academicJournals

Vol. 12(19), pp. 1612-1617, 11 May, 2017
DOI: 10.5897/AJAR2016.11265
Article Number: 50487EB64139
ISSN 1991-637X

http://www.academicjournals.org/AJAR

African Journal of Agricultural Research

Review

Role of phytohormones (indol acetic acid, jasmonic acid, salcylic acid, and ethylene) in nematode-plant interactions

Degife Asefa Zebire

Department of Plant Sciences, College of Agricultural Science, Arba Minch University, P. O. Box 21, Arba Minch, Ethiopia.

Received 27 May, 2016; Accepted 9 August, 2016

Plant parasitic nematodes are among the most destructive major pest of crop plants. Root knot nematode species and cyst nematodes are well studied species of nematode with various ranges of host. The common means of control for these pests mostly rely usually on use of chemicals like nematicides, which are environmentally unfriend and costly especially in large scale agricultural production systems. However, recent advances showed that there are other alternative strategies, such as development of resistant crop varieties and exploiting natural resistance genes of plants in conventional breeding programs. Molecular basis of biotechnological applications are also considered as an effective measure to control these pests. Many studies showed the existence of complex gene expression and hormone signaling for both compatible and incompatible interaction with the process of plant-nematode interaction. This review provides information on the overview of recent knowledges on the role of plant hormones mediating feeding site development through plant-parasitic nematodes and the role of phytohormones resistance against nematodes.

Key words: Plant parasitic nematode, phytohormones, nematode-plant interaction.

INTRODUCTION

Plant parasitic nematodes (PPNs) cause huge damage in many plant species and agriculturally important crops. However, there are major differences in the suitability of particular plant species and varieties as a host for each nematode. Many studies showed that root knot nematode species (RKNs), which causes a major economic loss throughout the world, have very broad host ranges. Some other species of nematode are limited to one or a few hosts (Djian-Caporalino et al., 2011; Gleason et al., 2008). For example, cyst nematodes usually have narrower host ranges. *Globodera rostochiensis* and *Globodera pallida* are the most common cyst nematode species damaging potato. Potato cyst nematodes reproduce mainly on potato and related Solanaceous

*Corresponding author. E-mail: asefadegife@gmail.com.

species, whereas soybean cyst nematodes can cause huge yield losses on soybean, which are commonly limited to this crop (Lilley et al., 2007).

Parasitic nematodes can be distinguished by their different feeding habits as ectoparasitic and endoparasitic. They feed essentially on the living plant cell cytoplasm (Bar-Or et al., 2005). Sedentary plant-parasitic nematodes of the family Heteroderidae cause most of the damage. These nematodes transform differentiated plant root cells into nematode feeding sites (NFS). Sedentary endoparasites have a distinctive and complex interaction with their hosts in order to establish parasitism. RKNs and cyst nematodes cause a host cell to be stressed through suitable interaction. Once these nematode species form suitable interaction, it is easy for penetration and the establishment of a feeding site, due to their migration into the host cells (Hewezi and Baum, 2013). Plant induces defense responses during the process of nematode infection. Plant cells respond to nematode attack by the production of toxins such as super oxide and hydrogen peroxide. Compatible interaction between tomato and RKN in the cells surrounding migrating nematodes and feeding sites can be affected by hydrogen peroxide (Melillo et al., 2006).

There are several strategies that sedentary endoparasitic nematodes use to interact with their host. Sedentary endoparasitic nematodes are obligate biotrophs that can establish successful relationships with their host plants by inducing redifferentiation of root cells into specific feeding cells (Jaouannet et al., 2012). The development of nematodes is determined by their success in establishing feeding cells. Manipulation of the essential elements of cell development enables RKNs to induce feeding cell formation in a wide range of their host (Caillaud et al., 2008). Feeding cells are converted into multinucleate giant cells via simultaneous nuclear divisions in the absence of cell division. These enlarged giant cells containing many nuclei may undergo extensive endo-reduplication. Extensive reprogramming of gene expression results in the formation of giant cells (Caillaud et al., 2008). Nematodes secrete effectors which play a critical role in parasitism by manipulating the recipient host plant. Nematode responsive plant cells enable RKNs to alter root development to induce and maintain giant cells (Bellafiore and Briggs, 2010).

