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ScienceNews

MAGAZINE OF THE SOCIETY FOR SCIENCE & THE PUBLIC ■ AUGUST 15, 2009

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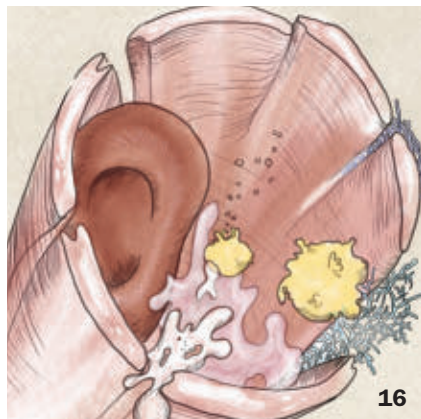
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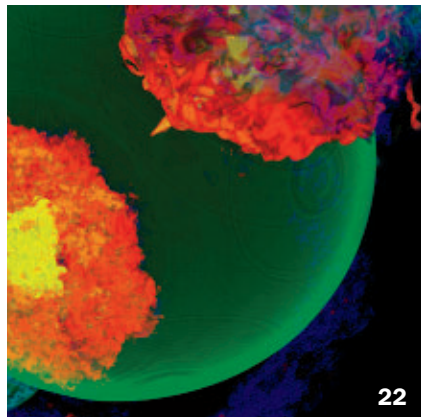
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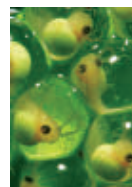
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COVER Three-day-old frog embryos respond to changes in oxygen levels—remarkably advanced behavior for unhatched eggs. *Photo by Karen M. Warkentin/Boston University*

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Subscriptions subs@sciencenews.org • *Editorial/Letters* editors@sciencenews.org

Advertising/Business snsales@sciencenews.org

✱ *Texterity* Digital edition provided by Texterity, www.texterity.com

Science News (ISSN 0036-8423) is published biweekly, for \$54.50 for 1 year or

\$98 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: PO Box 1205, Williamsport, PA 17703-1205.

For new subscriptions and customer service, call 1-800-552-4412.

Postmaster: Send address changes to *Science News*, PO Box 1205, Williamsport, PA 17703-

1205. Two to four weeks' notice is required. Old and new addresses, including zip codes,

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FROM THE EDITOR

Lessons from cosmic past tell about future, present



Astronomers are the ultimate historians. By peering into the depths of space, they perceive ongoing in the universe from eons long gone by, thanks to the time it takes the light emanating from faraway events to travel vast distances.

Some of those events are so spectacularly bright that their light tells a story billions of years old. Consequently astrohistorians can deduce details of the cosmic past that seemed forever lost, as nobody was around to write a blog about occurrences back then.

Most recently (well, about a decade ago), and most surprisingly, astronomers have used old light to discover that the expansion of the universe is speeding up. This light signals stellar explosions known as supernovas (the type 1a variety). Distant type 1a supernovas appear dimmer than they ought to be, so they must be farther away than expected. That means the expansion rate has been getting faster rather than slower.

This much is well established. But when astrohistorians become astroprognosticators, forecasting the future of the universe, they find that knowledge of accelerating expansion is not enough. They need a precise measure of the degree of acceleration, which requires knowing just exactly how bright those supernovas are supposed to be in the first place. So intense efforts are underway to decipher the physics behind the thermonuclear detonations that give 1a supernovas their radiance, as Ron Cowen reports in this issue (Page 22).

Depending on the outcome of such efforts, astronomers may someday be able to say whether everything in the universe will eventually be ripped to shreds or merely get colder and colder, leaving space emptier and emptier.

Of course, some still hold out hope for an ultimate cosmic collapse, the “Big Crunch” mirror to the original Big Bang. Followed, perhaps, by another bang for another try. But so far the lessons from studying cosmic history suggest that the future will not repeat the past.

In any case, reliving the past or foreseeing the future is not really the chief benefit of such endeavors. It's more about understanding the present. Astronomers have repeatedly revealed unexpected insights into the nature of reality. By investigating objects and events remote in time and space, astronomers have become not only historians but also detectives, discovering key clues to the natural laws governing today's universe — including the Earth and everything on it, even life. — *Tom Siegfried, Editor in Chief*

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Scientific Observations

"There will [be] more and more 'hybrid' studies that don't easily fit under conventional labels or approaches. These papers often have a more difficult time being fairly reviewed and published in a top-notch journal. In the review process, there are often the 'biology' expert and the 'physicist' expert, but sometimes no one really understands fully all the parts of the work. In general, journals could help by being enlightened about this type of hybrid work, and try to promote these new approaches and directions."

MICROBIOLOGIST FRED CHANG OF COLUMBIA UNIVERSITY MEDICAL CENTER IN THE JULY 14 *CURRENT BIOLOGY*



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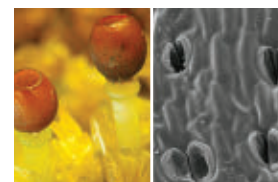
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EARTH

In a serendipitous find, researchers have hit upon a molecule that seems to soak up carbon dioxide from the air. Read "CO₂ sponge."

BOTANY

Dehydration, not pressure, causes tiny pores in the *Sphagnum* moss (shown) to spew spores, research shows, overturning a long-held notion. See "Pseudo pores help fling spores."



Science Past | FROM THE ISSUE OF AUGUST 15, 1959

COMPLEX "MOON" SUCCEEDS — Explorer VI, sent up on Aug. 7, is the most complex satellite launched by the United States. The 142-pound satellite orbits the earth from 150 miles at its lowest point to some 25,000 miles at its farthest... This highly elliptical flight path means that the satellite's instruments will cover a larger volume of space near earth for a longer time than any previous satellite. Scientifically, probably the most interesting information to be radioed



back from space ... will come from the three devices contained in the satellite to map the radiation belt ringing the earth.... From their telemetered readings scientists should be able to determine exactly how dangerous the earth's natural radiation belts will be for future space travelers.

Science Future

August 31–September 4

Scientists and policy makers meet at the World Climate Conference-3 in Geneva. Visit www.wmo.int/wcc3

September 2–6

IEEE Engineering in Medicine and Biology Society meeting in Minneapolis. See www.embc09.org

September 12–16

Educators explore new teaching methods at the Astronomical Society of the Pacific meeting in Millbrae, Calif. See www.astrosci.org/events/meeting.html

MATTER & ENERGY

A model shows a new way renewable energy could be produced from salt water, freshwater and electrodes. Read "Salty water power."

ATOM & COSMOS

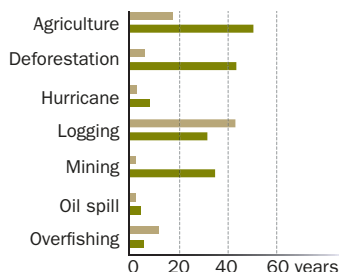
Rotation could explain how the universe's dimmest, most mysterious galaxies formed. See "Rotation may solve cosmic mystery."

Science Stats

Ecosystem convalescence

■ Animal ■ Plant

Average time needed for communities to recover based on disturbance type



SOURCE: H. JONES AND O. SCHMITZ/PLOS ONE 2009



For Daily Use

Another reason to take that multivitamin: At least in women, regular consumption of the pills seems to ward off potential chromosome damage, according to research in the June 1 *American Journal of Clinical Nutrition*. A study of 586 women found that telomeres, regions of DNA that protect the ends of chromosomes, were 5.1 percent longer among regular multivitamin users than nonusers, after controlling for age and other variables. Shorter telomeres have been associated with chromosome damage and aging. The vitamins' value could come from their antioxidants, which neutralize chemicals that might damage DNA, the researchers suggest.

“ I suspect that few people have ever seen a toucan without wondering, ‘Why the beak?’ ” — WINSTON LANCASTER, PAGE 13

Genes & Cells Path to wiener dog

Body & Brain Cheap shots at typhoid

Matter & Energy The splatter of raindrops

Life Whopping bill air-conditions

Earth Two auroras don't align

Numbers Laser generates random digits

In the News

STORY ONE

Virus can cause AIDS-like illness in chimpanzees

New study documents SIV's effects in a wild population

By Rachel Ehrenberg

A nonhuman primate version of the virus that causes AIDS was long thought to be harmless in its African hosts, but chimpanzees have not been spared after all.

Along-term study of a wild population has found that chimpanzees naturally infected with simian immunodeficiency virus, or SIV, die early and their babies die within a year of birth. In one instance, a female died with all the hallmarks of end-stage AIDS. The work, reported in the July 23 *Nature*, could help researchers understand the pathogenicity and species-to-species transmission of immunodeficiency viruses that, up until now, have appeared to pose serious health hazards primarily to humans.

“Great apes have so many disease threats — Ebola, anthrax, respiratory diseases spread from people — the last thing they need is something resembling AIDS,” comments Craig Stanford, codirector of the Jane Goodall Research Center at the University of Southern California in Los Angeles.

As well as boding ill for chimps, the research also emphasizes how the fate of those infected depends not only on characteristics of the virus itself, but also those of the host, including genetic makeup and



A new study reveals that chimps naturally infected with SIV die early. Chimp 006, the first to test positive in the study, went missing in 2007 and is presumed dead.

immune response, says Robin Weiss of University College London.

The sick chimps are from a population of the chimp subspecies *Pan troglodytes schweinfurthii* that lives in Gombe National Park in Tanzania. Of the four subspecies of chimpanzees, only two are known to harbor the chimp version of SIV, dubbed SIVcpz. The other subspecies that carries the virus, *Pan troglodytes troglodytes*, lives west of this region. It is from this second subspecies that SIV is believed to have jumped to humans on three separate occasions, yielding three types of HIV-1. One of these types is responsible for a majority of cases of AIDS in humans.

An AIDS-like illness has not been observed in the western chimps despite the presence of SIVcpz in their population. Perhaps SIVcpz is not that virulent in the western chimps but becomes more so in a different host such as the eastern chimps or humans, Weiss speculates.

Pathogenicity may also be affected by how long a host species has interacted with a virus. If the sick eastern chimps acquired SIVcpz from their western relatives fairly recently, then the chimps may have had naive, weaker immune responses to the virus, Weiss says.

“It might be quite a new virus in that population,” Weiss says.

Similarly, HIV-1 may be particularly devastating in people because of the virus's relatively recent move to humans. Since the AIDS pandemic began, research has shown a range of susceptibility, resistance and disease progression from individual to individual. Past work has already identified primate genes that hinder infection, and the new work could help clarify what causes differences in pathogenicity among species and individuals.

The sick chimpanzees were part of a population that has been under study by Jane Goodall and her colleagues since the 1960s. Without the animals' familiarity



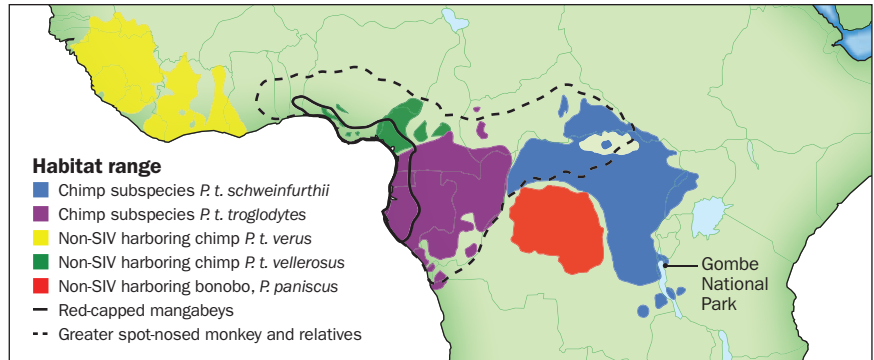
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with researchers, the work would not have been possible, says Beatrice Hahn, a virologist specializing in AIDS at the University of Alabama at Birmingham, who led the new study.

For nine years, the researchers followed 94 chimps from two communities in Gombe National Park. The scientists tested for virus antibodies in 1,153 chimp poop and 226 urine samples. Samples that tested positive for SIV antibodies — immune system proteins that the host produces in response to the virus — were then tested for viral RNA.

Seventeen chimpanzees tested positive for antibodies against SIVcpz and 14 of these animals had viral RNA in their feces. Two of the antibody-positive chimps were babies of infected moms. Oddly, one of these moms was SIVcpz-negative when her baby was born but became infected 10 to 15 months after giving birth, suggesting that transmission through breast milk is possible. Seven of the 17 infected chimps died or went missing (and were presumed dead) in the course of the study.

A comparison of survival rates revealed that infected chimps had a 10-



Only two of the four subspecies of chimpanzee, *Pan troglodytes troglodytes* and *Pan troglodytes schweinfurthii*, are known to harbor SIV. The eastern chimps in a new study may have acquired SIV from their western relatives. Previous work suggests the SIV version in the western chimps is a mix of versions from red-capped mangabeys and greater spot-nosed monkeys and relatives.

to 16-fold greater death rate than uninfected chimps. And uninfected females were three times as likely to give birth as infected females were, the researchers report. All four infants born to infected mothers died before their first birthday.

Analyses of the spleens and some lymph nodes of some of the chimps showed that in infected individuals, disease-fighting white blood cells were depleted, akin to the changes seen in people with AIDS. One female who died

within three years of becoming SIVcpz-positive also had several abscesses in her abdomen from nematode infection, and her skeletal muscle had wasted away, findings consistent with end-stage AIDS.

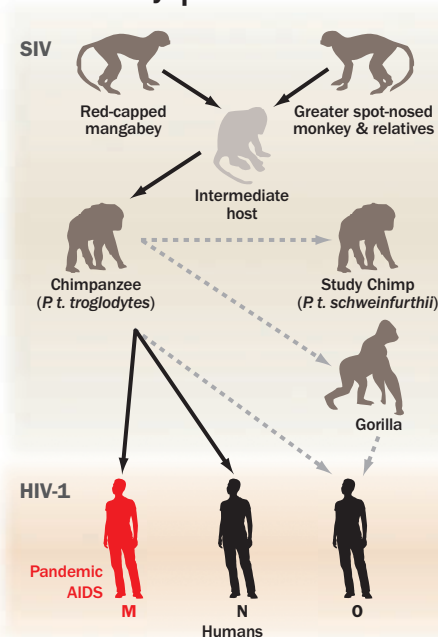
While troubling, the work suggests that SIVcpz in this subspecies isn't as serious a threat as HIV is in humans, Hahn says. Yet the discovery of the sick chimps offers what could be a better model for studying pathogenic lentiviruses, a group of viruses including SIV and HIV that are characterized by very long incubation periods.

