FEBRUARY 18, 2006 PAGES 97-112 VOL. 169, NO. 7

SCIENCE NEWS

THE WEEKLY NEWSMAGAZINE OF SCIENCE

virulent gut microbe spreads

sick-spouse syndrome

new class of rotating stars?

fear the flatworm!

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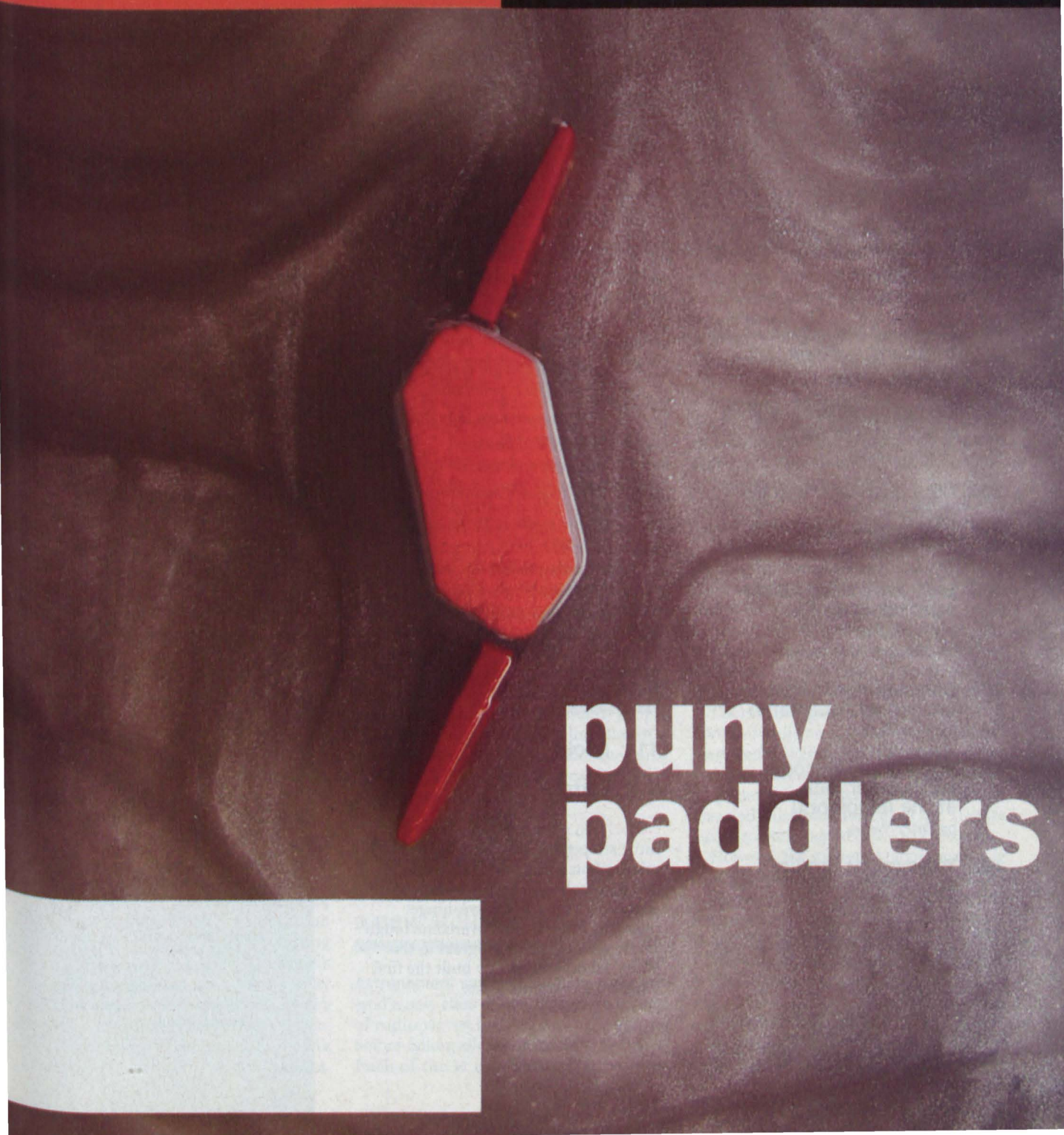

puny paddlers

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FEBRUARY 18, 2006 VOL. 169, NO. 7

Features

This Week

Of Note

Departments

Cover In science fiction flicks and futurists' predictions, medical microrobots patrol within the human body. In reality, designs—such as this pocket-size model of a two-hinge microswimmer—must overcome tough challenges. Marking progress in this effort, scientists recently built the first swimming micromachine.
(B. Chan/MIT) Page 107

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RUSSELL KIGHTLY MEDIA

In Sickness and in Death

Spouses' ills imperil partners' survival

Among elderly people, a spouse's hospitalization for certain ailments substantially raises his or her partner's likelihood of dying, according to the largest study ever to quantify such effects. The risk is especially great within the first month after the spouse enters the hospital.

Partners died most frequently following their spouses' hospitalizations for particularly disabling conditions, such as dementia, psychiatric illness, and hip or other bone fractures, say medical sociologist Nicholas A. Christakis of Harvard Medical School in Boston and sociologist Paul D. Allison of the University of Pennsylvania in Philadelphia.

QUOTE

"Your chances of dying increase not just when your partner dies, but when your partner becomes seriously ill."
NICHOLAS CHRISTAKIS, Harvard Medical School

"Our study shows that your chances of dying increase not just when your partner dies, but when your partner becomes seriously ill," Christakis says. After climbing rapidly in the first weeks after a spouse's hospitalization, a partner's risk of death declines to slightly above normal for a few months before rising again for the rest of the time examined, the scientists report in the Feb. 16 *New England Journal of Medicine*. The length of the spouse's hospitalization didn't affect the death risk.

For elderly people whose spouse had been hospitalized, the short-term risk of dying approaches that of elderly people after a spouse's death, Christakis and Allison assert. In the new study, a wife's hospitalization increased her husband's chances of dying within a month by 35 percent. A husband's hospitalization boosted his wife's mortality risk by 44 percent. In comparison, a wife's death increased her husband's 1-month mortality risk by 53 percent, and a husband's death raised his partner's risk by 61 percent.

A spouse's illness or death may hasten a partner's demise by causing severe stress and removing a primary source of emotional, financial, or practical support, Christakis suggests. These effects can undermine the body's immune system and intensify preexisting health problems.

The researchers analyzed hospitalizations and deaths from 1993 to 2002 among 518,240 married couples, ages 65 to 98, who were enrolled in Medicare. Roughly three-quarters of the husbands and two-thirds of the wives were hospitalized at least once during the study period. About half the husbands and 30 percent of the wives died.

A partner's risk of dying depended on the illness that afflicted his or her hospitalized spouse. For instance, among men, 8.6 percent died within 1 year of a spouse's hospitalization for dementia, 7.5 percent died within 1 year of a spouse's hospitalization for psychiatric disease, and 6.9 percent died within 1 year of a spouse's hospitalization for stroke. However, in men whose wives had been hospitalized for most forms of cancer, mortality rates were close to the 5.6 percent death rate measured among men whose wives hadn't been hospitalized.

A similar mortality pattern appeared among wives of hospitalized men, although their death rates were slightly lower than those of men.

The unprecedented size and scope of the new study make its findings "very exciting," remarks sociologist Linda J. Waite of the University of Chicago.

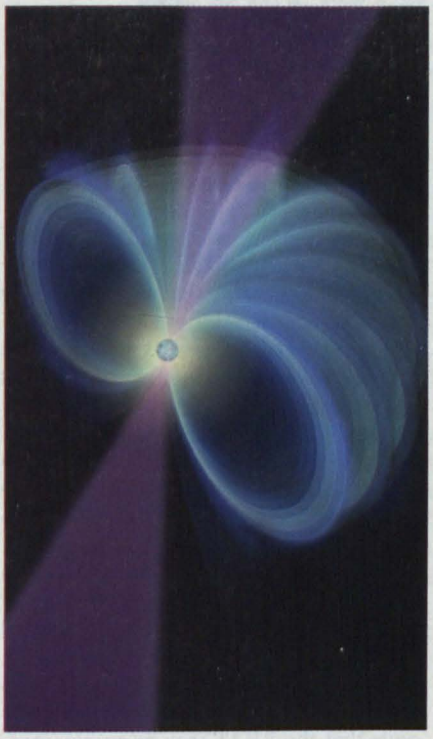
Psychologist Camille Wortman of the State University of New York at Stony Brook regards the dramatically increased risk of death among partners of recently hospitalized spouses as "amazing." Women tend to have more social support than men do, so it's surprising that hospitalization of wives wasn't harder on men in the short run than vice versa, she adds.

The new findings show that physicians and family members need to address the social and practical needs of elderly people whose spouses develop disabling illnesses, notes gerontologist Suzanne E. Salamon of Beth Israel Deaconess Medical Center in Boston. —B. BOWER

Radio Daze

Staccato pulses suggest a new stellar class

Astronomers say that they have discovered a new class of star that emits a burst of radio waves for 2 to 30 milliseconds before falling silent for minutes to hours. Each of the 11 newfound objects ranks among the strongest known sources of radio waves in the universe.



STELLAR CLASS Magnetic field lines (blue and green) loop around a rotating neutron star, while radio beams (purple) shoot out from it. Astronomers report a new class of such stars.

