

SCIENCE NEWS

THE WEEKLY NEWSMAGAZINE OF SCIENCE

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feathered filchers
tb in human ancestor
milky way gets new halo
stem cells for diabetes

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plain remains

GRASSLAND RESTORATION
EFFORTS ENCOUNTER HURDLES



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Cover The last remnants of America's once-great grasslands are yielding clues to those working to conserve and restore the prairie ecosystem. Biodiversity appears critical, but figuring out how to re-create and maintain it is proving to be a challenge. (iStockphoto) **Page 376**

THIS WEEK ONLINE

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Food for Thought Youngsters are developing peanut allergies earlier because of exposures in babyhood.

MathTrek A new study raises doubts about fractal patterns in animal behavior.

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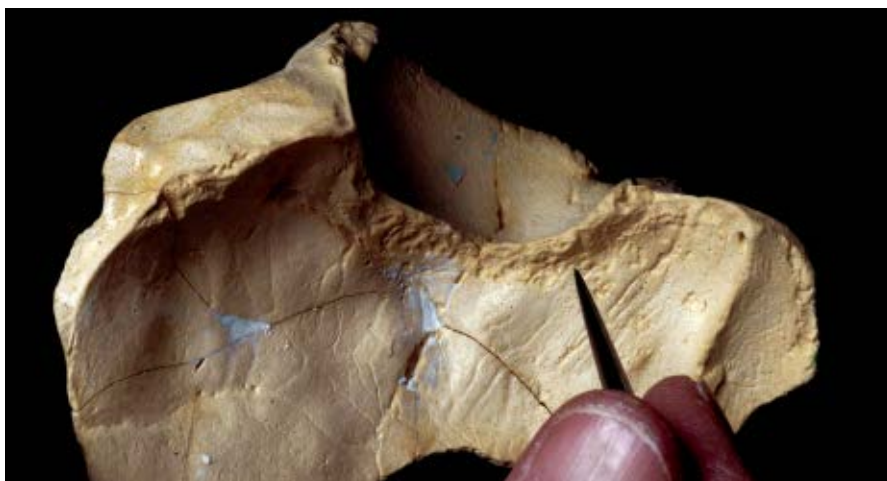
Ancient Ailment?

Early human may have
carried tuberculosis

Check your tile countertop for fossils. A consumptive *Homo erectus*—or at least a piece of him—might be trapped there.

While cutting coveted travertine into tiles, a saw operator in Turkey sliced through a fossilized skull and gave the pieces to his supervisor. The fragments from the 500,000-year-old rock sat on a shelf behind the supervisor's desk until a local geologist visiting the fossil-rich site claimed them.

"The workers didn't know what it was," says John Kappelman of the University of Texas at Austin, who studied the fossil. "The first saw cut took off a bit of the top of the [skull] and the second saw cut went through the middle of the eye orbit."



HOMO TUBERCULOSIS A stippling of tiny pits (pointer) heralds a rare form of tuberculosis in this plaster cast of a skull fragment from a 500,000-year-old *Homo erectus*, claims one anthropologist. If true, it would be by far the oldest case of the disease.

The partial skull is the first *H. erectus* fossil found in Turkey, Kappelman and colleagues report online and in an upcoming *American Journal of Physical Anthropology*.

A wildly successful species that predated modern humans, *H. erectus* walked out of Africa all the way to China, Indonesia, and the Republic of Georgia starting about 2 million years ago, other fossils show. Whether the tall tool users ever arrived in Europe remains controversial, but the new find suggests they at least got close.

Kappelman says the skull's heavy brow ridge and sharply sloped forehead mark it as *H. erectus*.

Moreover, he says the inside of the skull displays telltale signs of tuberculosis, which

in rare cases infects the lining of the brain. If confirmed, the find would push back the origin of the disease in hominins—the anthropological term describing human and near-human predecessors—back hundreds of thousands of years.

Until now, the oldest direct evidence of tuberculosis came from a 5,400-year-old Egyptian mummy. In 2005, genetic analysis of several strains suggested the disease originated about 3 million years ago in East Africa, the cradle of early human evolution.

The Turkish travertine traveler physically buttresses the claims of an early origin of the disease, Kappelman says.

When Kappelman initially examined the fossil, he missed the signs of tuberculosis—

LETTER FROM THE PUBLISHER

Dear Reader of *Science News*:

It gives me great pleasure to introduce you to the new Editor in Chief of *Science News*, Tom Siegfried. As one would expect for the venerable *Science News*, Tom is one of the nation's leading science journalists, with a long track record as both writer and editor. He was the science editor at the *Dallas Morning News*, leading its science section to the top ranks of newspaper science departments. In addition, Tom has written several well-received books, most recently *A Beautiful*

Math: John Nash, Game Theory, and the Modern Quest for a Code of Nature.

The future of our beloved magazine must keep faith with the core mission of *Science News*: to provide concise and credible news across a broad range of scientific disciplines. The historic strength of *Science News* is that faithful readers stay abreast of important developments in science efficiently, and with total

confidence in the source. That will always be true. At the same time, the way people find and use information has

changed dramatically, and *Science News* must change also. Tom Siegfried will be instrumental in helping *Science News* navigate new but exciting waters, delivering the same quality content in formats that promise to reach a broader community.

The most obvious opportunity is the explosion of online sources of science news and information. The public's hunger for developments in science is keener than ever, but many satisfy their hunger online rather than on the printed page. In order to thrive in this environment, every publisher must bring information to its audience in the form the

audience wants, including in print, on the Web, via e-mail, through podcasts, or by other rapidly evolving forms of information delivery. Our challenge is to do that while maintaining the high standards our readers have expected from *Science News* in print for more than 80 years. We feel fortunate to have found a new Editor in Chief who will usher in an era in which we will do just that.

We will look forward, with you, to watching and enjoying as Tom and the inimitable *Science News* staff bring us a version of *Science News* that embodies old virtues for a new time.

— ELIZABETH MARINCOLA,
PUBLISHER, *SCIENCE NEWS*



Tom Siegfried

a stippling of tiny pits around the eye orbit. But when he showed the fossil to paleopathologist Michael Schultz of the Georg-August University in Göttingen, Germany, Schultz recognized the pattern.

It matched what Schultz had seen in the skull of a 19th-century Austrian man who died from tuberculosis of the meninges, the membranes sheathing the brain. When the disease invades this covering, its characteristic tubercles, or grains, press tiny pits into the front of the skull, near the eyes.

"The imagery of that [Austrian] case is an exact match for what we have," says Kappelman.

The diagnosis arrives millennia too late for the adult male *H. erectus*, but it's just in time to set off a scientific controversy.

Two other paleopathologists, Pia Ben- nike of the University of Copenhagen and George Armelagos of Emory University in Atlanta, are skeptical of the claim. They want to see more of the ancient individual—such as his spine—to confirm that he indeed carried tuberculosis.

Kappelman hopes to find more of the early man in the quarry's scrap heap. He might make a few trips to Home Depot too. "Back in the tile section, they have travertine from Turkey," he says. "Honestly, it's a case where the rest of this thing might be in somebody's kitchen." —B. VASTAG

Hatch a Thief

Brains incline birds toward a life of crime

It's not brawn but brains that matter in the rise of crime families among birds.

That's one of the conclusions from a study of traits shared by families of bird species notorious for stealing food, says Julie Morand-Ferron of the University of Québec in Montréal.

The propensity to pilfer turns up in some lineages of birds more than in others, and ideas abound about what bird features typically accompany thievery and might have favored its evolution. Morand-Ferron and her colleagues tested proposed criminal profiles by analyzing 856 reports in the scientific literature of bird-on-bird food snatching.

Having a big body by itself didn't turn out to be strongly associated with thievish families. But having a larger than usual brain for a particular body size did appear to be a suspicious trait, Morand-Ferron



WATCH YOUR LUNCH The golden eagle has a rap sheet of food thefts and belongs to a family with food-stealer traits: large brains for their body size plus a taste for open habitats and vertebrate prey.

and her colleagues report in the December *Animal Behaviour*.

