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INTERACTION OF PLANT PARASITIC NEMATODE WITH FUSARIUM WILT A DISEASE COMPLEX

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A B S T R A C T

Plant disease complexes are caused by variety of interactions between fungal plant pathogens and plant parasitic nematodes but the most significant and frequent association is *Fusarium oxysporum* and Root-knot nematodes (*Meloidogyne* spp). Nematode-fungal interactions are frequently categorized as either synergistic or additive. Different plant-parasitic nematodes caused damage to the host plants depending on their life cycle and feeding habit. Nematode feeding sites are areas with intense metabolic activity, such as syncytia or giant cells, and have a large number of mitochondria and golgi bodies. In addition, cytoplasm is dense and packed with ribosomes. Injuries of host plants are vulnerable to infection by fungi that need assistance to enter their host. Nematode-instigated physiological changes can be fundamental but they still have a lot of detrimental effects. Some nematodes make plant tissues more favorable for pathogen that negatively affects the host by causing them to grow and reproduce more rapidly. Moreover, they alter the root exudates of host plant, promoting the various phases of fungal life cycle like germination, development and reproduction. The importance of nematode-fungus synergistic complex in the development of distinct crop species that exhibit resistance to various biotic or abiotic stresses is only infrequently, if ever described, although numerous studies reveal that resistance breaks down during concomitant infection.

Keywords: Plant Parasitic Nematode; Fusarium Wilt; Host; Disease

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INTRODUCTION

Fusarium wilt is caused by *Fusarium oxysporum. F. oxysporum* is a ubiquitous soil-borne plant pathogen (Prado et al., 2015) that causes severe vascular wilt in more than 100 plant species and poses a serious threat to important economical crops like banana, cotton, tomato (Prado et al., 2015). Tomato wilt is caused by *Fusarium oxysporum* f. sp. *lycopersici* which produces a significant yield losses of tomato (Srinivas et al., 2019). *F. oxysporum* is a cosmopolitan filamentous fungus belonging to the genus *Fusarium*, (Baayen et al., 2000) class ascomycetes and have a key role in agriculture production because it cause many diseases (Nisa et al., 2015). Fusarium produces canker, rot, wilt, and blight-like symptoms on many horticultural crops, field crops (Waller and Brayford, 1990), ornamental plants, and forest tress that directly and indirectly affects the agricultural and natural ecosystems (Ramdial et al., 2017). Mainly *Fusarium* species produce sexual (meiotic) and asexual (mitotic) spores (Figure 1) (Ohara and Tsuge, 2004). All species do not produce both types of spores (Hornok et al., 2007). Generally, most species produce asexual spores; only 20 % of *Fusarium* species have a sexual spore (Ohara and Tsuge, 2004). *F. oxysporum* produces typically three types of asexual (mitotic) spores*:* i) microconidia (one or two-celled) mostly produced in xylem vessels of diseased plants, ii) macroconidia (three to five celled) generally found on the upper surface of leaves and stem in the form of sporodochia like structure gradually pointed and curved in shape and iii) chlamydospores (either single or two celled) these are thick fenced spores mostly formed either critically and intercalary on mature hyphae. *F. oxysporum* multiplies its mycelium on various solid agar

medium as well as in liquid medium have varying appearances such as creamy white purple, yellow depend on the strain (Galimberti et al., 2012). If genus *Fusarium* produce abundant sporodochia the culture plate looks like dark creamy or orange in colour (Joffe, 1974).

Mode of infection

F. oxysporum enters the roots of plants through the epidermis layer, later migrating towards vascular bundles and spreads in xylem vessels, causing the development of tylosis, clogging the vessels and causes plant wilting (Kressin, 2018; Ndambi et al., 2012). The disease symptoms are identified on the plant's above-ground part by yellowing and wilting like symptoms on one side or the whole plant. The progress of *F. oxysporum* infection is a complicated phenomenon that comprises the following steps i) detection of roots through host-pathogen signals, ii) attachments of hyphal propagation to the surface of root hair iii) invasion of hypha into root cortex and vascular cells differentiation with in xylem iv) finally production of mycotoxin and causal factors. Colonization and establishment of the mycelium led to the formation of tylosis and wilting appeared on the infected host plant (Srinivas et al., 2019). The appressorium of fungus first adheres to the root surface before penetration. The hyphae enter into root cortical cells intercellularly and move into vascular bundles over the xylem vessels (Galimberti et al., 2012). At the base of stem, the fungus begins to form microconidia inside xylem vessels, which are then carried upward by the flow of water and nutrients. Furthermore, microconidia germinate and migrate towards upper vessels (Dimond, 1970). The wilting and yellowing-like symptoms appeared due to blockage of xylem vessels, the abundant mass of fungal hyphae, and host-pathogen suitable interaction such as mycotoxins, gums enzymes, and gels (Okungbowa and Shittu, 2012). The typical *Fusarium* infections on leaves are leaf epinasty, yellowing, vein clearing, and defoliation ultimately wilting proceeds towards death (Okungbowa and Shittu, 2012; Rania et al., 2016). Due to the attack of *F. oxysporum* f.sp. *lycopersici* first lower leaves of infected plants become yellow often on one side of the plant or one whole branch (Gleason and Edmunds, 2005). When the stem of infected plants is cut or split lengthwise, the browning of the vascular tissues can perceive closest to the skin while the pith center of the stem remains unaffected (Okungbowa and Shittu, 2012).

