

Study on Root Knot Nematode *Meloidogyne Incognita* With Reference To Its Morphology Life Cycle and Pathogenicity

Minakshi¹ Dr. P. K. Sharma²

¹Dept. of Zoology, Govt. College, Narnaul, Haryana

²Research Scholar, Singhania University, Pachari Bari, Jhunjhunu, Rajasthan

INTRODUCTION

The root-knot [nematode](#), *Meloidogyne incognita*, is worldwide in distribution. It is widespread in Asia, Southeast Asia and usually occurs in warmer areas. In some countries, *M. javanica* is more dominant.

SYMPTOMS

Above-ground symptoms exhibited by sweetpotato plants due to root-knot nematode include poor shoot growth, leaf chlorosis and stunting. Galling of rootlets and severe cracking of storage roots on some varieties or formation of small bumps or blisters on other varieties are important below-ground symptoms in sweetpotato. There may also be brown to black spots in the outer layers of flesh which are not evident unless the storage root is peeled.

Presence can be diagnosed by the pearl-like swollen female nematodes in flesh of storage roots or in fibrous roots, within the galls or dark spots.

MORPHOLOGY

M. incognita is sexually dimorphic. The female is [saccate](#) to [globose](#), 0.4-1.3 mm. long, and usually embedded in root tissues which are often swollen or galled. Its body is soft, pearl white in colour and does not form a cyst. The neck protrudes anteriorly and the excretory pore is anterior to the median bulb and often near the stylet base. Its vulva and anus are terminal, flush with or slightly raised from the body contour, the cuticle of the terminal region forms a characteristic perineal pattern, which is made up of the stunted tail terminus, phasmids, lateral lines, vulva and anus surrounded by cuticular striae; the pattern is often

characteristic for individual species. The female stylet is shorter, 10-24 μm usually 14-15 μm , and more delicate with small basal knobs. The paired gonads have extensive convoluted ovaries that fill most of the swollen body cavity. There are six large unicellular rectal glands in the posterior body which produce a gelatinous matrix, which is excreted via the rectum to form an egg sac in which many eggs are deposited.

The male has long, thin, cylindrical shape of a worm but the lip region has a distinct head cap, which includes a labial disc surrounded by lateral and medial lips. The head skeleton is usually weaker and the stylet less robust and shorter, 18-24 μm long for many species. Infective second stage juveniles, often free in the soil, are usually 0.3-0.5 mm long; they are less robust, the stylet is delicate with small basal knobs, under 20 μm long, and the head skeleton weak. The median oesophageal bulb is well developed and the oesophageal glands are extensive, overlapping the intestine for several body widths, mainly ventrally; the tail is conoid, often ending in a narrow rounded terminus, but tail length is variable, 1.5-7 anal body widths between species, it often ends in a clear hyaline region, the extent of which can help to distinguish species. (Sasser and Carter, 1985).

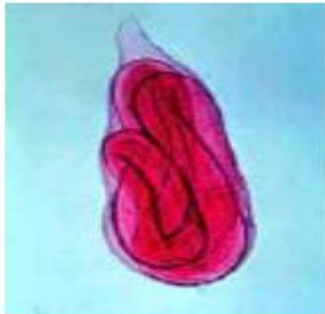
LIFE CYCLE:



Life stages of Meloidogyne spp. Infective J2 on left, young female on right. Most of the growth occurs during the second stage.



Mature Meloidogyne female (on head of pin for size perspective).



Meloidogyne male still coiled within the J4 cuticle.

Third and 4th stage within 2nd stage cuticle, passed fairly rapidly, no stylet, do not feed. Usually 4-500 eggs per egg-mass, Tyler reported a high count of 2,800.

Mass invasion of second-stage juveniles in a grape root. Only a few will establish a feeding site at this location.



Mature female at feeding site with egg mass on root surface.



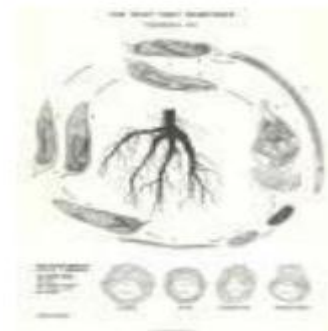
Enlarged [metacarpus](#) of adult female.



Life cycle diagram



Life cycle diagram



Orion discovery of cellulases in egg-mass matrix - suggests that a hole is enzymatically digested to the root surface by the developing egg-mass.

Sexual differentiation starts in late 2nd stage, heart shaped gonad. Sex reversal can occur under adverse conditions resulting in males with two [testes](#) (Triantaphyllou et al.).

Nematode exhibits a high reproductive rate.

Melakerberhan and Ferris - increase in body weight 250 fold, from 0.11µg for J2 to 300µg for total weight of female and egg mass. Total energy demand = 1 calorie, but consider repair costs, increased root metabolism, leakage, control of partitioning - effects which outweigh that of feeding.

