SN
SCIENCE NEWS MAGAZINE
SOCIETY FOR SCIENCE & THE PUBLIC
JULY 23, 2016

Map to Planet Nine

A Healing Ozone Hole

Evolution in the City

Mitochondrial Sabotage

Aging's Future

SPECIAL REPORT

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SOCIETY UPDATE
Meet alumni of SSP science competitions

COVER Extending life span may have limits, but prolonging health has real possibilities. *David Curtis*

Still mysterious, aging may prove malleable

Aging happens to each of us, everywhere, all the time. It is so ever-present and slow that we tend to take little notice of it. Until we do. Those small losses in function and health eventually accumulate into life-changers.

Despite its constancy in our lives, aging remains mysterious on a fundamental level. Scientists still struggle to fully explain its root causes and its myriad effects. Even as discoveries pile up (*SN: 12/26/15, p. 20*), a clear picture has yet to emerge. Debates continue about whether individual life spans and the problems associated with aging are programmed into our bodies, like ticking time bombs we carry from birth. Others see the process as a buildup of tiny failures, a chaotic and runaway deterioration that steals vim and vigor, if not health and life itself. There is no unified theory of aging. That means that there is no one way to stop it. As longtime aging researcher Caleb Finch put it in an interview with *Science News*: Aging is still a black box.

The issue is an urgent one. The globe's population has never been older. According to the U.S. Census Bureau's 2015 *An Aging World* report, by 2020 the number of people 65 and older worldwide will outnumber children 5 and under for the first time in history. Seniors will make up 22.1 percent of the U.S. population in 2050, and nearly 17 percent globally (a whopping 1.6 billion people), the demographers predict. Worldwide, the 80-and-above crowd will grow from 126 million to 447 million. It's a population sea change that will have ripple effects on culture, economics, medicine and society.

Scientists working at the frontiers of the field do agree that there are probably many ways to slow aging, Tina Hesman Saey reports on Page 16 of this special issue. Saey sums up current thinking on the actors of aging, as well as a number of intriguing approaches that might well tame aging's effects. The goal, most agree, is not to find a fountain of youth but the keys to prolonging health.

It turns out that healthy aging in people does occur naturally. It is, however, in the words of Ali Torkamani, "an extremely rare phenotype." Torkamani leads a genetic study of people 80 and older who are living free of chronic disease, described by Saey in her story. He and his team failed to find a single set of genes that protect these "wellderly." Instead, the people studied carry a plethora of different genetic variants. They do share a lower risk of heart disease and Alzheimer's. And, he says, the data hint that gene variants linked to key cognitive areas may be at play, leading him to ask: "Is cognitive health just one of the components of healthy aging? Or is there something about having a healthy brain that protects against other signs of aging?"

Exactly what happens in the brain as we age is a question Laura Sanders takes up on Page 22. An intriguing idea is that the brain begins to lose the specialization that makes it so efficient in its prime, she reports. Further afield, Susan Milius considers (on Page 26) a hydra and a weed, examining what these outliers of aging can tell us about how aging evolved and how flexible it truly is. Her answer: Very. The sheer diversity in life cycles and declines (see Page 36) gives credence to arguments that while death may come for all of us, a robust old age could well be in the cards for more of us. — *Eva Emerson, Editor in Chief*

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1719 N Street NW, Washington, DC 20036
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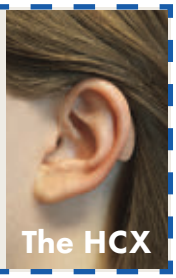
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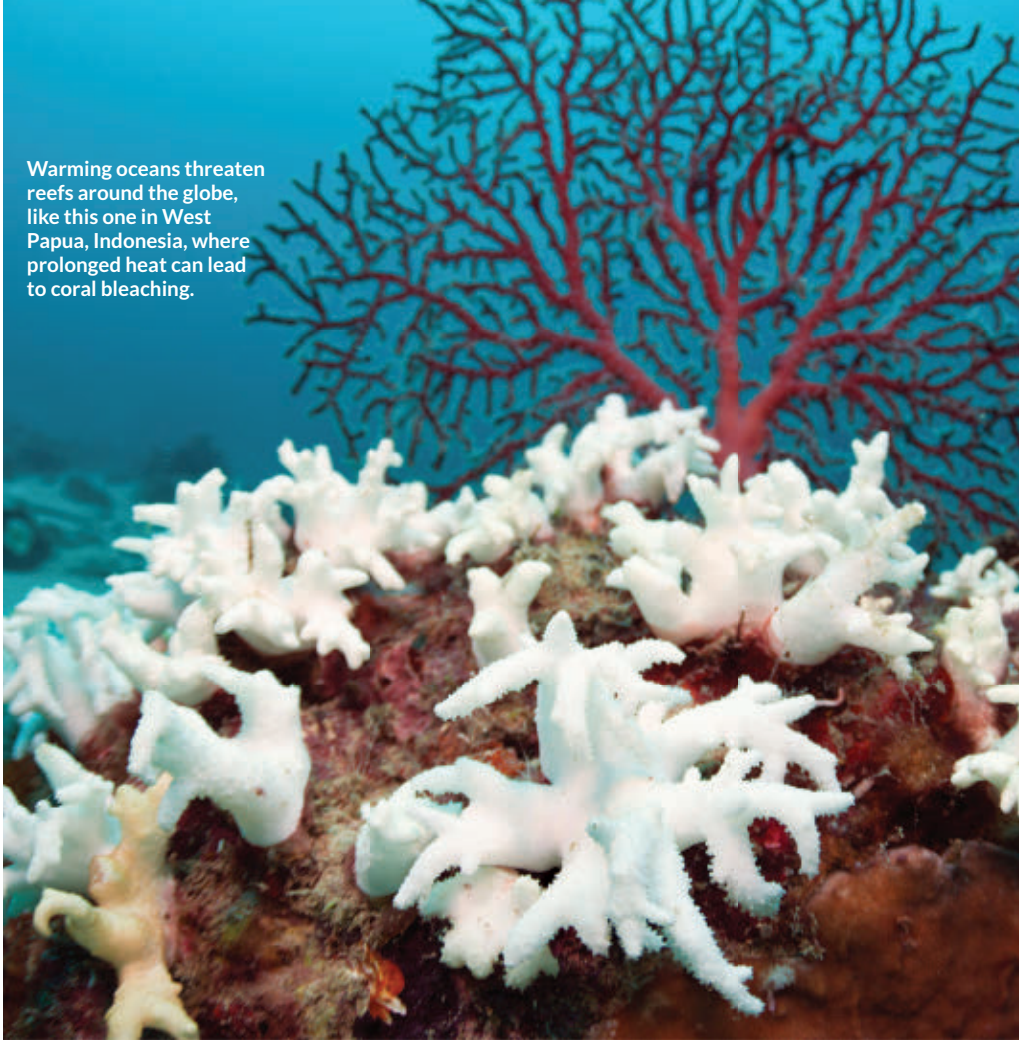
Excerpt from the July 23, 1966 issue of *Science News*

50 YEARS AGO

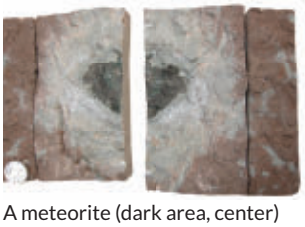
'Hearing' Electrically

A deaf person has been able to "hear" as a result of direct electrical excitation of auditory nerve fibers. But what he can hear may not make much sense.... Although speech-modulated stimuli were not understood, they were unerringly recognized as speech, mainly by rhythm and volume cues.... Optimism about the possibility of an artificial sense organ is now slightly more justifiable.

UPDATE: First developed in the late 1970s, cochlear implants became available in 1984 for deaf people who wanted to hear. Today, more than 300,000 people worldwide wear the small electronic devices, which translate sound into electrical signals that travel directly to the brain via the auditory nerve. The implants don't fully restore hearing, but they enable processing of human speech. The next wave of implants may replace electrical signals with lasers and LEDs to boost the frequencies users can hear and more accurately reproduce sound.



Warming oceans threaten reefs around the globe, like this one in West Papua, Indonesia, where prolonged heat can lead to coral bleaching.



A meteorite (dark area, center) embedded in red limestone may be from the same space crash that formed one of the most abundant meteorite groups found on Earth.

MYSTERY SOLVED

Missing "extinct" meteorite found in Sweden

A long-lost sibling to one of Earth's most common kinds of meteorites has finally been found. The discovery could help scientists piece together a half-billion-year-old hit-and-run, the researchers propose June 14 in *Nature Communications*.

Searching a Swedish quarry, researchers discovered a new variety of meteorite that may have originated from the same cosmic collision that formed L chondrites, which make up around 40 percent of all known meteorites. Scientists think L chondrites are the shards of a giant space rock — maybe an asteroid, maybe not — that collided with an asteroid around 470 million years ago. Although L chondrites are abundant, fragments of the mystery asteroid that hit the L chondrites' parent body have been elusive, making this explanation tough to prove.

Geologist Birger Schmitz of Lund University in Sweden and colleagues uncovered the newly discovered meteorite, nicknamed Öst 65, alongside more than 100 L chondrites. Dating showed that Öst 65 formed within a million years of the L chondrite–forming collision. That timing is close enough to suggest that the two types of meteorites could have been born from the same smashup.

Öst 65 may be the first documented example of an "extinct" meteorite, the researchers say. L chondrites still fall to Earth, but rocks like Öst 65 don't seem to; its parent body may have been obliterated by collisions long ago. — *Thomas Sumner*

FROM TOP: © REINHARD DIRSCHERL/ALAMY STOCK PHOTO; B. SCHMITZ

THE -EST

Coral bleaching event is longest on record

Coral reefs won't be out of hot water anytime soon. A global bleaching event that began in June 2014 is the largest and longest on record. What's worse, it shows no signs of ending.

Global warming exacerbated by the latest El Niño is to blame, National Oceanic and Atmospheric Administration scientists reported June 20 at the International Coral Reef Symposium in Honolulu. Since 1979, periodic mass bleachings over hundreds of coastal kilometers have lasted for "a year or so," says Mark Eakin, coordinator of NOAA's Coral Reef Watch. But recent warm conditions have dragged on for two years, threatening more than 40 percent of reefs globally and more than 70 percent of U.S. reefs.

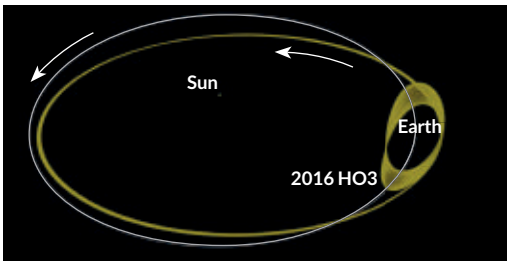
When corals are stressed by heat, they reject the colorful algae living inside them and turn a ghostly white. Without the nutrients and energy provided by the algae, reefs are weaker and can die.

NOAA scientists aren't sure what will end this episode. It could extend into 2017, and more frequent events are possible in the future. "Climate models suggest that most coral reefs may be seeing bleaching every other year by midcentury," Eakin adds. "How much worse that gets will depend on how we deal with global warming."
— Amy McDermott

SAY WHAT?

Quasisatellite \KWAH-zee-SAT-ah-lite\ n.

A body that orbits the sun and appears to orbit Earth



Asteroid 2016 HO3 appears to orbit Earth, but that's just an illusion. As the space rock loops around the sun in an orbit very similar to Earth's (see illustration), it plays leapfrog with our planet, sometimes speeding ahead, sometimes falling behind. The asteroid's suncentric orbit keeps it from qualifying as a full-fledged moon of Earth, but its constant proximity to us is enough to make it the only known "quasisatellite" of our world.

This tagalong was discovered on April 27 in images from the Pan-STARRS observatory in Hawaii. One year on 2016 HO3 is just about 16 hours longer than an Earth year. Earth's gravity keeps the asteroid from wandering; it never strays farther than about 40 million kilometers from Earth and never comes closer than about 14 million kilometers (38 times Earth's distance from the moon).

The tiny rock — no more than about 100 meters across — has probably tagged along with Earth for about a century, and orbital calculations suggest that it will continue to do so for several centuries to come. *— Christopher Crockett*

FOR DAILY USE

Tests turn up dicey bagged ice

Ice isn't always nice.

Tests of 156 bags of ice sold in grocery stores, liquor stores and gas stations across Southern California found that 19 percent exceeded recommended thresholds for bacterial contamination. Researchers also found that 56 percent had detectable levels of mold or yeast. The research was presented in Boston June 17 at ASM Microbe 2016.

About 2 billion bags of ice are sold every year, according to the International Packaged Ice Association, which sponsored the research. IPIA sets ice handling standards, including requiring that ice contain fewer than 500 microbial colonies per milliliter of thawed ice.

The greatest contamination occurred in a sample that contained 24,000 colonies per milliliter, according to Justin Lee, a master's degree student at Cal Poly Pomona in California who presented the independent research. None of the ice adhering to IPIA requirements exceeded acceptable levels of microbes. About 500 of roughly 700 North American companies that sell ice follow IPIA practices and pay a membership fee for testing and auditing to put the IPIA seal (below) on their products. *— Laura Beil*

All bagged ice that meets IPIA standards carries this label.

FROM TOP: JPL-CALTECH/NASA; IPIA; M. UNAL OZMEN/SHUTTERSTOCK

GENES & CELLS

Protein ruins dad's mitochondria

Study of worm sperm sheds light on inheritance mystery

BY LAUREL HAMERS

Scientists have found a clue to why one type of DNA is passed down to children by their mothers — but not their fathers.

DNA inside energy-producing organelles called mitochondria is destroyed in a worm dad's sperm shortly after it fertilizes an egg, researchers report online June 23 in *Science*. A protein called CPS-6 cuts apart the mitochondrial DNA in the male sperm, blocking production of the proteins that the mitochondria need to power the cell. Lingering paternal mitochondrial DNA might hurt developing embryos, the researchers say.

"This is a very long-standing mystery in biology — why in so many organisms, [only] the maternal mitochondria are inherited," says Ding Xue, a geneticist at the University of Colorado Boulder who led the work.

Scientists think that millions of years ago, mitochondria were their own simple cells. Now they produce energy for more complex cells, but they've held on to their own genomes. Their DNA is simpler and shorter than the regular DNA found in the nucleus of the cell.

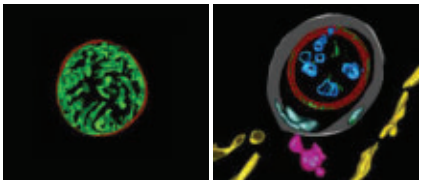
Xue and his collaborators used electron microscopes to watch as sperm from the worm *Caenorhabditis elegans* fertilized eggs. The images showed the paternal mitochondria breaking down from the inside out. To figure out what gene is responsible, the team looked at how that breakdown changed when certain genes weren't working. The identified culprit gene produces the protein CPS-6.

CPS-6 normally controls a process of programmed cell death that helps organisms keep old cells and new cells in balance. But after fertilization, Xue's team found, CPS-6 can also move into the innermost part of the mitochondria and chop the mitochondrial DNA stored there into pieces. That DNA spells out instructions for crucial tasks carried out by the mitochondria. Without the instructions, the mitochondria can't do their job.

CPS-6 doesn't work alone. Other scientists had previously identified a different process, called autophagy, that helps break down paternal mitochondria after fertilization (*SN: 1/1/00, p. 5*). Autophagy recruits specialized structures in the egg that carry away pieces of the paternal mitochondria and break them down, like a garbage collection team. The two seem to work together, the new study shows: Without CPS-6 acting as a flag, the autophagy machinery didn't cart away the unwanted mitochondria as quickly.

"Our study for the first time shows that paternal mitochondria actually cooperate with maternal degradation machinery to ensure that they're all removed," Xue says.

When the mitochondria removal process was delayed, embryos were more likely to die. That finding hints that paternal mitochondrial DNA interferes with normal development, but scientists don't yet know how or why that might be.



Like most cells, worm sperm contain energy-producing mitochondria (one shown with its membrane, red circle, left). Once a sperm fertilizes an egg, the sperm's mitochondria break down inside that membrane (right).

It's also not yet clear whether the mechanism also occurs in humans. "You could imagine there's a similar mechanism, but there's no demonstration yet," says Vincent Galy, a biologist at the Pierre and Marie Curie University in Paris.

The CPS-6 protein is similar to one found in humans that controls cell death in a similar way. Research in flies and mice suggests that exactly when sperm lose their mitochondria varies from species to species, so the process itself probably varies slightly. ■

EARTH & ENVIRONMENT

Antarctic ozone hole now recovering

Simulation filters out natural variation in long-term trend

BY THOMAS SUMNER

A gaping wound in Earth's atmosphere is healing. Since 2000, the average size of the Antarctic ozone hole in September has shrunk by about 4.5 million square kilometers, an area larger than India, researchers report online June 30 in *Science*.

