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REVIEW ARTICLE

Plant defense responses in monocotyledonous and dicotyledonous host plants during root-knot nematode infection

Arnika Przybylska · Aleksandra Obrępalska-Stęplowska

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Abstract

Background Root-knot nematodes (RKNs) – *Meloidogyne* spp. – are a group of nematodes distributed worldwide that infect monocotyledonous and dicotyledonous crop species. Plant responses to RKNs have been described in many studies of various host plants. In the course of parasitism, RKNs induce the transcriptional reprogramming of host cells to establish giant cells. Nematode attack induces many mechanisms in host plants, including pathogen-associated molecular pattern (PAMP)-triggered immunity (PTI) and effector-triggered immunity (ETI). Research on plant-RKN interactions has shown the activation and suppression of the expression of genes encoding various defense-related proteins.

Scope and conclusions In this review, our goal is to critically summarize current knowledge on monocotyledonous and dicotyledonous plant-*Meloidogyne* interactions, including data on the role of RKN effectors and nematode PAMPs in host plant defense responses.

Keywords Plant-nematode interaction · Plant defense · Effectors · *Meloidogyne* · Root-knot nematodes (RKNs)

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A. Przybylska · A. Obrępalska-Stęplowska (✉)
Department of Molecular Biology and Biotechnology, Institute of Plant Protection – National Research Institute, 20 Węgorka Street, 60-318 Poznań, Poland
e-mail: olaob@o2.pl

Introduction

Economic significance of root-knot nematodes (RKNs)

Meloidogyne spp. Göldi 1887 (Tylenchidae: Tylenchus) nematodes, also known as RKNs, are a group of nematodes distributed worldwide that contains more than 60 described species. These polyphagous and highly adapted plant parasites have a very wide host range. The most economically important RKN species include *Meloidogyne hapla*, *M. incognita*, *M. javanica,* and *M. arenaria* (Jones and Goto 2011; Moens et al. 2009). Another well-analyzed *Meloidogyne* species is *M. graminicola*, which is primarily a pathogen of rice (Kumari et al. 2016; Kumari et al. 2017; Kyndt et al. 2012; Nahar et al. 2011). There are also two quarantined RKN species included on the EPPO A2 list: *M. chitwoodi* and *M. fallax* (A2 List of pests recommended for regulation of quarantine pests 2017). These two species parasitize monocotyledons and dicotyledons, including several species of crop plants, such as potatoes, carrots and tomatoes (EPPO 2016). *M. chitwoodi* has been reported in Argentina, Belgium, France, Germany, the Netherlands, Portugal, the USA, Mexico, and South Africa, while *M. fallax* has been reported in Belgium, France, Germany, the UK, Switzerland, New Zealand, Australia, and South Africa (EPPO 2016). The main crops infected by RKNs in fields or in greenhouses are potato, tomato, carrot, rice, sunflower, corn, sugar beet, pepper, and tobacco; however, the damage caused by RKNs is often overlooked, and information on their economic impact on agriculture is limited (Wesemael et al. 2011).

The responses of plants to *Meloidogyne* infection have been analyzed by many research groups using different host plants. In this review, we summarize the information obtained so far on plant-*Meloidogyne* interactions. The role of nematode effectors in this process is also addressed.

Infection process

RKNs in the J2 invasive stage migrate in the soil, penetrate the root tip epidermis, and move inside roots through intercellular spaces. They establish giant cells by the transcriptional reprogramming of the parenchymal cells surrounding the phloem (Jones 1981). Giant cells are formed in a similar way for all *Meloidogyne* species and they have a feeding function. They are generated without cytokinesis but through sequential mitoses what leads to an increase of a size of a cell and a number of nuclei (Rodiuc et al. 2014). After J2 invasion, the expression of a broad spectrum of genes with many different functions is induced, which is correlated with wound and defense responses; changes in the cell wall, cell cycle and cytoskeleton organization; and morphological symptoms (Gheysen and Fenoll 2002).

Chemical and physical barriers and cell metabolism reprogramming

The first line of the plant defense system after RKN infection includes physical barriers, such as the cell wall, waxes or hairs, and chemical barriers, including enzymes or secondary metabolites (Jones and Dangl 2006). The first barrier to overcome by nematodes is usually the cell wall, which could also be covered with a cuticle. To overcome this obstacle, nematodes use a combination of mechanical penetration with its hollow mouth stylet together with an arsenal of virulence factors, including many cell wall-degrading enzymes (Jones and Goto 2011; Malinovsky et al. 2014). Among them, the presence and expression of plant cell wall degrading factors, such as β-1,4-endoglucanase, a functional polygalacturonase or a pectate lyase, was confirmed (Huang et al. 2005; Jaubert et al. 2002; Rosso et al. 1999). The primary cell wall is composed of hemicellulose polysaccharides and the heteropolysaccharide pectin interwoven within a cellulose microfibril network (Endler and Persson 2011). The walls of the different types of plant cells are specifically adapted to particular functions (Bradley et al. 1992). The composition of the plant cell wall can be altered by environmental stimuli in response to biological stresses (Bradley et al. 1992). Thus, mechanical wounding, infection, or elicitors obtained from pests and pathogens can stimulate the synthesis of lignin in peripheral tissues. These changes in cell wall composition constitute a part of inducible defense mechanisms (Malinovsky et al. 2014). In response to an attack, plants may deposit phenolic and callose complexes which have a reinforcing function, and produce toxic compounds (Hückelhoven 2007). When this barrier is overcome, other plant surveillance systems are activated (Malinovsky et al. 2014). Interestingly, Shah et al. (2017) hypothesized that RKN do not cause damage during their migration inside the root. Moreover, Teixeira et al. (2016) found that Arabidopsis lines with altered damage perception do not show any change in susceptibility to RKNs infection. Another recent study investigated the role of suberin and lignin-based Casparian strips and the fate of endodermis in roots during RKN infection. On the basis of the obtained results, the authors assumed that a functional endodermis is an obstacle to nematode penetration and contributes to defense mechanisms against RKNs (Holbein et al. 2019).

During feeding site formation (giant cells), nematodes induce the reprogramming of the metabolism of the roots by adjusting the expression patterns of root tip-specific genes or induce the expression of genes that are not usually expressed in the roots. A characteristic feature of giant cells is their outstanding isotropic growth (Sobczak et al. 2011). Such cell expansion requires extensive and coordinated cell wall remodeling. The expression of many cell wall-modifying enzymes, including expansins, endoglucanases, extensins, hydrolases, and structural proteins, is changed after *Meloidogyne* spp. infection (Sobczak et al. 2011). Wall extension involves the loosening of the cellulose/cross-linking glycan network, which is achieved through endo-β-D-glucanases, expansins and xyloglucan endotransglycosylases (Caillaud et al. 2008). The concomitant deposition of newly synthesized cell wall material is associated with this loosening process, and throughout feeding site development, further modifications to the cell wall result in wall thickening and the development of wall ingrowths (Caillaud et al. 2008).

