Bacteria, virus form diabolical partnership

By Bruce Goldman

A common bacterial pathogen called Pseudomonas aeruginosa produces a virus that substantially increases the pathogen’s ability to infect us, according to a study by investigators at the School of Medicine.
P. aeruginosa weaponizes its resident virus to exploit the immune system’s distinct responses to bacterial versus viral infections. This marks the first time a bacteria-infecting virus, otherwise known as a bacteriophage or simply a phage, has been observed inducing the immune system to mount an antiviral response and, in doing so, causing it to ignore the bacterial infection. When the scientists generated a vaccine directed at the virus, they showed that it dramatically lowered the bacteria’s ability to infect wounds in mice.

Detailed in a study published March 29 in Science, the findings could fuel new ways of preventing chronic, intractable infections by keeping antibiotic-resistant bacteria from getting a foothold in the first place. The discovery that phages foster bacterial infections also adds a previously unexpected layer of complexity to the relationship between us and the billions of bacteria inhabiting our gut and other organs.

Paul Bollyky, MD, PhD, assistant professor of infectious diseases and of microbiology and immunology, is the study’s senior author. The lead author is former graduate student Johanna Sweere, PhD.

Quadrillions of phages in body

“We’ve long known that you’ve got up to 10 quadrillion phages in your body, but we just figured whatever they were doing was strictly between them and your commensal bacteria,” Bollyky said. “Now we know that phages can get inside your cells, too, and make you sick.”

There’s currently no approved vaccine targeting P. aeruginosa, an increasingly drug-resistant pathogen that infects the lungs of most adults with cystic fibrosis and accounts for a sizeable percentage of all infections of diabetic ulcers, bedsores and burn wounds.

In 2017, the World Health Association named P. aeruginosa one of the “critical priority” pathogens posing the greatest threat to human health.

“I see this every day in my clinical practice,” Bollyky said. “What starts off as a little cut can’t heal as a result of a persistent, drug-resistant bacterial infection. The toll in terms of sickness, death and dollars is enormous.” Infected diabetic foot ulcers are the single biggest cause of amputation, he said.

Study: Starting colorectal cancer screening at 45 would prevent deaths

But screening more older adults would have even greater benefits

By Amy Jeter Hansen

Starting routine colorectal cancer screening at age 45 rather than 50 would decrease U.S. cancer deaths by as much as 11,100 over five years, according to a new study led by researchers at the School of Medicine.
The move would also decrease the number of cancer cases nationwide by up to 29,400 over that time period. However, screening...

By blocking a protein’s activity with antibodies, School of Medicine investigators were able to improve cognitive behavior in aging mice.

A paper describing the finding was published online April 3 in Nature. Tony Wyss-Coray, PhD, professor of neurology and neurological sciences, is the senior author. The lead author is MD-PhD student John Fluvinegan.

Wyss-Coray has been working for several years on the question of what causes the brain to lose its acuity with advancing age. One focus of his research has been a class of brain cells called microglia, which serve both as the brain’s immune cells and its garbage crew. Among the many different things microglia do to keep the brain healthy is scarfing up bits of cellular debris and protein deposits that build up in the course of normal metabolic activity.

On average, the garbage-collecting performance of microglia diminishes in aging brains. Why this happens, and the extent to which the faulty garbage service is actually responsible for age-related cognitive losses, are unclear. But it’s a decent bet that one way or another, microglial malperformance plays a role in neurodegeneration, said Wyss-Coray, the D. H. Chen Professor II and a senior research career scientist at the Veterans Affairs Palo Alto Health Care System.

“Many of the genes whose high-risk variants have recently been linked to Alzheimer’s disease are known to be active in the brain only in microglia,” he said. “Microglial genes’ activation patterns are abnormal in Alzheimer’s patients, and in other neurodegenerative disorders including Parkinson’s disease and amyotrophic lateral sclerosis.”

“We think we may have discovered a way to get those cells back on track and make them work the way they used to when we were young,” Bollyky said. “Now we know that phages can get inside your cells, too, and make you sick.”

