Virus fans the flames of desire in infected crickets

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Love may be a battlefield, but most wouldn't expect the fighters to be a parasitic virus and its cricket host. Just like a common cold changes our behavior, sick crickets typically lose interest in everyday activities. But when Dr. Shelley Adamo of Dalhousie University found her cricket colony decimated by a pathogen, she was shocked that the dying insects didn't act sick. Not only had the infected crickets lost their usual starvation response, but they also continued to mate. A lot. How were the pathogen and the exuberant amorous behavior in the sick crickets connected?

Dr. Adamo soon realized that she had stumbled onto a classic parasite-host struggle. As a neurobiologist, Dr. Adamo is especially interested in parasites that control the behavior of their hosts, known as parasite manipulators. "Parasite manipulators have something to teach us about how brains work," says Adamo, as they are able to exert fine-grained control over their hosts' brains in ways that human neurobiologists can only dream of. Parasites use hormones, neurotransmitters and other proteins to disconnect the host brain and the immune system, altering host behaviors to increase the survival and reproduction of the parasite. By studying the various avenues parasites use to control their hosts, neurobiologists hope to gain new understanding of how nervous systems produce behavior.

Dr. Adamo set out to determine the mechanisms by which the virus changed cricket behavior. As presented recently at the Society for Integrative and Comparative Biology annual conference in Austin, Adamo and colleagues found that the virus invaded and reproduced in the crickets' fat body, a vital organ that controls insect energy reserves.

By attacking this single organ, the virus impacted the host in multiple ways. In sick crickets, almost all of the hosts' resources were channeled into producing proteins for the virus, and the fat body became engorged with viral particles. As a result, infected crickets showed signs of sterility, as females produced few if any eggs, and male sperm showed low or no mobility. But attacking the fat body doesn't only help the virus gain host resources, so it seems that sterility is just collateral damage in the fight between the virus and the crickets.

Besides its role in making fats, the fat body also creates proteins that contribute to the crickets' immune responses. Dr. Adamo and colleagues found that infected crickets had overall lower levels of proteins in their blood, including a key component of the crickets' immune response. By hijacking the cricket fat body, the virus established itself more easily by knocking out part of the cricket immune response, a necessary tactic for any parasite to avoid being detected and destroyed by their host.

And what about the ardent mating that Dr. Adamo first observed in her sick crickets? The virus was the culprit, acting like other parasitic manipulators to change cricket mating behavior for its own gain. While it was unusual to find that sick female crickets acted just like healthy females, Adamo's biggest surprise was in sick male crickets. They actually outperformed healthy control males in mating, courting females more quickly and enthusiastically. Because the virus can be contracted through sexual contact, more matings means more opportunities for the virus to spread.

While Dr. Adamo was happy that she could turn the lemon of her dying cricket colony into an interesting study of this parasitic virus manipulator, she doesn't have any future plans to study this virus. Instead, she is moving on to other larger and more manageable parasite manipulators of crickets. By using the smaller and less complex cricket brain as a model, Dr. Adamo hopes to uncover more broad patterns of nervous system function in both immune responses and, of course, behavior.

Provided by Society for Integrative & Comparative Biology