"I was amazed at the amount of data mining that they did to come up with this," says David Shealer of Loras College in Dubuque, Iowa. "It's a study that nobody wants to do, but it needed to be done."

Morand-Ferron says that she started the project after watching Carib grackles in Barbados sneaking dry dog food out of unattended bowls and then deftly snatching pellets from each other. She searched out other accounts of food theft, or kleptoparasitism, and accumulated abundant data even when she limited her search to birds stealing food out of the immediate possession of another bird species. She read about thieves' acrobatic midair grabs, zigzaggy chases, and harassment of a successful hunter until some of the booty was regurgitated for the thief.

Out of the world's 9,672 known bird species, 197, or about 2 percent, have criminal records for swiping food from another species, Morand-Ferron says. Among certain bird families, such as falcons, eagles, and pelicans, kleptoparasites number far more than 2 percent of the species. Others, especially songbird families such as kinglets, count a smaller percentage among their kin.

The researchers took into account biases arising from kleptoparasites' shared lineages or from the fact that some families have been particularly well studied.

Overall, the feathered thieves tend to include other vertebrates in the menu, Morand-Ferron says. A fish or mouse, for example, yields a sizable meal well worth stealing, but requires a considerable effort to chase down.

The species of kleptoparasitic families also tend to live in open habitats such as ocean shores, where they can easily spot their marks.

The results of the analysis make sense, particularly the emphasis on brains over body size, says Shealer, who has studied food theft among roseate terns. He has seen terns rob larger species, and he has seen outright trickery—a female tern that flirted with fish-carrying males only long enough to sidle close to grab their food. So he welcomes new respect for kleptoparasites.

"There's this stigma attached to individuals who steal things to make a living: that they can't catch fish or forage on their own," he says. Among the terns he studied, the thieves "were far and away the best parents." —S. MILIUS

Pulling Together

Mitotic ring self-assembly revealed

During cell division, a ring of proteins forms around a cell's "equator" and then contracts to pinch the cell in half. These proteins arrange themselves into an orderly ring by a random process of searching, grabbing, and pulling each other, scientists have discovered.

Finding this ring-forming mechanism answers a basic question of cell biology from the 1970s, when researchers learned that the ring of proteins contains actin and myosin, the same molecules that generate contractions in muscle cells.

Only recently have modern genetics, cell imaging, and computing techniques made it possible to figure out how the ring assembles itself, says Thomas D. Pollard of Yale University. He and his colleagues proposed mechanisms for how this self-assembly

ISTOCKPHOTO

occurs based on microscope observations of dividing yeast cells. By flagging actin and myosin with fluorescent proteins, the researchers could see the movements of these proteins. They used this information to create simulations of the process. The team then looked at whether the movements of these proteins in real cells matched the behavior produced in the simulations. "The mechanism was tested by computer simulations at every step," Pollard says. "It's actually a little scary how close [the simulation is] to the real thing."

To form the ring, approximately 60 small nodules containing myosin and other proteins first gather at the cell's outer membrane, near the equator. As the time for cell division approaches, each nodule spawns a straight filament of actin, which extends in a random direction by rapidly tacking more units of actin on to its end.

If a filament happens to pass close to another nodule, myosin in that nodule will grab on to the filament and begin pulling, drawing the two nodules closer together.

Surprisingly, myosin in yeast cells spontaneously lets go after about 20 seconds, and the actin-sprouting nodule generates a new filament in another random direction. "It seems counterproductive," Pollard says. "You've got everybody hooked up, so why don't you just go ahead and keep on pulling?"

When the researchers tried a continuous-pulling scenario in their computer simulations, the nodules failed to form an orderly ring. Instead, they gathered into randomly spaced clumps.

Letting go and reaching out in a new direction over and over again allowed the nodules to uniformly draw together into a ring instead of merely huddling with their nearest neighbors, the researchers report online and in an upcoming *Science*.

"It's truly pioneering stuff," comments Alexander Mogilner of the University of California, Davis who has performed research on the self-organization of actin and myosin. "The insight they got is incredibly detailed and vivid." —P. BARRY

Stellar Opposites

Sky survey reveals new halo of stars

The Milky Way galaxy possesses a distinct outer halo that orbits in the opposite direction from its inner halo and the rest of the galaxy, researchers say. This second halo

contains some of the most primitive stars in the universe, offering new evidence about how the galaxy formed.

Some scientists had previously suspected that a portion of the stars in the Milky Way travel in a different direction from the rest. But data on such stars were too sparse to conclude that an entire second halo existed.

Now, an international team of scientists including Timothy Beers of Michigan State University in East Lansing has discovered stronger evidence for a double halo. Beers and his colleagues analyzed more than 20,000 stars as part of the Sloan Digital

Sky Survey, an astronomical effort to create a three-dimensional map of about a million galaxies.

The scientists noticed that stars more than 50,000 light-years away from the center of the Milky Way move in the opposite direction from closer ones, have distinctive chemical compositions, and travel around the galaxy at different speeds.

"It was certainly a surprise to my team how well the two populations were resolved from one another with the new data," says Beers.

Stars in the Milky Way's inner disk, where the Earth is located, orbit at 200 kilometers per second (kps). The inner halo moves in the same direction as this disk, but at 25 kps. Stars in the outer halo appear to speed around the galaxy in the opposite direction at about 50 kps.

Beers observes that the existence of small, metal-poor stars in the outer halo suggests a new story of the evolution of the galaxy.

The dense central region of the galaxy and the inner halo that surrounds it probably formed first, says Beers, as heavy, metal-rich stars clumped together to create the Milky Way. Dwarflike galaxies left behind merged together later to create the outer halo, he hypothesizes.

Though this halo formed after the inner halo, its stars are deficient in heavy metals, implying that they are older. These stars likely formed from gas that existed early in the universe, before all elements existed plentifully, says Beers.

The new findings, which appear in the Dec. 13 *Nature*, do not alter the inference that an invisible form of dark matter occupies the galaxy's halo region, Beers says. The stars in both halos are so far away from the center of the galaxy that their orbital velocity requires more gravity than visible matter supplies.

Rosie Wyse of Johns Hopkins University in Baltimore says the new data provide compelling statistical evidence in support of a distinct outer halo of the Milky Way. But she says questions remain about the nature and formation of the halo.

For example, the Milky Way's central bulge contains stars as old as those identified in the outer halo. Wyse says scientists must understand the relationship between this bulge and the outer halo to tell the full history of the galaxy.

"We need future, large, dedicated spectroscopic surveys of stars in our galaxy to answer the many outstanding questions," she says. —S. WILLIAMS

Run of the Mill

Finding galactic building blocks in early universe

Hunters of distant galaxies, meet Joe Average.

For more than 3 decades, astronomers have scoured the skies for tiny, ultrafaint galaxies that could be the early building blocks of the massive galaxies common in the universe today. Now researchers report that they have found 27 remote galaxies that appear to fill the bill.

These galaxies have low rates of star formation and appear to be 20 times as numerous as other, larger galaxies previously found from the same early era, when the universe was just 2 billion years old. Their properties suggest that the newly discovered bodies are part of a long-sought population of average-size galaxies that merged to form larger galaxies like the Milky Way.

Using the European Southern Observatory's Very Large Telescope (VLT) in Paranal, Chile, Michael Rauch of the Carnegie Observatories in Pasadena, Calif., and his colleagues studied a tiny patch of sky for an unprecedented 92 hours, recording extraordinarily faint levels of a particular wavelength of light. That wavelength, known as Lyman-alpha, is emitted when energetic radiation from newborn, massive stars bombards hydrogen gas within galaxies, causing the gas to glow.

As observed from Earth, the ultraviolet Lyman-alpha radiation is shifted to longer, or redder, wavelengths by the expansion of the universe. The more distant the galaxy, the greater the redshift. The redshifted Lyman-alpha radiation detected by Rauch and his colleagues indicates that the 27 galaxies reside nearly 12 billion light-years from Earth, the team will report in the March 1, 2008 *Astrophysical Journal*.

