INHERITANCE OF RESISTANCE TO ROOT-KNOT NEMATODE, *MELOIDOGYNE JAVANICA* (TREUB) CHITWOOD, AND SOME YIELD CHARACTERS IN TOMATO, *LYCOPERSICON ESCULENTUM* MILL.

BY

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DECLARATION

This thesis is my original work and has not been presented for a degree in any other University.

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ABSTRACT

The most common root-knot nematode species in the field at Thika comprising about 99% was found to be *Meloidogyne javanica.* Separate trials were conducted to determine the yield loss and the inheritance of resistance to this root-knot nematode species using three parents; T7 and T4 (resistant lines from U.S.A.) and Moneymaker the popular commercial (susceptible) fresh market tomato cultivar grown in Kenya.

Based on galling scores two resistance genes, one recessive, the other dominant were found in T7 and T4 and were designated as $LMjr_1$ and $LMjR_2$, respectively. These genes are non-allelic and could be located in the same chromosome distant apart or in different chromosomes. Although no linkage studies were done it is reasonable to assume that the $LMjR_2$ dominant resistant gene in T4 is either identical or closely linked to the *Mi* gene of Gilbert and McGuire (1956) found in chromosome VI. Histopathological studies showed that there was reduced larval penetration in T7 and T4 compared to susceptible' tomato, Moneymaker due probably to toxic chemical substances in their roots. However,

in case of T7 some female nematodes developed to maturity and laid eggs.

It seems that host resistance and galling response in T7 appears to be controlled by separate genetic mechanisms. Hence it was not surprising when yield losses in terms of fresh fruit weight in both Moneymaker and T7 (31.9% and 28.8% respectively) and fruit numbers (38.7% in Moneymaker and 27.8% in T7) were noticed.

The $\underline{LMjR_2}$ resistance gene in T4 seems to be the more reliable and could be transferred to the susceptible commercial Moneymaker by using the backcross-pedigree method.

In the inheritance of yield and component characters the means of six populations, namely $\bar{P}_1$, $\bar{P}_2$, $\bar{F}_1$, $\bar{F}_2$, $\bar{B}_1$ ($\bar{P}_1\bar{F}_1$) and $\bar{B}_2$($\bar{P}_2\bar{F}_1$) were used to estimate the various gene effects using the method of Gamble (1962). It was found that for yield; additive, dominant and dominant x dominant gene effects played a major role in two out of the three crosses. In the fruit size characters (fruit diameter, length and locule number) the dominance and dominance x dominance gene effects contributed more than the additive and additive x additive gene effects in all the three crosses.

CHAPTER 1

INTRODUCTION

Tomato, Lycopersicon esculentum Mill, is grown in Kenya mainly for local consumption as fresh or processed products such as juice, paste, sauce, ketchup and whole tomato. Recently, production has gone up such that it is exported to earn substantial foreign exchange (Appendix 1). The present population growth rate of 3.5% in Kenya and the rapidly expanding tourist industry will further enhance the demand for tomatoes. The number of vegetable processing factories are also expanding and they all need high quality tomatoes. The factories dealing with tomatoes are mainly Kabazi Canners Ltd, Trufoods Ltd, and Kenya Orchards Limited.

The tomato production in Kenya is concentrated in Eastern and Central Provinces (Appendix 2 and 3). It can be seen from Appendix 2 that the area under tomatoes is increasing each year although there are some fluctuations depending on the year and season. The districts of Kiambu, Meru and Machakos produce the most tomatoes. Around Lake Nakuru they grow mainly to supply the Kabazi Canners Limited while the Taita Hills (around Wundanyi) supply the Mombasa market.

Among the diseases and pathogens attacking tomato and causing considerable yield losses in Kenya the most important ones are root-knot nematodes, late blight, bacterial canker and bacterial wilt. The root-knot nematodes attack a number of crops other than tomato and in some cases they cause considerable damage. Their presence in the roots of infested plants is associated with the characteristic galling of the affected roots. The plants remain stunted and in severe cases they can die. Hainsworth (1962) claimed that 10% of the agricultural produce of Kenya was lost due to nematode attacks principally Meloidogyne spp. Hollis (1962) reported a loss of 50-100% on potatoes while Parlevliet (1970) reported a yield loss of up to 50% in pyrethrum. Ngundo and Taylor (1974) reported a yield loss of up to 60% in beans. Other crops affected by root-knot nematodes in Kenya are pineapple, cauliflower, cabbage, pepper, vegetable marrow, eggplant, okra, onion, carrot, passion fruit and tree tomato (Whitehead, 1957).

The commonest root-knot nematode species found in Kenya according to Whitehead et al. (1960) are Meloidogyne javanica and M. incognita, the other species are rare. Out of these species M. javanica is the commonest and is mostly found between 1500m

and 2000m above sea level. In the higher altitudes above 2000m M. hapla becomes the commonest species while in the lower altitudes below 1500m it is mostly M. incognita. In the medium altitude where M. javanica is mostly found the soils are friable clays with iron and aluminium concretions and according to Hollis (1962) these are ideal for the nematode development.

Although the root-knot nematodes are reported in all tomato producing areas of Kenya no work has been done to assess the magnitude of the losses and their control. However, according to Southey (1965) the recommended control measures are:

(1) Use of resistant or tolerant cultivars.

(2) Use of soil fumigants such as DD, EDB and Nemagon.

(3) Rotation of Crops.

(4) Dry fallow.

The soil fumigants are expensive hence not economical to use. Rotation is difficult to practise because most of the crops are susceptible to root-knot nematodes and dry fallow means the land has to be left unproductive for a time hence farmers do not favour it. Use of resistant varieties seems to be the most reliable and economical method of control.

The current popular cultivars such as Moneymaker for fresh market and Roma, San-Marzano, Heinz 1350, Eilon and Mecheast for processing are all susceptible to root-knot nematodes. Some tomato lines reported to be resistant have been obtained from the U.S.A. and are being maintained at the National Horticultural Research Station, Thika. However, these tomato lines have not been screened for resistance to the nematode mixed species found in the field at Thika. Also the degree and nature of their resistance are not known. If found resistant they could be used in breeding varieties resistant to common root-knot nematodes in Kenya. This study was therefore undertaken with the following objectives:

(1) To identify the common root-knot nematode species attacking susceptible cultivars in Thika area.

(2) To screen the local and introduced parental tomat cultivars for resistance to common root-knot nematode, Meloidogyne javanica.

(3) To estimate the yield loss due to susceptibility to root-knot nematodes.

(4) To study the genetics of resistance in crosses between local susceptible and introduced resistant lines.

(5) To characterise the mechanism of resistance in the host plants.

(6) To study the inheritance of yield and quality characters and their relationship to resistance.

(7) To identify the promising segregants in crosses involving the local susceptible cultivar and resistant lines.

(8) To formulate breeding programmes aiming at developing high yielding tomato varieties resistant to root-knot nematodes.

CHAPTER 2

REVIEW OF LITERATURE

2.1. Tomato Parasitic Nematodes

The tomato, Lycopersicon esculentum Mill, is a host of 19 nematode genera (appendix 4) representing 40 species (Goodey et al. 1965). Out of these are included 7 species of root-knot nematode belonging to the genera Meloidogyne namely, Meloidogyne acronea Coetze, M. arenaria (Neal) Chitwood, M. arenaria thamesi Chitwood, M. hapla Chitwood, M. incognita (Kofoid & White) Chitwood, M. incognita acrita Chitwood, and M. javanica (Treub) Chitwood. This means that the commonest and well-known species, M. incognita, M. javanica, M. arenaria and M. hapla included in Chitwood's 1949 revision of the genus are all parasites of the common tomato.

In East Africa at least 25 genera of known and suspected plant parasitic nematodes have been recorded by Whitehead (1957). Out of these he reported 3 genera; Meloidogyne, Rotylenchulus and Helicotylenchus as being parasitic on tomatoes. He also reported that M. javanica is the commonest species in East Africa while M. hapla is common in the

pyrethrum growing areas of Kenya and that M. incognita and its variants are uncommon.

In the Thika area the plant nematologist, Mr. Kanyagia, S.T. (personal communication, 1979) reports that 75% of the root-knot nematodes belong to the species M. javanica and the remaining ones mainly to M. incognita.

2.2. The importance of root-knot nematodes in tomato cultivation

Chlorosis, stunting and aggravation of nutrient deficiencies are symptoms frequently associated with infection by the root-knot nematodes Meloidogyne spp.

Chitwood (1951) provided the first quantitative data to show that Meloidogyne hapla suppresses growth and yield of tomatoes. Since then much has been done to try and correlate initial nematode populations and subsequent yield losses. Sayre and Toyama (1964) provided data from field tests showing that low and medium densities of M. hapla (220 and 1980 larvae/kg soil respectively) increased numbers and weights of processing tomatoes compared to the control. Barker et al. (1976) reported that M. incognita and M. hapla

caused maximum yield losses of 20-30% with populations of 0-12, 500 eggs and larvae/500cm^3 of soil (sandy loam, temperature ca 20.7^{o}C) in one trial and yield losses of up to 85% and 50% due to M. incognita and M. hapla, respectively in the second trial (loamy sand, temperature ca. 24.8^{o}C) compared to non-infested control. In the second trial the yield loss was exaggerated because M. incognita predisposed tomato plants to the early blight fungus. Olthof and Potter (1977) showed that 260 and 1840 M. hapla larvae/kg of soil on tomato stimulated while 6120 and 27950 M. hapla larvae/kg of soil suppressed vegetative plant growth. At the two highest densities the cumulative fruit production (wt.) was suppressed by 10%, and 40% respectively. The same workers postulated that the increase in growth and yield at the lower densities was due to an increase in the size of the root system while at the higher densities yield was no longer directly related to root weight. They concluded that initial densities larger than 2000 larvae/kg of soil may require control. Wisnuwardana (1978) showed that at low inoculum densities (<100 larvae/kg soil) root growth was stimulated but at higher densities (>500 larvae) root growth was inhibited, flowering,

fruiting and ripening occurred earlier and yields were reduced. At the highest densities (up to 100,000 nematodes/kg soil) crop losses exceeded 50%.

Of late, more work has been done which throws some light on the possible causes for the growth and yield reduction. Wallace (1974) showed that nematodes have an effect on the photosynthetic rate of the plant. This effect is not linear for with an inoculum of 250 larvae, photosynthetic rate was less than in uninfected control plants but as inoculum level increased there was a rise and then a fall in photosynthesis. His results did not support the hypothesis that creation of metabolic sinks in the roots, caused by the formation of syncytia and galls, have a major influence on top growth. The inhibition of upward translocation of water and nutrients may be more important. Bird and Loveys (1975) concluded that organic nutrients required by the nematode originate as products of photosynthesis and that the nematode acts as a metabolic sink while the giant cells act as transfer cells. McClure (1977) confirmed the conclusion of Bird and Loveys.

Root-knot nematodes do not act alone in the field and what is called a yield loss may be due to an interraction of many factors including the nematodes themselves. These factors may range from low fertility of soil, to interractions of nematode-nematode, bacterium-nematode, fungus-nematode to a combination of all of them. Conroy et al. (1974) found no increase in both infection incidence and symptom severity in concomitant infection of tomato seedlings by Verticillium albo-atrum and the root-knot nematode Meloidogyne incognita acrita. Moura et al. (1975) reported that bacterial canker was more severe in tomato var. Manapal inoculated simultaneously with Corynebacterium michiganense (E.F. Sm.) H.L. Jens and M. incognita than those inoculated with the bacterium alone. Golden et al. (1975) reported that Rhizoctonia solani was specifically attracted to nematode gall tissue. Sclerotia were selectively formed on nematode galls. Shoemaker et al. (1979) reported that M. incognita had no synergistic effect on verticillium wilt of tomato.

2.3. Control Methods

Over the years a number of methods to control nematodes have been developed. Southey (1965) has conveniently placed these methods under six groups,

viz, cultural, biological, chemical, physical, regulatory control methods and use of resistant varieties. Sometimes more than one control method is used at a time. In the control of root-knot nematodes, Meloidogyne spp. on tomatoes 4 methods namely; cultural, biological, chemical and use of resistant varieties have been developed as the main ones.

