Biomarkers tied to chronic fatigue severity

By Bruce Goldman

Researchers at the School of Medicine have linked chronic fatigue syndrome to variations in 17 immune-system signaling proteins, or cytokines, whose concentrations in the blood correlate with the disease's severity.

The findings provide evidence that inflammation is a powerful driver of this mysterious condition, whose underpinnings have eluded researchers for 35 years.

The findings, described in a study published online July 31 in the Proceedings of the National Academy of Sciences, could lead to further understanding of this condition and be used to improve the diagnosis and treatment of the disorder, which has been notably difficult.

More than 1 million people in the United States suffer from chronic fatigue syndrome, also known as myalgic encephalomyelitis and designated by the acronym ME/CFS. It is a disease with no known cure or even reliably effective treatments. Three of every four ME/CFS patients are women, for reasons that are not understood. It characteristically arises in two major waves: among adolescents between the ages of 15 and 20, and in adults between 30 and 35. The condition typically persists for decades.

“Chronic fatigue syndrome can turn a life of productive activity into one of dependency and desolation,” said Jose Montoya, MD, professor of infectious diseases and director of Stanford's Institute for Immunology, Transplantation and Infection.

**‘Solid basis for a diagnostic blood test’**

“There's been a great deal of controversy and confusion surrounding ME/CFS — even whether it is an actual disease,” said Davis. “Our findings show clearly that it's an inflammatory disease and provide a solid basis for a diagnostic blood test.”

Many, but not all, ME/CFS patients experience flu-like symptoms common in inflammation-driven diseases, Montoya said. But because its symptoms are so diffuse — sometimes manifesting as heart problems, sometimes as mental impairment nicknamed “brain fog,” other times as indigestion, diarrhea, constipation, muscle pain, tender lymph nodes and so forth — it often goes undiagnosed, even among patients who’ve visited a half-dozen or more different specialists in an effort to determine what’s wrong with them.

Montoya, who oversees the Stanford ME/CFS Initiative, came across his first ME/CFS patient in 2004, an experience he said he’s never forgotten.

“I have seen the horrors of this disease, multiplied by...”

Imaging reveals how well patients with PTSD will respond to psychotherapy, study finds

By Emma Hiolski

A pair of studies led by researchers at the School of Medicine demonstrates that scientists can predict, with a high degree of accuracy, which patients with post-traumatic stress disorder will respond to a method of psychotherapy often used to treat the condition.

The researchers showed how the treatment, prolonged exposure therapy, works in the brains of PTSD patients and linked brain activity patterns to how well patients responded. The results could lead to personalized treatment for PTSD. The studies were published online July 18 in The American Journal of Psychiatry.

“We understand vanishingly little about how psychotherapy works and for whom it works well,” said Amit Erkin, MD, PhD, the senior author of both studies and an associate professor of psychiatry and behavioral sciences at Stanford. “It’s not even a knowledge gap — more like a knowledge ravine. This is especially an issue for PTSD because the only effective treatment is psychotherapy.”

Lead authorship of the papers is shared by Stanford postdoctoral scholar Gregory Fonzo, PhD, and former Stanford postdoctoral scholar Madeleine Goodkind, PhD, a psychologist at the New Mexico Veterans Affairs Health Care System and an adjunct assistant professor of psychiatry at the University of New Mexico School of Medicine.

**See CFS, page 6**

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**See PTSD, page 7**

**Is it nature or nurture? New study shows both affect behavior in mice**

By Bruce Goldman

School of Medicine investigators have identified a cluster of nerve cells in the male mouse’s brain that, when activated, triggers territorial rage in a variety of situations. Activating the same cluster in the female mouse’s brain produces a similar behavior.

Moreover, whether a male mouse displays territorial aggression depends on whether it’s recently been the sole occupant of a bachelor pad or living in the mouse equivalent of boarding school. The latter makes for good manners; the former, not so much.

In a study published online July 27 in Neuron, the researchers used sophisticated laboratory techniques to determine how much this aggressive behavior owed to environmental...
Somatic cell and gene therapy hold promise for stem cell and gene-based therapeutics to their patients, and these therapies are also becoming more widely used, and these often require freezing. But physicians have been unsure whether freezing fresh embryos changes pregnancy rates. Until now, only small studies have been done, and some have been inconclusive.