The weak Lyman-alpha emission indicates that these galaxies are forming stars at a sluggish rate, equaling a tenth of the sun's mass every year. In addition, the density of the galaxies found in such a small area of sky suggests that the galaxies are about 20 times as common as a well-documented collection of brighter but equally remote galaxies that make stars more prodigiously. Those galaxies, known as Lyman-break,

QUOTE



It's actually a little scary how close [the simulation is] to the real thing."

THOMAS D. POLLARD,
Yale University

were found using a different detection technique. They are not only rarer than the new group but also more massive, Rauch says.

Hints of the newly found building blocks for larger galaxies emerged when astronomers began studying the detailed spectra of the brilliant beacons known as quasars. The spectra revealed that as the quasar light journeyed to Earth, some of the radiation was absorbed by intervening blobs of hydrogen gas. Rauch and his collaborators now suggest that those blobs, previously revealed only as shadows on the quasar light, are the small, Lyman-alpha-emitting galaxies his team has detected.

Images of another sky region, studied intensively with the Hubble Space Telescope and known as the Hubble Ultra Deep Field, may also show signs of these run-of-the-mill galaxies, notes Rychard Bouwens of the University of California, Santa Cruz.

The 92-hour VLT study, pieced together from odd corners of the night over several years, “is a heroic observation, and I hope that it represents the start of an era rather than something the world decides is too expensive to repeat,” comments David Weinberg of the Ohio State University in Columbus. Although the objects are detected at a low signal-to-noise ratio and the sample is small, the team “most likely is detecting star formation in small galaxies, mapping out the iceberg of which the previously known Lyman-break galaxies are the tip,” says Weinberg. “Knowing what is going on for these more run-of-the-mill systems will be valuable for testing theories of the [early] galaxy population.”

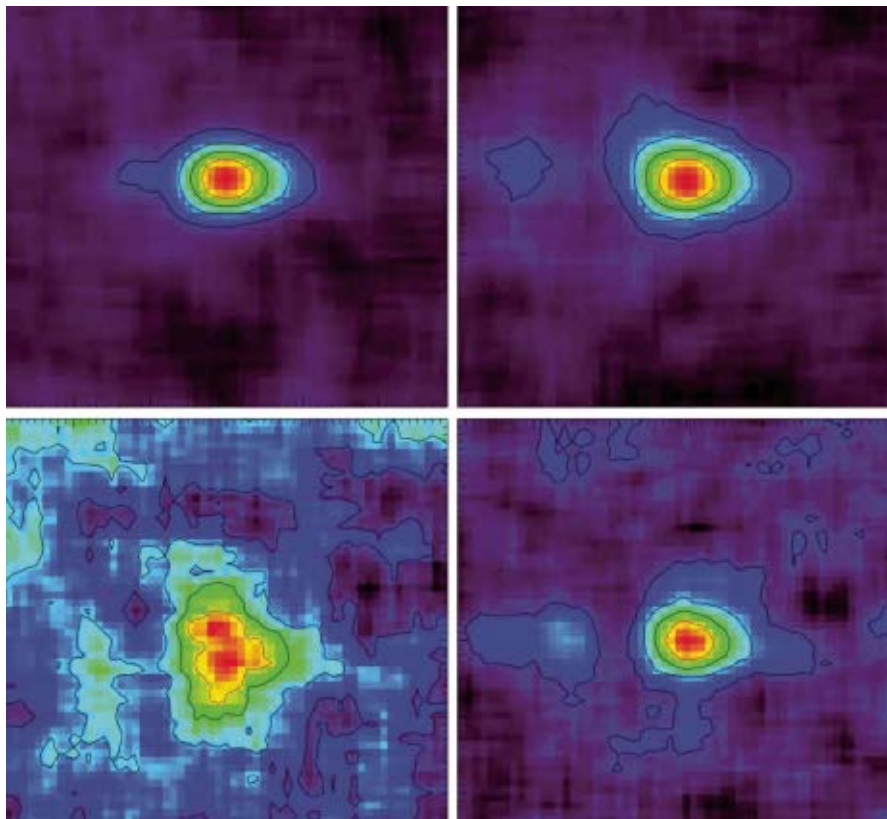
The observation bodes well for finding many more of the building blocks with ground-based telescopes, rather than having to conduct such studies in space, says Rauch. —R. COWEN

Light Swell

Optical rogue waves resemble oceanic ones

Slightly noisy signals can turn into rare large spikes in an optical fiber's output, in much the same way as unpredictable weather conditions occasionally create monstrous, isolated oceanic waves, researchers have found.

The new technique for creating such “rogue waves” in the lab might help physicists understand them as a general phe-



LITTLE GUYS A glow of hydrogen gas emanates from a population of low-mass, weakly star-forming galaxies believed to be the building blocks of bright, present-day galaxies.

nomenon, in the hope of predicting the risks for vessels at sea.

Rogue waves—waves significantly higher than the local average at a given time—belonged to seafarer lore long before scientists conclusively demonstrated their existence in the mid-1990s.

Theoretical studies and computer simulations have shown that rogue waves can originate from what physicists call nonlinearity. Two waves crossing each other's path ordinarily add up in height so that the new peaks' heights equal the sum of the original waves' heights. After this linear interaction, the waves will then proceed unperturbed, each on its way. Occasionally, however, the overlap will result in waves that are shorter in length but have heights larger than the sum of the original waves. Some scientists believe that in the ocean, such nonlinearity can create the waves of 30 meters or more that have sometimes been observed.

These waves will be very short-lived, although winds may increase their lifetime, as Christian Kharif of Aix-Marseille University in France and his team recently observed by creating artificial rogue waves in a 30-m-long water tank. Their results will appear in the *Journal of Fluid Mechanics*.

A rogue wave will appear “at a random location, at a random time,” says Bahram Jalali, an electrical engineer at the University of California, Los Angeles (UCLA), who developed an interest in rogue waves while spending time on his 36-foot sailboat.

Jalali and his collaborators examined laser light propagating in an optical fiber using a so-called time-stretch digitizer. This instrument allowed the researchers to analyze the waveforms of light and resolve spikes that lasted less than a trillionth of a second. Some of the random spikes, which occurred thousands of times per second, were up to 30 times more intense than average, says team member Daniel Solli of UCLA. The report appears in the Dec. 13 *Nature*.

The team's own computer simulations showed that noise in the input laser waves should indeed result in rogue light waves occurring about as often as they do in the lab. Although the physics is different, this nonlinear amplification is analogous to the idea that a butterfly flapping its wings in one continent can trigger a storm in another continent.

Although the optical system is essentially one-dimensional, it “shows the growth of freak waves very similar to what you see in the ocean,” says Mattias Marklund, a theoretical physicist at Umeå University in Sweden.

Marklund adds that optical experiments will allow physicists to test their theories with greater control over the variables.

Jalali says that he himself has never seen a rogue wave while sailing, which is probably a good thing. But the possibility tickles his scientist's curiosity. “To be honest, I do hope I see one,” he says. —D. CASTELVECCHI

S. RAUCH ET AL.

Find Out about the Spiritual Leaders Millions Embrace

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Researchers put restoration to the test

BY LESLIE ALLEN

It took less than a century after John Deere unveiled his steel-bladed plow in 1837 for the North American prairie to all but disappear. For 20 million years, a nearly 1,000-mile-wide swath of unbroken grassland belted the continent's midsection from northern Canada to Mexico. Now, only about 5 percent is left, mainly as mixed and shortgrass prairie in the Plains states. To the east, less than 1 percent of the original lush tallgrass remains, most of it as remnants in pioneer cemeteries and old railroad rights-of-way.

Plowed up, paved over, and little lamented, the vanishing prairie found few early champions. Among them were naturalists Aldo Leopold and John Curtis, who began using Civilian Conservation Corps enlistees in the 1930s to help restore more than 110 acres at the University of Wisconsin–Madison Arboretum. One of the earliest attempts at habitat restoration, the site today has hundreds of species of native plants, birds, and small mammals.