2.3.1. Chemical Control Method*

Under this method one can either treat the plant or the soil. The former includes the use of systemics and plant dips while the latter involves chemical soil sterilization which can be achieved by injecting volatile compounds into the soil such as DD(1,3-Dichloropropene - 1,2 Dichloropropane), EDB (Ethylene Dibromide), chloropicrin, DBCP (1,2-dibromo-3-chloropropane) or mixing into the soil parent compounds which break down to produce fumigant gases such as sodium N-methyl dithiocarbamate (metham-sodium) whose principal active breakdown product is methyl isothiocyanate. The extent to which the fumigant gases can spread depends on soil porosity, moisture, temperature, composition and properties

*Appendix 5 gives a list of the chemicals and their active ingredients.

of the chemical and the way of application.

Peacock (1960) reported that foliar applications of maleic hydrazide inhibited root-knot development, although it had little effect on the nematodes in vitro. Radewald, et al. (1970) indicated that oxamyl (carbamoyl oximes) applied as foliar sprays to tobacco, sugar pumpkins, tomatoes and sweet potatoes offered systemic protection from the nematode. Bindra et al. (1971) reported some control of Meloidogyne incognita on tomatoes with parathion and dimethoate as root-dip treatments; malathion, fenitrothion, gardona, formothion, disulfoton and carbofuran were ineffective. Navarro (1971) also confirmed that oxamyl was effective against Meloidogyne sp. on tomato.

Treating the soil is more common than treating the plant. Mukhopadhyaya (1970) reported that DD, DBCP and V-C 13 reduced the numbers of nematode larvae in a potato and tomato field. Reddy et al. (1971)reported that pre-and post-inoculation soil treatment with thionazin and aldicarb (Carbamoyl oximes) completely freed tomato from nematodes, whereas BAY 25141, methomyl, and carbofuran were effective only at dosages that were phytotoxic. Kyrou (1973) reported that DD at 40

litres per $1000m^2$ significantly reduced the root-knot incidence and increased the yield in tomatoes. Hough and Thomason (1975) reported that infection of sugarbeet and tomato seedlings by larvae of M. javanica was inhibited by the use of aldicarb at 1µg/ml. Mcleod (1977) working with granular nematicides showed that aldicarb, ethoprop, oxamyl and phenamiphos controlled galling equally well, resulting in yield increases of 20-40%. Baker et al. (1977) reported that basamid (granular nematicide), Di-Trapex and chloropicrin + EDB are used to control nematodes on tomatoes in the North Coast of New South Wales. Orum et al. (1979) reported that oryzalin and BAS 083 reduced root-knot infection in tomato roots when applied as a soil drench at 20 ppm and 10,000 ppm respectively. It is not yet a common practice to use nematicides to control nematodes in tomato fields in Kenya because of the high cost involved.

2.3.2. Cultural Control Method

Cultural methods are attempts to adapt husbandry practices so as to minimize the effect of nematodes. Before deciding on the best means of combating a nematode, its life history, population dynamics, host range and the efficiency and susceptibility of its main host must be considered. These methods are sub-divided into 4 groups, viz, crop rotation, prevention of spread, selection of healthy propagating material and manuring. Crop rotation and manuring have been widely tried in tomato culture while the other two are of minor significance.

2.3.2.1. Crop Rotation

Le Roux *et al*. (1939) showed that one crop of tomatoes could be grown economically in South Africa on plots where control of nematodes has been achieved by clean cultivation. Sellschop *et al*. (1948) recommended that tomatoes should not be planted more than once in every 3 or 4 years with cereals or maize in the rotation. Peacock (1957) found that a cultivated bare fallow was most effective against root-knot nematodes and that it was most effective

during the dry season. Oostenbrink (1960) observed yield increases of at least 40% for several crops with corresponding reduction of population of Pratylenchus penetrans, P. crenatus and Tylenchorhynchus dubius by growing the Marigold, Tagetes patula. Daulton and Curtis (1963) obtained control of root-knot of tobacco in fields where T. patula, T. erecta and T. minuta preceded tobacco while Good et al. (1965) on the basis of rotational trials, reported that marigolds and Crotolaria spp. were most efficient in reducing a wide range of soil nematodes. Khan et al. (1971) reported that T. erecta when grown with different varieties of tomato during winter brought about reduction in root-knot development. Sinnadurai (1973) showed that in a two course rotation, tomato following a fallow was superior to that of tomato followed by cowpea, bambara nut or tomato in nematode infested soil. Recently, Adamson et al. (1975) reported that root-knot resistant roselle (Hibiscus sabdariffa) failed to reduce populations of Meloidogyne incognita acrita and M. javanica to allow crops of kenaf (H. cannabinus) to be grown in following years. He also showed that continuous cultivation of

roselle for 3 years failed to reduce the root-knot larval population significantly compared to the level found after one year.

2.3.2.2. Manuring

Linford et al. (1938) was the first to observe the beneficial effect of organic matter on the reduction of nematode populations. It was believed that decomposition of the organic matter resulted in the build up of nematode capturing fungi, non-trapping fungal parasites of nematodes, predacious nematodes and predacious mites. Oostenbrink (1950) observed that organic manures suppressed the rate of infestation and reproduction of several plant-parasitic nematodes. Duddington (1957), Lear (1959) and Hutchinson et al. (1960) also reported that adding certain kinds of organic matter to soil reduces nematode populations although Duddington (1957), Linford et al. (1938) and Hutchinson et al. (1960) found no apparent correlation between the numbers of predacious nematodes and nematode-trapping fungi in areas in which parasitic nematode populations were lower due to the presence of organic residues. Johnson (1959) reported 75%

control of root-knot on tomatoes when 1% oat straw is added. Lownsberry (1961) and Mankau (1961) failed to control nematode populations by adding organic matter with or without supplemental nematode trapping fungi. Johnson (1962) showed that oat straw and lespedeza hay residues significantly controlled M. incognita when the soil and residue were incubated at 25-30°C, then put under a medium moisture level at a pH of 4.6 - 7.0. Singh (1964) reported beneficial effect of various organic amendments in reducing the infestation of root-knot nematode on tomato. Recently, Hameed (1970) showed that organic additives from neem, Melia azadirachta L. and chrysanthemum followed by marigold profoundly minimized the incidence of the nematodes and also increased the plant growth appreciably. Goswami and Swarup (1971) got an appreciable check in nematode population along with an increase in the vigour of tomato plants with Karanj, Pongamia glabra and groundnut cakes. Sitaramaiah (1978) showed that roots of tomato grown in soils amended with margosa, Azadirachta indica cake or sawdust supplemented with NPK had a higher total phenol content than plants grown in non-amended soil. This imparted

some resistance to invasion by M. javanica larvae.

Practical control of root-knot nematodes under field conditions by amending the soil with organic materials is yet to be realized. If a high rate of residue addition (25 tons/ hectare) is required, its value as an effective means of practical control might be limited.

2.3.3. Biological Control

This involves the use of enemy plants, trap cropping, nematophagous fungi, virus and predacious and parasitic animals. It is in a way very much interrelated to the cultural control method and the two are usually used in a combination (Oostenbrink, 1960; Daulton et al., 1963 and Good et al.,1965). In the control of root-knot nematodes enemy plants and trap cropping have been used successfully.

2.3.3.1. Enemy Plants

In a few cases plants have been found to reduce nematode numbers by producing materials in their roots that are toxic to nematodes. These are the enemy plants. Rohde (1972) reports that Asparagus officinalis produces a glycoside in its roots, stems and

leaves which is toxic to Trichodorus chistiei. Tagetes patula and T. erecta produce thienyl compounds that are toxic to Meloidogyne spp. and Pratylenchus spp. The marigolds (T. patula and T. erecta) are commonly used in rotations to lower root-knot nematode populations in heavily infested fields in the temperate countries.

2.3.3.2. Trap Cropping

Trap crops are usually heavily invaded by parasitic larvae, thus reducing the total number of larvae in the soil. Since no reproduction occurs in these non-hosts, the population does not increase. Barrons (1940) demonstrated that Meloidogyne larvae freely entered the roots of crotolaria but failed to survive. In this case the possibility of toxic action cannot be ruled out but the main effect is probably that of a non-host crop (Van der Linde, 1956; McBeth & Taylor, 1944).

The use of highly susceptible crops has been proposed as a means of trapping nematodes. Such plants have to be destroyed before the nematodes reach maturity and begin reproducing. To do this one has to have a

thorough knowledge of the nematode life cycle which has to be determined in each particular situation because of many inherent factors involved. It is a difficult method and can only be used where it is backed with technical knowledge.

It has been reported recently by Motsinger et al. (1977) that certain French marigold cultivars serve as a trap crop rather than as producers of toxins from their roots.

2.3.4. Use of Resistant Varieties

Resistance to Meloidogyne spp. is usually the type where larvae penetrate resistant plants but no reproduction of the eelworms takes place. Goplen and Stanford (1959) however, reported that resistance to M. hapla in one lucerne stock was due to complete failure of larvae to penetrate the roots.

Since the recovery of homozygous lines from a cross of Lycopersicon peruvianum (L.) Mill and L. esculentum Mill many root-knot nematode resistant tomatoes have been developed especially in the temperate countries. These include cultivars such as Nemared, Nematex and hybrids, e.g. Beefeater

(VFN), Small Fry VFN.

2.4. The Source of Resistance to Root-knot Nematodes on Tomatoes

The early work on breeding for root-knot nematode resistance on tomatoes was both disappointing and discouraging as there was no information on the source of resistance. It was not until 1939 when Barrons listed tomato varieties with varying degrees of resistance. Bailey (1941) reported a high level of resistance to root-knot nematodes in wild peruvian tomato, Lycopersicon peruvianum (L) Mill. Romshe (1942), Ellis (1943), McFarlane et al. (1946) and Watts (1947) confirmed Bailey's finding that a high degree of resistance to root-knot nematodes occurs in strains of L. peruvianum.

Taylor and Chitwood (1951), Sasser (1954), and Thomason and Smith (1957) reported the reactions of different collections of L. peruvianum to different species of Meloidogyne. Attempts to use this source of resistance failed due to the incompatibility of L. peruvianum and L. esculentum. The breakthrough was in 1944 when Smith produced an inter-specific hybrid between L. esculentum

(Michigan State Forcing) and L. peruvianum (PI 128657) by using the embryo culture technique. It was however, self-sterile and back-crossed with difficulty to L. esculentum parent. Since then Porte and Walker (1945), Alexander (1956), Choudhury (1957) and others have successfully produced interspecific hybrids using the same technique. Frazier and Dennett (1949) in Hawaii back-crossed Smith's hybrid to L. esculentum and isolated tomato lines which were homozygous for resistance to root-knot caused by M. incognita. Gilbert and McGuire (1952) confirmed the resistance of this material. Since the isolation of these homozygous resistant lines various tomato cultivars resistant to different Meloidogyne species have been developed. Although varieties resistant to M. javanica, M. incognita and M. arenaria have been developed there are very few reports of varieties resistant to M. hapla. Efforts must be made to locate a source of resistance to this species in different strains of L. peruvianum.

2.5. The Inheritance of Resistance to Root-knot Nematode on Tomatoes

The genetics of resistance in tomatoes (Lycopersicon esculentum) against the root-knot

nematodes (Meloidogyne sp.) has been studied by several workers . McFarlane et al. (1946) reported that resistance to root-knot nematode was dominant in comparison with susceptibility. Watts (1947) suggested two dominant genes for resistance to M. incognita in the early stage of plant growth. Frazier and Dennett (1949) postulated that one or two dominant genes were responsible for host resistance and later Gilbert and McGuire (1956) identified a single dominant gene for resistance in linkage group VI which they designated as Mi gene. Barham and Sasser (1956), Barham and Winstead (1957), Thomason and Smith (1957), Winstead and Barhan (1957) and Hernandez et al. (1965) independently reported that resistance to root-knot nematodes in tomato is controlled by a single dominant gene. In particular Barham and Winstead (1957) reported the Mi R-gene to be incompletely dominant while Winstead and Barham (1957) indicated that some genes control resistance to different species of Meloidogyne and that these genes are dominant. Harrison (1960) showed that the resistance to Meloidogyne sp. in tomatoes was controlled by a dominant gene or a block of genes acting as a unit. Cordner, Thompson and Galeotti (1965) reported that a single

completely dominant gene controlls root-knot resistance in the tomato Nemared.