There are at least 40 different simian immunodeficiency viruses. Previously, Asian macaques, a type of monkey, were the only nonhuman primates known to regularly develop full-blown AIDS when infected with SIV. In their natural habitat, however, Asian species do not come into contact with the virus.

Now that researchers are aware of SIV's pathogenicity in the wild, they should screen sanctuary chimps for the virus, Hahn says. If carriers, these chimps could provide additional information on the virus's relationship with its host.

The new findings underscore how long-term studies can reveal what might be right under researchers' noses, she adds. Typically, "the only time you know something is up is if you have a lot of dead bodies." ■

Back Story | UNTANGLING SIV AND HIV-1



The version of SIV found in the chimpanzee *P. t. troglodytes* appears to have evolved from a mix of SIV from red-capped mangabeys and greater spot-nosed monkeys and their relatives, maybe via an intermediate host.

This chimp version may have jumped to another chimp subspecies and gorillas. It also may have jumped to humans at least three different times, giving rise to HIV-1 groups M (responsible for most AIDS cases), N and O. Group O resembles the SIV recently found to infect gorillas, who may have been an intermediate host. An additional HIV-1 group, P, also similar to gorilla SIV, has recently been proposed.

A new estimate published in *PLoS Computational Biology* suggests that SIV may have infected its nonhuman hosts for hundreds of years — much shorter than previously thought — before jumping to humans as HIV-1 in the 1900s.

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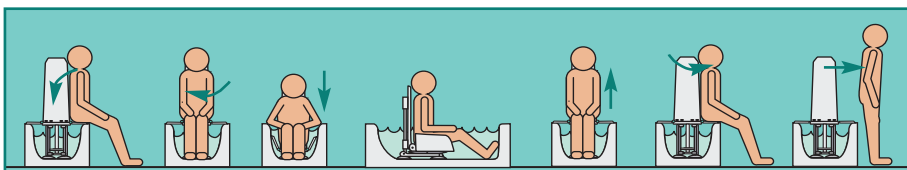
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Researchers traced the dachshund's short, curved legs to a retrogene called *fgf4*.



Old gene learns new short leg trick

Diminutive dogs owe stature to duplicated stretch of DNA

By Laura Sanders

It's hard to say how the zebra got its stripes, but scientists now know how the wiener dog got its short legs. Height-challenged dog breeds — including dachshunds, corgis and basset hounds — can thank an extra copy of a normal gene for their diminutive stature.

"It's stunning to see a genetic modification like this," developmental geneticist Douglas Mortlock of Vanderbilt University in Nashville says of the study, published online July 16 in *Science*.


In the new study, researchers focused on eight breeds from more than a dozen known to have a trait called chondrodysplasia — having legs that are short (relative to body size), curved and heavier-boned than normal.

Heidi Parker of the National Human Genome Research Institute in Bethesda, Md., and colleagues first analyzed the genomes of 95 dogs from the eight short-legged breeds and the genomes of 702 dogs from 64 breeds without the trait. Then the researchers pinpointed an extra stretch of DNA on chromosome 18 in every dog from the short-legged breeds. The extra DNA did not turn up in a closer analysis of 204 of the control dogs.

The DNA is almost identical to another gene important for limb development, called *FGF4*. Located at the opposite end of chromosome 18 in dogs, the original *FGF4* gene was duplicated at some point in the dog lineage, creating a new copy elsewhere called the *fgf4* retrogene, the researchers say in their report.

In rare cases, messenger RNA — molecules that carry information from DNA to cellular machinery for making proteins — can get turned back into DNA. If this DNA then gets plopped back into the genome in a new neighborhood, and conditions are right for it to become active, it becomes a retrogene.

"Finding a retrogene that is active and associated with certain traits is very rare," Parker says. "We believe that they're out there and that it happens, but they haven't been identified. We may find more."

Most pieces of DNA that hop around the genome aren't functional, partly because they may land in an inhospitable location. But this retrogene found a sweet spot in the genome, apparently influencing dog leg length. "This is very important because it's the first time a clear morphological effect of a retrogene was detected," says evolutionary geneticist Manyuan Long of the University of Chicago. 

Protein protects cells from cancer with 1-2-3 punch

Scientists find third strategy for tumor suppressor p53

By Tina Hesman Saey

Cells, do you need layers of cancer protection but hate juggling multiple proteins? Then an important tumor suppressor is for you. This cancer-controlling protein, p53, does not one, not two, but three different jobs, all in one convenient package, a new study shows.

Previous studies have demonstrated that p53 stops cancer from developing by sensing stress, such as DNA damage, and turning on genes that prevent cells from dividing. The protein, which is a normal component of cells, also teams up with other molecules to trigger apoptosis, a type of cellular suicide, in over-stressed cells.

Now, researchers from the University of Tokyo and colleagues report in the July 23 *Nature* that p53 helps slice RNA into small regulatory molecules called microRNAs. These microRNAs help control production of proteins, including some involved in cell proliferation. Proliferation can lead to cancer if unchecked.

This newly discovered function of p53 is surprising, says geneticist Franck Toledo of the Curie Institute's research center in Paris. He and others have studied p53's other two roles, but no one suspected the protein might also participate in the slicing and dicing of RNAs.

All three cancer-related functions require a part of the protein that latches on to specific DNA segments. Most cancers involve some disruption in p53's action, either a mutation or inactivation of the protein, Toledo says. In many tumors, the mutation lies in the portion of p53 called the DNA-binding domain.

Scientists thought that p53 could

direct the production of long RNAs, called primary transcripts, which eventually are broken up into microRNAs. Previous work has shown that the p53 protein turns on production of a long RNA molecule that gets chopped into a particular microRNA.

Mutations in the protein would disrupt production of the primary transcripts, ultimately leading to lower levels of microRNAs. But the Japanese team found that primary transcripts of several microRNAs associated with cancer are made as usual in DNA-damaged cells, indicating that p53 doesn't play a role in primary transcript production. But it was still possible that p53 mutations might disrupt the machinery that chops RNA into microRNA, says Kohei Miyazono of the University of Tokyo.

In the new study, Miyazono and colleagues show that p53's DNA-binding

domain interacts with Drosha and p68, proteins in an assemblage responsible for snipping primary transcripts into hairpin-shaped molecules. Another group of proteins, which includes Dicer, chops the hairpins into the final, mature microRNAs. Levels of both the hairpin-shaped intermediates and mature microRNAs were lower in cells in which p53 was mutated, the team found.


Some of the affected microRNAs control production of proteins involved in cell proliferation. Having a dearth of these microRNAs could allow too much of the growth-promoting proteins to be made, leading to uncontrolled growth and cancer.

"What's really exciting is that they show mutant p53 is actually worse than no p53 at all," Toledo says.

The normal form of p53 serves as a molecular matchmaker, bringing

Drosha and p68 together slightly more efficiently than the two proteins find each other on their own, the researchers showed. But altered forms of p53 spirit away p68, interrupting its interaction with Drosha.

People who have low levels of p53 develop cancer earlier and have poorer prognoses after treatment, Toledo says. It is possible that other variations in the protein may affect the ability to process microRNAs, which could make some people more vulnerable to cancer, he says.

The new "findings suggest that, in human cancers, mutations that affect the DNA-binding domain of p53 essentially perform a hat trick by hitting three tumor-suppressive functions at once," Toledo and colleague Boris Bardot, also at the Curie Institute in Paris, write in a commentary published in the same issue of *Nature*. 

Springtail dries out for the winter

Study shows genetic details of arthropod's extreme survival

By Laura Sanders

Instead of chopping firewood and pulling out the mittens to prepare for chilly winter days, the Arctic springtail hunkers down and dries itself out. A study published online July 21 in *BMC Genomics* identifies some of the genes that permit the millimeters-long arthropod's extreme wintering stunt.

When temperatures drop below freezing, Arctic springtails (*Megaphorura arctica*) lose massive amounts of water and shrivel into little husks. By this method—cryoprotective dehydration—the critters ride out harsh winters. When conditions improve, the animals rehydrate, dust themselves off and crawl away.

"This whole question of what happens during dehydration is terribly interesting," says David Denlinger, an insect physiologist at Ohio State University in Columbus. "Here is an animal that has


figured out how to survive—dried out."

Study leader Melody Clark of the British Antarctic Survey, based in Cambridge, and colleagues collected springtails from mossy areas beneath cliffs in the Arctic. At their laboratory in England, the researchers monitored which of the springtails' genes are active during dehydration and rehydration.

"We've tried to emulate what happens in winter," Clark says. She and colleagues kept the springtails at about

5°C Celsius and gradually chilled them to -7°C. Between zero and -2°C, the springtails darkened and shriveled, eventually entering dormancy. Some of the springtails were allowed to gradually warm back up to 5°C over the course of 18 hours.

Using a custom-made gene chip, the researchers found genes active in shriveled, dry animals and genes active after the animals had just rehydrated.

Understanding animals' strategies for getting through freezing temperatures may be relevant for medical needs such as long-term tissue storage. "It would be a wonderful thing if we could do that to transplant tissues," Denlinger says. 



Arctic springtails dry out to survive the winter. These scanning electron microscope images show a hydrated springtail (left) and one that's ready to go dormant (right).

Body & Brain



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Typhoid vaccine proves effective

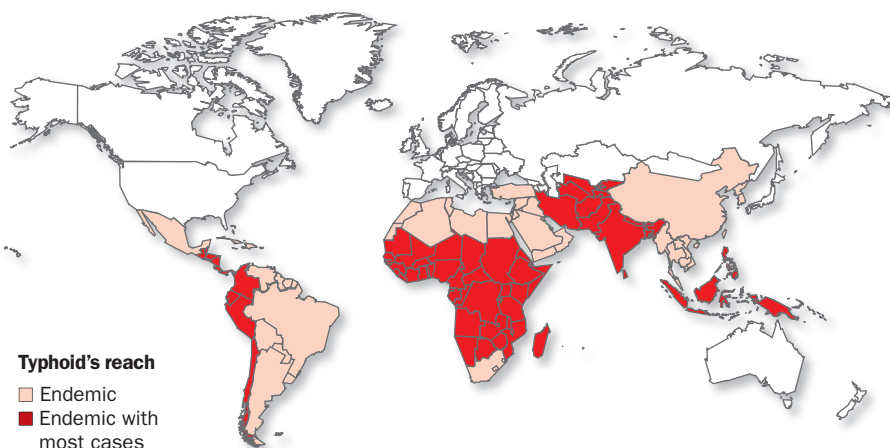
Study finds that shot confers immunity across age groups

By Nathan Seppa

An inexpensive vaccine against typhoid fever offers protection across age groups, a large trial in India finds. The study also shows that vaccinating half the people in a neighborhood confers significant protection throughout its population, researchers report in the July 23 *New England Journal of Medicine*.

Despite the availability of two approved vaccines, many countries have lagged in efforts to confront typhoid, which strikes 21 million people and causes 200,000 to 600,000 deaths worldwide each year.

In the new study, an international team vaccinated more than 37,000 people in the slums of Kolkata (Calcutta). Half the people in some neighborhoods received an injection of a typhoid vaccine called Vi, while half the people in other neighbor-



This map depicts areas around the world where typhoid fever is endemic, or naturally recurring. A large study in India shows that vaccination against typhoid can limit the disease's spread in entire neighborhoods, even among people in the neighborhood who aren't vaccinated.

hoods got hepatitis A vaccinations. Clinics tracked subsequent cases of fever.

After two years, the group receiving the typhoid vaccine had 61 percent fewer cases of the disease than the group getting the hepatitis shot, says coauthor John Clemens of the International Vaccine Institute and Seoul National University in South Korea.

Unvaccinated neighbors and relatives

also gained some level of protection.

"It makes sense that this could happen with typhoid," says Myron Levine of the University of Maryland School of Medicine in Baltimore. Researchers have seen similar indirect protection with pneumococcal vaccines, he says.

Clemens adds: "We believe that this study will strengthen the case for vaccinating against typhoid."

Spinal fluid test for early Alzheimer's

Study finds certain compounds predict risk in some cases

By Nathan Seppa

Elderly people with mild cognitive losses are at a heightened risk of progressing to Alzheimer's disease if they have a combination of telltale compounds in their spinal fluid, researchers report in the July 22/29 *Journal of the American Medical Association*.

By testing for a shortage of a sticky compound called amyloid-beta in the spinal fluid and for excess amounts of two kinds of a protein called tau, the scientists could identify people at greatest risk.

The test isn't foolproof, and a positive reading still warns of a disease for which there is no cure. But scientists

are heartened by this and earlier studies (*SN: 9/20/03, p. 179*) because Alzheimer's disease is difficult to foresee and its early symptoms are often mistaken for routine cognitive losses caused by aging.

Niklas Mattsson of a University of Gothenburg-affiliated hospital in Mölndal, Sweden, and an international group of scientists recruited 750 elderly people in Europe and the United States from 1990 to 2007. At the time of enrollment, the volunteers had mild cognitive impairment that wasn't attributable to aging alone but fell short of Alzheimer's disease. Each volunteer contributed a cerebrospinal fluid sample by undergoing a spinal puncture. The participants,

around age 69, were monitored for about three years during the study.

Those who developed Alzheimer's disease were more likely to have had less amyloid-beta or more tau in their spinal fluid than those who didn't develop Alzheimer's. People who had both low amyloid-beta and high tau levels were five times as likely to develop Alzheimer's disease during the study as were those with normal spinal fluid profiles, Mattsson says. The screening test correctly predicted incipient Alzheimer's disease 83 percent of the time.

In the future, studies that track elderly patients longer may show an increased accuracy rate because patients whose spinal fluid tested positive may develop Alzheimer's disease later, says Ronald Petersen, a neurologist at the Mayo Clinic in Rochester, Minn.

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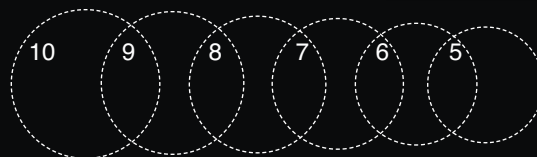
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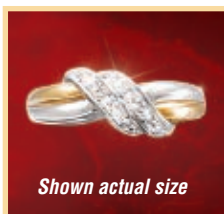
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Matter & Energy



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Green beetles are masters of manipulation

Study finds cells in the shell bend light in a particular way

By Laura Sanders

Jeweled beetles' resplendent shells have physicists green with envy. Intricate arrangements of cells on the beetles' outer layers manipulate light in a special way, a study published in the July 24 *Science* reveals. Understanding the shell's structure might prove useful for designing new optical devices.

