FEATURES

188 Editorial: Ink and LED
Stanley Maloy

201 Antibiotic Resistance Spreads through Diverse Species and Habitats, Part I
Shannon Weiman
The public health threat amplifies as drug-resistant pathogens move freely through various environments and species

209 PCR for Everything—Seeking Value in Speed
Bert K. Lopansri
With faster and more comprehensive diagnostic tests becoming widely available, we need to think more carefully about whether they truly improve patient care

FORUM

190 Setting Sail for the Future
Stefano Bertuzzi
In a fast-moving world, ASM has a big role to play to fulfill its mission of advancing and promoting the microbial sciences

CURRENT TOPICS

NEW FROM ASM

192 Tuna Can Harbor Histamine-Producing Bacteria
193 Repurposing Drugs, and Fresh Outcomes from Other Familiar Sources
195 Sequencing, not Culture, Proved Fast Way To Find New Hot Spring Virus
198 Highlights from Recent ASM Journals

RESEARCH ADVANCES

194 Infecting Pregnant Mice Disrupts Fetal Brain, Inducing Autism
196 Unexpected Taxa and Mixotrophy Help To “Sink” Carbon in Oceans

DEPARTMENTS

189 Letters
227 Reviews and Resources
229 Application Deadlines
230 Calendar
231 Employment
232 Small Things Considered
NEXT MONTH

The Ocean Microbiome: Metabolic Engine of the Marine Carbon Cycle
Elizabeth B. Kujawinski, Mary Ann Moran, Aron Stubbins, and Rob Fatland

Sea-surface microorganisms fix carbon dioxide, fueling a dynamic community of heterotrophic bacteria at the surface and in the depths of the ocean.

The International Space Station: An Extreme Environment for Key Microbial and Host-Microbe Discoveries
C. Mark Ott, Thomas Marshburn, and Cheryl A. Nickerson

The extreme environment of microgravity encountered during spaceflight helps to determine how various forces influence microbes and their interactions with the host and environment.

Food Sources Harbor Antibiotic-Resistant Pathogens
Shannon Weiman

Antibiotic-resistant strains from food reservoirs at the local level can quickly disseminate, leading to worldwide public health risks.
Ink and LED

Stanley Maloy

This edition of Microbe magazine marks a new experiment with digital publishing. It is our first online-only edition. It has become common for people to do more and more reading on digital devices, whether reading scientific articles at our desk or catching up with the latest news on our mobile devices.

In the past, Microbe magazine has primarily focused on the print edition. This year, we have leapt forward in the digital realm. At the beginning of the year, we launched the magazine on a fresh template on ASMScience.org. In March we launched the ASM Mobile app, which can be downloaded for free from the Apple or Google Play store. You can now bring Microbe magazine with you on your tablet or phone.

Now that we have robust digital platforms, we want to take advantage of the different strengths of digital and print media. On the digital platform we are beginning to offer multimedia content with the launch of the Microbe magazine podcast. Through the podcast, you can listen to smart and engaging interviews with featured authors. We look forward to exploring additional ways to enhance the digital content in future editions. We have more flexibility in the digital publishing cycle as compared to print. We plan to continue to print all of the sections of Microbe online monthly. The ASM Science platform gives us the opportunity to publish more frequent updates, and later in the year we will be publishing some Current Topics ahead of the issue release, so that our readers can get the latest news in a more timely fashion. Please subscribe for content alerts on the ASMScience.org site to make sure that you don’t miss the latest news from Microbe magazine.

We know from reader surveys that ASM members love their print magazine and often share it with others. This is why we are keeping a quarterly print schedule through 2016. Starting in June we gather a selection of Current Topics, feature articles, and special sections like Microbe Mentor into Summer, Fall, and Winter editions. You will be able to share these special editions with your colleagues and students. The move to different publishing schedules online and in print lets us be more cost-effective and more environmentally friendly, while still providing all of the great content that Microbe magazine is known for and continuing to add more enticing features to Microbe magazine.

I am really excited about the content we have in development for upcoming issues. In June, we are planning features on microbiological research on the International Space Station, antibiotic resistance reservoirs and the ocean microbiome, and we will continue the popular Microbe Mentor column as well as out other features. Looking forward, the Editorial Board is working on articles about partnerships between academia and industry. Of course we will continue to bring you updates about ASM as well.

I encourage you to be part of our digital movement! Please subscribe to content alerts on the ASM Science platform and download the app to your phone or tablet today.

Stanley Maloy, Center for Microbial Sciences, San Diego State University, is the Editor in Chief of Microbe and Chair of the Microbe Editorial Board.
The Postdoc Life

I am not a scientist. But I’ve observed a scientist’s life—including late-night time points and stress over preliminary exams—since I first started dating my now-husband when he was a Ph.D. candidate at the University of North Carolina.

He’s currently a Research Scientist studying bacterial pathogenesis at Yale University, having started out as a postdoc. He began his stint there in 2008 and, while he is incredibly fulfilled by his work there, has been actively on the job market for the past three years, looking at both academic and industry posts.

I’ve been living the life of a postdoc spouse since we arrived in New Haven, a strange and transient lifestyle with the constant awareness that our family, which includes my husband and I, three children and two dogs, may need to relocate for his profession.

It means maintaining a holding pattern in ways that are both meaningful and trivial; I love my oldest daughter’s elementary school, but I’ve never gotten involved with the PTA or even tried too hard to get to know many other parents, despite the fact that I’m a very outgoing person. “What’s the point?” I ask myself.

On a much less serious note, we waited until last year to replace a subpar dishwasher (it somehow made our dishes dirtier), thinking, “We can survive with this one. After all, we’ll be moving soon.”

Our situation also means answering questions from bewildered family and friends, because although it’s familiar territory for the scientists involved, the postdoc stint and challenges of the associated job search are foreign ideas to nonscientists. “Why can’t he stay in his current position?” they ask. “Doesn’t he like it there?”

We live near family, who are a tremendous help with our children. We invested in the purchase of a small, but lovely house. I have a job I love. We’ve made good friends. I get it: why would we move?

But, I explain, we undertook this life knowing it was temporary; that even in the unlikely event that we ended up staying local, my husband’s job wasn’t meant to last forever.

I guess we just thought it would be, well, more temporary. Three years, maybe five, tops. But postdocs, according to newspaper pieces I’ve read and anecdotal evidence I’ve heard from other researchers, are getting longer and longer. The months in New Haven marched on. Then the years.

I began echoing a familiar refrain with friends when they asked questions like, “What are you thinking for kindergarten next year?” or “What about summer camps?” I’d tell them our plans, adding, “If we’re still here then, of course.” But lately, I’ve grown tired of constantly repeating the sentiment. Once an exciting prospect, it’s now an overdone refrain.

The limbo is difficult, and it’s unsettling to be in the seemingly helpless partner role, waiting for someone else’s life to so radically shape my own.

But that’s what love and marriage means, after all, and our life story—ever-so-slowly revealing itself—isn’t one I’d change.

Our postdoc life has, indeed, provided myriad benefits. I’ve stretched the boundaries of my own tolerance, and have increased my sense of adventure, too. Although it’s tough to continually ask, “What’s next?” I realize that we’re so very lucky to sit on the brink of that question. In our late thirties, having already had children, the journey is still far from over.

Scientists, I’ve learned, are patient people. They are quiet optimists. Concocting experiments over days that carry out over weeks and then, it turns out, reveal nothing particularly interesting. Still, they carry on, awaiting that one outstanding outcome.

“I could never do it,” I say to my husband and his coworkers. “I’d give up the moment I experienced my first failure.”

Perhaps I’m more resilient than I give myself credit for, though. I’ve grown much more accepting of the waiting game over the years, recognizing that a long-awaited result could be just around the corner.

And knowing that the effort spent getting there will make it all the more wonderful.

Cara McDonough
New Haven, Conn.
Setting Sail for the Future

In a fast-moving world, ASM has a big role to play to fulfill its mission of advancing and promoting the microbial sciences

Stefano Bertuzzi

It was with great excitement that I took the helm of the great ASM schooner as the new Chief Executive Officer at the beginning of January. It has been only a few months, but I would like to share my first impressions and the excitement that I feel.

One of the reasons I was drawn to ASM is the renaissance of microbial sciences, which I had been witnessing from my outside vantage point, prior to joining ASM. I saw technology, in particular metagenomics, lifting the curtain on a world of unseen microbes, which we knew existed but which were not culturable and could not be studied in any tractable way. My reasoning was perhaps naïve—if a drop of ocean water contains approximately 50,000 different microorganisms and if we can culture only 1% of them, then, even if we assume that most of these microorganisms are really boring (which they are certainly not), there is an enormous number of exciting things not only to catalogue, but to study and to learn from.

I knew that sailing aboard the ASM ship would be fascinating, with the winds of science to carry us along on a great adventure. What I did not know was that the winds would pick up speed so quickly and in such exciting ways, so that “all hands on deck” would be required at ASM. Since I decided to join ASM, the Nobel Prize in Physiology or Medicine was awarded for microbial sciences advances in the area of antiparasitic drugs; antimicrobial resistance received attention as one of the world’s top priorities at the G-7 and World Economic Forum; the Zika epidemic exploded in South America demanding global attention to find remedies for this significant global health security threat; and understanding microbiomes is now one of the top priorities of the U.S. research agenda. ASM has responded to these advances by launching a coalition on antimicrobial resistance, which will help to facilitate collaboration and discussion about these critical topics, and by planning a colloquium on microbiomes. I am excited that Lynn Enquist and Sean Whelan came to the helm to help ASM organize, in record time, an @ASM Conference to address the basic virology of flaviviruses in collaboration with the American Society of Virology (ASV). We also recently convened a group of 14 leading science reporters who discussed with ASM President, Dr. Enquist, and other scientists the status of the Zika epidemic and what needs to happen to confront this latest outbreak, but most importantly what needs to happen to be better prepared and equipped for outbreaks to come.

It is very clear to me that in a world of science moving at warp speed, an organization like ASM has a big role to play to fulfill its mission of advancing and promoting the microbial sciences. ASM is uniquely positioned to be a game changer. We have enormous human resources, with 48,000 members in 122 countries. We are a global organization with enormous intellectual capital that can be used to change the world. We have amazing leadership, who show engagement and dedication at a level that I have seen rarely seen in other organizations, if at all.

With this in mind, it is perhaps not surprising that Bill Gates, who as a philanthropist devotes his time and wealth to solve really tough problems in health and education, decided to take time to address the great migration of microbiologists descending on Boston for the 2016 ASM
Microbe meeting on June 16. I find this to be really important because we will not be able to make significant socio-economic progress as a global community if fundamental problems such as food and energy security are not addressed and if constantly emerging infectious diseases cannot be diagnosed, prevented, and effectively cured. This requires not only a complex infrastructure of surveillance and preparedness, but also a fundamental understanding of the basic molecular mechanisms and ecological aspects of microbes, both benign and pathogenic.

ASM offers a venue and a think tank not only to meld together, but most importantly to help shape the microbial sciences coming from different disciplines and from different organizations with policies from different governments and international organizations. ASM with its broad expertise can be the convener, sometimes serving as the catalyst and other times as the reactant, to make reactions happen. It is a big responsibility; I see a future with a highly engaged and forward-thinking ASM, which taps into assets such as the American Academy of Microbiology to address these really big problems, providing leadership and facilitating the global discourse in making the world a better and more secure place. This is why I am particularly excited by Bill Gates coming to the ASM Microbe 2016 meeting.

Ultimately, this is also why ASM is in the process of important governance restructuring—because we want to make sure that the organization is as inclusive and transparent as it can possibly be, knowing and serving its members while engaging them in solving pieces of these daunting problems for which science, the professional practice, education, policy, and advocacy are essential. No one can do it alone, but if we combine 48,000 vivid thinkers with an agile and nimble structure, then we can quickly deploy resources and intellectual capacity to move the needle and help the world as it moves forward.

I could not be more excited to sail with you at this time when the microbial sciences are burgeoning and we are working together to refurbish this beautiful, 100-plus-year-old ship with a glorious past and present to ensure it can weather the choppy seas in the years to come—and perhaps one day the seas will also become less choppy because of ASM’s effective actions!

Stefano Bertuzzi, Ph.D., M.P.H., is the CEO of ASM.
Upcoming ASM Meetings

**ASM Microbe 2016**
June 16–20, 2016 | Boston, Massachusetts

Integrating ASM’s General Meeting and ICAAC, this unmatched meeting showcases the best microbial sciences in the world, and explores the complete spectrum of microbiology.

www.asm.org/microbe2016

**ASM Biodefense and Emerging Diseases Research Meeting**
February 6–8, 2017 | Washington, DC

This premier event focuses on the collaborative efforts to manage biothreat agents, pathogens, and global surveillance.

**33rd Clinical Virology Symposium**
May 7–10, 2017 | Savannah, Georgia

This international symposium delves into the relationship between rapid viral diagnosis, clinical course of viral infections, and preventive and therapeutic modalities for viral infections.

Upcoming ASM Conferences

**@ASM Conference—Special President’s Edition on What Does the Biology of Flaviviruses Tell Us About Zika: The Importance of Fundamental Virus Biology**
June 1, 2016 | Washington, DC

**5th ASM Conference on Salmonella**
August 29–September 1, 2016 | Potsdam, Germany

**6th ASM Conference on Beneficial Microbes**
September 9–12, 2016 | Seattle, Washington

**ASM Conference on Streptococcal Genetics**
July 31–August 3, 2016 | Washington, DC

**ASM Conference on Infection and Cancer**
October 24–27, 2016 | Washington, DC

**2nd ASM Conference on Experimental Microbial Evolution**
August 4–7, 2016 | Washington, DC

**ASM Conference on Antibacterial Development**
December 11–14, 2016 | Washington, DC

www.asm.org/conferences
Tuna fish may carry bacteria that, even at lowered temperatures, grow and retain histidine decarboxylase (HDC) enzyme activity, raising questions about the potential for those enzymes producing histamines in refrigerated fresh tuna, according to Kristín Björnsdóttir-Butler, of the Gulf Coast Seafood Laboratory, a Food and Drug Administration research facility on Dauphin Island, Ala., and her collaborators. These cold-tolerant, histamine-producing bacteria are indigenous to the fish, not contaminants introduced during handling, the researchers note. Details appeared 29 January 2016 in *Applied and Environmental Microbiology* (doi:10.1128/AEM.02833–15/).

Björnsdóttir-Butler and her collaborators used swabs to obtain samples from gills, skin, and anal vents of yellowfin, skipjack, and albacore tuna. Swabs from the anal vents contained *Photobacterium angustum* and *Photobacterium kishitanii* bacteria among other species, she says. Unexpectedly, these bacteria can grow at 5°C (41°F) as well as at higher temperatures. Moreover, both these species carry and express the histidine decarboxylase gene, which is necessary for producing histamine. However, not all members of these fish-associated bacterial species carry the histidine decarboxylase gene; nor is it known whether the gene is plasmid- or chromosome-based, according to the report.