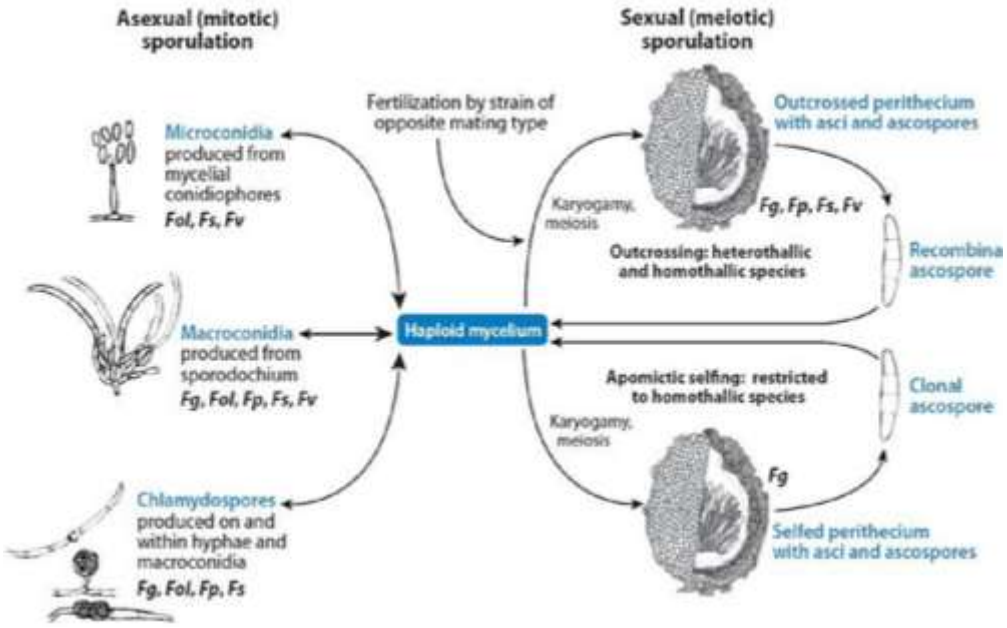

Figure 1. Life cycle of *F. oxysporum* (asexual and sexual spores) (Booth, 1971).

The cellular structure of plants infected by *F. oxysporum* has been examined under the scanning electron microscope, fluorescence light and the transmission electron microscope. The longitudinal and transverse sections of contaminated dried stems of the tomato plant had a large number of microconidia in xylem vessels, which germinate into mycelium and enter the vessels through the cortex after 10-14 days of inoculation (Gleason and Edmunds, 2005). However, the mycelium became thicker in diameter and spread through xylem vessel pits. On the other hand, the contact parenchyma tissues unsheathing the vascular cells and develop callose deposition. These parenchyma tissues play an important part

in managing storage, vascular elements, and defense-related functions in plants. The ultrastructure of parenchyma cells of *F. oxysporum* infected tomato plant under the transmission electron microscope (TEM) shows the callose deposition, wall apposition were associated with vesiculation and blistering of the plasmalemma and contain round bodies later these appeared in the form of striated and marbled (Mueller and Beckman, 1988).

Morphological and molecular characterization of *F. oxysporum*

The morphological identification of *F. oxysporum* was accomplished based on colony color, pattern, and single spore formation (Figure 2) (Ruiz-Roldán et al., 2010; Woudt et al., 1995).

Kornerup and Wancher (1978) studied *F. oxysporum* isolates morphologically. For microscopic characteristics observation, fungal isolates were cultured on the carnation leaf. Fifty chlamydospores of each isolate were observed under microscope shape, length, and width of macroconidia were measured. While for visual macroscopic observation (colony formation and pigmentation), the isolates were grown on PDA. The Methuen Handbook of the color chart was used to determine and confirm isolate type.

