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ScienceNews

MAGAZINE OF THE SOCIETY FOR SCIENCE & THE PUBLIC ▪ AUGUST 16, 2008

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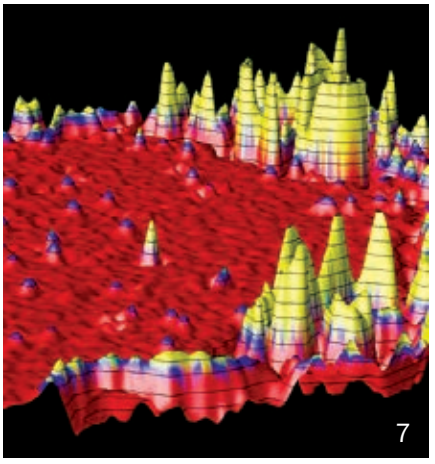
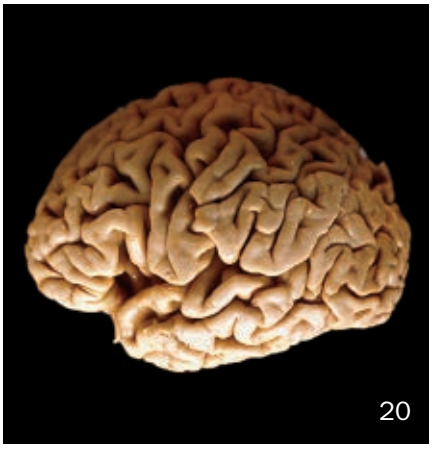
ScienceNews





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In the News

5 **STORY ONE**
Scientists enlist nanomagnets to kill cancerous cells

7 **MATTER & ENERGY**
Blame water's behavior on quantum uncertainty

With help from graphene, scientists get images of hydrogen atoms

8 **BODY & BRAIN**
SIRT1 ties metabolism to body's daily rhythms

Low-carb dieters lose more than those on low-fat plan

Stomach bacterium could decrease asthma risk

Viagra gives some women a sexual boost too

Gene variant increases risk of muscle pain for those on statins

10 **LIFE**
Bumblebee escapees may spread disease

Molecule tuned to magnetism may help migrating animals get oriented

No matter the branch in the snake tree of life, all fangs have a common origin

Parasitic vine's sucking strategy may provide the means for a counterattack

12 **HUMANS**
Kiddie-cam tracks toddlers' progress as they take developmental leaps

Words not required for thinking about quantities

Features

14 **BUILT FOR SPEED**
COVER STORY: If zoo inhabitants could compete in the Olympics, humans would have a hard time winning any medals.
By Susan Milius

20 **NEURON KILLERS**
Circumstantial evidence suggests that certain proteins gang up to murder neurons and cause neurodegenerative diseases. But scientists haven't yet established guilt beyond all doubt.
By Tina Hesman Saey

24 **WELCOME TO THE QUANTUM INTERNET**
Researchers are beginning to develop the first quantum computer networks, opening a world of weird communication possibilities.
By Davide Castelvecchi

Departments

2 **FROM THE EDITOR**

4 **NOTEBOOK**

30 **FEEDBACK**

31 **BOOKSHELF**

32 **COMMENT**
Better communication between astronomers and the public is needed to improve the world's awareness of the European Space Agency's achievements, scientists said at a recent forum.

On the cover:
From a standing start, bushbabies can easily outjump humans.
Photo: John Downer Productions

ScienceNews
MAGAZINE OF THE SOCIETY FOR SCIENCE & THE PUBLIC

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Letters: editors@sciencenews.org

Science News (ISSN 0036-8423) is published biweekly, for $54.50 for 1 year or $98.00 for 2 years (international rate $80.50 for 1 year or $161 for 2 years) by Society for Science & the Public, 1719 N Street NW, Washington, DC 20036. Preferred periodicals postage paid at Washington, DC, and an additional mailing office.

Subscription Department: P.O. Box 1205, Williamsport, PA 17703-1205. For new subscriptions and customer service, call 1-800-552-4412.

Postmaster: Send address changes to Science News, P.O. Box 1205, Williamsport, PA 17703-1205. Two to four weeks' notice is required. Old and new addresses, including zip codes, must be provided. Printed in U.S.A. on recycled paper.

It's a heavy burden to report on heavy water

If you pay attention in school, you can learn a lot about science.

Surely you remember, for example, that hydrogen is the simplest atom — a single proton surrounded by a single electron. And that hydrogen has a heavier form, with the proton joined by a neutron, called deuterium. And sooner or later the secret leaked out that water molecules, consisting of two hydrogen atoms plus an oxygen, might sometimes be built from deuterium instead of the ordinary lighter variety.

On a more advanced level — maybe we're in high school now — water displays some mysterious properties. Its density drops as it freezes, for instance, and it exhibits dozens of other anomalies compared with other liquids (*SN: 1/26/08, p. 58*).

You probably learned that many of water's oddities could be blamed on the curious "hydrogen bonds" weakly linking one water molecule to another. But it's unlikely that anybody ever mentioned what happens to hydrogen bonds in "heavy water," made with deuterium.

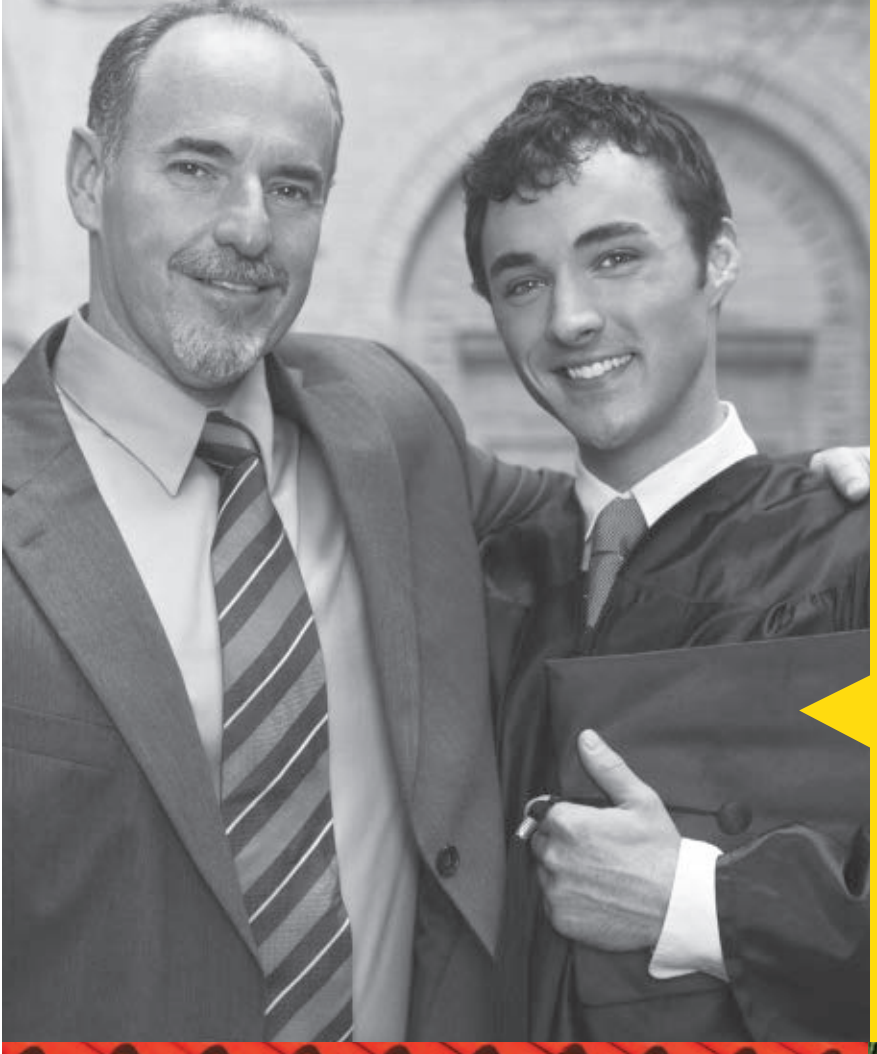
The answer: The hydrogen bonds are longer, by about 4 percent when D_2O is in the liquid state. At the same time, the bonds between D and O within the molecules are 3 percent shorter than those between H and O. You will not have encountered such molecular intelligence in any textbook. For that you need *Science News*.

You can read about the dimensional differences distinguishing heavy from light water in a story by Davide Castelvecchi on Page 7 in this issue (or for a longer version, online at www.sciencenews.org, under Matter & Energy). The study reporting the new data, being published in *Physical Review Letters*, provides intriguing new clues to some of water's deep mysteries. It's a step toward answering some of the questions that chemists and physicists have long asked themselves (and dodged when posed by their students).

Such steps are not headline-makers, but they are nevertheless worthy of reporting. For those who paid attention in school, and for anyone eager to stay informed about the state of scientific knowledge, these are the nuggets of news that illustrate the subtlety of science's substance and the nuances of the scientific process. Results of this sort rarely break into the news lineup of general mass media. You won't see them in the typical daily newspaper or on that little message line scrolling across the bottom of the TV screen tuned to CNN.

You will, of course, find them reported here. *Science News* has different priorities.

— Tom Siegfried, Editor in Chief



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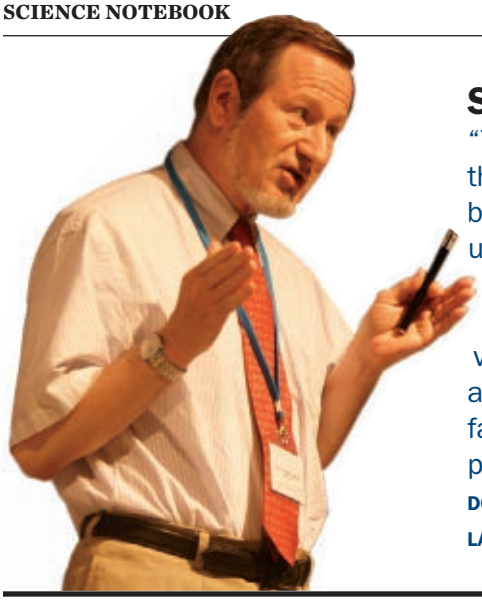

Scientific Observations

"Those discoveries that most change the way we think about nature cannot be anticipated.... Beware of subtle, unexplained behavior; don't dismiss it. Frequently nature does not knock with a very loud sound but rather a very soft whisper, and you have to be aware of subtle behavior which may in fact be a sign that there is interesting physics to be had." —NOBEL LAUREATE DOUGLAS OSHEROFF, SPEAKING IN JULY AT THE NOBEL LAUREATE MEETINGS AT LINDAU IN GERMANY.

Science Past: 50 Years Ago

From Science News Letter, August 16, 1958

WATERBIRDS DISPOSSESSED — Marshes, swamps and wetlands throughout the United States are on their way out. Going with them are hundreds of our waterbirds. Ducks, geese, herons, grebes, coots and many other birds that depend on wetlands for nesting, shelter and food are being dispossessed. As water and land are being taken over for farming, industrial development or to give expanding cities room, whole populations of waterbirds are disappearing, many going north and south of the border. Three scientists, studying a small marsh in Utah, have been able to measure the direct effects of lost wetlands on waterbird populations. Of 17 species found nesting or living in the marsh in 1950, six did not nest there at all in 1955; four species were still around but had two-thirds fewer nests, while nests of the other species were also present in reduced numbers.

Science Future

September 10–13

First International Ragweed Conference in Budapest. Visit www.nki.hu/ragweed

September 15

An Evening with Frank Wilczek: *The Lightness of Being.* Reception and book signing by the physics Nobel laureate at the New York Academy of Sciences. Visit www.nyas.org/events

October 4

Great Insect Fair at Pennsylvania State University. Visit www.ento.psu.edu/scied/fair.html

SN Online

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SN TODAY

Learn what a deep-ocean survey found off Ireland's coast in "Seafloor chronicles" in the Earth section.

MATH TREK

In designing the National Aquatics Center in Beijing, newly built for the Olympics, architects tapped advanced mathematics to make the building look like froth. See "A building of bubbles" by Julie Rehmeyer.

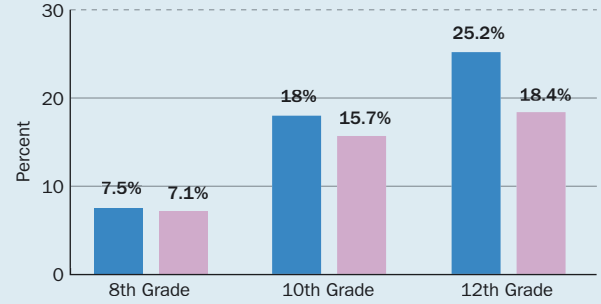
SCIENCE & THE PUBLIC

Policy writer and SN blogger Janet Raloff offers a nuanced discussion of animal rights activism in "Animal rights and wrongs," and she also gives us the skinny on trans fats in "This trans fat is vindicated."

Science Stats

GENDER DRUG GAP

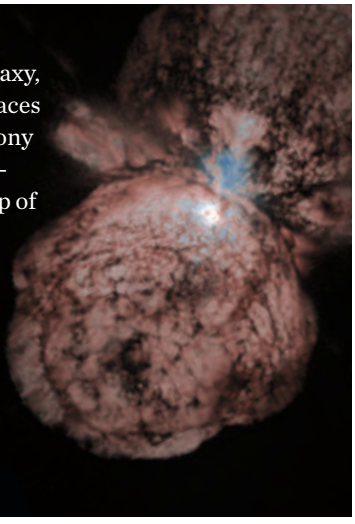
Percentage of U.S. 8th-, 10th- and 12th-graders who admit to using illicit drugs in the past 30 days, by gender ■ Male ■ Female



SOURCE: CHILDSTATS.GOV, 2007

The (-est):

The brightest star in the galaxy, Eta Carinae (shown), now faces a tough competitor. The Peony nebula star, which researchers scrutinized with the help of NASA's Spitzer Space Telescope, blazes with the light of 3.2 million suns. Though Eta retains her reign, shining as bright as 4.7 million suns, the scientists say pinning down exact luminosities in the Milky Way's dusty center is difficult.