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Paul Bollyky is the senior author of a study that found a possible method for reducing harm caused by a dangerous bacteria called Pseudomonas aeruginosa.
By Helen Santoro

How well-connected a particular brain network is, and how successfully memories are formed, may determine which patients with post-traumatic stress disorder benefit from behavioral therapy, researchers at the School of Medicine have found.

The finding could indicate a biological subtype of PTSD whose clinical relevance only becomes obvious when patients undergo a treatment, the researchers said. Furthermore, by replicating data via physiological monitoring of the creatures’ brain cells. It is a relatively easy experiment to do. The scientists who thought it up won a Nobel Prize for it in 2014. (The work was done under the leadership of a pair of Norwegian researchers under whom Giocomo did a postdoctoral fellowship.)

“Though animals don’t typically walk around inside big black boxes in the hope of hoovering up Cheerios dust,” Giocomo said. “You could have a goal of deciding to design a situation that would stimulate that goal-directedness but would also be able to relate whatever we found to what had been studied for the past 15 years or so.”

That meant shifting experimental animals between two alternate environments, one encouraging random meandering and the other one fostering goal-driven behavior.

A new box

To test goal-driven behavior, Giocomo and her colleagues designed a big box that was exactly the same size and shape as the one in the traditional experimental setup. But, instead of a food or a drink, the box contained crushed Cheerios. During experiments in either box, animals could forage freely and eat any Cheerio bits they found. But there was an important difference. The second box had a big red, roughly 8-by-10-inch “reward zone” in a fixed location on its Cheerios-strewn floor. The test animals were permitted to forage freely in this second box just as they did in the first box, but if, in response to an auditory cue, they navigated to the reward zone, they’d get a guaranteed, good-sized crushed-cereal reward. This reward was available only intermittently, and only for a short period after the cue.

Picture a “free lunch” counter in a supermarket. The counter is only open some of the time — but when it is, a storewide advertisement braves the news to hungry shoppers over the public address system.

The investigators implanted electrodes in several hundred nerve cells in the medial entorhinal cortex of rats that were placed in each of the two environments. The electrodes were connected to long cables, so the researchers could monitor individual nerve cells’ electrical activity as the animals roved freely inside whichever box they were put in.

“Rats’ spatial mapping system is the same as ours,” Giocomo said. “For rodents, they’re pretty smart. They like to move around. And they love Cheerios.”

Giocomo’s team collected and analyzed massive amounts of data, which enabled them to identify individual cells in each rat’s medial entorhinal cortex that served as compasses, speedometers and position detectors. They also observed that once a rat had learned enough about how the two environments differed — mainly, that one featured an occasional, well-advertised “free lunch” — several types of spatial-map-related cells in its medial entorhinal cortex changed their firing patterns whenever the animal got within a foot of a “free-lunch” counter.

For example, if a rat learned to go within about a foot of the center of the reward zone, whether or not the free-lunch counter was open, its position-signifying cells fired faster, and the position-signifying cells that were firing were spaced closer together, indicating higher spatial resolution.

“This tells us the rat’s brains are making a new map of space, in response to their experience of a reward, that reflects the importance of the place where they got it by providing a more accurate representation of its position,” Giocomo said.

Rats sensing a drug of abuse, she said, the improved accuracy at the center of this reward-based map could enable an addict’s habit.

For the study, researchers inserted electrodes through some nondescript side street in a strange neighbor- hood in search of a parking spot, remind yourself to gobble a chunk of chocolate as you’re getting out of your car. It might make it easier for you to remember where you parked.

Lisa Giocomo, PhD, assistant professor of neurobi- ology, is the study’s senior author. Lead authorship is shared by postdoctoral scholar William Butler, PhD, and graduate student Kiah Hardcastle.

Brain area crucial to navigation

“Every time you check your Google map for a par- ticular address or restaurant name or pair of grid coord- inates, you get the same map regardless of why you’re looking at it,” Giocomo said. “The GPS that generates spatial coordinates, or boundary and landmark detectors.