Sidhu and Webster (1973) showed that the resistance was monogenic and dominant in the tomato cultivars Nematex, and Small Fry and recessive in cultivar Cold Set. They tentatively designated the 3 R-genes possessed by the cultivars Nematex, Small Fry, and Cold Set as L MiR1, LMiR2 and LMiR3 respectively. It is the opinion of Sidhu and Webster (1975) that some of the tomato cultivars shown to possess the Mi gene may eventually be found to have different but very closely linked resistance genes or pseudoalleles depending upon their duration of association with the parasite and breeding background. Fatunia and Salu (1976) reported that resistance in Rossol and Nematex is separately conditioned by single genes. The genes are incompletely dominant and non-allelic. Recently, Kalloo et al. (1978) showed the resistance to M. javanica and M. incognita in tomato cultivars Nematex and 'R-2' to be controlled by a single dominant gene.

2.6. The Nature of the Resistance

The host-parasite interractions in plants resisting attack by root-knot nematodes may include the following characters; lack of root attraction, reduced larval penetration, failure of host response to the parasite, tolerance and hypersensitive tissue reactions.

Wiesser (1955) proved that the attractiveness of roots to larvae of Meloidogyne hapla is dependent upon rate of growth and degree of maturation of the root. He found that the apical 2mm (root-cap and meristematic region) of the excised root tips was repellant to the nematode. The next 6mm (region of elongation) was attractive and the remaining portion, up to 16mm behind the root apex, was either neutral or slightly repellant to the nematode. Dean and Struble (1953) reported reduced larval penetration by M. incognita larvae in the roots of resistant L. peruvianum compared to susceptible tomato, L. esculentum (Marglobe). Similar results were reported by Peacock (1959) who also reported that the larvae of M. incognita were attracted to excised root tips of L. peruvianum

a little less strongly compared to L. esculentum. He also noticed either a slower rate of development or little or no development of the larvae and swelling in L. peruvianum compared to susceptible tomato, L. esculentum. Gowen et al. (1969) reported that significantly fewer larvae entered the roots of Nemared compared to other tested varieties. He found no galls in the resistant Nemared 28 days after inoculation and the hybrids had significantly fewer galls than the susceptible tomato varieties tested.

Hypersensitive tissue reactions in tomato were reported by Webster and Paulson (1972). They postulated that in a resistant plant, the oesophageal secretions may activate a gene that produces a thermolabile enzyme that rapidly releases substances from the vacuoles and lysosomes which give the hypersensitive response. The effect of this enzyme is overcome by exogenous kinetin, thus supporting the role of cytokinins in the host response.

It is possible that unknown chemicals in plants may be responsible for resistance of the plant to root-knot nematode attack. These are:

(1) Masking the attractant substance in the root or by actively repelling the

nematode.

(ii) Killing the nematode on entry or retarding its development.

(iii) Neutralizing the effect of nematode saliva on giant cell formation.

(iv) Changing the composition of the cell-wall so that nematode saliva is no longer effective, or the cell-wall is impenetrable to the nematode stylet.

(v) Upsetting the sex-ratio of the nematode, either physiologically or by eliminating the females.

Toxic compounds in no case act as the sole mechanism of resistance but they work in conjunction with other factors (Rohde, 1972). Plants containing toxins are attacked and injured but development of the nematode is retarded and populations rapidly decline. Rohde (1972) reports that Marigolds, Tagetes patula and T. erecta are resistant to two genera of nematodes namely, Meloidogyne and Pratylenchus. The toxic compounds produced by these plants are thienyl compounds particularly α - terthienyl and 5-(3-buten-1-inyl)-2, 2'-bithienyl. Motsinger et al. (1977) reported that certain French Marigold cultivars serve as a trap crop rather than as a producer of nematicidal materials from their roots.

To understand the mechanism of resistance to root-knot nematode infection in host plants, it is necessary to study the metabolic make up of resistant and susceptible host plants, as well as a result of infection.

Metabolic changes which take place in host tissues as a result of root-knot nematode infection give rise to giant cell formation and the characteristic galling of roots. In tomato, such changes have been studied in detail by Myuge (1956), Owens and Navotny (1960), Bird (1962), Owens and Specht (1964, 1966) and Owens and Bottino (1966). They all observed that the internal plant-root environment has an important influence on nematode development through its reaction with nematode secretions injected at the time of entry of larvae into the host, or through the influence of exuded plant substances on nematodes. This suggests that resistant and susceptible plants have different chemical make-up. Meon (1979) reported decreased cytokinin activity in the xylem of tomato plants infested with M. javanica and increased abscisic acid concentrations particularly tops of infected plants. Gibberellin concetrations were not affected by the nematode.

Pi & Rohde (1967), Chia-Ling Hung and Rohde (1973), Singh and Choudhury (1973) and Alam et al. (1976) all reported an increase in phenolics with increase in degree of resistance in the tomato roots. According to Singh and Choudhury (1973) susceptible cultivars had 40-44 µg phenolics/gm of roots while cultivars with greater resistance had 79 - 117 µg phenolics/gm of root. They also found that the dry matter content was significantly lower in susceptible cultivars than in tolerant, resistant and immune cultivars and related wild species. The phosphorus content in the roots was significantly lower in tolerant, resistant or immune cultivars while there was no clear cut distinction in the total nitrogen and potash content of the different tomato roots. They also found the same number and kind of free and bound amino acids irrespective of the tomato reaction.

2.7. The Inheritance of quality Characteristics in tomatoes

The most important quality characters in tomatoes are flavour (which is due to sugars and acidity), Vitamin C content, fruit size and shape, firmness and colour.

Walkof and Hyde (1963) reported that high

acidity was dominant and controlled by a single gene. Lower and Thompson (1967) however, reported that the inheritance of acidity was largely quantitative, but a single major gene conditioning high acidity appeared to be segregating within his two genetic populations. Mittal *et al*. (1979) estimated gene effects on the total soluble solids in tomato and showed epistasis in a majority of of the crosses. Additive gene effect was significant in only three crosses while dominance was significant only in one cross. Allen *et al*. (1979) reported that improved tomato flavour can be achieved via increased sugar and acid content.

Reynard and Kanapaux (1942) showed that the wild species of *Lycopersicon peruvianum* (L) Mill possesses a high Vitamin C content compared to *L. esculentum* Mill. Reynard *et al*. (1942) demonstrated a negative correlation between fruit size and vitamin C content. Currence *et al*., 1951 as quoted by Mital *et al*. concluded that three genes controlled the quality of vitamin C while Poole, 1956 as quoted by Mital *et al*. reported that genes for high ascorbic acid content (Vitamin C) were largely dominant and that its concentration was negatively correlated with fruit size.

Young and MacArther, 1945 as quoted by Mital *et al*. reported that the size depends on 8-15 genes. Haughtaling (1935) showed that fruit size is related to cell size as well as cell number and that cell number does not increase after anthesis. Dempsey *et al*. (1956) showed that the number of seeds per fruit was significantly correlated with fruit weight and that each additional seed increased fruit weight by about one gram.

According to Rick and Butler, 1956 as quoted by Mital *et al*. the fruit shape is controlled by the interraction of two different pairs of genes, *o* and *lc*. *OLc* gives globular or round fruits. *oLc* plum-shaped fruits, *Olc* oblate or flattish fruits and *olc* gives giant plum fruits (rare).

CHAPTER 3

MATERIALS AND METHODS

The trials were laid out at the National Horticultural Research Station, Thika, about 50 km North of Nairobi City; centre coordinates $37^{o}04'E$; $0^{o}59'S$ at an altitude of 1550 metres above sea level. The soils are red to strong friable clays. The pH falls in the medium acidic range (5.3-5.9), available calcium is low while phosphorus is deficient. The carbon percentage varied from 1.94-2.18 in the experimental plots.

3.1. Identification of Nematode Species

The material consisted of galled roots of tomato cultivar Moneymaker grown at the National Horticultural Research Station, Thika. Fifteen heavily galled roots were washed thoroughly and the mature female nematodes were extracted. These were used as specimens for cutting of perineal patterns as described by s'Jacob and Bezooijen (1977). The specimens were mounted on slides and identified with the help of Professor Adeniji, a visiting Professor at the Department of Crop Science, University of Nairobi.

3.2. Resistance Scores

Plants from the different trials were uprooted and scored for nematode resistance using the classification of Gilbert and McGuire (1952):

Class 1: No visible galling (Fig. 1).

Class 2: Very light galling (Fig. 2).

Class 3: Moderate galling with no large galls but with a larger number of small galls than in class 2 (Fig 3).

Class 4: A wide distribution of small galls and/or some of larger size than in the lower classes (Fig. 4).

Class 5: Very heavy galling typical of the control plants (Fig 5).

Classes 1 to 3 were considered resistant while classes 4 and 5 as susceptible.

3.3. Screening of parents in the Field and Greenhouse

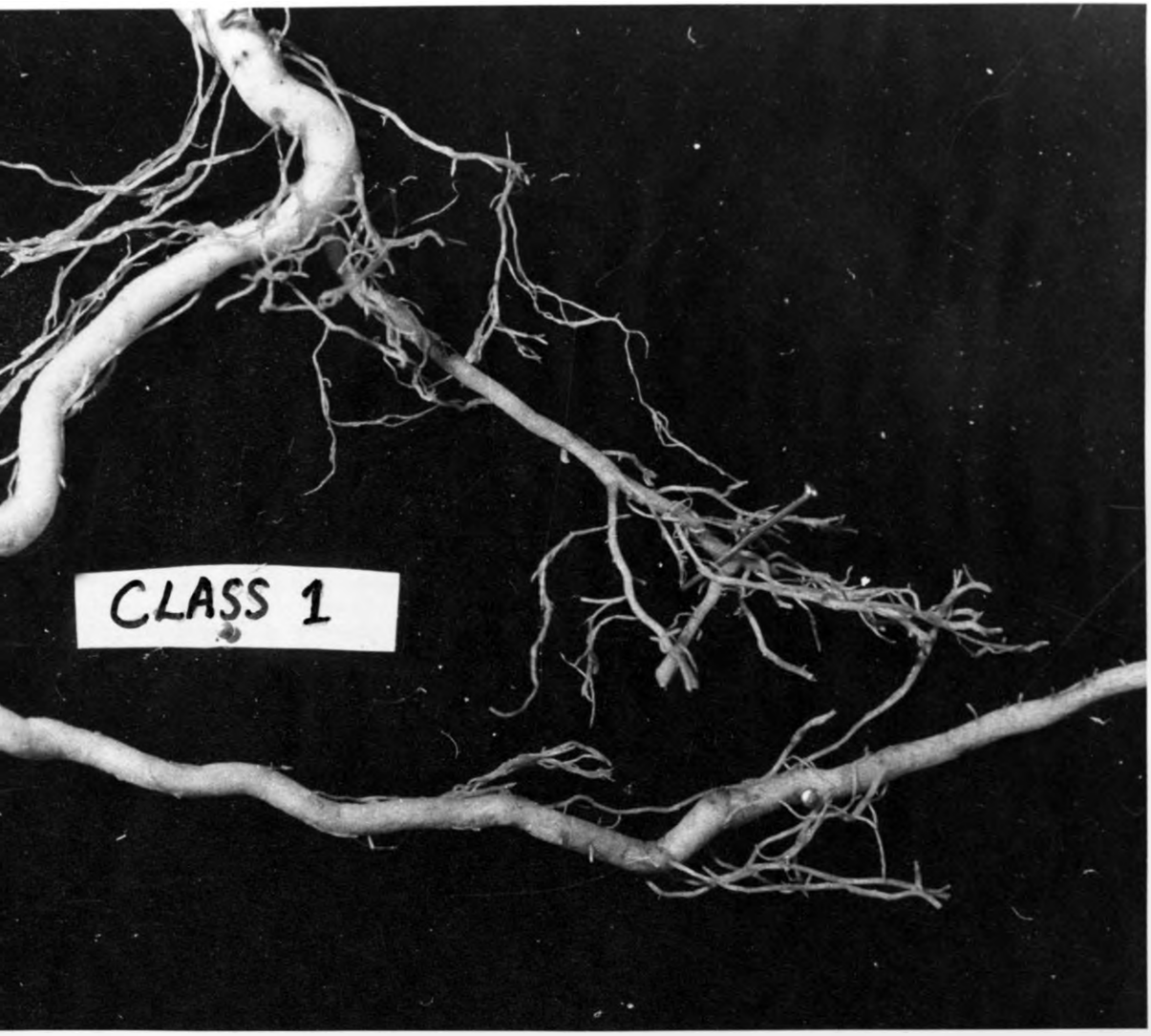
The objective of this trial was to ascertain the degree of susceptibility and resistance of the parental materials to be utilized in follow up trials.

