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Fungus lures male flies into having sex with dead females

Finding could lead to new ways to control fly populations

By Erik Stokstad

If you see a dead housefly on a windowsill surrounded by a ghostly halo of tiny white spores, it's a death trap. The insect was invaded by a fungus that took over its brain, manipulating the fly to find the highest perch it could. From there, the fungus launched its spores into the air to infect as many healthy flies as possible. Even weirder: Males try to mate with dead, fungus-swollen females. Now, a study has revealed the fungus creates a love potion by releasing chemicals that lure flies to increase their chances of infection.

The new paper "pretty conclusively shows … another way that the fungus helps to disseminate to new hosts," says Carolyn Elya, a molecular biologist and postdoctoral researcher at Harvard University who studies the fungus but was not involved. "They've made a big step forward here."

A male housefly eats fungal spores on the dead body of a female fly. **Fillipo Castelucci**

Prior to the new study, some researchers had observed male houseflies trying to mate with the corpses of females that had died of the fungus, *Entomophthora muscae*. It made sense that this kind of intimacy might help the fungus spread, but it wasn't clear whether the fungus somehow attracted the males.

Henrik de Fine Licht, an evolutionary ecologist at the University of Copenhagen, and Andreas Naundrup Hansen, a Ph.D. student, tested whether the attraction is sexual and the fungus is luring healthy males to the dead females. First, Naundrup infected female flies with the fungus, and just after they died, he placed them one by one in petri dishes. Each time, he added a healthy male to the dish and recorded whether it approached the dead female, how long it spent nearby, and whether it tried to mate. He did control experiments that included uninfected females he had killed by freezing to death.

The males were about five times as likely to try to mate when the female had died of the fungus, the team reported last month in a preprint posted on bioRxiv. Sometimes, vigorous mating let loose a cloud of spores, but even simple contact was enough to infect a healthy male, Naundrup showed.

In another experiment, healthy males could choose between two dead females in the same dish, one infected and the other not. The males tried to mate more often (compared with when neither female was infected), but they did not distinguish between the females. Naundrup suspects the fungus releases some sort of mating cue. "It's almost like an aphrodisiac, maybe driving his sexual behaviors to a supernormal level," he says.

Then, Naundrup checked whether males were indeed attracted to the fungal spores. He placed four male flies in a small chamber containing two opaque petri dishes. Inside each petri dish, which had a fly-size entrance in its lid, was a piece of fly paper, one dusted with fungal spores and the other not. In 43 trials, all four flies landed on the paper with fungal spores. The other paper caught all four flies in only 17 trials.

"It really is a beautiful study," says Matthew Kasson of West Virginia University, who specializes in insect-killing fungi. Kasson collaborates with de Fine Licht on a study of the fungus' genome, but he was not involved in this study.

The team suspected the strong odor of the fungus—a grassy, somewhat sweet smell—was part of the appeal. By placing an electrode on the tip of a fly's antennae, Naundrup showed whiffs of the fungus stimulated an electrical current in the brain. To find out

what chemicals the fungus releases, he extracted compounds from dead flies with a solvent. Working with chemical ecologists at the Swedish University of Agricultural Sciences, the team found flies infected with the fungus contained many more chemicals than did healthy flies, and the presence and abundance of several of these varied with how long the fly had been infected.

Some of the chemicals, called methyl-branched alkanes, have previously been found to stimulate male houseflies to mate. The researchers couldn't identify the fungus' specific chemical attractant, but they say if it could be isolated and manufactured, it might be useful as a lure to trap houseflies. But meanwhile, the researchers say they are astonished by the fungus' ability to manipulate its host. "I'm really impressed and amazed by the extent of the adaptation it shows," de Fine Licht says.

The fungal attraction can be spotted indoors or outdoors, where dead houseflies are perched with their wings spread, Naundrup says. "If people are interested in this, my advice would be to stop and—I wouldn't say smell the flowers—but stop and watch the flies."

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COVID-19 Vaccines Provide 5 Times the Protection of Natural Immunity, CDC Study Says

Unvaccinated people who had a recent infection 5X more likely to be reinfected with coronavirus compared to the fully vaccinated

Carolyn Crist

Unvaccinated people who had a recent infection were five times more likely to be reinfected with the coronavirus compared to those who were fully vaccinated and didn't have a prior infection, according to a new study published Friday in the CDC's *Morbidity and Mortality Weekly Report*.

The research team concluded that vaccination can provide a higher, stronger, and more consistent level of immunity against COVID-19 hospitalization than infection alone for at least six months.

"We now have additional evidence that reaffirms the importance of COVID-19 vaccines, even if you have had prior infection," Rochelle Walensky, MD, director of the CDC, said in a statement.

"This study adds more to the body of knowledge demonstrating the protection of vaccines against severe disease from COVID-19," she said. "The best way to stop COVID-19, including the emergence of variants, is with widespread COVID-19 vaccination and with disease prevention actions such as mask wearing, washing hands often, physical distancing and staying home when sick."

Researchers looked at data from the VISION Network, which included more than 201,000 hospitalizations for COVID-like illness at 187 hospitals across nine states between Jan. 1 to Sept. 2. Among those, more than 94,000 had rapid testing for the coronavirus, and 7,300 had a lab-confirmed test for COVID-19.

The research team found that unvaccinated people with a prior infection within 3 to 6 months were about 5-1/2 times more likely to have laboratory-confirmed COVID-19 than those who were fully vaccinated within 3 to 6 months with the Pfizer or Moderna shots. They found similar results when looking at the months that the Delta variant was the dominant strain of the coronavirus.

Protection from the Moderna vaccine "appeared to be higher" than for the Pfizer vaccine, the study authors wrote. The boost in protection also "trended higher" among older adults, as compared to those under age 65.

Importantly, the research team noted, these estimates may change over time as immunity wanes. Future studies should consider infection-induced and vaccine-induced immunity as time passes during the pandemic, they wrote.

Additional research is also needed for the Johnson & Johnson vaccine, they wrote. Those who have received the Johnson & Johnson vaccine are currently recommended to receive a booster shot at least two months after the first shot.

Overall, "all eligible persons should be vaccinated against COVID-19 as soon as possible, including unvaccinated persons previously infected," the research team concluded.

Sources :

CDC: "Morbidity and Mortality Weekly Report: Laboratory-Confirmed COVID-19 Among Adults Hospitalized with COVID-19-Like Illness with Infection-Induced or mRNA Vaccine-Induced SARS-CoV-2 Immunity — Nine States, January-September 2021." "New CDC Study: Vaccination Offers Higher Protection than Previous COVID-19 Infection."

https://go.nature.com/3wlJJtX

Scientists: don't feed the doubt machine

From climate to COVID, naivety about how science is hijacked promotes more of the same.

Cecília Tomori

Researchers at the COP26 climate talks this month know well how doubt can be weaponized to delay action — something many COVID-19 scientists have taken too long to appreciate. They point out problematic methods, poor study design and unjustified claims, but their efforts would be much more effective if they first considered a larger strategy: how 'sciency-ness' is used to distract from reality and hinder effective policy.

Much of my own work focuses on how industry exploits scientific credentials to bolster false claims that undermine breastfeeding to increase sales of formula milk and, ultimately, damage health. The strategies and patterns recur across industries: they have been documented in tobacco, fossil fuels, pharmaceuticals, food and more. This influence is so powerful that public-health researchers consider it a distinct area of study: 'commercial determinants of health'.

Throughout the pandemic, I've been saddened at how science has been hijacked. Arguments around herd immunity exemplify this: proponents claimed that acquiring immunity by infection was fine for most people and also that communities were well on their way to achieving herd immunity. The messages downplayed dangers for those with high risks of exposure or severe illness. Technical arguments over infection rates silently cemented the assumption that disabled or immunocompromised people did not merit collective protective action; nor did the workers whose jobs required dangerous public contact.

Although many scientific champions did provide appropriate context, I watched several respected colleagues step into debates on when, or if, society would reach herd immunity without realizing that the discussion was not simply a scientific debate. Their too-narrow focus unintentionally helped to promote controversy and doubt, and that ultimately impeded an effective public-health response. The same happened around mask use, vaccination and school policies. This helped to shift public opinion on which public-health measures were 'acceptable': the fewer the better.

The field of agnotology (the study of deliberate spreading of confusion) shows how ignorance and doubt can be purposefully manufactured. Famous scholars include David Michaels, Marion Nestle and Naomi Oreskes. In September, Katharine Hayhoe, chief scientist at the Nature Conservancy, a non-profit organization based in Arlington, Virginia, quoted environmentalist Bill McKibben on Twitter in regard to climate change: "We spent a long time thinking we were engaged in an argument about data and reason …. But now we realize it's a fight over money and power." Hayhoe elaborated: "'Objections' were always, entirely, professionally, and verrrry cleverly couched in scientific terms. They [industry] focused their lasers on the science and like cats we followed their pointer and their lead." Some elements of manufactured doubt in this pandemic might seem fuzzier, especially when vested interests are not always clear. Nonetheless, the same lessons apply.

How can researchers keep from being distracted like cats? By gaining a better understanding of how strategies are deployed to manufacture doubt and ignorance.

First, researchers must learn to identify authors of research, and their relationships with industry and with non-profit groups that have specialized agendas. How the tobacco industry paid scientists and physicians to serve as advisers and consultants to undermine the body of evidence pointing to the harms of tobacco is extensively documented. More recent examples abound. For instance, the non-profit International Life Sciences Institute, based in Washington DC and funded by leading companies in the food and chemical industries, promotes doubt about science that links ultraprocessed foods with health concerns, and provides experts to promote personal responsibility rather than regulations on junk food in policies to combat obesity.

Second, scientists should consider what kinds of argument the data and conclusions serve. How might these shape public opinion? What policy decisions might they affect? A review of corporate determinants of health highlights how media ownership can shape coverage and frame whether health is seen as a matter of 'personal responsibility', which suits corporate interests, or a communal and governmental responsibility (M. McKee and D. Stuckler *Am. J. Public Health* **108**, 1167–1170; 2018). This has a key role in whether individual decisions are cast as a matter of 'freedom' versus 'solidarity', and regulations as restriction or protection. Scientists can point out these framings when talking to reporters or on social media.

Third, scientists can consistently highlight correct information and avoid serving as inadvertent amplifiers of flawed information; they can encourage journalists to do the same. Avoid links to news articles or commentaries that highlight poor studies or otherwise use science irresponsibly. Provoking outrage and controversy helps misleading arguments to spread, which serves to manufacture doubt. And, as documented in anti-vaccine movements and climate denial, controversy around one article can generate attention that legitimizes problematic arguments.

The scientists who gum up the doubt machine do so by constantly pointing to the broader context, by acknowledging genuine scientific debate, by being alert to researchers' political and commercial connections, and by staying educated on how denialism works. If more scientists did the same, these distorting strategies would be stymied.

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https://bit.ly/3o6e77R

Signs of Dementia Are Written in the Blood: 33 Metabolic Compounds May Be Key to New Treatments

33 metabolic compounds linked to dementia could be key to new methods of diagnosis and treatment.

