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Nematode Secreted Peptides Modulate the Plant Immunity by Mimicking the Host Factor

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ABSTRACT

Plant-Parasitic Nematodes (PPNs) are a major concern for farmers and agricultural producers around the world, as they cause considerable economic losses and food insecurity worldwide. Peptides are short chains of amino acids having signaling properties, play a crucial role in various physiological processes in plants, including stress response, development, and pathogen defense. Recent studies revealed that plant-parasitic nematodes produce secreted peptide interact with plant receptor kinases and modify their activity to promote nematode infection and feeding by suppress plant immune responses to the nematodes. The CLE peptides are a family of small signaling molecules in plants they are perceived by specific receptor kinases in plant cells, triggers downstream signaling pathways to regulate plant development. The nematode secreted peptides are mimic to plant CLE peptides have been discovered in plants which are known to interact with specific receptor kinases in plant cells and trigger downstream signaling pathways that promote nematode parasitism and feeding. The identified nematode secreted peptides share similarity with plant CLAVATA3/ESR (CLE) peptides, but genomic analyses have also identified nematode secreted peptides homologous to other classes of plant peptide hormones, which are used by PPNs to counteract plant immunity. This review focuses on the recent discovery of secreted peptides by PPNs that mimic endogenous plant peptides to promote parasitism.

Keywords: Nematodes; Peptides; CLAVATA; CLE; Ligand mimicry; Peptide hormone

Abbreviations: CLEs: CLAVATA3/ESR-like effector proteins; CLV2: CLAVATA2; CRN: CORYNE; DGA: Dorsal Gland Ampulla

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INTRODUCTION

With the ongoing nature of the socio-economic importance of agriculture, the global needs for sustainable and mounting food production to suffice the increased human population. Thus, it is essential that issues associated with a full spectrum of crop production restrictions and losses are soundly solved. Plant-Parasitic Nematodes (PPNs) rank high among other crop pests and pathogens that constitute major constraints to agricultural production. Estimated crop losses due to PPNs for the 20 life-sustaining crops averaged 12.6% of worldwide crop yield which equaled USD 215.77 billion of annual yield. An additional 20 crops with significant values for food and export have also a 14.45% annual yield loss which equaled USD 142.47 billion. The total 40 crops sustain an average of 13.5% losses which are estimated at USD 358.24 billion annually [1].

Phytopathogens have evolved a diverse range of mechanisms to increase ability to infect host plants. The host-nematode molecular interaction is relatively little known, Some PPNs have employed multiple molecular mechanisms to suppress host immunity and enhance the virulence of PPNs during infections. PPNs release numerous effectors through the esophageal gland and stylet, these effectors can reprogram host cell to create nutrient-rich feeding site, by interfering the plant cell walls to make them more permeable to nutrients or alter the plant's hormone signaling pathways to promote cell division and expansion at the feeding site. Although some PPNs effectors have been well studied [2-4] there is still a lot is unknown about how these effectors work and which specific host targets they interact with. Understanding the host targets of these effectors is key to developing strategies for controlling PPNs and reducing their impact on crop production.

Plant-Parasitic Nematodes (PPNs) produce effectors that mimic the structure or function of plant proteins, allowing the nematodes to manipulate the host cellular functions to their advantage. By mimicking the host proteins, PPNs can evade the host immune system and establish successful infection. Mimicry is a common strategy employed in bacterial and viral pathogens in various animal systems information about this phenomenon in phyto pathogens is relatively limited [5,6].

Root-knot and cyst root-knot nematodes are types of Plant-Parasitic Nematodes (PPNs) that employ molecular mimicry for parasitism [6]. Cyst nematodes, in particular, secrete effector proteins that contain a conserved 12-amino acid C-terminal motif that closely resembles the plant CLAVATA3/ ESR (CLE) ligand peptides. This C-terminal motif is known as the CLE-like motif, is believed to mimic the plant CLE peptides and aid in nematode parasitism [7].

The Meloidogyne Avirulence Protein (MAP) family is secreted protein sarecrucial during interaction between the nematode and its host plant, contains CLE-like motifs, which mimics the host CLE peptides to modulate plant cell division and differentiation. The MAP proteins secreted by Plant Parasitic Nematodes (PPNs) have been shown to interact with plant receptor kinases, triggering downstream signaling pathways that facilitate nematode parasitism [8]. Huang, et al., demonstrated that a MAP protein secreted by the soybean cyst nematode interacts with a soybean receptor kinase, leading to the activation of downstream signaling pathways involved in nematode infection [9]. Similarly, Vanholme, et al., showed that a MAP protein secreted by the root-knot nematode interacts with a tomato receptor kinase, resulting in the activation of downstream signaling pathways that promote nematode parasitism [10]. The discovery of these CLE-like motifs in the MAP family highlights the importance of molecular mimicry in the host-parasite interaction between PPNs and plants.