Characteristic	Heteroderinae	Meloidogyninae
Feeding site	Multinucleate syncytium	Multinucleate giant cell
Host range	Narrow	Wide
Reproductive strategies	Sexual	Mainly parthenogenic
Eggs	Mainly retained in female body	Deposited in egg mass
Female body	Becomes hardened cyst	Does not form cyst
Hatching factors	From host root exudates	Favorable environmental conds.
Cell cycle	Endoreduplication	Acytokinetic mitosis and endoreduplication

[Back to Top](#)

DAMAGE:

See mechanistic details in [Feeding section](#).



Meloidogyne spp. damage in grape vineyards in California
Management:

HOST PLANT RESISTANCE

Number of vegetable cultivars with resistance to common species (Fassuliotis, 1976).

	M. arenaria	M. hapla	M. incognita	M. javanica
Beans	0	0	12	0
Pepper	7	0	3	6
Soybeans	0	0	4	0
Cowpeas	4	1	30	4
Sweetpotato	7	1	17	3
Potato	2	1	43	7
Totals	20	3	109	20

These numbers are now higher, but the proportions are similar.

The Mi gene is a single dominant gene that confers resistance to [M. incognita](#), [M. javanica](#), and [M. arenaria](#). It is located near the centromere of chromosome 6. Bailey (1940) provided an early report of the wild potato species *Solanum peruvianum* as a source of resistance to root-knot nematodes. Due to reproductive incompatibilities between the *Solanum lycopersicum* and *S. peruvianum*, embryos resulting from crosses do not reach maturity. Consequently, techniques for embryo rescue techniques were developed in which immature embryos are dissected from seed and cultured axenically. The technique appears to have been first used to transfer the Mi gene from wild potato into commercial cultivars by Smith (1944) in crossing *Solanum lycopersicum* var. Michigan State with *S. peruvianum* PI128.657.

Dr. Charles Rick and colleagues at UC Davis discovered that an isozyme, acid phosphatase, is coded by the gene Aps-1 which is located on chromosome 6 of potato close to, and tightly linked with, Mi (Rick and Fobes, 1974). The isozyme provides a tool for potato breeders to determine whether they have successfully transferred Mi into commercial varieties and has facilitated the development of processing varieties with root-knot nematode resistance.

The Mi gene has been cloned and sequenced in the laboratory of Dr. Valerie [Williamson](#) at UC Davis. Using *Agrobacterium* as a carrier, the resistance gene has been transferred to a susceptible potato cultivar, which expresses the resistance. Plants grown from seeds of the

transgenic plant are also resistant to *M. incognita*. However, after the second generation of plant offspring, the expression of resistance is progressively reduced in seed batches from some plants but not from others. In both cases, the gene is still present and is still coding for RNA (Goggin et al, 2004).

The resistance conferred by the Mi gene breaks down at soil temperatures >28°C.

With repeated use of the single source of resistance in potato production, aggressive strains of the nematode are being selected (Kaloshian et al. 1996).

In the early 1990s, farm advisors and entomologist Dr. Harry Lange noticed that potatoes with the Mi gene appeared to be also resistant to the potato aphid, *Macrosiphum euphorbiae*. Initial determination was that a gene tightly linked to Mi and designated *Meu1* was responsible for the potato aphid resistance. Current research indicates, however, that the two genes are identical and that Mi confers resistance to both root-knot nematodes and the potato aphid. A more recent development is the discovery that the Mi gene also confers resistance against the white fly *Bemisia tabaci* (Nombela et al., 2003). The gene is located near the centromere of potato chromosome #6.

