NEWS & VIEWS

PLANETARY SCIENCE

The Solar System's extended shelf life

Gregory Laughlin

Simulations show that orbital chaos can lead to collisions between Earth and the inner planets. But Einstein's tweaks to Newton's theory of gravity render these ruinous outcomes unlikely in the next few billion years.

In the midst of a seemingly endless torrent of baleful economic and environmental news, a dispatch from the field of celestial dynamics manages to sound a note of definite cheer. On page 817 of this issue, Laskar and Gastineau[1] report the outcome of a huge array of computer simulations. Their work shows that the orbits of the terrestrial planets — Mercury, Venus, Earth and Mars — have a roughly 99% chance of maintaining their current, well-ordered clockwork for the roughly 5 billion years that remain before the Sun evolves into a red giant and engulfs the inner Solar System.

The constant interplay of gravitational attractions between planets acts to degrade their repetitive and predictable motions. Over time, a system of orbits can become increasingly disordered, and, like a poorly balanced tyre that tears itself off the axle of a moving car, planets might fling each other out into space or into their parent star, or collide with each other. The census of extrasolar planets has revealed instances (such as the outer two planets orbiting the nearby star Upsilon Andromedae) where we seem to be observing a system in which one of the original planets has been ejected, leaving evidence of the catastrophe in the form of an ongoing back-and-forth exchange of angular momentum between the survivors[2].

To all appearances, our Solar System seems a model of stability. Phenomena such as eclipses can be pinpointed over the millennia, and the motions of the planets themselves can be charted with confidence tens of millions of years into the future. An ironclad evaluation of the Solar System's stability, however, eluded mathematicians and astronomers for nearly three centuries.

In the seventeenth century, Isaac Newton was bothered by his inability to fully account for the observed motions of Jupiter and Saturn. The nonlinearity of the gravitational few-body problem led him to conclude[3] that, "to consider simultaneously all these causes of motion and to define these motions by exact laws admitting of easy calculation exceeds, if I am not mistaken, the force of any human mind".

During the 1700s, Continental mathematicians, including Leonhard Euler, Joseph-Louis Lagrange and Pierre-Simon Laplace, developed elegant perturbative methods to describe long-term orbital evolution. Their approach met notable success with Laplace's demonstration that the so-called *grande inégalité* in the motions of Jupiter and Saturn could be attributed to the fact that the orbital period of Jupiter is close to two-fifths that of Saturn, resulting in a near 5:2 orbital resonance between them. Laplace believed that the planetary orbits would be stable and predictable for all time, an attitude that probably contributed to his formulation of a rational determinism[4] — the belief that initial conditions and physical laws precisely determine the future.

By the 1850s, however, it was recognized that the higher-order terms in the planetary 'disturbing function' could not be neglected, and consideration of these terms revived the question of orbital stability. In 1889, Henri Poincaré demonstrated that even the gravitational three-body problem cannot be solved by analytic integration, thereby eliminating any possibility that an analytic solution for the entire future motion of the eight planets could be found. Poincaré's work anticipated the now-familiar concept of dynamical chaos and the sensitive dependence of nonlinear systems on initial conditions[5].

In recent decades, computers have reinvigorated celestial mechanics. Orbital predictions obtained from numerical integration of the planets' equations of motion demonstrated that the planetary orbits will indeed become chaotic, with typical Lyapunov times — the time required for chaos to significantly degrade the predictability of a system — of the order of 5 million years. Statements regarding the stability of the Solar System must therefore be expressed in terms of probabilities. Computers are now fast enough to be able to produce forward models of the Solar System throughout the Sun's remaining 5-billion-year hydrogen-burning lifetime. One insight that has emerged is that, from a dynamical point of view, the Solar System is effectively two systems of planets. The gas giants — Jupiter, Saturn, Uranus and Neptune — constitute an extremely stable constellation, whereas the rocky terrestrial planets are on a far less solid footing. Were one