Now, prairie restoration is attracting widespread interest among environmental scientists, conservation groups, and even the U.S. government. The first federal grassland preserve, Midewin National Tallgrass Prairie, opened 3 years ago on the grounds of the former Joliet Army Ammunition Plant near Chicago. Thousands of ordinary midwesterners are also rediscovering their long-spurned heritage, working to preserve or restore patches of prairie in fallow cornfields, quarter-acre backyard plots, and an expanding network of preserves. How-to Web sites instruct landowners in restoration techniques, and seed companies specializing in prairie species are thriving. Prairies now rank among the most popular ecosystems targeted for restoration anywhere, especially the tallgrass of the Midwest's eastern third.

But researchers are left wondering how well the prairie renaissance is really succeeding and whether it's actually possible to recreate a prairie. Until recently, little long-term monitoring had quantified the success rates of common restoration techniques, and few studies had compared even the most careful restorations with scarce remnant prairie habitat.

Broad-scale comparisons are complicated by the fact that restorations serve a range of different purposes. Beyond bringing back native plants, some restorations focus on conserving fresh water or creating habitat for birds. About 40 percent of North American bird species are native to prairie.

Because restoring prairies is both labor- and cost-intensive, some restorations are seeded with only a fraction of the plants that a remnant prairie holds. Seed mixes usually contain relatively few species and some of those species are difficult to grow from seed.

MEASURING SUCCESS Real prairies are highly diverse. "In a remnant prairie, you can find 150 to 180 species of plants," says Deborah Marr, a plant ecologist at Indiana University in Bloomington. In western Indiana, Marr and her colleagues have been comparing restored prairie with slices of the original in nature preserves and along railway rights-of-way. Across the board, the remnants have more native-plant species. Over a 4-year study period, plant diversity increased in the restored prairies, but the proportion of grasses and flowering broad-leaved plants diverged from that found in remnant prairies.

A study during the 1990s of sites around the Fermi National Accelerator Laboratory in Batavia, Ill.,

where restoration efforts began in 1975, also found that species richness declined over time in restored sites, but not in remnants. The Fermi restorations had never achieved the biodiversity of remnants to begin with. High diversity, a Holy Grail to prairie ecologists, so far eludes their restorations, but no one is sure why.

"A remnant is very complex," notes Marr. Blazing wildflowers and rippling bluestems only hint at the complexity below ground. "It takes a long time for soils to build up," she adds. Unlike cropland, prairie soils are rich in fungi, which appear to be an essential component of high diversity. In August, at a conference

in San Jose, Calif., Indiana University researcher Peggy Schultz reported on field trials that suggest that adding soil from prairie remnants, or at least inoculating restorations with the kind of fungi found in remnants, can allow hard-to-establish plants to take hold.

SEED SELECTIONS Obtaining the variety of seed needed to start a restoration can be an arduous, months-long task involving painstaking hand picking by squads of volunteers. As a result, many restorations rely on mail-order seeds that typically include grass cultivars or wildflowers from various sources. The grass cultivars were originally bred by the U.S. Department of Agriculture to hold topsoil in place. They are now planted on millions of acres as part of the USDA's Conservation Reserve Program, which pays farmers to plant erosion-taming native grasses on land removed from agriculture.

In the late 1990s, though, plant biologist Sara Baer, then a graduate student at Kansas State University in Manhattan, began noticing something unexpected while doing research at the 3,487-hectare Konza Prairie Biological Station in northeastern



YARDSTICK OF REPRODUCTIVE SUCCESS — Wild indigo's height in an Indiana restoration yields comparisons to remnant prairie for researcher Deborah Marr.

Kansas, part of the largest remnant tallgrass prairie in North America. The grass cultivars in her sites were germinating readily and growing fast and tall. In some situations, that would be desirable, but here, the robust grasses were crowding out slower-growing native flowering plants and disrupting the balance of species. Productivity was easy to restore in the prairie; diversity much less so.

At around the same time, plant ecologist David Gibson and his students at Southern Illinois University Carbondale (SIUC) began finding major genetic differences between cultivars and wild seeds. Their photosynthesis rates also differed. Furthermore, genetic differences existed between local and nonlocal wild seed.

Since then, some prairie enthusiasts, passionate about making restorations as faithful to the original undisturbed prairie as possible, have begun avoiding mail-order seed mixes, instead hand gathering wild seed only within 200 miles of their site. Gibson cautions that little research supports any particular approach.

With a 5-year grant from the National Science Foundation, Gibson and Baer (now also at SIUC) hope to tease out some guiding principles for restorations. At three sites in Kansas and Illinois, the scientists are planting wild seeds and cultivars of prairie grasses and wildflowers in multiple plots and in various proportions. "No one has ever put the same plant species, but from different seed sources, in a common environment before," says Baer.

Cultivated seed will make up from 4 percent to 97 percent of each plot's mix. The idea, says Baer, is to see whether tinkering with proportions can help establish and maintain a truly diverse prairie.

BURNING QUESTIONS "Production of prairie seeds is a big business, and there are all these species that people can choose to put in," Baer says. "Restorations are unique, in that by our decisions, we humans are an integral filter."

Historically, bison grazing and fire were the two natural filters that shaped and maintained the prairie. Until they were nearly extirpated in the 19th century, along with the prairie itself, bison by the tens of millions ranged across North American grasslands, often in herds so big that observers compared them to roaring avalanches. Fires, set by lightning strikes and later by Native Americans, would attract bison and other herbivores, because the burned patches sprouted fresh green grasses that the animals prefer to graze on. At the same time, bison avoided the tender broad-leaved plants, or forbs. This kind of preferential grazing established a system of checks and balances, which kept grasses under control and allowed many plant species to flourish.

Researchers at Kansas' Konza Prairie and elsewhere have begun to see how bison encourage habitat diversity by grazing very heavily on burned patches and avoiding other areas altogether. Heavily disturbed habitats, for example, attract some native birds. Other native birds prefer completely undisturbed habitats; and still others, such as prairie chickens, require a mix of habitats. The same holds true for insects and small mammals.

Over time, fire drove the bison's behavior, which in turn shaped the prairie's biodiversity. But fire by itself is not enough to restore diversity to the prairie, says ecologist Scott Collins of the University of New Mexico in Albuquerque. Various studies by Collins and his colleagues have shown that frequent burning by itself can reduce biodiversity. Collins' work at Konza shows that species diversity rises in areas that are grazed and infrequently burned, and falls in frequently burned, ungrazed areas. "Diversity is much higher at all levels in the grazed areas," he says.

"If I had a prairie to restore," says Collins, "my recommendation would be that some kind of grazing, or at least mowing, to eliminate the big, thick grass canopy and create more light, take place." Studies by other researchers indicate that bison and other native herbivores like to eat many nonnative, exotic plants, which helps suppress the invasions that plague grasslands.

Deron Burkepille of Yale University, who studies native herbivores in North American and South African grasslands, says, "I think that grazing is essential for restoration. More and more people are starting to adapt that mind-set."

Those findings are borne out by the sight of newly installed bison herds silhouetted against the sky at a growing number of prairie preserves. Still, little is known, even now, about eons-old grazing

patterns and fire frequencies. If annual burning causes plant diversity to fall, then how often should controlled burns occur? Studies indicate that burning every 4 years probably isn't frequent enough to keep out trees and woody shrubs.



RESTORED TO THE RANGE — Bison promote biodiversity by grazing on new grasses but ignoring tender forbs.

ADAPTING TO THE FUTURE

Ironically, figuring out historical fire frequency at Konza may not be relevant today, says Collins. Human-driven environmental change imposes new conditions on prairies that could make restoration more challenging than ever. Already, encroachment by nonnative shrubs, bushes, and other woody plants is afflicting

grasslands around the world, with or without controlled burning.