Plant-nematode interaction influences the association between suppression of plant defenses and nematode feeding site development during gene expression. As a result, during infection of *Arabidopsis thaliana* with RKN, 70% of the genes involved in defense pathways were found to be locally repressed (Jammes et al., 2005). Resistance gene and signaling pathways overwhelms defense mechanisms in plant. For instance, salicylic acid (SA) pathways in the root can be suppressed by successful parasitism between cyst nematode and *A. thaliana,* whereas, susceptibility of plants to nematodes can be increased by ethylene signal transduction (Wubben et al., 2008). Susceptibility of tomato to RKN is facilitated by jasmonate signaling pathway (Bhattarai et al., 2008). Thus, plant hormones play a vital role in effective interactions between plants and nematodes. The objective of this paper was to review current published articles and journals on plant hormones in plant-nematode interaction. The review mainly focuses on research papers available in the online literature database using Scopus and Google scholars searched information by using key words of the content. For instance, “nematode effectors”, “plant parasitic nematode”, nematode-plant interaction”, “plant hormone signaling pathway” and so forth. Research papers used in this review were not older than 2005. This paper basically reviews plant-nematode interaction, role of hormones in feeding cells and role of hormone in plant resistance to plant parasitic nematodes. It aims to provide information to people about research carried out on defense response of plants to plant parasitic nematodes and nematode effectors

THE ROLE OF PLANT HORMONE IN FEEDING SITE ESTABLISHMENT

The cyst nematode *Heterodera schachti* and the root-knot nematode *Meloidogyne incognita* infect the roots of *A. thaliana*. But these two nematode species are distantly related and the feeding sites they induce are dissimilar in nature. During infection, the pre-parasitic second-stage juveniles (preparasitic J2) penetrate the roots of a suitable host. The cyst and root-knot nematodes then migrate intracellularly or intercellularly, respectively towards the vascular cylinder to find a competent plant cell for the induction of a multinuclear feeding cell complex. Cyst nematodes induce syncytia, whereas root-knot nematodes induce giant cells as a feeding site. The similarities between these two nematode species are that they induce Death receptor 5 (DR5) activation patterns in *A. thaliana* root. Transcriptional regulation of synthetic auxin-responsive promoter element DR5 used to study the role of plant hormone (auxin) in induction of nematode feeding cell. DR5 promoter element was administered using a model plant *Arabidopsis thaliana* roots infected with *H. schachtii* or *M. incognita*. Strong and specific activation of DR5 using gusA reporter observed inside the feeding cells at 18 hour post inoculation (hpi), showing an increase apparent auxin concentration. Therefore, the phytohormone indol acetic acid (IAA) might give an important clue in feeding cell induction by PPNs due to an increase in apparent IAA concentration in the initial feeder cell (Caillaud et al., 2008; Karczmarek et al., 2004).

Ethylene and auxin are important plant hormones involved in the regulation of many important plant processes. For instance, cell differentiation, cell expansion, and responses of a plant to abiotic stresses

mediated by phytohormones (Davies, 2010). In addition, these hormones play vital roles in many plant-pathogen interactions, including manipulation of plant defense responses and development of symptoms. Sedentary plant-parasitic nematodes, requires the formation of a complex feeding site within the host roots as an important pre-requisite. A great change in host cell morphology and gene expression occurs during the formation of nematode-induced feeding sites. These changes are probably mediated by phytohormones (Gutierrez et al., 2009).

