the quakes were only taking place where there is still olivine.

At a depth of 400 kilometers, that includes much of the thickness of the slab. But by the time the slab reaches depths of 600 kilometers and more, untransformed olivine, capable of breeding deep quakes, should linger only in a layer perhaps 10 kilometers thick at the center of the slab, where it is coolest. And there's the rub. The rupture that generated the massive Bolivian quake extended across a plane measuring 30 by 50 kilometers. And the increasing abundance of modern seismographs in South America and around the world allowed seismologists to pin down the large rupture's precise orientation—it cut more or less horizontally across the steeply inclined slab, extending well beyond the supposed olivine layer.

One possible explanation is that the slab and its shrinking olivine core have somehow crumpled and thickened at that depth. Stephen Kirby of the U.S. Geological Survey in Menlo Park, California, suggested at the AGU meeting that they might have done so as the slab ran into the more resistant lower mantle, which begins at 670 kilometers. But Silver is skeptical. He argues that the fuzzy images of slabs revealed by seismic waves suggest they deform only in the lower mantle, and when they do, it is by accordion-like folding, not by thickening. Even if a slab did thicken, says Silver, the heat generated by the deformation would wipe out the olivine core before it was thick enough to generate a quake like the one beneath Bolivia.

Seismologist Hiroo Kanamori of the California Institute of Technology offered another alternative. "It's very possible," he says, "that the rupture was triggered by some mechanism" like transformational faulting within a thin slab core, then sustained outside the core by some other means. And Kanamori thinks he sees a possible mechanism: "If frictional melting occurs on the rupture, it is probably possible to sustain faulting over a large zone. I think melting is very likely; it's very difficult not to melt" the fault as its two sides slide by each other under high pressure.

That would make for two mechanisms, at least, to explain the deepest earthquakes, which "doesn't seem very economical" to Silver. He and Charles Meade of Carnegie's Geophysical Laboratory are pursuing the possibility that a single alternative mechanism—the failure at great depth of faults formed millions of years ago when the rock was still at the surface—is at work. Green, however, is setting up new lab experiments to test Kanamori's proposal that a fracture touched off by transformational faulting can continue to propagate through unfavorable conditions. Either way, the Bolivian earthquake has left its mark. "The earth has spoken," Green says. "I think it's going to be productive."

–Richard A. Kerr

ZOOLOGY

St. Louis Meeting Showcases "Creature Features"

The 92-year-old American Society of Zoologists (ASZ) is preparing for a metamorphosis that will expand its focus from organism-level research to comparative studies ranging from the molecular to the ecosystem level. To mark the change, society officials hope to adopt a new name—the Society of Integrative and Comparative Biology—this year. But hints of change could already be seen in the ASZ's annual meeting, held in St. Louis from 4 to 8 January, where symposia spanned topics from DNA sequencing to ecological experiments.

New Evidence About Feminized Alligators

To the casual eye, the young male alligators dwelling in Florida's Lake Apopka may look perfectly normal. But they aren't: They have low levels of testosterone, high levels of estrogen, and unusually small penises. They've been feminized, apparently due to a major pesticide spill in the lake in 1980. Their plight has often been considered one of the best documented examples of the effects of environmental estrogens, compounds that mimic the female hormone estrogen. But just which chemicals are at fault, and how they exert their unfortunate effects on the alligators, has been something of a mystery.

HOWARD SUZUKI

Anti-androgen victim? If male, this Lake Apopka alligator hatchling may be sexually abnormal.

But new evidence presented at a special ASZ symposium on environmental endocrine disrupters by toxicologist L. Earl Gray of the Environmental Protection Agency (EPA) now suggests that the alligators aren't suffering from an excess of estrogenlike compounds after all. The culprit in Lake Apopka may instead be a compound called *p,p'*-DDE that exerts its feminizing effects by blocking the effects of androgen, the male hormone. *p,p'*-DDE had been linked to the alligators' hormonal problems before, but researchers had been puzzled by the fact that the compound's chemical activity was not similar to estrogen's. "Now I can explain what I'm seeing," says reproductive endocrinologist Louis Guillette Jr. of the University of Florida, who has been studying the Lake Apopka alligators with his co-workers. "DDE is not an estrogen, but an endocrine disrupter."