While the hole won't close completely until at least midcentury, the researchers say the results are a testament to the Montreal Protocol. Implemented in 1989, it banned ozone-depleting chemicals called chlorofluorocarbons worldwide.

Tracking the ozone layer's recovery is tricky because natural phenomena such as weather can alter the size of the hole.

MIT atmospheric scientist Susan Solomon and colleagues used a 3-D atmospheric simulation to distinguish between the forces acting on atmospheric ozone. The work suggests that about half of the ozone hole's recent shrinkage resulted from a drop in chlorofluorocarbons in the atmosphere; the remainder stemmed from weather changes.

Volcanic eruptions can obscure healing. Last October, the ozone hole reached a record-setting average size of 25.3 million square kilometers thanks to an eruption of Chile's Calbuco volcano. Without the temporary 4.2-million-square-kilometer boost from the volcano, the hole's average size would have peaked at 21.1 million square kilometers, the researchers estimate. In 2000, the ozone hole's peak monthly average was 24.8 million square kilometers. ■

Q. ZHOU ET AL/SCIENCE 2016

New clues in search for Planet Nine

Location, brightness estimates raise hope of seeing far-off world

BY CHRISTOPHER CROCKETT

More clues about where to search for a possible ninth planet lurking in the fringes of our solar system are emerging from the Kuiper belt, the icy debris field beyond Neptune. And new calculations suggest that the putative planet might be brighter — and a bit easier to find — than once thought.

Evidence for the existence of Planet Nine is scant, based on apparent alignments among the orbits of the six most distant denizens of the Kuiper belt (*SN: 2/20/16, p. 6*). Their oval orbits all point in roughly the same direction and lie in about the same plane, suggesting that a hidden planet, about five to 20 times as massive as Earth, has herded them onto similar trajectories.

Planetary scientists Mike Brown and Konstantin Batygin, both at Caltech, announced this evidence in January. Now they've used it to refine Planet Nine's properties and narrow in on where it might be hiding. Their results appear in the June 20 *Astrophysical Journal Letters*.

Planet Nine's average distance from the sun is most likely between 500 and 600 times as far as Earth's, Brown and Batygin report. Its orbit is highly stretched and tipped by about 30 degrees relative to the rest of the solar system, taking it well above and below the orbits of the eight known planets. And right now, it's probably near its farthest point from the sun — possibly as far as 250 billion kilometers away — in a large patch of sky around the constellation Orion.

But the evidence rests on orbital oddities among just six frozen worlds. "The argument that a planet is there is not ironclad," says Renu Malhotra, a planetary scientist at the University of Arizona in Tucson. "I think it's worth studying. There's enough there to not ignore this evidence," she adds. "We just shouldn't get depressed if the planet's not there."

Malhotra and colleagues have been looking for independent evidence for a ninth planet. And they think they've found some: The orbital periods of those six bodies are roughly synced to one another, her team reports in the same journal. For example, the most distant one, the dwarf planet Sedna, goes around the sun five times in about the same amount of time that its neighbor, 2010 GB174, completes eight orbits. Synced orbits usually hint at some gravitational link among all the bodies involved. But these minuscule worlds are too tiny to affect one another, says Malhotra, suggesting there's a more massive culprit.

A planet at least 10 times as massive as Earth and orbiting the sun once every 17,117 years would be in sync with four of these bodies, Malhotra and colleagues find. That puts Planet Nine, on average, about 100 billion kilometers from the sun, or roughly 665 times the distance between the sun and Earth.

The synced orbits are "very intriguing," says planetary scientist Scott Sheppard of the Carnegie Institution for Science in Washington, D.C. "But they need more of these objects to say if it's statistically significant or not." Sheppard and Chad Trujillo of the Gemini Observatory in Hilo, Hawaii, also suggested, in 2014, that a ninth planet could explain the orbits of a dozen worlds (including the aforementioned six) in the Kuiper belt (*SN: 11/29/14, p. 18*).

"We want to discover more of these smaller [bodies], which are more numerous and can lead to the big one," says Sheppard. He and Trujillo are hunting for remote Kuiper belt objects with telescopes in Chile and Hawaii. They've had some success, adding a few to the census of far-flung lumps of ice. And the orbits of these new discoveries show hints of being aligned with each other and the previously found dozen, he says. But more observations and analysis are needed to be sure. Sheppard and Trujillo have already been allotted several weeks on telescopes this fall, when the constellation Orion (the best guess at Planet Nine's location) is visible. "We'll be looking for more [distant] objects," he says. "And possibly a large planet as well."

If new objects help astronomers zero in on Planet Nine's location, there's a chance that it could be directly seen. Its chilled atmosphere (colder than about −220° Celsius) might contain only hydrogen and helium gas, which are good at reflecting light, Jonathan Fortney, a planetary scientist at the University of California, Santa Cruz, and colleagues report, also in the June 20 *Astrophysical Journal Letters*.

"We expect the planet, if it's there, to be a kind of mirror," Fortney says. Its atmosphere could reflect as much as 75 percent of the sunlight that reaches the planet, Fortney and collaborators report. That would make Planet Nine, depending on its size, bright enough to be detected by the Dark Energy Survey, a project that is scanning for galaxies and supernovas in the southern sky but can also spy on cosmic wanderers closer to home.

"The real problem is knowing where to look," Fortney says.

Brown and Batygin think they've narrowed it down to roughly 2,000 square degrees of sky near Orion. The Subaru telescope in Hawaii could cover that swath in about 20 nights, Batygin says.

"It's a big ask," he says. "But the reward is an expansion of our planetary family." ■

R. HURT/IPAC, CALTECH

Researchers are trying to pin down where to find a hypothetical ninth planet in the solar system (illustrated).

EARTH & ENVIRONMENT

Scientists up tally of deep-sea vents

Hydrothermal fissures may be more common than thought

BY THOMAS SUMNER

The deep, dark ocean bottom teems with far more oases of life than once thought.

Searching along the sunless seafloor where tectonic plates pull apart — regions known as spreading ridges — researchers discovered that heat-spewing hydrothermal vents are at least three to six times as abundant as previously estimated. The finding also boosts the likely number of marine ecosystems huddled around vents, the researchers report in the Sept. 1 *Earth and Planetary Science Letters*.

"The common knowledge of vent field distribution — that they're typically separated by tens or hundreds of kilometers — was not telling the whole story," says Edward Baker, an oceanographer at the University of Washington in Seattle. In reality, vents are spaced around three to 20 kilometers apart along spreading ridges, Baker and colleagues found.

Hydrothermal vents are underwater hot springs. Near tectonic plate boundaries, seawater seeps through the ocean floor and gets heated by molten rock. The hot water then erupts back into the ocean, bringing dissolved minerals such as iron along for the ride. The expunged minerals build smokestack-like towers that host bizarre ecosystems of giant tube worms, eyeless shrimps and ghostly white crabs that thrive in the hot, nutrient-rich water (*SN: 7/25/15, p. 4*).

Discovery of additional vents resolves a mystery about how vent-dwelling sea life spreads, says Duke University oceanographer Cindy Van Dover, who was not involved in the work. Critters that call vents home aren't very mobile and their offspring can't travel far, yet scientists find genetically related communities far apart. The new results show that "there are even more stepping stones than we thought," she says. "This helps us understand the resilience of these communities and how they relocate."

Hydrothermal vents, and associated deep-sea ecosystems (giant tube worms, shown), are more abundant than scientists thought.

Vent hunters typically search for visual signs of the smokelike streams of particles belched by many hydrothermal vents. Puffs of these smoke signals can extend for tens of kilometers horizontally, though, making it difficult to discern between individual vents in closely grouped clusters. Also, many vents at relatively lower temperatures emit few particles, making the vents difficult to spot.

Using a new kind of sensor, Baker and colleagues scanned for short-lived chemicals that all vents expel, such as unoxidized iron and sulfur. The researchers looked for slight changes in the electrical properties of seawater

MATTER & ENERGY

Quantum fragility may help guide birds

Magnetic field's effect on retinal chemistry could aid navigation

BY EMILY CONOVER

Harnessing the weirdness of the quantum world is difficult — fragile quantum properties quickly degrade under typical conditions. But such fragility could help migrating birds find their way, scientists report in the June *New Journal of Physics*. Some scientists think birds navigate with quantum-mechanical compasses, and the new study suggests quantum fragility would enhance birds' sense of direction.

Molecules known as cryptochromes, found within avian retinas, may be behind birds' uncanny navigational skills (*SN: 5/9/09, p. 26*). When light hits cryptochromes, they undergo chemical reactions that may be influenced by the direction of Earth's magnetic field, providing a signal of the bird's orientation.

"At first sight, you wouldn't expect any chemical reaction to be affected by a magnetic field as weak as the Earth's," says study coauthor Peter Hore, a chemist at the University of Oxford. Quantum properties can strengthen a cryptochrome's magnetic sensitivity, but their effect sticks around only for fractions of a second. Any chemical reactions that could signal the bird would have to happen fast enough to skirt a quick breakdown.

But Hore and colleagues' simulations of cryptochromes show that a little bit of quantum deterioration can actually enhance the strength of the magnetic field's effect on the chemical reactions.

According to scientists' theories, light striking a cryptochrome produces a pair of radicals — molecules with a singleton electron. These unpartnered electrons feel the tug of magnetic fields, thanks to a quantum property known as spin, which makes them act a bit like tiny bar magnets. But those magnets are not enough to serve as a compass — instead, the electrons' magnetic sensitivity is the result of a strange quantum dance.

The two radicals' electrons can spin in either the same or opposite directions. But rather than choosing one option, the electrons pick both at once, a condition called quantum superposition. Quantum mechanics can describe only the odds that the electrons would be found in each configuration if forced to choose. As time passes, these probabilities oscillate up and down in a pattern swayed by Earth's magnetic field. These oscillations then affect the rate of further chemical reactions — the details of which are not well understood — which signal to the bird which direction it's facing.

As the electrons interact with their

NOAA

caused by the vent-emitted chemicals. Since the chemicals don't extend as far as the smokelike particle plumes, the researchers could discern between vents about a kilometer or more apart.

Over several trips, the researchers scoured 1,470 kilometers along the East Pacific Rise. The sensor, towed by a ship, drifted within a few hundred meters of the seafloor. In total, 184 distinct vent sites were found, far more than expected based on previous inventories. About a quarter of the sites were particle-poor vents overlooked in previous studies. The finding could also apply to other spreading ridges, such as those in the Atlantic and Indian oceans.

"They're not all big, they're not all the iconic black smokers, but they're places that likely support ecosystems, so there's way more places on the seafloor where animals can survive," Baker says. The impact of the hydrothermal vents on life extends beyond the ocean floor, Baker adds. Iron released from the vents can travel thousands of kilometers and have a global impact on availability of the nutrient. ■

environment, their coordinated oscillations dissipate, weakening their magnetic sensitivity. Hore and colleagues show that some loss of quantumness may be useful. "Not only does it not hurt the compass signal, it can make it stronger," says physicist Erik Gauger of Heriot-Watt University in Edinburgh.

That's because the magnetic field's direction also determines how quickly electrons lose their coordination, further enhancing the difference in the chemical reaction rates based on the bird's direction in the magnetic field. So the magnetic field would do double duty: affect chemical reaction rates by altering the oscillating states of the electrons and determine when they break off their oscillation.

It's still not certain that birds navigate with cryptochromes at all, says computational biophysicist Klaus Schulten of the University of Illinois at Urbana-Champaign. More work is needed to understand how they function. ■

D. EVANS/*JOURNAL OF ARCHAEOLOGICAL SCIENCE* 2016

HUMANS & SOCIETY

Lasers map hidden cities in Cambodia

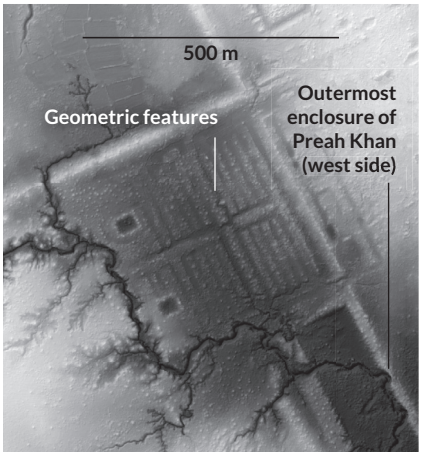
Aerial surveys are revealing extent of ancient Khmer Empire

BY BRUCE BOWER

Thanks to modern technology, Southeast Asia's Khmer Empire is rising from forest floors for the first time in centuries.

New findings show the remarkable extent to which Khmer people built cities and transformed landscapes from at least the fifth to the 15th century, and perhaps for several hundred years after that, says archaeologist Damian Evans of Cambodia's Siem Reap Center. Laser mapping in 2015 of about 1,910 square kilometers of largely forested land in northern Cambodia indicates that gridded city streets and extensive canals emerged surprisingly early, by around the year 500. Evans reports the findings online June 13 in the *Journal of Archaeological Science*. Researchers have generally assumed that large-scale urban development began later at Greater Angkor, capital of the Khmer Empire from the ninth to 15th centuries (*SN: 5/14/16, p. 22*).

A helicopter carrying light detection and ranging equipment, lidar for short, flew sorties over seven Khmer sites in the vicinity of Greater Angkor. Lidar's laser pulses gathered data on the contours of vegetation-covered land. Lidar maps revealed city blocks, canals and other remnants of past settlements.



A laser map of Preah Khan, a 12th century Khmer city in Cambodia, reveals huge earthen embankments arranged in geometric patterns. Similar structures have been found at the Khmer capital, Greater Angkor.

Mysterious ground features previously identified at Angkor Wat temple in Greater Angkor turned up at several sites, some located as many as 100 kilometers from Greater Angkor. Those sites include the eighth to ninth century city of Mahendraparvata and a 12th century city, Preah Khan of Kompong Svay. Fields of precisely arranged earthen mounds may have been used to collect rainwater, Evans speculates. Earthen embankments forming coiled or spiral patterns might have been gardens or ceremonial spaces.

"It's humbling to see the lidar data and realize how much was previously missed in ground surveys," says Mitch Hendrickson of the University of Illinois at Chicago. Hendrickson conducts research at Preah Khan, one of several ancient cities that provided food and other services to Greater Angkor.

Before the 2015 lidar survey, Mahendraparvata was known "only from inscription texts and a few bits of broken-down masonry," adds Charles Higham, an archaeologist at the University of Otago in Dunedin, New Zealand. Mahendraparvata's laser-traced layout indicates it was an early, small-scale version of Greater Angkor, Higham says.

A military invasion and sacking of Greater Angkor in the 15th century apparently did not result in most of its roughly 750,000 residents abandoning the site, as many investigators have thought. Lidar data from 2015 indicate that Khmer capitals established after Greater Angkor's defeat, such as Longvek and Oudong, show no signs of dense populations created by mass relocations from the former capital, Evans says.

That suggests that the political state collapsed at Greater Angkor, but hundreds of thousands of rice farmers carried on, Hendrickson says. "Lots of fish and rice were still available," he says. "Local farmers were more resilient than the state was." ■

BODY & BRAIN

Young brain must learn to feed itself

In infant mice, hungry nerve cells may aid development

BY LAURA SANDERS

Busy nerve cells in the brain are hungry and beckon oxygen-rich blood for sustenance. But in newborn mouse brains, active nerve cells can't yet make this request, and their silence leaves them hungry, scientists report in the June 22 *Journal of Neuroscience*.

Instead of being a dismal starvation diet, this lean time may actually spur the brain to develop properly. The new results, though, muddy the interpretation of the brain imaging technique called functional MRI when it is used on infants.

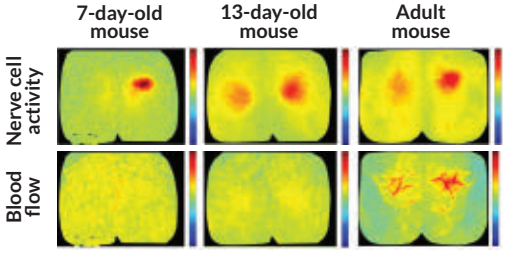
Most researchers had assumed that all busy nerve cells, or neurons, replenish themselves by signaling nearby blood vessels. But there were hints from fMRI studies of young children that their brains don't always follow this rule. "The newborn brain is doing something weird," says study coauthor Elizabeth Hillman of Columbia University.