The complex interacting signaling networks are regulated and modulated by the hormonal balance which also dictates the responses of resistance or susceptibility. Three biological compounds, including SA, jasmonic acid (JA), and ethylene (ET) regulate the resistance gene-mediated and induced basal defense responses. Experiments on gene expression profiling of hormone signaling pathways indicate that there is a significant overlap among these pathways (Li et al., 2006). The signaling of JA and ET generally showed synergistic interactions where negative pathway crosstalk may occur between the JA and SA. The JA signaling pathway is essential for tomato susceptibility to root knot nematodes (Bhattarai et al., 2008).

Important aspects of developmental processes in plants can be facilitated by phytohormones. Phytohormones may also participate in various aspects of plant-nematode interactions. Several reports revealed that nematode infection interferes with the accumulation and transport of auxin in the plant. Auxin is an essential plant hormone involved in the formation of feeding site (Curtis, 2007). Ethylene is another plant hormone which might be involved in the modulation of many cellular processes during feeding site formation. Auxin is involved in plant-nematode interaction by acting as a signalling molecule by inducing changes on the surface of the cuticle, making it more susceptible and can also change the behavior of *Meloidogyne* species. The change in the surface of the cuticle and behavior of these nematode species might be important for infection (Curtis, 2007). Involvement of SA on *A. thaliana* mutant affects the activity of cyst nematode. (Wubben et al., 2008) reported that *A. thaliana* mutants deficient in SA were more susceptible than the wild types to cyst nematode. SA also plays a significant role in the reduction of parasitism between cyst nematode and plant (Wubben et al., 2008); can be increased by the presence of protease inhibitors, senescence associated and seed specific proteins associated with JA (Uehara et al., 2010). Ethylene-induced genes activated by ripping and allow compatible interaction between plants and PPNs (Uehara et al., 2010). Susceptibility of plants to cyst nematodes can also be facilitated by ET signaling transduction (Uehara et al., 2010; Wubben et al., 2008). Plant hormones like ET and JA have the ability to interfere with tomato SA-inducible PCN resistance pathway in susceptible cultivars. In addition, auxin response factor induces the compatible interaction between plant and PCN (Uehara et al., 2010). Thus, phytohormones involved in many processes of plant nematode parasitism like the involvement of invasion plant cell and induction of syncytium.

SA triggers the resistance mechanism to plant cyst nematode. But the SA-inducible pathway in susceptible cultivars, one way or another affected by other hormones includes JA, auxin and ET (Uehara et al., 2010) (Figure 1).

Auxin and ethylene are involved in the formation of syncytia and host susceptibility. Cyst nematodes commonly complete their life cycle inside the root of the plant. They are associated with the plant by feeding on a nematode induced conglomerate of metabolically active root cells (Gutierrez et al., 2009). Cell enlargements in the plant after infection by nematodes are a common characteristic. Cell enlargement results in the formation of multicellular giant cells and this hypertrophy in the surrounding tissue in turn results in the formation of typical galls of the infected root. Enlargement and division of cell is mainly associated with increase in IAA perception. Auxin is also involved in programming the neighboring cells for integration into the developing syncytium (Karczmarek et al., 2004). The initiation and development of feeding sites of sedentary PPN is facilitated by plant signaling molecules of auxin (Grunewald, 2009). Auxin is also involved in the activation of cell division needed to keep up with the growing cells.

THE ROLE OF HORMONES IN PLANT PARASITIC NEMATODES RESISTANCE

Naturally, plants have the ability to protect themselves from pathogen attack. Resistance mechanisms are one of the ways by which plants respond to stimuli and activate the production of defensive compounds. The hypersensitive response results from the death of host cell and blocks the development and reproduction of the pathogen, and can be involved in various types of resistances (Goggin et al., 2006). Plant resistance to the most destructive nematode species such as RKN and cyst nematodes could be based on dominant, recessive or additive effects and can be found in many crop species. Therefore, the use of well-studied genetic resistance, which is environmentally friendly, may pave the way for breeding programs aimed at controlling nematodes (Djian-Caporalino et al., 2011). Several studies revealed the requirement of hormone signaling in response to resistance. The complex interaction of signaling networks is regulated and modulated by the hormonal3balance which also dictates the responses of resistance. SA, JA, and ET regulate the resistance gene-mediated and induced basal defense responses (Li et al., 2006).

