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www.jhsph.edu
Fall 2012

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Next issue
The end

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Inflammation
the body's friendly fire

Gravesite in Sri Lanka
Shehzad Noorani

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The Road to Hell

What would happen if every six days a 747 loaded with passengers crashed, killing everyone aboard?

Imagine the concern, the outrage, the media firestorm…. We would demand immediate solutions to prevent deaths and save lives. And yet, Americans remain strangely quiet about the 27,000 unintentional prescription drug overdose deaths each year in the U.S.—almost as many deaths as result from motor vehicle injuries. And this is just the tip of the iceberg. For every such death from prescription painkillers, 10 people are admitted for treatment of abuse and 130 people abuse or are dependent on such drugs.

It’s clear that we are in the midst of an epidemic of overdoses of prescription drugs—opioids, sedatives and stimulants. How did we get here?

Well, there’s plenty of blame to go around. Take opioids, for example. In the 1980s and 1990s, there was a growing realization by the medical community that many patients with pain were treated inadequately. Clinical protocols were revised to ensure that physicians assess every patient’s pain levels and treat pain aggressively. In response to the need to better control pain, pharmaceutical companies developed new formulations of narcotics that relieved pain, but unfortunately also had a high potential for abuse.

The result has been astonishing. From 1997 to 2007, U.S. sales of opioid drugs like Vicodin and Percocet increased by more than 600 percent. Concomitantly, prescription drug misuse and abuse increased, necessitating more than 475,000 emergency department visits in 2009 alone—almost twice as many as in 2004.

When I was a practicing physician, I had a healthy respect for the addictive power of narcotics as well as their ability to relieve pain. But you don’t have to be a medical provider to know that narcotic use can ensnare people. We all are familiar with news accounts of celebrities like Rush Limbaugh who become addicted to prescription drugs. Or stars like Heath Ledger who die from prescription drug overdoses.

This problem developed because people from many sectors were trying to do the right thing: help patients in pain. But, as my second-grade teacher used to say, “the road to hell is paved with good intentions.” In an attempt to relieve suffering, the threshold for use of narcotics was lowered. Problems presented by the more frequent prescription of opioids were exacerbated by unethical physicians who operated “pill mills,” patients who practiced “doctor shopping” (collecting prescriptions from different doctors), and our balkanized medical “system” that impedes checks and balances in prescription medication tracking.

As I said, there’s plenty of blame to go around; the question is how to solve the problem.

First we have to define it. The old metaphor of the blind men and the elephant comes to mind. Is it a regulatory problem? Yes. Is it a clinical problem? Yes. A patient education problem? Yes. A law enforcement problem? Yes. In fact, it’s all of these.

Because the problem is complex, solutions can have unintended consequences. In 2011, for example, a Maryland physician was disciplined for writing excessive numbers of medically unwarranted prescriptions for narcotics. An epidemic of withdrawal followed when his patients’ supply was cut off.

We need a systems approach to solutions that involve all the stakeholders. Public health has a long track record of attacking and solving problems like this. For example, injury researchers worked with policymakers, car manufacturers, law enforcement and others to make our roads dramatically safer. Annual traffic deaths in the U.S. fell from nearly 51,000 in 1966 to about 33,000 in 2010, even as our population increased by almost 60 percent.

We need the same kind of effective collaboration today to reduce prescription drug deaths. Patients and the public need to understand the risks, as well as the benefits, of taking these medications, how to safely store them, and methods of proper disposal when they are no longer needed. New drugs with less addiction potential must be developed. All states should have Prescription Drug Monitoring Programs that track prescription drug use. Even better, these state-based databases have to talk to one another. We also need to improve communications among clinicians, health agencies, pharmacists, drug manufacturers and law enforcement. Lastly, we need rational policies that promote judicious use of powerful prescription medications while limiting misuse that can lead to addiction and death.

Academic centers like the Bloomberg School can and should play the role of a trusted partner that brings groups together to find effective answers. By convening all stakeholders, we can devise better solutions.

To move this important agenda forward, I have asked our faculty from the Center for Injury Research and Policy as well as our soon-to-be-launched Center for Drug Safety and Effectiveness to bring together stakeholders for a symposium next spring to engage in a candid discussion about the problem and possible solutions.

It’s time we recognize that the prescription drug abuse problem is an epidemic. We must respond with the urgency and focus that such a public health crisis demands.
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(Photograph: Chris Hartlove, August 9, 2012)
Cover illustration by Michael Glenwood
Hyperlocal Hotspots

Each spring, Rio de Janeiro attracts well-heeled partygoers to its annual bacchanalia, Carnival. The city of 6 million has another, darker side however; one-fifth of its population lives in favelas, or slums—hotbeds for diseases that tend to cluster around the poor, such as tuberculosis and HIV.

Yet, until recently, public health researchers tended to treat Rio and most other large cities with extremely varied populations as single units with homogeneous inhabitants who had the same risk and incidence of disease.

Such thinking is slowly undergoing an evolution, says David Dowdy, MD, PhD, ScM, an assistant professor in Epidemiology. “There’s a growing awareness in tuberculosis and other diseases that we can’t just adopt one-size-fits-all strategies,” he says.

Consequently, Dowdy and other researchers are shifting tactics, focusing on far smaller areas to study diseases. By blocking off such disease “hotspots”—even to the neighborhood level—investigators are gathering fresh data that could eventually lead to novel ways to combat age-old diseases.

Smaller and Smaller

This hyperlocal focus isn’t totally new to public health, Dowdy explains. More than 150 years ago, John Snow famously traced the source of a cholera epidemic to a single London water pump by plotting cases in a neighborhood on a map. Since then, the hotspot approach has been a mainstay for a variety of infectious diseases, notably sexually transmitted diseases (STDs). To combat STDs, health workers focus on tried-and-true reservoirs—sex workers, for example, or people unlikely to take precautions for safe sex, such as teenagers.

“For STDs, parasitic infections and many other infectious diseases, there’s a widely known 80/20 rule,” says Dowdy. “Twenty percent of the population is responsible for 80 percent of transmission.”
In infectious disease outbreaks, those 80/20 diseases have provided natural targets for interventions. Henrik Salje, a doctoral candidate in Epidemiology, explains that it’s been significantly trickier, however, to focus intervention efforts in endemic settings because numerous overlapping transmission chains occur in the same areas and it is unclear who is responsible for the majority of infections. This has discouraged researchers from even bothering to look for disease hotspots or clusters—until now. Recently, he and other investigators have started examining disease transmission and prevention in smaller and smaller areas—tracking dengue transmission within neighborhoods, for example.

Why now? It’s all a matter of technology, explains Salje’s colleague Justin Lessler, PhD ’08, MHS ’08, MS, an assistant professor in Epidemiology. “There’s been this great increase in our ability to collect really fine-scale spatial data,” he says. “There is now wide availability of cheap and accurate GPS systems … most people have them on their phones by default.”

Combining GPS with better software to organize multiple types of data now allows researchers to easily “put a dot on a map,” and combine information in ways that they hadn’t been able to before, says Salje. “We can plot the location of a particular case and the time it happened, and even include genetic information of the culprit organism,” he adds. Genetic information is much more available because of increasingly sophisticated, cheaper technology.

**Surprisingly Focal**

Such work is exactly what Salje, Lessler, and Derek Cummings, PhD, MPH, MSc, assistant professor in Epidemiology and International Health, are doing with dengue. In a study published in the May 28 Proceedings of the National Academy of Sciences, they used geocoding to better understand how dengue, and immunity to this virus, spreads throughout individuals in Bangkok.

The researchers used data gathered over five years from a Bangkok children’s hospital. When patients with dengue-like symptoms came to the hospital for treatment, care providers there drew blood and sent it off for diagnoses—and, if it was positive for dengue, checked which of four viral serotypes caused the infection. Additionally, patients provided basic demographic information, including their addresses.

Once someone has been exposed to a single dengue serotype, they’re immune to that particular serotype for life, Salje explains. Preliminary but decades-old research suggests that they’re also immune to the other three for a stretch of several more months. But dengue is unusual in that if former patients are exposed to any of the other serotypes after this grace period, the resulting disease is much more severe.

To confirm this research and learn just how close cases cluster, the researchers used three basic pieces of information about the patient: the time they became sick, where they lived, and which serotype caused the infection. Using these data and geocoding technology to plot thousands of cases on maps over time, the researchers found that cases of the same serotype—suggesting that they might come from a single lineage of infection, passed from individual to individual—were occurring in areas smaller than a square kilometer. Dengue in these communities followed the same track as previous research suggested. Neighborhoods had a localized outbreak of a single serotype, followed by a period with few or no cases lasting many months, and then they were more likely to have severe disease caused by other serotypes.

Though Bangkok is full of commuters who could easily pass infections all over the city, the disease still clustered around homes, Lessler says. “It’s far more focal than we would have realized without these data,” he adds. The finding eventually could allow researchers better ways to implement prevention efforts or test whether vaccines in development are working.

**Better Targets**

Similarly, in Rio de Janeiro, Dowdy’s work on tuberculosis is showing that a small reservoir of individuals could be the key for slowing or stopping this disease’s spread throughout the entire city.

He and his colleagues used past surveillance data to narrow their focus to three areas—comprising about 6 percent of Rio’s population—that appeared to be hotspots for the disease, with TB rates at least double those of the rest of the city. Using additional data on how TB passes through populations from other cities, they constructed computer models to see how tuberculosis transmitted throughout the city, the disease still clustered around homes, Lessler says. “It’s far more focal than we would have realized without these data,” he adds. The finding eventually could allow researchers better ways to implement prevention efforts or test whether vaccines in development are working.

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Ideas Incubating in the Social Innovation Lab

Like many great inspirations, the Social Innovation Lab (SIL) emerged from a casual kitchen conversation. Jessica Ladd, MPH ’11, and her then roommate Mélodie Kinet, MPH ’11, MBA, were swapping stories about their nonprofit efforts.

In the quest for sustainability for her project combating STDs, Ladd discovered “there was not a lot of support as to how to proceed”—despite a growing number of students interested in creating nonprofits. Ladd’s project, Sexual Health Innovations, and Kinet’s Sustainable Roots initiative (see box) were radically different, but they had the same needs crucial to any nonprofit launch: everything from how to draft a mission statement to how to raise funds.

Ladd and Kinet’s initial discussion evolved into the peer-driven Social Innovation Lab, a student incubator whose primary purpose is to help “students already working on projects focused on social good to get to the next stage with their projects,” says Ladd, now a third-year PhD candidate in Epidemiology. “Social innovation comes from the fringes, from people coming together and exchanging ideas,” says Ladd. “We need [collaborators in] business, computer science, graphic design, medical fact checkers, pro bono legal services—[areas] that the School of Public Health didn’t have.” Next year, Ladd hopes to add local experts and faculty mentors who are also drawn from these specialties.

Health-related SIL projects range from the local to the international, from technology based to community based. “The best thing about SIL for me has been connecting with peers from different schools [who are] interested in pursuing the same kinds of endeavors—and connecting with those people to get from idea to research to real world setting,” says medical student Ralph Passarella, who’s launching a Web app called ReifyHealth.

SIL is “like a clearinghouse that connects people with like interests,” says Khadijah Mitchell, a medical student and the founder of Junior Biomedical Scholars. “It’s like the Jessica magic; she can connect people.”

Mitchell says that SIL introduced her to nonprofit leaders from around the city, helped her target financial support from medical organizations and taught her to formulate a pitch about her project and “to refine—not define—[my] mission.”

SIL also has the enthusiastic support of Marie Diener-West, PhD ’84, chair of the School’s Master of Public Health Program and the Abbey-Merrell Professor of Biostatistics Education. “What I think is so exciting about the Social Innovation Lab is that it is a student-run initiative for enabling social change,” says Diener-West. “It fosters cross-school partnerships,” she notes, and promotes the transfer of valuable skills and information that students won’t necessarily learn in the classroom.

Says Ladd, “It’s great from a learning perspective. If you have your own project, everything else you are learning [in school] becomes relevant. And when you can take the information you’ve learned that took days upon days to accumulate and can give it to someone else in five minutes … that’s really satisfying and really efficient.”

—Mary K. Zajac
Leading Liberian Health

The civil war burning through much of Liberia reached Tolbert Nyenswah and his family in the mid-1990s. It uprooted them from their home in the southern province of Sinoe and forced them to relocate to a refugee camp in Tabou, Ivory Coast.

“We walked for three months [to reach the camp], hiding in bushes and eating roots to survive,” he says. In the camp, he saw children die from diarrhea and disease.

The experience still echoes within, motivating him to become what he calls “a champion of global health with emphasis on community-based health services.”

Just before he graduated in May, Nyenswah, LLB, MPH ’12, received a letter from President Ellen Johnson Sirleaf appointing him assistant minister of Health and Social Welfare/deputy chief medical officer of Preventive Services.

It’s been a long journey to his dream. After Nyenswah and his family returned to Liberia from Ivory Coast, he earned degrees in biology and chemistry and a law degree. He became deputy program manager of Liberia’s National Malaria Control Program. There, he crafted policies for the treatment, prevention and control of the disease that affects 3.5 million Liberians and is the leading cause of death for pregnant women and children under 5. Although the program distributed more than 5 million mosquito nets and treated 2.5 million malaria episodes in 2010–2011, Nyenswah came to the Bloomberg School after deciding that he needed formal study in public health in order to be an even stronger health advocate.

Even so, he brought a wealth of direct experience about malaria treatment and prevention to the classroom. Tolbert was “more of a colleague [than a student] in the field of malaria,” says his capstone advisor William Brieger, DrPH ’92, MPH, an International Health professor and a senior malaria specialist with Jhpiego.

In his new position, Nyenswah says he is supervising an expanded immunization program and collaborating on maternal and child health issues. “Something I’m taking home is [the idea of] making change through policy,” says Nyenswah, whose new position, he says, puts him “at the epicenter for the implementation of the essential package of health services that is the cornerstone of Liberia’s 10-year National Health Plan and Policy [2011–2021].”