JON MORSE/UNIV. OF COLORADO, NASA; ©ARUP+BEN MCMILLAN; C. FLEMMING/LINDAU NOBEL LAUREATE MEETINGS

“Think of it sort of as a spiderweb, and the atoms you want to view are flies on the spiderweb.” —ALEX ZETTL, PAGE 7

Matter & Energy Quantum matters in water
Imaging the smallest of the small

Body & Brain Metabolism runs like clockwork
Low-carb keeps weight off

Life When diseased bees get loose
Fangs formed first and then moved

Humans Little tykes learn to see like adults
Counting is so overrated

In the News

STORY ONE

Fatal attraction: Nanomagnets tackle disease

Technique uses heat to kill cancerous cells

By Janet Raloff

A researcher at MagForce Nanotechnologies AG transfers a ferrofluid containing specially coated magnetic nanoparticles into a tube. The Berlin-based company was the first to begin clinical trials of the tiny magnetic beads as a cancer therapy. A U.S. company expects to begin trials in 2009.

MAGFORCE NANOTECHNOLOGIES

A new wave of therapies can exert a magnetic hold over disease—literally. The therapies employ powerful, roughly spherical magnets to help kill carefully targeted diseased cells and nothing else. What makes these magnets special is their size. Each is about a thousandth the diameter of a human hair.

Most researchers in the field are designing these billionth-of-a-meter–scale magnets to serve as highly localized space heaters. Under the influence of an external magnetic field, the magnetic particles will warm to temperatures that will kill immediately adjacent cells.

Two U.S. research groups recently reported success in developing high-performance iron-cobalt nanomagnets for cancer therapy. New studies by another group describe the ability to target, track and deliver killer heat with a weaker, but potentially less toxic, class of cobalt-free magnetic nanoparticles.

If these nanonuggets and their ilk perform as expected, they should increase cancer survival rates and lower the toxicity associated with conventional therapies. Indeed, MagForce Nanotechnologies AG, based in Berlin, is exploring the idea of making its tiny magnetic beads do double duty: heat-treat tumors in the body and at the same time deliver drugs directly into malignancies. Direct delivery should largely eliminate the poisoning of healthy tissue—a primary side effect of most existing cancer treatments.

Some dozen teams around the world are developing these therapeutic beads, notes Robert Ivkov of Johns Hopkins University in Baltimore. He and others have established the technology’s proof of concept in test-tube and animal studies.

MagForce, the only group to have tested nanomagnet therapy in people, appears closest to commercialization. Over the past five years, it has conducted trials, enrolling patients with at least eight tumor types, according to Uwe Maschek, the company’s chief executive officer. The most advanced trial is currently studying some 65 patients with late-stage, recurrent glioblastoma multiforme, a type of brain cancer. Individuals with this cancer typically survive no more than seven months, he notes.

By next year the company hopes to establish whether its nanomagnetic therapy lengthens survival by at least three months. If it does, Maschek says MagForce could receive regulatory approval to market its technology in the European Union by the first quarter of 2010.

On June 2, Triton BioSystems Inc., Ivkov’s former employer, merged with another company to form Aduro Biotech, based in Berkeley, Calif. The new firm’s website describes a planned 2009 trial that would administer therapeutic nanomagnets to U.S. cancer patients. »

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SN Today at **www.sciencenews.org**

» MagForce founder Andreas Jordan began exploring nanomagnet cancer treatment some 20 years ago. He aimed to use hyperthermia — essentially inducing highly localized 44° Celsius to 50°C fevers to kill diseased tissue. Not only are cancer cells much more sensitive to heat, but radiation and cancer drugs also tend to work better on heat-stressed cells.

In fact, researchers have long been interested in using heat to treat disease. A research team at Presbyterian-St. Luke's Hospital in Chicago led by R.K. Gilchrist reported a promising new approach — a full half century ago.

The surgeons injected a fine, iron-oxide powder into lymph nodes suspected of hosting metastases — the seeds of new cancers — and applied a magnetic field to heat the micromagnets. It worked like a charm, the researchers reported in a 1957 *Annals of Surgery* paper. "The possible application of such a tool," Gilchrist's group concluded, "requires little imagination."

Yet the technology languished for much of the next four decades. Ivkov says it required something that was unusual in the 1950s — research teams that integrated chemists, materials scientists, cell biologists and physicists. Today such collective efforts tailor tinier and more effective magnets, and are perfecting strategies to activate the nanonuggets without burning healthy tissues along the way.

Nearly all research groups work with iron-oxide nanomagnets. But in the April 1 *Journal of Applied Physics*, Michael McHenry's group at Carnegie Mellon University in Pittsburgh reported developing a non-oxide iron-cobalt particle with a magnetic strength five to 10 times that of oxide magnets. This could permit treatment using fewer magnetic nanoparticles, McHenry says, or a lower-powered external field to heat the nanobeads.

Ultimately, those beads will receive a coating to shield the potentially toxic cobalt and to keep the nanonuggets from looking like foreign objects that the body should mark for disposal. This coating can also be studded with antibodies to selectively bind to receptors found on the surface of a target, such as a cancer cell.

Blue fluid with therapeutic nanomagnets targets tumor cells (right). But the nanomagnets leave healthy cells (left) alone.

In a *Journal of the American Chemical Society* paper posted online in mid-July, Kenneth Scarberry and his colleagues at the Georgia Institute of Technology in Atlanta describe an oxide version of the iron-cobalt recipe for their nanobeads.

They gave their nanomagnets a "sugar coating" of polygalacturonic acid, Scarberry says, and then linked tiny proteinlike structures to the coating. The attached peptides serve as hooks to grab onto a receptor that's only present on ovarian cancer cells.

The scientists report that by placing a big magnet on the skin of a treated mouse, they can pull injected nanobeads to the other side of the skin, which could facilitate eventual nanobead removal. But the application the researchers are most excited about, Scarberry says, is a dialysis-like system. It would pump liquids from inside the body through a tube outside the body. Nanomagnets treated with ovarian cancer cell "hooks" would line the inside of the tube. The beads would catch and hold passing metastatic cells, filtering the blood before it is returned to the body.

Scientists at the University of California, Davis School of Medicine and the former Triton BioSystems collaborated for several years on related studies using a different nanoparticle model. Instead of creating sugar-coated magnets, they essentially created sugar balls studded throughout with magnetic iron-oxide "raisins," explains Ivkov.

His group attached antibodies that bind to receptors on breast cancer cells. Then they injected the nanomagnets into mice that had been seeded with those cancer cells and heated the beads for 20 minutes. Tumors in the treated animals shrank. Far more so, in fact, than predicted.

But cancer treatment is far from the only medical application being eyed for these nanomagnets. Scarberry first became interested in the technology a couple of years ago when he realized it might offer a clever adjunct to standard therapy for HIV — the AIDS virus. He won't say much except that his preliminary data on this "look promising." ■

Back Story Magnetism, then and now

ca. 575 B.C.
Thales of Miletus argues that magnets' power to move things must mean they are alive.

1269
Frenchman Peter Peregrinus writes a major treatise on magnets, assigning poles to a lodestone.

1600
English physician William Gilbert publishes *De Magnete*, a comprehensive guide to magnetism.

1957
A Chicago team proposes heating magnets (injected into the body) with an external field to kill tumor cells.

2003
MagForce launches the first clinical trials with nanomagnetic particles to treat disease.

Today
Researchers explore the use of nanomagnets to fight infection, deliver drugs and clean up oil spills.

MAGFORCE NANOTECHNOLOGIES

Matter & Energy

For longer versions of these and other Matter & Energy stories, visit **www.sciencenews.org**

Quantum effects make H_2O weird

Bond lengths are different in heavy, light water molecules

By Davide Castelvecchi

Heavy water is not just heavier. Swapping each H in H_2O with a D—hydrogen's isotope deuterium—changes water's properties. The deuterium version is mildly poisonous, and its freezing point is 4° Celsius, instead of 0°C. Such differences reveal that quantum effects, which aren't usually manifest to the naked eye, rule in ordinary water, researchers suggest.

Alan Soper of the Rutherford Appleton Laboratory in Didcot, England, and Chris Benmore of Argonne National Laboratory in Illinois found that, in the liquid state, the distance between oxygen and deuterium nuclei in a D_2O molecule is 3 percent shorter than the distance between oxygen and hydrogen in an H_2O molecule. Conversely, hydrogen bonds—relatively weak bonds connecting the oxygen in one molecule with the hydrogen or the deuterium in another—are 4 percent longer in heavy water than in light, the team reports in an upcoming *Physical Review Letters*. These differences are less than 1 percent in water vapor, where molecules are isolated.

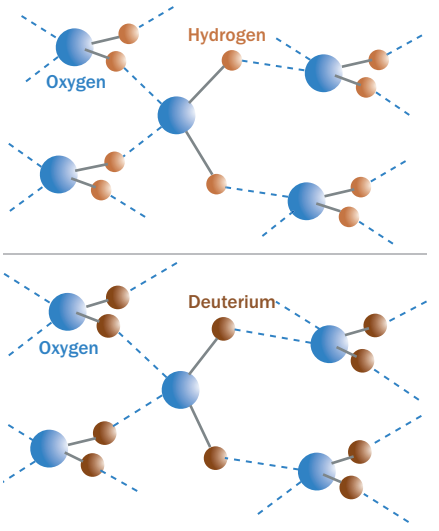
"A 4 percent change in bond length is quite a bit," says Michael Rübhausen of the University of Hamburg in Germany.

The deuterium nucleus, which contains a neutron in addition to the usual single proton, is heavier than the hydrogen nucleus. That makes deuterium nuclei behave more like classical objects, so the positions of the deuterium nuclei in space suffer less from the quantum uncertainty that "smears out" a proton's location. These nuclei stick closer to the oxygen nuclei within the heavy water molecule, and an oxygen atom from a nearby heavy water molecule exerts a weaker pull.

The susceptibility of ordinary water to quantum effects may explain some of its unusual features, such as its high surface tension and the fact that its density decreases when it freezes, Soper suggests. "Probably all the properties of water are affected by the hydrogen bond length," he says.

Rübhausen says the difference could also help explain some surprising results he and his collaborators reported last year. They compared RNA made with ordinary organic molecules with RNA made of those molecules' mirror images to understand why life always uses one type and not the other. Chemically, the molecules and their mirror images should be identical. But the researchers found small differences in the energy it took to excite electrons in the two types of RNA when the RNA molecules were in ordinary water. The differences disappeared in heavy water.

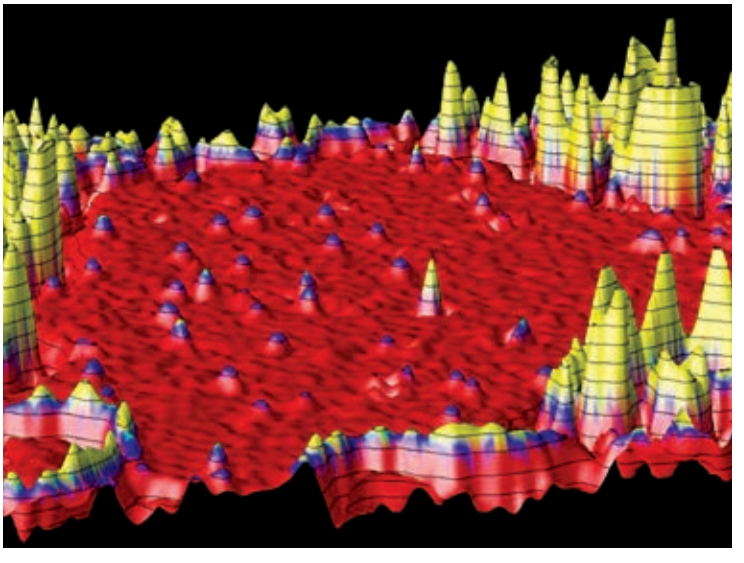
Different bond lengths in D_2O could somehow mask or enhance the energy differences in the two types of RNA, Rübhausen speculates.



Bond lengths within and between molecules of water (top) differ from those in heavy water (bottom) due to quantum effects, researchers find.

Spotting the smallest atoms

No room is left at the bottom: A team of physicists has used a transmission electron microscope to spot single hydrogen atoms—the smallest atoms of them all. Previously, such microscopes had trouble imaging single atoms lighter than carbon. The University of California, Berkeley team visualized defects and impurities—including atoms of hydrogen (purple peaks)—on graphene, the thin nets of carbon that stack up to form graphite. Graphene's uniformity improved contrast. "Think of it sort of as a spiderweb," says study coauthor Alex Zettl, "and the atoms you want to view are flies on the spiderweb." Using graphene could enable scientists to understand the structure of molecules that have been difficult to image with other techniques such as X-ray diffraction. And thanks to graphene's sturdiness, single-molecule motions and chemical reactions could be filmed as they happen, the team suggests in the July 17 *Nature*. —*Davide Castelvecchi*



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Body & Brain

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Protein links metabolism to clock

Work could lead to drugs for obesity, aging and jet lag

By Tina Hesman Saey

Cue stomach rumbles
SIRT1 sets internal clock
To metabolism

Timing is everything, especially when it comes to basic biological functions such as eating, sleeping and liver activity. Scientists have known for ages that metabolism is tied to the body's daily rhythms but have not known how.

Now, two groups of researchers report in the July 25 *Cell* the discovery of a molecule that links metabolism to the circadian clock in mice. The missing link turns out to be a protein called sirtuin 1, or SIRT1, which is also a key regulator of aging.

Uncovering the mechanism that links metabolism and circadian rhythms could lead to drugs for combating obesity, aging and jet lag and for helping shift workers reset their body clocks.

"It's an interesting connection," says Herman Wijnen, a circadian geneticist at the University of Virginia in Charlottesville who was not involved in the new studies. "It helps us understand one important aspect of how clocks and metabolism relate to each other."

SIRT1 has also been a source of research interest because of its involvement in the effects of resveratrol, a molecule found in red wine and other foods that mimics the health benefits of a nutritious, calorie-restricted diet.

Body rhythms are governed by molecular clocks that take about a day to complete a full cycle, hence the label circadian. The clocks are composed of proteins whose concentrations or levels of activity rise and fall like the tides.

Most animals have a main pacemaker in the brain. Triggered by light, this clock can reset within a couple of days. But almost every cell in the body contains a clock, and these clocks are reset by the introduction of food, by a change in body temperature or by other metabolic signals.

For the body to function normally, all the cellular clocks must synchronize with the main clock in the head, says Ueli Schibler of the University of Geneva in Switzerland and coauthor of one of the studies. But the cellular clocks take longer to reset, a week or more. This mismatch between the cellular clocks and the brain clock is one reason for jet lag.

That's probably as it should be, Schibler says. "Imagine if you stand up in the middle of the night and eat a sandwich. You don't want your clock reset just because of one sandwich."

