

be an shRNA vector that was depleted after shRNA induction in the mutant cells, but not their unmutated counterparts. Libraries of shRNAs should continue to improve, increasing genomic coverage and the degree of target inhibition, and so becoming ever more powerful tools in this endeavour. Sociologists have noted that some technological advances enable scientists to become 'communal harvesters' rather than 'hunter-gatherers'. With luck, the approach described by Staudt and colleagues will yield a bountiful harvest of cancer-drug targets that exploit weaknesses created by cancer-associated mutations. ■

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PLANETARY SCIENCE

A new spin on Saturn

David J. Stevenson

Measuring the rotation of a gaseous planet is no easy task. For Saturn, do observations of its magnetic field — which indicate that it is spinning more slowly than thought — mark a revolution in our understanding?

According to the International Astronomical Union (IAU), Saturn rotates once every 10 hours, 39 minutes and 22.4 seconds. Astronomical observations as far back as William Herschel's in the late eighteenth century¹ had suggested values for Saturn's rotational period of ten or so hours, but the IAU's seemingly precise value was defined by a periodicity in the kilometre-wavelength radio signal sent out by Saturn and detected by NASA's Voyager spacecraft in 1980. Surprising though it may seem, these radio emissions are still not well understood and — unlike radio emissions from pulsars or from Jupiter — turn out to be imprecisely periodic.

In this issue, Giampieri and colleagues (page 62)² report the observation, in data from the Cassini mission currently investigating the Saturn system, of a signal in Saturn's magnetic field of period about 10 hours 47 minutes. The authors suggest that this might be the planet's true rotation period.

So why should we care? One obvious reason is that rotation period is a fundamental property of a planet and tells us something about the conditions — such as the total angular momentum — when it formed. But that does not require a highly precise value. A less evident reason is that, with an equatorial radius some 10% greater than its polar radius, Saturn is the most distorted planet in the Solar System. Both the rotation and internal structure of a celestial object contribute to such an 'equatorial bulge', and the discrepancy of about 8 minutes (more than 1%) between the accepted and the new value for Saturn's rotational period will thus also affect our estimates of the size of its

likely inner core of rock and ice. Although the 'gas giant' Saturn is made mostly of hydrogen and helium, understanding the origin and evolution of such planets depends crucially on the nature and internal distribution of their minor constituents³. Saturn's rotation rate also provides the essential reference frame within which to talk about the dynamics of its atmosphere, and in particular the velocity of its very strong east–west winds. And this last point highlights a central issue: what does the rotation rate of a fluid planet actually mean?

The rotation rate of a planet such as Earth is conventionally defined to be that of its solid mantle, and can be measured to exquisite precision using geodetic methods such as the Global Positioning System. Tectonic movements of Earth's solid parts are, on average, slower than its rotation speed by 12 orders of magnitude, but are readily measurable. Larger effects arise from the interplay of tides and Earth's rotation, which causes an inexorable extraction of spin angular momentum, and the Moon to recede from Earth. Even larger, but fluctuating, effects arise from the exchange of angular momentum between Earth's solid regions and its fluid parts — the atmosphere, oceans and core. But the largest of these is still only a millionth of a hypothetical 1% saturnian change over the past 20 years.

Such a large variation in Saturn's rotation might be explained by its atmospheric features, in particular its strong easterly winds of up to 400 m s⁻¹ relative to the IAU period. But a 1% difference in rotation rate of an atmospheric feature corresponds to a 'wind' of around 100 m s⁻¹, and, although Saturn's

winds may have changed, it is unlikely to have been by that much.

A more reliable measure of a fluid planet's spin than cloud patterns on its surface comes from its magnetic field. If this field is large and emanates principally from a dipole whose axis is tilted with respect to the axis of rotation, there will be a distinctive, readily detectable periodicity in the field and any resulting radio emissions. Because this magnetic field is generated deep down, its lines are embedded deeply in a large fraction of the planetary mass where large variable rotational motions should not occur. An east–west motion of just 1 m s⁻¹ would, for instance, generate a toroidal magnetic field orders of magnitude larger than the observed dipole field, causing a Lorentz force to act and enforce uniform rotation again.