To compare success rates of transferring fresh versus frozen embryos, researchers used a large database maintained by Celmatix that contains records of hundreds of thousands of IVF treatments performed at 12 fertility treatment centers in the United States. From the database, two cohorts of 1,455 IVF transfers were selected to study: one group in which all the embryos were frozen before transfer, and another group in which fresh embryos were transferred. Patients in the two cohorts had similar ages, causes of infertility, reproductive histories, numbers of eggs retrieved, number of embryos created and levels of reproductive hormones.

A 73 percent higher pregnancy rate
Fifty-two percent of the embryo transfers performed after embryos were frozen had better pregnancy rates, whereas 45.3 percent of fresh transfers led to ongoing pregnancies. After analyzing all IVF transfers together, the researchers performed separate comparisons of women with lower and higher progesterone levels, as well as of women who were younger and older than 35. Women with lower progesterone levels who received previously frozen embryos did not experience better pregnancy outcomes, regardless of their age. However, among women with higher progesterone levels at the time of egg retrieval, transferring previously frozen embryos resulted in more pregnancies both in younger and older patients. The difference was greatest in patients with high progesterone levels who were older than 35. In this group, 48.4 percent of transfers using previously frozen embryos resulted in pregnancies, compared with 35.2 percent of fresh transfers. In other words, for older women with high progesterone levels, the odds of pregnancy were 73 percent greater following transfer of previously frozen embryos than of fresh embryos.

The new findings may prompt physicians to suggest that patients with high progesterone levels at egg retrieval freeze their embryos and wait for a subsequent cycle to transfer them, said Wang. But other factors also influence the decision about which protocol to use, she said.

“This finding is important because it may suggest a group of women that benefits more from freeze-all IVF,” Wang said. “In cases, as D. Smith Professor in Stem Cell and Regenerative Medicine, and professor of pediatrics and of medicine. "It is a privilege to lead the center and to leverage my previous experience to build Stanford’s preeminence in stem cell and gene therapies," said Roncarolo, who is also chief of pediatric stem cell transplantation and regenerative medicine, co-director of the Bass Center for Childhood Cancer and Blood Diseases and co-director of the Stanford Institute for Stem Cell and Regenerative Medicine.
Kelly Ormond on setting rules for germline editing

A team of genetics experts has issued a policy statement recommending that research on editing human genes in eggs, sperm, and early embryos continue, provided the work does not result in a human pregnancy.

Kelly Ormond, MD, professor of genetics at the Stanford School of Medicine, is one of the signatories of the statement, which provides a framework for regulating germline gene editing of human germ cells. Germ cells, a tiny subset of all the cells in the body, give rise to eggs and sperm. Edits to the genes of germ cells are passed on to offspring.

The American Society of Human Genetics was jointly prepared by the American Society for Human Genetics and four other human genetic organizations, including the National Society of Genetic Counselors, and endorsed by another six, including societies in the United Kingdom, Canada, Australia, Africa, and Asia.

A single gene editing raises a host of ethical and technical questions, that for now, remain largely unanswered. The ASHG policy statement proposes that federal funding for germline genome editing research not be prohibited; that germline editing not be prohibited for research that would develop inside a woman and that future clinical germline genome editing in humans not proceed without a compelling medical rationale, evidence supporting clinical use, ethical justification, and a process incorporating input from all stakeholders, patients, and their families, and other social and scientific principles.

Ormond recently discussed the issues that prompted the statement's creation with writer Jenifer DuShane.

1 Why did you think it was important to issue a statement?

ORMOND: Much of the interest arose a couple of years ago when a group of researchers in China did a proof-of-principle study demonstrating that they could edit the genome of human embryos.

The embryos weren't viable [meaning they could not lead to a baby], but I think that paper worried people. Gene editing in human germ cells is not technically easy, and it's not likely to be a top choice for correcting genetic mutations. Still, it worried us that something was starting to do it.

We've been able to alter genes for many years now, but the new techniques, such as CRISPR/Cas9, that have come out in the past couple of years provide a framework for regulating germline gene editing of human germ cells. Germ cells, a tiny subset of all the cells in the body, give rise to eggs and sperm. Edits to the genes of germ cells are passed on to offspring.

