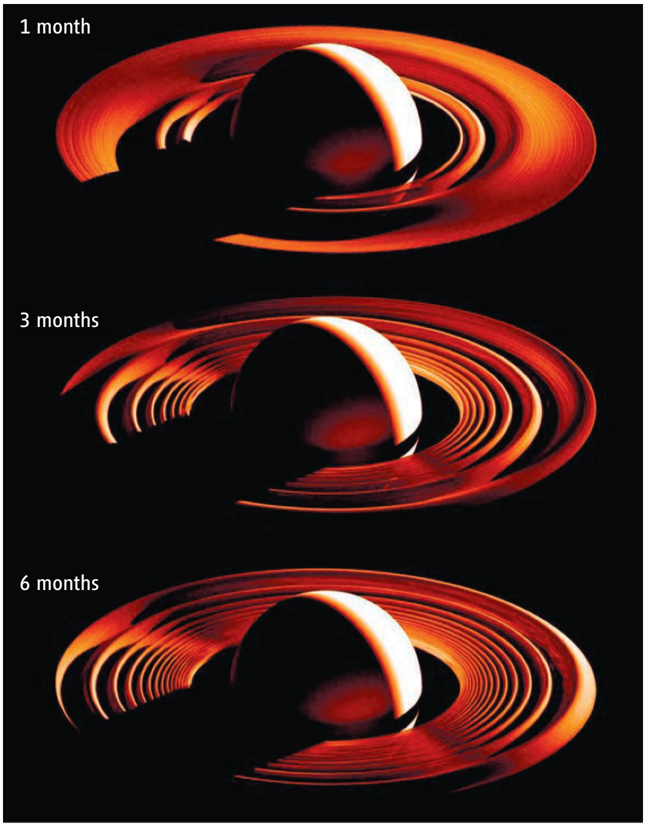
ASTRONOMY

Twisted Disks

Ripple patterns in Saturn's and Jupiter's rings result from collisions with comets.

Heikki Salo

Astrophysical disks come in many sizes but, as vertically thin rotation-supported systems, are all susceptible to both external perturbations and internal instabilities. One spectacular form of deformation is vertical bending. For decades, it has been clear that many galactic disks, including our own Galaxy (*1*), are warped. This manifests as up and down bends of the outer disks of edge-on galaxies and, in more face-on galaxies, by distortions in the neutral hydrogen velocity fields (*2*, *3*). Warps are also seen in circumstellar dust disks [e.g., β Pictoris (*4*)] and are hypothesized to be present in accretion disks around massive objects (*5*). Nevertheless, the most striking twisted disks reside in our solar system. This is vividly demonstrated by Hedman *et al*. (*6*) and Showalter *et al*. (*7*) on pages 708 and 711 of this issue, analyzing the vertical corrugation patterns in the rings of Saturn and Jupiter with imaging data from the Cassini and Galileo/New Horizon spacecrafts, respectively. The new data confirms the previously suspected (*8*) vertical undulations in Jupiter's main ring. In Saturn's rings, the coherent pattern extends over hundreds of wavelengths, covering not only parts of the D ring (*9*) but also the entire C ring with an inferred vertical amplitude on the order of a few meters.



Rippling through. Evolution of a corrugation pattern after the entire toy ring has been tilted along a constant line by 0.5°. A leading one-armed spiral pattern forms due to the winding of orbital planes. Realistic values for Saturn's gravity moments are used, and after ~30 years the pattern would appear as tight as observed in Saturn's rings. However, the vertical amplitude is exaggerated by a factor of ~100,000. The bending of the local ring plane leads to brightness variations: In the plot, the slopes exceed the 5° illumination elevation, and parts of the ring are in its own shadow (alternating darker zones lit by multiple reflections). In Saturn's rings, the slopes are a factor ~2000 smaller (and limited to the D and C rings), indicating very subtle brightness variations discernible only when the Sun is shining near the ring plane (*6*). In Jupiter's tenuous rings, there is no shadowing, and brightness variations arise due to the larger number of scatters when the line-of-sight is along the local slopes, made possible when the rings are viewed near edge-on (*7*).