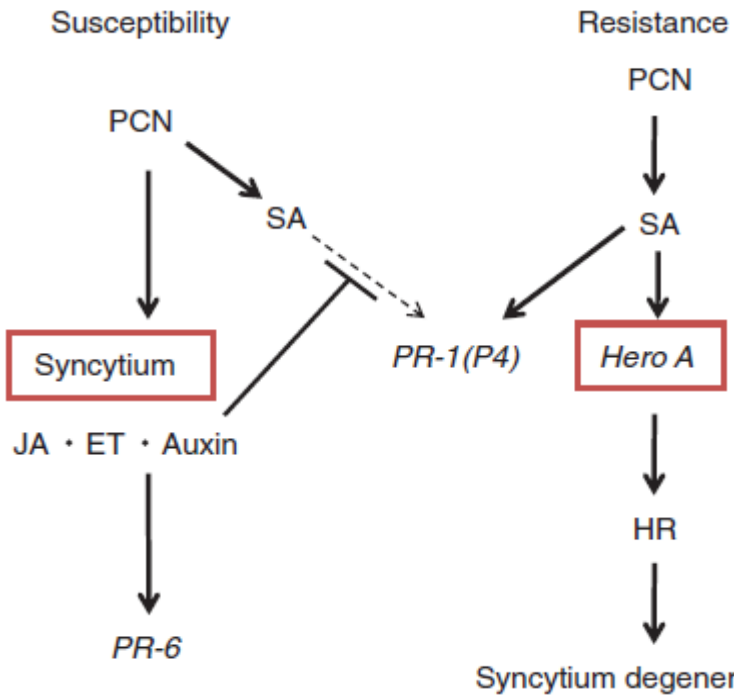

Figure 1. Model to explain resistance and susceptibility of tomato cultivars to potato cyst nematode. R-gene-mediated resistance is induced through the SA-mediated signaling pathway. Signaling pathways mediated by other hormones such as auxin, ethylene (ET) and jasmonic acid (JA), which possibly interferes with the SA pathway, may be activated by syncytium formation because some hormones are involved in the formation of syncytia. Arrows indicate positive regulation; the blunt end indicates negative regulation (Uehara et al., 2010).

SA plays a major role in activation of defenses against biotrophic pathogens. ET provokes the SA-dependent resistance against biotrophic pathogens together with JA. Therefore, hormones such as JA and ET in the susceptible cultivars are expected to interfere with the tomato SA-inducible PCN resistance pathway (Uehara et al., 2010). Over accumulation of the defense signal molecule SA commonly displays plants morphological phenotypes. SA acid could interfere with auxin responses due to reminiscent of auxin-deficient or auxin-insensetive mutants. Wang et al. (2007) reported that SA causes global repression of auxin related genes. This results in balancing the AUX/IAA repressor proteins and inhibition of auxin responses. SA-mediated disease resistance mechanism is due the inhibitory effect on auxin signaling.

Foliar application of systemic JA to plants induces a systemic effect that can suppress RKN infestation. This shows that jasmonate plays a significant role in foliar defenses. The results also revealed that jasmonates has an important role of in the protection of root tissues against root herbivorous. The resistance induced by JA is heat stable. In addition to this, JA treatments have the capacity to enhance the control of avirulent nematodes on resistance cultivars. The existence of this situation shows that Mi-mediated resistance and JA-induced resistance may have an additive effect. Induction of JA through systemic means could have a significant influence in protecting crops from damage caused by nematodes. This in turn can reduce the use of hazardous nematicides which are environmentally dangerous chemicals (Cooper et al., 2005).

