Dermatologists with access to free samples write costlier prescriptions, researchers say

By Krista Conger

Dermatologists with access to free drug samples are more likely than those without access to samples to write prescriptions for drugs that are more expensive, according to a study by researchers at the School of Medicine.

Although studies have shown that most physicians do not believe that the availability of free samples affects their behavior or recommendations for patients, the researchers found that the average retail cost of the prescriptions written by dermatologists at an academic medical center where such samples are prohibited.

The results of the study, which was published April 16 in JAMA Dermatology, are likely to add fuel to an ongoing debate about whether free drug samples are beneficial or instead skew the prescription tendencies for genetic counselors, clinicians and individual patients, as well as for clinical-trial designers. It could also help shed light on the underlying causes of Alzheimer’s disease.

Seven of 15 patients developed severe liver toxicity and died after taking a hepatitis B drug as part of a clinical trial in the early 1990s. Had a special variety of laboratory mouse been available then, that outcome could have been avoided, according to a study by researchers at the School of Medicine.

A few years ago, Gary Pelz, MD, PhD, professor of anesthesia, in collaboration with researchers at the National Academy of Sciences determined that the drug administered in that tragic trial two decades ago — which an investigation conducted by the National Academy of Sciences found to be dangerous during rigorous preclinical toxicity tests — caused severe liver toxicity when given to the bioengineered mice with humanized livers. This observation would have served as a bright red stop signal that would have prevented the drug from being administered to humans.

The discovery holds implications for genetic counselors, clinicians and individual patients, as well as for clinical-trial designers. It could also help shed light on the underlying causes of Alzheimer’s disease — a progressive neurological syndrome that robs its victims of their memory and ability to reason. Its incidence increases exponentially with age, and women are at higher risk for Alzheimer’s disease.

The scientists arrived at their findings by analyzing data on large numbers of older individuals who were tracked over time and noting whether they had progressed from good health to mild cognitive impairment — from which many move on to develop Alzheimer’s disease within a few years — or to Alzheimer’s disease.

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Short home videos, such as those posted on YouTube, may become a powerful tool for diagnosing autism, according to a study whose senior author is a scientist at the School of Medicine.

No biochemical or physical tests for autism have been established, so the developmental disorder is diagnosed by observing a child for such traits as repetitive behaviors, poor language skills and lack of eye contact. On average, children with autism are diagnosed at age 4, though their parents often suspect they have autism by age 2 or 3.

Dennis Wall is senior author of a paper whose finding suggests that when started early, at age 2 or 3.

Vincent Fusaro, PhD, a research associate author of the paper, which was published April 16 in "Our new paper supports the hypothesis that we can videos of children in natural settings, the study found. A low-cost empowerment program decreases incidence of rape in Kenya

Study: Home videos could be powerful tool for diagnosing autism

By Erin Digita

Numerous women in Kenya slums sharply curtail rape and sexual harassment of these girls, who live in an environment where women have low status and are frequently attacked, a large new study shows.

The findings, by researchers at the School of Medicine, Lucile Packard Children's Hospital Stanford and the nongovernmental organization Ujamasia-Africa/ No Means No Worldwide, validated the program's effectiveness in combating an appallingly common hazard among girls living in the slums of Nairobi's rape. The researchers found that nearly 18 percent of participants had been raped in the year before their program began.

"This study in very poor neighborhoods in Africa demonstrated that there is a very high baseline rate of gender-based violence, but a simple intervention empowered girls to take responsibility for protecting themselves, and this leads to a major decrease in incidence against those girls," said Yvonne Maldonado, MD, the senior author of the study, published April 13 in Pediatrics. Maldonado is professor of pediatrics at Stanford and chief of pediatric infectious diseases at the children's hospital.

The intervention involves teaching girls verbal and physical techniques to prevent sexual violence and assault. The study, which evaluated the effectiveness of these techniques for 1,978 adolescents living in Nairobi slums, confirmed the success of a smaller pilot study of the same program that Stanford and No Means No Worldwide published last year.

Funding off attacks

In the new study, more than half of the girls in the intervention group who had learned in their training to fend off rape or stop sexual harassment, halting 817 assaults and 957 harassment situations.

The rate of rape dropped from 17.9 per 100 person-years to 11.1 per 100 person-years in the girls who received the training. (The rate was 17.9 per person-year to account for the fact that questionnaires given to the girls before and after training asked about different lengths of time: The one used to document the girls' experience before training asked about the prior 12 months, while the follow-up questionnaire asked about the 10.5-month period since the girls had begun participating in training.)