The annexin-like effector from cyst nematodes known to mimic a plant protein to subvert the defense response, having low amino acid sequence similarity with *Arabidopsis* annexin1 but can complement the Arabidopsis annexin1 mutant and perform similar functions [11]. This indicates the effector may mimic the host annexin1 protein to manipulate the host cell's functions for the benefit of the nematode. The current review highlights the mimicry functions of effectors secreted by plant-parasitic nematodes and specifically focuses on the secreted peptides that mimic host factors in the sequence of disease development.

LITERATURE REVIEW

The Discovery of Plant Parasitic Nematode-Encoded Mimics

Multiple nematode encoded mimics have been identified, including the annexin-like effector discussed earlier, including various effectors that mimic the activities of plant growth regulators such as auxin and cytokinins. The discovery of nematode encoded mimics expanded our understanding of the mechanisms underlying plant-nematode interactions and this opened up a new avenue for developing novel methods to manage plant parasitic nematodes.

Horizontal Gene Transfer (HGT) is a possible explanation for the origin of certain nematode effectors that show similarity to host proteins. Few studies have shown that plant hosts could be a source of mimics through HGT in some plant-parasitic nematodes. In the study by Scholl, et al., the researchers proposed that an HGT event from a host plant led to the acquisition of a gene encoding a nematode effector with a functional mimic of a plant protein [12]. This effector, called SPRYSEC-19, was found to suppress host defense responses and promote nematode parasitism. The presence of a plant-like domain in the SPRYSEC-19 effector provided a strong source for the involvement of HGT in the acquisition of this effector by the nematode.

Convergent evolution refers to the process by which unrelated organisms develop similar traits or functions independently in reaction to similar environmental pressures. In the case of nematode encoded mimics, parasitic nematodes evolved to acquire functions or structures that mimic those of host plants to subvert the host immune system and facilitate their parasitism and this occurs without the exchange of genetic material between the nematodes and their hosts. Cyst

nematode effector 10A07 is a good example of how plant-parasitic nematodes can use structural mimicry to manipulate host post-translational modification mechanisms. The effector contains a sequence motif that structurally resembles a plant kinase phosphorylation site, allowing it to be recognized and phosphorylated by the host plant kinase. Once phosphorylated, the effector can interact with host receptors and disrupt the host cellular processes to benefit the nematode's parasitic lifestyle [13].

In 2005, Wang, et al., and colleagues reported the identification of the first CLE signaling peptide encoded by a gene expressed in the sub ventraloesophagal gland cell of *Heterodera schachtii* during parasitism, representing the first instance of a non-plant species utilizing CLE signalling. This discovery marked the first evidence of molecular mimicry by a nematode effector. Several other plant-parasitic nematodes, such as *Globodera rostochiensis*, *Globodera pallida*, and reniform nematodes, have also been discovered to secrete CLE peptides, in addition to *H. glycines*. These findings were reported in studies conducted by Vieira, et al.; Lu, et al.; Gheysen, et al., these peptides are believed to mimic the endogenous CLE peptides of plants and regulate plant cell division and differentiation, contributing to the development of specialized feeding structures by the nematodes [14].

By secreting CLE-like peptides, the nematode acquired a special feature to manipulate host plants to their advantage. CLE peptides are known to play significant functions in the plant such as regulating cell differentiation and proliferation [15]. Nematodes have the ability to process single or multiple CLE motifs from a single protein, which increases the complexity of the mimicry strategy used by these parasites, as reported by Wang, et al. The study by Zhang, et al., reported the first functional study of Ralf mimics in animals, which provided a new paradigm for understanding host-pathogen interactions [16]. The researchers showed that a nematode secreted Ralf mimic can mimic the function of a host Ralf peptide to manipulate plant development and suppress plant immune responses.

Plant Nematode Interaction

Plant-parasitic nematodes use various sensory mechanisms to perceive host cues and locate suitable hosts. Nematodes rely on chemosensation as a critical mechanism, and their sensitivities to specific chemicals in root exudates can vary among different species. For example, the potato cyst nematodes and soybean cyst nematodes are known to respond strongly to specific chemicals, while the root-knot nematodes rely on more general cues like low pH and CO_2 gradients. In addition to chemosensation, nematodes may also use other sensory mechanisms to locate and recognize their hosts such as mechanosensing, thermosensing, and electro sensing [17].