Scientists in Japan have identified metabolic compounds within the blood that are associated with dementia.

The study revealed that the levels of 33 metabolites differed in patients with dementia, compared to elderly people with no existing health conditions. Their findings, published recently in *PNAS*, could one day aid diagnosis and treatment of dementia.

"Metabolites are chemical substances produced by vital chemical reactions that occur within cells and tissues," said first author Dr. Takayuki Teruya, who works in the G0 Cell Unit at the Okinawa Institute of Science and Technology Graduate University (OIST). "Our body normally keeps these levels in balance, but as we age and if we develop diseases like dementia, these levels can fluctuate and change."

Dementia is not just a single disease, but a general term used to describe a set of symptoms, including a slow but typically irreversible decline in the ability to remember, think, make decisions or perform day-to-day activities. Of all aging-associated diseases, dementia is one of the most serious, not only for the patients and their family but for society as a whole, with an

estimated 55 million people living with the disease worldwide.

33 dementia markers			Dementia								HE							
			#1	#2	#3	#4	#5	#6	#7	#8	#9	#10	#11	#12	#13	#14	#15	#16
Higher in dementia (7)	A	Quinolinic acid	52.4	59.9	56.2	48.0	51.6	68.9	76.9	45.6	38.4	53.8	39.4	36.8	48.2	46.0	40.6	64.6
		Dimethyl-guanosine	49.2	63.0	51.8	42.3	60.9	79.7	58.3	62.2	42.7	49.8	52.7	38.4	52.8	47.9	50.8	47.0
		Pseudouridine	53.3	56.8	54.1	45.3	55.7	84.4	65.2	54.8	41.5	54.3	50.0	38.8	53.9	49.2	49.2	49.4
		Indoxyl-sulfate	57.1	63.4	53.2	53.6	43.7	78.1	58.8	57.2	37.3	59.9	47.4	38.1	52.8	52.4	39.1	38.7
		Kynurenine	67.0	50.9	56.7	37.6	63.3	53.5	57.2	60.1	47.9	52.6	50.9	22.8	44.4	55.3	44.4	51.5
		N6-Acetyl-lysine	85.1	46.3	45.4	49.7	57.8	48.4	71.7	44.9	45.8	48.2	42.9	44.5	47.8	44.8	43.8	46.0
		Adenosine	69.8	51.9	43.1	44.0	50.3	50.9	45.6	51.5	43.0	45.8	43.4	46.3	43.1	50.4	40.2	39.5
Lower in dementia (26)	B	S-Methyl-ergothioneine	40.7	40.2	44.2	40.5	41.2	38.9	40.2	42.0	52.5	45.2	57.6	70.9	45.2	57.7	79.0	45.5
		Ergothioneine	40.5	42.8	41.8	43.4	42.0	41.5	37.3	49.9	52.0	44.6	53.1	71.0	49.9	68.0	77.8	45.3
		Trimethyl-histidine	38.9	48.4	51.5	44.1	42.2	43.6	37.4	44.6	65.5	54.0	46.3	69.2	64.4	55.1	77.5	45.6
		Trimethyl-tryptophan	41.1	43.0	42.0	43.0	42.0	41.2	46.3	43.2	56.6	76.3	44.5	59.8	67.6	65.3	51.8	47.2
		Trimethyl-phenylalanine	46.9	46.9	46.9	46.9	46.9	46.9	46.9	47.0	47.2	46.9	47.2	78.2	86.2	49.0	47.1	46.9
		Trimethyl-tyrosine	45.5	45.7	45.7	45.8	45.7	45.5	45.5	45.5	45.6	45.9	47.4	46.9	93.6	47.4	57.5	45.9
	C	Pantothenate	42.0	41.6	40.1	39.1	43.9	38.6	59.7	50.1	69.2	77.2	45.5	51.3	57.7	47.7	52.5	45.6
		Gluconate	43.8	44.4	64.1	33.6	42.5	32.2	56.1	39.8	50.9	53.4	49.5	51.5	76.4	59.1	52.1	60.5
		S-Adenosyl-methionine	42.5	41.3	41.2	41.3	60.6	42.1	43.9	43.6	53.7	46.4	45.8	51.0	44.7	53.1	43.9	45.9
		NADP+	35.1	40.4	33.6	31.6	48.0	42.2	47.2	44.8	55.5	45.2	57.7	58.5	46.8	45.4	53.7	45.3
		Glutathione disulfide	37.3	51.5	22.0	42.0	46.0	46.5	36.2	56.3	62.3	58.7	61.2	58.5	47.2	50.1	58.4	44.0
		ATP	40.8	45.4	30.9	31.9	48.0	46.3	47.1	51.0	42.3	51.5	60.2	60.7	58.5	50.8	61.9	51.0
	D	Methionine	35.3	52.9	40.2	39.8	41.2	39.5	30.7	69.3	54.1	52.1	65.4	56.4	51.4	48.0	50.5	53.5
		Tryptophan	36.9	53.9	43.6	44.7	42.1	40.7	22.5	58.4	52.5	52.9	56.8	49.3	44.7	61.4	61.9	49.8
		Glutamine	44.6	45.2	33.1	40.6	41.2	53.6	33.6	66.9	49.3	51.3	54.8	54.9	60.9	54.9	52.6	67.6
		Betaine	45.4	52.5	44.4	45.8	40.7	42.6	27.3	52.4	52.8	59.5	53.1	56.0	62.3	46.3	60.8	51.3
		Phenylalanine	36.9	59.2	41.6	41.4	37.8	48.5	41.7	58.3	47.2	59.3	69.5	47.6	47.5	59.4	58.8	58.5
		Tyrosine	41.3	38.6	39.6	44.9	44.7	40.5	39.3	59.7	44.5	67.3	60.5	53.5	52.9	56.4	53.7	51.2
		Histidine	44.4	49.4	48.4	49.0	41.8	33.9	30.6	55.6	43.5	64.7	58.4	42.7	76.6	65.2	53.9	52.5
		Uridine	42.9	43.0	45.7	42.9	37.6	33.8	34.2	55.9	53.9	48.2	49.1	49.1	59.8	68.3	62.7	51.5
		Keto(iso)leucine	40.8	46.1	46.1	43.2	31.7	51.8	47.8	46.0	56.2	56.3	65.3	41.1	50.3	69.4	56.8	50.0
		Glycerophosphocholine	48.8	48.8	43.4	35.5	43.6	49.5	37.8	43.3	52.0	50.5	45.6	46.2	55.7	53.5	47.0	50.6
		2-Hydroxybutyrate	48.3	46.9	51.9	40.9	34.7	47.0	66.3	48.6	48.8	41.3	74.7	61.5	56.0	57.0	53.2	53.6
		Dodecanoyl-carnitine	39.5	40.0	39.5	42.0	44.7	55.5	55.1	39.7	54.5	51.0	59.6	44.9	40.0	46.6	60.3	62.2
	E	Caffeine	44.8	43.5	43.6	44.4	43.4	43.7	44.7	45.2	45.7	54.3	55.5	43.3	62.7	66.8	49.2	45.6
		Dimethyl-xanthine	57.7	40.4	40.7	40.7	40.1	40.4	40.3	42.3	45.5	60.7	59.2	39.8	64.2	71.1	55.6	44.3

A heat-map, where red shows high levels of a compound, and blue shows low levels of a compound, reveals the link between certain metabolites and dementia. Compounds in sub-group A were typically higher in dementia patients and lower in healthy elderly people. Compounds in sub-group B-E showed the opposite effect. **Credit: OIST**

While scientists know that dementia is caused by damage to nerves, the exact cause of this damage, and methods as to how it can be detected and treated have remained elusive.

In the study, the research team analyzed samples of blood collected from eight patients with dementia, as well as eight healthy elderly people. They also collected samples from eight healthy young people to use as a reference. Unlike most studies analyzing blood metabolites, this research included compounds found within red blood cells.

"Blood cells are difficult to handle because they undergo metabolic changes if left untreated even for a short period of time," explained Dr. Teruya.

However, the research team recently developed a way to stabilize metabolites in red blood cells, allowing them to examine for the first time the relationship between red blood cell activity and dementia.

The scientists measured the levels of 124 different metabolites in whole blood and found that 33 metabolites, split into 5 different sub-groups, correlated with dementia. Seven of these compounds increased in dementia patients, whilst 26 of these compounds showed a decrease in levels. 20, including nine that were abundant in red blood cells, of these compounds had not previously been linked to dementia.

"Identification of these compounds means that we are one step closer to being able to molecularly diagnose dementia," said senior author of the study, Professor Mitsuhiro Yanagida, who leads the G0 Cell Unit at OIST.

The seven metabolites that showed increased levels in patients with dementia were found within the blood plasma and belonged to sub-group A of metabolites. Importantly, some of these compounds are believed to have toxic effects on the central nervous system.

"It's still too early to say, but it could suggest a possible mechanistic cause of dementia as these compounds may lead to impairment of the brain," said Prof. Yanagida.

The research team plans to test this idea in the next steps of their research, by seeing if increases in these metabolites can induce dementia in animal models, like mice.

The remaining 26 compounds that decreased in patients with dementia, compared to healthy elderly people, belonged to four

other metabolite sub-groups, B-E.

Six metabolites that decreased in dementia patients were classified into sub-group B, due to their similar structure. These metabolic compounds are antioxidants, which protect cells and tissues by reducing damage caused by free radicals – unstable molecules produced by chemical reactions in cells. The researchers found that these antioxidant compounds derived from food were highly abundant in red blood cells of healthy elderly people.

“It could be that red blood cells deliver not only oxygen but also crucial metabolites that protect the nervous system from damage,” said Dr. Teruya.

The remaining sub-groups contain compounds that the researchers believe play a role in supplying nutrients, maintaining energy reserves and protecting neurons from damage.

“In the future, we hope to start some intervention studies, either by supplementing dementia patients with metabolic compounds in sub-groups B-E, or by inhibiting the neurotoxins from sub-group A, to see if that can slow, prevent, or even reverse symptoms of dementia,” said Prof. Yanagida.

Reference: “Whole-blood metabolomics of dementia patients reveal classes of disease-linked metabolites” by Takayuki Teruya, Yung-Ju Chen, Hiroshi Kondoh, Yasuhide Fukuji and Mitsuhiro Yanagida, 7 September 2021, Proceedings of the National Academy of Sciences. DOI: 10.1073/pnas.2022857118

The research was conducted by the Okinawa Institute of Science and Technology Graduate University, along with the National Ryukyu Hospital, Okinawa and Kyoto University.

https://wb.md/3qaqzX5

People Who Believe in COVID-19 Conspiracies More Likely to Catch Virus: Study

More likely to catch the virus, lose their jobs, and be socially isolated

People who believe in COVID-19 conspiracy theories are more likely to catch the virus, lose their jobs, and be socially isolated, according to a new study published in *Psychological Medicine*, a peer-reviewed medical journal by Cambridge University Press.