The results demonstrate that the program is both effective and scalable to large populations, the study's authors said.

"We're teaching girls that it is OK to say no without feeling guilty, teaching them 'I have permission to defend myself,'" said Lee Paiva, a co-author of the study and co-founder of No Means No Worldwide, who helped develop the intervention curriculum. "As in many cultures, the topics of rape and sexual assault are usually treated with silence and shame in the girls' communities, she said. "We're countering that, with the intervention curricular material that I just go through as women.'"

No Means No Worldwide is studying empowerment training for girls in the context of its larger efforts to stop gender-based violence in Africa and around the world. The organization has also developed a program for boys called Your Moment of Truth, which aims to change "gender scripts" that have been identified as a leading cause of violence against women. In addition, they teach the defense techniques to Kenyan grandchildren, another population vulnerable to sexual assault.

"This is the first time anyone's proven that an intervention can be delivered with a low-cost, simple intervention," said Jake Sinclair, MD, who co-founded No Means No Worldwide with Paiva, at Stanford Program, the National Institutes of Health and the National Library of Medicine. Stanford's Department of Pediatrics also supported the research.

The study's subjects were the current year's 2,406 high school girls, ages 13 to 20, attending schools in impoverished Nairobi slums: 1,978 received 12 hours of empowerment training over six weeks, as well as two-hour refreshing courses at three-, six- and 10-month intervals. A 428 in a comparison group received a 90-minute life-skills class that is the current national standard in Kenya. The empowerment program included lessons on self-efficacy, boundaries and personal awareness; assertive communication skills; assertive sexual boundaries; and a variety of physical skills for defending against and escaping from single or multiple attackers. Even after the training began, both groups answered anonymous questionnaires about their recent experiences of rape and sexual assault.

Puting the skills to use

At the start of the study, nearly one in five girls from the intervention group reported that they had been forced to have sex in the prior six months. By the end of the 10.5-month study, the rate had dropped by more than a third.

Girls who received training, 52.3 percent fended off rape in the subsequent 10.5 months. Of these girls, 45 percent used verbal skills alone, 30 percent used physical skills, 23.4 percent used a combination of verbal and physical skills.

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Scientists develop technique to reverse-engineer developing lung

By Tom Abate

Consider the marvel of the embryo. It begins as a glob of identical cells that change shape and function as they mature to become the cells of lungs, muscles, nerves and all the other specialized tissues of the body. Now, in a feat of reverse tissue engineering, Stanford researchers have begun to untangle the complex genetic code that allows embryonic cells to proliferate and differentiate into all of the specialized cells that perform myriad biological tasks.

A team of interdisciplinary researchers took lung cells from the embryos of mice, choosing samples at different points in the development cycle. Using the new technique of single-cell genomic analysis, they recorded what genes were active in each cell at each point. Though they studied lung cells, their technique is applicable in any type of cell.

"This lays out a playbook for how to do reverse tissue engineering," said Stephen Quake, PhD, the Lee Otterson Professor in the School of Engineering and a Howard Hughes Medical Institute investigator.

The researchers’ findings are described in a paper published online April 13 in Nature, Quake, who also is a professor of molecular and genetic medicine, is the senior author. The lead authors are postdoctoral scholars Barbara Treutlein, MD, PhD, and Doug Brownfield, PhD.

The researchers used the reverse-engineering technique to study the cells in the alveoli, the small, balloon-like structures at the tips of the airways in the lungs. To capture individual alveoli, the researchers used standard enzymatic techniques to dissolve the proteins that hold the lung cells together in tissue form, then sorted out the specific alveolar cell types that were studied.

Their next steps involved newer techniques at the heart of their reverse-engineering process.

Recall how eyepieces work. Squeeze the rubber bulb to expand the lens, and it begins to fill with fluid. Squeeze the bulb again to force the fluid out. In recent years, biologists have used those basic principles to develop microfluidic devices of such precision that they can capture a single cell out of solution and isolate it in a chamber to analyze its genetic material.

Quake’s lab has pioneered the use of microfluidic devices to study single cells. In this study, they used microfluidic devices to sort a sample lung cells. Then they used single-cell genomic sequencing to ensure which genes were active in each cell at each time.