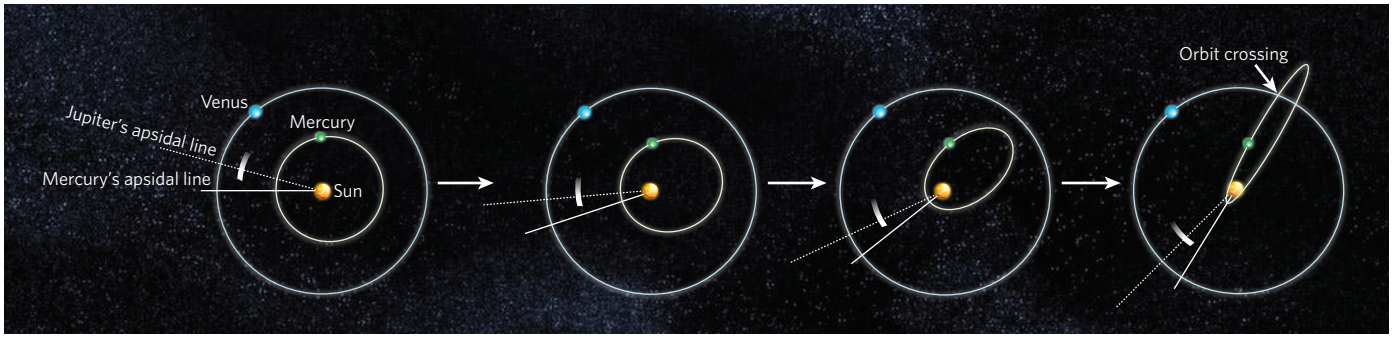

Figure 1 | Trouble with Mercury. Numerical simulations by Laskar and Gastineau[1] indicate that there is a roughly 1% chance that the inner Solar System will become destabilized during the next 5 billion years. The Solar System's Achilles heel is a secular resonance between Jupiter and Mercury, in which the orbits of the two planets, and hence their apsidal lines — the lines that connect the Sun to the point of closest approach of the planetary orbit — precess at the same rate. If the resonance is established, the eccentricity of Mercury's orbit increases steadily over a million-year timescale, and eventually crosses that of Venus. Once orbit crossing occurs, a variety of disastrous outcomes are possible, several of which are detailed by the authors[1].

to eliminate the Sun's eventual mass loss and its damaging encounters with passing stars in the far future, the outer planets would evolve with substantially unaltered orbits for about 10^{18} years before succumbing to a weak orbital resonance, in which the perturbative attractions between Jupiter, Saturn and Uranus would generate large-scale instability[6].

Laskar and Gastineau's work[1] is the culmination of a long-running effort to establish a probabilistic assessment of the orbital stability of the terrestrial planets. They report highly detailed numerical simulations of the evolution of the whole Solar System using the most accurate available planetary ephemeredes (a table of the precise positions and velocities of the planets at a specific time). The simulations indicate that Mercury, in spite of its diminutive size, poses the greatest risk to the present order. In a small but disturbing subset of possible future trajectories, Mercury becomes trapped in a 'secular resonance' with Jupiter, a state of affairs in which the elliptical figure of Mercury's orbit rotates in synchrony with Jupiter's orbital precession (Fig. 1). If the Jupiter–Mercury resonance is established, Mercury's orbital eccentricity will increase to the point at which it intersects the orbit of Venus, setting the stage for catastrophe.

But the odds of Mercury entering a secular resonance are greatly reduced by the small modifications that Einstein's theory of general relativity imparts to the planetary motions. Famously, 43 arcseconds per century of Mercury's total precession is due to the effect of general relativity. This correction effectively detunes the Mercury–Jupiter interaction, and decreases the chance that resonance will occur in the next 5 billion years to roughly 1%. This is fortunate indeed, as the Laskar and Gastineau paper[1] also relates the precise and grisly details of one case in which Mercury's destabilization leads to a wholesale exchange of angular momentum between the inner and outer Solar System. (Readers of the paper can see for themselves the consequences of this, but suffice it to say here that Earth does not fare well in the resulting interplanetary melee.)

Laskar and Gastineau's work brings closure to one of the most illustrious and long-running problems in astronomy, and in a sense the result is as satisfying as one could wish. With 99% certainty, we can rely on the clockwork of the celestial rhythm — but with the remaining 1% we are afforded a vicarious thrill of danger. What now remains is to understand the extent to which the hand of dynamical chaos that so lightly touches our Solar System has moulded the Galactic planetary census. ■

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CELL CYCLE

Cell division brought down to size

Kenneth E. Sawin

Cells normally divide on reaching a fairly specific size, but how cell size dictates the timing of cell division remains obscure. In fission yeast, a spatial gradient of a cell-tip protein may provide an answer.