Molecular mechanisms of plant defense

The presence of plant pathogens is revealed by recognizable molecular signals called pathogen-associated

molecular patterns (PAMPs) located on their surfaces as well as by damage-associated molecular patterns (DAMPs) released by the disrupted host plant tissues. PAMPs are often perceived at low concentrations and induce specific defense responses (Manosalva et al. 2015). The first layer of defense is designated innate immunity or basal resistance, which can be triggered once the plant cells recognize PAMPs and DAMPs. PAMPs and DAMPs are recognized by pattern recognition receptors (PRRs), resulting in pattern-triggered immunity (PTI), which can halt further colonization by RKNs (Jones and Dangl 2006). One of the first PRRs described for nematodes was a leucine-rich repeat (LRR)-RLK encoded by the *Arabidopsis thaliana nilr1* gene (nematode-induced LRR-RLK 1), which is widely conserved among dicots and monocots (Mendy et al. 2017). When innate immunity is suppressed by pest effectors, effector-triggered susceptibility (ETS) is induced, and a given effector is recognized by the appropriate proteins in plants, resulting in effector-triggered immunity (ETI) (Jones and Dangl 2006). On the other hand, when the host presents disease resistance, ETI is accelerated, and the PTI response is amplified. Moreover, it usually results in a hypersensitive cell death response (HR) at the infection site (Jones and Dangl 2006). RKNs evolved to avoid ETI by acquiring additional effectors that may suppress ETI as well as by shedding or diversifying the recognized effector gene. The suppression of ETI by effectors is effective in a susceptible host and results in ETS, which allows the nematode to infect the plant (Jones and Dangl 2006).

Jasmonic acid and salicylic acid pathways

Part of the PTI response induced by PAMPs during plant resistance occurs via conserved signal transduction mechanisms, including the activation of mitogen-activated protein kinases (MAPKs), generation of reactive oxygen species (ROS) and activation of the salicylic acid (SA) and jasmonic acid (JA) signaling pathways (Manosalva et al. 2015). SA regulates many genes encoding pathogenesis-related (PR) proteins, including PR1 and most acidic PR proteins, while the JA signaling pathway affects the expression of genes encoding defensin, thionin, PR3 and PR8 proteins (Reymond and Farmer 1998). Recent studies have suggested that JA may play a dominant role in plant-pathogen interactions in the roots of monocotyledons and dicotyledonous plants, including plant-RKN interactions (Fujimoto et al. 2011; Mendy et al. 2017; Nahar et al. 2011). Cross talk between the JA/ET and SA defense signaling pathways has been shown to optimize the response against a single attacker (Spoel et al. 2007).

The pathways induced during the plant response to nematode infection are summarized in the scheme below (Fig. 1).

Plant resistance genes against RKNs

As described above, the HR reaction can be activated by the gene-for-gene resistance mechanism. The gene-for-gene concept assumes that for each gene that causes a reaction in the host, there is a corresponding gene in the parasite that is responsible for pathogenicity (Flor 1971). The appropriate activation of host defense responses is mediated by disease resistance (*R*) genes that have a dual function of directly or indirectly recognizing specific avirulence (*avr*) factors and subsequently activating host signal transduction pathways, leading to physiological changes that make conditions in the host plant unfavorable for nematode survival (Kaloshian et al. 2011). The most well-known plant *R* gene against RKNs is the *Mi-1.2* gene from tomato. In plants carrying this gene, HR stops the nematode from establishing a feeding site. *Mi-1.2*-mediated resistance can dramatically reduce RKN survival, reproduction and gall induction (Corbett et al. 2011). The *Mi-1.2* gene is present in many tomato cultivars and confers resistance to three common RKN species, *M. incognita, M. javanica* and *M. arenaria*, and three insect species, potato aphid (*Macrosiphum euphorbiae*), sweet potato whitefly (*Bemisia tabaci*) and tomato psyllid (*Bactericera cockerelli*) (Corbett et al. 2011). Importantly, *Mi-1.2*-mediated resistance has some limitations. Tomato plant resistance is dependent on soil temperature. At temperatures higher than 28 °C, the *Mi-1.2* gene becomes inactive, and at 32 °C, the plant becomes sensitive (Dropkin 1969). Moreover, *Mi-1.2*-mediated resistance is effective only against the three aforementioned species but is ineffective against other harmful RKN species, such as *M. enterolobii* and *M. hapla* (El-Sappah et al. 2019; Kiewnick et al. 2009). There are also reports on nine other root-knot nematode resistance genes in wild species of tomato: *Mi-2* to *Mi-9* and *MiHT*, but unlike *Mi-1.2,* they have not been successfully transferred to cultivated tomatoes (Rashid et al. 2017; Wu et al. 2009). There are also some studies that indicate that the *Me* resistance gene in pepper (*Capsicum annuum*) confers resistance to RKNs (Djian-Caporalino et al. 2007)

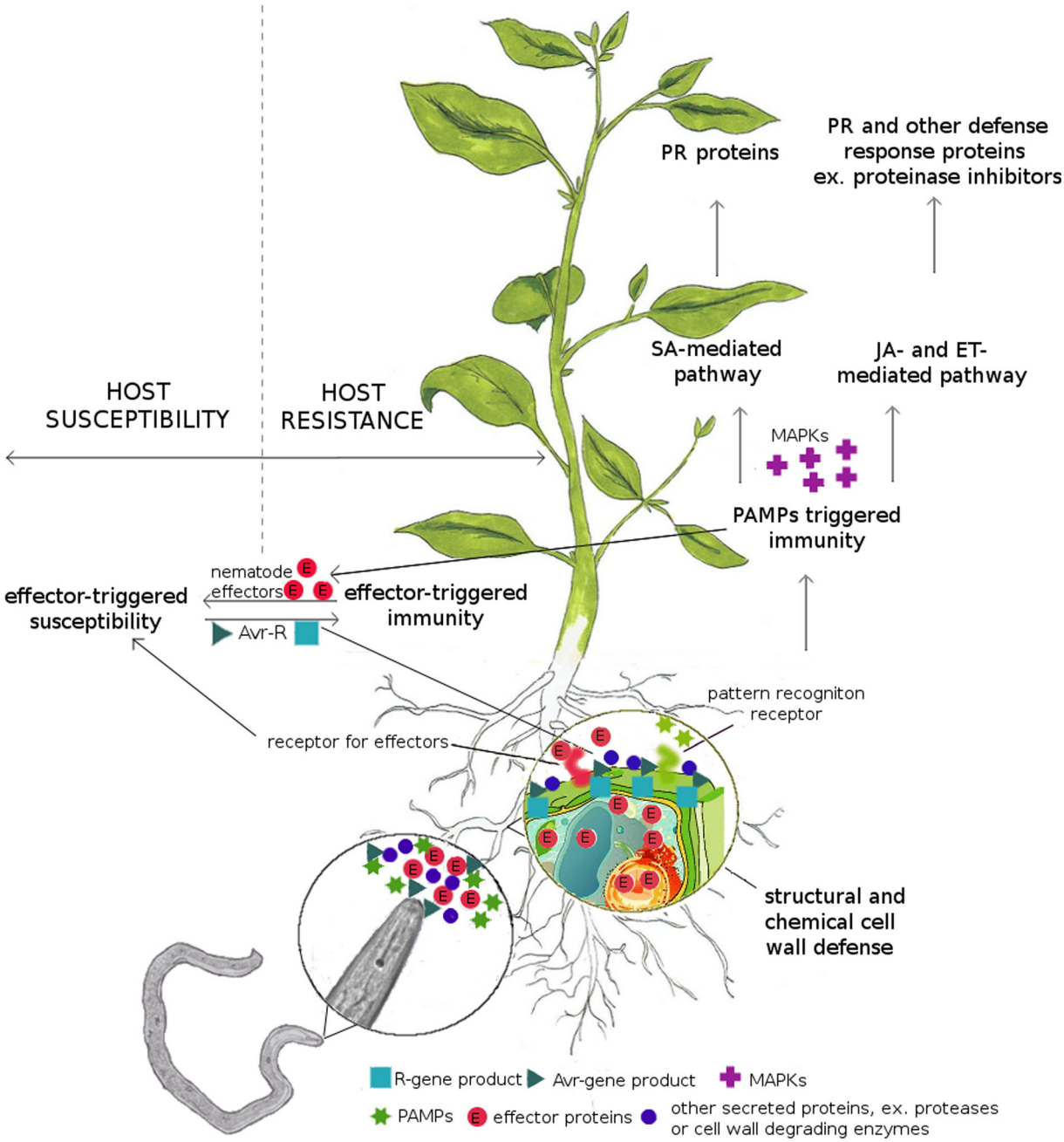

Fig. 1 Scheme of the pathways induced during the plant response to nematode infection. PR proteins – pathogen-related proteins, SA – salicylic acid, JA – jasmonic acid, ET – ethylene, *R* gene – plant resistance gene, *avr*-gene – gene encoding an RKN avirulence factor, MAPKs - mitogen-activated protein kinases, PAMPs - pathogen-associated molecular patterns

and that the *Mex-1* gene in coffee (*Coffea arabica*) confers resistance to *M. exigua* (Noir et al. 2003).