The finding could indicate a biological subtype of PTSD whose clinical relevance only becomes obvious when patients undergo a treatment, the researchers said. Furthermore, by replicating data via physiological monitoring of the creatures’ brain cells. It is a relatively easy experiment to do. The scientists who thought it up won a Nobel Prize for it in 2014. (The work was done under the leadership of a pair of Norwegian researchers under whom Giocomo did a postdoctoral fellowship.)

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Stanford acquires archive of palliative care pioneer Elisabeth Kübler-Ross

Elisabeth Kübler-Ross (1926-2004) was a Swiss psychiatrist who is best known for her work on the five stages of dying, which she detailed in her 1969 book, ‘On Death and Dying.’ Her work has had a profound impact on the way that people think about death and dying, and has been influential in the development of hospice and palliative care. Kübler-Ross was also a prolific writer, publishing multiple books and educational videos on the topic of death and dying. In 2019, Stanford acquired an archive of Kübler-Ross’s papers, which includes letters, manuscripts, audio and video recordings, and photographs. The archive is housed in Stanford Libraries and is available for research and study.

PTSD continued from page 2

In a second study, the researchers considered 245 combat veterans, 125 of whom were trauma-exposed but healthy and 128 of whom had been diagnosed with PTSD. They underwent behavioral and clinical assessments, including the verbal memory test and an fMRI scan of the brain.

In both studies, the findings revealed that a subgroup of participants with PTSD had both reduced verbal memory and VAN functioning compared with the rest of the PTSD participants and healthy control subjects. The researchers observed that participants with PTSD did respond to therapy, whereas the other participants with PTSD did not respond to therapy.

The finding emphasizes the importance of considering patient symptomology in developing a treatment for their PTSD, O’Hara said. “Our study shows that this specific combination of a deficit in VAN communication and poor memory leads to a failure to respond to exposure therapy, above and beyond their specific symptoms,” she said.

A deeper look into the VAN

In another study reported in the same paper, the veterans also underwent a direct brain activity test that used simultaneous transcranial magnetic stimulation and electroencephalography, a test that measures brain wave activity.

TMS is used to noninvasively manipulate activity in a targeted brain region and the networks to which it’s connected. Unlike fMRI, which shows a correlation between a cognitive task and brain activity, TMS allows scientists to transiently disrupt information processing in a particular brain region through magnetic stimulation, which behavior or brain function is affected. To detect these effects, participants were also hooked up to an EEG machine, which picks up the communication between neurons using sensors that are placed on a participant’s scalp.

The TMS and EEG tests revealed that the brains of veterans with poor connectivity in their ventral attention network took twice as long to recover from the TMS magnetic pulse than the brains of patients with good communication in their VAN, indicating a deficit in information flow within this brain network.

Toward a more personalized treatment

Etkin hopes to use this information as a means to develop more specialized mental health care. Down the road, clinicians could potentially perform repetitive TMS — a therapeutic tool that is currently used to help treat depression — to stimulate the VAN and ease PTSD symptoms.

A consistent challenge in creating personalized treatments for psychiatric conditions is that the Diagnostic and Statistical Manual of Mental Disorders, which classifies all mental disorders, is constantly evolving. But if clinicians can one day use biological measures instead, they will no longer have to deal with these varying definitions of disease, Etkin said.

Etkin and his team aim to translate their findings into treatments for PTSD. Brain-imaging studies of psychiatric disorders often stop short of that translation, he said. “To me, that’s not satisfying,” he said. “If you were to take these findings, how would you set up a study that would allow you to get to an FDA-approved biomarker quickly — not in 20 years, but in five years? That’s what we are now focused on.”

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 and treat disease in the ill.

O’Hara and Etkin are members of the Wu Tsai Neuroscience Institute at Stanford and investigators at the Stanford Mental Illness Research, Education and Clinical Center of the Sierra Pacific at the Veterans Affairs Palo Alto Health Care System. O’Hara is also a member of the Stanford Maternal & Child Health Research Institute.