Materials

(i) Two tomato lines from U.S.A. code named T7 and T4. T7 is a semi-determinate

Fig. 1.

Class 1: No visible galling


CLASS 1

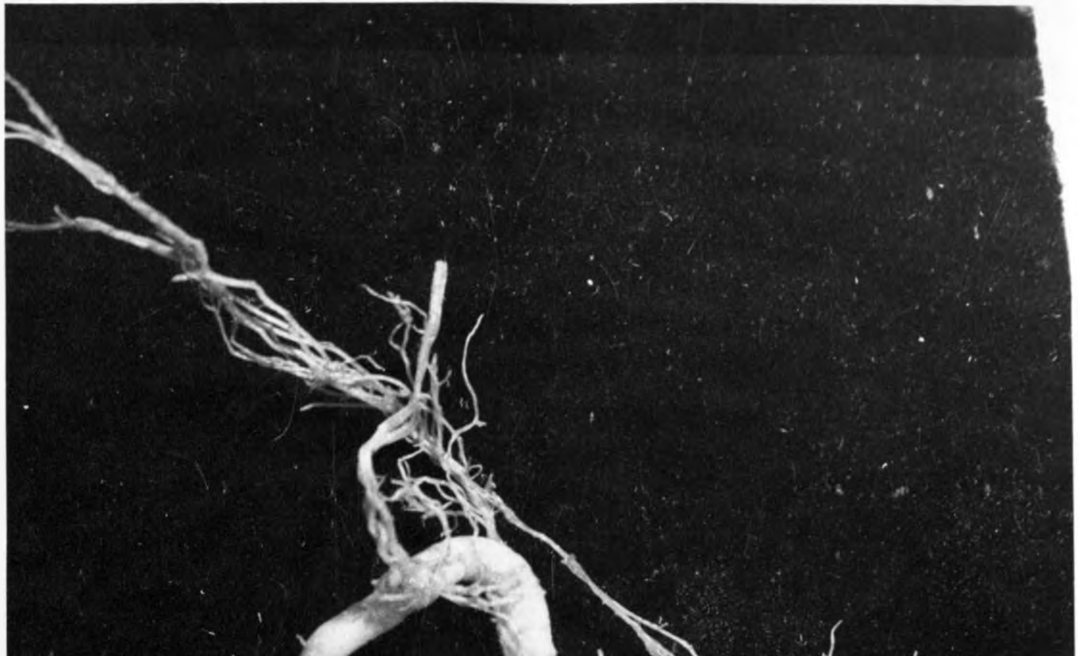


Fig. 2.

Class 2: Very light galling

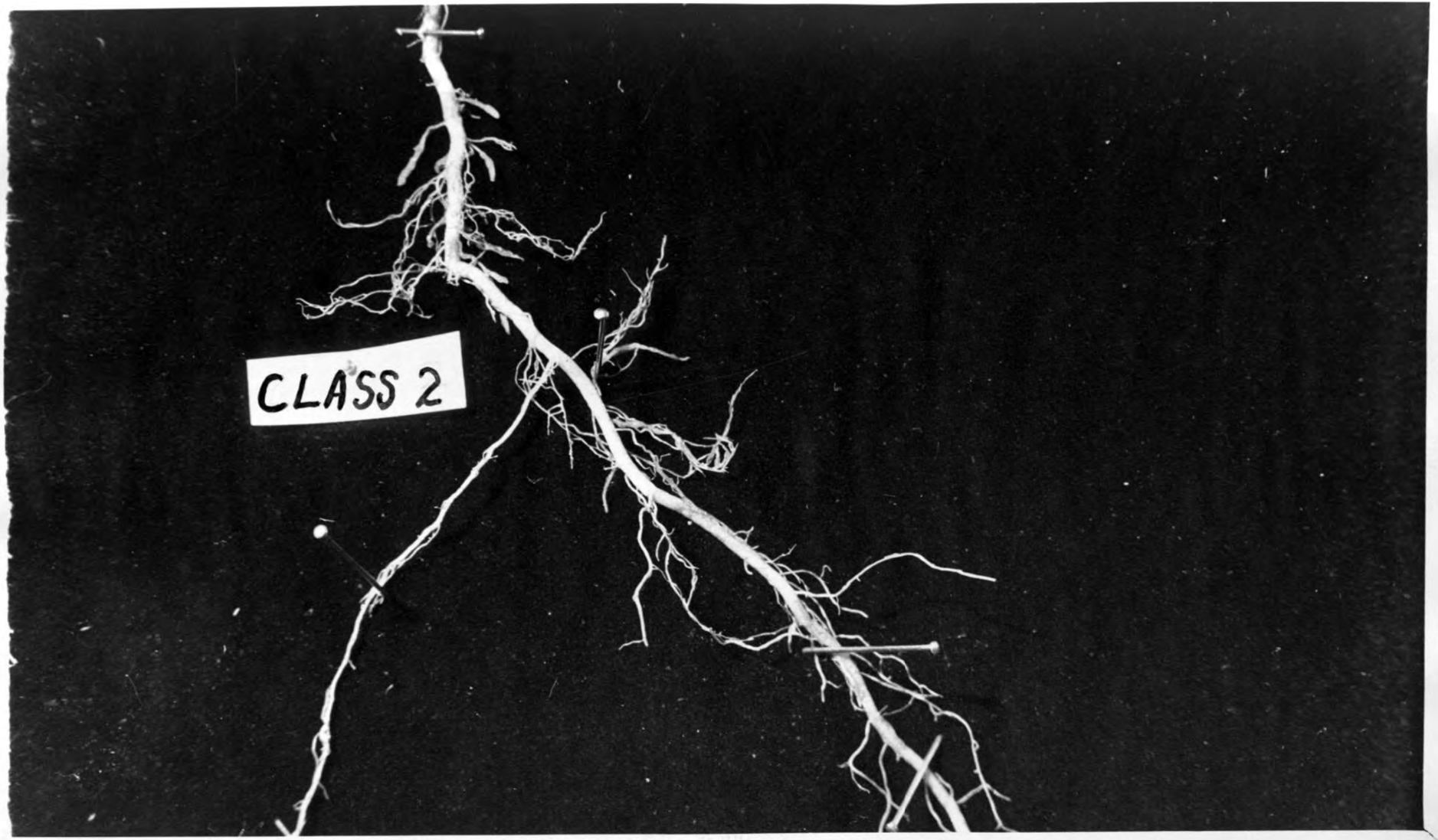
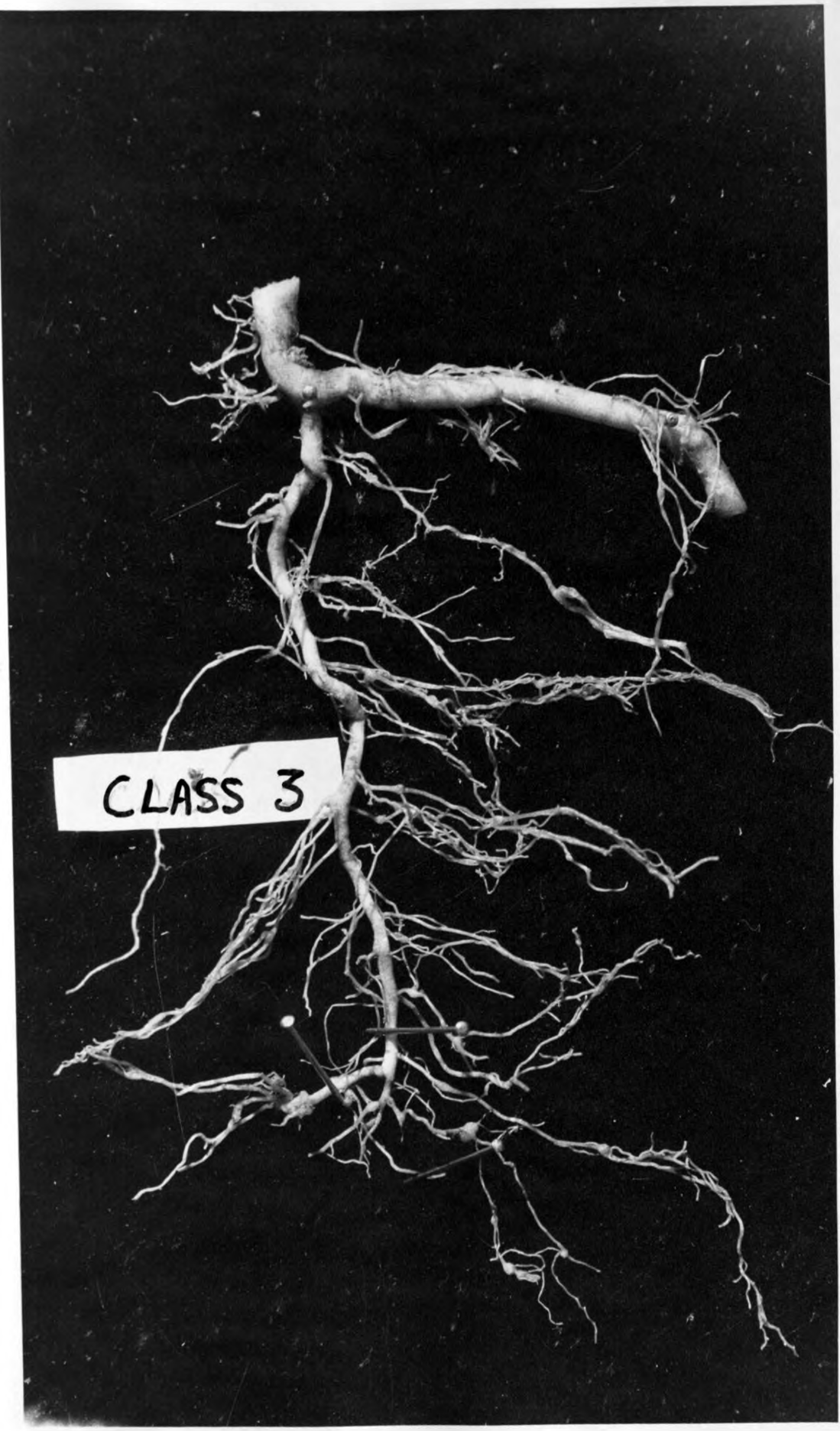
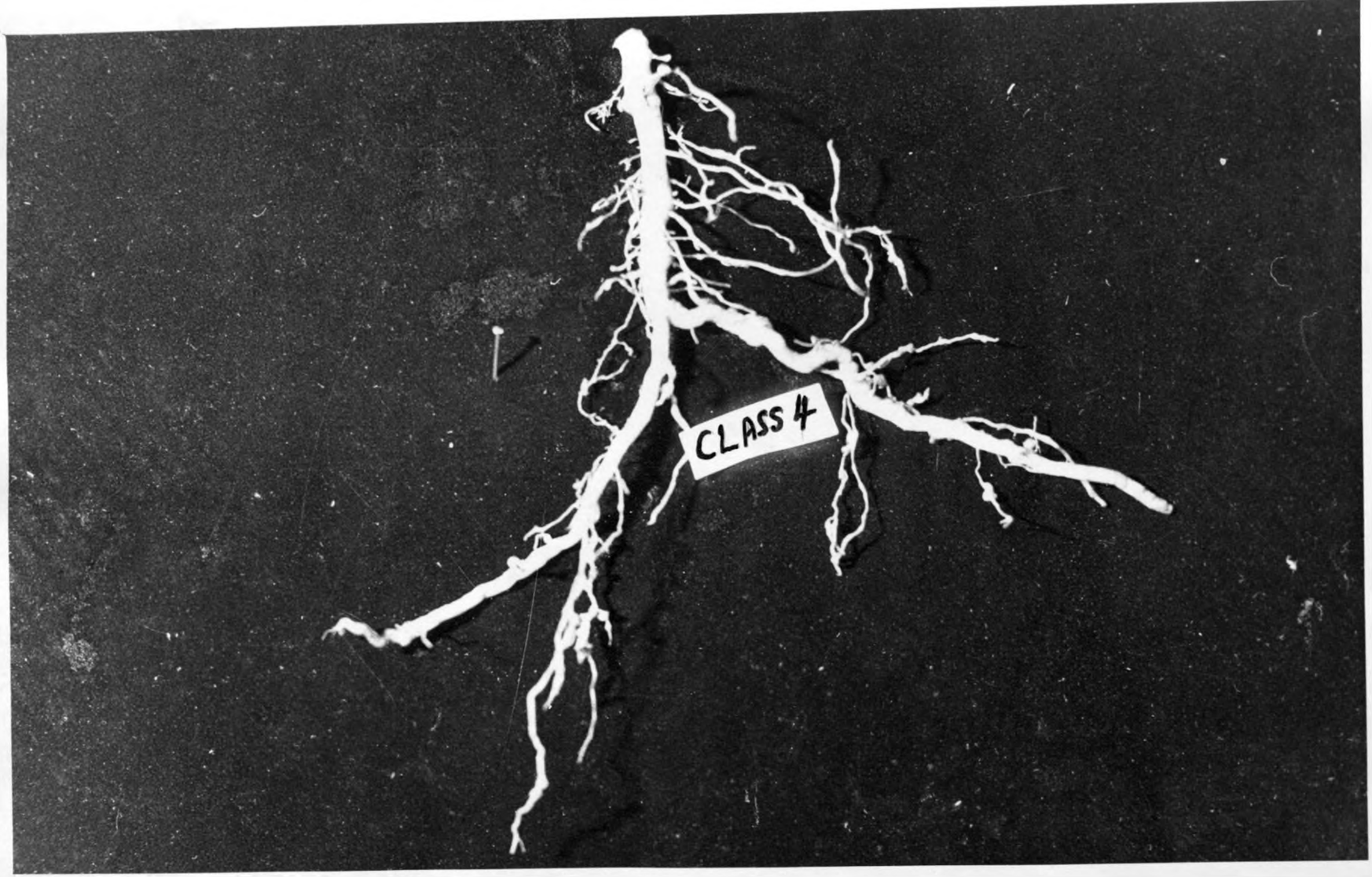
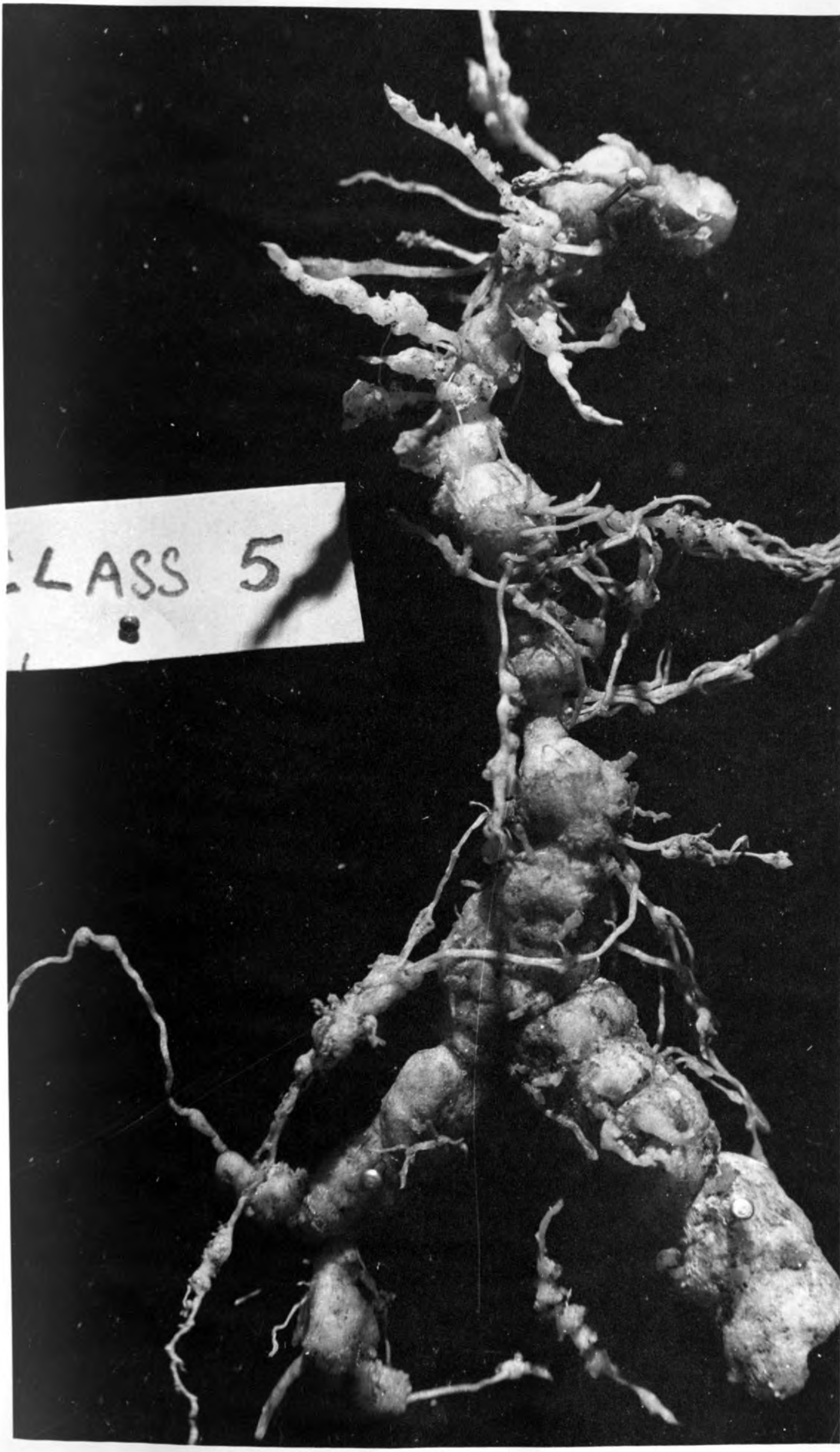