Several review articles address the interactions between plants carrying resistance genes and RKN species (Barbary et al. 2015; El-Sappah et al. 2019; Rashid et al. 2017; Roberts 1992; Seid et al. 2015). In addition, there are also recent review articles presenting in a broader sense some topics associated with the field of plant parasitic nematode-plant interactions (Kaloshian and Teixeira 2019; Sato et al. 2019; Siddique and Grundler

2018), including nematode effectors and other secreted proteins (Mejias et al. 2019; Vieira and Gleason 2019). Here, in addition to general aspects of plant-RKN interactions, we focused largely on discussing the processes taking place separately in monocotyledonous and dicotyledonous hosts and RKNs.

Nematode PAMPs that activate the host plant PTI response

Ascarosides and elicitor(s) presented in NemaWater (water obtained after removing the preinfected J2 larvae after 24 h of incubation (Mendy et al. 2017)) are the only nematode PAMPs that have been described so far. This PAMPs activate the initial of PTI responses in their hosts (Mendy et al. 2017).

Ascarosides constitute an important group of proteins produced by RKNs that may affect plant defense responses (Manosalva et al. 2015). More than 200 different ascarosides derived from over 20 nematode species have been described, indicating that ascarosides are a highly conserved group of nematode proteins. Ascarosides are recognized by plants as NAMPs (nematode-associated molecular patterns), which induce the activation of conserved immune responses in host plants and, as consequence enhance plant resistance to a wide spectrum of pests and pathogens. The protein designated asrc#18 was analyzed in more detail. The authors who studied ascr#18 suggested that the systemic induction of defense genes and RKN resistance in leaves following root application of ascr#18 may indicate that this protein moves from the roots to the leaves and/or that ascr#18 induces the synthesis of a mobile signal in the roots that is then transmitted to the leaves to activate immune responses (Manosalva et al. 2015).

A study on NemaWater showed that NemaWater treatment triggers PTI responses, such as immune gene expression and ROS burst and, as a consequence, can inhibit seedling growth (Mendy et al. 2017). The obtained results may suggest the presence of putative elicitor(s) in NemaWater. The identity of the elicitor(s) remains unknown, but according to the results of the study, it may be a heat-sensitive protein(s) (Mendy et al. 2017). Using NemaWater is a relatively new and unique approach and may have some limitations in the accurate representation of in vivo infection processes.

Role of RKN effector proteins in host plant defense responses

Nematodes have three large specialized secretory gland cells in the esophagous, one dorsal and two subventral, and these cells are the principal sources of effectors essential for phytonematodes to parasitize plants (Hussey 1989). In recent years, several research papers have focused on the characterization of nematode effectors. The first nematode parasitic genes expressed in the esophageal gland cells were identified in the cyst nematodes *Heterodera* spp. and *Globodera* spp., which encode β-1,4-endoglucanases (cellulases) (Smant et al. 1998; Yan et al. 1998). It has also been suggested that effectors secreted by nematodes contribute to plant defense suppression during infection (Davis et al. 2008; Rosso et al. 2012). Huang et al. (2003) obtained a profile of 37 cDNA sequences encoding candidate effector proteins expressed exclusively within the secretory esophageal gland cells of *M. incognita* throughout the parasitic cycle by combining the expressed sequence tag (EST) analysis of a gland-cell LD-RT-PCR cDNA library with high-throughput in situ screening of the clones encoding a signal peptide for secretion. To date, several effectors encoded by RKNs have been reported. Their mode of action and different functions in plant-RKN interactions are briefly described below, and the effectors are also discussed in other review articles in additional detail (Mejias et al. 2019; Vieira and Gleason 2019).

One of the most important modes of action of RKN effectors is the suppression of plant cell death associated with PTI or/and ETI response. To effectors with such a function belong the effector Mi-CRT, calreticulin, described for *M. incognita* (Dubreuil et al. 2009; Jaouannet et al. 2013). The knockdown of gene encoding Mi-CRT in preparasitic infective juveniles resulted in a reduced ability of the nematodes to induce galls on tomato (Dubreuil et al. 2009). In addition, Jaouannet et al. (2013) in their research on *A. thaliana* provided evidence for the manipulation of the plant basal immune response by the Mi-CRT protein, with a direct effect on PTI suppression. They observed that the induction of marker defense genes was strongly suppressed in plants in which Mi-CRT was secreted by the nematodes to the apoplast. Another *M. incognita* effector that is able to manipulate plant immunity is the Msp40 protein (Niu et al. 2016). It was demonstrated that Msp40 may suppress PTI and ETI by targeting

common components, such as the elements of MAPK cascades, or by interacting with diverse components of these two immunity pathways in *A. thaliana* plants. The expression of another protein, the MiSGCR1 effector, which is secreted by *M. incognita* and is specifically involved in the early stages of plant-nematode interaction, may suppress cell death (Nguyen et al. 2018). There is also an effector, MeTCTP, described for *M. enterolobii* (Zhuo et al. 2017). The TCTP family of proteins is highly conserved and has been suggested to have various functions, such as calcium binding, histamine release, protection against stress, antiapoptosis and microtubule stabilization (Zhuo et al. 2017). The effector MeTCTP is expressed specifically within the nematode dorsal esophageal gland and is localized to the cytoplasm of plant cells. The authors discovered that host susceptibility increased with an increased expression level of MeTCTP, suggesting that the MeTCTP effector contributes to *M. enterolobii* virulence. Additionally, this protein has been suggested to function in the suppression of defense-related host cell death. Interesting results were also obtained for RKN lectin, which was experimentally demonstrated to be secreted into host plants as an effector, e.g., Mg01965 from *M. graminicola*. This protein is released during the early stages of infection and accumulates in the apoplast, where it can suppress PTI, possibly by binding to certain apoplastic sugars and by interfering with sugar signals (Zhuo et al. 2019). Effector proteins can also be conserved between some economically important *Meloidogyne* species, such as *M. hapla*, *M. incognita*, and *M. graminicola*, as described in the example of the Msp18 protein (Grossi-de-Sa et al. 2019). The results of this study suggested that this effector suppresses programmed cell death mediated by immune defenses to achieve successful parasitism in the host plant.