Upon reaching the root surface, plant-parasitic nematodes secrete enzymes to facilitate their entry into the plant tissue, where they establish a feeding site. In migratory nematodes, this involves the development of a feeding cell or syncytium, which is formed by the reprogramming of host cells into a nutrient-rich environment that can support the nematode's growth and development. In sedentary nematodes, a feeding site is established by the nematode inducing the development of a specialized structure called a nurse cell, which provides nutrients to the nematode through a specialized feeding structure called a stylet.

Molecular Mechanism of Plant Parasitic Nematode Interaction

A hypothetical model demonstrates plant nematode interaction, nematode invasion causes damage to the cell wall and produces Damage-Associated Molecular Patterns (DAMPs) to activate plant basal defence responses *via* DAMP-receptors such as WAK1. In addition, the nematode produces Cell Wall Degrading Enzymes (CWDEs) such as Polygalacturonases (PG) to hydrolyse the plant cell wall [18]. In turn, the plant produces PG-Inhibiting Proteins (PGIPs), which interact with PG and promote the development of small cell wall fragments, and Oligogalacturonides (OGs) that activate the DAMP-associated immunity.

Conserved Nematode-Associated Molecular Patterns (NAMPs) are released from nematodes in the apoplast and are perceived by plasma membrane localized Pattern Recognition Receptors (PRRs) to trigger Pattern-Triggered Immunity (PTI) including the release of Reactive Oxygen Species (ROS), callose, and lignin [18]. Some PRRs are related to the Leucine-Rich Repeat Receptor-Like Kinase family (LRR-RLKs), which detect typically proteinaceous ligands. LRR-RLKs recruit the BRI1-associated receptor-like kinase 1 (BAK1) as a co-receptor to initiate PTI. Nematodes, in turn, secrete apoplastic and cytoplasmic effectors to perturb the DAMP or NAMP based immunity [19-22].

Apoplastic effectors interfere with the detection of PAMPs or DAMPs by PRRs and cytoplasmic effectors may disturb downstream signalling which acts of PTI. Plants, in turn, carry *R-genes* (Nucleotide-binding Leucine-Rich repeat, NLRs) that recognize effectors either directly or indirectly and initiate Effector-Triggered Immunity (ETI). In addition to NLRs, non-NLRs type *R-genes* also exist against nematodes. *Indicates additional nuclear localization for effectors.

Nematode-Secreted Peptides Modulate Plant Immunity

Nematodes secreted effector proteins, mimic plant C-terminally Encoded Peptides (CEPs) to manipulate the plant nitrogen response and promote nematode feeding site development. The nematode CEP mimics bind to CEPR1 and CEPR2, triggering the downstream signalling cascade and altering the plant's nitrogen utilization, promoting the growth and development of the nematode. Activation of CEPRs (CEP Receptor Kinases) by either plant CEPs or nematode CEP mimics can induce nitrogen demand signals that increase the expression of nitrogen transporters in the plant. Furthermore, the activation of CEPRs can also inhibit primary root elongation and initiate lateral root development to take up nitrogen. The biotrophic interaction of nematodes with the

host plant prevents the loss of nutrient drainage, ensuring their survival and minimizing damage to the host plant. This strategy allows nematodes to extract nutrients from plants while minimizing harm to the host plant [23].

Plant pathogens such as *Pseudomonas syringae* pv. Tomato (Pst) reported that secrets molecular mimics modulate plant signalling pathways. For example, Pst produces corona tine, which is structurally and functionally similar to jasmonate-L-isoleucine (JA-Ile). This mimicry allows the pathogen to hijack the plant's defence response and promote its own survival and growth within the plant. Nematodes produce CLE peptides that mimic plant CLEs to modulate the plant signalling pathway. These CLEs are recognized by the plant CLAVATA1 (CLV1)/CLV2 heterodimer or a Treachery element Differentiation Inhibitory Factor (TDIF) Receptor (TDR), leading to altered plant physiology to benefits the nematode.

Fusarium oxysporum a fungal pathogen produces Rapid Alkalinization Factor (RALF) mimics that interfere with the plants receptor kinase by disrupting the extracellular alkalinization and cell expansion regulation [24]. This could promote fungal multiplication and infection by altering the physiological conditions of the host. Additionally, the fungal Ralf mimic may directly respond to Feronia (FER) receptor, leading to downstream signalling events that benefit the fungus. However, further studies are required to understand the molecular mechanism underlying the interaction of the fungal Ralf mimic and the plant host.