There is a simple kinematic description for a warp pattern. Particles orbiting in a spherical potential maintain fixed orbital planes. However, if the potential is flattened with respect to a central plane (e.g., around an oblate planet), the vertical motion with respect to this plane has a shorter period than the azimuthal rotation. Imagine a swarm of particles on inclined orbits, each initially crossing the central plane along a common nodal line. On each orbit, the particles cross the central plane earlier and earlier, indicating that their orbital nodes regress in the direction opposite to rotation. This twisting of orbits is faster closer to the planet, leading to a warp, and if the twisting goes on, to a wavelike vertical corrugation pattern (see the figure). Such a pattern, with wavelengths decreasing inversely proportional to time, describes well the corrugations reported (*6*, *7*), although the small amplitude of the patterns requires the advantage of very special observational geometries.

The origin of galaxy warps, with their small amount of winding, is poorly understood (*10*). The warps are common and are thus either very long-lived or continuously regenerated. Current explanations invoke a tilt between the disk and triaxial dark matter halo, or a continuous infall of material with angular momentum misaligned with that of the disk. Galaxy disks can also sustain vertical oscillations due to their self-gravity, but no permanent global mode can be maintained (*11*) unless fed by external forcing. Again, Saturn's rings provide the best example of externally forced permanent warp: the spiral bending waves (*12*) associated with resonance locations of Saturn's slightly inclined moons, known since the Voyager fly-bys in 1981. In contrast, the present corrugation patterns are truly transient features whose origin can be dated to within a few months, based on their observed degree of winding. Furthermore, this winding will proceed to make them eventually indiscernible.

Hedman *et al*. make a thorough analysis of how to provide the initial tilt responsible for the pattern. They argue that a sudden turning of Saturn's rotation axis is improbable and conclude that the rings themselves have been inclined, in a hit by an interplanetary debris cloud in Fall 1983. Such debris clouds, whose off-axis angular momentum transfers much more effectively to the ring than that of a single solid body, could be produced by comets disrupted in close planetary passages. The study by Showalter *et al*. adds a crucial piece of evidence by linking the Jupiter ring corrugations directly to the debris associated with the comet Shoemaker-Levy 9, which disintegrated during its close passage with Jupiter in 1992 and whose largest fragments hit the planet in 1994.

Besides providing fresh examples of the rapid dynamic processes in planetary rings (*13*), the implications of the vertical cor-

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rugations are that they add a valuable tool for measuring the local ring properties: Although the ring's self-gravity has no role in exciting or maintaining the corrugations, the local nodal regression rate due to Saturn's oblateness increases by the extra gravity of ring particles. Hedman *et al*. verify this feeble effect and obtain a new estimate of the C ring's surface density. Concerning the dynamical evolution of the outer solar system, the amount of cometary debris is probably larger than previously anticipated. The rings, with their enormous surface area, thus provide an effective flypaper detector for interplanetary debris.

