Biology

Caterpillar bodies not built for speed

U.S. Dept. of Agriculture

Gypsy moth caterpillars

A caterpillar's soft, squishy body doesn't just make a juicy meal for birds; it also limits the caterpillar's crawling to an inefficient creep.

That's the conclusion of ecological physiologist Timothy M. Casey, who measured the oxygen consumption of gypsy moth caterpillars as they "sprinted" to keep up with a tiny treadmill. From these data, he calculated how efficiently the caterpillars convert energy into motion – an analysis similar to figuring the fuel economy of a car. The results: Caterpillars are gas guzzlers, and remarkably slow ones at that.

In the April 5 SCIENCE, Casey reports that these caterpillars need about 4.5 times more energy to travel a given distance than do animals with solid skeletons. At their speediest, they wriggle along at about 100 yards an hour.

Casey, of Cook College at Rutgers University in New Brunswick, N.J., says his findings clarify an observation made in a number of previous energy-efficiency studies: Regardless of body shape or size, creatures as varied as humans, rats and cockroaches use similar amounts of energy to move a given weight a given distance. Those animals differ from caterpillars in one important respect, however: They all possess a solid internal or external skeleton, whereas caterpillars rely entirely on fluid-driven locomotion.

The inefficiency of this "hydraulic skeleton" may explain the caterpillar's exceptionally uneconomical movement, says Casey, who describes the system as "a tube within a tube." When an outer muscle layer contracts, the fluid between the two tubes forces each body segment forward.

Studies of how much energy gypsy moth caterpillars spend on different activities may help researchers assess the insects' "overall energy expenditure," he adds. This provides an estimate of how much food (i.e., leaves) the caterpillar needs to eat, which in turn helps scientists gauge the best way to control the proliferation of these voracious pests.

Insects bugged by 'jumping genes'

Scientists have discovered that two virus-like agents proliferate in the genetic material of a variety of insect species, raising the prospect of designing new strategies for controlling such pests as cockroaches, gypsy moths and mosquitoes.

The agents are retrotransposons – DNA fragments that insert copies of themselves into an organism's chromosomes, disrupting any genes into which they happen to land, says Thomas H. Eickbush, an insect molecular biologist at the University of Rochester in New York. In the April 15 PROCEEDINGS of the NATIONAL ACADEMY of SCIENCES, he and his colleagues report finding two of these fragments, dubbed R1 and R2, in "virtually every insect" they examined.

Because retrotransposons are inherited, scientists could use R1 and R2 to deliver damaging genes into an insect population, Eickbush suggests. The introduced genes might persist indefinitely, providing a means of pest control, he says.

Similar attempts to use other "jumping genes" have proved unsuccessful because researchers have been unable to coax these agents into insects other than fruit flies. However, Eickbush has already identified R1 and R2 in 43 different insect species. He says he plans to try out this novel bug-battling strategy in fruit flies – the insect version of the laboratory rat – before moving on to more destructive pests.

Genetics

Narcoleptics may have an immune disease

They sleep fitfully at night, drop off unexpectedly during the day, and sometimes fall slumbering face first into their plates at mealtimes. The exact cause of the disorder, called narcolepsy, has eluded researchers so far, but a California group now suggests the problem may involve a defect in one of the genes that control the immune system.

A team led by Emmanuel Mignot of Stanford University School of Medicine's Sleep Disorders Research Center has uncovered evidence in dogs that narcolepsy arises from a mutation in a gene similar to one that spurs the production of antibodies in humans. They report their results in the April 15 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES.

The immune system has been implicated before in narcolepsy. In 1983, a Japanese study linked the disease with a particular class of cell-surface proteins that enable the immune system to distinguish the body's own tissue from foreign invaders. But subsequent studies found that only 90 percent of narcoleptics carried the protein, suggesting the disease results from more than one factor.

In the new study, the Stanford researchers examined DNA from two inbred families of Dobermans and Labradors, roughly half of which had narcolepsy. Two-thirds of the narcoleptic dogs had pieces of DNA that matched human DNA known to control the production of antibodies.

Mignot suggests that the dogs' DNA could be part of a mutant antibody gene. Antibodies made by such a gene, he theorizes, could contribute to narcolepsy by interfering with unidentified cells or molecules necessary for healthy sleep.

Narcolepsy researcher Shiva M. Singh, from the University of Western Ontario in London, Ontario, believes Mignot's group could be on the right track. "There is probably more than one genetic cause for narcolepsy," he says.

Creutzfeldt-Jakob: Culinary or genetic?

The cannibalistic culinary habits of the Fore people of New Guinea – and particularly their taste for human brains – was discovered in the early 1970s to underlie their high incidence of kuru, a transmissible neurodegenerative disorder related to Creutzfeldt-Jakob disease. When Libyan Jews were recently found to have a frequency of Creutzfeldt-Jakob disease 100 times that of most other populations, physicians wrote it up to their fondness for lightly cooked sheep brains.

Now, a team of researchers from California, Israel and Italy has partially exonerated the cerebral delicacy as the cause of the Libyan Jews' high rates of the disease. They report in the April 18 NEW ENGLAND JOURNAL OF MEDICINE that 11 Libyan Jews with Creutzfeldt-Jakob disease have an identical mutation, which points to a genetic cause for their disorder.

The work was led by Stanley B. Prusiner of the University of California, San Francisco, the neurologist who first proposed that the illness was caused by an infectious agent with genes made of protein instead of DNA. In a 1985 study of healthy people, Prusiner and others identified a gene whose mutant produces such an agent, dubbed a "prion." Prusiner theorized that Creutzfeldt-Jakob results when a mutation in this gene activates the production of prions, which can then infect others. In a controversial 1989 report, he announced finding evidence of the prion in the brain tissue of people who died of Creutzfeldt-Jakob or kuru and in sheep with a similar disease called scrapie.

In the new study, Prusiner's group found that all of the Libyan Jews with Creutzfeldt-Jakob disease had an identical mutation in their prion proteins, which was lacking in 37 healthy Libyan Jews and in a Moroccan Jew with the disease. "These results dispel the widely held belief that [the Libyan Jews'] disease is due to cultural or culinary habits," the researchers write.