How did they decode genomic activity in a single cell? DNA in the nucleus of every cell contains the full genome for that organism. That’s why it’s possible to build an organism from a single cell. But only some of those genes are active in any given cell at any given time. That’s why lung cells are different than hair cells; each cell has a different set of active genes directing its functions.

Genes direct cellular activity by making proteins or “expressing” messenger RNA. Each mRNA instructs the cell to make a particular protein. Cells are essentially a group of interacting proteins. Therefore knowing which mRNAs are active in a cell can tell us a lot about how it works when it was captured in the microfluidic device.

Using this process the Stanford researchers revealed for the first time precisely which genes regulate the development of these particular lung cells at each stage along the way to mature alveoli.

One important finding involved the development of the alveoli, where the lung meets blood to perform the gas exchange that keeps us alive.

Alveolar type-1 cells are the flattest cells in the body. Blood cells dock alongside them to deliver oxygen or pick up carbon dioxide. The thickness of the cell is vital for the function of the lung. The researchers found that the alveolar cells express a large set of genes that allow them to do this.

Alveolar type-2 cells are compact and cuboidal. They secrete proteins to keep the alveoli from collapsing. They also build the lining of the lung, through which oxygen and carbon dioxide can move.

Using single-cell genomics allows the researchers to reverse engineer the development process to show how a single progenitor cell type gives rise to both of these different, mature alveolar cells.

The researchers also captured cells in transition from the progenitor to mature cell state, gaining crucial insights into the mechanisms of alveolar cell differentiation. The reverse-engineering technique — capturing individual cells at different stages of embryonic development and assessing gene activity through mRNA sequencing — can be used to reverse-engineer all tissues.

In addition to studying embryonic development, the technique could be used in clinical settings. For example, clinicians could study differences between individual cells in a tumor, improving our understanding of the stages of cancers and leading to better, more targeted therapies.

“Technology represents a quintessential leap forward in our ability to apprehend the full diversity of cells in a given population, including rare ones that could have special functions,” said Tushar Desai, MD, MPH, assistant professor of pulmonary and critical care medicine at Stanford and co-author of the paper. “Because a comprehensive molecular characterization of each cell is possible, we now can study how they send and receive, a snapshot of the communication between individual cells will also emerge and may suggest therapeutic targets in disease.”

The other Stanford co-authors of the paper are Mark Krasnow, MD, PhD, professor of biochemistry and an HHMI investigator; postdoctoral scholar Angela Wu, MD, and Armeeta N Nikolov, MD, MPH, a clinical fellow in the Department of Bioengineering; research associate Gary Mantalas; and F. Hernan Espinosa, PhD, an HHMI research specialist.

The research was supported by the National Heart, Lung and Blood Institute, the National Institutes of Health, the Parkinson’s Disease Foundation and the Howard Hughes Medical Institute. The work was also supported by the Department of Bioengineering, which is jointly operated by the School of Engineering and the School of Medicine.

By Kathleen J. Sullivan

“In an era in which people are living and working longer, universities may have a new role to play in society by offering programs designed to revitalize, re-engage and reconnect people who are midway through their life journeys and are looking for new pathways in their professional and personal lives,” said Stephen Quake, PhD, the senior author. The lead authors are postdoctoral scholars Barbara Treutlein, MD, PhD, and Doug Brownfield, PhD.

Typical fellows will have a 20- to 30-year career of achievements and contributions in the public and private sectors. They will be leaders who are eager to join their peers for a program of personal renewal, intellectual exploration, physical recalibration and societal engagement.

The institute will welcome its inaugural class in January of 2015. The program of transformation that will connect established leaders with experienced individuals who are ready for new models for the role of universities in teaching and learning, helping to create new pathways to education, will be able to audit Stanford classes. Each fellow will have a faculty adviser. With the help of those advisers, fellows will learn how to create scholarly pathways designed to help them achieve their goals.

The fellows will participate in a core program that includes weekly discussions of seminars with faculty on a broad range of topics, and weekly receptions for fellows that share learning that they have learned and consider issues of life transitions from a variety of disciplinary perspectives.

The program will include one- to two-day meetings on key social and intellectual challenges, such as the brain and behavior across the life journey; the transition to sustainability; the societal and ethical implications of technology and the widening economic gap in the United States.

Monthly dinner soirées will be held with faculty scholars, as well as leaders from the broader Stanford and Silicon Valley communities.

The program will emphasize community building by offering dedicated office space on campus that facilitates networking among fellows and with Stanford faculty, students and staff. The program also will encourage interactions with undergraduate and graduate students in research, academic and service projects that foster intergenerational learning.

