



Review Article

Entomopathogenic and plant pathogenic nematodes as opposing forces in agriculture



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ABSTRACT

Plant-parasitic nematodes are responsible for substantial damages within the agriculture industry every year, which is a challenge that has thus far gone largely unimpeded. Chemical nematicides have been employed with varying degrees of success, but their implementation can be cumbersome, and furthermore they could potentially be neutralising an otherwise positive effect from the entomopathogenic nematodes that coexist with plant-parasitic nematodes in soil environments and provide protection for plants against insect pests. Recent research has explored the potential of employing entomopathogenic nematodes to protect plants from plant-parasitic nematodes, while providing their standard degree of protection against insects. The interactions involved are highly complex, due to both the three-organism system and the assortment of variables present in a soil environment, but a strong collection of evidence has accumulated regarding the suppressive capacity of certain entomopathogenic nematodes and their mutualistic bacteria, in the context of limiting the infectivity of plant-parasitic nematodes. Specific factors produced by certain entomopathogenic nematode complexes during the process of insect infection appear to have a selectively nematicidal, or at least repellent, effect on plant-parasitic nematodes. Using this information, an opportunity has formed to adapt this relationship to large-scale, field conditions and potentially relieve the agricultural industry of one of its most substantial burdens.

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1. Introduction

Nematodes are a fairly vast phylum, and many of the species in the group also happen to be parasitic, opportunistically inhabiting a range of hosts that include plants, insects and animals (Dillman and Sternberg, 2012; L'Ollivier and Piarroux, 2013; Quist et al., 2015). Naturally, depending on the type of organism a nematode has infected and the context in which the infection is taking place, a nematode's success in terms of survival and parasitism can either be in line with, or sharply opposed to, human health or economic interests. Research into the interactions between the host immune system and nematode virulence mechanisms have therefore garnered considerable interest and support in the hope that these interactions can be mediated beneficially (Castillo et al., 2011; Babu and Nutman, 2014; Govere and Smant, 2014).

In particular, nematodes can have a large impact on agriculture through their effect on populations of insects and plants alike. Those nematodes that are entomopathogenic, or insect parasitic, can generally be thought of as advantageous, and the *Heterorhabditis*

and *Steinernema* genera of entomopathogenic nematodes (EPNs) have been employed specifically and intentionally as biocontrol agents for insect pests (Ehlers, 2001; Ffrench-Constant et al., 2007). Plant-infectious nematodes, on the other hand, account for approximately 5% of crop yield loss by limiting root growth, plant size and photosynthetic rate (Gheysen and Mitchum, 2011; Kyndt et al., 2013). One of the confounding issues with this situation then is that because nematodes present in a plant's environment may be having opposite effects, an unspecific nematicidal treatment is eliminated as a viable strategy for crop protection, especially if EPNs are maintaining a population of insect pests below a harmful threshold, which may not be immediately apparent. These two kinds of nematodes may also have little competitive effect on each other, as efforts to suppress plant-parasitic *Meloidogyne paratytla* nematodes with *Steinernema feltiae* produced inconsistent and marginal results (Shapiro-Ilan et al., 2006), although this may be a species-dependent effect, as other studies using different pairings of EPNs and plant-parasitic nematodes (PPNs) have shown more affirmative findings (Molina et al., 2007).

With this dynamic in place, guiding nematode interactions to a desired result may require a significant amount of tact and subtlety that will rely on a thorough knowledge of plant and insect

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immunity as it relates to nematode infection. It is important to consider as well that these two forms of immune response will have strong and fundamental differences. In general, insect innate immunity consists of mechanisms that lead to the production of antimicrobial peptides (AMPs) and reactive oxygen species (ROS), and cellular functions involving phagocytosis, encapsulation and nodulation (Welchman et al., 2009; Viljakainen, 2015), but plants, lacking a cellular response, will be incapable of responding to nematodes with functions such as encapsulation, which is the most common insect immune response to a metazoan invader (Jones and Dang, 2006; Muthamilarasan and Prasad, 2013; Honti et al., 2014; Vlisidou and Wood, 2015). A further examination of these two immune systems and their respective responses to nematode parasites will outline the primary mechanisms of insect and plant resistance.

