induced arrest, and in recombination[6–9]. For instance, mutations in the gene encoding Srs2 lead to excessive recombination[7]. Krejci *et al.*[1] and Veaute *et al.*[2] now provide biochemical evidence that Srs2 actively inhibits a key step in one particular recombination process — homologous recombination, or genetic exchange between two matching DNA regions.

During homologous recombination, single-stranded DNA must be produced, and the Rad51 protein binds this DNA to form so-called Rad51 nucleofilaments. Rad51 then mediates the exchange of this strand with a complementary tract of DNA. Krejci *et al.* and Veaute *et al.* show that Srs2, as well as acting as a helicase, also has a 'translocase' activity: it dislodges Rad51 from these filaments, thereby preventing recombination.

These findings explain why alterations in Srs2 are associated with hyper-recombination in yeast[7] (which is reminiscent of the excessive recombination seen in cancer cells). The results might also provide an explanation for other previous findings — and they raise new questions.

For instance, single-stranded DNA might signal the presence of DNA damage[4,9], leading to the recruitment of specialized proteins that activate the checkpoint response. The checkpoint then delays the cell cycle, allowing time for the damage to be repaired by various processes, some of which are mediated by Rad51. Srs2 is known to be involved here: it is phosphorylated in response to DNA damage[8] and, in its absence, cells manifest obvious checkpoint alterations[8,9], such as a hyperactive checkpoint that stops the cell cycle from restarting even when the damage has been repaired[9].

Perhaps the newly discovered inability of Srs2 mutants to dislodge Rad51 from nucleofilaments can explain this aberrant checkpoint: it might be necessary to remove Rad51 after recombination-mediated DNA repair so that the proteins that activate the damage-induced checkpoint can also be removed[9]. Veaute *et al.* also suggest that the Rad51 nucleofilaments themselves could be a checkpoint-activating signal, and hence that the removal of Rad51 by Srs2 is necessary to tell the cell that division can begin again. This is plausible, although cells depleted of Rad51 can still promote checkpoint activation. But regardless of whether they signal to the checkpoint, Rad51 nucleofilaments can clearly form during chromosome repair, making it essential that they be dismantled by Srs2 during recovery.

Srs2 also bears a relationship with Sgs1, the yeast counterpart of the human helicases that are defective in Werner, Bloom and Rothmund–Thomson syndromes[10]. When both Sgs1 and Srs2 are mutated, Rad51-mediated recombination causes cell death[10]. Although the functional interaction between these two helicases remains unknown, it is possible that they have a similar role in processing Rad51 filaments. If these proteins do have the same biochemical function, it is not surprising that they can sometimes substitute for each other[11]. But they do not seem to be redundant, hinting that they might act at different cell-cycle stages or different steps in replication. Perhaps Sgs1 is involved during the replication of damaged chromosomes by antagonizing the formation of Rad51 intermediates and hence promoting specialized, replication-specific repair processes. Srs2, by contrast, could preferentially contribute to the processing of Rad51 filaments after the passage of the replication fork, and later on in the cell cycle.

Another question is whether Srs2's translocase activity is implicated in other cellular pathways involving protein–DNA complexes. Support for this idea comes from Veaute and colleagues' finding[2] that Srs2 can also remove RecA, an *Escherichia coli* relative of Rad51, from DNA.

In addition, both genetic and physical approaches have shown that yeast cells with mutant Srs2 are defective in certain types of recombination event[9,12,13]. The implication is that Srs2, as well as preventing an early step in homologous recombination, might actively promote specific recombination subpathways. This apparent paradox could be resolved if this alternative role of Srs2 is more closely related to its helicase activity, or is controlled by its phosphorylation state, or is influenced by the formation of complexes between Srs2 and other proteins or by the type of DNA damage.

Finally, it remains to be seen how Srs2 itself is regulated. Recombination, of course, is often essential, and so must not always be prevented. ■

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1. Krejci, L. *et al. Nature* **423,** 305–309 (2003).
2. Veaute, X. *et al. Nature* **423,** 309–312 (2003).
3. Paques, F. & Haber, J. E. *Microbiol. Mol. Biol. Rev.* **63,** 349–404 (1999).
4. Sogo, J. M., Lopes, M. & Foiani, M. *Science* **297,** 599–602 (2002).
5. Hickson, I. D. *Nature Rev. Cancer* **3,** 169–178 (2003).
6. Rong, L., Palladino, F., Aguilera, A. & Klein, H. L. *Genetics* **127,** 75–85 (1991).
7. Chanet, R., Heude, M., Adouda, A., Maloisel, L. & Fabre, F. *Mol. Cell. Biol.* **16,** 4782–4789 (1996).
8. Liberi, G. *et al. EMBO J.* **19,** 5027–5038 (2000).
9. Vaze, M. *et al. Mol. Cell* **10,** 373–385 (2002).
10. Gangloff, S., Soustelle, C. & Fabre, F. *Nature Genet.* **25,** 192–194 (2000).
11. Mankoury, H., Craig, T. J. & Morgan, A. *Nucleic Acids Res.* **30,** 1103–1113 (2002).
12. Paques, F. & Haber, J. E. *Mol. Cell. Biol.* **17,** 6765–6771 (1997).
13. Hegde, V. & Klein, H. *Nucleic Acids Res.* **28,** 2779–2783 (2000).

