

PHYSICS

Triplets Proliferate

If a normal material is sandwiched between two superconductors, Cooper pairs—the electron pairs of opposite spin that cause superconductivity—can tunnel through it, forming a supercurrent. If, however, the middle material is a ferromagnet, it will tend to align the spins of the electron pairs, drastically reducing the supercurrent. One way around this is to use an inhomogeneously magnetic material, which can help to create a triplet supercurrent, where the pairs have parallel spin—a phenomenon rarely observed in natural materials. Klose *et al.* show that by applying an in-plane magnetic field to a junction with two magnetic materials, Co and Ni, the value of the supercurrent can be increased up to 20 times. When the magnetizations of the junction materials are parallel, no triplet supercurrent should be observed; the more orthogonal they are, the larger the supercurrent. Polarized scattering and microscopy experiments showed that the in-plane field caused a spin-flop transition in the Co layers, enhancing orthogonality with the Ni layers and thus increasing the supercurrent, making it possible to control these junctions with external magnetic fields. — JS

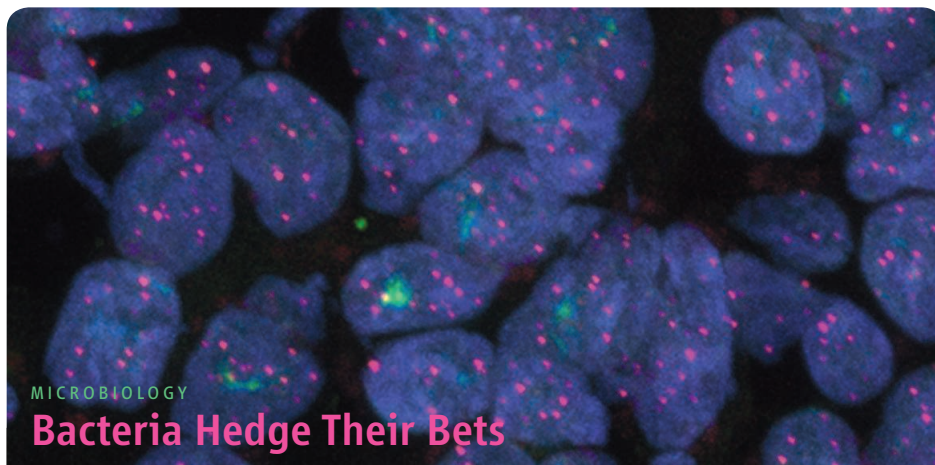
Phys. Rev. Lett. **108**, 127002 (2012).

MOLECULAR BIOLOGY

No Fix for Broken Ends

Cells that suffer DNA damage temporarily stop dividing until the damage has been repaired or removed. If DNA damage cannot be repaired, cells enter a state of cellular senescence to avoid progressing to a tumorigenic state. Cellular senescence, however, has also been causally linked with aging.

In an effort to understand possible sources of persistent DNA damage, Fumagalli *et al.* show that our genomes are differentially susceptible to being repaired. Although senescent cells are capable of repairing damage, they cannot do so when the damage is localized near the ends of our linear chromosomes. Chromosomes are capped by specialized structures known as telomeres, which consist of multiple DNA repeats bound by a protein complex (shelterin) that protects the ends from inadvertently being recognized as DNA damage. Indeed, the presence of telomeric DNA repeats or one of the shelterin proteins (TRF2) near DNA damage interferes with the normal repair process, which results in persistent DNA damage signaling. Persistent damage also accumulates at telomeres (which themselves have



MICROBIOLOGY

Bacteria Hedge Their Bets

Antibiotic treatment may be thwarted by bacteria, not only because molecular mechanisms of resistance are acquired and selected, but also because persister cells withstand the onslaught by becoming dormant. We know persister-cell formation occurs as bacterial populations move from log to stationary-phase growth, but little else. Vega *et al.* have implicated the bacterial signalling molecule indole in the enhancement of persistence to various antibiotic treatments by monitoring the formation of persisters and their transcriptional responses to indole. Gene expression in the oxidative stress regulator and phage-shock pathways increased, but drug efflux systems did not, suggesting that molecular antibiotic resistance pathways were not being induced in these conditions. Knocking out these genes resulted in less persistence. Activation of these responses, via indole signalling induced by low-level antibiotic exposure or other stress, may thus prepare a proportion of the bacterial population for further unpredictable and potentially harmful events—a form of “bet hedging.” — CA

Nat. Chem Biol. **8**, 10.1038/nchembio.915 (2012).

not become critically shortened) in aging baboons, linking DNA damage at repair-resistant telomeres with aging. — GR

Nat. Cell Biol. **14**, 10.1038/ncb2466 (2012).

MICROBIOLOGY

Cave of Forgotten Fungi

A fungal outbreak has been threatening the 15,000-year-old Paleolithic rock art found in Lascaux Cave, France, since 2001, when intrusive white mycelia growths were first discovered in the cave. Unfortunately, the situation only worsened after varying biocide and mechanical treatments that removed the white overgrowths but resulted in the appearance and spread of large black fungal stains on the walls. Martin-Sanchez *et al.*



isolated the most abundant species of fungi growing in the black stains between 2008 and 2011, before and after biocide treatments, to determine how the community structure has changed over time. Clone libraries constructed from samples taken in 2008 showed that fungal communities were dominated by one species; however, after treatment, fungal diversity substantially increased. In 2010 and 2011, the communities were again different, with the new

community dominated by, among others, closely related black yeasts in the family Herpotrichiellaceae. Thus, the biocide treatments were at least partially responsible for the new fungal outbreaks. Careful testing of treatment methods or limiting tourism activities near Lascaux and other cave paintings (see Saiz-jimenez *et al.*, Policy Forum, 7 October 2011, p. 42) may hopefully prevent a similar fate for other caves. — NW
Environ. Sci. Technol. 10.1021/es2040625 (2012).