Synergistic interactions between the signaling molecules JA and ET is due to the negative pathway crosstalk that could occur between the JA and SA (Fujimoto et al., 2011). At the early stages of *Meloidogyne*-tomato plant incompatible interactions, SA was not found to be responsible for inhibition of catalase. Branch et al. (2004) reported that SA has a key role in the resistance response of tomato to RKN. They also investigated various resistance genes against *M. incognita* such as *Mi*-1. This SA showed positive effect of resistance response to the RKN *Meloidogyne javanica* in transformed tomato carrying Mi-1 with construct expressing NahG. NahG encodes a bacterial enzyme salicylate hydroxylase that has the capacity to degrade SA into catechol. In addition, they produce SA, which would lead to an increase in the levels of catechol, which is toxic for both resistance and susceptible tomato lines. SA have a positive contribution both in hypersensitive reactions of plants to incompatible RKN and systemic

acquired resistance (Molinari and Loffredo, 2006). Tomato plants might not be affected by signaling of SA concerning the inhibition of catalyze during early stage of incompatible interaction between *Meloidogyne* spp. However, catalase have a role in subsequent lesion formation which is part of *Mi-1*-mediated resistance to RKNs (Molinari and Loffredo, 2006).

Uehara et al. (2010) found that the induced SA-dependent pathogenesis-related genes mediate resistance in incompatible interactions between tomato and potato cyst nematode *G. rostochiensis* at 3 days-post-inoculation (dpi) when compared with compatible interaction. As direct evidence to show the link between salicylic acid and the *Hero A*-dependent *PR-1 (P4)* gene transcript, they produced *Hero A-NahG* tomato. The transcript accumulation of *PR-1 (P4)* was inhibited by the expression of *NahG* after potato cyst nematode infection of transformed tomato roots. Additionally, the reproduction of potato cyst nematodes on *Hero A-NahG* tomato was higher than on the *Hero A* tomato plants. This indicates that SA directly controls the *Hero A*-mediated potato cyst nematode resistance (Uehara et al., 2010).

Wubben et al. (2008) reported that wild type cultivars were more resistant to the cyst nematode than SA-deficient mutants. Moreover, SA application reduced parasitism by cyst nematodes. Wubben et al. (2008) demonstrated that SA plays more or less the same roles in resistance to the cyst nematode. According to Uehara et al. (2010), cultivars resistance to PCNs is elicited by SA, resulting in an elevated level of *PR-1* (P4*)*, a hallmark gene for SA-inducible type of resistance. However, the SA-inducible pathway in the susceptible cultivars is somehow disturbed, possibly by other hormones such as JA, auxin and ET.

JA application in two near-isogenic lines of tomato with and without *Mi-1.2* resistance gene helps in activation of induced resistance. This result shows a significant role of jasmonate in the defense mechanism of root tissues against root herbivorous. The JA treatment gives only partial resistance to susceptible tomato cultivars. The reproductions of avirulent RKNs were almost completely inhibited by *Mi-1.2* gene response. On the other hand, the effects of *Mi*-mediated resistance response were reduced at a temperature of 32°C. Though, the most effective result was obtained in the combined treatment of JA and the presence of *Mi*-1.2 gene at a temperature of 25°C (Cooper et al., 2005). Gene expressions and induced resistance response to *M. incognita* through foliar application of methyl jasmonate treatment at four different levels and timing were studied to realize the effects of exogenous optimum JA on resistance responses by Fujimoto et al. (2011). This result revealed that there was a positive correlation between the application of methyl jasmonate at 0.5 mM. This leads to a significant reduction of plant infection by RKNs.