And the past itself is a moving target, points out Alan Knapp, a plant ecologist at Colorado State University in Fort Collins who is also doing studies at Konza Prairie. "We have a romantic, snapshot view of the prairie when Europeans settled it," he says. "But ecological systems are always dynamic, always changing."

Prairies evolved under blazing summers, harsh winters, and extreme fluctuations of temperature and rainfall from year to year and within growing seasons. To adapt, prairie plants developed underground-storage structures and extensive root systems. Scientists have recently discovered that prairie species grow more variably from year to year, depending on rainfall variations, than do plants in any other North American biome. That variability makes remnant prairies, such as Konza, good natural laboratories for studying the likely effects of future climate change.

Most climate models predict extreme and variable rainfall patterns and future temperature increases. To study those hypothetical effects, scientists at Konza Prairie are manipulating rainfall and temperature under canopied shelters where native prairie grasses, such as big bluestem and Indian grass, grow. Altering the timing of rainfall from current norms can lead to significant declines in the plants' productivity. "It's surprising how rapid the changes have been," says researcher Melinda Smith of Yale University.

In a new, related study at Konza Prairie, Smith is profiling the genetic activity of two grasses under simulated climate-change conditions. Some regulatory genes may become less active when the grasses are stressed by alterations in precipitation. Smith and colleagues hope to identify specific genetic changes linked to the plants' responses to environmental changes. That should, in turn, reveal implications for large-scale ecosystem processes.

One early surprise is the large amount of genetic diversity that already exists within populations of native dominant species. "You can find 14 genotypes of big bluestem in 1 square meter," says Smith. "Diversity within dominant species is often ignored."

Tapping that genetic diversity may in time offer the best shot for keeping grasslands vibrant under future conditions that will be vastly different from those of today. ■

THE LONG ROAD TO BETA CELLS

Quest for type 1 diabetes cure inches forward

BY BRIAN VASTAG

In 2000, researchers in Canada reported a possible breakthrough in the treatment of type 1 diabetes. By transfusing insulin-producing cells from donated pancreases into patients, the researchers provided what looked like cures. Within a week after the procedure, all of the first six patients were liberated from daily insulin injections.

For a time, the team that developed the procedure, at the University of Alberta in Edmonton, appeared to have succeeded where many others had failed.

But after a year or two, the transplants began to falter. Last year, the latest report on the Edmonton protocol found that of 36 patients, 21 initially were able to ditch their insulin needles. Two years after transplantation, though, 16 of those patients were back on insulin. Another report on 65 patients found that only 6 were insulin-independent at 5 years post-transplant.

While ultimately disappointing, the Edmonton protocol is “clearly orders of magnitude better than previous attempts at [pancreatic-cell] transplantation,” two diabetes researchers wrote in 2006 in the *New England Journal of Medicine*.

Since the 1970s, researchers had been implanting pancreatic cells from cadavers into type 1 diabetes patients with little luck. By the mid-1990s, after some 450 transplants worldwide, the success rate hovered at a dismal 2 percent.

The field was ready to give up. But the Edmonton team, led by transplant surgeon James Shapiro, studied every previous transplantation and theorized why the earlier procedures had failed: Patients weren’t receiving enough cells and the older immune-suppressing drugs given after the infusions were actually hurting the implanted cells. Shapiro and his team engineered a more efficient way of extracting islets—the working zones of the pancreas—from cadavers and replaced the older immune-suppressing drugs with newer ones.

Islets contain beta cells, which secrete insulin in response to glucose, maintaining healthy concentrations of blood sugar. In type 1 diabetes, the immune system attacks and destroys islets. After 60 to 80 percent of the small, round structures disappear, symptoms arise, starting with severe thirst and hypoglycemia. Over the long term, toxic acids can build up in the blood and cause blindness, kidney failure, nerve damage, and accelerated blood vessel hardening. Early death from heart attack or stroke often results.

Between 1 million and 2 million people in the United States have type 1 diabetes, with about 30,000 new cases each year. While the disease is often called juvenile diabetes, it can strike at any age. Type 2 diabetes is a more common yet distinct disease in which beta cells generally continue making insulin, but the body’s other cells lose the ability to use it.

When Shapiro began treating patients with the Edmonton protocol in 1999, he infused huge numbers of islets into the large vein that feeds the liver. There, the cells nest and deposit insulin directly into the blood, each clump acting like a little pancreas.

The early success of the protocol “galvanized the medical community,” says Alan Colman, an embryonic stem cell researcher and executive director of Singapore Stem Cell Consortium. Stem cell researchers noted the results because they provided “proof of principle,” says Colman, that cell replacement could treat type 1 diabetes.

After scientists first grew human embryonic stem cells in the laboratory in 1998, many researchers waxed about an imminent era of cellular-replacement therapy, where the blank slate embryonic cells would be transformed into an array of tissues useful for treating Parkinson’s disease, heart failure, diabetes, and other conditions.

To date, though, there is little evidence that cell replacement works in people. The Edmonton protocol is the sole exception. For

“It’s turning out to be an extraordinarily difficult cell to make.”

— ALAN COLMAN,
SINGAPORE STEM CELL
CONSORTIUM

cell replacement to ultimately succeed in type 1 diabetes, though, researchers first must fix the long-term failure of the cell transfusions. (One prominent diabetes researcher recently published data suggesting how to do so.) Second, clinicians will require a vast supply of insulin-making cells. With a chronic donor shortage, very few pancreases become available for islet or whole-organ transplants. (Whole pancreas transplants often cure type 1 diabetes, but in 2006, just 1,367 people in the

United States received pancreas or pancreas-kidney transplants, many for conditions other than diabetes.)

Enter embryonic stem cells. When cared for properly, the cells double in population every 18 hours or so. If a magic formula to coax embryonic cells to form beta cells could be found, scientists could conjure bottomless vats of potential diabetes cures.

But the road from embryonic cell to beta cell is proving to be long and treacherous. Dead ends and wrong turns plague the travelers. Since 2001, several research teams have announced the creation of insulin-producing cells only to watch their results evaporate under scrutiny.

“It’s turning out to be an extraordinarily difficult cell to make,” says Colman, who knows how to persevere—he worked on the team that in 1997 cloned Dolly the sheep from a skin cell after 277 failed attempts.

At a recent meeting organized by the private New York Stem Cell Foundation, however, three teams reported progress down the beta cell road. “I think it’s going to be possible ... to turn human embryonic stem cells into fully functional beta cells,” says Douglas Melton, a diabetes researcher at the Harvard Stem Cell Institute and the Howard Hughes Medical Institute in Cambridge.

DEAD ENDS In 2001, a team headed by a National Institutes of Health researcher, Ron McKay, announced success in making insulin-producing cells from mouse embryonic stem cells.

But Melton and his Harvard team found something peculiar when they tried to replicate the results. While insulin appeared on the surface of the cells, the team found no evidence that the cells churned out the messenger RNA needed to make insulin. Puzzled, the researchers grew more batches of the putative insulin-making cells—but this time, unlike McKay, they used a growth medium devoid of insulin. Now there was no insulin on the surface of the cells either. In 2003, the Harvard group concluded that the cells were not making insulin; they were simply sucking in small amounts from their surroundings. The work was a dead end.

Beginning in 2000 and continuing through this year, other research groups periodically announced potential shortcuts: Cells in the human spleen, liver, bone marrow, or near the pancreas that also could be grown into insulin-making cells. The announcements unfailingly generated excitement—if such cells existed, they could provide a quicker route to a cure, obviating the scarce and finicky embryonic cells.

But Melton and other top stem cell researchers reject such claims. “Routinely, I open a newspaper and find a report on a new cell that can replace a pancreatic beta cell,” says Melton. “There isn’t any ... reason to believe those reports are correct.”

Most organs—including the brain, heart, skin, eyes, liver, and bone marrow—harbor small reservoirs of organ-specific, or adult, stem cells. From these, a trickle of new tissue repairs normal wear and tear. But the pancreas behaves differently, says Melton. There is no adult stem cell for the pancreas, he argues. Instead, he says, new beta cells arise from existing beta cells.