The beetles, *Chrysina gloriosa*, get their greenish color from microstructures in their exoskeleton rather than from pigment. Study coauthor Mohan Srinivasarao of the Georgia Institute of Technology in Atlanta and his colleagues have found that these structures are also



Jeweled beetles' shells polarize light with a clockwise twist, researchers find.


responsible for the beetles' light-bending tricks. Light hitting the shell is reflected by microstructures, and the reflections create an electric field that forms a clockwise helix. Humans cannot see this property of light — known as circular polarization — but can see a green hue.

To find out how the beetle can shape light in this way, Srinivasarao and his colleagues examined the beetle's exoskeleton under high-powered microscopes.

Under the scope, the beetle's body appears as a "richly decorated mosaic of cusps and color," the authors report.

"The details are just stunning," says Srinivasarao. "There are all of these patterns on the beetle."

Cells with five, six and seven sides create intricate clusters. Cells gradually twist as layers get deeper, creating microstructures that look like snail shells turned on their sides. These regions — called focal conic domains — twist the reflected light to make it circularly polarized.

Stealing the beetles' tricks may help researchers design materials with desirable optical properties, comments Michael Barnes of the University of Massachusetts Amherst. Although it's too early to say what specific devices might be created with inspiration from the beetle shell, "the scientific goal is to understand the 'what' and the 'how' of micro- and nanoscale structures in natural systems," he says, "so that we can design our own systems for specific purposes." 

Each falling raindrop may go it alone

Study suggests drips shatter to produce a variety of sizes

By Laura Sanders

Raindrops keep falling on your head in all different sizes, and now researchers know why. The shattering of single raindrops after they leave clouds is enough to explain the wide variety of drop sizes, a study appearing online July 20 in *Nature Physics* shows, overturning the notion that complicated interactions between falling drops are to blame.

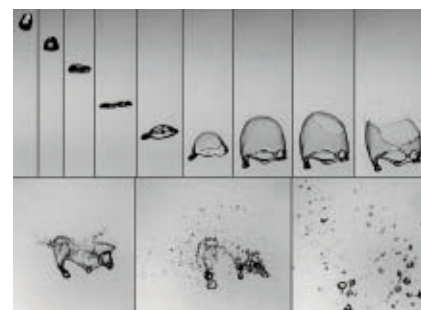
Explaining the details of raindrop shattering "is a very nice result," says Howard Stone of Princeton University, since "apparently, no one had connected a theory with the measurements."

Scientists used to think that the assortment in raindrop sizes was a result of splitting and coalescing as the drips bumped

into each other during their descent. But study coauthor Emmanuel Villermaux thought that collisions would be too rare to explain the size distribution.


Seeking an alternative explanation, Villermaux and Benjamin Bossa, both of Aix-Marseille University in France, conducted experiments on isolated water droplets. A high-speed camera captured each contortion of a solitary drop as it fell a few meters. An upward air current simulated the experience of a raindrop during its fall from the sky.

Over tens of milliseconds, each drop flattened like a pancake because of drag. Next, water in the pancake shifted to the outer rim, forming what's called a ligament shape. For some drops, these shapes looked like upside-down bags. Eventually,



In lab tests, one drop breaks into many (shown in this 60-millisecond sequence).

the ligament shape dramatically shattered into many smaller globules.

After creating mathematical equations to describe this shattering, the researchers found that the breakup of individual drops alone could explain the staggering variety of raindrops. "You don't need this interaction ingredient to understand how drops fragment," Villermaux says. "You just need a single drop." 

FROM TOP: GARY MEEK, GEORGIA TECH; E. VILLERMAUX



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Bird's distinct bill offers a big chill

Study identifies new function for toucan's sizable front end

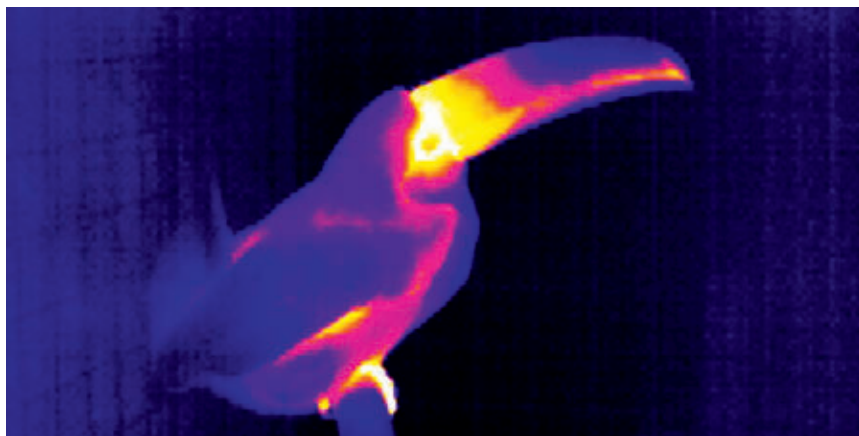
By Susan Milius

Toco toucans have a huge cooling bill. But it's the good kind.

While overheated people crank up their air conditioning, toucans increase the blood flow to their supersized, uninsulated bills, report Glenn Tattersall of Brock University in St. Catharines, Canada, and his colleagues. Extra heat radiating from the bill keeps the bird comfortable, the researchers report in the July 24 *Science*.

This study adds an “entirely plausible” twist to a long-standing debate, says Winston Lancaster of California State University in Sacramento. “I suspect that few people have ever seen a toucan without wondering, ‘Why the beak?’”

But documenting cooling powers for the toucan bill doesn't mean other ideas for its function are wrong, says coauthor Denis Andrade of the São Paulo State University's campus in Rio Claro, Brazil.



A toucan toucan can release heat through its bill, as seen here in an infrared thermography image. Warmer areas are shown in yellow, and cooler areas are in purple.

“The bill has many functions,” he says.

To test the cooling properties of bills, the researchers focused on the toucans with the largest bill, the toco toucan (*Ramphastos toco*). The bill of an adult bird can account for up to half of its body surface area.

Researchers put birds one at a time into a temperature-controlled chamber. Over the course of six hours, the chamber warmed 10 degrees Celsius. Thermal imaging showed the researchers how various parts of the birds' bodies changed temperature during the warm-up.

As the temperature increased, the birds' bills warmed too, a change that the researchers interpret as a sign that the birds were flooding their beaks' blood vessels with extra blood. Yet the unfeathered skin around the birds' eyes, an indicator of core body temperature, stayed about the same.

Toucans also spent a night in the chamber, and researchers monitored them during sleep. When the birds settled down, their bills warmed up, suggesting they were dumping heat. Animals typically cool down as they fall asleep. 🐦



First all-American honeybee

North America did too have a native honeybee. A roughly 14-million-year-old fossil unearthed in Nevada preserves what's clearly a member of the honeybee, or *Apis*, genus, says Michael Engel of the University of Kansas in Lawrence. The fossil (left) shows the somewhat jumbled parts of a honeybee, recognizable in part by its distinctive pattern of wing veins (arrow). The Americas have plenty of other kinds of bees, but all previously known honeybees come from Asia or Europe. Even the *Apis mellifera* honeybee that has pollinated crops and made honey across the Americas for several centuries arrived with European colonists some 400 years ago. “This rewrites the history of honeybee evolution,” Engel says, turning over the long-held view of Europe and Asia as the native land of all honeybees. The newly discovered bee, found squashed and preserved in shale, no longer exists as a living species, Engel says. To a specialist's eye, it looks closest to another extinct honeybee from Germany, *A. armbrusteri*. Engel and his colleagues christen the new honeybee *Apis nearctica* in the current, May 7, issue of *Proceedings of the California Academy of Sciences*. — Susan Milius 🐝

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Earth



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Lopsided lights grace the poles

Observations reveal that auroras are not symmetrical

By Sid Perkins

The serendipitous observations of two Earth-orbiting satellites, one passing high over the North Pole while the other whizzed over Antarctica, have revealed that Earth's auroras aren't symmetrical.

Auroras, commonly called the northern and southern lights, are caused when charged particles from space slam into gas molecules in the upper atmosphere. These phenomena are most often seen at high latitudes because the particles follow the lines of Earth's magnetic field, which pierce the atmosphere in polar regions.

Scientists have presumed that the aurora encircling Earth's northern magnetic pole mirrors that seen in the south because magnetic field lines connect the two hemispheres, says Nikolai Østgaard of the University of Bergen in Norway. But new observations, reported by Østgaard and Bergen colleague Karl Laundal in the July 23 *Nature*, reveal that the intensity and pattern of the northern and southern auroras can differ substantially.



Northern Hemisphere



Southern Hemisphere

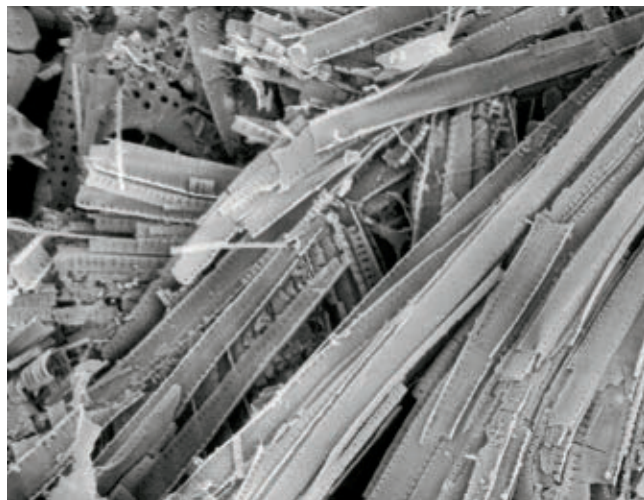
As seen on May 12, 2001, the northern lights aren't a mirror image of the southern lights. The find suggests an electrical current may connect the two hemispheres.

The team took data from satellites occasionally positioned so that both poles are observed simultaneously. Images taken the evening of May 12, 2001, London time, reveal that while post-sunset areas of Antarctica had a bright, persistent aurora, the brightest parts of the northern lights were transient and located over predawn areas. Known differences in Earth's magnetic field strength don't explain the new observations, Østgaard says.

The auroral asymmetries may be driven by large numbers of charged particles flowing between Earth's Northern and Southern hemispheres along mag-

netic field lines. An electrical current of this type has been previously proposed but never observed. According to theory, says Østgaard, those currents arise because sunlight-driven reactions in the atmosphere over a fully lit hemisphere cause the electrical conductivity there to differ from that over the darkened hemisphere half a world away.

Though the instruments viewing the two auroras were of different types, the asymmetries seem to be real, says Arthur Richmond of the National Center for Atmospheric Research in Boulder, Colo.



Sign of ancient sea ice

Fossils of marine algae (shown) reveal that Arctic sea ice, which is today very much an endangered species, formed before about 47.5 million years ago. Catherine Stickley of the University of Tromsø in Norway and colleagues examined sediments drilled from a site on a submarine ridge about 250 kilometers from the North Pole. In sediments deposited 47.5 million years ago, the team found fossils of algae whose modern cousins live in sea ice, pushing back the known date that ice first appeared by 1.25 million years, the researchers report in the July 16 *Nature*. Remains of those needle-shaped microorganisms, part of the genus *Synedropsis*, make up as much as 61 percent of the fossils in those sediments. The team's findings provide new details about the pace of global cooling occurring at the time, Stickley notes. — Sid Perkins

FROM TOP: K. LAUNDAL; C. STICKLEY/UNIV. OF TROMSØ, RICHARD PEARCE/NATIONAL OCEANOGRAPHIC CENTRE, SOUTHAMPTON



Laser beam, simple math employed to produce random numbers faster

New technique can generate 12.5 gigabits per second

By Laura Sanders

With a laser, a mirror and some simple calculations, researchers have created a fast, reliable way to produce long strings of random numbers. This speedy method, reported in the July 10 *Physical Review Letters*, may one day be used to improve data encryption, computer simulations and even gambling software.

Generating truly random numbers isn't easy. Many techniques rely on computer algorithms to create seemingly unpredictable chains of bits—0s and 1s. But such methods, says study coauthor Michael Rosenbluh, are not actually random. Under certain conditions, anyone with the same computer program could reproduce an identical series. Despite this flaw, many of today's encryption programs still rely on such computer-generated numbers.

Other techniques, based on the inherent messiness of physical processes, generate truly random bits but work too slowly to be practical. "If it takes you 10 years to generate a gigabit, it's not very helpful," says Rosenbluh, of Bar-Ilan University in Israel. "What we've shown is that you can generate very random numbers at very high rates."

In the new study, researchers harnessed chaos from a laser to generate 12.5 gigabits per second. That's more than twice the data a standard audio CD holds. (There are eight bits to a byte.) The rate, the researchers say, beats the 1.7 gigabits per second of another laser-based method reported by a different group last year and is on par with the speed of computer-based number generators.

Rosenbluh and his colleagues Igor Reidler, Yaara Aviad and Ido Kanter, all also of Bar-Ilan, shone a laser beam at a mirror a few meters away so that the


beam bounced back into itself. Light originating from the laser combined with the returning laser light to make a chaotic, unpredictable system that emits varying light intensities. In this system, Rosenbluh says, the laser “just goes wild.” Detectors then measured the varying intensities of laser light, and an analog-to-digital converter turned the light information into digital bits.

This wild system alone, though, is not totally wild. The time it takes a beam of light to travel from the laser to the mirror and back imposes a nonrandom structure on the system. “The problem,”

Rosenbluh says, “is that the randomness repeats itself every round-trip time.”


To eliminate this predictable pattern, the detector converted light to bits at irregular time intervals. The team fed this data into a computer program, which performed simple and quick mathematical manipulations, including subtracting bits from bits and throwing out some of the bits. These safeguard calculations appeared to eliminate all of the periodicity, resulting in a string of completely random bits, Rosenbluh says.

It remains to be seen whether this new random number generator will prove useful and replace computer-based systems. Mathematician Peter Hellekalek of the University of Salzburg in Austria questions the utility of the new method. "A laser setup is less practical than portable software," he says. ■



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Venom hunters

Scientists probe toxins, revealing the healing powers of biochemical weapons

By Laura Sanders

When the monitor lizard chomped into Bryan Fry, it did more than turn his hand into a bloody mess. Besides ripping skin and severing tendons, the lizard delivered noxious venom into Fry's body, injecting molecules that quickly thinned his blood and dilated his vessels.

As the tiny toxic assassins dispersed throughout his circulatory system, they hit their targets with speed and precision, ultimately causing more blood to gush from Fry's wound. Over millions of years, evolution has meticulously shaped these toxins into powerful weapons, and Fry was feeling the devastating consequences firsthand.

"I've never seen arterial bleeding before, and I really don't want to ever see it again. Especially coming out of my own arm," says Fry, a venom researcher at the University of Melbourne in Australia.