To promote future longevity and success, each fellow will have the opportunity for a health assessment, including a health, exercise and personal well-being plan.

Sponsors and partners of fellows also will be welcome to participate. Applicati- ons for the initial fellowships are available on the institute’s website: http://dci.stanford.edu — and are now being accepted.

Kathleen J. Sullivan is a public informa- tion specialist in the Office of University Communications.
Gene panel effectively screens for cancer-related mutations

By Krista Conger

As many as 10 percent of women with a personal or family history of breast or ovarian cancer have at least one genetic mutation that increases their risk of developing cancer, but they don’t always receive recommendations to change their care, according to a new study by researchers at Stanford School of Medicine.

The women in the study did not have mutations in BRCA1 or BRCA2 (mutations in these genes are strongly associated with hereditary breast and ovarian cancer), but they did have mutations in other cancer-associated genes. They were recruited using what’s known as a multiple-gene panel to quickly and cheaply screen just a few variants of uncertain significance out of several genes simultaneously.

The findings illustrate a form of care recommended by researchers based on what is known about a disease. Although such panels are being widely clinically available, it’s not been clear whether their use can help patients or affect medical recommendations.

A middle ground

“Although whole-genome sequencing can be effective in identifying disease-causing mutations, it may be premature to consider doing on everyone,” said James Ford, MD, a dermatologist, assistant professor of medicine (dermatology) and of genetics, is the senior author of the study, published April 14 in the Journal of the American Medical Association.

Gene panels offer a middle ground between sequencing just a single gene like BRCA1 that we are already doing, and attempting to sequence every genome in the gene. It’s a focused approach that should allow us to capture the most relevant information,” Ford said.

Ford, an associate professor of medicine and of genetics, is the senior author of the study, published April 14 in the Journal of Clinical Oncology. Alison Kurian, MD, associate professor of medicine, and the Stanford Cancer Institute’s research and policy associate director of the Clinical Cancer Genetics Program, is the study’s lead author.

Ford was a co-author of a recent paper in The Journal of the American Medical Association that highlighted the challenges and opportunities of making whole-genome sequencing clinically available for seemingly healthy people. Although that study showed that whole-genome sequencing can be potentially life-saving, the challenges involved in sequenced samples and the costs to make up all of a person’s DNA, and then translating the results into clinical care recommendations is significant.

“This indicates that using gene panels to screen for potentially harmful variants can be clinically useful in certain groups of patients,” said Kurian. “It also shows that patients or some of whom have been given blood samples for research as many as 10 years earlier, are willing and interested in receiving this type of follow-up information and to incorporate it into their health care plans.”

Quicker, easier and cheaper

Gene panels allow researchers to learn the sequences of several genes simultaneously from a single blood sample. It stands to reason that screening for mutations in just a few select genes is quicker, easier and cheaper than whole-genome sequencing. The technique usually focuses on fewer than 100 of the approximately 21,000 human genes. But until now, few studies have investigated whether homeing in on a pre-determined panel of suspects can actually help people.

In the study, Kurian and Ford assessed the sequences of 42 genes known to be associated with the development of breast or other cancers, or involved in DNA repair pathways that nipping potentially dangerous mutations in the bud. Blood samples in the study came from 198 women who underwent BRCA1 and BRCA2 testing at the Stanford Cancer Genetics Program between 2002 and 2012. At the time of the testing, the women were asked if they would like to donate an additional blood sample for future research.

Of the 198 women, 57 carried BRCA1/2 mutations. Ford and Kurian found that 14 of the 141 women without a BRCA1/2 mutation had clinically actionable mutations in one of the 42 genes assessed by the panel. (An actionable mutation is a genetic variation correlated with an increased risk that clinicians would recommend a change in routine care — such as increased screening — for carriers.)

Eleven of the 14 women were reachable by telephone, and 10 accepted a follow-up appointment with a genetic counselor and an oncologist to discuss the new findings. The family members of one woman, who had died since giving consent, were also invited to have regular screenings for gastrointestinal cancer.

Six participants were advised to schedule annual breast MRIs, and six were advised to have regular screenings for gastrointestinal cancer.

One woman with a history of both breast and endometrial cancer, learned she had a mutation that causes Lynch syndrome, a condition that increases the risk of many types of cancers. As a result, she had her ovaries removed and underwent a colonoscopy, which identified an early precancerous polyp for removal.