Other compounds considered to be environmental estrogens have been linked to feminized fish in British rivers and birds in the Great Lakes, and some researchers have been concerned that the chemicals also pose a risk to humans. These connections have been controversial, however (*Science*, 15 July 1994, p. 308), and the work of Gray and his colleagues will further complicate efforts to pin down the reproductive effects of environmental contaminants, because it implies that assessing the estrogenic effects of a suspicious compound is not sufficient; researchers must also probe whether the chemical blocks androgens, says Guillette.

Gray and colleagues didn't start out working on Lake Apopka animals. Rather, for the past few years, they have been studying Vinclozolin, a pesticide that blocks the effects of natural androgen by binding to the receptor through which the hormone works. Male rats exposed to Vinclozolin in utero have delayed puberty and malformed reproductive organs. In his ASZ talk, Gray explained that his work took a new direction when he heard Guillette speak about the Lake Apopka alligators at a meeting last April—and realized that they had abnormalities that "looked just like our Vinclozolin rats." Gray went home and began experiments to find out whether *p,p'*-DDE might also act as an antiandrogen. The results suggest it does.

Gray and his collaborators, William Kelce and Susan Laws, also of EPA, and Elizabeth Wilson of the University of North Carolina, showed that *p,p'*-DDE binds to both human and rat androgen receptors, blocking binding by natural androgens like testosterone. The team also showed that *p,p'*-DDE prevents androgens from turning on the genes they normally activate. Finally, they showed that young male rats treated with *p,p'*-DDE have delayed puberty.

And *p,p'*-DDE may not be the only envi-

ronmental contaminant with anti-androgen effects, says Gray. His co-authors Kelce and Laws have found, he says, that every estrogenic compound they've looked at so far, including the artificial estrogen DES, binds to the androgen receptor. So studies which ascribed sexual effects to the estrogenlike activity of these chemicals may have focused on the wrong mode of action, says Guillette.

Guillette, for one, is convinced *p,p'*-DDE is the "smoking gun" that demasculinized the Apopka alligators, but other leading endocrine disruption experts are eager to see more data. After Gray's talk, Howard Bern of the University of California, Berkeley, a pioneer in the field who worked on the effects of DES, pointed out that embryos might have a different androgen receptor than adults do. If so, then *p,p'*-DDE's action in embryos might be somewhat different from the receptor-blocking exemplified by Vinclozolin.

All in all, the scientists agreed that the new results demand additional attention for androgen-blocking agents. "People always ask me if there are environmental androgens, and I've always said no," says John McLachlan, a leading environmental endocrine researcher now at Tulane University. "Now perhaps the question should be expanded to, 'Are there environmental anti-androgens?' "—and the answer appears to be yes.

The Case of the Missing Mussel

Although marine conservationists have long been concerned about the loss of marine species, they've had an uphill battle in rallying support, because only a handful of modern marine extinctions have been documented (*Science*, 18 February 1994, p. 918). But at a special ASZ symposium on molecular techniques, Jonathan Geller of the University of North Carolina, Wilmington, aided the cause. He presented new DNA sequence data showing that a common marine species, a mussel that formerly lived off the coast of southern California, has become extinct there. And no one noticed.

The reason the extinction could take place so quietly is that the native species was replaced, sometime during the last 50 years, by a related mussel, a biological invader from the Mediterranean. "Here's one of the commonest, well-studied organisms, and it went regionally extinct under our noses and we didn't even know it," says Geller. "This suggests that we should not be complacent about the possibility of marine extinctions."

In addition to sounding a warning, Geller's study also illustrates the potential of molecular techniques in ecology, says marine biologist Steven Palumbi of the University of Hawaii, who helped National Institutes of Health biologist Joan Ferraris arrange part of the ASZ symposium. "It's an excellent example of how these molecular techniques let you look back into the past in ways we weren't able to do before."