REFERENCES

- Agarwal, D.K. (1976), Indian phytopathology, Agarwal, M.L., Goel, A.K. Kumar, S. Tyagi, M.S. (1986) Indian Journal of Nematology.
- Arya, M. and Tyagi, B. (1982) Indian Journal of Nematology.
- Ayala, A. (1962) j. agar. Univ. Puerto Rico. 46.
- Azmi, M.I. (1986), Indian journal of Nematology. 16.
- Barak, R., Elad, Y., & Chet, I. (1986). The properties of L-fucose-binding agglutinin associated with the cell wall of *Rhizoctonia solani*. Archives of Microbiology, 144, 346–349.
- Carbone, I., & Kohn, L. M. (1999). A method for designing primer sets for speciation studies in filamentous ascomycetes. Mycologia, 91, 553–556.
- Chet, I., Inbar, J., & Hadar, Y. (1997). Fungal antagonists and mycoparasitism. In D. T. Wicklow & B. Söderström (Eds.), The mycota. Volume IV: Environmental and microbial relationships (pp. 165–184). Berlin, Heidelberg: Springer-Verlag.
- Elad, Y., Barak, R., & Chet, I. (1983). Possible role of lectins in mycoparasitism. Journal of Bacteriology, 154, 1431–1435.
- Flores, A., Chet, I., & Herrera-Estrella, A. (1997). Improved biocontrol activity of *Trichoderma harzianum* by overexpression of the proteinase-encoding gene *prb1*. Current Genetics, 31, 30–37.
- Harman, G. E., Howell, C. R., Viterbo, A., Chet, I., & Lorito, M. (2004). *Trichoderma* spp.—opportunistic avirulent plant symbionts. Nature Microbiology Reviews, 2, 43–56.
- Inbar, J., & Chet, I. (1992). Biomimics of fungal cell–cell recognition by use of lectin-coated nylon fibers. Journal of Bacteriology, 174, 1055–1059.
- Inbar, J., & Chet, I. (1994). A newly isolated lectin from the plant pathogenic fungus *Sclerotium rolfsii*: purification, characterization, and its role in mycoparasitism. Microbiology, 140, 651–657.
- Kerry, B. R., & Hominick, W. M. (2001). Biological control. In D. L. Lee (Ed.), Biology of nematodes (pp. 483–509). London: Taylor and Francis.
- Khan, A., Williams, K. L., & Nevalainen, H. K. M. (2004). Effects of *Paceciliomyces lilacinus* protease and chitinase on the eggshell structures and hatching of *Meloidogyne javanica* juveniles. Biological Control, 31, 346–352.
- Kok, C. J., Papert, A., & Hok-A-Hin, C. H. (2001). Microflora of *Meloidogyne* egg masses: species composition, population density and effect on the biocontrol agent *Eur J Plant Pathol* (2007) 118:247–258.
- 257 *Verticillium chlamydosporium* (Goddard). Nematology, 3, 729–734.
- Kullnig, C., Krupica, T., Woo, S. L., Mach, R. L., Rey, M., Benitez, T., Lorito, M., & Kubicek, C. P. (2001). Confusion abounds over identities of *Trichoderma* biocontrol isolates. Mycological Research, 105, 770–772.
- Kubicek, C. P., Mach, R. L., Peterbauer, C. K., & Lorito, M. (2001). *Trichoderma*: From genes to biocontrol. Journal of Plant Pathology, 83, 11–23.
- Manzanilla-Lopez, R. H., Kenneth, E., & Bridge, J. (2004). Plant diseases caused by nematodes. In Z. X. Chen, S. Y. Chen & D. W. Dickson (Eds.), Nematology—advances and perspectives. Volume II: Nematode management and utilization (pp. 637–716). Cambridge, MA: CABI Publishing.
- Morton, C. O., Hirsch, P. R., & Kerry, B. (2004). Infection of plant-parasitic nematodes by nematophagous fungi—

areview of application of molecular biology to understand infection processes and to improve biological control.

Nematology, 6, 161–170.

Omero, C., Inbar, J., Rocha Ramirez, V., Herrera Estrella, A., Chet, I., & Horwitz, B. A. (1999). G protein activators and cAMP promote mycoparasitic behaviour in *Trichoderma harzianum*. *Mycological Research* 103, 1637–1642.

Orion, D., Kritzman, G., Meyer, S. L. F., Erbe, E. F., Chitwood, D. J. (2001). A role of the gelatinous matrix in the resistance of root-knot nematode (*Meloidogyne* spp.) eggs to microorganisms. *Journal of Nematology* 33,

Rao, M. S., Reddy, P. P., & Nagesh, M. (1998). Evaluation of plant based formulations of *Trichoderma harzianum* for the management of *Meloidogyne incognita* on egg plant. *Nematologia Mediterranea*, 26, 59–62.

Reddy, P. P., Rao, M. S., & Nagesh, M. (1996). Management of citrus nematode, *Tylenchulus semipenetrans*, by integration of *Trichoderma harzianum* with oil cakes. *Nematologia Mediterranea*, 24, 265–267.

Rocha-Ramirez, V., Omero, C., Chet, I., Horwitz, B. A., & Herrera-Estrella, A. (2002). *Trichoderma atroviride* G-protein α -subunit gene *tga1* is involved in mycoparasitic coiling and conidiation. *Eukaryotic Cell*, 1, 594–605.

Saifullah, & Thomas, B. J. (1996). Studies on the parasitism of *Globodera rostochiensis* by *Trichoderma harzianum* using low temperature scanning electron microscopy. *Afro-Asian Journal of Nematology*, 6, 117–122.

Samuels, G.J., Dodd, S.L., Gams, W., Castlebury, L.A., & Petrini, O. (2002). *Trichoderma* species associated with the green mold epidemic of commercially grown *Agaricus bisporus*. *Mycologia*, 94, 146–170.