That weirdness, she suspected, might be explained by an immature communication system. To find out, she and colleagues looked for neuron-blood connections in mice as they grew.

When 7-day-old mice were touched on their hind paws, a small group of neurons in the brain responded instantly, firing off messages in a flurry of activity. Yet no fresh blood arrived. By 13 days, the nerve cell reaction got bigger, spreading across a wider stretch of the brain. Still, the blood didn't come. But by the time the mice reached adulthood, neural activity prompted an influx of blood. The results show that young mouse brains lack the ability to send blood to busy neurons, a skill that influences how the brain operates (*SN: 11/14/15, p. 22*).

Showing that oxygen demands are unheeded during early development is interesting, says neuroscientist Matthew Colonnese of George Washington University School of Medicine and Health Sciences in Washington, D.C. More studies are needed to say whether human infant brains behave similarly and, if so, how this process might sculpt the brain.

The results don't mean that fMRI data from young children aren't valuable, Hillman says. "What we are begging people to do is to make room for this hypothesis, and actually treat it as an opportunity." Although blood flow data may not track with neural activity in newborns, such data may be measuring an important aspect of normal brain development, she says. ■



Wait for it After paw stimulation, nerve cells in the brain of a 7-day-old mouse become active (top row, left), but blood doesn't show up (bottom row, left). A 13-day-old mouse has larger nerve cell responses, but still no blood. The ability to call for blood doesn't appear until adulthood (bottom row, right). Red signals higher nerve cell activity and more blood flow.

BODY & BRAIN

Cocaine users are creatures of habit

Addicts have hard time adjusting their everyday behaviors

BY LAUREL HAMERS

People hooked on cocaine are more likely to stick to other habits, too. They're also less sensitive to negative feedback that tends to push nonaddicts away from harmful habitual behaviors, researchers suggest in the June 17 *Science*.

The findings may help explain why cocaine addicts will do nearly anything to keep using the drug, despite awareness of negative consequences. Treatments that encourage new, healthier habits in place of drug use might be most effective.

"It's such a devastating situation," says psychologist Karen Ersche of the University of Cambridge. Drug users will "tell you they want to change, but still they carry on using the drug."

Habits can be helpful; they free up brainpower for other things. A new driver has to think through every push of the pedal and flick of the turn signal, while experienced drivers can perform these actions almost effortlessly, allowing them to also carry on a conversation. People can snap out of that automation when necessary, slamming on the brakes when a deer darts across the road. But it's harder for cocaine addicts to get off autopilot.

Ersche and colleagues showed sets of animal pictures to 125 people (some cocaine-dependent, others not). In one set of tests, participants learned through trial and error that certain responses to specific pictures would earn them points. In another, responding correctly let them escape an electrical shock.

When pictures that once scored many points no longer did, participants who weren't drug users adjusted and were less likely to choose those pictures. Cocaine users didn't adjust their behavior in the same way. They were also less successful at avoiding shocks.

Scientists don't know whether people who form and keep habits more easily are more likely to become addicts in the first place, or whether drug addiction makes the brain more susceptible to habitual behavior. Charlotte Boettiger, a psychologist at the University of North Carolina at Chapel Hill who has found similar results in her own work on drug and alcohol abusers' habits, suspects both might contribute. Understanding habitual behavior and its neurological basis might someday help scientists develop new medications to supplement behavior-based addiction treatments. ■

HILLMAN LAB/MORTIMER B. ZUCKERMAN MIND BRAIN BEHAVIOR INST./COLUMBIA UNIV.

BODY & BRAIN

Pets could spread drug resistance

Hard-to-treat urinary tract infections on the rise in dogs, cats

BY LAURA BEIL

An increase in drug-resistant urinary tract infections in pets is raising concerns that companion animals may serve as microbe reservoirs that could contribute to the spread of potential superbugs among people and pets.

About four in 10 U.S. households own dogs, which sleep with us, eat off our plates, lick our faces and leave plenty of poop to scoop. Cat ownership is nearly as prevalent.

It's not clear whether pets are picking up the resistant microbes from their owners, or vice versa, said Cátia Marques, a veterinary medicine doctoral candidate at the University of Lisbon in Portugal. More research is needed to answer that question, she said. Marques presented the new research June 20.

Either way, scientists worry that companion animals provide a haven for bacteria to mingle and pick up genes that give them resistance to drugs, said Michael Schmidt of the Medical University of South Carolina in Charleston, who was not involved in the new work. "It is a substantial issue," he said, because pets could contribute to the problem of antibiotic resistance.

Other research has examined human-pet sharing of bacteria, but the subject has been little explored for urinary tract infections, which are extremely common. The new research found a growing resistance in veterinary infections to antibiotics crucial for treating human illness. In one study, samples of the bacterium *Proteus mirabilis* taken from dogs and cats over 16 years in Portugal showed a steady climb in the prevalence of resistant strains. An example: Resistance to a class of drugs known as third-generation cephalosporins grew from 2 percent of samples in 1999–2006 to 20 percent in 2007–2014.

Other research presented by Marques found worrisome multidrug resistance in infections caused by *Klebsiella*. In a third study, which tested for resistance in urinary tract infections in pets across Europe, patterns of drug resistance in dogs and cats tracked that of humans, the researchers found.

In humans, doctors have watched warily as resistance to antibiotics that treat urinary tract infections has grown. In May, scientists reported the discovery of a woman in Pennsylvania with a urinary tract infection resistant to colistin, a rarely used drug of last resort (*SN Online: 5/27/16*). It's not clear how the patient contracted the resistance, but given colistin's role as a last-ditch drug, it raised the specter of an unstoppable microbe.

While the new studies are broader, they aren't the first to raise concerns about difficult-to-treat urinary tract infections in pets. In 2013, German researchers writing in the *Journal of Antimicrobial Chemotherapy* described finding carbapenem-resistant *Escherichia coli* and *Klebsiella* urinary tract infections in six dogs — a discovery later called a phenomenon "of great concern" in a commentary in the same journal. *E. coli* and *P. mirabilis* are the two biggest causes of urinary tract infections in people. Carbapenem, which Marques and colleagues did not test for resistance to, is also considered a drug of last resort for urinary tract infections.

Whether pets are passing resistant microorganisms to their owners or the opposite is true, the findings emphasize that the battle against resistance needs a global strategy that involves veterinarians along with doctors and human patients, Marques said. "We need to have a common public health approach."

Schmidt also cautioned that people who are especially vulnerable to urinary tract infections, such as pregnant women, should take extra care around pets, especially when cleaning up after them.

"If you do have a companion animal and you're prone to these infections," he said, "be very strict with your hand hygiene before you eat." ■

MEETING NOTES

Benign bacterium turns deadly

A deadly infection is puzzling disease investigators. The illness is caused by *Elizabethkingia anophelis*, a bacterium found in soil and water that, until now, has rarely caused problems.

Authorities in Wisconsin reported the outbreak in January. Six people were ill with symptoms that included fever, difficulty breathing and skin inflammation. The outbreak has now spread to 63 people in Wisconsin, one in Illinois and one in Michigan. Most patients are over 65. About 30 percent have died from the infection.

An investigation led by the U.S. Centers for Disease Control and Prevention has not determined how *E. anophelis* spreads or why it has become so fatal. "I don't have any answer for you about the source of this outbreak," said the CDC's Maroya Walters, who discussed the infection June 19. — *Laura Beil*

Tooth decay microbes take many routes to kids' mouths

Moms get blamed for a lot — including their kids' cavities. But new data show that the most common cause of tooth decay, the bacterium *Streptococcus mutans*, doesn't always come from mother-to-child transmission.

Researchers at the University of Alabama at Birmingham studied 119 children and 414 of their household contacts. Contrary to expectation, 40 percent of the kids did not share any *S. mutans* strains with their moms. And 72 percent carried a strain that no other family member had, probably picked up at school, day care or other locations. The research was presented June 17.

The ultimate goal of the research is to learn whether certain strains of *S. mutans* pose a greater hazard for dental health. Knowing that could help identify children who might be in need of more aggressive dental hygiene. — *Laura Beil*

LIFE & EVOLUTION

Cities alter plant, animal evolution

Urban living forces wildlife to adapt at human timescales

BY SUSAN MILIUS

Cities have become great unintentional experiments in evolution. Urban life can alter the basic biological traits of its plant and animal residents, down to the taste of leaves or the stickiness of toes, researchers reported.

For white clover (*Trifolium repens*), leaf taste matters as a defense against grasshoppers and other enemies, Kenneth Thompson of the University of Toronto Mississauga said June 19. Variations in two genes let clover booby-trap its leaves and stem to release a warning burst of cyanide when bitten. A little taste doesn't kill a nibbler but can send it spitting away to another plant.

Clover's cyanide-doping genes are more common in warmer locations. So in Toronto, Thompson expected the elevated temperatures typical of cities to mean more bitter clover downtown than toward the outskirts. He was startled to find just the opposite. The results couldn't be explained by fewer clover eaters downtown. Grazers were no more likely to damage clover at the urban edge than toward the city center, the researchers found.

The same unexpected pattern of cyanide defense increasing away from the city center also showed up in Boston and New York but not Montreal. The explanation, Thompson and his colleagues now propose, lies in what happens during winter.

Urban snow cover can insulate plants from the worst of winter's stinging cold, a special threat for cyanide-carrying clover. Cyanide is toxic to plant tissue as well as to its predators. At temperatures above freezing, though, clover leaves and stems aren't at risk because cells contain only the molecules that will eventually form cyanide. Stored separately, the ingredients usually don't come together in their dangerous form until an animal bites, crushing and ripping cells. Such a defense damages some plant tissue, but the sacrifice can save the rest of the plant. Freezing, however, ruptures the safety system and sets the cyanide loose on the clover for no apparent benefit.

A prodigious snow cover insulates, but in Toronto, Boston and New York, the buildup of urban heat thins the protective snow cover, more so downtown than toward city margins. The closer to the city center a clover plant grows, the less likely it is to set up a cyanide defense and risk poisoning itself during hard freezes, Thompson said. Montreal, however, has such a thick snow cover that even downtown clover can stay pretty well insulated.

The clover study with its multiple cities illustrates one opportunity that analyzing urban adaptation provides. "It's basically a replicated evolution experiment," says Jason Munshi-South of Fordham University in Armonk, N.Y., where his lab group studies local rodents and pigeons. Comparing cities "is where we need to go," he says.

Urban evolution, says Kristin Winchell of the University of Massachusetts Boston, "is a very young field, growing very fast." She presented her latest data June 20 from her ongoing project to put together a comprehensive picture of how a lizard is adapting to urbanization. Puerto Rico's agile, abundant *Anolis cristatellus* lizards have colonized dense cities despite the perilous open expanses between urban trees. Legs of city lizards tend to be 2 to 5 percent longer than those of their forest counterparts. In the urbanites, the toe pads that help the lizards skitter up walls also have more of the specialized gripping scales called lamellae, she and her colleagues reported in the May *Evolution*.

Urban lizards may need all the anatomical advantages they can get to race around cities: The smoothest thing a lizard copes with in the wild, a leaf, is much rougher and easier to grip than a smooth metal surface. Videos of lizards dashing up slanting lab racetracks show varying footwork abilities, typically better among urban residents. While the lizards' origins weren't indicated in the presentation, in one video, a lizard darts up a slippery unpainted aluminum surface, pauses, then shoots to the end, beating a lizard that also stopped and then steadily slid backward.

Differences between city and country lizards persist even in a generation raised in the same environment, Winchell reported. That might mean the two groups of lizards have somewhat different genetic makeups. She still has to test the alternative hypothesis that some carryover effect in gene regulation that mothers pass to their offspring is responsible for the difference.

Winchell has a way to go before putting together the whole picture of how cities change lizards. But however these stories turn out, they have a special pull: They take place on human-scale time. And as Munshi-South puts it, the evolutionary history of urban organisms "is one we created ourselves." ■

After generations of city life, white clover and the widespread tropical lizard *Anolis cristatellus* are not quite the same as their country cousins, though not always in ways that scientists expect.

FROM LEFT: FRANK VINCENT/WIKIMEDIA COMMONS (CC BY-SA 3.0 MIGRATED); GEOFF GALLICE/WIKIMEDIA COMMONS (CC BY 2.0)

Scales share origins with hair, feathers

Skin bumps on reptile embryos resolve evolutionary debate

Bumps (blue dots) on the skin of developing mammals, birds and reptiles point to a shared ancestral structure for hair, feathers and scales. Mouse (left) and Nile crocodile embryos shown.

BY AMY MCDERMOTT

Hair, scales and feathers arose from one ancestral structure, a new study finds.

Studies in fetal Nile crocodiles, bearded dragon lizards and corn snakes appear to have settled a long-standing debate on the rise of skin coverings. Special skin bumps long known to direct the development of hair in mammals and feathers in birds also signal scale growth in reptiles, implying that all three structures evolved from a shared ancestor, scientists report June 24 in *Science Advances*.

In embryonic birds and mammals, some areas of the skin thicken into raised bumps. Since birds evolved from ancient reptiles, scientists expected that modern reptiles would have the same structures. A study last year found that one protein important in hair and feather development is also active in the skin of developing alligators. But the team did not find the telltale skin thickening. Without that evidence, scientists thought the bumps had been lost in reptiles or maybe birds and mammals had evolved them independently using the same set of genes.

The new results are "a relief," says Michel Milinkovitch, whose lab at the University of Geneva led the new study. Scientists had come up with a variety of complicated ideas to explain how birds and mammals could share a structure that reptiles lack.

Clues from a mutant lizard inspired Milinkovitch's team to probe the mystery. Study coauthor Nicolas Di-Poï, now at the University of Helsinki, found that a hair-development gene called *EDA* was present, but disrupted, in scaleless bearded dragons. Di-Poï and Milinkovitch searched for similar molecular signals in normal reptile embryos and found genes and proteins associated with hair and feather growth studding the skin. Cell staining revealed characteristic skin thickening at those signal centers.

Reptilian skin bumps eluded previous researchers because they appear briefly and don't all come in at once as they do in mammals, Milinkovitch speculates.

The next step is to understand how hair, feathers and scales diversified from the same ancestral structure. That primordial body covering wasn't necessarily a scale, says Günter Wagner, an evolutionary biologist at Yale University who coauthored the 2015 study. ■

N. DI-POÏ, M. MILINKOVITCH, ATHANASIA TZIKA/UNIGE 2016

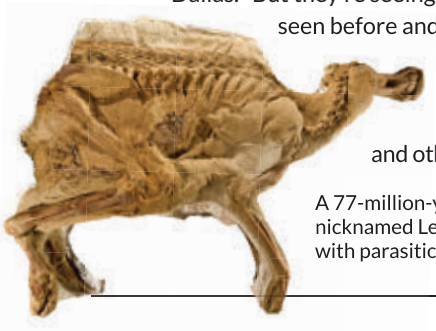
LIFE & EVOLUTION

Parasites wormed way into dino's gut

Inside the blackened gut of a 77-million-year-old dinosaur, scientists have spotted a surprise: the once slimy traces of parasitic worms.

Needlelike burrows snaking through the stomach of a duck-billed dino offer the first hard evidence that gut parasites infected dinosaurs, paleontologist Justin Tweet and colleagues report online June 16 in the *Journal of Paleontology*.

"Maybe they're right, maybe they're not," says paleontologist Anthony Fiorillo of the Perot Museum of Nature and Science in Dallas. "But they're seeing something no one else has seen before and that's pretty awesome."



A 77-million-year-old duck-billed dinosaur nicknamed Leonardo may have been infected with parasitic worms.

Scientists had suspected that, like animals living today, dinosaurs hosted parasites and other microscopic organisms.

"But that doesn't mean that anybody ever expected to see them," says Tweet, a former researcher at the University of Colorado Boulder who now consults for the National Park Service.

Eight years ago, Tweet and colleagues reported the probable stomach contents of a hadrosaur (*Brachylophosaurus canadensis*) nicknamed Leonardo. Leo's gut held small dark flakes that may have once been chewed leaves.

Now, Tweet and colleagues have examined squiggly white tracks, an average of 0.3 millimeters in diameter, among the flakes — 280 tracks in 19 samples of gut material. Closer inspection revealed that the tracks look like tunnels, some marked with thin lines, as if little hairs had once brushed by.

The team also found chemical clues that mucus lined the tunnels and left behind a fossilized trail of slime. Tweet thinks tiny, mucus-secreting worms with fine bristles might have once burrowed in Leonardo's belly.

Reexamining old fossils could reveal if other dinosaurs also show signs of possible worms, Fiorillo says. Tweet's paper "illustrates the beauty of the fossil record and how much more we still have to learn from it — if we just keep our eyes open." *— Meghan Rosen*

BODY & BRAIN

Well-timed exercise aids memory

If you want to lock new information into your brain, try working up a sweat four hours after first encountering the info.