Other effectors can interfere with SA- and/or JA-mediated pathways. An example of that kind of protein can be the *M. incognita* Misp12 effector examined on *Nicotiana benthamiana* plants. This protein was suggested to be involved in the downregulation of SA- and JA-dependent defense response genes to enhance *M. incognita* parasitism during the mature stages of the nematode life cycle (Xie et al. 2016). On the other hand, Wang et al. (2018) characterized a novel chorismate mutase from *M. incognita*, Mi-CM3, which regulated the SA pathway in such a way that enhanced nematode parasitism. Mi-CM3 disrupts SA biosynthesis and SA-mediated defense in the plant tissues in which this gene is expressed.The presence of a chorismate mutase was also reported in *M. javanica* species (MjCM-1), and the authors assumed that this protein has the potential to be a multifunctional enzyme responsible for promoting nematode pathogenicity (Doyle and Lambert 2003). In addition, the MiISE5 effector, described in *M. incognita,* may participate in the manipulation of several pathways in host plants during the infection process, such as the regulation of transcription and the inhibition of the expression of multiple marker genes in response to various biotic and abiotic stimuli. In addition, the overexpression of MiISE5 in *Arabidopsis* also modified host hormones from both the JA- and SA-mediated signaling pathways. These lines of evidence suggest that MiISE5 plays an important role in nematode parasitism (Shi et al. 2018b). The same authors described that the MiISE6 effector protein, which was highly expressed in the early parasitism stages, has a functional signal peptide and can localize to the nucleus. It was suggested that MiISE6 can enhance nematode parasitism by interfering with multiple signaling pathways in plants, especially by the upregulation of genes encoding the jasmonate ZIM-domain (JAZ) protein family, which are known to be repressors of the JA signaling pathway (Shi et al. 2018a).

Some other effector proteins can also bind actin monomers to manipulate plant actin in conjunction with the enhancement of the expression of the genes encoding endogenous actin-depolymerizing factor (ADF), as described for MiPFN3 in *M. incognita* by Leelarasamee et al. (2018). Their results suggested that diminishing actin network density was important to facilitate nematode feeding. There is also a report about the Mh265 effector protein from *M. hapla* (Gleason et al. 2017). The authors found that *A. thaliana* expressing this protein exhibited enhanced susceptibility to RKNs, which may indicate the role of this protein in plant defense mechanisms. Moreover, the effector protein MjTTL5 from *M. javanica* promotes plant ROS-scavenging activity and suppresses host defenses (Lin et al. 2016). For another effector protein, Mg16820, encoded by *M. graminicola* and localized in the apoplast, cytoplasm and nucleus of plant cells, the potential target protein in plants is dehydration-stress inducible protein 1 (DIP1), which was reported as an ABA-responsive gene (Naalden et al. 2018). Mg16820 functions in the apoplast and in the cytoplasm by interacting with components important in ROS defense signaling, which may suggest that this protein is able to

interfere with two different mechanisms to suppress the immune system of the plant. Another mode of action has MgGPP protein from *M. graminicola*. Experimental evidence has suggested that MgGPP may be secreted into host plants during parasitism; first, MgGPP is secreted into the cell apoplast before entering the cells, then it is targeted to the endoplasmic reticulum (ER), where N-glycosylation and C-terminal proteolysis occur, and finally, it is translocated from the ER to the nucleus (Chen et al. 2017). Effector proteins can also play a role in the suppression of basal defense in plants in other ways, such as MgMO237 encoded by *M. graminicola*. This effector interacts with three rice proteins (OsGSC, OsCRRSP55 and OsBetvI) that are all host defense-related proteins (Chen et al. 2018).

The described RKN effectors and their localization and function in parasitism are summarized in Table 1.

Plant defense mechanisms induced in monocotyledonous plants

Many studies have been conducted to elucidate the plant response to *Meloidogyne* infection for both mono- and dicotyledonous plants; however, most of the research has been conducted on dicotyledonous plants (Table 1). Two types of interactions with different host varieties can be considered: compatible, where the host plant is susceptible to RKN infection, and incompatible, where the host plant is resistant.

Compatible interactions

Most of the studies on the response of monocotyledons to RKN infection utilized the *M. graminicola-Oryza sativa* pathosystem. One study showed that *M. graminicola* and *M. incognita* nematodes were able to suppress the expression of rice basal defense genes at early stages after infection (Nguyễn et al. 2014). The authors determined that the gene *MAPK5a* encoding the kinase involved in phosphorylation cascades in PTI and ETI was downregulated after infection. Other studies conducted on rice reported that part of the PTI signaling response, which is a JA-mediated pathway, plays an important role in plant defense responses. In addition, the consistent local downregulation of major defense-related genes, such as *PR10*, *WRKY45* or *PR1b,* in nematode-induced root galls at an early stage of infection has been reported (Nahar et al. 2011). Some authors also indicated that both JA- and SA-mediated rice defenses are activated during the early stages of *M. graminicola* infection, but these responses were suppressed during the later stages of infection (Kumari et al. 2016, 2017). In another study conducted on rice, the genes encoding *AOS2* (allene oxide synthase, one of the enzymes in the JA biosynthesis pathway), *PAD4* (phytoalexin deficient 4, part of the SA-dependent response) and *WRKY13* (positive transcriptional regulator of defense genes) were downregulated after nematode infection (Nguyễn et al. 2014). Interestingly, the significant upregulation of ABA response marker genes, such as *OsZEP* or *OsLip9*, in the sensitive variety of *O. sativa* infected by *M. graminicola* was observed (Kyndt et al. 2017). On the other hand, during *M. arenaria* infection in maize, changes in the expression levels of genes encoding proteins involved in both the SA and JA pathways and the downregulation of other defense-related genes, such as peroxidase, catalase or superoxide dismutase, were reported at the early stage of nematode infection (as early as 1 dpi) (Przybylska et al. 2018). The results obtained for *Musa acuminate* provided evidence for the early host defense responses that involved the downregulation of the expression level of the genes encoding ROS and JA/ET signaling-related proteins. In addition, Kyndt et al. (2012) found evidence that RKNs suppress the SA-mediated pathway in the systemic tissues of infected rice plants. Moreover, RKNs have been shown to modulate the ET pathway and induce the production of methyl jasmonate (MeJA) to promote successful infection of the plant. Among studies performed on *Zea mays*, Gao et al. (2008) tested mutants lacking lipoxygenase (*ZmLOX3*) and found that there was an increased level of JA in the roots but not in the leaves and increased susceptibility to *M. incognita*.

Incompatible interactions

There are several studies on resistant varieties of rice revealing that both JA- and SA-mediated host defenses are activated 2 days postinfection, and in contrast to sensitive varieties, this activity can still be observed in later stages of infection (Kumari et al. 2016, 2017). Moreover, a study performed on maize infected with *M. arenaria* showed changes in the expression levels of genes encoding the PR3, PR4 and PR5 proteins, which may also suggest the role of JA- and SA-mediated pathways in host resistance (Przybylska et al. 2018). Alternatively, Starr et al. (2014) analyzed the expression

