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**Genomic mechanisms accounting for the adaptation to parasitism in nematode-trapping fungi**

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Nematode-trapping fungi form unique infection structures, traps, to capture and kill free-living nematodes. The traps have evolved differently along several lineages and include adhesive traps (knobs, nets or branches) and constricting rings. We show, by genome sequencing of the knob-forming species *Monacrosporium haptotylum* and comparison with the net-forming species *Arthrobotrys oligospora*, that two genomic mechanisms are likely to have been important for the adaptation to parasitism in these fungi. Firstly, the expansion of protein domain families and the large number of species-specific genes indicated that gene duplication followed by functional diversification had a major role in the evolution of the nematode-trapping fungi. Gene expression indicated that many of these genes are important for pathogenicity. Secondly, gene expression of orthologs between the two fungi during infection indicated that differential regulation was an important mechanism for the evolution of parasitism in nematode-trapping fungi. Many of the highly expressed and highly upregulated *M. haptotylum* transcripts during the early stages of nematode infection were species-specific and encoded small secreted proteins (SSPs) that were affected by repeat-induced point mutations (RIP). The high expression and rapid divergence of SSPs indicate a striking similarity in the infection mechanisms of nematode-trapping fungi and plant and insect pathogens from the crown groups of the filamentous ascomycetes (Pezizomycotina). The patterns of gene family expansions in the nematode-trapping fungi were more similar to plant pathogens than to insect and animal pathogens. The RIP activity in the Orbiliomycetes suggested that this mechanism was present early in the evolution of the filamentous ascomycetes.