"Imagine if you stand up in the middle of the night and eat a sandwich. You don't want your clock reset just because of one sandwich."
UELI SCHIBLER

In 2006, researchers led by Paolo Sassone-Corsi, a molecular biologist at the University of California, Irvine and coauthor on the other *Cell* study, reported that a protein named CLOCK is a component in cellular clocks. It drums out the beat of circadian rhythm by chemically modifying a histone protein, which packages DNA in the cell. CLOCK transfers an organic molecule called acetyl to a histone protein. That action causes DNA to open up, helping to turn on the genes contained within the DNA.

Such chemical alterations of DNA and its associated proteins are called epigenetic modifications. They help control development, behavior and metabolic processes in the body.

In order for epigenetic modifications to be most effective they should be reversible, so cells can switch genes off and back on again when needed, such as when a person eats a sandwich and needs to make hormones to tell the brain that the stomach is full or to deal with the sudden influx of energy.

No one knew what CLOCK's counterpoint — a protein that would remove the acetyl and turn genes off — might be. But Sassone-Corsi and his colleagues suspected that sirtuins might be involved because the proteins respond to a cell's energy state by plucking acetyl groups from histones and other proteins. The team hypothesized that sirtuins might also interact with cellular clocks.

In the new study, Sassone-Corsi's group shows that SIRT1 indeed acts as tick to CLOCK's tock, removing an acetyl group from histones and also from CLOCK's partner, BMAL1.

Schibler and colleagues report similar results, demonstrating that SIRT1 levels rise and fall throughout the day, and that SIRT1, CLOCK and BMAL1 interact in a circadian manner. Schibler's group also found that SIRT1 is involved in removing acetyl groups from another clock component, a protein called PER2. That action leads to degradation of PER2, one of the gears that keeps the clock moving.

Both groups found that SIRT1 is active in liver clocks. The liver performs many of its functions, such as detoxifying harmful substances and processing fat and cholesterol, on a schedule.

Tying the liver's clock to metabolic activity makes sense, says Wijnen, and SIRT1's connection to the clock may be important for timing the organ's functions. Breakdowns in the body's clocks could put those clocks out of sync with the brain's timer, possibly leading to disease.

Metabolic links to gene activity and circadian rhythms may help explain some mysteries of obesity and aging, but the researchers say they still don't know exactly how SIRT1 keeps clocks ticking.

"The clock really dominates all of our physiology," says Sassone-Corsi, so it's not surprising to find molecules that are involved in metabolism, aging and obesity linked to the circadian rhythms . "But it is important to find the molecular basis of this mechanism," he adds. ■

29.7 million

The number of people in the United States taking statins in 2005; that number increased from 15.8 million in 2000

Go against the grains, diet study suggests

Low-carb beats low-fat in weight loss, cholesterol

By Nathan Seppa

Talk about taking a hit in the breadbasket. A new study finds that a low-carb diet results in greater weight loss and better cholesterol than a low-fat regimen that promotes a lot of grains and fruits. A Mediterranean diet yielded results between the two, researchers in Israel report in the July 17 *New England Journal of Medicine*.

By conducting a trial within a single workplace, the scientists kept 85 percent of study participants on their respective diets for a full two years, a coup among diet studies. High dropout rates have historically skewed the results of such studies.

While people lost at least some weight on all three diets, the differences were statistically significant. "The old food pyramid is going to get turned on an angle," says study coauthor Iris Shai, a nutritional epidemiologist at Ben-Gurion University of the Negev. "Maybe now it's a little more questionable that we should be basing our diets on carbohydrates."

The team recruited 322 overweight people, average age 52 and mostly men, and randomly assigned them to one of the diets. Some 272 completed the study.

The low-fat diet closely adhered to guidelines developed by the American Heart Association, which recommends plenty of low-fat grains, vegetables, fruits and legumes. Dietitians counseled participants to strictly limit fats and meats and to keep daily calorie intake under 1,800 a day for men and 1,500 for women.

The Mediterranean diet had the same overall calorie limits, but participants could eat fats, mainly olive oil and nuts, in moderation. These dieters also ate poultry and fish but little red meat.

The low-carbohydrate group ate an Atkins diet, in which they could consume all they wanted, provided very little of it was carbohydrates. Protein and fat intake weren't limited, but dietitians urged participants to choose vegetarian foods when available. All groups avoided trans fats.

Because the study participants ate lunch at the same cafeteria, they obtained food that fit their diets for the midday meal, the largest of the day.

After two years, low-carb dieters had lost an average of 5.5 kilograms, compared with the 3.3 kilograms lost by the low-fat dieters. Mediterranean dieters showed results between the two.

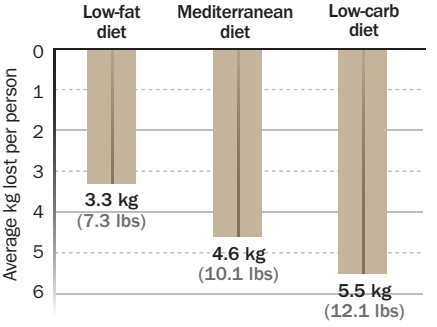
Low-carb dieters also increased their average HDL cholesterol, the good kind, by 8.4 points — 2 points more than the others. LDL, the bad cholesterol, didn't change significantly in any group. Also, the low-carb and Mediterranean dieters lowered blood levels of triglycerides (fats) significantly more than the low-fat group.

Other studies have tackled the low-fat versus low-carb issue, with mixed results. Some found that low-carb diets induced quick weight loss but that early gains faded after six months.

This study and previous reports "are showing repeatedly that higher-fat diets do not worsen the overall blood cholesterol profile," says internist William Yancy Jr. of Duke University in Durham, N.C.

The Atkins Foundation provided partial support for the study.

Leaving the bread behind



Data from 272 people show that on average low-carb dieters lost more weight than low-fat dieters after two years.

AVIK NANDY

NEWS BRIEFS

Asthma oddity

Children infected with a common stomach bacterium are less likely to have asthma than other kids, according to a study in the Aug. 15 *Journal of Infectious Diseases*. Microbiologist Martin Blaser of New York University and colleague Yu Chen analyzed data from a health information database and found children ages 3 to 13 who had a *Helicobacter pylori* infection were less than half as likely to have had asthma as were kids without an infection. It's possible that the infection might quell the immune system or lead to the production of compounds that do, Blaser says. — *Nathan Seppa*

Viagra for women

A new study in the July 23/30 *Journal of the American Medical Association* suggests Viagra, well known for treating male impotence, can relieve sexual difficulties in women caused by antidepressant use. Women on serotonin reuptake inhibitors who took Viagra reported improved sexual function after eight weeks, compared with those who took a placebo. It remains unclear why Viagra would work for women on these drugs but has not worked for other women, says study coauthor Julia Heiman of the Kinsey Institute at Indiana University in Bloomington. — *Nathan Seppa*

Statin snag

Cholesterol-reducing drugs called statins do their job with remarkable efficiency, but in rare cases they can cause serious muscle pain, called myopathy. In the August 21 *New England Journal of Medicine*, researchers will report the discovery of a variant in the gene *SLCO1B1* that places people at risk. The variant prevents a protein from shuttling compounds, including statins, from the bloodstream into the liver for processing, says study coauthor Rory Collins of the University of Oxford in England. — *Nathan Seppa*

Life

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Bumblebees deliver disease

Hitchhiking pathogens threaten wild populations

By Susan Milius

Bumblebees sneak out from work, too, and fugitives buzzing away from their greenhouse jobs could contribute to pollinator declines.

Greenhouse growers use bumblebees for crops that need what's called "buzz pollination," a strong vibration that shakes loose the pollen. Honeybees don't give the buzz, but bumblebees do.

Some commercial bumblebees escape their greenhouses and forage among the flowers outdoors, mingling with local wild bees. Observations and a new model of how a bee disease spreads now support the idea that these fugitives transport pathogens to the bumblebee species in the wild, Michael Otterstatter and James Thomson of the University of Toronto in Canada report in the July 23 *PLoS ONE*.

"This paper makes a convincing case that pathogen spillover could be contributing to declines" of North America's bumblebees, says Rachael Winfree of Rutgers, the State University of New Jersey in New Brunswick.

Bumblebees are important pollinators, says Winfree, who has studied bees pollinating fields of watermelons. Of 50 visiting species of native bees, a single bumblebee species, *Bombus impatiens,* did more than half the work.

When *Bombus impatiens* escapes from greenhouses, it can carry diseases to other bees.

Bumblebees buzzing in the neighborhood of big commercial greenhouses were more likely to have three out of four major bee parasites than were bees far from the operations, Otterstatter found in an earlier survey. The most clear-cut case came from the gut parasite *Crithidia bombi,* which Otterstatter and Thomson modeled for the new study of pathogen spillover.

Lab tests, combined with bee literature, provided the basis for a mathematical model of how *C. bombi* spreads. As the model predicted, infection rates near Canadian greenhouses rose as wild bees and fugitives mingled during the summer.

The spread didn't build into a major epidemic wave, Otterstatter says, but probably only because the wild bumblebee season was too short. In warmer climates with longer seasons for bees, epidemic waves certainly could form, he says.

Molecule makes magnetic sense

Cryptochrome could help explain animal orientation

By Ashley Yeager

Scientists have identified a molecule that fruit flies need to sense magnetic fields. It is the first time this molecule has been linked conclusively to magnetic sensitivity in any animal, researchers say online July 20 in *Nature*.

The molecule, cryptochrome, is sensitive to blue and ultraviolet light and is found in fly photoreceptor cells.

"This is a huge finding. It really says that cryptochrome, which is linked to animals' circadian clock, is also involved in a living animal's response to magnetic fields," says study coauthor Steven M. Reppert, a neurobiologist at the University of Massachusetts Medical School in Worcester.

"Cryptochrome really is involved in animals' detection of magnetic fields."
KENNETH LOHMANN

The study shows "quite convincingly that cryptochrome really is involved in animals' detection of magnetic fields," says Kenneth Lohmann, a neurobiologist at the University of North Carolina at Chapel Hill, who was not involved in the study.

But the study's authors note that it's difficult to relate their findings directly to the use of geomagnetic fields in animals with a more complex cryptochrome system. The results may, however, help scientists understand how migratory birds, which use geomagnetic sensing for navigation and orientation, could use cryptochrome to know where they are going, Reppert says.

To test the molecule's role in fruit flies' orientation, Robert Gegear, a postdoctoral researcher in Reppert's lab, built a T-shaped maze, with coils that emit magnetic fields on either side, for the fruit flies to navigate. Both coils were turned on, but only one emitted the magnetic field. Some flies were then trained to associate the magnetic coil with a food reward. When exposed to visible and ultraviolet light and the magnetic field, the trained and untrained flies responded to the magnetized coil.

But when the researchers blocked the blue and ultraviolet wavelengths that activate the flies' cryptochrome, none of the flies flew toward the magnetic field. Mutant flies with damaged or nonexistent cryptochrome light receptors did not respond to the magnetic field either.

"Cryptochrome is therefore necessary for magnetic sensitivity in fruit flies," Reppert says. "Our study even suggests that the molecule is the actual receptor that detects the magnetism."

DAVID CAPPAERT/MICHIGAN STATE UNIV.; BUGWOOD.ORG

> “This will become a textbook example in evolutionary biology.” —KEN KARDONG

Fangs sprang from one source

Embryos disclose history of snake venom delivery

By Amy Maxmen

By studying snake embryos, including an 18-day-old African night adder removed from its egg (left), Freek Vonk (right with king cobra) and colleagues examined how fangs evolved.

“How’d you get those newfangled teeth?” hissed the petite garter snake to the venomous cobra. “Same way that you got yours,” cobra replied. All fangs — no matter their size, shape or position — descend from a single evolutionary event, new evidence from snake embryos suggests.

“I’m sky high on this piece of work,” comments Ken Kardong of Washington State University in Pullman, who has been studying snake evolution for more than 30 years. “This will become a textbook example in evolutionary biology.”

The new study, led by Freek Vonk of Leiden University in the Netherlands, reveals that snakes didn’t reinvent the wheel with each new version of their venom-delivery systems. The report appears in the July 31 *Nature*.

“They’ve shown there is a single underlying way of building things that has been elaborated different ways, in different groups,” says biologist Rick Shine of the University of Sydney in Australia.

Dagger-sharp frontal fangs allow cobras and vipers to prey on feisty mammals. Garter snakes and others that hunt less wily creatures have fangs in the back of their mouths. What confused biologists was learning that the front-fanged snakes don’t fall into a neat group. In the snake tree of life, rear-fanged snakes are scattered on evolutionary branches between the cobras and vipers, suggesting fangs evolved at least a couple of times.

Such multiple origins pose a problem for evolutionary theories that say that complex structures like fangs — multipart weapons consisting of a sharp tooth connected to a venomous gland — don’t just come and go.

The new study by Vonk is the first in decades to compare how front and back fangs develop within snake embryos. In snake “gums,” a gene called *sonic hedgehog* lays out the toothy part of the body plan. Vonk and colleagues tracked down where that gene was expressed in embryo mouths of eight snake species and discovered that the initial plans for fang formation consistently began at the back of the mouth, even in front-fanged snakes.

A critical event millions of years ago set the stage for fangs to form, the researchers suggest. Tooth-forming tissue at the back of the jaw became uncoupled from the front, so the back area was free to change while the front still grew teeth for grabbing prey. Thus, the primitive snake could survive while under construction.

“It supports the idea that there’s an evolutionarily common origin for the fang,” explains Michael K. Richardson of Leiden University.

RAKEFET DAVID-SCHWARTZ; F. VONK, M. RICHARDSON; F. VONK

Death to dodder

The parasitic plant known as dodder really sucks. The vine (shown wrapped around a green stem) pierces the tissue of other plants, extracting water and nutrients. But it also consumes molecules that scientists could manipulate to design “attack RNA” that could interfere with dodder’s growth and bring on its demise. Some RNA molecules siphoned from the host plant remain stable in dodder, traveling several centimeters within the parasite, Neelima Sinha of the University of California, Davis and colleagues report in an upcoming *New Phytologist*. “This is very exciting from the point of view of controlling parasitic plants,” says James Westwood of Virginia Tech in Blacksburg. —*Rachel Ehrenberg*

Humans

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Toddler worldview shifts at age 2

Study looks at leap in object recognition and thinking

By Bruce Bower

WASHINGTON — Something extraordinary happens between 18 and 24 months of age. Toddlers take giant strides in their ability to visually inspect, recognize and manipulate items, putting the kids on par with adults given comparable tasks.