Because the objects broadcast for only about a tenth of a second each day, they're extraordinarily difficult to detect. The Milky Way may harbor several hundred thousand of these elusive stars, Andrew G. Lyne of the University of Manchester in England and his colleagues report in the Feb. 16 *Nature*.

That estimate would make these stars two to three times as numerous as the relatively common stars called radio pulsars. Ultracompact neutron stars, they produce regular pulses of radio waves as they rotate. The newfound objects, dubbed rotating radio transients (RRATs), may be cousins of the radio pulsars, the discovery team suggests.

The researchers initially detected isolated bursts of radio emissions using data from the wide-field Parkes Radio Telescope in New South Wales, Australia. They then searched for patterns in the arrival times of the signals and calculated that 10 of the 11 stars have a rotation period of 0.4 to 7 seconds.

However, the stars emit radio waves only during a few of those rotations. "It's as if, following [a burst], a RRAT has to gather its strength for perhaps a thousand rotations before it can do it again," says Lyne.

The rotation rate and intermittent release of radio waves suggests that these stars are neutron stars but not radio pulsars, which emit regular, frequent pulses.

Although the team may have uncovered a new kind of neutron star, theorist Robert

Duncan of the University of Texas at Austin says that "a more reliable and conservative interpretation" is that the researchers have detected elderly rotating neutron stars—either radio pulsars or magnetars, which have the strongest known magnetic fields of any star.

Theorist Maxim Lyutikov of the University of British Columbia in Vancouver concurs, noting that radio pulsars generate weaker electric fields as they age and therefore fewer radio waves.

In separate research, Lyne and other colleagues report online (*http://xxx.lanl.gov/abs/astro-ph/0512379*) that they identified X-ray emissions from one of the RRATs. The weakness of the X rays, along with the star's unusually strong magnetic field, suggests that the RRAT may be an old, inactive magnetar, Duncan says.

This star and at least two other RRATs are slowing their spins, further suggesting that they might be old radio pulsars or fading magnetars, Duncan notes.

With the results so new, "it will take time—and more data—to gain a full understanding," he adds. —R. COWEN

Killer Flatworm

New species hunts with puffer fish toxin

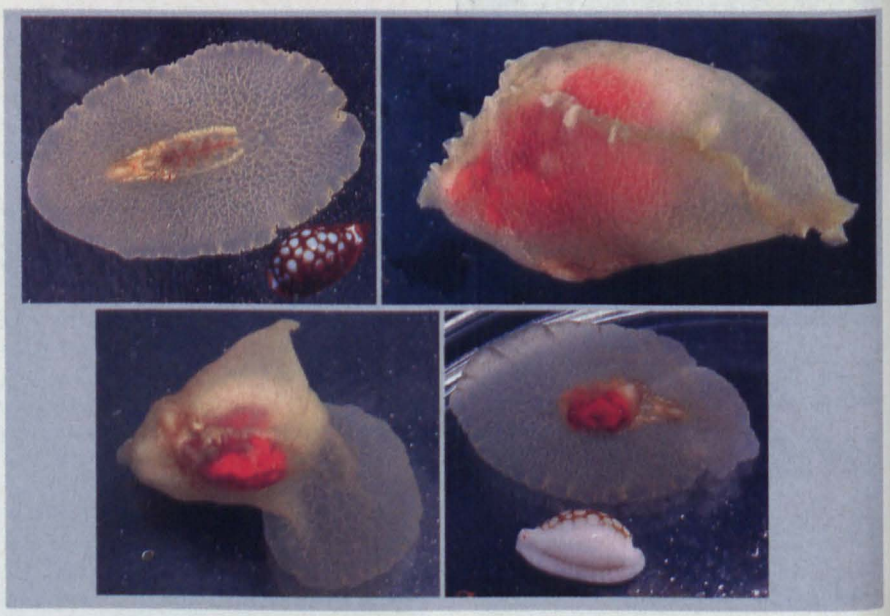
A newly discovered saltwater flatworm, pale yellow and about the size of a silver dollar, can take down mollusks in their shells, thanks to a powerful neurotoxin also found in puffer fish.

Yet this formidable hunting tool flops as a defense against fish eating the flatworm, says Raphael Ritson-Williams of the Smithsonian Marine Station in Fort Pierce, Fla.

The puffer fish poison, tetrodotoxin, has turned up in a variety of other creatures that spend at least part of their lives in water. These include other flatworms, frogs, North America's rough-skinned newt, and the blue-ringed octopus.

Tetrodotoxin and other toxins from marine organisms have caught the attention of biomedical researchers for basic research and drug development. However, Ritson-Williams and a few other biologists are studying what animals actually are doing with the poisons. His new experiments provide an unusual look at how a flatworm uses its formidable chemistry.

A small-scale mystery led Ritson-Williams to recognize the new flatworm's predatory use of toxin. Five years ago, while collecting flatworms in Guam, he picked up a cowrie to give to a friend who was doing a research project on that speckled-shell mollusk. He put the cowrie in a container with the new flatworm. When Ritson-Williams got back to his base camp, the flatworm "was really fat," he says. And all that remained of the cowrie was an empty shell.



LUNCH DATE A newly discovered flatworm from Guam wraps its body around a cowrie in its shell, paralyzes it with a nerve toxin, and pulls out the red body in about half an hour.

Since flatworms don't have teeth or any obvious weapons, Ritson-Williams' Smithsonian colleague Valerie Paul suspected poison. Ritson-Williams sent samples of the worms to chemist Mari Yotsu-Yamashita of Tohoku University in Sendai, Japan. The flatworm has tetrodotoxin along with some closely related chemicals, the researchers report in an upcoming *Proceedings of the National Academy of Sciences*.

To see how the flatworm uses its toxic arsenal, Ritson-Williams offered it various mollusks. The flatworm killed at least 30 species, including ones with trapdoors that block out most danger. By engulfing its prey or covering the victim's trapdoor, the flatworm presumably seals its quarry in a pocket of toxin-laced water (for Ritson-Williams' video, go to *www.sciencenews.org/articles/20060218/worm.mov*).

Ritson-Williams next tested for defense capabilities by offering flatworms to fish swimming free in the wild. Many of the fish readily swallowed the worms. He says that he doesn't know whether the fish suffered any long-term ill effects but points out that the poison failed as a defense against being eaten.

The flatworm's tetrodotoxin probably comes from bacteria that live in its body, says Marian Litvaitis of the University of New Hampshire in Durham. Marine flatworms are good at borrowing weaponry, she notes. The ones that she studies acquire their toxins from sponges and sea squirts. —S. MILIUS

Model for Madness

Engineered mice have schizophrenia-like symptoms

Scientists have genetically altered mice so that they mimic the deficits in short-term memory and attention of schizophrenic patients. The new animal model could shed light on the causes and treatment of these symptoms, which respond only minimally to current drugs.

Memory and attention problems are only part of schizophrenia, a complex disorder that also includes hallucinations, delusions, and social isolation. Scientists suspect that many of schizophrenia's symptoms stem from hyperactivity in the brain's machinery for using the signal transmitter dopamine.

Previous research had shown that schizophrenics have slightly more of a particular dopamine receptor, known as the D2 receptor, on nerve cells within a central part of the brain called the striatum. Scientists weren't sure whether this increase in D2 receptors was a cause of schizophrenic symptoms or a result of drugs used to treat the disorder, notes Eleanor Simpson, a neuroscientist at Columbia University.

Rather than study schizophrenia by crafting mice that have all the characteristics of the disease, she and her colleagues, led by Columbia's Eric Kandel, decided to make mice that simply had more D2 receptors in

their striata than normal. "We said, 'Let's ... see what aspects of the disorder happen with that change,'" Simpson says.

Kandel's team genetically engineered mice to have about a 15 percent excess of D2 receptors in their striata. A wrinkle in the genetic strategy also gave the researchers the option of shutting off the extra receptors by feeding the animals an antibiotic called doxycycline.

The engineered mice behaved much as normal ones did in tests of general cognition, such as learning the location of a platform hidden under water. However, the researchers noticed some differences when they tested the animals' working memory—a type of short-term memory held only during a task and briefly afterward.

By using working memory, normal mice quickly learned that researchers were alternating in which side of a T-shaped maze they hid food. In contrast, the engineered mice floundered at the test, taking significantly more trials to master the game.

An aspect of attention also diminished in the engineered mice. When the researchers hid a treat under one of two piles of bedding with different scents and then switched the scent hiding the treat, normal mice quickly learned which pile would yield the reward. The engineered mice dug in the original pile for significantly longer before trying the other one.

When researchers shut off the extra D2 receptors by feeding engineered mice doxycycline, they found no change in these animals' memory and attention deficits. The finding suggests that extra D2 receptors cause permanent brain damage, Kandel says.

That damage probably occurs while the brain is still developing and might be permanent, says Kandel. If so, he adds, it could explain why antipsychotic drugs that target D2 receptors don't improve schizophrenic patients' cognitive deficits.

The mouse findings, reported in the Feb. 16 *Neuron*, could help researchers develop new drugs to prevent such early brain damage from occurring in people who may be susceptible to schizophrenia, says neuroscientist Solomon Snyder of Johns Hopkins Medical Institutions in Baltimore. —C. BROWNLEE

STATS

2.2 million

Number of adults in the U.S. with schizophrenia

Not So Sweet

Cancers in rats that consumed aspartame

A large, new test in rats suggests that the artificial sweetener aspartame may be a carcinogen. But scientists not affiliated with the research express doubts about the study's validity and point to earlier trials that produced the opposite result.

