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Rooting Out the Mechanisms of Root-Knot Nematode–Plant Interactions

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Abstract

Root-knot nematodes (RKNs; *Meloidogyne* spp.) engage in complex parasitic interactions with many different host plants around the world, initiating elaborate feeding sites and disrupting host root architecture. Although RKNs have been the focus of research for many decades, new molecular tools have provided useful insights into the biological mechanisms these pests use to infect and manipulate their hosts. From identifying host defense mechanisms underlying resistance to RKNs to characterizing nematode effectors that alter host cellular functions, the past decade of research has significantly expanded our understanding of RKN–plant interactions, and the increasing number of quality parasite and host genomes promises to enhance future research efforts into RKNs. In this review, we have highlighted recent discoveries, summarized the current understanding within the field, and provided links to new and useful resources for researchers. Our goal is to offer insights and tools to support the study of molecular RKN–plant interactions.

1. INTRODUCTION

Root-knot nematodes (RKNs) are the most economically important plant-parasitic nematodes. RKNs have broad host ranges, infect the majority of flowering plants, and cause significant losses in crops (228). RKNs engage in complex biotrophic interactions with their host plants (**Figure 1**). They hatch from eggs as motile second-stage juveniles (J2s), which migrate to host-plant roots. When the infective J2 reaches the host plant, it penetrates behind the root tip and then migrates intercellularly to the vascular cylinder of the root. Using its stylet, the RKN injects secretions that contain effectors into the plant tissues. Effectors help facilitate parasitism by suppressing host defense responses and modifying host cell processes to accommodate the nematode. The nematodes use their effectors to convert 6–8 protoxylem cells into enlarged, multinucleate feeding sites called giant cells. The host cells surrounding the nematodes and giant cells also undergo changes, often exhibiting hypertrophic cell division resulting in the formation of root galls. RKNs must invade the roots of a susceptible host plant and establish their feeding sites to survive.

There is still a lot to be uncovered about the complex interaction between plants and RKNs. In this review, we discuss the latest research in RKN biology, their genomes, and the mechanisms by which these nematodes suppress host defenses to become successful plant parasites. We also discuss molecular aspects of the host-plant response to RKN infections and the genes that help plants resist these nematodes.

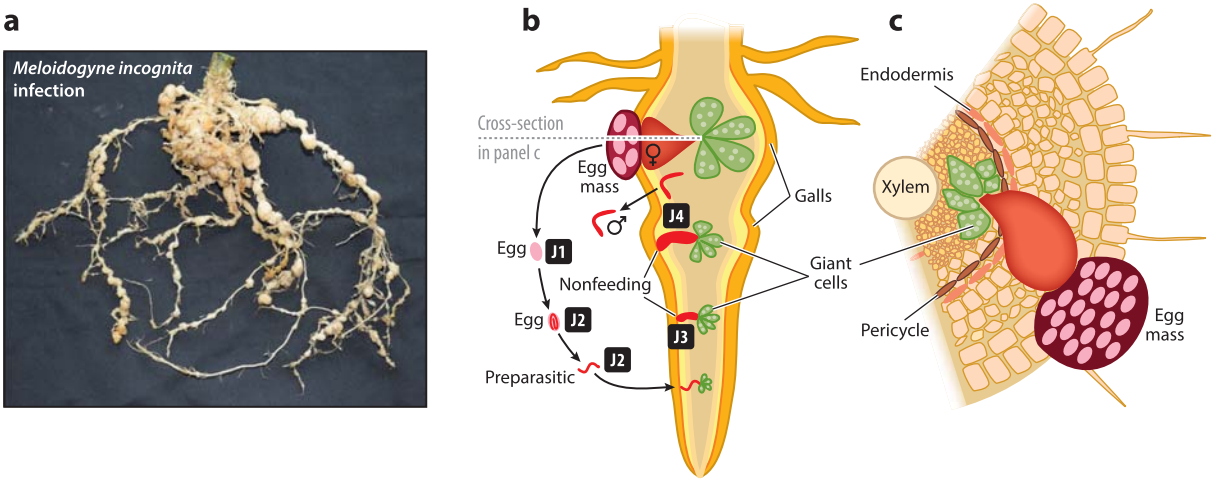


Figure 1

Root-knot nematode (RKN) symptoms and life cycle. (*a*) RKN root symptoms. Summer squash roots with galling caused by *Meloidogyne incognita* infection. (*b*) The life cycle of RKNs. The life cycle of RKNs spans approximately four weeks, depending on environmental conditions. The nematodes molt once within the egg and hatch as second-stage juveniles (J2). The infective J2 nematode penetrates behind the root tip and migrates intercellularly within the vascular cylinder, where it initiates a permanent feeding site adjacent to the vascular tissue. The nematode transforms 6–8 protoxylem cells into specialized multinucleated giant cells. The parasitic J2s feed consecutively from each giant cell until they gain enough energy to continue their life cycle, at which point they cease feeding and undergo three successive molts, either producing ovaries and enlarging into a bulbus adult female or developing into a much larger vermiform male. The males do not feed and leave the roots. Each adult female can produce hundreds of eggs, which she deposits in a gelatinous matrix, forming an egg mass that often works its way out of the root gall to become visible on the root surface. Eggs can develop and begin hatching in a matter of days under ideal conditions, resulting in multiple cycles of reinfection of the same host. If a J2 nematode is not able to establish and maintain its giant cells, the nematode may produce few eggs or even die.

2. GENERAL FEATURES OF ROOT-KNOT NEMATODE GENOMES

The advent of second generation and longer-read third generation sequencing technologies, as well as more advanced bioinformatic tools, has greatly enriched our understanding of plant-parasitic nematode genome structures. As of December 2021, there are at least 22 genome assemblies generated from 10 RKN species (**Table 1**). Improved genome sequencing and assembly pipelines have led to more complete and accurate genome assemblies. For example, the first version of the *Meloidogyne incognita* iso. Morelos genome, published in 2008, was 82 Mb long, had an N50 of 13 kb, and was predicted to contain just 19,212 genes [77% complete, CEGMA (core eukaryotic genes mapping approach) score] (1). Fast-forward to 2017 and the newest assembly of that same *M. incognita* isolate has expanded to 183 Mb, had an N50 of 39 kb, and was predicted to contain 45,351 gene annotations (97% complete, CEGMA score) (22). The newest RKN genomes all have N50s that are measured in megabases and are predicted to contain upward of 90% of the core genes (as measured by CEGMA). The availability of these accurate genomes from different RKN species and isolates has facilitated comparative genomics studies that have led to new insights into RKN biology and pathogenesis.

Studies comparing RKN genomes have already begun to answer long-standing questions about how different RKN species evolved. Some of the most cosmopolitan and damaging RKN species are mitotically parthenogenetic, reproducing exclusively asexually. Multiple studies have now confirmed that three of these asexual species, *M. incognita*, *Meloidogyne javanica*, and *Meloidogyne arenaria*, are all triploids that likely arose from a common ancestral hybridization event (22, 139, 203, 218). Despite the lack of sexual recombination within these species, they have demonstrated a remarkable ability to diversify and adapt into different pathotypes that infect different hosts and/or overcome host resistance (33, 87). It has been suggested that the increased number and diversity of parasitism genes obtained from these hybridization events may have enhanced the ability of these species to become such successful plant parasites (22).

Before the availability of genomic tools, it was hypothesized that different RKN pathotypes shared a common ancestral state and could therefore be distinguished via phylogenetic relationships. Interestingly, a recent population genomics study compared the genomic variations across 11 *M. incognita* isolates from around the world. The study revealed that *M. incognita* pathotypes do not share an evolutionarily common ancestor; instead, it is likely that the same pathotypes have arisen independently multiple times in different regions (123). These studies have implications for how new pathotypes may arise in the field and stress the need for further research into the genetic determinants of pathogenesis. However, it is still a matter of speculation how these asexual RKN species can maintain their diversity and maintain the ability to shift virulence in the absence of sexual recombination.

Evidence from fungal plant pathogens suggests that transposable elements (TEs) can contribute to pathogenic plasticity and evolution, thereby compensating for the loss of sexual recombination (72). The polyploid genomes from parthenogenic RKN species contain a high number of repetitive TEs relative to some diploid sexual species (22). Additionally, a study comparing different isolates of *M. incognita* identified TE polymorphisms within genic regions that could contribute to functional differences (124). Based on this work, it is likely that repetitive elements such as TEs do facilitate genetic plasticity within RKNs in a manner similar to what has been observed in other plant pathogens.

