# Treatment blocks sites for dental bacteria

Armed with toothbrushes, toothpastes, and floss, people wage a daily war against cavity-causing bacteria. Now, researchers in England have found another way to defeat those microscopic foes. Teeth treated with a new synthetic molecule remain free of the feared bacteria for up to 4 months, they report.

Most cavities are caused by the bacterium *Streptococcus mutans*, which binds to receptor proteins on the surface of teeth and collects into the film of plaque that dentists warn their patients about. Unlike other bacteria in the mouth, *S. mutans* produces lactic acid, which erodes tooth enamel.

"If you can prevent infection with *Streptococcus mutans*, you will actually prevent tooth decay," says Charles G. Kelly of the Guy's, King's, and St. Thomas' Hospitals Medical and Dental School in London.

Kelly and his colleagues pursued this goal by creating a peptide, or short sequence of amino acids, that blocks the receptors and thus prevents *S. mutans* from sticking to teeth. Earlier work had shown that *S. mutans* possesses a large protein, called adhesin, that binds to receptors. Kelly's team identified and synthesized a critical 20–amino acid portion of adhesin that, in the test tube, successfully binds to the receptors. The researchers then tested how well the synthetic peptide prevents *S. mutans* from colonizing human teeth.

The researchers first treated three groups of four volunteers with an antiseptic mouthwash for 9 days to remove all microbes from their mouths. Over the following 3 weeks, a solution containing the peptide was dripped twice a week onto the teeth of one group, which also used a daily mouthwash with the peptide. The other two groups received similar treatments with a different peptide or no peptide at all. The researchers then monitored growth of *S. mutans* on the volunteers' teeth.

Those who received the binding peptide remained free of *S. mutans* for at least 3 months. The bacteria appeared on the teeth of the others within 3 weeks, however. Kelly and his colleagues report their findings in the January NATURE BIOTECHNOLOGY.

After treatment, the peptide remains in the mouth for only about 6 hours, Kelly says, but it appears to exert long-term antimicrobial effects. "If you can hinder [*S. mutans*] colonization initially, other bacteria occupy the niche," Kelly says. Plaque formed by harmless bacteria acts as a protective film, crowding out the acid-producing *S. mutans*.

The results of the study are "quite striking," says Randall T. Irvin of the University of Alberta in Edmonton. "If this is indicative of what will happen in a larger group, it's encouraging." He expects that bacteria subjected to this treatment would evolve resistance less readily than when attacked with antibiotics.

This approach could be applied to other microbial targets, Irvin says, "but it will take a lot of work." Receptor binding often triggers normal cell processes, so the peptides would have to be designed to deflect bacteria without interfering with those effects. *—C. Wu*

# Hormone helps ring internal alarm clock

Some people find setting an alarm clock a waste of time—they can somehow simply decide when to wake up. This curious ability has been noted for at least 100 years, says William Moorcroft of Luther College in Decorah, Iowa, who in 1997 published a study documenting the phenomenon.

A hormonal surge that begins about an hour before a person's anticipated awakening may play a role in this enviable talent, Jan Born of the University of Lübeck in Germany and his colleagues now report in the Jan. 7 NATURE.

"Born and colleagues have provided the first evidence of a biological basis for what may be an internal alarm clock," says Mark R. Opp of the University of Texas Medical Branch in Galveston.

Like Opp, the German researchers investigate how certain hormones, such as cortisol, adrenocorticotropin (ACTH), and corticotropin-releasing hormone, help a body react to or prepare for stressful events, which include awakening. During sleep, the amounts of ACTH and cortisol in the blood gradually increase.

In people who are awake, anticipation of a stressful event can prompt the release of these hormones. Born's team wondered whether expecting to get up at a particular time influences the secretion of the hormones during sleep.

The scientists monitored 15 volunteers during three nights of sleep. For one night, the volunteers were told they could sleep until 6 a.m. The other two nights, they were informed that someone would rouse them at 9 a.m.—well beyond their normal wake-up times. On one of those nights, however, they were unexpectedly awakened at 6 a.m.