The study, conducted by researchers in the Netherlands, found that those who believe in COVID-19 conspiracies are less likely to be tested for COVID-19. But they're more likely to get infected and test positive.

"One basic property of conspiracy theories is that they are consequential: Even if a conspiracy theory is extremely implausible according to logic or scientific evidence, if it seems real to a perceiver, it has a genuine impact on attitudes, emotions, and behavior," the study authors wrote.

The research team surveyed 5,745 people to provide a large sample of observations from a cross-section of Dutch residents. They contacted people in April 2020 and December 2020 to examine whether conspiracy beliefs early in the pandemic would predict health and well-being outcomes later in the year.

The researchers asked about four COVID-19 conspiracy beliefs, including whether the coronavirus is a "bioweapon engineered by scientists," whether the coronavirus is a "conspiracy to take away citizens' rights for good and establish an authoritarian government," whether the coronavirus is a "hoax invented by interest groups for financial gains," and whether the coronavirus was "created as a cover-up for the impending global economic crash."

They found that conspiracy beliefs predicted an increased likelihood of violating coronavirus regulations, experiencing social rejection, having economic problems such as job loss or reduced income, and having lower overall well-being. Most of the effects generalized to a broader susceptibility to conspiracy theories or a conspiracy mentality overall.

The research team also found that conspiracy beliefs predicted an increased chance of disrupted social relationships. People who scored "low" in conspiracy beliefs were more likely to reject people

who scored "high" in conspiracy beliefs. Publicly endorsing conspiracy beliefs can lead to stigmas and reduce people's social support network, the authors wrote.

"These findings suggest that conspiracy beliefs are associated with a myriad of negative life outcomes in the long run," the study authors wrote.

"Conspiracy beliefs predict how well people cope with the challenges of a global pandemic and therefore has substantial implications for private and public health, as well as perceivers' economic and social well-being," they concluded.

Source *Psychological Medicine: "Conspiracy beliefs prospectively predict health behavior and well-being during a pandemic."*

https://bit.ly/3bMuscs

Two Planets in HD 3167 System are on Perpendicular Orbits, Astronomers Say

Super-Earth HD 3167b is close to orbiting within the stellar equatorial plane, while the mini-Neptune HD 3167c orbits above the poles of the host star

by Natali Anderson

The super-Earth HD 3167b is close to orbiting within the stellar equatorial plane, while the mini-Neptune HD 3167c orbits above the poles of the host star, and the orbits of the two planets are nearly perpendicular (mutual inclination 102.3 degrees), according to a team of astronomers led by the Observatoire Astronomique de l'Universite de Geneve.

HD 3167 is a bright K0-type star some 149 light-years away in the constellation of Pisces. Also known as EPIC 220383386 and 2MASS J00345752+0422531, it has a radius and a mass roughly 86% that of the Sun, and is approximately 8 billion years old.

HD 3167 hosts at least three exoplanets: HD 3167b, c and d.

The innermost planet, HD 3167b, is a super-Earth on an ultrashort period of 0.96 days and the outermost one, HD 3167c, is a mini-Neptune on a 29.84 days orbit.

While HD 3167b and HD 3167c transit, this is not the case for HD 3167d, which orbits in between them on a 8.51 days orbit.

"Theories of the origin of planetary systems predict that planets form in the equatorial plane of their star and continue to evolve there, unless disturbed by special events," said Dr. Vincent Bourrier from the Observatoire Astronomique de l'Universite de Geneve and colleagues.

"This is not the case in the Solar System, where our planets lie close to the solar equatorial plane. In this case, the planets are said to be aligned with their star. However, a 2019 study showed that two of the three planets around HD 3167 are not aligned with it. HD 3167c and HD 3167d actually pass over the star's poles, nearly 90 degrees from its equatorial plane."

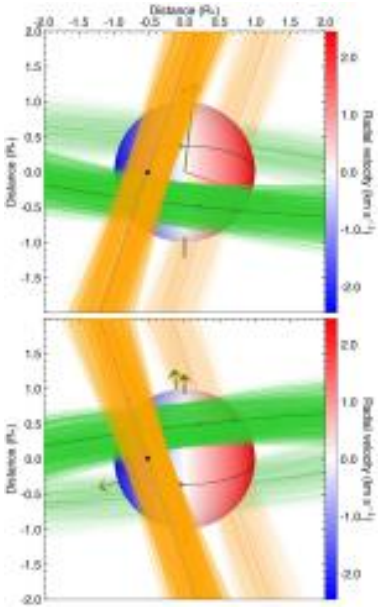


Fig. 15 ***Projection of HD 3167 in the plane of sky for the best-fit orbital architecture (the top and bottom panels show configurations A and B, respectively; see text). The stellar spin axis is displayed as a black arrow extending from the north pole. The stellar equator is represented as a black line. The stellar disk is colored as a function of its surface RV field. Normals to the orbital planes of HD 3167b and HD 3167c are shown as green and brown arrows, respectively. Thick solid curves with corresponding colors represent the best-fit orbital trajectories. The thin lines surrounding them show orbits obtained for orbital inclination, semi-major axis and sky-projected obliquity values drawn randomly within 1σ from their probability distributions. The star, the planets HD 3167b and HD 3167c (black disks), and their orbits are shown to scale. HD 3167d, which orbits in between HD 3167b and HD 3167c, is not shown because of its unknown inclination.*** **V. Bourrier et al.**

In the new study, the astronomers observed the HD 3167 system

with the ESPRESSO spectrograph on ESO's Very Large Telescope and the HARPS-N spectrograph on the 3.58-m Telescopio Nazionale Galileo. Using the Rossiter-McLaughlin effect Revolutions (RMR) technique, they were able to measure the spin-orbit angles of HD 3167b and HD 3167c.

"The two planets are on perpendicular orbits," the researchers said.

"This could be explained by HD 3167b being strongly coupled to the star and retaining its primordial alignment, whereas HD 3167c would have been brought to a nearly polar orbit via secular gravitational interactions with an outer companion."

"Follow-up observations of the system and simulations of its dynamical evolution are required to search for this companion and explore the likelihood of this scenario."

"HD 3167b is the smallest exoplanet with a confirmed spectroscopic Rossiter-McLaughlin signal." "The RMR technique opens the way to determining the orbital architectures of the super-Earth and Earth-sized planet populations." The team's paper was published in the journal *Astronomy & Astrophysics*.

V. Bourrier et al. 2021. The Rossiter-McLaughlin effect revolutions: an ultra-short period planet and a warm mini-Neptune on perpendicular orbits. A&A 654, A152; doi: 10.1051/0004-6361/202141527

https://bit.ly/3006BTW

Achieving Type 2 Diabetes Reversal Seems Way More Common Than Scientists Realized

Research in recent years has demonstrated that type 2 diabetes can be reversed in the body, with dieting methods and other lifestyle interventions sending the disease into remission.

Peter Dockrill

About 1.5 million Americans are diagnosed with diabetes every year. The vast majority of cases (90–95 percent) will be type 2 diabetes, a chronic health condition that can lead to heart disease, kidney disease, vision loss, and more.

For a subset of these patients, it doesn't have to be that way.

A huge amount of research in recent years has demonstrated that type 2 diabetes can be reversed in the body, with a range of dieting methods and other kinds of lifestyle interventions sending the disease into remission.

It is, however, quite hard to know for sure how many people are able to successfully pull off such a reversal. After all, hundreds of millions of people around the world are currently diabetic, but millions of them aren't even aware they have the condition.

Against such a backdrop – and outside of scientific experiments specifically measuring type 2 diabetes remission – it's difficult to say how many people might develop the condition before going on to successfully reverse it.

Nonetheless, a new study from Scotland suggests the phenomenon might be more common than we realized, even without things like scientific interventions and invasive procedures such as bariatric surgery.

"We have been able to show, for the first time, that one in 20 people in Scotland with type 2 diabetes achieves remission," says clinical diabetes researcher Mireille Captieux from the University of Edinburgh. "This is higher than expected and indicates a need for updated guidelines to support clinicians in recognizing and supporting these individuals."

In their study, Captieux and her co-authors assessed a national Scottish diabetes registry, containing data for over 99.5 percent of people with a diagnosis of the condition in the country.

They identified 162,316 individuals over the age of 30 with type 2 diabetes on the basis of HbA1c (glycated hemoglobin) readings in the diabetic range.

From this cohort, during the study window (the calendar year of 2019), a total of 7,710 people went into remission on the basis of their HbA1c reading dropping below the diabetic range of 48

mmol/mol (6.5 percent), representing approximately 4.8 percent of the group.

Individuals who were more likely to go into remission were older, had lost weight since their diagnosis, had no history of glucose lowering therapy or bariatric surgery, and generally had healthier blood readings at the time of their diagnosis.

"Our prevalence estimates suggest that a reasonably large proportion of people achieve remission of type 2 diabetes in routine clinical care outside trial or bariatric surgery settings," the researchers write in their paper.

"The immediate implications for practice are that these people should be recognized and coded appropriately so they can be given adequate support and followed up to ensure continued care consistent with diabetes management guidelines. It is important to recognize that remission of diabetes may not be permanent."

Beyond helping us to support people who appear to successfully reverse their type 2 diabetes on their own, the findings could go some way to helping researchers and health workers identify which patients might be most likely to achieve and maintain remission.

It's as yet unclear how these results from Scotland might apply to communities elsewhere, but one thing's for sure.

With estimates predicting that today's population of roughly 460 million diabetics worldwide will expand to some 700 million people by 2045, we need plenty more insights on how to turn this disease around, and soon.

The findings are reported in *PLOS Medicine*.

https://bit.ly/3kejqkO

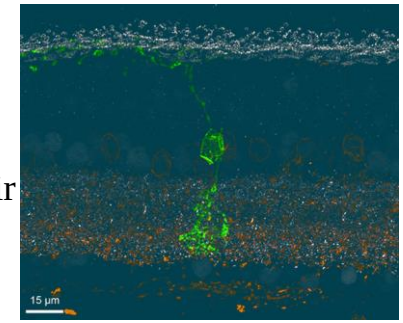
New Type of Neuron Discovered in Mammalian Retina

Newly-discovered retinal neurons don't fit into any existing mammalian retinal cell class

Cell classes are the building blocks for the central nervous system. It is widely believed that major classes of neurons have been identified in the retina, although some types have not been fully characterized. The newly-discovered retinal neurons don't fit into any existing mammalian retinal cell class.

"Photons entering the eye are detected by photoreceptors and processed through a set of function-specific synaptic pathways in the retina," said University of Utah's Dr. Ning Tian and colleagues.

"The structural basis of these pathways are the synaptic connections among five major classes of retinal neurons: photoreceptors, horizontal cells, bipolar cells, amacrine cells, and retinal ganglion cells. Identifying each of the retinal cell classes and understanding their synaptic connections is crucial for understanding how the retina processes visual signals."