Aspartame, sold under the brand names Equal and NutraSweet, is used in thousands of products, including diet soft drinks and sugarfree gum. The acceptable daily intake set by the Food and Drug Administration is 50 milligrams per kilogram body weight (mg/kg) per day, the equivalent of about 20 cans of diet soda.

Eight past studies looked at whether the sweetener causes cancer in lab animals, says veterinary pathologist James Swenberg of the University of North Carolina at Chapel Hill. "There's one equivocal study, and the rest are clearly negative for this endpoint [cancer]," he says.

But medical oncologist Morando Soffritti says that the earlier trials were small, funded by aspartame's manufacturer, or never published in detail in the scientific literature. Soffritti led the new study, which appears in the March *Environmental Health Perspectives*. He works at a nonprofit organization, the European Ramazzini Foundation of Oncology and Environmental Sciences in Bologna, Italy.

Soffritti and his colleagues examined 1,800 rats, making their study larger than the past aspartame-carcinogenicity tests. The researchers divided the animals into seven groups and gave each group drinking water with either no aspartame or a daily dose of aspartame between 4 mg/kg and 5,000 mg/kg.

The experiment continued until the animals died, which took an average of 2 years. Pathologists dissected each animal to detect any cancers.

The researchers found more cancers, particularly more lymphomas and leukemias, in animals exposed to aspartame than in unexposed animals. The number of cancers detected increased in proportion to the amount of the sweetener that the animal had ingested, the team reports.

That dose-response relationship is a strong sign that aspartame causes cancer, Soffritti says. "We have shown that aspartame is a carcinogenic agent," he concludes.

Because the study is so large and includes a wide range of doses, it has "a fair amount of power," says toxicologist John Bucher of the National Toxicology Program in Research Triangle Park, N.C. The study might detect a subtle carcinogenic effect that had been invisible in previous, smaller studies, he says.

However, the relationship between aspartame dose and cancer incidence reported by the Ramazzini researchers was "not overly strong," Bucher adds. "It's not like [what] one would get with a frank, strong carcinogen."

Lois Swirsky Gold, who directs the Carcinogenic Potency Database at the University of California, Berkeley, says that she isn't convinced that the data reflect an increase in tumors with dose.

Furthermore, says Swenberg, the Ramazzini researchers bucked the convention that laboratory animals be sacrificed at a predetermined point to ensure that their tissues get preserved immediately upon death. The team presumably ended up with less-than-well-preserved tissues, in which identification of cancers would be difficult, he says.

He and Gold both recommend that an independent group of pathologists reevaluate the tissues identified as tumors.

The FDA says that it has requested that the Ramazzini researchers share all their raw data. —B. HARDER

New View

Speedy microscope takes fuller look at the nanoworld

Although the atomic-force microscope is a workhorse for nanoscale measurements and manipulations, it's neither the fastest nor the most informative of instruments. Used widely in biological and materials research, as well as in microelectronics manufacturing and other industries, the instrument provides minute topographical details of a sample but not much else.

A team of engineers has now unveiled a radically revised version of the device. The inventors claim that it operates 100 times as fast as its conventional cousins do, raising the prospect that now-rare videos of molecular interactions could become routine. What's more, as the new instrument examines the topography of an object, it can simultaneously measure other properties, such elasticity, stiffness, and stickiness, the scientists report.

"This is really powerful stuff," claims mechanical engineer F. Levent Degertekin of the Georgia Institute of Technology in Atlanta. He and his colleagues describe their new device in the February *Review of Scientific Instruments*.

A standard atomic-force microscope, or AFM, probes a sample by means of a tiny cantilever with a sharp, downward-pointing tip (*SN: 1/1/05, p. 12*). The instrument drags or taps the tip along a sample's surface. By raising and lowering the cantilever to maintain a constant force on the tip,

the instrument maps out the surface's ups and downs.

"Right now, it's a blind man's world. We're just feeling with the cantilever," says physicist Thomas G. Thundat of Oak Ridge (Tenn.) National Laboratory, who works with AFM but was not on the design team for the new device.

Instead of a cantilever, the business end of the new-style AFM is a conventional tip mounted at the center of a circular membrane roughly the size of the period at the end of this sentence. The setup facilitates quick scanning because the membrane immediately flexes in response to an electrical signal within the device, Degertekin explains. Ordinary AFMs require more time to mechanically raise and lower the bulky apparatus that holds the cantilever.

Because the pliant membrane is also more responsive than the cantilever to forces imposed by a sample's surface, its motions yield information about properties that affect those forces, Degertekin adds.

"If you're going to look at nanostructures, you want a tool that will do everything," says team member Calvin F. Quate of Stanford University, who coinvented the AFM in 1986. The membrane-based probe could be that all-purpose tool, he adds.

The next challenge is to run the new AFM in water to show whether it's applicable to biological studies, comments AFM microscopist Paul K. Hansma of the University of California, Santa Barbara.

That shouldn't be a problem, Degertekin says, because versions of the new AFM's membrane have already served as ultrasound sensors in liquids. —P. WEISS

Looking Ahead

Tests might predict Alzheimer's risk

In the century since German physician Alois Alzheimer first described the devastating brain disease that bears his name, the illness has resisted cure and its origins have remained elusive. Now, two teams of scientists report that years before symptoms appear, there are indications of who will develop the disease.

U.S. researchers show that brain scans can spot warning signs of cognitive decline, and a Swedish team reports that spinal fluid contains markers of Alzheimer's risk. Such early diagnostic tests could increase the time available to treat the disease.

"Many people believe that we're going to have treatments [for Alzheimer's] in 5 years," says William Jagust, a physician and neuroscientist at the University of California, Berkeley. "And there's virtual unanimity of opinion that the drugs will be far more effective if given to people before they develop symptoms."

Jagust and his colleagues performed positron emission-tomography scans and magnetic resonance imaging on 60 volunteers who had no cognitive impairments and were ages 60 to 100. Annual tests of cognition over the next 4 years showed memory declines or other mental losses in six of the participants. Scans of those participants taken at the start of the study revealed that specific areas of the brain had shrunk or become less active. Brain scans of the other 54 participants showed less shrinkage and more activity in these areas.

The locations affected in the six participants with mental declines matched spots that autopsies commonly reveal as damaged in Alzheimer's patients. "It's guilt by association," Jagust says. "We think it's very suspicious" that the same regions are damaged in both groups.

The report will appear in an upcoming *Annals of Neurology*.

"This is a unique study," says neurologist Charles DeCarli of the University of California, Davis. "These are important pieces of information to have available. Their true impact will be seen when we have therapies" for Alzheimer's disease, he says.

A team of Swedish researchers took a different approach. They performed spinal taps on 137 people ages 50 to 86 who had mild cognitive impairments—beyond the normal challenges that come with aging—that affected their language, memory, or critical thinking. The team tested the spinal fluid for concentrations of tau protein and beta-amyloid, two substances implicated in Alzheimer's disease.

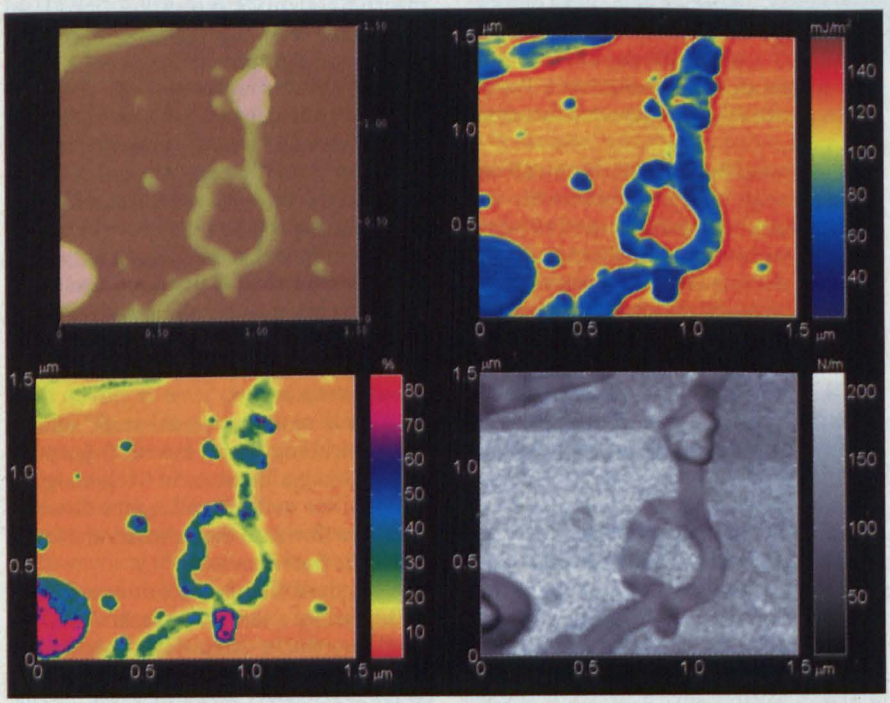
Over 4 years, 57 of the people developed Alzheimer's disease. Those whose spinal fluid at the start had abnormally high tau protein and low beta-amyloid concentrations were nearly 18 times as likely to develop Alzheimer's disease as were people with normal concentrations, says physician Kaj Blennow of Gothenburg University in Sweden. Almost all the people in the high tau–low beta-amyloid group were diagnosed with Alzheimer's disease during the study.

The report will appear in the March *Lancet Neurology*.

The new work shows that "even in the early stages, you can find evidence that the disease is there or is coming," says Steven T. DeKosky, a neurologist at the University of Pittsburgh.