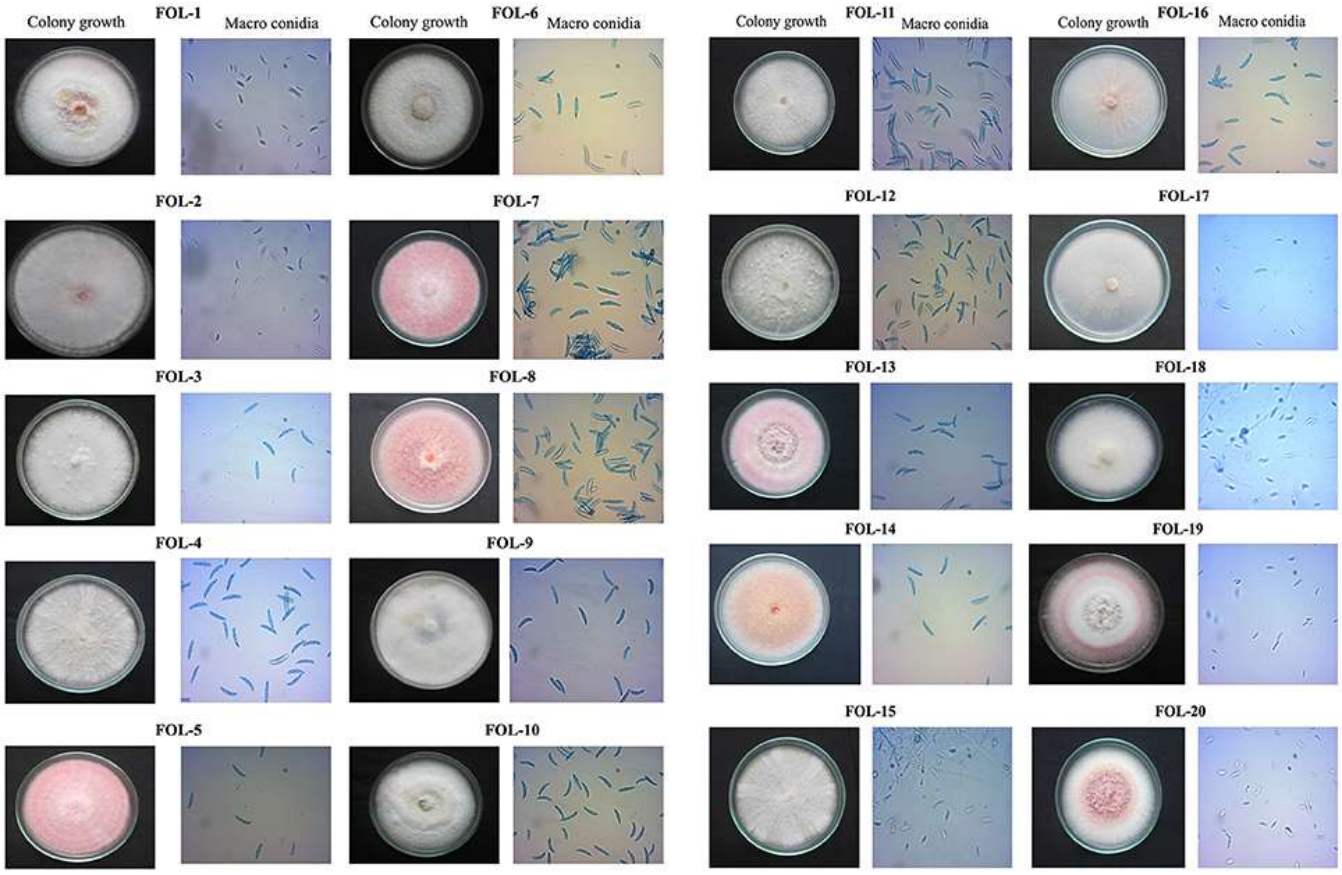

Figure 2. Colony color and spore shape formation of *F. oxysporum* f.sp. *lycopersici* (Manikandan et al., 2018).

Root knot nematode

The most harmful and severe parasites are plant parasitic nematodes, which substantially reduce yields in horticulture crops all over world. (Root-knot nematodes (*Meloidogyne* spp.) are the most destructive genera and obligate parasites of more than hundreds of different plant species (Pulavarty *et al.* 2021). This genus has more than 90 species with several damaging and non-damaging races (Pulavarty *et al.* 2021). Four species of *Meloidogyne* (*M. incognita*, *M. arenaria*, *M. javanica*, and *M. hapla*) are major parasites of various crops across the world (Coyne et al., 2018).