Planetary science

Jupiter's moonopoly

Douglas P. Hamilton

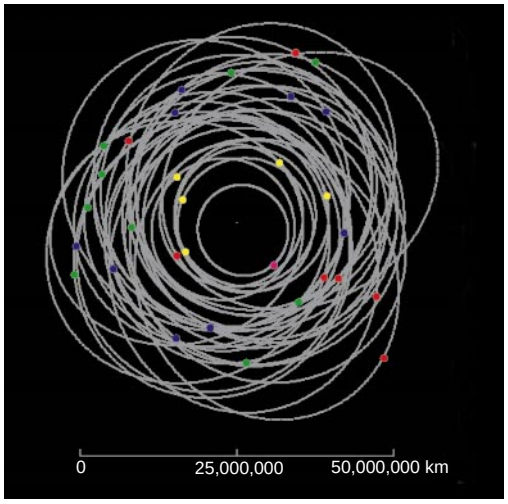
A further 23 satellites have been discovered in orbit around Jupiter. With diameters of between two and eight kilometres, the moons are the smallest yet spotted around any planet.

Swinging serenely around the Sun, mighty Jupiter has reason to be pleased: its pre-eminence as the planet with the largest number of natural satellites, or moons, has been dramatically and decisively re-established. Fending off strong challenges from rival Saturn and wild card Uranus, the Solar System's largest planet now has nearly as many known moons as all of its competitors combined. Satellite-seekers Scott Sheppard and David Jewitt are responsible for returning Jupiter to its dominant status — on page 261 of this issue[1], they report the discovery of nearly two dozen new jovian moons.

The search for planetary satellites has a long history, dating back to 1610 and Galileo Galilei's discovery of four star-like objects orbiting Jupiter — Io, Europa, Ganymede and Callisto. Saturn's splendid ring system

Planet	Number of satellites	Number of irregular satellites	Largest irregular satellite and radius (km)	
Earth	1	—		
Mars	2	—		
Jupiter	60	52	Himalia	85
Saturn	31	14	Phoebe	110
Uranus	22	6	Sycorax	80
Neptune	11	5	Triton	1,353
Pluto	1	—		

Figure 1 **Planets and satellites. Irregular, or distant, satellites are found only around the giant planets and are thought to have been captured during the final stages of planetary formation. Total numbers are continually updated at ref. 4 and include this year's findings, up to April 2003: 20 moons at Jupiter, 1 at Saturn and 3 at Neptune. The smallest objects spotted at Jupiter are barely 2 km across[1].**



M. ASBURY

Figure 2 **The orbits of Jupiter's outer satellites. The giant planet is at the centre of the image (taken from an animation program[13]), with 31 of its distant satellites grouped into the families identified by Sheppard and Jewitt[1]. Prograde objects (pink and yellow) circle Jupiter in the anticlockwise direction when viewed from above Jupiter's north pole, while the three retrograde families (blue, green and red) circle clockwise. The few known prograde orbits nestle inside the far more numerous retrograde ones.**

and its largest moon, haze-enshrouded Titan, were first seen by Christiaan Huygens about 50 years later, and, in 1684, the discoveries of icy Dione and Tethys established Saturn as the planet with the most moons, a title it held for 230 years. Jupiter's Sinope, spotted in 1914, evened the score at nine known moons apiece, and two additional findings in 1938 allowed the giant planet to surge into the lead. Saturn staged a surprise comeback in 1980, when seven new satellites were spotted by the Voyager spacecraft and ground-based observers. Then came the great upset of 1999: dark horse Uranus revealed three additional outer satellites[2] and vaulted to the forefront. But the title has since been reclaimed — first by Saturn, with a dozen new discoveries reported[3] in 2000, and now by Jupiter, with the 23 findings detailed in this issue[1].

Currently, the number of known planetary moons stands[4] at 128 (Fig. 1). More than half of this total has been added since 1997, when Brett Gladman and colleagues found the first two distant, or 'irregular', satellites of Uranus[5]. The large number of satellite discoveries over the past six years, at an ever quickening pace, is reminiscent of the situation following Jewitt and Luu's 1992 discovery[6] of the first trans-neptunian (or Kuiper belt) objects. Both population explosions have been fuelled by major improvements in digital-camera technology[7].

