health’s Center for Rehabilitation Services, Griffin borrowed an approach that originated in public health research: asking patients to narrate their perspectives with a method called photovoice.

Many of Griffin’s patients have complex regional pain syndrome, in which pain from an injury spirals out of control. The nervous system magnifies sensory input so that the slightest touch can cause fiery pain. Young people with the condition stop attending school, hanging out with friends, playing sports, taking music lessons, feeling like normal kids. The pain becomes its own disease.

“I am not in their bodies and I can’t possibly know what that experience is like,” Griffin said. “I wanted to get a better understanding of what they’re experi- encing and how it impacts their lives.”

The photovoice project is supported by a seed grant from the School of Medicine’s Medicine and the Muse program, which helped fund the purchase of cameras for patients to use. At the conclusion of the rehabilita- tion program — which lasts four to 12 weeks and includes medical evaluation and treatment, physical and occupational therapy, and individual, group and family sessions with pain psychologists — Griffin has each patient pick a “before” and “after” photo and explain them. Here are two examples: a third is available online at http://stan.md/2kJZU8j.

Painting with her feet

“That was one of my very first days in the program,” said Laura, 14, of the photo of herself making blue footsteps. A dancer before she fractured her left foot at age 10, Laura (a pseudonym) had been in pain for three years by the time she started the pain rehabilitation pro- gram in May 2015. The pain had spread all the way to her left hip, leaving her unable to walk without a cane. Dancing, singing and other arts forms she’d once loved had fallen out of her life.

Painting with her feet was kind of fun. “But I couldn’t concentrate on the fun,” Laura said. “I was just constantly making sure I wouldn’t fall, focusing on the pain in my foot.”

Partway through the program, Laura videotaped her- self and several other patients dancing. “When I was having a really bad pain flare-up, I’d go back and watch that video,” she said. “I’d think, oh yeah, I can do this! The pain isn’t everything. It isn’t me.”

By the time Laura’s parents took her to her grandma’s farm to celebrate her rehabilitation, she had progressed from walking without a cane to walking without a limp to running. With her complex regional pain syndrome in remission, she could also sit in the grass and feed her grandma’s goats, which would have been impossible a few months earlier.

“In the second photo I was finally at peace with myself,” she said. “I wasn’t battling my foot.”

Potency of priming

Mice given the priming protein recover muscle function more quickly after damage, their skin heals more rapidly and even the shaved area around the injury regrows hair more quickly, the study found. Harnessing the power of this protein may one day help people recover more quickly from surgery or restore youthfulness to aging stem cells.

“We’re trying to better understand wound healing in response to trauma and aging,” said Thomas Rando, MD, PhD, professor of neurology and neu- rological sciences. “We’ve shown that muscle and bone marrow stem cells enter a stage of alertness in response to distant injury that allows them to spring into action more quickly. Now we’ve pin- pointed the protein responsible for prim- ing them to do what they do better and faster.”

Rando, who also directs Stanford’s Glenn Center for the Biology of Aging, is the senior author of the study, which was published April 18 in Cell Reports. Former postdoctoral scholar Joseph Rodgers, PhD, is the lead author. Rodgers is now an assistant professor of stem cell biology and regenerative medicine at the University of Southern California.

Potential therapy

“Our research shows that by priming the body before an injury you can speed the process of tissue repair and recovery, similar to how a vaccine prepares the body to a fight infection,” Rodgers said. “We believe this could be a therapeutic approach to improve recovery in situa- tions where injuries can be anticipated, such as surgery, combat or sports.”

Normally, adult, tissue-specific stem cells hold their fire because of a deep freeze called quiescence to un- necessary cell division in the absence of injury. In a 2014 paper published in Nature, Rodgers and Rando showed in laboratory mice that an injury to the muscle of one leg caused a change in the muscle stem cells of the other leg. Those cells entered what the researchers called an “alert” state of the cell cycle that is distinct from either fully resting or fully active stem cells.

The fact that muscle stem cells distant from the injury were alerted indicated that the damaged muscle must release a soluble factor that can travel throughout the body to wake up quiescent stem cells. Rodgers and his colleagues found that a protein called hepatocyte growth factor, which exists in a latent form in the spaces between muscle cells and tissue, can activate a critical signal- ing pathway in the cells by binding to their surfaces. This pathway stimulates the production of proteins im- portant in alerting the stem cells. But it wasn’t known how HGF itself became activated.