*P. kishitanii* are better known because of their affiliation with deep-water fish species in which they form symbioses within the light organs of their hosts, enabling them to be bioluminescent in exchange for nutrients and travel privileges. The presence of these bacteria in tuna is likely due to the fish feeding on other finned fish with such light organs or, alternatively, on those crustaceans and squid that may carry *Photobacterium* species, according to Björnsdóttir-Butler and her collaborators. The presence of these *Photobacterium* species in freshly caught tuna indicates that they are indigenous to the tuna and not present as a result of post-harvest contamination during handling and processing of the fish, she points out. Scombroid fish, such as tuna and mackerel, and some other finned fish species, such as mahi-mahi, are more likely than other fish to accumulate histamine because they carry high levels of histidine in their muscle tissues.

Although histamine poisoning from fish is well known, the potential for its production by fish-associated bacteria at low temperatures is a new finding, suggesting seafood safety experts should now take this possibility into account, according to William Fenical of the Scripps Institution of Oceanography in La Jolla, Calif., who was not involved in the research. “Histamine is not a toxin per se,” he says, explaining that it does not block sodium channels, nerve transmission, or similarly vital functions. Even so, he adds, the industry takes the risk of scombroid fish-associated histamine “rather casually.” Its effects when ingested are similar to those of an allergic reaction in terms of symptoms and mechanism. Indeed, histamine is the same compound that,
when produced endogenously, triggers allergic reactions. “You can get sick, but it would be difficult to die from it unless you are prone to anaphylactic shock,” he says.

In contrast, shellfish poisoning is much more virulent for those who dine on contaminated servings of it, Fenical continues. Typically, the toxins associated with shellfish are complex organic chemicals produced by dinoflagellates, diatoms, or other phytoplankton that shellfish accumulate via filter feeding.

David Holzman is a freelance writer in Lexington, Mass.

NEW FROM ASM

Repurposing Drugs, and Fresh Outcomes from Other Familiar Sources

Jeffrey L. Fox

In the face of new and emerging infectious disease threats, it is prudent to look back at old or even ancient sources to uncover a drug or entity that might work anew or in a different context, according to several researchers who spoke during the 2016 ASM Biodefense and Emerging Diseases Research Meeting last February. Here, let us consider the old in three specific ways: re-visiting licensed drugs, using old phage for new purposes, and looking at old beasts for new antimicrobials.

Inspired in part by a 2014 National Academy of Sciences workshop report, Rob Davey of the Texas Biomedical Research Institute in San Antonio and his collaborators examined many kinds of approved drugs, looking for any with activity against Ebola virus that might be “repurposed” as antiviral agents. A broad drug-repurposing effort sponsored by the Defense Threat Reduction Agency targeting this virus traces back even earlier to 2006, Davey says. This strategy seeks to curb the “huge failure rate” everyone faces in developing drugs from scratch, only 5% of which ever make it to clinical trials, and still more of which fail from that point onward, he notes.

In practice, however, teaching old drugs to perform new tricks is not so easy, Davey continues. First, there are no libraries from which to check out a Food and Drug Administration (FDA)-approved set of approximately 4,000 chemical entities bearing the agency stamp of approval. Furthermore, about half the companies he contacted “don’t want their drugs re-tested, and that’s a big issue because they’re worried you’ll discover something ‘funny’ or adverse,” he says. On the plus side, among those FDA-approved drugs that companies agree to subject to retesting, the hit rate is “15-fold higher than it is with randomly selected compounds.”

Although various repurposed drugs show activity against the Ebola virus, many do so at concentrations that would not be healthy for the human taking them, according to Davey. Even some more promising candidates from among channel-blocking drugs proved unworkable, however, because not only are they potentially lethal for the host but they also do not block the right viral target, he says.

Yet another channel blocker, although potent against the Ebola virus when used in nanomolar concentrations, requires further tinkering because of its hypotensive activity, Davey says. “Can we separate its blood-pressure-lowering activity from its Ebola activity, and optimize the latter?” Moreover, the underlying mechanism for its viral-killing activity is “still a bit of a mystery,” he adds. Although some
MINITOPIC

AAM Report: High Impact of Next-Gen Sequencing in Clinical Microbiology

The ASM American Academy of Microbiology (AAM) in February released a report, “Applications of Clinical Microbial Next-Generation Sequencing,” predicting that next-generation DNA sequencing (NGS) will have a big impact on clinical microbiology. This technology, when used for identifying microbial pathogens, “has the capacity to provide crucial clinical benefits in patient care, patient outcomes, and public health,” according to the report. “Clinical laboratories must find ways to overcome operational, technical, regulatory, and strategic challenges in order to effectively employ NGS-based diagnostic tests,” it further notes. “Moving the NGS process from the research lab to widespread clinical application is the payoff of the remarkable odyssey of DNA sequencing breakthroughs over the last decade,” says George Weinstock of the Jackson Laboratory in Farmington, Conn., who chaired the AAM Colloquium Steering Committee whose members wrote the report. “But there are major obstacles, which the report lays out and suggests how to combat.” To download the report, go to http://academy.asm.org/images/Colloquia-report/NGS_Report.pdf.

derivatives appear to enhance that activity, “we have no toxicology information on those derivatives. It’s worth pursuing but not really much of a shortcut, at least not as envisioned. Still, it’s a new platform.”

Before treating patients with pathogen-specific antibiotic agents—whether standard, novel, or repurposed—one should know precisely what pathogen is causing their infections and to which antibiotics it is susceptible. Bacteriophage can provide a rapid and highly specific way to determine those susceptibilities, according to Herbert Schweitzer of the University of Florida, Gainesville. For instance, he maintains a collection of phages that target the bacterial pathogen and select agent Burkholderia pseudomallei. By adding appropriate drugs to plates growing this pathogen and then adding phages, which replicate “only on live cells,” he says, “we can detect [drug-resistant] Burkholderia very rapidly.” This drug susceptibility test can be run with rapid diagnostic procedures such as MALDI-TOF mass spectrometry to first identify the bacterial pathogen.

Komodo dragons and alligators, ancient species dating back some 55 million years, are a source of potentially useful, and in their own way novel, antimicrobial agents, according to Ryan Blower of George Mason University in Manassas, Va. The blood of these species carries hundreds of cationic antimicrobial peptides, which Blower and his collaborators are screening for further development. They then combine the more promising among these peptides with lipid-specific particles to enhance their interactions with microbial membranes, he says. Small chemical changes can further improve the antimicrobial activity, which depends mainly on membrane disruption. Fearing that some active agents are lost in transit from alligator farms in Florida, he adds, the George Mason group is considering farming its own animals locally to provide fresher blood samples that perhaps will contain novel activities.

Jeffrey L. Fox is the Microbe Current Topics and Features Editor.

RESEARCH ADVANCES

Infecting Pregnant Mouse Disrupts Fetal Brain, Inducing Autism

Shannon Weiman

Infecting pregnant mice can activate immune responses that disrupt fetal brain development, causing autism spectrum disorder (ASD)-like syndrome in newborn mice, according to Gloria Choi of Massachusetts Institute of Technology in Cambridge, Mass., and her collaborators there and at several other institutions. The inflammatory cytokine IL-17a orchestrates that pathology, a finding with implications for preventive and therapeutic strategies if the mechanism in mice applies to humans, they say. Details appeared online 28 January 2016 in Science (doi: 10.1126/science.aad0314).

“Numerous animal studies demonstrate that prenatal infections can result in acute and persistent neurological and behavioral abnormalities in offspring resembling ASD or schizophrenia,” says Brian Lee of Drexel University in Philadelphia, Pa., who was not involved in this research. “This work provides a really intriguing chain of causation, from immunological mediators, to abnormal brain development, to behavioral abnormalities, that together elegantly explains the epidemiological observation that maternal viral infection during pregnancy increases risk of offspring autism.”

Prenatal infections with rubella virus, cytomegalovirus, and the parasite Toxoplasma gondii can exert teratogenic effects on brain development. However, despite well-recognized correlations since the 1970s between pregnant women becoming infected with viruses such as the measles, mumps, or influenza viruses and their babies having ASD, the mechanism underlying this phenomenon remains elusive. Further, children of mothers hospitalized for infection of any kind during pregnancy have a 30% higher risk of ASD, Lee reports. Recent evidence implicates diverse infectious agents, including viruses, bacteria, and fungi, rather than specific neurotropic viruses—suggesting an unspecific disease process underlying these associations, he says.

Searching for a molecular mechanism, Choi and colleagues injected pregnant mouse with synthetic double-
Zika Virus Update: Research, Models, Diagnostics

Amid rising concern over the rapid geographic spread of the Zika virus and expanding public health risks from outbreaks that it is causing, related research and other activities are surging. Recent examples include:

- Analysis of blood samples from patients who developed Guillain-Barré syndrome (GBS) during a 2013–2014 Zika virus outbreak in French Polynesia supports the view that infections with this virus cause GBS, according to Arnaud Fontanet from the Institut Pasteur, Paris, France, and his collaborators. Details appeared 29 February 2016 in The Lancet (doi: http://dx.doi.org/10.1016/S0140–6736(16)00562–6).
- Case studies of pregnant women indicate that the Zika virus crosses the placental barrier, according to Ana de Filippis of the Oswaldo Cruz Institute in Rio de Janeiro, Brazil, and her collaborators. Details appeared 17 February 2016 in The Lancet Infectious Diseases (doi:http://dx.doi.org/10.1016/S1473–3099(16)00095–5).
- A three-dimensional, cell-based model of the human placenta that resists infection by viruses and the parasite Toxoplasma gondii could be used to study whether and how Zika virus or other pathogens cross the placenta to cause birth defects, according to Carolyn Coyne of the University of Pittsburgh School of Medicine and her collaborators. Details appeared 4 March 2016 in Science Advances (doi: 10.1126/sciadv.1501462).
- In another model study, Zika virus “efficiently infects” human neuronal cells in vitro that are counterparts to those that form the cortex during brain development, according to Guo-li Ming and Hongjun Song of the Johns Hopkins University School of Medicine in Baltimore, Md., and Hengli Tang of Florida State University in Tallahassee. Details appeared 4 March 2016 in Cell Stem Cell (doi:10.1016/j.stem.2016.02.016).
- The gene-editing capabilities of the CRISPR-Cas9 system, if used to drive “male-determining” genes through mosquito populations, could make it feasible to control Zika-virus-carrying mosquitoes, according to Zach Adelman and Zhijian Tu at Virginia Tech in Blacksburg. Details appeared March 2016 in Trends in Parasitology (http://dx.doi.org/10.1016/j.pt.2015.12.003).
- Siemens Healthcare Diagnostics of Berlin, Germany, in March said it would release its PCR-based test for Zika virus; the test is for research use only and, although designed to run on the Siemens Versant system, it also can run on other commercially available PCR systems, the company said.
- Responding to a request from the Centers for Disease Control and Prevention, Food and Drug Administration (FDA) officials in February authorized for emergency use an antibody capture-based ELISA test for detecting Zika virus in blood specimens.

stranded RNA—to mimic a viral infection—inducing ASD-like symptoms in pups, including abnormalities in brain development as well as behavior and social interactions that persist into adulthood. These symptoms in newborn mice are accompanied by alterations in immune signaling—specifically, IL-17a receptors are upregulated in cortical layers of the brains, where structural defects arise, of the developing fetuses. Expression of IL-17a also is enhanced in maternal mononuclear cells throughout the placenta and uterine fluids during development due to dysregulated levels of cytokines during development, according to Guo-li Ming and Hongjun Song of the Johns Hopkins University School of Medicine and her collaborators. Details appeared 4 March 2016 in Science Advances (doi: 10.1126/sciadv.1501462).

These findings raise the possibility that modulation of IL-17a in the CNS can influence neuronal development, with implications as to specific neuronal cell types and their connectivity,” she says.

Other evidence implicates cytokines and IL-17a in human cases of ASD, according to Lee. “Dysregulated levels of various inflammatory markers in maternal sera or amniotic fluid during pregnancy are associated with increased risk of ASD,” he says. Further, cytokines influence neuronal migration, synapse formation, and neuronal survival. “Dysregulated levels of cytokines during development due to maternal infections therefore may adversely affect neurobehavioral function,” he says.

Children with ASD have similar immune abnormalities, Choi adds. “Elevated levels of IL-17a have been detected in the serum of a subset of autistic children.” IL-17 is also implicated in numerous autoimmune diseases in humans, including rheumatoid arthritis, irritable bowel syndrome, and multiple sclerosis. Based on results in mice with ASD, therapeutics against these diseases, including antibodies against IL-17 and the upstream IL-6 receptor, might prove useful in treating ASD, according to Choi.

Shannon Weiman is a freelance writer in San Francisco, Calif.

NEW FROM ASM

Sequencing, Not Culture, Proved Fast Way To Find New Hot Spring Virus

Carol Potera

Culture-independent methods are making it possible to discover and partly characterize viruses and their hosts straight from hot springs such as those in Yellowstone National Park (YNP)—short-circuiting the traditional approach that depends on first growing samples in vitro, according to Rebecca Hochstein at Montana State
University (MSU) in Bozeman and her collaborators. In this way, they discovered the *Acidianus* tailed spindle virus (ATSV), which infects archaeal species found in the Crater Hills Thermal Basin of YNP. Details appeared 13 January 2016 in the *Journal of Virology* (doi: 10.1128/JVI.03098–15).

“We started with the virus, not the host,” Hochstein says. “That’s unique. The viral genome gave us the first hint of what was there. This virus-centric approach enables researchers to go after a specific virus of interest, instead of whatever virus is most amenable to culturing.” This approach “could be used to discover viruses in oceans, lakes, soils, and almost any environment you could think of,” adds study leader Mark Young of MSU. “It greatly expands our understanding of viral diversity on the planet and the role viruses play in the ecology and evolution of life.”

We in biology “know so little about the diversity of viruses on Earth and their effect on microbial populations,” says Rachel Whitaker at the University of Illinois, Urbana, who was not involved in the MSU research. “This type of work makes a strong impact on expanding our view of viruses in this natural model system for virus ecology and evolution.”

In addition to sequencing the viral genetic material, the MSU researchers used an assortment of other molecular methods, including CRISPR/Cas, viral FISH, 16S rRNA, quantitative PCR, and transmission electron microscopy, to build a picture of ATSV and identify its host, which they say is *Acidianus hospitalis* or a closely related species. Members of this genus, recognized in the mid-1980s, are thermoacidophilic archaeabacteria, generally found in solfatara fields and marine hydrothermal systems; they grow as facultative aerobes by lithotrophic oxidation and reduction of sulfur dioxide.

ATSV is a new member of the group of archaeal large spindle viruses that includes *Acidianus* two-tailed virus, *Sulfolobus tengchongensis* spindle-shaped virus 1 and 2, and *Sulfolobus* monocaudavirus, according to Hochstein. All are found in hot springs worldwide, replicate in archaeal hosts, and share similar shapes and a set of seven core genes, including genes that encode a coat protein, an integrase, and multiple ATPases. Strikingly, ATSV has a large head and a very long tail that, when magnified, looks like ropes of protein molecules. In part for that reason, the MSU team proposes that ATSV and its relatives constitute a new family, which they call *Fusellocaudaviridae* based on their shape and tail.