From blood samples drawn every 15 minutes while the volunteers slept, the scientists found that the amount of ACTH usually began to rise sharply around 4:30 a.m. in the people expecting to get up early. The hormone's concentration only increased gradually in the people awakened by surprise and in those who slept until 9 a.m., never surging to the same heights.

The investigators conclude that the expectation of arising at a specific time sets a neurological timer that continues into the sleeping state and that the ACTH surge marks the body's preparations for the alarm to sound. Born hopes to monitor electrical activity in the brain during sleep to isolate where this alarm clock resides and better explain how it gets set.

Some people visualize a clock or repeatedly think of a wake-up time, notes Moorcroft, but most report no fixed strategy to set their internal alarm. *—J. Travis*

# Formaldehyde: Some surprises at home

It's hard to avoid exposure to formaldehyde, a respiratory irritant and suspected carcinogen. It protects latex paints from mildew and inhibits wrinkles in permanent-press fabrics. It's also a key ingredient in many insulating foams, durable automotive resins, and glued-wood construction materials.

A new study finds that although manufacturers have in recent years cut formaldehyde emissions from some of its most notorious sources—such as particleboard—many common consumer products still release copious amounts. Indeed, one of the big surprises was the amount coming from certain floor finishes, observes Thomas J. Kelly, a chemist who led the new analysis.

Under contract to the state of California, Kelly's team at Battelle Memorial Institute in Columbus, Ohio, measured 24-hour formaldehyde emissions from 55 domestic consumer and construction products. While polyurethane floor finishes don't emit the toxicant, he found, the more durable acid-cured resin finishes do. Until they dry, they can spew up to 1.2 grams per square meter per hour—nearly 1,000 times more than bare particleboard.

*Nail polish and, especially, nail hardeners are potent, though short-lived, sources of formaldehyde emissions.*

Moreover, he notes, refinishing a home's floors with this product could saturate other surfaces—walls, furniture, carpeting, even toys—with formaldehyde, allowing it to reenter the air long after the floors had dried.

Wet fingernail hardeners and polishes also proved to be big emitters. A 3-inch-square coating emitted far more formaldehyde—between 50 and 800 micrograms—than did an equal area of particleboard or veneer-covered plywood, the Battelle scientists report in the Jan. 1 ENVIRONMENTAL SCIENCE & TECHNOLOGY. While this can offer individuals—from fashion-conscious teens to professional manicurists—a big slug of the toxicant, nail treatments coat small areas and the exposures are short-lived. By contrast, plywood, particleboard cabinets, and new plastic-laminate counters not only cover relatively large areas but also emit measurable formaldehyde continuously for days to weeks.

Other major sources of formaldehyde included permanent-press shirts and draperies. While a single washing reduced a shirt's formaldehyde emissions by 60 percent, Kelly notes that draperies might never get washed. Pre-pasted wallpaper, while wet, also emitted substantial amounts of the toxicant—nearly 700 µg per square meter per hour.

Kelly's data on the effect of washing permanent-press fabric "is nice information that we can pass along," notes Peggy L. Jenkins, who manages the Indoor Air Quality Program at California's Air Resources Board in Sacramento. The main use of the new data, she says, "will be to improve and revise our guidance to the public" regarding what to buy.

While the recommended exposure limit of 0.5 parts per million in air will not change, she says, "we have, with this new information, a little more meat and potatoes" on where big exposures may occur. For instance, she says, consumers may want to ask more questions about floor finishes or cabinet materials, instructing their contractor to use products that emit less formaldehyde. *—J. Raloff*

# Young plants prepare to see the light

Researchers have found what may be the key steps that allow newborn seedlings to burst out of the soil without a spot of ray-catching chlorophyll but still start using light.

While underground, seedlings rely on lunches their mothers packed, nutrients in the seed. Although conifer sprouts under soil prepare for daylight by making chlorophyll, flowering plant sprouts cannot synthesize it in the dark.