Other Stanford co-authors of the study are postdoctoral scholars Adi Maron-Katz, PhD; Gregory Fonzio, PhD; Corey Keller, MD, PhD; and Julia Huenter, PhD; instructor Wei Wu, PhD; software engineer Brian Patenaude, PhD; research specialist Yevgeniya Zaiouk; research coordinators Kathy Peng, Emmanuel Shpigel, Parker Longwell, Raph Edelstein and Irene Akingbade; graduate student Russ Toll; associate professors of psychiatry and behavioral sciences Allison Thompson, PhD; and Steven Lindley, PhD; Sanno Zach, PhD; clinical associate professor of psychiatry and behavioral sciences; clinical psychologist Elizabeth Weiss, PsyD; program manager Jillian Aurea; and professors of psychiatry and behavioral sciences Bruce Arnold, PhD, and Joachim Hallmayer, MD.

Researchers from the VA-Palo Alto, Sierra Pacific Mental Illness, Research, Education and Clinical Center; South China University of Technology; New York University Langone School of Medicine; University of Cambridge; Cambridgeshire and Peterborough NHS Foundation Trust; Lausanne University Hospital; Seattle Healthcap; New Mexico Veterans Affairs Health Care System; University of New Mexico; University of Pennsylvania; Pontificia Universidad Católica de Chile; and Emory University School of Medicine also contributed to this work.
Researcher investigates why some pain becomes agonizing and chronic

By Nicolleta Lanese

Stacey Morris remembers being roused in the early morning one summer day in 2008. “I took too many pills,” she told the hospital staff. “I don’t know what I took.”

Morris, whose name is changed for this article to protect her privacy, was kept overnight on a psychiatric ward because the doctors thought she may have attempted suicide. But that wasn’t the case, she insists. She said she had accidentally eaten a small glass of wine. She was nearly a statistic, illustrating the vast majority of pain research has been done in mice. “You can think of them as turning up the volume on the pain pathway in mice that are bred to lack an essential microglial protein. She also is experimenting with disabling this component with an injectable drug engineeri

Compounding the problem, the vast majority of pain research has been done in mice because of the same genetic strain, even though chronic pain affects far more women than men.

Tawfik has tried to deal with these concerns by building an animal study that closely replicates what she sees in her patients, most of whom are women. Many of them first fracture a bone; then, after the cast comes off, the fracture pain lingers and develops into complex regional pain syndrome. Tawfik reproduces this scenario in her mouse experiments to study symptoms she sees in humans.

Making do with mice

Pain is a complex interaction of physical, emotional and psychological factors, and scientists studying mice have yet to figure out how to ask a mouse how it feels emotionally. Researchers can only study painlike behavior and nociception — how the nervous system reacts to painful stimuli — in animals. For instance, a researcher may prod a mouse’s injured paw and note whether it pulls away and how quickly.

Now, Tawfik hopes to understand how these cells contribute to chronic pain and how to stop it.

Microglia

In her Stanford lab, Tawfik is using genetic engineering technology to disable different genes along the pain pathway in mice that are bred to lack an essential microglial protein. She also is experimenting with disabling this component with an injectable drug to investigate how different levels of microglial activation correspond to the intensity of pain symptoms.

The classic symptom of complex regional pain syndrome is long-lasting pain that is stronger than expected given the injury that triggered it. Other symptoms include muscle tremors and weakness, bristle hairs, slow-growing hair, swelling, redness and unexplained coldness in the affected limb. Those with the syndrome may become hypersensitive: A minor cut or scrape can cause severe pain. More commonly, pain becomes chronic. But no one knows exactly how. By studying glial cells found only in the brain and spinal cord, called microglia, Tawfik hopes to understand how these cells contribute to chronic pain and how to stop it.

Turning up the volume on pain

But as technologies were developed to better study glia, scientists found evidence that the cells fire — often friendly fire — as scientists increasingly argue that pain experiments in mice have little relevance in human disease. While pain signals move through mice similarly, it’s not possible to re-create the suite of emotional, psychological and physical aspects of human pain in a rodent.

Still, animal studies are irreplaceable pieces in the pain research jigsaw, Tawfik said. For patients like Morris, these studies offer some hope for relief. In the 10 years since Morris’ shoulder surgery, her pain has lingered for longer than three months. Morris’ pain syndrome is a relatively rare form of chronic pain, with about 55,000 newly diagnosed cases each year. The pain subsides for some and persists for years in others.