Nearly two-thirds of the known moons (including all of the recent discoveries) are irregular satellites, orbiting far from their planets along highly tilted, elliptical paths. These objects are believed to have been captured by their planets from independent orbits around the Sun early in the history of the Solar System. Regular satellites, by contrast, have much smaller, untilted, circular orbits, and were probably formed out of the disks of gas and dust that surrounded the giant planets in their youth. Energy dissipation in these early accretion disks also acted to facilitate the capture of the irregular satellites[8].

The orbital distributions of irregular satellites at Jupiter (Fig. 2) and Saturn, particularly their tilt angles, show several intriguing patterns that hint at past dynamical and collisional processes. No moons have yet been found on orbits tipped by more than about 55° to the planet's orbital plane. This is due to gravitational forcing from so-called solar tides, which is the largest perturber of distant planetary satellites[9–11]. Objects with greater tilts experience large, correlated changes in their inclinations and eccentricities which put them on orbits that penetrate deep into the inner jovian system. Such objects are lost as a result of destabilizing gravitational kicks from large regular satellites and, in some cases, through direct collisions[11].

The lack of highly tilted orbits means that the orbits may be cleanly divided into prograde (those that circulate in the same direction as the planet's motion around the Sun), and retrograde (those that move in the opposite direction). At Saturn and Neptune, the number of distant, prograde moons is comparable to the number of retrograde objects, but at Jupiter and Uranus, the retrogrades dominate. Also in this issue (page 264), Astakhov and colleagues[12] proffer an explanation for the striking Jupiter–Saturn dichotomy. Through numerical integrations of the capture process, they find that interactions with Jupiter's large regular satellite Callisto preferentially remove moons with prograde orbits, which penetrate closer to Jupiter than retrogrades (see, for example, ref. 10). Titan, closer to Saturn than Callisto is to Jupiter, is less efficient at removing the prograde population. Although alternative explanations for the dichotomy cannot be ruled out, the orbital properties of the extant satellites certainly put important constraints on conditions that existed during the capture process[12].

The prograde and retrograde groups can be further divided into families of objects that share similar characteristics, such as the size, shape and tilt of their orbits. At least five such families are evident[1,11] at Jupiter and four are seen[3] at Saturn. Like asteroid families, each satellite family appears to have been formed from the collisional breakup of a larger object[1,3,11].

So satellites are occasionally shattered by breakups, but do moons ever merge? Although retrograde satellites at Jupiter now vastly outnumber the prograde irregulars, the latter (primarily Himalia) account for nearly 98% of the mass of the distant jovian satellites. And at Saturn, despite similar numbers of prograde and retrograde irregulars, retrograde Phoebe dominates with approximately 99.5% of the orbiting mass. Perhaps Himalia and Phoebe are merely the largest surviving members of much bigger primordial populations. Alternatively, these two large satellites may have grown significantly by cannibalizing some of their smaller neighbours, or by accreting inwardly migrating solids in the latter stages of giant-planet growth. This hypothesis is supported by the fact that both Phoebe and Himalia lie near the inner edge of the zone of irregular satellites (where collisional accumulation rates are fastest) and have relatively low orbital eccentricities and tilts. These and other satellite evolutionary processes need to be explored more fully.

Although ever smaller moons around the giant planets will undoubtedly be discovered, it is likely that Jupiter, whose satellites appear brightest because of their proximity to the Earth and the Sun, will continue to dominate the moon count for the foreseeable future. ■

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1. Sheppard, S. S. & Jewitt, D. C. *Nature* **423,** 261–263 (2003).
2. Gladman, B. J. *et al. Icarus* **147,** 320–324 (2000).
3. Gladman, B. J. *et al. Nature* **412,** 163–166 (2001).
4. http://ssd.jpl.nasa.gov/sat_discovery.html
5. Gladman, B. J. *et al. Nature* **392,** 897–899 (1998).
6. Jewitt, D. C. & Luu, J. *Nature* **392,** 730–732 (1993).
7. Hamilton, D. P. *Nature* **412,** 132–133 (2001).
8. Pollack, J. B., Burns, J. A. & Tauber, M. E. *Icarus* **37,** 587–611 (1979).
9. Kozai, Y. *Astron. J.* **102,** 1213–1225 (1962).
10. Hamilton, D. P. & Burns, J. A. *Icarus* **92,** 118–131 (1991).
11. Carruba, V. *et al. Icarus* **158,** 434–449 (2002).
12. Astakhov, S. A., Burbanks, A. D., Wiggins, S. & Farrelly, D. *Nature* **423,** 264–267 (2003).
13. http://janus.astro.umd.edu/javadir/orbits/moons.html

Corrections

● In the News and Views in Brief item "Cell biology: Muscle mice" (*Nature* **422,** 393; 2003), the adult stem cells referred to were derived not from human bone marrow but from synovial membrane.

● The correctly spelt name of the author whose work was discussed by Ian Stewart in "Mathematics: Regime change in meteorology" (*Nature* **422,** 571–573; 2003) is Daan Crommelin.