CLASS 2

Fig. 3.

Class 3: Moderate galling with no large galls but with a larger number of small galls than in class 2.


CLASS 3


CLASS 4


LASS 5

tomato reaching a height of 1.5-2.0m. The fruits are large and oblate. It is reported to be resistant to blight, root-knot nematodes, Fusarium, Verticillium and Tomato Mosaic Virus. T4 is also semi-determinate and reaches a height of 1.5-1.8m. the fruits are very large and oblate. It is reported to have resistance to blight, nematodes and Verticillium wilt.

(ii) The susceptible popular fresh market tomato Moneymaker code named MM. It has small to medium sized round fruits. It is high yielding and indeterminate in growth habit. It reaches a height of 1.7-2.0m. It is susceptible to all the maladies known in tomatoes including root-knot nematodes.

Methods.

One month old seedlings of all parents raised in separate boxes filled with sterilized soil obtained from the Plant Quarantine Station, Muguga were used for (a) Field Screening, and (b) Greenhouse Screening.

(a) Field Screening

Seedlings were transplanted in a field heavily infested with root-knot nematodes from the previous tomato crop. To make sure that the inoculum was high, heavily galled roots of the previous tomato crop were chopped up and thoroughly mixed with the soil before transplanting. Random plants were uprooted and infestation grade recorded as well as photographed at weekly intervals upto four weeks.

(b) Greenhouse Screening

To increase the nematode population susceptible tomato plants were first grown in the pots containing pure cultures of *Meloidogyne javanica* obtained from the Nematologist at Thika. When they matured the galled roots were chopped up and thoroughly mixed with pot soils. The infected soil was then filled into a number of plastic pots size 18 x 16½ cm. One month old seedlings of parents were then transplanted into the plastic pots.

For examination and grading random seedlings were uprooted at weekly intervals upto four weeks.

3.4. Yield Loss Assessment Trial

The trial was conducted to find out the yield reduction and effects on the tomato quality due to infestation of root-knot nematodes.

Materials

(i) Cultivars: T7 and Moneymaker (MM).

(ii) Treatments: (1) T7-F(F=fumigated plots)
(2) T7-I(I=plots inoculated with root-knot nematodes)
(3) MM-F, and
(4) MM-I.

Method

The fumigated plots were injected with 6 ml of DD soil fumigant per planting hole on 8th May 1978, Inoculated plots were prepared by mixing with 156 grammes of chopped galled roots of eggplant, Solanum melongena, to each planting hole.

One month old seedlings of MM and T7 raised in sterilized soil were transplanted in a randomized complete block design with

six replications in fumigated and inoculated plots on 6th June 1978. The plot size was 3.6m x 6m and the sample area was 1.8m x 3.6m (9 plants/plot). The spacing between rows and plants were 1.2m and 0.6m, respectively. Weekly sprays of copper fungicide 4kg/ha alternated with Dithane M.45 2kg/ha were used to control late blight and any other fungal diseases that might have risen. Endosulfan 35% M.L. at the rate of 4 litres/ha was used against the American bollworm. Tobacco Whitefly was controlled by spraying with dimethoate 32% M.L. at 600 ml/ha. Diammonium phosphate at the rate of 400kg/ha was applied in two dozes, the first at 100 kg/ha during transplanting and the remaining 300 kg/ha at flowering. Calcium was added to the soil in the form of agricultural lime at the rate of 200 kg/ha at transplanting. Natural rainfall supplemented with sprinkler irrigation was enough to maintain required moisture. The experiment was kept completely free of weeds. The plants were pruned to a single stem system. The first pruning was done forty days after transplanting followed by three fortnightly prunnings afterwards. Harvesting of ripe fruits was started on 11th September

1978. Fruits attacked by blossom-end-rot were considered as diseased and were recorded separately from the marketable fruits. The records were taken on (1) fruit numbers, (2) fruit weight, (3) fruit diameter, (4) fruit length, (5) sugar content (6) acidity and (7) vitamin C. Twenty random fruits per plot were used in the determination of fruit diameter, length, sugar, acidity and vitamin C. After the final harvest the nine plants from the sample area were uprooted and scored for resistance using the classification of Gilbert and McGuire (1952).

3.5. Inheritance of Resistance to Root-knot nematodes

Materials

The materials of this trial consisted of the following progenies:

Parents: MM, T7, T4.

F_1'S: MMxT7, T7xT4, T4xMM.

F_2'S: MMxT7, T7xT4, T4xMM.

B_1'S: F_1(MMxT7)xMM; F_1(T7xT4)xT7; F_1(T4xMM)xT4.

B_2'S: F_1(MMxT7)xT7; F_1(T7xT4)xT4; F_1(T4xMM)xMM.

One month old seedlings of MM, T7 and T4 raised in sterilized soil were transplanted in buckets placed in a greenhouse on 30th June 1977. At flowering crosses were made between MM x T7, T7 x T4 and T4 x MM and labeled. The seeds from the ripe crossed fruits were removed by fermenting the pulp for about four days and washed through a sieve. The seeds were dried on filter paper.