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2 Are there regulations now in place that prevent re-

searchers from editing human embryos that could result in pregnancy and birth?

ORMOND: Regulations vary from country to country, so research that is illegal in one country could be legal in another. That's part of the challenge and why we thought it was important to have multiple countries involved in this statement.

Also, since 1995 the United States has had regulations against federal funding for research that creates or manipulates embryos by fertilization or any other means that restricts federal funding on things like germline editing will drive the research underground so there's less regulation and less transparency. We felt it was really important to say that we support federal funding for this kind of research.

3 Is germline editing in humans useful and valuable?

ORMOND: Germline editing doesn't have many immediate uses. A lot of people argue that if you're trying to prevent genetic disease (as opposed to treating it), there are many other ways to do that. We have options like prenatal testing or IVF and pre-implantation genetic testing and then selecting only those embryos that aren't affected. For the vast majority of situations, those are feasible options for parents concerned about a genetic disease.

There are a number of situations where you couldn't use pre-implantation genetic diagnosis to avoid having an affected child so for example, if a parent was what we call a homozygote for a dominant condition such as BRCA1 or Huntington's disease, or if both members of the couple were affected in the same recessive condition like cystic fibrosis or sickle cell anemia, it wouldn't be possible to have a biologically related child that didn't carry that gene, not unless germline editing was used.

4 What makes germline editing controversial?

ORMOND: There are families out there who see germline editing as a solution to some genetic conditions. For example, during a National Academy of Sciences meeting in December of 2015, one parent stood up and said, "I have a child who has a genetic condition. Please let this move forward; this is something that could help." But I also work in disability studies, so it relates to genetic testing, and there are many individuals who feel strongly that genetic testing or changing genes in any way makes a negative statement about them and their worth. So this topic really edges into concerns about eugenics and about the way making genes can happen once we have the ability to change our own.

Germline gene editing impacts not just the individual whose genes are edited, but their future offspring and future generations. We need to listen to all of those voices and try to set a path that takes all of them into account.

That's a huge debate right now. A lot of people say, "Let's not mess around with the germline. Let's only edit genes after a person is born with a medical condition." We're seeing an exciting medical condition is different from changing someone's genes from the start, in the germline, when you don't know what else you're going to influence.

5 There was a paper recently about gene editing that caused mutations in exome numbers of targeted genes, so called "off-target effects." Did that re-

result surprise you or change anything about what you think?

ORMOND: No. And, again, I think part of the problem is that this research is moving very fast. One of our biggest challenges was that you can't do a good ethical assessment of the risks and benefits of a treatment or technology if you don't know what the risks are.

We keep learning about potential risks, includ-

ing off-target mutations and other unintended conse-

quences. Before anyone ever tries to do germline gene editing in humans, it is very important that we do animal studies where the animals are followed through multiple generations, so that we can see what happens in the long term. There's just a lot that we don't know.

There are so many unknowns that we don't even know what guidelines to set. For example, what's an appropriate new mutation level in some of these tech-

nologies? What is the risk we're willing to take as we move forward into clinical trials, both in somatic cells [cells of the body, such as skin cells, neurons, blood cells] and in germ cells.

It's really hard because, of course, we're talking about, for the most part, bad diseases that significantly impact quality of life. So if you're talking about a really serious disease, why are you willing to take more risk there, and these new mutations aren't likely to be as bad as the genetic condition you already have. But we don't know, right?

We haven't had any public dialogue about any of this, and that's what we need to have. We need to find a way to use the public and scientists about all of these issues so people can have informed discussions and really come together as this moves forward, so that we're not in that reactive place when it potentially becomes a real choice.

And that goes back to your first question, which is why did we feel like we needed to have a statement now? We wanted to get these conversations going.
Reversing signaling imbalance in brains of mice eases symptoms of autism

By Bruce Goldman

A study by Stanford investigators sug-
gests that key features of autism reflect
an imbalance in signaling from excita-
tory and inhibitory neurons in a por-
tion of the forebrain, and that reverting
the imbalance could alleviate some of
its hallmark symptoms.

