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War of the worms: how plants fight underground attacks

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Sedentary plant-parasitic nematodes (PPNs) establish specialized feeding cells within roots to maintain long-term relationships with their hosts. However, feeding cells degenerate prematurely in plants that harbor resistance (*R*) genes against these parasites reducing their life span and ability to reproduce. Recognition of the nematode, mediated directly or indirectly by plant R proteins, occurs via nematode secreted effectors and evokes a resistance response, which is referred to as effector-triggered immunity (ETI). Recent breakthroughs in nematode effector biology shed new light on key players mediating ETI and have identified those involved in plant defense suppression as novel targets for engineering resistance in transgenic plants. Advances in plant genetics and genomics has facilitated the discovery of *R* genes to nematodes. Nevertheless, underlying resistance mechanisms remain poorly understood and are confounded by recently identified *R* genes that do not fit previously proposed paradigms. Thus, there is still much to be learned about how plants fight off underground attacks from PPNs. In coming years, we can expect breakthroughs in our understanding of the nature and mechanisms of plant resistance and nematode virulence as we explore these novel *R* genes.

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Introduction

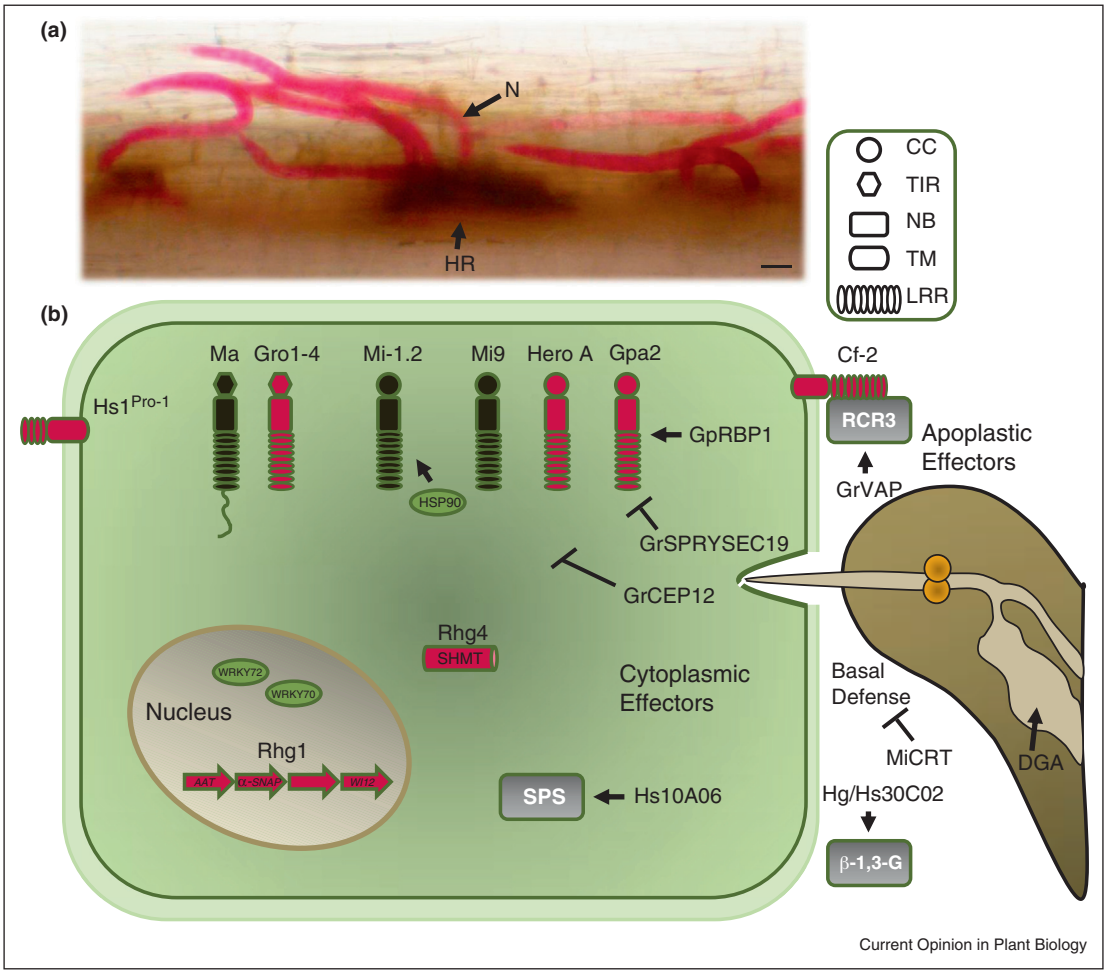
PPNs cause more than $100 billion in worldwide crop losses [1]. Current control measures rely heavily on natural resistance in plant species. Only a handful of nematode *R* genes have been identified in plants and even less is known about the plant resistance mechanisms to these agronomically important pests. Most work has focused on two groups of PPNs, the cyst (*Heterodera* and *Globodera* spp.) and root-knot nematodes (RKN, *Meloidogyne* spp.) due to their major worldwide economic impact. The success of these sedentary endoparasites as pathogens stems from their ability to manipulate fundamental aspects of plant biology. During the infection process, these nematodes use stylet-secreted effector proteins to induce the formation of unique and specialized plant cell structures within host roots to serve as metabolically active nutrient sinks (i.e. feeding cell) (reviewed in [2]). Successful formation of the feeding cell coupled with subsequent growth and development of the nematode is described as a compatible interaction. In contrast, an incompatible interaction occurs when feeding cells are not well formed resulting in cessation of nematode development. The resistance response, which is localized to the feeding cells or immediately surrounding cells, has been described as a rapid necrotic reaction similar to the hypersensitive cell death response (HR) that occurs in response to microbial pathogens, and leads to feeding cell collapse (Figure 1a) [3,4••]. In this review, we focus on the latest breakthroughs in our understanding of plant and nematode genes underlying incompatible interactions between sedentary endoparasites and their hosts.

