Biology

Ants hint at how social species evolve

Two populations of the same species that live far apart and don't mate can develop such different traits that they become separate species, researchers believe. Moreover, sympatric speciation—in which new species evolve from populations that live close together but don't mate—may also occur.

New findings now support a controversial version of that model stating that different social behaviors in groups of a social species, such as ants, living close together may also give rise to new species. D. DeWayne Shoemaker of the University of Rochester (N.Y.) and Kenneth G. Ross of the University of Georgia in Athens describe the study in the Oct. 17 NATURE.

"As far as we are aware, Shoemaker and Ross are the first to demonstrate that social behavior influences sympatric speciation," assert Ross H. Crozier of La Trobe University in Bundoora, Australia, and Pekka Pamilo of the University of Uppsala in Sweden in an accompanying commentary.

Shoemaker and Ross studied differences in the social behavior and genetic makeup of two populations of fire ants (*Solenopsis invicta*) in northern Georgia. Specifically, they looked at segments of mitochondrial DNA, which only mothers pass on to their offspring, and segments of two nuclear genes. One group of ants, called the monogyne form, has one queen per nest. The polygene nest may contain 200 or more.

The genetic differences that the researchers found support their observations of who is—or more important, who is not—mating with whom, they contend. The only genetic link between the two populations is that monogyne males sometimes mate with polygyne females, they assert.

Many roadblocks bar the two groups from mixing their genes. Workers reject queens from the different form, so queens stay away from the other group's nests. Few fertile polygyne males exist, and they appear to mate only with females in a nest.

If monogyne males cease mating with polygyne females altogether, the two types may become distinct species. This could happen if, for example, the females begin mating only in the comfort of their nests and never in the open skies, where they now meet monogyne males. Such a change of heart has occurred among other species of ants, the authors note.

Ross

A monogyne fire ant queen and her workers.

The frequencies crickets love and fear

Field crickets (*Teleogryllus oceanicus*) typically call at a frequency of 4 to 5 kilohertz (kHz), while their archenemies, bats, produce ultrasound ranging from 25 to 80 kHz. Sounds above 16 kHz alarm crickets, giving them a wide margin of safety, Robert A. Wyttenbach of Cornell University and his colleagues report in the Sept. 13 SCIENCE.

To determine how crickets respond to sound, they exposed the animals to 20 kHz sounds, which normally cause the insects to flee. However, they repeated the sound so often that the insects' fear disappeared.

Other studies have shown that only a novel sound will reinstate a cricket's normal response. The team tested the animals with a variety of frequencies and found that sounds below 16 kHz did the trick. Sounds above 16 kHz were too similar to 20 kHz for the crickets to discriminate.

This suggests that crickets, like other animals, hear only broad categories of frequencies, although sound, like color, varies continuously.

The mutant genes that stymie HIV

A new study confirms earlier reports that people with two mutant copies of a particular gene are largely resistant to infection by HIV, the virus that causes AIDS. More important, the study provides evidence that people who have just one mutant copy of the gene, although not protected from HIV infection, do take longer than usual to progress to AIDS.

The crucial gene encodes a protein called CC-CKR-5, which sits on the surface of immune cells and is used by HIV to infect the cells. People normally have two copies of the gene, but investigators have found that about 1 percent of the white population has a large deletion, or missing DNA segment, in both of their *CC-CKR-5* genes. The most common strains of HIV cannot infect such people (SN: 8/17/96, p. 103).

After studying 1,955 people whose lifestyles put them at high risk for HIV infection, Stephen J. O'Brien of the National Cancer Institute in Frederick, Md., and his colleagues have found that those with the deletion in only one *CC-CKR-5* gene enjoy no protection from HIV infection. Yet if infected, they generally survive several years longer than infected people with two normal genes, the team reports in the Sept. 27 SCIENCE.

O'Brien notes that about 20 percent of white people appear to harbor the deletion in at least one *CC-CKR-5* gene. "That's more frequent than red hair. That kind of frequency doesn't arise randomly," he says. To explain why the deletion is now so common, O'Brien suggests that long ago, the relatively few Europeans who had the *CC-CKR-5* deletion may have survived a version of AIDS or some other fatal illness that wiped out much of the population.

Smoking leaves fingerprints on DNA

Few people outside the tobacco industry remain skeptical about the link between lung cancer, the leading cancer killer in the United States, and smoking. A new study examining the DNA damage caused by a single compound in tobacco smoke should cast out any lingering doubts.

When physicians study people with lung cancer, they find the same genes mutated in patient after patient. In particular, about 60 percent of lung cancer patients have mutations in the *p53* gene. The protein encoded by this gene normally prevents cells from growing out of control and can even command a cell to commit suicide if it becomes cancerous.

In recent years, physicians have documented the curious fact that *p53* mutations in lung cancer patients usually occur at three specific sites, known as codons 157, 248, and 273, on the gene's DNA sequence. Those genetic hot spots are exactly where a common compound in tobacco smoke prefers to mutate *p53*, scientists now report in the Oct. 18 SCIENCE.

A research group headed by Gerd P. Pfeifer of the Beckman Research Institute of the City of Hope in Duarte, Calif., treated lung and other kinds of cells with benzo[*a*]pyrene, a component of tobacco smoke that belongs to a family of cancer-causing agents known as polycyclic aromatic hydrocarbons. The scientists found that *p53* genes in such cells were laden with mutation-causing adducts, sites where a metabolite of benzo[*a*]pyrene had attached itself to the gene's DNA. When they examined the location of the adducts, the scientists found that codons 157, 248, and 273 were affected most often.

While epidemiological studies have provided all but conclusive evidence that tobacco smoking causes lung cancer, the new results appear to provide the first direct evidence associating carcinogens in smoke with specific lung cancer mutations. "It's a bit harder to deny the connection now," says Pfeifer. His group intends to study the patterns of *p53* adducts formed by other suspected carcinogens to see whether the compounds are linked to additional types of cancer. Examining how such adducts form is also a priority, says Pfeifer.