As soon as they emerge, these pale sprouts can nevertheless harness light to power their shift to adult metabolism, thanks to a novel complex of compounds, report Christiane Reinbothe of Bayreuth University in Germany, and her colleagues. They and other researchers had identified some bits of the system. Now, coauthor Steffen Reinbothe says, "we put them together to find a way out of the dark." The results appear in the Jan. 7 NATURE.

Studying barley seedlings grown in darkness, the researchers focused on two enzymes called PORA and PORB, which catalyze a step in the transformation of precursor pigments into chlorophyll. Researchers had not known how the activity of these two forms of the enzyme protochlorophyllide oxidoreductase differs. Reinbothe was surprised to find that PORA and PORB bind different chlorophyll precursor pigments having extremely similar structures. "It was very unexpected," he says.

Reinbothe's group proposes that in a seedling, the two enzymes and the pigments form part of a complex that waits for the first blast of light. Mixing ingredients in the laboratory, the researchers did find such a complex. When they flashed light that only the pigment bound by PORA could absorb, they saw reactions in the PORB-pigment pair. They suggest that the pigment bound by PORA acts as an antenna, soaking up rays and passing along energy to power PORB's step in making chlorophyll.

The work "took a number of leaps over the current dogma," says Robert Willows of Macquarie University in Sydney, Australia. He says the most dramatic change is the new role for protochlorophyllide *b*, the pigment bound by PORA. Most physiologists assumed it didn't show up so early in development, Willows says. However, he speculates that "the main reason no one's found it is that no one's looked very hard."

Timothy W. McNellis of the University of California, Berkeley was intrigued by the way the complex protects seedlings. Without chlorophyll, "it's as if an albino person goes out in bright sunlight without sunblock," he says. Reinbothe, however, found that if protochlorophyllide *b* receives more energy than the complex can use, it fluoresces, emitting the excess harmlessly. *—S. Milius*

# Fossil ape's grasp gets two thumbs way up

Ancient apes, like their modern counterparts, typically had hands equipped for tree climbing and branch swinging. But a little-studied set of fossil remains tells a gripping tale of surprisingly deft digits in an apelike creature that lived 9 million to 7 million years ago on what was once a Mediterranean island.

The animal, known as *Oreopithecus bambolii*, boasted an opposable thumb and a grasping ability much like that exhibited by members of the human evolutionary family 3 million to 4 million years old, according to a new report.

Possession of such a hand, which may have given this primate an advantage in gathering sometimes scarce food supplies, laid the groundwork for the evolution of its ability to walk upright (SN: 10/18/97, p. 244), propose anthropologist Salvador Moyà-Solà of the M. Crusafont Paleontological Institute in Sabadell, Spain, and his colleagues.

A two-legged gait may have evolved similarly in ancient human ancestors, such as the australopithecines, after they developed a grip suitable for extensive food gathering and rapid feeding, the scientists suggest.

Moyà-Solà's team studied *Oreopithecus* fossils previously found at an Italian site and now held at the Natural History Museum in Basel, Switzerland. The collection includes many isolated hand bones, several partial hands, and a nearly complete right hand.

Several features of the *Oreopithecus* hand signify the presence of a thumb-assisted grip capable of precise manipulations, the researchers report in the Jan. 5 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES. First, short hands relative to estimated body weight combine with long thumbs, considered essential for forming a humanlike grip. *Oreopithecus* thumb bones also exhibit large, deep pits for the attachment of what was apparently an unusually strong muscle for flexing that digit.

Moreover, the fossil ape's finger joints show no evidence of having supported knuckle-walking, the investigators say.

Overall, *Oreopithecus* hands display evidence of the improved finger control and greater ability to exert force observed in early members of the human evolutionary family, they contend.

"I have doubts about that conclusion," remarks anthropologist Peter Andrews of the British Museum of Natural History in London.

Hand and foot bones that in some ways resemble those of australopithecines do not conclusively show that *Oreopithecus* preferred to walk upright and use its hands for precise manipulations, Andrews argues. The few hand bones recovered from other fossil apes look much like those of *Oreopithecus*, he notes, although those creatures are generally thought to have dwelled in trees and knuckle-walked while on the ground. *—B. Bower*