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PATHOGENESIS

Sacre bleu — un assassin de nématode!

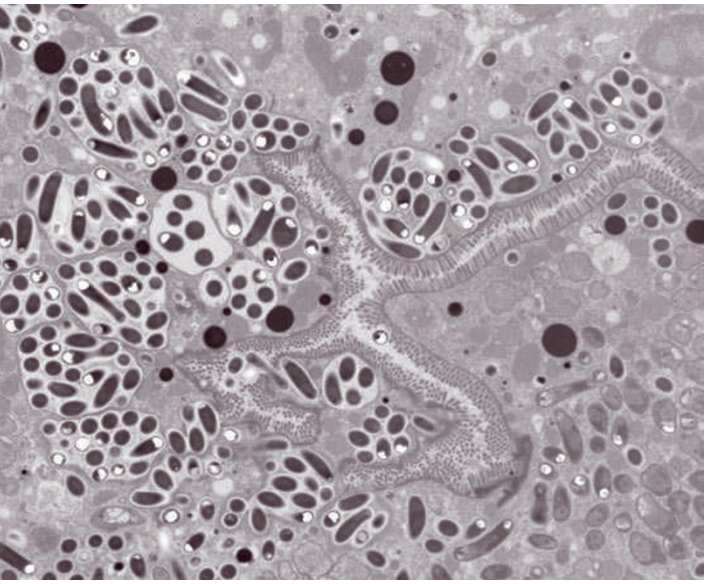
In recent years, the nematode worm *Caenorhabditis elegans* has become a model for studying innate immunity and intestinal infection. In an article published in PloS Biology, Ausubel, Troemel and colleagues now identify a novel genus of microsporidia that infects intestinal cells of wild-caught *C. elegans* worms.

As *C. elegans* lacks professional immune cells, it largely relies on innate immunity to fight pathogen infection. In the laboratory, infection with a number of pathogens has been shown to trigger innate immune signalling systems. However, no pathogen of wild-caught *C. elegans* worms has ever been identified. Ausubel and colleagues isolated a wild-caught *C. elegans* strain from a compost pit in France that was found to harbour small, rod-shaped microorganisms in its intestinal cells. Although the infection was not transmitted from infected hermaphroditic parents to their progeny, incubation of infected worms with uninfected worms led to horizontal transmission at a high frequency and all infected worms ultimately died prematurely. PCR analysis of rDNA identified sequences that were related to *Ovavesicula popilliae*, a beetle-infecting microsporidian species. The microsporidia are a group of intracellular eukaryotic parasites that can survive outside of cells by producing spores. Differences in rDNA sequences and spore morphology between the *C. elegans* microsporidian and *O. popilliae* led the authors to name the new species of microsporidian *Nematocida parisii*, meaning nematode killer from Paris.

Using transmission electron microscopy, the authors followed the different stages of *N. parisii* infection. Intestinal epithelial cells of *C. elegans* have a brush border on the apical face that is decorated with cytoskeletal structures known as microvilli, each of which is anchored at the base by a conserved structure known as the terminal web. After infection with *N. parisii*, grooved regions appeared on the intestinal cells. The grooves contained irregular multinucleate meronts (cell wall-deficient microsporidian cells) that were often attached to intracellular vesicles. Later in infection, the meronts differentiated into spores. Interestingly, in worms infected with *N. parisii* spores, damage to the cytoskeletal intermediate filaments and terminal web was observed, hinting at a possible exit strategy for the pathogen. Mutations in immunity signalling pathways, which had previously been shown to alter resistance to bacterial and fungal infection, had little effect on *N. parisii* infection, suggesting that the worm makes distinct host responses to this natural pathogen. Finally, the authors isolated related microsporidia from other wild-caught *C. elegans* strains from France, Portugal and India, which indicates that microsporidian infections of worms are widespread.

The discovery of a natural pathogen for *C. elegans* will enhance the use of this model for studying infection and immunity and could lead to novel strategies for biocontrol of the parasitic nematode, a common agricultural pest.

Andrew Jermy



Small rod-shaped spores of *Nematocida parisii* in the intestines of *Caenorhabditis elegans* worms. Figure reproduced from Troemel, E. R. *et al.* Microsporidia are natural intracellular parasites of the nematode *Caenorhabditis elegans*. *PLoS Biol.* **6**, e309 (2008).

ORIGINAL RESEARCH PAPER Troemel, E. R. *et al.* Microsporidia are natural intracellular parasites of the nematode *Caenorhabditis elegans*. *PLoS Biol.* **6**, e309 (2008)