In addition to genome alterations, epigenetic changes could also be a source of phenotypic plasticity within RKNs. An in-depth study that investigated genes encoding epigenetic regulatory machinery within RKN genomes found that these nematodes do not possess any of the DNA methyltransferases typically involved in epigenetic regulation (188). However, (de)acetylation and

Table 1 Summary of sequenced root-knot nematode genomes

Meloidogyne species	Isolate	Assembly size (Mb)	N50	Number of contigs or scaffolds	Annotated genes	CEGMA (%complete)	Accession	Primary reference
M. incognita	Morelos	82.1	13 Kb	9,538	19,212	77	GCA_000180415.1	1
M. incognita	Morelos	183.5	39 Kb	12,091	45,351	97	GCA_900182535.1	22
M. incognita	W1	121.96	17 Kb	33,351	24,714	83	GCA_003693645.1	218
M. incognita	V3	183.53	39 Kb	12,091	45,351	97	GCA_900182535.1	22
M. javanica	VW4	150.35	14 Kb	34,316	26,917	90	GCA_003693625.1	218
M. javanica	Avignon	235.8	10 Kb	31,341	98,578	96	GCA_900003945.1	22
M. arenaria	A2-O	284	204 Kb	2,224	NA	95	GCA_003133805.1	202
M. arenaria	HarA	163.75	11 Kb	46,436	30,308	91	GCA_003693565.1	218
M. arenaria	Guadeloupe	258.07	16 Kb	26,196	103,001	95	GCA_900003985.1	22
M. enterolobii	L30	162.97	11 Kb	42,008	31,051	81	GCA_003693675.1	218
M. enterolobii	Swiss	240	143 Kb	4,437	63,841	95	GCA_903797545.1	122
M. floridensis	JB5	99.9	3.5 Kb	81,111	NA	56	GCA_000751915.1	139
M. floridensis	SjF1	74.9	13 Kb	9,134	14,144	77	GCA_003693605.1	218
M. luci	V13	209.16	1,711 Kb	327	NA	95	ERS3574357	217
M. exigua	Mex-1	42.1	1,882 Kb	260	NA	96	JAGUQR010000000	223
M. hapla	VW9	53.6	83.6 Kb	1,523	14,420	94	GCA_000172435.1	174
M. chitwoodi	PNW-Mc1	47.47	2,451 Kb	30	10,441	NA	JACZZP000000000	10
M. chitwoodi	PNW-Mc2	46.98	2,317 Kb	39	10,424	NA	JACZZO000000000	10
M. chitwoodi	PNW-Mc1-Roza	47.78	2,363 Kb	38	10,660	NA	JACZZN000000000	10
M. graminicola	IARI	38.19	20 Kb	4,304	10,196	84	GCA_002778205.1	212
M. graminicola	IARI	36.86	105 Kb	514	14,062	89	PRJNA411966	211
M. graminicola	Mg-VN18	41.5	294 Kb	283	10,284	96	JABEBT010000000	181

Abbreviations: CEGMA, core eukaryotic genes mapping approach; NA, not available.

(de)methylation pathways are present in RKNs and conserved with those pathways in the model free-living nematode *Caenorhabditis elegans*. Moreover, parthenogenetic RKNs also have unique classes of noncoding RNAs (ncRNAs) that are expressed during parasitic stages, which may indicate that they have unique roles related to parasitism (188). A recent deep sequencing analysis of RKNs during infection of cotton also revealed that specific microRNAs, a class of ncRNAs, are expressed during certain nematode developmental stages (137). Although certainly an intriguing area of study, more work is needed to determine the role that epigenetic pathways play in RKN development and pathogenesis.

The growing number of RKN genomes has expanded our ability to identify virulence factors (primarily effectors) that have the potential to act during parasitism. However, few effectors have been characterized in RKNs, making it increasingly clear that more work is needed to grasp the complete effectomes deployed by RKNs. As new genome sequences from additional RKN species and isolates become available, they will provide insight into which effector genes have arisen specifically in different species and how they may work together to facilitate RKN pathogenesis in different host crops.

3. ROOT-KNOT NEMATODE EFFECTORS

3.1. Identifying Nematode Effectors

RKN effectors are secreted by secretory organs, which include the cuticle, amphids, and rectal gland, but the most well-studied secretory organs are the two subventral glands and one dorsal esophageal gland. Because proteins made in the esophageal glands can be secreted through the stylet into the plant cell apoplast and/or cytoplasm, a majority of effector studies has focused on these esophageal glands. In 2003, an esophageal gland cDNA library helped to identify 185 transcripts encoding secreted proteins, including the MSP (*Meloidogyne* secretory protein) effectors (101). Putative effectors were also identified from expressed sequence tags of *Meloidogyne chitwoodi* (196). More recently, transcriptome analyses of preparasitic and parasitic nematodes have helped in identifying genes that are upregulated during parasitism (42, 74, 134, 180, 259). The typical pipeline for nematode effector studies is to identify nematode genes upregulated during parasitism that encode proteins containing a secretion signal peptide and lack transmembrane domains. These criteria are computational evidence of proteins destined for secretion. In situ hybridization experiments are often performed to determine whether the candidate effector is made in a nematode secretory organ (typically the esophageal glands). An alternative approach has been to directly analyze the nematode secretions by mass spectrometry. Using this direct approach, Bellafiore et al. (14) identified approximately 500 proteins secreted by *M. incognita*.

Transcriptome analyses have shown that there is transcriptional regulation of nematode genes involved in development and parasitism. Previous work in cyst and pinewood nematodes indicates that conserved promoter motifs drive effector expression in specific gland cells (67–69). By coupling *M. incognita* genomic and transcriptomic data, a recent analysis identified a *cis*-regulatory motif called Mel-DOG (49). Similar to the DOG box first identified in cyst nematode genomes, the Mel-DOG motif is associated with the promoters of genes encoding dorsal gland–secreted effectors. Because Mel-DOG is broadly conserved across the *Meloidogyne* genus, the presence of Mel-DOG in gene promoters can be used as a criterion to predict effectors in other *Meloidogyne* genomes. Da Rocha et al. (49) also monitored the expression of genes with Mel-DOG *cis* elements at different *M. incognita* life stages. They found an enrichment of Mel-DOGs in the promoters of genes encoding proteins with specific functions in pathogenicity, such as enzymes related to cell wall degradation (49). As additional RKN genomes and transcriptomes become available, it will be

possible to identify other *cis* elements that can help predict effectors associated with tissue-specific and/or life-stage-specific expression.

Some genome-wide analyses have focused on expanded effector gene families in plant-parasitic nematodes. Increased diversity within these effector families is indicative of rapid evolution, a feature of many pathogen effectors that need to adapt rapidly to evade the host immune system (55). For example, there has been a large expansion of SPRY domain–containing effectors in cyst nematodes. The *Globodera pallida* genome contains 300 SPRY domain–containing proteins, some of which are secreted effectors that have been shown to suppress the plant innate immune system (3, 56). *M. incognita* also has proteins with SPRY domains but far fewer than what is found in cyst nematodes and none have signal peptides (56, 148). However, gene expansion has been observed in other RKN effector families, particularly in the MAP-1 effector family. Genome analyses of *map-1* genes show evidence of duplications and intragenic domain losses and gains. The variation in the number/arrangement of internal repeats in the *map-1* genes correlates with the (a)virulence of *M. incognita* near-isogenic isolates on resistant tomato (34). The *map-1* gene family is restricted to *Meloidogyne* species that reproduce by mitotic parthenogenesis, with the exception of *Meloidogyne floridensis* (225). Although the functional role of MAP-1 in parasitism is unclear, intraspecies conservation of the *map-1* among *M. incognita* isolates suggests that strong selection pressure is acting to maintain these genes and that they may have roles in host specialization (225).

Some effectors can be assigned function based on homology to known proteins, such as those with homology to cell wall–degrading enzymes (CWDEs). In fact, nematode genes with homology to bacterial genes led to the hypothesis that nematodes acquired genes by horizontal gene transfer (HGT) from bacteria and fungi (86). Within the genome of *M. graminicola*, 67 genes were identified as possibly originating from HGTs (181). Up to 3.34% of the genes within all RKN genomes are predicted to have originated from HGTs (176). The conserved biological functions of the transferred genes between the donor and receiver organisms help identify nematode effectors that are cell wall–modifying enzymes (e.g., polygalacturonase, pectate lyase) (50), act to suppress host defenses (e.g., chorismate mutases and cyanate lyases), and process nutrients (e.g., bioB biotin synthase, GH32 invertase) (21).

3.2. Characterizing Nematode Effectors

Many putative effector genes have no homology to any genes in public databases, so uncovering their roles in nematode parasitism has been challenging. RKNs are not yet amenable to genetic manipulation using current approaches, which adds to the challenge of characterizing effectors (125). Instead, heterologous expression in plants and RNAi knockdowns of nematode transcripts have provided insights into effector gene functions. Based on these functional effector characterizations, RKN effectors can be divided into two broad categories: effectors with roles in host defense suppression and effectors that alter the host to generate and maintain their giant cells. However, these two processes are not necessarily mutually exclusive (**Figure 2**) (see **Table 2** for a current summary of RKN effectors).

