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WEEKLY 7 March 2026

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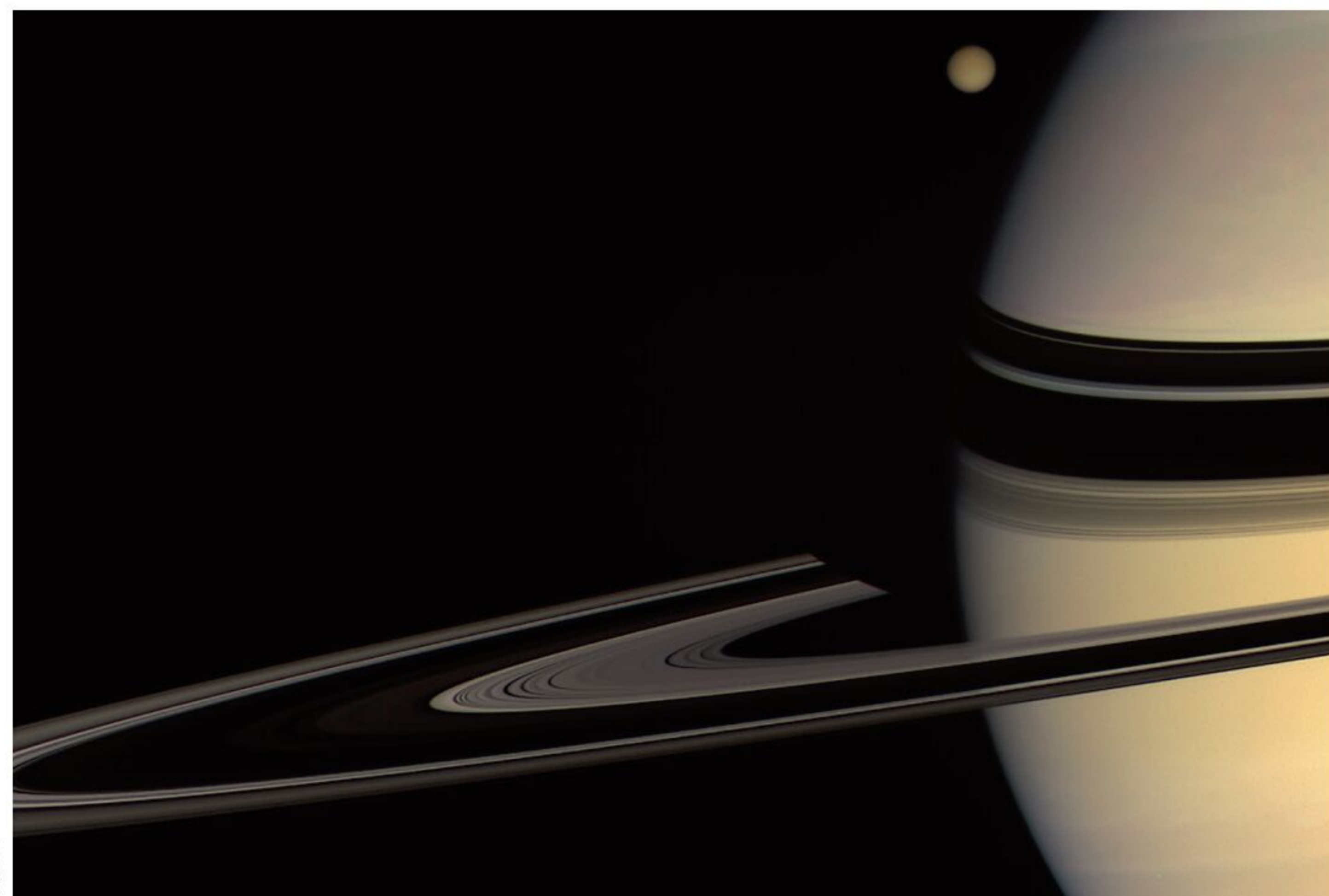
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Solar system

Is this how Saturn's rings were formed?