Drugs currently available for Alzheimer's disease can ease symptoms but don't slow disease progression. That's likely to change, DeCarli says. "It's my understanding that every major pharmaceutical company is working on an Alzheimer's drug or has one in the pipeline," he says.

When drugs become available to fight Alzheimer's, "there will be a very high demand for diagnostic methods," Blennow predicts. —N. SEPPA



NANOPORTRAITS A single scan by an enhanced atomic-force microscope reveals (clockwise from upper left) the topography, adhesion, elasticity, and softness of a loop of carbon nanotubes.

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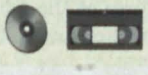

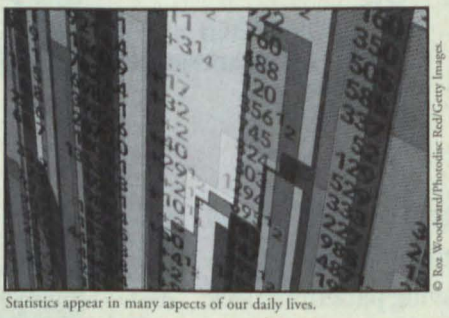

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Statistics appear in many aspects of our daily lives.

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Hospitals struggle with a serious new gut microbe

BY BEN HARDER

About 3 years ago, physicians in Quebec noticed an alarming pattern in patients with diarrhea. "All of a sudden, we were having patients so sick that they needed the ICU [intensive-care unit]," says doctor and epidemiologist Sandra Dial. The same illness was also sending patients to the morgue as never before. They weren't succumbing to the ailment that brought them to the hospital. Instead, they seemed to have gotten sick from their antibiotic treatment.

As an antibiotic attacks its target, it can also kill harmless flora, the term for the billions of bacteria that live in healthy intestines. Invaders, mainly the pathogen *Clostridium difficile*, can flock to the colon's open real estate.

Doctors treat *C. difficile* by suspending the culpable antibiotic therapy and administering a different one. While usually curable, the diarrheal disease recurs repeatedly in some patients and occasionally causes life-threatening inflammation of the colon.

C-diff, as it's called, is a rising problem across North America and in parts of Europe. A virulent, once-rare strain has driven a string of recent outbreaks and is suspected to be responsible for much of the overall increase in the disease. New resistance to widely used antibiotics seems to have made the microbe more likely to cause diarrhea in sick and elderly people.

At the same time, scattered cases of *C. difficile* among people not normally susceptible to it have researchers concerned.

Before December 2002, Dial had seen only three cases of *C. difficile* in 8 years at the McGill University–affiliated ICUs where she works. Then, she says, "we had five patients in 1 month."

Parallel epidemics were soon under way elsewhere in the region. More than 7,000 *C. difficile* cases occurred in Quebec hospitals in 2003, affecting up to 2.5 percent of patients. By early 2005, 30 centers in the province were seeing at least five times as many cases as they had identified in previous years.

In the United States, "the rate of C-diff in hospital patients doubled between 2000 and 2003," says L. Clifford McDonald of the Centers for Disease Control and Prevention (CDC) in Atlanta.

In response, researchers have identified unique characteristics of the newly virulent strain—and have revealed a grimmer picture of *C. difficile* than the one familiar to most physicians.

"Ten years ago, we didn't believe people died of this," Dial says. "It was very unusual. Now, unfortunately, it's not unusual."

UNPRECEDENTED ILLNESS Previously, elderly people staying in health care facilities and nursing homes were virtually the only targets of *C. difficile*. Now, the bacterium is gaining notoriety for illnesses in seemingly vigorous people.

Last year, for instance, the pathogen felled a 31-year-old Pennsylvania woman who was pregnant with twins. Early in her second trimester, she went to the emergency room with worsening diarrhea. Despite treatment that night and during two subsequent hospital stays, within a month she delivered stillborn fetuses and died of complications of colon inflammation.

In the Dec. 2, 2005 *Morbidity and Mortality Weekly Report*, McDonald and his collaborators in four states describe this case and 34 other recent illnesses caused by *C. difficile* contracted outside health care facilities.

To tease out the changing characteristics of C-diff, Jacques Pépin and his colleagues at the University of Sherbrooke Hospital Center in Quebec combed through data on 5,619 hospital patients treated between January 2003 and June 2004. Nearly 300 of the patients had developed the disease.

Compared with the other patients, those who'd contracted *C. difficile* tended to be older and to have had longer hospital stays. Those infected were also more likely to have recently used an antibiotic of the cephalosporin family, the team found.

> "Ten years ago, we didn't believe people died of this.... Now, unfortunately, it's not unusual."
>
> — SANDRA DIAL, MCGILL UNIVERSITY

Those antibiotics had been previously linked to outbreaks. The diarrhea-causing bacterium is usually resistant to cephalosporins, but normal colon bacteria are not. Therefore, the drugs create a wide-open opportunity for *C. difficile*. Swallowing a spore of the bacterium during cephalosporin therapy could result in an infection that otherwise wouldn't have gained a foothold.

In the Nov. 1, 2005 *Clinical Infectious Diseases*, the Sherbrooke researchers also link *C. difficile* to a class of antibiotics that had only infrequently turned up in earlier work. Fluoroquinolones, the most widely used antimicrobials in the United States, fight infections of the respiratory and urinary tracts, the skin, and other tissues. Ciprofloxacin is the main drug in this class.

In early 2004, researchers at Sherbrooke and 11 other Quebec hospitals joined forces to investigate what they regarded as the province's plague. Led by microbiologist Vivian G. Loo of McGill, they ultimately studied 1,703 patients who acquired *C. difficile* during the first half of that year.

The infected patients represented 2.25 percent of all people hospitalized at the 12 centers during the study. That reflects an infection rate four times as high as that in earlier data.

Almost 7 percent of the 2004 infections were fatal. That rate, too, is at least four times as high as that found in the past, Loo says.

As in the Sherbrooke study, patients who developed C-diff were more likely than other patients to have used fluoroquinolones while hospitalized, the researchers report in the Dec. 8, 2005 *New England Journal of Medicine* (*NEJM*).

More than 80 percent of the infected patients had acquired a single, fluoroquinolone-resistant form of the pathogen, making that strain primarily responsible for the Quebec outbreaks, Loo's team concludes.

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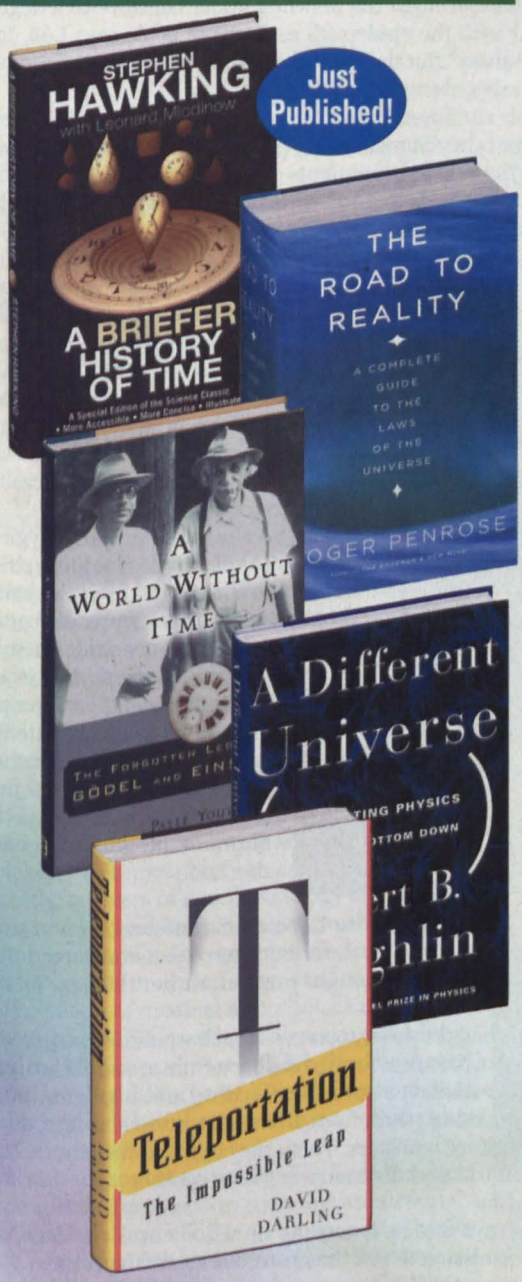

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A NOVEL MUTANT To characterize the guilty strain, Pépin, McDonald, and six collaborators analyzed 124 *C. difficile* samples taken from Sherbrooke patients in 2004 and early 2005. They also looked at 30 recent samples from other patients in Quebec, the United States, and the United Kingdom.

Disease-causing *C. difficile* produce at least two colon-inflaming substances, called toxin A and toxin B. Rare strains that make neither toxin don't result in illness. Scientists have occasionally identified strains that produce a third toxin, called binary toxin.

The new outbreak strain always produces binary toxin, the researchers determined. Furthermore, they found, it produces 16 times as much toxin A and 23 times as much toxin B as typical hospital strains of *C. difficile* do.

Genetic traits also set apart the dangerous strain. For example, the researchers identified a standard *C. difficile* gene that was uniformly mutated in the samples. The mutation may account for the excess production of toxins A and B, the team suggested in the Sept. 24, 2005 *Lancet*. Extra toxin may lead to more severe diarrhea, and, therefore, greater spread of the pathogen in hospitals.