This precisely timed trick, described online June 16 in *Current Biology*, comes courtesy of 72 people who learned the locations of 90 objects on a computer screen. Some people then watched relaxing nature videos; others worked up a sweat on stationary bikes, alternating between hard and easy pedaling for 35 minutes. This workout came either soon after the cram session or four hours later.

Compared with the couch potatoes and the immediate exercisers, people who worked out four hours after learning better remembered the objects' locations two days later. The delayed exercisers also had more consistent activity in the brain's hippocampus, an area important for memory, when they remembered correctly. That consistency indicates that the memories were stronger, Eelco van Dongen of the Donders Institute in the Netherlands and colleagues propose.

The researchers don't yet know how exercise works its memory magic. But they suspect molecules sparked by aerobic exercise, including the neural messenger dopamine and the protein BDNF, help solidify memories by reorganizing brain cell connections. *— Laura Sanders*

EARTH & ENVIRONMENT

Successful helium hunt suggests element is not yet running out

The world's known helium reserves just ballooned. Applying techniques from the oil industry, scientists uncovered a reservoir of over a trillion liters of helium gas beneath Tanzania. That's enough to satisfy the world's helium needs for about seven years, the researchers announced June 28 at the Goldschmidt Conference, a geochemistry meeting held in Yokohama, Japan. The find may allay fears that a global helium shortage will hit when the U.S. Federal Helium Reserve — currently the world's largest helium source — runs dry in the next few years.

Helium accumulates underground during the radioactive decay of unstable elements. That helium can be liberated when surrounding rock melts during volcanic activity. Using this information and seismic imaging of gas-trapping formations, Diveena Danabalan of Durham University in England and colleagues found five spots in a volcanic region of Tanzania where water and helium-rich gas bubble to the surface from underground reservoirs. The team predicted that it can find more helium reservoirs elsewhere.

Society's helium needs go far beyond making balloons float and voices sound squeaky: Helium is essential for scientific research and is a crucial component of MRI medical scanners' cooling systems. *— Thomas Sumner*

ATOM & COSMOS

Juno now in orbit around Jupiter

Jupiter has a new visitor. After traveling 2.8 billion kilometers across space, NASA's Juno probe — a mission to investigate Jupiter's deep interior (*SN: 6/25/16, p. 16*) — arrived at the giant planet July 4.

Juno's scientific instruments were switched off on June 29 before the probe slid into its first of 37 orbits, so there are no pictures to celebrate its arrival. Scientists won't get their first intimate look until Juno swoops in again on August 27, with all of its instruments operating.

After one more 53-day loop around Jupiter, Juno will start a series of 14-day orbits in October that will take the spacecraft over the north and south poles while soaring just 5,000 kilometers from the tops of the clouds that enshroud the planet. *— Christopher Crockett*

INTRODUCING

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SLOWING TIME

Fighting aging may enhance health, not longevity

By Tina Hesman Saey

On the inevitability scale, death and taxes are at the top. Aging is close behind.

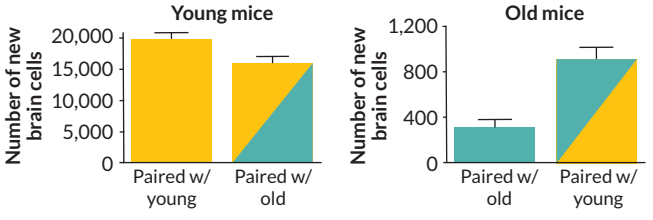
It's unlikely that scientists will ever find a way to avoid death. And taxes are completely out of their hands. But aging, recent research suggests, is a problem that science just might be able to fix.

As biological scientists see it, aging isn't just accumulating more candles on your birthday cake. It's the gradual deterioration of proteins and cells over time until they no longer function and can't replenish themselves. In humans, aging manifests itself outwardly as gray hair, wrinkles and frail, stooped bodies. Inside, the breakdown can lead to diabetes, heart disease, cancer, Alzheimer's disease and a host of other problems.

Scientists have long passionately debated why cells don't stay vigorous forever. Research in mice, fruit flies, worms and other lab organisms has turned up many potential causes of aging. Some experts blame aging on the corrosive capability of chemically reactive oxygen molecules or "oxidants" churned out by mitochondria inside cells. DNA damage, including the shortening of chromosome endcaps (called telomeres) is also a prime suspect. Chronic, low-grade inflammation, which tends to get worse the older people get, wreaks so much havoc on tissues that some researchers believe it is aging's prime cause, referring to aging as "inflammaging." All these things and more have been proposed to be at the root of aging.

Some researchers, like UCLA's Steve Horvath, view aging as a biological program written on our DNA. He has seen evidence of a biological clock that marks milestones along life's path. Some people reach those milestones more quickly than others, making them older biologically than the calendar suggests. Others take a more leisurely stroll, becoming biological youngsters compared with their chronological ages.

Odd couples Scientists sewed together mice to share blood supplies. Young mice paired with old mice (left chart, two-toned) made fewer new cells in the brain's hippocampus than when paired with another young mouse (yellow). Old mice made hundreds more new brain cells when paired with a young mouse (right chart, two-toned) than when paired with an old mouse (teal). SOURCE: S. VILLEDA *ET AL*/*NATURE* 2011



OPPOSITE: DAVID CURTIS

Special Report: Aging's Future

The latest studies reveal exciting prospects for slowing the effects of aging. But scientists are still divided on some fundamentals: What is aging? How does it change the brain? How did different life histories evolve? This special report addresses those questions and more, here and on the following pages:

Many others, including Richard Miller, a geroscientist at the University of Michigan, deny that aging is programmed. Granted, a biological clock may measure the days of our lives, but it's not a ticking time bomb set to go off on a particular date. After all, humans aren't like salmon, which spawn, age and die on a schedule.

Instead, aging is a "by-product of running the engine of life," says biodemographer Jay Olshansky of the University of Illinois at Chicago. Eventually bodies just wear out. That breakdown may be predictable, but it's not premeditated.

Despite all the disputes about what aging is or isn't, scientists have reached one radical consensus: You can do something about it. Aging can be slowed (maybe even stopped or reversed). But exactly how to accomplish such a counterattack is itself hotly debated. Biotechnology and drug companies are developing several different potential remedies. Academic scientists are investigating many antiaging strategies in animal experiments. (Most of the research is still being done on mice and other organisms because human tests will take decades to complete.)

Even researchers who think they have finally come up with real antiaging elixirs say they don't have the recipe for immortality, though. Life span and health span, new research suggests, are two entirely separate things. Most researchers who work on aging aren't bothered by that revelation. Their goal is not necessarily extending life span, but prolonging health span — the length of time people live without frailty and major diseases.

Aging as disease

Many health problems are so commonly associated with aging that some researchers take the highly controversial stance that

aging itself is a disease, says Saul Villeda of the University of California, San Francisco.

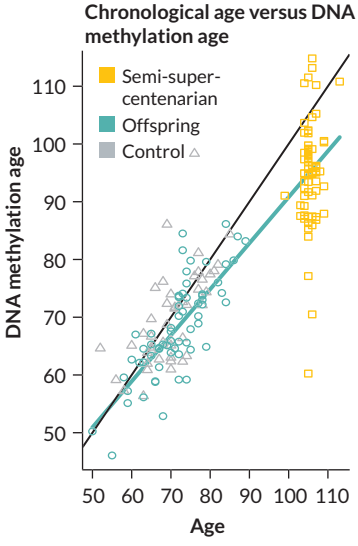
If aging is a disease, in Villeda's lab it's almost a contagious one: He can artificially spread aging from old lab mice to young ones. One mode of aging transmission is to give genetically identical mice transfusions of young or old blood. In another approach, researchers sew together pairs of mice so that their blood vessels will join up and link their circulatory systems.

This artificial joining of two separate animals, known as parabiosis, was a staple of physiology experiments for over a century before Irina Conboy got the idea to pair an old mouse with a young one. Conboy, a stem cell researcher at the University of California, Berkeley, made headlines with her experiments.

Those headlines focused on the good news: Young blood rejuvenated old mice. In further studies by other researchers, infusions of young blood made broken bones in old mice heal better (*SN Online: 5/19/15*), gave their muscles extra spring and improved their memories (*SN: 5/31/14, p. 8*). Apparently some substances in the blood triggered the rejuvenation. Some candidates for those rejuvenation factors have been identified, although none are universally agreed on.

But news accounts mostly ignored the flip side of the experiment: Being tethered to an old mouse made young mice age faster. One substance in the blood of old mice, a protein called Beta-2-microglobulin, or B2M, seemed to prematurely age the young ones, Villeda and colleagues reported last year in *Nature Medicine* (*SN: 8/8/15, p. 10*). Parabiosis experiments don't last very long, so no one knows whether youth or decrepitude will win in the end — or if the two mice would have settled into middle age together.

UCLA's Horvath has evidence that the mice may never totally sync. He monitors aging by examining molecular tags called methyl groups, which attach to various locations on DNA in a process called methylation. Methylation is an epigenetic modification of DNA. Such modifications work something like flagging passages in a book with sticky notes. Attaching a tag doesn't change the information in the book — it just draws attention to some passages and signals that others should be ignored.



Taking their time
Semi-supercentenarians (people who live to be 105 to 109, yellow) tend to be biologically younger than their chronological age. So do their children (teal) compared with other people (gray). The rate of aging of the semi-supercentenarians and their children (teal line) is slower than normal (black line). SOURCE: S. HORVATH *ET AL/ AGING* 2015

Like other supercentenarians, Emma Morano, 116, seems to age more slowly than other people.

Horvath measures DNA methylation changes at 353 different spots in the human genetic instruction book, or genome. As people age, 193 locations accumulate tags, like playbills plastered on urban buildings. At 160 others, methylation is gradually stripped away with age. Knowing how much methylation is normally found at each spot at a given chronological age allows Horvath to calculate biological age.

Some people age at different rates than others, he discovered. For instance, semi-supercentenarians — people who live to be 105 to 109 — are about 8.6 years younger epigenetically than their chronological age. Their children are slow to age, too, though not as slow as their parents. Epigenetic clocks indicate that the offspring are about five years younger biologically than other people of the same chronological age.

People often joke about certain abilities, such as eyesight, memory or hearing being "the first to go." Some of Horvath's work suggests that the notion isn't entirely far-fetched. He calculated the epigenetic age of specific organs and discovered that body parts can age at different rates. The cerebellum, the part of the brain that sits at the top of the brain stem and helps coordinate movement, speech and other activities, ages the slowest of the brain regions that Horvath analyzed. While there are natural differences in organ aging, some conditions, such as HIV infection and obesity, can prematurely age certain organs, Horvath and colleagues have found.

These experiments demonstrate that aging and its effects are malleable. "Aging is really plastic — it's not set in stone," says Conboy. Consequently, she and other researchers agree, something can be done to slow aging, or perhaps turn it around entirely. But exactly what can be done is vigorously disputed.

Interpret with caution

Most scientists working on aging urge caution in extrapolating promising results in animal studies to humans. For instance, one of the most promising early candidates for a rejuvenation factor from young blood was a protein called GDF11. Reports in 2013 and 2014 concluded that GDF11 levels in blood decline with age; restoring the protein in old animals could reverse some heart problems, improve muscle strength and spur nerve cell growth in the brain. Since those reports, other researchers have disputed the protein's revitalizing powers. In a recent study, researchers measured GDF11 levels in 140 people aged 21 to 93. Levels of the protein didn't decline with age, Mayo Clinic researchers reported in the June 14 *Cell Metabolism*.

FROM TOP: ANTONINO DI MARCO/ANSA VIA AP; M. TELFER

A rushed, maybe risky, telomere test

Elizabeth Parrish was growing old before her time. The 45-year-old chief executive of BioViva USA Inc. had the telomeres of a 65-year-old.

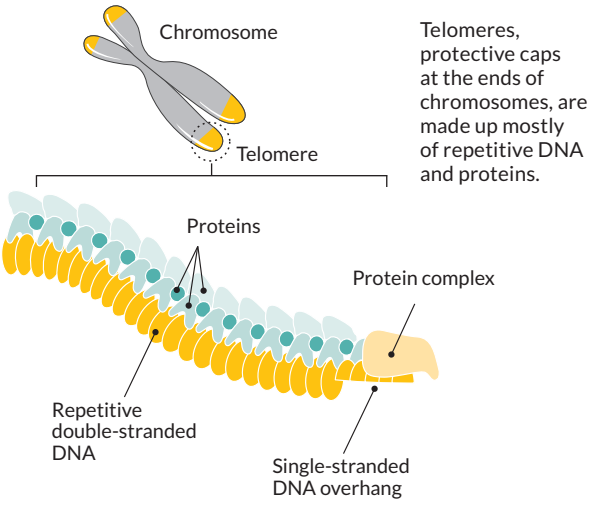
Telomeres are long stretches of repetitive DNA at the ends of chromosomes. They work like plastic aglets on shoelaces, preventing chromosomes from fraying and getting chewed up. When telomeres get too short, the risk of dying increases (*SN: 12/15/12, p. 13*). Mice in which telomeres have been lengthened by gene therapy tended to live longer in some studies than mice that didn't get the treatment.

Parrish and researchers at her company decided it was time to see if stretching telomeres in people could also lengthen life. Too impatient to wait for Food and Drug Administration approval for clinical trials in people, Parrish went to Colombia to test her company's antiaging gene therapy on herself.

An intravenous infusion delivered viruses carrying a gene for a telomere-lengthening enzyme into her bloodstream. The gene therapy was also designed to inhibit action of the myostatin gene, which stops muscle cells from growing. Inhibiting that gene may allow muscles to better repair themselves. Parrish said she had "myriad injections we didn't even count — we would have lost count if we had" — into her muscles and other parts of her body. Her aim was to demonstrate that the therapy is safe. "I felt like we really had to take the first risk," she says.

In April, BioViva reported in a news release that telomeres in Parrish's white blood cells were longer, now consistent with those of a 45-year-old instead of those of a retiree. No one knows how the treatment will affect Parrish's health or life span. Some scientists say her risk of cancer may increase, a concern the company dismisses.

"It's absolutely premature" to conduct telomere therapy in people, says Matt Kaeberlein, a geroscientist at the University of Washington. "We don't have the technology to do this in a safe way. We don't even understand the biology that well," he says. "It's absurd for somebody to be doing this, and trying to get other people to participate is dangerous, in my view." — *Tina Hesman Saey*



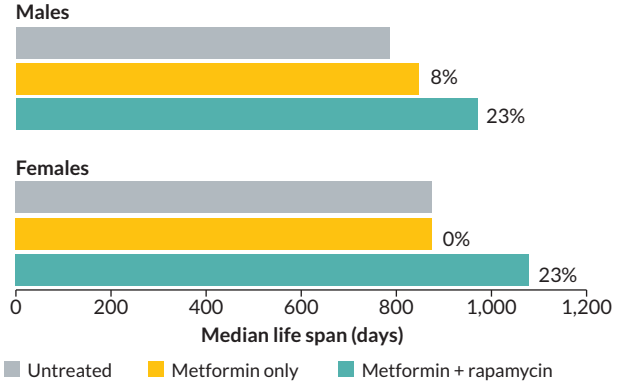
Telomeres, protective caps at the ends of chromosomes, are made up mostly of repetitive DNA and proteins.

Previous researchers may have gotten GDF11 mixed up with a similar protein called myostatin, which does dip as people get older. Not only does GDF11 not decline with age, having too much of it could be bad, the Mayo team found. People with higher blood levels of the protein were more likely to be frail, have diabetes and heart problems, and have a more difficult time recovering from surgery than people with lower levels of the protein.

Beyond the blood experiments, scientists have examined various ideas about what goes wrong in aging and have devised strategies to counteract it. For instance, some evidence suggests that stem cells run out of steam as they get older. Restoring old stem cells to youthful vigor may enable them to repair or replace damaged tissues and turn back the biological clock. Keeping stem cells youthful may involve sheltering them from inflammation or things that could damage their DNA.

One way to keep stem cells and other cells working is to avoid the loss of telomeres capping the ends of chromosomes. As cells divide, their telomeres grow shorter until they are so short that chromosomes can no longer safely replicate. That may be a signal for the cell to shut down or die. So some researchers think that lengthening telomeres could give cells the protection needed to survive longer.