Much less is known about geothermal viruses, especially the archaeal viruses, than about the bacteria that are recovered from hot springs at YNP. Viruses that thrive at near-boiling temperatures and acidic conditions have unusual morphologies and a wealth of genes with possible industrial applications, according to Hochstein. “The coat proteins of these viruses offer options for making nanomaterials, such as cages to deliver drugs or other materials,” she says.

The ecosystem that drives the sequestering of carbon in oceans “remains largely uncharacterized,” note Guidi and his collaborators. To develop a better understanding of that ecosystem, they reexamined environmental and metagenomic data gathered during 2009–2013 on the Tara Oceans expedition. A key step was applying an original systems biology approach, linking organisms from all domains and networks with which they are associated to carbon export at 150 meters depth, according to Guidi. “The scale of both environmental and genomics datasets collected by Tara Oceans enabled us to perform these associations for the first time at a global planetary scale,” he says.

Based on that analysis, it appears that taxa such as Radiolaria and alveolate parasites, as well as Synechococcus and their phages, are among the lineages most strongly associated with carbon export in subtropical, nutrient-depleted, oligotrophic regions of the ocean, Guidi continues. “Understanding the structure of these networks and the function of the genes linked to carbon export opens up a wide range of possibilities, especially for modeling the biological processes associated with the oceanic carbon cycle. It should therefore be possible to test the robustness of these networks in various climate simulations, to better understand how different planktonic species affect the carbon cycle and climate regulation.”

Meanwhile, according to modeling by Ward and Follows, mixotrophy proves a more important lifestyle for plankton than anyone appreciated, they note. In the Atlantic, for example, as much as 95% of grazing on bacteria is done by species that also conduct photosynthesis, they point out. This “mixotrophy enhances the transfer of biomass to larger size classes further up the food chain, leading to an approximately threefold increase in global mean organism size and about a 35% increase in sinking carbon flux.”

“While the two studies clearly address different issues using very different methodologies, I think the key similarity is that they both emphasize the importance of ecological interactions in determining ocean carbon exports,” says Ward, referring to his and Follows’ as well as Guidi’s reports. “This is in contrast to a view that biological productivity and export are primarily governed by physical processes, especially light conditions and nutrient supply.” Adds Guidi, “Both these papers . . . bring new information about the functioning of the biological carbon pump”—his term for how photosynthesis behaves in the oceans.

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MINITOPIC
Microbial Findings of Note: Peculiar, Historical, or in Extremes

Here are several recent microbial findings of note, including in peculiar, historical, or extreme situations and environments:

- Contrary to the widely held view that human-associated microorganisms outnumber host cells 10 to 1, a recent recount puts that ratio closer to 1 to 1, with the prototype human adult estimated as carrying nearly 40 trillion bacterial cells compared to about 30 trillion human cells, according to Ron Milo, Shai Fuchs, and Ron Sender of the Weizmann Institute of Science in Rehovot, Israel. Moreover, the host and microbial cell numbers are so close, they note, “that each defecation event may flip the ratio to favor human cells over bacteria.” Details appeared 28 January 2016 in Cell (doi:http://dx.doi.org/10.1016/j.cell.2016.01.013).

- The youngest example of a terrestrial fossil is a fungus, called Tortotubus, that dates back 440 million years, according to Martin Smith, formerly of the University of Cambridge and now at Durham University in the United Kingdom. Its morphology and pattern of growth suggest “an affinity with the ‘higher’ fungi,” he notes. Details appeared 2 March 2016 in the Botanical Journal of the Linnean Society (doi:10.1111/boj.12389).

- The enzyme RNA polymerase from bacteria “scrunches” the DNA that it binds and then transcribes—unwinding the DNA strands and pulling the unwound polymer into itself until it engages the transcription start site, according to Bryce Nickels and Richard Ebright from Rutgers, the State University of New Jersey in New Brunswick, Deanne M. Taylor at the Children’s Hospital of Philadelphia, and their collaborators. Details appeared 4 March 2016 in Science (http://doi.org/10.1126/science.aad6881).

- Graphene oxide, mixed with low levels of calcium chloride, is an effective and non-toxic antiseptic agent, active in vitro against both gram-negative and -positive bacterial pathogens, according to Valentina Palmieri at the Università Cattolica del Sacro Cuore in Rome, Italy, and her collaborators. They presented their findings during the annual meeting of the Biophysical Society, held in Los Angeles in March (abstract: http://tinyurl.com/zzgsfu).
NEW FROM ASM
Microbial Communication over the Airwaves

Scientists at the Institut Pasteur in Paris, France, were surprised to discover that an airborne volatile compound released by the bacterium Pseudomonas aeruginosa can promote growth of the fungus Aspergillus fumagatus. First author Benoît Briard, working with senior scientist Jean-Paul Latgé, made the discovery. “That microbes ‘smell’ other microbes is not new,” says Latgé. “But that one species of bacteria is stimulating the growth of a fungus by ‘smell’—this is totally new.” The compound, identified as dimethyl sulfoxide by mass spectrometry, contains sulfur, which the fungus can use to grow. The findings will be useful when studying coinfections of the lung, where both P. aeruginosa and A. fumagatus can cause disease.

Briard B, Heddergott C, Latgé J-P. Volatile compounds emitted by Pseudomonas aeruginosa stimulate growth of the fungal pathogen Aspergillus fumigatus. mBio. Published online 15 March 2016; doi: 10.1128/mBio.00219–16

NEW FROM ASM
One Drug to Both Sensitize and Kill Drug-Resistant Malaria

Researchers at the National University of Singapore in Singapore are working hard to combat chloroquine resistance in Plasmodium falciparum, a major cause of malaria. Two newly discovered molecules show promise in simultaneously sensitizing chloroquine-resistant parasites and inhibiting parasite growth. Now published in Antimicrobial Agents and Chemotherapy, the work takes advantage of previously characterized chemosensitizers, which are able to confer sensitivity to resistant malaria parasites. First author Aicha Boudhar, working with senior scientists Brian Dymock and Kevin Tan, first identified the compounds in a screen of novel hybrid molecules combining the active structures of chemosensitizers and chloroquine. Both molecules inhibit growth of several clinical isolates, including drug-resistant strains. The next step will require animal models to assure safety and efficacy in a host.


NEW FROM ASM
A biofilm model that accounts for cell aggregates

A new paper published in mBio addresses the difference between biofilms initiated with single planktonic cells versus those initiated with groups of cell aggregates. Researchers at the University of Copenhagen in Copenhagen, Denmark, the University of Texas in Austin, Tex., and the University of Edinburgh in Edinburgh, United Kingdom used both in silico modeling and Pseudomonas aeruginosa biofilm experiments to test aggregated cells, which sit atop one another and gain structural height compared to single cells. Co-first authors Kasper Kragh, Jaime Hutchinson, and Gavin Melaugh, working with co-senior authors Rosalind Allen, Vernita Gordon, and Thomas Bjarnsholt, found that elevated cells, whether single or in aggregates, grow more quickly than those at lower elevation, likely due to differential access to oxygen. The authors propose a new model based on this information that accounts for the role of cell aggregates in biofilm formation and dispersal.

Microbiology—Volume 11, Number 5, 2016

NEW FROM ASM

A Respiratory Syncytial Virus-Like Particle Vaccine Shows Promise

Respiratory syncytial virus (RSV) is the most common viral cause of pneumonia and has no approved vaccine. First author Velasco Cimica, working with lead scientist Jose Galarza, is working to change that with a virus-like particle (VLP)-based vaccine. The researchers tested VLPs containing the RSV F glycoprotein, which has two major conformations during viral envelope fusion. Researchers at TechnoVax, Inc, in Tarrytown, NY, working with the City College of New York in New York, NY, compared VLPs containing prefusion F protein, postfusion F protein, or a combination of both conformations. All three VLP vaccines conferred complete protection in mice challenged with RSV, and induced a strong Th1 cytokine response, with little disease-exacerbating Th2 cytokine induction. Inflammation was lowest in animals vaccinated with postfusion or combination conforma- tions VLPs, which was reflected in animal lung pathology. These VLPs show potential toward clinical development.


NEW FROM ASM

Nonpathogenic Viruses Are Transferred during Fecal Microbiota Transplants

Fecal microbiota transplants (FMTs) are increasingly being used for refractory Clostridium difficile infections, but scientists at the University of Pennsylvania in Philadelphia, Penn., have found more than bacteria can be transferred between patients. “We could see bacterial viruses moving between humans and we were able to learn some viruses through transmission, but we did not see any viruses that grow on animal cells that may be of concern for infecting and harming patients,” says senior author Frederic Bushman, who published the results in mBio. He and first author Christel Chehoud observed mostly temperate bacteriophage, which are latently located in the bacterial genome but can be induced during stress to replicate at higher concentrations. Future studies will investigate how viruses move through similar scenarios, such as organ transplantations.


NEW FROM ASM

Targeting the Gut Microbiome to Fight Heart Disease

Researchers have found that resveratrol, a compound in red wine, reduces the risk of heart disease by altering the gut microbiome. Researchers at the Third Military Medical University in Chongqing, China, found that resveratrol reduces levels of trimethylamine-N-oxide (TMAO), a known contributor to development of atherosclerosis. “Resveratrol reduces TMAO levels by inhibiting the gut microbial TMA formation via remodeling gut microbiota,” says senior author Man- tian Mi. TMA is necessary for the production of trimethylamine-N-oxide (TMAO)-induced ratios, inhibits Prevotella growth, and promotes Bacteroides, Lactobacillus, Bifidobacterium, and Akkermansia growth in mice. Now available in mBio, the work may lead to prescription probiotics or prebiotics for patients with cardiovascular disease.


NEW FROM ASM

New Fish Virus Identified in Tilapia Farms

Scientists have found the viral cause of disease outbreaks in tilapia farms and published their results in mBio. Collaborators at Tel Aviv University in Tel Aviv, Israel, Columbia University in New York, NY, the New York Genome Center in New York, NY, the University of Edinburgh in Edinburgh, Scotland, and St. George’s University in Grenada, West Indies identified a new RNA virus in the Orthomyxoviridae family. Co-first authors Eran Bacharach and Nischay Mishra, working with senior scientists Avi Eldar and Ian Lipkin, identified the viral genome by negative selection. First the non-ribosomal RNA from diseased fish samples was sequenced, and then the sequences matching known fish or other contaminating genes were subtracted. The leftover sequences produced the genome of the virus, which was named the Tilapia Lake Virus (TiLV). “This will be a key preventative tool to stop the transfer of infected stock and stop the disease from spreading,” said Eldar.

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Antibiotic Resistance Spreads through Diverse Species and Habitats, Part I

The public health threat amplifies as drug-resistant pathogens move freely through various environments and species

Shannon Weiman

Antibiotic-resistant bacteria continue to spread across the globe—directly affecting human and animal patients while also establishing reservoirs from which those strains continue to emerge long into the future, according to several researchers who spoke during the 2015 ICAAC conference held in San Diego last September. Their findings paint a broader and, in some ways, more alarming picture of the expanding influence and dynamic nature of antibiotic resistance than experts previously drew.

Antibiotic resistance genes and bacterial strains carrying them are infiltrating diverse environments, threatening to cause long-term havoc along with the current challenges for which they are so well known. The reservoirs for such strains in both terrestrial and aquatic habitats, as well as within wild and domesticated animals, foster the accumulation, spread, and reintroduction of such drug resistance into humans, posing serious long-term public health threats.

In particular, several types of pathogens that infect humans, including extended-spectrum β-lactamase producing *Escherichia coli* (ESBL-*E. coli*), carbapenem-resistant Enterobacteriaceae (CRE), and methicillin-resistant *Staphylococcus aureus* (MRSA), are highly adaptable and move freely among diverse animal hosts before they jump back into humans. Researchers are now documenting environmental excursions by these and other pathogens—monitoring environmental reservoirs that they occupy while also identifying how they are being transmitted from one host species to another. These tracking studies are part of a larger effort to predict and eventually prevent further spread of drug-resistant pathogenic strains into and within human populations.

Soil Microbes as a Source and Reservoir of Antibiotic Resistance

Soil environments are a likely source for novel antibiotic-resistance genes, according to Fiona Walsh of Maynooth University in Ireland, who spoke during the 2015 ICAAC session “Surrounded by the Enemy—the Environment and Foodstuffs as Sources or Reservoirs of Antimicrobial Resistance Threats.” Within soil, resident bacterial species constantly battle one another over turf, she says. Some species produce toxins or growth inhibitors that enable them to thwart and thus outcompete their neighbors. Humans co-opted some of those same compounds for use as antibiotics. Meanwhile, neighboring microbial species in soils developed the means to resist toxins from aggressor microbial species. Buried in soil, those resistance mechanisms are of little consequence to humans. However, serious public health consequences can arise when soil bacteria transmit their resistance genes to microbial species living within humans or animals (Fig. 1).

SUMMARY

➤ Antibiotic resistance genes and bacterial strains carrying them are infiltrating diverse terrestrial and aquatic environments, threatening to cause long-term havoc along with the challenges for which they are currently known.

➤ Soil environments are a likely source for novel antibiotic resistance genes, whereas water provides a more dynamic reservoir, accumulating and also dispersing bacteria carrying familiar antibiotic resistance factors.

➤ Wildlife species as well as domesticated animals are spreading multidrug resistant strains across vast geographic areas.

➤ Pets are reservoirs for antibiotic-resistant strains, acting as conduits between humans and the environment, and disseminating resistance factors into other animal species for further spread.
Walsh’s research focuses on uncovering the hidden presence of such resistance genes in soils from both rural and urban sources. She identifies novel resistance genes that act against the quinolone antibiotic naladixic acid, and tries to identify how each works before they exert any impact on public health. “If they ever emerge from the soil and transfer to human clinical pathogens, it will be important to characterize their activity,” she says.

There is precedent for such concern, says Dereje Dadi Gudeta of the University of Copenhagen, Denmark, who spoke during the session, “Emergence of Resistance: Environmental and Food Chain Threats.” Such an environmental transfer likely began the worldwide spread of the Klebsiella pneumoniae carbapenemase (KPC) from that bacterial species to diverse other gram-negative pathogens, including Salmonella enterica, E. coli, and Pseudomonas spp. Gudeta finds ancestral homologues of KPC in various Chromobacterium species, gram-negative bacteria found in both soil and water that only rarely infect mammals. “The recovery in three distinct Chromobacterium species of functional class A β-lactamases with up to 76% amino acid identity to KPC suggests that [these enzymes] may have evolved from possible ancestor genes resident on the chromosome of members of this genus,” he says. The ancestral genes then likely transferred to strains of other gram-negative bacteria that are...
common pathogens of humans when transposon Tn4401 was mobilized.

Soil microbes also harbor resistance genes that are commonly known for their clinical significance among human pathogens, including more than a dozen β-lactamases and various carbapenemases, according to Walsh. “We identified the multidrug-resistant (MDR) nature of soil bacteria by selecting them on one antibiotic,” she says. “In this group of soil bacteria, greater than 80% of isolates are resistant to 16–23 antibiotics.”