To track this developmental leap for the first time, researchers at Indiana University in Bloomington created a special head-mounted camera that toddlers wear as they play with toys. The device allows the team to monitor what children look at while they explore objects, thus measuring the expansion of children's ability to recognize objects.

That expansion feeds into rapid advances made during the same time period in the ability to learn objects' names and to engage in pretend play, a basic form of symbolic thinking, Indiana's Linda Smith said July 24 during a meeting of the Cognitive Science Society.

"Something very important happens in visual object recognition during this six-month period that then affects many other developing cognitive systems," Smith said.

The latest findings support the view that general mental capacities, such as perceiving and exploring objects, give rise to specific skills, such as recognizing objects' names, said psychologist Jeffrey Elman of the University of California, San Diego. An opposing view holds that higher-order cognition stands apart from visual perception and other basic mental functions.

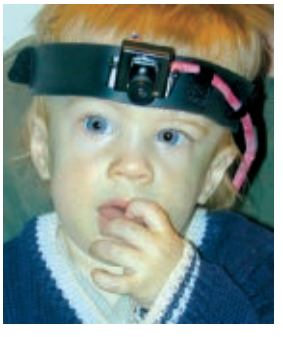
One head-cam study, led by Indiana's Alfredo Pereira, found that from 15 to 30 months of age, children begin to prefer viewing objects from the same angles that adults do. This visual orientation, known as the planar view, focuses on an object's top, bottom or side.

The researchers gave each of 30 toddlers objects to play with. Half were familiar, such as a toy airplane and a cup. The rest preserved the basic structure of familiar items but lacked identifying details.

Head-cam data indicated that, from 15 to 30 months of age, children — especially those with vocabularies of at least 100 nouns — increasingly favored planar views for both types of objects. Planar views may offer a prime look at buttons to be pushed, handles to be grabbed and other clues to objects' functions. That knowledge fuels learning of objects' names, Smith said.

A second study, led by Indiana's Sandra Street, examined the ability of 42 toddlers, 18 months and 24 months, to insert objects into appropriately shaped openings. The younger kids "failed miserably," Smith said. Older children immediately succeeded, performing at an adult level. Only the older kids looked at objects with a consistently planar view.

Intriguingly, pretend play — say, using a laundry basket as if it's a car — also emerges during that time. Recognizing objects with abstract shapes may prepare toddlers for symbolic play, Smith hypothesized.



A team at Indiana University in Bloomington used a head-mounted camera (left) to study how toddlers look at objects that are familiar and at similar objects that lacked identifying details (right).

COGNITIVE DEVELOPMENT LAB/INDIANA UNIV. BLOOMINGTON

No counting necessary

People track quantities even when distracted

By Bruce Bower

WASHINGTON — Shhh. Listen — that's the sound of people keeping track of quantities without using or thinking of number words.

English speakers can identify small numbers of items even as they perform a task that interferes with counting, according to a study presented on July 25 at a Cognitive Science Society meeting. The finding adds to evidence that language is not required for thinking about numbers of objects, said study coauthor Michael Frank of MIT. Instead, number words are abstract-thinking tools that help people manipulate and remember quantities with greater efficiency by building on basic, nonverbal knowledge, Frank proposed.

MIT's Edward Gibson, who directed the investigation, and his team already reported that the Pirahã, an Amazonian hunter-gatherer group, lack number words but could accurately place small numbers of uninflated balloons next to corresponding spools on a table (*SN: 7/19/08, p. 5*).

Participants in the new study listened to radio clips and repeated what was said as quickly as possible while matching balloons to spools. The participants performed much as the Pirahã did on the same tasks, though English speakers did better at remembering quantities greater than four, Frank said.

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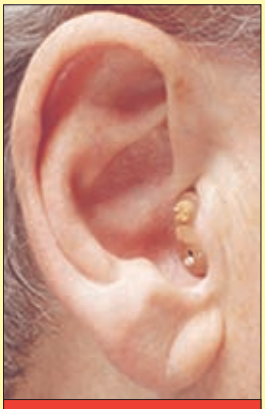

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At 8 a.m., before the day turns shirt-clinging muggy, bystanders gather in hopes of seeing some of the world's really fast runners, soon to appear on the outdoor course for training sprints. A boy a bit bigger than his backpack fidgets against the railing, but the rest of the small crowd stands quietly, cameras ready. From behind a grassy rise on the far side of the course comes the slamming of metal doors, and suddenly the runners lope into sight, their long yellow tails kinked behind them.

Travel costs being what they are these days, this report on what makes an athlete extraordinary is not brought to you from Beijing. Instead, a $1.35 fare (off-peak) on the Washington, D.C., subway leads from the *Science News* offices to the grounds of Smithsonian's National Zoo.

That's fine actually. The zoo has the better athletes by far. In this assemblage of contestants in a physiologist's fantasy Olympics, plenty of species can outrun, outdistance, out-hop and out-scurry poor old *Homo sapiens*. And researchers around the world are analyzing how these alternative gaits work and why some are so fast.

The zoo really does arrange morning training sprints for its cheetahs, but not at the top speed recorded for the species. The restriction comes in part from concerns for safety on the running path. A cord moving along a series of ankle-high guideposts pulls a lure in a snaking path through the domain of the three young cheetah brothers out today. The cord curves between clumps of tall grass and swerves around a perching log. With all these switchbacks to keep the exercise interesting, "acceleration is no problem; stopping is a problem," says zoo cheetah biologist Craig Saffoe. So for a cheetah on the longest straightaway in the course, Saffoe keeps the speed lively but still safe, no more than 20 meters per second (about 45 miles per hour) — a speed that would smash world records in Beijing.

Of course, these cheetahs will race after a little swatch of rabbit fur on a motorized string. So there's some consolation in remembering who controls the motor.

FRANS LANTING/CORBIS

peed

Animals would prove fierce competitors at the Olympics — if only they would stay in their lanes **By Susan Milius**

Cheetahs may look skinny, but they've got the muscles, cardiovascular system and stride to blur past a world-class human sprinter.

Run, Spot, run

An animal sprinting along a measured course marks a high point for testing animal abilities. "Many of the animal speeds given in encyclopedias, et cetera, are little better than guesses," laments longtime locomotion researcher McNeill Alexander of the University of Leeds in England.

Even speeds timed on a measured course have their limitations, as does Alexander's report of 7.5 m/s (almost 17 mph) for a white rhino at a briskish run, he acknowledges. The measurement derives from video that Alexander shot of the 2-ton-plus rhino being urged forward, respectfully, by a Jeep. That's almost certainly not the top speed for the animal, he cautions. "You're not going to hassle a rhino too much."

Despite Alexander's general skepticism about speed measurements, he does accept the cheetah as probably the fastest known running species. The measurement he finds most reliable, 29 m/s (about 65 mph), comes from a 1997 record along a 200-meter course clocked by an experienced timekeeper for athletic races.

Cheetahs in the wild hunt by stalking their prey and then sprinting after it in a brief blur. Saffoe says cheetahs can accelerate to 20 m/s (45 mph) in 2.5 seconds.

To see if he can inspire a little sprint this morning, Saffoe sets the cord humming around the course as soon as the cheetah brothers appear. The fur swatch flicks invitingly along the straightaway, but the brothers ignore it , trotting along a corner of the fence with a view of a female cheetah next door. After some long looks, the brothers turn to their own yard, where there are logs to be sniffed and marked, and — Hey! Small fleeing fur! One brother starts after it nose down, his stride lengthening.

The lure swerves back into the high grasses, and a different brother takes up the chase as it emerges. Saffoe has the lure burning down the straightaway now, but

the cheetah appears casual — not even trying — as his long legs close some distance.

The cheetahs look skinny, but Saffoe says that much of their sprinting muscle is found on their backs. Disproportionately large hearts and even large nasal passages feed extra oxygen to those muscles.

After several minutes of Saffoe's best feints and dodges, the brothers lose interest and flop in the shade, twitching their tails and waiting for breakfast. At best, Saffoe estimates, we saw a burst of a little more than 13 m/s (30 mph), not fabulous for a cheetah but fast for other species. At the lesser pace of 10.29 m/s, Jamaican runner Usain Bolt sprinted 100 meters in 9.72 seconds in May, challenging the human world record.

At least some humans can outrun small rodents. Alexander awards an honorable mention to the kangaroo rat, which is quick for its size. No relation to kangaroos, the little handful of fur is faster than a somewhat annoyed rhino and can hop at 8.9 m/s (almost 20 mph).

Alexander himself sounds somewhat annoyed at the mystique surrounding another supposedly prodigious hopper, the flea. "Do not be impressed by popular books that compare a flea's 30-centimeter jump to a man jumping over St. Paul's Cathedral," he says. "Theory tells us that jump height should not fall in proportion to body size as animals get smaller.... Fleas are actually rather poor jumpers."

Fakes, goes left

A stroll eastward from the cheetahs' home reveals some underappreciated terrestrial runners: flightless birds.

In a shaded outdoor pen, two rheas step delicately around their bathtub-sized pond. Tall as people, they're mostly legs and necks. Their cocoa-brown, egg-shaped bodies look startlingly wide, almost precarious, on such long legs.

Odd as a leggy flightless bird's body plan looks to a human, it works well for running. Ostriches, for example, can sprint about as fast as horses. Alexander has timed an ostrich keeping pace beside his Jeep at 17 m/s (38 mph). And the birds prove nimble, switching directions while running at speed, says Devin Jindrich of Arizona State University in Tempe.

Jindrich began studying the birds after examining cockroaches for a military-funded robot project. To analyze how the roaches coped with sudden jolts, he devised miniature jet-packs that he fastened onto the roaches' backs. And to study maneuverability he borrowed an engineering technique for detecting stresses. "I was running cockroaches over Jell-O," he says. Gelatin — he actually used unflavored brands — undergoes structural shifts when disturbed, changing the polarization of light passing through. Even the footfalls of roaches leave telltale signs for analysis.

To compare maneuverability in a different kind of runner, Jindrich visited the Royal Veterinary College's Hawkshead campus in England to experiment with eight ostriches. The birds hadn't reached full adult size but already looked the 6-foot-1 Jindrich eye-to-eye. "Fortunately they were friendly birds," he says.

This was no job for Jell-O, though. The ostriches ran along a fenced runway and crossed a platform instrumented to measure the forces of foot stomps. Jindrich placed a box just beyond the platform so ostriches had to dodge the obstacle.

The egg-shaped ostrich body is less likely to over-rotate during fast turns than humans' more columnar body shape, he and his colleagues reported last year in *The Journal of Experimental Biology*.

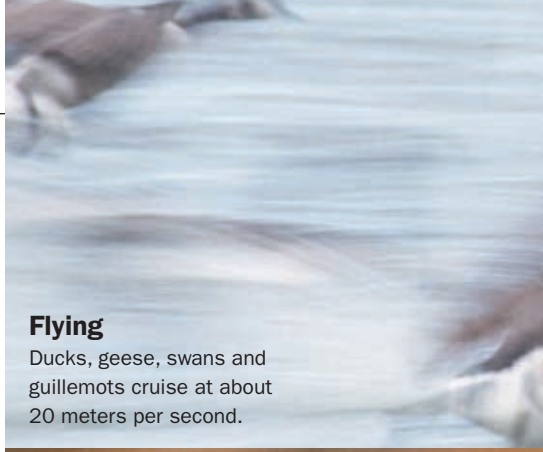
For an animal Olympics then, Jindrich muses about ostriches playing soccer. They do kick, he says. Albeit backward.

Stand up and run

Australia's dragon lizards can run bipedally too, although they use four legs to walk.

Or so says science. The three dragon lizards in the National Zoo, of the *Pogona minor* species, are using all of their legs to cling in perfect stillness to logs. The plastic-dinosaur immobility does offer a good chance to admire their skin, studies in grays and browns with delicate fringing around the strong-jawed heads.

These lizards do move, says Christofer Clemente of the University of Cambridge in England. What interests him are the explanations for why four-legged walkers rear up on occasion to run. He and his

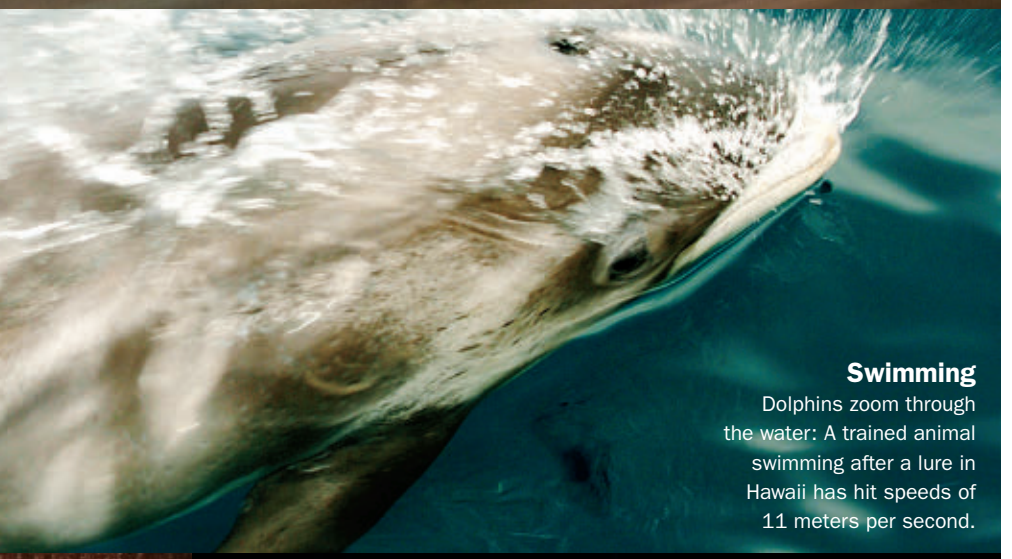


Flying
Ducks, geese, swans and guillemots cruise at about 20 meters per second.