One biotechnology company executive flew from the United States to Colombia to try out her company's gene therapy for lengthening telomeres (see sidebar, above). That decision bypassed U.S. government and



Life extension elixir? Male mice receiving the diabetes drug metformin (top, yellow) had median life spans (the age at which half the population has died) that were slightly longer (8 percent) than untreated male mice (top, gray). Metformin did not increase life spans of female mice. Combined with rapamycin, metformin (teal) boosted median life span in both sexes about 23 percent, but the result wasn't statistically different from life span boosts in mice given rapamycin alone in previous studies.
SOURCE: R. STRONG *ET AL/AGING CELL* 2016

FROM TOP: E. BLACKBURN *ET AL/SCIENCE* 2015; M. TELFER

other safety measures designed to protect human study participants. And no one knows whether it will work or doom her to cancer, which often relies on long telomeres to keep growing.

Other researchers are exploring more measured approaches to antiaging therapies. One study in dogs is testing rapamycin, the first drug shown to lengthen mouse life spans. Rapamycin is an immune suppressant that also has anticancer effects. The rationale for using it came from research on caloric restriction, the world-champion method for making animals live longer. Animals on calorie-restricted diets typically eat at least 25 percent fewer calories than normal. Such low-cal treatment has increased life spans in mice, dogs, fruit flies, yeast, worms and other lab organisms. Results from primate studies have been mixed (*SN: 8/1/09, p. 9; SN: 10/6/12, p. 8*). Some people have put themselves on caloric-restriction regimes (*SN: 10/25/08, p. 17*). A handful of studies suggest that those people have better health, but it's too soon to know whether they will outlive their peers.

Exactly why drastically reducing food intake can extend life isn't known. But researchers have good evidence that a series of biochemical reactions known as the mTOR pathway is involved. The protein mTOR helps monitor nutrient levels in cells and regulates cell movement, protein production, and cell growth and survival. When starvation sets in, cells turn off mTOR's activity, which allows a self-cannibalizing process called autophagy to scavenge nutrients by digesting some of the cell's internal organs. This internal garbage disposal and recycling method also removes old, worn-out mitochondria and proteins that may otherwise keep cells from functioning efficiently. That process and other cellular activities governed by mTOR may be responsible for making cells, and organisms, live longer.

Rapamycin gave mTOR its name — mechanistic target of rapamycin. Giving the drug might do what caloric restriction does without requiring superstrict diets (*SN: 6/4/11, p. 22*). Matt Kaeberlein, a geroscientist at the University of Washington, and colleagues conducted a safety study of the drug last year in 24 dogs. The study was only 10 weeks long, so the researchers can't yet draw any conclusions about long-term effects on aging. But the dogs had no major side effects from taking low doses of the drug, a worry because rapamycin impairs immune system function and could make animals (including people) who take it more vulnerable to infection or cancer.

Rapamycin's drawbacks make it unattractive for human studies. The diabetes drug metformin may instead be the antiaging drug of choice for people, says gerontologist Nir Barzilai. In addition to mTOR, metformin targets an insulin-like growth protein known as IGF-1. That protein has been implicated in a variety of biological processes that promote aging.

Barzilai, of Albert Einstein College of Medicine in New York City, and researchers at more than a dozen centers around the country plan to test metformin for its ability to fight aging in people 65 to 79. Barzilai and colleagues laid out the case for using metformin in the June 14 *Cell Metabolism*. Metformin is generally safe, with few major side effects. It has been shown to improve a variety of health measures and to impair cancer development in people with type 2 diabetes (*SN: 11/30/13, p. 18*). Barzilai says the drug may help people who don't have diabetes also live healthier when they are elderly. If it does, commercials touting metformin might have to add another disclaimer, he jokes. "The commercials will go on: 'This will make you healthy, but we have to apologize because you might live longer.'"

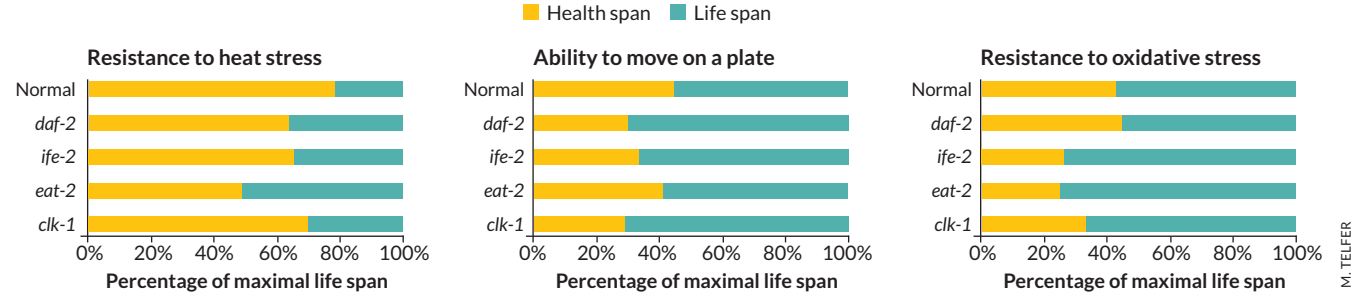
But studies of mice suggest that disclaimer may not be necessary. Research by Miller and others suggests that metformin may not prolong life. They have been dosing mice with various chemicals, including metformin and rapamycin, looking for drugs that will make mice healthier and live longer. In a new study, published online June 16 in *Aging Cell*, Miller and colleagues fed mice metformin starting when the rodents were 9 months old — middle age for a mouse. Combining metformin and rapamycin didn't make the mice live much longer than rapamycin alone did in previous trials.

Cellular zombies

Other researchers are hoping to stave off death by getting rid of the undead. Cellular zombies called senescent cells are stressed cells that have entered a type of stasis — they're not dead, but they're not functioning either. Stress for cells usually means

Live longer, but frailer Mutations in certain genes (*daf-2*, *ife-2*, *eat-2* and *clk-1*) can make *Caenorhabditis elegans* worms live longer than normal worms without these mutations. But researchers examining health measures, such as the worms' ability to move or to withstand heat or oxidative stress, found that long-lived mutant worms spend a smaller proportion of their lives as healthy animals (indicated in yellow).

SOURCE: A. BANSAL *ET AL*/*PNAS* 2015



M. TELFER

severe DNA damage that could produce cancer, critically short telomeres or other molecular catastrophes that trigger shutdown mode.

That lockdown is for the greater good, says aging researcher Judith Campisi, who studies senescence at the Buck Institute for Research on Aging in Novato, Calif. "It's protective," she says. "You don't want defective cells to propagate." (When damaged cells continue to grow they may become cancerous.)

Unfortunately, says Campisi, the senescent cells don't die. Instead they send out messages to neighboring cells: "Hey, there's a problem. Be prepared. What happened to me could happen to you." Such messages are probably intended as public service announcements, but they could trigger mass panic and inflammation. Like zombies putting the bite on the living, senescent cells damage surrounding cells and accelerate aging.

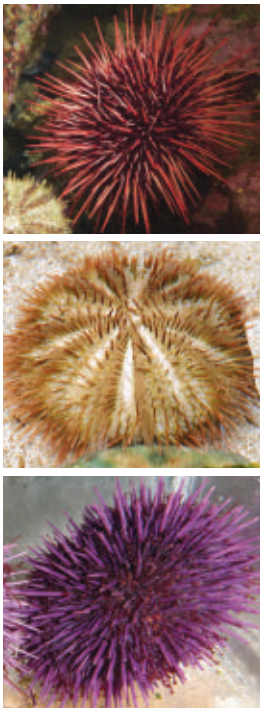
Researchers have worked out methods for hitting the zombie cells with genetic shots to the head, effectively destroying the cells and removing them from the body. Mice from which senescent cells have been removed had increased median life span and improved health, researchers reported in *Nature* in February (*SN: 3/5/16, p. 8*).

Campisi and other researchers are working on ways to clear senescent cells from humans, too. But no antiaging treatment makes mice or any other animal live forever. Researchers have yet to increase a mouse's life span (which rarely goes above two years) to five years, although one mouse fell just short of that mark.

Much research suggests that things that extend life span, such as rapamycin, might not stretch health spans. Mutations that make millimeter-long transparent worms known as *Caenorhabditis elegans* live longer also extend the proportion of their lives the worms spend being frail, Heidi Tissenbaum of the University of Massachusetts Medical School in Worcester and colleagues reported last year in the *Proceedings of the National Academy of Sciences*.

But living healthy doesn't guarantee longevity either, a new study of sea urchins suggests. Red sea urchins (*Mesocentrotus franciscanus*) live well past 100 years old in the wild, while purple sea urchins (*Strongylocentrotus purpuratus*) make it to 50. But variegated (also called "green") sea urchins (*Lytechinus variegatus*) normally die after four years.

The difference in the species' life spans might be due to different rates of aging, thought aging researcher Andrea Bodnar at the Bermuda Institute of Ocean Sciences in St. George's and developmental biologist James Coffman of the MDI Biological Laboratory in Salisbury Cove, Maine. Instead, they found, none of the species seem to age at all. Young and old members of each species are similar in their abilities to reproduce and to regenerate spines and tube feet, the researchers reported online April 20 in *Aging Cell*. Even though the short-lived variegated urchins have no signs of slowing down, they still die. Why is a mystery, Coffman says.



Sea urchins have vastly different life spans, although none seem to age. Red sea urchins (*Mesocentrotus franciscanus*, top) live over 100 years. Variegated sea urchins (*Lytechinus variegatus*, middle) live only about four years. Purple sea urchins (*Strongylocentrotus purpuratus*, bottom) die after about 50 years.

FROM TOP: C. SHUSTER; © HANS HILLEWAERT/WIKIMEDIA COMMONS (CC BY-SA 4.0); C. SHUSTER

Ways to be wellderly

A similar paradox is also seen in "wellderly" people that geneticist Ali Torkamani has been studying at the Scripps Research Institute in La Jolla, Calif. About eight years ago, Torkamani started bringing in people over 80 who had made it to an advanced age without any sign of chronic disease. The idea was to study their DNA and learn the secrets of healthy aging.

Despite living healthy, the wellderly didn't carry genetic variants connected with extremely long lives, Torkamani and colleagues discovered. The wellderly also had no genetic advantage when it comes to cancer, stroke or diabetes. What they did have was a lower risk of getting Alzheimer's and heart disease. Each of the wellderly seemed to have their own genetic recipe for success, suggesting there are lots of ways to stay healthy into old age. The researchers didn't rule out that diet and lifestyle also help. "There's hope for everybody," Torkamani declares.

But his cloud of optimism may have a tarnished lining. His findings, along with the sea urchin and worm results, suggest that aging and longevity aren't the same things. If that's the case, it would mean that stopping aging would not extend human life span by much. The oldest (verified) person to have ever lived was Jeanne Louise Calment, a French woman who died at age 122 in 1997. People might top out at 130 if aging is controlled (and most people still would not make it that long because they just don't have the necessary makeup). As a species, humans probably can't go further without changing whatever controls longevity too, some researchers think.

Exactly how long people can live won't be answered until proven antiaging therapies are developed. If aging and longevity are linked, then treating aging could very well make people live longer, healthier lives. If they are separate phenomena, then people could forgo the cancer, heart disease and other ailments of aging, but they would still have limited life spans. In that case *Star Trek*'s Mr. Spock might need to revise his usual parting words. When talking to humans, he should wish that they will live long *or* prosper. We may not get both. ■

Explore more

- Carlos López-Otín *et al*. "The Hallmarks of Aging." *Cell*. June 6, 2013.

The mature mind

When it comes to the brain, aging starts early

By Laura Sanders

If you've ever watched a baby purse her lips to hoot for the first time, or flash a big, gummy grin when she sees you, or surprise herself by rolling over, you've glimpsed the developing brain in action. A baby's brain constructs itself into something that controls the body, learns and connects socially.

Spending time with an older person, you may notice signs of slippage. An elderly man might forget why he went into the kitchen, or fail to anticipate the cyclist crossing the road, or muddle medications with awkward and unfamiliar names. These are the signs of the gentle yet unrelenting neural erosion that comes with normal aging.

These two seemingly distinct processes — development and aging — may actually be linked. Hidden in the brain-building process, some scientists now suspect, are the blueprints for the brain's demise. The way the brain is built, recent research suggests, informs how it will decline in old age.

That the end can be traced to the beginning sounds absurd: A sturdily constructed brain stays strong for decades. During childhood, neural pathways make connections in a carefully choreographed order. But in old age, this sequence plays in reverse, brain scans reveal. In both appearance and behavior, old brains seem to drift backward toward earlier stages of development. What's more, some of the same cellular tools are involved in both processes.

Probing the connections between growing and aging may reveal how time affects the brain. And with a deeper understanding of brain aging, and the tools involved, scientists might be able to slow — or even stop — mental decline.

That's a lofty goal, made even more challenging by the multitude of theories from a diversity of researchers that aim to explain why and how the brain ages. Everybody focuses on a different aspect of the aging brain, leaving no one with a sense of the whole process, says epigeneticist Art Petronis of the Center for Addiction and Mental Health in Toronto. It's like people trying to put together a giant jigsaw puzzle from separate rooms, each with only a few pieces in hand. So far, people studying how the brain ages have found only the evidence they can grab.

Petronis and others are intrigued by the idea that the brain's early life holds clues to its end. "You see blips here and blips there," he says. "This critical mass is accumulating."

Other scientists, including Caleb Finch of the University of Southern California, in Los Angeles, caution against falling for

As part of the Lothian Birth Cohort, John Scott took an intelligence test as a child in 1947. When compared with later testing, the results have provided clues about how the brain ages. Here, Scott, a retired miner, looks at a model of his white matter tracts.

NATIONAL MUSEUMS SCOTLAND

appealing but overly simple explanations for aging. As a gerontologist who has been thinking about aging for 50 years, he has seen aging theories come and go, a perspective that makes him skeptical that the complex process can be reduced to the notion that it's just development in reverse. "The more we poke into biology, the more wondrously complex it is," he says.

Nonetheless, there's something to the notion that aging starts early. "We are born dying," Finch says. And poking at that idea just might lead somewhere.

Head start

When the human brain makes its first appearance in the third week of gestation, it is no more than a minuscule smear of indistinct cells. This glob then grows at a furious rate up through the preschool years. At the same time, these accumulating brain cells begin to take on specific jobs, changing from generalists to specialists. Nerve cells are born and migrate to their final destinations, linking up in precise order to form the high-speed neural connections that enable memory, emotion and thought. And scientists now realize that the way the brain is built has lifelong effects.

In 1932 and 1947, nearly every Scottish 11-year-old sat down to take an intelligence test. Decades later, their scores have matured into academic gold, offering scientists a rare opportunity to see how intelligence fares with age. In 1999, scientists led by Ian Deary of the University of Edinburgh got back in touch with as many of the long-ago test takers as possible, forming a group of more than 1,000 people — ranging in age from 80 to 95 — called the Lothian Birth Cohort. Deary and colleagues have studied the group in detail, and one factor rises above the rest: People with higher intelligence scores at age 11 were more likely to have better thinking skills in old age.

> "There is a large range of mostly small influences on people's cognitive aging."
>
> IAN DEARY

Childhood intelligence wasn't the only factor, though. From the start, Deary and his colleagues cast a wide net, imaging participants' brains and examining genetics, lifestyles, health and social factors. "We were right to do so, because there is a large range of mostly small influences on people's cognitive aging," he says. But the fact that intelligence at age 11 can partially predict who will be sharp into their 90s suggests that a long-lasting brain must be solidly constructed.

One way in which the brain is built well involves its white matter — tracts of tissue that connect distant brain regions, allowing for quick communication. And in fact, members of the Lothian Birth Cohort with healthier white matter in old age, measured by an MRI-based brain scan method called diffusion tensor imaging, performed better on tests of brain function, Deary and colleagues found.

Mature neural highways take decades to develop. Brain areas are still solidifying into a person's thirties. The later-blooming brain regions oversee jobs like impulse control and judgment, two well-known weak spots among teenagers.

These slow-to-grow brain networks are the first to go in old age, neuroscientist Gwenaëlle Douaud of the University of Oxford and colleagues found. Networks of nerve cells (the gray matter) are guided by a "last in, first out" rule, brain scans of 484 people from 8 to 85 years of age indicate. "What we show is a precise mirroring for these regions," she says.

These networks, which reach their peak around age 39 for men and age 41 for women, handle sophisticated jobs, like merging multiple kinds of information together, she says. And sure enough, people with seemingly healthier neural connections had better memories, Douaud's team reported in the *Proceedings of the National Academy of Sciences* in 2014.