Host production of reactive oxygen species (ROS) is often associated with plant defenses. Many RKNs have effectors that suppress ROS production, activate host scavenging of ROS, or inhibit defenses downstream of ROS. For example, Mg16820 is an effector secreted from *M. graminicola* into the plant cell cytoplasm and apoplast, and in the apoplast, it suppresses flg22-induced ROS production (160). Recent work in *M. graminicola* has also identified the effector MgMO289 (213). MgMO289 interacts with the rice metallochaperone protein OsHPP04, which in turn targets a rice copper/zinc superoxide dismutase, ultimately boosting the rice superoxide radical scavenging system and suppressing host immunity (213). Similarly, the *M. javanica* effector TTL5

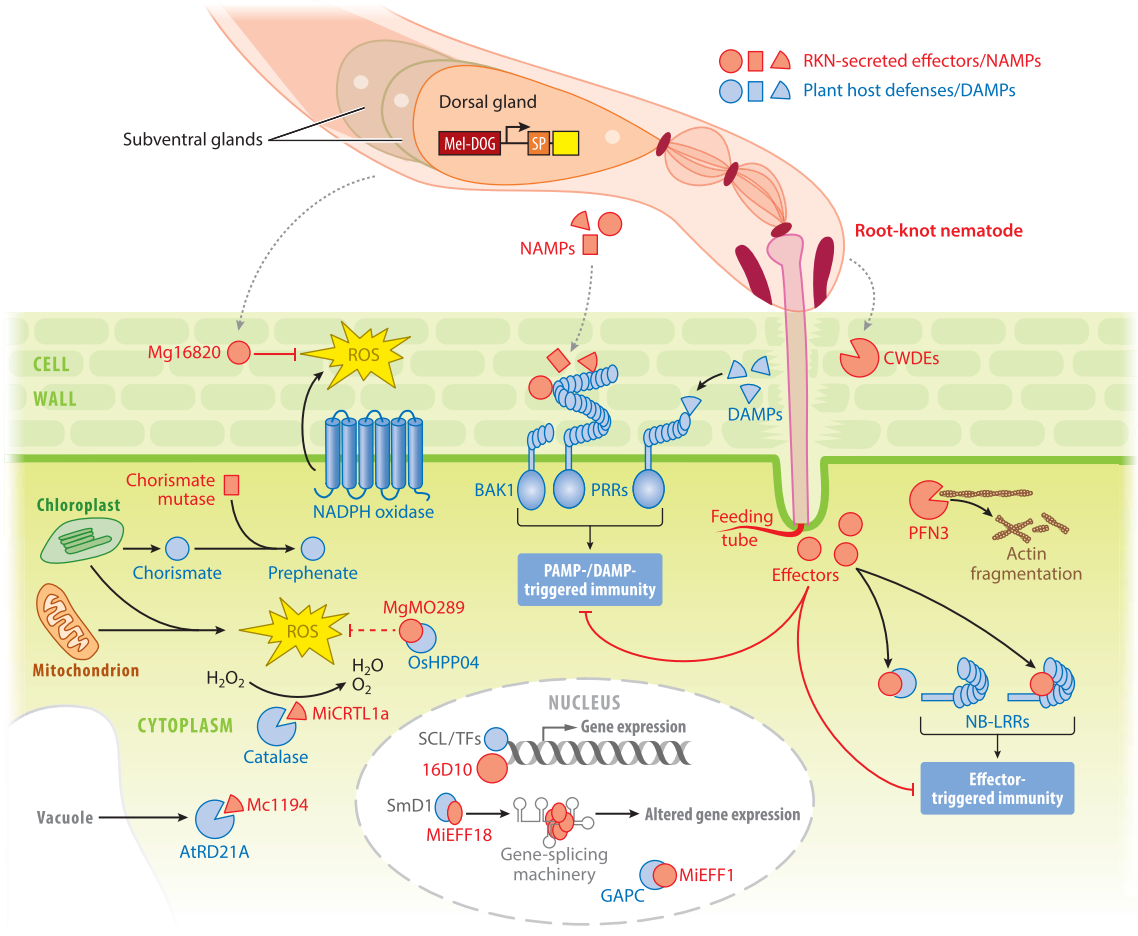


Figure 2

Advances in root-knot nematode (RKN)–plant interactions. RKNs possess a stylet that mechanically penetrates host-plant cells for feeding and effector secretion. Effectors are expressed in the esophageal glands, where they are secreted into the apoplast or directly into the cytoplasm. The *cis*-regulatory motif, Mel-DOG, is enriched in effectors expressed within the dorsal gland cell of *Meloidogyne incognita*. Apoplastic CWDEs are secreted to degrade and breach the plant cell walls. However, the host can perceive cell wall fragments, as well as NAMPs, that are released during infection and mount defense responses. In turn, RKNs secrete an arsenal of effectors to overcome plant defenses. The apoplastic effector Mg16820 interferes with PAMP-triggered immunity (PTI)-mediated production of reactive oxygen species (ROS). Additionally, RKNs balance cytoplasmic ROS abundance during infection by modulating ROS scavenging (*dashed red line*), as demonstrated with the effectors MgO289 and MiCRTL1a. Many effectors manipulate different plant immunity components to suppress defenses. The RKN effector Mc1194 targets the plant defense protease RD21A (Responsive to Desiccation 21) as a means to overcome host defenses. Chorismate mutases have also been shown to suppress defense responses. Effectors are also involved in giant cell formation and maintenance to acquire nutrients. PFN3 disrupts actin polymer formation during early giant cell formation. The interaction between host GAPCs and effector MiEFF1 in giant cells is also required for parasitism. Furthermore, the RKN effectors MiEFF18 and 16D10 hijack the host gene expression and the gene-splicing machinery, respectively, to modulate gene expression within giant cells. More studies are required to identify effectors that aid the nematode in evading components of PTI and effector-triggered immunity (ETI) as well as those recognized by host resistance genes. Abbreviations: CWDEs, cell wall–degrading enzymes; DAMPs, damage-associated molecular patterns; GAPCs, glyceraldehyde-3-phosphate dehydrogenases; NAMPs, nematode-associated molecular patterns; NB-LRRs, nucleotide binding site–leucine-rich repeats; PAMPs, pathogen-associated molecular patterns; PRRs, pathogen recognition receptors; SCL, SCARECROW-LIKE; SP, signal peptide sequence; TFs, transcription factors.

Table 2 Effectors from root-knot nematodes

Functional category	Gene name	*Meloidogyne* species[a]	Function	Secretory organ	Primary reference(s)
Defense suppression	*MiISE6*	*M. incognita*	Interferes with various metabolic and signaling pathways to facilitate nematode parasitism	Subventral	207
	MiISE5	*M. incognita*	Suppresses pathogen-induced cell death	Subventral	208
	Mh265	*M. hapla*	Suppresses the host defenses	Subventral	80
	TTL5	*M. javanica*	Encodes transthyretin-like protein; suppression of the host defenses and manipulation of the host ROS-scavenging system	Subventral	135
	MiCRT	*M. incognita*	Encodes a calreticulin that suppresses basal plant immunity	Subventral	106
	MSP40	*M. incognita*	Suppresses aspects of PAMP and effector-triggered immune responses	Subventral	166
	MgGPP	*M. graminicola*	Subject to proteolytical processing in the host cell and is thereby able to suppress host defense responses, including *R*-gene-mediated HR	Subventral	39
	MiCTL1	*M. incognita*	Targets host catalase to fine-tune ROS and promote parasitism	Subventral	263
	MgPDI	*M. graminicola*	Plays roles in reproduction and pathogenicity; protects nematode from exogenous H_2O_2 stress	Subventral	224
	Mg16820	*M. graminicola*	Secreted in both the apoplast and the cytoplasm; suppresses PTI and ETI responses and targets a protein involved in the stress response	Subventral	160
	Mc1194	*M. chitwoodi*	Interacts with host papain cysteine protease	Subventral	52
	MjCM-1	*M. javanica*	Encodes nematode chorismate mutase; may be involved in altering auxin and/or SA biosynthesis	Subventral	62
	Minc00344	*M. incognita*	Targets GmHub10 protein to disrupt cell cycle or plant defense responses	Subventral	82, 198
	Mi-gst-1	*M. incognita*	Protects against ROS	Subventral	63
	Mg01965	*M. graminicola*	Encodes C-type lectin, which suppresses basal immune response in apoplast	Subventral	269
	VAP	*M. incognita*	Encodes a venom allergen involved in early defense suppression and early parasitic processes	Subventral	58
	Mj2G02	*M. javanica*	Promotes parasitism by interfering with the host JA signaling pathway	Dorsal	214
	MgMO237	*M. graminicola*	Interacts with multiple host defense proteins to suppress plant defenses	Dorsal	37
	MiSGCR1	*M. incognita*	Suppresses pathogen-induced cell death	Dorsal	165
	MgMO289	*M. graminicola*	Interacts with OsHPP04 in rice; suppresses host innate immunity	Dorsal	214
	Misp12	*M. incognita*	Suppresses host defense responses at later stages of nematode parasitism	Dorsal	247
	MeTCTP	*M. enterolobii*	Suppresses programmed cell death in host plants	Dorsal	268
	Mj-NULG1	*M. javanica*	Interacts with GmHub10; suppresses plant defenses	Dorsal	82, 136

(*Continued*)

Table 2 (*Continued*)

