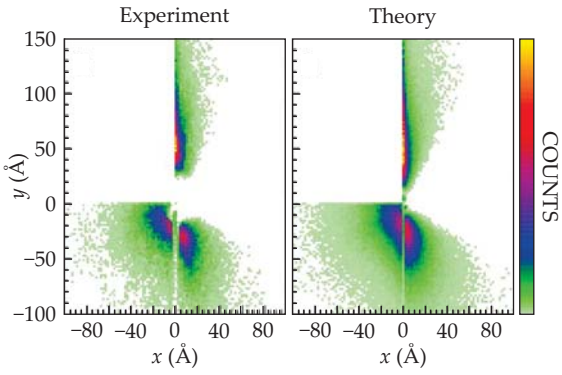


**Figure 3. The structure** of the helium Efimov trimer is an elongated triangle, with one atom far removed from the other two. The panel on the left shows the experimental data: For each ionization event that was identified as an Efimov trimer, the reconstructed structure was positioned so that its center of mass was at the origin, the axis of its smallest moment of inertia lay along the vertical axis, and the upper left quadrant was empty. The panel on the right shows a simulation of the same process using the theoretically predicted Efimov structure. (Adapted from ref. 3.)

structure. As Dörner asks, "Why should three identical particles form such a highly asymmetric system?" Dörte Blume (Washington State University), who provided theoretical support for the project, points out that the experiments agree well with the structure that theorists have known about for years. "However, a lot of researchers have been studying the Efimov effect without analyzing or appreciating the structures," she says. "My hope is that this will now change."

## Next steps

Having gotten a first glimpse of the Efimov state, Dörner and colleagues next want to study its dynamics. Unlike ordinary molecules—but like the dimer and ground-state trimer—the Efimov trimer has no rotational or vibrational substates. Any attempt to set its constituent atoms in motion will tear it apart. Instead, the researchers plan to create coherent superpositions of the ground-state and Efimov trimers and look at how their wavefunctions change with time.

They also hope to study larger systems. Theory predicts that the Efimov effect should have an impact on clusters of four or more particles. (See the article by Chris Greene, PHYSICS TODAY, March 2010, page 40.) The He tetramer, for example, should have both a ground state and an excited, Efimov-like state. And Dörner and his group are looking for them.

**Johanna Miller**

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# Mechanical rupture explains a bacterium's puzzling "pop"

**Experiments suggest that dividing *Staphylococcus* cells are designed to crack under pressure.**

*Staphylococcus* bacteria undergo cell division roughly once every 30 minutes. But when the time comes, the actual splitting of one cell into two happens in less than the blink of an eye—about a millisecond, to be precise. The event, known as "popping," is too swift to be explained by the enzyme-mediated processes thought to govern conventional cell division. Now Julie Theriot and coworkers at Stanford University have made the case that popping is driven not by biochemistry but by mechanics.[1]

As a *Staphylococcus* cell prepares to divide, it constructs a double-layered septum that partitions the spherical cell into two hemispheres—the soon-to-be daughter cells. Theriot and her colleagues postulate that as the cell contin-

ues to grow, stress builds along the peripheral ring where the septum meets the cell wall until, eventually, the ring cracks. As the crack grows in both directions around the cell's circumference, the daughter cells tear away from one another, each taking a layer of the septum with it. Based on previous measurements of crack-propagation speeds in synthetic analogues of cell walls,[2] such a rupture could easily account for popping's short time scale.

Although the popping events happen too quickly for the Stanford team to image them in detail, Theriot's graduate student Xiaoxue Zhou was able to use a fluorescence imaging technique known as structured illumination microscopy to investigate how the wall of a parent cell evolves during the division process. She labeled a population of *Staphylococcus aureus* cells with wheat germ agglutinin, a fluorescent protein that binds to the cell's wall but can't penetrate it, and then imaged 40 popping events.

After each event, the old cell wall appeared to have hinged open like a clam shell, consistent with division mediated by a single propagating crack. In all but one case, the two halves remained momentarily connected at the hinge point. Scanning electron microscope images such as those shown in the figure below painted a similar picture of *S. aureus*'s division process. It's likely that the other members of the *Staphylococcus* genus divide in the same manner.