The mutant strain, which the researchers labeled NAP1/027, had infected patients in all three countries in the study.

It has also caused recent outbreaks in the Netherlands and Belgium, says Dale N. Gerding of Hines Veterans Affairs Medical Center in Illinois.

Until recently, NAP1/027 was rare, he says. "It was definitely present in other hospitals 20 years ago, but it wasn't causing epidemics," he says.

Gerding, McDonald, and their collaborators made that assessment after comparing 187 recent samples of *C. difficile*, collected between 2001 and 2003, with more than 6,000 older samples. About half of the recent samples were NAP1/027.

Genetic similarities indicated that 14 older samples dating to 1984 were of the same, newly troublesome strain. NAP1/027 made binary toxin in the 1980s, but it was not resistant to most fluoroquinolones before 2001, the researchers report in the Dec. 8, 2005 *NEJM*.

"Fluoroquinolone resistance is unusual and new for this strain," Gerding says. Use of those antibiotics in hospitals has increased tremendously over the past 2 decades, which could explain the success of the newly resistant variant, he says.

But Dial suggests that antibiotics aren't the only medications contributing to the epidemic. She and three McGill colleagues have found possible links between *C. difficile* infection and some commonly used drugs, particularly acid reducers that treat heartburn, indigestion, and stomach ulcers in millions of people.

In studying a U.K. database that doesn't track individual strains, Dial's team found that people who acquired *C. difficile* outside the hospital were three times as likely as uninfected people to use proton pump inhibitors (PPIs), such as Prilosec, and twice as likely to use H2 blockers, an older class of acid reducers.

Dormant *C. difficile* spores are resistant to acid, but replicating cells are susceptible. So, normal concentrations of stomach acid could suppress the organism's activity—until a person takes an acid reducer, Dial says.

Use of nonsteroidal anti-inflammatory drugs is also slightly elevated among the people with *C. difficile*, her team reports in the Dec. 21, 2005 *Journal of the American Medical Association*.

Gerding, along with doctors from a Portland, Maine, hospital, separately found an association between PPIs and *C. difficile*. Robert C. Owens Jr. of the Maine Medical Center presented that finding at the Interscience Conference on Antimicrobial Agents and Chemotherapy in Washington, D.C., last December.

But Gerding says that it's "an open question" whether the acid reducers contribute to infection. Data sets analyzed by Loo and by Pépin don't suggest a link.

In any case, says Gerding, "antibiotics are still the overwhelming risk factor."

MANIFOLD RESPONSES In the United States, the newly troublesome strain continues to spread. "CDC has documented the strain in 16 states," McDonald says.

In Quebec, the worst-hit Canadian province, diligent efforts in hospitals have brought the new bug under control—to a degree. Compared with the epidemic's early-2003 peak, says Loo, "our rates are halved." But they're still significantly elevated from pre-epidemic rates, she notes.

Hospitals employed a multifaceted approach to rein in the epidemic. First, they increased the frequency of cleanings. Rooms with *C. difficile*–infected patients now get scrubbed down twice a day, Loo says. And to fight *C. difficile*'s hardy spores, bleach has been substituted for gentler agents.

Second, hospitals spent new funds from the Canadian government to buy more of their most frequently used medical items, such as blood pressure cuffs, so that equipment that might pick up spores doesn't get shuttled from room to room. "We also eliminated rectal thermometers," Loo says.

Furthermore, hospitals installed more sinks to encourage hand washing and created new rooms to reduce patient crowding in some older hospitals.

"We also changed our hand-hygiene practice," Loo says. "Alcohol hand rinses decrease the concentration of the bacteria, but washing at the sink was still better."

Finally, she says, the hospitals made an effort to educate physicians and medical residents on appropriate antibiotic use. That effort has somewhat reduced unnecessary administration of fluoroquinolones and other drugs, but more progress is needed, says Loo.

MONSTER OF MEDICINE — *Clostridium difficile* bacteria, shown here isolated from a stool sample, are causing more severe diarrheal illness in hospitals than they have in the past.

FIRE WITH FIRE At the December conference on antimicrobial agents, Gerding described new tests conducted on a strain of *C. difficile* that might prevent, rather than cause, disease.

Scientists had previously observed that hospitalized people who already carry a harmless form of the bacterium are unlikely to pick up a disease-causing strain. So, Gerding and his team took a non–toxin-making strain from a healthy person and introduced it into laboratory hamsters. Then, they exposed the animals to a strain that normally kills hamsters within 48 hours. The hamsters stayed healthy.

Gerding says that he is seeking the Food and Drug Administration's permission to test the protective strain in people.

While the approach is "counterintuitive," says McDonald, "there's a couple of lines of evidence to suggest it holds promise."

Other experimental approaches aim to strengthen the gut's defenses. The closest to fruition, according to McDonald, is a strategy in which a polymer binds to and inactivates the bacterium's toxins. Genzyme Corp. of Cambridge, Mass., developed the polymer, which it calls tolevamer. After promising preliminary results, the company launched a treatment trial that it projects will include more than 1,000 *C. difficile*–infected volunteers by the end of this year.

A positive outcome could lead to the first treatment for the antibiotic-associated illness that does not employ an antibiotic. ■

ARTIFICIAL ANIMALCULES

In the microscopic realm, machines learn to swim

BY PETER WEISS

Some 300 years ago, microscope inventor Antony van Leeuwenhoek stunned the world when he became the first to observe "animalcules" that were "very prettily a-moving" in human saliva and other excretions. Now, a human-rigged device has joined the menagerie. To a red blood cell, scientists in France have attached a wavy tail that responds to magnetic fields. When changes in those fields wiggle the tail, the red blood cell swims.

Experiments with that device have given researchers insights into the physics that tiny swimming creatures have to master. Along with other micromachines making their way from the drawing board to the real world, such devices may someday prove useful in disease research or industry.

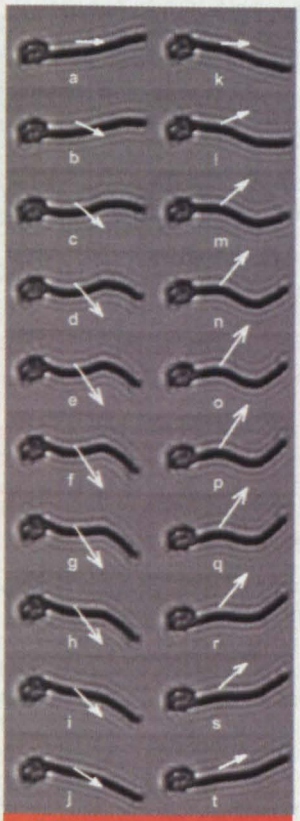
A tinge of science fiction, as well as hype, colors the endeavor. Notions of microscopic underwater craft trigger flashbacks to the 1966 movie *Fantastic Voyage*, in which a literally downsized medical team journeys aboard a tiny submarine inside a stricken diplomat's body to save his life. More recently, boosters of nanotechnology have repeatedly promised minuscule rovers capable of navigating human bloodstreams and dispensing medical treatments or reporting on local conditions.

The vision of medical microbots is not all fantasy, say researchers of scaled-down swimming gadgets. However, the task of devising these craft is challenging work, beset by complications that go far beyond the inherent difficulty of fabricating tiny gizmos.

Water—or any other surrounding liquid—affects micrometer-size objects in ways that are strikingly different from how it influences large-scale objects such as people, fish, or submarines. Researchers inventing propulsion mechanisms to exploit those unfamiliar circumstances find their own experiences in water to be of little help.

"Our intuition [about swimming] is based on the way objects our size move," notes Joseph E. Avron of the Technion–Israel Institute of Technology in Haifa. That intuition is "very bad for [understanding] small creatures."

"You have to ditch your previous concepts of how things swim," agrees mechanical engineer Anette E. Hosoi of the Massachusetts Institute of Technology (MIT).



TAIL GAIT — In a series of microscope images of a tiny, artificial swimmer (a to t), the pivoting of a magnetic field's orientation (white arrow) causes undulations of the swimmer's tail. The wiggling tail propels an attached red blood cell gradually to the right. The microswimmer travels at a leisurely speed of up to one red blood cell diameter per second.

DREYFUS ET AL./NATURE

LIFE'S A DRAG The underwater world of bacteria and swimming microcontraptions is so different from our world that it might as well be on another planet. Although the laws of physics remain the same for swimmers big and small, being as tiny as a bacterium makes them seem dramatically changed.

Why are microscale conditions so strange? Consider first the frictionlike force known as drag. Arising from contact between a fluid and a swimmer's surface, drag opposes motion through the fluid. The more viscous—or resistant to flow—a fluid is, the more drag it exerts.

Another characteristic, called inertia, resists changes in velocity. Inertia increases in proportion to the mass of a moving object.

A bacterium, say an *Escherichia coli*, has roughly a million times more surface area in proportion to its volume than does a person. So the effects of drag, compared with those of inertia, are much greater on an *E. coli* than on a person.

At the macroscopic scale, where inertia is large and drag is small, things in motion tend to stay in motion. In a pool, a person thrusts a hand or foot so that it launches water backward and pushes the swimmer forward in accordance with Newton's laws. Inertia then prolongs the swimmer's glide, although drag eventually slows it.

Under the topsy-turvy conditions of the microscale, however, swimmers can't launch liquid away from themselves because they can't overcome the drag of the water. And without much inertia, they don't glide. Swimming on the microscale is less like doing laps in the local swimming pool than it is like taking a dip in the asphalt goo of the La Brea tar pits on a warm day.