Since Nyenswah’s return to Liberia in June, he has been deeply involved with creating community-based health care services, particularly in communities where public health infrastructure has been decimated by violence. His MPH studies, he says, have given him a newfound “confidence that you can deliver the necessary services anywhere.”

It’s this kind of optimism that inspires Henry B. Perry, MD, PhD, MPH ’71, senior associate in International Health and Nyenswah’s mentor. “What I learned from [Tolbert] was that it really is possible for an individual to make a difference in government programs,” says Perry of Nyenswah’s work with Liberia’s malaria program. “Tolbert is an exceptional person with a strong personal commitment. Even though he is not a physician, he obtained specialized training and will put it to good use as a strong leader with a focus on engaging communities.”

Although Nyenswah looks forward to working further with American colleagues and institutions to promote global health in both Liberia and in Africa, he is pleased to be home in Monrovia, he says, “serving my country and my people.”

—Mary K. Zajac
HIV’s Confounding Superpowers

As a founder of the Rakai Health Sciences Program in Uganda, Maria Wawer, MD, MHSc, has led some of the most influential HIV/AIDS research in the past two decades. Still, the cunning of the HIV virus sometimes astonishes even her.

New research from Rakai upends conventional thinking about the virus’s powers of superinfection, a condition in which an HIV-infected person later acquires a second, new viral strain.

The first study of superinfection in a general population, published online June 5 in the Journal of Infectious Diseases, suggests that it is more common than initially thought and is not limited to groups at high risk for HIV, such as sex workers and intravenous drug users.

“The study shows that superinfection … is not rare, and the implications for vaccine and other prevention research may be quite substantial. This is one clever virus.” —Maria Wawer, PhD, staff scientist at the National Institute of Allergy and Infectious Diseases and lead author of the new study.

For answers, he turned to the Rakai Program, which follows the health of a 14,000-member cohort and has a massive data repository of blood samples and interviews to better understand HIV transmission and prevention.

“The monitoring of this population for 18 years gave us the statistical power and samples to take a look at this question in a general population,” says Redd. “It’s an ideal place to compare rates of diseases because people aren’t selected on risk factors or certain characteristics. If you live in this village, you’re asked to join the study.”

Researchers tested HIV-positive blood specimens collected between 1998 and 2004 from 149 randomly selected members of the Rakai cohort. They looked at two samples: one taken at the time of initial HIV diagnosis and the second up to eight years later, but prior to when the patients began antiretroviral (ARV) therapy.

Using a highly sensitive ultra-deep virus sequencing technology, scientists captured “snapshots” of the entire virus population and identified seven cases of superinfection. The observed superinfection rate in this study was 1.4 per 100 person years.

The finding raises important questions about the immune system response of the initial HIV virus and may have implications for future vaccine design, Redd says.

Says Wawer: “We need to do a lot more work with our immunology colleagues to try to understand why the initial infection does not protect persons from superinfection.”

From a clinical standpoint, based on existing research, a diagnosis of superinfection doesn’t appear to diminish the effectiveness of ARV therapy, the standard treatment for HIV. However, scientists have voiced other concerns, says Redd. It’s unclear whether superinfection leads to an accelerated progression of HIV to AIDS or whether superinfection increases the transmissibility of the virus. And the potential for superinfection with an ARV-resistant strain is another unknown.

In the meantime, Redd says that it makes sense for clinicians to discuss superinfection with patients newly diagnosed as HIV positive.

“You don’t have to be high risk to be at risk for superinfection,” he says.

—Jackie Powder
The X Factor

If the skin you’re in is in good shape, you may need to thank the X.

A surprising X-shaped discovery made by Bloomberg School scientists not only reveals the core chemical architecture of nanofibers inside of skin cells but also may provide new insight about how skin manages to be an effective portal, one that’s both protective and permeable.

“From form follows function,” says Pierre Coulombe, PhD, the E.V. McCollum Professor and Chair of the Department of Biochemistry and Molecular Biology. “We found a form that was unexpected. And now we are in the early stages of revealing a new type of function for these nanofibers.”

His team was shocked to find a specific type of chemical bond called a cystine linkage lurking inside cells.

“Cystine linkages are quite abundant in biological systems, but outside cells, not in them,” Coulombe says.

The particular nanofibers that he studies originate from coiled coils of keratins, which are protein components manufactured by 70 genes. He discovered in 1991 that a mutation in one of these genes causes a disease (epidermolysis bullosa simplex) characterized by skin so fragile that it can be shredded by even minimal friction—the kind caused by well-fitting shoes. In a high-tech search for clues to reveal the mechanism, his team scrutinized the nanofiber’s most basic structural makeup using X-ray crystallography, a sophisticated technique accomplished with the assistance of Daniel Leahy and Min-Sung Kim of the Johns Hopkins School of Medicine.

Focusing on mouse skin, the researchers obtained data to suggest that the X-shaped orientation of keratin nanofibers and the associated cystine linkages concentrate around the cell’s nucleus, which houses the genome.

“This is the first evidence that the size and shape of nuclei may be impacted by a keratin nanofiber network,” says Coulombe, who credits the perseverance of Chang-Hun Lee, a former postdoc and first author of the study published in June in Nature Structural & Molecular Biology.

“Nine out of 10 people would have said the odd discovery of the X shape was an artifact and not bothered trying to pursue it,” he recalls. “But Chang-Hun did bother, and we found a new form that’s leading us to uncover new function.”

The next step for Coulombe’s lab is to use genetic engineering to investigate the consequences of preventing the formation of these cystine linkages and the associated X-shaped orientation of keratin nanofibers in their natural context in the skin.

The big-picture question, ultimately, is how do the sizes and shapes of nuclei—of not only skin cells, but all kinds—affect cellular processes.

“This is an area of biology that’s beginning to get traction,” Coulombe says. “For one thing, it’s going to help us understand how the skin achieves a form that allows it to be an effective interface, fostering a healthy relationship with our environment.”

—Maryalice Yakutchik
As a boy, Dave Love raised oysters with his family in the marshes of Virginia. As a grad student in North Carolina, he studied shellfish and water quality. And as project director for the Public Health and Sustainable Aquaculture Project at the Johns Hopkins Center for a Livable Future (CLF), he chose oysters to star in some of his latest research.

Love, PhD, MSPH, is a systems guy. Public health, diet, food production and the environment are pieces of a single puzzle, he says, linked in constant interplay. With pressures like population growth, climate change and resource depletion, Love and his CLF colleagues work to better understand the food system and how to achieve balance.

Convinced that aquaculture—growing seafood in controlled conditions—is a powerful strategy to restore that equilibrium if sustainable methods are used, he sought a research project that could help make his case.

Oysters, a “perfect aquaculture species,” have simple needs and deliver large benefits—when properly treated.

In Maryland’s Chesapeake Bay, polluted waters are off-limits for oystering. So when Love heard that people were still occasionally getting sick from eating oysters, he wondered if the shellfish were being illegally harvested in contaminated areas. The resulting paper, just published in the *Journal of Shellfish Research*, sheds light on poaching patterns that highlight the need for more intensive enforcement and reveal challenges within Maryland’s wild-caught oyster industry—challenges that oyster aquaculture could solve, say Love and five co-authors.

Digging through citations issued by the state dating back to 1959, the authors found that poaching does have the potential to make people sick; about 6 percent of the tickets were for harvesting oysters out of season (contamination is more likely in warmer months) or from contaminated areas, or storing them inappropriately.

But poaching poses a greater danger to the Bay’s oyster population itself: 75 percent of citations were for harvesting too-small oysters. When oysters are harvested below market size, the population can’t rebound, Love says. Oysters are essential for a healthy Bay for two reasons: they filter the water when they eat, and they build reefs that other organisms use.

The state of Maryland has already initiated a two-pronged approach to restore the oyster population. Oyster bars in areas off-limits for harvest allow oysters to flourish, says Kennedy Paynter, a co-author and research associate professor at the University of Maryland. The bars are often intentionally located near sewage outfalls so that these oysters—not meant for consumption—can benefit from the accompanying nutrients; but any oysters poached there may be contaminated.

Without stronger enforcement, the potential for an outbreak of disease is significant.

The state is also using loan incentives to drive the shift to sustainable aquaculture. Love’s team hopes their paper will encourage such efforts to balance the interests of watermen, resource managers and public health by switching from a wild-harvest approach (which leaves openings for poachers) to a farm-raised approach, where individuals tend their own sites.

“We wanted to say to the General Assembly, ‘Here’s some evidence that shows you guys are headed in the right direction in strengthening laws for harvesting from the Bay,’” says Love.

—Rachel Wallach
Cigarette smoking. Drunk driving. Lead poisoning. They’re public health problems that trigger immediate associations with injury, illness and death. Ozone pollution, however, generally doesn’t arouse similar concern.

Environmental Health Sciences (EHS) doctoral student Jesse Berman hopes to help alter that perception of ozone, a pollutant formed by chemical reactions in sunlight and the main ingredient in smog. Decades of studies have found that exposure to unhealthy ozone levels can lead to decreased lung function, exacerbated asthma symptoms and more hospital and emergency room visits. Especially at risk are children and the elderly.

Although an individual can choose to quit smoking, abstain from alcohol before driving and protect a child from lead exposure, Berman points out that people have few options for avoiding ozone pollution.

“You can’t really do anything as an individual to reduce exposure except stay inside when the air quality is bad,” he says.

In a new study, Berman reports that exposure to ozone levels in excess of federal limits accounted for as many as 2,480 preventable deaths between 2005 and 2007. And, if more stringent regulations had been in place, he estimates that nearly 8,000 ozone-related deaths could have been avoided during the three-year period.

“The results give firm, quantifiable numbers of avoided deaths and illnesses if we [had actually met] the existing air quality standards, and the [health] effects under proposed standards,” says Berman, lead author of the study, published online July 18 in Environmental Health Perspectives.

Investigators also determined that there would have been 3 million fewer cases of acute respiratory problems and 1 million fewer lost school days if current EPA ozone regulations had been met over the last two years.

In 2010, the EPA’s Clean Air Scientific Advisory Committee recommended lowering ozone standards from 75 parts per billion to between 60 and 70 parts per billion. A year later, President Barack Obama rejected the proposal and deferred additional review until 2013, according to the study.

As EPA moves forward next year with further review of ozone standards, Frank Curriero, PhD, MA, a study co-author and EHS associate professor, suggests that the new research will be especially relevant to the process. “I can’t imagine going into any kind of discussion or review, whether from the EPA’s side or the president’s side, without this article in hand,” he says.

—Jackie Powder

Solar Powered

Catching rays. It’s the latest strategy in the Bloomberg School’s efforts to create a greener campus. Ken Uhl (left, recently retired) and Joe Bentz of the School’s Facilities Department stand among the 110 newly installed rooftop solar panels on the Wolfe Street Building. (Another 54 solar panels top Hampton House.) The solar arrays are projected to generate 64,379 kilowatt hours of electricity per year and shrink the School’s carbon footprint by about 74,230 pounds of carbon dioxide annually. The project is part of a wide-ranging University plan to reduce carbon dioxide gases through conservation measures and new technologies.
The words “parasite,” “bacteria” and “digestive system” do not necessarily give comfort when nestled alongside one another—unless, that is, they are being used to describe the work of Marcelo Jacobs-Lorena, PhD.

Ten years ago, Jacobs-Lorena, a professor of Molecular Microbiology and Immunology, began genetically engineering mosquitoes to make them resistant to *Plasmodium*, the parasite that causes malaria—a disease that kills more than 800,000 people each year. The approach held great promise. Drug and insecticide resistance have stymied efforts to manage both *Plasmodium* and the female *Anopheles* mosquito that transmits it, and there is no vaccine.

Unfortunately, while Jacobs-Lorena’s transgenic mosquitoes performed well in the lab, there was convincing evidence that their malaria-fighting genes would be difficult to propagate in the field. So he and his colleagues at the Johns Hopkins Malaria Research Institute began searching for other ways to foil *Plasmodium*.

Their latest solution, reported in July in the *Proceedings of the National Academy of Sciences*, involves tinkering not with the mosquitoes but rather with the bacteria that live in their digestive systems—more specifically, in the midgut, a tiny tube that could become ground zero in the war against malaria.

When a female *Anopheles* mosquito bites an infected human, she ingests both blood and parasites. The latter mate in her midgut, yielding a handful of thick-skinned offspring called ookinetes. These, in turn, cross the midgut and transform into oocysts, each of which then spawns thousands of progeny that migrate to the mosquito’s salivary glands, where they stand ready to infect the next person she bites. Consequently, says Jacobs-Lorena, “the best way to interfere with the parasite is before it becomes an oocyst.”

Fortunately, nature has provided an opportunity to do just that.

“It just turns out that, like us humans, the mosquito carries lots of bacteria in its midgut,” Jacobs-Lorena says. “And every time the mosquito feeds on blood, the bacteria increase tremendously in number.”

Hence the new approach: Rather than genetically modifying the mosquitoes to knock out *Plasmodium*, modify their midgut bacteria to do the job instead.

Jacobs-Lorena and his team engineered *Pantoea agglomerans*, a bacterium commonly found in the midgut of *Anopheles* mosquitoes, to secrete five antimalarial proteins. The two most effective proteins thwarted oocyst formation of *Plasmodium falciparum* (the parasite that causes the deadliest form of malaria) by 98 percent. The technique also worked well against *P. berghei*, a species of *Plasmodium* that infects rodents, suggesting that it might work against any variety of the parasite.

In theory, this new strategy should sidestep the gene propagation problem, since distributing engineered bacteria among wild mosquitoes ought to be easier than replacing an existing mosquito population with a transgenic one. Indeed, spreading the antimalarial microbes could be as easy as baiting jars with cotton balls that have been soaked in bacteria and sugar water. (Female *Anopheles* need blood proteins to produce eggs, but also feed on nectar.)