Tawfik aims to help her chronic pain patients with a variety of treats, including physical therapy, sessions in pain psychology, pain-relieving drugs and procedures such as nerve block injections. But many of Tawfik’s patients tick straight through that list and remain wracked with pain. They tote around packed pillboxes; swallow their empty promises of freedom from pain; and are left exhausted, foggy and constipated, rather than relieved. The stakes are even higher for women between the ages of 45 and 55, who have the highest risk of dying from a prescription painkiller overdose.

At the heart of the issue is a question that has plagued medicine for many years: Why does some pain dissipate after an injury is healed, while other pain hangs around long after the fact? If pain physicists knew that, they could prevent the onset of chronic pain, rather than trying to numb patients to stop it.

Tawfik, an assistant professor of anesthesiaology, perioperative and pain medicine at the School of Medicine, hopes to someday figure that out. In addition to caring for patients, she is studying the transition from normal, short-term pain to chronic pain, which is how it becomes chronic.

Studies offer hope for relief

Neuroscientists have known that cells called microglia amplify pain signals on their way to the brain. If this boost persists after the painful injury has healed, it may lead to chronic pain. If this is the case, Tawfik hopes she might be able to alter the activity of microglia, tone down the incoming pain signals and reduce the pain.

Tawfik’s challenge will be moving her research from mice to humans. Pain researchers have been under
Researchers outline the possible role of a deep brain structure in concussion.

By Taylor Kubota

Concussion researchers have long suggested that damage to the corpus callosum, a thick band of tissue that connects the brain’s two halves, could result in some common side effects of concussion, like dizziness or vision problems. The assumption is straightforward — that damage to the corpus callosum could affect coordination between the two halves — but difficult to prove. “Concussion is a big, vague term, and we need to start breaking it down,” said former graduate student Fidel Hernandez, PhD, a lead author of the paper. “One way we can do that is to study individual structures that would be likely to cause traditional concussion symptoms if they were injured.”

**Evaluation, three ways**

This research is built on data from mouth guards worn by football players. Each mouth guard records head movement and acceleration in six directions through an integrated accelerometer and gyroscope. Analyzing 115 impacts recorded by these mouth guards, the researchers found two associated with concussion diagnoses. By applying the mouth guard measurements to a simulation of the neck, head and brain, the researchers saw instances in which the corpus callosum was pulled around by a structure above it called the falx.

The falx sits like a mohawk hairstyle between the brain’s two halves and is stiffer than the rest of the brain, called the falx. “Concussion is a big, vague term, and we need to start breaking it down.”

Michael Zeineh, the study’s other co-senior author, and his lab assessed magnetic resonance imaging scans from two athletes who had been diagnosed with concussion. “Concussion is a big, vague term, and we need to start breaking it down.”

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**Microglia**

Continued from page 1

...and whose activity levels either increase or decrease substantially with age.

**Blocking genes’ functionality**

The investigators picked about 3,000 genes encoding proteins that they judged could be targeted by drugs to block microglia as on those of younger mice, concluding that microglial phagocytosis substantially changed with advancing age.

In a parallel experiment, the investigators determined which of those approximately 3,000 genes are poorly trained staff? Or is the street in bad shape? “An unanticipated garbage overflow? Lazy or poorly performing garbage-hauling company,” Wyss-Coray said. “We wanted to examine the books of the garbage-managers.”

“Now we had a tentative suspect, a gene that had not previously been implicated in microglial garbage removal,” Wyss-Coray said. So they zeroed in on this gene, called CD22, which is found in both mice and humans.

In a follow-on experiment, the CD22 protein turned up three times as often on the surface of older mice’s microglia, compared with younger mice — as if the gene were complacent. Older microglia produced far more copies of this gene — a proxy for upregulated production of the protein for which the gene is a blueprint — than younger ones did, and knocking out its function greatly improved microglial phagocytosis.

“With obesity being such a big concern, we need to develop interventions that will improve the health of these cells,” Wyss-Coray said.