Whereas a macroscale propeller takes a mass of water and throws it backward, Avron says, the helical appendage, or flagellum, that propels an *E. coli* "is more like a corkscrew moving through cork." Microscale swimmers scramble against the liquid or squirm through it in some manner that takes advantage of the fluid's viscosity.

WIGGLE ROOM Although scientists have known how viscosity, drag, and inertia act on microscale objects for decades, no one had found a way to exploit that knowledge to make an artificial swimmer of micrometer-scale proportions—until last year.

A group at the École Supérieure de Physique et de Chimie Industrielle in Paris was devising a way to straighten out loops in long, stringy biomolecules, such as RNA and DNA, to be used on microchips that detect gene activity. Rémi Dreyfus first created filaments composed of microscopic magnetic beads

joined by bundled DNA strands. Then, he used magnetic fields to make the beads pull apart, exerting tension on the DNA.

When Dreyfus watched videos taken with a camera-equipped microscope, he made a startling observation. "I could see that some filaments were swimming," he recalls. "It was really a surprise."

Dreyfus and his colleagues, including Howard A. Stone of Harvard University, soon found that the combination of steady and varying magnetic fields used to manipulate the beads was creating waves along the filaments that made a few of those chains swim around. The swimming chains had defects that made them asymmetrical, the team discovered.

To deliberately create asymmetry, the researchers attached one end of a chain to a red blood cell, which was an appropriate size and easy to affix.

By manipulating magnetic fields, scientists might direct their souped-up cell to a particular location. In this way, the team demonstrated that a swimming microdevice might do something useful, Dreyfus says. Instead of a red blood cell, the chain might deliver a vesicle filled with drug molecules or some other useful load.

Dreyfus and his colleagues "succeeded in attaching a mechanical engine to a biological creature. This was a wonderful piece of work," comments Avron.

To transform this first microswimmer into a device that carries out a specific task in the bloodstream would require many improvements, notes Jérôme Bibette of the French group. To avoid having to remotely apply changing magnetic fields, researchers might need to endow the device with its own on-board motor and fuel supply and some amount of intelligence to navigate and report on its location. So far, the team has not secured funding to support such development.

Still, the device is more than just a toy. In the report in the Oct. 6, 2005 *Nature* in which the researchers unveiled the chain-driven red-blood cell, they also reported using it to confirm theoretical predictions correlating viscosity, tail elasticity, and other factors to speeds of spermatozoa in a saline solution.

Bibette says that the wavy filaments also look promising for potential use in disease research. As simple strands unattached to red blood cells, they might model other common biological machines, hairlike cilia. These structures are prevalent in microorganisms as a means of propulsion. The human body uses them, for instance, to clear mucus from airways. Malfunctioning cilia can lead to serious lung and kidney problems.

Bibette says that the French team plans to measure cilia-driven fluid flow while altering cilia properties, such as elasticity. Such studies could shed further light on microswimming, since moving fluid with cilia or moving bacteria that bear cilia are analogous processes, he notes.

SIMPLE STROKES While the first microswimming machine resembles one of nature's designs for a tiny swimmer, many other swimmers now on drawing boards stray far from what's known from biology. Physicists have dreamed up Tinkertoy-like designs as they have vied to meet the challenge in the simplest way.

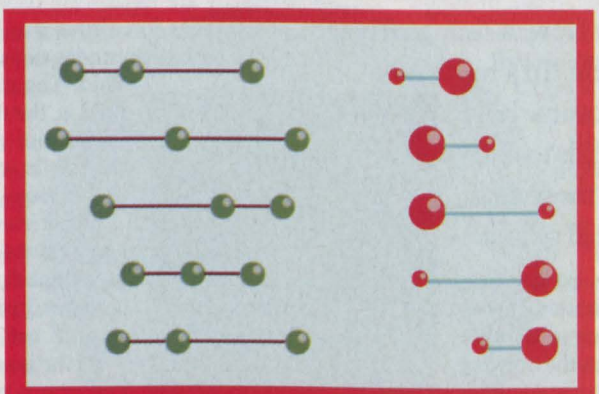
In an oft-cited 1970s lecture on microscale swimmers, Nobel prize–winning physicist Edward M. Purcell proposed a "two-hinged swimmer" as the simplest such gadget. The device would have three panels connected in series by two hinges. The outer two panels would move relative to the middle panel to propel the device forward.

Purcell suggested a series of movements of the microscale device's arms that would enable it to wiggle along a straight line in a specified direction. However, he left the details of why it would go that direction as "an exercise for the student."

In a recent investigation at MIT, Hosoi and Brian Chan tested Purcell's claims experimentally. They built a scaled-up model of the two-hinge apparatus, powered its arms with a windup spring, and immersed it in thick silicone oil to mimic the viscous conditions of the microworld. As can be seen in videos on the researchers' Web site (*web.mit.edu/chosetec/www/robo/3link/*), the model swims straight and in the direction Purcell claimed.

However, other researchers, including Harvard's Stone, have tackled Purcell's exercise with a theoretical analysis and found that the direction isn't as straightforward as Purcell imagined or as the mechanical testing indicates. In a 22-page report in 2003, the scientists concluded that although Purcell's gizmo should go straight, its direction depends on the size of the strokes made by its arms, a factor that Purcell didn't investigate.

Another Tinkertoy-style device proposed in 2004 consists of three balls connected in a line by arms that telescope in and out. The three-ball gizmo would move by a series of transformations in which each arm elongated or contracted, changing the spacing between the balls and propelling the device forward, reported Iranian physicists Ramin Golestanian of the Institute for Advanced Studies in Basic Sciences in Zanjan and Ali Najafi, now at Zanjan University.



NEW BALL GAME — A hypothetical microswimmer composed of three rigid balls connected by telescoping rods (left) could locomote to the right by executing the shown cycle of rod extensions and retractions, theorists claim. To also head to the right, another proposed contraption—two inflatable balloons joined by a flexible tube (right)—cycles through its own series of swelling, shrinking, and tube-length changing.

Microswimmer designers have examined how quickly and efficiently the devices might move. For instance, Stone and his colleagues considered efficiency when they evaluated Purcell's two-hinge swimmer. Their calculations showed that the venerable device would be only a tenth as efficient as common microscale biological-propulsion structures, such as an undulating tail and a rotating helical flagellum.

Yet nature's solutions are not automatically the best. Indeed, there may be good reason to avoid mimicking biology, Avron suggests. "The history of flight was held back many years because at the beginning, everyone wanted to emulate nature and flap wings," he notes.

Avron and his Technion colleagues broke with natural design with a hypothetical construct that resembles two balloons connected by a thin, stretchy tube. The device propels itself in a series of choreographed steps in which each balloon is alternatively inflated and deflated as the tube elongates and retracts.

This device can theoretically outperform spermlike swimmers with beating tails, the inventors report in the November 2005 online *New Journal of Physics*. Given the reputation of sperm cells for speed, "it's like a turtle beating a hare," Avron says.

Studying the two-balloon device might yield some insights into biology because its movements vaguely resemble contortions that microbes known as *euglena* use to propel themselves, he adds.

A theoretical device propelled by couple of balloons is a long way from the miniaturized but complex technology that amazed viewers of *Fantastic Voyage*. Researchers have to find answers to many questions before even the simplest devices make their debuts.

How quickly and efficiently does the gadget move? How readily might it be built with current technology? Has nature already invented something better?

With each answer, researchers move closer to creating a new breed of animalcules to swim the microrealm. ■

OF NOTE

ENVIRONMENT

Global warming may already be a killer

Earth's rising temperatures may be a precipitating factor in the extinctions of dozens of tropical frog species, according to new research.

At least 110 species of harlequin frogs once lived in Central and South America, but two-thirds of them went extinct in the past 2 decades. Scientists have puzzled over these and other amphibian disappearances in seemingly pristine areas.

CROAKING? This harlequin frog belongs to a group of species that is rapidly going extinct in Central and South America.

F. BREM

Years ago, scientists found that chytrid fungus (*Batrachozchytrium dendrobatidis*) had infected many dead frogs found in tropical regions (*SN: 2/26/00, p. 133*). In a new study, J. Alan Pounds of the Monteverde Cloud Forest Preserve in Costa Rica and his colleagues propose that global warming could be promoting the fungus' growth.

Pounds' team matched records of air and sea-surface temperatures with data on frog disappearances. The researchers found that species tended to vanish during years with the warmest average temperatures.

Warm periods enhance cloud formation over the tropics, which makes days cooler and nights warmer. Temperatures thus stay in the narrow range in which the fungus thrives, which could explain massive amphibian die-offs, says Pounds.

The researchers report their hypothesis in the Jan. 12 *Nature*. —C.B.

BIOLOGY

Soil microbes are reservoir for antibiotic resistance

Bacteria that live in dirt are surprisingly resistant to antibiotics, even those drugs they presumably have never encountered before, according to new research.

The majority of medical antibiotics originally came from soil bacteria, which produce chemicals to kill off other microbial species that compete for the same resources. To one-up the competition, many of these bacteria have evolved ways to detoxify antibiotics that neighboring species secrete.

Few of these soil bacteria are species that make people sick. However, notes Gerald Wright of McMaster University in Hamilton, Ontario, scientists had never examined the extent of antibiotic resistance in soil bacteria. Such knowledge might give researchers clues to how infection-causing microbes develop antibiotic resistance.

