don't travel far, says Reaka-Kudla. In general, small species are less likely than large ones to have long-lived planktonic larvae, she says. Therefore, she argues, small species —which are more numerous and also more likely to be overlooked—have more restricted geographic ranges and are more vulnerable to extinction. Sea slugs also fit that picture, says Clark. One-third of the 33 species he's studied in the Florida Keys have either no free-floating larval stage or their larvae spend only a few hours in the plankton.

For many organisms, the question of exactly how far their larvae travel has not been tested. But larvae or no larvae, researchers have evidence that at least a few marine species have relatively narrow geographic distributions—making them vulnerable to extinction due to local disturbances. For example, Michael Smith, senior research scientist at the Center for Marine Conservation in Washington, D.C., analyzed the ranges of 500 Caribbean fishes, using recently published literature. While most did have broad ranges, he found that 16% were restricted to the Caribbean Sea alone or to smaller geographical areas.

Among invertebrates, isolated cases of such narrow distributions are also trickling in, says Knowlton. For example, the Kumamoto oyster, *Crassostrea sikamea*, has planktonic larvae, but its native range was apparently restricted to the southernmost island of Japan, according to Dennis Hedgecock of the University of California. Although farmed for the restaurant trade on the U.S. West Coast—you'll pay extra for its delicate flavor—the Kumamoto now appears to be extinct in Japan, says Hedgecock.

Laws of the sea. Despite such tales, few marine biologists claim that marine extinctions are occurring at the same rate as those on land. Most biologists agree that marine species probably do have an extra measure of resilience. But biologists such as coral reef specialist Robert Buddemeier of the University of Kansas warn that numbers of extinctions in the sea may not serve as the same kind of ecological damage indicator as they do on land. "You don't want to get trapped into a linear comparison of terrestrial and marine ecosystems," says Buddemeier. "The marine system is less extinction-prone, but if you do start getting extinctions, it means you've got a problem on a much larger scale. The rules *are* different in the sea."

For example, in January a coral reef research and monitoring panel convened by the Department of State reported that many coral reefs in close proximity to large human populations are in decline; a colloquium on coral reefs held last June in Miami came to a similar conclusion, according to the draft report of the meeting. Reef biologists suggest that increases in nutrients, intense fishing (which decreases the numbers of predatory fish), plus natural disturbances such as storms combine to put reefs under stress. Specifically, says Jeremy Jackson of STRI, these factors tip the ecological balance in favor of corals' chief competitor, algae. The end result, already seen at some locations: large, fleshy macroalgae thriving atop dead coral.

"I can't point to a single species of coral which has gone extinct in the Caribbean," says Jackson. "But that doesn't mean that corals aren't in decline. I see it everywhere: The corals look like they need a shave—they're being overgrown with algae."

The visible danger sign is not the extinction of geographically restricted species, as might happen in a rain forest, but rather a widespread change in abundance of species, explains Knowlton. "It's different from a forest. You can chop a forest down, but the pieces you don't chop are more or less OK. In the ocean, it's more interconnected. You can have the whole system slowly drifting downward."

For those convinced that marine habitats are at risk, the next step is to find ways to protect them. In the Florida Keys, a new sanctuary has been set up to protect reefs. But how to manage a watery park poses a whole new set of questions, since neither currents nor organisms respect park boundaries, says Buddemeier. Although not everyone is convinced of the need for a more organized approach, many scientists are hoping that efforts like that of the NRC will outline the research needed to understand—and perhaps protect—the diversity of marine life.

–Elizabeth Culotta

Additional Reading

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NEUROBIOLOGY

All Strung Out at the Synapse

Just as understanding how a telephone system works requires knowing what goes on inside the switchboards, understanding the nervous system requires knowing what happens inside the nerve endings. It's there—at the synapses where adjoining neurons talk to one another—that much of the biochemical machinery of communication is found. Although this machinery is complex, over the last few years, some progress has been made in understanding it. Neurobiologists have found well over a dozen proteins sitting in the chattering tips of the nerve cell. Synapses, however, are loaded with proteins, and finding one there is often the easy part; the tough part is pinpointing its role in communication.