Functional category	Gene name	*Meloidogyne* species[a]	Function	Secretory organ	Primary reference(s)
	Mi-MSP18	*M. incognita*	Suppresses defense-related cell death	Dorsal	84, 100
	MiMIF-2	*M. incognita*	Interacts with plant annexins to suppress plant immunity	Hypodermis	262
	Mj-FAR-1	*M. javanica*	Manipulates lipid-based plant defenses	Hypodermis and cuticle	104
Giant cell formation/ maintenance	*Mi8D05*	*M. incognita*	Interacts with the host protein TIP2; involved in water/solute transport	Subventral	249
	16D10	*M. incognita*	Targets host plant transcription factors; regulates feeding site formation	Subventral	99
	PFN3	*M. incognita*	Encodes profilin; binds to monomeric actin; expression in plant cells disrupts actin filaments	Subventral	133
	MiPM	*M. incognita*	Encodes small protein with cell-penetrating properties and interacts with regulatory component of the ubiquitin/proteasome system	ND	26
	MiEFF1	*M. incognita*	Interacts with a cytosolic GAPCs in the nucleus of giant cells	Dorsal	107, 229
	MiEFF18	*M. incognita*	Interacts with small ribonucleoprotein particle SmD1 and is required for giant cell formation	Subventral	149
	Map1	*M. incognita*	Encodes expansin-like protein; may be involved in initiation and/or maintenance of the feeding site	Amphids	205
	Mi-Msp2	*M. incognita*	Promotes parasitism; ortholog of *M. javanica* 2G02	Subventral	101
Peptide mimics	*Cep*	*M. hapla*	Encodes CEP plant hormone mimic	ND	24
	RALF	*M. incognita*	Encodes rapid alkalinization factor that modulates plant immune responses	Esophageal	261
	MiIDL	*M. incognita*	Encodes IDA-like peptide; functions as a host IDA mimic	ND	230
Cell wall–modifying enzymes	*Mi-eng-1*	*M. incognita*	Encodes a β-1,4-endoglucanase	Subventral	195
	Mj-pel-1	*M. javanica*	Encodes pectate lyase	Subventral	61
	MI-PG-1	*M. incognita*	Encodes a polygalacturonase	Subventral	108
	Mi-xyl1	*M. incognita*	Encodes an endo-1,4-β-xylanase	Subventral	154
	MI-CBP-1	*M. incognita*	Encodes cellulose-binding protein	Subventral	57
Unknown function in parasitism	*SXP/RAL-2*	*M. incognita*	Encodes an SXP/RAL-2 protein family with unknown function	Subventral	231
	7H08	*M. incognita*	Travels to nucleus to activate transcription	ND	257

[a]The species in which the effector was first identified and/or characterized.
Abbreviations: CEP, C-TERMINALLY ENCODED PEPTIDE; ETI, effector-triggered immunity; GAPCs, glyceraldehyde-3-phosphate dehydrogenases; HR, hypersensitive response; IDA, INFLORESCENCE DEFICIENT IN ABSCISSION; JA, jasmonic acid; ND, no data; PAMP, pathogen-associated molecular patterns; PTI, PAMP-triggered immunity, ROS, reactive oxygen species; SA, salicylic acid.

interacts with a host protein that increases the host's ROS-scavenging activity, reducing host resistance (135). The *M. incognita* protein disulfide isomerase–like effector interacts with a tomato stress-associated protein (SlSAP12), which may act as a redox sensor to tune plant resistance responses and promote disease (224). Recently, a C-type lectin (CTL)-like effector from *M. incognita*

(MiCTL1a) was shown to interact with a plant H_2O_2 catalase. The binding of MiCTL1a with this catalase suppresses the enzyme's H_2O_2-degrading activity (263). Although this may seem counterintuitive, work in cyst nematodes has shown that during the early infection stage ROS play a positive role in infection processes (209). For RKNs, secretion of effectors that either enhance or suppress ROS production may reflect the nematode's need to keep the ROS levels under tight control, avoiding the ROS-mediated defenses while harnessing the positive role ROS can play in the infection process.

Plant hub proteins are often targeted by pathogens to promote their success in the host (32). For example, the papain-like cysteine proteases (PLCPs) play important roles in plant immunity and are targeted by effectors from diverse pathogens. *Ustilago maydis*, *Ralstonia solanacearum*, *Cladosporium fulvum*, and *Phytophthora infestans* all secrete effector proteins, sometimes multiple, that target common host-plant PLCPs (16, 206, 215). Interestingly, some of these same proteases are also targets for plant-parasitic nematode effectors. The defense protease RCR3Pim in tomato is targeted by the *Globodera rostochiensis* effector VAP1 (138), and both the RKN effector Mc1194 and the cyst nematode effector Hs4E02 target the *Arabidopsis* PLCP Responsive to Dehydration 21A (RD21A) (52, 185). In the case of Mc1194, the nematode effector interacts with RD21A in the host-plant cell and suppresses RD21A's role in plant defenses (52).

Effectors also have roles in giant cell formation and maintenance. During giant cell organogenesis, there are dramatic changes to the cytoskeleton of the chosen host cells. These cytoskeletal changes include a rearrangement of the microtubule arrays and cytoplasmic actin filaments, both of which are required for the proper development of the giant cells (53, 54). RKNs secrete an effector called Profilin 3 (PFN3) that binds to actin monomers and disrupts actin polymerization, which ultimately helps drive parasitic success (133). One effector that may be involved in giant cell formation is MiEFF1. MiEFF1 interacts with a cytosolic glyceraldehyde-3-phosphate dehydrogenase (GAPC) in the nucleus of giant cells, where GAPC's nonmetabolic function helps with nematode infections (229). Another RKN effector, MiEFF18, also localizes to the nuclei of giant cells, where it interacts with the nuclear ribonucleoprotein SmD1 and affects the expression of genes involved in giant cell development (149).

Nematodes secrete effectors that mimic plant peptides to suppress plant defenses and cause developmental changes that are necessary for giant cell formation. In plants, the rapid alkalinization factors (RALFs) are peptides involved in plant growth, defense, and stress responses. RKNs secrete a RALF-like peptide that binds to a host receptor-like kinase FERONIA (261). The recognition of the nematode RALF-like peptide by the plant helps to modulate plant immune responses and potentially regulates cell wall expansion to facilitate giant cell development (261). RKNs also secrete mimics of INFLORESCENCE DEFICIENT IN ABSCISSION (IDA) peptides. *M. incognita* has two IDA-like genes, *MiIDL1* and *MiIDL2*, that can functionally complement the *ida* mutant phenotype of *Arabidopsis* (97, 119, 230). The exact role of the IDA-like peptides in facilitating parasitism is unknown but based on the role of plant IDA peptides in abscission and lateral root emergence (127), it has been suggested the nematode IDA-like peptides may be involved in the cell wall modifications needed for gall formation. Because IDA peptides are negative regulators of stress responses (238), the nematode IDA-like peptides may also be involved in the suppression of plant defense/stress responses.

Another family of plant peptide hormones known as the CLAVATA3 (CLV3)/EMBRYO SURROUNDING REGION–related (CLE) family play important roles in development as well as symbiosis and abiotic stress responses. CLEs act as cell-to-cell signals with their specific biological function dependent on the combination of CLE peptides and their cognate receptors (253). Cyst nematodes secrete CLE-like peptides that can functionally mimic endogenous plant CLEs and bind to and activate plant CLE receptors (244). This process is necessary for feeding

site formation and may act by priming neighboring cells for incorporation into the expanding syncytium (242, 244). In RKNs, CLE-like motifs have been found embedded within *M. incognita* MAP effector proteins (198). The RKN effector 16D10 also has some sequence similarity to the CLE motif (99). RNAi silencing of 16D10 showed that this gene is crucial for RKN infectivity of plants. However, unlike cyst CLEs, 16D10 cannot complement the *Arabidopsis clv3–1* mutant phenotype, suggesting that it is not a plant CLE mimic. Moreover, the 16D10 effector is targeted to the nucleus to interact with plant SCARECROW-LIKE (SCL) transcription factors, also suggesting that RKN CLE-like effectors are not localized to the same compartment and do not follow the same trafficking pathway of cyst nematode CLEs (99, 153). Instead, RKN CLEs are likely involved in regulating gene expression needed for giant cell formation.

Plants secrete small peptides called C-TERMINALLY ENCODED PEPTIDES (CEPs) that have roles in plant root development and architecture (168) and can act as long-distance signals regulating nitrogen-demand signaling responses (219). CEPs have been reported in the genomes of both RKNs and reniform nematodes (21, 70). Transcriptome analysis has demonstrated that all MhCEPs were detected at either the egg stage, J2 stage, or three weeks postinoculation, but MhCEP11 was highly expressed at the J2 stage. The specific role of RKN CEP-like effector mimics in nematode parasitism is still unknown. In reniform nematodes, the RrCEPs are made in the dorsal glands and may have roles in feeding site formation (70). Interestingly, RrCEPs contain a variable domain similar to the apoplastic trafficking domains in cyst nematode CLEs effectors (241, 242). Not only does this indicate that the RrCEPs contain a motif for trafficking, but it also suggests conservation of a trafficking motif in different effectors and in different genera of nematodes (78). However, RrCEPs show no sequence similarity to CEPs in RKNs outside the 15-amino-acid CEP domain itself. Considering that reniform nematodes are more closely related to cyst nematodes, which lack CEPs, it is thought that the CEPs arose independently in reniform nematodes and RKNs. Typically, CEPs are posttranslationally modified, which is necessary for their storage, transport, and function. Although gland cell expression or the function of an RKN CEP-like effector remains unknown, it would be interesting to explore the role of posttranslational modifications for the function of RKN CEPs.

4. PLANT RESISTANCE RESPONSES TO ROOT-KNOT NEMATODES

4.1. Preparasitic Resistance

Plants have chemical defenses that impede RKNs from finding and accessing the host root. Preparasitic J2s rely on chemical cues (root exudates) to find and penetrate host roots. Recent work has demonstrated that changes in the ethylene response pathway in plants alter root exudate composition, leading to changes in the attractiveness of roots to RKNs as well as other nematodes (66, 75). RKN infection itself also alters the attractiveness of the roots to facilitate reinfection by RKNs over time (118). Early experiments demonstrated that the galls induced by RKNs actually become more attractive to preparasitic juveniles compared to other portions of the same root (20). Plants release many compounds into the rhizosphere, and only recently have some of the specific volatile compounds that attract RKNs been identified (35, 173). Much research is still needed to understand how root exudates affect RKN–host interactions. What is clear is that changes in root exudates influence the nematode's ability to find and penetrate roots.