Colorectal cancer continued from page 1

A greater number of older and high-risk adults would aververt nearly three times as many diagnoses and deaths at a lower cost, the study found. The researchers conclude that the potential effects of a 2018 change to the American Cancer Society (ACS) recommendations point to the work that...
Ralph Greco, MD, a pioneer in the movement to support medical school wellness for trainees and the former chief of the School of Medicine’s division of general surgery and director of its general surgery residency program, died March 31 at his home on the Stanford campus, surrounded by family. He was 76.

The cause of death was prostate cancer, said his wife, Rajasthan MD.

Sparted by the suicide of a former resident, Greco created the first program to promote well-being among surgical residents in the United States and pushed successfully for changes at the national level to require residency programs to support work-life balance for their trainees.

“He was fearless about taking on the surgical establishment,” said Claudia Mueller, MD, a Stanford pediatric surgeon who was associate director of the Balance in Life Program Greco established when it was launched in 2011. She now directs the program, “Ralph’s ideas about wellness for trainees were not popular among old-school surgeons. It’s a macho culture. But he was so committed to supporting trainees and so clever and farsighted about how to establish a program that he made it happen. It was stunning.”

Greco was a product of that same macho culture. “You had to be a certain way to be successful in surgery,” said Bryan Bohman, MD, associate dean for medical education at the Stanford School of Medicine. “His efforts have benefited not only physicians and their spouses, but also the leaders of other training programs, who have come to recognize the value of work-life balance for surgeons. Many have emulated the program he created here.”

“We get calls every month asking how we do it. They think we have something golden,” said Mueller, one of the original faculty members who have been invited to other academic medical centers to discuss how the program works and its impact.

**Bronx native**

Greco was born and raised in the Bronx, a borough of New York City. His mother was an elementary school teacher, and his father designed women’s clothing. He went to college at Fordham University, graduating cum laude, and then earned a medical degree at Yale in 1968.

Greco completed his internship and residency at Yale New Haven Hospital, where he was chief resident in 1972-73; served as a staff surgeon in the military from 1973-1975 — at the U.S. Army Hospital in Seoul, South Korea, and at Kimmrough Army Hospital in Fort Meade, Maryland; and joined the faculty of Rutgers Medical School (now the Robert Wood Johnson Medical School), where he specialized in surgical oncology and became chief of general surgery in 1982. He became chief of surgery at Robert Wood Johnson University Hospital in 1997.

While at Rutgers, Greco began research on the clinical use of biomaterials, which he continued at Stanford. Over his career, he co-authored more than 100 papers in the scientific literature. He met Wapnick, now a professor of surgery at Stanford, during her fellowship in breast surgery at Robert Wood Johnson University Hospital in 1997.

Ralph Greco with former Stanford surgical residents Yale Popowich (left) and Geoffrey Salles in Haiti in 2014. Greco established an elective rotation in Haiti for surgical residents.

In recognition of his accomplishments, the Accreditation Council for Graduate Medical Education bestowed on him in 2012 the organization’s highest honor, the John C. Gienapp Award, for his work as a surgery program director, humanitaria and innovator of a surgery curriculum focused on resident wellness and well-being for the medical student.
Phage continued from page 1

P. aeruginosa is itself frequently infected with a phage called Pf. This phage lives inside the bacterium but can be shed from the bacterial surface into the surrounding environment (such as a wound) like the virus herpes lives in our cells and is shed from cold sores. In the study, Bollyky’s team showed Pf was common in wounds infected with P. aeruginosa. The researchers cultured 111 patients with microbially infected, non-healing wounds and found that 37 of them were infected with P. aeruginosa. Two-thirds of those wounds infected with P. aeruginosa were carrying Pf — a fraction that grew the longer a wound persisted.

