

Nicotinoid and fungal disease team up to break down termites' tough defenses

May 20 2015, by Natalie Van Hoose

Purdue University research shows that a small amount of nicotinoid pesticide substantially weakens termites' ability to fight off fungal diseases, a finding that could lead to more effective methods of pest control.

The study also provides clues into termites' robust defense systems and how nicotinoids affect [social insects](#).

A team led by Michael Scharf, the O.W. Rollins/Orkin Chair and professor of entomology, found that a sublethal dose of imidacloprid knocked out key microbes in the termite gut and suppressed the social hygiene habits that help keep a termite colony healthy. Their defenses weakened, the termites became vulnerable to a fungal pathogen that normally poses little threat. The combination of pesticide and pathogen wiped out laboratory colonies in seven days.

"A termite colony can tolerate this dose of imidacloprid and fungal pathogen independently, but put them together, and they really have deleterious effects," Scharf said. "Understanding how to cripple termite defenses could lead us to new, safer control technologies."

Termites rarely get sick, despite living in moist, underground environments and in close contact with thousands of fellow colony members - conditions that are ideal for disease development.

While termites contain the disease defense genes common among all

insects, they also have unique, non-genetic ways of protecting themselves from pathogenic bacteria and fungi, Scharf said.

Termites build up "social immunity" by grooming pathogens off of one another and transfer disease resistance throughout the colony by feeding on each other's secretions, said study co-author Drion Boucias, a professor of insect pathology at the University of Florida who has been researching termite immunity and response to disease for several decades.

"Social cleaning and grooming are critical," he said. "A solitary termite is susceptible to anything."

Termites also protect themselves by cultivating mutually beneficial relationships with microorganisms. The termite gut houses what Boucias called a "microbial garden" - a rich community of thousands of beneficial bacteria and protists, simple microorganisms whose symbiotic relationship coevolved with termites over millions of years. These microbes allow termites to digest cellulose, the tough material that gives plants their ability to stand upright. But they also appear to play an important role in disease defense.

Previous research suggests that some of these protists produce an enzyme that fatally punctures the cell wall of pathogenic invaders.

When Scharf and Boucias's team treated [termite colonies](#) with a small dose of imidacloprid, the protists began to die. The pesticide also had a druglike effect on termites, suppressing the grooming behaviors necessary to keep colony members from being infected with a [fungal disease](#).

Applying a sublethal amount of a [fungal pathogen](#) quickly destroyed the imidacloprid-treated colonies. The pathogen penetrated the termites'

outer cuticle and dissolved their muscles and organs.

"The termites became little fuzzy piles of mush," Scharf said. "We don't typically see this in colonies in the wild unless they are severely stressed."

The researchers studied the termites' gut metatranscriptome - all termite and microbe genes that are being expressed at a given moment - to measure the decline of the [gut microbes](#) and better understand which genes are involved in termite defense.

Unexpectedly, the pesticide and fungus did not trigger the "stereotypical" immunity genes that they do in other insect species such as bees, Scharf said. The finding could indicate that termites rely almost exclusively on their gut microbes and social immunity to protect their health.

Future control measures may target these defenses, opening the door for [termites](#)' natural enemies to finish the job, Boucias said.

More information: The paper was published in *PloS One* and is available at [journals.plos.org/plosone/arti ... journal.pone.0123391](https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0123391)

Provided by Purdue University

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