Wright and his colleagues first collected 480 strains of soil bacteria from a variety of locations, including urban, agricultural, and forest areas. They then treated cultures of the microbes with high concentrations of 21 commonly prescribed antibiotics. These included drugs that have been used for decades, such as penicillin and erythromycin, as well as drugs that were only recently invented and marketed, such as telithromycin and tigecycline.

Each of the bacteria was resistant to at least seven of the antibiotics. A couple of strains were resistant to 15 of the drugs, the researchers report in the Jan. 20 *Science*.

Wright notes that he and his team don't yet know whether soil bacteria can transfer their antibiotic-resisting capabilities to infectious bacteria. However, some of the strains that his team studied seem to have novel ways to fight the drugs with which they were treated. "If you can see how they get around these drugs, you might be able to identify what mechanisms could eventually evolve in [infectious] bacteria," he adds. —C.B.

EPIDEMIOLOGY

SUVs no safer for kids than passenger cars

Children in sport utility vehicles (SUVs) are just as likely as children in passenger cars to be injured in an accident, despite the SUVs' greater weight, a study finds.

Scientists analyzed accidents in 16 states and the District of Columbia that involved 3,922 children in SUVs or passenger cars. The vehicles were model-year 1998 or newer. All crashes had been reported to State Farm Mutual Automobile Insurance Co. between 2000 and 2003.

Researchers also interviewed the drivers of vehicles in which a child suffered an injury requiring treatment, such as a concussion, laceration, broken limb, or internal-organ injury. For both passenger cars and SUVs, children sustained such injuries in less than 2 percent of the crashes.

Heavy vehicles generally fared better in accidents than lighter vehicles did. However, SUVs rolled over more than twice as often as passenger cars did, and roll-over crashes were three times as likely to cause child injuries as were other crashes, the researchers report in the January *Pediatrics*.

SUVs' protection afforded by weight "is undermined by a roll-over tendency," says coauthor Dennis R. Durbin, a physician and epidemiologist at Children's Hospital of Philadelphia and the University of Pennsylvania School of Medicine in Philadelphia.

Children in cars or SUVs not wearing seat belts were four times as likely to be injured in a crash as belted kids were. Unrestrained children in SUVs that rolled over were 25 times as likely to be injured as were belted-in children, the researchers report. —N.S.

NUTRITION

Of taters and tots

Diets rich in french fries may be toxic—at least to little girls, according to a new study. Researchers found that for each serving of french fries that a preschool girl typically consumed per week, her adult risk of developing breast cancer climbed 27 percent.

The long-running Nurses' Health Studies I and II have been following nearly 240,000 female nurses born between 1921 and 1963. In the new analysis, epidemiologists looked for an association between foods that study participants consumed as children and their subsequent risk of breast cancer. Researchers analyzed information that had been provided by mothers of 582 participants who developed breast cancer and of 1,569 who didn't. They had estimated how much of 30 foods—from apples to hot dogs—their daughters had regularly eaten between the ages of 3 and 5.

At the start of the test, "I didn't have my money on any particular food or nutrient," says study leader Karin B. Michels of the Harvard School of Public Health in Boston. "So, I was surprised that one food so distinctly stood out" as a risk factor, she adds. Michels' team published the finding in the Feb. 1 *International Journal of Cancer*.

Michels warns that people should interpret these provocative results cautiously.

OF NOTE

In exploring cancer-risk factors for years, "we and other groups have been very unsuccessful in linking adult women's diets to breast cancer," she notes.

Her team explored childhood eating patterns, she explains, because other studies had shown that breast carcinogens, such as radiation, have their greatest impact before puberty. —J.R.

METEOROLOGY

How to rate a snowstorm

For decades, meteorologists have rated weather phenomena such as tornadoes and hurricanes on a scale of 1 to 5, but they've never really had a good rating system for snowstorms.

A new scale, unveiled on Jan. 30 at the annual meeting of the American Meteorological Society in Atlanta, enables scientists to rank each snowstorm affecting the northeastern United States according to amount of its snowfall, the size of the region it covers, and the population of the affected area. Each storm ranks in one of five categories: notable, significant, major, crippling, or extreme.

The new system is primarily intended to gauge the potential physical and economic impacts of a storm that has just occurred, says Louis Uccellini, director of the National Centers for Environmental Prediction in Camp Springs, Md. The researchers focused on the 13 states of the Northeast because that's where the most significant impact from U.S. snowstorms occurs.

Only two snowstorms in that area in the past century—one in March 1993, the other in January 1996—would have earned an extreme rating, says Uccellini. Such storms affect more than 65 million people and blanket much of a 775,000-square-kilometer area with at least 50 centimeters of snow. Using the new scale, meteorologists rated the blizzard that swept through the Northeast last weekend as a category 3, or major, snowstorm. —S.P.

PLANETARY SCIENCE

Stellar passage yields Charon's girth

On July 10, 2005, astronomers watched as Pluto's moon Charon passed in front of a star. The event lasted less than a minute, but that was long enough for researchers operating telescopes in Chile and Brazil to use the star as a backlight to obtain new, more accurate measurements of Charon's radius, density, and atmosphere.

In the Jan. 5 *Nature*, two teams report that Charon's radius is 606 kilometers. Combined with Hubble Space Telescope measurements of Charon's mass, the new size estimate reveals that the moon has a density 1.71 times that of water—and about one-third the density of Earth.

The rare stellar passage could be seen only from a 980-km stretch of South America. Observations as Charon's disk passed the star also indicate that if the moon has any atmosphere at all, its density is less than one-millionth that of Earth's atmosphere, according to Amanda Gulbis of the Massachusetts Institute of Technology and her collaborators. A team led by Bruno Sicardy of the Paris Observatory describes similar results.

FROZEN MOON Researchers recently obtained precise measurements of the density and radius of Pluto's moon Charon, seen here between the planet and the distant sun in an artist's rendering.

The lack of a substantial atmosphere supports the theory that Charon was released when an object struck Pluto. Scientists have similarly proposed that Earth's moon formed when a giant object struck the young Earth. —R.C.

EARTH SCIENCE

Krakatoa stifled sea level rise for decades

Ocean cooling caused by the volcanic eruption of Krakatoa in 1883 kept sea level worldwide in check well into the 20th century, a new analysis suggests.

When the Indonesian volcano exploded, it hurled immense amounts of ash and other particles into the stratosphere. For up to 2 years, those aerosols blocked about 1 percent of the sunlight that had previously reached Earth, says Peter J. Gleckler, an atmospheric scientist at Lawrence Livermore (Calif.) National Laboratory. The resulting decrease in absorbed radiation caused the upper layers of the oceans to cool and contract. Worldwide, sea level dropped.

Gleckler and his colleagues used modern oceanographic data to confirm the accuracy of six computer models that consider the effects of volcanic aerosols and other factors on Earth's oceans. On average, those models suggest that, between 1955 and 1998, sea level rose about 1.7 centimeters because of the warming of ocean waters, the researchers note in the Feb. 9 *Nature*.

Applying those models to look farther back in time, the team detected a drop in sea level after the eruption of Krakatoa. In fact, even though the oceans were gradually warming because of changes in Earth's climate, sea level wouldn't have returned to its pre-Krakatoa height until around 1950, says Gleckler.

Thanks to rapidly rising concentrations of greenhouse gases, the heat content of Earth's oceans is increasing much faster today than it did early in the 20th century. The models suggest that the drop in sea level caused by the 1991 eruption of Mount Pinatubo, which lofted a comparable amount of aerosols as Krakatoa did, lasted only a decade or so. —S.P.

BIOMEDICINE

Alzheimer's drug shows staying power

Since its U.S. approval in 2003, the drug memantine has been prescribed to slow mental decline in people with moderate-to-advanced Alzheimer's disease. But studies hadn't addressed the drug's long-term effectiveness.

Researchers report in the January *Archives of Neurology* that 66 people taking memantine for Alzheimer's disease continued to benefit for a year, the longest test of the drug to date, says coauthor Barry Reisberg, a geriatric psychiatrist at New York University School of Medicine.

During the first 6 months of the study, the benefits were assessed in comparison with Alzheimer's patients randomly assigned to take placebos (*SN: 4/5/03, p. 211*). People who switched from placebo to memantine after 6 months also benefited by 1 year.

Memantine slows mental decline in Alzheimer's but doesn't reverse it, which is also the case for cholinesterase inhibitors, which doctors prescribe for patients in the early stages of the disease.

Researchers are investigating whether patients can get more benefit from using memantine along with cholinesterase inhibitors, Reisberg says. —N.S.