This argument agrees with our understanding of Earth. In Earth's liquid core, movements at depth are undetectable, but the movements at the top, in part revealed by magnetic-field changes over decades, indicate only tiny differential motions. Even the seismologically detected superrotation of Earth's inner core⁴ corresponds to only about one part in 10¹⁰ of Earth's total rotation.

Giampieri and colleagues² have found a stable periodicity in Saturn's magnetic field. Their observations are, however, incompatible with the expected inverse cubic dependence of field strength on distance from the planet that is expected from a tilted dipole. Saturn's magnetic field (Fig. 1) certainly lacks the substantial dipole tilt that we see so strikingly in Earth, Jupiter and, in a more complicated way, in Uranus and Neptune; but it would be astonishing if Saturn's field was exactly symmetric about the rotation axis. There is no reasonable model for field structure that can explain the observed behaviour solely through the internal field.

The observed periodicity might instead result from something that is a proxy for the internal rotation, for example a disturbance generated in Saturn's magnetosphere by a field anomaly (not necessarily dipolar) that is rotating with the core of the planet. The stability of the period would certainly be easiest to explain with the rotation of such a massive object. But although Saturn's deep interior would undoubtedly be a good clock, some region of smaller inertia would not necessarily be a bad one: the observed periodicity might be an indication of some wave-like or advected feature of the planet's ionosphere, and so be quite possibly unrelated to a rotation of the whole planet. In this case, it is curious that all the observed atmospheric motions rotate faster than the proposed rotation.

But the central puzzle would seem to be why Saturn behaves differently from Jupiter. Jupiter has a tilted dipole, a magnetic field that is much larger than Saturn's (even after taking account of the different-sized metallic region at its core), and winds that move both with and against the sense of the planet's rotation.

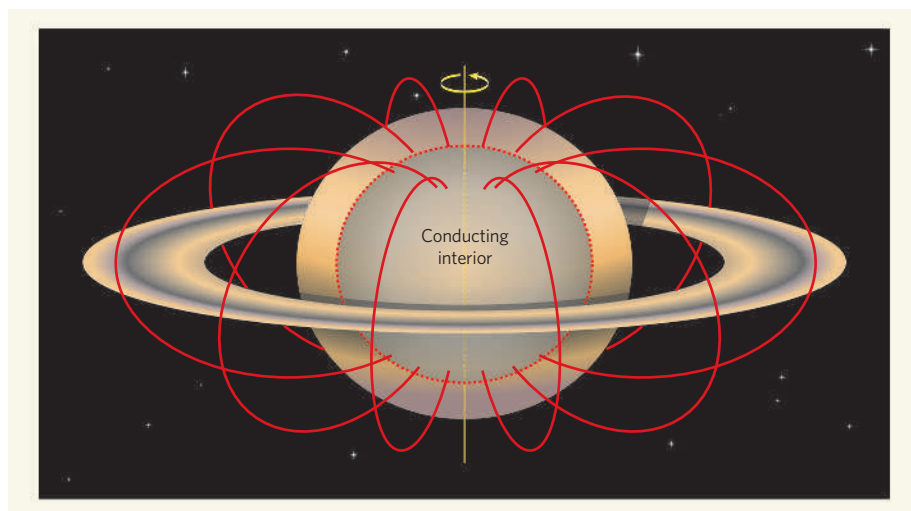


Figure 1 | Straight-up rotator. Saturn's magnetic field is almost exactly aligned with its rotation axis, and the dipole field lines emanate from a massive electrically conducting metallic hydrogen core (both axes are tilted by about 27° with respect to the plane of its orbit). Giampieri and colleagues' observation² of a periodic field variation, although not fully understood, could indicate that Saturn is rotating more slowly than had been assumed.

Owing to the much greater tilt of its axis of rotation (27° against 3°), Saturn has appreciable seasons, whereas Jupiter does not; perhaps we need a longer time base to appreciate fully the differences arising from this. (The orbital periods of the two planets are 29.5 years and 12 years, respectively.) However, this can only explain superficial differences. Maybe the Jupiter–Saturn differences are just another example of planetary diversity: the remarkable richness of planets in the Solar System alone

has arguably been the most striking outcome of the planetary exploration programme. ■ David J. Stevenson is in the Division of Geological and Planetary Sciences, California Institute of Technology, Pasadena, California 91125, USA. e-mail: djs@gps.caltech.edu

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MOLECULAR BIOLOGY

Chromosome guardians on duty

Paul Megee

Curiously, in cell division the proper separation of chromosomes into daughter cells needs set periods when they are stuck together. So how do they come apart at the right time and place? Their 'guardian spirits' intercede.