Hurdles remain, such as securing regulatory approval to release genetically modified organisms into the wild. In the meantime, Jacobs-Lorena and his collaborators have already identified a different bacterium that holds even greater promise than *Pantoea*. And they are trying to engineer a single gene that will enable the microbe to produce several anti-malarial compounds at once.

Jacobs-Lorena suspects that, unlike his transgenic mosquitoes, these *Plasmodium*-fighting bacteria will work even better in the field than they do in the lab. If so, they will make a potent addition to an antimalarial arsenal that is in sore need of new blood.

—Alexander Gelfand
In a new alliance with a leading online education provider, the Bloomberg School is refreshing its commitment to free online public health education.

Johns Hopkins is among 12 top-ranked universities that partnered in July with Coursera to make high-quality education available worldwide. The School is the first Hopkins division to offer classes through Coursera, founded a year ago by two Stanford University professors with four university partners. “It’s part of our mission to disseminate our knowledge,” says James Yager, PhD, senior associate dean for Academic Affairs and the Edyth H. Schoenrich Professor in Preventive Medicine. “We felt that this level of visibility, particularly with these other institutions, was the right thing to do at the right time.”

With the first course to launch in late September, the School’s Coursera offerings include Biostatistics Bootcamp, Introduction to the U.S. Food System, and Vaccine Trials: Methods and Best Practices. At presstime, more than 100,000 people had registered for the noncredit courses.

The collaboration augments the School’s Internet-based offerings, which include 106 online credit courses and OpenCourseWare (OCW), a Web resource that makes the content from 107 courses accessible at no cost to users worldwide on a noncredit basis. OCW does not offer assignments or exams. “[Coursera] is addressing another way that people learn,” Yager says, “and we’re reaching out to provide knowledge in a slightly more formal way than OCW that will hopefully help people who could never come here.”

The target audience is “anyone in the world who has access to the Internet and who has an interest in the course topics.” Yager says that Coursera’s approach to open learning differs from the OCW model in that it offers “mini-courses” that may include quizzes and assignments. Students can complete class evaluations and may post questions to faculty via an online bulletin board. They can also answer each other’s questions and create their own social networks of learners with shared interests.

“You can actually determine how well you’re learning by taking quizzes and getting a little bit of faculty input,” he says.

Describing the Coursera partnership as an “experiment,” Yager says that School officials will assess the extent of global interest in the curriculum, review student feedback and determine the demands on faculty.

Yager says that the commitment would be worthwhile if students found the courses to be of value and “if we were able to attract some new students to take full courses and/or enter one of our degree programs.”

—Jackie Powder
Courting Health

Stephen Teret trains students to use a potent lifesaving tool: the law.
In 1978, Stephen Teret was a trial lawyer turned MPH student when he first thought of using litigation to advance public health. He explored the idea in a paper for an injury prevention course taught by Health Policy and Management (HPM) professor and injury expert Sue Baker, MPH ’68. The published paper was the first in a series of reports connecting litigation and public health. In a provocative article for a trial lawyers’ magazine, Teret suggested that carmakers be held liable for failing to offer airbags as a safety option. Litigators eagerly took up the idea. By 1985, Ford Motor Company, facing $1.1 billion in airbag litigation claims, began offering airbags in cars.

Teret, JD, MPH ’79, has pursued the integration of public health and the law ever since. Now he’s launching the Johns Hopkins Clinic in Public Health Law and Policy, a first-of-its-kind initiative to teach students to use the law to solve public health problems. “I would hope that in years to come, all schools of public health develop similar clinics and that ours will be seen as the prototype,” says Teret, an HPM professor. He spoke this summer with writer Jackie Powder about the emerging discipline and his hopes for the new clinic.

How did the field develop after the airbag litigation?

We thought, here’s an example of where litigation actually seemed to be successful in changing the behavior of product manufacturers. We then expanded that to deal with litigation involving guns, and litigation involving other consumer products. With guns, we encouraged lawyers to sue gun makers for failing to use existing safety technologies that could prevent some shootings. Another example would be litigation against the maker of an outboard engine for a boat for failing to place a guard around the propeller. It’s work that was done at this School that developed the idea of using litigation as a tool to protect the public’s health and safety.

Is public health law a growth area within the legal arena?

There are a lot of people now who are interested in public health law, much more than there were 10, 20 or certainly 30 years ago. It’s a growth area in that foundations now are supporting work in public health law. For instance, the Robert Wood Johnson Foundation, one of the largest foundations with regard to health issues, has invested a great deal of money in the last few years in enhancing the quality of public health law and increasing the human capital, people who are working in public health law. Lawyers may not be the first group of people that one thinks about when one is thinking about public health, but now there’s widespread acceptance of the value of public health litigation as one of the most potent tools for enhancing the public’s health.

What’s the idea behind the new clinic?

When I graduated from this school a very long time ago, I didn’t really know well enough how I could solve actual public health problems in a practical way, and I think it’s still true for some students today. They’ve learned a lot about key elements of public health—epidemiology, biostatistics, health policy, health behavior, biological sciences—but they haven’t necessarily learned how to go out and solve a public health problem. We’ll present students with a public health problem and they’ll bring together what they’ve learned to actually solve it by the use of law and policy.

But to be effective in public health law, students would need a law degree, wouldn’t they?

No. Students don’t need a law degree. Students in the clinic are going to be fortunate in that they will have at least six faculty members working with them, all of whom have law degrees as well as an understanding of public health. The faculty will help with technical legal issues, although we hope that some of our students who are either lawyers getting their MPH degree or in a JD/MPH joint degree program will also participate and help their colleagues to understand how the law can be used as a tool in public health.

Why did you select salt consumption as the clinic’s first issue?

It’s a compelling public health problem for which there is not a solution immediately in sight. Working on salt consumption will present our students with a problem that has interdisciplinary aspects to it—the biology of how the body deals with sodium, the epidemiology and the costs of hypertension disease, the behaviors of individuals regarding sodium consumption and the behaviors of businesses that make and market foods.

Most of the salt that people get is not from a saltshaker. It’s the salt’s already in prepared foods at restaurants and supermarkets. You can go to a fast food or chain restaurant and order a meal that may give you 7,500 mg of sodium (one of the elements of salt)—multiple times the recommended daily dietary allowance of sodium—and there’s no law or regulation that requires the restaurant to give you a warning that you might be placing yourself at risk: “Before you eat that, watch out, you’re getting 7,500 milligrams of sodium.”

What legal and policy solutions might the students propose for dealing with this issue?

They might decide that a regulatory agency like a health department should require restaurants to post how much sodium is in a given menu item, and write up a model regulation. They might draft model legislation that sets limits on how much salt is permissible in foods. If they think that litigation is going to be the most effective tool, then they could write up the documents necessary for the commencement of a lawsuit. We want our proposed solutions to actually be implemented, so, ultimately, we’ll work with legislators, regulators, litigators and other policy people to see how what we’ve developed can be used to address a problem that is estimated to cause 150,000 excess deaths a year in the U.S. from hypertension and related diseases.

What will determine whether the clinic is a success?

We have a wonderful group of dedicated public health lawyers who are all excited to teach in the clinic, and we have the best public health students in the world. I don’t foresee any barriers to success.
“It could have been the flu, pink-eye, or hell, let me be stung by a bee. But Oh no, I was so lucky... I caught HIV.”

—From the poem “My Experience” by Tavon Vinson
Tavon Vinson just did not have time for this call.

The popular gay 18-year-old, class president of Edmondson-Westside High, an actor, an athlete … his life was too vital, too full, to have the gears suddenly stripped from an engine that loved running at full throttle. And yet the voice on the other end of the line was attempting to do just that. A doctor, an old friend, a comforting face that normally Tavon enjoyed seeing. But now, in this moment, in this context, he was disembodied, his tone somber, his words halting yet imploring. It was as though the cell phone was discharging volts of fear along with the message:

“Tavon… you need to come in.”
“My test, MY TEST! Tell me, TELL ME!” pleaded Tavon.
“I can’t. Not over the phone. Please, just come in and we’ll talk. It’s OK.”
“Look, I have a track meet to run tomorrow. It’ll ruin my focus if I don’t know. Tell me. TELL ME!”

This wasn’t a negotiation. The young man was desperate, consumed by dread. He’d always protected himself during sex. And he went for regular HIV tests. And he’d had no partners at all since his last test, so what was going on?

To the doctor, the cause wasn’t the immediate concern. The result was. It came down to compassion versus clinical guidelines. In the end, compassion won out. The doc couldn’t string this young man along, not for one more minute. And so, in a sentence, he forever changed the young man’s life.

“Tavon. The test. It … was positive.”

On the other end of the line, silence. Then screaming. Then cursing. And through it all, like a mantra against the torrent of pain, the doctor repeating, again and again, “I’m sorry, Tavon. I’m sorry. I’m sorry.”

The phone call ended. Tavon, in a daze, instinctively went to the place that had always brought him comfort. Across the field from his home was the track. As the sun set, he sat cross-legged on the infield, surrounded by the gravel lanes and chalked lines that had brought him glory.

And he cried.

And he cried.

The numbers have remained consistent and horrifying through eight years and three separate research cycles by Bloomberg School scientists and colleagues from the Maryland Department of Health and Mental Hygiene.

According to the Behavioral Surveillance Research Study (BeSURE) sampling completed last December, 48 percent of African-American men who have sex with men (MSM) in Baltimore City are HIV positive. That’s far higher than the average of 28 percent found in the 20 other cities participating in the National HIV Behavioral Surveillance in 2008. (Researchers in Baltimore, like those in the other cities, recruited most MSM study participants from clubs and venues that might attract people who engage in higher HIV risk behaviors.)

The prevalence is even more troubling when compared to the two other cohorts in the BeSURE sampling: African-American MSM surveyed in 2011 were infected with HIV at a proportion more than three times that of injection drug users, and eight times that of heterosexuals considered at high risk for infection. Baltimore’s MSM community is, in one sense, a microcosm of a global issue; a recent *Lancet* article authored by Bloomberg School faculty noted that in countries like France, Australia, and the U.K., the overall HIV rate is declining in populations with the exception of one group: MSM.

In Baltimore, a key challenge for reducing HIV among the MSM community is testing. More than two-thirds of those surveyed with HIV had no idea they were infected prior to testing in the study. That means many African-American MSM are not receiving critical HIV care services, says BeSURE principal investigator Danielle German, PhD ’09, MPH. “It is also a challenge for preventing HIV transmission to partners,” she says. BeSURE participants “get the full scope of pre- and post-test counseling, referral services and linkage to care. That aspect of our activities is as much of a priority for us as the data gathering.”

BeSURE is working on the problem because there’s little doubt that knowing one’s status greatly lowers transmission risk. The transmission rate for people who know
their status is 2.7 percent, versus 10.4 percent for those unaware, says David Holgrave, PhD, an HIV researcher and chair of the Department of Health, Behavior and Society (HBS). But the appalling infection rates don’t seem to be attracting larger community or public health attention. “If the rate of infection in the African-American inner city male population was the same in medical doctors in Baltimore,” says Carl Latkin, PhD, an HBS professor, “it would be considered a national emergency and a huge amount of attention and effort would be put into combating it. And from the inside, there’s a lack of effective grassroots organizations that have demanded that the city address it as a public health issue.”

That lack of community cohesiveness is perhaps the natural outcome of how MSM are seen by those in Baltimore’s African-American culture. To delve into that culture, to understand the stigma that in many influential corners is still attached to homosexuality, is to glimpse the nature of the challenge facing public health workers trying to encourage MSM to know their status and protect both their health and the health of the community.

At 49, Carlton R. Smith is old enough to remember when HIV was a death sentence. That it’s not anymore—at least for those who get tested and treated—is a message he conveys via the organization he founded in 2002, Baltimore Black Pride.

Consider the picture that Smith paints of the rejection a young, gay African-American male often encounters in the inner city. “It comes some out of the churches,” says Smith. “They preach out of the book of Leviticus that you’re an abomination. People who don’t have an understanding of the [religious] context hear that word and think they’re a blight on the planet. Sometimes, there’s a community sense of, ‘How dare you!’ You’re taught as an African American that you need to be a man and create and take care of your family. And now you’re going to come out and say you’re what? You’re gay?” — Carlton R. Smith

“You’re taught as an African American that you need to be a man and create and take care of your family. And now you’re going to come out and say you’re what? You’re gay?” — Carlton R. Smith

Some of the factors that impair self-esteem are not unique to the African-American community. But racism, poverty and high unemployment are endemic in the inner city. Access to resources—and even public transportation—is an additional barrier to testing and care. For many young men, the hurdles to determining one’s status, let alone accessing care, are too discouraging to consider. “When I talk to a lot of young people, they’re not worried about testing,” says activist Keith Holt, 26, who is HIV-negative. “They’re worried about living … ‘where am I staying at tomorrow? Will I have something to eat?’ It’s about what’s important to you at that moment. With HIV, it’s not something you think is affecting you right in the here and now, even though it is.”

And just as easily, discouragement can turn to anger, with deadly effects. “For people who have been kicked out of their house for being gay, or positive, or they can’t get insurance or medication or don’t know how to get it, they’re afraid they’re going to die, they’ve been through a lot,” says one HIV-positive community activist. “When you’re not getting help, and you have no one to talk to and you’re dealing with it by yourself … there’s a reason that people go crazy and have unprotected sex: They just don’t care anymore.”

And so the wildfire continues to rage.

For those on public health’s frontlines, spreading the test-and-treat message among Baltimore’s African-American MSM community is no easy task. That may be due to a lack of urgency on the part of younger MSM, who haven’t seen their peers die from AIDS in the numbers that older MSM have. Another barrier is that the African-American MSM community is so fragmented that an outreach effort or a single ad campaign is unlikely to reach all MSM.