An image of an isolated Campana cell (green) in a marmoset retina. The Campana cell receives input from photoreceptor synapses (white, top) in the primate retina. **Image credit: University of Utah.**

"While all the major retinal classes are believed to be defined, we identified a previously undescribed retinal interneuron."

The newly-identified interneuron shares fundamental morphological, physiological, and molecular features with bipolar cells. "Based on its morphology, physiology, and genetic properties, this cell doesn't fit into the five classes of retinal neurons first identified more than 100 years ago," Dr. Tian said. "We propose they might belong to a new retinal neuron class by themselves."

The authors named their discovery the Campana cell after its shape, which resembles a hand bell.

These cells relay visual signals from both types of light-sensing rod and cone photoreceptors to the retinal ganglion cells, but their precise purpose is the subject of ongoing research.

Experiments showed Campana cells remain activated for an

unusually long time — as long as 30 seconds — in response to a 10 millisecond light flash stimulation.

"In the brain, persistent firing cells are believed to be involved in memory and learning," Dr. Tian said. "Since Campana cells have a similar behavior, we theorize they could play a role in prompting a temporal 'memory' of a recent stimulation."

The discovery is reported in a paper in the *Proceedings of the National Academy of Sciences*.

Brent K. Young et al. 2021. An uncommon neuronal class conveys visual signals from rods and cones to retinal ganglion cells. PNAS 118 (44): e2104884118; doi: 10.1073/pnas.2104884118

https://bit.ly/3BZ2vsA

Samoan Medicinal Plant May Be as Effective as Ibuprofen, Study Says

The poorly understood 'matalafi' is widely used in Samoan traditional medicine to treat inflammation

The poorly understood 'matalafi' — the homogenate of *Psychotria insularum* (ボチョウジ属) leaves (fresh leaf juice) — is widely used in Samoan traditional medicine to treat inflammation associated with fever, body aches, swellings, wounds, elephantiasis, incontinence, skin infections, vomiting, respiratory infections, and abdominal distress.

***Psychotria insularum*. Image credit: Victoria University of Wellington.**

Compounds from natural resources are reliable lead templates of new pharmaceuticals, having persisted through evolutionary selection to control fundamental molecular pathways.

Of the 1,562 newly approved drugs from 1981 to 2019, 64% were either natural products, derived from natural products, or based upon natural product scaffolds, biological macromolecules, or botanical drugs.

Psychotria insularum is a small tree approximately 2 m in height with small white flowers and glossy red berries.

Known locally as matalafi, the homogenate of *Psychotria insularum* leaves is used in Samoan traditional medicine to treat inflammation associated with fever, body aches, swelling, wounds, incontinence, skin infections, elephantiasis, vomiting, respiratory infections, and abdominal distress.

"Matalafi is used in two ways in Samoa: to treat illnesses attributed to ghosts, and to treat various forms of inflammation," said indigenous Samoan Dr. Seeseei Molimau-Samasoni, a researcher at the Scientific Research Organisation of Samoa and the Victoria University of Wellington.

"We used chemical genomic analyses in the model organism *Saccharomyces cerevisiae* (baker's yeast) to identify and characterize an iron homeostasis mechanism of action in the traditional medicine as an unfractionated entity to emulate its traditional use."

The researchers found that matalafi interacts with the iron within cells of the body. They identified bioactive compounds — namely rutin and nicotiflorin — which both act to bind iron in a process called iron chelation. They also found that matalafi exhibits anti-inflammatory activity remarkably similar to that of ibuprofen.

Iron chelators like matalafi also have the potential to treat iron overload associated with transfusions, and have also been identified as prospective agents against common diseases like cancer, neurodegenerative diseases, cardiovascular diseases, and diabetes.

"This raises the possibility for applications of matalafi beyond traditional use," said Dr. Helen Woolner, a researcher at the Victoria University of Wellington.

"Our findings also highlighted the sensitivity of the RIM101 gene deletion to the *Psychotria insularum* homogenate," said Dr.

Andrew Munkacsi, also from the Victoria University of Wellington. "This gene is a major regulator of lipotoxicity which underlies obesity."

"Also, molecular studies published last year predicted rutin as a strong contender in inhibiting the viral replication of the SARS-CoV-2 virus that causes COVID-19." "This project is unique in integrating traditional knowledge with different types of biological and chemical methodologies," Dr. Molimau-Samasoni said.

The findings were published in the *Proceedings of the National Academy of Sciences*.

Seeseei Molimau-Samasoni et al. 2021. Functional genomics and metabolomics advance the ethnobotany of the Samoan traditional medicine 'matalafi.' PNAS 118 (45): e2100880118; doi: 10.1073/pnas.2100880118

https://bit.ly/3ERqcou

Specks of dust on the microscope slide? No, we are looking at the building blocks of our genome

"Microchromosomes" are almost identical, and represent ancient chromosomes of a spineless animal ancestor that lived 684 million years ago.

Jenny Graves*

If you look at cells from a human or other mammal under a microscope, you'll see big fat molecular complexes called chromosomes that contain our DNA. If the cells are from a bird or reptile, you'll see a few of these chunky chromosomes but also a flotilla of tiny specks that look like broken-down pieces of chromosomes or even specks of dust.

Those specks turned out to be tiny chromosomes, but their significance has been a mystery for decades. I assembled a talented team of young genome scientists to show that these "microchromosomes" are almost identical, and they represent the ancient chromosomes of a spineless animal ancestor that lived 684 million years ago.

The human genome and human chromosomes

The human genome comprises about 3 billion base pairs of DNA, each one like a rung on a long, twisted ladder. If you stretched the whole genome out, it would be about 1 metre long. It contains about 20,000 genes and a lots of repetitive sequences of DNA with few known functions.

Our genome is broken up into 23 bits. We can see these bits when a cell divides into two, because during this process the DNA condenses with proteins into chromosomes (literally "staining bodies") which we can see under the microscope. We have two copies of the genome in each of our cells (one from our mum and one from our dad), so we see 46 chromosomes in each cell.

Other mammals have pretty much the same set of genes on a similar length of DNA, but it is broken up differently. Some animals have lots of small chromosomes (there is a South American rat with 51) and others have a few big ones (the swamp wallaby has only 5).

Surprisingly, other higher vertebrates (birds and reptiles), though they have somewhat smaller genomes (1 or 2 billion base pairs) have pretty much the same sets of genes – as do frogs and even fish. The genomes of all vertebrates are amazingly similar.

The story of microchromosomes

When we look at the *chromosomes* of birds, turtles and squamates (snakes and lizards), however, we see big differences from those of mammals. They have between six and nine normal-looking chromosome pairs, but also lots of tiny elements that at first were thought to be degraded bits of chromosome or even dust on the microscope slide.

However, it proved that these elements were present in a constant – and even – number. Most birds have 62, representing 31 pairs of tiny "microchromosomes".

Although microchromosomes are tiny, they have the same ends

(telomeres) and attachment points (centromeres) as larger chromosomes. Curiously, they seem to hang out together in the centre of the cell.

The real surprise came when it became possible to sequence bits of chicken microchromosome DNA and check out the genes they contained.

It turned out that chick microchromosomes carry a big share of the genes and contain far fewer repetitive sequences than the large "macrochromosomes". In fact, about half the chicken genes lie on microchromosomes. This implied that microchromosomes are important parts of the bird genome.

But the mystery remained. Why are there two such distinct size classes of chromosomes in birds and other reptiles? And why do you always see microchromosomes huddled together in the centre of the cell?

Microchromosomes are highly conserved across birds and reptiles

Thanks to huge improvements in DNA sequencing technology, there are now well-assembled end-to-end or "telomere-to-telomere" sequences of many birds and reptiles.

In our new work, we have lined up DNA sequences of macro- and microchromosomes between several birds, turtles and squamates. We see startling similarities in the sequences. Emus and pigeons are only distantly related to chickens, as birds go, but they have virtually the same chromosomes. Turtles and squamates have fewer microchromosomes than birds, but the ones they do have are very similar within each group.

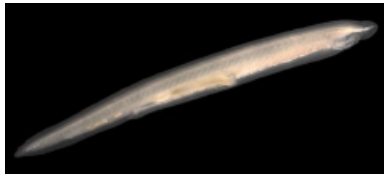
About half the genes of a chicken are carried in microchromosomes. **Fernando de Sousa, CC BY-SA**

When we compared sequences between emus, turtles and squamates, we saw a high degree of homology in microchromosome DNA sequences stretching over the nearly 300 million years since these species last shared a common ancestor. Turtles and squamates each carry different subsets of emu microchromosomes. We could see the lost microchromosomes; they had fused with each other or with macrochromosomes.

This suggested that 31 bird microchromosomes was present in the genome of a common ancestor of birds and reptiles about 300 million years ago, and turtles and squamates independently lost different subsets of these.

We used new techniques to reveal which bits of DNA are physically closest to which in the DNA tangle of a non-dividing cell. This showed that microchromosomes play tag with each other, and not with macrochromosomes.

This gives molecular reality to the old observations that microchromosomes lie close together in bird and reptile cells. It looks like microchromosomes form a compartment in the cell that might help the genes work together.



The tiny chromosomes of the amphioxus or lancelet are the building blocks of the genomes of modern vertebrates. **Hans Hillewaert, CC BY**

Microchromosomes are ancient genetic elements

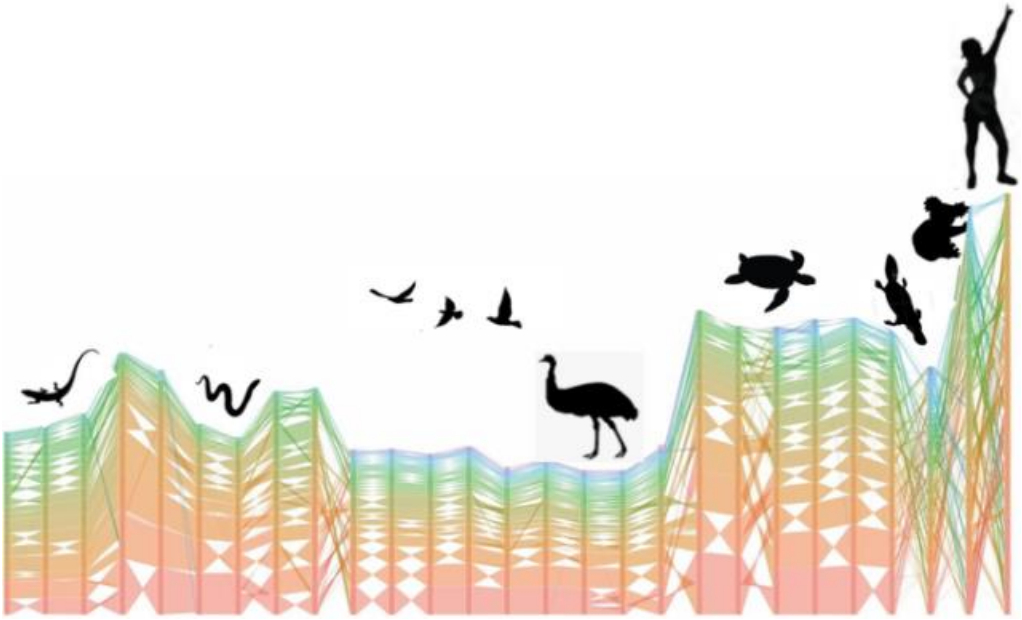
As it turns out, microchromosomes go back far, far further than the ancestral reptile: all the way to the tiny chromosomes of a very distantly related animal called the amphioxus or lancelet. Lancelets are small fish-like invertebrates that last shared a common ancestor with vertebrates 684 million years ago, long before the spine evolved.

