A New Gap in the Antibiotic Arsenal

Not long ago, to demonstrate how staphylococcus bacteria invade the body, instructors would have medical students roll up their sleeves and smear the microbes on their forearms. The instructors then pricked the skin. Within days, the students developed infections—which the instructors treated with antibiotics. Point made.

They don't do that anymore.

Like other bacteria, some staph strains are showing resistance to many antibiotics. Indeed, concerned scientists have kept an uneasy vigil, watching for a microbe that would repel all drugs.

Now, a strain of staphylococcus infecting a 4-month-old Japanese boy has withstood a pharmaceutical onslaught from vancomycin, physicians' lone remaining surefire drug against the bacterium. The microbe, a strain of *Staphylococcus aureus*, had already acquired resistance to every other antibiotic.

The unsettling news that vancomycin had failed after 29 days of treatment has the federal Centers for Disease Control and Prevention (CDC) in Atlanta scrambling to warn U.S. hospitals to redouble their vigilance against the spread of staphylococcus.

The report has also sent a shudder through the research community, which some scientists claim has become complacent about resistant microbes.

At worst, the *S. aureus* could signal the rise of a microbe that no drug on the U.S. market can handle.

The CDC terms the Japanese infection "intermediate resistance"—somewhat short of full resistance, a medical red alert. Nonetheless, scientists consider the case ominous. *S. aureus* is a potentially lethal staph that occurs naturally in humans, residing on the skin and along the mucous membranes. This opportunistic microbe typically needs a break in the skin to invade the body. It can form an abscess at that spot or travel in the bloodstream to infect kidneys, bones, or other tissues.

Vancomycin routinely stops it and probably still will for a time, says Stuart Levy, director of the Center for Adaptation Genetics and Drug Resistance at Tufts University School of Medicine in Boston. However, Levy and others expect the resistant strain to spread.

"Bacteria do not sit around," he says. "They are moving targets."

The fact that this strain was found in only one individual "doesn't make any difference," says Anthony S. Fauci, director of the National Institute of Allergy and Infectious Diseases. "It's not a question of whether it's going to happen here, it's a question of when."

Despite a recent research push, the U.S. pharmaceutical industry could be caught flat-footed. Work on new antibiotics slowed in the 1980s and early 1990s. It can take a decade to bring a new antibiotic to market (SN: 5/17/97, p. 310).

"We have a few drugs in the pipeline that appear effective against vancomycin-resistant microbes," Fauci says. "But they are only in the development stage."

The staphylococcus group of bacteria is especially hardy and difficult to wipe out with normal precautions. Hospitals fight a running battle against the microbes, which can withstand hot water and some disinfectants, says Gail H. Cassell, a microbiologist at the University of Alabama at Birmingham.

Scientists have worried that staph would gain resistance by swapping genes with other bacteria. Of the enterococci, less virulent bacteria that inhabit the intestines but seldom cause problems in healthy people, as many as one in four strains has proved resistant to vancomycin, report researchers at the University of Pennsylvania Medical Center in Philadelphia in the Feb. 19 JOURNAL OF THE AMERICAN CHEMICAL SOCIETY.

Instead of swapping genes, *S. aureus* has apparently become resistant by mutating its own genes, Levy says. Exactly how *S. aureus* did so remains unknown.

Thus far, the story in Japan has a happy ending. Doctors gave the boy a combination of other drugs—including some that aren't commercially available in the United States—and he warded off the infection. Japanese officials are withholding details, awaiting publication of a report on the case in the July JOURNAL OF ANTIMICROBIAL CHEMOTHERAPY. *—N. Seppa*

Patients savor this brain disorder

Swiss researchers report finding a new brain disorder in a small percentage of people who have suffered strokes, brain tumors, and head traumas. In each case, the damage has produced a persistent behavioral effect. Yet none of the victims desires a cure. Indeed, they're enjoying the fallout: a craving for fine foods.

Marianne Regard of University Hospital in Zurich and Theodor Landis of Geneva University call this benign disorder gourmand syndrome.

Regard first encountered the condition 8 years ago in a 48-year-old political journalist who had been hospitalized with a stroke. Scans of the man's brain identified a lesion around the middle cerebral artery in the right hemisphere. The wound produced a temporary weakness on the left side of his body, making him unable to walk. Even so, Regard recalls, "he didn't complain about that." Instead, he griped about hospital meals.

"Since most people complain about hospital food, we initially took no notice," the neuropsychologist admits. But when she asked him to keep a diary of his thoughts, the man exhibited an inordinate preoccupation with food. Before the stroke, he had had an overwhelming interest in politics and had shown no particular food preferences. Afterwards, he lived for food. Indeed, as soon as he returned to work, he abandoned politics to become a columnist on fine dining.

When she observed a businessman hospitalized for stroke who also exhibited a newfound "lusting" after food, Regard says, she began investigating the role of the brain damage. After studying 723 patients suspected of having a discrete lesion in the brain, she and Landis identified 34 more instances of gourmand syndrome. Each patient had brain damage, usually in the right frontal region.

What constitutes fine food has proved "very individual," Regard says, with no single cuisine or taste—such as sweet or salty—driving the compulsion.

Most patients exhibited additional symptoms at first, such as spatial memory problems or diminished control over impulsive behaviors. During 8 years of follow-up, these symptoms disappeared for the most part, but the passion for food remained. Although preoccupied with shopping, dining rituals, and food preparation, the patients did not become overweight.

Many types of brain damage have been linked to altered eating behaviors, from insatiability to anorexia, but none seems to stem from this region of the brain, the authors note in the May NEUROLOGY.

The new findings "coincide with work that we and others have done with brain degenerations that affect primarily the right side of the brain," says neurologist Jeffrey Cummings of the University of California, Los Angeles School of Medicine. Some patients, he's found, experience a behavior-altering "heightened sensitivity" to and appreciation for particular stimuli. *—J. Raloff*