In the new study, Rodgers and his colleagues identi- fied the activating factor by injecting unjured animals with muscle tissue and serum isolated from animals with an induced muscle injury. (Mice were anesthetized prior to a local injec- tion of muscle-damaging toxin; they were given pain relief and antibiotics during the recovery period.) After 2.5 days, the researchers found that muscle stem cells from the recipient animals were in an alert state and completed their first cell division much more quickly than occurred in animals that had re- ceived blood serum from uninjured mice.

Increased levels of a protein

“Clearly, blood from the injured ani- mal contains a factor that alerts the stem cells,” said Rando. “We wanted to know, what is it in the blood that is doing this?”

The researchers found that the serum from the injured animals had the same levels of HGF as the control serum. However, it did have increased levels of a protein called HGFA. By snipping it into two pieces. Treating the serum with an antibody that blocked the activity of HGFA elimi- nated the recovery benefit of pretreatment, the researchers found.

In a related experiment, exposing the animals to a single intravenous dose of HGFA alone two days prior to injury helped the mice recover more quickly. They scampered around on their wheels sooner and their skin healed more quickly than mice that received a control injection. They also regrew their hair around the shaved sur- gical site more completely than did the control animals.

“Just like in the muscles, we saw the responses in the skin were dramatically improved when the stem cells were alerted,” Rando said.

In addition to pinpointing possible ways to prepare people for surgeries or other situations in which they might sustain wounds, the researchers are in- tigued by the role of HGFA. See RANDO, page 5.
Huddled over a virtual dissection table, Stanford medical students zoomed in on glistening muscles and nerves in the neck by swiping their fingers across the giant touchscreen designed to visualize an entire body in three dimensions. What they were looking at, however, were not virtual renderings of human anatomy, or even images of the real thing: rather, they were examining high-resolution photographs of wax models made between the mid-17th and mid-19th centuries.

“We were shocked to know that they were real waxes,” said Shayan Fakurnejad, a second-year medical student and teaching assistant in a clinical anatomy class where the digital images are used. “It is really interesting to be able to manipulate them in 3-D space.”

Paul Brown, DDS, a consulting associate professor of anatomy at Stanford, led the effort of photographing about 200 of the more than 1,400 wax figures at La Specola, a natural history museum in Florence, Italy, in an effort to make them more accessible. Most of the wax figures were created to demonstrate one body part or system, although some demonstrate more.

A surgeon would dissect a body, and an artist would then cover the body part in plaster to create a mold. Then, the artist would pour colored wax into the molds, and add more detail by arranging silk threads to exactly reproduce capillaries and nerves. The workshop obtained the images have been used in a variety of courses as visual aids to enhance learning.

Steeped in history

“The Venus sculpture is stunning,” said Brown, as he swiped through computer images of a wax model of a woman, reposing on a crimson silk bed. Her brown hair framed her face perfectly. She wore only a pearl necklace. In the next image, the model’s chest plate was off, revealing her rib cage, breast and the subcutaneous muscles.

The following images exposed her lungs, heart and uterus. Brown continued. “Look at the detail. Isn’t that phenomenal?” The final layer of the anatomical Venus de Medicis revealed the inside of her heart, as well as a fetus in her uterus.

La Specola, home of the Venus sculpture and other wax figures, is one of the oldest scientific museums in the world. Between 1775 and 1850, the museum was host to a wax modeling, or retopologizing, workshop for the purposes of both art and medicine. “This is not a morbid collection,” said Claudia Corti, a zoologist and curator of the wax collection at La Specola. “It’s made for scientific public education. It came out during the Enlightenment period, when [artists] were trying to work precisely on a scientific basis.”

Since its inception, the museum has attracted medical students and the general public alike. “Every once in a while, someone will faint,” Brown said. The waxes are still invaluable for people who want to take an elaborate look at the human body is put together, Corti said.

Better than an app

Medical students agree. Although they dissect real cadavers, images of the waxes help illustrate anatomical features, said Karthik Nathan, another second-year medical student and teaching assistant. For example, real cadavers have a lot of connective tissue and layers of fat between the muscles, which makes it hard to distinguish as a fine level. The waxes, on the other hand, show clear muscle borders, Nathan said. “Also, some of the smaller arteries, veins and nerves are a lot more difficult to dissect,” he said.