The seeds of F_1 crosses were divided into equal halves. One half was planted in plastic cups on 21st. November 1977 while the other half was left as reserve seed. To produce backcrosses parents were planted at the same time. One month old seedlings were transplanted in buckets keeping one seedling per bucket. Each F_1 was backcrossed to produce B_1(F_1 crossed to first parent) and B_2(F_1 crossed to second parent). The F_1'S were used as females in all backcrosses.

Method

One month old seedlings of 15 progenies (3 parents, 3 F_1's ,3F_2's,3B_1's and 3B_2's) raised in sterilized soil were transplanted in a randomized block design with four

replicates in a field heavily infested with namatodes. Chopped galled roots of brinjal and okra (250 gm/hill) were mixed at each planting hole. Usual agronomic practices were applied. There were a total of about 80 plants in each of segregating populations of F_2 and backcrosses except B_1 of F_1 (MM x T7) x MM, which had about 20 plants. The parents and F_1's had about 40 plants in the experiment.

Inheritance of resistance was determined by resistance scores of F_1, F_2, B_1 and B_2 progenies. The reaction of F_1 progenies indicated whether the resistance was determined by a recessive or dominant gene(s). Segregation ratios in F_2, B_1 and B_2 were confirmed by X^2 - test of goodness of fit.

3.6. Histology and Development of Nematodes

Three weeks old seedlings of the parents raised in sterilized soil were transplanted in plastic cups (7.5cm x 9.5cm) filled with sterilized soil. Twenty egg masses of root-knot nematodes were poured around the roots of each seedling at the transplanting time. The soil temperatures were recorded. One seedling of each parent was uprooted after 12, 24, 36 and 48 days. The roots were

washed thoroughly and three most heavily galled roots from each seedling were selected and stained using the method described by Mcbeth *et al*. (1941). The number of nematodes in the roots and their development stages were recorded.

For the histological studies the roots were dehydrated by using the methods of Johansen (1940) and Jensen (1962). Microtome sections were cut and mounted on slides for detailed observations.

3.7. Inheritance of yield and component characters

Using means of six populations, namely $\bar{P}1$, $\bar{P}_2$, $\bar{F}_1$, $\bar{F}_2$, $\bar{B}_1$ $(P_1\bar{F}_1)$ and $\bar{B}_2$ $(P_2\bar{F}_1)$ estimates of the various gene effects were obtained using the following relationships (Gamble, 1962):

$$m = \bar{F}_2$$

$$a = \bar{P}_1\bar{F}_1 - \bar{P}_2\bar{F}_1$$

$$d = -\tfrac{1}{2}\bar{P}_1 - \tfrac{1}{2}\bar{P}_2 + \bar{F}_1 - 4\bar{F}_2 + 2\bar{P}_1\bar{F}_1 + 2\bar{P}_2\bar{F}_1$$

$$aa = -4\bar{F}_2 + 2\bar{P}_1\bar{F}_1 + 2\bar{P}_2\bar{F}_1$$

$$ad = -\tfrac{1}{2}\bar{P}_1 + \tfrac{1}{2}\bar{P}_2 + \bar{P}_1\bar{F}_1 - \bar{P}_2\bar{F}_1$$

$$dd = \bar{P}_1 + \bar{P}_2 + 2\bar{F}_1 + 4\bar{F}_2 - 4\bar{P}_1\bar{F}_1 - 4\bar{P}_2\bar{F}_1$$

Where the parameters m,a,d,aa,ad,dd refer to mean effects, additive, dominance, additive× additive, additive × dominance, and dominance × dominance gene effects, respectively.

Significance of gene effects were tested by their respective standard errors using 't' test. The variances of these estimates were obtained in the usual manner for example,

$$Vd = \tfrac{1}{4}V\bar{P}_1 + \tfrac{1}{4}V\bar{P}_2 + V\bar{F}_1 + 16V\bar{F}_2 + 4V\bar{P}_1\bar{F}_1 + 4V\bar{P}_2\bar{F}_1$$

where V = Variance.

CHAPTER 4

RESULTS

4.1. Identification of Nematode Species

A total of sixty two perineal patterns were cut and examined. Out of these 98.4% were identified as *Meloidogyne javanica* while only 1.6% belonged to *M. incognita.* It was therefore concluded that *M. javanica* species is predominant in the Thika area.

4.2. Screening of Parents

Three tomato parental lines, Moneymaker, T7 and T4 were screened for resistance to *Meloidogyne javanica* species of root-knot nematode under greenhouse as well as field conditions. About 99% of the root-knot nematodes found in the fields at Thika belong to the species *M. javanica* (See section 4.1), hence field as well as greenhouse screening gave similar results. The relative swelling and galling in the roots during the first four weeks after transplanting in the inoculated pots and field was used as a criterion for classification of a parent to be susceptible or resistant on a 1-5 scale (see section 3.2.)

A score of resistance for parents is given in Table 1. The parent Moneymaker was found to be highly susceptible (score 4.75). Swelling in

Table 1. Mean galling scores of parents under greenhouse and field screening on 1-5 scale.

Week	MM		T7		T4	
	Greenhouse	Field	Greenhouse	Field	Greenhouse*	Field
First	4	4	1	1	4 + 1**	4
Second	5	5	1	1	4	4
Third	5	5	1	1	5	4
Fourth	5	5	2	2	5 + 2**	5
Mean	4.75	4.75	1.25	1.25	4.5 + 1.5	4.25

* The two class scores in the first and fourth week in the greenhouse refer to different plants.

** The Number of T4 plants in each class were:-
4 + 1 = $\frac{3}{5}$; 5 + 2 = $\frac{2}{6}$.

the root started in the beginning of the first week (Fig. 6a). By the fourth week there was profuse galling and swelling in the roots giving it a 'beaded' appearance (Fig. 6b). The second parent T7 was clear of swelling or galling in the root from first (Fig. 7a) to fourth week (Fig. 7b) indicating a high level of resistance. The mean galling score in case of T7 was 1.25. In case of T4 both resistant and susceptible plants were found. Susceptible plants showed some swelling in the first week (Fig. 8a) and profuse swelling in the fourth week (Fig. 8b). Such plants scored a mean grade of 4.38 (field and greenhouse). Those plants which were classified as resistant T4 were fairly free from galling from first to fourth week as shown in Figs 9a and 9b, respectively. These plants had a mean score of 1.25.

4.3. Yield Loss Assessment

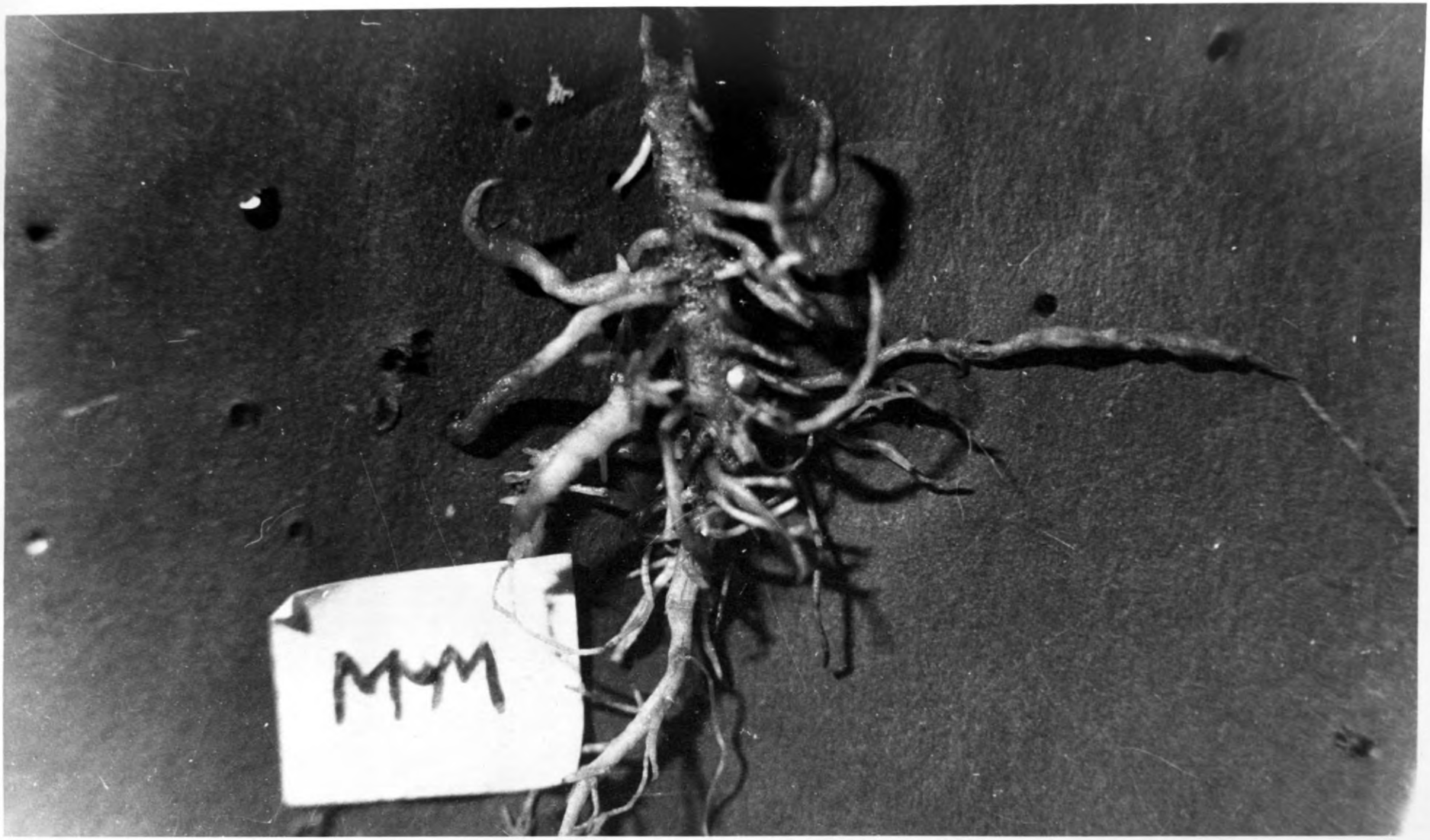
The yield loss was assessed in two ways, i.e.

(i) Fruit yield (kg/plot),

(ii) Fruit numbers/plot. The effects of root-knot nematodes on,

(iii) Fruit size and fruit quality characters were also examined.

Fig. 6a. Swollen roots of Moneymaker one week after infection.