Lancelets have a very small genome (520 million base pairs) cut up into 19 tiny, gene-dense chromosomes. This genome was duplicated twice during the evolution of the fish that gave rise to

animals with four limbs (tetrapods).

We found that most emu microchromosomes aligned with a single lancelet chromosome, or sometimes with two. So the tiny lancelet chromosomes have survived almost unchanged as bird and reptile microchromosomes. The rest of the vertebrate genome is made up of copies of these chromosomes, diluted with enormous amounts of repetitive DNA.

This means that the tiny lancelet chromosomes, represented today by bird and reptile microchromosomes, were the original building blocks of vertebrate genomes.



Genomes of lizards and snakes, birds, turtles and mammals (vertical lines show genome size) with DNA sequences lined up between chromosomes (coloured by size, microchromosomes in blue/green). Chromosomes have stayed the same in birds and reptiles but gone mad in mammals. **Genome array by Hardip Patel, Paul Waters, Nick Lister. Author provided**

Mammal genomes have gone mad

Some reptile and bird groups seem to have lost all or most of their microchromosomes. We show that, in these exceptional genomes, microchromosomes fused with each other (as in crocodiles) or with macrochromosomes (as in eagles and their relatives).

But mammals are the real exceptions. They have no microchromosomes. When we lined up emu sequence against the human and koala genomes (representing the marsupial and placental branches of the mammal family tree), we could find only small patches of homology with microchromosomes, scattered all over the genome.

However, in monotremes (egg-laying mammals that represent a third, and the oldest, branch of mammals), we saw that four platypus chromosomes are composed entirely of fused microchromosomes.

This implies that microchromosomes fused together into large blocks in a reptile-like mammal ancestor more than 200 million years ago. The chromosomes stayed that way in monotremes. But in our own lineage (therian mammals that diverged into marsupials and placental mammals), blocks of micro- and macrochromosomes were rearranged, obliterating their origins.

After this rearrangement, marsupial chromosomes stayed quite conserved, 19 large blocks of genes being shifted around in simple ways. However, the chromosomes of placental mammals have gone quite mad, rearranging multiple times in many lineages.

Such dizzying chromosome variation is unusual in vertebrates.

So the tiny microchromosomes of birds and reptiles are really the "normal" chromosomes rather than our big, fat mammal chromosomes that are scrambled and inflated by repetitive DNA sequences.

***** *Distinguished Professor of Genetics and Vice Chancellor's Fellow, La Trobe University*

Disclosure statement

Jenny Graves receives funding from the Australian Research Council.

https://bit.ly/3GVPkfv

Psilocybin reduced depression symptoms as much as a leading antidepressant

New research compared the "magic mushrooms" component to Lexapro

Soren Emerson

Since their introduction in the late 1980s, selective-serotonin reuptake inhibitors (SSRIs) have become the go-to treatment for major depression. SSRIs, however, have a number of limitations: they take several weeks to start working, can cause a variety of side-effects, and do not help some people with depression. A series of recent clinical investigations suggest that psilocybin, the active compound in magic mushrooms, may be an effective alternative. One question that these studies left unanswered, however, is how effective psilocybin treatment is compared to SSRIs.

In a first-of-its-kind study recently published in *The New England Journal of Medicine*, researchers at the Center for Psychedelic Research at Imperial College London compared psilocybin and escitalopram, an SSRI drug sold under the name Lexapro, as treatments for major depression. The six-week long study enrolled 59 volunteers with moderate-to-severe major depression. They were randomly and blindly assigned to receive treatment with psilocybin and an escitalopram control, or escitalopram and a psilocybin control. All the participants also received psychological support.

Psilocybin and escitalopram both reduce depression symptoms

To evaluate the two treatments, the researchers compared the change from baseline on the 16-item Quick Inventory of Depressive Symptomatology–Self-Report (QIDS-SR-16), a basic clinical measure of depression symptoms. Based on results of the QIDS-SR-16, psilocybin and escitalopram both reduce depression symptoms. The researchers did not detect a statistically significant difference between the two treatments.

The results of other measures taken in the study, however, suggest that psilocybin may be more effective than escitalopram. When designing the study, the researchers determined that the QIDS-SR-16 most directly addressed their experimental question and would therefore be the primary outcome measure, but they also evaluated depression symptoms with a number of additional scales. Nearly all secondary outcome measures favored psilocybin over escitalopram, but their results hold less weight than the QIDS-SR-16 because of how the study was designed.

The study was also limited by its small size, non-random enrollment of interested volunteers, and the possibility that participants may have been unblinded by the strong subjective effects of psilocybin or the well-known side-effects of SSRIs. Nonetheless, as the most rigorous evaluation of the therapeutic potential of psilocybin conducted to date, the results provide a benchmark for the design of future investigations.

https://bit.ly/3EVxMy6

Scientists Discover a Novel Therapeutic Target To Treat Fatty Liver Disease

About 80 million Americans have fatty liver disease unrelated to alcohol abuse.

Nonalcoholic fatty liver disease is associated with obesity and diabetes, and can lead to more severe liver damage such as nonalcoholic steatohepatitis (NASH), cirrhosis and liver cancer. Cardiovascular disease, colorectal cancer and breast cancer actually are the major causes of death in patients with fatty liver disease.

Several drugs in advanced stages of development have failed because of the complexity of the disease, low efficacy, or the toxicity of drugs. Although several clinical trials were conducted in past decades, currently there is no FDA-approved pharmaceutical therapy for NASH.

To understand the complexity of the progression of fatty liver

disease, a team of USC scientists explored the molecular mechanism in experimental NAFL/NASH. The project led to the discovery of a plausible therapeutic target gene, SH3BP5, also known as SAB. "The finding is the culmination of years of work by the team including USC bioinformatics specialists, pathologist, students, visiting scholars and collaborators," said Sanda Win, MD, PhD, assistant professor of research medicine in the GI/Liver division in the Department of Medicine at the Keck School of Medicine of USC.

As Win explains, SAB is an outer membrane protein of mitochondria, which is known as the powerhouse of the cell. The biological function of SAB was not known until the USC researchers first discovered it 10 years ago. SAB is a pivotal protein, and the level of SAB determines the severity of liver damage in an acetaminophen-induced liver injury model and a tumor-necrotic factor (TNF) induced acute liver failure model. SAB is a target of stress- activated kinase (JNK) which then leads to impaired mitochondrial function and an increase in toxic reactive oxygen species. Interestingly, SAB gene activation and protein levels increase in a diet-induced fatty liver and correlate with progression of the disease in experimental models and human fatty liver disease, Win added.

"We could prevent that whole progression by knocking out the SAB gene in the liver early on in these experiments in adult animals that were then fed a high-fat diet," said Neil Kaplowitz, MD, professor of medicine and the Thomas H. Brem Chair in Medicine at the Keck School. The project was initiated by a pilot grant to Win funded by USC Research Center for Liver Diseases, and the Donald E. and Delia Baxter Foundation Faculty Fellows award. The research recently was published in Hepatology, a journal by the American Association for the Study of Liver Diseases.

The mice were fed — overfed, really — a diet of high fat food pellets with added sucrose and fructose water. The long-term feeding of high-fat, high-sugar diet causes obesity, diabetes and fatty liver diseases. But even in mice that had been fed the high-fat, high-sugar diet for a year, "if we introduced this antisense targeting the liver cells, when the mice already had established disease with inflammation and fibrosis in the liver, we could reverse the entire thing, normalize their insulin resistance, and markedly decrease the fat accumulation in the liver and also the inflammation and fibrosis in the liver," Kaplowitz said.

One benefit, Win said, is "we don't need to delete or knock down, or knock out, the SAB protein entirely. Giving the dose, just to maintain the normal level of SAB prevents or reverses the disease progress." With the advantage of advanced science in antisense oligonucleotides (ASO), designed and synthesized by collaborators of Ionis Pharmaceuticals Inc., of Carlsbad, California, the team is optimistic about SAB-targeting DNA therapy.

The research shows just how much damage to the liver — from dietary choices — could be avoided through modest changes in behavior. Giving the mice antisense therapy during the first six months actually helped them lose weight. The authors caution that studies involving mice don't always translate to assumptions about humans.

"There's no question that many things that have been successful in mice don't work in humans," Kaplowitz said. But "our data suggests that this is a really strong potential therapeutic target, and we don't see any downside to directly interfering with SAB when lowering it."

About the study

This research was supported by NIH grants R01DK067215 (NK), the Veronica Garrie Budnick Chair in Liver Disease (NK), the Donald E. and Delia Baxter Foundation Faculty Fellows award (SW), a pilot project award (SW) by USC Research Center for Liver Diseases (P30DK048522), and a pilot project grant funding (SW) by the Rodent Metabolic Core of the Saban Research Institute of Children's Hospital Los Angeles.

https://bit.ly/3046qat

Spiny Mice Appear to Regenerate Damaged Kidneys

The mice, already known to regenerate skin, seem to avoid the tissue scarring that leads to organ failure in other animals.

Dan Robitzski

A peculiar rodent called the spiny mouse seems to be able to regenerate kidney tissue, according to research published today (November 3) in *iScience*. After damaging their kidneys to simulate kidney disease, the scientists found that the spiny mice not only regenerated the structure and function of nephrons, the tiny filters that make up the kidney, but they did so without the dangerous scarring that normally occurs in mammals.

Spiny mice, a collection of several species in the genus *Acomys*, are famous for their stiff coats of hair that resemble a hedgehog's quills. The critters were already important to scientists studying regeneration, as they have an unusual defense mechanism in which they shed their skin to escape predators. A 2012 study in *Nature* revealed that spiny mice regenerate all of the tissue they give up, including vasculature and hair follicles, without any scarring, a process that subsequent research found may stem from a reduced inflammatory immune response to injuries. In the new study, researchers set out to determine whether the mice could pull the same regenerative trick with their internal organs.

Lead study author and regenerative medicine researcher Mark Majesky and his team at the University of Washington and Seattle Children's Research Institute contrasted how spiny mice and house mice (*Mus musculus*) responded to kidney injuries. To do so, they operated on the mice to obstruct urine flow into the kidney and also directly damaged tissue, then watched to see whether organ structure and function returned. The process seems to trigger "scarless, regenerative wound healing" in the spiny mice, Majesky tells *The Scientist* in an email.