Leah Crane



A COLLISION involving Titan, the largest moon of Saturn, could have set into motion the series of events that formed Saturn's iconic rings and altered both the planet's wobble and the orbits of its moons.

Saturn is awash in mysteries. Its rings seem to be younger than expected, the planet's wobble isn't tied to the motion of Neptune as simulations have suggested it ought to be, and its small moon Iapetus has a strangely tilted orbit. Titan itself has remarkably few craters and an oval, or eccentric, orbit.

A huge collision that created the Titan we see today could explain all of these elements. "This is sort of a grand unified theory that covers all of the major problems," says Matija Ćuk at the SETI Institute in California.

In 2022, a hypothesised extra moon called Chrysalis in the outer reaches of the system was proposed to explain how Saturn's wobble got decoupled from Neptune. The idea was that Chrysalis got tossed towards Saturn and broke up to form the rings, destabilising Saturn's wobble and Iapetus's orbit in the process. However, Ćuk and

Saturn's biggest moon, Titan, emerges from behind the planet

his colleagues noticed that, in simulations, the most likely outcome was that Chrysalis would collide with Titan.

That is a problem, says Ćuk. "If there was a collision with Titan, it could not have become the rings." So he and his team calculated what would happen if Chrysalis did smash into Titan. They found that such a collision about 400 million years ago would erase Titan's craters and push its then-circular orbit to become eccentric, as well as creating a shower of debris (arXiv, doi.org/hbn8gb).

Over time, Titan's changing orbit would have destabilised the small inner moons and sent them careening into one another, grinding each other down into the tiny particles that now make up Saturn's rings.

"If a collision with Titan 1.0 can explain many other things about the Saturn system, then I think that would really centre Titan as being pivotal to how we see the system today," says Sarah Hörst at Johns Hopkins University in Maryland. ■

Health

Rapamycin can add years to your life, or none at all

James Woodford

THE longevity benefits of fasting or taking rapamycin are more like a lottery than a sure bet. The interventions were linked to a robustly extended lifespan less than a year ago, but a reanalysis of the data suggests that the benefits vary hugely between individuals.

"[They] might increase lifespan by a little bit or [they] might increase it by a lot," says Tahlia Fulton at the University of Sydney in Australia.

The 2025 study analysed 167 research papers across eight non-human species, including fish, mice, rats and rhesus monkeys. Fulton and her colleagues found that these animals lived longer, on average, if they were given rapamycin – a potential anti-ageing drug – or were subject to a calorie-restriction regime, which has been linked to longevity. The results led the team to conclude that the same probably applied to people.

Now, the researchers have looked at the spread of the responses to the longevity interventions among the individual animals and have found that the benefits were variable (*Biology Letters*, DOI: 10.1098/rsbl.2025.0651). This means that at an individual level, either taking rapamycin or doing dietary restriction with

the aim of living longer is "likely beneficial, but you don't know how beneficial", says Fulton.

"Some individuals will be much longer lived, some will be a little longer lived and some might not live any longer than they would have anyway," she says. "You've got a bit of a lottery happening."