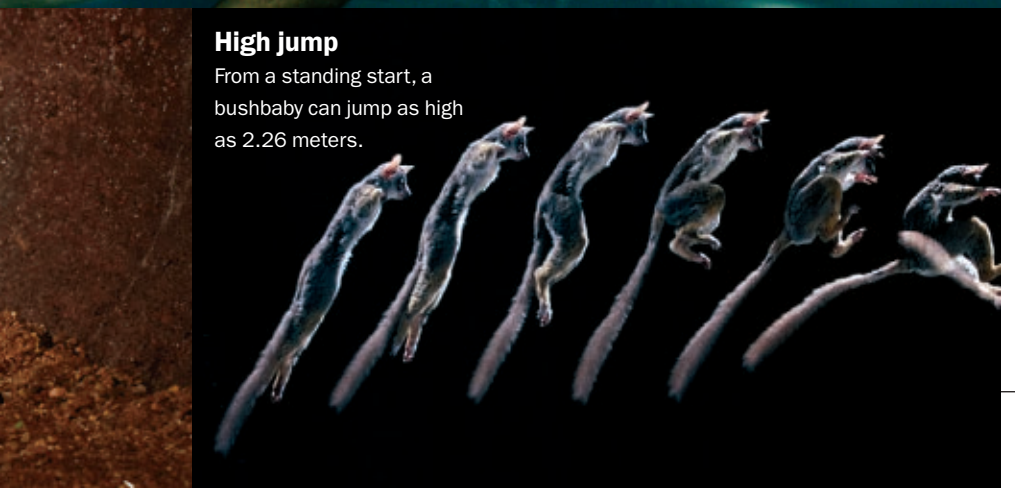
Gymnastics
Dragon lizards can accelerate so fast that they rise off all fours and run on two legs in a reptilian wheelie.

SOURCES: ALEXANDER, CLEMENTE; IMAGES, FROM TOP: PAUL A. SOUDERS/CORBIS; ART WOLFE/THE IMAGE BANK; ISTOCK/DEZOMBO; SIMON PYNT; JOHN DOWNER PRODUCTIONS

Distance running
The pronghorn antelope, according to one field observation, can run 11 kilometers in 10 minutes.



Swimming
Dolphins zoom through the water: A trained animal swimming after a lure in Hawaii has hit speeds of 11 meters per second.



High jump
From a standing start, a bushbaby can jump as high as 2.26 meters.

colleagues caught species of the lizards, including *P. minor*, in Australia and set them running on a treadmill.

Theorists had proposed that bipedal running was more efficient. Yet Clemente established that the lizards got exhausted sooner when they loped along bipedally than when moving on all fours. So he finds it unlikely that upright running saves energy for the lizards.

Instead, Clemente is exploring an idea proposed in 2003 by Peter Aerts of the University of Antwerp in Belgium that ties lizard two-legged running to acceleration. During a rapid start, the upper body of the lizard would just leave the ground in a reptilian wheelie.

The treadmill tests support this idea. Clemente measured lizard accelerations as high as 30 m/s/s, which he estimates roughly doubles a human sprinter's bursts. Speedy pickup went with lifting up to run on two legs, he and colleagues report in the July 1 *Journal of Experimental Biology*.

One scenario has lizards rearing upright as a side effect of a shift toward more maneuverable bodies. Less weight in the skull and front parts of the body would improve a lizard's ability to turn on a pebble. Yet that light front end tends to lift off the ground during rocket-start acceleration.

The side effect notion fit for one kind of lizard, which went bipedal when the acceleration model predicted it would. Other species, though, went bipedal at lower accelerations than physics would necessitate. These lizards seemed to be shifting their center of gravity behaviorally, by tucking in their arms or repositioning their tail.

However fascinating his lizards are, Clemente acknowledges that they aren't that fast. They manage only 5 to 6 m/s (12 mph or so). So he proposes an Olympics rule-change: measuring races in body lengths. A human athlete covers some six lengths per second. Dragon lizards would beat that — covering 30.

Splash speeds

No funny business with the rules is necessary for fantasy fish Olympics.

"I have told my zoology class that we should put a Speedo logo on the side of an albacore in the Olympics and have it race,"

Flightless, but fleet of foot In studies of maneuverability and body shape, running ostriches dodge a big box by sidestepping (in two images at left) or crossing over a leg (images at right). Egg-shaped birds turn easily, requiring less braking while switching directions than humans require.

says Frank Fish of West Chester University in Pennsylvania. His research focuses on dolphins and other aquatic animals, but he says he watches a lot of human competitive swimming. "I'm amazed at how pathetic human swimmers are compared to other animals," he says. In March, Alain Bernard set a world record by swimming 50 meters at an average speed of 2.3 m/s (5.1 mph). Big tuna, Fish points out, can do 20 m/s (about 45 mph).

Albacore and other tuna get the shape right, close to the teardrop that minimizes drag. Tuna, which cover huge distances on their migrations, pack a lot of high-powered aerobic muscle into that teardrop.

Tuna represent an "extreme design" for a swimming animal, says Robert Shadwick of the University of British Columbia in Vancouver, Canada. They and some other fast predators like lamnid sharks have converged in highly streamlined bodies with muscles tuned for aerobic performance.

In yellowfin tuna, the rippling muscles don't bend the body so much as transmit power to the tail, Shadwick and Douglas Syme of the University of Calgary in Canada report in the May 15 *Journal of Experimental Biology*. Muscles and a system of tendons create strong, well-timed strokes of the tail fin for near-maximal power.

Some hunting whales can sprint too. "Cheetahs of the deep sea" is what Natacha Aguilar Soto of La Laguna University in Tenerife, Spain, calls short-finned pilot whales in a paper published online April 28 in the *Journal of Animal Ecology*.

Electronic tags on 23 whales tracked their diving behavior. In the deepest plunges, down as far as 1,019 meters, whales reached speeds of 9 m/s (20 mph), as if racing after prey. Like cheetahs, the pilot whales could be putting their energy into sprints after big targets.

Jetting around

At the zoo's invertebrate display, zoogoers can see swimmers taking a different approach. In two big, dimly lit tanks, chambered nautiluses the size of saucers bob gently against the glass. When they move, they squirt out gusts that drive them in the opposite direction by jet propulsion.

Nautiluses are slow, but jet propulsion can be quite fast, says Hans-Otto Pörtner of the Alfred Wegener Institute for Polar and Marine Research in Bremerhaven, Germany. The champions are the temperate-latitude, upper-ocean squid. The shortfin squid (*Illex illecebrosus*) and relatives have the sprint power to hunt with the fish.

Pörtner calculates that jetting around so much takes five to 10 times the amount of oxygen that fishy swimming does. Also, "they're not just the jet set; they're blue bloods," he says. When oxygenated, their blood turns blue with the reactions of hemocyanin molecules. These bulky oxygen-carrying molecules have only a third to a fifth of the capacity of the hemoglobin that does the same job in vertebrate blood.

Offsetting these disadvantages, thin skin lets squid take in some 60 to 80 percent of their oxygen directly from surrounding water, Pörtner and colleagues have found. And the muscles that most need the oxygen lie just under the skin.

Pörtner calls the shortfin squid "marine invertebrate athletes," but he's not daydreaming of squid Olympics. Any food will distract them, he says. "I don't think you could keep them in the lanes."

Panda acrobats

The most famous residents of the National Zoo aren't swimmers, but they are visiting from the 2008 Olympic host country. Near the top of the zoo's main hill sits a complex of boulder-filled yards inhabited by three pandas.

For all their teddy bear looks, pandas do have strength and agility. Visitors rarely see it, but male pandas naturally back their rumps up to trees and then walk their legs up the trunk. Their short forelimbs are muscular enough for a male to back his rear legs so high he is doing a handstand, albeit with feet propped against a tree.

It's an extreme version of a dog at a fire hydrant. A handstanding panda leaves a urine trace on the bark as part of his species' community bulletin board system.

A hot, summer day in Washington doesn't lend itself to handstands. Even the youngest of the pandas, 3-year-old Tai Shan, has moved to his indoor, air-conditioned apartment. He sprawls in a panda-sized dip on one of the indoor rocks, one paw hooked under his chin. So, in homage to the official Olympics and their host country, be it noted that a panda can flop in the shade and lounge as fast as any cheetah. ■

Explore more

■ R. McNeill Alexander. *Principles of Animal Locomotion*. Princeton University Press, 2003.

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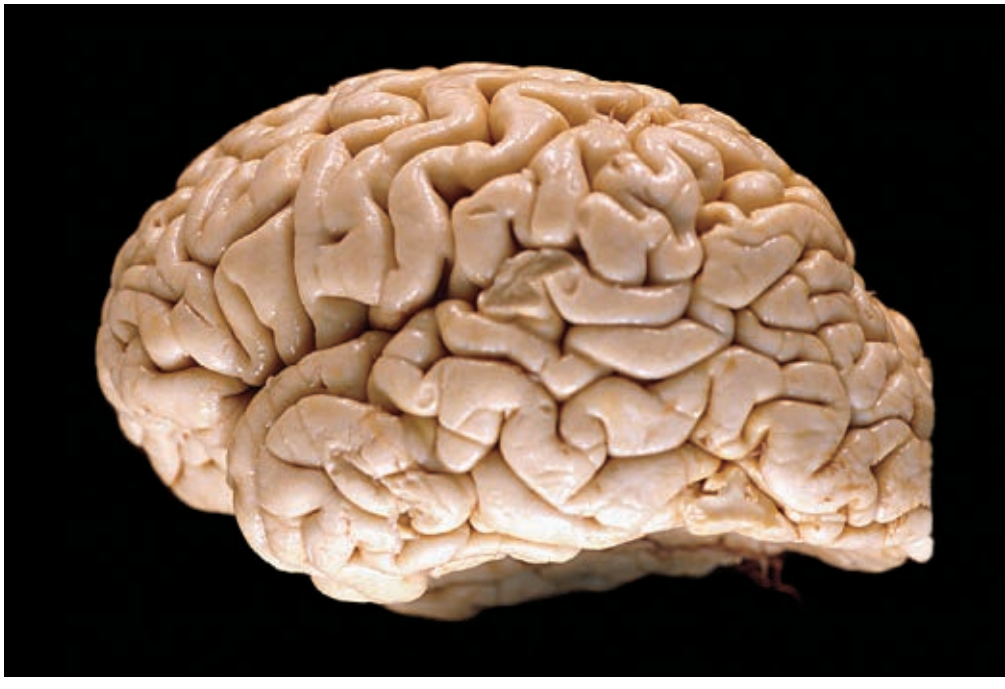
Neuron Ki



As open-and-shut cases go, Alzheimer's disease should top the list. The victim is clear. Suspects are in custody. Wherever neurons die due to Alzheimer's disease, a protein known as amyloid-beta is always found at the scene of the crime, hanging around in large, tough gangs called plaques. Parkinson's and Huntington's diseases; amyotrophic lateral sclerosis (which goes by its initials ALS or the alias Lou Gehrig's disease); and prion diseases, such as scrapie in sheep, mad cow disease in cattle and Creutzfeldt-Jakob disease in humans, all have similar stories.

Scientific investigators have pieced together this much: A seemingly mild-mannered brain protein falls in with a bad crowd, the corrupted protein and its cronies gang up and mob violence results in the death of a brain cell. It's a scene repeated over and over again in different neighborhoods of the brain, by different proteins, but all with the same result — the death of neurons and rise of disease.

But no one has convicted these suspected neuron killers. So far, cases mostly rely on circumstantial evidence, with large holes in the web of proof. There's no smoking gun, no motive and no eyewitness to corroborate what scientists suspect. And there's no cure for the diseases that slowly break down brains and spinal cords, robbing victims of memories or mobility.

No one has observed all the steps of a neuron's demise, so no one is sure exactly what the murder weapon is or who dealt the final blow. But scientists acting like shamuses on the scent of a killer have picked up tantalizing clues about how neurons meet their end, and protein aggregation is almost certainly involved.

"It seems unlikely that coincidence is at work here," says Bradley Hyman, a neurologist at Harvard Medical School and Massachusetts General Hospital-East in Charlestown, Mass. Recent research from Hyman and colleagues shows that plaques develop more rapidly in the brains of mice prone to Alzheimer's disease than had been thought. The discovery, published February 7 in *Nature*, suggests that there may be many years between the appearance of plaques and the onset of disease, providing a window of time for doctors to take action and stop the death of neurons.

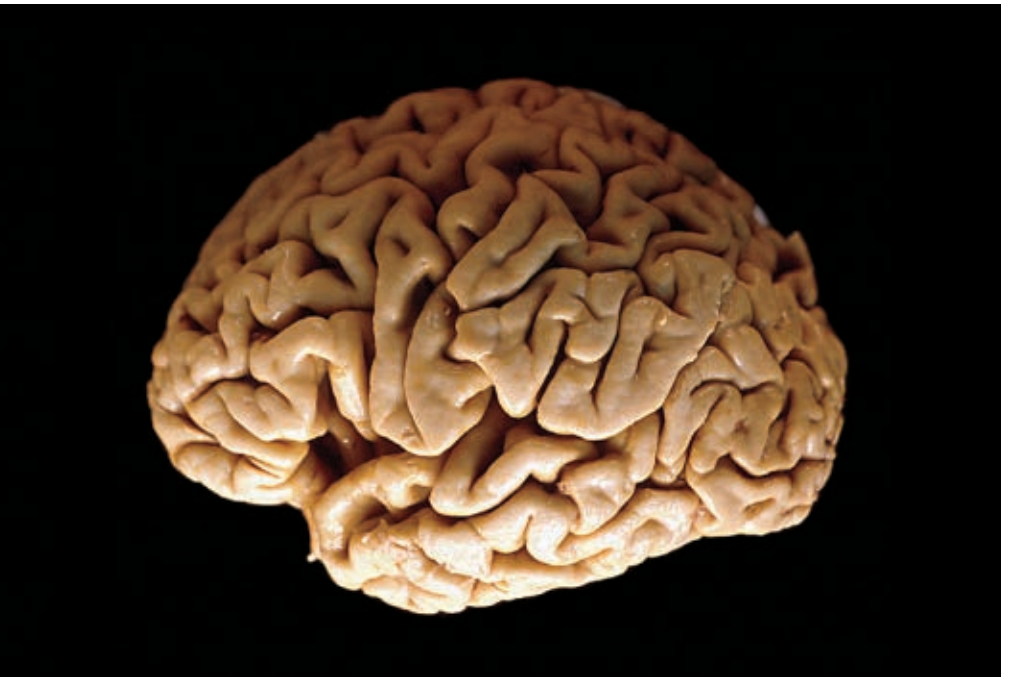
Other researchers have recently reported progress on developing molecules that may help protect the brain against proteins-gone-bad. And other new research shows that the perpetrator in some cases of neurodegenerative disease may not be one of the usual suspects.