The same injuries from which the spiny mice seemingly escaped unscathed led to scarring in the house mice. As in human organ damage, that scarring can build up over time and cause fatal organ failure down the road—suggesting that unlocking the secrets of mammalian regeneration could someday prove invaluable to medicine.

"We used the term 'functional regeneration' because spiny mice sustain severe kidney injury initially but then completely restore kidney function within two weeks. This differs from many kinds of 'repair' responses, including fibrotic repair, that restore tissue continuity but do so with variable degrees of loss of organ function," Majesky writes in his email.

"It sounds as if this new paper does show true restoration of injured *Acomys* kidney tissue, which I would explain by the immunodeficient status of this unusual species allowing patterning of new nephrons rather than scarring," Anthony Mescher, an emeritus professor of anatomy and cell biology at Indiana University School of Medicine who didn't work on the new study, emails *The Scientist*. The discovery, he adds, is "rather remarkable."

Spiny mice aren't the only mammals with regenerative capabilities, explains Rachel Sarig, a molecular cell biologist and regeneration expert at the Weizmann Institute of Science in Israel who didn't work on the paper. They're joined by animals like deer, which can grow new antlers, and MRL mice, which can regrow skin, hair, ears, and even some organs all without scarring. Even neonate mice can regenerate heart tissue but lose the ability during the first week of life.

Still, regeneration is typically not mammals' forte, and examples of the phenomenon in these animals are so rare that they're inevitably met with excitement. Regeneration is far more common among animals like the zebrafish, which can regrow pieces of its heart

even after 20 percent has been removed, and salamanders, which famously regrow entire limbs. But fish, reptiles, and amphibians rely on a different biological process to regenerate lost limbs or tissue than do mammals, and more work is needed to uncover the exact mechanisms at play in spiny mice and what other organs or tissues they might apply to. "In our lab, we definitely will try to see what else these mice can regenerate—maybe their hearts," Sarig says. "Maybe we can learn from them what is missing for us"—that is, why humans don't have the same regenerative capacity.

Multiple researchers tell *The Scientist* that getting to the bottom of mammalian regeneration could prove invaluable for developing new treatments for organ damage, whether it stems from severe injury or disease.

However, University of Kentucky animal regeneration researcher Ashley Seifert, who was not involved in the new study but was one of the researchers behind the 2012 skin regeneration paper, says that some key aspects of the paper gave him pause, and he will be interested to see what happens when other researchers attempt to replicate the study.

One potential issue, Seifert notes, is that it's particularly difficult to conduct a surgical procedure on two different animal species and ensure that the same injury produces the same initial effect in both.

"One thing that troubled me about this particular paper . . . is that it almost looks like they never caused any damage whatsoever" to the spiny mice, Seifert tells *The Scientist*. Seifert points out that the researchers found almost no collagen buildup in the spiny mice after injury. The authors pointed to that lack of collagen as a sign that the mice were healing without forming scars, but collagen is an expected part of healing and recovery that even other regenerating animals experience, Seifert explains.

Seifert adds that the paper's methodology lacks details that would be helpful in evaluating and replicating the work. For instance, the study authors say they experimented on adult spiny and house mice, but because the species have significantly different lifespans, a spiny and house mouse of the same age may be at different stages of life. Perhaps the authors controlled for that discrepancy—and Seifert adds that he believes the authors' methods were sound—but without clarification in the paper, it's impossible to tell whether there were issues or errors. "At the end of the day, science needs to be reproduced to be worthwhile," Seifert says.

Similarly, Sarig adds that she hopes researchers will conduct a more precise analysis of the genetic and epigenetic mechanisms responsible for the apparent regeneration in order to paint a clearer picture of what's going on at a molecular level.

While much research remains to be done before the finding might be translated to anything of clinical relevance to humans, Sarig says papers of this sort give her hope for a future where regenerative medicine can treat diseases or perhaps prevent organ failure in people.

"A decade ago, when [scientists] started to study heart regeneration, it was like science fiction; it seemed impossible," Sarig says. "Now we know it is possible. During those ten years, we found several factors, several strategies, which we can use to induce adult mammalian heart repair. So we know it is possible, we just need to find the right factors—the right signal."

Seifert adds that even if future researchers and clinicians never quite figure out true regeneration for human tissue, taking lessons from the spiny mouse or other animals with incredible healing abilities could still prove valuable for emergency medicine situations. Finding new ways to induce healing, whether or not that involves scarless regeneration of tissue, could lead to therapeutics that keep hospitalized heart failure patients alive, he explains.

"We [know] that over 600,000 Americans have kidney failure and over 450,000 patients, including children, are currently on dialysis,"

Majesky writes. “Most of those patients have progressive kidney fibrosis leading to kidney failure. We conducted our research with those individuals in mind.”

https://bit.ly/3wnywJn

A Vaccine Is Urgently Needed For Infection Killing 150,000 Babies Annually, WHO Warns

WHO call for development of a vaccine against Group B Streptococcus, responsible for 150,000 stillbirths and infant deaths yearly

The World Health Organization (WHO) on Wednesday called for the urgent development of a vaccine against a bacterial infection responsible for nearly 150,000 stillbirths and infant deaths each year.

A fresh report by the UN health agency and the London School of Hygiene and Tropical Medicine found that the impact of Group B Streptococcus infection (GBS), which is estimated to live harmlessly in the intestinal tracts of up to a third of all adults, is a far bigger cause of preterm births and disability than previously thought.

The report confirmed a previous devastating finding from 2017 that the bacterium causes almost 100,000 newborn deaths and close to 50,000 stillbirths each year, although it pointed to significant data gaps suggesting the true figures could be higher.

And for the first time it quantified the impact on preterm births, finding that GBS is behind more than half a million early deliveries each year, leading to significant long-term disability. In light of such staggering numbers, the report authors lamented that more progress had not been made towards developing a vaccine.

"This new research shows that Group B strep is a major and underappreciated threat to newborn survival and wellbeing, bringing devastating impacts for so many families globally," Phillipp Lambach of WHO's immunization department said in a statement. "WHO joins partners in calling for urgent development of a maternal GBS vaccine, which would have profound benefits in countries worldwide."

Professor Joy Lawn, who heads LSHTM's maternal adolescent, reproductive and child health center, agreed. "Maternal vaccination could save the lives of hundreds of thousands of babies in the years to come," she said, decrying the lack of progress since the idea of developing a jab against GBS was first raised three decades ago.

On average, 15 percent of pregnant women worldwide, or nearly 20 million annually, carry the GBS bacterium in their vagina.

But even though most of these cases show no symptoms, an infected pregnant woman can pass GBS to her fetus via the amniotic fluid, or during birth as the infant passes through the vaginal canal.

Babies and fetuses are particularly vulnerable because their immune systems are not strong enough to fight the multiplying bacteria.

If untreated, GBS can lead to meningitis and septicemia, which can be deadly. Babies that survive may develop cerebral palsy, or permanent sight and hearing problems.

Wednesday's report showed that the bacterium leaves some 40,000 infants each year with neurological impairments.

Currently, women with GBS are given antibiotics during labor to reduce the chance of it passing to their baby. But this approach poses problems in places where screening and antibiotic administration during labor are less accessible.

Tellingly, the highest rates of maternal GBS are found in sub-Saharan Africa – which alone accounts for around half of the global burden – and Eastern and South-Eastern Asia, the study showed.

It suggested that a GBS vaccine that could be administered to pregnant women during routine pregnancy checkups and that reached over 70 percent of pregnant women could avert 50,000 infant and fetus deaths each year.

https://bit.ly/3wptpZt

HPV vaccine slashes cervical cancer rates by 87% among women in the UK

A study finds that the vaccine is most effective when given to children between the ages of 12 and 13.

By Yasemin Saplakoglu

The human papillomavirus (HPV) vaccine reduced cervical cancer cases by 87% among women in the U.K. who received the vaccine when they were 12 or 13 years old, according to a new study.

These new findings are based on follow-up data from a vaccination program that began in the U.K. in 2008. The strains of human papillomavirus that are transmitted through sexual contact are extremely common around the world, and most people are infected at some point in their lives, typically soon after becoming sexually active, according to The World Health Organization (WHO).

Many HPV infections clear up on their own without causing issues, but some infections can lead to cervical cancer. In fact, nearly all cervical cancer cases around the world are due to an infection with HPV, according to the WHO. Now, more than 100 countries, including the U.K. and the U.S., offer HPV vaccinations to young girls, and some countries are also offering them to young boys to prevent genital warts and cancer.

As part of the U.K. program, girls between the ages of 12 and 13 were given an HPV vaccine called Cervavix, which protects against the two most common types of HPV that account for 70 to 80% of all cervical cancers, according to a statement. The vaccine was also offered to women up to age 18 as a"catch-up" vaccination.

In the new study, the researchers analyzed data collected between 2006 and 2019 from a cancer registry. They compared cervical cancer rates between women who were vaccinated with Cervavix when they were younger and those who weren't; the researchers further divided those who were vaccinated into groups based on their age of vaccination.

Between 2006 and 2019, there were 28,000 cervical cancer diagnoses in the U.K. and 300,000 cases of non-invasive cervical carcinoma (CINN3), abnormal cells on the cervix that can turn into cancer if left untreated. That's about 450 cases of cervical cancer and 17,200 fewer cases of cervical pre-cancer than expected in the general population, according to the statement.

The researchers found that the vaccine was most effective when given to the younger cohort; women who were vaccinated with Cervavix between the ages of 12 and 13 had 87% fewer cases of cervical cancer compared with those who weren't vaccinated. Women vaccinated between the ages of 14 and 16 and those between the ages of 16 and 18, there was a 62% and a 34% reduction in cases compared to the unvaccinated population, respectively. (The vaccine was less effective for girls vaccinated at older ages because more of them were sexually active and therefore exposed to the virus before getting vaccinated. The vaccines work best before people are exposed to the virus.)

"Although previous studies have shown the usefulness of HPV vaccination in preventing HPV infection in England, direct evidence on cervical cancer prevention was limited," senior author Peter Sasieni, a professor in the King's College London said in the statement. Early modeling had predicted that HPV vaccination would reduce cervical cancer rates substantially in young women, he said. "The observed impact is even greater than the models predicted," he added.

There are some limitations to the study, including that the vaccinated population is still young and so it's still early to understand the full impact of the HPV vaccination program, according to the statement.

Since 2012, the U.K. has been using another HPV vaccine called Gardasil that protects against four different types of HPV and

wasn't evaluated in this paper. The U.S. is administering Gardasil-9, which protects against nine different HPV types. All three vaccines protect against the the two most common HPV types that cause cancer.

The Centers for Disease Control and Prevention (CDC) recommends that two doses of the HPV vaccine be given 6 to 12 months apart to children between the ages of 11 and 12, but can be given as early as age 9. Those who are 15 years of age or older need three doses over 6 months.

https://bit.ly/3bWzQcJ

COVID Alpha Variant Detected in Dogs and Cats – Pets Had Acute Onset of Cardiac Disease, Including Severe Myocarditis

First identification of the SARS-CoV-2 alpha variant in domestic pets - many owners of these pets had also tested positive for COVID-19.