The vast majority of these strains carry efflux pump resistance mechanisms, Walsh continues. Where these genes originated is not known, but possibly they are native to soil bacterial populations or were acquired after mingling with human- and animal-associated microbial strains, which contaminate soil environments in various ways, including via wastewater, farm runoff, and manure, she says. “To minimize the risks to human health posed by resistance genes in the environment and the risk to the environment by waste containing resistance genes, we must identify the critical points of control that are resistance gene hotspots.”

Water Exposes Humans to Antibiotic-Resistant Bacteria

Water is a dynamic reservoir, accumulating and also dispersing antibiotic-resistance bacteria. In particular, municipal wastewater treatment facilities foster the mixing of bacterial strains shed from human populations, leading to rampant exchanges of resistance genes among microorganisms that congregate there, according to Amy R. Sapkota of the University of Maryland School of Public Health. “Water is a complex and diverse reservoir of antibiotic-resistant bacteria,” she says. “Municipal wastewater is the richest aquatic habitat in terms of known antibiotic resistance genes.”

Jill Hoelle of the Environmental Protection Agency (EPA) in Washington, D.C., concurs, noting that MDR bacteria are present in wastewater-treatment plants across the United States (US). While most E. coli stains in these facilities are antibiotic susceptible, those that do carry resistance are frequently MDR strains, she says. “Some of these organisms have become resistant to all or almost all antibiotics, including last-resort drugs [like] carbapenems.” A disconcerting 41% of such MDR E. coli strains are carbapenem resistant, belonging mostly to phylogenetic subgroups B2 and D that are known for causing invasive human infections. These strains were most prevalent in samples taken from urban wastewater treatment facilities, suggesting that such urban reservoirs may pose greater public health risks than rural agricultural sources.

While wastewater treatment plants are intended to remove such contaminants, they are not always successful, according to Sapkota. For example, MRSA strains can survive treatment systems that do not use chlorination and then can pose risks to public health. Specifically, those individuals who are commonly exposed to reclaimed water, such as spray irrigation workers, are more likely to carry MDR S. aureus in their nasal cavities than are others, and could become a source for further spread of such strains among human populations, she says. There are no federal regulations regarding reclaimed water testing or use, and few studies to indicate public health risks, she points out. Further, droughts tend to exacerbate the situation, increasing reclaimed water use on farms and gardens, with the unintended consequences of possibly introducing and disseminating MDR clinical strains into the environment and increasing human exposures to them.

In addition, wastewater from pharmaceutical plants can release by-products into the environment that select for antibiotic-resistant pathogens, according to Avemaria Obasi of the University of Lagos, Nigeria. “Wastewater effluent systems represent a protective niche for commensals and pathogens favoring the horizontal transfer of genes encoding for resistance factors,” she says. ESBL-producing K. pneumoniae and Pseudomonas aeruginosa strains contaminate wastewater from pharmaceutical plants in Nigeria, she finds. In the lab, these strains can pass their resistance genes to other opportunistic pathogens, including E. coli. One of the ESBL resistance genes that she identified in wastewater isolates is also found in clinical isolates from patients with urinary tract infections in the region, she notes. However, further analyses are needed to determine whether such transmissions are occurring and whether they are contributing to public health risks in Nigeria.
Wildlife Spreads MDR Strains across Vast Geographic Areas

MDR bacteria within water reservoirs also can be disseminated to wildlife, in effect generating animal reservoirs for those bacteria that further disperse resistant strains to geographically distant sites and increase the chances for humans to be exposed to them. For example, an MRSA strain found in wild boars and deer in Spain traces to a local water source, according to Mark Holmes of Cambridge University in England, who spoke during the session, “Animal-Human Transmission of Methicillin-Resistant Staphylococci.”

“Presence of MRSA in the river water highlights the potential role of water in the dissemination” of strains carrying resistance, says Holmes. Indeed, many antibiotic-resistant strains made their way into diverse animal species across the globe, including the Iberian wolf, lynx, red fox, beaver, Spanish slug, and numerous fish and bird species. “These are indicators of environmental pollution, which contributes to spread and expansion of antibiotic resistant bacteria in fragile niches,” says Andrea Endimiani of the University of Bern in Bern, Switzerland.

One major source of such “pollutants” is livestock farms, Endimiani continues, citing findings by Christina von Salviata of the University of Berlin, Germany, and her collaborators (Fig. 2). Some 25% of flies and 33% of mice on a pig farm in Germany carry ESBL- \(E.\ coli\), according to von Salviata. Because such organisms are near the bottom of the food chain, they carry enormous potential to disseminate these MDR strains to larger mammals, birds of prey, and other predators that can further spread antibiotic resistance factors regionally and, eventually, globally. In addition, 75% of green plants surrounding the pig farm were contaminated with antibiotic-resistant microbial strains, potentially exposing any animals that eat such vegetation.

Thus, the pig farm that von Salviata surveyed was seeding multiple environmental reservoirs in its immediate vicinity and beyond, Endimiani says. As one remedy to these amplifying effects, he supports the One Health concept, in which the...
combined effects of human, animal, environmental, food production, and veterinary health are recognized for being intertwined and dependent on one another. Our surveillance systems and public health efforts to control MDR bacteria need to take into account this interconnectedness, he says.

Furthermore, environmental contamination can be self-perpetuating, with frequent exchanges of resistant bacteria between wild animals and aquatic habitats seeding additional environmental reservoirs. Wild animals can carry resistant bacteria in their feces, contaminating fresh water sources even in apparently pristine places, according to Endimiani. For instance, 36% of rivers and lakes in Switzerland contain ESBL-producing Enterobacteriaceae, which in turn make their way into 19% of fish inhabiting those waters, he reports.

Wild birds in particular, especially migratory species, are of great concern for further disseminating antibiotic-resistant bacterial strains. “Migratory birds are the global spreaders,” says Endimiani, referring to various types of ESBL-producing Enterobacteriaceae in black vultures and E. coli identified across the globe.” They retain, as gut carriers, human MDR bacteria and plasmids acquired from dumps in corresponding regions,” he says. “Thus bird migration could contribute to the dissemination of MDR E. coli over the globe.

He also notes that cormorants and mallards carry ESBL E. coli with quinolone resistance genes across Europe, while migratory wild birds in Germany carry carbapenemase-producing S. enterica originating from Asiatic regions. MDR strains have even made their way into wild bird populations in remote and developing regions of South America, says Alice Batalha-Jesus of Universidade Federal de Rio de Janeiro, Brazil, who documents ESBL-E. coli in black vultures and tropical screech owls in Brazil.

Because of their scavenging activities, omnivorous diets, and close interactions with humans, gulls and crows are particularly prone to being colonized with MDR pathogens of humans, says Ivana Jamborova of the University of Veterinary and Pharmaceutical Sciences in Brno, Czech Republic. In the US, for example, 11% of fecal samples from crows contain AmpC-producing E. coli, while 2% contain ESBL E. coli, she adds. “We identified E. coli clones and plasmids previously documented in humans and food animals, most of which are potentially pathogenic for humans or animals. . . including E. coli strains of hyperepidemic sequence type (ST) 131, ST405, and ST648. This constitutes alarming environmental contamination by MDR bacteria.” Their extensive interactions with humans and wild animals make these bird species efficient transmitters of such pathogenic strains across various human, animal, and environmental reservoirs, further amplifying this already serious public health threat.

**Companion Animals Carry MDR Strains between Humans and the Environment**

Dogs, cats, and other pets are yet another conduit for transmitting MDR microbial strains within and between species. The intimate interactions between pets and the humans who take care of them foster colonization with community strains, which can spread back to human populations, serving as sources for recurrent human infections. In addition, pets shed these resistant strains into the environment (Fig. 3).

“Pets and wild animals are important reservoirs...that contribute to the spread and expansion of MDR pathogens, particularly for CTX-M-15-producing E. coli and newly emerging OXA-48- and NDM-producing E. coli and K. pneumoniae,” says Endimiani, referring to various types of β-lactamases. While data is scarce on precise colonization rates, studies in Western Europe suggest that approximately 18% of pets are colonized with these types of drug-resistant strains of E. coli. In parts of Asia, these rates are higher—estimated at 24.5% for mainland China and a whopping 87–92% in Hong Kong.

“There is increasing recognition that companion animals may act as a reservoir for community-associated MDR pathogens,” says Thomas Gottlieb of the University of Sydney, Australia. While reports of CRE in cats, dogs, and horses are common across the globe, animals in Australia remained free of CRE until recently. The resistance gene IMP-4 was first detected in Acinetobacter baumannii in Hong Kong in 1997, and first recognized in Australia in 2002 during a clonal outbreak in several urban hospitals. It continues to spread within humans, based on isolates from hospitals across eastern Australia.

During a localized outbreak in a Sydney cat shelter, Gottlieb identified such animals as an
unexpected source of *S. enterica* strains carrying this resistance gene. While strains harboring IMP-4 and infecting humans were documented in Australia during the past decade, this recent finding is the first evidence of its spread to other animal species, he says. “No CREs had been reported in companion animals or livestock in Australia, either as pathogens or commensals.” The IMP-4 carbapenemase gene likely moved from human clinical *E. coli* and *E. cloacae* isolates, and then spread to a wide variety of other Enterobacteriaceae via highly promiscuous plasmids such as IncA/C, he adds. “The maintenance of IMP-4 in broad-range plasmids may contribute to diffusion and maintenance in different bacteria in the Asia-Pacific [region].”

Some cats are asymptomatic carriers of IMP-4-carrying *S. enterica*, making them difficult to track and facilitating further spread of the clone, according to Gottlieb. He says that implementing infection control measures and better controls over antibiotic use in veterinary hospitals and human clinics could help to minimize the unwitting spread of these MDR strains. Meanwhile, the feral nature of many cats and their predatory activities against wild birds and mammals make them an efficient bridge between humans and other wild animal species. For example, the IMP-4 resistance gene moved relatively recently into flocks of silver gulls within southeastern Australia, he found. The colonization of these wild birds likely will accelerate the spread of these MDR microbial strains throughout Australia. “Gulls and other birds can be an efficient means of spread between waste dumps, which may include hospital waste and MDR flora, and to the community,” he says.

Companion animals also serve as reservoirs of MRSA strains, which can colonize skin and nasal mucosa, and are easily passed between humans and dogs, according to Holmes of Cambridge University. For example, dog owners sometimes become chronically infected with MRSA because their pets reinfect them following antibiotic treatment, he says, citing findings from Farrin Manian of St. John’s Mercy Medical Center in St. Louis, Mo. Similarly, more than 8% of infections due to MRSA in an outpatient facility were traced to
family pets serving as reservoirs for these pathogens, according to Vance Fowler of Duke University in Durham, N.C.

This path for MRSA transmissions is a two-way street, with humans sometimes infecting dogs, Holmes points out, describing the chain of transmission of a particular strain of pathogen, called ST22, involving staff and animals at a particular animal hospital. “Direct transmission occurred in both directions, substantiating the view that ST22 has a broad host range and behaves as a nosocomial pathogen within a veterinary health care setting just as it does within human hospitals,” he says.

Such strains can circulate among humans, dogs, cats, and even horses—sometimes merely colonizing their animal hosts and other times causing fulminant illnesses, Holmes continues. “A shared population of an important and globally disseminated lineage of MRSA can infect humans and companion animals without undergoing host adaptation. This study furthers the One Health view of infectious diseases that the pathogen pool of human and animal populations are intrinsically linked and provides evidence that antibiotic usage in animal medicine is shaping the population of a major human pathogen.”

Part 2 of this article will appear in the next issue of Microbe.

Shannon Weiman is a freelance writer in San Francisco, Calif.
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PCR for Everything—Seeking Value in Speed

With faster and more comprehensive diagnostic tests becoming widely available, we need to think more carefully about whether they truly improve patient care

Bert K. Lopansri

Medicine is a science of uncertainty and an art of probability.
— Sir William Osler

In the summer of 2013, a patient arrived in our emergency department with complaints of high fever and abdominal pain. She came to us straight from the airport following her return from a 3-month visit to India. Identified as septic, she was “pan-cultured,” meaning readily obtained fluids and substances were sent immediately to begin cultures. Due to concerns for bacterial gastroenteritis, she received ceftriaxone in the emergency department and ciprofloxacin after admission. Fevers continued and blood cultures yielded *Escherichia coli*, which was phenotypically resistant to most antibiotics, including ceftriaxone and ciprofloxacin, but susceptible to “last-resort” carbapenems, which were then used to treat her.

Before that stage, however, we asked whether she might be infected with a carbapenemase-producing *E. coli*—such as a New Delhi metallo-β-lactamase, which would inactivate the “last-resort” antibiotics. Pending clinical lab findings, should this patient be placed into contact isolation and consigned to yellow-gowned health care workers to prevent spread to other hospitalized patients? Should her antibiotics be switched to an expanded-spectrum cephalosporin, carbapenem, or colistin, an uncommonly used antibiotic with significant risk for nephrotoxicity?

These questions were swirling before antibiotic susceptibility results became available. Fortunately, conventional microbiology methods confirmed that, although the microorganism infecting her was resistant to most antibiotics, it produced “only” an extended-spectrum β-lactamase (ESBL), one that cleaves most such antibiotics as well as aztreonam but spares carbapenems.

When we encountered this patient, we were participating in a clinical trial evaluating a commercially produced molecular diagnostic instrument—the Verigene® Gram-Negative Blood Culture Test (BC-GN), by Nanosphere, Inc. of Northbrook, Ill.—that identifies bacterial pathogens and many of the resistance markers that they carry within 3 hours of a blood culture turning positive. Our retrospective testing of this isolate revealed the absence of carbapenemase genes and the presence of the CTX-M type ESBL, which was consistent with results obtained by phenotypic testing.

After achieving microbial clarity, we posed another critical question: would the patient have improved more quickly and could the length of stay have been shortened if we had known sooner that she was infected with an ESBL?

**Antibiotic-Resistant Bacteria Challenge Clinical Medicine in Many Ways**

This vignette illustrates the latest challenges and uncertainties we face in clinical medicine from antibiotic-resistant bacteria. Methicillin-resistant *Staphylococcus aureus* (MRSA) was long the poster child for the “resistance movement.”

**SUMMARY**

- When evaluating patients, clinicians and clinical microbiologists should consider whether results from rapid diagnostic tests will improve how those patients are treated.
- Molecular diagnostic tests shorten turnaround times but also can increase costs.
- Rapid organism identification with molecular tests can improve antibiotic use and patient outcome with bloodstream infection, but increase cost and add little value without an infrastructure to translate rapid results into action.
- The newest syndromic tools, designed to detect pathogens that cause gastroenteritis, may reduce many uncertainties but also can confuse clinicians, especially when several targets test positive.
However, the unholy band of drug-resistant nasty germs continues to grow. Vancomycin-resistant Enterococcus faecium remains a problem among some patients, such as those who are immunocompromised. The numbers of carbapenem-resistant Acinetobacter baumannii, ESBL and carbapenemase-producing Enterobacteriaceae (CRE) exploded during the past decade—further reinforcing the standard clinical practice of treating patients with broad-spectrum antibiotics before their infecting pathogens are identified. As history shows us repeatedly, this practice contributes to antibiotic resistance and increases patients’ risk for becoming infected with Clostridium difficile.