Geller made his discovery while tracking the spread of the mussel *Mytilus galloprovincialis*, one of three *Mytilus* species that look similar but are genetically distinct. *M. galloprovincialis* is native to the Mediterranean but has invaded coasts around the world, apparently by hitching rides in ships' ballast water. Geller set out to document the history of its conquest of the western Pacific, using analyses of the mussel's mitochondrial genome to distinguish it from the other *Mytilus* species.

His data confirmed previous enzyme studies showing that while the native species, *M. trossulus*, still rules north of San Francisco Bay, the invading *M. galloprovincialis* is the only one of the three species found south of the bay. But when did the invader arrive, and what happened to the native *Mytilus* species?

To find out, Geller developed techniques to sequence DNA from dried and pickled museum specimens. Those sequences reveal that mussels taken from Santa Catalina Island off the coast of Los Angeles in about 1900 were more closely related to *M. trossulus* than to *M. galloprovincialis*. But by 1947, mussels from the Los Angeles area were clearly the Mediterranean type. The obvious conclusion: *M. galloprovincialis* invaded sometime between 1900 and 1947.

More importantly, the data show that sometime after 1900, the native species went regionally extinct along the southern California coast, possibly because of competition from the invader. Of course, the replacement of one mussel species by an ecologically similar one may have had little effect on the coastal ecosystem. But, says Geller, the finding is still important, as it's only the second documented regional extinction of a marine invertebrate. (The first was the Atlantic eelgrass limpet, published in 1991.)

The extinction is especially sobering because all three species of *Mytilus* are superabundant, found in a wide range of habitats, and very fecund. "You'd think you couldn't drive a species like that extinct if you tried," says Geller. But because this regional extinction did happen, he says, other marine species may also be at risk.

JONATHAN GELLER

Look-alike. No one noticed this Mediterranean mussel displacing a California species.

The Parasite *X-Files*

Death-dealing parasites are a staple of science fiction from the movie *Alien* to TV's trendy *X-Files*. But the tale told by ecologist Kevin Lafferty at another ASZ symposium, about a parasite that castrates one host and then renders the next more vulnerable to predation by modifying its behavior, needed no fictional embellishment.

The idea that parasites might evolve strategies to make their hosts more susceptible to predation is not new; such a strategy could help facilitate the development and spread of the parasite. But the work by Lafferty, a postdoc at the University of California, Santa Barbara (UCSB), is one of only a few field studies to show directly that behavioral changes induced by a parasite increase the host's chances of being eaten, said Janice Moore of Colorado State University, an expert in parasitic control of host behavior.

Lafferty studied a common flatworm, *Euhaplorchis californiensis*, whose life cycle begins when birds infested with the worm shed parasite eggs into a salt marsh with their feces. The parasite then invades a marine snail and castrates it by devouring the snail's sex organs. Next, this wandering killer swims from the snail to the killifish *Fundulus parvipinnis*, where it forms cysts on the fish's brain. The cycle is closed when a bird such as a heron or egret eats the killifish, allowing adult worms to develop inside the bird.

In lab studies, Lafferty and UCSB undergraduate Kimo Morris showed that the parasites clearly alter the behavior of infected fish. They were far more likely to jerk, shimmy, and swim at the surface—antics that might well attract predatory birds.

To find out whether that was true, the researchers put equal-sized fish from infected and uninfected populations into large outdoor pens to see whether birds were more likely to snap up parasitized fish. The results were dramatic: After 20 days, 80% of the severely infected fish had been eaten, compared to only 20% of less severely infected fish. But only 2% of uninfected fish were eaten. "The thing I didn't expect was the degree to which this strategy is effective," says Armand Kuris, Lafferty's former thesis adviser at UCSB. "Parasitized fish were more than 10 times more susceptible! That's altered my thinking. In some systems, predators might not be successful without these parasites."

Ecologists have been saying for years that parasitism ought to be given more attention in ecosystem models, but few have actually done so, says predator-prey ecologist Andy Sih of the University of Kentucky. But Lafferty's work, completed only a few weeks ago, should nudge the field in the direction of looking into its own horrifying *X-Files*.

–Elizabeth Culotta