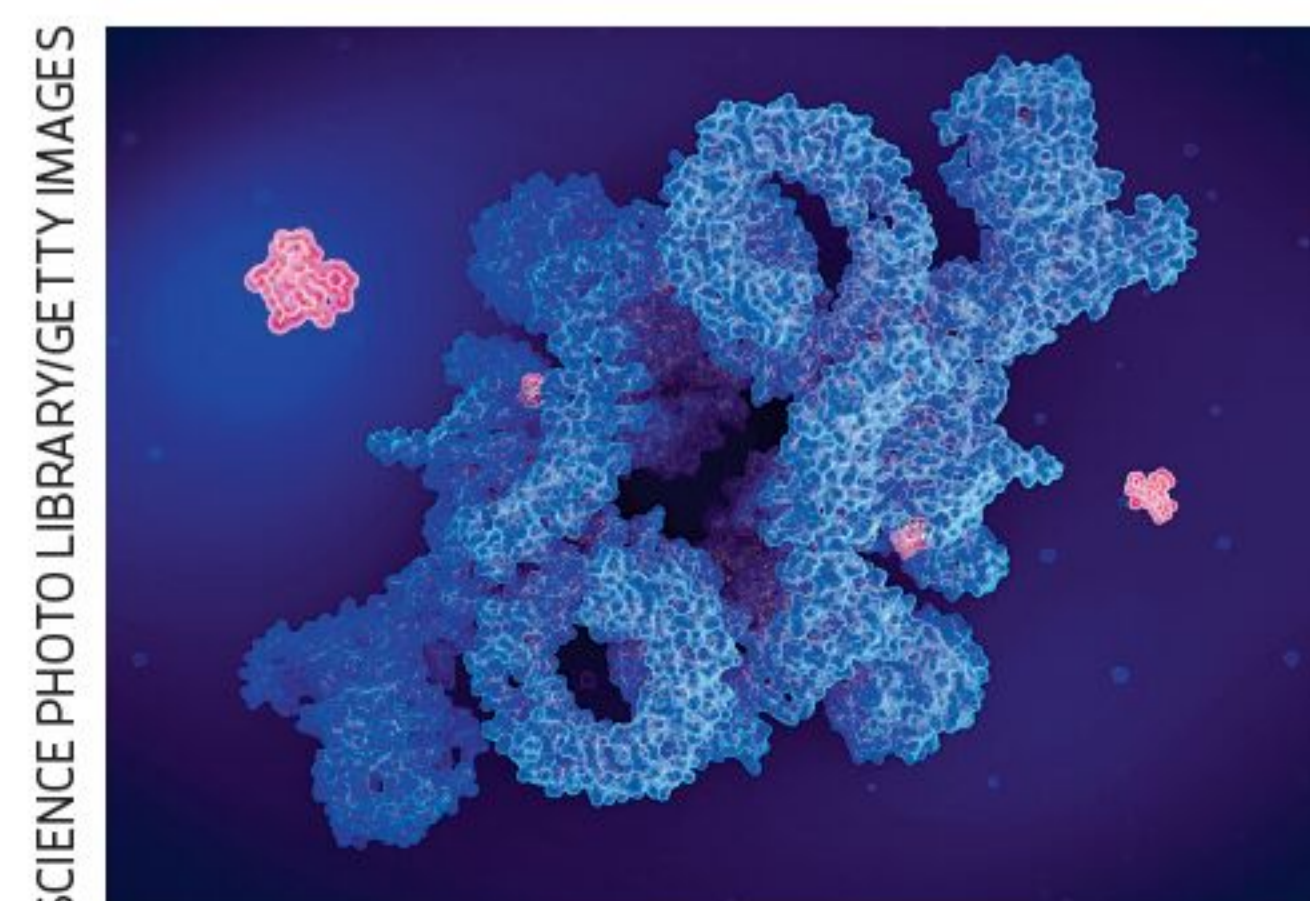
Fulton says the goal of a longevity intervention is to square the curve of a graph showing population size versus lifespan. This means that more people would live longer, rather than just a few, as seen with a sloping curve. "Squaring the survival curve means that everybody lives a really long, happy life, let's say, until 100 years old, and then you pretty reliably die at 100 years old," she says.

The latest research shows that neither dietary restriction nor rapamycin squares the curve. Off the back of this, Fulton says expectations need to be tempered until more research is undertaken to learn who benefits from these approaches most.

Matt Kaeberlein at the University of Washington in Seattle points out that squaring the curve doesn't necessarily improve people's years of healthy life. He says a more interesting question is whether "healthspan inequality" increases or decreases with longevity interventions, such as exercise.

Originally developed as an immunosuppressant for people undergoing organ transplants, rapamycin blocks the action of the mTOR protein, which is key in cell growth and division. At low doses, it has been shown to increase lifespan in some animals, possibly by protecting against DNA damage. ■

Rapamycin works by blocking the action of the mTOR protein



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