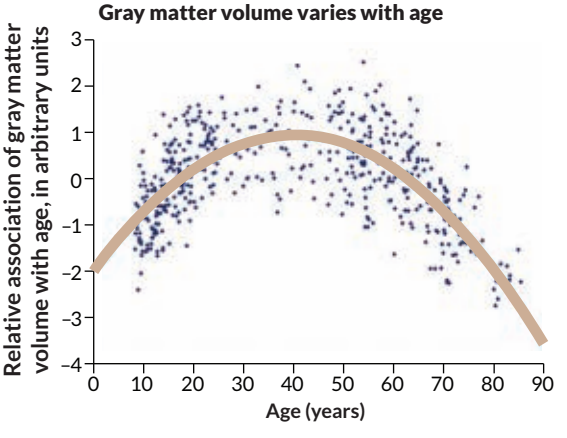
Special no more

As neural connections come and go with age, brain cells themselves change in a way that harkens to the brain's early days. Human brain cells are a dazzlingly diverse crew that handle a variety of jobs, from sending crucial signals to clearing out clutter. Yet these workers come from common ancestors that eventually specialize as the brain matures. In old age, some of these specialists seem to revert, becoming more similar to one another once again.

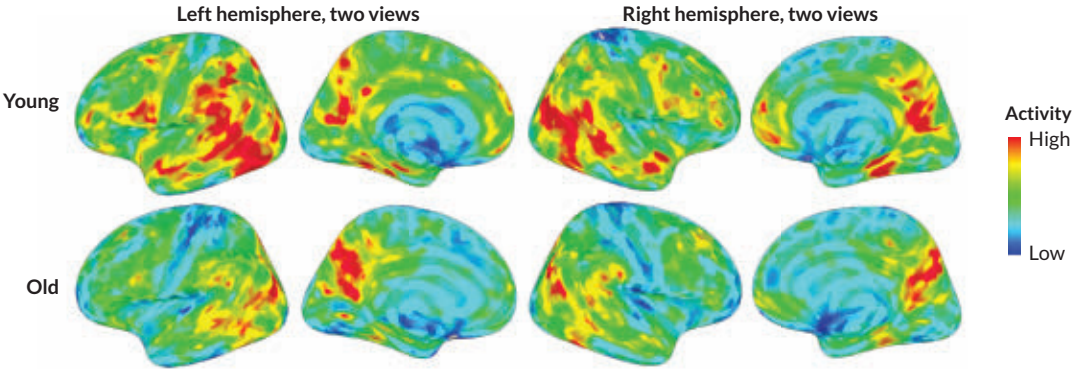
Cells are controlled by genes, but those genes don't always behave the same way across a life span. Markers on cells' DNA can dial activity up or down, controlling how much protein is made from a particular gene. In the case of brain cells, these epigenetic marks, many of which are laid down early in life in response to the environment, are one of the things that make nerve cells distinct from one another. So a nerve cell in the hippocampus, a structure important for memory, has an epigenetic fingerprint that's distinct from that of a nerve cell in the cerebellum, a part of the brain important for movement.

But with age, these marks become less distinct, both

Last in, first out Gray matter volume, made of nerve cells, peaks within a select number of neural networks around age 40. Structural brain scans of people ages 8 to 85 suggest that these brain areas that are late to develop are the first to degenerate. Each dot represents a single person. SOURCE: G. DOUAUD *ET AL*/*PNAS* 2014



Sharper recall
Young people's brains (top row) had sharper and more distinct reactions as they watched and then later remembered video snippets than brains of older people (bottom row). Red indicates brain areas that had higher activity.



between regions in a single brain and even among different people, Petronis says. After age 75, brain cells become more similar to one another, both in their epigenetic marks and their genes' behaviors, he and colleagues reported April 28 in *Genome Biology*.

That was a big surprise, he says. It contradicts a popular concept called epigenetic drift, which says that with time, epigenetic stamps accumulate on cells, making the cells more distinct. But Petronis' results suggest that once nerve cells hit a certain age, they begin to experience a different kind of drift, back toward sameness.

Petronis cautions that his results are preliminary and need to be reproduced. But he says they point to the link between development and aging. "Developmental epigenetic marks and aging epigenetic marks seem to be overlapping to some extent," he says.

It's not just nerve cells that show tendencies toward conformity in old age. Microglia do too, researchers recently found. These brain cells have multiple job descriptions, including fighting off pathogens, snipping unnecessary neural connections and hoovering up cellular debris. Microglia in different parts of the brain use their genes in specific ways — making more or fewer proteins as needed. This protein customization helps the microglia do their diverse jobs.

But this specificity diminishes with age. Microglia in the hippocampus actually become less diverse as mice get older, neuroscientist Barry McColl of the University of Edinburgh and colleagues reported in the March *Nature Neuroscience*. "It wasn't something we were looking for at all," McColl says.

The unexpected results hint that a slow loss of specialization might cause trouble during aging by hindering cells as they try to do their particular jobs, McColl says. "That's the overriding — but quite speculative — theory we've got at the moment."

Loss of specialization with age may happen not just in single brain cells, but in the networks they form. Any time a person sees, hears or feels something, the brain fires off a pattern of highly specific neural responses. Cognitive neuroscientist Bradley Buchsbaum of Baycrest Health Sciences in Toronto and colleagues wondered if elderly brains might lose the ability to form these sharp neural reflexes.

For Buchsbaum's study, 28 adults — half young and half old — watched video clips while undergoing functional MRI brain scans, which detect changes in blood flow that represent the activity of big collections of nerve cells. As participants watched snippets of President Barack Obama giving a speech, a skateboarding dog and a meat slicer in action, their brains responded to the sights and sounds. Later, they were asked to remember the videos.

In people ages 21 to 32, each type of video evoked a specific and sharp neural fingerprint, both as people saw the videos for the first time and remembered them later. The sharper the fingerprint, the better the memory, the researchers reported in 2014 in the *Journal of Neuroscience*.

But in people 64 to 78, the neural signatures became fuzzy and less distinct, particularly when participants tried to remember the videos. Buchsbaum calls this fuzziness dedifferentiation. "In the beginning, you've got this blank slate," he says. But along the way, brain areas diversify and connect in intricate ways. Dedifferentiation is an about-face toward that blank slate.

Other observations of the old brain seem to fit this idea. Language, for instance, is handled by the left side of the young adult brain. But in elderly people, both hemispheres are required to handle the job. And in older people, remembering can activate both sides of the frontal cortex, instead of just one as in younger people.

Some cognitive psychologists caution against making too much of these signs of generalization. Understanding spoken language is one of the tasks that scientists thought might become hazier in the brain with age. But when psychologist Karen Campbell of Harvard University and colleagues asked old people to simply listen to language while in a scanner, without any additional tests, the task elicited brain responses that looked similar to the specialized responses of younger people.

Campbell's results, published May 11 in the *Journal of Neuroscience*, suggest that the extra work of experimental tests — and not the task itself — may take more brainpower in older people, an addition that may confound simple interpretations. Her results are "a challenge to other scientists," she says. "Try a more natural approach."

M. ST-LAURENT *ET AL/J. NEUROSCI.* 2014

Snip early and snip late

Although scientists are still probing the relationship between brain construction and deconstruction, it's becoming clear that the brain relies on some of the same tools for both jobs.

One of the most tantalizing finds has to do with microglia. The synaptic pruning that these cells do is crucial for a growing brain, shaping the tangle of new nerve cells into an efficient, elegantly connected information processor.

This snipping may happen late in life, too, and that may not be a good thing. Synapses in the hippocampi of mice and humans become sparser with age. But when mice were engineered to lack a protein that helps mark synapses for destruction, old mice no longer showed synapse thinning, neuroscientist Cynthia Lemere of Brigham and Women's Hospital in Boston and colleagues reported last year in the *Journal of Neuroscience*. These lucky mice with an abundance of synapses performed better on memory tests and learning, too.

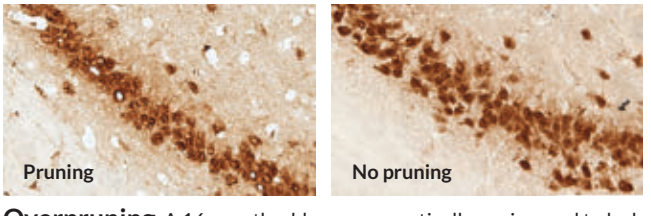
Other recent results from neuroscientist Beth Stevens' lab at Harvard hint that excessive synapse pruning may play a role in Alzheimer's disease (*SN: 4/30/16, p. 6*) and schizophrenia, though what kicks off the pruning is a mystery. "One of the really big questions is what turns this pathway on in aging, or in Alzheimer's or other diseases?" she says. "Are they the same kind of signals that we've identified in development, or could they be completely different?"

Finding those signals and other molecules in the body that could stall some of the brain's aging processes might lead to better treatments for Alzheimer's, schizophrenia or even the mental decline that comes with healthy aging.

But just because things appear to be similar doesn't mean that they are the same thing, cautions neurologist Tony Wyss-Coray of Stanford University. Finding a developmental process that's also at work during aging "doesn't mean that we are triggering a developmental program," he says. A protein that becomes active again later in life is not necessarily trying to restart development.

A lack of clarity on brain aging hasn't stopped scientists from floating ideas for delaying the mental trouble that comes with age. One notion is to wipe out age-related epigenetic changes on brain cells, a concept called "epigenetic rejuvenation." Scientists might be able to overwrite epigenetic changes using the same cellular tools that manage those marks.

Other researchers are looking to the blood for answers.



Overpruning A 16-month-old mouse genetically engineered to lack a protein that helps prune synapses had more nerve cells in part of its brain (right) along with better performance on learning and memory tasks than a mouse with intact synapse snipping (left). The results implicate synapse pruning in the aging process.

FROM TOP: ICONS: E. OTWELL; Q. SHI *ET AL/J. NEUROSCI.* 2015

Aiming for a youthful mind

You can thank your parents for about a quarter of how your brain handles aging. That leaves a lot that's open for a possible upgrade. Scientists have found links between certain habits and healthy brain aging. The tips below may seem like no-brainers, but they've got stronger evidence behind them than other behaviors, such as dietary changes, sleep and mental training.

Move. People who exercise during middle age are more likely to have sharper memories later in life. A study of 387 older women in Australia found that those who reported doing more physical activity during middle age formed stronger memories of a list of 10 unrelated words half an hour after seeing them. Researchers don't yet know how exercise wards off memory decline, but it may protect the blood vessels that feed the brain.

Don't smoke. Lothian Birth Cohort members who still smoked at age 70 performed worse on tests of general brain ability. The good news is that kicking the habit seems to help at any age. Ex-smokers performed just as well on brain tests as those who had never smoked.

Socialize. Healthy brains depend on others to stay sharp. Social butterflies are at lower risk of brain decline and dementia with age than loners, studies have found. The benefits of an active social life may come from reductions in stress, pressure to pursue better health care or even from the cognitive work it takes to maintain relationships. — *Laura Sanders*

Wyss-Coray and others have turned up tantalizing evidence that some mysterious contents in young blood can rejuvenate the older brain. Young blood spurred more neural connections and stimulated the birth of newborn nerve cells in mice. The brain changes came with better memory and a sharpened sense of smell (*SN: 5/31/14, p. 8*). The researchers are trying to figure out which blood components led to the improvements, described in *Nature Medicine* in 2014, and are testing whether plasma from young people can help the brains of older people with Alzheimer's disease.

Given the parallels emerging between development and aging, these approaches that borrow from youth to stave off decrepitude start to make sense. "Sometimes things start converging," Petronis says, "and it's very interesting to see that process." ■

Explore more

- Lothian Birth Cohort website: www.lothianbirthcohort.ed.ac.uk/

Aging's WILD SIDE

Escape artists and suicidal reproducers offer clues to how senescence evolved

By Susan Milius

The scene was stranger than it looked, even by Las Vegas standards: Two young men pull up in a U-Haul truck to a motel outside the city. They check in and move a cooler into their room. They appear to be handling something of importance, and look to see if the ice needs replenishing. Inside the cooler is not the makings of epic hangovers but instead an experiment in eternal youth.

Tucked within, protected from the desert heat, are more than a hundred tiny pond invertebrates. One of the men, Daniel Martínez, with a Ph.D. in ecology and evolution a month or so old, is rearing these little organisms to test a claim that they somehow stay young all their lives, no more likely to die as years go by as they are early on. They *can* die, however, from high temperatures or starvation. Leaving the animals on their own for more than a day invites disaster, so if Martínez travels, even stopping for sightseeing with his brother in Las Vegas, all the animals in the aging experiment travel, too.

Their road trip was in 1993, when the "dogma," as Martínez recalls, was that evolution would not allow any multicelled organism to escape aging. Just as humans age, the thinking was, other organisms also decline in health as time goes by, with death becoming more and more likely. Yet few people at the time were bothering to document aging in any creatures other than a few standard lab residents.

Biologists have long tracked aging in fruit flies and lab mice (see Page 16), but a bloom of recent data from more diverse organisms is stirring up discussion about how aging could have evolved — and if it's inevitable. The ongoing studies of Martínez's pampered pond invertebrates and a massive effort to study aging in a roadside weed are good examples of these provocative approaches. They're shaking up basic assumptions of a long-standing theory and inspiring new thinking to explain why there's so much crazy variety in how life deteriorates — or maybe doesn't.

The pond invertebrate *Hydra vulgaris* is the star of a study looking for signs of eternal youth.

Old ideas

Deciding whether an organism is aging can get tricky. For humans, the slowing and graying, the wrinkling and creaking are all too obvious. But what about plants? Or fungi? For a metric that applies across many species, evolutionary biologists often focus on how the number of deaths in a population changes over a particular period of time. If this death rate increases as time passes, the organism ages. (In this scheme, life span is irrelevant. A hypothetical species that lives for just a few months but keeps its death rate flat until the end would still be considered "biologically immortal.")

Early evolutionary thinkers proposed that aging followed by death is a good thing, another marvel of the mindless force of natural selection. Built into individuals, this inevitable decline kept feeble parents from sapping resources from the young.

But the idea that aging evolved as a boon for the next generation "is really nonsense," says Axel Kowald of Newcastle University in England, a biochemist who specializes in the bioinformatics of aging. Among the many objections: It's hard to see why a lucky few that could live a bit longer and continue to reproduce wouldn't overtake a population. With more offspring, they'd spread more of their genes. Over time, then, genes for aging should be few, fewer, gone.

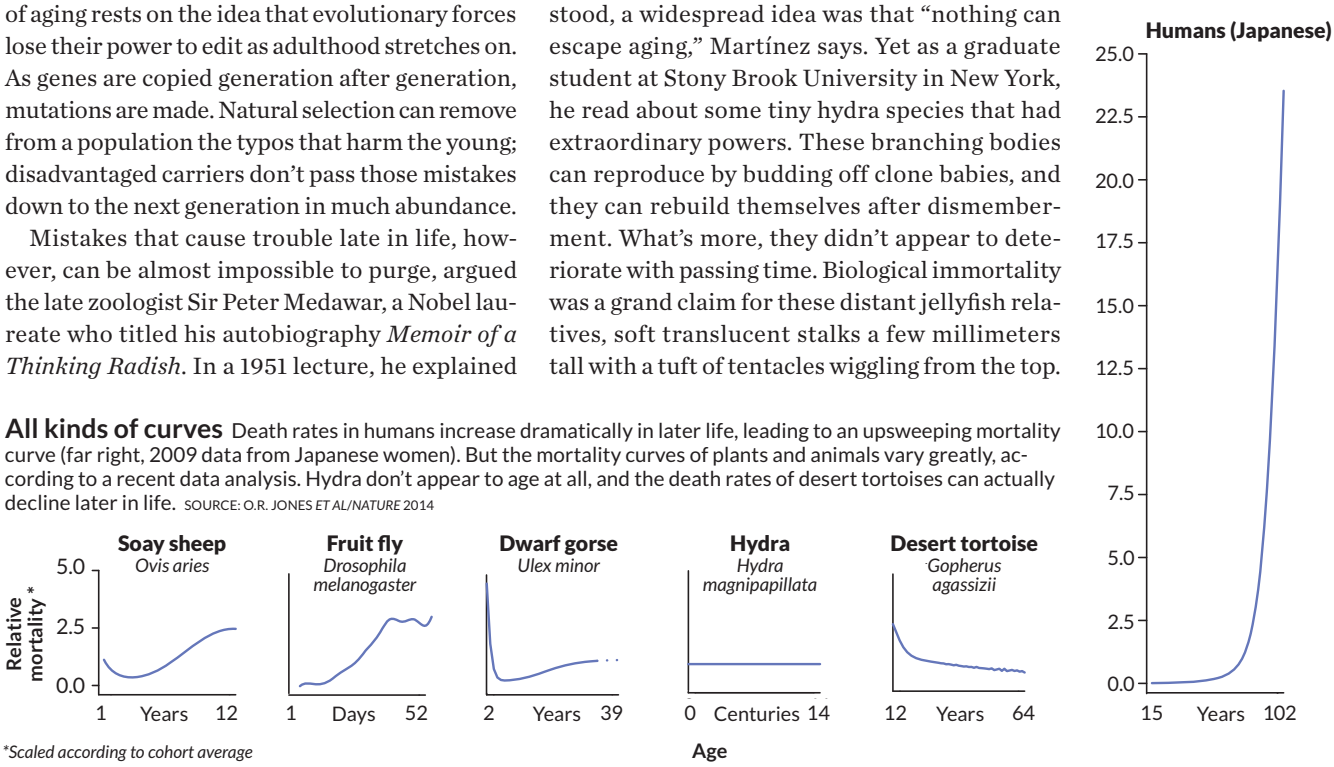
One of the modern mainstream explanations of aging rests on the idea that evolutionary forces lose their power to edit as adulthood stretches on. As genes are copied generation after generation, mutations are made. Natural selection can remove from a population the typos that harm the young; disadvantaged carriers don't pass those mistakes down to the next generation in much abundance.

