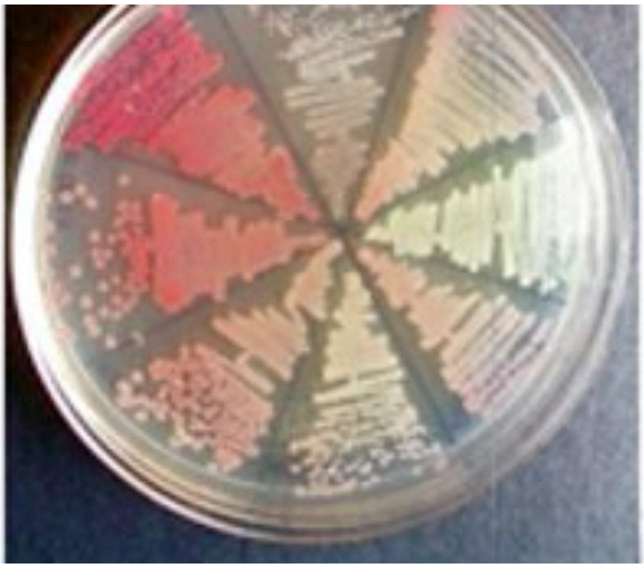
THE SCIENCES

Miraculous Microbes: They Make Holy Statues "Bleed"--and Can Be Deadly, Too

A sinister bacterium implicated in Catholic miracles and "blood"-tainted polenta also kills coral, insects, and are even are up to no good in your contact lens case

By Jennifer Frazer on November 11, 2011



Credit: Robert Shanks, University of Pittsburgh School of Medicine

The Killer Bacteria Hall of Fame no doubt houses the usual suspects: *Yersinia pestis,* perpetrator of the Plague; *Treponema pallidum,* the spiral-shaped culprit in syphilis; and *Vibrio cholerae*, the swimmer that causes cholera. But you have probably never heard of one of the inductees.

Serratia marcescens is a forgotten but ubiquitous bacterium that can produce a red pigment called prodigiosin and likes to hang out as a pink film in the shower grout and toilet bowls of less-than-scrupulously clean homes. The pigment is so persistent that giant amoebas called slime molds that dine on *S. marcescens* turn red just as flamingoes that eat shrimp turn pink. Yet the picture emerging of this unsung organism is increasingly sinister.

These bacterium first attracted scientific attention in early modern times when it was found oozing out of damp Italian statues, communion wafers and, of all things, polenta doing its best impersonation of "blood." And blood it was taken to be—usually miraculously—until a pharmacist named Bartolomeo Bizio started trying to get to the bottom of what peasants declared to be an outbreak of diabolically cursed polenta in 1819.

Bizio believed a microorganism was responsible. In the test chamber, he found the bacterium happily chowing down on polenta while cranking out red pigment. Believing it to be a fungus, he named it *Serratia* in honor of Italian physicist Serafino Serrati, and *marcescens* because of the pigment's tendency to fade or decay rapidly.

Fast forward to the middle of the 20th century. In the early 1950s the U.S. government decided it would be a good idea to use *S. marcescens* in a bioweapon dispersal experiment dubbed Operation Sea-Spray. They burst balloons filled with *Serratia* over San Francisco Bay. Chosen because the red pigment makes it easily traceable, the supposedly innocuous bacterium so generously sprinkled over the bay was subsequently linked to several respiratory infections and at least one death.

Since then the bacterium has been widely found to be an opportunistic human pathogen, capitalizing on its prowess in forming tight-knit surface communities called biofilms wherever it can. It infects urethras through catheters, lungs via respirators, and premature babies by way of hospital caregivers. *S. marcescens* turns out to be one of the top 10 causes of all hospital-acquired respiratory, neonatal and surgical infections, said Robert Shanks, associate professor of ophthalmology at the University of Pittsburgh School of Medicine, who studies *S. marcescens*.

It has also been found irritating or infecting the corneas of contact lens wearers who fail to clean their cases with enough diligence (or at all). "What I think is sort of strange about *S. marcescens* is so many people have them in their contact lens cases," said Regis Kowalski, an ocular microbiologist at the University of Pittsburgh Medical Center. Although it often lives there harmlessly, *S. marcescens* is the third-most common cause of ocular keratitis, a corneal infection usually caused by poorly cleaned cases.

Disturbingly, the bacterium also seems to thrive in soap and other aggressive cleaners. In your hand soap, you might have *Serratia* living in it," Shanks said. "We actually had a bottle of Triton X-100 that was contaminated with it. It was really hard to believe because it's a strong detergent." The many hospital outbreaks of *Serratia*, he added, are almost always traced to contaminated cleaning solutions.

Yet *S. marcescens* also has a benign side. Certain strains are a normal, harmless component of mammalian guts, water and soil, and probably pose little risk to your average healthy human who cleans his or her lens case properly.