In addition, *Mi-1* plants transfected with *NahG,* which leads to the suppression of SA production, did not show any increase in susceptibility to RKN which is in contrast to the results obtained by Branch et al. (2004). They proposed that the low levels of basal SA, which has a crosstalk interaction with JA, could be appropriate for *Mi-1* mediated resistance to RKN because all *Mi-1 NahG* plants showed complete resistance (Bhattarai et al., 2008). These differences between the two studies can be due to different analysis techniques used in the experiment.

Conclusion

Plant parasitic nematodes have the ability to infest several plant species by manipulation of the basic elements of plant cell development. They can also participate in establishment of feeding site, including multinucleate cell induction, which is the main source of nutrients for reproduction, multiplication and development of nematodes. There are several processes involved in establishment of feeding site and plants also have a defense mechanism to protect the pressure of plant parasitic nematodes. Phytohormones like ethylene and auxin regulate many important processes in the plants such as cell differentiation, cell expansion and responses to abiotic and biotic stresses. They also have a significant role in several plants-pathogen interactions, including regulation of plant defense responses and the development of symptoms. Sedentary parasitic plant nematodes are the world most destructive plant pathogens, need hormones for the formation of a complex feeding site within the host root system. Feeding sites induced by nematodes result in a dramatic change in the host cell morphology and gene expression. These changes are most likely mediated by phytohormones. The development of resistance cultivars through introduction natural resistance gene to the plant is the environmentally friendly way of nematode pest control. This method leads to loss of susceptibility factors in plant responses to plant parasitic nematodes. Several complex gene expressions and subsequent hormone signaling like JA and SA signaling pathways during incompatible interactions between plants and nematodes are considerable aspects to understand the mechanism underlying resistance responses as well as helping to conduct further studies for more details. Furthermore, the importance of plant hormone signaling in the determination of plant-nematode interactions is supported by several evidences. Apart from the contribution of directly defense response, plants also regulate hormone signaling pathway in order to control its vital processes for resistance. The role of phytohormones in establishment of feeding site and plant response in defense mechanisms of nematode is complex. Therefore, further investigation is needed to clearly understand this complex process.

CONFLICT OF INTERESTS

The author declared that there is no any conflict of interest

REFERENCES

Bar-Or C, Kapulnik Y, Koltai H (2005). "A broad characterization of the transcriptional profile of the compatible tomato response to the plant parasitic root knot nematode Meloidogyne javanica" European J. Plant Pathol. 111:181-192.

Bellafiore S, Briggs SP (2010). "Nematode effectors and plant responses to infection" Curr. Opin. Plant Biol. 13:442-448.

Bhattarai KK, Xie QG, Mantelin S, Bishnoi U, Girke T, Navarre DA, Kaloshian I (2008). Tomato susceptibility to root-knot nematodes requires an intact jasmonic acid signaling pathway. Mol. Plant-Microbe Interact. 21:1205-1214.

Branch C, Hwang C-F, Navarre DA, Williamson VM (2004). "Salicylic acid is part of the Mi-1-mediated defense response to root-knot nematode in tomato". Mol. Plant-Microbe Interact. 17:351-356.

Caillaud MC, Dubreuil G, Quentin M, Perfus-Barbeoch L, Lecomte P, de Almeida Engler J, Abad P, Rosso MN, Favery B (2008). Root-knot nematodes manipulate plant cell functions during a compatible interaction. J. Plant Physiol. 165:104-113.

Cooper W, Jia L, Goggin L (2005). Effects of jasmonate-induced defenses on root-knot nematode infection of resistant and susceptible tomato cultivars. J. Chem. Ecol. 31:1953-1967.

Curtis RH (2007). "Do phytohormones influence nematode invasion and feeding site establishment"? Nematology 9:155-160.

Davies PJ (2010). "The plant hormones: their nature, occurrence, and functions" In Plant hormones (pp. 1-15). Springer Netherlands.

Djian-Caporalino C, Molinari S, Palloix A, Ciancio A., Fazari A, Marteu N, Ris N, Castagnone-Sereno P (2011). "The reproductive potential of the root-knot nematode Meloidogyne incognita is affected by selection for virulence against major resistance genes from tomato and pepper. Eur. J. Plant Pathol. 131:431-440.