“You’re taught as an African American that you need to be a man and create and take care of your family. And now you’re going to come out and say you’re what? You’re gay?” — Carlton R. Smith

The conundrum of programs since I’ve worked in them here, in Seattle and New York, is that when we say we’re having a ‘gay’ event, we get men who identify as gay and it’s usually white or Asian gay men who make a certain amount of money,” says Anthony Morgan, an HIV health educator with the Lighthouse, a Bloomberg School project that conducts community-based disease reduction research and education in Baltimore. “And when you look around the room you notice that maybe there’s one or two Latino guys, and maybe a black guy. So do you say we’re just having a black men’s program, and see who is engaging in a certain type of sex? Do you say ‘MSM’ and risk that no one will connect with it? Or is that term too objectifying; does that just reduce you as a human to where you put your penis?”

There’s no shortage of groups that fall under the MSM banner. There are the out and open, who may well be in the minority; these include the club-goers who were recruitment targets of BeSURE’s cohort, and members of the ballroom scene, where “houses” compete in lavishly costumed and elaborately produced shows reminiscent of French haute couture runways.

But many African-American MSM lead fully or semi-closeted lives: professionals, college students, bisexuals, men on the
“Isn’t this some shit,
I’m track captain, president of my class,
But now everybody won’t see that,
just the gay black boy
that’s gonna die fast.”
—From the poem “My Experience” by Taavon Vinson
“A dear friend became positive, and he never knew until he had full-blown AIDS. It changed my life, and got me out in the community to get people tested.”
—Adrian Ross

Committed to the cause: Ross and Carlton R. Smith (below)
“down-low” who meet regularly for sex with men in a tight-knit group that is never mentioned elsewhere. These groups are far harder to reach. In some cases, the reticence to discuss homosexual sex is so pervasive, it’s not even acknowledged by men who engage in sex with men as part of their work.

“One focus group we did was with men who sold sex,” says Latkin, an HBS professor. “And one guy brought in [male prostitutes] from The Block where they sold sex. What I found fascinating about that discussion was that here were six guys who knew each other, and nobody ever mentioned having sex with a man; they might say ‘a client,’ or ‘somebody,’ or this and that, but even in a group that was completely aware of each other’s behaviors, there was still a high level of stigma, of even talking about [MSM] for that group.”

Given this ongoing reluctance to self-identify sexual preferences or which subgroups one identifies with, behavioral interventionist Karin Tobin, PhD, says the best course might be to focus on education. She and Latkin have been piloting a program called Unity in Diversity, which targets African-American MSM (including MSM who also sleep with women) to educate them about their status, risk behaviors such as frequency of condom use and, most importantly, to encourage them to pass that information on to their social networks regardless of how they self-identify.

Tobin reports that three months after the seven group meetings, participants reported a reduction in the number of male sex partners and an increase in condom use compared to those in the control group, which had just a single risk-reduction counseling session. But what also caught her ear was how participants didn’t readily verbally identify with being gay or part of an MSM subculture. “In our experience with guys in Unity in Diversity, if we don’t bring it up, they’re not bringing it up. I’m not going to force a label on anyone,” says Tobin. “I think folks come in waiting for that shoe to drop, for you to put a label on them. And even if someone were to think, looking from the outside, that a person identifies as gay, he may never use the term, ‘I’m a gay man.’”

Activist Adrian Ross, 26, agrees that a simple, broad test-and-treat message is probably best: “Take a message targeted to the ballroom community. What is a D.L. [down-low] or ‘straight-identifying’ [MSM] going to know about the ballroom scene? Nothing. What he does, he does in the dark, fast and quick.”

Whether Tobin’s efforts have a long-term effect on Baltimore’s MSM HIV crisis remains to be seen, but what all sides agree on is that any inroads will need tremendous community buy-in. Identifying and gaining the trust of the key stakeholders is where the game is really at. MSM are taking leadership positions in the community for their friends and other young men who need support.

In Baltimore, being educated, tested and treated for HIV is a hit-or-miss proposition. The city’s school system barely touches upon the subject. For the inner-city African-American MSM community, comprehensive, one stop test-and-treat shops sensitive to their issues are hard to find. Single-focus, free clinics can be so obvious that to be seen there is the scarlet-letter equivalent of saying, “Oh … you, too.”

It’s no wonder Latkin says, of the overall academic, city and state public health push to engage the African-American MSM community in HIV awareness, there’s no coherent plan. “There’s inadequate monitoring of what’s going on in the community. We don’t know when people drop through the cracks,” says Latkin. “If someone is treated at Hopkins but then goes to University of Maryland, we don’t know, they may be lost. Maybe there’s too much testing in the community and not enough linkage to care. Or maybe one group has been inundated with [“get tested”] messages every day and another hasn’t gotten a message in months. It’s completely fragmented, how we approach it, and this leads to huge inefficiencies and lack of effectiveness.”

Acting Baltimore City Health Department deputy commissioner Patrick Chaulk, MD, MPH ’89, says the city is doing its utmost to provide testing and care. As in other U.S. cities, individuals who receive a positive diagnosis at a Baltimore City health facility are referred to a care provider. “But we take it a step further; if you get diagnosed we have same day referral in which we drive you to a provider, wait for you to get everything done, and then drive you home,” says Chaulk. “We do two of those visits in the first six months [after an HIV diagnosis]. I don’t know of any other city that does that.”

Still, with Baltimore missing a major grassroots African-American MSM-oriented organization like Washington, D.C.’s Us Helping Us, the movement to bring a message of hope and cohesion to those most at-risk has landed squarely on the shoulders of those with the most energy and awareness: the city’s African-American gay youth.

Before he could become an AIDS activist, Tavon Vinson first had to survive the emotional plunge following his diagnosis. Vinson and his doctor believe he was infected by a contaminated needle during...
Tin Tun rides his motorbike through the Thai border town of Mae Sot.

He cruises past rice fields on the outskirts until he reaches the warehouse of a corn-processing plant. A wiry man with a broad forehead and receding black hair, he ducks into a passageway that leads to an encampment for Burmese migrant workers.

Tin Tun must travel here each week to counsel Yi Yi (not her real name) because she is afraid to leave the workers’ compound. Like other undocumented Burmese, Yi Yi fears the Thai police, who often demand bribes to ignore migrants who lack the proper papers.

Tin Tun’s counseling session is part of a study by the Bloomberg School’s Applied Mental Health Research (AMHR) group. Its aim: to test whether lay counselors with brief training and close supervision can provide effective therapy to people within their communities who have suffered from trauma or violence.

For Yi Yi and the roughly 2 million other Burmese who have crossed into Thailand, harrowing stories are commonplace. Since 2011, a quasi-civilian government in Burma (also known as Myanmar) has eased repression. However, these reforms follow on nearly 50 years of military rule that gutted the economy, outlawed dissent and waged war on ethnic groups seeking self-determination. The junta packed the prisons with dissidents.

The army burned thousands of villages, destroyed crops and forced people to do...
dangerous work without pay; Tin Tun’s colleague Mya Mya Win, for example, counseled a woman whose brother died doing forced labor.

Tin Tun himself has faced prolonged hardship. He was arrested for political activities the day after his daughter’s birth. Torturers deafened his right ear and scarred his body.

When the regime released him in 2009, his baby daughter was 19 years old.

Today, it is Yi Yi’s suffering that he will address. Tin Tun walks down the trash-strewn lane between two rows of houses pieced together from bamboo, corrugated zinc, plastic sheeting and empty rice sacks. Yi Yi emerges into the glare to greet Tin Tun. She is 45, has short black hair and dresses in stylish capris. Tin Tun kicks off his flip-flops and stoops to follow her into the wobbly 8-by-10-foot shack with a roof made of leaves. He sits cross-legged on a woven mat facing Yi Yi, and they begin to talk.

The purpose of therapy with Yi Yi and others is to help them recognize the connections between thoughts, feelings and behavior and to use this understanding to feel better. In eight to 12 sessions, Tin Tun and 16 other counselors teach their clients to notice negative or self-defeating thoughts and behaviors and then to reconsider them. The counselors guide clients in moderating their intense emotional and physical reactions to memories of trauma.

More is at stake than the clients’ psychological distress: depression and anxiety can lead to physical illness, risky behavior, fractured relationships, injuries, lost wages, even suicide.

Today Yi Yi talks to Tin Tun about her divorce. She thought it was all her fault, but Tin Tun has helped her see that her husband’s drug addiction played a role. When Yi Yi began therapy, she recalls, “I felt so hopeless. I had no future.” These days, she says, “I have many goals.” Each day, she and her new husband manage to spend only $1.50 of the $5 to $7 he earns loading trucks. “When we go back to Burma, we can open our own rice shop in Yangon,” she says.

Another of Tin Tun’s clients was a young man who was furious with his father for the political activism in Burma that landed him in jail. The son felt abandoned. Tin Tun says he helped his client to recognize that the father had been attentive before his imprisonment, and that after his release, the father had helped his son to find work in Thailand. At the start of therapy, the young man “felt as if a stick was stuck in his heart,” says Tin Tun. “Now he feels he can take out the stick.”

Tin Tun’s own children refuse to see him because they resent his absence while in prison. To avoid obsessing over his grief, Tin Tun applies to himself the coping skills he teaches to his clients. He redirects his thoughts and seeks positive interpretations for events.

Tin Tun’s supervisor, Kyaw Soe Win, and his colleagues learned a structured method of counseling from assistant scientist Laura Murray, PhD. She and two colleagues spent two weeks in Mae Sot in early 2011 training the counselors, mostly high school or college graduates.

Research is scarce regarding the mental health of Burmese in Thailand, but two studies, one of ethnic refugees and one of exiled dissidents, both found that four in 10 were depressed. Among the dissidents, one in four had PTSD. The Thai health care system provides little in the way of counseling, and Burmese without papers generally don’t feel safe going to a government clinic.

The project in Thailand is one of eight linked studies in Asia and Africa under AMHR’s aegis. The research, supported by the USAID Victims of Torture Fund, tests strategies for using local counselors to help people with problems such as anxiety or depression stemming from extreme poverty, trauma or systematic violence. Other study sites are in Zambia, the Democratic Republic of Congo and Iraq.

In all eight studies, “the principle is always the same,” says International Health associate scientist Paul Bolton, MBBS, MSc, the group’s leader. “Can we make therapeutic interventions that work? Can we train people who don’t have a mental health background to provide them?” The unmet need is stark: a 2011 Lancet commentary reported that more than 75 percent of people with neuropsychiatric problems in poor countries get no treatment. To help fill that gap effectively, says Bolton, researchers must first test interventions through randomized studies like these.

So far, he says, results in all sites show that most people in the communities accept counseling and that “task-shifting” works: that is, paraprofessionals like Tin Tun can learn to conduct it correctly. But do the therapies help the clients themselves? Bolton says it’s too early to judge.

The Thailand study, led by International Health assistant professor Courtland Robinson, PhD ’04, and doctoral student Catherine Lee, took unexpectedly long to enroll 154 clients and 131 controls (who can get free counseling after waiting 10 weeks). Counseling for people without severe psychiatric illness is virtually unknown in Burma. As Kyaw Soe Win explains, “Most people think counseling is for people who are crazy. I finally got the idea of telling the community, ‘If you were crazy, we couldn’t give any counseling to you.’ ”

Ultimately, he and several other counselors want to return home and provide therapy in Burma if the recent reforms prove to be genuine and lasting. “Many prisoners have been released, and they have been tortured,” says Mya Mya Win. “I want to counsel them.” Among the ex-prisoners are two of her own sisters.

Meanwhile, Kyaw Soe Win says he will look for money to keep counselors working in Mae Sot after the Hopkins study closes at year’s end. “I’d like to do this for the migrant community,” he says. “They have faced so much suffering.”

Cathy Shufro reported from Thailand this spring on a fellowship from the International Reporting Project, an independent journalism program in Washington, D.C.
FRIENDLY FIRE
A new wave of research links inflammation to almost all chronic disease

Story Maryalice Yakutchik
Illustrations Michael Glenwood

The inflammatory system is like the ocean. It’s beautiful, but also deadly. —Josef Coresh
Sixty years ago, Patricia Mabe took her first-ever breath, inflating new pink lungs with the air of Carbondale, Pennsylvania, home of the nation’s first underground mine; a town that today, despite a long-ago demise of the anthracite industry, still smolders from burning veins of coal.

Mabe’s subsequent inhalations—even before she acquired, at age 15, a smoking habit that would be lifelong—no doubt contained vestiges of a variety of toxins, no matter whether she was outside or in. Her dad, who worked in a mine, was a smoker. Her mother smoked, too.

Whenever Mabe inhaled anything noxious, her body’s defense system recognized foreign molecules in her lungs as the interlopers they were and mounted a functional inflammatory response. Brawny cells called macrophages, among others, stormed her lungs to do away with pathogens and debris while unleashing a barrage of molecular messengers that orchestrated strategic battles and mended damaged cells.

Then—and this is every bit as important as their SWAT-team-like arrival—the macrophages would leave with all their assorted artillery in tow once the mission was over. In a healthy system, inflammatory cells are both highly regulated and self-disciplined; they go where ordered, expertly distinguish friend from foe, exert only necessary firepower, tidy up after themselves and then retreat, ultimately restoring calm—also known as homeostasis.

Precise though the immune system may be, some level of self-attack occurs even when all parts are working well. The key is keeping the collateral damage in check.

In fact, how effectively and efficiently any one person controls inflammation is a key determinant of health and disease. Some individuals regulate inflammation better than others. It’s likely that many of us are able to control inflammation at certain times of our lives better than at other times. Why—and how—is the focus of intriguing research that’s implicating how nutrition and inflammation interact in just about every major chronic disease.

“[It’s not simply the presence or amount of inflammation that’s important. It’s the texture of it. That’s where you find the causes of diseases.” —Noel Rose

Intimately related to how we meet and greet and defend ourselves from potential invaders,” says Keith West, DrPH ’86, MPH ’79, the George G. Graham Professor of Infant and Child Nutrition. “We get less good at that as life goes on.”