How does a cell decide that it is big enough to divide into two daughter cells? From *E. coli* to elephants, long-term sustainability depends on coordinating cell growth (an increase in total mass) with cell division. Divide too soon, and cells become progressively smaller with each generation; divide too late, and they become progressively larger. Coordination of cell size and cell division involves homeostatic mechanisms at multiple levels[1], but one question that has been particularly difficult to answer is how cell size per se affects the decision to enter mitosis — the stage in the cell cycle at which duplicated genomes separate and the cell divides in two. On pages 852 and 857 of this issue, two studies, one by Martin and Berthelot-Grosjean[2] and the other by Moseley *et al.*[3], independently provide evidence that in fission yeast (*Schizosaccharomyces pombe*) a spatial gradient of a protein emanating from cell tips may regulate mitotic entry, thereby directly linking cell size with cell division.

Pioneering work[4] on the cell cycle in the 1980s revealed that the key enzyme regulating entry into mitosis is cyclin-dependent kinase (CDK), a complex made up of the protein kinase Cdk1 (also known as Cdc2) and its regulatory subunit, cyclin. Protein kinases phosphorylate other proteins, thereby changing their properties. Active CDK, with the help of several other protein kinases, phosphorylates multiple substrates to drive the events of mitosis and cell division. During interphase — the stage of the cell cycle preceding mitosis — CDK activity is held in check by several mechanisms[5], including inhibitory phosphorylation of Cdk1 by another protein kinase, Wee1. Entry into mitosis accordingly depends on a protein phosphatase, Cdc25, that removes the inhibitory phosphorylation from Cdk1. Wee1 is itself inhibited by two related protein kinases, Cdr1 (also known as Nim1) and Cdr2 (refs 6, 7).

The unique properties of fission yeast make it an ideal model for cell-cycle research. Fission-yeast cells are cylindrical and maintain a constant width as their tips grow; as a result, cell length reflects both cell-cycle stage and cell size. Increasing the amount of the Cdk1 inhibitor Wee1 causes fission-yeast cells to divide at increased lengths, whereas increasing the amount of the Cdk1 activator Cdc25 leads to cell division at decreased lengths[4]. That these cell-cycle regulators can change the length 'set-point' for entry into mitosis suggests that cell size might be linked to mitotic entry by modulating the balance of Wee1 and Cdc25 activities in growing cells. But how might this modulation occur?

Martin and Berthelot-Grosjean[2] and Moseley *et al.*[3] harness the power of yeast genetic analysis to pinpoint the involvement of another protein kinase, Pom1 (ref. 8). Both groups[2,3] show that when the *pom1* gene is deleted, cells divide at a slightly reduced length. Increasing the amounts of Pom1 increases the length at which cells divide, implying that Pom1 inhibits entry into mitosis. Additional experiments indicate that Pom1 exerts its effects on division length by affecting the Cdr2–Cdr1–Wee1 pathway, possibly through Pom1 phosphorylating Cdr2, which Martin and Berthelot-Grosjean[2] show occurs *in vitro*. Inhibitory phosphorylation of Cdr2 would prevent inhibition of Wee1, allowing active Wee1 to inhibit Cdk2 and delay cell division.

The link between Pom1 and Cdr2 becomes even more intriguing on observing the cellular localization of these proteins. Cdr2 is found in unusual structures known as interphase nodes, which form a band in the cortex (the region just under the cell membrane) in the middle of the cell, overlying the cell nucleus[9]. Until now, these nodes had been thought to function primarily in linking the position of the nucleus to the cell-division plane during cytokinesis — the physical separation of daughter cells after mitosis[10]. However, the new work[2,3], particularly that by Moseley *et al.*[3], shows that Cdr1 and Wee1, as well as several other proteins, are also localized to these nodes. Moreover, when the *cdr2* gene is deleted, most or all of the proteins are no longer found in nodes. Thus, the medial cell cortex contains a Cdr2–Cdr1–Wee1 regulatory network, whose spatial positioning is determined by Cdr2.

During interphase, Pom1 is localized to cell tips, in the cell cortex[8]. Martin and Berthelot-