“It is really interesting to be able to manipulate them in 3-D space.”

The images help show a complete product if students don’t have enough time to finish their dissections, Nathan added. “The waxes, students said, are also far better than the simulated images they access, which are typically more realistic.”

HGF and HGFA might play in aging

Rando

HGF and HGFA might play in aging. It’s known that stem cell activity diminishes with advancing age, said Robert Chase, MD, professor emeritus of the cardiovascular institute and neurology and neurosurgery.

“Stem cell activity diminishes with advancing age, and older people heal more slowly and less effectively than younger people. Might it be possible to restore function.” The work is an example of Stanford Medicine’s focus on precision health, the goal of which is to anticipate and prevent disease in the healthy and precisely diagnose disease in the sick.

Rando is a member of Stanford’s Bio-X, Neurovascular Institute and Cardiovascular Institute. Other Stanford co-authors are research associates Matthew Schroeder and Chantia Ma.

The research was supported by the National Institutes of Health, the Department of Veterans Affairs, The Donald E. and Delia B. Baxter Foundation and the Glenn Foundation for Medical Research.

Swap

HGF and HGFA might play in aging. It’s known that stem cell activity diminishes with advancing age, and older people heal more slowly and less effectively than younger people. Might it be possible to restore function.

The initial lab swap event, held in conjunction with the School of Medicine, brought together over 100 labs and saved an estimated $100,000 in research-related costs. With that success, Cisneros and Sahai knew that this behavior was already happening at a smaller scale,” he said.

According to Sahai, the first lab swap diverted enough waste to fill a small swimming pool. “The amount of detail is astounding.”

Better than an app

According to Cisneros, an important goal of the lab swap is to make for a richer experience. “Anatomy is really body geography — where it is and what do you call it,” he said. With detailed notes, the waxes will offer a distinct edge over traditional methods of learning human anatomy, he said.

The following year, the event partnered with the Stanford BioScience Student Association and was co-sponsored by Peninsula Sanitary Service Inc. and the Stanford Property Management Office. At the end of the event, PSSI collected leftover materials and either disposed of the equipment properly or donated it to local high schools.

Spreading sustainability

In addition to diverting waste and saving money for researchers, a major aim of the lab swap is to build an awareness of sustainability in labs.

“Anatomy is really body geography — where it is and what do you call it.”

Current year’s event was just — a sharing of not only materials, but of ideas, conversations and laughs at some of the outdated equipment.

“We can use sustainability to create community,” Sahai said. “That’s always fun to see.”

HGF and HGFA might play in aging.

By Devika G. Bansal

Continued from page 4

HGF and HGFA might play in aging.

Rando continued from page 4

HGF and HGFA might play in aging.

Swap continued from page 3

HGF and HGFA might play in aging.

The following year, the event partnered with the School of Engineering, had about 75 labs in attendance and saved researchers an estimated $100,000.

This year’s event was hosted by the Stanford BioScience Student Association and was co-sponsored by Peninsula Sanitary Service Inc. and the Stanford Property Management Office. At the end of the event, PSSI collected leftover materials and either disposed of the equipment properly or donated it to local high schools.
Our findings suggest a limited understanding among physicians and patients of the meaning of genetic testing results," said Allison Kurian, MD, associate professor of medicine and of health research and policy at Stanford, and principal investigator of a pilot study designed to educate patients about uncertain variants—variants of uncertain significance should not be considered to confer high cancer risk, and that patients with these variants should not be subjected to unnecessary and unproven cancer screening tests. "These results suggest that patients are not aware of the role of genetic counselors or their availability to explain the implications of uncertain variants to known or at-risk individuals," Kurian said. Kurian is the lead author of the study, which was published online April 12 in the Journal of Clinical Oncology. "The findings come on the heels of a February study by many of the same researchers showing that physicians often fail to recommend genetic testing for breast cancer patients who have high risk for breast cancer because of a family history. The results are difficult to interpret without the help of a trained genetic counselor," Kurian said.

The implications of this study's findings are significant because 90 percent of women with breast cancer who have genetic testing ever discussed their test results with a genetic counselor, and between one-quarter and one-half of the surveyed breast cancer surgeons indicated they treat women with VUS differently than women with known cancer-associated mutations, the researchers found. Furthermore, some women undergo surgery prior to receiving genetic testing or seeing the results.