The bacterial pathogen *Xanthomonas oryzae* pv. *Oryzae* (Xoo) produces a sulphated peptide called RaxX-sY, which is similar peptide hormone PSY (plant peptide containing sulphated tyrosine) of the plant. This pathogenic peptide initiates PSY signalling to enhance plant growth and development. However, XA21 a rice receptor specifically recognizes microbial RaxX and triggers the immune response to combat the pathogen. Straight lines indicate the secretion of pathogen molecules. Dashed lines indicate products of the endogenous factor from the plant. Question marks indicate pathways that have not yet been fully elucidated.

Plant and PPNs Secreted Peptides

The CLE genes encode CLE peptides that exhibit structural variability, with each peptide performing distinct functions in the plant. CLE peptides are two types: A-type and B-type, having specific peptide structures and functions. CLAVATA3 (CLV3)/Endosperm Surrounding Region (ESR) (CLE) peptides are known to have significant roles in plant development, including shoot meristem differentiation, vascular development and root growth [25]. CLV3 is a well-known that promotes stem cell differentiation at the root apical meristem, and several other CLEs have been identified that performa similar function. On the other hand, CLEs 41, 42, and 44 are known to inhibit tracheary element differentiation during vascular development.

Nematode CLEs are synthesized in the dorsal oesophagal gland cell and secreted from the stylet into plant tissues where they mimic plant CLE ligands to promote differentiation of feeding cells. This allows for establishing a feeding site and obtaining nutrients from the host [26]. Intriguingly, PPNs can secrete molecules that mimic plant peptides, such as CLEs and CEPs, to promote infection and establish a feeding site within the plant. This is occur by manipulating the plant signalling pathways involved in root development and nutrient uptake, the *Heterodera glycines*, a soybean cyst nematode produces CLE propeptides that are transported into the host and function as mimics of host CLE peptides in the apoplast, promoting the synthesis of nematode feeding cells showed how nematodes use mimicry of plant signalling molecules to manipulate plant growth and development to promote their own parasitic success The Variable Domain (VD) of HgCLE2 contains a cryptic ER translocation signal, but the exact motif responsible for directing the CLE peptide to the apoplast need to be identified [27].

Mimics of CLE (CLAVATA/embryo surrounding region) peptides are best studied in cyst nematodes. In that group of PPNs, CLE-like peptides are delivered as propeptides into the host cytoplasm through the nematode stylet. The propeptides are then post-translationally modified and proteolytically processed by host enzymes for secretion into the host's extracellular space as mature CLE-like peptides. The N-terminal variable domain of the propeptide is needed for the secretion process of nematode CLEs. Studies revealed that deletion or alteration of this domain significantly impairs the secretion of the peptide.

CLE Peptide Secretion Pathways in Cyst-Nematode Feeding Cells

The exact secretion pathway of CLE peptides in nematode feeding cells is still under investigation. However, it is believed that the CLE peptides may be secreted *via* exosomes or Extracellular Vesicles (EVs) that are released by the nematode into the host cell cytoplasm. Another proposed mechanism, a nematode may manipulate the host plant's Endoplasmic Reticulum (ER) golgi secretory pathway to secrete the CLE peptides. Further research is required to understand the secretion pathway of CLE peptides in nematode feeding cells. In soybean cyst nematode, the CLE peptides HgCLE2 and HgCLE3 are detected in the cytoplasm and cell wall of the feeding cells, indicating that they are secreted into the apoplast, likely *via* a non-classical ER-golgi-independent pathway. This pathway may involve vesicles derived from the plasma membrane or endocytic compartments.

Meloidogyne incognita Peptide and its Mimicry

Root-Knot Nematodes (RKNs) have been found to encode peptides that mimic other peptide hormones, such as CEP (C-terminally Encoded Peptides), RALF (Rapid Alkalization Factors), and IDA (inflorescence deficient in abscission) peptides, as a strategy to promote parasitism [28-32]. Unlike cyst nematode CLEs, which have a variable domain and require processing to become active, all the peptide hormone mimics characterized in root-knot nematodes lack a variable domain and may be secreted as mature peptides directly into

the host's extracellular space. This makes them more similar to plant peptide hormones than cyst nematode CLEs.

The discovery of two putative secreted peptides with similar sequences to *Arabidopsis* Inflorescence Deficient in Abscission (IDA) from *Meloidogyne incognita* is an important finding in the field of plant-nematode interactions. The two *IDA*-like genes, MiIDL1 and MiIDL2, encode small proteins having an N-terminal signal, indicating these peptides are secreted outside of the nematode. The *IDA* signalling peptides function as a lateral root emergence and mediating cell separation for floral organ abscission has been reported in several studies. The identification of *IDA*-like genes in *M. incognita* shows that the nematode may use similar mechanisms to induce plant cell separation during infection. Recent researchers revealed that *M. incognita* secretes effector proteins that modulate plant hormone signalling pathways [33].