Meloidogyne spp. parasitized different field crops, pastures, grasses to horticultural, ornamental, and vegetables. If root-knot nematodes are found in deep-rooted, perennial crops, control is difficult and options are constrained. Intrusions of juvenile nematode mostly during incompatible interaction among non-virulent pathogens and resistant tomato varieties trigger a defense cascade, resulting in hypersensitive reactions. The nematodes access the roots of plants and change plant cells into the complex; multinuclear feeding

sites termed as giant cells (Figure 3) (Bartlem et al., 2014). During giant cell organogenesis, progenitor plant cells undergo a variety of morphological alterations, including cytoplasmic actin, which gives the cells a fragmented and chaotic appearance. Plants regulate their assembly of actin filament by expressing actin-binding protein-like profilins. The infectious root-knot nematode juveniles express profiling called MiPFN3 that acts as an effector and makes the plant more susceptible to parasitism (Leelarasamee et al., 2018).

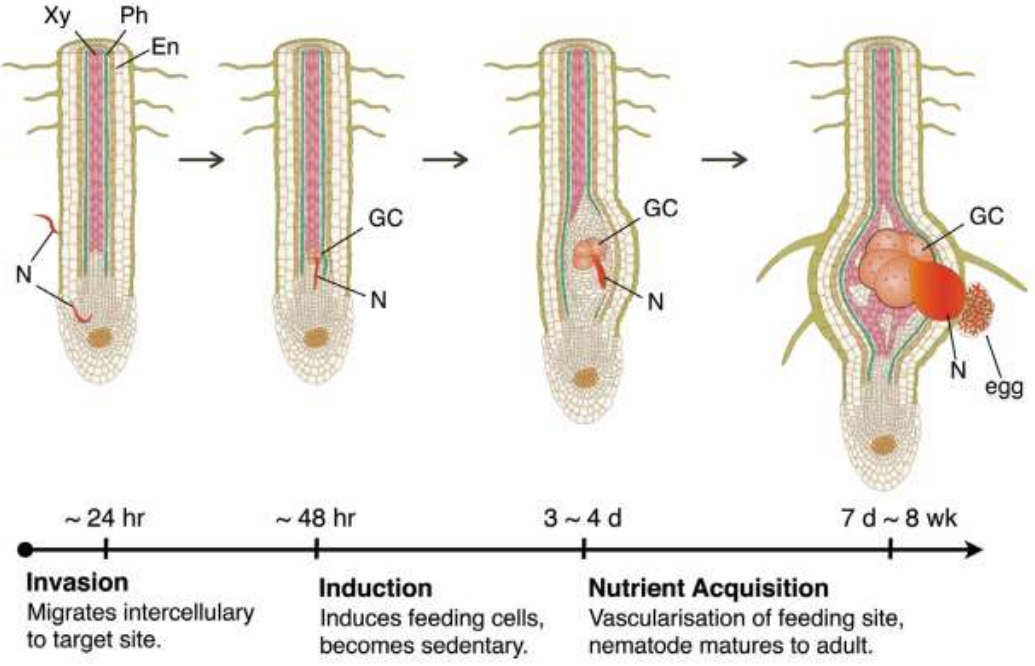

Figure 3. Schematic demonstration of the root-knot nematode (RKN) infestation life-cycle to form giant cells (Bartlem et al., 2014).

Morphologically male and female counterparts of root-knot nematodes are distinct. Males are mostly eel shape worm-like, about 1.2cm -1.5mm long and 30-36µm in diameter. The female nematode is pear in shape, about 0.40-1.30mm long and 0.27-0.75mm wide. Approximately every female of root-knot nematode lay 500 to 600 eggs in a gelatinous mass. The 2nd stage juveniles' eel shape is only the infective stage that emerges from the egg. If it extends the susceptible host plant roots, the J2 becomes sedentary when invaded by the host plant roots (Figure. 3) and profuse like a sausage shape. The juvenile inserts its style in cells around his head and secrete saliva into it. This saliva softens and liquefies the contents of cells and feeds on them. After that, nematode undergoes the 2nd molt and gives rise to the third stage juvenile (J3), which is firmer and goes through the 3rd molting stage and gives rise to the fourth stage juvenile (J4) and distinguished either male or female. In the 4th molting stage, male nematodes emerge from the roots and become free squanders in the soil, while female nematodes grow in length and width and look like a pear (Figure 4). After some time, female swelled and laid the egg in a gelatinous matrix with or without fertilization with the male, these egg masses are embedded in the root cortex. The eggs may hatch after 3 -15 days or over winter till the next spring season. The life cycle of root-knot nematode is mostly complete within 30-35days at 27°C$\pm$2 but proceeds longer at lower temperatures. After hatching, J2 migrates to the adjacent root part and causes a new infection in the same root or other root parts. Mostly root-knot nematodes are found in rhizosphere area 5-25cm below the surface (Karssen et al., 2013).