2. The insect immune response to EPNs

A nematode infection of an insect host begins when a nematode of the infectious juvenile (IJ) stage attaches to the cuticle of the insect, penetrates through the various natural openings, such as the spiracles or mouth, and establishes itself in the hemolymph after advancing into the body cavity (Griffin, 2012). Once established, the nematode will release its mutualistic bacteria into the host hemolymph, *Photorhabdus* bacteria in *Heterorhabditis* nematodes and *Xenorhabdus* bacteria in *Steinernema* nematodes, either through regurgitation or esophageal pumping of the bacteria down through the intestine and out of the anus (Goodrich-Blair, 2007; Waterfield et al., 2009). These bacteria then go on to release toxic and immunosuppressive compounds, eventually leading to the death of the host by septicemia (Ffrench-Constant et al., 2007; Herbert and Goodrich-Blair, 2007). The release of nematode mutualistic bacteria does not occur immediately, however, and is instead delayed, by 30 min in the case of *Heterorhabditis*, and 4–6 h for *Steinernema* (Li et al., 2007). This means that the host insect has a window, granted of a variable timeframe depending on the species, in which it may neutralise the parasite before being forced to compensate for the additional challenge of the bacterial infection. In a general way, nematode avirulence is primarily achieved by initiating hemolymph clotting, activating a melanisation reaction, and encapsulating the nematode in layers of hemocytes. Clot formation is based on the activity of soluble factors in the hemolymph, including transglutaminase, which will bind foreign bodies, including *Photorhabdus* (in the case of transglutaminase), and form microclots that can be incorporated into networks of fibers produced by hemoclectin and triggerin that will further isolate the pathogen (Hyrsl et al., 2011; Toubarro et al., 2013). The melanisation reaction, which is technically part of the humoral response, although it functions in close association with the cellular response, is comprised of the conversion of the inactive precursor prophenoloxidase to active phenoloxidase (Eleftherianos and Revenis, 2011), which generates the indole groups used to form melanin that then binds the nematode and supports the destruction of the parasite with ROS (Castillo et al., 2011). The cellular response, although thus far underrepresented, then plays perhaps the most crucial role by rapidly encapsulating the pathogen in compacted layers of hemocytes, which is a process that can be initiated within minutes of exposure, and potentially prevents the release of bacteria into the hemolymph (Satyavathi et al., 2014).

The release of bacteria by nematodes is clearly a challenge to the host that, once initiated, is difficult to overcome, as each nematode can release 50–200 bacteria directly into the hemolymph (Goodrich-Blair, 2007; Wu et al., 2014). The products of these

bacteria can undermine much of the immune system, both cellular and humoral, by releasing toxic components that are capable of damaging hemocytes, and enzymes such as the RTX-like metalloprotease of *Photorhabdus* that can cleave hemolymph proteins involved in regulating host immune effector genes (Bowen et al., 2003; Rodou et al., 2010; Vlisidou et al., 2012). The success of the insect immune system in overcoming an infection by nematode parasites is therefore based largely on its ability to prevent the release of these bacteria and their immunosuppressive products, and indeed, a correlation between survival and the degree of encapsulation has been demonstrated (Li et al., 2007; Eleftherianos et al., 2010). A number of variables in the immune response, as viewed between different species of insects, reflect this point through their ability to influence host survival (Castillo et al., 2011). One such variable is the starting point of encapsulation. As mentioned previously, nematodes eject their mutualistic bacteria from the mouth and anus (Snyder et al., 2007), and therefore it would follow that the most effective method of encapsulation would be to cover these openings first in order to prevent the release of bacteria. Concordantly, encapsulation of *Heterorhabditis* by *Manduca sexta* hemocytes is initiated at the head and tail of the nematode (Li et al., 2007). In the Colorado potato beetle, *Leptinotarsa decemlineata*, however, encapsulation is initiated in the middle of the nematode, near the esophageal region, possibly in response to secretory-excretory pore exudates (Ebrahimi et al., 2011). This mechanism could represent a disadvantage for the insect, as the nematode could potentially eject bacteria during the encapsulation process and well before the process is complete. The time frame of the encapsulation process measured against the timing of bacterial release is also important, as both are represented by a spectrum of a fairly wide range. Encapsulation may begin within minutes of exposure, but completion of the task involves multiple stages, each with their own timing (Stanley et al., 2009; Jiang et al., 2010). Depending on the species involved in the interaction, the formation of multiple layers of hemocytes can take 45 min – 2 h, the complete encapsulation of the nematode 2–4 h, and partial melanisation 16–24 h, as was observed with combinations of *Heterorhabditis bacteriophora*, *L. decemlineata*, and *Galleria mellonella* (Ebrahimi et al., 2011). These differences could be crucial in determining the survival of the insect based on the delay of bacterial release by the nematode, which as mentioned previously can be as brief as 30 min or as long as 4–6 h.

The insect immune response has been described in some detail, but one factor that has not yet been discussed is that nematode parasites are also capable of evading detection by the immune system, in which case the mechanics of a cellular response would be largely irrelevant (Brivio et al., 2005; Castillo et al., 2011). Lytic surface coat proteins, hydrophobic exudates, and lipopolysaccharide-like binding proteins produced by nematodes can all facilitate the parasite's evasion of encapsulation (Li et al., 2009; Brivio et al., 2010; Mastore et al., 2014). Overall, the interaction can then be characterised as a highly complex interplay between the genotypes of the insect, the nematode and its mutualistic bacteria, which although perhaps difficult to predict, does provide a number of potential targets for control that could be beneficial to agriculture if applied appropriately. In the context of eliminating insect pests, strains could be developed that produce the proteins necessary for evasion, or nematodes could be generated that have significantly decreased delays in the ejection of their bacterial endosymbionts. Future research may do well to investigate the factors that generate differences in ejection timing, as nematodes that can overwhelm the insect immune system before being encapsulated would likely serve as much more efficient biocontrol agents.

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