In a series of experiments conducted
on a mouse model of the disorder, the
scientists showed that reducing the ra-
tio of excitatory to inhibitory signaling
counteracted hyperactivity and deficits in
social ability, two classic symptoms of
autism in humans.

The study was published Aug. 2 in
Science Translational Medicine. Karl De-
isseroth, professor of bioengineering and
of psychiatry and behavioral sciences, is
the study’s senior author. The lead au-
thor is former graduate student Ashish Selimbeyoglu, PhD.

In 2011, Deisseroth’s group pub-
lished a study in Nature showing that
autismlike behavioral deficits could be
induced in ordinary mice by elevating
the ratio of excitatory to inhibitory neu-
ronal firing patterns in the mice’s medial
prefrontal cortex.

The new study shows that decreasing
that ratio restores normal behavior
patterns in a strain of lab mice bio-
geneered to mimic human autism. These
mice carry a mutation equivalent to
a corresponding mutation in humans
that is associated with autism spectrum
disorder.

Autism incidence increasing

For reasons that are not understood,
the incidence of autism spectrum disor-
der has increased steadily in recent years,
said Deisseroth, a practicing psychiatrist.

Around 1 in 80 American children may
be diagnosed with the disorder, which
is characterized by repetitive behaviors
difficulty with social interaction. To
date, there are no medications that treat
the fundamental underpinnings of the
disorder.

“In all of psychiatry, there’s no lab test
that can diagnose this condition,” said
Deisseroth. “It’s been associated with nu-
meros new genetic variants, many of which
appear to exert only small individual
influences.”

Deisseroth, who holds the D.H. Chen
Professorship, notes that UCSF psychia-
trist John Rubenstein and his colleagues,
has found that raising the excitatory-to-
inhibitory ratio in the brain may reduce
symptoms of a form of autism.

As for the Rothschilds, they say their goal is to con-
tribute to knowledge in the field.

“We just hope to be able to affect things in a posi-
tive way — have some impact,” Jeffrey Rothschild said.

“That’s all you ever can hope for,” he
said.
Scar painter Ted Meyer to lecture and lead workshops at Stanford

By Rosanne Spector

Ted Meyer's career as an artist was succeeding beyond his dreams. Though he was often in great physical pain — a result of Gaucher disease he had faced since childhood — his paintings were critically acclaimed, shown in galleries around the world.

Then, in his mid-30s, he lost his music. He began a newly available treatment for Gaucher disease, and his symptoms vanished.

"All of a sudden, everything that had been the motivation for my art work disappeared. I wasn't in pain or fatigued. I wasn't worried about dying young," said Meyer, whose paintings had been expressions of his anguish. He felt great, he said, but as an artist he was lost — until a conversation at an art opening with a woman in a wheelchair wearing a backless dress that showed a long scar running down her spine. Though partly paralyzed, she was nonetheless an actress and dancer.

Their meeting set him on a new course: creating art that tells the stories of survivors of health crises.

"Scarred for Life"

Meyer, this year's Sterling Visitor Professor in the Department of Chemical & Systems Biology, will be at Stanford Aug. 14-17 to speak and lead workshops that continue telling those stories. His aim, he said, is to round out the medical profession's view of patients' lives.

Attendees will learn about his project "Scarred for Life: Mono-prints of Human Scars." The project began when Meyer printed the dancer's scar — a result of surgeries to repair damage from the zip line accident that had paralyzed her. He added details to the print with paint and color pencil and paired the result with a photograph of the dancer showing the scar slathered with paint on her back.

Now 59, Meyer still paints. He also teaches medical students. As the artist in residence at the Keck School of Medicine of USC, he brings artists with chronic illnesses to the medical school to exhibit their work and meet with students.

"My hope is that over the month each show is up, it is not only beautiful or compelling but it tells the story of what the life is like for an artist," said Meyer.

"You can see a lot of things besides, 'I was in pain for five years.' If the med students see it over and over, I hope it will remind them that they're really dealing with patients and not a pile of symptoms."

The talks and workshops at Stanford, listed below, are open to the public:

• Talk — A Patient Life, 4-6 p.m. Aug. 14 in Munzer Auditorium at the Beckman Center.

