How bad-tasting species got their markings

Someone who sports dark glasses, a hat pulled down low, a bulging coat pocket, and a swagger is sending a clear message: Leave me alone if you value your life.

Other members of the animal kingdom, such as bumblebees, employ a similar strategy. They have striking colors and markings that warn their predators that they taste bad and may even be poisonous. A recent study provides new details about how these warning, or aposematic, signals evolved. It suggests that unpalatable creatures living in family groups were the first to display them, Rauno V. Alatalo and Johanna Mappes of the University of Jyväskylä in Finland report in the Aug. 22 NATURE.

The Finnish scientists "succeed in uncovering selective forces involved in the initial evolution of anti-predator warning signals," Tim Guilford and Candy Rowe of the University of Oxford, England, say in an accompanying comment.

Many animals with aposematic markings now live in groups. Some researchers have argued that aposematic signals first evolved in unpalatable species that lived in families, as their predators would eat one member of the group but then stay away from similarly marked relatives. But other scientists say that unpalatable creatures could have evolved aposematic markings well before they took to group living.

In their study, Alatalo and Mappes examined the responses of 16 birds called great tits (*Parus major*) to artificial prey, both palatable and unpalatable, that the scientists had marked with novel symbols. Because the birds had no experience with these markings, the researchers assert that the tits' initial reactions probably resemble animals' responses to early aposematic colors and patterns.

Stalks of rye with fat in their hollow stems served as prey for the birds. The investigators dipped some of these stalks in chloroquine, a bitter tasting antimalarial drug. To each stalk they attached a piece of paper marked with a symbol. All palatable items were tagged with shapes that matched patterns on the cage floor; the unpalatable items had markings that either blended with or stood out against the floor.

For one hour every day for three days, the birds searched for the palatable food among different arrangements of the artificial prey. Some items lay 20 to 30 centimeters apart, while others were placed in groups of similarly marked and flavored food.

The palatable food, as expected, proved generally more popular than the chloroquine-flavored prey. The tits, however, ate fewer of the tasty items that lay alone than in groups. By contrast, the chloroquine-flavored items that stood alone were tried more often than those in groups.

Furthermore, among the unpalatable, solitary prey, the birds were at first more likely to taste the conspicuously marked items than those that didn't stand out.

"Altogether, this suggests that initially aggregation would have been beneficial for the aposematic [conspicuously marked] prey," the authors explain.

Eventually, the tits learned to largely avoid the conspicuously marked prey.

The second trial tested how the birds would react to another "species" of prey—almond pieces—equipped with the same markings as the rye and, in some cases, dipped in chloroquine. The tits continued to avoid the conspicuously marked prey. This time, however, they hit on as many conspicuous items that were in groups as those that lay alone.

The experiments show that aposematism probably evolved first in groups of unpalatable prey, but that other species that later adopt similar markings may not benefit from group living, the authors conclude.

"Yet we suspect that the controversy is not altogether over," Guilford and Rowe note. Predators may become so abundant in some cases that their aposematic prey still benefit, as other species do, from living in groups. Also, species may happen to evolve new markings during times of low predator populations and not need the additional benefit of a group. — *T. Adler*

Marie Read

Douglas C. Ferguson

*In the lives of many insects and other animals, yellow and black scream "stay away," such as in the case of the harlequin frog (*Atelopus varius*) from Panama (top) and the cinnabar moth (*Tyria jacobaeae*) caterpillar (bottom), shown here resting on a tansy ragwort in Quilcene, Wash.*

Is NO a good cop or bad actor in malaria?

Nitric oxide has suffered from an unsavory reputation for years, with good reason. Not only is the highly reactive chemical guilty by association—it is an ingredient of smog and cigarette smoke—but excess production by brain cells damages nerves during a stroke.

More recently, however, nitric oxide has emerged as the biochemical linchpin in a host of essential bodily processes. Among other things, it conveys molecular messages, helps control blood pressure, aids digestion, boosts erections, and kills tumor cells.

Now researchers in the United States and Tanzania have provocative evidence suggesting that nitric oxide may guard against the ravages of malaria, a mosquito-borne parasitic disease that kills up to 2 million people a year. Their study is the latest to suggest that nitric oxide may be a formidable bulwark in the body's fortifications against infectious disease.

The findings, reported in the August JOURNAL OF EXPERIMENTAL MEDICINE, can be stated simply. "The higher the NO [nitric oxide] concentration [in blood], the lower the parasite load and the milder the illness," says J. Brice Weinberg, a hematologist at Duke University Medical Center in Durham, N.C., and a study author.

The role of nitric oxide in human chemistry is anything but simple, however, and the study, although it is "a nice piece of clinical research," raises many questions, says Lee Hall of the National Institute of Allergy and Infectious Diseases.

The Duke study focused on *Plasmodium falciparum,* the deadliest of the four malaria parasites. These parasites infest red blood cells, where they feast on oxygen-bearing hemoglobin. *P. falciparum* is more dangerous than the others, because a small fraction of the people who are infected develop a severe complication known as cerebral malaria. It is fatal in one third of the cases where it occurs.

Some researchers postulate that cerebral malaria occurs because the parasites alter the red blood cell's membrane, making the cells more likely to clump together against the blood vessel walls. If this blockage cuts off the blood that carries oxygen in vessels nourishing the brain, coma results.

The problem with this theory is that survivors of cerebral malaria usually emerge from their comas without the brain damage typical of oxygen starvation. Other researchers, including Ian Clark of the Australian National University in Canberra, put forth another scenario, in which nitric oxide plays a starring role.

This view hinges on the discovery that *P. falciparum* infection provokes white blood cells to produce tumor necrosis factor, a protein that stimulates the release of nitric oxide. Clark contends that excess nitric oxide disrupts the normal flow of nerve signals within the brain, thereby causing coma or death.

In keeping with this theory, Weinberg