Some specific root exudates also actively repel juvenile RKNs. Polythienyls secreted from the roots of marigolds provide the most well-known example of an RKN repellent (40, 183, 243). These ornamental plants have been deployed as effective biocontrol against RKNs and other nematodes under specific conditions (95, 184). Cucurbitacin A, secreted by cucumbers, can also act as a repellent. Cucumber genotypes that produce this compound are less attractive to *M. incognita*

(90, 114), although not to a degree that would confer complete resistance. Numerous in vitro studies have identified promising plant-derived compounds that have repellent or nematicidal effects on RKNs, but the significance of these compounds with regard to RKN–host interactions in the soil is largely unknown.

The physical structure of the root can thwart the ability of nematodes to move through the root tissue while attempting to establish a feeding site. Many studies have reported reductions in the numbers of J2s penetrating the roots of RKN-resistant genotypes in multiple crop species, including pepper (88), grape (6), peanut (15), and rice (30). Other studies have found that RKN penetration of resistant hosts increases the accumulation of cell wall–strengthening compounds, including lignin, suberin, and callose (9). Similarly, host mutations and defense priming compounds that affect the cell wall composition often lead to changes in J2 penetration (76, 102, 162, 246). However, it is difficult to disentangle the effects of these physical cell wall barriers to nematode penetration from the host's chemical defense responses to RKN infection.

The root endodermis provides a clear physical barrier to RKN infection. This tissue contains the Casparian strip, with highly lignified and suberin-reinforced cell walls that separate the inner vascular cortex from the outer stele. It has been observed that RKN J2s avoid the hardened tissues of the endodermis by navigating to the undifferentiated zone of elongation near the root tip, performing an end run around the endodermis before traveling back up through the cortical tissues to establish a feeding site (85). Recent work in *Arabidopsis* has shown that host mutations that disrupt the Casparian strip and/or suberin deposition within the endodermis allow for increased parasitism by *M. incognita* (94). Conversely, rice varieties with constitutively higher suberin deposition in their roots show reduced penetration by *M. graminicola* (210). In addition to altering RKN penetration, the feeding sites and galls are also reduced in roots that have stronger endodermal tissues, suggesting that structural reinforcement of the endodermis enhances resistance to RKNs throughout the parasite's life cycle.

4.2. Pathogen-Associated Molecular Pattern–Mediated Resistance to Nematodes

RKNs must also overcome the plant's basal immunity. In this form of plant immunity, the plant perceives pathogen-derived molecules (e.g., proteins, carbohydrates, lipids, and nucleic acids) called pathogen-associated molecular patterns (PAMPs) (27, 41). Recognition of PAMPs is mediated through plant membrane–bound pathogen recognition receptors (PRRs). The host recognition of the PAMPs triggers both local and systemic defense responses that include increased callose deposition, calcium flux, a burst of ROS, and defense gene expression (179, 270). Altogether, these responses constitute the basal plant immune response referred to as PAMP-triggered immunity (PTI). There is limited information about the PTI response to RKNs. However, when *Arabidopsis* plants were treated with the flagellin-derived flg22 peptide, a well-studied bacterially derived PAMP, the treatment enhanced plant resistance against RKNs, with the flg22-treated plants showing a 75% reduction in the number of juveniles inside the roots compared to the control (222). The data show that if PTI is elicited prior to nematode infection, the plant can mount a defense against the RKNs. Interestingly, the mutant in FLS2, the plant receptor for flg22, did not show any changes in the number of root galls compared to the wild type, indicating that this receptor was not recognizing nematode PAMPs or any bacteria associated with the nematodes (222).

The first nematode PAMP [also referred to as a nematode-associated molecular pattern (NAMP)] was the nematode pheromone ascaroside #18 (ascr#18) (142). Ascarosides are derivatives of the 3,6-dideoxy-L-sugar ascarylose that are modified with fatty acid–derived side chains and are found in both parasitic and free-living nematodes (115, 204). There are more than 200 characterized ascarosides produced by different nematode species, each with a numerical

designation determined by the number of carbons on its side chain (204), and specific blends of these molecules function to regulate nematode aggregation, mating, development, and foraging behaviors (47, 159, 177). ascr#18 is the most abundant ascaroside in plant-parasitic nematodes. When plant leaves or roots are treated with purified ascr#18, local and systemic defense responses are induced, including increased PTI marker gene expression and induction of both salicylic acid (SA)- and jasmonic acid (JA)-mediated defense signaling (142). The ascr#18 treatment increases *Arabidopsis* resistance to not only plant-parasitic nematodes but also to a range of bacterial, viral, fungal, and oomycete pathogens. The data indicate that ascr#18 triggers basal plant immune responses that result in broad-spectrum resistance (142).

The host receptor for ascr#18 is still unknown, but a follow-up study found that plants metabolize ascr#18 into ascr#9. Although this processing step was required to elicit enhanced nematode resistance, bacterial resistance and defense gene expression were independent of this processing step (141). This surprising result unlinks ascr#18-mediated plant defense signaling pathways and nematode resistance. Because ascarosides can be involved in social cueing, Manohar et al. (141) looked at the role of ascr#18 and ascr#9 in attracting nematodes to the plant. They discovered that a blend of ascr#18 and ascr#9 is involved in repelling the nematodes from plant roots. Therefore, the plants are recognizing and metabolizing ascr#18, and as a result, the combination of ascarosides that are produced deter plant-parasitic nematode infections.

In a search for additional NAMPs, Mendy et al. (151) created NemaWater by placing sterile J2s in water and removing the nematodes after 24 hours. NemaWater from either the cyst (*Heterodera schachtii*) or the RKN (*M. incognita*) could trigger a PTI-dependent ROS burst in roots, suggesting that the NAMPs in NemaWater are conserved among plant-parasitic nematodes (151). Heat and Proteinase K treatments decreased NemaWater activity, which suggests that proteins in the NemaWater are being detected by the host plant (151). However, the specific NAMPs in NemaWater have yet to be identified. Another recent study also suggested that bacteria found in nematode-suppressive soil may also play a role as a defense elicitor. The bacteria associated with the juvenile RKN cuticle released PAMPs that elicited plant immune responses and suppressed nematode infections (226). It is intriguing to consider that nematode-associated microbes may initiate PTI that is effective against the nematode.

Researchers are interested in identifying NAMP receptors. A screen of *Arabidopsis* defense receptor mutants found that an LRR-RLK (leucine-rich repeat receptor-like kinase) mutant, termed NILR1 (NEMATODE-INDUCED LRR-RLK 1), was significantly more susceptible to the sugar beet cyst nematode *H. schachtii* (151). Altogether, the data point to NILR1 as a NAMP receptor. Considering that the NILR1 receptor is largely conserved in the genomes of several dicotyledonous and monocotyledonous plant species, a wide range of plants may be detecting NAMPs and triggering NILR1-dependent PTI. Work in *Arabidopsis* defense mutants showed that NemaWater-induced responses were dependent on BAK1, a coreceptor for LRR-type PRRs (151), pointing to conserved downstream PTI components that are turned on by the recognition of a NemaWater protein(s) by NILR1.

4.3. Damage-Associated Molecular Pattern–Mediated Resistance to Nematodes

Although RKN penetration and intercellular migration within the roots are relatively gentle compared to cyst nematodes, they can still cause a degree of damage to the roots. Plant damage results in the release of cell wall fragments and other damage-related saccharides, peptides, and nucleotides. These plant-derived molecules function as danger signals called damage-associated molecular patterns (DAMPs) (96, 216, 221, 251, 265). DAMPs are recognized by cell surface receptors and trigger downstream defense pathways that overlap with PTI (25). One type of DAMP

is the small defense plant elicitor peptides (Peps), which are encoded by PROPEP genes (103). Plant damage causes the transcriptional activation of PROPEP genes (13). The PROPEP precursors are then cleaved into Peps and released from damaged plant cells, where they are recognized by PEP receptors (PEPRs) on neighboring cells. Recognition of Peps by their cognate receptor triggers downstream plant defense responses both locally and systemically (126, 252). The exogenous application of soybean and potato Peps has proven effective in enhancing RKN resistance in those respective plants (132, 258), indicating that activating DAMP-mediated immune responses can prime plant defenses for improved plant resistance to RKNs. Interestingly, single *Arabidopsis* mutants in the DAMP receptor genes *dorn1*, *pepr1*, and *pepr2* or the double mutant *pepr1 pepr2* did not significantly affect the number of juveniles inside the roots or total galling compared to the wild-type controls (222). Other DAMPs may play roles in regulating plant responses to nematodes, masking the effects of these specific receptor mutants. Although we are still learning about the DAMPs that are released during RKN infections, it seems that DAMP-triggered immunity can offer some plant protection.

RKNs use their stylets in combination with cell wall–modifying effectors to facilitate root penetration and migration (190). The CWDEs identified in RKNs include β-1,4-endoglucanases (195), glycosyl hydrolase family cellulases, and pectin-degrading enzymes such as pectin methylesterases, pectate lyases, and polygalacturonases (155, 191). Although the RKN-secreted CWDEs act as virulence factors, the damage they cause can release DAMPs that trigger host immune responses. For example, the *M. graminicola* pectate lyase (Mg-PEL1) acts as a virulence factor and promotes nematode infection (38). However, data show that the pectate lyase activity of Mg-PEL1 results in the release of cell wall components from the plant, which elicited DAMP-mediated immune responses. Chen et al. (38) speculated that the Mg-PEL1 may work in combination with other virulence factors to promote nematode infections, but on its own, the enzyme causes cell wall damage that induces DAMP-triggered immunity.