But *Serratia* has doubled down the menace in the past few years. Just this year, nine patients died and another 10 were sickened in Alabama by feeding tubes and bags contaminated with *S. marcescens*. And you may recall the debacle that ensued in 2004 when Chiron Corp. had to deep-six some 48 million doses of flu—half that year's —at the beginning of the flu season due to unspecified contamination. The contaminant? *Serratia marcescens*.

And lately, rampaging *Serratia* have turned up in some even less expected places. In 2002 scientists discovered the "white pox" pathogen devouring elkhorn coral in the Caribbean was none other than *S. marcescens*. Although *Serratia* is a common inhabitant of beaches, canals and some shore-dwelling animals, it is not typically found in seawater, so discovering it there was a surprise, said Kathryn Sutherland, associate professor of biology at Rollins College in Winter Park, Fla., who unmasked the pathogen.

After extensive testing of *Serratia* strains from nearly every conceivable source, Sutherland and her colleagues concluded that the coral-killing strain was an exact match with one of the many strains found in human excrement. In a paper published in *PloS ONE* in August, they showed that this strain of bacteria experimentally caused white pox on elkhorn coral infected in the lab (although other factors such as another pathogen, pollution and rising water temperatures may also contribute to the disease, she noted).

Released from leaking septic systems ill-suited to the local geology of the Florida Keys, the bacterium by chance happened to be able to both survive in saltwater and dine on elkhorn coral, an unhappy accident for both us and it, because about 90 percent of the species in those waters have vanished in the last 15 years. "I call them elkhorn graveyards," she said of the ghostly fields of departed coral.

The story does not stop there. In 2010 scientists reported a bacterium from the genus *Serratia* partnering with microscopic roundworms called nematodes from the genus *Caenorhabditis*—the genus to which *C. elegans*, a much-loved experimental subject, belongs—to take out insects. Recent research had already indicated *C. elegans* was not the sweet little free-living soil dweller scientists may long have thought they had cultured. Instead, nematodes in this genus make a living by hitching rides on insects to travel between food sources or by living on them and patiently waiting for them to die so they can feast on the corpse.

But a chance encounter revealed a darker story. Discovered accidentally in a wax moth larvae–baited nematode trap in South Africa, scientists discovered a new species of roundworm called

C. briggsae had partnered with a species of *Serratia*. In these sorts of relationships, which also occur in other nematode genera, symbiotic bacteria are carried inside the nematode's digestive tract, sometimes in pouches especially for the purpose.

Bacteria-loaded nematodes invade an insect through its own digestive openings or cuticle pores. Once inside, the roundworms release the bacteria, which start releasing toxins. "The bacteria does the work of the killing and changing the whole thing into a septic soup," said Eyualem Abebe, a biologist at Elizabeth City State University in North Carolina and lead author of the study that discovered the Serratia–Caenorhabditis partnership. The nematodes, in turn, feast on the bacteria in an arrangement that could be looked at as a twisted agricultural scheme. Intriguingly, the researchers also found that by adding the requisite strain of *Serratia* to five other non–insect infecting *Caenorhabditis* species—including the venerable *C. elegans*—they were able to turn all these freeloaders into killers.

How is it that *Serratia* can survive in so many different environments and opportunistically infect so many unrelated hosts? Shanks thinks it is because *Serratia* is a classic bacterial generalist. It has a big genome packing enough genes to consume practically any carbon (food) source and to resist virtually any antibiotic—traits acquired through countless generations of selection in bacterial soil wars. "It's got so many enzymes it can eat just about anything," he said.

Which brings up one final question: Just what is that red pigment that *Serratia* sometimes secretes, and why does it make it? Until recently, few had bothered to investigate that question. The research of Pryce Haddix, associate professor of biology at

Auburn University at Montgomery, suggests the bacterium may be using the pigment to slow energy production in the form of ATP (adenosine triphosphate) and limit damage from free radicals caused by oxygen's presence during ATP synthesis as it prepares for rest or dormancy, he said.

But why red? Does the bloody hue have a physiologic purpose or is the bacteria's sinister appearance merely a chemical coincidence? "That's an excellent question," he said. "I don't have a clue really."

ABOUT THE AUTHOR(S)

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Jennifer Frazer is a AAAS Science Journalism Award-winning science writer. She has degrees in biology, plant pathology/mycology, and science writing, and has spent many happy hours studying life *in situ*.

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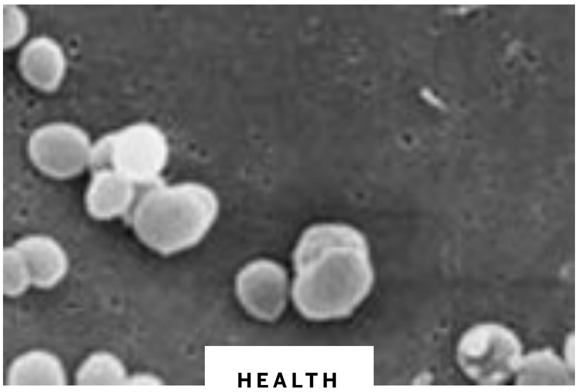

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