• Workshop — Scarred for Life, 10 a.m. Aug. 15 in rooms 4105-4107 of the Center for Clinical Sciences Research. The event will feature a roundtable discussion on the representation of scars and the meaning and stories behind them, as well as the creation of prints of workshop participants' scars.

• Workshop — The Collective Experience: Charting Illnesses that Have Touched You, Your Family and Friends, 10 a.m. Aug. 16 rooms 4105-4107 of the Center for Clinical Sciences Research.

• Talk and presentation — Art and Medicine, 4-6 p.m. Aug. 17 in Munzer Auditorium.

Deisseroth continued from page 4

Deisseroth is a member of the Stanford Neurosciences Institute and of Stanford Bio-X, an interdisciplinary consortium of physical and medical scientists and engineers.

Other Stanford study co-authors are postdoctoral scholars Christina Kim, PhD, and Matsunori Inoue, PhD; former postdoctoral scholars Soo Yeun Lee, PhD, and Thomas Davidson, PhD; laboratory technician Alice Hong; graduate student Juau Kauvar; laboratory manager Charu Ramakrishnan; former graduate student Lief Fennis, PhD; and psychiatry instructor Matthew Wright, MD, PhD.

The study was funded by the National Institute of Mental Health, the National Institute on Drug Abuse, the U.S. Defense Advanced Research Projects Agency, the Simmons Foundation, the Wiegert Foundation, the Gary Schmidt Foundation, the National Institute of Mental Health, and the National Institute on Drug Abuse.

The Department of Bioengineering and of Psychiatry and Behavioral Sciences also supported the work.

The Department of Bioengineering is jointly operated by the School of Medicine and the School of Engineering.
Shah continued from page 1

factors and how much was genetically hand-wired. Their findings suggest that social forces can override genetically programmed behavior. The findings also could potentially help explain the ill effects of solitary confinement on prison-ers, as well as what underlies psychiatric disorders characterized by bursts of vio-lent anger.

Male mice are naturally territorial. In the wild or in the lab, they attack other male mice even if plenty of room, food and females are available. A typical male mouse who’s been dug in for a while as the sole occupant of a chunk of turf will normally attack any other male placed into that territory, said Nirao Shah, MD, PhD, professor of psy-chiatry and behavioral sciences and of neurobiology.

But selectively activating just this tiny cluster — about 5,000 nerve cells in a brain with 80 million nerve cells — escalates the level and extent of male mice’s aggression enormously, said Shah, the study’s senior author. The lead author is postdoctoral scholar Taehong Yang, PhD.

The cells, designated as PR+ VMHvl nerve cells, are found in a part of a brain structure called the ventromedial hypo-thalamus and are distinguished by the fact that they contain receptors for sex hormones.

Turning mouse Jekyll into mouse Hyde

Stimulating PR+ VMHvl cells in a male lab mouse who’s spent a week to 10 days in a cage of its own triggers force displays of territorial aggression even when such behavior would rarely occur, the scientists found. This mouse not only will attack a female, which male mice normally never do, or a member of another species, which mice rarely do, but will threaten a mirror placed in its cage or even an inflated surgical glove. Even if neutered, the resident male will launch an attack on another mouse introduced to its cage. (Circulating tes-tosterone is normally indispensable for displays of male territorial aggression.) In effect, stimulating these cells makes the solitary male lash out violently and indiscriminately.

In an earlier study, Shah and his col-leagues showed that selectively killing PR+ VMHvl nerve cells radically reduces territorial aggression in male mice. This cluster therefore appears to be essential to a male mouse’s display of territorial aggressiveness. The new study indicates that the circuitry’s activation is also suf-ficient to trigger territorial aggression in a broad range of circumstances. Nevertheless, this genetically hand-wired behavior seems to be subject to complex social etiquette. For one thing, the male showed, a solitary-resident male mouse wouldn’t start exhibiting displays of aggression if it was all alone, regardless of how angered up its PR+ VMHvl circuitry was. But the sight of an unfamiliar object — even the nonef-fec-tive threat of a mirror — was enough to trigger tail-twitching threats. Such a mouse would also attack when in-serted into the turf of another aggressive, solitary male. On the other hand, a male mouse that had been housed with other males would not attack the aggressive solitary male on the latter’s home turf even when the researchers revved up PR+ VMHvl activity in the socially housed male. Something about social housing. Shah said, seems to powerfully temper male mice’s aggressiveness — so much so that even directly activating the intru-sors’ “rage center” wasn’t enough to coax it into attacking.