Books

A selection of new and notable books of scientific interest

DISEASES OF TREES AND SHRUBS: Second Edition

WAYNE A. SINCLAIR AND HOWARD H. LYON

With more than 2,200 color images and 350 black-and-white photographs and drawings, this revised guide offers a comprehensive survey of the diseases of North American trees and shrubs. Originally published in 1987, this reference book by Sinclair, professor emeritus of plant pathology at Cornell University, and Lyon, a photographer from Cornell, has been heralded as one of the best horticultural books of the century. The oversize book is organized by disease-causing agents and includes symptoms and signs of hundreds of diseases, from blights to rust to rots. The book includes a searchable CD-ROM with a detailed bibliography of further references. This guide will be useful to horticulturists, plant-health specialists, and students. ***Cornell, 2005, 660 p., b&w and color photos, hardcover, $85.00.***

EXTINCTION: How Life on Earth Nearly Ended 250 Million Years Ago

DOUGLAS H. ERWIN

While most people are aware of the cataclysmic meteor impact that led to the extinction of the dinosaurs 65 million years ago, few know of the earlier, far greater, extinction of 95 percent of life on Earth. Erwin, curator of the Smithsonian's National Museum of Natural History, describes how life on Earth was nearly destroyed at the end of the Permian period, 250 million years ago. He first introduces the common perpetrators of extinction throughout Earth's history: massive volcano eruptions, global cooling, and extraterrestrial impacts. He then reviews the late-Permian fossil record, which includes only a few species but is virtually identical in places as far-flung as Utah and Iran. The author completes the story by explaining what this paleontological, as well as geological, evidence can tell scientists about the dramatic and deadly shift in the Earth's environment. ***Princeton, 2006, 320 p., hardcover, $24.95.***

THE DANCE OF MOLECULES: How Nanotechnology Is Changing Our Lives

TED SARGENT

Nanotechnology is all the rage among materials scientists today, but few laypersons are fully aware of its potential, Sargent asserts. A professor of nanotechnology at the Massachusetts Institute of Technology, he trumpets the promise of his field throughout science and technology. Nanotechnologists are manipulating structures that would have to be blown up 1,000 times even to be seen in an optical microscope, he explains. The book offers a detailed history of nanotechnology and the development of remarkable structures such as carbon nanotubes. Possible applications for such devices include diagnosing cancerous tendencies of individual cells, controlling a drug's delivery with implanted microchips, making better solar panels than exist today, and creating computers that mimic biological systems. Going beyond a mere thought exercise, Sargent calls on scientists to embrace this new technology in a responsible way that will improve people's lives without threatening their health or the environment. ***Avalon, 2006, 234 p., b&w photos, hardcover, $25.00.***

BREAKING THE SPELL: Religion as a Natural Phenomenon

DANIEL C. DENNETT

Religion and science can be considered mutually exclusive ways of viewing the world. But what would happen if the explicative power of science were applied to religion? Can it be done? Should it be done? Philosopher Dennett, author of the Pulitzer Prize–nominated book *Darwin's Dangerous Idea*, dares to address these questions and more in this unusual volume. He attempts to analyze religion from the perspective of the dispassionate observer, delving into its cultural and psychological origins, its careful cultivation over millennia, its dissemination within and among populations, and its role in forming allegiances among peoples and nations. Dennett ponders whether religion is good for people and whether it should form the basis of morality. In the end, the author asserts a pressing need to protect democracy and free thought from people who threaten human rights on the basis of blind religious faith. ***Penguin, 2006, 464 p., hardcover, $25.95.***

THE WEATHER MAKERS: How Man Is Changing the Climate and What It Means for Life on Earth

TIM FLANNERY

About 8,000 years ago, Earth entered what some scientists call the Anthropocene era, the period in which people radically alter Earth's environment. The mammoth and accelerating production of methane and carbon dioxide by agriculture and industry has set into motion climatic changes that are having wide-ranging and devastating effects, Flannery writes. The author, a conservationist and academic, says he was once skeptical about greenhouse gases and global warming. But after spending years researching the evidence, which he presents here, he has become convinced that energy use by people is ruining the planet. He points to the increasing destructive power of hurricanes and other storms, the significant warming of the Pacific Ocean and the poles, glacial melting, and the slow disappearance of coral reefs and other organisms. Flannery examines what these patterns could mean for the future of Earth's environment. For instance, global temperature changes in foreseeable ranges could cause the collapse of the Gulf Stream or the destruction of the Amazon rainforest. Individuals and nations must turn to solar and wind power and to hybrid and carbon-free transportation to stop the climate-change crisis, the author asserts. ***Grove/Atlantic, 2006, 320 p., color plates, hardcover, $24.00.***

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LETTERS

Pain, pain, go away

I'm pleased that images are now available to prove that self-control over pain works ("Brain Training Puts Big Hurt on Intense Pain: Volunteers learn to translate imaging data into neural-control tool," *SN: 12/17/05, p. 390*). Actually, I and many other moms could have helped the researchers. During childbirth, we simply focused on various breathing techniques and discovered that the pain became manageable or disappeared. I've continued to use these breathing techniques in the dentist's chair for 40 years. Chronic pain is, of course, a different situation. It is interesting, however, to wonder if practiced-breathing and relaxation techniques would be useful for those suffering from this awful situation. Having good science back up anecdotal evidence is surely moving in the best direction.

LINDA MANGELSDORF, NEWBURGH, N.Y.

Airing differences

"Changes in the Air: Variations in atmospheric oxygen have affected evolution in big ways" (*SN: 12/17/05, p. 395*) raises the question of how oxygen levels have changed over the past 2 centuries, when carbon dioxide has been increasing.

JOHN MILLS, DECATUR, ALA.

There is a problem in this interesting article. The graph of oxygen content versus time doesn't agree with the text. Specific example: "About 255 million years ago ... the oxygen concentration stood at 30 percent." The graph shows this concentration at about 290 million years ago. Which is correct, graph or text?

ALAN SOBEL, EVANSTON, ILL.

I have long wondered about giant insects in the Carboniferous period. What about barometric pressure? Even with increased oxygen, would such insects be able to fly unless air pressure was higher as well?

LINDON DURVIN, RICHMOND, VA.

Data gathered during the past decade show that the seasonal variations in the concentration of oxygen mirror those of carbon dioxide. Some discrepancies noted here arose because ancient concentrations of atmospheric oxygen have been estimated by various methods, including isotopic analyses of sediments. Also, the graph depicts new data that hadn't been available to the other scientists cited in the text. Regarding insect flight, experiments with modern insects suggest that oxygen availability, not air density, is the factor that determines whether insects can fly. —S. PERKINS

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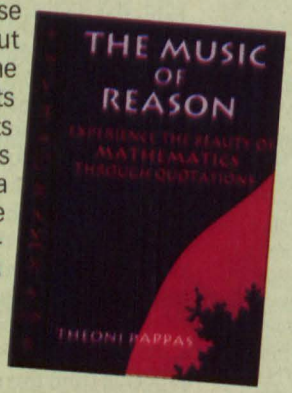
MUSIC OF REASON



The power of mathematics has literally defined the limits of the universe and all it contains. But equally certain is that the science has perplexed its share of people with its unique brand of logic. This collection of quotes—a mixed bag of the humorous and the philosophical from sources as diverse as Alice in Wonderland author Lewis Carroll, Albert Einstein, and Napoleon Bonaparte—is a thought-provoking sampler on the subject.

Some of the musings included are:

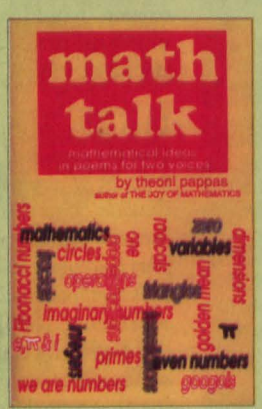
- Benjamin Disraeli: "There are three kinds of lies: lies, damned lies, and statistics."
- Henri Poincaré: "The mathematician does not study pure mathematics because it is useful; he studies it because he delights in it and he delights in it because it is beautiful."
- Huckleberry Finn: "I had been to school ... and could say the multiplication table up to 6x7=35, and don't reckon I could ever get any further than that if I was to live forever. I don't take stock in mathematics, anyway."

Consists of 15 chapters of quotes organized by themes such as Mathematics & Art or Mathematics & the Imagination, with wonderful graphics sprinkled throughout this gem of a book.

—Wide World Publishing/Tetra, 5" x 7", 140 pages, paperback $9.95

MATH TALK

Mathematics and poetry? Why not? Pappas delivers a new way to enjoy and learn some sublimely abstract notions, such as tessellations, fractals, googols, imaginary numbers, and much more. Creative juices were certainly at work when this book of mathematical dialogues was created. It presents a new way to enjoy and learn mathematical ideas via poetic dialogues read by two people. Discover and enjoy a new twist to mathematical themes with this refreshing volume of mathematical poems for two voices.



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MATH-A-DAY

Pappas has come up with yet another way to make math part of your life. Not a calendar, not a reference book, but a compendium of mathematical information that will give you your math fix everyday. *Each day -*

- kick starts your brain with a problem or puzzle (detailed solutions are included)
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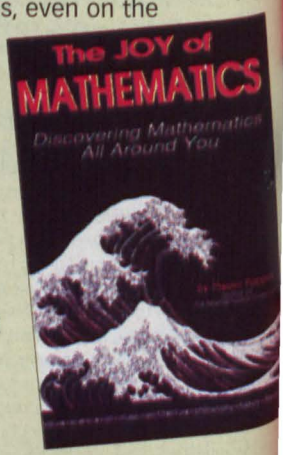
—Wide World Publishing, 5½" x 8½", 250 pages, paperback $12.95

THE JOY OF MATHEMATICS



Part of the joy of math is that it's everywhere—in soap bubbles, electricity, da Vinci masterpieces, even on the pool table. This collection of over 200 clearly illustrated puzzles and tips demonstrates mathematical concepts like Fibonacci numbers, probability and magic squares, and shows where they turn up in the "real" world.

You'll find out what a googol is, invent numerical palindromes and read a thorny problem of logic that was stumping them back in the eighth century. Dive right in and open this book at random, and you'll enjoy such sections as the Paradox of the Unexpected Exam and Lewis Carroll's decidedly Tangled Tale— THE JOY OF MATHEMATICS adds up to the most fun you've had in a long time.

—Wide World Publishing/Tetra, 5½" x 8½", 242 pages, paperback $10.95

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