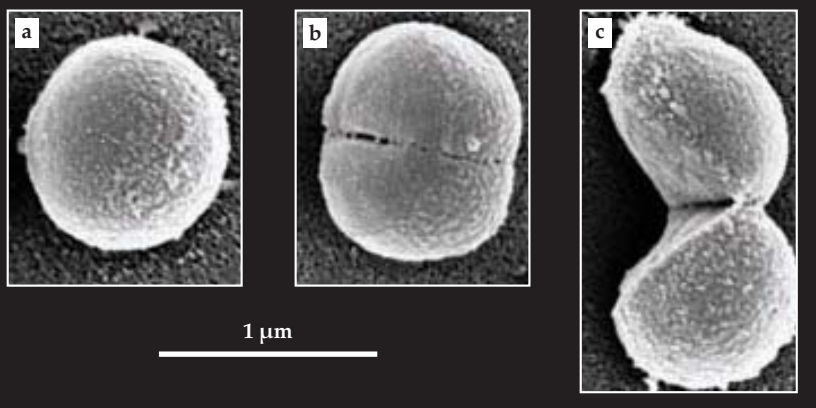
Enrique Rojas, a postdoc in Theriot's lab, captured further experimental evidence of popping's mechanical origins: He found that he could induce popping in a colony of *S. aureus* by suddenly diluting its growth medium. Such dilution elicits a sharp jump in the cells' turgor pressure, the outward pressure on the cell walls, as the bacteria take up water to restore their osmotic balance. The manipulation also worked the other way around; Rojas could suppress popping by rapidly concentrating the growth medium.

## Stretching and necking

At tens of nanometers, the walls of *Staphylococcus* cells are thick by microbial standards. It's unlikely that turgor pressure alone would cause them to tear. The figure's panel b, which shows a cell on the verge of popping, hints at a possible explanation. It reveals nanometer-sized perforations along the ring where the cell will eventually split. Presumably, those weak spots serve as potential nucleation sites for cracks. Might the perforations be the work of enzymes?

According to the Stanford group, probably not. Although *Staphylococcus* cells often use hydrolase enzymes to break down and remodel their cell walls, the researchers found that mutants lacking *atl*—the gene that encodes the bacterium's main hydrolase—popped at the same rates and exhibited the same pattern of perforations as cells that had the gene.

The researchers think the perforations are more likely attributable to necking, a generic mechanical instability in which a band of ductile material, stretched taut, thins and weakens at random, localized regions along its length. The team's numerical simulations suggest that during the late stages of a *Staphylococcus* cell's division cycle,




**Scanning electron microscope** images capture *Staphylococcus aureus* cells **(a)** well before, **(b)** just before, and **(c)** just after division. The bacterium's split is swift; it takes only a millisecond or so for a single cell to separate into two. That process, known as popping, is thought to be mediated by a stress-induced crack in the cell wall. (Adapted from ref. 1.)

the tension along the ring joining the septum with the cell wall may indeed be great enough for the instability to kick in.

The proposed mechanism could help to explain another of *Staphylococcus*'s morphological mysteries—the tendency of its cells to cluster not into neat arrays but into irregular, grapelike bunches.[3] Because the location and orientation of the hinge axis is random from cell to cell, so are the relative arrangements of each daughter-cell pair.

Cell division by mechanical rupture may not be unique to *Staphylococcus*. "We looked at *Micrococcus*, and they seem to pop as well," Theriot says. "If you talk to people who study *cerevisiae*, a budding yeast, they all talk about a point in cell division called cracking, where the bud cracks off. *Mycobacterium*, which causes tuberculosis, does something very similar."

Theriot sees those and other examples as a sort of cautionary tale: "There's often a starting assumption that complex biological processes—such as a cell deciding when and where to divide—must involve genes and proteins, so we often try to inhibit unwanted processes by targeting the genome." But for mechanically driven phenomena such as popping, she says, that approach wouldn't necessarily work. "In these cases, the cell is simply taking advantage of the fact that it's a material object. It's just obeying the laws of physics."