M+M


MM

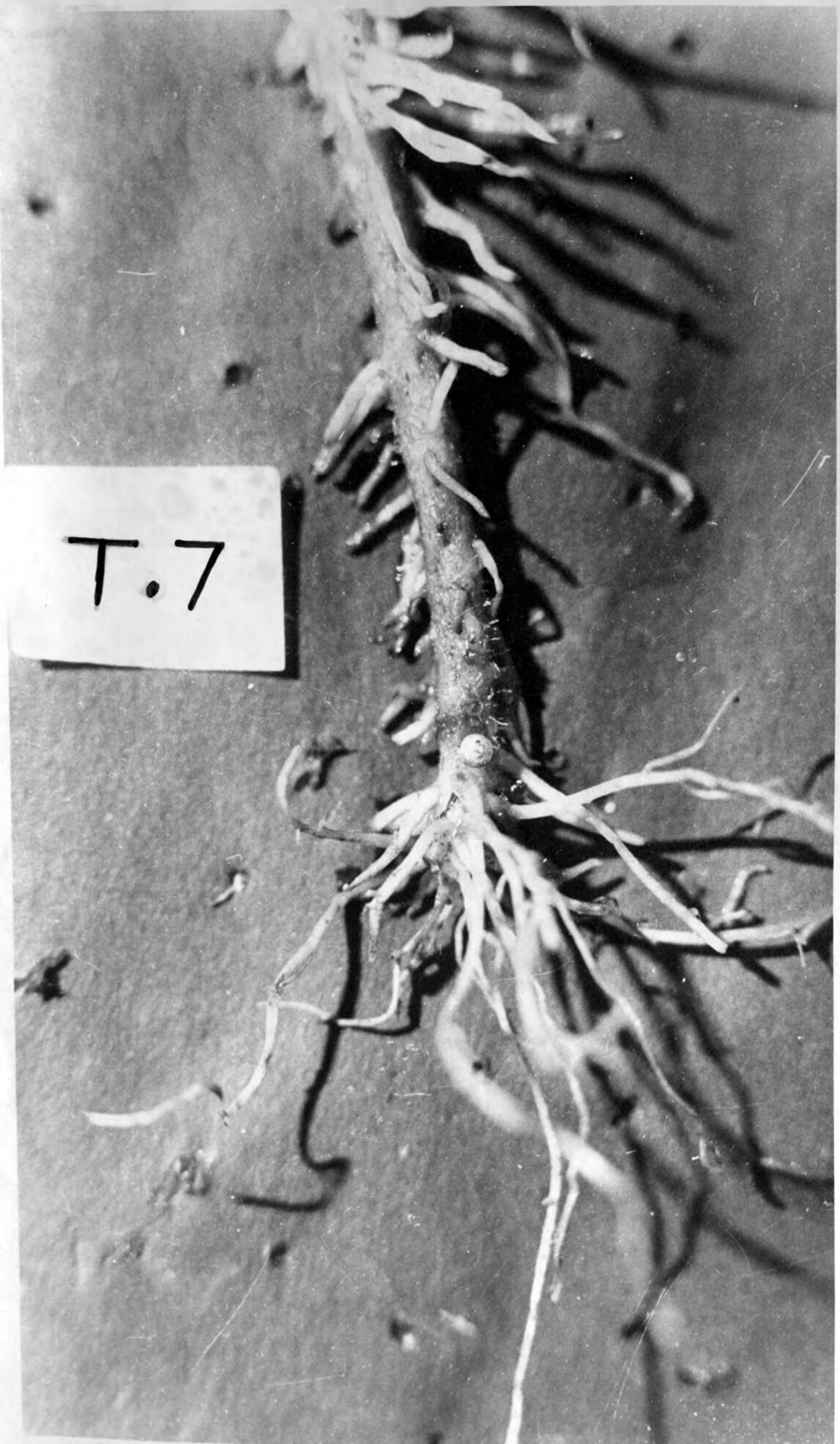
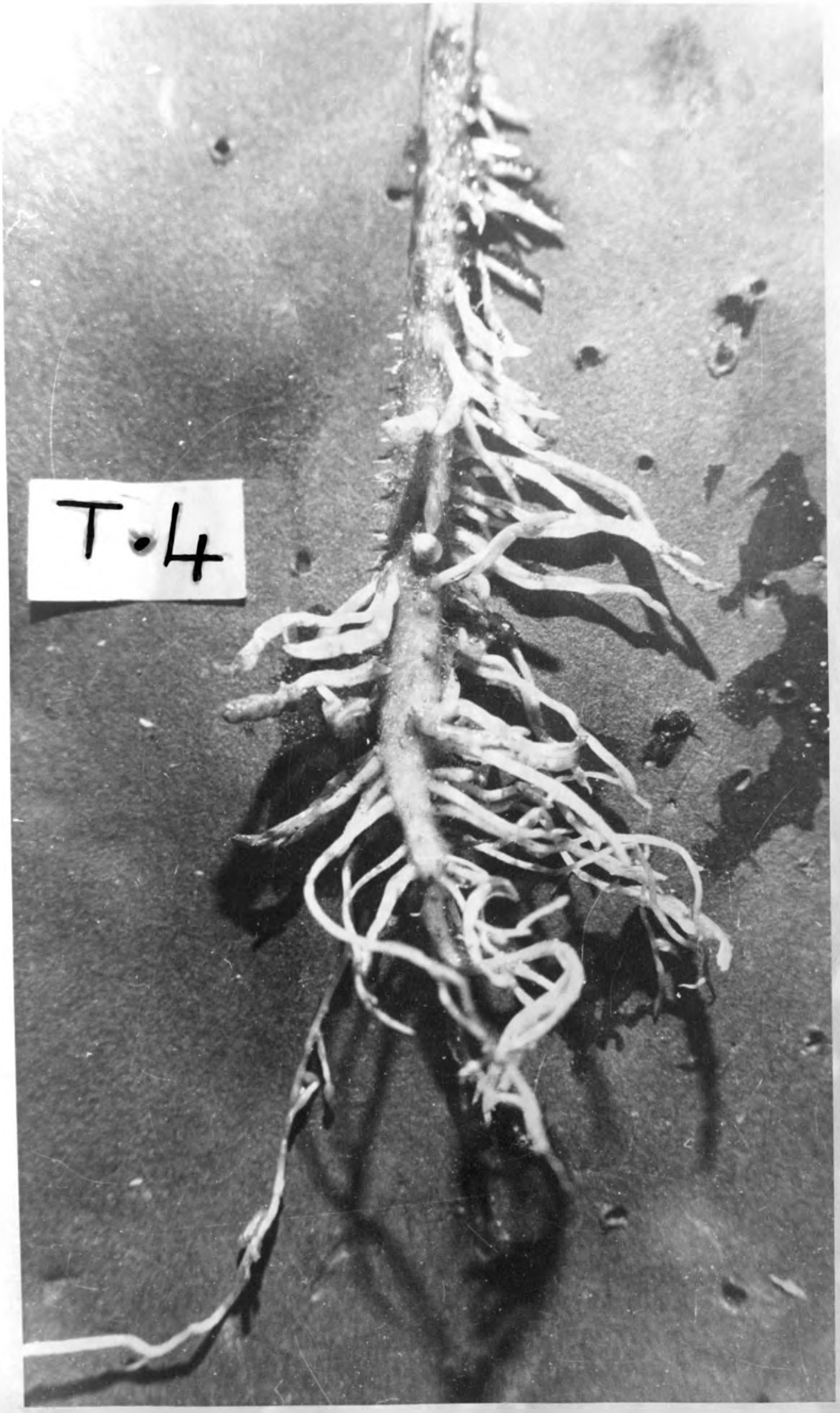

T.7

Fig. 7b. Clean roots of T7 four weeks after infection.

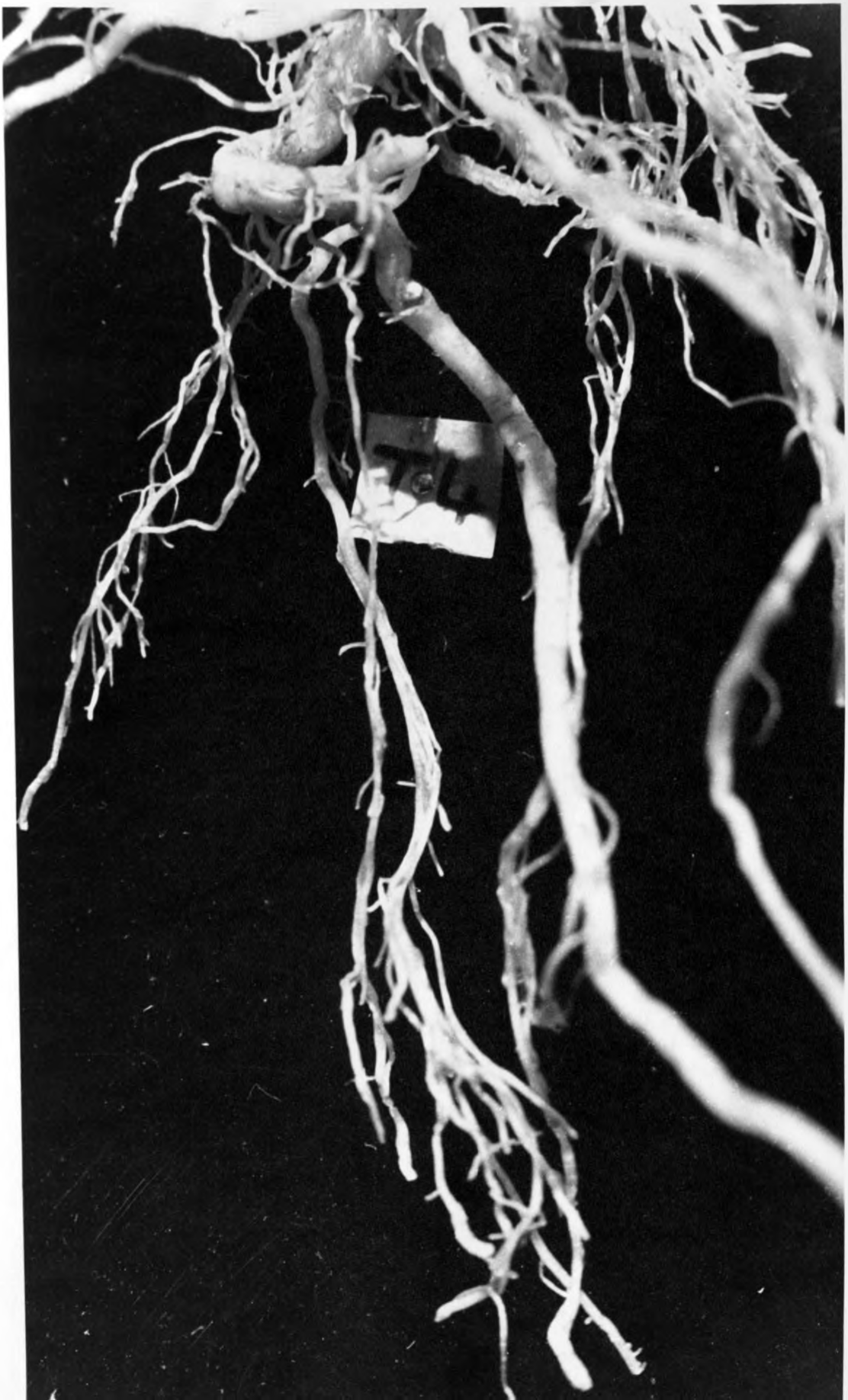
T07


T·4


4 TO 4


T·4

Fig. 9b. Clean roots of T4 four weeks after infection.



4.3.1. Fruit Yield (kg/plot)

The results of fruit yield loss are shown in Table 2. There was a signigicant reduction in total fruit yield in both Moneymaker and T7 due to root-knot nematode attack. The marketable fruit yield was however not affected in both Moneymaker and T7. The diseased fruit was significantly more on the fumigated plots of Moneymaker and T7 compared to their respective inoculated plots. Table 3 shows that the two parents T7 and MM did not differ significantly in their yielding ability.

4.3.2. Fruit Numbers

Reduction in fruit number was observed on total fruit, marketable fruit and diseased fruit. The root-knot nematodes significantly reduced the fruit numbers per plot in both Moneymaker and T7 (Table 4), except marketable fruit number in T7. The fumigated plots of both Moneymaker and T7 had more fruits attacked by blossom - end-rot as can be seen in the same table.

Among the two parents Moneymaker had significantly more total as well as marketable and diseased fruits (Table 5).

Table 2. Mean fruit yields of Moneymaker and T7 under fumigation (F) and and inoculation (I) in kg/plot (6.48 sq. m.)

Yield		MM-F	MM-I	Difference	Percentage Loss
Total Yield	:	21.785	14.845	6.940	31.9*
Marketable fruit	:	18.433	13.547	4.886	26.5
Diseased fruit	:	3.352	1.299	2.053	61.2**
		T7-F	T7-I	Difference	Percentage Loss
Total Yield	:	24.911	17.740	7.171	28.8*
Marketable fruit	:	21.399	15.539	5.860	27.4
Diseased fruit	:	3.512	2.201	1.311	37.3*

* Significant at 5% level (based on orthogonal contrasts)

** Significant at 1% level (based on orthogonal contrasts)

Table 3. Mean fruit yields of the two parents (kg/plot).

Yield		MM	T7	Difference
Total yield	:	18.315	21.326	3.011
Marketable fruit	:	15.990	18.469	2.479
Diseased fruit	:	2.326	2.857	0.531

Table 4. Mean fruit numbers/plot (6.48 sq. m.) of Moneymaker and T7 under fumigation (F) and inoculation (I).

