Biology

Eggs or live young: A thrips'll try both

Chickens lay eggs; humans make babies. And the rule has always been that an animal species must settle on one reproductive mode or the other. Not so for the tiny, tree-living thrips, the first known animal that can switch between laying eggs and bearing live young, says biologist Bernard J. Crespi, who discovered the thrips' reproductive versatility while at the University of Michigan's Museum of Zoology in Ann Arbor.

Crespi says his finding will give scientists the first animal with which to test theories of the ecological and social factors important in the evolution of the two kinds of reproduction.

Crespi first demonstrated that the 5-millimeter *Elaphrothrips tuberculatus* uses two separate reproductive modes by dissecting the ovaries of more than 738 pregnant females and finding them specialized for either egg-laying or bearing live young. Then, to find out whether individual females can switch between the two modes, he watched summer-generation females, which live long enough to reproduce several times, breed on dead oak leaves inside nylon bags. In both 1986 and 1987, about one-third used both reproductive modes, he reports in the Jan. 26 NATURE.

Crespi also noticed that all 1,051 larvae from the 42 egg-laying females were female and that all 202 non-egg larvae from 32 live-young-producing females were male. The male-bearing females produced fewer offspring than those laying eggs, but proportionally more of their young survived.

Crespi went on to look at other thrips species. "The most exciting thing is that [this reproductive versatility occurs] in not just one species but a dozen," he says.

Before Crespi's discovery, scientists had assumed all *E. tuberculatus* lay eggs, but they knew some females became pregnant without producing eggs. Crespi sexed newborns from eggs and realized they were all female. "We knew the males existed but had no idea where they were coming from," he says.

Crespi says he had been studying this species of thrips for more than a year before he figured it out. Upon dissecting non-egg-laying females with curiously large abdomens, he found that "lo and behold, [they were] full of larvae."

Dogwoods fight fungus; fungus wins

A deadly fungus is attacking flowering dogwoods, and new research indicates the trees have little chance of resisting it. "Dogwoods have more problems than this disease, but none is quite as deadly as anthracnose," says plant geneticist Frank S. Santamour of the Department of Agriculture's National Arboretum in Washington, D.C.

Dogwood anthracnose was first noted in the late 1970s in New York, Pennsylvania, New Jersey and the Pacific Northwest. Since then, the disease has spread the length of the Appalachian Mountains, but its full extent is unknown.

To test whether dogwoods from any region could resist the disease, Santamour grew seedlings from trees from 17 states and then planted them in Maryland's Catoctin Mountains, where they would be naturally infected. After 2½ years, all the trees "were completely gone," he says. "So the disease is not going to be confined by any [genetic] resistance and is just going to spread. And it will depend on weather and about 10,000 other things how fast and how much it spreads."

The origin of the disease remains obscure. "Whether it's a native American fungus that remained dormant for years or [was imported] from abroad, we don't know," Santamour says. Pathologists have not even identified the species of the fungus, he adds, and no fungicidal treatment has yet been found.

The disease seems confined to the native U.S. dogwoods. When Santamour tested the Asian dogwood species, *Cornus kousa*, it showed some leaf spotting but resisted the full-blown disease.

Biomedicine

Blood cells yield cystic fibrosis clues

Scientists have discovered the molecular defect thought to cause cystic fibrosis in white blood cells, a finding that may provide a laboratory model for studying this lethal genetic disease and lead to a screening test for genetic carriers.

Researchers believe patients with cystic fibrosis have a faulty chloride channel, a gate in the cell membrane that regulates the entry of chloride. Previous work has shown that epithelial cells lining the body's airways contain the malfunctioning channel, but these cells are difficult to obtain and culture, a problem that has stymied research.

Jennifer H. Chen, Howard Schulman and Phyllis Gardner of Stanford University report in the Feb. 3 SCIENCE that they have found the defective chloride channel in white blood cells taken from cystic fibrosis patients. The work, if verified, would give scientists an easier way to study the channel because white blood cells can be obtained by drawing blood and are easily cultured.

The researchers say their work may lead to a simple blood test for carriers of the cystic fibrosis gene. At present, people with a family history of the disease can undergo DNA analyses and counseling to get an idea of their risk of having a child with cystic fibrosis. One out of every 20 people in the United States carries the gene without showing symptoms. Cystic fibrosis patients inherit a defective gene from each parent.

People with the disease have abnormally thick secretions in the lungs and other organs. Researchers suspect the faulty chloride channel leads to the heavy mucus that blocks airway passages and organs, eventually causing death. The average life expectancy for a child born with cystic fibrosis is 25, although advances in treatment have kept some patients alive much longer.

Hardy viruses survive drug assault

Researchers are detecting drug-impervious viral infections in some AIDS patients, a trend that makes treatment extremely difficult and suggests that viruses common in the general population may be outwitting the few drugs scientists have developed to combat them.

Two reports in the Feb. 2 NEW ENGLAND JOURNAL OF MEDICINE describe AIDS patients with drug-resistant herpes simplex virus and cytomegalovirus, both potentially lethal for AIDS patients. In one study, Kim S. Erlich at the University of California, San Francisco, and his colleagues found drug-resistant herpes simplex strains that caused painful skin ulcers in 12 AIDS patients. Most people who get a herpes simplex viral infection can be treated with the drug acyclovir, but Erlich's dozen AIDS patients failed to respond.

In the second report, Alejo Erice at the University of Minnesota Health Sciences Center in Minneapolis and colleagues describe resistant cytomegalovirus infections in three AIDS patients. Infection by cytomegalovirus, a member of the herpesvirus family, rarely leads to disease in healthy adults, but causes pneumonia and inflammation of the liver, kidneys, colon and retina in AIDS patients with damaged immune systems. Doctors typically treat these infections with ganciclovir, but the three patients studied by Erice proved resistant to the antiviral drug.

"There appears to be an emerging problem, primarily in the AIDS population," says Martin S. Hirsch of Massachusetts General Hospital in Boston. Hirsch worries that the emergence of drug-resistant viruses in AIDS patients may signal a problem for the general population, where reports of drug-resistant viruses have been rare so far. Scientists must develop a better arsenal of antiviral drugs, he says. But that may be a difficult task, because drugs that attack viruses often end up damaging the host cell as well, he adds.