Over time—likely in response to the toxicity and persistence of cigarette smoke in her lungs—Mabe’s inflammatory response turned pathological. The composition of immune molecules and cells became qualitatively and quantitatively different. Inflammation became too too: There was too much of it; it was too strong for too long. Complicating that scenario, macrophages also became unified and lingered incompetently in her lungs, clogging her airways.

What once defended her from disease now was causing it.

There’s a new buzz among scientists about inflammation, even though it’s been known for ages to be one of the first responses by the immune system to infection and irritation. Inflammation is hot. And not in just a classical “calor” kind of way. Calor—as in heat or fever—is one of four characteristics of inflammation recorded in De medicina, an ancient Roman medical text. The remaining three—dolor, rubor and tumor—translate to pain, redness and swelling, respectively.

“Inflammation is something we have rediscovered in the 21st century,” explains Shyam Biswal, PhD, MS, a professor in Environmental Health Sciences (EHS), “specifically, inflammation as a focal point of chronic disease. People used to think of it as a bystander of disease. But it’s actually a driver.”

If attention to inflammation is spreading like wildfire throughout the research world, an early spark was ignited by Noel Rose, MD, PhD, director of the Center for Autoimmune Disease Research. Having introduced the concept of autoimmunity as a cause of chronic thyroiditis in 1956, Rose now is investigating the causes of magnified inflammatory responses in the hearts of young men for whom transplant is the only cure. A muscle that needs to pump, Rose concedes, is a bad location for excessive scarring to occur as a result of inflammation.

“We’re now looking at the details of inflammation,” he says, “and realizing the benefits from targeting specific parts of the inflammatory response. You have to know which part is doing good and which is doing bad and tailor drugs accordingly.”

It’s not simply the presence or amount of inflammation that’s important, Rose says: “It’s the texture of it; the makeup of the cells that are attracted, the ways in which they are stimulated and the products they release. If you’re getting down to that level, that’s where you find the causes of diseases.”

Among chronic, noninfectious disorders now commonly regarded as “inflammatory” is atherosclerosis, often called hardening of the arteries, says Josef Coresh, MD, PhD ’92, MHS ’92, director of the George W. Comstock Center for Public Health Research and Prevention.

“We’ve known for a long time that inflammation is central to atherosclerosis,” says Coresh, principal investigator of the Johns Hopkins Field Center of the Atherosclerosis Risk in Communities (ARIC) study. “You can see inflammatory cells in the lesions.” (Known as plaques, lesions form in the arteries, hardening them.)

The prospective ARIC study, which first examined 15,792 Americans in 1987 and has followed them ever since, is a key resource for the investigation of many inflammation-related chronic diseases—few of which show their hands so obviously as atherosclerosis.

Researchers who are sleuthing the origins of cancers and diabetes, for instance, have found themselves stumbling time and again on inflammatory roots. Apparently, long ago resolved and seemingly unrelated infections buried deep in people’s pasts might tip the precarious immune system balance, imperceptibly if irrevocably reprogramming it. So too might the persistently simmering, subclinical kind of
inflammation caused by excess body fat, for example. One investigative focus is finding the mechanisms that link long ago infections to inflammation and later, chronic diseases. Another is dissecting and tinkering with processes that can block, enhance or otherwise regulate inflammation once the SWAT team has mutinied.

For instance, Andy Pekosz, PhD, with collaborators from Johns Hopkins Medicine, has demonstrated that epithelial cells harvested from the noses of patients suffering from chronic sinusitis “remember” many generations later that they are different. These cells have comparatively heightened inflammatory responses to various stimuli a month after leaving a diseased nasal environment—despite having been grown and propagated under the same conditions as healthy cells in lab culture dishes.

“This tells us there’s something about these cells that has changed,” explains Pekosz, an associate professor in the W. Harry Feinstone Department of Molecular Microbiology and Immunology (MMI). “The detection machinery or the circuitry in the cells from sinusitis patients is reprogrammed to respond differently to factors that stimulate inflammation.”

Understanding this reprogramming of the inflammatory response itself is the holy grail for Pekosz.

“If we could understand how to tune down that heightened response by the epithelial cells, we could relieve the chronic sinusitis,” he says. “The flip side is, if we could, at an opportune and early time point, find a way to increase the inflammatory responses, we might have a very powerful broad tool to use against a number of different viruses, for instance.”

Fiddling even a bit with any discrete part of a delicate and complex system is not without unforeseen consequences, many of which could be perilous, if not immediately, then sometime in the future. (Case in point: Although it provided sweet relief for many with osteoarthritis pain, the anti-inflammatory drug Vioxx, which works by inhibiting an enzyme in the inflammatory pathway called COX-2, was withdrawn from the market in 2004 after a study showed it doubled patients’ risk of heart attacks and strokes after 18 months of use.)

The inflammatory system is like the ocean, according to Coresh: “It’s beautiful, but also deadly,” he says. “As long as it’s calm, you can conduct commerce and fish on it, and without it, you’re dead. But, if it storms, it’s incredibly powerful and can kill you.

“When there’s imbalance in one thing, it’s like a wave that pushes on other things and you get a whole cluster of effects.”

Among the “things” that both cause and react to system imbalances are messenger proteins called cytokines. The immune system communicates through these versatile factors that float around in the bloodstream. Unlike neurons in the hard-wired nervous system, cells in the immune system are not physically connected to one another.

“If you think of the nervous system as a landline, then the immune system is a cell phone,” says Jay Bream, PhD, an MMI associate professor. “Different cocktails of cytokines, in various abundances, play a central role in determining immune responses.”

A phenomenon known as a “cytokine storm” can occur if the reaction of the immune system to a pathogen is wildly exaggerated and stimulates too many of the messenger molecules, which in turn activate the same cells that stimulated them, resulting in a dangerous feedback loop. For example, cytokine storms are associated with severe bacterial infections and the onset of septic shock as well as avian influenza (H5N1) infection. Likewise, infection with the deadly Ebola virus is associated with a cytokine storm leading to uncontrolled inflammation.

Cytokine storms can happen in tissues throughout the body. That’s because the component parts of the immune system spread far and wide, from the top layer of skin to the deepest recesses of the bowels.

Bream, whose mission is linking cytokines with disease outcomes, studies Interleukin-10 (IL-10): “a lynchpin” he says, “at the nexus of inflammation.”

When the volume of IL-10 is turned down low, inflammation happens. When it’s blasting, inflammation is tamped down. Bream, co-director of the Becton Dickinson Immune Function Laboratory at the Bloomberg School, has shown that mice prone to express higher levels of IL-10 are susceptible to certain types of persistent infections because they can’t mount appropriate inflammatory responses. If there’s an under-abundance of IL-10, they are susceptible to immunopathology caused by collateral damage from the excessive immune response. In this scenario, the original infections clear, but the animals’ inflammatory responses set them up for autoimmune diseases and cancers. Animals whose IL-10 gene has been knocked out have extremely compromised anti-inflammatory responses. For example, under conditions that mimic septic shock, 100 percent die within 48 hours due to the cytokine storm, Bream says.

“One of the main roles of IL-10 is to control expression of pro-inflammatory cytokines,” Bream explains. “That’s how it tries to right the ship, by reducing inflammation to acceptable levels.”

If inflammation, which dictates how we respond to vaccines and infectious pathogens, is not under stringent genetic control, the result is disease.

“In terms of public health, I see all these diseases—from heart disease to autoimmune disease to cancer to schizophrenia—as gene regulation issues, related to inflammation,” he says. “Some people who get influenza end up hospitalized and die while others recover. Some people get colds and can go to work while others are bedridden. It’s this diversity in the human population of response to disease threats that is at the center of my research program.”

To find out why some individuals control inflammation better than others, he’s

“When there’s imbalance in one thing, it’s like a wave that pushes on other things and you get a whole cluster of effects.”

—Josef Coresh
looking at tiny genetic variations (known as single nucleotide polymorphisms, or SNPs) that make each human a unique individual, and noting how they affect the levels of IL-10 in various tissues, ultimately exerting control over inflammation and disease.

Because he wants to know how IL-10 works in people, Bream has inserted chunks of DNA containing the human IL-10 gene into the mice he's using. Some mice get human genes with variations associated with high IL-10 expression, and some get human genes with variations associated with low IL-10 expression.

Among other things, he’s discovered that location is all-important: Where he manipulates IL-10—that is, which tissue type—matters. If, for instance, Bream turns down the IL-10-producing ability of a subset of cells in a very specific area of the colon, just below the surface cell layer, those mice end up with severe colitis.

“IL-10 is an attractive target for therapeutic interventions that either add back or neutralize IL-10,” says Bream, who's now testing different expression levels of the human gene across various tissue types in response to different kinds of infectious pathogens in mice. “By identifying the triggers and genetic variations that regulate IL-10 levels, it will be feasible to develop more personalized therapies that restrict or enhance IL-10 in tissues where inflammation is occurring. But it’s extremely complicated.”

Another group of researchers at the Bloomberg School is looking at the anti-inflammatory IL-10 cytokine in the context of frailty in older adults. Some elderly people get frail in a clinical sense, meaning they spiral into a vicious cycle of decline characterized by exhaustion, slowness, weakness and muscle loss.

“It’s hypothesized that there’s at least a subset of older adults in whom inflammation essentially gets turned on all the time,” says Karen Bandeen-Roche, PhD, the Frank Hurley and Catharine Dorrier Professor and Chair of Biostatistics, and co-principal investigator of the Older Americans Independence Center. “It’s associated with muscle wasting and other adverse outcomes.”

Bandeen-Roche is collaborating with Jeremy Walston, MD, a Johns Hopkins professor of Medicine and co-director of the Biology of Healthy Aging Program, who has developed a frail mouse model by knocking out expression of the IL-10 gene.

“In human studies, again and again, associations of high inflammation and adverse outcomes have been revealed, with frailty prominently among them,” says Bandeen-Roche. “Pro-inflammation is thought by many to be one of the key hallmarks in a cycle of multisystem dysregulation that leads to frailty.”

Human studies designed to discover how inflammation works and reveal its links to diseases require not only big funding and endless approvals but also plenty of participants, willing subjects who are healthy, as well as those who are sick.

People like Patricia Mabe, for instance.

FIVE YEARS AGO, Mabe visited her doctor complaining of asthma-like symptoms. Disconcerting as that was, it didn’t affect her everyday life. An inhaler was prescribed, and for a while, she used it only occasionally. Then, two years ago, it was like a switch had flipped.

“I was always very active—some people might say hyper,” she says. “Then, all of a sudden, I didn’t have any energy.”

An avid walker, Mabe grew depressed when she couldn’t exercise with Buddy, her Yorkshire terrier, and Holly, her English bulldog. Already petite, she lost weight, falling to an alarming 86 pounds.

Quite a few members of Mabe’s extended family had breathing issues. Her extended family had breathing issues. Her...
parents, both lifelong smokers, needed course after course of antibiotics to fight recurring infections before they died, one year apart, from chronic obstructive pulmonary disorder (COPD). Given her heredity and behavior—despite attempts to quit, Mabe still smokes—she envisioned a bleak future. COPD is an umbrella term that includes chronic bronchitis, emphysema and chronic asthma or asthmatic bronchitis. Most people have a little of this, a little of that, according to Robert Wise, MD, a Johns Hopkins School of Medicine professor of Pulmonary and Critical Care Medicine who holds a joint appointment in EHS at the Bloomberg School. All suffer declining lung function that can contribute to coughing, panic and death. COPD has no cure. Although smoking is a major risk factor, only one in seven smokers ends up with the disease.

Why Mabe’s parents? Why her?

Eager for answers, Mabe signed up for a research study investigating the link between genetics and COPD. More recently, she participated in a clinical trial instigated by Shyam Biswal’s research involving a new therapeutic agent: sulforaphane, a compound from broccoli sprout extract that was discovered in 1992 by Paul Talalay, MD, a professor in Pharmacology and Molecular Sciences who has a joint appointment in International Health at the Bloomberg School.

If scientists can prove in this study and successive clinical trials that sulforaphane works in people—like they previously demonstrated it worked in mice and in human cells in a dish—they will have found a potent intervention for a largely ignored public health issue affecting millions of Americans. (COPD is the No. 3 cause of death nationwide.) In addition, billions of people worldwide also might stand to benefit, mostly women and children whose lungs are chronically compromised from indoor air pollution caused by cooking fires fueled by cow dung and brush.

Sulforaphane works differently than existing anti-inflammatory agents, Biswal explains. Rather than tamp down inflammation by interfering with its various pathways, it ramps up a host’s defense system that’s been compromised by chronic inflammation. It breathes new life into Nrf2, a vital molecular player that’s effectively strangled by the “bad” inflammation that underpins not only COPD but also nearly every chronic disease imaginable.

Already, Biswal’s group has published research demonstrating in the lungs of COPD patients that a defect in the host defense results from a decrease in Nrf2.

Additionally, they have shown that sulforaphane boosts Nrf2 levels and this enhances host defense in the lungs by improving the ability of macrophages to kill bacteria and making them more responsive to anti-inflammatory drugs such as steroids.

The ongoing clinical trial in which Mabe participated is double-blind, meaning that nobody knows yet whether their purple pills are placebos or contain high or low doses of broccoli sprout extract. But Mabe has a sneaking suspicion that she ingested sulforaphane and that it helped her. During the month-long trial when she dutifully swallowed her pills daily, she felt different. “It seemed like my air passages opened up more,” she reports. “I didn’t have any flare-ups during that time, and it’s odd that I wasn’t on my nebulizer for a whole month.”

The multicenter trial, coordinated here at Johns Hopkins and taking place at Temple University and SUNY Buffalo, to date has enrolled about half of the 90 participants needed to test whether feeding sulforaphane to people alters Nrf2 activity as assessed by anti-oxidant enzymes in macrophages. Reducing inflammation is ancillary.