4.4. Roles of Defense Hormones Salicylic Acid and Jasmonic Acid in Plant–Nematode Interactions

During plant–pathogen interactions, plant defenses are regulated by one or more phytohormones, including SA and JA. Although they are not as well studied, other hormones such as auxin, cytokinin, gibberellic acid, and abscisic acid also have roles in plant–nematode interactions and plant defense (78). Because RKNs are biotrophs, the prevailing thought is that plant defenses are mediated primarily through SA-responsive pathways. Exogenous application of SA or its analogs on tomato can reduce RKN infections (156). SA plays a role in transcriptional reprogramming and controls the expression of some defense genes, including pathogenesis-related (PR) genes (201). In *Arabidopsis*, plants that overexpressed PR1 were more resistant to RKN infection compared to the wild type (89). Interestingly, transgenic NahG tomato lines that lack SA accumulation were not more susceptible to RKNs. One possible explanation for this discrepancy is that residual SA in the NahG plants was sufficient for basal levels of nematode resistance (18). Another player in SA-mediated defenses is NONEXPRESSOR OF PATHOGENESIS-RELATED GENES 1 (NPR1), which activates *PR1* gene expression and induces the plant immune response called systemic acquired resistance (SAR). Transgenic tobacco plants expressing *AtNPR1* exhibited enhanced resistance to RKNs, and this resistance was associated with constitutive basal expression of the PR genes (*PR-1* and *PR-5*), and their expression was further enhanced after RKN infections (189). Phytoalexin deficient 4 (*PAD4*) encodes a nucleocytoplasmic lipase-like protein, which promotes the accumulation of SA and helps drive SAR (110). Overexpression of *AtPAD4* in soybeans conferred resistance to RKNs, and the resistance was concurrent with an increase

in *GmPR1* transcripts (255). Overall, the data indicate that SA-mediated responses play a role in host defenses against RKNs.

ROS are involved in both upstream and downstream SA-mediated responses. A ROS burst can trigger SA-mediated signaling and SA responses mediate redox dynamics within the cell (92). When PAMPs are recognized by the host plant, a ROS burst occurs. Therefore, nematode-triggered PTI responses may lead to rapid production of ROS. ROS are potentially toxic to the nematodes, but ROS are also involved in cell wall strengthening in addition to their roles in modulating defense gene expression (113). The accumulation of ROS during plant defense occurs via the activation of NADPH oxidases in the plasma membrane. Mutants in NADPH oxidase (RbohD and RbohF) have been found to be more susceptible to RKN infections (43, 209, 222), pointing to the importance of ROS in plant defense against RKNs. Interestingly, the opposite was found with cyst nematodes; RbohD- and RbohF-deficient plants are more resistant to cyst nematodes. The data suggest that cyst nematodes can tune ROS levels to coordinate the host metabolism for their own benefit and that differences in temporal and spatial induction of ROS by cysts and RKNs lead to two different feeding structures (209).

JA is also a player in plant–nematode interactions. Several groups have shown that exogenous application of JA or its volatile derivative methyl jasmonate can enhance RKN resistance in a variety of plants, including tomato, rice, soybean, and *Arabidopsis* (48, 77, 79, 98, 161, 236). Plant mutants lacking JA biosynthesis or JA-mediated signaling responses are typically more susceptible to RKNs. The *Arabidopsis* mutants in the JA biosynthesis enzymes allene oxide synthase and allene oxide cyclase were more susceptible to RKNs (79, 163). In the compatible interaction with potato, several genes in the JA pathway were downregulated at both 3- and 7-days postinoculation, showing that suppression of JA defense responses correlates with susceptibility to RKNs (140). Tomato plants overexpressing miR319 exhibited reduced endogenous levels of JA and were more susceptible to *M. incognita* (264). A mutant in the JA biosynthetic 13-lipoxygenase gene (*LOX3*) was less susceptible to RKNs compared to the control (175). Conversely, overexpression of JA biosynthesis genes was associated with plant resistance against RKNs. For example, rice plants that overexpressed the allene oxide synthase were significantly less susceptible to RKN infection than wild-type plants (129).

Gleason et al. (79) tried to further define the response using *Arabidopsis* mutants in the JA biosynthetic pathway. The data suggested that the JA precursor 12-oxo-phytodienoic acid (OPDA), in addition to JA, can regulate basal plant defenses against RKNs. More recently, Wang et al. (240) asked where the JA that was important in plant defenses against RKNs was synthesized in the plant. Through a series of grafting experiments, they concluded that basal resistance of roots against RKNs is largely dependent on JA synthesis in shoots but not in roots (240). In fact, their work suggests that the JA produced locally cannot induce an effective basal resistance response. Moreover, during nematode attack, there are systemic electrical and redox signals from roots to leaves that result in increased leaf JA biosynthesis. The JA is then transported from the leaves to the roots, where it helps trigger defense responses against RKNs.

To complicate matters, the data are not always consistent with the role of JA in plant defense against nematodes. The *Arabidopsis lox4* mutant, which exhibited higher levels of JA and increased expression of JA-related genes, was more susceptible to RKNs (175). One possibility is that because the JA biosynthesis pathway has many branches, various electrophilic compounds produced during JA biosynthesis, such as OPDA, could have varying effects on plant resistance to nematodes (78, 79). The different mutants used to assess the role of JA in plant–nematode interactions may be affected in more than just JA levels, leading to confounding infection results (78). This does not fully explain why some groups find that certain mutants (e.g., the JA receptor mutants) are more resistant to RKNs, other groups find them more susceptible to RKNs, and still others have seen no

effect of such a mutation on RKN infections (18, 79, 266). As pointed out by Gheysen & Mitchum (78), some of this confusion may be due to differential experimental setups and the evaluation of different nematode phenotypes as a measure of susceptibility (i.e., galling versus number of females).

The role of JA in plant–nematode interactions may be complicated because of its various functions in the plant. In addition to its role in plant biotic responses, JA plays an important role in plant development and root regeneration, the process by which plants regenerate damaged tissues (266). When RKNs infected *Arabidopsis* roots containing a JAS9-VENUS promoter fusion, the JA-responsive reporter was turned on during early penetration stages and was then reduced to an intermediate level of expression during feeding site formation. This suggests that RKNs are turning on JA-responsive genes during early infection processes. The induction of the JA-responsive reporter also corresponded to increased expression of two genes (*ERF115* and *CYCD6;1*) involved in the tissue regeneration pathway in roots (266). Based on these data, the nematodes are triggering JA-mediated root regeneration pathways, which are required for nematode reproductive success (266). These data also lend credence to the idea that JA can act as a susceptibility factor, although it should be noted that JA acts synergistically with auxin in regulating regeneration responses in roots (248). Localized auxin accumulation is a necessary component of nematode development and giant cell formation (128, 172). Thus, a careful interplay of plant hormones may be regulating host susceptibility to RKNs (192, 260).

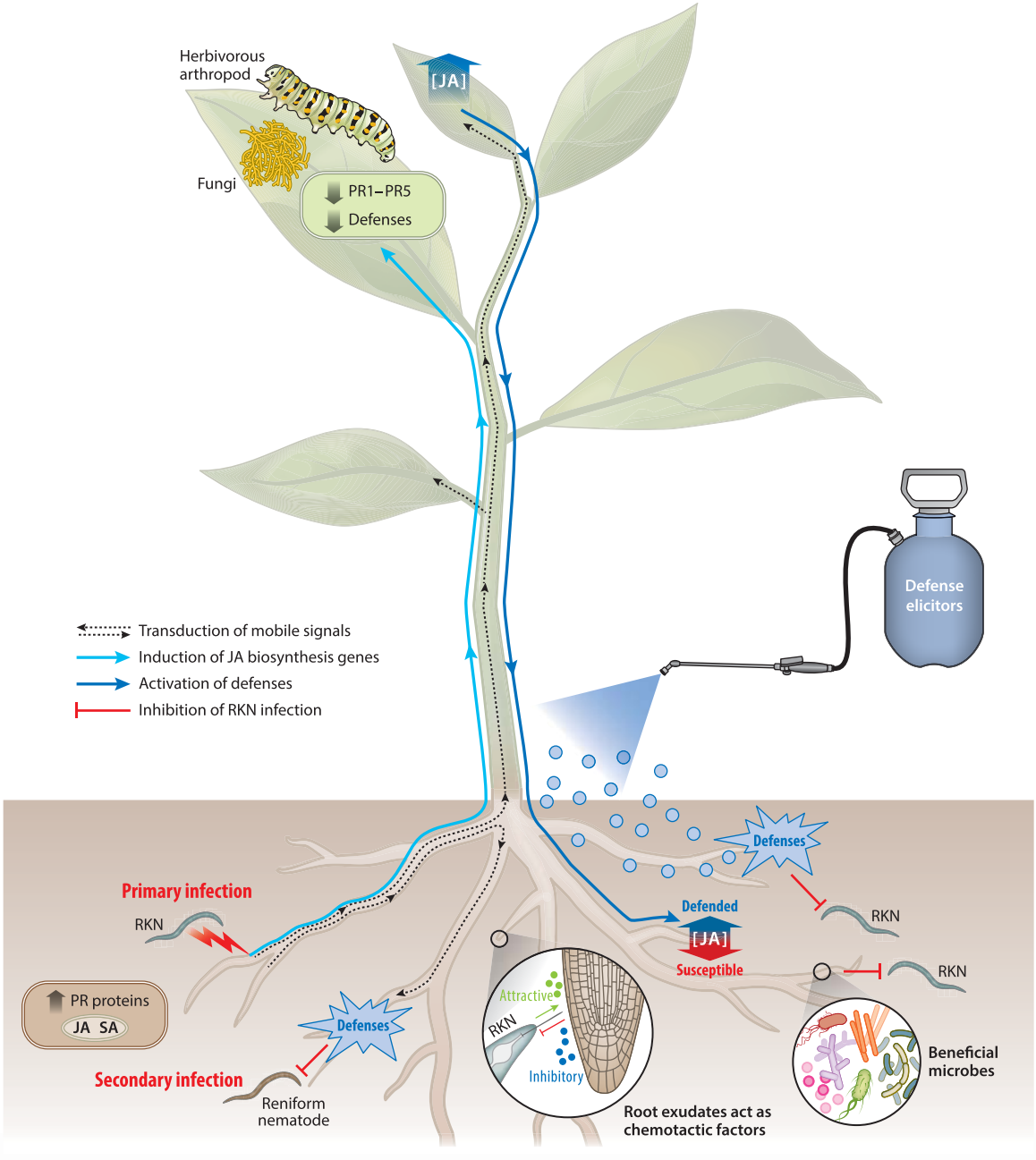
Taken together, a model emerges in which a wounding response triggered by nematode root penetration elicits a systemic signal that leads to JA transport from the leaves to the roots, and depending on the JA concentration and its balance with other hormones, hormone derivatives, ROS, etc., in the cell, the JA can trigger gene expression involved in either basal defenses or host susceptibility (**Figure 3**).