To support his claim, Melton points to work he published in the September *Journal of Clinical Investigation* with investigators at the Hebrew University in Jerusalem. The team engineered a strain of mice whose beta cells contain a “Trojan horse” gene for the diphtheria toxin. When activated by an antibiotic such as doxycycline, the toxin destroys its host beta cell. Feeding the mice doxycycline-spiked water makes them diabetic.

“The surprise was, when we removed the doxycycline, the animals recovered,” says Melton. Necropsies of the once-diabetic animals revealed that their pancreases held almost as many beta cells as normal mice. To see where these new cells came from, the team shoehorned another gene, a marker, into newborn animals’ beta cells. This marker appears in any new beta cells spun off from the originals. After repeating the experiments, the team found that all of the new beta cells displayed the marker—they all came from existing beta cells, not from some pancreatic adult stem cell.

In the May *Developmental Cell*, a team at Children’s Hospital of Philadelphia reported similar results (*SN*: 6/2/07, p. 350).

“There is no evidence for beta cells coming from adult stem cells,” says Melton. Instead, “during the life of a type 1 diabetic, beta cells are constantly replicating and then they’re being smacked down by the immune system.”

Jake Kushner, who led the work in Philadelphia, says that “if you could understand the biology of the beta cells that do grow, maybe you could make them grow” faster to treat diabetes. Melton says NIH and other funders should stop paying for work focused on finding pancreatic adult stem cells—he thinks it’s a false short-cut. He says researchers should instead look for treatments that increase the replication rate of existing beta cells.

Melton’s mouse experiments may also explain the Edmonton protocol’s long-term failures. When the diabetic animals received the two immune-suppressing drugs used in transplants, sirolimus and tacrolimus, the mice failed to spontaneously recover. Melton surmises that the very drugs that suppress the immune system also prevent beta cells from replicating—and such replication apparently contributes to the early success of the cell transfusions. Melton says that Edmonton protocol practitioners “would be well advised to look for immunosuppressants that don’t block beta cell regeneration.”

Shapiro, who developed the Edmonton protocol, agrees that “the drugs we use are not ideal. We can certainly improve on them.” He adds that his team is now testing a new drug regimen that employs lower doses of

immune-suppressing drugs. “We’re continuing to improve it,” he says.

BACKING UP While a few researchers continue searching for pancreatic stem cells, the leaders in the field have backed up. “I don’t believe [adult stem cells] are really going to work. Let’s move to the embryonic stem cell and start from the beginning,” says Emmanuel Baetge of San Diego-based Novocell.

The beta-cell creation strategy now in vogue seeks to retrace the development of the pancreas from embryo to organ.

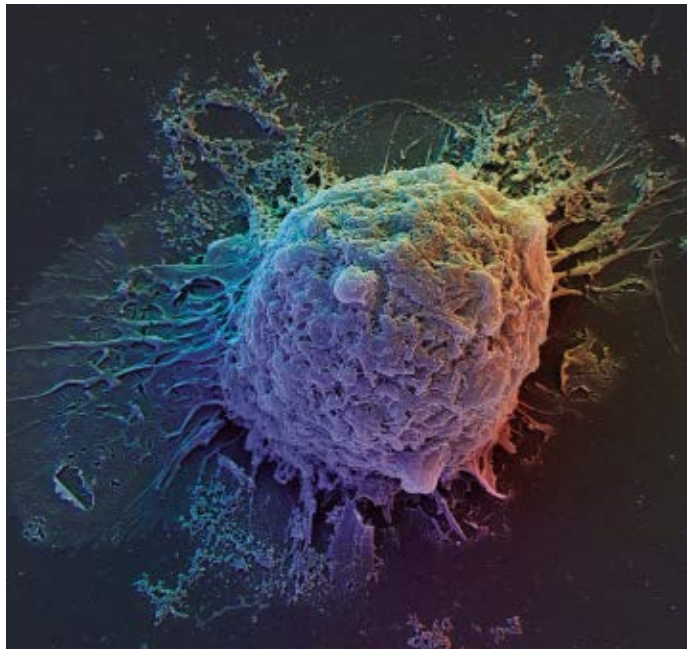
To visualize this journey, imagine that the drive from Washington, D.C., to New York City represents the trip from embryonic stem cell to beta cell. Along the route there are obvious stops—Baltimore, Philadelphia, Newark—corresponding to well-defined fetal tissue types.

The first stop, call it Baltimore, is endoderm, a thin layer that appears a few days after fertilization. It eventually forms most of the digestive tract, including the pancreas.

In 2005, Baetge’s team published a simple formula for turning human embryonic stem cells into endoderm. The team also identified a marker that distinguishes endoderm from what Baetge calls its “somewhat evil twin,” extra-embryonic endoderm. This tissue looks and acts almost exactly like endoderm, but it doesn’t grow into a pancreas. Instead, it forms the yolk sac that feeds the embryo. “It was a major problem,” says Baetge, often leaving him and other researchers thinking they’d reached Baltimore when in fact they’d veered left to Pittsburgh.

With Baetge’s formula, researchers can reliably concoct endless dishes of real endoderm in just 2 days.

The next stop, Philadelphia, corresponds to a cell type that, in fetuses, buds into the beginnings of a pancreas. Baetge and



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Colman say they can turn about half of a dish of embryonic stem cells into these cells. Melton's group can also make them. The cells are committed to becoming a pancreas, but they don't produce insulin. They "represent a major intermediate population," says Colman. "And we can reliably produce them."

Then, in October 2006 in *Nature Biotechnology*, Baetge and his Novocell colleagues published a five-step recipe to drive embryonic cells almost all the way to Manhattan. Drawing from the developmental biology literature, the researchers determined which growth factors and other molecules to sprinkle on the cells to move them along. Baetge says the cells at the end of the journey produce insulin and other pancreatic hormones. However, they do not respond to glucose—a key shortcoming. "What we have is a not fully formed, but betalike" cell, Baetge says. It's like being stuck in Newark.

Colman says his lab has not been able to reproduce Novocell's recipe, despite trying with nine embryonic stem cell lines. A member of Colman's team will soon travel to Novocell to observe the technique. "This is sincere, and it's been sealed by a tribal oath and a pint of bitters," the Scot says of the scientist exchange.

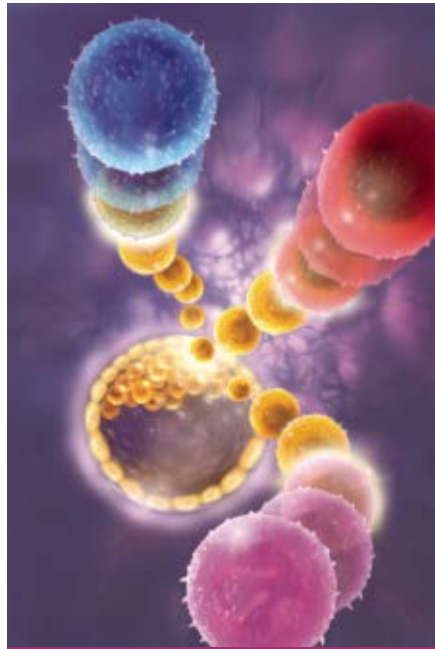
When Baetge tried implanting the betalike cells, the Newark cells, into diabetic mice, the animals remained diabetic. So he backed up and tried again with less-developed cells, or Philadelphia cells. The idea: Let the animal's body tell

the immature cells how to grow into fully functional beta cells. At the New York Stem Cell Foundation meeting, Baetge reported new, unpublished work that suggests this is exactly what happens. In the experiment, Baetge's team implanted a million or more Philadelphia cells under the skin, near the kidney, or in the fat of 24 diabetic mice. In two of the mice, the graft didn't take. But in the others, the cells switched on a critical beta-cell gene, churned out insulin in response to glucose, and cured the animals. "By transplanting [cells of] this earlier state and allowing the *in vivo* environment to finish the job, we can make structures that look very much like islets," says Baetge. "We think that's a remarkable efficiency."