“If we can’t hit this target, then we’ll have to step back and say, well, this works great in mice and in the test tube, but not when people ingest sulforaphane,” says Wise, who’s heading up the broccoli sprout extract clinical trial.

This wouldn’t be the first inflammation-related wonder-compound to fall apart in a human trial. But Wise is confident in the predictive quality of Biswal’s previous research. In addition, others have shown Nrf2 was increased in the nasal tissue of people who eat broccoli sprouts.

“We think if you can show that in the nose, we can show it in the lung,” Wise says. That this strategy potentially may benefit nonsmokers who have chronic inflammation in the lungs is notable in the context of public health. Not incidentally, the WHO lists indoor air pollution from primitive household cooking fires as the leading environmental cause of death in the world.

“There’s no question that women and children in South America, Africa and South Asia are exposed for many hours a day in cooking huts to amazingly high levels of particulates from burning biofuels like cow dung, and that this leads to a condition that is akin to COPD,” Wise says. “They develop chronic cough and mucus production. They have airflow obstruction and die early.”

How their lungs are similar to or different from those compromised by tobacco-related COPD is unknown, prompting Biswal to remain hot on the Nrf2 trail.

Currently, he’s implanting particulate collected from cooking huts in India into the lungs of mice and testing how manipulations of the Nrf2 pathway affect disease outcomes. Next, he’s working on developing a breathing chamber for mice that would be analogous to the interior of a cooking hut.

“Our understanding is very weak in this area,” Biswal says, “and half the world’s population is at risk.”

If sulforaphane does, in fact, tackle COPD by boosting the defense system and rendering the immune system once again competent, complete with robust macrophages, what’s to prevent it from doing the same for those suffering from a gamut of inflammation-related diseases, including cystic fibrosis, HIV, cancer, asthma, psoriasis, sepsis, schizophrenia, atherosclerosis …?

“Nothing,” says Biswal. “Our discovery is not restricted to lung diseases only. If this pans out, it could be a big thing for public health.”

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*Johns Hopkins Public Health / Fall 2012 31*
A few months back, Sivaprakash Ramalingam, PhD, focused his microscope on a crowded clump of human stem cells and saw a reddish glow—a chemical signal he had successfully inserted a gene into a “safe harbor” site in the cells’ DNA where it wouldn’t interrupt vital functions.

It was a crucial step in a drive by the postdoc and his advisor, Environmental Health Sciences (EHS) Professor Srinivasan Chandrasegaran, PhD, to develop a practical cure for sickle cell disease. The painful and debilitating genetic illness affects millions of people around the world, including in the U.S. and some of the poorest regions of India.

Ramalingam, 34, began his life on a small plot of land near the Bay of Bengal in southeastern India. While his father toiled in the family’s banana grove, sugar cane field and rice paddy, young Siva helped out by milking the family cow.

Today, Ramalingam works with his mentor Chandrasegaran—also a native of the southern Indian state of Tamil Nadu—on the frontiers of genetic medicine, trying to find gene-based cures for major health challenges like cystic fibrosis and HIV, as well as sickle cell disease. In their common quest, it’s hard not to see a torch passing from one generation of
scientists to the next, from basic science to applied medical research and from Western institutions to young researchers from the rapidly advancing scientific institutions of the developing world.

The two public health scientists are collaborating with stem cell expert Curt I. Civin, MD, of the University of Maryland, on the sickle cell project. They are racing with labs around the world pursuing similar goals. In May, the Maryland Stem Cell Research Fund awarded Ramalingam one of 17 grants worth up to $200,000 over the next two years for his sickle cell work, as part of a program to support Maryland scientists pursuing novel approaches to stem cell therapies.

Ramalingam and Chandrasegaran say the painstaking research could take two or three more years before it is ready for testing in animals, in preparation for human trials. Asked whether they worried about the intense competition, Ramalingam admits that he sometimes loses sleep over the publication of an important paper by a rival lab.

Chandrasegaran just smiles. The 30-year veteran scientist, who pioneered the development of man-made gene-editing tools called zinc finger nucleases, takes a philosophical approach. “If you’re asking me, do you want to be first? Yes. But it’s not in our hands. If others do it, we will be happy that it was done since it will help a lot of people,” he says.

There is no guarantee of success in this latest assault on the scourge of sickle cell. Except for mice and yeast, the DNA of most animals, including humans, is notoriously difficult to fiddle with and many efforts to repair human genes have failed.

Some early gene therapy patients died when viruses carrying engineered DNA inserted it at random locations on the genome and switched on genes that caused cancer.

Chandrasegaran says he’s leery of overselling his lab’s progress. “I want to keep it low-key,” he says. “Let’s take it one step at a time, and do careful science.”

But for Chandrasegaran, as for many other scientists, the relatively recent discovery that stem cells can be “induced” or derived from adult cells has opened exciting new avenues for medical research. “I hope that I can see it in my lifetime,” he says. “I’d like to see people cured of HIV, cured of sickle cell—any monogenic disease where you can replace the cells. It will help a lot of people, and that’s the ultimate goal.”

**Death’s Crescent**

Sickle cell is among the most common disorders caused by a single genetic defect, and it can be devastating.

People with the disease produce crescent-shaped red blood cells that are stiff, sticky and prone to piling up or breaking apart, clogging small vessels. These misshapen cells only live about one-tenth as long as normal blood cells, and a patient’s bone marrow can’t make replacements fast enough to keep delivering sufficient oxygen to the body.

Clogged vessels often trigger attacks, called “crises,” that produce acute pain in the back, chest, arms or legs and can last for hours or days. Patients may suffer leg ulcers, small strokes, blindness, and kidney failure and be prone to lethal infections.

The disease is found in certain populations around the world but is most common in Africa, parts of the Middle East, India, Central America and the Caribbean. It affects an estimated 90,000 to 100,000 people in the U.S., including about 1 in 500 African Americans.

The mutation that causes the disease is thought to have evolved in the tropics. For those carriers of a single gene (said to have “sickle cell trait”), most do not have symptoms of sickle cell disease but do have some protection against malaria.

In the U.S., the universal screening of newborns and early, aggressive treatment of sickle cell disease with blood transfusions, antibiotics and other drugs have helped reduce infant mortality and prolong lives. But the disease can still have a devastating impact on patients: Life expectancy in the U.S. for women with the disorder is still only about 48 years. For men, it’s 42.

In recent years, doctors have cured sickle cell disease in a few hundred patients using a technique that combines stem cells from healthy donors with bone marrow transplants. But the procedure is expensive and risky, Chandrasegaran says, while matching patients with healthy donors can be very difficult.

So Ramalingam and Chandrasegaran, working with Civin, are trying a different approach. Instead of using donors, they plan to take a sickle cell patient’s own stem cells, repair the faulty gene, and turn the repaired stem cells into blood and blood-producing...
cells. The hope is that these healthy cells, put back in the body, will outlast and replace the diseased ones without the need for a bone marrow transplant.

The aim is to make the repair of a patient’s sickle cell gene safer, simpler and cheaper, putting the procedure within the reach of more patients. “We’d like everybody to have access to it, so we want to make it as inexpensive as possible,” says Chandrasegaran.

If the technique works, the senior researcher says it could have wide applications. The biotech company Sangamo Biosciences of California has licensed some of Chandrasegaran’s work and is using a similar strategy to knock out a gene known as CCR5 with zinc finger nucleases, eliminating a route HIV uses to invade and hijack the body’s immune system.

The Mentor Chain
When he was still in India, Ramalingam, who earned his doctorate in molecular biology from the University of Madras, studied strategies for boosting the iron content of rice through manipulating the crop’s genes.

That’s how he heard about Chandrasegaran’s groundbreaking work on zinc finger nucleases, called ZFNs, as a tool for tweaking DNA. “Chandra was the expert,” the younger scientist says. “I sent him my CV and wrote that I was interested. I was very fortunate to work with him.” He came to Hopkins in 2008 as a postdoc to work with Chandrasegaran in EHS.

One advantage of ZFNs and similar gene-editing technologies, Chandrasegaran says, is that, made carefully, they can be targeted at one and only one point in the genome, avoiding the damage that can be caused by random insertion. (Sickle cell disease is caused by an error in a single chemical “letter” in the 3.2-billion-letter-long library of human DNA.)

But making these precise tools for cutting and editing DNA isn’t always simple. Ramalingam says he probably faces another two or three years of working on this crucial phase of the effort. “The success rate is very, very low here,” he says. “So you need a lot of patience doing this research.”

The postdoc, who is married with a 10-month-old son, says he was very proud when his parents traveled the 7,000 miles from his tiny home village of Kullampalayam to visit the Baltimore lab. “They were very excited, they were very happy,” he says. “I tried to explain it to them and the basics, they understand. But the technology, they may not yet.”

While the sickle cell project could accelerate Ramalingam’s career, his senior partner in the lab is considering retiring after three decades at the School.

Chandrasegaran, who grew up as one of 10 children, is the son of a customs official working in what was then the French colonial city of Pondicherry on the Bay of Bengal. Accepted to an elite state-run military secondary school, Chandrasegaran rose to the rank of house captain, excelled at physics and graduated with honors. “All my friends who right now are in India? They’re generals and air marshals,” he says.

But he decided to become a scientist rather than an officer, earning a degree in chemistry from the University of Madras in India and his doctorate from Georgetown. He came to the School as a postdoc in late 1981 and joined the faculty in 1986.

At Johns Hopkins, Chandrasegaran learned molecular biology at the bench of Hamilton Smith, professor emeritus at the School of Medicine and a key scientific strategist for a private company that published a working draft of the human genome in 2001. Smith shared the 1978 Nobel in physiology or medicine with Hopkins’ Dan Nathans and a Swiss scientist, Werner Arber, for the discovery of restriction enzymes, the first chemical tools for editing DNA.

It was Smith, in fact, who suggested that Chandrasegaran pursue the synthesis for new gene-editing tools. That suggestion eventually led to Chandrasegaran’s groundbreaking work on ZFNs—technology that, Smith notes, “is now leading to discoveries of several new ways to cleave DNA in site-specific fashion without using restriction enzymes. It’s a hot new field with implications for gene therapy and genome engineering.”


Ramalingam, in turn, says Chandrasegaran has inspired him by spending long hours in the lab and generously sharing his skills. “Whenever I end up with some problems, I discuss them with him,” he says. “He’s a great advisor to me.”

“Whenever I end up with problems, I discuss them with [Chandra]. He’s a great advisor.” —Sivaprakash Ramalingam
On a spring-like Saturday morning in February of last year, Jeanette Walke drove her silver Honda Civic northwest on University Parkway near Johns Hopkins University’s Homewood campus and made a right turn across a bicycle lane into the driveway of her apartment house. Police say she cut off 20-year-old Nathan Krasнопoler—science fiction fan, chess player, enthusiastic amateur cook and Hopkins computer science student—who was carrying a bag of produce home from the Waverly Farmers Market on his Trek bicycle. A police reconstruction of the accident said Krasнопoler swerved, collided with Walke’s car and was thrown in front of it, trapping him underneath. Badly injured and apparently unable to breathe, he was caught between the searing heat of the engine and the pavement. He was still wearing his bike helmet, according to police, but his lungs had collapsed. His broken glasses were found at the scene.

Walke, then 83 years old, climbed out and sat on a low wall as passers-by gathered. A witness told police she held her purse on her lap and seemed to be staring into space until someone asked her to switch off the engine. “I started to turn into the alley, then I heard a crunch like metal crumbling,” Walke later told police investigators. “Then I
saw a limb like an arm and then I saw a head and I stopped and realized that the person was under my car.” By the time Baltimore firefighters managed to pull Krasnopoler out, he had a broken collarbone, fractured ribs, two collapsed lungs and severe burns to the face. He suffered extensive brain damage from a lack of oxygen and died six months later.

While most media reports emphasized Walke’s age—“Elderly Woman Ticketed in Crash with Hopkins Bicyclist” was a typical headline—Walke told police she was in good overall health. She reported having had glaucoma surgery in 2009 in both eyes, but told police she had visited the ophthalmologist the previous month and was given “a good report.” Walke could not be reached for comment, but her attorney says he did not believe her age played any role in the incident.

Still, the tragic death of Nathan Krasnopoler bore some of the hallmarks of collisions involving older motorists. Walke, who was charged with negligent driving, told police she looked but didn’t see Krasnopoler riding in the bike lane on her right as she approached her driveway. “I kept checking,” she said, according to the police investigation. Experts say that drivers older than 80 or so who are involved in collisions are more likely to report never having seen the other vehicle.

In America and affluent societies around the world, driving has come to be regarded not just as a symbol of youth and independence, but perceived as a basic human right. Giving it up can be hard. If we live long enough, most of us will face increasing mental and physical problems that can affect our ability to drive. Yet many older drivers with declining skills fiercely resist giving up their licenses. Meanwhile some studies suggest that giving up driving can increase social isolation, raise the risk of depression and restrict access to health care—though these problems may be aggravated by other age-related health issues.

Researchers are seeking ways to help keep older people behind the wheel for as long as they can drive safely and to prepare them to call it quits if they can no longer do so. The goal: Help governments, families and society improve road safety while respecting the rights of older citizens.

Answers, however, have been elusive. “The evidence is really just not there yet on what policies and programs are most effective, and much more needs to be done in the area of older driver research,” says Andrea Gielen, ScD ’89, ScM ’79, director of the Center for Injury Research and Policy (CIRP).

States grappling with the issue have no clear path ahead, says John Kuo, administrator of Maryland’s Motor Vehicle Administration and the governor’s highway safety coordinator. “There’s no norm or best practice that’s surfacing. This is a national dilemma,” says Kuo. “We must develop a strategy that meets their needs and keeps them safely on our roadways.”

Last year saw the first of the baby boomers turning 65. Older Americans are now the fastest-growing segment of the driving population. Today, about one in seven motorists is age 65 or over. By 2025, that figure will be one in four.

