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How gypsy moth is kept high to die

Virus gene found to control caterpillar behaviour.

Lee Sweetlove

Caterpillars infected with baculovirus climb to the tops of trees, where they melt and drip the virus onto foliage below.
Michael Grove

The genetic mechanism behind a virus's ability to change the behaviour of a caterpillar is revealed today in a study published in Science1.

European gypsy moth caterpillars (Lymantria dispar) have an arduous existence, scaling trees at night to feed on leaves then trekking all the way back down to the safety of the ground during the day. But a baculovirus can dramatically change this behaviour — a caterpillar in the final stages of infection no longer returns to the ground.

Death is then swift — the virus unleashes a battery of enzymes that digest the caterpillar from the inside out, releasing a rain of new viral particles from the liquefying animal. Because this happens up high, the virus is dispersed widely.

But how a simple virus could control caterpillar behaviour was a mystery. Kelli Hoover of Pennsylvania State University in University Park, who led the research, had a hunch that a gene in the virus called egt could be responsible.

The gene encodes an enzyme known to inactivate the caterpillar hormone 20-hydroxyecdysone, which triggers moulting.

Because the caterpillars normally descend the tree to moult, Hoover figured that their tendency to remain high up when infected by the virus might be because the moulting hormone was being blocked. Her team tested this idea by disrupting the egt gene in the virus. Caterpillars infected with the modified virus returned to the bottom of a tall experimental bottle to die, whereas those infected with the normal, egt-containing, virus died high up the side of the bottle.

The work provides a neat explanation for how a viral gene can affect caterpillar behaviour. "Interfering with the hormonal system is the most elegant way to influence complex traits such as behaviour," says Hoover.

Parasitologist Glenn McConkey from the University of Leeds, UK, agrees. "For a parasite gene to affect something as complex as host behaviour, it must be modifying something neural and that means either hormones or neurotransmission," he says.

The extended phenotype

The baculovirus provides a graphic illustration of the concept of an 'extended phenotype', first proposed by Richard Dawkins in his 1982 book of the same name. Dawkins argues that the definition of phenotype should be extended to include all effects that a gene has on its environment, inside or outside the body of the organism it comes from.

There are several other examples of parasites that have extended phenotypes, including fungi that turn ants into 'zombies' clamped onto leaves in the ideal position for fungal spore dispersal2, and a Toxoplasma parasite that causes rats to lose their fear of cats, making the rats more likely to be eaten, thereby enhancing transmission of the parasite to the cat, its main host3.

Until now, however, the final piece of the extended phenotype concept — a definitive link to a specific gene — had been missing. Co-author David Hughes, also from Pennsylvania State University, has also worked on the zombie ants and is in no doubt as to the importance of the new work. "To me this moves the concept of the extended phenotype forward in quite a dramatic fashion," he says. "This is the first empirical evidence that a gene in the body of one organism can have a direct effect on another organism."

McConkey, however, is more cautious, explaining that coincidental behavioural changes need to be ruled out. He uses the example of malaria to illustrate his point. "If you are ill with malaria you tend to lie down a lot and this makes you more likely to get bitten by a mosquito. This apparent benefit to the malaria parasite is entirely coincidental."

References:

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