ganism found in forests, they discovered that diacylglycerol, in conjunction with a still-unknown protein, stimulates individual actin monomers to nucleate—come together at a site near the plasma membranes of the cell. This figures to set the stage for polymerization, since three monomers must link up in an actin trimer before the long filaments can grow.

Until recently, another molecule, in some respects the parent of diacylglycerol, captured most of the attention in the actin community, but Shariff and Luna's work may change that. This molecule, a phospholipid called PIP_2 (for phosphatidy-linositol-4,5-bisphosphate), gives birth to diacylglycerol when it is split into two pieces by cellular enzymes. PIP_2 drew the spotlight when a number of proteins that bind to actin, such as gelsolin and profilin, were found to bind also to PIP_2, raising the possibility that the lipid was at the heart of all the signals that controlled assembly and disassembly of actin filaments within the cell. While interactions between PIP_2 and actin are certainly observed, questions about their importance and timing in regards to cell movement have grown, especially as some inconsistencies have arisen in further research. "We simply do not have enough information about the biochemistry or events in live cells," says Pollard. "No one has enough data to prove any one theory correct."

The unknown protein. For now, Pollard may be right, but Shariff and Luna think the case for diacylglycerol's importance will be bolstered once they nail down a mysterious protein their work revealed. Their experiments clearly indicated that diacylglycerol did not directly provide nucleation sites but needed to interact with a mystery protein, which they contend is also bound in the plasma membrane. Indeed, they hazarded a guess—that the enzyme protein kinase C, known to be a popular target of diacylglycerol's action, was the quarry. But a number of tests the duo did seemed to rule out that possibility. So now all they are able to say is that the mystery protein may contain a diacylglycerol-binding site similar to that of protein kinase C and may work by regulating the activity of ponticulin, an actin-binding membrane protein implicated in nucleation. And then there's another possibility, says Luna: that this mystery protein directly promotes nucleation. To test the two alternatives, the two scientists plan to examine the effect of diacylglycerol on membranes that do not contain ponticulin.

Luna sees her paper as a wake-up call that should draw interest to diacylglycerol. Most of the actin world has been thinking of actin regulation in terms of PIP_2, or possibly other compounds such as inositol triphosphate or calcium, she says. "This will shake things up." Many of the scientists in the field, including Luna, hesitate to polarize the issue into a PIP_2-diacylglycerol contest. They point out that still undiscovered mechanisms may be important. Indeed, two or three new actin-binding proteins are discovered each year.

Actin at work. A thick network of actin filaments and other proteins allows cells to move about.

More important, says Pollard, the work on diacylglycerol "is not necessarily in conflict with PIP_2 theories." For instance, while diacylglycerol may control actin nucleation, PIP_2 could regulate how filaments assemble and disassemble.

All this uncertainty in the theories about actin polymerization is a source of real frustration to researchers. If the myriad of cellular signals involved can be cleared up, the mysteries of how amoebas crawl and cancers spread may finally reveal their secrets. Now, Luna says, "the race is on to find this [new] protein." Perhaps when Shariff and Luna find their mystery protein that works with diacylglycerol, it will be an effective weed killer for misguided theories.

–John Travis

NUCLEAR PHYSICS

Cluster Fusion: Close, But No Cigar

Another fusion dream is hanging by a thread. In September 1989, a research trio at Brookhaven National Laboratory (BNL) reported what looked to them like a promising new route to nuclear fusion. When clusters of hundreds of heavy-water molecules are accelerated into targets loaded with deuterium (heavy hydrogen), the researchers argued, the energy of the collision was somehow getting concentrated into a few of the deuterium atoms, spurring them to fuse. The output wasn't dramatic—this was no cold fusion—but the Brookhaven team did claim that minuscule amounts of energy had been produced from what they dubbed cluster-impact fusion. So, while fusion power from their discovery might be but a remote prospect, it was at least a prospect, the researchers suggested. And while skeptics quickly emerged, a number of physicists bought the argument and set up their own experiments.

No surprise, then, that for 2 years the trio worked diligently to strengthen their position and deflect the skeptics' contentions that experimental or interpretational errors were lurking behind the exciting claim (*Science*, 25 October 1991, p. 515). But the skeptics were on to something, the BNL team of Robert Beuhler, Lewis Friedman, and Gerhart Friedlander now concedes.

In an erratum in the 30 March *Physical Review Letters*, the researchers write that artifacts in the accelerated cluster beams "are primarily responsible for events that have been ascribed to cluster-impact fusion." The sobering results, they say, come from experiments done "during the last several months" with collaborator Y.K. Bae, who joined the BNL team last year, in which small artifact ions in the beam were deflected from the target by a magnetic field. The beam lost as much as half of its mass as it passed through the field, and the fusion rate dropped at least a hundredfold. Apparently, the fusion events that had tantalized the group were triggered by small, still unidentified ions slamming into the target, not some exotic energy-concentration mechanism involving the clusters.

Richard Petrasso, a plasma physicist at the Massachusetts Institute of Technology, isn't surprised; he and his colleagues had predicted as much. On the page just before the BNL team's erratum, Petrasso and his colleagues published a "comment" in which they argue that small, highly accelerated artifacts in the beam could have yielded the observed fusion rates. But Petrasso is "staggered" by something that's not mentioned in the BNL erratum: his own team's comment.

Petrasso had showed a draft of it to the BNL team as early as last November, and he even spoke with Friedman on the phone about the comment in early February. Friedman defends the omission, in part by saying that his team had the magnetic deflection experiments on the agenda for a year, well before Petrasso's team worked up its objections. Petrasso isn't impressed.

Meanwhile, the debate over cluster-impact fusion isn't quite over: Fusion research has a knack for showing different sides to different people. For skeptics like Petrasso, cluster-impact fusion is just another busted fusion claim. For fellow physicist Robert Vandenbosch of the University of Washington, there's still hope that it is a real phenomenon—though his own cluster-impact fusion experiments will now move to a back burner. And for Friedman the new evidence is a setback, but not the end of the road: "We were not as cautious as we could have been," he told *Science*, then added that "it's premature to say there's nothing left."

–Ivan Amato