backward in a computer simulation, Charnoz *et al*. (*1*) demonstrate that the spiral arose as a localized cloud, about 300 km in radial extent, in early 2004. In other words, the spiral began with an event. But whereas the clumps imaged by Voyager dissipated on time scales of months or less, this pattern has persisted for at least 1.5 years and is likely to continue for years more. (The spiral strands will continue to wind ever tighter until, eventually, they blend into a more uniform skirt around the F ring.)

The discovery of spiral structure reopens the debate over external versus internal collisions. Charnoz *et al*. (*1*) emphasize the role of S/2004 S6, although they acknowledge that its mass is far too small to create such an immense pattern. They suggest instead that scattering of ring particles off the surface of S/2004 S6 may be the cause of the spiral. As for interactions of the ring with other moons, Prometheus is large enough, but we know that it has not collided with the F ring in the recent past.

So what is left as an explanation? When particles collide, the energy of impact is distributed among particles typically comprising 10 to 100 times as much mass as that of the impactor. As a result, any impactor that produced the spiral probably came from a distance far greater than 300 km. So an alternative to the model of Charnoz *et al*. (*1*) is that we are seeing the outcome of a rare, very large impact into the F ring. By this argument, S/2004 S6 may still play a role but, as a consequence, not a cause—it may be a particularly large shard left over from the impact, which now finds itself on a very different orbit from the rest of the ring.

Further monitoring by Cassini should enable us to distinguish between these models. If S/2004 S6 has a continuing role, then we would expect a new spiral to form when its orbital orientation again intersects the ring's core. If Cassini is still operational in 2009, then we might observe the formation of something similar when Prometheus begins to skim the inner edge of the F ring. On the other hand, meteoroid impacts would be expected to occur randomly, with a broad distribution of sizes from the numerous small, clump-forming events to the very rare, large, spiral-forming events.

Perhaps the most fundamental questions concern how the F ring came to exist and why it is so strange. A few factors are important. First, the ring orbits at the edge of the Roche limit, the boundary that separates rings from moons. Inside the Roche limit, Saturn's tidal forces overcome self-gravity, preventing moons from accreting. In the F ring, some reaccretion is possible, so ring bodies are continuously breaking up and joining back together. Second, it is faint and narrow, so that small injections of new dust are quite noticeable; in the A ring, the equivalent of a spiral-forming event might pass unnoticed. Third, the ring and its nearby "shepherds" all follow highly eccentric orbits, which means that the ring is perturbed quite radically as the moons approach and recede on each orbit. (Here "shepherd" is probably a misnomer; elsewhere in the solar system, nearby moons act to confine rings, whereas the perturbations by Prometheus and Pandora are quite disruptive.) The ring also presumably contains a large mass of its own; how else could it maintain a fixed eccentricity against the tendency of ring particles to precess at different speeds? The picture that emerges is that of a ring that arose from the disruption of a small moon—perhaps the size of Prometheus—that lives in an environment too severely perturbed to ever settle down into a uniform, circular ring. If history is any guide, the F ring harbors a few more surprises that are awaiting Cassini's instruments and science teams.

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DEVELOPMENTAL BIOLOGY

Encountering MicroRNAs in Cell Fate Signaling

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Organisms must tightly regulate where and when each of their genes is expressed, lest their development goes awry with potentially lethal consequences. The mechanisms that control these important time and place decisions have been of great investigative interest. Hence, it came as a huge surprise that a major level of gene regulation was completely unknown until the recent discovery of a class of small regulatory RNA molecules known as microRNAs (*1*, *2*). Ever since, we have been racing to understand microRNA function during the development of multicellular animals and plants. On page 1330 in this issue, Yoo and Greenwald (*3*) describe a direct connection between the miR-61 microRNA and LIN-12/Notch, the cell surface receptor that controls a fundamental and highly conserved signaling pathway. The link marks an important advance in our understanding of the role of microRNAs in developmental processes.

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Mature microRNAs are small RNAs, about 22 nucleotides in length, that are encoded in the genomes of every multicellular organism examined so far. MicroRNAs block gene expression by binding to complementary sequences in the 3′ untranslated region (3′ UTR) of messenger RNAs and directing either degradation of the messenger RNAs or inhibition of their translation (*2*). MicroRNAs were first discovered in the microscopic roundworm *Caenorhabditis elegans* as important regulators of developmental timing. They have since been implicated in other aspects of development in plants (*4*) and animals, including vertebrates and invertebrates (*1*, *5*). However, little is known about the precise role that microRNAs play in many important developmental decisions. For example, communication between cells via molecular signals is a universal mechanism to coordinate cell fate decisions during animal development. What role do microRNAs play, if any, in signaling pathways? Using *C. elegans* vulval development as a model system, Yoo and Greenwald provide an answer.

The vulva is a specialized adult structure that provides a connection from the uterus of the worm to the external environment (*6*) (see the figure). In wild-type larvae, three vulval precursor cells, called P5.p, P6.p, and P7.p, are specified to eventually form the adult vulva. Each of these cells adopts one of two vulval cell fates—primary (1°) or secondary (2°)—in the precise spatial pattern 2°-1°-2° (see the figure). LIN-12 (the worm homolog of Notch) specifies the 2° fate in P5.p and P7.p, whereas the epidermal growth factor signaling pathway specifies the 1° fate in P6.p. Cross-talk between the two pathways ensures the spatial precision of these cell fate decisions (*6*). To understand how LIN-12 signaling specifies the 2° fate, one must identify the target