What is the alternative? One major step is to treat infected patients from the outset with effective and appropriate antibiotics—a step that requires rapid, reliable, and accurate detection of the specific pathogen responsible for each patient’s illness. In other words, we need to take better advantage of advances in diagnostic microbiology.

Three major developments are helping to shorten the time to identifying infecting pathogens: (i) molecular methods that identify bacteria within 1–3 hours of a positive blood culture bottle turning positive (Table 1); (ii) matrix-assisted laser desorption/ionization time of flight (MALDI-TOF) microorganism identification using mass spectrometry (MS); and (iii) syndrome-specific molecular methods that detect pathogens directly from primary specimens. Although these methods minimize sample preparation steps and shorten turnaround times, the requisite instruments are expensive and, in the case of molecular testing, each test comes with an added unit cost.

### Rapidly Identifying Organisms and Assessing the Value of That Speediness

One important advance is shortening the time to identifying what might be causing bacteremia or fungemia, whether by MALDI-TOF MS or by molecular methods. Traditional methods for de-
Detecting pathogens in blood cultures and determining their drug susceptibilities require many steps and can take 72 hours (Fig. 1). Newer technologies shorten that turnaround time to one to three hours after a blood culture turns positive (Table 1). Although not cleared by the Food and Drug Administration (FDA) for direct use on positive blood culture broth, many labs, including ours, have internally validated and are using MALDI-TOF MS for this purpose. Its main drawbacks are an inability to detect antibiotic resistance markers sooner and low sensitivity for identifying organisms in polymicrobial samples.

What is the value of these advances, and does this increased speed matter? If the main value from faster diagnostic tests is improving overall antibiotic use, the answer is unequivocally yes. The emerging consensus is that rapid testing, when paired with antimicrobial stewardship, will improve antibiotic use, especially in cases where patients are carrying antibiotic-resistant bacteria, according to Debra Goff of the Ohio State University Medical Center, James Musser of the Methodist Hospital in Houston, Melissa Miller of the University of North Carolina, and their respective collaborators.

However, without ways to promptly communicate rapid test results to take action on the clinical side, such molecular testing would increase health care costs but add little value in terms of how patients are being treated, cau-

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**TABLE 2. Proposed Benefits of Rapid Bacterial Identification, Resistance Detection and Improved Antibiotic Use**

<table>
<thead>
<tr>
<th>Benefit</th>
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<tbody>
<tr>
<td>Decreased mortality</td>
</tr>
<tr>
<td>Decreased length of stay</td>
</tr>
<tr>
<td>Decreased risk for <em>Clostridium difficile</em> infection</td>
</tr>
<tr>
<td>Decreased antibiotic exposure for blood cultures deemed to be clinically insignificant or a contaminated specimen</td>
</tr>
<tr>
<td>Decreased time to implementation of infection control procedures for antibiotic resistant bacteria</td>
</tr>
<tr>
<td>Reduced selection pressure for development of antibiotic resistance</td>
</tr>
</tbody>
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**FIGURE 1**

Traditional process for blood cultures which require incubation of blood culture bottles, subculture onto solid media and identification with antibiotic susceptibility testing using automated methods. Rapid tests by differing methods allow for identification directly from positive blood culture broths. To date the only rapid test that identifies organisms before blood cultures turn positive is for *Candida* spp. (T2 Biosystems; Lexington, MA).
Lopansri: Between Thailand and the U.S., Focused on Infectious Disease and Diagnosis

Bert Lopansri's parents raised him and his two brothers to believe that they could be anything they wanted to be—as long as they became doctors. “Fortunately for my parents, one of the three developed an interest in science and medicine,” he says, referring to his career in medicine, specializing in infectious diseases and microbiology.

Today, Lopansri, 45, is an infectious diseases expert who spends his time doing patient care, research, and teaching. He is clinical associate professor at the University of Utah School of Medicine, and Chief of the Division of Clinical Epidemiology and Infectious Diseases at LDS Hospital and Intermountain Medical Center in Salt Lake City. His research now encompasses health care epidemiology and clinical microbiology, particularly the impact of rapid diagnostics and novel ways of communicating rapid results to clinicians. He also has a longtime interest in global health, including tropical diseases, which has involved numerous visits to Africa and Southeast Asia.

Born in Chiang Mai, a city in mountainous northern Thailand, Lopansri moved with his family to the Midwestern United States when he was a baby. However, he visited Thailand every summer until he started college. “On summer trips during high school and undergraduate studies, my aunt would take me to the teaching hospital where she worked and had me round with her,” he says. “She would tell me about patients who died from things like rabies, tetanus, malaria.” Later, seeing hospitalized patients with dengue, yellow fever, measles, HIV, and dysentery, he adds, “as an undergraduate, I was amazed by how the clinicians pieced together the clinical presentation to make a diagnosis, and lab tests were not the major pieces of the equation.”

Lopansri received his B.S. degree in biology in 1993 from the University of Illinois at Urbana-Champaign, and his M.D. in 1997 from Loyola University Stritch School of Medicine in Maywood, a suburb of Chicago, where he also completed his internship and residency in internal medicine. Later, he completed an Infectious Diseases fellowship at the University of Utah, where he joined the faculty as an assistant professor in 2003 and was part of a team investigating the pathophysiology of severe malaria in African children and Asian adults. He developed a passion for clinical research during his Fellowship when he was tasked to measure arginine levels in very small volumes of plasma collected from African children suffering from cerebral malaria. Once he solved a self-induced technical problem, he says, “While processing samples in a blinded manner, I quickly realized that I could predict which ones were from healthy children and which ones were from those with cerebral malaria. It was at that moment that I realized a career in research was in the cards.”

He left in 2007 for Chicago to pursue a career in academia where he continued malaria research and was involved with hospital epidemiology and infection control. He returned to Salt Lake City in 2011 and, in addition to his patient care duties, has been involved in clinical microbiology as a medical director. “I have had the good fortune to be involved with transformations occurring in diagnostic microbiology and evaluating how cutting-edge technology can improve patient care.”

Lopansri’s wife is a director of project management for the pharmaceutical company Quintiles. They have an 11-year-old son and a 12-year-old daughter. “I enjoy athletics greatly and now spend time watching my kids compete and coaching them. I also enjoy the outdoors—hiking, camping, skiing—anything that gets me outside, although I do draw the line at anything that gets me outside, although I do draw the line at activities in which death is a potential outcome.” He plans to travel with his wife and children to explore parts of the world they have not seen. “We’ve only made it to Thailand, so we have a long way to go,” he says. “My kids let me know about it regularly.”

Marlene Cimons
Marlene Cimons lives and writes in Bethesda, Md.
cording to Goff, Musser, and their respective collaborators. However, there was no benefit in other types of bacteremia, according to Miller of the University of North Carolina and her collaborators. We conducted a small quality study to assess the impact of a multiplex panel in patients with *S. aureus* bacteremia using matched, historical controls as comparators. In our study, we showed improvement in antibiotic use with a roughly $4,500 cost savings per patient for those with MSSA bacteremia (unpublished). Based on available studies and personal experiences with rapid diagnostic testing, I conclude that some but not all patients with septicemia will experience a cost reduction when rapid diagnostic tests are applied and acted on. The key question is how many patients need to benefit to prove value when using cost and mortality as metrics.

The final value consideration is not readily measurable and relates to the influence on a clinician’s approach to a patient with septicemia. Knowing the identity of a pathogen sooner has tremendous value to a clinician as it can set the course not only for antibiotic treatment but for identifying unsuspected sources of infection. As an example, *S. aureus* septicemia has different implications than other causes of bacteremia, including other *Staphylococcus* spp., and often requires more aggressive search and control strategies. A 24-hour head start makes a major difference to a hospitalized patient as many things can occur within that short period.

**Questions about Multiplex, Molecular Syndromic Panels**

Last summer, I was asked to see a patient admitted to the hospital with sepsis, severe abdominal pain and cramping, profuse diarrhea, and severe inflammation of the colon. We learned later that she was one of several individuals who developed diarrhea after attending an event the evening before. A multiplex, respiratory panel by PCR, which detects 20 respiratory pathogens in a single test, was negative. A stool sample tested positive for leukocytes, indicating an inflammatory diarrhea, but cultures were negative. In the ER, she was treated initially with ceftriaxone and, later after being admitted, ciprofloxacin, on which she improved.

At the time of this encounter, we were validating two FDA-cleared molecular panels for detecting gastrointestinal pathogens, including *Shigella* species. Even with this pathogen in mind, our repeat cultures of the initial sample were negative. However, an independent lab with a multiplex panel confirmed *Shigella*. If we had known earlier that this patient was infected with *Shigella*, could we have treated her with different antibiotics, and avoided admitting her to the hospital? Why was a respiratory panel ordered initially, and how many times have respiratory panels been used in this capacity?

This vignette reflects the trend in diagnostic molecular microbiology to follow a multiplex “syndromic” approach. It also perfectly captures how my enthusiasm for these tests weighs against my reservations about their potential overuse and misuse.

The newest syndromic tools are designed to detect pathogens that cause gastroenteritis (Table 3). Unlike bloodstream infection panels, which start with a positive culture and when positive almost always provide actionable information, syndromic tests start with primary specimens. Proposed advantages with the gastrointestinal (GI) panels include automation with >90% sensitivity and specificity and a turnaround time 2–3 days faster than stool cultures. When compared to antigen detection tests that are available for many GI pathogens, and other singleplex stool tests such as *Clostridium difficile* tests by nucleic acid detection, however, multiplex molecular panels provide little benefit with respect to turnaround time.

When viewed from a laboratory perspective, one important value from using these panels is that they greatly improve efficiency of laboratory workflow, especially for stool cultures. For example, clinicians who submit stool samples typically order multiple tests. Thus, taking the syndromic approach can be more cost-effective and efficient. Why order a stool culture, multiple antigen tests, and a *C. difficile* test when you can order a single test covering all these targets?

When viewed from a clinical perspective, however, the value of these broad panels becomes less certain. Keeping patients from being admitted and shortening their hospital stays are important value metrics. However, I am less certain about the impact such testing can have on antibiotic use and clinical outcomes. Supportive care with fluid replacement is most important for treating patients with diarrhea, while antibiotics play a limited role. Unlike blood culture panels,
which can influence antibiotic use, the GI panels are configured to diagnose infections that either cannot be treated with antibiotics (rotavirus, norovirus, sapovirus, adenovirus, astrovirus), should not be treated (STEC, *E. coli* O157), may or may not be treated depending on severity of illness (*Campylobacter* spp., *Salmonella* spp., *Plesiomonas shigelloides, Yersinia enterocolitica, Vibrio* spp., *Clostridium difficile* toxin A/B, *E. coli* O157, STEC, *Enteroaggregative* *E. coli*, *Enteropathogenic* *E. coli*, *Enterotoxigenic E. coli*)

One other important consideration is that clinical presentations and epidemiologic factors differ among gastrointestinal pathogens, even when symptoms overlap. While these tests will reduce many uncertainties, they also can confuse clinicians, especially when several targets test positive. For example, what does a clinician do with test results that are positive for *Enteroaggregative* *E. coli* and *C. difficile*, when there are no risk factors for the latter?

A colleague recently described a patient whom she suspected to be infected with norovirus (personal communication from Dr. Dascomb). However, the multiplex GI panel confirmed norovirus but was also positive for *C. difficile* and *E. coli* O157, with negative shiga toxins. Is the *C. difficile* result real? What is the significance of O157 positive with a negative shiga toxin? She elected to trust the clinical presentation, ignore the O157 result, and not to act on the *C difficile* result. The patient improved without antibiotic treatment for *C difficile*.

Important questions to consider going forward are: must we test simply because we have the molecular means to identify more enteric pathogens, and how will these tests integrate with other tests currently being used in most labs such as culture, singleplex molecular and antigen-based detection methods? In the United States, there are about 179 million cases of diarrhea each year, according to Herbert DuPont of the University of Texas School of Public Health and Medical School in Houston. Testing a mere 5% of those affected individuals, at $75-$300 per episode depending on what tests are ordered, will come with a hefty price tag to the system. To control such costs, we need to consider patient needs first. Development of care process models to guide the approach to a patient with acute diarrhea has the potential to influence use of diagnostic tests. We must also carefully monitor how these new multiplex molecular diagnostic panels are being used and how these tests integrate with other diagnostic tests available.

In the PCR-for-everything age, clinicians need to know what they are looking for when ordering a test. They are still trained to take detailed histories and to perform physical examinations in order to understand what might be causing a patient’s illness. Laboratory testing is an adjunct that may throw light on suspicions and guide therapy but should not become the sole means for diagnosis. Despite value in using rapid methods to identify bacteria and drag resistance markers in patients with bloodstream infections, an appropriate infra-

<p>| TABLE 3. Clinically Available Commercial, Multiplex, Molecular Gastrointestinal Pathogens Tests |</p>
<table>
<thead>
<tr>
<th>Test</th>
<th>Bacteria</th>
<th>Viruses</th>
<th>Parasites</th>
</tr>
</thead>
<tbody>
<tr>
<td>BioFire GI PCR</td>
<td><em>Salmonella</em> spp., <em>Shigella/Enteroinvasive E. coli</em>, <em>Campylobacter</em>, <em>Plesiomonas shigelloides, Yersinia enterocolitica, Vibrio</em> spp., <em>Clostridium difficile</em> toxin A/B, <em>E. coli</em> O157, STEC, <em>Enteroaggregative</em> <em>E. coli</em>, <em>Enteropathogenic</em> <em>E. coli</em>, <em>Enterotoxigenic</em> <em>E. coli</em></td>
<td><em>Adenovirus</em> 40/41, <em>Norovirus</em> GI/GII, <em>Astrovirus</em> Rotavirus, <em>Sapovirus</em> (I, II, III, IV)</td>
<td><em>Cryptosporidium</em>, <em>Cyclospora cayetanensis</em>, <em>Entamoeba histolytica</em></td>
</tr>
<tr>
<td>Verigene Enteric Pathogens Test</td>
<td><em>Salmonella</em> spp., <em>Shigella</em> spp., <em>Campylobacter</em> spp., <em>Vibrio</em> Group, <em>Yersinia enterocolitica</em>, <em>Shiga Toxin</em> 1, <em>Shiga Toxin</em> 2</td>
<td><em>Norovirus</em> GI/GII, <em>Rotavirus</em> A</td>
<td>None</td>
</tr>
<tr>
<td>Luminex xTAG® (Luminex, Corp; Austin, TX)</td>
<td><em>Salmonella</em> spp., <em>Shigella</em> spp., <em>Campylobacter</em> spp., <em>Clostridium difficile</em> toxin A/B, STEC, <em>E. coli</em> O157, <em>Enteroaggregative E. coli</em>, <em>Enteroopathogenic E. coli</em></td>
<td><em>Norovirus</em> GI/GII, <em>Rotavirus</em> A</td>
<td><em>Giardia lamblia</em>, <em>Cryptosporidium</em></td>
</tr>
<tr>
<td>BD Max™</td>
<td><em>Salmonella</em> spp., <em>Shigella</em> spp., <em>Enteroinvasive E. coli</em>, <em>Campylobacter</em> spp., <em>STEC</em></td>
<td><em>Norovirus</em> GI/GII, <em>Rotavirus</em></td>
<td><em>Cryptosporidium</em>, <em>Entamoeba histolytica</em>, <em>Giardia lamblia</em></td>
</tr>
</tbody>
</table>

*STEC, shiga toxin-producing E. coli*
structure is needed, such as an antimicrobial stewardship program, to allow prompt action based on results from such testing. The value of multiplex molecular GI panels is less certain and needs to be evaluated. As we navigate the ever-expanding menu of broad, multiplex panels, we must be cautious about letting the test-for-everything approach become the norm.