To avoid cell death or genetic diseases such as cancer, chromosomes must be transmitted to progeny cells with high fidelity. During cell division, chromosomes are duplicated to form sister chromatids, which must then be divided into two equal groups and separated into daughter cells (a process termed segregation). To prevent random segregation, sister chromatids are held together along their lengths by a ring-shaped protein complex called cohesin¹. At the cell-cycle stage known as anaphase, cohesins are cleaved and cohesion between sister chromatids dissolves, triggering their segregation. So the chromosomal association and dissociation of cohesin must be tightly regulated for proper segregation. In this issue, Kitajima *et al.* (page 46)² and Riedel *et al.* (page 53)³ describe how proteins known as

shugoshins — Japanese for 'guardian spirits' — and an associated regulatory enzyme temporally and spatially control the removal of cohesins from chromosomes.

The association of cohesins with the chromosome is particularly robust near the centromere⁴. This specialized region of the chromosome mediates assembly of the kinetochore complex that attaches the chromosome to the 'spindle', the cellular structure that pulls sister chromatids apart once cohesion has dissolved. Strong cohesion flanking the kinetochores is crucial, because it promotes the attachment of the two kinetochores on each chromatid pair to spindle fibres that originate from opposite sides of the dividing cell. It also opposes the splitting forces exerted by spindle fibres, preventing

premature separation of sister chromatids.

There are two types of cell division — meiosis, a specialized process that generates reproductive cells, and mitosis, which produces all other cells. Each has a pathway to deal with cohesin removal. In vertebrate mitosis, cohesins are detached from the chromosome arms first, in prophase (early mitosis), but the cohesins around the centromere are retained until the onset of anaphase (late mitosis) (Fig. 1, overleaf). At anaphase, an enzyme called separase cleaves the cohesin subunit known as Mcd1/Sccl, causing cohesins to dissociate from the centromeres⁵. How cohesins are removed from the chromosomal arms during prophase is less clear, other than it does not require separase and it is triggered by the phosphorylation (addition of a phosphate group) of a cohesin subunit called Scc3/SA (ref. 6).

In meiosis, DNA replication is followed by two successive rounds of chromosome segregation and cell division, producing four egg or sperm cells with half the chromosomal content of the other cells in the body (so that the fusion of two cells at fertilization gives the correct genetic complement). Maternally and paternally derived copies of chromosomes — referred to as homologues — are segregated in the first round, and sister chromatids are segregated in the second round. Before the first meiotic division, homologues usually undergo 'recombination', the reciprocal exchange of chromosomal segments. Recombination produces crossovers, which are physical links between homologues that can only be untangled by the release of sister-chromatid cohesion (Fig. 1). However, complete removal of cohesins at this stage would lead to random segregation of sister chromatids during the second division, so cohesin association persists in centromeric regions until the second division. At the first division, cohesins are removed in prophase by a pathway that is probably similar to that operating in mitosis⁷, and by the separase-mediated cleavage of Rec8, a meiosis-specific variant of Mcd1. Cohesin removal by these pathways seems to be enhanced by phosphorylation of cohesin subunits^{7,8}. As in mitosis, the removal of centromeric cohesins is dependent on separase.

How cohesin binding is stabilized specifically in centromeric regions is unclear. But hints came from the fruitfly protein MEI-S332, which, when inactivated, leads to defective centromeric cohesion⁹. MEI-S332 is the founding member of a family of proteins called shugoshins, whose localization to the kinetochore is essential for the persistence of centromeric cohesin^{10–12}. Shugoshin family members exist in organisms from yeast to humans, implying that there is an evolutionarily conserved mechanism to protect centromeric cohesin.

To determine how shugoshins protect centromeric cohesin, Kitajima *et al.*², Riedel *et al.*³ and Tang *et al.*¹³ set out to identify proteins that interact with shugoshin in mitotic and