Kurian said the results confirm findings of a study published online November 2011 in the journal Breast Cancer Research and Treatment. The study was supported by the National Institutes of Health, the California Department of Public Health and the Centers for Disease Control and Prevention.

Kurian has received research funding from Invitae, Myriad Genetics, Ambry Genetics, GenDx and Genomic Health. Stanford's departments of Medicine and of Health Research and Policy also supported the work.

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The need for genetic testing

The findings come on the heels of a February study by many of the same researchers showing that physicians often fail to recommend genetic testing for breast cancer patients who have high risk for breast cancer because of a family history. The results are difficult to interpret without the help of a trained genetic counselor. "The gaps identified in this study are striking," said Jagsi, who is a professor of medicine and of health management and policy at the University of Michigan.

Expertise to interpret the results

Although genetic testing has become more common and less costly, it’s also become more confusing. The advent of multiplex gene panels that simultaneously test for mutations or variations in many different genes can be daunting for patients. "You’re being inundated with information and need help of a trained genetic counselor. Uncertainties as to the meaning of test results may lead less-experienced patients to recommend aggressive treatment in the form of bilateral mastectomies, or cause women to opt out of treatment," Kurian said.

Conversely, high-risk women who do carry dangerous mutations need this information to make informed decisions about their health care choices. “The gaps identified in this study are striking,” said Jagsi, professor and deputy chair of radiation oncology at the University of Michigan. “It is critical to ensure that patients at high risk for known cancer-associated mutations are fully informed of the potential benefits of genetic testing, and counseled accurately about the meaning of test results.”

“WE’re learning that clinicians’ knowledge of breast cancer genetics can be highly variable.”

Mastectomy continued from page 1

For this study, the research team also investigated and compared women for whom the test results were confirmed by a second independent test. “The goal of our research was to determine whether patients would want to undergo cancer treatment if they learned their cancer was the result of a genetic mutation, and whether patients who have undergone genetic testing and who have genetic counselors also want them to refer women to genetic counselors,” said Kurian.

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Blood

continued from page 1

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The Jarvik 2015 ventricular assist device is designed for young children with advanced heart failure who are awaiting a transplant. Stanford will be one of the sites testing the device.

Christopher Almond on pediatric heart pump trial

Christopher Almond

During the wait for a heart transplant, patients with advanced heart failure can be supported with a ventricular assist device, an artificial pump that helps the heart move blood through the body. But the VAD now used for babies and small children — the Berlin Heart — has drawbacks. The pump carries a 30% rate of stroke and is unwieldy: The driver, which sits outside the body, is about the size of a shopping cart. For these reasons, children supported with the Berlin Heart must stay in the hospital until a donor heart becomes available. This can take months.

In recent years, researchers have developed a replacement for the Berlin Heart called the Jarvik 2015. The new device is a completely implantable pump that is much smaller than current models, weighs about 5 pounds, and can power the heart 24 hours a day. In May, Stanford researchers reported the findings of a clinical trial in which children who are on the heart transplant waitlist are implanted with the Jarvik 2015. The device supports the child’s native heart in the same way an adult VAD does, but the Jarvik 2015 is about the size of a shopping cart. Unlike adult VADs, which can only support the heart for up to a year, the Jarvik 2015 pumps can help children with advanced heart failure survive long enough to receive a heart transplant. The device is named for Dr. Robert Jarvik, whose team was the first to implant a total artificial heart into a human — a feat that earned him worldwide fame.

The Jarvik 2015 has several advantages over other VADs. The main challenge of miniaturizing any pump technology is to design it in a way that does not crush fragile red blood cells as they pass through the device, which is estimated to occur millions of times per minute. An earlier version of the Jarvik 2015 pump tended to break red blood cells and had to be redesigned. Interestingly, a relatively minor design change caused a big reduction in the pumps red cell breakage.

A second challenge is that the flow rate through miniaturized pumps is much lower than for adult VADs, which greatly increasing the chance that clots can form inside the pump. When clots form, they can break off and travel through the bloodstream to the brain, causing stroke. Jarvik 2015 pumps are designed to make it much less likely to clot. However, the Jarvik 2015 pump has a relatively low risk of clot formation.

How will the trial work?

How does Stanford’s leadership role in this trial fit in with our larger history as a center of innovation in heart transplant?