Table 1 Nematode effectors and their role in parasitism

Nematode	Effector protein	Localization	Function	Reference
M. incognita	Mi-CRT	Apoplast	Overproduction in plant cells increases plant resistance to RKNs	Jaouannet et al. 2013
	Msp40	Cytoplasm and nucleus	Suppresses ETI-associated cell death	Niu et al. 2016
	Misp12	Cytoplasm	Participates in the maintenance of giant cells during parasitism	Xie et al. 2016
	Mi-CM3	Cytoplasm and nucleus	Suppresses plant immunity by manipulating the SA pathway at the early stage of nematode parasitism	Wang et al. 2018
	MiPFN3	Unknown	Binds actin monomers to manipulate plantactin, which must undergo reorganization for giant cell formation	Leelarasamee et al. 2018
	MiSGCR1	Cytoplasm and nucleus	Suppresses plant cell death	Nguyen et al. 2018
	MiISE5	Cytoplasm	Interferes with various metabolic and signaling pathways, especially SA- and JA-mediated signaling pathways	Shi et al. 2018b
	MiISE6	Nucleus	Interferes with various metabolic and signaling pathways, especially the JA signaling pathway	Shi et al. 2018a
M. javanica	MjTTL5	Plastids	Encodes a transthyretin-like protein that may suppress host defenses	Lin et al. 2015
M. hapla	Mn265	Cytoplasm	Suppresses host defenses	Gleason et al. 2017
M. enterolobii	MeTCTP	Cytoplasm	Suppresses programmed cell death in host plants	Zhuo et al. 2017
M. graminicola	MgGPP	Nucleus	Suppresses host defenses and enhances nematode parasitism	Chen et al. 2017
	MgMO237	Cytoplasm and nucleus	Overexpression in plants increases plant susceptibility to RKNs by suppressing the host defense responses	Chen et al. 2018
	Mg16820	Apoplast, cytoplasm and nucleus	Suppresses both the PTI and ETI response	Naalden et al. 2018
	Mg01965	Apoplast	Suppresses the PTI response	Zhuo et al. 2019
Conserved among *M. hapla, M. floridensis, M. incognita, M. javanica* and *M. graminicola*	Msp18	Cytoplasm and nucleus	Suppresses defense-related programmed cell death	Grossi-de-Sa et al. 2019

level of the *ZmPAL4* gene, which encodes phenylalanine ammonia lyase (the key enzyme in phenylpropanoid metabolism in plants, which is involved in plant responses to biotic and abiotic stresses), in maize varieties with different susceptibilities to *M. incognita* infection, and the results of this study indicated that the most tolerant inbred variety had the highest expression of this gene, which may suggest that the *PAL* gene plays a role in modulating the susceptibility of maize to *M. incognita*. Factors involved in the monocotyledonous host response to *Meloidogyne* infection and the processes analyzed in these interactions are listed in Table 2. Moreover the interaction between the *Aloe vera* plant and *M. incognita* and *M. javanica* was described by (Palomares-Rius et al. 2015), but the mechanism of the plant response has not been analyzed in detail. However, scientists have discovered that aloe plants possess a possible defense mechanism that affects the

development and viability of RKN eggs laid inside the root. They suggested that this effect may be associated with the presence of certain phenolic compounds in infected tissues.

Plant defense mechanisms induced in dicotyledonous plants

Compatible interactions

Substantially more RKN interactions have been studied in dicotyledonous plants than in monocotyledonous plants. Many studies have indicated that the ET/JA pathway plays an important role in plant-nematode interactions. One such study carried out on tomato (*S. lycopersicum*) and *M. javanica* suggested that the expression pattern of the ethylene-responsive elements may reflect the involvement of ethylene in RKN parasitism (Bar-Or et al. 2005). Alternatively, Fan et al. (2015) stated that endogenous JA and exogenous MeJA are potent inducers of systemic root defense against *M. incognita* attack in this host. Fujimoto et al. (2011) found that multicystatin (*MC*) and proteinase inhibitors (*PIs*), the JA-responsive genes in tomato infected with *M. incognita*, may be important for RKN invasion and infection because a smaller number of egg masses were observed when RKNs were inoculated into plants overexpressing *MC* and *PIs*. In addition, there have been studies suggesting the role of nitric oxide (NO) in the JA-dependent defense against *M. incognita* in tomato. NO is an essential regulatory molecule that has multiple functions in plants and has been widely observed in different plant species and organs under various biotic stress conditions (Zhou et al. 2015). Moreover, a study on soybean (*Glycine max*) showed that all of the allene oxide synthase family members were significantly downregulated in addition to other genes encoding enzymes involved in the JA signaling pathway (Ibrahim et al. 2011). It has also been shown that abscisic acid (ABA) synthesis and the JA- and ET-mediated signaling pathways were downregulated in a susceptible variety of peanut (*Arachis hypogea*) (Clevenger et al. 2017). Therefore, the authors concluded that the downregulation of the JA/ET signaling pathways may enhance nematode susceptibility in peanut.

Alternatively, there have been reports that also showed an important role of SA and SAR induction in plants following nematode infection in tomato (Molinari et al. 2014). Research conducted on the tomato-*M. incognita* model showed that the components of ET, ABA and SA signaling were differentially regulated (Shukla et al. 2018). In addition, the genes encoding enzymes related to oxidative stress, including glutathione S-transferases, peroxidases and thioredoxins, were largely downregulated during the later stages of infection (Shukla et al. 2018). Studies on *A. thaliana* showed that *M. incognita* induced both SA-dependent and JA-dependent pathways in the roots of infected plants, which was confirmed by the high levels of mRNA transcripts of *PR-2*, *PR-3* and *PR-5* in the roots of *M. incognita*-infected plants (Hamamouch et al. 2011). The results of studies performed on peanut indicated that the defense responses in plants were highly conserved and involved cross talk between JA, SA and ethylene signaling cascades, as demonstrated by the upregulation of the gene encoding 6,7-dimethyl-8-ribityllumazine synthase, which catalyzes the penultimate step in the biosynthesis of riboflavin and may influence the cross talk among the ABA, SA, JA and ET signal cascade pathways in various biotic and abiotic stress environments (Tirumalaraju et al. 2011).

In addition, the involvement of transcription factors and gene expression regulators in the plant response to *Meloidogyne* infection was observed in several studies. Jammes et al. (2005), in their global analysis of *A. thaliana* infected with *M. incognita*, found that the successful establishment of this RKN was associated with the suppression of the plant defense mechanisms. In nematode-infected tissues, 17 of the 21 WRKY genes identified were downregulated, as was the case for other genes encoding other proteins involved in plant defense mechanisms, such as lipoxygenase or peroxidase (Jammes et al. 2005).

Among other proteins with very important roles in plants, RKNs interact with disease resistance proteins from the TIR-NBS class (Toll/interleukin-1 receptor-nucleotide-binding site) or the LRR (leucine-reach repeat) family proteins described in the *A. thaliana-M. incognita* pathosystem (Fuller et al. 2007). The authors observed the upregulation of genes encoding these proteins in the later stages of infection in susceptible varieties.

Other studies indicated an involvement of cell wall-modifying proteins, auxin-related proteins and the ROS-scavenging system in response to the invasion of

Table 2 Host factors and their role in the plant-nematode interaction in monocotyledonous and dicotyledonous plants