That something, said Shah, is likely to be related to mice’s acute ability to sense pheromones, which are chemical signals released by members of a species to signal their social and reproductive status to other members of the species. You can tell male and female mice apart by the way they smell,” said Shah, an experienced rodent researcher. Unlike female mice, male mice “hormone reactive.” Ponder the scent of moldy socks and dirty T-shirts in a teenage boy’s gym locker.

Previous work showed that a solitary male hanging out on his home turf attacks a male intruder because of the intruder’s pheromones, the study team showed that pheromones exuding from such a solitary male on his home turf deter aggression from a socially housed male intruder. Strikingly, if the socially housed male could no longer sense the solitary male resident’s pheromones, he would attack the resident once his PR+ VMHvl cells were reactivated.

‘Nature versus nurture is a false dichotomy’

‘Nature versus nurture is a false dichotomy,’ said Shah, on the one hand, that genetically pro-grammed circuitry massively influences behavior. ‘But then, on the other hand, that, under certain circumstances, nar-row wins: Your social conditions can override your natural impulse to fight.’

Human beings, like the mouse, also features a ventromedial hypo-thalamus. While this brain region’s not been directly studied in humans, Shah said, case studies suggest at least a degree of similarity to those of a rat’s VMHvl.

The study’s findings, therefore, may bear on the question of whether solitary confinement affects male human behaviors. Further research is needed to test this hypothesis.

The findings hold implications for the design of future studies of disease, including, for example, studies of interpersonal violence and of inter-personal violence and of interpersonal violence and of interpersonal violence and of interpersonal violence and of interpersonal violence and of interpersonal violence and of interpersonal violence and of interpersonal violence and of interpersonal violence.

Study funders

The study was funded by the National Institutes of Health, the Stanford ME/CFS Initiative Fund and an anonymous donor.

Nirao Shah says the new findings show that both genetics and environment can affect behavior in mice. ‘Nature versus nurture is a false dichotomy,’ he adds.

is real. Antivirals, anti-inflammatory drugs and modulating drugs have led to symptomatic improvement in some cases, Montoya said. But no single pathogenic agent that can be fingered as the ultimate ME/CFS trigger has yet been isolated, while previous efforts to identify immunological abnormalities behind the disease have met with con-flicting and confusing results. Still, the sporadic effectiveness of antiviral and anti-inflammatory drugs spurred Montoya to undertake a systematic study to see if the inflammation that’s been a will-o’-the-wisp in those previous searches could be definitively pinned down.

To attack this problem, he called on Davis, who helped create the Human Immunodeficiency Virus (HIV) in the 1970s to bring his team. Since its inception a decade ago, the center has served as an engine for large-scale, data-intensive immunological analysis of ME/CFS cases.

The investigators analyzed blood samples from 192 of Montoya’s patients, as well as from 392 healthy control subjects. The median age of the patients and controls was about 50. Patients’ average duration of symptoms was somewhat more than 10 years.

Importantly, the study design took into account patients’ disease severity and duration. The scientists found that some cytokine levels were lower in pa-tients with mild forms of ME/CFS than in the control subjects, but elevated in ME/CFS patients with relatively severe manifestations. Averaging the results for patients versus controls with respect to those cytokines would have obscured this phenomenon, which Montoya said he thinks may reflect different genetic pre-dispositions, among patients, to progress to mild versus severe disease.

When comparing patients versus con-trol subjects, the researchers found that only two of the 51 cytokines they mea-sured were different. Tumor growth fac-tor beta was higher and resinifer was lower in ME/CFS patients compared to control subjects. The scientists found that the concentrations of 17 of the cytokines tracked disease se-verity. Thirteen of those 17 cytokines are pro-inflammatory. TGF-beta is often thought of as an anti-inflammatory rather than a pro-inflammation cytokine. But it’s known that’s been a will-o’-the-wisp in those searches could be definitively pinned down.