Stanford has been a pioneer in developing cardiac transplantation and therapies for children with end-stage heart failure. In 1984, we performed one of the world’s first heart transplants in a young child. We have been refining pediatric heart-transplant techniques ever since.

Stanford has also been a pioneer in the field of heart pumps. In 2004, Packard Children’s was one of the very first U.S. hospitals to use the Berlin Heart. Until that time, the Berlin Heart had been used primarily in Europe. Under the direction of pediatrician David Rosenblatt, Packard Children’s petitioned the FDA to import the device from Germany for a 5-month-old boy, who was then the youngest child in the world to receive the device. The heart was discovered to be failing; the Berlin Heart was then the only pump available for that age. By contrast, the Jarvik 2015 is a fully implantable pediatric VAD.

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Stanford collaboration with pharmaceutical company funds two new projects

Two research projects have been selected for one-year funding by the Stanford Center for Clinical Research and the pharmaceutical company AstaZeneica.

The second year of a three-year collaboration between the organizations. The focus of the collaboration is cardio-metabolic and respiratory diseases, oncology, mobile health, innovations in clinical trial design and operations, and education and training initiatives.

The 2017-18 grantees and their projects are:
- Daniel Rubin, MD, MS, associate professor of biomedical data science, of radiology and of medicine, who is using electronic health records from multiple institutions to build statistical models that will relate patient treatments to outcomes. Focusing on metastatic breast cancer, he will compute predictive statistical models to identify best treatments, such as the decision about when to switch from endocrine therapy to chemotherapy, which chemotherapy drug to administer and when to stop one treatment or start another. Ultimately, he hopes to expand this infrastructure to develop decision models for a variety of diseases.
- Tina Hernandez-Boussard, PhD, associate professor of medicine, of surgery and of biomedical data science, who proposes building a tool that will automate the manual and extremely labor-intensive staging of prostate cancer using both clinical and pathological data captured in the electronic health record. She hopes such automated staging will improve care coordination, physician workload, hospital certification and national cancer surveillance efforts.

The Stanford-Astra-Zeneca collaboration is distributing $2 million over three years to support six innovative research projects by Stanford investigators. The projects each receive $260,000.

Trials led by Mark Genovese wins Clinical Research Forum award

By Bruce Goldman

A clinical trial led by Mark Genovese, MD, Stanford professor of immunology and rheumatology, has been recognized by the Clinical Research Forum as one of the top 10 clinical studies of 2016. The trial, which focuses on improving care being conducted at nearly 60 research institutions and hospitals across the United States, as well as at partner institutions around the world.

Known as the RA-REACON trial, the multicenter study headed by Genovese, who holds the James W. Raitt, MD, Professorship, tested a new rheumatoid arthritis drug, baricitinib, in more than 500 patients for whom other therapies had failed. The new drug was found to be safe and effective, significantly improving the conditions of more than 50 percent of the patients within 12 weeks. The improvements seen in all groups of patients treated with baricitinib largely remained at 24 weeks.

The Clinical Research Forum is dedicated to providing leadership to the clinical and translational research enterprise and to promoting understanding and support for clinical research and its impact on health and health care.

The organization conducts annual competitions to determine the 10 outstanding research accomplishments in the United States. Winners are chosen based on the degree of innovation and novelty involved in the advancement of science; contribution to the understanding of human disease or health; and the potential of the investigation to the diagnosis, prevention and/or treatment of disease.

The awards were presented April 18 at a ceremony at the National Press Club in Washington, D.C. Members of the RA-REACON research team and the Ellen Congressional representatives on Capitol Hill on April 19 to brief them on findings of the trial and the critical and necessary role of federal funding for clinical research.

Andrew Hoffman, Ann Weinacker appointed senior vice chairs in Department of Medicine

Professors of medicine Andrew Hoffman, MD, and Ann Weinacker, MD, have been named senior vice chairs in the Department of Medicine.

The appointments are part of an effort to strengthen ties between the Department of Medicine and other organizations and departments within the School of Medicine.

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Weinacker, a pulmonologist and critical-care specialist who serves as the associate chief medical officer of patient care services at Stanford Health Care, has been named senior vice chair for clinical operations. She will represent the department on clinical issues and collaborate with Stanford Health Care.

Twelve new members will be inducted into the National Academy of Medicine, including eight current faculty members who will be promoted to the rank of full professor from the ranks of associate professor or professor.

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