Guidance for care decisions

“An important question about the use of these gene panels is whether they can allow us to provide additional genetic guidance and screening,” Kurian said. “We found that the participants were interested and willing to receive the additional information, and they were generally pleased at the results, which helped them make decisions about their clinical care.”

Screening with gene panels does not, however, eliminate the problem of variants of uncertain significance. This term is used when a gene sequence deviates from the consensus, but the clinical effect of that change is unknown. Each of the 141 women in the study had about two variants of uncertain significance in the 42 genes studied.

“This problem is shared with whole-genome sequencing,” Ford said, “and should suffice as we gather even more data on the effect of specific mutations in these genes.”

The National Institutes of Health-sponsored Clinical Genome Research Program was created to speed the identification and aid in the interpretation of clinically important variants.

Yet even though the study shows that gene panels can be useful in some groups, it may be some time before they could be routinely used in the general population, the researchers said.

“It’s a slippery slope at the moment,” Ford said. “We need to know how prevalent these cancer-associated genes are in the general population.”

Other Stanford co-authors are genetic counseling program manager Meredith Mills; genetic counselor Kerry Kingham; research associate Michelle McPherson, PhD; professor of health research and policy Alice Whittemore, PhD; senior research scientist Valerie McGuire, PhD; and associate professor of medicine Uri Lada- baum, MD. Researchers from the San Francisco-based Invitae Corp., a genetic testing company, were also co-authors. Invitae sequenced the genes in the study and helped to fund the study.

Additional funding came from the Breast Cancer Research Foundation, a Stanford Cancer Institute Developmental Research Award in Population Sciences, the Jan Weiner Junior Faculty Chair in Breast Oncology at Stanford and the National Institutes of Health. Stanford’s Department of Medicine and Department of Genetics also supported the work.

Neural activity promotes brain plasticity through myelin growth

By Christopher Vaughan

The brain is a wonderfully flexible and adaptive learning tool. For decades, researchers have known that if you lose a function, called plasticity, coming from selective strengthening of well-used synapses — the connections between nerve cells.

Now, researchers at the School of Medicine have demonstrated that brain plasticity also comes from another mechanism: activity-dependent changes in the cells that insulate neural fibers and make them more transmissible. These cells form a specialized type of insulation called myelin.

“Myelin plasticity is a fascinating concept that may help us to understand the brain’s capacity to recover from experience or training,” said Michelle Monje, MD, PhD, assistant professor of neurology and neurosurgery.

The researchers’ findings are described in a paper published online April 10 in Science Express.

In recent years, researchers have learned that neural plasticity based in myelin, and future work on the molecular mechanisms responsible may ultimately shed light on a broad range of neurological and psychiatric diseases,” said Monje, senior author of the paper. The lead author of the study is Stanford postdoctoral research scholar Erin Gibson, PhD, and graduate student David Purger.

“Nerve cells extending neural impulses quickly down a long nerve fiber requires insulation with myelin, which is formed by a cell called an oligodendrocyte that wraps itself around a neuron. Even small changes in the structure of this insulating sheath, such as changes in its thickness, can dramatically affect the speed of neural-impulse conduction. Demyelinating disorders, such as multiple sclerosis, can leave these cells and degrade nerve transmission, especially over long distances.

Myelin-insulated nerve fibers make up the “white matter” of the brain, compared to the “gray matter” that makes up the active neural circuit, making signal transmission along the neural fiber more efficient. It’s much like a system for improving traffic flow along roads that are heavily used, Monje said. And as with a transportation system, improving the routes that are most productive makes the whole system more efficient.

In recent years, researchers have seen clues that nerve cell activity could promote the growth of myelin insulation. There have been studies that showed a correlation between experience and myelin dynamics, and studies of isolated cells in a dish suggesting a relationship between neuronal activity and myelination. But there has been no way to show that neuronal activity directly causes myelin changes in an intact brain. “You can’t really implant an electrode in the brain to answer this question because the resulting injury changes the behavior of the cells,” Monje said.