4.5. Induced Resistance and Nematode Infections

When plants are treated with pathogens or chemicals, there is an induced resistance response in which a signal from the primary localized infection travels systemically throughout the plant, priming the uninfected tissue for defense against subsequent infections. There are two main forms of induced resistance: SAR, which is induced by pathogens, and induced systemic resistance (ISR), which is induced by beneficial microbes (120, 239).

One of the main characteristics of SAR is that it involves the accumulation of SA (or SA-like compounds) and activation of PR genes in the plant. RKN infections can elicit SAR in cotton (7), and tomato pretreated with SA gained effective SAR against subsequent RKN infections (157). SAR can be triggered by pathogens or it can be triggered by the exogenous application of certain chemical elicitors (47, 227, 239). Some of these SAR elicitors are analogs of SA, including 2,6-dichloroisonicotinic acid (INA) and benzol(1,2,3) thiadiazole-7-carbothioic acid S-methyl ester (BTH). Pretreating the roots of tomato, eggplant, or pepper plants with SA, INA, or BTH resulted in decreased root galling and nematode reproduction (156). However, foliar sprays of these SAR elicitors were not as effective in priming the plants against RKNs, which may be related to the poor transport of signaling compounds from leaves to roots (150, 156). A pretreatment of plants with the nonprotein amino acid β-aminobutyric acid, through either a foliar spray or root drench, also induced resistance against RKNs in plants, such as tomato (169), cucumber (199), rice (109), and potato (158). In addition to SAR elicitors, a report looking at nematode infections of cotton showed that when *M. incognita* infected roots two weeks prior to *Rotylenchulus reniformis* inoculation, the plants were most resistant to the reniform nematode (7), indicating that nematodes can trigger an SAR that is effective against another nematode species (**Figure 3**).

ISR is triggered by soil-dwelling organisms (bacteria and fungi) that can serve as nematode biocontrol agents (187). One of the most well-studied examples is fungi in the genus *Trichoderma*. Although *Trichoderma* can act as a direct antagonist against RKNs, it can also stimulate plant resistance against RKNs by priming SA- and JA-dependent immune responses (145) (**Figure 3**).



(*Caption appears on following page*)

Figure 3 (*Figure appears on preceding page*)

Above- and belowground defense responses against root-knot nematodes (RKNs). The hormones salicylic acid (SA) and jasmonic acid (JA) are involved during RKN infection. However, their diverse roles in plant development and immunity have provided challenges in isolating their responses solely attributed to RKN infection. Briefly, during nematode attack in the roots, there is increased expression of pathogenesis-related (PR) proteins that serve as marker genes for the hormones SA and JA. Secondly, a mobile signal (*dashed black arrows*) is transduced to distal above- and belowground tissues. In the aboveground tissue, genes involved in JA biosynthesis are induced (*orange arrows*). JA is shuttled back to the roots, where, depending on JA concentrations, it can either activate defenses or act as a susceptibility factor (*blue arrows*). Interestingly, studies have also shown that SA and JA marker genes are reduced in the leaves of RKN-infected plants, leading to increased susceptibility to fungal and herbivorous pathogens. Within distal root tissue, mobile signals have been shown to activate defenses against reniform nematodes. RKNs' potential to modulate hormone concentrations may be dependent on multiple factors, including their infective stage and host genotype. Plants possess chemical cues (root exudates) that vary in their composition and are necessary for RKNs to find and access plant roots. The attractiveness of the roots to a particular RKN species varies depending on the host genotype, infection status, and physiological status. An application composed of defense elicitors induces defense responses within the plant, preventing RKN infection. Additionally, beneficial microbes present in nematode-suppressive soils help prime plant immune defenses against RKNs.

When looking at the effects of RKN root infections on aboveground tissue, nematode infections typically enhance susceptibility to shoot pathogens (19). For example, when RKNs infect rice roots, the decrease in plant immunity in the aboveground tissue makes the rice plant more susceptible to rice blast (131). RKN infections made tomato more susceptible to *Fusarium oxysporum*, with the RKN-infected plants exhibiting more aboveground wilting (93). Work in *Fusarium*-resistant pigeonpea indicated the RKN infections lead to increased aboveground *Fusarium* wilting symptoms (144). At the molecular level, Hamamouch et al. (89) found that RKN infections of *Arabidopsis* roots led to increased expression of SA and JA marker genes in the roots, but these genes were downregulated in the leaves of the infected plants (**Figure 3**). Work in rice showed that by three days post RKN inoculation, there was an attenuation of genes related to ethylene and jasmonate biosynthesis pathways in shoot tissues (130).

Because of the attenuation of defenses in aboveground tissue of RKN-infected roots, there has been interest in whether plants with RKN infestations influence infections with insect herbivores. Most studies indicate that RKN and cyst nematode infections have either a positive influence or no influence on leaf-chewing herbivore performance (19, 245). For example, *M. incognita* infections of tobacco plants increased the weight of the foliar-feeding insect herbivore *Trichoplusia ni*, possibly because the nematodes influence foliar nicotine dynamics, but RKN infections had no effect on performance (116). On cotton, RKN infestations had no effect on its defenses against corn earworm (171). When looking at the effects of RKN root infections on caterpillar (*Spodoptera exigua*) herbivory, researchers found that when roots exhibited visible galling, there was enhanced *S. exigua* performance on the leaves (147). This work indicates that nematode life stage may be influencing the leaf antiherbivore defenses. The influence of RKNs on sap-sucking insect performance is an interesting area of study because both organisms are competing for sugars and metabolites in the phloem as their food source (147); however, research has indicated that RKN infections have variable effects on sap-sucking insect performance. For example, *Brassica nigra* infested with *Meloidogyne hapla* had increased cabbage aphid preference and population growth compared to those on control plants (234). Whereas in *Nicotiana tabacum*, RKNs feeding on roots caused reduced growth and fecundity of aphids (117). When both potato aphids and RKNs coinfected tomato, the RKNs conferred only subtle changes in major defense compounds, and, overall, there was no effect of RKN infection on the reproduction of potato aphid nymphs on leaves (146). Nematode life stage, the order of infection by the different pathogens, and other factors influence the complex systemic defense responses in plants and the outcomes of those responses.

4.6. Effector-Triggered Immunity

Effector-triggered immunity (ETI) is typically mediated by dominant resistance (*R*)-genes that encode intracellular receptors that perceive pathogen effectors and initiate programmed cell death within the host (121). Most of the identified plant *R*-genes encode some variation of nucleotide binding site–leucine-rich repeat receptors (NLRs), which have diversified to detect a wide variety of pathogen effectors (111). These canonical receptors either directly or indirectly interact with secreted pathogen effectors and then trigger a signaling cascade that leads to hypersensitive cell death. Although the full mechanisms are still largely unknown, and likely vary to some degree between receptors, it has become clear that multiple NLRs, receptors, and coreceptors work together to perceive specific pathogen proteins and initiate a cell death response (112). In the context of RKN–plant interactions, *R*-gene-mediated responses generally occur within the cells of the nematode's feeding site, leading to the death of the nematode. But different RKN *R*-genes can vary widely with respect to their specificities, mechanisms, and durability in the field.