Mistakes that cause trouble late in life, however, can be almost impossible to purge, argued the late zoologist Sir Peter Medawar, a Nobel laureate who titled his autobiography *Memoir of a Thinking Radish*. In a 1951 lecture, he explained this approach to aging by whimsically tracing the perilous lives of laboratory test tubes. The mortality rate of these hypothetical test tubes, which for the sake of explanation reproduced more than once in their lives, allowed few tubes to reach old age. Test tubes that don't reach old age don't reveal detrimental effects from mutations that act only late in life. Therefore natural selection didn't have a chance to stop those mutations from being passed down to test tube babies. In a scenario now called mutation accumulation, the late-acting mutations could thus build up and cause the declines of aging, also known as senescence. Natural selection doesn't weed out these mutations because, Medawar said, wild organisms "simply do not live that long."

In a perverse twist on this idea, natural selection might not just allow genes that bring late-life decrepitude to accumulate but also might favor those genes. Evolutionary biologist George C. Williams, later eulogized as a quiet and deep thinker with the look of Abraham Lincoln, argued in 1957 that genes with split personalities, like Jekyll and Hyde, could help explain aging. The benefits of these genes appear early in life and the gene is thus passed to the next generation, with its downside revealed as frailty only late in life.

Till death do us part

In the 1990s, as the theories were then understood, a widespread idea was that "nothing can escape aging," Martínez says. Yet as a graduate student at Stony Brook University in New York, he read about some tiny hydra species that had extraordinary powers. These branching bodies can reproduce by budding off clone babies, and they can rebuild themselves after dismemberment. What's more, they didn't appear to deteriorate with passing time. Biological immortality was a grand claim for these distant jellyfish relatives, soft translucent stalks a few millimeters tall with a tuft of tentacles wiggling from the top.



All kinds of curves Death rates in humans increase dramatically in later life, leading to an upsweeping mortality curve (far right, 2009 data from Japanese women). But the mortality curves of plants and animals vary greatly, according to a recent data analysis. Hydra don't appear to age at all, and the death rates of desert tortoises can actually decline later in life. SOURCE: O.R. JONES *ET AL*/*NATURE* 2014

But no one had done a rigorous test collecting the hydra and tracking their death rates.

Martínez eventually set up 145 *Hydra vulgaris* in laboratory luxury, where no predator could reach them and they could enjoy catered food all their lives. "When I started doing the experiment, I thought that I was going to prove that hydra could not escape aging," he says. "A year and a half later I got my Ph.D. — the hydra were still with me." The expected rise in death rate that characterizes aging organisms still hadn't started. "I got a postdoc at University of California, Irvine," he says, "so I crossed the country in a U-Haul truck with the hydra and all my furniture."

The truck was supposed to be air-conditioned but wasn't, and with a hot engine right under the cab, driving a southern route pulling a trailer, Martínez had to keep careful track of ice for the hydra cooler. Plus, there was all the changing of water, the feeding, the raising brine shrimp so the hydra had live prey. This was when the whole party visited Las Vegas.

The hydra made it. (Martínez, however, no longer even considers a hydra project without a technician to manage their care.) In 1998 he published results of four years of hydra watching. His title was cautious: "Mortality patterns suggest lack of senescence in hydra."

"I published the paper and forgot about it," says Martínez, now at Pomona College in Claremont, Calif.

Opinions about the inevitability of aging continued to run strong — as demographer James Vaupel of the Max Planck Institute for Demographic Research in Rostock, Germany, discovered in 2002. At a workshop on aging in nonhuman species, Vaupel stood up to say that the paper he had found most interesting was one describing mortality rates in a roadside weed. The rates appeared to drop as time passed, leading Vaupel to propose it as a possible case of what he called "negative senescence."

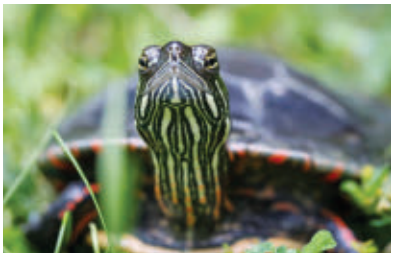
"My remark was met with ululations of horror, cries of derision, hisses and boos," Vaupel says. Eminent biogerontologists said that theorist William Hamilton had proved decades earlier that mortality, at least in repeatedly reproductive adults, universally rises with age and "there was no need for the audience to listen to a demographer who didn't understand biology."

Further data on the weed showed a more complex story, but the meeting outcry had a meaningful effect. As soon as Vaupel got back to his Rostock lab, he asked Annette Baudisch, then a new Ph.D. student, to "figure out why Hamilton's proof was wrong."

Baudisch published her critique of Hamilton in the *Proceedings of the National Academy of Sciences* in 2005. Hamilton's proof could not explain the full diversity of aging, she argued. Some species that keep growing throughout adulthood, tortoises and many plants, for example, might not be included.

Immortal tales

Some animals are known for their extreme longevity, or their attention-grabbing ability to escape death. But as with most mysteries in biology, the truth is often more nuanced than the tall tales. — *Elizabeth Quill*



Turtles
Turtles and tortoises have come to symbolize longevity, because some species can live over 100 years and don't appear to age once they reach maturity. But in a recent study tracking painted turtles (one shown above) for more than two decades, researchers found that reproduction and survival rates did in fact decline as the turtles got older. The team attributed the change to human impacts, including roads and boating activity.

Jellies
The "immortal jellyfish" (*Turritopsis spp.*) has received a lot of attention, including a profile in the *New York Times Magazine*. But this hydrozoan, common in the western Mediterranean and Adriatic seas, is something of a cheat. It doesn't escape aging the way the hydra does, but it can instead reverse its life cycle and then clone itself — a mature medusa reverts to a colonial polyp and then buds off new medusae.

Honeybee queens
Honeybees haven't received as much popular credit as they deserve for their antiaging tactics. Despite sharing a genome with other bees in the hive, the queen bee survives 10 times as long and continues to reproduce throughout her life. She somehow avoids the common trade-off between longevity and reproduction and, along with other social insects, poses a challenge for certain aging theories.

FROM LEFT: DAVE HUTH/FLICKR (CC BY-NC 2.0); MUZINA_SHANGHAI/FLICKR (CC BY-NC-SA 2.0); USGS

Though some researchers believe there's still much truth to Hamilton's approach, Vaupel took this conclusion as a cue to go questing for examples of prolonged youth. He talked Martínez into redoing the hydra experiment — but bigger. Instead of four years, the test ran for eight. Instead of 145 animals, the team had multiyear data from 2,256.

The resulting paper came out last year in the *Proceedings of the National Academy of Sciences*. Two species of hydra, with their many representatives divided between Claremont and Rostock for raising, had continued their usual low-drama lives, feeding and budding off babies but not showing any upsweep in their mortality rates. In 10 of the 12 groups, the annual probability of death stayed around 0.6 percent, and two groups held steady with an even lower annual rate of 0.09 percent.

Continuing the tests until the whole study population died, which would be ideal for tracking the hydra's entire life history, would take more than 1,000 years, researchers calculate. But eight years of data gave Martínez and Vaupel confidence. The old view that aging is inevitable, the paper declared, "is no longer tenable."

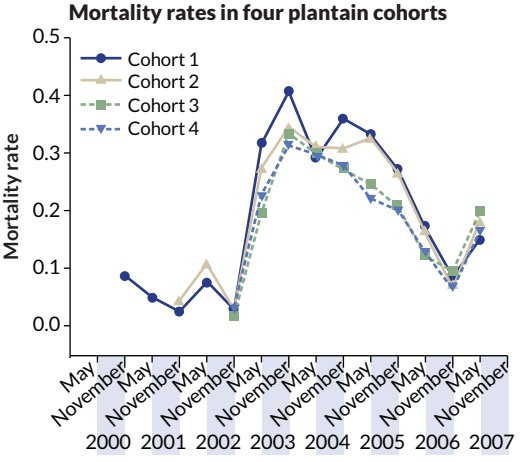
The hydra results so far are "solid evidence" that not all species age, Kowald says. And there are other, less-studied candidates for what's called negligible senescence, too: three-toed box turtles and bristlecone pines, for instance.

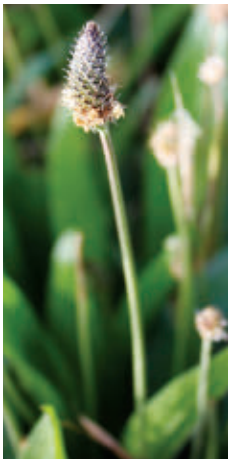
Into the wild

The lab, of course, isn't where evolution shapes life. Biologists seeking to understand how aging evolved need to know if and how organisms age in the wild, research that is likewise challenging Medawar's pronouncements.

The plant study that caused a ruckus for Vaupel was an early version of a test by Deborah Roach, a plant evolutionary biologist now at the University of Virginia in Charlottesville. Her results from 4,476 ribwort plantains (*Plantago lanceolata*), set out at a long-term research site in Durham, N.C., had suggested that this common roadside weed was escaping the supposedly inevitable decline of aging. But construction of a new art museum wiped out those plots after less than five years.

After moving to Virginia and summoning the resolve to start the experiment again, Roach selected meadows at Thomas Jefferson's birthplace, close to Charlottesville and under the protection of local historical preservationists. During the years 2000 through 2002, an army of undergraduates set out 30,000 plantains, all of known genetic heritage and marked for individual monitoring.





Showing their age From 2003 to 2006, ribwort plantains (shown at right) in plots in Virginia faced an uptick in mortality rates. Though rates were mostly similar among age cohorts before and after these years, the mortality rates of older plantains increased more than those of younger plantains during this period. Older cohorts appeared to be suffering more from a stressful environment — clear evidence of aging.
SOURCE: D.A. ROACH, C.E. RIDLEY AND J.L. DUDYCHA/*ECOLOGY* 2009

After collecting seven years of data on when plants died, Roach picked up a subtle signal she hadn't observed in Durham. At first, plantains of different ages had about the same mortality rates, all relatively low, with six-month mortality rates at less than 10 percent. But during the three years that followed, the plantains clearly struggled. Roach suspects soggy winters plus competition from neighboring foliage were to blame. Death rates rose to around 30 percent and — this was the important bit — the death rates climbed higher for the older plants. A population that had looked as if it weren't physically declining with age showed signs of senescence when the going got tough.

The results contradict Medawar's fundamental assumption that life is so short and brutal in the wild that the possibility of seeing frail, aged organisms there would be exceedingly rare. "Now we have a great body of literature showing that in fact there are these old animals out there, these old plants," Roach says.

A 2013 tally by Dan Nussey of the University of Edinburgh and an international array of colleagues documented more examples of aged organisms in the wild — 175 species, in fact, including Dall sheep, antler flies and great tits, among others. A study of painted turtles published June 7 in the *Proceedings of the National Academy of Sciences* added that species to the list, showing that human impacts might inadvertently be nudging an Illinois population toward senescence.

Across the tree of life, aging now looks more varied than old ideas predicted. Drawing on data

for 46 species, Vaupel, Baudisch (now at the University of Southern Denmark in Odense) and 12 coauthors published a paper in 2014 featuring a full page of mortality curves, which track how the number of deaths in a given group changes over time. Many of the organisms show the expected upsweep with time, but other curves are idiosyncratic. The curve of Soay sheep curls concavely downward during early adulthood before rising again to a rounded hilltop in old age. Alpine swifts' curve looks like a side view of a lawn chair relaxed way back for a summer snooze.

With mortality data on so few of the species on Earth, it's too early to pronounce big trends. So far, body size and life span don't appear to dictate the shape of the curve: The curves of water fleas and lions look remarkably similar. Organisms from different kingdoms can also have similar curves: The curves for desert tortoises and netleaf oaks both tilt downward.

"We're going to have to figure out what it is about the biology of these species that explains the variety," Roach says. The new reports, she adds, "are putting a big, bold spotlight on the theories and saying, 'Hey guys, we need to update.'"

Think again

Last year in *Experimental Gerontology,* Kowald and Thomas Kirkwood of the Institute for Ageing at Newcastle University proclaimed that Medawar's idea about natural selection losing its power appears to be "difficult to reconcile" with new research. The discussion on that point isn't over yet, though.

Baudisch, for her part, would like theoretical frameworks that describe aging (or its lack) as part of the whole topography of change during a life. "Theories that just deal with the end of life don't speak to all this diversity," she says. "Physicists don't make theories that only apply on Sundays."

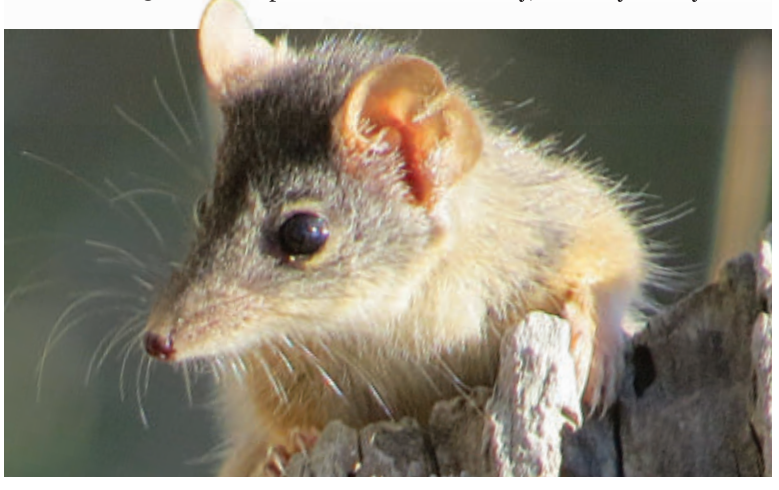
One long-standing approach does offer more of a whole-life framework. Kirkwood proposed the approach, called disposable soma theory, back in 1977. Wild organisms have to split their limited resources between reproduction and maintaining the soma, the nonreproducing parts of the body, he noted. In many cases, the best strategy demands such liberal spending on reproduction that there's not enough left for full upkeep of the rest of the body. Aging is, in this interpretation, the sum of deferred maintenance.

Australian *Antechinus* marsupials and members of two related genera offer the most dramatic example of mammals that forgo upkeep for extravagant reproduction. The climate where these marsupials live supplies a surge of insect nourishment for nursing moms only once a year. The males, roughly the size of mice or rats, grow disproportionately large testes and devote all their resources to vying for fatherhood, Diana Fisher of the University of Queensland in Australia and her colleagues reported in 2013. After healthy males reach adulthood, they stop producing new sperm, start mating and, a few weeks later, are all dead. Their immune systems collapse. A once-per-lifetime bout of intense competition leads males to what Fisher calls "suicidal reproduction."

The hydra species in the big lab test pursue a different strategy. Because they can regenerate and reproduce through budding, there is no distinction between reproductive cells and soma. Kirkwood says that the lack of senescence in hydra fits easily with the disposable soma approach. He would bet on their immortality.

He also thinks the ideas proposed by Medawar and Hamilton still have great value, with "central relevance to understanding aging." To explain all the recently uncovered variety in aging, researchers may simply need more than these theories. They might need to know, for example, the particulars of a species' home, be it pond, meadow or bug-rich forest.

Understanding these details may or may not allow humankind to do much about the process of aging. But creating better theories might finally reveal how some pale brainless squiggles of pond life may have achieved perpetual youth when humankind, despite all its apparent sophistication, has not. ■



In *Antechinus* marsupials (one species shown), males invest nearly all their energy in reproduction, dying soon after a mating frenzy. Females live longer.