The solution was a relatively new and radical technique called optogenetics. Scientists insert genes for a light-sensitive ion channel into a specific group of neurons. Those neurons can be made to fire when exposed to particular wavelengths of light. In the study, Monje and her colleagues used mice with light-sensitive ion channels in an area of their brains that con-
Neural activity promotes brain plasticity through myelin growth

Inside Stanford Medicine
April 21, 2014

how does that contribute to disease?" Monje
and colleagues, how is that relevant?"
In the absence of other treatments, such as multiple sclerosis, the leukodystrophies and spinal cord injury.

promote myelin repair in diseases in which myelin is degraded, decisions that were meant to increase myelin. Such a molecular understanding could help researchers develop therapeutic strategies that promote myelin.

activating a probe directly next to the neurons, which would allow researchers to "read" certain movement behaviors in the mice by turning on and off the light. Because the light diffuses from a source placed at the surface of the brain down to the depths, researchers didn't need to insert a probe directly next to the neurons, which would have created an injury.

In the absence of other treatments, such as multiple sclerosis, the leukodystrophies and spinal cord injury.

...he was the first to perform such a treatment, and the practice has since become the standard... "It could be a clinician who found a drug that can be repurposed for multiple uses, or a scientist who knocked out a gene and is wondering how to use the phenotype to treat a disease, or a physician interested in finding some of their patients. Many of these applications come from students who have enrolled in development classes in the Chemical and Systems Biology Department. One such student is currently working on an alternative botanical treatment for an inflammatory bowel disease.

Matching students with mentors who can guide their progress is another benefit of the SPARK program, Garner said. Once a SPARKee himself, he now advises a student group working on a treatment for a lysosomal storage disorder.

In recent years, the program has sparked similar and identical initiatives outside Stanford, fulfilling another of Mochly-Rosen's original visions for the program. Several universities in the United States — in Vermont, New York, New Mexico, and across the globe — in Taiwan, Japan and South Korea — have now launched their own SPARK programs. And many more are in the pipeline.


Mochly-Rosen's vision for SPARK extends beyond the confines of an educational institution. She hopes one day to be able to integrate various institutions' SPARK programs under a single brand that will encourage and attract more companies to invest in early-stage discoveries, and open the doors for better academia-industry collaborations.

A yearly SPARK conference that would showcase successful projects from around the world to enable faster licensing is another goal, she said.

Mochly-Rosen ultimately hopes that programs like SPARK would help make translational research "second nature" to scientists "so that they can come up with better drugs faster," she said.

"We have a social responsibility to bring our discoveries as far as we can to patients," she said.

This year, SPARK applications will be accepted starting in late June. The deadline will be in September. More information on SPARK can be found at http://sparkmed.stanford.edu.

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Gary Peltz helped bioengineer a line of mice whose livers were largely replaced with human liver cells. The mice can help researchers better assess if drugs would be toxic to human livers.

"FAIUI was supposed to be a revolutionary drug," Peltz said. "It looked very promising in preclinical tests. In phase 1, when the drug was administered to subjects for a short period of time, the human subjects seemed to do fairly well." But the phase-2 trial was stopped after 13 weeks, when it became clear that FAIUI was destroying patients’ livers. Before advancing to clinical trials, FAIUI had been tested for as long as six months in mice, rats, dogs and monkeys without any trace of toxicity.

Some years ago, Peltz, working with scientists at the Central Institute for Experimental Animals under the direction of the late Tatsujir Nomura, MD, PhD, bio-engineered mice by very selectively destroying most of their liver cells and repopulating the resulting cavity with human liver cells. The new cells took hold, multiplied and formed structures typical of the human organ. This was possible because these mice deficient immune systems, so the human cells were not rejected. Studies have shown that these mice’s humanized livers express the batteries of enzymes that human livers do and that they mimic the pattern of drug metabolism seen in humans. Except for their impaired immune systems and altered livers, these mice are quite normal. That makes them ideal for liver-toxicity tests.

“When we first made these mice, my goal was to create one that could help us more accurately predict human toxicity," Peltz said. Where better to demonstrate this than by turning back the clock to the preclinical toxicity studies that preceded the disastrous FAIUI trial!

Trough virtual reality and computer simulations, researchers can create逼真 environments that simulate real-life situations. The use of this technology has helped improve the accuracy of drug testing by allowing researchers to test drugs in a controlled environment, reducing the number of animals needed for testing. This has led to significant cost savings and ethical benefits.

The joint Stanford-Oxford initiative on human liver models, which is supported by the Li Ka Shing Foundation, has awarded approximately $807,000 in seed grants to 12 projects. The aims are to improve understanding and research in the data sciences.

High-throughput sequencing serology for infectious disease tracking and microbiome monitoring.

Principal investigator: Scott Boyd (Stanford).