In many ways, older people make ideal drivers, says Vanya Jones, PhD ’06, MPH, an assistant professor in Health, Behavior and Society and a CIRP faculty member. “They don’t drink and drive. They wear their seat belts and tend to stay within the speed limit. They do the good stuff,” says Jones who is a passionate advocate for the elderly. The problem, she and other researchers
say, comes when age-related cognitive and physical changes start to affect the complex task of threading a one- or two-ton vehicle through a maze of moving traffic.

Jones is keenly aware of the impact that quitting driving can have on the elderly. She vividly remembers the day her grandfather reached his own painful decision to stop driving. “For me, as a child, he was sort of this larger than life man,” she says. “When he gave up his car, that was one of the few times in his life that I saw him cry.”

Later, while in college in Ohio, she was standing at an intersection when a car hit a pedestrian in front of her. She saw how a life could have been saved if the light had changed a few seconds later or if the driver or pedestrian had slightly altered their behavior. “Personal injuries and motor vehicle crashes are a huge, huge issue for me personally,” she says, saying her concern led to her work with colleagues at CIRP.

Some elderly drivers are as good or better than the average middle-aged motorist. But as we age, researchers say, we experience a gradual erosion of our vision, hearing, response time, mobility, strength and coordination, cognition and judgment. We can also develop a host of age-related illnesses, from glaucoma to diabetes to dementia. To treat what ails us, we may take an array of drugs that separately or in combination can cloud our judgment or slow our reflexes. These changes come sooner for some and later for others, but whenever they come they affect our ability to drive.

“I think there’s often a natural progression as we age,” says Jones, adding that the problem of declining driving skills is “one that we all will probably have to face if we live to be old enough.”

Teenagers and young adults have the worst crash statistics, victims of a cocktail of immaturity and inexperience. But as they spend time behind the wheel, their crash rates go down. Starting around age 75 or so, the process reverses gears, and fatal motor vehicle crashes involving elderly drivers begin to rise sharply. According to the Insurance Institute for Highway Safety, the crash rate per mile for drivers 85 and older is roughly the same as for teenagers. The rate of fatal collisions per mile traveled is close to double that for teens.

Given the physical effects of age, no one is in greater peril in a crash than an older motorist. Someone 80 years of age or older is six times more likely to die in a collision than...
someone 35 to 54 years old. Kuo points out that, nationally, while drivers 65 and over rack up just 8 percent of miles traveled, they account for 17 percent of traffic fatalities.

Because of this increased risk of injury-related deaths, Jones says, researchers and traffic safety experts need to find strategies to reduce collisions involving older adults. “You don’t want to be injured in a crash and you don’t want to injure someone else,” she says. “These are really terrifying things.”

The graying of America’s driving population seldom draws much attention until a high-profile tragedy strikes, like the case of George Weller, who in 2003 at age 86 killed 10 and injured 70 when his car barreled through a farmers market in California. In Texas, there were calls for tougher licensing regulations for the elderly after 90-year-old Elizabeth Grimes ran a red light in Dallas in 2006 and slammed into a car driven by 17-year-old student Katie Bolka, who died of her injuries. Likewise, the Krasnopolier case has inspired a call for Maryland to review its policies affecting older drivers.

A recent report by the Trust for America’s Health found that 33 states and Washington, D.C., had some limits for mature drivers, including required vision tests, shorter times between license renewals and limits on online or mailed renewals. That means about a third of states have no such requirements. Some safety activists want to see more restrictive laws on licensing older drivers, including mandatory age-related screening exams or road tests.

Following Nathan’s death in 2011, his grieving parents—lawyer Susan Cohen, an assistant attorney general for Maryland at the time, and her husband, engineer Mitchell Krasnopolier—launched a campaign to advocate new licensing rules. In response to public concern, the state Legislature has directed the MVA to conduct a two-year study of older drivers.

According to the Foundation for Traffic Safety at the American Automobile Association (AAA), Maryland requires drivers over age 40 who are renewing their license by mail to submit a report from a vision specialist and requires new drivers over age 70 to provide a medical report. The Krasnopoliers want to go further and require drivers, as they age, to take routine cognitive screening exams that may help spot high-risk motorists before they have catastrophic crashes. But state governments are reluctant.

**Carl Thistel, 80**

I’ve become more gentle in my driving, not like an old person but like a prudent person. That’s hard; I still have this 17-year-old inside me that likes to get behind the wheel and really see what this sucker will do. I don’t do that anymore.
to demand additional testing for seniors until there is more data showing that these tests work. The Trust for America’s Health, in a May 2012 report, warned against passage of “reactive, unscientific legislation that overly restricts the driving privileges of older drivers.”

The American Association of Retired Persons (AARP) supports tightened testing policies and prelicense screening exams, but not if they’re required based on age. “The only screening method that has been identified that helps reduce crashes among older people is in-person license renewal, and AARP supports in-person renewal across the life span,” says Nancy Thompson, an AARP spokeswoman. “The issue about driving is about health, not age.”

Most older drivers now do what safety experts call “self-regulate,” limiting their driving to match their skills. Edward Ryan, an 89-year-old former engineer at Fort Meade, Maryland, avoids busy streets, the Baltimore Beltway and driving at night. He enjoys short hauls to nearby shopping centers but has no interest in driving on longer trips or in heavy traffic. “I don’t think I miss it, to tell the truth,” he says.

One of Vanya Jones’ chief goals is to find ways to encourage drivers to fine-tune their driving to match their skills and help them prepare to stop driving altogether if the time comes when they are no longer safe on the roads. “We are trying to help adults plan to retire from driving in the same way they would plan to retire from their jobs or change their housing,” she says.

Persuading drivers to take a hard look at their own abilities is not necessarily a straightforward matter.

In a study published in the Journal of Applied Gerontology in December, Jones and a team of researchers from CIRP, the Maryland Highway Safety Office, the Maryland MVA and others administered three standard computer-based cognitive and physical screening exams to 67 older Baltimore County motorists in a laboratory setting. Nine of the drivers, or about 13 percent, were unable to complete or failed two or more of the screening tests and were judged to be at high risk for a crash. (Another 20 were ranked as medium risk because they couldn’t successfully complete one of the screening tests.)

As a group, the nine older drivers judged at high risk had the most trouble with the test that measures the ability to process and sort information.

One of the goals in the study was to see how the high-risk group reacted to being told test results indicated they had a driving-related impairment and should seek medical advice. Jones and her colleagues wanted to learn what participants did with the information, if they would accept the results and seek medical advice or voluntarily stop driving.

From the public health perspective, the results were not encouraging. Of the four drivers who later agreed to in-depth interviews, all said they were uncomfortable with at least one aspect of the testing experience. One told researchers: “Trying to search for a proper word. Disappointed, I guess. Disappointed and [pause] I couldn’t understand why I failed because everybody tells me I’m a good driver.”

Importantly, none of the four who failed the tests disclosed the results to a
physician, and only one surrendered his or her license. The one participant who voluntarily gave up driving said: “I think probably subconsciously it was the reason I gave up my car, because I realized that my reflexes were not as good as they were.”

Despite the small sample, Jones says the study demonstrates how difficult it is to deliver unwelcome news to older drivers in a way that encourages them to act. But she wasn’t surprised, because of the importance of driving to many older people.

When it comes to competency behind the wheel, gerontologists say that chronological age isn’t as important as what is called “functional age.” Steven Gambert, MD, director of Geriatric Medicine at the University of Maryland Medical Center and R Adams Cowley Shock Trauma Center and an authority on the mental and physical effects of aging, recalls testing a former military pilot in his 60s who, as a younger man, had landed a crippled plane armed with a nuclear weapon on the deck of an aircraft carrier. The man’s exceptional skills seemed unaffected by his age. “This guy had superhuman hearing and reflexes,” says Gambert. “He tested off the charts. We couldn’t believe it.”

While many older motorists are highly skilled, Gambert says, others experience a sharp decline starting around age 80. At Shock Trauma, he all too frequently deals with the tragic results.

While Gambert describes himself as an advocate for the elderly, he says that perhaps drivers at a very advanced age, starting in their mid-80s, should be subject to screening that goes beyond an eye test. Drivers with medical conditions or a record of accidents that raises concerns, he says, may need screening earlier. “The reality is, the older you get, probably you’ll get to the point where you’ll need a driver’s assessment,” he says.
Some drivers stay on the road long past the time when they should no longer be behind the wheel. When George W. Rebok, PhD, a professor in Mental Health, was a postdoc studying dementia patients at Hopkins in the 1980s, he discovered that some of his study subjects were driving guided by directions shouted at them by their passengers. Others manipulated the pedals while their spouses steered.

Rebok’s father, Jack Rebok, a retired nuclear power plant engineer, fiercely resisted surrendering his car keys after developing Parkinson’s disease in his early 80s. His family took away his keys, but he had extras hidden around the house. When the family disabled Jack’s beloved Plymouth sedan, a buddy helped him fix it. After Jack Rebok’s doctor reported his declining skills to the state, as required in Pennsylvania, Jack flunked the driver’s test three times and lost his license. But when George saw that his father had visited the barber several miles away, Jack admitted he was driving without a license.

Finally, Jack’s family hid the car and told him it was in the shop for repairs.

While acknowledging such experiences, public health researchers say it is also important to try to keep competent older drivers on the road. Studies have shown that those who stop driving are five times more likely to enter long-term-care facilities and those who stop driving are five times more likely to die within three years. In part, Rebok says, that’s probably because many drivers quit as their health declines. But he also says that the depression, isolation and loss of control that come with giving up driving may—by themselves—cause health problems. In a 2009 study of 690 current and former drivers published in the Journal of Gerontology, Rebok and other researchers found that at the point older motorists quit driving, they reported a sharp, immediate drop in their physical functioning, social activities and general health. Quitting also accelerated the rate at which their health was declining.

Experts say research is needed into the relationship between giving up driving and household activities, including studies to identify and test coping strategies.

When it comes to cognitive problems, some researchers say new training programs may be able to help older drivers stay safer. AARP and AAA offer behind-the-wheel courses designed to help older drivers sharpen their skills. Several commercial firms have produced so-called “brain-training” programs designed to improve driver performance.

In a widely cited study published in Nature in 2010, one team of researchers concluded that thousands of volunteers ages 18 to 60 who played brain-training games online for six weeks did not improve their overall memory or reasoning. Instead, the study found, they improved their skill at taking a particular test.

But Rebok and others believe that an intensive cognitive training program can produce changes that carry over into real life. He is part of a team of researchers participating in a large, long-term, multicenter study called ACTIVE (Advanced Cognitive Training for Independent and Vital Elderly), which in 2006 reported finding evidence that a 10-week program could improve memory, reasoning and speed of mental processing in older adults. In the study of 2,832 volunteers, participants on average reported improvements in their performance of everyday tasks, including driving, that with booster sessions persisted for up to five years after the training ended. The ACTIVE study’s 10-year follow-up was completed last year and the results have not been published, but Rebok says that the training program had a significant impact on cognitive fitness through at least five years.

“I think the results we were getting with the speed of processing in particular shows a lot of promise in terms of extending driver life spans, letting people stay on the road longer and more safely, and shows evidence of actually reducing crashes,” he says. On the other hand, Rebok says, the severely cognitively impaired may reach a point where “there may not be much you can do to bring [them] back to where they can safely operate a motor vehicle.”

A year after Nathan Krasnopolser’s death, the “ghost bike” that his family bought for $30 and painted white still sits chained to a signpost under an elm tree on the sidewalk a few steps from the driveway where he was injured. Walke, now 84, did not respond to a request for an interview, but her lawyer, Robert H. Bouse Jr., says she was deeply affected by Nathan’s death. “It devastated her, it truly did,” he says.

Walke was cited for negligent driving and failing to yield the right of way to a rider in a bike lane, court records show. She pleaded guilty and was fined $220. The Krasnopolser family filed a $10 million lawsuit that Walke settled for what the Krasnopolser’s lawyer called a “substantial” sum. The family says they took legal action only after they learned Walke had continued to drive after the accident, and they insisted she surrender her license as part of the settlement.

Now Cohen has left her job with the state Attorney General’s office and plans to use the money from the lawsuit to set up a nonprofit foundation called Safe Roads USA. Cohen says she will dedicate the rest of her life to an effort to promote research, education and legislation to address the problem of older motorists and traffic safety. “I plan to go for laws across this nation,” she says.
Lawrence J. Appel, MD, MPH ’89, professor, Medicine, Epidemiology and International Health (IH), and director of the Welch Center for Prevention, Epidemiology and Clinical Research, received the National Award for Career Achievement and Contribution to Clinical and Translational Science.

Four Johns Hopkins Center for Injury Research and Policy (CIRP) faculty are among 20 injury prevention leaders honored by the CDC National Center for Injury Prevention and Control as part of its 20th anniversary celebration:

- Susan P. Baker, MPH ’68, professor, Health Policy and Management (HPM), and CIRP founding director;
- Jacquelyn Campbell, PhD, MSN, Anna D. Wolf Chair and Professor in Nursing, Andrea Gielen, ScD ’89, ScM ’79, professor, Health, Behavior and Society (HBS), and CIRP director; and
- Ellen MacKenzie, PhD ’79, Fred and Julie Soper Professor and Chair, HPM, and past CIRP director.

The first recipient of the endowed Susan P. Baker and Stephen P. Teret Chair in Violence Prevention at the University of California at Davis is Garen J. Wintemute, MD, MPH ’83. Baker and Teret, MPH ’79, JD, professor, HPM, were mentors to Wintemute.

Abdullah Baqui, DrPH ’90, MPH ‘85, professor, IH, was honored by the Bangladesh Medical Association of North America for his outstanding contribution to clinical research in Bangladesh. He also received the 2012 CORE Group’s Dory Storms Child Survival Recognition Award.

Eric B. Bass, MD, MPH, professor, HPM, Epidemiology and Medicine, was named president-elect of the Society of General Internal Medicine.

Lee Bone, MPH ’77, RN, associate professor, HBS, is the inaugural recipient of the Crenson-Hertz Award for Community-Based Learning and Participatory Research.