Bert K. Lopansri, M.D., Division of Infectious Diseases and Clinical Epidemiology, Intermountain Medical Center, is Medical Director, Intermountain Healthcare Central Microbiology Laboratory, and Associate Professor of Medicine, University of Utah School of Medicine.

Suggested Reading


32nd Clinical Virology Symposium
A meeting of the America Society for Microbiology
May 19–22, 2016 | Daytona Beach, Florida

Secure Your Seat Today!

Join ASM for the 32nd Clinical Virology Symposium to explore the latest discoveries in the research, care, and prevention of viral infections. Stay current and expand your connections with fellow laboratorians, physicians, and biomedical researchers involved in patient care and public health through focused plenary sessions, novel posters, engaging case presentations and dynamic networking events.

www.asm.org/cvs2016

Arrive early and delve into the practical aspects of molecular diagnostics during
PASCV’s Molecular Virology Workshop
Wednesday, May 18, 2016!
Ocean Station ALOHA, University of Hawaii at Mānoa Designated as a Milestones in Microbiology Site

Ocean Station ALOHA (A Long-term Oligotrophic Habitat Assessment), University of Hawaii (UH) at Mānoa, the microbiological research site 100 km north of Oahu, Hawaii, was officially named a Milestones in Microbiology site by ASM on 17 November 2015, in recognition of its historic and visionary contributions to the science of microbial oceanography. Research conducted at the site has fundamentally changed the understanding of microbes in the sea, and has played a central role in defining the discipline of microbial oceanography, which studies the roles and interactions of microorganisms in the structure and function of marine ecosystems.

Research Programs. From its origins, research at Ocean Station ALOHA has been conducted by scientists from around the world and has featured interdisciplinary collaboration between individuals who in the past did not typically interact (microbiologists, physical scientists, oceanographers, mathematicians, and educators). These collaborations have resulted in the discovery of novel microorganisms, unprecedented metabolic pathways, and a wealth of information regarding complex microbial interactions, and have significantly enhanced understanding of the impacts of climate change on marine ecosystems.

Much of the research at Ocean Station ALOHA is accomplished through the HOT (Hawaii Ocean Time-series) program, launched in 1988, which aims to provide a comprehensive description of the ocean at a site (Ocean Station ALOHA) representative of the North Pacific subtropical gyre through repeated observations of the hydrography, chemistry, and biology of the water column there throughout the year. Monthly cruises are made to the deep-water Station, where measurements are taken of the thermohaline structure, water column chemistry, currents, optical properties, primary production, plankton community structure, and rates of particle export.

In 2006, the range of the HOT work was expanded through the creation of C-MORE (Center for Microbial Oceanography: Research and Education), a National Science Foundation Science and Technology Center, with the mission of investigating the identities, roles, and impacts of microorganisms to global environmental variability and climate change. Studies investigating how microorganisms and phytoplankton control the oceanic carbon, nitrogen, phosphorus, and iron cycles have yielded significant insights into global climate change.

The SCOPE (Simons Collaboration on Ocean Processes and Ecology) program was launched in 2014 to specifically investigate microbially mediated processes that govern the flow of matter and energy.

Education and Public Outreach. In addition to its basic research mission, C-MORE has an educational mission through which it develops undergraduate and graduate-level curricula and training programs to increase the number of students and teachers engaged in quantitative sciences and engineering, focusing on underrepresented groups, especially Native Hawaiians and Pacific Islanders. Its international course “Microbial Oceanography: Genomes to Biomes” has trained over 150 early career scientists in the growing discipline of microbial oceanography.

Through its public and private partnerships with the National Science Foundation, the Gordon and Betty Moore Foundation, and the Simons Foundation, Ocean Station ALOHA conducts public outreach to increase awareness of the science and its global importance. Each of its research programs hosts detailed websites that provide public access to news releases and multimedia educational materials, including educational videos and the “ALOHA-HOTEL” (ALOHA HOT Electronic Library), a comprehensive bibliography of more than 600 scientific papers, book chapters, and review articles.
The Milestones Ceremony. The Milestones ceremony was held in conjunction with the inaugural lecture in the Pavel Distinguished Lecture Series, “Waypoints in Microbial Oceanography.” The lecture, “Climate, Oceans, and Human Health: The Cholera Chronicle,” was presented by Rita Colwell, former director of the National Science Foundation and former ASM president, who commented, “Ocean science can no longer be viewed as an esoteric, ‘offshore’ discipline. It is mainland and mainstream. The health and bounty of our oceans are an issue of planetary survival.”

The Milestones plaque was presented by Tim Donohue, ASM past president, to Alexander Shor, Associate Dean for Research, UH School of Ocean and Earth Science and Technology, David Karl, Co-Founder of the HOT program that established Ocean Station ALOHA, and Co-Founder and Co-Director of C-MORE and SCOPE, Edward DeLong, Co-Director and Co-Founder of C-MORE and SCOPE, and Matthew Church, Professor and Senior Researcher, C-MORE, and current lead principal investigator of the HOT program. “This open-ocean research station has played a key role in defining the discipline of microbial oceanography and educating the public about the vital role of marine microbes in global ecosystems,” said Donohue. “It is my opinion that we are in a renaissance period for microbiology, a time where we are poised to gain new insight into the myriad of ways in which microbes impact the world that we inhabit and will pass on to future generations. We can look to Ocean Station ALOHA for examples of how to explain the science that excites us and its potential to solve problems relevant to society today and in the future.”

The Milestones event was well attended by faculty, students, ASM Hawaii Branch members, university leaders, including David Lassner, President of the University of Hawaii System and Donna Vuchinich, President and CEO of the University of Hawaii Foundation, and ASM guests, including Doug Eveleigh, chair of Milestones, and John Meyers, ASM Membership Services Director. Other highlights of the Milestones celebration and Pavel Lecture were a seminar, “The Science of Ocean Station ALOHA” delivered by David Karl, Edward DeLong, and Matthew Church, a tour of C-More Hale, and a view-

Urine Collection, Storage and Preservation Laboratory Practice Survey

ASM and the Centers for Disease Control and Prevention (CDC) are conducting a survey to understand laboratories’ current practices related to the collection, storage and preservation of urine for microbiological culture. The goal of the study is to improve the diagnosis and management of patients with urinary tract infection. This survey will take approximately 20 minutes to complete; participation is voluntary.

The results from the survey will be compiled and shared in aggregate as a learning tool, presented at professional conferences, and potentially published in a professional journal in the field of laboratory science. All information collected in this survey will be kept in a secure manner; your IP address will NOT be retained.

Visit https://www.surveymonkey.com/r/CDN26NN to complete the survey. Thank you for taking the time to complete this survey by June 1, 2016. Your feedback is important for guiding ASM and CDC’s efforts to improve laboratory practice. Contact clinmicro@asmusa.org with questions or concerns.
ing of the award-winning film, “The Invisible Seas” which was produced in the 1970s by Rita Colwell. Publicity after the event include the issuance of a first-day cover with ASM, University of Hawaii and Ocean Station ALOHA logos, and the local TV broadcast of a 30-minute video (produced by Jay Fidell of “Think Tech Hawaii”) featuring event highlights and interviews with Colwell and Donohue (now available on YouTube at https://www.youtube.com/watch?v=iprHO5wrflA).

To learn more about Ocean Station ALOHA and to view videos of the Milestones ceremony and Pavel Lecture, go to http://cmore.soest.hawaii.edu/.

The Milestones in Microbiology program recognizes and honors institutions (and the scientists who worked there) that have made significant contributions toward advancing the science of microbiology. For more information, visit www.asm.org/milestones-in-microbiology.
ASM Public Affairs

ASM Comments on Select Agent Regulations

In March, the ASM sent comments to the Centers for Disease Control and Prevention (CDC) and the Animal and Plant Health Inspection Service (APHIS) regarding changes to the list of Select Agents and Toxins and enhanced biosafety requirements, in response to the Biennial Reviews published in the Federal Register on 19 January 2016. The comments addressed the removal of certain agents and proposed new requirements to clarify the regulatory language and to address concerns related to inactivation of select agents, biosafety procedures, and confirmed identification of a select agent. To read the ASM comments and the Federal Register notices go to http://www.asm.org/index.php/publicpolicy-2/statements-testimony/137-policy/documents/statements-and-testimony/94078-sa-3-16.

ASM Submits Congressional Testimony on FY 2017 Research and Public Health Funding

In March, ASM submitted written testimony to the House and Senate appropriations subcommittees on FY 2017 funding for federal research and public health programs impacting microbiology. The statements included recommendations for the National Institutes of Health, Food and Drug Administration, Department of Agriculture Research, Department of Energy, Office of Science, Centers for Disease Control and Prevention, National Science Foundation, and Department of Defense medical research programs. To read the testimony, go to http://www.asm.org/index.php/publicpolicy-2/statements-testimony/137-policy/documents/statements-and-testimony/94075-fy-2017.

ASM Participates in S-FAR Congressional Briefing

On 24 February, ASM joined the other members of the U.S. Stakeholder Forum on Antimicrobial Resistance (S-FAR) in a briefing discussing the public health crisis of antimicrobial resistance (AR) and related programs in the President’s Budget Request for Fiscal Year 2017. Attendees had the opportunity to hear clinician and patient perspectives on infections with AR organisms and were able to interact with federal agency officials who are working to implement the “National Action Plan for Combating Antibiotic-Resistant Bacteria.” S-FAR is a national partnership and was convened on the principle that any U.S. government strategy to address antimicrobial resistance should involve sustained and meaningful engagement of nongovernmental experts and stakeholders throughout the development and implementation process. Visit http://s-far.org/.

ASM Attends Congressional Hearing on Zika Virus

On 24 February, ASM staff attended “The Zika Virus: Coordination of a Multi-Agency Response” hearing held by the House Committee on Oversight and Government Reform. Presenting the history of Zika virus and current federal government strategies were Anne Schuchat, Principal Deputy Director of the Centers for Disease Control and Prevention, and Anthony Fauci, Director of the National Institute of Allergy and Infectious Diseases. John Armstrong, the Surgeon General and Secretary of Health of the State of Florida, and Bill Moreau, Managing Director for Sports Medicine for the United States Olympic Committee, raised specific concerns about mosquito eradication and risks to U.S. athletes. To see the hearing in its entirety, see https://oversight.house.gov/hearing/the-zika-virus-coordination-of-a-multi-agency-response/.

ASM Attends FDA Pharmacy Compounding Advisory Committee Meeting

ASM staff attended the 8–9 March U.S. Food and Drug Administration Pharmacy Compounding Advisory Committee Meeting meeting in Silver Spring, Md. The portion of the meeting of interest to ASM members involved the discussion of quinacrine hydrochloride and its use as an antiparasitic treatment, an antilupus treatment, and for intrauterine sterilization. All materials from the meeting can be found at http://www.fda.gov/AdvisoryCommittees/CommitteesMeetingMaterials/Drugs/PharmacyCompoundingAdvisoryCommittee/ucm486094.htm.

PSAB Committees Co-Author CIDT Interim Guideline

Members of the ASM Public and Scientific Affairs Board (PSAB) Committees on Laboratory Practices and Professional Affairs joined the Association of Public Health Laboratories Culture-Independent Diagnostics Subcommittee to author “Interim Guideline: Submission of Enteric Pathogens from Positive Culture-Independent Diagnostic Test Specimens to Public Health.” This document is intended to assist clinical laboratory personnel in submitting required isolates to public health laboratories. The guidance applies to isolates and specimens that are positive by culture-independent diagnostic tests (CIDTs), since nationally recommended practices for the public health submission of specimens that test positive using CIDTs are not available to clinical microbiologists. To read the document, go to http://www.aphl.org/AboutAPHL/publications/Documents/FS-Enteric_Pathogens_Guidelines_0216.pdf.

NAS Gain-of-Function Workshop

The “Gain-of-Function Research: The Second Symposium” was held by the National Academies of Sciences, Engineering, and Medicine on 10–11
March. This public meeting was the Academies’ second gathering to provide a mechanism to engage the life sciences community and the broader public and solicit feedback on optimal approaches to ensure effective federal oversight of gain-of-function research. Among those participating were officials from the Office of Science and Technology Policy, the U.S. Food and Drug Administration, the National Institutes of Health, the Departments of Health and Human Services and Homeland Security, and the World Health Organization. View the meeting agenda by going to http://dels.nas.edu/resources/static-assets/bls/agenda/GOFagendafinal.pdf.

ASM Meetings and Conferences

ASM Microbe 2016: Register Now and Win!

Register now for the inaugural ASM Microbe 2016 (June 16–20, Boston), and you could win a $100 American Express gift card! This unmatched meeting will offer top-notch scientific sessions and posters, as well as unique learning and networking opportunities such as Scientific Track Hubs, Peer-to-Peer Exchange Zone, and wellness activities. There will be plenty of opportunities for you to find your home within the larger meeting. Be sure to attend the Opening Keynote Session on Thursday, June 16, featuring the legendary Bill Gates! Early bird rates end on May 5, 2016. Register today at www.asm.org/microbe2016.

Last Chance to Register for the 32nd Clinical Virology Symposium. Time is running out to secure your seat at the 32nd Clinical Virology Symposium (May 19–22, 2016, Daytona Beach, Fla.). This highly acclaimed international symposium will showcase the cutting-edge research findings on viral infections, and foster discussions with laboratorians, physicians, and biomedical researchers involved in patient care and public health. Arrive early on May 18 to attend the Pan American Society for Clinical Virology (PASCV) Molecular Virology Workshop. To learn more, visit www.asm.org/cvs2016.

Upcoming ASM Conferences. ASM Conferences address the needs of the diverse scientific interests of microbiologists by providing a forum for international groups of scientists to discuss their specific area of concentration. Mark your calendar for these upcoming ASM Conferences. For more information, visit www.asm.org/conferences.

@ASM Conference – Special President’s Edition on What Does the Biology of Flaviviruses Tell Us About Zika: The Importance of Fundamental Virus Biology (June 1, 2016, Washington, D.C.)

ASM Conference on Streptococcal Genetics (July 31–August 3, 2016, Washington, D.C.)


5th ASM Conference on Salmonella (August 29–September 1, 2016, Potsdam, Germany)

6th ASM Conference on Beneficial Microbes (September 9–12, 2016, Seattle, Wash.)


ASM Conference on Antibacterial Development (December 11–14, 2016, Washington, D.C.)

Education Board

ASM Represented at National Student and Educator Meetings

The strategic directions of the ASM Education Board include collaborating with national organizations to promote microbiology education at all levels. In winter 2015–2016, the Board sponsored the Society’s participation in several conferences focused on science students and educators.