To unlock the molecular secrets of venom, Fry and other researchers have pioneered a burgeoning field called

venomics. With cutting-edge methods, the scientists are teasing apart and cataloging venom's ingredients, some of which can paralyze muscles, make blood pressure plummet or induce seizures by scrambling brain signals. Researchers are also learning more about how these toxins work.

Discovering venom's tricks may allow scientists to rehabilitate these damaging molecules and convert them from destroyers to healers. Venom might be teeming with wonder drugs, for instance. After all, a perfect venom toxin works with lightning speed, remains stable for a long time and strikes its mark with surgical exactitude — attributes that drugmakers dream about.

Already, toxins from a Brazilian viper have provided the key molecule for blood pressure-lowering drugs known as ACE inhibitors, and a medication based on cone snail venom alleviates types of chronic pain that even morphine can't touch. George Miljanich, a researcher

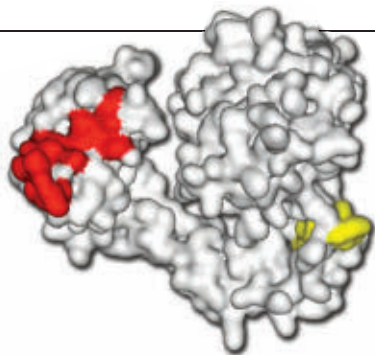
who helped develop the snail-derived drug, calls venom an "amazing soup" with "great potential as a source of new medicine."

What's more, researchers are stepping back in time to understand how the toxic proteins that make up venom evolved in different animals, revealing details on how beneficial proteins may have been recruited to the dark side and eventually become toxic. Such studies are also finding rapidly mutating toxin genes and describing how unique environmental conditions shape venoms in different animals.

Despite the occupational hazards, "It's a great time to be doing this kind of research," Fry says. "With the techniques we have today, it's astounding what we can learn."

What makes a venom

The "amazing soup" that is venom brims with proteins and smaller pieces of proteins called peptides. "Snake



The snake's 'meat tenderizer'

Venom from the western diamondback rattlesnake (*Crotalus atrox*, far left) contains a toxic protein (left) that targets blood vessel walls and kills tissue, earning it and similar toxins the nickname of “meat tenderizer.” By analyzing the sequences of amino acids that make up such toxins in related species of snakes, Stephen Mackessy of the University of Northern Colorado in Greeley and his colleagues found that the amino acid sequence changes more rapidly in certain regions (red) and less frequently in other regions (yellow) compared with the rest of the protein (white). Highly changeable regions enable toxins to diversify more quickly, the team proposes.

venom is virtually all protein, thick as honey,” says Christopher Shaw, a biological chemist at Queen’s University Belfast in Northern Ireland. Figuring out the long list of ingredients in these potent mixtures, and understanding the genetics behind the ingredients, are big challenges — ones that new research approaches are helping to address.

A multinational project called CONCO represents one effort to document venomous genes. In collaboration with the J. Craig Venter Institute in Rockville, Md., CONCO scientists are now sequencing the entire genome of the project’s namesake, the venomous marine cone snail *Conus consors*. Its genome is about the size of the human genome.

“The sequencing is moving ahead nicely,” but it is no small task, says Reto Stöcklin, a venom researcher at Atheris Laboratories in Geneva who leads the CONCO project.

With the decoded genome in hand, researchers will be able to quickly learn details about any toxin in *Conus consors* venom. “Once you have a genome, it makes it easier to know what you’re looking at,” says Baldomero Olivera, a cone snail expert at the University of Utah in Salt Lake City. But just because an organism’s DNA has the gene for a protein, that doesn’t mean the gene is active and the protein is produced. “As for which compounds you actually find in venom, there is much more play than we realized,” says Olivera.

To figure out which proteins and peptides are present in venom, scientists turn to several other approaches. One method relies on identifying messenger RNA, molecules created from DNA that carry a gene’s instructions to the cell’s

protein-building factories. Messenger RNA analysis was used to profile the toxins made by the Komodo dragon, a lizard only recently shown to be venomous. “With the techniques we have, we can point out what the dragon is making at the time, and say with absolute certainty,” says Fry, who led the analysis, which was published online May 18 in the *Proceedings of the National Academy of Sciences* (SN Online: 5/18/09). “We can almost obtain more data than we can process.”

In a study published online July 1 in *BMC Genomics*, researchers used a similar approach to identify toxins in the scorpion *Scorpiops jendeki*. The scorpion venom had 10 types of compounds that scientists already knew about, but surprisingly, nine unknown classes of molecules also turned up. These mystery molecules are unlike anything else in venom, the researchers write.

Researchers including Stöcklin rely on mass spectrometry, in which small pieces of proteins are identified by their motion through an electromagnetic field. This process results in a “chemical fingerprint,” which can be used to reconstruct the compounds in venom.

Taking venoms’ fingerprints has allowed researchers to make surprising finds about how venom composition can vary, even venom that comes from the same animal. For instance, in a study published in the *Journal of Proteomics*, Stöcklin and his colleagues showed that the composition of venom milked from live *C. consors* differed greatly from that of venom taken from dissected *C. consors* venom glands. The team hypothesizes that — similar to a snail ejecting venom in natural settings — the milking allows the cone snail to control venom compo-

sition by inserting some toxins into the venom and keeping others out.

Shape-shifting toxins

Venomous creatures are found throughout the animal kingdom. Everyone knows to beware of envenomed snakes, spiders and scorpions. But beware, too, of shrews, sea anemones and platypuses, to name a few. Researchers estimate there to be some 100,000 venomous species, each with its own blend of venom containing, in some cases, hundreds of different toxins. “It’s pretty clear that there are convergent features in all venoms,” says Olivera. “But each group has its own peculiarities.”

Researchers have found venom glands to be a rich source of information, not only for discerning differing molecular makeups of venoms (as in the cone snails), but also for anatomical comparisons. Such analyses could shed light on the evolution of various venomous creatures. In the Komodo dragon study, Fry and colleagues used an MRI scanner to reveal an intricate and unusual array of a dozen venom ducts, more than in other venomous lizards. The results show how the dragon’s venom system may have evolved from other, older lizard species, and help solidify the notion that Komodo dragons kill their prey with a combination of a powerful bite and venom injection.

Such a glimpse into the predatory life of a venomous creature has opened a research floodgate. “We’ve been chucking everything into the machine,” says Fry. “Vampire bats, cone snails, spiders, octopuses, you name it, we’re chucking it into the machine now and getting incredible images of the glands.”

Camilla Whittington of the Univer-

When venomous animals strike

Toxins in venom interact with a range of targets in the human circulatory and nervous systems, leading to a variety of effects.

Toxin producers

A handful of groups from the roughly 100,000 known venomous animals are shown.



Cone snails



Cephalopods



Irukandji jellyfish



Lampreys



Sea anemones



Leeches



Hookworms



Assassin bugs



Dipteran insects



Hymenopteran insects



Fleas



Ticks



Spiders



Frogs



Caterpillars



Scorpions



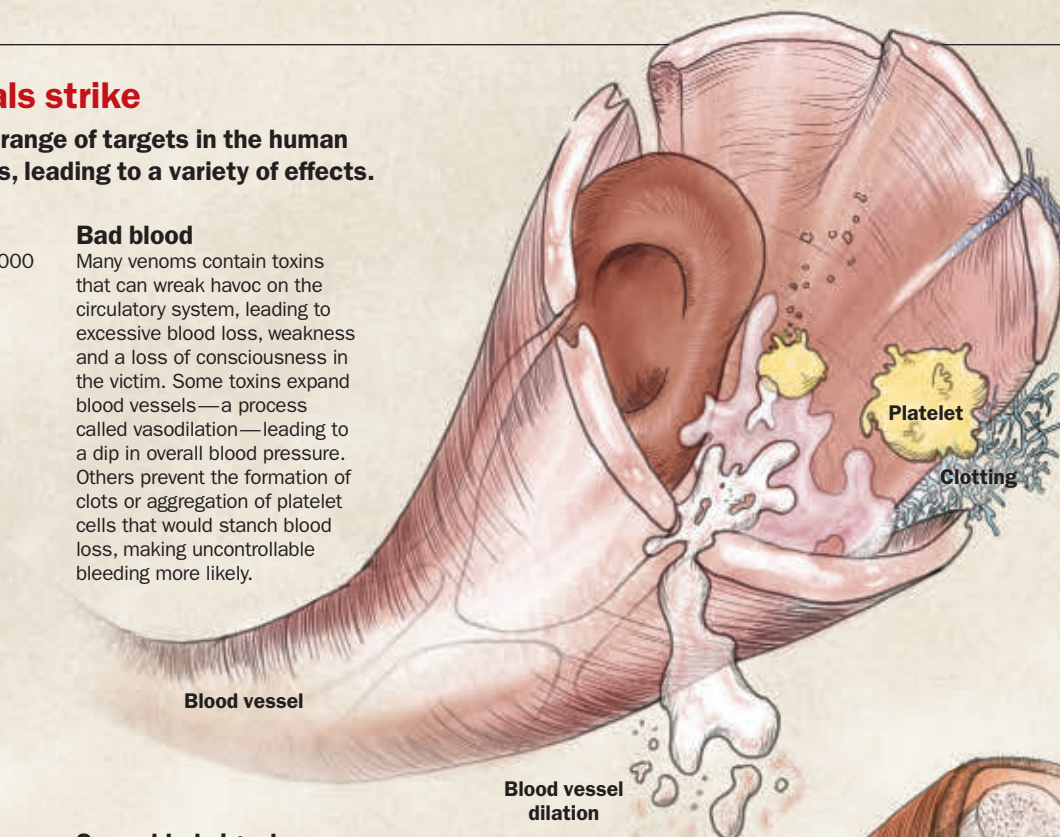
Snakes

Toxicoferan reptiles
(lizards and snakes)

Short-tailed shrews

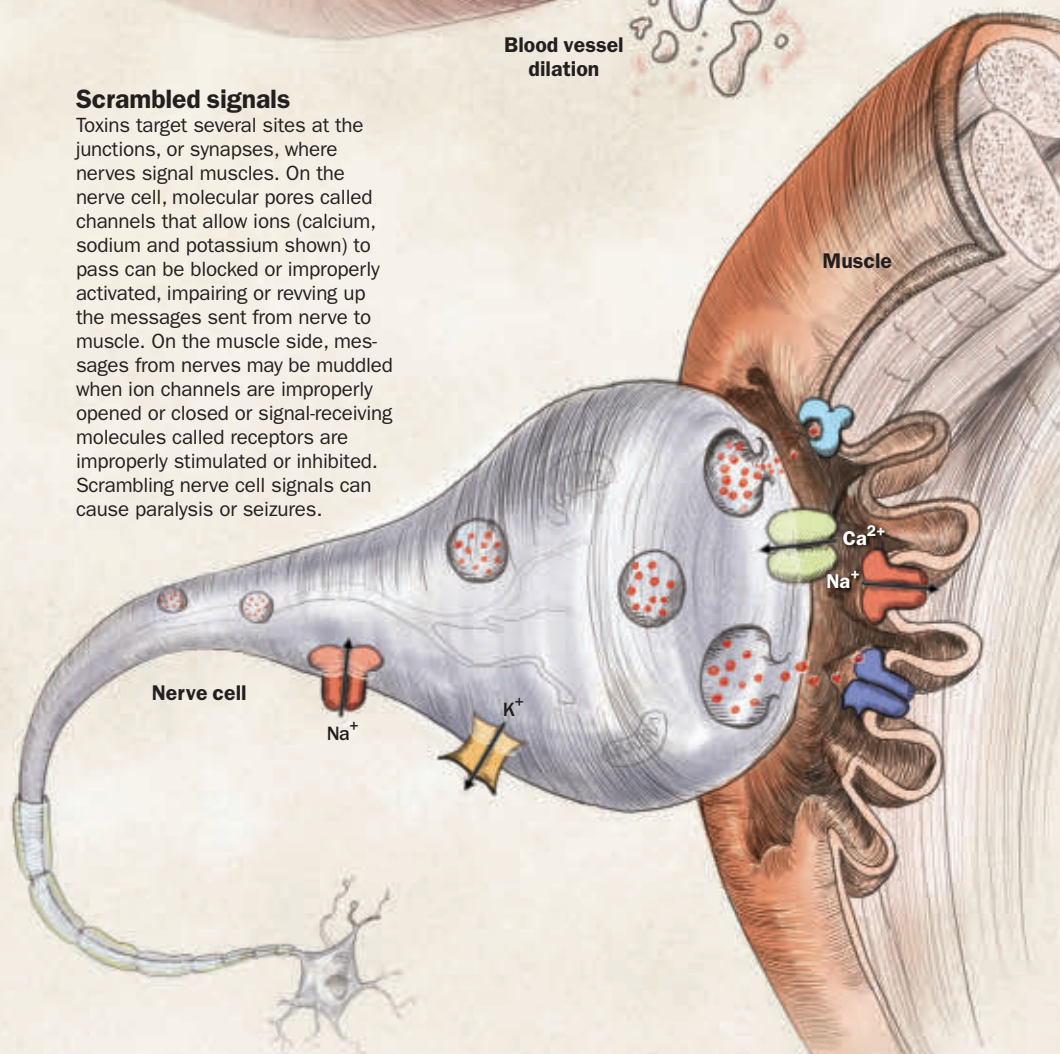
Bad blood

Many venoms contain toxins that can wreak havoc on the circulatory system, leading to excessive blood loss, weakness and a loss of consciousness in the victim. Some toxins expand blood vessels—a process called vasodilation—leading to a dip in overall blood pressure. Others prevent the formation of clots or aggregation of platelet cells that would stanch blood loss, making uncontrollable bleeding more likely.



Scrambled signals

Toxins target several sites at the junctions, or synapses, where nerves signal muscles. On the nerve cell, molecular pores called channels that allow ions (calcium, sodium and potassium shown) to pass can be blocked or improperly activated, impairing or revving up the messages sent from nerve to muscle. On the muscle side, messages from nerves may be muddled when ion channels are improperly opened or closed or signal-receiving molecules called receptors are improperly stimulated or inhibited. Scrambling nerve cell signals can cause paralysis or seizures.



Toxin targets:

Animal icons show venomous organisms that make each of the following known toxins.