DISCUSSION

In the study by Kim, et al., the exogenous application of a synthetic MiIDL1 peptide rescues the abscission phenotype of the *Arabidopsis* Ida mutant. This indicates that the MiIDL1 peptide has a functional mimicry of the *Arabidopsis IDA* peptide and can induce cell separation in plant tissues. This finding suggests that the nematode may use the MiIDL1 peptide to manipulate plant tissues during infection. Additional investigations are required to gain a more comprehensive understanding of the underlying molecular mechanisms between the MiIDL1 peptide and plant tissues during nematode infection. However, the identified functional mimicry of the MiIDL1 peptide in inducing cell separation in plants provides a new avenue for the molecular study of plant-nematode interactions and could be exploited for the development of sophisticated strategies to control nematode infestations in crops.

FER is a key play important role in plant-microbe interactions and is involved in both susceptibility and defense responses to certain pathogens. Specifically, loss-of-function mutations in FER have been found to increase susceptibility to powdery mildew and *Pseudomonas syringae* infections, while overexpression of FER increases the resistance to these pathogens [34]. Besides playing a role in plant-nematode interactions, the FER receptor kinase is also involved in mediating plant responses to other abiotic and biotic stresses. Recent studies have shown that FER can regulate plant responses to salt and drought stress by modulating ion transport and water use efficiency. FER has also been implicated in plant immunity against bacterial and fungal pathogens [35].

Root-knot nematodes encode Rapid Alkalinization Factors (RALF) peptide mimics and interact with plant Feronia (FER) kinase to facilitate RKN parasitism. By infecting the *Arabidopsis* plant with the RKN, *Meloidogyne incognita*, the researchers noticed that mutations in FER lead to significantly low susceptibility, suggesting the involvement of the RALF-FER complex in immune suppression. A total of 18 putative *RALF*-like genes were discovered in nematode genomes. Four of them are from *M. incognita* had high sequence similarities either between MiRALF1 and MiRALF2or between MiRALF3 and MiRALF4.

Sulfated tyrosine (PSYs) is plant peptides, promotes root growth *via* cell expansion and proliferation [36,37]. PSY1is an 18-amino acid tyrosine sulfated glycopeptide was first identified from the culture medium of *Arabidopsis* suspension cells. It is derived from a 75-amino acid precursor peptide, which is processed by proteolytic cleavage and post-translational modifications to yield the mature PSY1 peptide [38]. Tyrosine sulfation is catalyzed by Tyrosylprotein Sulfotransferase (TPST), which is encoded by a single gene in *Arabidopsis* (At1g08030). Exogenous application of AtPSY1 to seedlings has been shown to activate plasma membrane localized proton pumps, leading to the acidification of the extracellular space [39]. The activation of cell wall modifying enzymes causes a loosening and softening of the cell wall, allowing for cell growth and expansion [40].

Models Describing the CLE Peptide Signalling Pathways in PPNs

- Cyst nematode CLEs are produced in the oesophagal gland cell and transported as proproteins through the stylet into the cytoplasm of host cells. The variable domain facilitates the targeting of CLE proproteins to the apoplast of host cells through an unknown mechanism. In the apoplast, the proproteins are likely processed into 12-amino acid CLE peptides, function as ligand mimics of plant CLEs. These CLE peptides bind to extracellular leucine-rich repeat receptor kinases, redirecting developmental programs that are important for syncytium formation.
- Root-knot nematode CLE-like peptides have been proposed to be transported as 12-amino acid peptides directly to the apoplast of host cells *via* the stylet, where they interact with extracellular receptors to redirect developmental programs required for giant cell formation. Alternatively, or in addition, these CLE-like peptides may be localized to the cytoplasm and/or nucleus of giant cells, where they can interact with host transcription factors. This has been reported in root-knot nematode 16D10 CLE-like peptide.

CONCLUSION

The current review highlights the mimicry functions of effectors secreted by plant-parasitic nematodes specifically. The concept of molecular mimicry employed by parasitic nematodes to recognize and enter host plants has opened up new avenues for developing sophisticated innovative strategies for nematode control and also for manipulating plant physiology. Further research on the functions of peptide mimics derived from microorganisms can help identify potential targets for disease resistance and aid in the discovery of new fundamental mechanisms that can be applied in modern agriculture. Additionally, studying signalling peptides in both symbiotic and pathogenic

organisms can lead to new discoveries that have both fundamental and practical applications in agriculture.

DATA AVAILABILITY

No data were used for the research described in the article.

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