**Ashley G. Smart**

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# Artificial eardrums get real

**Three-dimensional printing can fabricate polymer scaffolds that mimic the orientation of collagen in the tympanic membrane.**

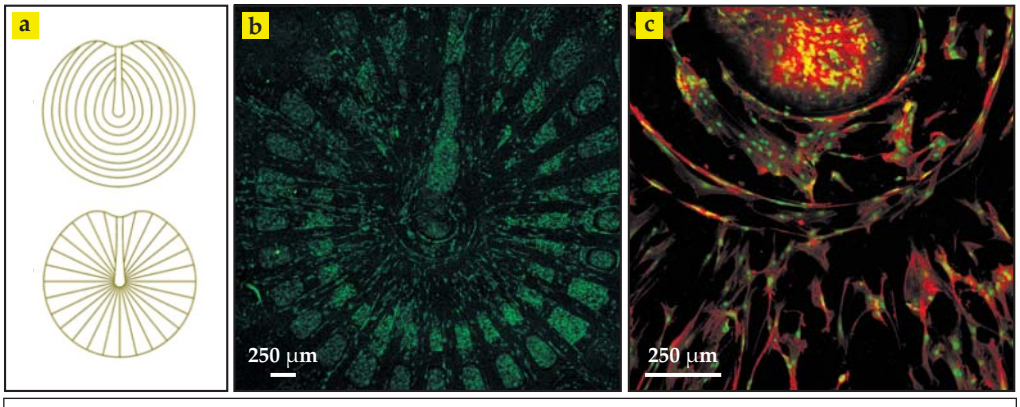
The human eardrum is astonishingly sensitive to sound. At the threshold of hearing, it vibrates with an amplitude of mere picometers. That's on a par with the membrane's spontaneous oscillation from the thermal motion of air molecules bouncing off it and water molecules sloshing around inside the cochlea, the ear's frequency analyzer and amplifier (see PHYSICS TODAY, April 2008, page 26). At the upper limit of hearing—near the pain threshold from a roaring jet engine, for instance—the eardrum vibrates through just tens of microns. For the 0- to 120-dB sound-pressure levels we normally experience, the range of vibratory motion spans six orders of magnitude.

Although typically less than 100 μm thick, the eardrum, otherwise known as a tympanic membrane, also serves as a protective barrier to the outside world. And its linkage to the chain of bones in the middle ear forms a mechanical lever that transmits sound waves from air to the liquid in the inner ear by reducing the impedance mismatch between the two media. Were it not for that mechanism, most of the energy in sound waves that enter our ears would simply be reflected.

Fortunately, most perforations of the eardrum, whether from a loud blast, say, or a rogue cotton swab, heal by themselves. But patients with chronic pathologies must endure surgeries that graft fascia (thin connective tissue), fat, or pieces of cartilage onto the eardrum—or replace it entirely—to serve as a scaffold for new tissue. The history of such intervention goes at least as far back as the 17th century, when a piece of pig bladder was the artificial eardrum of choice. And later physicians tried their luck with, among other options, vulcanized rubber, lint, tin or silver foil, or moistened cotton wool that a patient could insert daily, much like contact lenses.[1]

Not surprisingly, even today's replacements are sometimes poor substitutes. Good hearing requires a tympanic membrane that is light and elastic, yet strong enough to buffer potentially large static pressures. Our eardrums achieve those properties through a complex arrangement of collagen fibers that grow circumferentially in one of the central layers of the membrane and radially on an adjacent layer. The fibers' near-orthogonal orientation is more likely to resist tears and lesions than an isotropic one. And the high concentration of stiff radial tissue at the drum's center optimizes the transmission of sound waves to the middle ear's ossicular chain.

Carlos Mota (now a postdoc at Maastricht University in the Netherlands), Serena Danti (one of his PhD advisers at the University of Pisa in Italy), and their colleagues have now constructed polymer scaffolds that mimic




**Synthetic scaffolds.** The collagen fibers on adjacent layers of the human eardrum follow different patterns, **(a)** circular and radial. Carlos Mota and colleagues superposed the two patterns to form a 300-μm-thick polymer grid and topped it with a much thinner elecrospun mesh of the same material for growing biological tissue. The scaffold was seeded with human stem cells and bathed in a culture medium. **(b)** After eight days, it was stained and imaged using confocal microscopy. The green fluorophores reveal networks of cellular nuclei in the mesh between the grid lines. **(c)** This confocal microscope image of another scaffold stained with both green and red fluorophores reveals connective protein filaments known as f-actin. Near the center of the scaffold, the cell nuclei (green) and elongated protein filaments (red) appear to follow circular and radial outlines. (Adapted from ref. 2.)