Whether a similar strategy will work in people is, of course, unknown. Also unknown: whether researchers will ever be able to conjure beta cells in the lab. "Perhaps we're all asking too much too quickly," says Colman. Fetuses take 8 weeks to begin producing insulin, a figure Baetge's recipe cuts to 12 days.

Melton is taking a slower road. His team spent 3 years building a system to screen thousands of drugs and growth factors that might push embryonic cells along. "Our approach is perhaps too slow, but it's certainly systematic," he says. He's convinced such painstaking methods are the best

route to beta cell, a journey that he cautions will take "some years. We're just taking our first baby steps." ■



NOT YET — Scientists are trying to grow embryonic stem cells, like those seen here, into insulin-producing beta cells to treat diabetes, but progress has been slow.

CORBIS

OF NOTE

EPIDEMIOLOGY

Big kids at risk for heart disease

Being a weighty kid carries a heavy toll into adulthood. Overweight children grow up to have an elevated risk for blocked coronary arteries, a long-term Danish study reports.

"Our findings suggest that as children are becoming heavier worldwide, greater numbers of them are at risk" for heart disease, researchers from the Center for Health and Society in Copenhagen write in the Dec. 6 *New England Journal of Medicine*.

Drawing on government health records of 276,000 Danes born between 1930 and 1976, the team found that the older the child, the stronger the connection between excess

weight and adult heart disease. In 7-year-old boys, each 2 kilograms of excess weight conferred a 6 percent increase in risk of adult heart disease. But by age 13, the same relative amount of excess weight conferred a 15 percent increase in risk. The risks were somewhat lower for girls.

Obesity-promoting habits persist into adulthood, comments David S. Ludwig of Children's Hospital Boston. In addition, excess childhood weight "may elicit irreversible biological changes" in metabolism, which in turn increase heart disease risk. —B.V.

ZOOLOGY

Female antelopes take the lead in courtship

Among topi antelopes, it's the males that need convincing.

Topis, medium-sized antelopes in Africa, reverse the standard roles in

courtship, says Jakob Bro-Jørgensen of the Zoological Society of London. "When biologists talk about the battle of the sexes, normally it's between persistent males and resistant females," he says. Not so among topis, even though they live otherwise standard mammal lives with maternal care. A male topi stakes out his own small territory in a male cluster. Females visit during a frenzied 6-week mating season. Bro-Jørgensen says the desirable males find an abundance of mates. Each female becomes fertile for only 1 day per year, and Bro-Jørgensen reports females averaging 11 encounters with each of 4 males. It's not unusual for several hundred females to visit a cluster of only a dozen males.

A male presented with two fertile females prefers the newest arrival or the one he has mated with less often. Females spurned as old news readily lower their horns and charge a mating pair. Those lunges tend to shift a male away from his preferred newcomer and back to the repeat partner, Bro-Jørgensen says in the Dec. 18 *Current Biology*. —S.M.

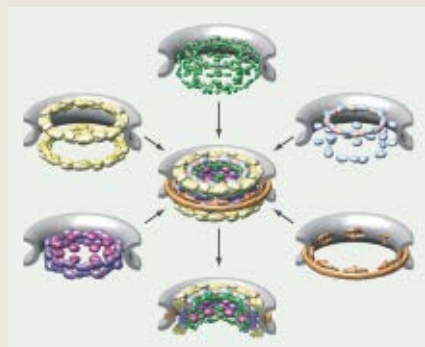
American Society for Cell Biology,
Washington, D.C.
Dec. 1–5

COMPUTATIONAL BIOLOGY

Cell's core pore structure solved

In a challenge that's like solving a three-dimensional jigsaw puzzle, scientists have worked out how 456 proteins fit together to form a doughnut-shaped gateway to the cell's nucleus.

Many such gateways cover the nucleus like polka dots, and they control what protein can pass through to reach the DNA inside. Knowing the structure of the gateway—called a nuclear-pore complex (NPC)—could improve scientists' understanding of why some large molecules can enter the nucleus and others cannot.



GRAND ENTRANCE The nuclear-pore complex (center) controls what can enter and exit a cell's nucleus. Cutaway views reveal the pore's elaborate architecture.

"[The NPC is] one of the biggest stable protein complexes in the cell," says Frank Alber of the University of California in San Francisco (UCSF). Even finding the structure of pores in a cell's outer membrane can be challenging, and those pores typically consist of less than a dozen proteins—far fewer than the NPC.

Solving the puzzle required pulling together many different kinds of data about the proteins. "None of the types of data on their own can tell us the structure" Alber says. "It's a bit like a crossword puzzle" in which each clue is ambiguous, but the overlapping answers restrict what the other answers can be, thus narrowing the possibilities to a single solution.

Alber and his colleagues at UCSF and Rockefeller University in New York used different kinds of data to place constraints on where each protein could be located relative to other proteins. For example, the researchers repeatedly fragmented the complex into small pieces, and then observed which clumps of proteins remained tightly bound to identify adjacent proteins. And electron microscope

images of an entire pore revealed its overall shape. Altogether, the scientists amassed about 10,000 constraints on the proteins' locations.

Starting from random swarms of proteins, the scientists then used computers to gradually find stable protein arrangements that met all of the constraints. Repeating this process thousands of times produced the most likely position for each protein.

The researchers studied NPCs from yeast cells, which have nuclei, just as human, animal, plant, and algae cells do. In principle, the new computational method could be applied to the human NPC, Alber says, although it is considerably larger than the yeast version. —P.B.

METHODS

Escaping flatland

Nothing is more iconic of biological research than the petri dish. Yet the idea that growing cells in a flat dish can sometimes lead scientists astray is gaining traction.

As an alternative, some researchers are experimenting on cells grown in gelatinous materials made from many of the same structural proteins that fill the spaces between cells in the body. The nutritive materials allow the cells to form three-dimensional structures, as in real tissues, rather than flattening into a single layer in a dish. The experiments are revealing the many ways that cells' immediate surroundings guide their behaviors.

"All of the sudden, half the field is jumping into these 3-D models," says Mina Bissell of the Lawrence Berkeley National Laboratory in Berkeley, Calif., who helped pioneer 3-D cell cultures about 30 years ago (*SN*: 8/30/97, p. 138).

For example, cancerous breast cells develop resistance to multiple chemotherapy drugs more readily when grown in 3-D conditions, better mimicking what happens in patients, according to research led by Valerie M. Weaver of the University of California, San Francisco. Weaver's team found that cancer cells grown in a 3-D environment had higher levels of a protein called nuclear receptor corepressor 2 (N-CoR2) than cells in flat dishes. While it's still not clear what environmental cue causes the cells to boost N-CoR2 production, adding N-CoR2 to the cells in dishes allowed those cells to develop drug resistance.

"Finding a way to treat these cells in 3-D would be beneficial for cancer therapy,"

says Chandrima Chatterjee, a member of Weaver's research team at the University of Pennsylvania in Philadelphia. —P.B.

EVOLUTION

Cells' innards may share origin

Despite their outward differences, many of the organelles within cells may have a common evolutionary heritage.

In a case of scientific serendipity, data gathered by separate research teams working on various organelles lend new support to the theory that a simpler cellular compartment gave rise to the organelles' diverse modern forms.

"We all had been looking at specific organelles, but sitting there [at the conference] listening to the other scientists speak, there seemed to be something common in all of them," says Damien Devos of the European Molecular Biology Laboratory in Heidelberg, Germany.

Several research groups had been studying proteins that guide the movements and interactions of organelles such as the Golgi apparatus, the endoplasmic reticulum (ER) and the nucleus.

"The data are contradictory if you look at one protein at a time," says Joel B. Dacks of the University of Cambridge in England. "But if you look at them together, it fits."

Each protein on an organelle has evolved at a different rate, so each tells a different story about how long ago that organelle might have diverged from an ancient, simpler organelle and begun developing unique functions.