Blood and blood vessels



Blood vessel dilators



eledoisin; tachykinin-like peptides



sialokinins



tachykinin-like peptides



blarinatoxin; kallikrein



peptidase S1 toxins



Anticlotting toxins



aegyptin; anopheline antiplatelet protein; apyrase; thrombostasins; anophelins



pallidipin and related lipocalins; infestin and related kazal-type proteins; rhodnius prolixus aggregation inhibitor-1



leech antiplatelet protein; saratin; hirudins; ornatin



snake venom metalloproteinases; C-type lectin toxins; factor Xa-like proteases; bothrojaracin



moubatin and related lipocalins; apyrase; savignin and related kunitz-type proteins; variegins; variabilin



apyrase



lopap, lonomin III

Nerves and muscles



Calcium-channel blockers



assassin bug toxins



ω -conotoxins



lamprey salivary CRISP



calcicludeine



ω -neurotoxins



CRISP toxins



Potassium-channel blockers



K-conotoxin



apamin



short scorpion toxins



cnidaria kunitz-type proteinase inhibitors; sea anemone type 3 potassium channel toxins



dendrotoxins



K-atracotoxins



CRISP toxins



Sodium-channel blockers



μ -O-conotoxins (pre- and post-synaptic); μ -conotoxins (pre- and post-synaptic)



hainantoxins; protoxin-II; huwentoxin-IV



Sodium-channel activators



β -toxins



μ -neurotoxins



Sodium-channel prolongers



δ -conotoxins



irukandji-toxins



α -toxins



sea anemone sodium channel inhibitory toxins



δ -atracotoxins



Nicotinic receptor antagonists



α -conotoxins



α -neurotoxins



Muscarinic receptor antagonists



uncharacterized toxin or toxins



three-finger toxins; phospholipase A₂ toxins

ILLUSTRATION BY NICOLLE RAGER FULLER;
SOURCE: B. FRY ET AL./ANN. REV. OF
GENOMICS AND HUMAN GENETICS 2009

sity of Sydney focuses her studies on the platypus, one of just a handful of venomous mammals. "Venom in mammals is very unusual, and to see how it evolved is interesting because it might lead to insights about mammalian evolution," says Whittington. Publishing last year in *Nature*, she and others used data from the platypus genome to show that some platypus toxins evolved independently from those in snake venom.

Even though platypus venom and snake venom arose separately, the way it happened might have been similar. Many researchers think that the genes for normal, "good" proteins may have been duplicated by accident, leaving the second copy free to encode what turned into a havoc-wreaking venomous molecule. For instance, immune system proteins called defensins, which normally help fight off invading pathogens, were turned into molecules with the ability to slice up "good" proteins in victims (usually other platypuses or dogs), Whittington and her colleagues suggest in their report.

To be king of the hill in any given environment, though, venomous animals are often forced to invest in more than one weapon. "It's like investing money in a business. No one puts all their money in a single option. It's best to diversify," says Juan Calvete, a venom researcher at the Institute of Biomedicine in Valencia, Spain. "It's the same philosophy in nature. A cocktail of toxins is better suited as an arsenal that can be used in quite different environments."

One way proteins diversify is through mutation. Some genes that code for venom proteins mutate faster than genes that code for most other proteins. A report published online June 30 in *BMC Evolutionary Biology* shows how a special mutation process in toxin genes causes some snake venom proteins to change rapidly. Called accelerated segment switch, this process can make a venom toxin recognize a different target, leading to greater variety and utility.

In a study published last year in the *Journal of Proteome Research*, Calvete and colleagues found that venom from *Bothrops asper* pit vipers in Costa Rica

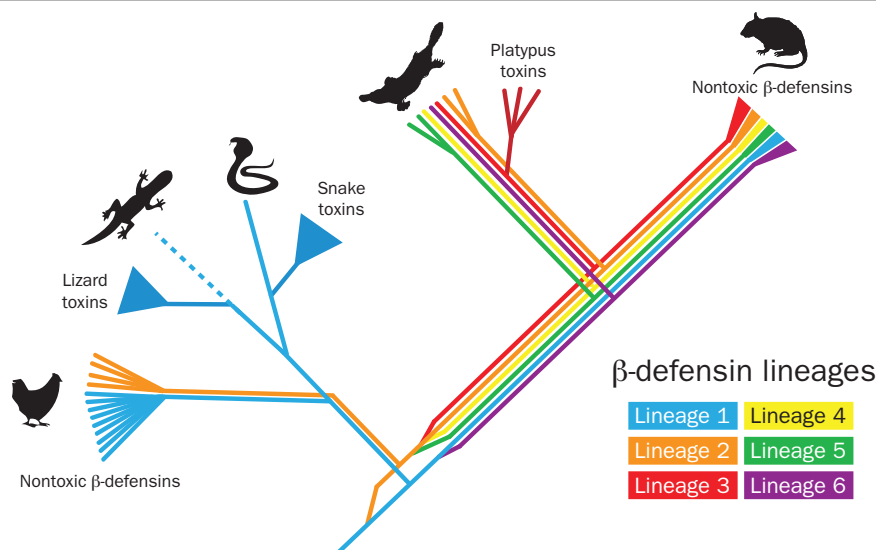
differed depending on the population's geographical location. Snakes that lived on one side of a steep mountain range had markedly different venom profiles from those of snakes on the other side. In the same way a particular Southern twang identifies a Texan, the composition of venom can reveal where a snake hails from, Calvete says.

Repurposing venom

The customized toxins in venom also make up a vast collection of potential weapons against diseases. "Venomous animals have an extraordinarily rich history in this regard," says Fry. "If you know anybody that takes high blood pressure medication, odds are they're taking a class of compounds called ACE inhibitors." The founding member of this class, says Fry, is a modified toxin from a pit viper—"one of the biggest, meanest, most horrible snakes in South America."

Another example comes from the cone snail *Conus magus*. In 2004, ziconotide, a drug based on the snail toxin omega-conopeptide MVIIa, was approved by the U.S. Food and Drug Administration to treat chronic pain. Years earlier, Olivera had given Miljanich cone snail toxins to help with experiments on nerve cell signaling. In the experiments, conducted at the University of Southern California in Los Angeles and later at Neurex Corp. in Menlo Park, Calif., Miljanich and his colleagues recognized that the omega-conopeptide MVIIa toxin blocked a specific protein crucial for moving pain signals through the spinal cord to the brain. Interfering with this protein, called the N-type calcium channel, offered a way to stop some kinds of pain better than even morphine.

"We've taken advantage of 50 million years of evolution of those N-channel toxins," says Miljanich, now the CEO of the pharmaceutical company Airmid Inc. in Redwood City, Calif. Miljanich and his team at Airmid are currently working with a sea anemone toxin that has potential as a therapy for autoimmune diseases such as multiple sclerosis, psoriasis and type 1 diabetes. This toxin, he says, appears to halt rogue immune cells



From defense to attack molecules

Some proteins in venomous creatures evolved into toxins when certain genes were duplicated. A family of molecules called beta-defensins—immune system heroes that help fight off disease-causing invaders by chopping up proteins—were repurposed as toxins in lizards and snakes and, independently, in platypuses. Nontoxic beta-defensins are common in chickens and mammals, including the platypus. This tree traces the various lineages of defensin molecules that led to the platypus toxin (dark red) as well as the lizard and snake defensin-derived toxins (blue) that probably share a common ancestor.

that are attacking the body's own tissue. The team is tweaking the toxin by adding or removing chemical groups to make the molecule more stable and effective.

A growing number of researchers are exploring the wealth of molecular resources venoms offer. "We don't want to leave any potential source [of medicines] off our radar," Miljanich says.

Beyond treating medical conditions, venom toxins may offer clues to deeper mysteries about the body and brain. "Venom has turned out to be very useful in telling us what's important about how the nervous system works," says Andres Villu Maricq, a neurobiologist and geneticist at the University of Utah.

While screening dozens of toxins from the fish-hunting cone snail *Conus striatus*, Stori Jensen, a student in Maricq's laboratory, hit upon one that inhibited a brain process called desensitization, which alters brain cell activity by dampening nerve cell cross talk. The toxin, the researchers found, clamps open a pore that is usually shut in the desensitized brain, making the cell respond to certain signals from other cells it normally would ignore.

Understanding how brain cells com-

municate and having a precise way to interrupt some of those messages may offer new ways to look at neurological conditions like Alzheimer's disease in the laboratory, says Maricq. "There were really no fresh approaches."

In the wild, *C. striatus* venom causes fish to spin around, as if chasing their tails, although Maricq says he doesn't yet know exactly why. The team, which included Olivera, named this new toxin con-ikot-ikot, which means "spinning" in Filipino, and published the results June 9 in *Current Biology*.

Olivera and other toxin hunters aim to identify more such molecules and figure out how they work. This is the next great challenge for his research, he says. "What we would like to do is be able to explore the whole biodiversity of venomous snails," says Olivera. "This opens up the possibility of a huge group of compounds that could be interesting. In my case, we've suddenly realized that looking at cone snails, what we've been looking at is only scratching the surface." ■

Explore more

- Read about the cone snail venomics CONCO project at www.conco.eu/



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Astronomers hope type Ia supernovas will help in quest to explain dark energy

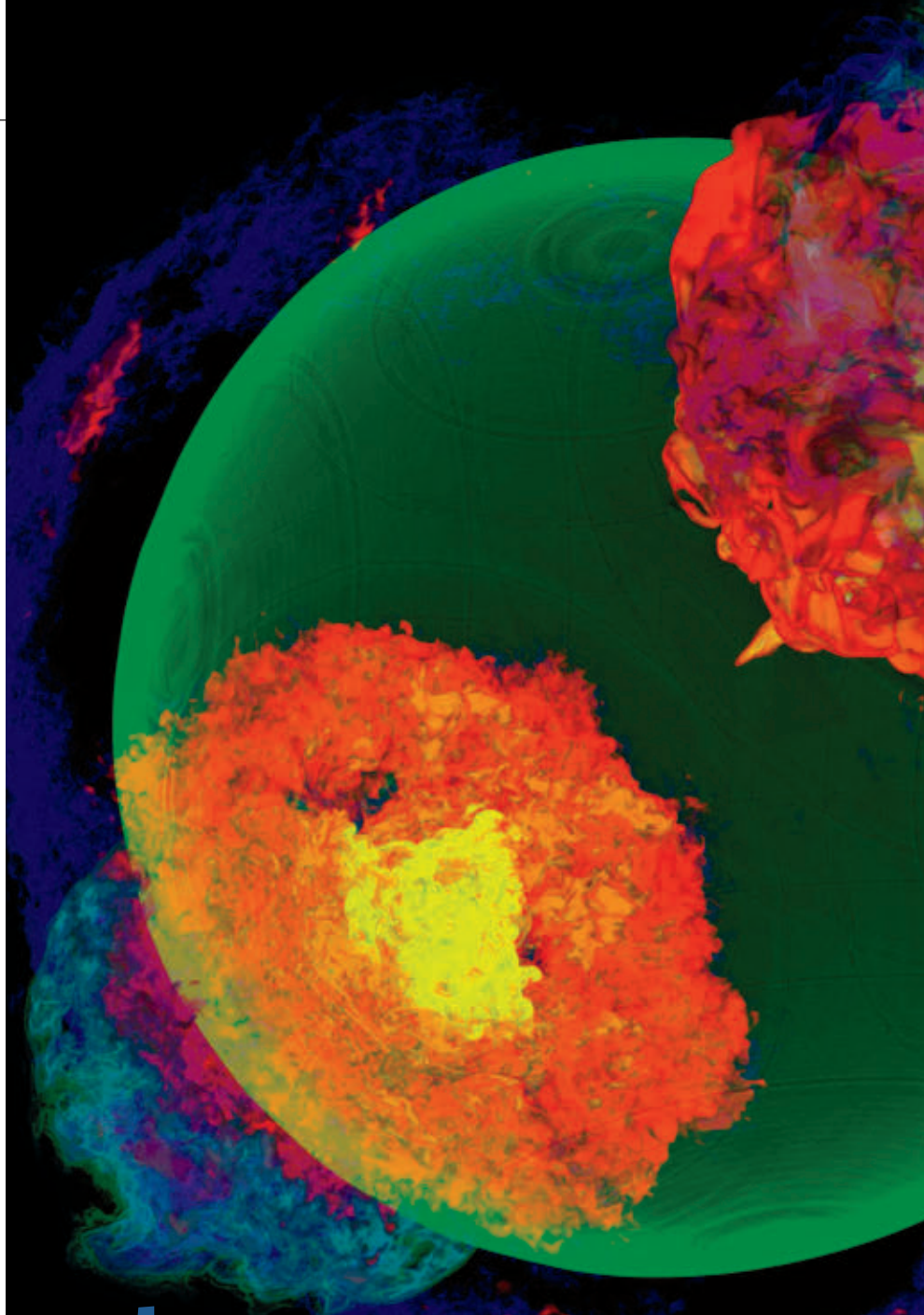
By Ron Cowen

At least once a second, a dim, elderly star somewhere in the cosmos turns into a thermonuclear bomb. Briefly outshining its home galaxy, the explosion, known as a type Ia supernova, unleashes the equivalent of 10^{28} megatons of TNT—enough energy to destroy an entire solar system.

Astronomers have marveled at these cosmic firecrackers for centuries. But so far nobody has explained in detail how these supernovas explode. Now, theorists are on the verge of attaining that understanding—and just in time, because astronomers are observing type Ia supernovas with a new urgency. In fact, the story these stars have to tell is a matter of cosmic life and death.

When astronomer Robert Kirshner, now at Harvard University, first began observing these cataclysmic explosions in 1972, it didn't matter that no one understood how they happen. A lack of knowledge about the explosion process didn't stop Kirshner and his colleagues, along with another team, from using type Ia supernovas to discover in 1998 that a mysterious entity, later dubbed dark energy, is accelerating the expansion of the universe (*SN: 2/2/08, p. 74*). But today, ignorance about type Ia supernovas is no longer bliss, say Kirshner and other astronomers. Researchers now are not only relying on supernovas as distance markers to deduce the presence of dark energy, but also to unveil its character.

One of the deepest mysteries in all of physics and astronomy, the nature of dark energy determines the fate of the universe. If its density across the universe increases over time, the cosmos will end in a Big Rip, with every atom torn asunder. If it somehow vanishes, cosmic expansion



stars go
KABO
spill
cosmic



OM ing secrets

An inwardly directed jet produced by the collision of hot ash along the surface of a white dwarf star penetrates the star and triggers detonation in this simulation. Green indicates the star surface, and yellow shows the hottest temperature.

will continue but at a slower rate. And if its strength remains fixed in time, akin to the cosmological constant that Einstein inserted into his equations of general relativity, every galaxy will someday become its own island universe.

To determine whether dark energy varies or remains the same throughout time, astronomers need to measure its equation of state, defined as the ratio of its density to its pressure. And to measure the equation of state at different epochs in the universe, researchers urgently need more detailed information on type Ia supernovas, says Don Lamb of the University of Chicago.