Although mostly root-knot nematode damage diagnoses occur underground parts of plants like roots tubers bulbs, and many symptoms appear in the above-ground part. Severely infected plants with a high nematode population density will cause wilting rapidly as root galling has limited the vascular bundles' ability to absorb water and nutrients of the whole plant. Even with sufficient water content, severely diseased plants showed permanent wilting, particularly in the afternoon. These symptoms were also confused with abiotic

stress (nutrients deficiency) due to lack of nutrient absorbance and transportation. That's why fertilization application in root-knot nematode-infested fields did not recover the wilting condition. Chlorosis, necrosis, stunting growth is frequently observed in host plants grown in the nematode-infested field leading to a reduction in yield. The death of plants mostly occurs in the early season of growing plants with minimum root mass. The attack of nematode is primarily found in patches (foci). Root-knot nematodes move slowly in the soil; parasitism will gradually radiate from the initial infestation point. Cultural practices rapidly spread the root-knot nematode in the whole field (Khan, 2003).

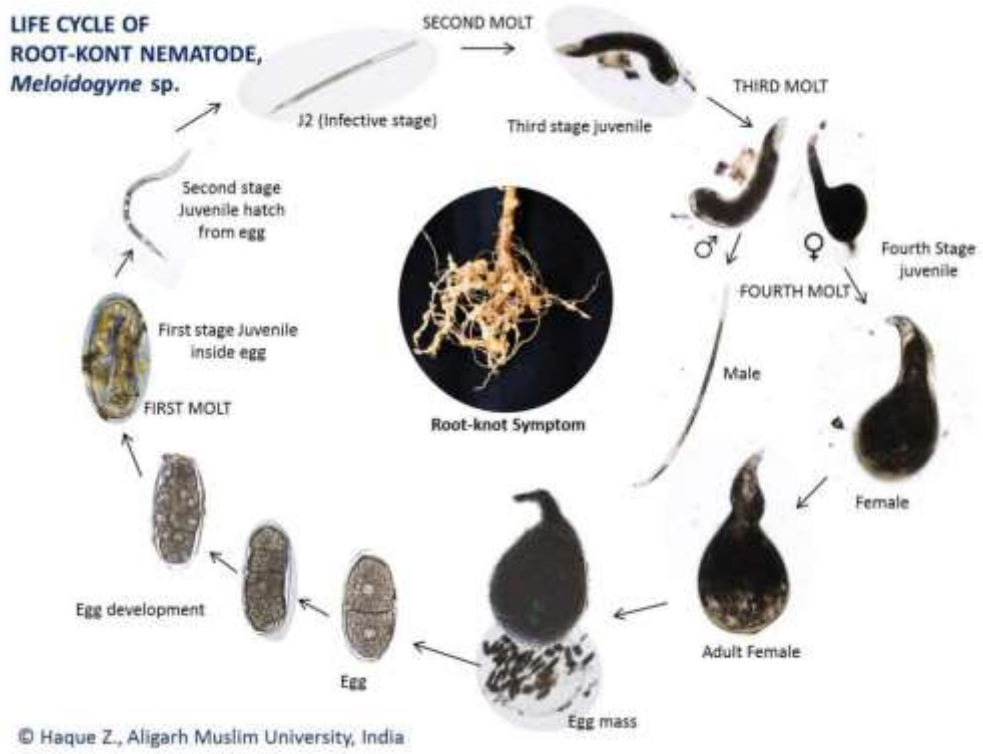

Figure 4. Life cycle of Root knot nematode (Dallavalle et al., 2005).

RKNs are increasingly organized parasites that sabotage host mechanisms to activate and maintain feeding cells within the host roots by secreting effector molecules to finish their life cycle within 4-6 weeks (Nguyen, 2016). The effector molecules contain peptides, enzymes and other biomolecules that have a crucial role in parasitism (Mitchum et al., 2004; Shukla et al., 2018). The immune system of plants suggested a zig-zag model that offers two divisions of plant defense response against pathogen induction: 1) ETI (Effector-triggered immunity) and PTI (Pattern triggered immunity) (Ávila Méndez and Romero, 2017; Haegeman et al., 2012). PAMPs (nematodes pathogen-associated molecular patterns) have recently been identified as individuals from a preserved set of the nematode pheromones (ascarosides) (Holbein et al., 2016). The resistance-associated R gene from tomato (Bhattarai et al., 2010) Mi-1.2, which belongs to the nucleotide-binding site–leucine-rich repeat (NBS-LRR) class, triggers ETI and deliberates resistance against three species of *Meloidogyne* (Ghosh et al., 2017; Rosen, 2012). Explicitly or implicitly, the correlation of the Mi gene with an avirulence (Avr) protein elicits still unknown, a cascade of signal transduction pathways that activate defense responses (Jones and Takemoto, 2004; Rossi et al., 1998). A comprehensive RNA sequencing method to explore the expression of the tomato plant and root-knot nematode genes in the roots of tomatoes at five different infection time intervals in susceptible cultivars and two infection time intervals in resistant cultivars. In susceptible cultivars, 1827 differentially expressed genes and 462 genes While in resistant cultivar 25 genes highly expressed in tomato and 160 in root knot nematode were identified (Shukla et al., 2018).