Root-knot nematode species	Host species	Analyzed genes/proteins (or group of genes/proteins)	Changes in expression	Method	Reference
Monocotyledonous plants					
M. graminicola	*Oryza sativa*	*JiOsPR10, OsWRKY45, OsPR1b, OsEin2b*	Downregulation in the early stage of infection in a susceptible variety	Real-time PCR	Nahar et al. 2011
		OsZEP, OsNCED3, OsLip9	Upregulation in the early stage of infection in a susceptible variety	Real-time PCR	Kyndt et al. 2012
		MAPK5a, MAPK6, MAPK20, EDS1, PAD4, AOS2, ACS1, ACO7, PR1a, PR10	Upregulation in the early stage of infection in a susceptible variety	Real-time PCR	Kumari et al. 2016, 2017
		MAPK20	Downregulation in the early stage of infection in a susceptible variety		
		PR1b, MAPK20, PAD4, NPR1, AOS2, ACO7, PR1b, WRKY13	Downregulation in the later stage of infection in a susceptible variety		
		MAPK6, RbohB, RAC1, PAL1, ICS1, EDS1, NPR1, EDS2, JAMYB, ECS1, EIN2, PR1a, PR1b, PR10, WRKY13, WRKY24	Upregulation in the early stage of infection in a resistant variety		
		MAPK6, MAPK20, PAL1, ICS1, EDS1, PAD4, AOS2, JMT1, JAMYB, ACS1, ACO7, EIN2, PR1a, PR1b, PR10, WRKY13, WRKY24	Upregulation in the later stage of infection in a resistant variety		
		PAD4, NIH1	Downregulation in the early stage of infection in a susceptible variety	Real-time PCR	Nguyễn et al. 2014
M. incognita		*NIH1*	Upregulation in the early stage of infection in a susceptible variety		
		MAPK5a, AOS2, PAD4, WRKY13	Downregulation in the early stage of infection in a susceptible variety		
		ZmPAL4	No expression in the highly susceptible variety, the highest level of expression in the most resistant variety	Semiquantitative RT-PCR	Starr et al. 2014
M. arenaria	*Zea mays*	*PR3, PR4, PR5*, peroxidase, catalase or superoxide dismutase	Downregulation in the early stage of infection in a resistant variety	Real-time PCR	Przybylska et al. 2018
M. incognita	*Musa acuminate*	LRR receptor-like serine/threonine protein kinases, peroxidases, WRKY TFs, snakins	Downregulation in the early stage of infection in a susceptible variety	NGS	Castañeda et al. 2017
		MYB TF proteins, JA and ET signaling-related proteins and TFs, programmed cell death-related proteins	Downregulation in both the early and later stages of infection in a susceptible variety		
Dicotyledonous plants					
M. javanica	*Solanum lycopersi-cum*	Peroxidase	Downregulation in both the early and late stages	Microarray confirmed by real-time PCR	Bar-Or et al. 2005

Table 2 (continued)

Root-knot nematode species	Host species	Analyzed genes/proteins (or group of genes/proteins)	Changes in expression	Method	Reference
			of infection in susceptible varieties		
		Homeotic protein VAHOXI	Downregulation in the early stages of infection in susceptible varieties		
		WRKY, plant defensin	Upregulation in both the early and later stages of infection in a susceptible variety		
		Ethylene-responsive transcriptional coactivator, hin1-like protein, gibberellin 2-oxidase-like protein	Upregulation in the later stage of infection in a susceptible variety		
M. incognita	*Solanum lycopersicum*	Transcription-related proteins, signaling, defense-related proteins	Upregulation in the early stage of infection in a resistant variety	Microarray	Bhattari et al. 2008
		Transcription-related, signaling, protein biosynthesis-related proteins	Downregulation in the early stage of infection in a resistant variety		
		Transcription-factor-related, signaling, cell-wall-related proteins	Upregulation in the early stage of infection in a susceptible variety		
		Transcription-related, protein biosynthesis-related, signaling proteins	Downregulation in the early stage of infection in a susceptible variety		
		LeLOXA, LeLOXC, LeLOXD, LeAOS1, LeAOS2, LeAOS3, LeOpr3, LeJA3, LeDes, LeAOC, LeZIP, LeCOI, LePrs, LeSR160	Gene expression systemically activated within 6–24 h and diminished over 24–72 h	Real-time PCR	Fan et al. 2015
		PR1, PR2, PR5	Upregulation in the early stage of infection in a resistant variety	Real-time PCR	Molinari et al. 2014
		EF1, collagen, *MAP1*, peptidase, C-type lectin	Downregulation in the later stage of infection in a susceptible variety	NGS confirmed by real-time PCR	Shukla et al. 2018
		MjNULG1a	Upregulation in the later stage of infection in a susceptible variety		
		UDP-glucosyltransferase	Upregulation in the early stage of infection and downregulation in the later stage of infection in a susceptible variety		
		Carboxylesterase	Upregulation in the later stage of infection in a susceptible variety		
		Carbohydrate-binding module family	Downregulation in the later stage of infection in a susceptible varietyss		
		Genes encoding aspartic, metallo and serine peptidases	Upregulation in the early stage of infection and downregulation in the		

Table 2 (continued)

Root-knot nematode species	Host species	Analyzed genes/proteins (or group of genes/proteins)	Changes in expression	Method	Reference
			later stage of infection in a susceptible variety		
		Genes encoding various peptidases	Upregulation in the later stage of infection in a susceptible variety		
		Genes encoding members of the GH family, genes encoding members of the PL family, genes encoding aspartic and cysteine peptidases	Upregulation in the early stage of infection in a resistant variety		
		Cuticle collagen, tubulin proteins	Upregulation in a later stage of infection in a susceptible variety		
		IFA-1	Upregulation during all stages in a susceptible variety		
		Calponin protein	Downregulation during all stages in a susceptible variety		
		Lipid transport proteins, cytosolic fatty acid-binding protein	Upregulation in the later stage of infection in a resistant variety		
		Catalase	Upregulation in the early stage of infection and downregulation in the later stage of infection in a susceptible variety		
		SOD	Upregulation in a later stage of infection in a susceptible variety		
M. incognita	*Arabidopsis thaliana*	*PR-1, PR-2, PR-3, PR-5*	Upregulation in the later stage of infection in a susceptible variety	Real-time PCR	Hamamouch et al. 2011
		LOB domain protein 41, wound-responsive family protein, lipid transfer protein, speckle-type POZ-related protein, zinc finger protein, kelch repeat-containing protein, histone H3, pathogenesis-related protein, signal peptide peptidase, photosystem I reaction center, leucine-rich repeat transmembrane protein kinase, arabinogalactan-protein, gibberellin-regulated protein 5, senescence-associated-related protein, universal stress protein, auxin-responsive-related protein	Upregulation in the later stage of infection in a susceptible variety	Microarray confirmed by real-time PCR	Fuller et al. 2007
		Adenosine-deaminase family, UDP-glucuronosyltransferase family protein, glycosyl hydrolase family protein 5, F-box family protein,	Downregulation in the later stage of infection in a susceptible variety		

Table 2 (continued)

Root-knot nematode species	Host species	Analyzed genes/proteins (or group of genes/proteins)	Changes in expression	Method	Reference
		RNA-binding protein, germin-like protein, ADP-ribosylation factor, cytochrome P450 71B29, protein kinase family protein, patatin, glutathione S--transferase, flavin-containing monooxygenase family protein, glycine cleavage system H protein 1, F-box family protein, metal transporter, CBL-interacting protein kinase 9, disease resistance protein, polygalacturonase, allergen V5, multicopper oxidase type I family, MIF4G domain-containing protein, lectin protein kinase family protein, glutathione S-transferase, no apical meristem family protein			
		Major intrinsic family protein, calcium-binding EF-hand, lipoxygenase, ethylene-responsive element-binding factor 2, ammonium transporter 1, glycosyl hydrolase family 3 protein	Downregulation in the later stage of infection in a susceptible variety	Microarray confirmed by real-time PCR	Jammes et al. 2005
		Pectate lyase family protein, expansin, beta-expansin, glycoside hydrolase family 28 protein, 3′ exoribonuclease family domain 1-containing protein, sulfate transporter, microtubule-associated protein, elongation factor 1-alpha, formin homology domain-containing family, amino acid transporter family protein, MYB family transcription factor, phosphate-responsive 1 family protein, gibberellin-regulated protein 4, pyruvate decarboxylase	Upregulation in the later stage of infection in a susceptible variety		
		miR157d, miR159a, miR159c, miR156h, miR167d, miR390a/b, miR398a, miR398b/c, miR408, miR831, miR833b, miR2111a-3p, miR2934-5p	Upregulation in the later stage of infection in a susceptible variety	miRNA sequencing	Medina et al. 2017
		miR163, miR164c, miR319c, miR399b/c, miR822, miR861-5p	Downregulation in the later stage of infection in a susceptible variety		
M. arenaria	*Arachis hypogaea*	PR protein, patatin, *USP*, MFS transporter, transmembrane transporter, expansin B1	Upregulation in all stages of infection in a resistant variety	SSH confirmed by real-time PCR	Tirumalaraju et al. 2011
		Catalase, Aux/IAA TF, XET, tetraspanin--LEL-like, integrin-like, phi-1	Upregulation in all stages of infection in a susceptible variety		
		Defense-responsive genes	Upregulation in a resistant variety and downregulation or unchanged in a susceptible variety	NGS	Clevenger et al. 2017