Bol, a film co-produced by the Center for Communication Programs under the USAID-funded Pakistan Initiative for Mothers and Newborns project, won Best Film, Best Female Actor and Best New Talent at the 2012 Asian Film Festival in London.

Marie Diener-West, PhD ’84, Helen Abbey and Margaret Merrell Professor of Biostatistics Education and chair of the MPH Program, was inducted as a Fellow of the Society for Clinical Trials.

Mary Fox, PhD ’01, MPH, assistant professor, HPM, has been selected to serve on the U.S. Environmental Protection Agency’s Science Advisory Board Ad-hoc Perchlorate Advisory Panel.

Shannon Frattaroli, PhD ’99, MPH ’94, assistant professor, HPM, was appointed to the Baltimore City Board of Fire Commissioners.

Robert H. Gilman, MD, professor, IH, was named an honorary professor at the Universidad Catolica in Santa Cruz, Bolivia.

Diane Griffin, MD, PhD, Alfred and Jill Sommer Professor and Chair, W. Harry Feinstone Department of Molecular Microbiology and Immunology, was elected to the Council of the Institute of Medicine.

John Groopman, PhD, Anna M. Baetjer Professor of Environmental Health, Environmental Health Sciences (EHS), was appointed by the U.S. Secretary of Defense to
Some people argue that the money expended on these conferences would be better spent on antiretroviral drugs or condoms. That is a very real argument. We don’t think we’ll have another conference this large. With social media and the Web, we feel we can have an ever-expanding audience for the science without necessarily having everybody physically together. That said, AIDS is unique in being a truly global pandemic. It is unique in requiring responses across sectors. We need the politicians, the researchers, people from the infected communities ... . As a global movement, we need to come together and come to consensus on what the goals are.

What will be your priorities when you lead IAS, starting in 2014?
My thinking is evolving. Certainly what our work has really been known for is highlighting the key populations most affected by HIV and their unmet needs for prevention, treatment and care. I think that very much will be a theme that I will bring to this.

How is CFAR going to change AIDS research at Hopkins?
It’s already starting to have an impact. We’re going to make the first round of developmental awards this year ... to support junior investigators with new ideas and hopefully bring some more senior investigators new to HIV into the field. We have been asked to take the lead in the CFAR African research network. [Africa is] obviously the center of the pandemic. It’s also a critical place for the next phases of research. [And] one of our aims is addressing the epidemic in Baltimore. We have already formed a community participatory board. We really want the community to be a partner.

What’s your leadership style?
If you stay really focused on the science, on the public health issues, the human rights issues, on the real concerns of real people, then other people are happy to participate. And you create a space where junior people feel they’re a part of something that really matters. Then they work enormously hard, and you build a team that can punch above its weight. ♦

the Defense Health Board, a federal advisory committee.

The Institute for Global Tobacco Control received the 2011 All-Star Award from Constant Contact, Inc., for exemplary communications results.

Rafael Irizarry, PhD, professor, Biostatistics, was chosen as the 2012 Myrto Lefkopoulou Distinguished Lecturer at the Harvard School of Public Health.

Ruth Karron, MD, professor, IH, was appointed to the Advisory Committee on Immunization Practices of the CDC by the U.S. Department of Health and Human Services Secretary.

Jonathan Links, PhD ’83, professor, EHS, received the 2012 Ernest L. Stedman Faculty Award.

Paul Locke, DrPH, MPH, JD, associate professor, EHS, was appointed to the National Research Council committee, Lessons Learned from the Fukushima Nuclear Accident for Improving Safety and Security of U.S. Nuclear Plants.

Jill Marsteller, PhD, MPP, associate professor, HPM, and Lainie Rutkow, PhD ’09, MPH ’05, JD, assistant professor, HPM, received Advising, Mentoring and Teaching Recognition Awards (AMTRA).

Cindy Parker, MD, MPH ’00, associate professor, EHS, was appointed to the Baltimore Commission on Sustainability.

Keshia Pollack, PhD ’06, MPH, associate professor, HPM, was awarded the Mid-Career Outstanding Service Award by the Injury Control Emergency Health Services Group.

Elizabeth Selvin, PhD ’04, MPH, associate professor, Epidemiology, was elected a Fellow of the American Heart Association. She also received an AMTRA.

Moyses Szklo, MD, DrPH, MPH, professor, Epidemiology and Medicine, was elected to the Brazilian Academy of Sciences.

Roland J. Thorpe Jr., PhD, MS, associate scientist, HPM, has been named a Fellow of the Gerontological Society of America.

Zhibin Wang, PhD, assistant professor, EHS, was named to the 2012 Kimmel Scholar Program by the Sidney Kimmel Foundation for Cancer Research.

Daniel Webster, ScD ’91, professor, HPM, has been appointed to the Institute of Medicine’s Planning Committee for the Evidentiary Base for Violence Prevention Across the Lifespan and Around the World Workshop.

Albert Wu, MD, MPH, professor, HPM, and director of the Center for Health Outcomes Research, has been selected to serve on the National Quality Forum’s Patient-Reported Outcomes Expert Panel.

A SOURCE for Service
Five JHSPH faculty recently were selected as SOURCE (Student Outreach Resource Center) Service-Learning Faculty Fellows and received awards to implement service-learning into academic courses: Daniela Lewy, MPH ’06, research associate, IH; Vanya Jones, PhD ’06, MPH, assistant professor, HBS; Roni Neff, PhD ’06, MS, assistant scientist, EHS; Beth Resnick, MPH ’95, CPH, assistant scientist, HPM; and Carey Borkoski, PhD, MA, instructor, HPM.
a neighborhood cleanup. Regardless of how HIV got into his body, he still had to deal
with it. He was nearly driven to suicide by the isolation he felt, but Vinson rallied,
thanks in large measure to being invited to a gathering of positive-status youth in Denver
by Melody Lynch, an outreach coordinator at Hopkins’ Harriet Lane clinic.

There, Vinson discovered that his status wasn’t a reason to be distraught, that all around him were hundreds of vital young people who could even joke about their status while taking care of themselves. At a talent show there, he read a poem he’d written. It would change his life. The poem, which chronicled his experiences post-diagnosis, won him national recognition and led to a book of poems on lifestyle and HIV titled *Positively Me*.

Vinson, who was part of the club scene when he first came out, recalls that HIV hung
over the crowd like a specter, acknowledged but unspoken. “We were aware, but we
didn’t talk about it,” says Vinson, now 22. “And if people were positive, many weren’t
sick of you targeting the MSM community, perhaps looking for other HIV at-risk groups more willing to be tested. “In this city, with this kind of [MSM] work, a lot of people aren’t really dedicated, in my opinion. The last organization I worked at … I was told ‘I’m sick of you targeting the MSM community,’ even though MSMs have the highest risk.”

Taken as a group, these young MSM advocates hit on some common themes: Show commitment. Build trust. Have compassion. Give us professionals who can relate to our world.

It is a message folks such as Danielle German, Karin Tobin, Carl Latkin and Anthony Morgan are hearing loud and clear. By identifying and engaging young, well-connected advocates—think of it as finding the tipping point for MSM at risk for HIV—Karin Tobin says the prevention message can spread organically, from within the community. “We are all embedded within naturally occurring social networks; we are not only influenced by everybody we know, but we are influencing them,” says Tobin. “And so if we can train anybody within this network to educate others, they’re going to influence somebody else; it may well have a broader impact.”

suddenly turn serious and warn the audience that he’s about to show them what someone with HIV looks like. “I’ll say, ‘Now, if your stomach is weak you should probably leave.’” Then Vinson hits the button, and there’s his handsome, smiling, healthy-looking face, eating an ice cream cone. “Cracks ‘em up every time,” he laughs.

Keith Holt has used his grin to reach at-risk MSM as well. It can be seen on posters for the campaign the 26-year-old helped come up with, “Have Balls, Get Tested,” a Baltimore City Health Department outreach effort aimed at the city’s ballroom community, of which Holt is a part.

To Holt, the idea of a community-gathering spot in Baltimore for young gay
African-Americans is a key toward building a safer, healthier community. “When I came
out at 17, I realized right then there was no support, no place for me to go. There was
Project Olympus, part of HERO [the Health Education Resource Organization, which
lost its funding]. I’d like to start something like that again. Some place people know they
could come, chill, afterward put on some music and vogue, get to know your peers,
and talk about what you’re going through. It’s amazing there’s no place like that here
now. You know, the world is coming along in accepting people and their sexuality. I would
think the support here would have increased versus decreased.”

For Adrian Ross, activism was triggered by simple observation. “I saw people I was
hanging out with all coming up positive. A dear friend became positive, and he
never knew until he had full-blown AIDS. It changed my life, and got me out in the
community to get people tested,” says Ross. He’s also been shocked to find that
organizations he’s worked with have turned their back on the MSM community, perhaps
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connected advocates—think of it as finding the tipping point for MSM at risk for HIV—
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community to get people tested,” says Ross. They

you not to get people pregnant. It should be
mandatory that you take ‘HIV 101’ in high
school.”

Ross has found his message is best
conveyed through Facebook, where he receives at least two or three messages a
week from scared youth seeking education, support and testing information. Ross,
who has worked for several Baltimore HIV initiatives, says that many organizations are
out of touch with how youth truly interact with social media. “They need to change or
they won’t reach who they want to target,” he says. He’s also been shocked to find that
some federally funded city HIV-testing programs he’s worked with have turned their
back on the MSM community, perhaps

looking for other HIV at-risk groups more
willing to be tested. “In this city, with this
kind of [MSM] work, a lot of people aren’t
really dedicated, in my opinion. The last
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Continued from page 21
Driving for Life

Vanya Jones is an engaging, exuberant lecturer. The assistant professor of Health, Behavior and Society leavens theory-heavy discussions in courses like Program Planning for Health Behavior Change with examples from her research. Last January, I found myself in a Hampton House classroom, taking the program planning class as part of my MPH coursework. At one point, possibly while I was grappling with the slippery nuances of the Integrated Behavior Model, she mentioned that one of her projects focused on older drivers and safety issues.

I immediately wrote “older drivers” in my notes and circled it a few times. I knew there was a story there.

My father lived it. He was a great driver for more than six decades. A veteran Air Force pilot, he had an intuitive appreciation of safety. In his prime, he could fly 7 tons of metal at more than 600 miles per hour and bring it all home safely. Into his 60s, he piloted a Cessna 172 and other planes across the country. He was a natural pilot, with a keen sense of direction, astonishing mathematical skills and a smooth, sure touch at the controls.

All of those skills made him a safe, reliable driver. On car trips when I was a kid, he would say things like, “We’ll be there in 42 minutes.” And usually, he was right. Late into his retirement years, however, things began to change. He began having problems with his vision, hearing, reaction times and memory. (He would later be diagnosed with Alzheimer’s.) He became something unthinkable: a dangerous driver.

He held fiercely to driving. When polite suggestions and blunt reasoning failed, we turned to his physician. We gave the doc the facts and asked him to tell my dad to stop driving. A good military man to the end, dad respected authority and obeyed.

It was a hard time for my family, recognizing that age had imposed unforgiving limits on our father. Our consolation was in knowing that he wouldn’t hurt himself or anyone else while driving.

As writer Douglas Birch explains in the story on page 36, we live in an aging society and this issue will only become more critical in the coming years. We can only hope that research by Vanya Jones and others will lead us to better solutions.

BRIAN W. SIMPSON
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Letters to the Editor

A Father, a Daughter and Autism
I was very touched by this piece [“Open Mike,” Spring 2012]. Your message inspired me to connect with others about this issue and understand that we are a community working together as colleagues, friends and resource advocates to educate ourselves and others about pressing public health issues. Thanks for leading the way, Dr. Klag, by sharing this heartfelt message. It makes a positive difference, as you do for us.

Gail Wallace, PhD
University of Alabama at Birmingham
via Magazine Comments

Thank you, Dean Klag, for your editorial. It was interesting and moving and got me started thinking about children with special needs. I wish more people had the guts to share a personal story to call attention to a public health issue.

Magnus Borres, MD, PhD, MPH ’84
Medical Director, Immunodiagnostics
ThermoFisher Scientific
Uppsala, Sweden

Building Mentally Healthy Countries
I am delighted with your article on global mental health [“A Global Call for Mental Help,” Spring 2012]. Most nations have no information concerning the economic costs of NOT providing mental health programs. Ideally, nations would allocate 1 to 2 percent of their budget to mental health.

I would also like to see an educational program for mental health professionals on integrating mental health services and primary health care. We provided a one-week seminar to mental health professionals in Panama, using Skype. Similar programs can help other nations integrate mental health and primary care services, based on a country’s unique needs, resources and culture.

Leonard Feinberg, PhD
UN Representative
Faculty, W.A. White Institute
Associate Professor Emeritus, Iona College
Greenwich, Connecticut

A Silent Epidemic
Unless and until we find a way to speak about child sexual abuse (CSA)—without shying away or talking about “monsters,” as the article [“Reason versus Rage,” Spring 2012] points out—we will not be able to adequately make advances in prevention and treatment. A plethora of information about both CSA prevention and treatment is available. Unfortunately, nearly everyone is working without adequate resources to step up programming. As a more-than-concerned citizen, as a mother and as the founder of Stop the Silence: Stop Child Sexual Abuse (www.stopcsa.org), I implore the public to get involved, despite their discomfort, and ask policymakers to put the resources in place to address CSA as the public health epidemic—indeed, pandemic—that it is.

Pamela Pine, PhD
Glenn Dale, Maryland
via Magazine Comments

Enthralled? Appalled? Send us your comments: editor@jhsph.edu.
Wheels imply freedom. That freedom is not easily relinquished by aging drivers, no matter whether those wheels are affixed to a bicycle in Afghanistan, an electric motorbike in China or a car in the U.S. (See page 36.)

Photos:
above/Shehzad Noorani; below/Xu Haitong
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