The key to stopping the killing of neurons is figuring out what causes otherwise innocuous proteins to show their Mr. Hyde side, and discovering why the proteins flock together once they've turned. The method by which "bad" proteins bump off neurons is also a matter of dispute. Scientists are drawing ever closer to solutions for these mysteries, and what they discover may one day help head off these diseases or even repair some damage after rogue proteins have vandalized the brain or spinal cord.

lers

Misfolded, clumping proteins evade conviction, but they remain prime suspects in neurodegenerative diseases

By Tina Hesman Saey



In diseases like Alzheimer's, the persistent destruction of neurons leads to an obvious toll by the end of life, visible in this comparison of postmortem brains. The brain on the left was collected from a person without dementia; the right image shows the smaller, atrophied brain of an Alzheimer's patient.

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Cause or effect

Not everyone believes that protein aggregation is such a bad thing for neurons. Take those big plaques of amyloid-beta, or A-beta, found near dead and dying brain cells in Alzheimer's disease patients.

"Some people say it's a tombstone, others say it's not the cause," says Gang Yu, a neuroscientist and biochemist at the University of Texas Southwestern Medical Center at Dallas.

Big clusters of protein may be a cell's way of coping with otherwise harmful proteins, suggests Lila Gierasch, a biophysical chemist at the University of Massachusetts Amherst. Plaques are "like garbage dumps for insoluble proteins," she says. Indeed A-beta plaques contain remnants of other proteins, perhaps dumped in the plaque to avoid cluttering up a cell and gumming up its inner workings.

Brain images of healthy people reveal that A-beta plaques are common, even in people who don't have dementia. And mice that make a lot of A-beta have memory problems, but their neurons don't die, says Li-Huei Tsai, a neuroscientist at MIT. "The role of A-beta is still very, very controversial," she says. Some people think elevated levels of the protein may interfere with neuron communication. Others think that small aggregates, rather than large clumps, are toxic to cells.

Part of the difficulty in deciphering A-beta's role in Alzheimer's disease is that no one is sure what the protein's day job is. That's true of alpha-synuclein, a protein that forms clumps called Lewy bodies inside brain cells of people with Parkinson's disease, and of huntingtin, a protein which has been shown to be the causative agent of Huntington's disease. Alpha-synuclein, A-beta and the prion protein PrP probably aren't unemployed, but scientists have not yet established their roles.

On the surface, these proteins, as well as two proteins (TDP-43 and superoxide dismutase or SOD1) involved in ALS, have nothing in common, says Mark Goldberg, director of the Hope Center for Neurological Disorders at Washington University in St. Louis. The sequences of amino acids that compose the proteins aren't the same, nor are the normal shapes of the proteins. The neuron-killing proteins probably function differently too. But all of them go bad in a similar way, twisting from loose, flexible molecules into rigid, sticky formations known as beta-pleated sheets.

Every protein in the body probably has the ability to form beta-pleated sheets given the right (or wrong) circumstances, says Erich Wanker, a molecular biologist and biochemist at the Max Delbrück Center for Molecular Medicine in Berlin. Something about these proteins and others that cause amyloidosis — fatal diseases in which abnormally folded proteins build

up in organs — makes the proteins more prone to assuming the deadly conformation. Genetic mutations can tip the balance, but that doesn't explain why people who don't have mutations sometimes end up with the aggregates.

On the straight and narrow

Although the precipitating event that leads good proteins down the beta-pleated path isn't known, Wanker and his colleagues may have developed a way to stop the process, at least in the test tube. In a report published in the June *Nature Structural & Molecular Biology*, Wanker and his collaborators showed that a small molecule called (—)-epigallocatechin gallate (mercifully shortened to EGCG) can keep A-beta and alpha-synuclein from forming beta sheets. The group had previously shown that the compound could prevent huntingtin from aggregating.

EGCG latches on to the backbones of the amino acid chains that compose the proteins. With EGCG riding piggyback, the proteins form small clumps. But apparently the proteins never switch to the beta-sheet formation, so the little clumps aren't toxic to cells in the test tube.

Wanker doesn't know whether EGCG, found in green tea, would be an effective therapy for neurodegenerative diseases. The researchers have yet to demonstrate that the compound can dissolve existing aggregates. Also, the experiments used equal parts of the molecule to protein in order to stop the proteins from forming the toxic beta sheets, which may mean that therapies would require massive amounts of the compound to work effectively. It's also not known how well EGCG gets across the blood-brain barrier. If the molecule doesn't enter the brain easily, doses of EGCG needed to prevent disease might be too high to be practical.

Cells may already possess molecules that work in the same way EGCG does, Wanker says. Proteins called chaperones also help keep other proteins loose and ready for action. Some evidence suggests that defects in chaperones may be the blow that sets off brain-wasting diseases. "This mechanism may be more common than we think," Wanker says.

Other proteins may act as guardian angels to keep would-be neuron killers on the straight and narrow too. One such guardian may be a protein known as Pin1, which could keep another potential killer that stalks the brains of Alzheimer's disease patients from turning deadly.

While spotlights have been trained on A-beta as the most likely killer of neurons in Alzheimer's disease, Kun Ping Lu of Beth Israel Deaconess Medical Center in Boston thinks scientists may be ignoring a more deadly culprit, a protein called tau.

Tau is normally a hard-working protein that helps create the internal skeleton of the cell by binding to the cell's frame-supporting microtubules. If not for tau, the long fibers called axons that connect neurons across the brain would break down, severing communication as surely as cutting a fiber-optic cable to a building would. Dendrites, the neuron's branchlike projections that receive signals from other neurons, would also disintegrate without tau pinning microtubules in place.

People who have mutations in the gene that encodes tau develop a disease called frontotemporal dementia. The brains of people with this dementia look much like brains of people with Alzheimer's disease with one critical difference: Frontotemporal dementia patients don't have plaques in their brains. But they do have tangles of tau in brain cells, and their neurons are as dead as a person with Alzheimer's disease.

That leads Lu to believe that tau may be more directly involved in killing neurons than A-beta. In other words, A-beta may order the hit, but tau pulls the trigger. "If, on top of tangles, you add plaques or increase A-beta, now you have massive neurodegeneration," Lu says.

Lu lays out the scenario for brain-cell murder this way: A-beta builds up outside neurons, leading to inflammation in the brain. Inflammation prods enzymes called kinases to tack extra phosphates on to tau inside the cells. This causes tau to walk off the job and hang out in hard tangles with other tau molecules that have more phosphates hanging off them than groupies on a rock star. Hyperphosphorylated tau forms such tight bonds with its cronies, not even boiling it in detergent can untangle it, Lu says. After that, it's all over for the neuron as its axons and dendrites collapse.

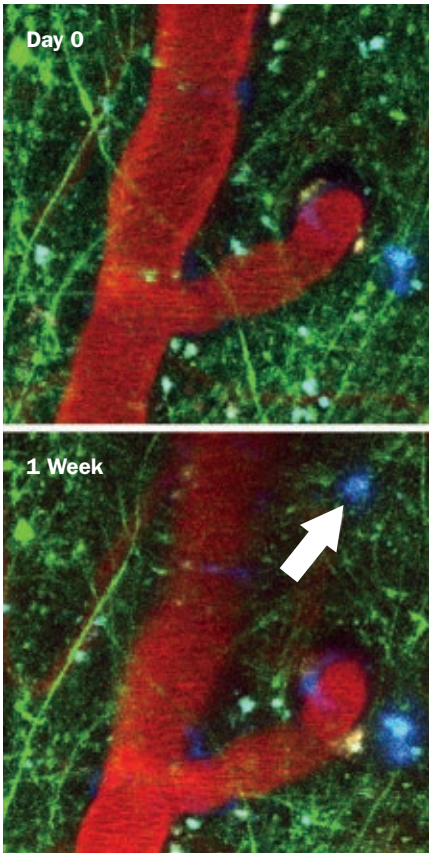
Normally, tau's protector, Pin1, keeps it from falling in with hardened tangles. Pin1 actually does double duty, watching over tau and APP, the protein precursor to A-beta. Mutations in the gene for Pin1 have now been linked to late-onset Alzheimer's disease, but not to early onset forms.

Lu and his colleagues have found a variation in the Pin1 promoter, a stretch of DNA that controls activity of the gene, associated with a five-year later onset of Alzheimer's disease. The researchers don't yet know if the variation increases Pin1 production. They do know that aging causes Pin1 production to fall.

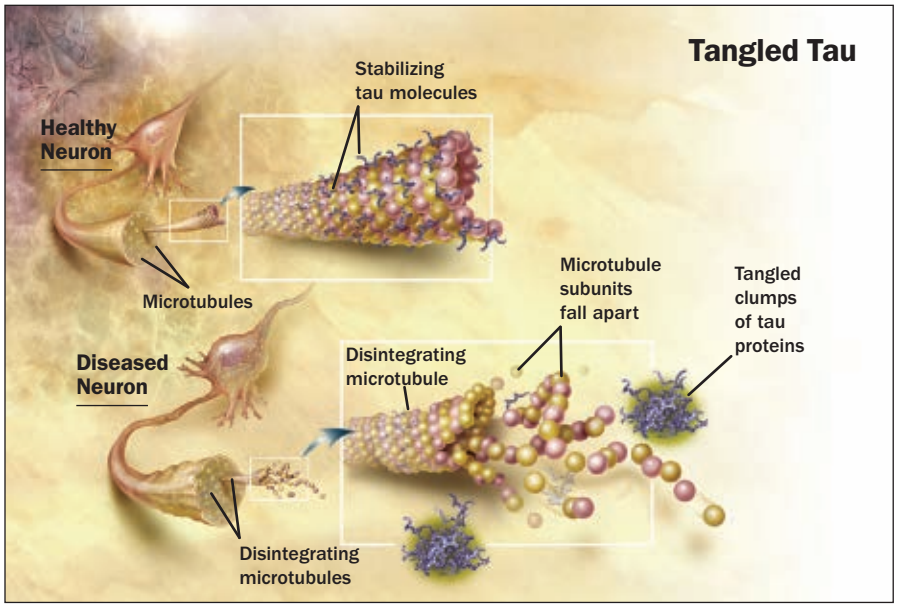
"As people get older and older, Pin1 levels drop, drop, drop," Lu says.

Boosting Pin1 levels may help untangle tau in people at risk of Alzheimer's disease, slowing the disease's progression or pre-

Contrary to previous thinking, amyloid-beta plaques can form in less than a week. At the start of an experiment by Bradley Hyman's group, a blood vessel (red) snakes through neurons (green). Just one week later, an amyloid-beta plaque (blue, marked by arrow) appears amid the neurons.



HYMAN *ET AL.*



Microtubules support neurons and act like tracks, guiding nutrients and molecules from the cell body down to the axon and back. The protein tau makes the microtubules stable. But in Alzheimer's disease, phosphate groups (not shown) are added to tau, allowing it to pair up and tangle. Then the microtubules disintegrate, collapsing the neuron's transport system, sometimes leading to cell death.

venting it altogether. Reporting in the May *Journal of Clinical Investigation*, Lu and colleagues showed that making more of the protein could help protect against tangle formation in mice. But the new research also shows that too much Pin1 can be a bad thing. When researchers increased Pin1 levels in mice carrying the P301L alteration in tau—found in people with frontotemporal dementia—more brain cells died than did in mice that carry the tau mutation but make normal levels of Pin1.

The poisoning blame game

Tau is not the only protein that may be getting away with neuron murder while a more high-profile suspect takes the rap. The antioxidant protein superoxide dismutase had been fingered as the killer of spinal cord neurons in people with ALS. A small subset of those with the disease have mutations in the gene for SOD1 that lead to clumping of the protein and the death of neurons that direct motion.

But recently scientists learned that nearly everyone with ALS has aggregates of a protein called TDP-43 (for TAR DNA binding protein) in their spinal neurons.

"If TDP-43 is the major pathway, then SOD1 was misdirecting us," says Christopher Shaw, a neurologist and neurogeneticist at King's College London. He estimates that about 1 percent of people with ALS have mutations in the gene for TDP-43. Shaw and his colleagues showed in a report published March 21 in *Science* that those mutations lead the protein to stick together more readily. Most cases are sporadic, not inherited, and occur when TDP-43 twists into a shape that favors aggregation. Scientists don't yet know what sets off the conversion, but Shaw says the tail of the molecule certainly plays a role.

The tail end of TDP-43, what scientists refer to as the c-terminus, "aggregates fantastically quickly," he says. "It's an extremely sticky little beast."

That stickiness is characteristic of all proteins that form neuron-killing beta sheets and may account for the speed at which plaques and other aggregates form. Although scientists have evidence that proteins become toxic after twisting into beta sheets and aggregating, just how clumps of protein poison neurons isn't clear.

For instance, even though SOD1 protein is made everywhere in the body, and mutations that lead to overproduction cause aggregation of the protein in many tissues, only spinal cord neurons degenerate to give rise to ALS. Similarly, neurons that produce dopamine, a chemical key to neural communication, are the victims of alpha-synuclein clumps in people with Parkinson's disease. And Alzheimer's plaques tend to congregate in parts of the brain that are active when people are daydreaming or thinking about nothing in particular (*SN Online: "Journey to the center of the brain," 6/30/08*).

The life cycle of a neuron might explain its susceptibility to damage, Shaw says. Most neurons last a lifetime. The cells don't divide after they are born and take their place in the brain. Some new neurons do develop in parts of the brain, but most of the 10 billion to 100 billion neurons are present before birth and last until death. The cells never get a day off and they have no backup or replacement.

Their long lives may lead neurons to produce proteins differently than other cells. "Maybe brain cells have a just-in-time policy," Shaw says. "You don't make a lot of protein and stack it up, so therefore you don't have the same rigorous protein turnover mechanisms." In other cells in the body, quality control would quickly recognize a misfolded protein and get rid of it before it could cause mischief. The lack of supervision in neurons could make them more vulnerable to rogue proteins.

On the other hand, neurons may process proteins correctly, but age may catch up with the neurons, making them weary of the constant effort against aggregation.

"There's an ongoing battle for many years, and ultimately the neuron gives up," speculates Yu from UT Southwestern. But scientists don't know what causes neurons to throw in the towel. The final straw could be the loss of chaperone proteins, which oversee protein-folding, or a strike by the cellular machinery that transports or breaks down proteins, causing crowding in the cell that foments aggregation.

"Theories abound," Yu says, "but none have been definitively proven." ■

Explore more

■ D.E. Ehrnhoefer *et al*. "EGCG redirects amyloidogenic polypeptides into unstructured, off-pathway oligomers." *Nature Structural & Molecular Biology*. June 2008.