Interaction of nematode and fungi

The disease severity in horticultural crops has relied on the triangle complex relationship: the host, the strain of the pathogen, and epidemiological conditions. In nature, plant disease frequently includes numerous species or

genotypes. The different microbes consolidate as a malady complex on different crops. The plant-parasitic nematode interaction with various soil-borne fungi has been demonstrated all over the world (Table 1). Among plant-parasitic nematode and fungus complex relationships, root-knot nematode (*M. incognita*) is involved in a disease complex relationship with the fungus. Previously, many researchers worked on it.

Table 1. List of Nematode- fungus combine effect on different crops.

Nematode	Fungus	Crop	Reference
Meloidogyne incognita	*Fusarium oxysporum f.sp. vasinfectum*	Okra	(Agbaglo et al., 2020)
Meloidogyne incognita	*Fusarium oxysporum*	Tomato	(Kassie *et al* 2020)
Meloidogyne javanica	*Fusarium oxysporum f.sp. lentis*	Lentil	(Haseeb et al., 2005)
Root-Lesion Nematodes	*Rhizoctonia Solani*	Potato	(Viketoft et al., 2020)
Pratylenchus spp	*Rhizoctonia solani*	Potato	(Björsell et al., 2017)
Meloidogyne incognita	*Fusarium oxysporum*	Carnation	(Meena et al., 2016)
Meloidogyne incognita	*Rhizoctonia solani*	Phaseolus vulgaris	(Al-Hazmi and Al-Nadary, 2015)
Meloidogyne incognita	*Rhizoctonia solani*	Okra	(Safiuddin and Shahab, 2012)
Meloidogyne arabicida	*Fusarium oxysporum*	Coffee	(Bertrand et al., 2002)
Meloidogyne incognita	*Rhizoctonia solani*	Peanut	(Abdel-Momen and Starr, 1998)
Meloidogyne incognita	*Fusarium oxysporum*	Cotton	(Starr et al., 1989)
Meloidogyne incognita	*Fusarium oxysporum*	Pigeonpea	(Marley and Hillocks, 1996)
Globodera rostochinesis	*Rhizoctonia solani*	Potato	(Back et al., 2010)
Meloidogyne incognita	*Fusarium oxysporum* f.sp. *lycopersici*	Tomato	(Ansari et al., 2012)

The combined effect of *Pythium aphanidermatum* and *M. incognita* was observed on five different cultivars of tobacco regarding variation in growth and physiological parameters. All cultivars showed susceptibility toward *M. incognita* and developed a galling index (1.33-3.00). On the other hand, in the combined application of *M. incognita* with *P. aphanidermatum* the root rot index was significantly increased in those varieties resistant to fungus previously. Variation in growth attributes like chlorophyll and carotenoid contents decreased significantly as root rot index progressed. Overall, combined salicylic acid and total phenolic contents in resistant cultivars were higher in combined inoculation than the control and single *M. incognita* infestation (Khan et al., 2011). (Björsell et al., 2017) studied the spatial relationship of *Pratylenchus*spp, *Trichodoridae*, and free-living fungus *Rhizoctonia solani* caused severity in stem canker. (Parkunan et al., 2016) observed the concomitant and sequential interaction of *M. incognita* and *Phytophthra capsici* caused root rot on bell paper during greenhouse and field conditions on different cultivars. The *P. capsici* has a negative correlation with the nematode population, while *M. incognita* did not affect the severity of *P. capsici*. *F. oxysporum* f. sp. *niveum* caused devastating Fusarium wilt on water melon. It has three races, but races 1 and 2 are commonly found in watermelon-growing areas all over the world. (Hua et al., 2019) investigated root-knot nematode break resistance in resistant cultivars of watermelon against races 1 and 2 of *F. oxysporum* f.sp *niveum*. Results showed early wilting symptoms with high disease severity rate were observed with co-inoculation and sequential inoculation. Gall indices were reduced in the presence of fungus. Contrarily single application of *M. incognita* did not significantly reduce plant growth parameters.