But that task is not impossible. And, just to prove the point, two papers in this issue of *Science* provide new clues about one protein's role in the intimate conversation between nerve cells. On page 977, Konrad Zinsmaier and Seymour Benzer of the California Institute of Technology and their colleagues provide direct evidence that a nerve protein, known as a "cysteine string protein" because it has several cysteine amino acids strung together in its middle, is essential for neural communication in the fruit fly. And on page 981, a team led by Cameron Gundersen of the University of California School of Medicine in Los Angeles reports a finding that helps explain what the protein's role in neuronal communication might be.

"Whenever people find a protein in the nerve terminal they assume it's essential, but the proof of that [typically] comes much more slowly," says fruit fly neurobiologist Tom Schwarz of Stanford University in Palo Alto. But the new results make it clear, he says, "that without the [cysteine string] protein, the terminal is not very happy."

These latest findings also mark the cysteine string protein's transformation from a mere novelty to a protein of potentially great neurobiological importance. The proteins were discovered in fruit flies in 1990 by Zinsmaier, then a graduate student with Erich Buchner at the University of Würzberg in Germany. The long string of cysteines was intriguing, says Zinsmaier, because "there's absolutely no sequence like that in any other synaptic protein." Moreover, because the protein was concentrated in the synapses, it was an excellent candidate for a neural communications protein.

That candidacy, however, remained purely speculative until the Benzer group's current study, in which the researchers bred fruit flies that had been genetically engineered to lack the cysteine string protein. All the mutant flies suffered from paralysis that killed most of them before they hatched; the few survivors died from the same paralysis as adults. The larvae that die appear to develop normally, Zinsmaier says, "but the normal muscle movement is not there, and they cannot hatch."

The adult survivors eventually died, because they, too, lost control of their muscles. "They get sluggish, then they can't fly, then they can't walk, and eventually they can't even retract their proboscis," says Zinsmaier. In spite of these symptoms, the demise of the mutant flies is not due to problems with their muscles. Those problems are in neuronal communication, a suspicion the Benzer team confirmed by measuring the ability of neurons in

the retinas of mutant flies to pass signals to one another. When they stimulated a neuron, that neuron responded—but its signal did not get transmitted to the next neuron. What's more, the tips of the nerves, at the synapse, appeared to have degenerated.

Other investigators are excited about the method the Benzer team used to show that the cysteine string protein actually has a crucial synaptic function. "This paralysis phenotype shows just how potent genetics can be in showing that a [synaptic] protein really is important," says Schwarz. The genetic method doesn't, however, show precisely what that function is—that is where the work of the Gundersen team comes in.

Their work suggests that the protein may be necessary for the release from the nerve tip of the neurotransmitters that carry the nerve signal across the synapse to the next neuron. The group came to this conclusion because they found that in nerve cells of a fish called the torpedo ray, the cysteine string protein is anchored to the surface of the synaptic vesicles, tiny membranous sacs which contain the neurotransmitters prior to their release into the synapse.

Gundersen speculates that the cysteine string protein on the surface of the vesicle may help open calcium ion channels in the tip of the neuron, triggering the calcium influx that is needed for the synaptic vesicles to spill their contents into the synapse. "One can imagine," he says, "that when the vesicle gets close to, or docks at, the presynaptic membrane, it (the cysteine string protein) interacts with the calcium ion channel." And, in fact, that fits with earlier work from the Gundersen lab that hinted that the string protein might regulate, and so possibly associate with, calcium ion channels.

If Gundersen's theory is correct, one would predict, he says, "that if you stimulate a nerve in a *Drosophila* mutant you should see inhibition of the calcium influx." And that's exactly what Zinsmaier intends to test next. Using calcium imaging techniques, he says, "we can visualize the presynaptic calcium influx, and see whether or not it is altered in our mutant flies." Meanwhile, the Gundersen team is trying another tack.

They want to know just how widespread cysteine string proteins are throughout the animal kingdom. If they find the protein and its gene in humans, says Gundersen, that will be a major cause for excitement. Efforts to understand neuronal degenerative diseases such as Parkinson's and Alzheimer's have been intense, but the underlying causes remain only poorly understood. If lack of the cysteine string protein causes neuronal degeneration in the fruit fly, he reasons, why not in humans, too? And if that proves to be the case, the once-obscure cysteine string protein will have truly arrived.