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CELL BIOLOGY

Ancient Neurons Regulate Immunity

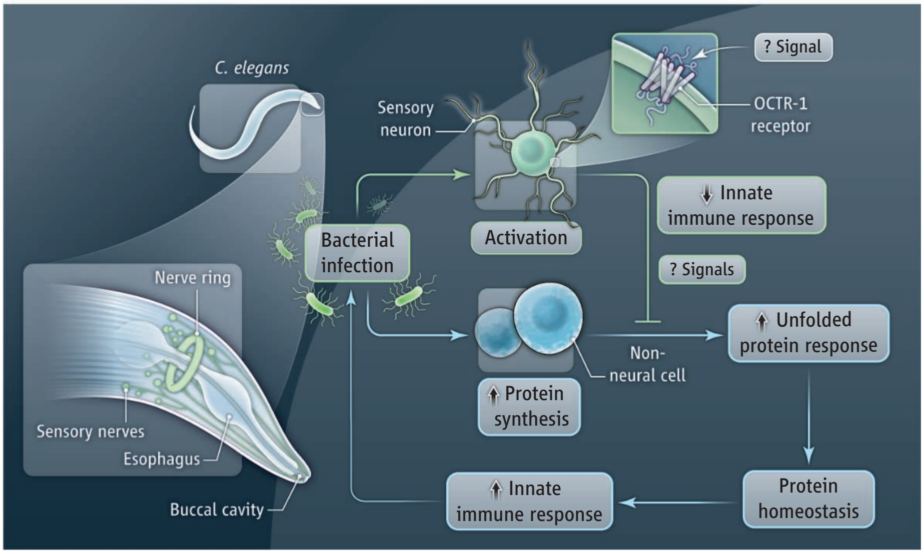
The innate immune system of multicellular animals is regulated by the nervous system.

Kevin J. Tracey

The most evolutionarily ancient type of immunity, called "innate," exists in all living multicellular species. When exposed to pathogens or cellular damage, cells of an organism's innate immune system activate responses that coordinate defense against the insult, and enhance the repair of tissue injury. There is a modern-day cost associated with these processes, however, because innate mechanisms can damage normal tissue and organs, potentially killing the host. Human life is a balance between dual threats of insufficient innate immune responses—which would allow pathogens to prevail—and overabundant innate immune responses—which would kill or impair directly. What has been the key to maintaining this balance throughout years of mammalian evolution? On page 729 of this issue, Sun *et al*. (*1*) report that neurons in a nematode worm can regulate innate immunity, a mechanism dating back to the early origins of the nervous system itself.

Research on the pathophysiology of infection in the late 20th century revealed that molecules produced by the innate immune system, not pathogens, account for the major physiological, metabolic, and pathological responses to infection in mammals. Cytokines and other molecules were associated with the signs and symptoms of infection, ranging from fever, anorexia, and fatigue, to lethal shock and tissue injury. By understanding the "cytokine theory of disease," it became possible to develop highly selective drugs that neutralize cytokines and experimentally modify the pathophysiology of infection (*2*). This same approach subsequently revolutionized the treatment of inflammatory disease in humans with other, noninfectious, but inflammatory conditions. Today, millions of patients with arthritis, colitis, and other inflammatory syndromes have benefited from therapy with cytokine-blocking agents.

These advances also underscored the importance in understanding mechanisms that control innate immunity and restrain it from injuring the host. Early work focused on soluble factors that control innate immune responses by inhibiting the synthesis or action of cytokines. This "protective mediator" list grew to include glucocorticoid hormones, soluble cytokine receptor fragments, and other anti-inflammatory factors (*3*). More unexpected, however, were later findings that information propagated in neurons controls the magnitude of the mammalian innate immune response. Action potentials traveling in the vagus nerve to the spleen and other organs culminate in the release of acetylcholine, an evolutionarily ancient molecule that effectively inhibits cytokine production by innate immune cells. The cytokine-blocking mechanism requires signal transduction through α7 nicotinic acetylcholine receptors expressed on macrophages and other cytokine-producing immune cells (*4*). Signals generated via this neural circuit tonically suppress innate immunity, because lesions in this pathway enhance the innate immune response to pathogens and injury



Innate innervation. Infection of *C. elegans* with a pathogen stimulates the innate immune response and activates the synthesis of new proteins, potentially causing the accumulation of unfolded proteins in host cells. To restore protein homeostasis, the unfolded protein response is activated. ASH and ASI sensory neurons negatively regulate the innate immune response to infection by blocking the unfolded protein response in nonneuronal cells. The OCTR-1 receptor in the sensory neurons is required for this effect.

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