Theorists are beginning to crack the riddle of supernova explosions by borrowing some of the techniques — and computer codes — applied to a surprisingly down-to-Earth system: combustion in gasoline engines. Thanks to these codes, which require the processing power of supercomputers, researchers can now view the full three-dimensional evolution of a stellar explosion instead of a muted, one-dimensional facsimile.

On the computer screen, “it’s like watching a fire consume a forest, you just see these flames working through the star, with all this structure to it,” says theoretical astrophysicist Daniel Kasen of the University of California, Santa Cruz.

Simulations developed by supernova expert Stan Woosley, also of UC Santa Cruz, along with Kasen, Fritz Röpke of the Max Planck Institute for Astrophysics in Garching, Germany, and others now suggest that supernovas that erupted a few billion years back in time may be different — intrinsically brighter — than those exploding today. The team has begun to identify several other features that may affect supernova brightness — such as how rapidly a star rotated before it exploded and its abundance of elements heavier than helium — which might confound dark energy measurements if overlooked.

“We’re starting to make meaningful

comments about how useful these supernovas can be for precision cosmology,” Woosley says.

Exploding stellar probes

Astronomers rely on type Ia supernovas to probe the expansion history of the universe because these explosions are almost perfect cosmic mile markers.

Since all Ia’s appear to have the same starting point — blowing up the same amount of mass — they all should have roughly the same luminosity. After adjusting for variations by applying the Phillips relation, which holds that intrinsically brighter supernovas take more time to fade than dimmer ones, researchers can, in principle, read off the wattage of these cosmic lightbulbs. Just as the apparent brightness of a 60-watt bulb predictably diminishes with distance, so too should the observed brightness of a supernova.

When astronomers applied this prescription, they found that light from distant supernovas appeared dimmer than it ought to be based on what had been the accepted model of the universe’s evolution. That unexpected result led in 1998 to an astonishing conclusion: Rather than slowing down, the cosmos has recently sped up its rate of expansion, putting extra distance between nearby and remote supernovas — and the galaxies in which they originated.

Now, astronomers want to know the inherent brightness of type Ia supernovas to within a few percent, rather than the previous error margin of 20 percent — and how that brightness varies among different populations. Suppose, for example, that supernovas containing a lower abundance of heavy elements — typical of stars earlier in the history of the universe — are on average intrinsically brighter than supernovas exploding today. The Phillips relation says that the supernovas with fewer metals should remain bright for a longer period of time than others. Indeed, models suggest that such cosmic

bulbs would last longer than younger supernovas, but not quite as long as the relation predicts, Woosley and collaborators now find. This effect cannot be ignored if researchers want to use type Ia's to measure distances to an accuracy of 1 or 2 percent, which will be required to assess whether or not dark energy varies with time, Kasen says.

If type Ia supernovas vary in brightness according to a random statistical distribution, with some explosions brighter and some dimmer than average, simply observing many more of them will beat down the error in using them as standard lightbulbs, Kasen says. But if some type Ia's, such as distant ones, are systematically different from others, as his team now suggests, a problem emerges.

If such properties aren't accounted for, "our errors would be greater than we really believe" in using type Ia supernovas to measure the expansion of the universe and the nature of dark energy, says Mike Zingale of Stony Brook University in New York.

Road to kaboom

Most astronomers agree that a type Ia supernova starts with a white dwarf—an aging star that crams as much mass as the sun into a volume no bigger than Earth. Most white dwarfs are cold and inert. But if the star has a companion, it will siphon

mass off the neighbor star until tipping the scales at about 1.4 solar masses. At that mass, the white dwarf becomes dense and hot enough to initiate an explosion.

No one really knows the nature of the partner stars, exactly how or where the initial nuclear flame is sparked, or how a relatively slow flame transitions into an inferno that races through the star at supersonic speeds. Because white dwarfs are so dim, astronomers have never even seen one right before it blows up as a supernova.

But based on the information they do have, theorists have developed dazzling if complex computer models to mimic and learn about these stellar bombs. Watching flames racing across the screen, it's easy to lose sight of one of the most important properties that researchers are now trying to pin down. A single number, the amount of nickel-56 forged at the core of the exploding star by the fusing together of lighter nuclei, determines a type Ia's luminosity.

Although the explosion itself lasts for only a few seconds, the slow radioactive decay of nickel-56, which generates photons that diffuse out of the exploded star's core and heat the outlying shrapnel, causes supernovas to glow brightly and linger in the sky for months. It's Kasen's task, in the UC Santa Cruz group, to determine if models reproduce the

observed amounts of nickel-56, how long photons would take to travel through the supernova debris and how bright the simulated supernova would appear to observers on Earth.

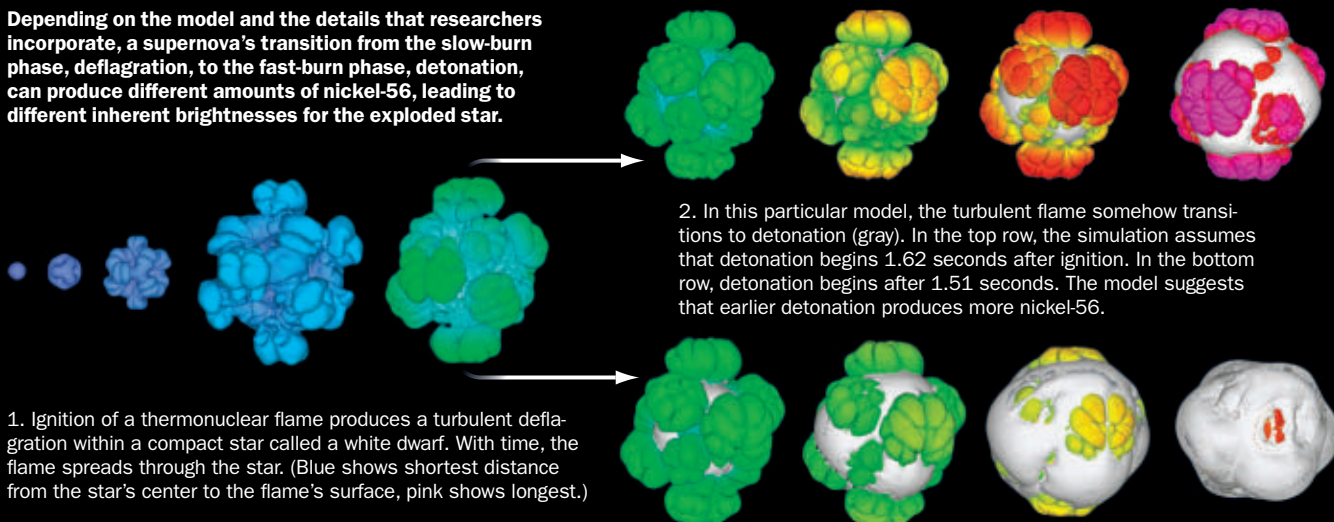
Different supercomputer models have to handle different aspects of a mind-boggling array of distance scales—from less than a millimeter to 2,000 kilometers. Also, the approximations embraced by physicists and astronomers for other computational problems do not apply to supernovas, which are highly asymmetrical, involve complex, turbulent flows, and explode under conditions of high density and extreme gravity.

"We've been learning a lot from the people who study combustion," Woosley says. Internal combustion engines exhibit two types of burning that supernovas, at least in theory, also exhibit. A car engine normally operates at a slow burn, with the flame ignited by the compression of gasoline and oxygen traveling at speeds considerably slower than the speed of sound through the fluid. That sluggish burning is known as deflagration. In car engines that knock, the flame travels supersonically, a burning known as detonation.

In exploding stars, models in which a thermonuclear flame travels exclusively at subsonic speeds produce a much dimmer explosion than telescopes have recorded. Also, such models leave too

Possible explosive paths

Depending on the model and the details that researchers incorporate, a supernova's transition from the slow-burn phase, deflagration, to the fast-burn phase, detonation, can produce different amounts of nickel-56, leading to different inherent brightnesses for the exploded star.



BELOW: V. GAMEZO, A. KHOKHLOV, E. ORAN, ASTROPHYSICAL JOURNAL 2005; FACING PAGE: A. RIESS/STSCI, NASA

much carbon and oxygen unburned. At the other extreme, simulations in which a flame travels only at supersonic speeds burn the white dwarf's material so rapidly and thoroughly that all the lighter-weight elements are squeezed together. This squeezing forges the heaviest elements a supernova can make in abundance — nickel, cobalt and iron. But that also doesn't match observations, which reveal intermediate-weight elements including magnesium, calcium and silicon in the supernova debris.

In the early '90s, Woosley and Alexei Khokhlov, now at the University of Chicago, independently proposed that a hybrid model, in which a supernova begins as a deflagration and transitions to the more rapid detonation, might be the most likely scenario. The original simulation, however, was only in one dimension, limiting its usefulness. In 2003, a team led by Elaine Oran and Vadim Gamezo, both of the Naval Research Laboratory in Washington, D.C., showed that the hybrid model, when extended to three dimensions, did indeed match observations. But the underlying physics that would cause a transition from deflagration to detonation remains unclear.

"It looks promising, but no one is there yet," Woosley says.

Determining how fast a thermonuclear flame moves and where it starts is critical, says Woosley. His team's most recent studies show that these properties affect how much nickel-56 will be produced and how bright a supernova can become.

For instance, Röpke now finds that if the flame originates as a slightly off-center deflagration, just 20 to 80 kilometers from the core, the star doesn't "puff up" as much in response to the slow-moving burning front. Then, when the burning switches to a detonation, the higher density of the exploding star makes it easier to fuse lighter nuclei into heavier ones to produce nickel-56.

Differences in the location of ignition,

which may vary from one white dwarf to another and result in a lopsided explosion, "may be the critical factor" for accounting for the diversity of type Ia supernovas, and why they don't all have exactly the same brightness, Kasen says. Because the central regions of the stars are so turbulent before they explode, "we

don't expect ignition to originate in the same way in every supernova," Kasen, Röpke and Woosley report their findings online at arXiv.org and in an upcoming *Nature*.

Building on previous studies, the team also finds that small deficits in a white dwarf's metal content — in this case a lack of elements heavier than oxygen and carbon — can generate slightly brighter supernovas by generat-

ing more nickel-56. In a few cases, the model created some supernovas that were as much as 10 percent more luminous than others, Kasen says. That's important because white dwarfs with few metals tend to hail from remote reaches of the universe, seen as they appeared farther back in time, before stars had a chance to produce an abundance of heavy elements. So type Ia's from more distant reaches of the universe might be systematically brighter than the nearby explosions. Observing more supernovas won't address these differences; it will only eliminate the statistical ones.

"We're at a point now where we can vary the properties of the white dwarf and get a sense of what the systematic errors [in brightness] might be," Kasen adds.

From slow to fast

Although theorists have made progress in simulating the two-step burning process, they're still debating how a slow-burn becomes a detonation. How this happens could have consequences for nickel-56 production, and ultimately how bright a Ia supernova can become.

In Woosley's view, the flame acts as a barrier, keeping apart hot ash and the cold, unburned carbon and oxygen fuel.

Late in the explosion, as the white dwarf expands and densities within the star become low enough, turbulent gases rip through the flame and quench it, allowing the hot ash and cold fuel to mix.

If the ash and fuel remain well mixed, they can combine into a large volume of material that ignites all at once, triggering a high-speed burning front, or detonation, says Woosley. "We don't completely understand the physics of [the transition] yet, but we understand the density when it would happen."

In another model, developed by Lamb and his colleagues at the University of Chicago's FLASH center in 2004, a series of ignition points within a white dwarf meld into a hot, burning bubble that rises rapidly, breaks through the surface of the star and spreads quickly across the surface. Waves of ash sloshing around the surface in opposite directions collide at high temperatures, creating a set of outwardly and inwardly directed jets. The inward jets penetrate the star's interior, generating temperatures and densities high enough to initiate a detonation.

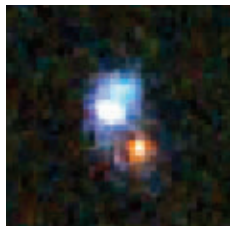
In an updated version of the FLASH model, reported last year, researchers demonstrated that the simulation produces a range of nickel-56 abundances that could explain observed variations in supernova brightness, Lamb says. In an upcoming *Astrophysical Journal*, the researchers show that the detonations can naturally occur in their three-dimensional models. In past versions, the detonation had to be added to the model.

At the Naval Research Laboratory, Oran and Gamezo are exploring how turbulent gases in a white dwarf might generate shock waves that force the transition. They expect to unveil a new simulation in a few months.