Committee on Minority Education member Beronda Montgomery and Education staff member Irene Hulede were invited speakers at the Emerging Researchers National Conference (ERN) in Science, Technology, Engineering, and Mathematics held 25–27 February 2016 in Washington, D.C. Sponsored by the American Association for the Advancement of Science and the National Science Foundation, the annual ERN conference targets participants of national programs that increase diversity and develop students in undergraduate STEM education. Montgomery and Hulede presented “Tips and Coaching for Effective Oral and Poster Presentations,” which (i) emphasized the importance of disseminating research findings at scientific conferences and meetings, (ii) provided strategies for navigating these gatherings, and (iii) shared approaches for communicating science clearly, concisely, and logically.

ASM Education Director Amy Chang and staff member Kelly Diggs-Andrews represented ASM at Understanding Interventions that Broaden Participation in Research Careers in Philadelphia, Pa., on 26–28 February. Diggs-Andrews was an invited speaker at the meeting, which was established to facilitate the dissemination of research on interventions and initiatives that broaden participation in science and engineering research careers. Her presentation, “Early-Career Fellowships for Networking and Research Initiation Improve Research Capacity and Undergraduate Research at Predominately Undergraduate Institutions,” described the ASM Leaders Inspiring Networks and Knowledge (LINK) Undergraduate Faculty Research Initiation (UFRI) fellowship program as a new model of support and professional development for faculty at undergraduate-serving and resource-limited institutions. She discussed barriers to research success for
undergraduate STEM faculty, potential strategies to overcome the barriers, and how the UFRI fellowship could serve as a scalable intervention program. Program outcomes—including improved networking skills, increased confidence and success in attracting scientific collaborators, and enhanced integration of authentic research into undergraduate courses—for the inaugural cohort of fellows were also reported.

Committee for K-12 Outreach chair Dave Westenberg, Education staff member Kari Wester, and Communications staff member Emily Dilger represented the Society at the National Association of Biology Teachers (NABT) Annual Meeting held in Providence, R.I., on 11–14 November. ASM sponsored two sessions at the meeting. “A Constructive Approach to Biology,” by Kristala L. J. Prather and Natalie Kuldell (Massachusetts Institute of Technology) focused on ways to use the BioBuilder curriculum to teach synthetic biology in the classroom, and “Lab Safety,” an interactive presentation by ASM member Ruth Gyure (Western Connecticut State University), shared best practices in biosafety when working with microbes in the lab. In the exhibits program, Wester and Dilger demonstrated ASM outreach activities and shared information about the Society’s faculty and student resources.

ASMCUE: Registration Ending Soon

Register today for the 2016 ASM Conference for Undergraduate Educators (ASMCUE), set for 21–24 July at the Bethesda North Marriott Hotel & Conference Center in Bethesda, Md. Don’t miss the chance to hear from ASM’s new CEO, Stefano Bertuzzi, Ph.D., M.P.H., who will be on hand to welcome attendees. ASMCUE will also feature intensive professional development sessions and inspiring talks by leaders at the forefront of science and teaching. Confirmed plenary lectures include the following:

**Integrating Modern Genomic Science into Practical Microbiology: The Case of Food Safety**, by Eric Brown, Ph.D., U.S. Food and Drug Administration

**Pathogenesis of Fungal Infections**, by mBio editor-in-chief Arturo Casadevall, Ph.D., Johns Hopkins University

**Increasing Access to Education and Careers in STEM Field**, by Shirley Malcom, Ph.D., American Association for the Advancement of Science

**The State of the Nation: What We Know About Learning Biology**, by 2016 Carski Foundation Distinguished Undergraduate Teaching Awardee Loretta Brancaccio-Taras, Ph.D., Kingsborough Community College

The lectures will complement discussions of advances in STEM education and research, lab safety guidelines, teaching tools, student learning, and more. The conference registration deadline is 13 June 2016. For full program details, visit http://www.asmcue.org.
Microbe Mentor

Career Activities at ASM Microbe 2016

In our second edition of a two-part series about Microbe 2016, we invited Dr. Laura Runyen-Janecyk, Professor in the Department of Biology at the University of Richmond, to provide insight on how students and postdocs can navigate the careers activities at Microbe 2016. Here is what she had to say:

Large meetings hosted by ASM (such as ASM Microbe 2016, which is the General Meeting and ICAAC combined) have plentiful opportunities to learn about cutting-edge microbial science and to draw ideas of inspiration for one’s own science. What many students and postdocs may be unaware of, however, are the equally exciting opportunities for career exploration and skill development alongside the super scientific discourse that is occurring daily. Whether you are an undergraduate student presenting your research for the first time at a national meeting, a graduate student contemplating future career options, or a postdoc wanting to develop a specific professional skill, ASM Microbe 2016 has just the workshop/session for you. These opportunities exist as half-day workshops the day before the scientific meeting begins and in sessions during ASM Microbe 2016. Registration and cost for workshops are available online and on site, and is separate from the ASM Microbe registration.

Finding Your Future: Sessions for Undergraduate and Graduate Students. For those attendees who are interested in learning more about careers in microbiology, there are two half-day workshops the day before the meeting officially starts that allow in-depth career exploration. The Microbiology Career Choices Workshop (June 16, 12:45–4:15 PM, Boston Convention & Exhibition Center [BCEC], Grand Ballroom East) is targeted to undergraduate and graduate students who will have the opportunity to participate in small-group discussions with microbiologists who have careers in and outside of academia. For those interested specifically in careers in industry, a workshop earlier in the day (Transitioning Science from Academia to Industry, June 16, 8:15–11:45 AM, BCEC, Meeting Room 109A) will examine the process of developing one’s science into a viable company. For those attendees who are not arriving until June 17, there will be short, 30-minute Career Talks from individuals in industry, government, clinical, teaching, policy, and communications, followed by a Q&A session on June 17, 10:45–1:45 PM and June 18–20, 12:30–2:45 PM in BCEC, Exhibit Hall in the Professional Development Zone. In the general program, there is a specific session in which five science communicators from a range of areas provide an overview of their working life and share some of their favorite work (Pixels, Paints, and Pop Culture: Unique Perspectives on Science Communication, June 19, 2:45–5:15 PM, BCEC, Meeting Room 257A). Finally, for those attendees, especially graduate students, who are eager to develop their own individual development plan (IDP) to identify career objectives and professional development activities, the session “Using an IDP to Plan a Successful Scientific Career” might be just the session for you (June 16, 12:45–4:15 PM, World Trade Center [WTC], Cityview Ballroom 1). The National Institutes of Health (NIH) strongly encourage the use of IDPs for graduate students supported by NIH awards.

Hone Your Communication Skills: Seven Sessions for All Levels. Not surprisingly, since communication is key in almost every profession, ASM is offering seven opportunities for attendees to hone this skill. There are two half-day workshops the day before the meeting officially starts. For written communication, during the “Effective and High-Impact Scientific Writing” workshop (June 16, 8:15 - 4:15 PM, BCEC, Meeting Room 206A), participants will receive formal training in strategies to communicate scientific ideas effectively in manuscripts using concepts that can also be applied to other works. For oral communication, which one is called upon to explain why their research is important to society, the “Science Storytelling: Engaging the Audience as You Advance Your Career” workshop (June 16, 12:45–4:15 PM, WTC, Harborview Ballroom
1) will provide attendees with practical skills for telling stories that are clear, concise, and compelling to students, colleagues, supervisors, donors, patients, news media, legislators, family, and friends. For those attendees who are interested in this topic but cannot attend the workshop, there will be a shorter, 45-minute session “Once Upon a Time, in a Lab Far Away…: Telling Your Science as a Story” during the scientific meeting (June 19, 8:15–9:00 AM, BCEC, Exhibit Hall in Professional Development Zone). Additionally, during the scientific meeting, there will be several other communication-themed events in the Professional Development Zone at BCEC: Lights, Camera, Science! Engaging Videos 101 (June 17, 8:15–9:00 AM), Science in 30 Seconds: Develop an Elevator Speech (June 18, 8:15–9:00 AM), How Can I Put This?: Creating Analogies (June 18, 9:15–10:00 AM), and Microbiology in the World of Social Media (June 19, 9:15–10:00 AM).

**Management of People and Projects: Two Workshops to Boost Your Knowledge.** One area in which many scientists report receiving little to no formal training, but wished they had some, is management of people and projects. Two workshops will provide attendees with this valuable training. In the workshop, “Effective Scientific Leadership: Developing Your People” (June 16, 8:15 - 11:45 AM, WTC, Amphitheater), participants will learn the essential communications skills valued in industry, and how to apply them in mentoring others, delegating responsibilities, and becoming an effective leader. In “Practical Project Management for Scientists” (June 16, 12:45 - 4:15 PM, WTC, Waterfront Ballroom 2), participants will learn the importance of tactical

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<thead>
<tr>
<th>Workshop/Session Title</th>
<th>Date</th>
<th>Time</th>
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<tr>
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<td>June 16</td>
<td>8:15–11:45 AM</td>
<td>BCEC, Meeting Room 204A</td>
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<tr>
<td>Getting Hired: Job Search Strategies and Crafting Your CV/Resume</td>
<td>June 18</td>
<td>8:15–10:45 AM</td>
<td>BCEC, Meeting Room 206A</td>
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<td>Effective Interviewing Skills and Job Offer Negotiation</td>
<td>June 18</td>
<td>2:45–5:15 PM</td>
<td>Westin, Marina II</td>
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<tr>
<td>Graduate School Recruitment Event</td>
<td>June 17</td>
<td>1:45 PM</td>
<td>TBD</td>
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*For the most up-to-date information, check www.asmscience.org.*
planning, communications, negotiation, and control in being an effective project manager, and will become familiar with the tools and language of project management so that they can more effectively fit into cross-matrixed teams, and in some cases even assume the role of project manager.

Undergraduate Teaching: A Professional Development Workshop. Many attendees at ASM Microbe may be considering a career in teaching at the undergraduate level, yet have no formal training in this area. The most competitive applicants for these positions in the current job market are individuals who have both formal training in pedagogy and experience in teaching. Early-career faculty may have obtained a position, but wish there were more opportunities for professional development in pedagogy at their institution. The workshop “Engaging and Inclusive Teaching in the Microbial Sciences” (June 16, 8:15–4:15 PM, BCEC, Meeting Room 204A) is just the opportunity to obtain such training. In this workshop, attendees will learn how implementing evidence-based instructional practices and the ASM Curriculum Guidelines can help improve student learning in their microbial sciences classroom and laboratory.

Moving to the Next Career Stage: Grad School Recruitment, Job Search, and Interviewing Skills. For attendees preparing to “move to the next career stage”, there are several sessions that will be of interest to you. In “Getting Hired: Job Search Strategies and Crafting Your CV/Resume” (June 18, 8:15–10:45 AM, BCEC, Meeting Room 206A), you will learn how to create an effective communication strategy to articulate your strengths, interests, and values in a way that shows potential employers you are the best solution to their problem. Topics will include the difference between a résumé and a CV, how to translate your CV into a nonacademic résumé, and how to write cover letters that tie your interests and those of the employer together. In “Effective Interviewing Skills and Job Offer Negotiation” (June 18, 2:45–5:15 PM, Westin, Marina II), you will focus on technical and behavioral ways to approach interviews and how to negotiate job offers and startup packages. Finally, for undergraduates, there will be a “Graduate School Recruitment Event” (June 17, 1:45 PM, Location: TBD), where students will have an opportunity to talk to graduate school representatives to learn about what schools are looking for in applicants, the requirements for graduate school, and the types of research programs.

Expanding Your Network: Introduce Yourself and Start a Conversation. In addition to the formal career opportunities workshops and sessions at ASM, with thousands of microbial scientists in attendance at Microbe 2016, there will be plenty of opportunities for informal networking. Helpful hints for networking can be found in the Microbe Mentor column of Microbe January 2016. Furthermore, tips to create your “elevator pitches” that will help you network can be found in the April 2016 Microbe Mentor column in Microbe. For some individuals, the idea of initiating a conversation with a stranger is somewhat stressful. However, my experience at ASM meetings is that (1) it gets easier with practice and (2) that most people are excited to respond to an initiated conversation. So, I challenge the students and postdocs attending Microbe 2016 to make a point of introducing yourself to at least one new person each day and initiate a conversation. Good things will come of it, I assure you.

Microbe 2016 will be a busy time for you as you engage in scientific exploration and discourse with other scientists. However, I encourage you to find the time to deliberately explore equally important topics in areas of career exploration and skill development. See you at ASM Microbe 2016 in Boston!

Laura Runyen-Janecky, Ph.D.

Laura Runyen-Janecky is a Professor in the Department of Biology at the University of Richmond, where she teaches in the areas of Microbiology and Genetics and has an active research program with undergraduate students. She is enthusiastically engaged in bringing evidence-based teaching into the classroom and is a member of the ASM Graduate and Postdoctoral Education Committee.

Have a question for Microbe Mentor or want to write for Microbe Mentor? E-mail microbementor@asmusa.org.
Microbe
SECOND EDITION

AUTHORS
Michele S. Swanson, Gemma Reguera,
Moselio Schaechter, Frederick C. Neidhardt

“This is a fantastic text! Written in a
comfortable, conversational style, it grabs the
reader’s attention immediately, sparking their
curiosity and keeping them engaged
throughout each chapter while they seek and
find answers to questions posed at the
beginning of each section. A true joy to read.
I recommend it highly for both traditional and
flipped classrooms.”

— Peggy Cotter, PhD, Professor,
Department of Microbiology and
Immunology, UNC School of Medicine

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Reviews and Resources

BOOKS

Life’s Engines: How Microbes Made Earth Habitable

This short book, less than 200 pages of text, is a delight. It’s not clear for whom it is written. Falkowsky says it’s an outreach beyond that of a textbook. I doubt if most of its contents aren’t familiar in broad outline to most professional microbiologists, but I’m sure all of them would be rewarded by reading it. I was. It’s sprinkled with intriguing historical vignettes: when 22, Darwin, collected fossils with Adam Sedgwick in north Wales; Darwin took a copy of Charles Lyell’s Principles of Geology along with the King James Bible on the HMS Beagle; Robert Hooke learned Dutch to read Van Leeuwenhoek, and wonderful sentences like, “In 1859, the same year that Big Ben chimed for the first time and the London publisher John Murray and Sons sent the first edition of The Origin of Species to press, on the other side of the Atlantic an American train conductor, Edwin Drake, drilled the first major oil well near Titusville, Pennsylvania.”

Falkowsky has intriguing ideas about a variety of microbiological happenings. He pays considerable attention to the delay between the emergence of oxygenic photosynthesis and the great oxygenation event, discounting the impact of iron’s being the major oxygen sump (as he used to believe), emphasizing instead that of sulfur and nitrogen. The topics are eclectic. He discusses, among others, the age of Earth, origin of life, pansperma, lateral gene transfer, climate change, and the consequences of our burgeoning human population. He’s intrigued by the origins and impact of biology’s “nanomachines;” particularly photosynthetic reaction centers and membrane-bound ATP synthases. Perhaps the book ought better to be titled A Chat with Paul Falkowsky; that, too, undoubtedly a delight.