A new study in the *Veterinary Record* reveals that pets can be infected with the alpha variant of SARS-CoV-2 (the virus that causes COVID-19 in humans), which was first detected in southeast England and is commonly known as the UK variant or B.1.1.7. This variant rapidly outcompeted pre-existing variants in England due to its increased transmissibility and infectivity.

The study describes the first identification of the SARS-CoV-2 alpha variant in domestic pets; two cats and one dog were positive on PCR test, while two additional cats and one dog displayed antibodies two to six weeks after they developed signs of cardiac disease. Many owners of these pets had developed respiratory symptoms several weeks before their pets became ill and had also tested positive for COVID-19.

All of these pets had an acute onset of cardiac disease, including severe myocarditis (inflammation of the heart muscle).

"Our study reports the first cases of cats and dogs affected by the COVID-19 alpha variant and highlights, more than ever, the risk that companion animals can become infected with SARS-CoV-2," said lead author Luca Ferasin, DVM, PhD, of The Ralph Veterinary Referral Centre, in the UK. "We also reported the atypical clinical manifestations characterized by severe heart abnormalities, which is a well-recognized complication in people affected by COVID-19 but has never described in pets before. However, COVID-19 infection in pets remains a relatively rare condition and, based on our observations, it seems that the transmission occurs from humans to pets, rather than vice versa."

Reference: "Infection with SARS-CoV-2 variant B.1.1.7 detected in a group of dogs and cats with suspected myocarditis" by Luca Ferasin, Matthieu Fritz, Heidi Ferasin, Pierre Becquart, Sandrine Corbet, Meriadeg Ar Gouilh, Vincent Legros and Eric M. Leroy, 4 November 2021, Veterinary Record. DOI: 10.1002/vetr.944

https://bbc.in/3kihEPE

Covid: Pfizer says antiviral pill 89% effective in high-risk cases

A pill to treat Covid developed by the US company Pfizer cuts the risk of hospitalisation or death by 89% in vulnerable adults, clinical trial results suggest.

By Jim Reed and Philippa Roxby

The drug - Paxlovid - is intended for use soon after symptoms develop in people at high risk of severe disease.

It comes a day after the UK medicines regulator approved a similar treatment from Merck Sharp and Dohme (MSD). Pfizer says it stopped trials early as the initial results were so positive.

The UK has already ordered 250,000 courses of the new Pfizer treatment, which has not yet been approved, along with another 480,000 courses of MSD's molnupiravir pill.

Health and Social Care Secretary Sajid Javid called the results "incredible", and said the UK's medicines regulator would now assess its safety and effectiveness. "If approved, this could be

another significant weapon in our armoury to fight the virus alongside our vaccines and other treatments," he said.

The Pfizer drug, known as a protease inhibitor, is designed to block an enzyme the virus needs in order to multiply. When taken alongside a low dose of another antiviral pill called ritonavir, it stays in the body for longer. Three pills are taken twice a day for five days. The combination treatment, which is still experimental because trials haven't finished, works slightly differently to the Merck pill which introduces errors into the genetic code of the virus.

Pfizer said it plans to submit interim trial results for its pill to US medicines regulator the FDA as part of the emergency use application it started last month. Full trial data has not yet been published by either company. The US has already secured millions of doses of the pill, according to President Joe Biden.

The company's chairman and chief executive Albert Bourla said the pill had "the potential to save patients' lives, reduce the severity of Covid-19 infections, and eliminate up to nine out of 10 hospitalisations".

Trial results

Vaccines against Covid-19 are seen as the best way of controlling the pandemic but there is also demand for treatments that can be taken at home, particularly for vulnerable people who become infected.

Interim data from trials of the treatment in 1,219 high-risk patients who had recently been infected with Covid found that 0.8% of those given Paxlovid were hospitalised, compared with 7% of patients who were given a placebo or dummy pill.

They were treated within three days of Covid symptoms starting.

Seven patients given the placebo died compared to none in the group given the pill.

When treated within five days of symptoms appearing, 1% given Paxlovid ended up in hospital and none died. This compared to 6.7% of the placebo group being hospitalised and 10 of them dying.

Patients in the trial, which has not yet been published or verified, were elderly or had an underlying health condition which put them at higher risk of serious illness from Covid. They all had mild to moderate symptoms of coronavirus.

Dr Stephen Griffin, associate professor in the School of Medicine at the University of Leeds, said: "The success of these antivirals potentially marks a new era in our ability to prevent the severe consequences of Sars-CoV2 [coronavirus] infection, and is also a vital element for the care of clinically vulnerable people who may be unable to either receive or respond to vaccines."

Pfizer is also studying the treatment's impact on people at low risk of Covid illness and on those who have already been exposed to the virus by someone in their household.

Analysis

By James Gallagher Health and science correspondent

Developing truly effective antiviral drugs is notoriously difficult, so having two that look highly potent against Covid is a remarkable feat. Viruses are much simpler beasts than bacteria or parasites.

That sounds like they should be easier to defeat, but in reality it means there are far fewer weak spots for drugs to exploit.

There is also a wide variety of different types of virus that exploit our bodies in different ways, which means scientists often have to go back to the drawing board for each one.

Then they hide inside our body's own cells, which means drugs that seem potent in the lab may not work as well in the body. There have been successes, notably in HIV, but reports concluded one antiviral for flu ended up being about as effective as paracetamol.

The question now is whether the success of these pills in clinical trials can be repeated in the real world, as people with Covid will have to be identified and treated within a matter of days of their symptoms developing.

https://bit.ly/3kghcRK

Giant, invasive spiders have taken over Georgia. Will they spread across the US?

The spiders arrived in 2014. Now, there are millions of them.

By Ben Turner 3 days ago

Joro spiders spin dense, gold-tinted webs **(Image credit: University of Georgia)**

Millions of giant spiders have invaded North Georgia, terrifying residents and spinning webs as thick as 10 feet (3 meters) deep.

Porches, power lines, mailboxes and vegetable patches across more than 25 counties in the state have been draped with the dense, wheel-shaped webs of the bright-yellow Joro spider (*Trichonephila clavata*), an invasive species originating in East Asia.

The first of the 3-inch (7.6 centimeters) spiders was spotted 80 miles (128 kilometers) northeast of Atlanta in 2014; it likely hitchhiked there inside a shipping container, its discoverer, Rick Hoebeke, the collections manager at the Georgia Museum of Natural History said in a statement.

Since then, the spider's population and range have expanded steadily across the state, but nothing prepared residents or researchers for the number of spiders they would face this year. Will Hudson, an entomologist at the University of Georgia, said his porch became unusable after being covered by a blanket of webs 10 feet (3 m) deep, and he claims to have killed more than 300 spiders.

"Last year, there were dozens of spiders, and they began to be something of a nuisance when I was doing yard work," Hudson said in the statement. "This year, I have several hundred, and they actually make the place look spooky with all the messy webs — like a scene out of 'Arachnophobia.'"

Common to China, Taiwan, Japan and Korea, Joro spiders are part of a group of spiders known as "orb weavers" because of their highly symmetrical, circular webs. Though they are venomous, they use the venom only to immobilise the prey they snare in their webs. The venom poses no threat to human beings, dogs or cats unless they are allergic to it. While the spiders may nip if threatened, their bites are not often strong enough to break the skin.

Most of Georgia's Joro spiders will probably die off by late November, but this is far from the last we will see of them. Now that the spiders have gained a foothold (or eight) in the U.S., experts believe that the arachnids could spread even farther into other states with similar climates. Female Joros lay egg sacs, spun together with silk, that contain at least 400 babies. When the hatchlings emerge in the spring, they ride the wind on a strand of silk, floating across enormous distances, much like the baby spiders in the E.B. White novel "Charlotte's Web".

A lot of invasive species tend to destabilize the ecosystems they enter, but some scientists are optimistic that the spiders could actually bring unexpected benefits. Nancy Hinkle, an entomologist at the University of Georgia, said Joro spiders kill off mosquitoes, biting flies and invasive brown marmorated stink bugs, which have no natural predators and are known for damaging crops.

"Joro spiders present us with excellent opportunities to suppress pests naturally, without chemicals, so I'm trying to convince people that having zillions of large spiders and their webs around is a good thing!" Hinkle said in the statement.

https://bit.ly/3o5cA1X

Researchers Call for Therapeutics To Treat Cholesterol Cousins Called Ceramides – Linked to Many Health Problems

Therapeutics that target lipids called ceramides might hold potential for treating cardiometabolic disease

Therapeutics that target lipids called ceramides might hold potential

for treating cardiometabolic disease, argues a review article published today (November 5th, 2021) in the journal *Trends in Pharmacological Sciences*.

The authors summarize evidence supporting a strong relationship between ceramides and a range of diseases in animals and humans and compare it to the decades of datasets that drove the creation of cholesterol-lowering pharmaceuticals.

"Scientists have a lot to do if we are going to realize the potential of ceramide-lowering therapies," says senior study author Scott Summers of the University of Utah College of Health.

"Our hope with the article was to compare the body of literature on ceramides with that of cholesterol in order to point out the critical gaps and emerging questions in the ceramide field.

Basically, we want to get as many labs as possible studying this important molecule."

One of the most widely prescribed drug classes is statins, which inhibit the synthesis of the lipid cholesterol to prevent coronary artery disease. Statins also reduce blood levels of other lipids such as ceramides.

Compared to what we know about cholesterol, much less is known about the role of ceramides in disease. But it is becoming increasingly clear that ceramides are linked to a broad swath of health problems.

Over the past couple of decades, studies in humans have shown that ceramides are standalone biomarkers of cardiovascular disease, independent of cholesterol.

Ceramides strongly predict major adverse cardiovascular events, including death due to coronary artery disease and acute coronary syndrome.

These results have been replicated across the world in different countries and ethnicities, highlighting the robust nature of the association.

Unlike cholesterol, ceramides have also been linked to metabolic conditions such as insulin resistance and diabetes in humans. Blood ceramides are now being measured clinically to assess disease risk.

Research in animals has provided evidence for a causal relationship and revealed potential disease mechanisms. For example, lowering ceramides through genetic or pharmacological interventions prevents cardiovascular disease and diabetes in rodents.

Other studies have shown that ceramides can lead to an increase in fat storage, a decrease in glucose use, and lower mitochondrial efficiency—hallmarks of metabolic syndrome.

Ultimately, these metabolic changes might lead to programmed cell death of pancreatic b-cells, thereby driving type 2 diabetes.

"Ceramides may prove to be as deleterious as cholesterol, as they elicit a non-overlapping spectrum of tissue defects and ultimately trigger cell death," Summers says.

Despite the accumulating evidence, many questions remain.

Currently, there is a lack of data to support specific clinical recommendations based on high ceramide scores.

More research is also needed to understand the genetic abnormalities that drive high ceramide levels and how ceramides damage cells and tissues.