HAZEL WATSON/FLICKR (CC BY-NC 2.0)

Explore more

■ Owen R. Jones *et al*. "Diversity of ageing across the tree of life." *Nature*. January 9, 2014.

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TELEVISION

A gorilla and her Penny

For the last four decades, Koko, the world's most famous gorilla, has lived in a trailer in Silicon Valley, the subject of the longest-running project on ape sign language. With a reported vocabulary of hundreds of signs, Koko has appeared to express feelings almost anyone can relate to — a love of kittens, a desire to be a mother.

A new PBS documentary argues that Koko's remarkable life "challenges what it is that makes humans unique." The problem, though, is that the film never really makes clear what "it" is. Rather than diving into the question of ape language and dissecting Koko's abilities, *Koko — The Gorilla Who Talks* focuses more on the relationship between Koko and researcher Penny Patterson.

Koko — The Gorilla Who Talks
AIRS AUGUST 3
PBS

Patterson began working with Koko in 1972 while a Ph.D. student at Stanford University, with the aim of conducting the first sign language experiment with a gorilla. Koko was an infant, living at the San Francisco Zoo. By 1977, Patterson had negotiated to take ownership of Koko.

After completing her Ph.D., Patterson drifted away from mainstream science, and her relationship with Koko seems to have morphed from researcher and study subject to mother and child. Patterson appears deeply attached to Koko, and she seems to genuinely believe Koko is communicating her thoughts and feelings.

Koko signs "good" as Penny Patterson hands her a kitten. A new documentary profiles the gorilla and researcher's decades-long relationship.

Skeptics interpret Koko's behavior differently. Columbia University psychologist Herbert Terrace, who appears in the film, has conducted his own research on primate communication and intelligence. He suggests Koko is largely mimicking Patterson to receive rewards. Patterson, he argues, has failed to produce any data that prove otherwise.

The reality is probably somewhere in between these extremes. It's difficult for anyone to really know what's going on inside an animal's head, but the idea of conversing with animals is deeply appealing. In the end, the film may reveal more about human behavior — our infinite capacity for empathy (*SN Online: 6/29/16*) and our yearning to bond with others — than it does about the capabilities of Koko or any of our other ape cousins. — *Erin Wayman*

Cracking the Aging Code
Josh Mitteldorf and Dorion Sagan
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BOOKSHELF

Aging examined from a different lens

A new book on aging starts with what sounds like a promise: "It is a common belief that aging is inevitable and universal. Nothing could be further from the truth." From this, you might expect the final pages to offer a list of options for fending off the ravages of time. But this is less a how-to guide and more of a dive into why aging happens.

The authors, theoretical biologist Josh Mitteldorf and writer Dorion Sagan, take an extensive stroll through evolutionary theory and aging research in support of an off-center view. After pointing out problems with several theories of why aging evolved, the authors present the controversial premise that aging is a programmed march toward oblivion that evolved as a form of population control. "Aging in animals enforces a common, predictable life span, helping to prevent the dominance of any one individual or one gene type. Diversity is preserved for the health of the community." Other researchers have been skeptical of that idea.

Aging, however, is unyielding. The authors describe how certain hardships — starvation, exertion, even small amounts of poison — can paradoxically lead to life extension in lab animals. From these findings, Mitteldorf and Sagan make antiaging recommendations that start with familiar medical advice: exercise, lose weight and take a daily aspirin or ibuprofen. But then they jump to suggestions that have not yet been proven, including supplementation with "huge doses of vitamin D" and melatonin, plus metformin (a diabetes drug) and selegiline (a drug used to treat early Parkinson's and depression). Next comes a list of herbs that could restore telomeres, the protective tips of chromosomes. The book spends much less real estate describing the research behind all of these recommendations, perhaps because the human studies haven't been done yet.

The crystal ball section of the book is an optimistic look at very preliminary research on the benefits of lengthening telomeres, removing senescent cells from the body and regrowing the shrinking thymus, the organ that produces immune system T cells. The authors may be onto something. But none of these ideas have yet had a chance to mature. — *Cori Vanchieri*

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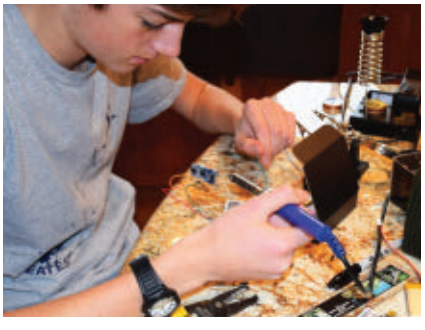
Featured alumni

JANIE KIM is no ordinary high school junior. The Broadcom MASTERS alumna (pictured left with famed comic book writer Stan Lee) was a finalist of the *Captain America: Civil War* — Girls Reforming the Future Challenge. Kim and over 1,000 other young women in the United States entered the competition, submitting projects that used science and technology to encourage positive world change. She developed a low-cost method to disinfect hospital rooms using surface acoustic wave devices.

"Participating in science fairs like Broadcom MASTERS definitely solidified my decision to go into STEM."

ALEXANDER WULFF (pictured right), a 2015 Intel ISEF finalist, developed and manufactured HaptoTech — an inexpensive electronic aid that helps the visually impaired navigate without guide sticks. As a junior in high school, he also founded Conifer Apps, a company that offers a variety of apps for mobile devices. Wulff received the 2016 Annese & Associates Award & Scholarship for Academics in Technology at the Central New York Young and Amazing Awards.

"I can do in my house what was once done in multimillion dollar computer laboratories. That's great justification for devoting so much of my time to pursuing hobbies involving technology."



MERYL NATOW (pictured left, center) was an Intel STS 2009 semifinalist and is the cofounder of Six Foods. The company produces chips made from crickets that have three times as much protein and two-fifths as much fat as traditional tortilla chips. Natow was recently named one of Forbes' 30 Under 30 in the social entrepreneurs category for her sustainable, alternative food.

"Presenting scientific research to the public taught me that it isn't all that scary to speak to a crowd, especially if you are passionate about what you are presenting."

Meet more of our amazing alumni: student.societyforscience.org/blog/doing-science

FROM TOP: COURTESY OF J. KIM; COURTESY OF A. WULFF; COURTESY OF M. NATOW

MAY 28, 2016

Particle parody

Scientists are intrigued by hints of a possible new subatomic particle that appeared in proton collisions at the Large Hadron Collider outside Geneva, writer **Emily Conover** reported in "Hints of new particle baffle physicists" (*SN: 5/28/16, p. 11*). Assuming the particle actually exists, one online reader suggested that researchers name it after one of the LHC's notorious foes — a weasel that briefly brought the collider to a standstill in April (*SN Online: 4/29/16*).

"Let's call it the Weaseltron."
Orlando Saint-Sebastien

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Oddball eyes

*The visual systems of sea urchins, mantis shrimp and other creatures are broadening scientists' understanding of what qualifies as an eye, **Susan Milius** reported in "Strange visions"* (SN: 5/28/16, p. 22).

"The article ... may be the best written and most fascinating article I have read in *Science News*, covering several decades," wrote **Patrick Roache**. Reader **RME76048** added: "Pardon the pun, but it was quite eye-opening."

Interest in animal vision goes beyond biology, reader **Paul Gorenstein** of the Harvard-Smithsonian Center for Astrophysics noted. Astronomers have developed X-ray telescopes inspired by crustacean eyes, which have a wide field of vision. "These telescopes can have nearly an all-sky field of view, which is ideal for detecting and positioning fast transient sources, which include gamma-ray and X-ray bursts plus X-rays that are expected to accompany gravity waves," he wrote.

Cool microbes

Pseudomonas syringae, *a microbe found in plants that is known for making artificial snow, uses a protein to rearrange water molecules so the liquid freezes at temperatures above those at which ice crystals normally form, **Sarah Schwartz** reported in "Bacteria use cool trick to make ice"* (SN: 5/28/16, p. 5).

"How does it benefit this bacterial species to rearrange nearby water molecules?" asked online reader **Maia**. "Does it make plants easier to infect?"

It appears so, **Schwartz** says. Raising the temperature at which ice crystals grow makes frost form more readily on and in plants. Once ice damages tissues, it's easier for bacteria to invade. "There have been some conflicting data on the particular role and extent of the bacterium's ice-making on plants, and not all strains of the bacterium infect plants," she says. "But *P. syringae*, which is especially damaging to woody plants including fruit trees, can be a costly threat for farmers and gardeners — despite its usefulness on the slopes."

More than words

*Researchers mapped words and their meanings to specific regions of the brain. The findings indicate that humans comprehend language in a way that's more complex than previously thought, **Meghan Rosen** reported in "Words' meanings mapped in brain"* (SN: 5/28/16, p. 15).

Online reader **Eugeno** wondered if brain maps could apply to animals.

Animals don't have the same language capacity that people do, says neuroscientist **Jack Gallant** of the University of California, Berkeley. "But to the extent that their calls have specific meanings, one could indeed map how that meaning is represented in an animal's brain." For example, vervet monkeys have separate alarm calls for different predators. By playing those alarm calls to a monkey in a brain scanner, scientists could map the representations of these concepts in the brain. "Of course," he says, "it might be difficult to convince a vervet monkey to lie still in the MRI machine."

Sleepy evolution

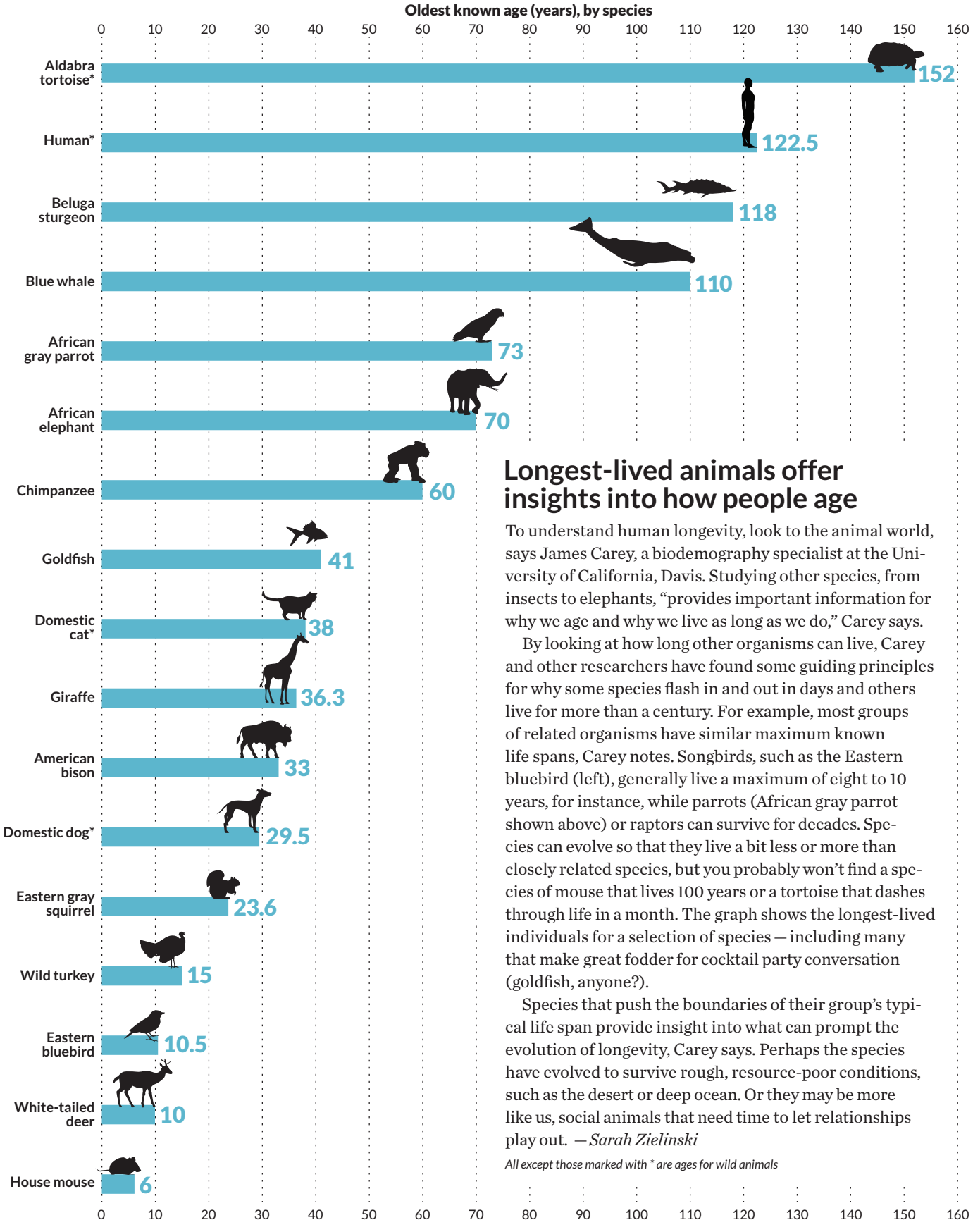
*Part of the brain's left hemisphere keeps watch when people sleep in unfamiliar places, **Laura Sanders** reported in "Left brain stands guard while sleeping away from home"* (SN: 5/28/16, p. 8).

Online reader **Robin** wondered about the evolutionary origins of this ability.

Sleeping with just half a brain offers a compromise between safety and sleep, **Sanders** says. Unihemispheric sleep allows aquatic mammals to surface to breathe and birds to detect predators, all while letting part of the brain rest. Scientists don't understand all of the evolutionary trade-offs. The relative rarity of unihemispheric sleep among mammals suggests that the partial shutdown comes with some unknown costs.

Correction

In "Juno's view to Jupiter" (*SN: 6/25/16, p. 16*), Jupiter's Great Red Spot is described as "a storm more than twice as wide as Earth." While the storm used to be that large, it has been shrinking and is now just a bit wider than Earth.



Longest-lived animals offer insights into how people age

To understand human longevity, look to the animal world, says James Carey, a biodemography specialist at the University of California, Davis. Studying other species, from insects to elephants, "provides important information for why we age and why we live as long as we do," Carey says.

By looking at how long other organisms can live, Carey and other researchers have found some guiding principles for why some species flash in and out in days and others live for more than a century. For example, most groups of related organisms have similar maximum known life spans, Carey notes. Songbirds, such as the Eastern bluebird (left), generally live a maximum of eight to 10 years, for instance, while parrots (African gray parrot shown above) or raptors can survive for decades. Species can evolve so that they live a bit less or more than closely related species, but you probably won't find a species of mouse that lives 100 years or a tortoise that dashes through life in a month. The graph shows the longest-lived individuals for a selection of species — including many that make great fodder for cocktail party conversation (goldfish, anyone?).

Species that push the boundaries of their group's typical life span provide insight into what can prompt the evolution of longevity, Carey says. Perhaps the species have evolved to survive rough, resource-poor conditions, such as the desert or deep ocean. Or they may be more like us, social animals that need time to let relationships play out. *—Sarah Zielinski*

*All except those marked with * are ages for wild animals*

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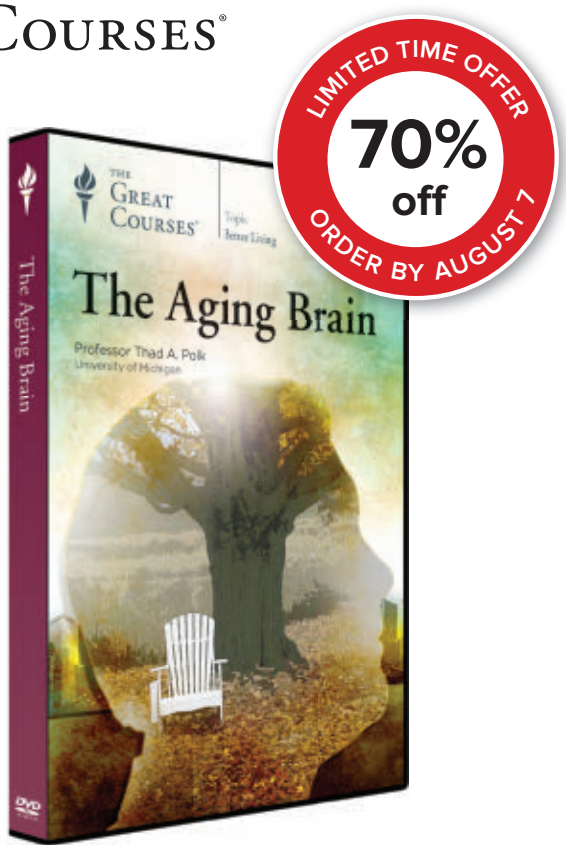

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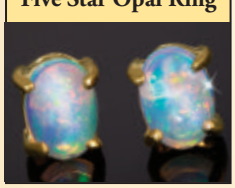

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