Welcome to the quantum Internet

Quantum encryption is here, but the laws of physics ca

A stylish new way of surfing the Internet is coming to Vienna this fall. Researchers plan to flip the switch on the next step toward a quantum version of the Internet. They will build a network allowing users to send each other messages as virtually unbreakable ciphers, with privacy protected by the laws of quantum physics.

The Vienna net is admittedly just a prototype for research purposes. It is also not yet a true quantum version of the Internet. Although it can transmit ordinary data with quantum security, it can't transfer quantum information, which encodes the states of objects that obey quantum rules. Such a breakthrough might be years off, but it's getting closer.

Truth be told, it's not completely clear what a fully quantum Internet would be good for. In fact, at first it even sounds like a really bad idea. Quantum information is notoriously wobbly. An object tends to live in a superposition of states — for example, an electron can spin in two directions at once, or an atom can be simultaneously in two different places — until interaction with the rest of the world forces the object to pick one state. Quantum reality is a limbo of coexisting possibilities.

And because any measurement done of a quantum system changes the system's state irreversibly, quantum information is different every time it's read. That makes it impossible, for example, to copy, broadcast or back up quantum data.

But the eccentric physics could also impart unique strengths to networks. While each data bit in an ordinary com-

Welcome to the quantum Internet

n do much more than protect privacy

By Davide Castelvecchi

puter takes the value 0 or 1, the units of quantum information, called quantum bits, or qubits for short, can take both values simultaneously. A quantum Internet could transfer software and data between future (and futuristic) quantum computers, which could outperform ordinary computers by running multiple operations at once, in superposition. And the network could lead to new kinds of social interactions — such as letting quantum physics pick a presidential candidate who pleases the most voters or allowing people to donate to a cause based on whether others donate as well — and do so with absolute secrecy.

Perhaps — and this inches toward *Star Trek* territory — some day a quantum net could even "beam up" a physical object. All the information needed to re-create the object, such as its shape and energy, would be transferred elsewhere, leaving just chaos behind.

In the meantime, when the switch is flipped October 8, the Vienna net will demonstrate how quantum physics can keep ordinary information, such as an e-mail or the balance of a checking account, safe from prying eyes.

This latest step toward the quantum Internet is a limited network backbone that will often run at the speed of a 1980s modem. To plug into it, a user would need to buy expensive gear and link an optical fiber to one of the backbone's five nodes. But it's a step.

Meanwhile, most of the basic technical ingredients of a truly quantum Internet have now been demonstrated, at

least in the lab. In particular, researchers have created various types of "quantum memory," in which light pulses traveling through an optical fiber essentially slow to a halt, a crucial requirement for the quantum version of an Internet router. So it may be just a matter of time before scientists can start beaming up stuff — or at least data.

"I'm optimistic that within a few years we'll be able to build at least a lab demonstration of a quantum network," says Mikhail Lukin of Harvard University.

A solid quantum key

In tunnels stretching under Vienna and the Danube river, pulses of light will be beamed this October along tens of kilometers of existing optical fibers owned by German engineering conglomerate Siemens. A collaboration of more than 40 universities, companies and research institutions will piece together technologies to link five Siemens buildings, four of them scattered across the city and one 85 kilometers away in the town of St. Pölten.

The building-to-building connections will use a number of quantum encryption systems to pass the information, many of them inspired by a version of quantum encryption first proposed in 1991 by Artur Ekert, now at the National University of Singapore. With Ekert's procedure, the sender and the receiver, conventionally called Alice and Bob, use both a quantum connection and a classical one, which could be the good-old Internet or a phone line.

Through the quantum connection, Alice and Bob establish a common encryption key — a secret sequence of data bits that Alice will use to scramble her message, and Bob to unscramble it. Alice can then send her scrambled message to Bob through the classical connection, for example as an e-mail attachment.

To someone who doesn't know the key, Alice's message would look like a random sequence of bits. Even the most sophisticated computer imaginable wouldn't be able to crack it. But Bob knows the key, so he can unscramble the message.

Keeping the key secret as they create it is the crucial part, and here's where Ekert exploits quantum physics — specifically, a weird phenomenon called quantum entanglement. In quantum physics, each of two objects can exist in its own state, or the objects' states can be entangled, meaning that, while separate, they are not independent of each other.

One power of entanglement is that it makes quantum teleportation possible. That's a nearly magical way of transferring the quantum state of an object onto another object, possibly far away.

Take photons, the elementary particles that form electromagnetic radiation, including light. Photons wiggle sideways as they zip along an optical fiber. Two photons can wiggle in independent directions, called linear polarizations. But two photons can also be entangled, so that, for example, when one photon is polarized vertically, the other must be polarized horizontally, and vice versa.

In Ekert-style encryption, a laser device creates pairs of entangled photons and sends (along the fiber-optic cable) one photon from each entangled pair to Alice and the other one to Bob.

Because photons in each pair have correlated polarizations, Alice and Bob could now turn that information into a common key, which for example could contain a 0 for each vertically polarized photon and a 1 for each horizontally polarized one.

However, Alice and Bob also want to be sure the photons they are using haven't been intercepted by an eavesdropper, inevitably referred to as Eve. Any Eve who intercepts the photons, trying to steal the key, will change the photons' states, or even destroy them, since it's impossible to measure the state of a quantum system without changing it irreversibly. Alice and Bob, over the phone, will then compare notes on their test photons. If they notice discrepancies, they'll know Eve was there, so they'll throw away the key and start again.

Quantum encryption systems are now available commercially. Some are owned by banking institutions, for example, and one was used last fall in Switzerland to transmit electoral data from an electronic polling station. So far, though, these links have mostly been point-to-point rather than networks with multiple users.

With a network of quantum-encrypted lines such as the one being built in Vienna, users will just need to link to the node closest to them. When one user wants to send a secret message to another, the message will travel in encrypted form from the first user to an entry node. There, the message will be decrypted and then encrypted again (using a new key) to be sent to the next node. The same will happen at every node in between, until the message reaches its destination.

Privacy will be guaranteed, as long as the locations of the sender, the receiver and the intermediate nodes stay protected from intrusion. (By routing messages through multiple nodes simultaneously and using some mathematical tricks, the network will actually guarantee privacy even if one of the nodes is broken into.)

This piecemeal encryption — a solution also adopted on a smaller scale in a Boston-area quantum network laid out in 2003 — is needed because of a fundamental limitation with transmitting photons.

Quantum RAM

Sharing an encryption key between any two users requires sending single photons — entangled photons in the case of Ekert's scheme. But something as small as a photon easily gets lost or absorbed even in the highest-quality optical fiber, says Norbert Lütkenhaus of the University of Waterloo in Canada, a physicist who helped design Vienna's quantum net. "You lose one-half of the photons every 15 kilometers," he says.

Establishing a key thus becomes exponentially slower as the distance increases. Lütkenhaus calculates that 25 kilometers is still a good distance for decently efficient quantum communication, but beyond that distance a different solution is needed.

In the case of ordinary optical communications, the problem of photon loss is easily solved by adding "repeaters" along

the line — gadgets that receive weakened laser pulses and replace them with stronger ones. But ordinary repeaters don't work for quantum systems such as single photons. For one thing, as Lukin points out, "If you sent a single photon, if it's lost, there's nothing left to amplify." And if the photon does arrive at the node, the laws of quantum physics forbid fully copying its quantum state, so some of the photon's information will inevitably be lost. In particular, if the photon was entangled with another photon somewhere else, the entanglement will be lost.

However, in 2001 Lukin and his collaborators envisioned a way to get around this problem by creating entangled pairs from photons that are far apart. If realized, their scheme would enable long-distance, quantum-encrypted communication.

If photons can be entangled over long distances, they could enable people to interact in ways that just aren't possible within the realm of classical physics.

One power of entanglement is that it makes quantum teleportation possible. That's a nearly magical way of transferring the quantum state of an object onto another object, possibly far away. Say Alice has a photon X, which she wants to teleport to Bob. Alice also has a photon Y, which is entangled with a photon Z owned by Bob. Alice then makes her two photons interact. That way, the state of X becomes entangled with the state of Y, and thus with the state of Z.

Making the perfect encryption key

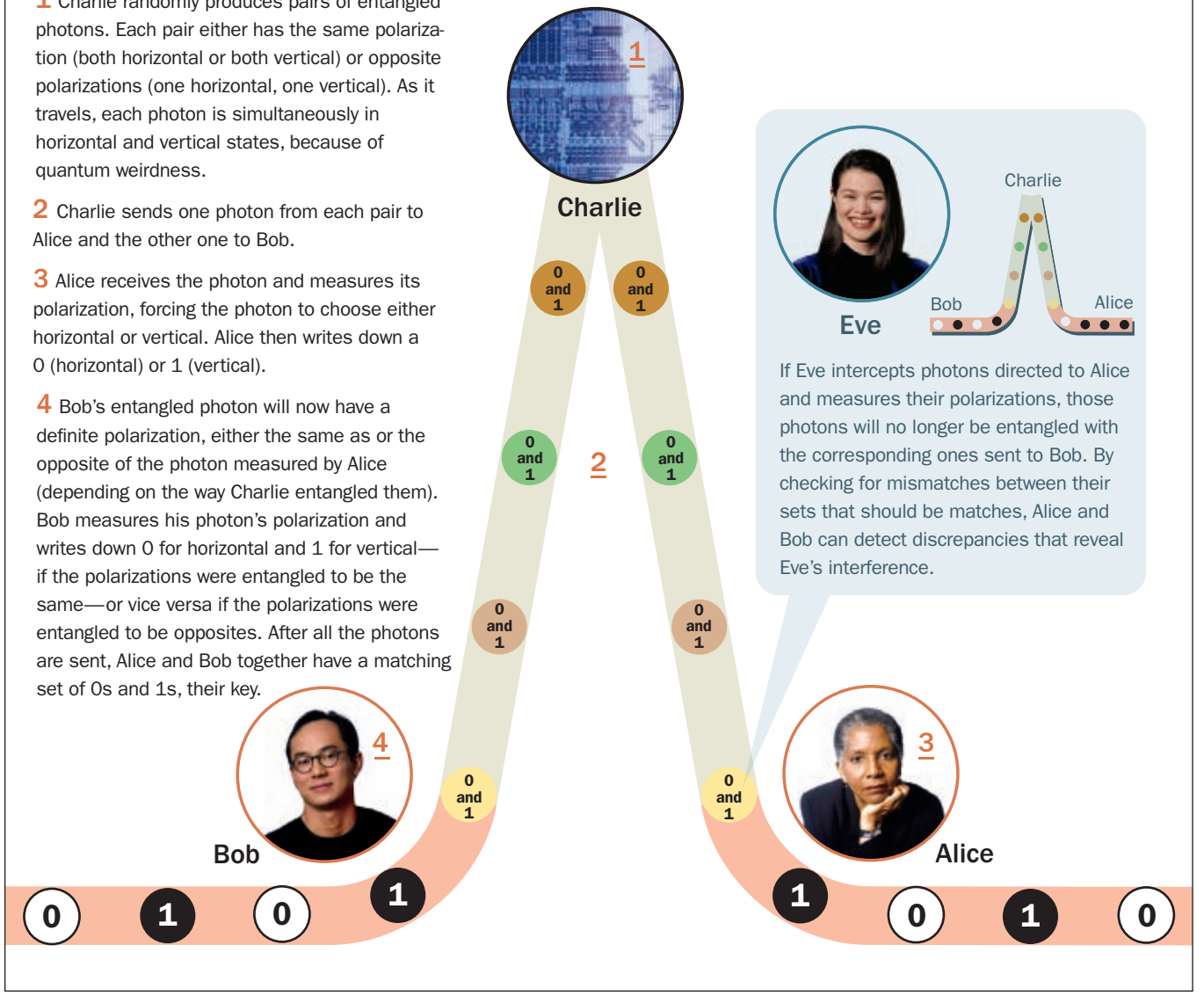
Entangled photons can enable Alice and Bob to create a secure common encryption key, a shared set of numbers that Alice can use to send an encrypted message to Bob and no one but Bob can decrypt. If Eve tries to steal the key by intercepting the entangled photons, Alice and Bob will know, thanks to quantum weirdness.

1 Charlie randomly produces pairs of entangled photons. Each pair either has the same polarization (both horizontal or both vertical) or opposite polarizations (one horizontal, one vertical). As it travels, each photon is simultaneously in horizontal and vertical states, because of quantum weirdness.

2 Charlie sends one photon from each pair to Alice and the other one to Bob.

3 Alice receives the photon and measures its polarization, forcing the photon to choose either horizontal or vertical. Alice then writes down a 0 (horizontal) or 1 (vertical).

4 Bob's entangled photon will now have a definite polarization, either the same as or the opposite of the photon measured by Alice (depending on the way Charlie entangled them). Bob measures his photon's polarization and writes down 0 for horizontal and 1 for vertical—if the polarizations were entangled to be the same—or vice versa if the polarizations were entangled to be opposites. After all the photons are sent, Alice and Bob together have a matching set of 0s and 1s, their key.



DESIGN: J. KORENBLAT; PHOTODISC/GETTY

Alice then destroys X and Y by measuring their states, and she calls up Bob to tell him the results. Using that information, Bob can now twist the state of Z to make it identical to the original state of X. Alice has sacrificed the two photons in her possession, but as a result, Bob now has an exact copy of the original photon, photon X.

Lukin's idea to create long-distance entanglement relies on yet another trick called entanglement swapping. In entanglement swapping, each of two sources produces a pair of entangled photons. The photons from the first source, say A and B, are not entangled with those from the second source, say C and D. Next, B and C are brought to the same detector. There, B and C interact and are destroyed, causing A and D to become entangled even though they were never close to each other.

Repeated applications of entanglement swapping over a chain of nodes can create pairs of entangled photons that are farther and farther from each other. Eventually, all photons are destroyed, except for the ones at the opposite ends of the chain. Those two end up entangled.

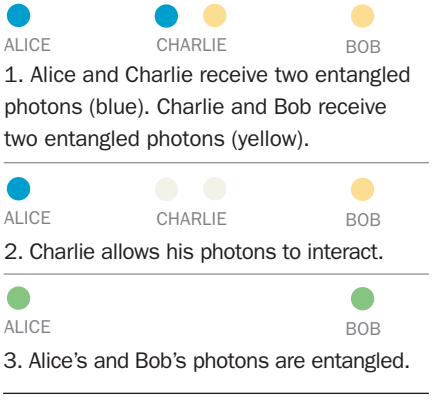
The method seems fail-safe on paper, but in practice, at each step at least some of the photons have a high chance of getting lost. But if one could somehow store pairs of photons that have successfully been entangled while other pairs are still being generated, long-distance entanglement would become possible at a reasonable speed. The key to quantum networking, then, is the ability to keep entangled photons in a sort of quantum RAM.