R-genes that confer resistance to RKNs have been identified in different crops over the past two decades (**Table 3**). The vast majority of these *R*-genes have yet to be cloned, with few exceptions: the *Mi1.2* gene from tomato was shown to encode an NLR that confers resistance to *M. incognita*, *M. javanica*, and *M. arenaria* (152). Interestingly, *Mi1.2* has also been shown to provide resistance to aphids and whiteflies (167, 194). The *Ma* gene from myrobian plum was also shown to encode an NLR receptor (45), and it is more broadly effective than any previously characterized source of RKN resistance, conferring resistance against all the species so far tested from *Meloidogyne* clade I, including *Meloidogyne enterolobii*, *M. floridensis*, and *Meloidogyne ethiopica* (45, 65). Although few other RKN *R*-genes have been cloned to date, many have been mapped to clusters of NLR genes. Upward of a dozen RKN *R*-genes have been identified in solanaceous crops alone, and nearly all have been mapped to colinear clusters of NLR genes within tomato, pepper, and potato genomes (59).

The specificity of *R*-genes can vary widely. Although some genes, like *Mi1.2* and *Ma*, provide resistance against multiple RKN species, other genes function only against specific species or even specific isolates. In pepper, the *Mech2* gene provides resistance against specific populations of *M. chitwoodi* (17), whereas multiple other *R*-genes map within the same NLR genomic region but provide resistance against *M. incognita*, *M. javanica*, and *M. arenaria* (36, 59, 233). The specific molecular mechanisms that underlie the specificities of these *R*-genes are still largely unknown, but based on evidence from other pathosystems the resistance is almost certainly dependent on the different effectors secreted by these RKN species.

Although most characterized *R*-genes induce a hypersensitive response to RKNs, they vary with respect to their timing and mode of action during parasitism. Histological comparisons of two *R*-genes from pepper, *Me1* and *Me3*, provided evidence that *M. incognita* tended to develop longer in roots carrying *Me1* resistance, but both genes reduce the number of J2 nematodes penetrating the root and both induce strong hypersensitive responses to the nematode (23, 88). In contrast, an *R*-gene in cowpea allows *M. incognita* to penetrate and develop feeding sites that subsequently degrade at later stages of infection, ostensibly without a classical hypersensitive response (51). It is thought that these differences in *R*-gene mode of action could affect the resistance durability against RKNs.

The durability of different *R*-genes against RKNs varies considerably. Over-reliance on the tomato *Mi1.2* gene to manage RKNs has resulted in the selection for virulent (resistance breaking) populations in the field (87, 232, 237), and similar effects have been noted for both the *N* and *Me3* genes in pepper (29, 193). However, the pepper *Me1* gene has been found to be substantially more robust than the former two *R*-genes, despite the fact that it resides in the same genomic region

Table 3 Root-knot nematode (RKN) resistance genes/loci

Gene/loci	Mapped	Cloned	Likely identity	Host plant species	Confirmed avirulent RKN species	Primary reference
Ma	Yes	Yes	NLR	Plum	*Meloidogyne incognita*, *Meloidogyne javanica*, *Meloidogyne arenaria*, *Meloidogyne enterolobii*, *Meloidogyne floridensis*, *Meloidogyne ethiopica*	45
RMia	Yes	No	NLR	Peach	*M. incognita*, *M. arenaria*, *M. ethiopica*	64
Rjap	Yes	No	NLR	Plum	*M. incognita*, *M. javanica*, *M. arenaria*, *M. floridensis*	44
RMja	Yes	No	NLR	Almond	*M. javanica*, *M. arenaria*, *M. enterolobii*, *M. ethiopica*	235
PkMi	Yes	No	NLR	Peach	*M. incognita*	31
Mf1	Yes	No	NLR	Peach	*M. floridensis*	143
mf3	Yes	No	Unknown	Peach	*M. floridensis*	143
Mi1.2	Yes	Yes	NLR	Tomato	*M. incognita*, *M. javanica*, *M. arenaria*, *Meloidogyne luci*, *M. ethiopica*	152
Mi-3	Yes	No	NLR	Tomato	*M. incognita*, *M. javanica*, *M. arenaria*	250
Mi9	Yes	No	NLR	Tomato	*M. incognita*, *M. javanica*, *M. arenaria*	4
RMC1	Yes	No	NLR	Potato	*Meloidogyne chitwoodi*	28
RMC2	Yes	No	NLR	Potato	*M. chitwoodi*	105
Mfa	Yes	No	NLR	Potato	*Meloidogyne fallax*	8
Mh-chcA	Yes	No	NLR	Potato	*Meloidogyne hapla*	220
Mh-tar	Yes	No	NLR	Potato	*M. hapla*	220
N	Yes	No	NLR	Pepper	*M. incognita*, *M. javanica*, *M. arenaria*	73
Me1	Yes	No	NLR	Pepper	*M. incognita*, *M. javanica*, *M. arenaria*	230
Me2	No	No	NLR	Pepper	*M. javanica*	91
Me3/7	Yes	No	NLR	Pepper	*M. incognita*, *M. javanica*, *M. arenaria*	36
Me4	Yes	No	NLR	Pepper	*M. arenaria*	59
Me5	No	No	NLR	Pepper	*M. javanica*	91
Mech1	Yes	No	NLR	Pepper	*M. chitwoodi*	59
Mech2	Yes	No	NLR	Pepper	*M. chitwoodi*	59
mj	Yes	No	Unknown	Cucumber	*M. javanica*	197
Rk-1	Yes	No	NLR	Cowpea	*M. incognita*, *M. javanica*, *M. arenaria*, *M. hapla*	200
Rk-2	Yes	No	NLR	Cowpea	*M. incognita*, *M. javanica*	164
Mj-1	Yes	No	NLR	Carrot	*M. incognita*	178
Mj-2	Yes	No	NLR	Carrot	*M. incognita*, *M. javanica*	2
NO NAME	Yes	No	Unknown	Sweet potato	*M. incognita*	170
GHNTR1	Yes	Yes	NLR	Tobacco	*M. incognita*	256
Rk1	Yes	No	NLR	Tobacco	*M. incognita*, *M. arenaria*	254
Rk2	No	No	NLR	Tobacco	*M. javanica*	186
Tifguard	Yes	No	NLR	Peanut	*M. arenaria*	46
Rkn-mn1	Yes	No	NLR	Wheat	*Meloidogyne naasi*	11
TRKR	Yes	No	NLR	Clover	*Meloidogyne trifoliophila*	12
SacMi	Yes	Yes	NLR	Eggplant	*M. incognita*	267
Mex-1	Yes	No	NLR	Coffee	*Meloidogyne exigua*	5

Abbreviation: NLR, nucleotide-binding site leucine-rich repeat.

and works against the same RKN species (29, 60). The durability of a specific *R*-gene is thought to be directly related to the fitness cost born by the pathogen, which must lose the matching avirulence (*avr*) gene (usually an effector) to avoid detection by the *R*-gene (182). Further research is needed to understand the mechanisms that underlie these variations observed in RKN *R*-gene durability.

The biggest hurdle to understanding ETI against RKNs has been the lack of known *avr* genes that trigger *R*-gene-dependent immunity. The sole exception is *Cg-1*, which when silenced via RNAi was shown to enhance the virulence of *M. javanica* on plants carrying the *Mi1.2* gene (81). *Cg-1* lies within a TE, and the virulent *M. javanica* isolate has a large deletion of at least 2 Kb that includes *Cg-1*. Although the mechanism is still unclear, it is possible that *Cg-1* regulates expression of a *cis*-linked effector that triggers *Mi1.2* resistance (83). Knowledge of nematode *avr* genes and the mechanisms that allow *R*-genes to detect them would enhance our understanding of the specificity and durability of RKN resistance and enhance our ability to design RKN resistance in plants.

CONCLUDING REMARKS

The intimate and prolonged interaction between RKNs and their host plants requires that these parasites carefully coordinate the production and secretion of their effectors to overcome the complex plant immune responses. Recent advances in genomics and transcriptomics have helped advance our identification of RKN effectors and enhanced our understanding of nematode evolution. There has been recent progress in RKN effector characterization, particularly by using the various in planta tools for functional analysis and methods for studying protein–protein interactions (71). There is much we still do not fully understand about how RKNs interact with their host plants (see Future Issues). As we increase the number of RKN genomes that are publicly available, comparative genomics and studies in effector diversity will help better our understanding of plant–RKN interactions.

FUTURE ISSUES

1. Many of the most damaging root-knot nematode (RKN) species are mitotically parthenogenetic. Questions remain as to how these species are able to maintain genetic diversity and plasticity and be so successful in such a wide variety of hosts and environments.
2. Factors that determine RKN host range and their ability to adapt to new environments at the genetic and epigenetic levels need to be further examined.
3. The host-plant targets of RKN effectors are still largely a mystery. Identifying these targets would help to unravel the steps needed for compatible RKN–plant interactions, as well as the cascade of defense responses involved in incompatible interactions, and could ultimately lead to strategies for designing plants with resistance to RKNs.
4. Because effector characterization tools tend to measure plant defense responses against nematodes, most characterized effectors have been described in terms of their effects on plant immunity. The new challenge will be to better characterize the effects of these effectors on giant cell formation and maintenance and identify whether and which conserved pathways are required for giant cell formation.

5. Methods for RKN transformation are needed and would not only greatly facilitate RKN research of RKN effectors but also provide important insight into RKN biology, such as factors involved in chemotaxis and sex differentiation.
6. Although genomes are becoming available for more RKN species, their complexity makes accurate genome assembly and gene annotation difficult. A concerted international effort among RKN experts is needed to develop better genome and transcriptome resources and would greatly benefit the community.

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