Co-investigators: Shilpa Joshi (Stanford), Benjamini Pinsk (Stanford), Andrew Pollard (Oxford), Robert Tibshirani (Stanford).

Accelerometers in U.K. biobanks: transforming data into meaningful health information.

Principal investigator: Aiden Doherty (Oxford).

Co-investigators: Charlie Foster (Oxford), Peltz and his colleagues set out to determine how their bioengineered mice would fare in comparison with regular lab mice — the kind routinely used in clinical toxicity studies — when each type was given the same drug. They administered four different doses of FAIUI — which has never seen approval for any use since its failure in 1993 - once a day, orally (just as in the clinical trials) then a half-dose over one mouse of each type.

First they dosed the regular mice for four weeks, without eliciting symptoms of liver damage. "We could have backed away from the clinic and repeated dosage without toxicit," Peltz said. He and his team bracketed themselves for what they thought they might be a long, drawn-out repetition with the bioengineered mice. "Within three days, the mice given the highest dose looked terrible, moved lethargically and ate poorly, just like people with liver trouble. By day four, we saw signs that the drug had killed rats, monkeys without any trace of toxicity.

"FAIUI would never have been given to humans if it had been tested in animal models that mimicked the conditions in our mice," Peltz said. "We think they should be used before humans are exposed to new drugs." The improved testing method has two advantages, he noted. First, as in the FAIUI case, drugs that are selectively toxic in human liver can be detected and a tragedy can be avoided. Second, we are less likely to lose a safe and effective drug that would be easily tolerated by liver, Peltz said. "We could have backed away from the clinic and repeated dosage without toxicit," Peltz said. He and his team bracketed themselves for what they thought they might be a long, drawn-out repetition with the bioengineered mice. "Within three days, the mice given the highest dose looked terrible, moved lethargically and ate poorly, just like people with liver trouble. By day four, we saw signs that the drug had killed rats, monkeys without any trace of toxicity.

Second, the high-throughput sequencing serology for infectious disease tracking and microbiome monitoring. The use of next-generation sequencing technology allows researchers to identify and quantify the presence of microorganisms in a sample, which can provide insights into the health status of an individual or a population.

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Alzheimer's disease is the most common form of dementia and affects millions of people worldwide. The development of new treatments and therapies for Alzheimer's disease is a major focus of medical research. The potential for genetic factors to influence the risk of developing Alzheimer's disease is of particular interest. The ApoE gene plays a role in the metabolism of fatty substances, and its three variants, ApoE4, ApoE2, and ApoE3, are associated with different risks of Alzheimer's disease. People who are carriers of the ApoE4 variant are at a higher risk of developing Alzheimer's disease compared to those without the variant. The ApoE4 variant is more common in women than in men, and this difference has been observed in various populations and research studies. For example, a study of women in China found that the ApoE4 variant was associated with a higher risk of Alzheimer's disease in women compared to men. The ApoE4 variant also appears to have a differential effect on cognitive decline, with women carrying at least one copy of ApoE4 having a greater risk of cognitive decline compared to men with the same genetic profile. This differential effect has been observed in multiple studies and is a topic of ongoing research. The genetic factors that influence the risk of Alzheimer's disease are complex and involve multiple genes, and understanding the role of genetic factors in the development of Alzheimer's disease is an important area of research. The potential for personalized medicine, which takes into account genetic and other individual characteristics, is being explored as a way to improve the treatment of Alzheimer's disease. However, the development of effective personalized treatments for Alzheimer's disease remains a challenge and further research is needed to identify effective strategies for prevention and treatment.
Maldonado tapped to lead faculty development, diversity efforts

By Patricia Waldron

Yvonne (Bonnie) Maldonado, MD, professor of pediatrics and of health research and policy, has been named senior associate dean for faculty development and diversity at the School of Medicine, effective May 1. Lloyd Minor, MD, dean of the medical school, said Maldonado is dedicated to nurturing future leaders. “Bonnie is an outstanding investigator and clinician who is committed to making Stanford Medicine a more diverse and inclusive community,” he said.

Maldonado, who is also chief of infectious diseases in the Department of Pediatrics and medical director of the Division of Infectious Diseases and Control at Lucile Packard Children’s Hospital Stanford, will head the Office of Faculty Development and Diversity, formerly the Office of Leadership and Diversity. She will work with medical school departments to recruit and retain a diverse and distinguished faculty.