Catch and release

In 2001, Lukin and his collaborators, and an independent Harvard group led by Lene Hau, created the first rudimentary quantum memory, essentially by slowing light to a crawl inside clouds of atoms (*SN: 1/27/01, p. 52*). Since then, several groups have performed ever-more-advanced quantum-memory tricks. For example, groups led by Lukin, Alex Kuzmich of the Georgia Institute of Technology in Atlanta and Jeff Kimble of the California Institute of Technology in Pasadena were able to take a photon emitted by one atom cloud and store it in another atom cloud. And last September, Christopher Monroe and his team at the University of Maryland in College Park were able to entangle two qubits made of single ions.

Long-distance relationship

One crucial tool for the quantum Internet could be entanglement swapping.



Most recently, in the March 6 *Nature*, a team led by Kimble described what may be the most advanced kind of quantum memory to date. The researchers captured two entangled photon states in atom clouds and were able to release the states on demand. The photon states remained entangled during the capture and release. "We put entanglement into matter and then read it out," says Kimble's coauthor Julien Laurat, who was then a colleague of Kimble's at Caltech but is now at the Pierre and Marie Curie University in Paris.

First, Kimble, Laurat and their colleagues shot photons one at a time at a semitransparent mirror. In this situation each photon, presented with the choice of bouncing off or zipping through, will not make up its mind right away. Instead, it will split its path into two, a superposition of both possibilities. Only when forced to interact, for example by running into a detector, will the photon appear all in one place or in the other. Because these two measurements are mutually exclusive rather than independent, the two paths are entangled states.

Next, the researchers trapped each of the virtual photons in a cloud of cesium atoms. Using a laser pulse, the physicists turned the clouds transparent, to allow the photons in. When the physicists turned the laser off, the clouds went back to opaque, trapping the photons inside. That forced the photons to virtually come to a stop, as their quantum states became enmeshed with the quantum states of the clouds. So the clouds themselves became entangled.

The team was able to store the quantum information — preserve the entanglement — for up to 10 microseconds. A second laser pulse made the gas transparent again, allowing the two virtual photons to escape and continue on their paths. The physicists were able to check that the two photon states were still entangled.

What's missing now, Laurat says, is the ability to entangle two separate qubits by entanglement swapping. Still, Lukin says, the Caltech result was "an important step."

In another recent result, Kuzmich and his collaborators induced a cloud of atoms to emit two photons at once, with wavelengths that were each optimized for different tasks — for transmission through an optical fiber and for storage in another qubit. Typically, single photons emitted by atom clouds tend to have wavelengths too short for efficient telecommunications, Kuzmich says.

According to Lukin, eventually, a practical quantum memory will need to store information on some kind of solid support. In this respect, he says, single-atom impurities in artificial diamond are one of the most promising candidates, since they would require no sophisticated laboratory to handle (*SN: 4/5/08, p. 216*).

Most of the pieces needed to put together a quantum Internet now exist, and the challenge will be to make them work together efficiently. With the best technology available so far, a working prototype might end up costing as much as $100 million, and might be able to send just one qubit per minute, Kuzmich says.

A more sensible question might be: What would a quantum Internet be good for? So far, the main motivation for researchers has been to provide secure communications. But a quantum Internet might some day do things that, until recently, would have sounded like complete science fiction. ■

Explore more

■ H.J. Kimble. "The Quantum Internet." *Nature*. June 19, 2008.

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Feedback

> "In my own case, the link between depression and chronic migraines was broken largely by physical therapy...."

Starry details

The article "Astronomers find distant star with a whole set of superEarths" (*SN: 7/05/08, p. 7*) leaves out some of the most interesting and important information. Is HD 40307 a G-type star like our sun? Which method was used to detect the planets? The article implies Doppler was used, but Doppler could not give the specific masses of planets in the article.

JOHN MYERS, SAN DIEGO, CALIF.

HD 40307 is a K-type star, spectral class K2.5V. You could call this an orange dwarf star. The researchers used the Doppler method. Technically their measurements give minimum masses. The team probably presented these as the actual masses because the researchers believe the angles of inclination of the orbits are close to 90 degrees and therefore the actual masses are close to the minimums. —RON COWEN

Breaking the link

I have observed the link between serious disease and depression—discussed in "Sick and down" (*SN: 7/19/08, p. 26*)—in two elderly relatives, now deceased. It caused one to engage in self-destructive behaviors and the other to skip medications. In my own case, the link between depression and chronic migraines was broken largely by physical therapy after surgery for repair of a torn ligament. To my astonishment, the depression lifted after about six weeks. Medications were also changed, so the extra exercise alone may not have done the trick. I am convinced it was a big part.

DIANA GAINER, GREENVILLE, TEXAS

Planning for hazards

In regard to "Challenges to building a disaster-resilient nation" (*SN: 6/21/08, p. 32*), the South Carolina Emergency Management Division—from which I am now retired—and many state counties use Hazards United States Multihazard, or HAZUS-MH, at the state and local levels. We have used this program for many years. We were the first state to update the infrastructure database. Working with FEMA and a consultant, the state is now using a web portal and consolidated data management system to update the infrastructure annually and perform hazards analysis. Planning hazard preparedness, response, recovery and mitigation is an ongoing and important program at SCEMD, state agencies and local governments, and for FEMA.

JOHN KNIGHT, RIDGEWAY, S.C.

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The Universe in a Mirror: The Saga of the Hubble Space Telescope and the Visionaries Who Built It

By Robert Zimmerman

"The truth is that what ordinary people really care about are things they can see, with their own eyes," writes Zimmerman, a science writer and historian.

The Hubble Space Telescope has let the public see the universe and has completely changed humanity's perception of the cosmos. *The Universe in a Mirror* explores the lives of the men and women who dreamed of, lobbied for and engineered the first optical, Earth-orbiting telescope—the one that made the view of the heavens clear.

Zimmerman begins the story with astronomers who were "condemned to look at the heavens as though they had bad vision and were forbidden from using glasses." The consequences of these cataracts, the author contends, were profound: Dreamers like Lyman Spitzer, hard-nosed realists like Nancy Roman, brilliant engineers like Jean Olivier and tenacious astronauts like Story Musgrave united to find a way to shoot a mirror-equipped metal can into space so that everyone could marvel at the universe's mysteries.

Zimmerman describes how the men and women who make Hubble fly risk their careers, families and lives to "build and fix what has undoubtedly been the most successful and important scientific instrument ever put into space."

The Universe in a Mirror is an epic biography of the Hubble telescope. But perhaps more poignant is the book's subtle reminder of all that will be lost in just a few years when Hubble falls from its orbit around Earth—and disintegrates.

—Ashley Yeager

Princeton Univ. Press, 2008, 287 p., $29.95.

Reframing Scopes: Journalists, Scientists, and Lost Photographs from the Trial of the Century

Marcel Chotkowski LaFollette

Watson Davis clipped a short article out of a newspaper on May 7, 1925. John Scopes had been arrested for discussing evolution in a Tennessee public high school. In the Scopes trial, Davis saw an opportunity for his young nonprofit organization, Science Service, to prove its worth.

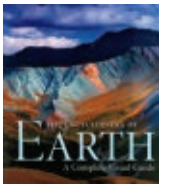
The Science Service Executive Committee agreed to give its reporters $1,000 to cover the trial. The committee also decided to reject neutrality, supporting the defense on the side of evolution. Davis and Frank Thone, the senior biology editor of Science Service's newsletters, acted as journalists as well as informal assistants to the defense, says historian LaFollette. They sought out top scientists to comment on the trial and lived among the scientists and biology teachers. Thone wrote: "All day and far into the night the rumble of scientific discussion and laughter issues forth from the Defense mansion, that pleasant old house … that has become the headquarters for the defenders of science, religion, and freedom."

Magazines and newspapers across the country ran columns published by Science Service, now Society for Science & the Public, publisher of *Science News*.

Rather than rehashing old stories from the trial, LaFollette uncovers unprocessed records and lost photographs from Science Service records stored in the Smithsonian's archives. She examines the role of the press in shaping the trial. She also reveals how different Davis and Thone were from others who sensationalized the trial or simply kept their distance. **—Amy Maxmen**

Univ. Press of Kansas, 2008, 172 p., $45.

The Encyclopedia of Earth: A Complete Visual Guide



Michael Allaby, Robert Coenraads, Stephen Hutchinson, Karen McGhee and John O'Byrne

The reference book for the planet, complete with thousands of photographs, illustrations, diagrams and maps.

Univ. of California Press, 2008, 608 p., $39.95.

Year of the Fires: The Story of the Great Fires of 1910

Stephen J. Pyne

A former firefighter describes the destructive blazes that shaped forest-fire policy in the United States.

Mountain Press Publishing Company, 2008, 325 p., $16.

One Minute Mysteries: 65 Short Mysteries You Solve With Science!

Eric Yoder and Natalie Yoder

With these short stories, kids ages 8 to 14 can have fun learning and solving puzzles.

Science, Naturally!, 2008, 176 p., $9.95.

The Future of the Internet — And How to Stop It

Jonathan Zittrain

The Internet's openness nourishes innovators but also makes it vulnerable to abuse.

Yale Univ. Press, 2008, 342 p., $30.

Primeval Kinship: How Pair-Bonding Gave Birth to Human Society

Bernard Chapais

An account of the evolutionary origins of human kinship, linking social and biological anthropology.

Harvard Univ. Press, 2008, 349 p., $39.95.

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Euroscience Open Forum

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In communicating science, Europe envies the U.S.

On July 21, at the Euroscience Open Forum in Barcelona, members of the European astronomy community participated in a discussion about why their space program has failed to engage public interest in a manner comparable to programs in the United States.

Organized by Dirk Lorenzen, a physicist turned journalist for German public radio, the session was titled "Reaching for the Stars: Research in Heaven, Communication in Hell." Lorenzen, a longtime reporter on space science and technology, began by pointing out that the public, both in Europe and elsewhere, knows little of the work of ESA, the European Space Agency. Yet NASA is known globally. Lorenzen blamed the overall attitude of European scientists, saying that most of them "don't regard scientific communication as important."

As an example, he pointed out that NASA's Hubble Space Telescope has generated hundreds of gorgeous photos from throughout the cosmos. The images are readily available on its website and can be downloaded without charge. By contrast, he said, ESA's powerful telescopes made only four Hubble-like space images available in the last year. Furthermore, journalists seeking to use ESA images must wait for official approval, which has sometimes been given only after a publication deadline has passed.

Lorenzen assembled a panel of three space science representatives to respond to these concerns. They were Claus Madsen, a foreign affairs officer for the European Southern Observatory; Rudolf Albrecht, of the Space Telescope European Coordinating Facility; and Mark Kidger, a researcher and communications specialist for the European Space Astronomy Centre near Madrid. Excerpts follow:

Madsen: The criticism is well placed and deserves consideration. Journalists know NASA, not ESA. There is a European malaise — we are afraid of showing our success stories. There are really three main issues: of perceptions, culture and institutional structure. Scientists often believe that they don't know how to talk to the media. But also they see it as beneath their intellectual level. I know one scientist who turned press officer. He called himself a failed scientist.

Institutionally, there is no career incentive to talk to the media. In a world of hypercommunication — Internet, cell phones, PDAs — it's strange that communication is so undervalued in science. Public communication is seen as a burden — something we have to do. It's not understood as what it should be — a service investment. Most industries make a 5 to 10 percent investment in public communication of their work. In science it's less than 1 percent.

> **In a world of hypercommunication — Internet, cell phones, PDAs — it's strange that communication is so undervalued in science.**
>
> **CLAUS MADSEN**

And, yes, ESA has taken its kicks. But let's not limit the problem to ESA. We aren't the only scientists with this attitude. I do claim that this is changing, that the wind is blowing in the right direction. But we started from a very low point, so it is hard for people to tell.

Albrecht: Without belittling scientific papers, they tend to be the holy cause of science. We have to get them in; if not, careers can be terminated. And they disseminate and build knowledge in our community. But scientific papers only reach the immediate community; they hardly cross the scientific disciplines. We have to also digest the information for the public.

Why? The public is powerful. In the United States, the Hubble Space Telescope servicing mission was canceled and astronomers were unhappy. If only astronomers were unhappy, the administration would not care. But all at once, there was a huge public outcry, and the mission was restored. You wouldn't see that in Europe.

In the U.S., of course, such public influence can also be dangerous to science. The regulation of stem cells is an example. But we have to remember that the media can be the university of the general public. In an ever more complex society, it's important for the public to be well informed. We want to avoid technophobia — if that develops, people will go in other directions like cults, astrology, they make strange decisions, they elect leaders ... well, I think I won't say anymore about that. But we should learn that the media can propagate rational concepts and thoughts.

Kidger: The attitude that's prevalent in Europe is "don't speak to him — he talks to journalists." It's changing, but slowly.

We need to be more confident about what we do. We work with Americans, collaborate, and the feeling is that we are lucky that NASA is willing to collaborate with us, not that we're willing to let them collaborate. We need to let people know that ESA has an incredible space record.

Of course, one problem is that out of 100 scientists, maybe five or 10 can really talk well at a public level. Those ones are pretty damn good. But the other 90 percent are a dead loss.

But we should keep encouraging scientists to do it. Really, it shouldn't be so hard. Scientists like to talk about what they're doing. Once they start, it's hard to get them to stop. ■

Deborah Blum, a freelance science writer and professor of journalism at the University of Wisconsin–Madison, provided this report from the meeting in Barcelona.

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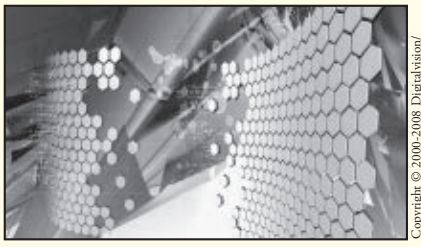

About Your Professor

Professor Steven Strogatz is the Jacob Gould Schurman Professor of Applied Mathematics and Professor of Theoretical and Applied Mechanics at Cornell University. He received his Ph.D. from Harvard University. Professor Strogatz is the author of *Nonlinear Dynamics and Chaos*, the most widely used textbook on chaos theory. In 2007, he received the Communications Award—a lifetime achievement award for the communication of mathematics to the general public—from the Joint Policy Board for Mathematics, which represents the four major American mathematical societies.

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