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Small reductions in childhood mea-
sles vaccinations in the United States would produce disproportionately large increases in outbreaks and in related public health costs, ac-
cording to a new study by researchers at the School of Medicine and at Baylor College of Medicine.

A 5 percent drop in the number of children ages 2 to 11 inoculated against the measles virus could triple the number of annual measles cases in this age group, the study found. The MMR vaccine is an inoculation against the three diseases.

The additional measles cases would incur annual public health expenditures by at least $2.1 million, or $20,000 per case of measles.

The study was published July 24 in JAMA Pediatrics.

“We focused on measles as an example of the effects of declining vaccine coverage because it is highly infectious,” said Nathan Lo, the study’s lead author and a Stanford MD-PhD student. “It’s likely to be the most common infectious disease for which we will see change, given the trend toward school-based vaccinations and age stratification for many vaccine doses.”

Lo said he hopes the findings will be considered by policymakers and others making decisions about vaccination policy.

“I think our study is a wake-up call for what we can expect in the coming months,” said the study’s senior author, Peter Hotez, MD, PhD, dean of the National School of Tropical Medi-
cine at Baylor.

The article’s title, “Small reductions in childhood DPT (diphtheria, pertussis, and tetanus) vaccinations in 2015 following a large measles outbreak are likely to increase cases of measles,” referred to a recent study in the journal JAMA Pediatrics that found that the number of measles cases in the United States increased substantially after a large outbreak of the disease in 2015.

The JAMA Pediatrics study focused on the effects of declining vaccine coverage in the United States and found that a 5 percent reduction in vaccination coverage could triple the number of annual measles cases. The study’s authors warned that the findings suggest that measles outbreaks may be more common in the future if vaccination coverage continues to decline.

The study also found that the effects of declining vaccine coverage are likely to be more pronounced in certain geographic areas, such as those with low vaccination coverage, where the disease is more likely to spread rapidly.

The authors of the JAMA Pediatrics study noted that their findings highlight the importance of maintaining high vaccination coverage to prevent measles outbreaks.

The study also noted that the effects of declining vaccine coverage are likely to be more pronounced in certain geographic areas, such as those with low vaccination coverage, where the disease is more likely to spread rapidly.

The study also found that the effects of declining vaccine coverage are likely to be more pronounced in certain geographic areas, such as those with low vaccination coverage, where the disease is more likely to spread rapidly.
Jennifer Cochran, PhD, has been appointed chair of Stanford’s Department of Bioengineering, which is jointly operated by the School of Medicine and School of Engineering. Her five-year term begins Sept. 1.

“This department has an amazing energy due to no small part to its faculty, students and staff,” said Cochran, associate professor of bioengineering. “These individuals — nearly 500 of them, in all — have an unwavering commitment to research, learning and service, and they create a spirit of collegiality and collaboration that permeates our department and the broader Stanford community.”

Cochran’s research is interdisciplinary, integrating chemistry, engineering and biophysics. Her laboratory focuses on protein-based drug discovery for applications including oncology and regenerative medicine, and the development of new technology for high-throughput protein analysis and engineering.

‘A superb scholar and educator’

“In addition to being a superb scholar and educator and a proponent of deeper connections with Silicon Valley’s burgeoning biotechnology activities, Jenni- Jennifer Cochran, PhD, has been appointed chair of Stanford’s Department of Bioengineering, which is jointly operated by the School of Medicine and School of Engineering. Her five-year term begins Sept. 1. nder full professor of neurology and neurobiology, of developmental biology and of neurology and neurological sci- ences, has received the Inge Grundke-Iqbal Award for Alzheimer’s Research from the Alzheimer’s Association. The honor recognizes the senior author of the most impactful study published in the journal. His work advanced the understand- ing of the role played by the apolipoprotein E gene in controlling the rate of synapse pruning by astrocytes in the brain.