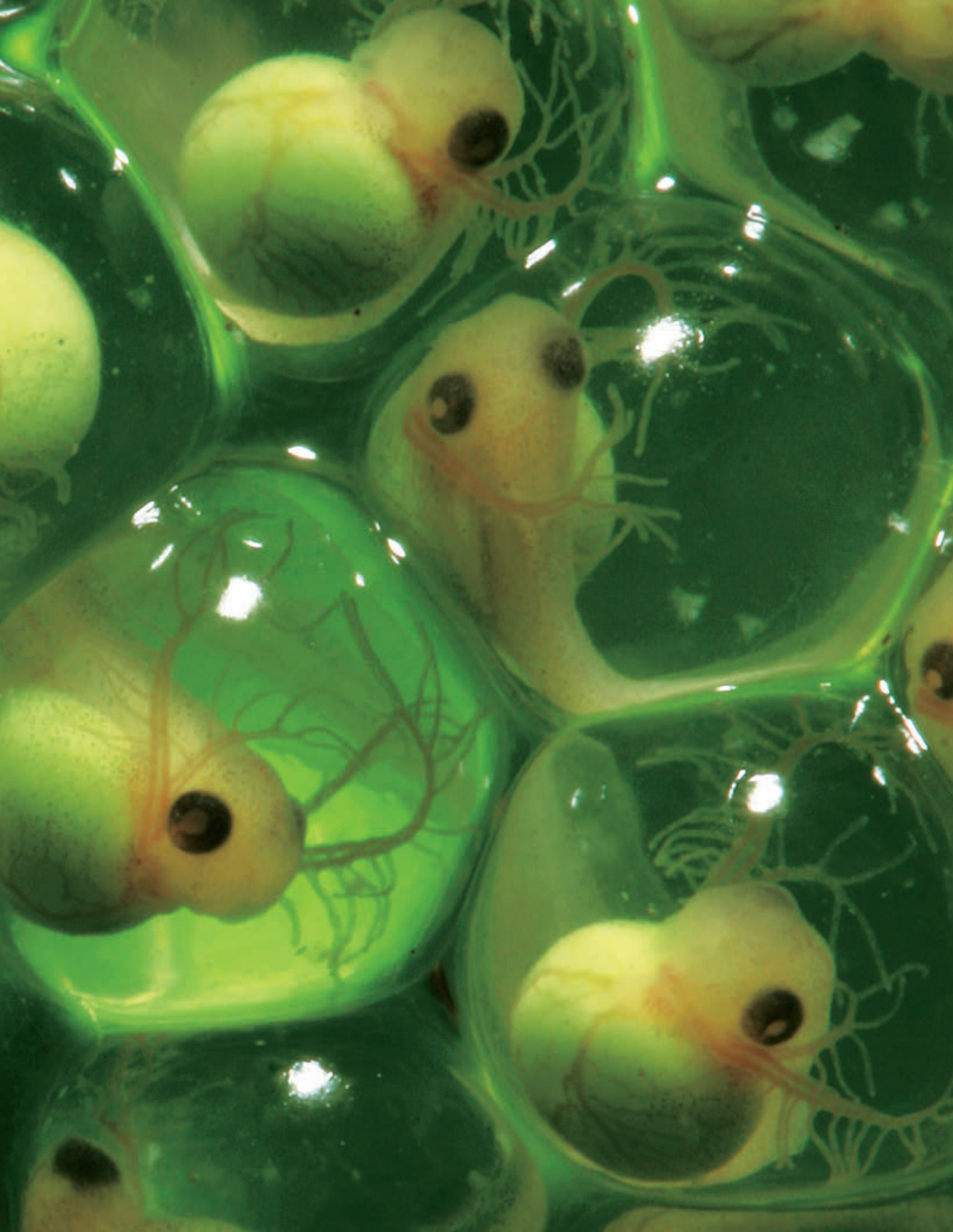
"We're [all] getting at the physical underpinnings of supernovas," says Kasen. Researchers are hoping that those details will prove to be a giant step forward in unmasking dark energy. ■

Explore more

■ NASA's multimedia presentation on dark energy: hubblesite.org/hubble_discoveries/dark_energy



This portrait shows a distant type Ia supernova (red). These explosions are used to measure cosmic expansion.



Smart from the start

Animal embryos get some respect for their survival skills **By Susan Milius**

Karen Warkentin speaks admiringly of the eggs of red-eyed tree frogs because, for one thing, they know what's shaking.

Masses of these glistening eggs hang on leaves that dangle over tropical ponds, and the eggs stay put even when branches thrash in storms. A hungry snake biting into one end of an egg mass can make the embryos' home dip and dance too. But at this jouncing, older embryos flee. They can't run, but they can hatch. A sudden burst of emergency hatching sends a rain of new tadpoles into the water, often saving some 80 percent of a clutch.

Pretty sophisticated for a glob of goo.

It turns out that frog eggs and other embryonic blobs possess a rather advanced repertoire for coping with the earliest stages of life. Embryos can react to perils both inside their eggs and out. Even while still within their shelter, they're learning lessons about eating. And being eaten. These and other recent findings are forcing researchers to expand their ideas of how capably and subtly these half-baked beings can act.

Maybe deft dodges and finely judged trade-offs should be expected: Life for the small and underpowered is dangerous, and plenty of good embryos die young. "That creates opportunities for natural selection to shape a response," says Warkentin, a biologist at Boston University. But there's still the lingering old view of early life stages as clods.

Three-day-old embryos of red-eyed tree frogs position their big, branching gills near the oxygen-rich egg surface.

Warkentin says: "We think, 'It's a frog egg, it's a snail embryo — what can it do?' That's what surprises us."

Even from inside its egg, a remarkably young embryo can do a thing or two to get what it needs.

Among red-eyed tree frogs, *Agalychnis callidryas*, embryos develop big feathery gills for extracting oxygen from the watery world inside their eggs. But just where an embryo's head-to-belly lies within its egg makes a difference in oxygen availability. The part of an egg closest to the air typically carries twice the oxygen concentration of the deep interior squeezed among neighboring eggs, Warkentin and her student Jessica Rogge found. When Rogge prodded embryos so their gills fell into the low-oxygen zone, most of the 3-day-olds twitched themselves back into place within 15 seconds.

Rogge even tested embryos just a day old. "They don't have blood. They don't have a heart," Warkentin says. They move only by beating a fuzz of tiny hair-like projections. Yet when Rogge nudged embryo front ends toward the downside of the oxygen gradient, more than half of the embryos she tested worked their fuzz to chug at least halfway back to the sweet spot within five minutes, she and Warkentin reported in 2008 in the *Journal of Experimental Biology*.

Red-eyed tree frogs, of course, could just have exceptionally smart babies. But research published last summer suggests pond snails also can fix a bad situation.

Masses of *Helisoma trivolvis* eggs stick to plants or other surfaces in freshwater streams. Like the youngest frog embryos, these snail embryos also depend on hair-

like projections to shift around in the egg. Sometimes the developing snails rotate slowly like little turning planets, and sometimes they wind up for a brisk spin. Researchers know that some of the snails' developing neurons respond to dissolved gases, so Jeffrey Goldberg of the University of Calgary in Canada proposed that the turning movements improve the aeration of fluid inside the eggs. In the *Journal of Experimental Biology* in 2008, he called the idea the "stir-bar hypothesis."

Baby bailouts

What's stirring outside the egg also can provoke embryos to action. Scientists are learning that embryos are pretty savvy when it comes to deciding when the coast is clear for them to hatch — and what to do when it's not.

Warkentin remembers facing a skeptical audience at a scientific meeting in the early 1990s when she was a grad student presenting her idea that some frog eggs panic-hatch to escape attacks — but then face extra perils as puny preemies in the water. Some scientists doubted that the little embryos could hatch fast enough to do themselves any good or that plunging into the water several days early would make much practical difference. Both hypotheses had grounding in the field of behavioral ecology, but the time periods involved were short and, come on, these were just frog eggs.

Her first field experiment still stands out in her memory. "It rained a lot, and frogs were bouncing into the ponds — it was very intense," she says. She let a snake bite a mass of eggs and saw them respond so strongly that she said to her-

self, “OK, I have a dissertation.”

Today, looking for changes in hatching time as a reaction to danger is becoming a cottage industry among biologists, Warkentin says. She and her team have now shown that red-eyed tree frog embryos can shorten their time in the egg by some 30 percent if attacked by snakes, wasps or killer molds. And embryos of at least 17 other species of amphibians can split open their eggs for an early escape in a crisis, according to various studies. So can two species of fish as well as a lizard.

Predator-sensitive hatching has even shown up in an animal where mom looks after the eggs: the spitting spider *Scytodes pallida*. She carries the eggs around in grasping jaw parts but has to put them down if she needs to defend herself by spitting goo at an attacker. Daiqin Li of the National University of Singapore threatened egg-carrying moms with a predatory *Portia labiata* jumping spider or its silk. Egg clutches of these imperiled females hatched sooner than eggs of mothers in safer spots, he reported in 2002 in *Proceedings of the Royal Society B*. But eggs exposed to spider scent without her care didn’t hatch appreciably early. Just what hurry-up message mom sends to the eggs or how she fast-forwards their development is a question for future research.

Staying in the egg’s safe haven can be a good idea too. In 1993, the year after Warkentin started wading around in snaky wetlands to test her idea, Andrew Sih, now at the University of California, Davis, published evidence of the reverse strategy, where embryos delay hatching

in dangerous places.

Water with the mere scent of predatory flatworms convinced streamside salamander embryos to linger in their eggs, Sih and Robert Moore, both then at the University of Kentucky in Lexington, reported in *The American Naturalist*. *Phagocotus gracilis* flatworms feast on the smallest salamander hatchlings, but larger hatchlings usually escape. Staying in the egg longer means growing larger, and salamanders that delayed their debut into the free-swimming world indeed improved their chances of survival.

Other hatching delays are turning up too. Embryos of two frog species just stay in the egg extra-long and grow to maximum size when leeches or egg-gulping fish are about.

Species-to-species differences in what embryos can do pose rich evolutionary puzzles. In 2008, Warkentin and her colleagues reported in *Ecological Monographs* on tests of crisis-hatching in relatives of the talented red-eyed tree frogs. As long as embryos had reached a minimum age, all the *Agalychnis* tree frog species tested hatched in a hurry if dunked in water. If they didn’t hatch, they would have drowned in their eggs, despite their large gills.

Even though these frog embryos can burst out of their eggs in the face of one menace, some species just sit there and perish when confronted by another danger, the researchers found. Embryos of one frog species that lives in the same snake-infested forests where Warkentin studied the red-eyed tree frog just lie on their leaf as a snake picks off mouthful

after mouthful. This snake-bait species coats its eggs in a tougher covering than the jiggly eggs of the red-eyed tree frog. So maybe warning vibes don’t travel as well through the tougher eggs, or early hatching would pose its own problems. Warkentin says she’s still musing about how to make sense of the evolutionary pattern.

Exactly how snaky shaking warns red-eyed tree embryos still holds some mysteries as well, Warkentin and her colleagues say in the Feb. 15 *Journal of Experimental Biology*. Earlier egg-vibration studies had established that the embryos don’t need scent or sight to tell a snake shake from a storm blast. Embryos distinguished between the two when researchers subjected eggs to vibrations only, based on leaf-movement recordings. The new work, in cooperation with Boston University’s mechanical engineering department, shows that the embryos are more likely to hatch at low-frequency vibrations but that frequency alone won’t distinguish threat from routine. Frequencies from benign and lethal causes overlap broadly, so the embryos must be combining multiple clues to make such clever choices.

Prehatch preschool

For the cleverest choices, embryos actually learn the latest about their environment’s opportunities and dangers. “This may not be how most people think of the learning they did in school,” says Alicia Mathis of Missouri State University in Springfield. She uses a broad definition: “Learning is experiencing something that causes you to change your behavior.”

In this sense, prenatal learning of smells and tastes has been a traditional line of embryo research. Exposing an embryo to a compound, sometimes by giving it to mom, sets up the baby to react to the substance after birth. After researchers added a dash of anise seed oil to food for pregnant dogs (golden retriev-



A female pale spitting spider can’t spit at predators while carrying an egg mass. In danger zones, the eggs hatch earlier than when mom and offspring are safe.

RICHARD SEAMAN

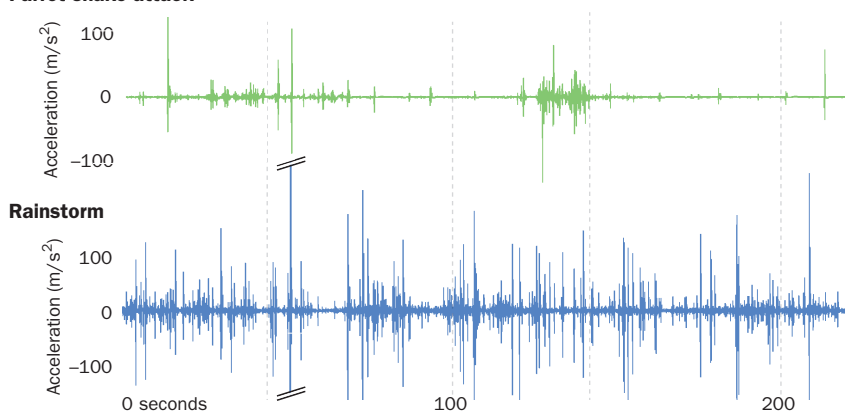


Embryos feel the vibes

Frog embryos know when to cut and run. As a cat-eyed snake bites into fertilized eggs of a red-eyed tree frog in Costa Rica (left), embryos that don't go down the snake's throat early in the attack escape by hatching and dropping off the leaf. In tests of hatching cues, research-

ers mimicked vibrations from a parrot snake attack (below, top) and a rainstorm (below, bottom). Embryos fled from the simulated snake but not from the storm, apparently assessing risk based on a combination of the duration, frequency and spacing of shake-ups.

Parrot snake attack



ers, Labradors and mutts), for example, the newborn pups tended to point their noses toward an anise-scented cotton swab instead of one wetted with plain water. Pups didn't have the same interest in vanilla swabs, showing they had learned to recognize the anise scent before they were born, researchers from Queen's University Belfast in Northern Ireland reported in 2006.

Similar prenatal exposures or lessons work in people, rats, sheep and rabbits as well as in some birds, reptiles, amphibians and even invertebrates.

Embryos can learn visually, too. As cuttlefish develop, the outer egg membrane turns clear enough for the embryos to peek out at the big world around them. To see whether the youngsters were paying attention, Ludovic Dickel at the University of Caen Lower Normandy in France and colleagues set crabs within sight of cuttlefish eggs. After hatching, the youngsters preferred to eat crabs, Dickel and his colleagues reported last summer in *Animal Behaviour*. Without that embryonic view of food, newly hatched cuttlefish preferred shrimp to crab.

One lesson from cuttlefish and other reports of prenatal learning is a new caution in describing animal behavior, says Mathieu Guibé, who works on the Dickel lab's cuttlefish project. "Now that we

know embryos can learn, scientists must avoid interpreting a behavior shown at birth as something innate," he says.

After birth, plenty of creatures can manage the fancier task of associative learning, like Pavlov's dogs drooling for food at the mere sound of a bell. To see if embryos can learn this way too, Mathis scared wood frog eggs. Research on stressful stimuli typically focuses on negative after-effects, she says, but "negative is not always bad." Perhaps scary experiences would teach an embryo some valuable lessons about danger.

Mathis, Douglas Chivers of the University of Saskatchewan in Canada, and colleagues let wood frog eggs get a good long taste of water dosed with a slurry of freshly ground-up wood frog tadpoles. To a little embryo on the brink of tadpolehood itself, this could be scary, and the researchers combined it with water from tanks of fire-belly salamanders. In nature, the salamanders don't live around wood frogs, so the scent by itself should have been ecologically irrelevant.

After the eggs hatched, though, tadpoles that had been exposed to a double-scent cocktail reacted to salamander scent alone as a threat, Mathis reported last year in *Proceedings of the Royal Society B*. Untraumatized tadpoles didn't.

In a twist on that lesson, Chivers and

Maud Ferrari, also of the University of Saskatchewan, exposed wood frog embryos to salamander scent, with no dead-tadpole spicing this time. Once the little ones hatched, the researchers gave them the full cocktail of ground-up tadpole plus salamander. In theory, such an alarming brew could give youngsters a long-term horror of the salamanders. Yet tadpoles that had been exposed to salamander scent as embryos did not develop a phobia after this treatment. Tadpoles without the early lesson did. An embryonic experience of harmlessness can be as important as a lesson in danger, the researchers say in the April 23 *Biology Letters*.