Fujimoto T, Tomitaka Y, Abe H, Tsuda S, Futai K, Mizukubo T (2011). Expression profile of jasmonic acid-induced genes and the induced resistance against the root-knot nematode (< i> Meloidogyne incognita</i>) in tomato plants (< i> Solanum lycopersicum</i>) after foliar treatment with methyl jasmonate. J. Plant Physiol. 168:1084-1097.

Gleason CA, Liu QL, Williamson VM (2008). Silencing a candidate nematode effector gene corresponding to the tomato resistance gene Mi-1 leads to acquisition of virulence. Mol. Plant-Microbe Interact. 21:576-585.

Goggin FL, Jia L, Shah G, Hebert S, Williamson VM, Ullman DE (2006). Heterologous expression of the Mi-1.2 gene from tomato confers resistance against nematodes but not aphids in eggplant. Mol. Plant-Microbe Interact. 19:383-388.

Gutierrez O, Wubben M, Howard M, Roberts B, Hanlon E, Wilkinson J (2009). The role of phytohormones ethylene and auxin in plant-nematode interactions. Russian J. Plant Physiol. 56:1-5.

Hewezi T, Baum TJ (2013). Manipulation of plant cells by cyst and root-knot nematode effectors. Mol. Plant-Microbe Interact. 26(1):9-16.

Jammes F, Lecomte P, Almeida-Engler J, Bitton F, Martin-Magniette ML, Renou JP, Abad P, Favery B (2005). Genome-wide expression profiling of the host response to root-knot nematode infection in Arabidopsisa. Plant J. 44:447-458.

Jaouannet M, Perfus-Barbeoch L, Deleury E, Magliano M, Engler G, Vieira P, Rosso MN (2012). A root-knot nematode-secreted protein is injected into giant cells and targeted to the nuclei. New Phytol. 194(4):924-931.

Karczmarek A, Overmars H, Helder J, Goverse A (2004). Feeding cell development by cyst and root-knot nematodes involves a similar early, local and transient activation of a specific auxin-inducible promoter element. Mol. Plant Pathol. 5:343-346.

Li Q, Xie QG, Smith-Becker J, Navarre DA, Kaloshian I (2006). Mi-1-mediated aphid resistance involves salicylic acid and mitogen-activated protein kinase signaling cascades. Mol. Plant-Microbe Interact. 19:655-664.

Lilley CJ, Bakhetia M, Charlton WL, Urwin PE (2007). Recent progress in the development of RNA interference for plant parasitic nematodes. Mol. Plant Pathol. 8:701-711.

Melillo MT, Leonetti P, Bongiovanni M, Castagnone-Sereno P, Bleve-Zacheo T (2006). Modulation of reactive oxygen species activities and H_2O_2 accumulation during compatible and incompatible tomato–root-knot nematode interactions. New Phytol. 170:501-512.

Molinari S, Loffredo E (2006). The role of salicylic acid in defense response of tomato to root-knot nematodes. Physiol. Mol. Plant Pathol. 68:69-78.

Uehara T, Sugiyama S, Matsuura H, Arie T, Masuta C (2010) Resistant and susceptible responses in tomato to cyst nematode are differentially regulated by salicylic acid. Plant Cell Physiol. 51:1524-1536.

Wang D, Pajerowska-Mukhtar K, Culler AH, Dong X (2007) Salicylic acid inhibits pathogen growth in plants through repression of the auxin signaling pathway. Curr. Biol. 17:1784-1790.

Wubben MJE, Jin J, Baum TJ (2008) Cyst nematode parasitism of Arabidopsis thaliana is inhibited by salicylic acid (SA) and elicits uncoupled SA-independent pathogenesis-related gene expression in roots. Mol. Plant-Microbe Interact. 21:424-432.