According to the article's authors, answering these questions might shed light on potential therapeutic approaches to safely and effectively lower ceramides and treat cardiometabolic disease.

"Hopefully help is on the horizon, either in the way of new therapeutics or new diet recommendations," Summers says.

Reference: "Cholesterol – the devil you know; ceramide – the devil you don't" by Tippetts et al., 5 November 2021, Trends in Pharmacological Sciences.
DOI: 10.1016/j.tips.2021.10.001

This work was supported by the National Institutes of Health, the Juvenile Diabetes Research Foundation, the American Diabetes Association, the American Heart Association, and the Margolis Foundation. Scott Summers is a consultant, co-founder, and shareholder of Centaurus Therapeutics.

https://bit.ly/3mUG2sd

Man donated his body to science; company sold $500 tickets to his dissection

The widow learned of the dissection from a news reporter.

Beth Mole

A Louisiana widow is left horrified at the news that her deceased husband was dissected in front of a live, paying audience after she donated his body to scientific research.

Elsie Saunders had carried out the wishes of her late husband, David Saunders, who wanted his body donated to help advance medical science, according to The Advocate. David Saunders, a World War II and Korean War veteran, died of COVID-19 on August 24 at the age of 98. Donating his body was his last act of patriotism, Elsie Saunders said.

But instead of being delivered to a research facility, David Saunders' body ended up in a Marriott Hotel ballroom in Portland, Oregon, where DeathScience.org held an "Oddities and Curiosities Expo." At the October 17 event, members of the public sat ringside from 9 am to 4 pm—with a break for lunch—to watch David Saunders' body be carefully dissected. Tickets for the dissection sold for up to $500 per person.

The Advocate noted that an online description of the event read: "From the external body exam to the removal of vital organs including the brain, we will find new perspectives on how the human body can tell a story… There will be several opportunities for attendees to get an up close and personal look at the cadaver."

"Horrible, unethical"

Elsie Saunders learned of the dissection from a Seattle-based reporter at KING 5, who was investigating the event and tracked her down. A photojournalist who attended undercover for KING 5 had noted that the body had a bracelet with the typed name "David Saunders."

"As far as I'm concerned, it's horrible, unethical, and I just don't have the words to describe it," Elsie Saunders told The Advocate. "I have all this paperwork that says his body would be used for science—nothing about this commercialization of his death."

Elsie Saunders explained that she had initially tried to donate the body to Louisiana State University. But LSU turned down the donation due to the COVID diagnosis. She then connected with a private company named Med Ed Labs in Las Vegas, which says it was "established to provide medical and surgical education and training for the advancement of medical and surgical innovation."

Med Ed Labs subsequently sold the body to Death Science. An administrator for Med Ed Labs, Obteen Nassiri, told KING 5 that DeathScience.org founder Jeremy Ciliberto was "beyond" dishonest about how the body would be used. Nassiri said he believed Ciliberto would use the body for a medical class. Meanwhile, Ciliberto said Med Ed was fully aware that the body would be used in an event attended by people who were "not exclusively medical students."

Elsie Saunders is now hoping to have David Saunders' remains returned. According to KING 5, the Louisiana-based company Church Funeral Services and Crematory, which prepared David Saunders' body before it was given to Med Ed Labs, told Elsie Saunders it would track down her husband's remains, cremate them for free, and return them to her. "We're extremely sad for his widow," Church Funeral Services owner Greg Clark told the Advocate. "This is not what her intentions were."

https://bit.ly/3EUJp8J

A 150-Year-Old Note From Darwin Is Changing How We Plant Forests

Studies of this "Darwin effect" have spawned vast ecological literature.

Rob Mackenzie And Christine Foyer, The Conversation

More than 150 years ago Victorian biologist Charles Darwin made a powerful observation: that a mixture of species planted together often grow more strongly than species planted individually.

It has taken a century and a half – ironically about as long as it can take to grow an oak to harvest – and a climate crisis to make policymakers and landowners take Darwin's idea seriously and apply it to trees.

There is no human technology that can compete with forests for take-up of atmospheric carbon dioxide and its storage. Darwin's idea of growing lots of different plants together to increase the overall yield is now being explored by leading academics, who research forests and climate change.

Scientists and policymakers from Australia, Canada, Germany, Italy, Nigeria, Pakistan, Sweden, Switzerland, the UK, and the US came together recently to discuss if Darwin's idea provides a way to plant new forests that absorb and store carbon securely.

Why plant more forests

Planting more forests is a potent tool for mitigating the climate crisis, but forests are like complex machines with millions of parts. Tree planting can cause ecological damage when carried out poorly, particularly if there is no commitment to diversity of planting. Following Darwin's thinking, there is growing awareness that the best, healthiest forests are ones with the greatest variety of trees - and trees of various ages.

Forests following this model promise to grow two to fourfold more strongly, maximizing carbon capture while also maximizing resilience to disease outbreaks, rapid climate change, and extreme weather.

In mixed forests, each species accesses different sources of nutrients from the others, leading to higher yields overall. And those thicker stems are made mostly of carbon.

Mixed forests are also often more resilient to disease by diluting populations of pests and pathogens, organisms that cause disease.

Darwin's prescient observation is tucked away in chapter four of his 1859 famous book *On the Origin of the Species*. Studies of this "Darwin effect" have spawned vast ecological literature. Yet it is still so outside of the mainstream thinking on forestry that, until now, little major funding has been available to prompt use of this technique.

Darwin also famously described evolution by natural selection, a process by which genes evolve to be fit for their environment. Unfortunately for the planet, human-induced environmental change outstrips the evolution of genes for larger, slower reproducing, organisms, like trees.

Modern gene-editing techniques – direct DNA surgery – can help speed things up once careful laboratory work identifies the key genes. But only evolution of human practice – that is, changing what we do – is fast and far-reaching enough to rebalance the carbon cycle and bring us back within safe planetary limits.

Healthier trees capture more carbon

At our meeting we discussed a study of Norbury Park estate in central England, which describes how – using the Darwin effect and other climate-sensitive measures – the estate now captures over 5,000 tons of carbon dioxide per year, making it quite possibly the most carbon-negative land in the UK. Such impressive statistics don't happen by accident or by sticking some trees in the ground and hoping; care and ecological nous is needed.

Trees of different ages also continuously provide harvestable timber and so steady jobs, in stark contrast to the other methods of forestry, where large areas are felled and cleared at the same time.

The UK government, like other administrations, has laid down requirements for responsible large-scale tree planting. These requirements continue to be revised and improved. There are still vital questions about which trees we should plant, where we should

plant them, and what to do with them once they've grown.

It has been said that it is impossible to plant a forest, but it should certainly be possible to design a plantation that will blossom into a forest for future generations. We need forests to be a practical, dependable, and just response to our climate and biodiversity crises, and Darwin has shown us the way.

https://bit.ly/3qdAKtW

Brain Implant Translates Paralyzed Man's Thoughts Into Text With 94% Accuracy

Man paralyzed from the neck down can communicate his thoughts, thanks to a brain implant system that translates his imagined handwriting into text.

Peter Dockrill

A man paralyzed from the neck down due to a spinal cord injury he sustained in 2007 has shown he can communicate his thoughts, thanks to a brain implant system that translates his imagined handwriting into actual text.

The device – part of a longstanding research collaboration called BrainGate – is a brain-computer interface (BCI), that uses artificial intelligence (AI) to interpret signals of neural activity generated during handwriting.

In this case, the man – called T5 in the study, and who was 65 years of age at the time of the research – wasn't doing any actual writing, as his hand, along with all his limbs, had been paralyzed for several years.

But during the experiment, reported in *Nature* earlier in the year, the man concentrated as if he were writing – effectively, thinking about making the letters with an imaginary pen and paper.

As he did this, electrodes implanted in his motor cortex recorded signals of his brain activity, which were then interpreted by algorithms running on an external computer, decoding T5's imaginary pen trajectories, which mentally traced the 26 letters of the alphabet and some basic punctuation marks.

"This new system uses both the rich neural activity recorded by intracortical electrodes and the power of language models that, when applied to the neurally decoded letters, can create rapid and accurate text," says first author of the study Frank Willett, a neural prosthetics researcher from Stanford University.

Similar systems developed as part of the BrainGate have been transcribing neural activity into text for several years, but many previous interfaces have focused on different cerebral metaphors for denoting which characters to write – such as point-and-click typing with a computer cursor controlled by the mind.

It wasn't known, however, how well the neural representations of handwriting – a more rapid and dexterous motor skill – might be retained in the brain, nor how well they might be leveraged to communicate with a brain-computer interface, or BCI.

Here, T5 showed just how much promise a virtual handwriting system could offer for people who have lost virtually all independent physical movement.

In tests, the man was able to achieve writing speeds of 90 characters per minute (about 18 words per minute), with approximately 94 percent accuracy (and up to 99 percent accuracy with autocorrect enabled).

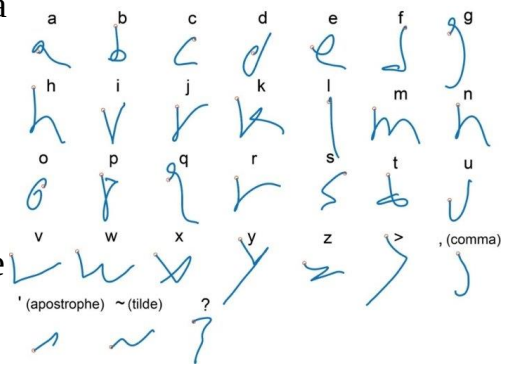
Not only is that rate significantly faster than previous BCI experiments (using things like virtual keyboards), but it's almost on par with the typing speed of smartphone users in the man's age group – which is about 115 characters or 23 words per minute, the researchers say.

"We've learned that the brain retains its ability to prescribe fine movements a full decade after the body has lost its ability to execute those movements," Willett says.

"And we've learned that complicated intended motions involving changing speeds and curved trajectories, like handwriting, can be

interpreted more easily and more rapidly by the artificial-intelligence algorithms we're using than can simpler intended motions like moving a cursor in a straight path at a steady speed."

Basically, the researchers say that alphabetical letters are very different from one another in shape, so the AI can decode the user's intention more rapidly as the characters are drawn, compared to other BCI systems that don't make use of dozens of different inputs in the same way.



The man's imagined handwriting, as interpreted by the system. **(Frank Willett)**

Despite the potential of this first-of-its-kind technology, the researchers emphasize that the current system is only a proof of concept so far, having only been shown to work with one participant, so it's definitely not a complete, clinically viable product as yet.

The next steps in the research could include training other people to use the interface, expanding the character set to include more symbols (such as capital letters), refining the sensitivity of the system, and adding more sophisticated editing tools for the user.

There's plenty of work to still be done, but we could be looking at an exciting new development here, giving the ability to communicate back to people who lost it.

"Our results open a new approach for BCIs and demonstrate the feasibility of accurately decoding rapid, dexterous movements years after paralysis," the researchers write.

"We believe that the future of intracortical BCIs is bright."

The findings are reported in *Nature*.