“I think we can always improve our diversity at all levels in terms of gender, race and ethnicity,” she said. She also said she hopes to expand professional development opportunities to help faculty at all levels succeed in their clinical and research endeavors, as well as to help them with teaching and mentoring students and trainees. She plans to provide these resources within the context of improving work-life flexibility and balance.

In particular, Maldonado said she wants to make Stanford a more nurturing environment for young faculty. “I want to see young faculty succeed, and I’m energized by the frontiers of new clinical research and teaching methods that we can use in medicine, especially at an institution like Stanford, where breaking new ground is the norm,” she said. “However, it takes years to develop a cohort of well-trained faculty, and I want to be a part of supporting the next generation of outstanding teachers, clinicians, scientists.”

Maldonado earned a medical degree from Stanford and completed a residency and a fellowship in pediatric infectious diseases at Johns Hopkins University. A second fellowship brought her to the Centers for Disease Control and Prevention, where she trained as an epidemiologist. She returned to Stanford in 1988.

During her 25 years on Stanford’s faculty, Maldonado has developed training programs for clinical residents and medical students in collaboration with the American Academy of Pediatrics, the Infectious Diseases Society of America, and the Pediatric Infectious Disease Society.

Through her research, she has become a leader in the prevention of HIV transmission from mothers to their infants. She currently studies how family-planning counseling and drug treatment may reduce HIV rates among infants at her research site in Zimbabwe. She has also led polio vaccine studies in Zimbabwe and Mexico to understand the best practices for vaccine use to help reach the worldwide goal of eradicating polio by 2018. “We are delighted that Bonnie has accepted this new role and look forward to her joining the Dean’s Office,” said vice dean Linda Boxer, MD, PhD. “In all her efforts, she will work closely with the departments to ensure that Stanford Medicine is a welcoming community where faculty flourish.”

Patricia Waldron is a science-writing intern for the School of Medicine’s Office of Communications & Public Affairs.

Medical Student Research Symposium set for May 1

Medical students will showcase their medical research projects at the 31st annual Medical Student Research Symposium, scheduled for May 1 in Berg Hall at the Li Ka Shing Center for Learning and Discovery.

More than 40 MD and MD/PhD students at Stanford will present posters on their original research.

The event is free and open to the public. Students will be available at their posters for informal discussions from 3 to 5:30 p.m.

At 5:45 p.m., after closing remarks by Lloyd Minor, MD, dean of the medical school, the Stanford University Medical Center Alumni Association will announce the winning poster presentations.

For more information about this event, contact Maria Visilanti at mmar@stanford.edu.

Ho-Am Prize in Medicine goes to Seung Kim for beta cell research

Seung Kim, MD, PhD, professor of developmental biology and a Howard Hughes Medical Institute investigator, has been named the winner of the 2014 Ho-Am Prize in Medicine.

The prize, which consists of a certificate, a gold medal and 300 million South Korean won (about $288,000), will be presented to Kim at an awards ceremony in Seoul on May 30. During the same week, Kim will lecture on his work throughout South Korea.

Kim studies the development of insulin-producing beta cells in the pancreas. He has identified key pathways involved in beta cell development and expansion, bringing scientists ever closer to being able to generate or regenerate functional human beta cells.

“During his clinical training at Harvard hospitals in Boston, Dr. Kim encountered patients suffering and dying from diseases rooted in the pancreas, including diabetes mellitus and pancreatic adenocarcinoma,” said Stanford President John Hennessy, PhD, in his letter to the Ho-Am committee nominating Kim for the award. “Convinced he could contribute to improving diagnosis and treatment of these diseases, he focused his research investigations on understanding mechanisms regulating development, growth and function of the pancreas. In the past decade he has become a leader in his field, making seminal contributions that have influenced diabetes and cancer research and strategies for clinical therapies.”

“It is a great honor to be recognized by the Ho-Am Foundation for this work,” said Kim. “It reminds me how grateful I am to those who nurtured, trained, guided and supported me, and to my students and research team for their hard work and dedication. That said, we still have a great deal left to accomplish, and the affirmation from this award will help us continue to pursue our goals.”

The Ho-Am Prize, which honors Samsung founder Byung-Chull Lee (nicknamed Ho-Am), is awarded in five categories — science, engineering, medicine, the arts and community service — to people of Korean descent. The community service prize, however, may be given to non-ethnic Koreans for outstanding contributions to Korea and Koreans at home and abroad.

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