John Ingraham
University of California, Davis

Metabolism and Bacterial Pathogenesis

I recently had the pleasure of reading this wonderful book. Metabolism and Bacterial Pathogenesis came at the right time, because I work on a human-exclusive pathogen for which some strains collected from patients are auxotrophic, making me wonder: how is it that a pathogen that is very effective at surviving in humans requires one of the very amino acids that is limiting in humans? Every chapter in this book, directly or indirectly, suggested to me that the answer I am looking for may be very near, and what I have to do is to dig through some of the numerous references listed. These references are so limited that it made me recall the frequent editorial restrictions on references—clearly, the contributors were encouraged to freely discuss the details in depth. The contributors also suggest provocative and challenging new concepts, e.g. “pathometabolism.” This term encompasses the complex metabolic interactions between host and bacterial pathogen, concepts that could lead to novel antimicrobial therapeutics.

Every chapter is well written and entertaining (in contrast to what a reader might expect from description of metabolic pathways). Rather than simply a review of biochemical details, the metabolic pathways explain how a pathogen energetically or nutritionally overcomes the challenge of invasion and survival within mammalian cells. For instance, how Mycobacterium tuberculosis survives within a granuloma and why it can be called an “essential pathogen,” a concept that helps explain the epidemiology of tuberculosis. This pathogen must cause symptomatic disease (cough) in order to be transmitted, but its persistence in the community is favored by a long-term latent state until a susceptible population becomes available.

In short, this book provides an insightful collection of reviews that describe how basic metabolic processes play critical roles in virulence, unveiling fundamental (although often ignored) molecular mechanisms of infectious disease.

Guido Mora
Escuela de Medicina
Universidad Andrés Bello
Santiago, Chile
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Generous funding for OpenStax is provided by Rice University, the Laura and John Arnold Foundation, The William and Flora Hewlett Foundation, Bill & Melinda Gates Foundation, the Maxfield Foundation, the Calvin K. Kazanjian Economics Foundation and the Michelson 20MM Foundation.
Application Deadlines

American Board of Medical Laboratory Immunology (ABMLI) Certification. Certifies the expertise of doctoral-level scientists seeking to direct laboratories engaged in the immunological diagnosis of human disease. ABMLI certification is the highest credential available to practicing medical laboratory immunologists and is recognized under CLIA ’88 as one of the acceptable personnel requirements for high complexity laboratory directors. ABMLI certification is achieved by passing an online multiple-choice exam that is offered daily in the month of August at testing centers worldwide.

WWW: www.asm.org/abmli.

Deadline: 1 June 2016.

Undergraduate Faculty Research Initiative Fellowships. Early-career (and future) undergraduate STEM educators are encouraged to apply for a 2016 ASM-NSF LINK Undergraduate Faculty Research Initiative (UFRI) Fellowship. This nascent professional development resource trains STEM faculty to develop undergraduate research programs by initiating successful research partnerships. As part of the fellowship, LINK will provide travel subsidies of up to $2,000 to (i) increase participation of undergraduate STEM educators at eight eligible ASM-sponsored research conferences, (ii) encourage networking and collaborations with potential research partners, and (iii) access resources and mentoring to advance undergraduate research programs. Fellowship applications are accepted on a rolling basis for each of the eight conferences. The deadline is 6 June to be considered for a UFRI fellowship for the ASM Conference on Streptococcal Genetics or the ASM Conference on Experimental Microbial Evolution (each takes place in Washington, D.C.)

WWW: http://www.asm-link.org/ufri.


ASM Grant Writing Course. Senior-level graduate students, postdoctoral scientists, and early-career scientists are invited to apply for the ASM Grant Writing Course (formerly the ASM Kadner Institute). Sponsored by the ASM Committee on Graduate and Postdoctoral Education to meet the growing need for guidance and support on grant applications, the course will take place 12–14 August 2016 in Washington, D.C. The course will emphasize excellence in grantsmanship, and participants will receive in-person mentoring, real-time constructive feedback, and best practice strategies for composing effective grant proposals. ASM offers the Grant Writing Course with partial support from the ASM-NSF Leaders Inspiring Networks and Knowledge (LINK) Program and the Burroughs Wellcome Fund. For additional program details, such as costs and eligibility criteria, please visit http://bit.ly/asmgw16nl.


Deadline: 30 June 2016.

ASM Scientific Writing and Publishing Course. The ASM Committee on Graduate and Postdoctoral Education welcomes applications to the ASM Scientific Writing and Publishing Course, an effort that supports beginning researchers in understanding the writing, publishing, and review processes for scientific journals. Set for 12–14 August 2016 in Washington, D.C., the course is led by ASM members who have published widely, reviewed manuscripts, and served on the editorial boards of major journals. Program benefits include one-on-one feedback from facilitators, writing practice, and stimulating group discussions and interactions. The course is open to senior-level graduate students, postdoctoral fellows, and early-career scientists who are ready for an immersive and intensive writing experience. ASM offers the Scientific Writing and Publishing Course with partial support from the Burroughs Wellcome Fund. For additional program details, such as costs and eligibility criteria, please visit http://bit.ly/swpc16nl.


Deadline: 30 June 2016.
ASM Meetings Calendar

19–22 May 2016.
32nd Clinical Virology Symposium.
Daytona Beach, Fla.
www.clinicalvirology symposium.org

1 June 2016.
ASM Conference—Special President’s Edition:
What Does the Biology of Flaviviruses Tell us
About Zika: The Importance of Fundamental
Virus Biology.
Washington, D.C.
http://conferences.asm.org

ASM Microbe 2016.
Boston, Mass.
www.asm.org/microbe2016

31 July–3 August 2016.
ASM Conference on Streptococcal Genetics.
Washington, D.C.
http://conferences.asm.org/

4–7 August 2016.
2nd ASM Conference on Experimental Microbial Evolution.
Washington, D.C.
http://conferences.asm.org/

29 August–1 September 2016.
5th ASM Conference on Salmonella.
Potsdam, Germany.
http://conferences.asm.org/

9–12 September 2016.
6th ASM Conference on Beneficial Microbes.
Seattle, Wash.
http://conferences.asm.org/

ASM Conference on Infection and Cancer.
Washington, D.C.
http://conferences.asm.org/

11–14 December 2016.
ASM Conference on Antibacterial Development.
Washington, D.C.
http://conferences.asm.org/

About the Calendar

The ASM Meetings Calendar is provided as a service to readers of Microbe. It includes annual meetings and conferences organized by the Society. Detailed information for these events is published in the ASM Meetings and Conferences insert, which appears bimonthly in the center of Microbe.

As an added benefit of membership in ASM, an online calendar of microbiology-related meetings hosted by ASM and by other organizations is available through the ASM website. Any organization may submit items for the online calendar provided that submissions are of obvious interest to microbiologists. ASM will not permit announcements to appear in the calendar when the subject matter and dates conflict with ASM meetings or workshops. The calendar is located at https://info.asm.org/index.php/meeting-and-event-calendar. All entries in the online calendar are limited to conference name, dates, location, website, and contact information (person, address, telephone, fax, and/or e-mail). When websites and e-mail addresses are provided, links to them will be established. Because of the volume of submissions received, ASM staff is unable to provide proofs or other confirmation of receipt of each listing. Submit items for the online calendar through the “Add a new event/deadline” link on the Meeting and Event Calendar page.
Employment

POSITIONS AVAILABLE

Professor, Associate Professor, or Assistant Professor

The Department of Laboratory Medicine, University of Washington, is recruiting a full-time Professor, Associate Professor, or Assistant Professor without tenure in clinical microbiology on the Clinician-Educator or Physician-Scientist pathway. This would be a 12-month, multi-year appointment. University of Washington faculty engage in teaching, research, and service. The primary service responsibility will be to participate in the direction of one or more of the Department’s clinical microbiology laboratories. Additional responsibilities include the teaching of residents, fellows, medical students, and medical laboratory scientist program undergraduates, and development of a suitable area of research or scholarship. Documented experience is required directing clinical laboratories and in the clinical interpretation of microbiological testing results. Applicants must have an M.D., D.O., Ph.D., or foreign equivalent and be board-certified or board-eligible in clinical or anatomic pathology by the American Board of Pathology, in clinical microbiology by the American Board of Medical Microbiology, or in infectious diseases by the American Boards of Internal Medicine or Pediatrics. In order to be eligible for University sponsorship for an H-1B visa, graduates of non-U.S. medical schools must show successful completion of all three steps of the U.S. Medical Licensing Exam (USMLE), or equivalent as determined by the Secretary of Health and Human Services. Salary will be commensurate with qualifications and experience. Applicants should submit CV, contact information for five references, and a brief statement of professional goals to Brad T. Cookson, M.D., Ph.D., c/o Karen Walter, Box 357110, University of Washington, Seattle, WA 98195–7110 (kwalter@uw.edu). The University of Washington is an affirmative action and equal opportunity employer. All qualified applicants will receive consideration for employment without regard to race, color, religion, sex, sexual orientation, gender identity, national origin, age, protected veteran or disabled status, or genetic information.

Employment Advertising

Microbe is published monthly and available to nearly 40,000 ASM members and institutional subscribers. Lead time for employment ads is about 3 weeks. Microbe is mailed around the 8th of the month of issue, but the delivery date is not guaranteed. Please consider delivery dates when setting application deadlines.

ASM does not accept classified advertisements that indicate a limitation, specification, or discrimination on the basis of race, religion, national origin, sex, mental or physical disability, age, or any other matters which may not be lawfully considered in making employment decisions. Employment notices that discriminate against microbiologists on the basis of a particular board certification or doctoral degree will not be accepted. Such advertisements will be rejected unless it can be established that the position by state or federal law or regulation requires a specific board certification or doctoral degree.

Classified

Classified ads must be typed, double spaced, with normal sentence capitalization (capital and lowercase letters). Microbe cannot accommodate requests for extra capitalization, boldface type, or other text or layout enhancements in classified ads.

Include the name and telephone and fax numbers of a contact person for questions about your ad copy. Incorrectly typed ads or ads with application deadlines earlier than the 15th of the publication month requested cannot be guaranteed placement in that issue.

Deadlines: Your ad must be received by the 1st of the month before the publication month to ensure timely publication (e.g., to appear in the July 2016 issue, your ad must be received by 1 June 2016).

 Classified ads should be sent (with payment) to Walchli-Tauber Group, 2225 Old Emmorton Road, Suite 201, Bel Air, MD 21015, attn: Rhonda Beamer, tel. (443) 512-8899x106; fax, (443) 512-8909; e-mail, rhonda.beamer@wt-group.com.

Rates:
1–50 words $185  201–250 words $910
50–100 words $350  251–300 words $1,070
101–150 words $535  >301 words $3.45 per word
151–200 words $720

Internet posting: All classified line advertising printed in Microbe also appears on the ASM website. Ads are posted to the website shortly before the issue mailing date and remain on the site for approximately 1 month. Hence, line ads placed for an issue of Microbe will be available to ASM website browsers around the beginning of the month and will overlap their print appearance in the magazine. All ads are replaced with the close of the next issue.

For display ad Internet posting costs, please contact Rhonda Beamer at the address given above.

Display

Display advertising closes the 1st of the month preceding publication. For specifications, rates, and deadlines for display ads, contact Rhonda Beamer at the address given above.
Small Things Considered

Phage Responses to the SOS Response
by Merry Youle

Bacterial DNA can be damaged by UV radiation, toxins, or by DNA replication error, but bacteria have sophisticated mechanisms to detect and repair such damage. One widely conserved system is the SOS response, which activates a network of genes to repair the damage. A key agent in recovery is the recombinase RecA. When regions of single-stranded DNA (ssDNA) result from damage, they must be protected from attack by cellular nucleases. Such regions eventually become coated by RecA monomers that polymerize into long filaments wrapping around the DNA. The filament form of RecA (RecA*) drives increased expression of the more than 43 genes that comprise the SOS regulon through its interaction with the repressor LexA.

During normal growth, LexA represses expression of the SOS regulon. During the SOS response, LexA dimers embed deeply into the RecA* filament groove, altering LexA’s conformation so as to stimulate its autocatalytic cleavage, thus ending its repressor activity.

The host’s SOS response invites exploitation by phages, and indeed some do use signals from the SOS response to trigger a switch from lysogeny to lytic replication as they hasten to assemble progeny while the host can still support phage replication. For example, phage λ, which infects E. coli, silences genes involved in replication and host lysis during lysogeny using its repressor CI. CI is similar to LexA in structure and function and is also prompted by RecA filaments to self-cleave, thereby flipping the switch from lysogeny to lytic replication.

Another mechanism for exploiting the SOS response has been found in Bacillus thuringiensis phage GIL01. Its prophage does not integrate during lysogeny into the host chromosome, but instead is maintained stably as an independent linear prophage. Nevertheless, GIL01 replication is induced when UV or mitomycin C (MMC) treatment elicits the SOS response, and this induction requires RecA. Researchers sought, but were unable to find, a phage-encoded repressor comparable to λ’s CI.

To pursue this puzzle further, they isolated clear plaque (CP) mutants that cannot establish lysogeny. Some mutants mapped to a region named dinBox1 (damage-inducible Box1), which was similar in sequence to the LexA-binding sites (SOS boxes). Introducing point mutations in this region abolished lysogeny. Did GIL01 perhaps use the host’s LexA as its repressor? To test this, the researchers generated a host strain with a noncleavable LexA. As expected, these cells were hypersensitive to DNA damage, since cleavage of the LexA proteins is necessary to mount an SOS response. Cleavage of LexA was also found to be necessary to induce the GIL01 prophage. Based on these and other experiments, the researchers concluded that LexA directly represses the lytic pathway for GIL01 by binding to dinBox1, an SOS box in the prophage genome. Treatment with MMC triggered LexA cleavage, inducing the GIL01 prophage to replicate and ultimately lyse the cell.

But this is not the whole story for GIL01, since other GIL01 CP mutants mapped to an ORF encoding Gp7 (gene product 7), suggesting it was required for lysogeny. They found that Gp7 formed a stable complex with LexA, thereby inhibiting LexA self-cleavage and repressing prophage induction. Gp7 also makes LexA bind more firmly to host SOS boxes, impeding the SOS response.

In subsequent experiments the author, Nadine Fornelos, found that overexpressing gp7 alone in B. thuringiensis actually increased cell survival by up to 10-fold following MMC treatment. To explain these surprising results, she looked for clues in the cellular genome and found a chromosomal prophage (CP) encoding a LexA-like repressor, as does phage λ. She noted that in her earlier experiments she induced GIL01 replication with a low concentration of MMC, while in the recent Gp7 expression studies she used a higher MMC concentration that markedly reduced host survival. She tentatively concluded that at the lower MMC concentration, GIL01 replicated without interference from the repressed CP. At the higher concentration, the CP was induced to replicate and lyse the cells. Gp7 expression inhibited this induction of CP, leading to increased cell survival. These studies make Gp7 the first phage factor known to interact directly with LexA to modulate both prophage induction and the host’s SOS response.

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