Biology

Rattlesnakes feel the final bite

Movies of rattlesnake strikes don't leave much time for popcorn. The snake can zap a lethal dose of venom into its scurrying victim, then jerk its head away in about half a second.

Yet in these shortest of movie shorts, the snakes display impressive accuracy, says Kenneth V. Kardong of Washington State University in Pullman. He and Vincent L. Bels of the Agronomic Center of Applied Research in Hainaut, Belgium, analyzed the strike motion in movies of northern Pacific rattlesnakes biting mice. The results appear in the Feb. 18 JOURNAL OF EXPERIMENTAL BIOLOGY.

In 20 out of 21 bites, the snakes landed a lethal strike on the first try. "They're really very good," Kardong says.

The snakes' sense of touch may play a role in that accuracy by guiding final adjustments, he suggests. Kardong did not see any midair, midstrike swerves that might have corrected the rattlers' course, although some other snakes do alter the trajectory of their heads as they strike. What he saw instead were shifts in fang position for optimal venom injection after the snake touched its victim.

The use of touch intrigued another maker of bite movies, William K. Hayes of Loma Linda (Calif.) University. "I don't think we've appreciated the importance of tactile stimuli," he says. —*S.M.*

A northern Pacific rattlesnake's quicker strikes last about 0.07 second, but injecting a full dose of venom requires almost a half second.

When the little guys win one

Underdogs of all species, take heart.

Ornithologists in Australia have documented that small birds win benefits by taking on a predator 20 times their size.

A variety of bird species will mob a predator such as a hawk, harassing it by raising a ruckus and repeatedly performing displays, swooping, or striking at the victim. There may be safety in numbers, but many of these so-called mobs number only two or three birds, says Chris R. Pavey, formerly of the University of Queensland in Brisbane.

Most studies of the benefits of mobbing have focused on birds that pick on somebody near their own size, he says. He's more interested in the likes of the grey butcherbird, all 75 grams of it, which mobs a species called the powerful owl. These hawkish-looking predators can weigh up to 1,700 g.

To a big owl, small birds pose no more danger than flying hors d'oeuvres. Yet pestering an owl at its daytime roost pays off for the smaller birds, say Pavey and Queensland colleague Anita K. Smyth. In 20 percent of mob scenes, the owl left, the researchers report in the February ANIMAL BEHAVIOUR.

This harassment may explain why owls so often roost in the rain forest, Pavey speculates. Only one of the seven mobbing species spends much time there, and owls snoozing in the thick vegetation suffer less than a third of the disturbances they endure elsewhere. Rain forest covers only 12 percent of the study area, yet the owls roost there about half the time.

By checking pellets regurgitated by owls, Pavey and Smyth estimated that nonmobbing birds were nine times more likely to be eaten as mobbing species. Although not convinced by those data, Keith L. Bildstein of Hawk Mountain Sanctuary in Kempton, Pa., is nevertheless impressed by the Australian mobsters' power. "It actually shapes the behavior of the predator," he says. —*S.M.*

Biomedicine

Heart benefits from sneaky calcium ions

Consider them the sparks of life—brief releases of calcium ions inside heart muscle cells that ultimately trigger the cells to start a contraction of the organ, a heartbeat. These intracellular sparks occur when a small rush of calcium ions enters the cells through channels in their outer membrane.

Scientists have now found to their surprise that this initial wave of calcium may not enter just through channels specifically designed for the ion. A study suggests that calcium ions can also sneak through the channels previously thought to be reserved for sodium ions, a finding that also challenges beliefs about how certain heart drugs work.

"People are stunned," says W.J. Lederer of the University of Maryland in Baltimore. "They sort of believe it because the evidence is compelling, but they are sort of worried because it shakes up some fundamental assumptions."

In the Feb. 13 SCIENCE, Lederer and his colleagues describe experiments that establish the existence of the unlikely calcium pathway. Scientists had previously noticed that even if they blocked all known calcium channels, they could still detect calcium releases in heart muscle cells and could observe the muscle contract. So Lederer's group decided to block the cells' sodium channels with the puffer fish toxin tetrodoxin. The calcium releases vanished.

The investigators then examined steroids similar to the drug digitalis, which helps people with heart failure by strengthening the contractions of the heart. These steroids were thought to work primarily by binding and inhibiting a cell structure called the sodium pump, which indirectly increases the amount of calcium inside the cell. Yet Lederer's group found that the steroids also convert the sodium channels into openings that allow calcium to pass through.

"This action may help these drugs regulate contraction under. . . conditions like heart failure," writes Dottie Hanck of the University of Chicago in an accompanying commentary.

Lederer notes that identifying this unexpected mode of action may help investigators design improved heart drugs. —*J.T.*

Stopping coughs . . . and cancer?

Noscapine, a cough suppressant long used in South Africa, Japan, and Sweden, may someday help combat a much more serious ailment—cancer. Investigators have unexpectedly found that this drug can induce dividing cells to commit suicide and can shrink tumors growing in mice.

"We're the first to find that this agent can be used as a cancer drug. A lot of companies are contacting us," says Keqiang Ye of the Emory University School of Medicine in Atlanta.

Ye and his colleagues, who describe their work in the Feb. 17 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES, believe that noscapine exerts its anticancer effects by binding tubulin, a protein that forms the cellular filaments called microtubules. Several other known cancer drugs, such as taxol, function similarly. While some of these drugs inhibit microtubule assembly and others stabilize the filaments, they all apparently interfere with a cell's ability to divide.

In the case of noscapine, this interference causes the cell, which is fruitlessly trying to divide, to kill itself. When the investigators gave noscapine to mice with solid tumors, including animals implanted with human breast or bladder tumors, the drug dramatically shrank the cancers. For example, a 3-week regimen of noscapine reduced the size of human breast tumors by 80 percent. Some tumors were eliminated.

Noscapine may be a particularly attractive cancer drug, says Ye, because it can be taken orally and has a proven safety record. Many of the other cancer drugs that bind tubulin must be injected and produce severe side effects, he notes. —*J.T.*