Table 2 (continued)

Root-knot nematode species	Host species	Analyzed genes/proteins (or group of genes/proteins)	Changes in expression	Method	Reference
		Genes involved in auxin homeostasis and the auxin signaling pathway, ceramidase and ceramide catabolic process	Downregulation in both varieties		
		ABA transport, glutamate metabolism,	Upregulation in a susceptible variety		
		ABA synthesis, JA- and ET-mediated signaling pathways, camalexin biosynthesis, oxylipin biosynthesis, linoleate 13S-lipoxygenase activity	Downregulation in a susceptible variety		
		Ubiquitination, clathrin-coated vesicle, phosphatidylcholine metabolism, phosphoinositol binding, phospholipase D activity, cyclic nucleotide binding, cyclic nucleotide-gated cation channel activity	Upregulation in a resistant variety		
		ABA biosynthetic process, mucilage metabolic pathways	Downregulation in a resistant variety		
	Arachis stenosperma	*AsALKBH2, AsAUX/IAA*	Downregulation in the later stages of infection in a resistant variety	NGS confirmed by real-time PCR	Guimaraes et al. 2015
		AsAOC3, AsBTB, AsERF6	Downregulation in early and later stages of infection in a resistant variety		
		AsGH3.1, AsSLP	Downregulation in the early stage of infection in a resistant variety		
		AsSAG, AsTIR-NBS-LRR, AsSAG	Downregulation in the early stage of infection and upregulation in the later stage of infection in a resistant variety		
		AsAraH8, AsATPase α, AsBap, AsCOX1, AsCWAH, AsCWAH2, AsMYB25, AsWRKY49	Upregulation during all stages of infection in a resistant variety		
		AsTAT	Upregulation in the early stage of infection in a resistant variety		
		AsBger, AsCHI2, AsCOX1, AsIOMT, AsUreD	Upregulation in the later stage of infection in a resistant variety		
M. incognita	*Glycine max*	*LOX1, OPR2/OPR3*	Upregulation in the early stage of infection and downregulation in the later stage of infection in a susceptible variety	Microarray confirmed by real-time PCR	Ibrachim et al. 2011
		PECT, CCOA-OMT, C3H, CDKB2, FSH	Upregulation in all stages of infection in a susceptible variety		
		AOS, CELL	Downregulation in all stages of infection in a susceptible variety		

Table 2 (continued)

Root-knot nematode species	Host species	Analyzed genes/proteins (or group of genes/proteins)	Changes in expression	Method	Reference
		AOC	Downregulation in the early stage of infection in a susceptible variety		
		CYCD3	Downregulation in the later stage of infection in a susceptible variety		
M. incognita	*Glycine max*	Chitinase	Upregulation in the later stage of infection in a resistant variety	Chitinase activity assay	Qtu et al. 1997
M. incognita	*Vigna unguiculata*	26S proteasome, aldo-keto reductase, spermidine synthase, patatin	Downregulation in the early stage of infection and upregulation in later stages of infection in a resistant variety	2-DE confirmed by real-time PCR	Villeth et al. 2015
		20S proteasome, nitrile-specifier protein 5, disease resistance protein RPP13, hydroxyacid oxidase, isopropylmalate dehydrogenase	Upregulation in the later stages of infection in a resistant variety		
	Nicotiana tabacum	*PMEU1, GUN9, BGL, PGLR4, E1312, Y5487, AUX/IAA, LAX5*	Downregulation in a resistant variety and upregulation in a susceptible variety	NGS confirmed by real-time PCR	Xing et al. 2017
		LRX4, NEK2, PRB1, PER11	Downregulation in a resistant variety		
		Kinase CLV1	Downregulation in both varieties		
		GH3, TGA21, GST23	Upregulation in a resistant variety and downregulation in a susceptible variety		
		PRB1	Upregulation in a resistant variety		
M. incognita	*Medicago sativa*	NB-ARC domain disease resistance protein, lipid transfer protein, calcium-binding EF-hand-like protein, cysteine-rich receptor-kinase-like protein, patatin-like phospholipase, transcription factor bHLH122-like protein	Downregulation in a susceptible variety	NGS confirmed by real-time PCR	Postnikova et al. 2015
		Matrixin family protein, flavonol synthase/flavanone 3-hydroxylase, transmembrane protein, heat shock cognate 70 kDa protein, F-ox/FBD-like domain protein,	Upregulation in a susceptible variety		
		Matrixin family protein, glutamate receptor 3.3, organelle transcript processing protein, heat shock cognate 70 kDa protein, endonuclease/exonuclease/phosphatase family protein, F-box/RNI/FBD-like domain protein, Harpin-inducing protein 1-like protein	Downregulation in a resistant variety		

Table 2 (continued)

Root-knot nematode species	Host species	Analyzed genes/proteins (or group of genes/proteins)	Changes in expression	Method	Reference
		Disease resistance protein (TIR-NBS-LRR class), receptor-like protein, DUF674 family protein, RNA-binding domain CCCH-type zinc finger protein	Upregulation in a resistant variety		
	Ipomoea batatas	*LOX*	Upregulation in the early stage of infection and downregulation in the later stage in a resistant variety	NGS confirmed by real-time PCR	Lee et al. 2019
		MYC2, EIL1, EIN4	Upregulation in the later stage in a resistant variety		
		TGA1	Downregulation in the early stage in a susceptible variety		
		LOX, JAZ1, EDS1, NPR1, SNRK2	Upregulation in the later stage in a susceptible variety		
		ERF1, LEA14	Upregulation in the later stage in both varieties		
		ABF3	Downregulation in the later stage in both varieties		

nematodes in tobacco (*N. tabacum*) (Xing et al. 2017). In addition, the results obtained for coffee suggested that the genes related to cell death, cell wall modification, oxidative burst, gene expression regulation and defense played a role in coffee plants as 'RKN-responsive' genes. Their common regulation in compatible and incompatible interactions suggested that their expression overlaps in the PTI and ETI responses (Albuquerque et al. 2017).

Incompatible interactions

Many studies have attempted to explain the mechanism of host resistance to RKN infection. The data from the transcriptome profiling of a resistant variety of cucumber (*Cucumis metuliferus*) infected with *M. incognita* suggested an important role for proteins involved in the first layer of plant defense (Ling et al. 2017). The authors discovered that the PAMP-associated genes, including *BAK1* and *ADFs,* may have been important for the resistance of this plant to RKNs that similarly utilize two pathogen-related signal genes: *MPK3* and *MPK6*.