Mechanism underlying synergistic interaction

Exploitation of nematode-induced injury by soil-borne pathogens

Different plant-parasitic nematodes caused damage to the host plants depending on their life cycle and feeding habit. For example, ectoparasite nematodes (*Trichodorus* spp. and *Tylenchorhynchus* spp.) mostly feed on epidermal tissues of roots and resulting in simple small puncture form injuries. Whereas migratory endoparasitic nematodes (*Pratylenchus* spp.) are distant more to their host plant roots, their stylet cut the cell wall with the help of its stylet to make a path and move intracellularly. The sedentary endoparasites *Meloidogyne*, *Globodera*, and *Heterodera* spp. have particular nourishing strategies. The second stage juvenile nematodes (J2) select invasion sites behind developing root tips (Grundler et al., 1992) and move either intracellularly by (*Globodera* and *Heterodera* spp.) or intercellularly by *(Meloidogyne* spp.) (Gravato-Nobre et al., 1999) to the

vascular cylinder, where specialized 'nurse cell systems' are initiated (Back et al., 2002). (Back et al., 2002) have investigated invasion sites of plant-parasitic nematodes (Akhtar and Malik, 2000) and the second stage adolescent nematodes (J2) select infiltration destinations behind developing root tips (Naalden et al., 2018) where specific 'nurture cell frameworks' are started (Teixeira, 2017). Some authors (Bergeson, 1972; Featonby-Smith and Van Staden, 1983; Orion and Hoestra, 1974) have studied nematode invasion sites and reported areas as unimportant in the etiology of fungal infections. Nevertheless, there are a lot of publications that perfectly demonstrate that nematode damage does have a role in the formation and growth of disease induced by soil-borne pathogens. (Inagaki and Powell, 1969; Lee and Lee, 2002) suggested the cracks and wounds formed from nematode invasion might be resourceful for secondary pathogen infection.

Nematode-instigate physiological changes to the host plant

The taking care of destinations of sedentary endoparasitic nematodes induced are zones of high metabolic action like (giant cells or syncytia), with an enormous number of Golgi bodies and mitochondria. At the same time, the cytoplasm is thick and contains numerous ribosomes (Escobar et al., 2015). Hence, nothing unexpected that these supplement-rich cells ought to turn into the substrate for parasitic colonization. The primary, objective evidence of this effect came from the treatment of tomato seedlings in gnotobiotic tissue culture with M. incognita and the subsequent removal of soil suspension from the rhizosphere of untreated tomato plants. Infection appraisal of these plants after 50 days and 85 days in the wake of planting demonstrated broad rot of their root framework. Several fungi (*F. oxysporum., R. solani*) were isolated from root galls of *M. incognita*.

Similarly, (Negron and Acosta, 1989) investigated that *F. oxysporum* f.sp. *coffee* instigated prolonged root rotting with the death of chlorophyll contents (yellowing) on the foliar part of coffee plants (cv. Bourbón) when the plants had been inoculated before 15 to 30 days with *M. incognita*. Root cross-sections taken from the plants pre-inoculated with *M. incognita* were discovered to be colonized by *F. oxysporum* f.sp. *coffee* in an exceptionally extraordinary manner from those where the fungus and nematode were either applied at the same time or where the organism was inoculated alone. In previous research, the mycelium of *F. oxysporum* f.sp. *coffee* were present in substantial amounts in vascular bundles and females of *M. incognita.* Many giant cells were in decay condition with depleted contents, where Fusarium spp colonized. The concurrent inoculation of *F. oxysporum* f.sp. *coffee* and *M. incognita* induced fewer giant cells with no mycelium within vascular bundles. Contrarily, pre-inoculation of *M. incognita* 3-4 weeks before Fusarium were induced to be disease complex linked with syncytial development in susceptible host plants (Negron and Acosta, 1989). (Parkunan et al., 2016) (Manzanilla-López and Starr, 2009) found that a decrease in the peanut yield was fundamentally more noteworthy in the coinfection of *M. javanica* and *R. solani* where a grouping of contagious development was found around the galled areas. These investigations demonstrate that certain plant microbes might effectively choose nematode-parasitized plant tissue. As indicated by (Back et al., 2002; Taylor et al., 1991), syncytia or giant cells contain more elevated levels of total protein, amino acids, lipids, DNA, and sugars valuable to numerous parasites. This would uphold the proposal that nematode contamination upgrades the dietary structure of bits of plants to organisms, yet the relationship stays doubtful. Just as these limited impacts, a few creators (Bird, 1967, 1974; Hussey and Williamson, 1998) have believed that nematode-induced physiological alterations may have a significant impact. In these situations, it is thought that the nematode-induced components or compounds beneficial to organisms can go into the plant (Khan, 1993). While researching this cycle, (Gomes et al., 2014; Hillocks, 1986; Katsantonis et al., 2003) used a technique known as "split-root" in which the root system of the mystery plant was divided into two different compartments, one of which was invaded by a connecting nematode species while the other was infected with a connecting infecting fungus.