Players in natural plant resistance to nematodes

Nematode resistance proteins identified to date are illustrated in Figure 1b. $HS1^{pro\text{-}1}$ from sugarbeet was the first nematode *R* gene cloned and encodes a putative extracellular leucine-rich repeat (LRR) protein with a transmembrane domain sharing little similarity to other R-proteins [5]. Since then, the majority of nematode *R* genes have been found to encode proteins that fall into the nucleotide binding (NB)-LRR class of proteins (reviewed in [6]). These include *Mi-1.2* [7], *Mi9* [8], *Hero A* [9], *Gpa2* [10] and *Gro1-4* [11] from solanaceous plant species and *Ma* [12•] from Myrobalan plum (*Prunus cerasifera*), the first nematode *R* gene recently cloned from a perennial plant. *Hero A*, *Gpa2*, *Mi-1.2* and *Mi9* encode proteins belonging to the coiled-coil (CC)-NB-LRR class of R proteins, whereas *Gro1-4* and *Ma* encode proteins with a toll interleukin receptor (TIR)-like domain at the N-terminus (TIR-NB-LRR). The Ma protein also has a 1088 amino acid post-LRR domain of unknown function. *Mi-1.2*, *Mi9*, and *Ma* confer resistance to RKNs, whereas $HS1^{pro\text{-}1}$, *Gpa2*, *Gro1-4* and *Hero A* confer resistance to cyst nematode species.

In soybean, a leguminous crop plant, resistance to soybean cyst nematode (SCN; *Heterodera glycines*) is controlled by multiple quantitative trait loci (QTL). Genome mapping studies have concentrated on *Rhg1* (for resistance to *Heterodera glycines* 1) and *Rhg4*, two major QTLs associated with resistance in the vast majority of resistant germplasm sources (known as plant introductions, PIs) examined to date [13]. In some cases, such as PI 88788, which serves as the source of resistance used in

Figure 1



(a) A picture of a root of the resistant soybean cultivar Forrest showing hypersensitive response (HR)-like cell death at the site of feeding by infective second-stage juveniles of soybean cyst nematode *Heterodera glycines* inbred population PA3 (HG type 0), which are stained pink with acid fuchsin (N). Bar, 50 μm. Image taken by Xiaohong Liu, University of Missouri **(b)** Nematode resistance genes and effectors involved in activation and suppression of plant defense. The findings illustrated in this figure come from research on root-knot and cyst nematodes. The structures and predicted subcellular localization of root-knot (black) and cyst (magenta) nematode resistance proteins are shown. These include $Hs1^{Pro-1}$ [5], Ma [12•], Gro1-4 [11], Mi-1.2 [7], Mi9 [8], Hero A [9], Gpa2 [10], Cf-2 [4••], and Rhg4 [22••]. The subcellular localization of proteins encoded by genes at the *Rhg1* locus is presently unknown. The multi-gene segment of DNA shown in the nucleus contains three genes found to contribute to resistance. These genes are predicted to encode an amino acid transporter (AAT), a soluble NSF (N-ethylmaleimide Sensitive Factor) attachment protein (α-SNAP), and a protein with a wound-inducible 12 region (WI12) [21••]. Several nematode stylet-secreted effectors for which the plant targets and defense suppression function have been characterized are depicted. These include GrVAP [4••], GpRBP1 [47], GrSPRYSEC19 [49•], CEP12, a 12-aa peptide derived from GrUBCEP12 [50•], MiCRT [51•], Hs10A06 [52], Hs/Hg30C02 [53]. Other putative nematode effectors with potential roles in activation of *Mi-1.2*-mediated resistance not pictured include amphid secreted MAP-1 [44] and CG-1 [46]. DGA, dorsal gland ampulla; SPS, spermidine synthase; β-1,3-G, beta-1,3-endoglucanase.

more than 90% of commercial soybean lines, resistance to SCN is controlled by *Rhg1* (referred to as the *rhg1-b* allele). In soybean cultivar (cv.) Forrest, which derives resistance from PI 548402 (also known as Peking), resistance to the same SCN population requires both *Rhg1* (referred to as the *rhg1-a* allele) and *Rhg4* to achieve effective resistance [14]. In both cases, the feeding cells ultimately degenerate; however, the resistance response is stronger and faster in Peking [15]. Initial mapping studies in Forrest identified a leucine rich repeat receptor-like kinase (LRR-RLK) at each locus, and these were claimed to be the *R* genes based solely on their similarity to known plant *R* genes [16]. Recent functional studies have yielded disparate results with regard to the Peking (*rhg1-a*) *LRR-RLK* allele in SCN resistance [17,18] warranting additional fine mapping efforts to define this locus. However, no evidence of a role for the PI 88788 (*rhg1-b*) *LRR-RLK* allele [17] or the Forrest *Rhg4 LRR-RLK* allele [19] in SCN resistance was found in functional studies and these results have been corroborated by fine