On the other hand, some authors investigated the role of JA, SA and ET signaling pathways. Research conducted on the tomato-*M. incognita* model showed that the components of ET, ABA and SA signaling were differentially regulated during both the susceptible and resistant responses (Clevenger et al. 2017). In addition, the authors observed that in resistant varieties of peanut, the number of induced proteins involved in plant stress and defense responses was comparatively higher than that in the susceptible varieties, including putative PR proteins, patatin-like proteins and other stress-related proteins (Tirumalaraju et al. 2011). Guimaraes et al. (2015) analyzed *M. arenaria* resistance in wild peanut (*Arachis stenosperma*) plants and found that both JA-dependent and SA-dependent defense genes and their regulators were triggered in wild peanut roots infected with *M. arenaria*. Additionally, Qtu et al. (1997) described an important role of chitinases during the *M. incognita* infection of a resistant variety of soybean, which may indicate an important role of both the JA and SA pathways in the soybean resistance response. On the other hand, in the tomato-*M. javanica* and tomato-*M. incognita* pathosystems, the authors observed that a

tomato mutant, which does not accumulate JA after wounding, did not exhibit reduced susceptibility, thus indicating that nematode susceptibility does not depend on JA biosynthesis (Bhattarai et al. 2008).

Other tomato genes with upregulated expression during incompatible interactions are those encoding the defensin protein and subtilisin-like protease, leading to the production of phytoalexins and stress-induced proteolysis (Shukla et al. 2018). Studies performed on coffee indicated that host resistance to *M. exigua* was not only due to the HR but also due to a set of defense responses that were both constitutive and induced after nematode penetration, that resulted in the inhibition of feeding site formation, and that provoked J2 migration or inhibited nematode development and reproduction (Silva et al. 2013). Moreover, the genes encoding LRR and TIR-NBS class proteins were upregulated in a resistant variety of alfalfa infected with *M. incognita* (Postnikova et al. 2015). Alternatively, Clevenger et al. (2017), in a study on wild peanut plants resistant to *M. arenaria*, reported the downregulation of these genes in the early stage of infection but upregulation during later stages of infection.

Additionally, the transcriptome analysis of two alfalfa (*Medicago sativa*) cultivars that were sensitive and resistant to *M. incognita* infection revealed nearly a thousand differentially expressed genes that were presumably involved in basal defense responses and in resistance pathways and revealed a number of transcripts potentially associated with resistance to RKNs (Postnikova et al. 2015). Another genome-wide transcriptome analysis with susceptible and resistant varieties was performed in sweet potato (*Ipomoea batatas*) infected with *M. incognita*. The authors indicated an increase in the activity of genes involved in defense signaling, including genes encoding transcription factors as well as these associated with JA, SA, ET and ABA pathways, which were upregulated in a resistant variety under RKN treatment (Lee et al. 2019).

Moreover, research conducted on *A. thaliana* suggested a role for the miRNA family, designated *miR159,* in the formation of galls during the plant response to *M. incognita* infection. A strong resistance to this nematode was observed in the *miR159abc* triple *Arabidopsis* mutant. The *miR159* family is known to regulate MYB transcription factors implicated in the ABA-mediated response and in interactions with other transcription factors (Medina et al. 2017).

The results of studies performed on dicotyledonous plants are summarized in Table 2.

Concluding remarks

Effectors of RKNs are multifunctional proteins that are involved in interactions with mono- and dicotyledonous hosts, and they are able to manipulate plant metabolism on many different levels. Most of the described effectors were analyzed, and their modes of action were studied for one species of nematode in which they are expressed. However, one of the described effectors (Msp18) is conserved among many nematode species, including *M. hapla, M. floridensis, M. incognita, M. javanica* and *M. graminicola*. Effectors encoded by RKNs with a broad spectrum of host plant species, such as *M. incognita*, were reported to play similar roles as the effectors encoded by *M. graminicola*, whose main crop host is rice.

The research conducted so far has shown that there are some differences between the responses of monocotyledonous and dicotyledonous plants to RKN infection. However, it is difficult to say whether these are real differences or simply a highly unbalanced number of publications on the reaction of monocotyledonous and dicotyledonous plants to RKN infection. The likely cause of this imbalance is the fact that more dicotyledonous than monocotyledonous plants have been observed to be RKN hosts (CABI 2020). Most studies conducted on monocotyledonous plants have suggested a role for both the JA/ET- and SA-mediated pathways in the plant response during the early stages of infection, but these responses may be suppressed during the later stages in compatible interactions in contrast to resistant varieties where the changes in expression of genes encoding proteins involved in these pathways can still be observed. This statement is true for RKNs with broad host ranges, such as *M. incognita* and *M. arenaria*, as well as for monocot-specific *M. graminicola* species. In studies on dicotyledonous plants, the authors primarily indicate the role of the JA/ET-mediated pathways in compatible interactions, while the SA-mediated pathway seems to play a supporting role, primarily in resistant varieties. Additional differences concerning the expression of genes encoding transcription factors were also observed as well as in the expression level of ABA-mediated pathway marker genes, which were significantly upregulated in susceptible varieties of rice infected by *M. graminicola*, while in peanut plants infected by

M. arenaria, the genes associated with ABA synthesis were downregulated.

The plant response to RKN infection is very extensive, occurs on multiple levels and involves the engagement of many different pathways. Many nematode species are able to overcome all barriers, including all activated defense mechanisms in both mono- and dicotyledonous plant species. When the infection process is successfully established, plant metabolism is reorganized at all possible levels, beginning from epigenetic changes (miRNA) to structural and morphological modifications (giant cells).

Future challenges and research directions

Although knowledge on the processes involved in RKN-plant interactions has significantly increased in recent years, there are still important aspects of these interactions that have not yet been thoroughly investigated. This category includes the regulation of signaling pathways and the involvement of posttranslational modifications, e.g., phosphorylation, in plant-*Meloidogyne* interactions, as has been studied for few nematode species (Hewezi 2015). Among other factors that should be elucidated further in future studies is the role of epigenetic changes in plant resistance/susceptibility to the infection process, as shown for cyst nematodes and soybean (Li et al. 2012). Another promising research direction is the induction of systemic resistance in host plants. There are many studies on the possible induction of systemic resistance by abiotic agents, such as BTH (benzo[1,2,3]thiadiaz*ole-7-carboth*ioic acid S-methyl ester) and its derivatives, BABA (β-aminobutyric acid) or MeJa (methyl jasmonate) (Frąckowiak et al. 2019; Fujimoto et al. 2011; Mutar and Fattah 2013; Veronico et al. 2018) as well as by microorganisms such as bacteria or fungi (El-Fattah and Sikora 2007; Siddiqui and Shaukat 2004; Vos et al. 2013). This topic, however, still has great investigation potential in nematode studies.

Moreover, a broader spectrum of data concerning the molecular mechanisms of plant immunity might be essential to develop resistant varieties of economically important RKN hosts.

It is very likely that in plant-*Meloidogyne* interactions, many other factors may play roles that have not yet been investigated in detail in this pathosystem. Studies conducted in recent years have provided substantial evidence for the important role of symbiotic microorganisms associated with various pests, especially insects, in the modulation of the outcomes of pest-plant interactions (Wielkopolan et al. 2018). Research on nematode-associated microbiota has also been performed for some nematode species, including *M. incognita*, and the microbes associated with their different life stages (Cao et al. 2015; Elhady et al. 2017), but for the majority of the *Meloidogyne* species, the role of symbionts has been subjected to limited analyses. However, this aspect might contribute to an enhanced understanding of the parasitism process involving RKNs in their host plants.

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