“His research examines the mechanisms of human neurodegenerative diseases including Parkinson’s disease and amylo- lateral spongiosis. Victoria Huns, PhD, postdoctoral scholar in developmental biology and in genetics, and Shaogeng (Steven) Tang, PhD, postdoctoral scholar in biochemistry, have been named Damon Runyon Fellows by the Damon Runyon Cancer Research Foundation. The fellowship provides $231,000 over four years to support basic and translational cancer research. Hung is examining how phos- pholipases of ribosomal components leads to the specialization of ribosomes. Tang is developing small-molecule inhib- itors drugs that target human immu- nopathogenetic pathways. Tang was also selected as a Merck Fellow by the foundation.

JIN MYONG LEE, PhD, was promoted to associate professor of neurology and neuro- logical sciences, of bioengineering and of anesthesiology, effective Feb. 1. His research focuses on analyzing and ma- nipulating brain circuits to develop new therapies for neurological diseases. AMY KRYSOSIK, PhD, postdoc- toral scholar in infectious diseases, was awarded the Robert E. Shope Interna- tional Fellowship from the American So- ciety of Tropical Medicine and Hygiene. The fellowship provides $25,000 for a short-term arbovirology or infectious disease research experience in the tropics. Krystosik plans to conduct field tests of a miniaturized, automated, whole-blood cellular analysis system to test immunity to arboviruses in Kenya. WINNIE KWOKF, assistant director of facilities operations for the Office of Facilities Planning & Management, has received the 2017 Pacesetter Award from APPA, an educational facilities professional organization. The honor recognizes individuals who have made significant contributions to the educa- tional facilities management profession and industry. DAVID MYUNG, MD, PhD, was ap- pointed assistant professor of ophthal- mology, effective June 1. He is the co-director of the Stanford Ophthalmic Innovation Program, a one-year, project- based fellowship. His research interests include regenerative medicine and drug delivery, as well as global health through translational and emerging smartphone- based diagnostic platforms. KATHLEEN SAKAMOTO, MD, PhD, pro- fessor of pediatrics, has been awarded a $100,000 grant from the Bear Necessi- ties Pediatric Cancer Foundation to de- velop new small-molecule compounds to target CURE-dependent pathways for the treatment of relapsed acute leukemia. She holds the Sheilag Gilligan Profes- sorship in the School of Medicine. PETER SANTA MARIA, MBBS, PhD, was appointed assistant professor of otolaryngology-head and neck surgery, effective June 1. His clinical focus is on adult and pediatric surgery for hearing, balance and facial nerve disorders. His research interest includes tympanic membrane regen- eration, biofilm treatment, oral wound healing and medical device development. DEBANT SENGUPTA, MD, PhD, postdoctoral scholar in radiation oncology, was given the Alavi-Mandell Award from the So- ciety of Nuclear Medicine and Molecu- lar Imaging for being the lead author of “Single-cell characterization of 18F-FLT uptake with radioluminescence micros- copy,” a paper published in the Journal of Nuclear Medicine in July 2016. The award recognizes a trainee who is the lead author of an outstanding paper pub- lished in the journal. GEOFFREY TABIN, MD, was appointed professor of ophthalmology, effective June 1. He is a cornea and specialist whose work focuses on reducing global blindness and developing systems of care in Asia and Africa. He is the co-founder of the Himalayan Cataract Project. KATHRYN TAYLOR, PhD, postdoc- toral scholar in neuroscience and neuro- logical sciences, was awarded a Damon Runyon-Stanford Pediatric Cancer Fellow- ship Award. The award provides $231,000 over four years to support research with the potential to significantly impact the prevention, diagnosis or treatment of pediatric can- cer. Taylor is investigating the effect of neural activ- ity on pediatric high-grade glioma progression.”

The Department of Bioengineering in- cludes more than 30 tenure-track faculty, 70 postdoctoral scholars and 200 graduate students with a variety of backgrounds and interests.

“Now, more than ever, interdisciplinary training and awareness is critically needed for bioengi- neering to be broadly productive and impactful. Stan- ford excels in this arena,” Cochran said.

“One of my goals is to work with students, staff and faculty to build stronger connections with Silicon Valley and the world at large,” she added. “I’d also like to help enable the department to tackle new technology devel- opments and innovations in biotechnology, medical de- vices, mobile health and data and measurement science, and forge new connections in areas such as agriculture, environment, humanities, policy and the arts.”

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