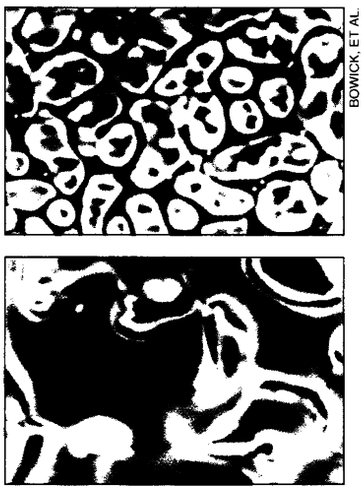
–Rachel Nowak

PHYSICS

Recreating the Universe's Fateful Flaws

Most people who gaze into crystals hope to see the future, but some physicists are now doing the reverse: peering into a drop of liquid crystal to see whether they can glimpse the beginning of time. As a liquid crystal cools, the material's rodlike molecules make an abrupt change from helter-skelter disarray to an orderly state, in which the molecules line up like logs in a raft. That process, some researchers believe, could be a model for a similar transition that took place in the first fraction of a second after the big bang. A liquid crystal, says Syracuse University physicist Mark Bowick, "is an excellent analogy [to the mathematics]."

On page 943 of this issue, Bowick and his Syracuse colleagues Eric Schiff and L. Chandar, along with Ajit Srivastava of the University of California, Santa Barbara, exploit that analogy as what Schiff calls a "reality check" on a cosmological theory. The theory holds that if various parts of the primordial universe made the transition independently, the change would have spawned "cosmic defects." These flaws in space-time could have seeded the growth of larger structures, such as galaxies, before gradually vanishing. Now, the Syracuse team has found that in the model universe of a liquid crystal, one kind of defect—threadlike "strings"—forms at about the rate implied by the theory. "It's nice to have experimental confirmation," says Brown University cosmologist Robert Brandenberger.



BOWICK, ET AL.

Microcosm. Liquid crystal "domains" *(top)* merge, forming strings.

The scenario that the Syracuse group set out to test was originally proposed in 1976 by theoretical physicist Thomas Kibble of Imperial College, London. Kibble's starting point was a widely accepted account of changes in the newborn universe. At first, the fundamental forces, such as the strong and weak forces, were indistinguishable. This "symmetry" broke as the universe cooled and distinct forces emerged. The symmetry didn't break on its own, however; helping the process along was a hypothetical force field called the Higgs field, which gave space-time an overall "direction"—a mathematical abstraction analogous to the overall orientation of molecules in a liquid crystal.

Kibble argued that this process took place independently in many different parts of the universe, like curds forming in milk. As a result, the Higgs-imposed direction of space-time could differ for each curd, or domain. As the universe cooled further, the domains jostled together and merged, but sometimes their directions differed too much for them to merge smoothly. And, as Bowick puts it, "When neighboring domains can't jiggle to make everything uniform, you get defects trapped." Strings, for example, might be created at points where three or more irreconcilable domains met. That would happen at a predictable rate, Kibble said, depending on the range of possible domain orientations.

Bowick and his colleagues realized they could give Kibble's idea a real-world test in liquid crystals, because they form separate bubbles of the ordered phase—analogous to the Higgs-inspired domains—during the transition to order. Three years ago, Bernard Yurke of AT&T Bell Laboratories, Neil Turok of Princeton, and their colleagues showed that when these bubbles merge, they spawn all manner of "cosmic" defects, including threadlike remnants of the disordered phase resembling cosmic strings. As a test of the Kibble mechanism, the Syracuse team decided to see whether it could predict the number of strings in a liquid crystal.

Finding out what the Kibble mechanism predicted, however, was no easy task, as Srivastava and Bowick found; complications such as the influence of the top and bottom of the cooling layer on domain orientation forced the researchers to build a simplified computer model of the material. The model predicted that strings would form at a rate of about 0.6 per bubble. Then it was a matter of painstaking observation, says Schiff: "You see bubbles form, count the number of bubbles, measure the number of strings per bubble."

The actual number was, indeed, about 0.6 strings per bubble. "It's the first experimental verification that the idea is qualitatively correct," says Bowick. Turok agrees, but he puts the emphasis on qualitative. "To be honest, I'm not a hundred percent convinced by the numbers," he says, noting they reflect a lot of assumptions and approximations. And he cautions that demonstrating the Kibble mechanism in a liquid crystal doesn't prove that strings existed in the early universe or shaped its structure. For scientists, like the rest of us, crystal gazing has its limits.

–Tim Appenzeller