To prove Pf actually promotes P. aeruginosa infections rather than merely co-exists with them, the scientists inoculated small wounds in the skin of mice with P. aeruginosa strains that either did or didn’t contain Pf. They observed that the two strains differed greatly in their ability to establish wound infections. The inoculation dose necessary to result in a reliable P. aeruginosa infection was 50 times larger if it lacked Pf. But the scientists looked to see what Pf might be doing to immune cells that could affect P. aeruginosa’s ability to sustain an infection. In a lab dish, they found that the presence of the phage in P. aeruginosa reduced by 10-fold the number of invading bacteria that were engulfed by either mouse or human phagocytes — immune cells that ingest, then digest, invading bacteria. “The phagocytes lost their appetite,” Bollyky said.

Tripping molecular detectors

Bollyky’s team determined that stretches of the phage’s genomic material trigger molecular detectors in the phagocytes, steering the immune system’s response from an antibacterial to an anti-phage one. When a phage encounters bacteria, the appropriate response is to gobble them up, chew them out and call in more troops. But phage response to virus is different, Bollyky said. “If you’re an immune cell, ingesting a virus is absurd. So when you’re surrounded by something you can do, because now you’re let in side of you — you’re infected by it.”

Bollyky suspects the reason for a phage that comes in contact with a virus to shut down phagocytes. The appropriate immune response involves the generation of antibodies to vagrily infected cells and to signal other types of immune cells to home in on and destroy any virus-carrying cell they come across.

What Pf does inside phagocytes, Bollyky said, is somehow pulling up fire alarm when they should have called the police. “If 20 fire engines pull up to the scene of the crime, it makes it easier for the thief to get away,” he said.

The investigators generated a vaccine containing a component of Pf protein and noted that it cut the incidence of wounds infected with Pf-positive P. aeruginosa by half. They also generated antibodies that specifically target the same protein component and showed that they worked at least as well as the vaccine.

Bollyky and his colleagues have filed for a patent on intellectual property associated with the vaccine, and they plan to test it in large animals as a step toward eventual clinical trials.

Bollyky’s vision is to vaccinate people against Pf when they’re first diagnosed with cystic fibrosis or diabetes, as well as people in nursing homes and hospitals, in order to protect them from P. aeruginosa infections. Since a vaccine takes time to develop, it allows for a step in large animals as a step toward eventual clinical trials.

The findings could fuel new ways of preventing chronic, intractable infections.

For instance, vaccine development needs time. But if there are ways to trick the immune system into thinking a phage is a virus, it may be possible to stop the immune system from reacting to the phage in a way that is harmful to the patient. If this is possible, it could be a major breakthrough in treating infections caused by P. aeruginosa.

Gozo Durmus, PhD, was appointed assistant professor (research) of radiology, effective Feb. 1. He specializes in developing tools to detect and investigate circulating biomarkers and rare cells from biological fluids for precision medicine.

Aaron Gilter, PhD, professor of genetics, will receive the 2019 Sheila Essey Award from the American Academy of Neurology, the ALS Association and the American Brain Foundation. The $50,000 prize recognizes significant contributions in the search for the causes of amyotrophic lateral sclerosis, also known as Lou Gehrig’s disease, as well as ways to prevent and cure the disease. His research uses genetic screening to focus on mechanisms of neurodegenerative diseases, including Parkinson’s, Alzheimer’s and ALS.

Jaime Lopez, MD, was promoted to professor of neurology and neurological sciences, effective Feb. 1. He founded and directs the Stanford Intraoperative Neuropsychologist Monitoring Program. His research focuses on developing techniques for monitoring the nervous system during surgical and endovascular procedures, and identifying how these techniques alter surgical management and patient outcomes.

Inaugural gathering set for Stanford Medicine Abilities Coalition

The Stanford Medicine Abilities Coalition will hold its inaugural event, a mixer that will include members of the Medical Students with Disability and Chronic Illness group, at 5:30 p.m. April 15 in Room 308 of the Li Ka Shing Center for Learning and Knowledge.

The debut gathering, sponsored by the Office of Faculty Development and Diversity, is free and open all members of the Stanford community. It will feature short presentations and a social mixer. Light refreshments will be served.

The Stanford Medicine Abilities Coalition is an initiative started by Peter Pouillo, MD, clinical associate professor of radiology and of medicine. Its goals are to foster equal treatment; advocate for accessibility, resources and services; and promote diversity and inclusivity at the medical school and hospitals.