Reduction of host resistance

In the advancement of different crop species that shows resistance from various biotic and abiotic stresses, the essentialness of nematode–fungus synergistic complex is only occasionally if at any point reported, yet various investigations report breakdown of resistance during associated infection (Katsantonis et al., 2003; Marley and Hillocks, 1994; Punja and Zhang, 1993; Shameer and Prasad, 2018). (Marley and Hillocks, 1994) reported, it is exasperating to the plant breeders to see his rewards for all the hard work come to nothing by the nematode–fungus intrigue'. Frequently, loss of resistance has been demonstrated using split-root procedures, as previously illustrated. Agents have received this kind of way to deal with deciding if the loss of microbe resistance actuated by nematode pervasion happens because of the breakdown of a foundational concoction safeguard framework inside the host plant. Two variet tomato, Rutgers and Homestead, are resistant to *F. oxysporum* f.sp. *lycopersici*, but progression

of wilting and rotting symptoms in combination with *M. incognita* (Prot, 1993). Furthermore, (Webster and Sidhu, 1977) performed grafting and root layering of the resistant scion of tomato to *M. incognita* with susceptible root stock of *F. oxysporum* f.sp. *lycopersici* to confirm the nematode is break-resistant or not. The results showed resistant scion free to *M. incognita* could block infection by *F. oxysporum* f.sp. *lycopersici.* Similar outcomes were seen (Vargas et al., 1996) on chili (*Capsicum annuum*), *Nacobbus aberrant* affected resistance capacity to *P. capsici*. The loss of resistance of *F. udum* in pigeon pea with nematode was demonstrated by Marley & Hillocks (1994). The loss of resistance capacity associated with the low level of cajanol, isoflavonoids, and phytoalexin especially cajanol reduced 62 % by combine infection of *F. udum* and *M. incognita.* Although this investigation shows that nematode invasion decreased a cellular defense component to fusarium in pigeon pea, it is yet unknown how nematodes action changed the plant physiology. (Hillocks et al., 2000; Marley and Hillocks, 1994) recommended that either the plants' overall metabolic process was slowed down or specific alterations were made to the mixture of isoflavonoids during nematode attack.

Certain scientists have a general understanding (Lindhout, 2002; Parlevliet, 1979) that in comparison to monogenic resistance, polygenic resistance is almost less constant. (Francl and Wheeler, 1993) express those plants with polygenic resistance from parasitic microbes are often found to get defense less to contagious assault during nematode pervasions. However, plants with a solitary prevailing quality for opposition are seldom influenced. This was seen by (Abawi and Barker, 1984) on tomatoes, where precautions from *F. oxysporum* f.sp. *lycopersici* was disturbed by pervasions of *M. incognita* on varities with polygenic resistance, yet not on those where a prevailing single I-gene expression connected resistance. Transgenic plants including quantitative attribute loci may have a more noteworthy limit with regards to giving strong resistance within the sight of collaborating microorganisms and PPN. (Back et al., 2002) hypothesized that healthy plants are carried without defense against bacteria through nematode physiological changes, which have no effect on the gene(s) responsible for encoding resistance. For instance, the cycle of intrusion by PPN may give soil-borne microorganisms entrances (Powell and Nusbaum, 1960) through a formerly impervious physical resistance chosen for in a plant defense system. Studies of illness edifices on various yield genotypes have shown that communications between types of nematodes and parasites can vary dramatically across plant species, cultivars, and lines (Khan and Husain, 1989). Subsequently, a few examinations have been not able to exhibit opposition misfortune (Jones et al., 1996; Ryan et al., 2000), but others have shown the contrary with mingling of nematode and fungus species that are difficult to differentiate. There are additionally other abiotic factors, for example, type of soil and its temperature, which have been appeared to influence cooperation (Umamaheswari and Chatterjee, 2008) and which may have differed between singular examinations on explicit malady edifice.

CONFLICTS OF INTEREST

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AUTHOR'S CONTRIBUTION

All authors contributed and supported towards writing of this manuscript.

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