Learning when not to panic also avoids unnecessary costs. When Warkentin's frog embryos escaped a snake attack, they faced consequences. They didn't become eggs for breakfast, but they were undersized and underpowered as tadpoles darting away from deadly fish or shrimp. And in wood frogs, youngsters that learn fear don't forage as freely or grow as fast. Trade-offs, trade-offs. Not a bad life lesson for smart embryos of any species. ■

Explore more

■ K.M. Warkentin. "Oxygen, gills, and embryo behavior: mechanisms of adaptive plasticity in hatching." *Comparative Biochemistry and Physiology* 2007.

Making tall or short of it

In your article “The genetic dimension of height and health” (*SN*: 5/9/09, p. 22), some medical consequences of being either taller or shorter than the median height of the study group are explained. To help us all extrapolate these findings to our own lives, don’t you think it would have been helpful to state what the average heights for men and women are for the general population?

Candy Shedden, Boca Raton, Fla.

Including information about average height was considered, but after trying and failing to find a straightforward way to do it, we decided to leave it out. The “average height” for each study and each type of disease mentioned was different, since each study looked at distinct populations and used different methods. So, comparing your height with the average wouldn’t tell you much about your disease risk. But, to answer your question, the U.S. Centers for Disease Control and Prevention’s data from 1999–2002 show the average height for U.S. men as 5 feet, 9.3 inches and for women as 5 feet, 3.8 inches.

—Solmaz Barazesh

Better controlling controls

A graph provided with the article “Think like a scientist” (*SN*: 6/20/09, p. 20) presents data about the understanding of control of variables by students from two different groups, those who received explicit instruction about the concept and those who were left to learn by an exploratory approach. However, the graph also appears to show that the students in the direct instruction group had a much higher mean score on prior knowledge of the concept. Especially for research about learning the concept of control of variables, I am surprised that better-matched test groups were not chosen for this research.

Greg Skala, Nanaimo, Canada

The difference between average scores of the two groups of children on a written pretest that’s apparent in the graph was not statistically significant. The statis-

tically significant difference between groups on control-of-variables knowledge emerged on a test administered shortly after each child received either explicit or exploratory instruction.

—Bruce Bower

Intentionally or not, the article “Think like a scientist” clearly illustrated the problem of teaching scientific reasoning. The statement about the control-of-variables strategy, “Researchers hold constant all changeable features in an experiment except for one of interest,” does not make sense to a young person. While some engineers and medical researchers do single factor experiments, a single factor experiment is only marginally better than a no factor experiment. The issue is to model a process and, as noted in the article, the concept of modeling (using math and experiments) is critical for the understanding needed to make predictions.

When teaching people, trying to present information and methods that, on the surface, contradict observation will not be successful, unless one clearly notes how the experimental model is correlated to reality. And reality involves more than one variable and lots of interactions. I would suspect the results in the “discovery” versus “direct” instruction groups had more to do with the presentation and reasoning ability of the teacher than the characteristics of either method. My personal opinion is that some of both are required to engage natural curiosity.

David Sweetman, Dyer, Nev.

More astronomy cover coverage

Readers of *Science News* are certainly a tough bunch — and evidently lovers of the perennial activity “what’s wrong with this picture?” The recent letters (“Astronomical art faux pas,” *SN*: 7/4/09, p. 30) taking umbrage at the “errors” on the May 23 cover might have noted, as was clear in the credits, that it is from a 1925 French publication for children, *Le Petit Inventeur*. Right or wrong is not the issue; the joy and romance of observing the heavens is — whether camper,

wizard, meridian circle professional or, evidently, a French civil servant at the Paris Observatory. My only quibble is that the address label obscured the fourth quadrant. I’d love to know what lurks beneath.

David DeVorkin, Washington, D.C.

DeVorkin is the senior curator of astronomy and the space sciences at the Smithsonian Institution’s National Air and Space Museum.

Speedy musings

Body-lengths per second as a measure of (relative) speed (as in “Hummingbird pulls Top Gun stunt,” *SN*: 7/4/09, p. 7) offers more entertainment than the incessant “miles per hour” reports about cheetahs, peregrine falcons, swordfish and such.

Decades of close observation have convinced me not only that spiders and insects hold all the body-length per second records, but also that many event categories must be established to make sense of their athletic skills. A leafhopper, for instance, may spring several hundred times its own body length in one second. However, like a flea, it doesn’t spring toward a target, and it may land on its head as well as its feet. This is “dumb-jumping.” A jumping spider, on the other hand, knows where it’s going. A zebra jumper one-eighth an inch long can cover 2.5 feet per second in a series of five jumps and hit its target every time. Water skaters introduce a new venue for competition: surface tension. Ponderosa pine bark beetle grubs are the heavyweight bite-pressure champions. Parnid beetle grubs can hold on to a relatively heavier rock than can an abalone.

Why do the smaller creatures do better, and what are the limits of this advantage?

Karl Staubach, Benicia, Calif.

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The Root of Thought: Unlocking Glia—the Brain Cell That Will Help Us Sharpen Our Wits, Heal Injury, and Treat Brain Disease

Andrew Koob

It is famously — and incorrectly — said that humans use only 10 percent of their brains. The claim stems from the observation that only about 10 percent of brain cells are neurons, electrically active cells thought to carry and store information. This book celebrates the other 90 percent, cells known as glia.

Once thought to merely hold the brain together, glia are now recognized as an important support system for neurons. But Koob, a neuroscientist, points out that glia are not just bit players. He glorifies them, especially star-shaped cells known as astrocytes, as

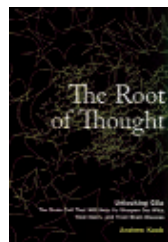
the real movers and shakers of thought and intelligence.

Koob shows disdain for neurons and the scientists who relegate glia to the background by advancing the theory that neurons alone are responsible for information transport and storage.

Astrocytes control neurons, not vice versa, Koob argues. He cites a wealth of studies to support this idea, but he also extrapolates beyond the findings, implicating glia in everything from dreaming to disease. According to Koob, Einstein's genius stemmed from an abundance of astrocytes populating brain areas involved in math and language.

Glia have certainly gotten short shrift for much of brain science's history, but it remains to be seen whether, as Koob claims, astrocytes are the true “nerve centers” and neurons only highways connecting the hubs of astrocyte activity. — *Tina Hesman Saey*

FT Press Science, 2009, 192 p., \$24.99.



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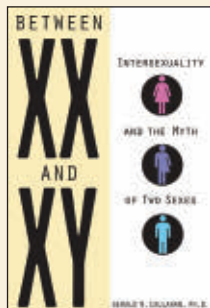
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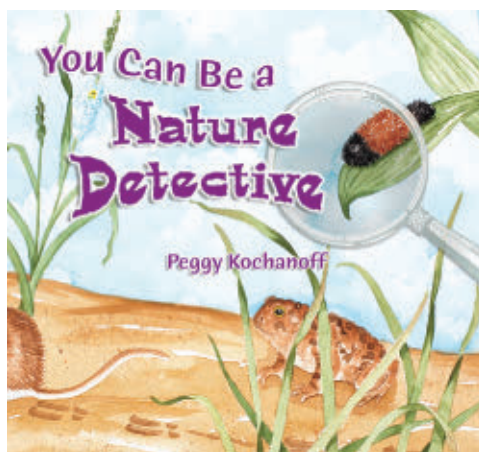
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Linda S. Birnbaum


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Tackling toxicology and environmental health

In January, toxicologist Linda S. Birnbaum became director of the National Institute of Environmental Health Sciences, home to the National Toxicology Program, in Research Triangle Park, N.C. Birnbaum recently spoke with Science News writer Rachel Ehrenberg.

What areas would you like to see the institute zoom in on?

One of the things I've been really working on is to increase our interaction with various federal partners as well as trying to involve the larger community in our actions and our activities. Scientists need to do a better job of helping the general public understand what we do, why it is important and what it means to them. Many scientists take the attitude that what they do is too complex, and in fact, my response to that is, "Then you don't really know what you are doing." So I think that we need to meet with our constituents, understand what their concerns are, listen to them, learn from them and then help them to understand what our findings mean. The dialog has to be a two-way street.

In terms of the scientific things, we need to focus on complex diseases — diabetes, heart disease, cancer, autism and ADHD. There appears to be a genetic component to a lot of these but there is a gene-environment interaction as well. What are the populations that are most susceptible? Is it the very young? Very elderly? We are past the one-gene, one-disease kind of paradigm. We need to think about testing smarter, testing differently, taking a more systems approach.

I am also very interested in the issue of differential susceptibility and in long-term effects of early exposures. I think we're understanding more and more and more that things that happen to you in utero or as a young child or even during puberty can ... come back

to haunt you 40 and 50 years later, and I think we need to be spending more attention on that.

I'm interested in what some people call low-dose exposures ... exposures that result in levels in our bodies that have some relevance to the real world. There has been a lot of criticism of a lot animal studies that they are done at very high doses. And in many cases, if you actually look at the internal dose in the animal — the blood level or the tissue concentration — what you find is it is not so very high. If it is exceedingly high there may be very little relevance to what's going on, but in many cases it is not that high compared to at least some people in our population.

Some have argued that you can't draw conclusions about the health effects of a chemical such as bisphenol A because studies examining BPA vary so much. Do we need guidelines for designing such studies?

I am not a fan of strict adherence to guideline studies. I think that it's a turn-the-crank mentality. For example, GLP [Good Laboratory Practice, federal regulations designed to ensure quality in lab studies] doesn't guarantee that you had a good study — it guarantees that there was good record keeping in the study ... the i's were dotted and t's were crossed and things weren't removed from the file, but it doesn't mean that the right question was being asked. I think that the guidelines should just be that — guidelines.

Many of the guidelines that we're using today were developed 20 and 30

years ago. We know a lot more. We have additional or different concerns. We need to be asking the right scientific questions. You know the saying, if your keys aren't under the light of the lamp-post and you only look under the light, you are never going to find them. Well,

it is the same thing in science: If you don't ask the right questions, you are not going to find an answer.

For example, does long-term adult exposure to a chemical cause cancer in rodents? Is that the question we really want the answer to? I would probably say no, we really want to know what's happening in people, but we want to know what's happening to susceptible people or people who are exposed at a susceptible period of time. So I think the problem is the guidelines studies were developed to answer a particular set of questions and we've moved beyond that set.

I look at the whole weight of evidence. If I see hundreds of studies showing effects in a couple of different species and I see a whole plethora of responses — that raises my level of concern. If I see that a given chemical causes one kind of effect in say, male rats only, in one tissue, I'm not as concerned as if I see a chemical that is appearing to affect lots of different kinds of tissues in different developmental or adult stages in a couple of different species. Then I begin to think, hmm, maybe there's some relevance to humans. Because nature is inherently conservative — animals may not be people, but people are animals. ■



If I see hundreds of studies showing effects in ... different species and ... a plethora of responses — that raises my level of concern.



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How Has Christianity Changed over 2,000 Years?

In the first centuries after Christ, there was no “official” New Testament. Instead, early Christians read and fervently followed a wide variety of scriptures—many more than we have today.

Relying on these writings, Christians held beliefs that today would be considered bizarre. Some believed that there were 2, 12, or as many as 30 gods. Some thought that a malicious deity, rather than the true God, created the world. Some maintained that Christ’s death and resurrection had nothing to do with salvation while others insisted that Christ never really died at all.

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"Henry Philip Hope would be impressed with the superfine deep blue of the Stauer *Eternal Hope* Necklace. Ever since the original first surfaced centuries ago, it has been shrouded in legend and mystery."

**— JAMES T. FENT,
Stauer GIA
Graduate Gemologist**

This colossal stone of more than 45 carats is enlarged to show details.

World's Most Famous Diamond Found Inside Toaster

Stauer introduces a 45½ carat, lab-created tribute to the legendary Hope Diamond!

It's a wonder that the *Hope Diamond* survived the 20th century. In the hands of eccentric millionaire heiress Evalyn Walsh McLean, the 45½ carat deep blue diamond was treated more like a fancy plaything than a precious gem. McLean wore it on rollercoasters, while swimming and when doing yard work. She hid it underneath couch cushions and from time to time, even kept it inside her toaster!

These days, the *Hope Diamond* is the superstar of the National Gem Collection at the Smithsonian's Museum of Natural History. And even though the spectacular blue stone attracts millions of visitors a year, it looks lonely stuck behind that 3-inch bulletproof glass. So we decided to set it free.

Science recreates a legend of luxury. We challenged our gemologists to reimagine the largest and most perfect blue diamond in existence. They came back with the magnificent *Eternal Hope Necklace*, a 45½ carat, lab-created beauty inspired by the \$350 million original. Using the legendary *Hope* as their guide, they carefully cut lab-created blue spinel to match the shape, the geometric angles and the exact carat weight of the world famous stone.

The *Hope Diamond* remains one of the world's most unattainable treasures, but we've set the price of our *Eternal Hope Necklace* at an unbelievably accessible \$195!

When being called a "jewel thief" is a badge of honor. The final product is so convincing that it will surely have museum curators double checking their vault. Our update keeps the allure of the original, the magic that captivated Maharajahs, kings and queens.

The custom-cut stone is brilliant cobalt blue lab-created spinel. Spinel is considered the greatest impostors in gemstone history. One of the most valuable gemstones in the world, the *Black Prince* in the British Crown Jewels, was only recently discovered to be a spinel. They are favored by jewelers because of their brilliance and hardness. And the structure of a spinel is similar to a diamond—they both have the same high symmetry (4/m bar 3 2/m).



Don't forget the eye-catching 6 ctw Eternal Hope Ring.

Add the world's most famous rock band. Enhance the look of your stunning Necklace with the *Eternal Hope Ring*, a gorgeous 6-carat version of the pendant stone, prong-set on a .925 sterling silver band. Flanked by 10 dazzling, lab-created DiamondAura®, the Ring perfectly complements the pendant.

Try the *Eternal Hope Necklace* for 30 days. If for any reason you are not satisfied with your purchase, simply return it to us for a full refund of the purchase price. Don't miss this chance to claim your own historic piece of Hope!

JEWELRY SPECS:

- 45½ carat faceted blue spinel center stone
- Stone's dimensions: 21 x 17 x 8½ mm
- DiamondAura® accents
- .925 sterling silver setting, bail and chain
- 18" cable chain with 2" extender
- Ring sizes 5-10

Eternal Hope Necklace

(45½ ctw) MSRP ~~\$1,110~~

Your price \$195 +s&p

Eternal Hope Ring

(6 ctw) MSRP ~~\$820~~

Your price \$145 +s&p

Call now to take advantage of this limited offer.

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