But Dacks suggests that because all the proteins on one organelle must function together, a change in even one protein could be enough to send the whole compartment off in a new evolutionary direction.

Viewed this way, the measured similarities among the versions of organelle proteins such as Rab, SNARE, and Adaptin suggest they all evolved from a compartment in an ancestral cell that lived long before multicellular life arose, Devos and Dacks say. Such a scenario would contradict the idea that organelles such as the Golgi apparatus and the ER independently evolved, perhaps from pockets in the cell's outer membrane.

"They all came from the same place," Dacks postulates. However, even if further research supports the new theory, it would not apply to energy-converting mitochondria or sunlight-absorbing chloroplasts, which are known to have evolved from ancient, independent-living bacteria that became incorporated into the cells. —P.B.

Books

A selection of new and notable books of scientific interest

BEYOND HUMAN: Living with Robots and Cyborgs

GREGORY BENFORD AND ELISABETH MALARTRE

In the future, robotic assistants will be as pervasive as personal computers are today. The technological



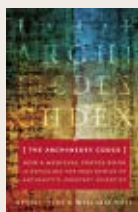
augmentation of humans that began with pacemakers and hearing aids will continue with smarter prosthetics and implants, eventually becoming voluntary enhancements that people seek out to give themselves an edge. Gradually, the line between man and machine will blur, and people will become acclimated to these

new roles of technology just as they've adjusted to carrying a mobile phone. In this book, University of California physicist and author Gregory Benford and a biologist who writes under the pen name Elisabeth Malartre offer an in-depth exploration of this imagined future filled with robots and cyborgs. Beyond speculation, the book cites numerous present-day examples of this trend toward everyday robotics and technology-enhanced humans. This brave new world is already here, the authors argue. *Forge*, 2007, 272 p., **hardcover, \$24.95.**

THE ARCHIMEDES CODEX: How a Medieval Prayer Book Is Revealing the True Genius of Antiquity's Greatest Scientist

REVELI NETZ AND WILLIAM NOEL

Some of the works of Archimedes—the Greek thinker and tinkerer who lived in 3rd-century B.C. Sicily and discovered the principle of buoyancy—



survive only in a single 8th-century copy. As Netz and Noel recount, the manuscript was lost and found multiple times, erased and recycled into a prayer book by a 13th-century monk, and lived through fire, mold, and forgers who covered some of its pages with fake medieval paintings. In 1998, a collector bought

the manuscript for \$2 million and entrusted it to Noel, a curator at the Walters Art Museum in Baltimore. Using pioneering technology, researchers have managed to read most of the book's content, allowing historians—including Netz—new glimpses into Archimedes' genius. *Da Capo*, 2007, 320 p., **color photos and b&w illus., hardcover, \$27.50.**

LOVE AND SEX WITH ROBOTS: The Evolution of Human-Robot Relationships

DAVID LEVY

Today, some people form fervent attachments to their Blackberry devices, electronic pets, and even laptops. Could true love be that far off? And what about relations more amorous? In a book aimed at a popular audience, David Levy foresees a time when humans' intimacy with artificially intelligent machines will grow even deeper, entering the realms of romance and the erotic. These machines,

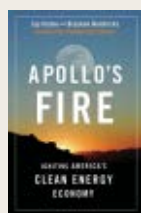
Levy argues, would be much more sophisticated than today's most advanced robots. The author describes the latest research in artificial intelligence and its implications for the developing human-robot relationship. Looking first at emotional bonds, he discusses research on the human drive for connection with other humans. He goes on to explore the close bonds people form with pets, inanimate objects, and, increasingly, electronic gadgets. The second half

of the book looks at the past, present, and possible future of humans' intimate relations with objects, machines, and robots. *HarperCollins*, 2007, 352 p., **hardcover, \$24.95.**

APOLLO'S FIRE: Igniting America's Clean Energy Economy

JAY INSLIE AND BRACKEN HENDRICKS

The authors present a manifesto for the Apollo Alliance, a clean-energy advocacy organization that Inslie, a congressman from Washington state,



helped found and where Hendricks is a senior fellow. Greening the U.S. economy is not only necessary to save the environment and wean us off Middle Eastern oil, the authors write. It will also create millions of "green-collar" jobs, which will be held by everyone from engineers developing better solar panels to the workers

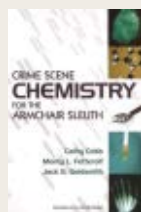
who will install them. The book evokes the national focus on reaching the moon in the 1960s to advocate a comprehensive array of policy and technological solutions. It also aims to allay fears of losing jobs to new regulations and to defuse tensions between trade unions and environmentalists, two traditionally Democratic constituencies. *Island Press*, 2007, 416 p., **b&w photos, hardcover, \$25.95.**

CRIME SCENE CHEMISTRY FOR THE ARMCHAIR SLEUTH

CATHY COBB, MONTY L. FETTEROLF, AND

JACK G. GOLDSMITH

From finding and collecting trace evidence to measuring blood alcohol levels and analyzing other bodily fluids, law enforcement depends on chemistry. In this book, chemists Cathy Cobb and Monty L. Fetterolf, along with chemist and reserve police officer Jack G. Goldsmith, take readers through the fascinating field of forensic chemistry. Requiring no prior



knowledge of chemistry, the chapters are arranged in a logical progression that showcases the depth and breadth of this burgeoning scientific discipline. Become an amateur sleuth by following the instructions for 25 hands-on demonstrations you can perform using ordinary household items and products. A

fictional minicase after each demonstration illustrates how its techniques are used to solve crime. Techniques reviewed include testing for the presence of drugs, collecting latent fingerprints, identifying body fluids, and analyzing soil, trace fibers, and gunshot residue. This comprehensive source provides insights into the painstaking work—rarely shown in most TV crime dramas—that goes into police investigations. *Prometheus Books*, 2007, 384 p., **b&w illus., hardcover, \$26.00.**

LETTERS

Fuzzy logic

Astronomer Masanori Iye of the National Observatory of Japan blames the blurry appearance of meteor trails at about 100 kilometers altitude on the fact that they were photographed with telescopes focused at infinity ("Out-of-focus find," *SN*: 9/29/07, p. 205). But optics teaches that any object much farther away than the focal length of the telescope is essentially "at infinity." Wouldn't a routine cause of fuzzy telescopic images—diffraction—be a more important reason for the blurred trails?

DAN WILKINS, OMAHA, NEB.

For the telescope used in these observations, calculations indicate that improper focus causes much more blurriness than diffraction does, says Iye. —S. PERKINS

Treating the symptoms

"Stimulant Inaction: ADHD drug's lift proves surprisingly weak" (*SN*: 11/3/07, p. 277) suggests that Ritalin fails to "cure" attention-deficit hyperactivity disorder. Ritalin for ADHD is like glasses for a vision problem. True, it aids some functions and not others—visual memory, not executive function, for example. But for the right child at the right time, Ritalin can be tremendously helpful, especially combined with other support the child needs.

HELEN F. NEVILLE, OAKLAND, CALIF.

Who don't you love?

"Tortoise Genes and Island Beings" (*SN*: 11/10/07, p. 298) refers to Lonesome George, the Galápagos tortoise, as "misanthropic"—meaning a hater of people. He certainly has good reason to dislike humans, but I wonder how the investigators could tell. Or did you mean that George doesn't like other tortoises, and is therefore antisocial?

ROMAN KOZAK, OMAHA, NEB.

Lonesome George's lack of gregariousness extends across species: He has shown next to no interest in other tortoises, and retreats to the far reaches of his enclosure when tourists and other strangers come to gawk. —B. NELSON

Correction "Flawed Stem Cells Yield Fragile X Clues" (*SN*: 11/17/07, p. 310) referred to boys inheriting a defective gene on the X chromosome from either parent. In fact, the X chromosome in males can come only from the mother. Also, the research described in the story was done by scientists at the Tel Aviv Sourasky Medical Center as well as the Hebrew University in Jerusalem.

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