Yield		MM-F	MM-I	Difference	Percentage Loss
Total fruit	:	508.00	311.25	196.75	38.7***
Marketable fruit	:	381.25	264.75	116.50	30.6**
Diseased fruit	:	126.75	46.50	80.25	63.3***
		T7-F	T7-I	Difference	Percentage Loss
Total fruit	:	317.75	229.50	88.25	27.8*
Marketable fruit	:	246.75	187.50	59.25	24.0
Diseased fruit	:	71.00	42.00	29.00	40.8

* Significant at 5% level (based on orthogonal contrasts).

** Significant at 1% level (based on orthogonal contrasts).

*** Significant at 0.1% level (based on orthogonal contrasts).

Table 5. Mean fruit numbers in MM and T7/plot

Yield		MM	T7	Difference
Total fruit	:	409.63	273.63	136.00***
Marketable fruit	:	323.00	217.13	105.87***
Diseased fruit	:	86.63	56.50	30.13**

** Significant at 1% level (based on orthogonal contrasts).

*** Significant at 0.1% level (based on orthogonal contrasts).

4.3.3. Fruit Size

The root-knot nematodes did not have any significant effect on the fruit length, diameter and locule number of Moneymaker and T7 as can be seen in Table 6.

4.3.4. Fruit Quality

The results show that the root-knot nematodes did not significantly affect the sugar and vitamin C content of both Moneymaker and T7 (Table 7). The nematodes had no effect on the titratable acidity of T7 fruits but they significantly increased the acidity of Moneymaker fruits as can be seen in the same table.

4.3.5. Effect of fumigation on gall formation in roots

In order to find out the effect of fumigation on resistant and susceptible cultivars roots of T7 and Moneymaker were examined in both fumigated and inoculated plots after the final harvest of the yield assessment trial. The resistance scores (1-5) are presented in Table 8. The results showed that fumigation with D-D soil fumigant significantly reduced the infestation of root-knot nematodes in the susceptible cultivar Moneymaker (mean score 1.4) as compared to

Table 6. Mean fruit length and diameter (cm) and locule number of Moneymaker and T7 under fumigation (F) and inoculation (I).

	MM-F	MM-I	Difference	Percentage Difference
Fruit Length :	4.1250	4.0817	0.0433	1.1
Fruit diameter :	5.5300	5.5250	0.0050	0.1
Locule Number :	2.3650	2.3917	0.0267	1.1

	T7-F	T7-I	Difference	Percentage Difference
Fruit length :	4.3100	4.2817	0.0283	0.7
Fruit diameter :	6.3617	6.2883	0.0734	1.2
Locule Number :	6.3767	6.0483	0.3284	5.2

Table 7. Mean Sugar, Vitamin C and titratable acid content of Moneymaker and T7 fruits under fumigation (F) and inoculation (I).

		MM-I	MM-F	Difference	Percentage increase over MM-F
Sugar (gm/100ml)	:	3.1167	2.7833	0.3334	12
Vitamin C (mg/100ml):	:	20.0167	19.0000	1.0167	5.4
Titratable acid (% as citric)	:	0.6783	0.5133	0.1650	32.1***
		T7-I	T7-F	Difference	Percentage increase over T7-F
Sugar (gm/100ml)	:	2.4833	2.3000	0.1833	80
Vitamin C (mg/100ml)	:	15.7500	13.5167	2.2333	16.5
Titratable acid (% as citric)	:	0.5283	0.5050	0.0233	4.6

*** Significant at 0.1% level (based on orthogonal contrasts).

Table 8. Mean nematode galling scores (1-5) in the yield loss assessment trial

Treatment	Replication						Mean Score
	I	II	III	IV	V	VI	
MM-I	4.7	4.8	5.0	5.0	5.0	4.9	4.9
MM-F	1.4	1.1	1.1	1.2	1.3	2.1	1.4
T7-I	1.0	1.0	1.0	1.0	1.3	1.1	1.1
T7-F	1.0	1.0	1.0	1.0	1.0	1.0	1.0

MM-I Moneymaker inoculated
MM-F Moneymaker fumigated
T7-I T7 inoculated
T7-F T7 fumigated

inoculated roots (4.9). Mean galling score in case of resistant cultivar T7, on the other hand, was very low (1.0) in both fumigated and inoculated plants. This suggests that fumigation in case of resistant cultivars appears not to be of any economic value.

4.4. Inheritance of Resistance to root-knot nematode *Meloidogyne javanica*

It was indicated in section 4.2 that T7 was found to be resistant and Moneymaker was susceptible. T4, on the other hand, was found to be segregating. It was therefore decided to study the inheritance in three crosses, Moneymaker x T7, T7 x resistant T4, and Moneymaker x reistant T4.

(i) Cross MM x T7

The data of segregation with respect to resistance in the cross MM x T7 together with its F_2 and backcrosses are given in Table 9a. F_1 was susceptible indicating the susceptibility of MM was dominant over resistance of T7. In the F_2 generation a ratio of 3 susceptible: 1 resistant was obtained, hence confirms that the resistance in T7 is controlled by a

Table 9a. Reaction of parents and F'_1S, and segregation ratios in F_2 and back-cross progenies for resistance to root-knot nematode, Meloidogyne javanica in a tomato cross, MM x T7.

Parent		F_1	F_2 Segregation	X^2	Backcross segregation		X^2
MM	T7				B_1 (MMXT7) x MM	B_2(MMxT7)xT7)	
S	R	S	Obs.20R: 56S Exp. (1:3)19:57	0.017* (P=0.900)	S	Obs.39R:34S Exp.(1:1):36.5:36:5	0.220* (P=0.500-0.750)

R = Resistant S = Susceptible

*Ratio shows a good fit (after Yate's, 1934 correction for ldf).

recessive gene. This was further confirmed by the 1 resistant: 1 susceptible ratio in the back-cross generation, F_1 (MM x T7) x T7. It was therefore concluded that the reaction of resistance to Meloidogyne javanica as found in parent T7 is controlled by a single pair of recessive gene designated as LMjr_1 (i.e. Lycopersicon esculentum host, Meloidogyne javanica parasite, recessive gene for resistance number one).

(ii) Crosses T7 x T4 and T4 x MM

The F_1's of the crosses, T7 x T4 and T4 x MM showed unexpected segregation into resistant and susceptible plants suggesting that the T4 resistant plants used in crosses were heterozygous and that their resistance was dominant over susceptibility. These F_1's therefore could be treated as back-crosses i.e., B'_1(= F_1(T4) x susceptible MM) and B'_2 = (F_1(T4 x resistant T7) and

hence an expected ratio of 1 resistant: 1 susceptible in both cases. Such results were obtained (Table 9b). The good fit to a ratio of 1 resistant: 1 susceptible in the F_1 of the cross T7 x T4 confirmed that the resistance in T4 was monogenic and dominant and this gene was designated $\underline{LMjR_2}$ (i.e. Lycopersicon esculentum host, Meloidogyne javanica parasite, dominant gene for resistance number two). The resistant T4 plants therefore were heterozygous, $LMjR_2/LMjr_2$. Similarly F_1 of the cross T4 x MM was expected to fit a 1:1 ratio but this was not achieved although this may be due to the relatively few plants that were sampled.

From the results it was apparent that there are two types of resistant genes, one recessive pair of genes as found in T7 and another dominant pair of genes as found in T4. These two gene pairs could be located in the same chromosome or in different

Table 9b. Reaction of parents and F_1'S of the crosses T7 x T4 and T4 x MM.

Parent		$F_1(=B'_1)$	X^2	Parent		$F_1(=B'_2)$	X^2
T4	MM			T7	T4		
Segregating	S	Obs.5R:34S	20.102	R	Segregating	Obs. 16R: 24S	1.226*
		Exp (1:1)19.5R:19.5S	(P=0.005)			Exp(1:1):20R:20S	(P=0.25-0.50)

R = Resistant; S = Susceptible

* Ratio shows a good fit.

B'_1 = F_1 crossed to susceptible parent MM, B'_2 = F_1 crossed to resistant parent, T7

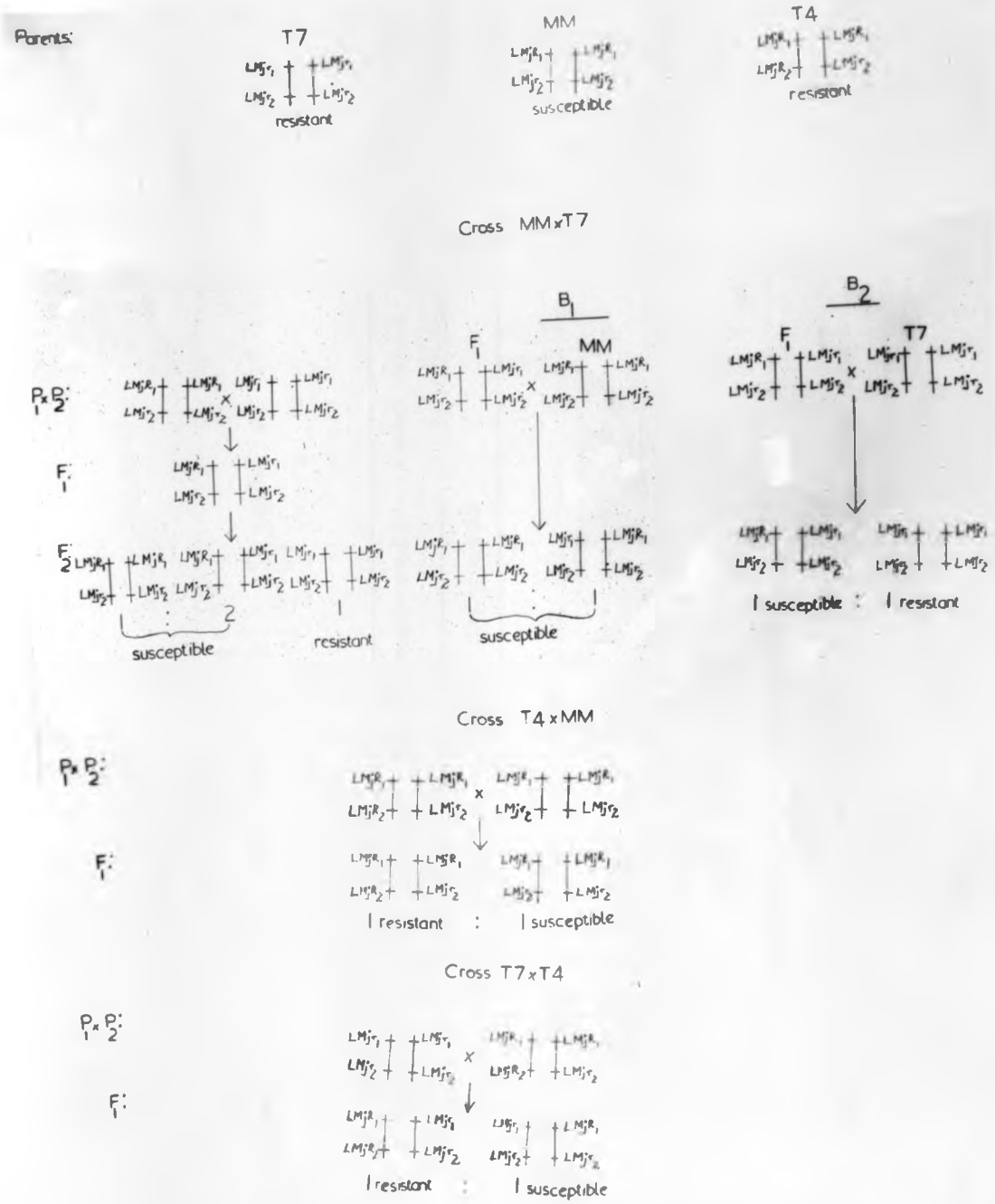


Fig. 10. Genotypes of parents and their F_1, F_2, B_1 and B_2 progenies

Table 10. Mean measurements of the various cell tissues (μ) of resistant and susceptible tomato cultivars

	Diameter			Epidermal cell		Mean cell layers (Endodermis)	Endodermal cell (Diameter)	Epidermal Surface
	Epidermis	Endodermis	Stele	Length	Diameter			
MM-D3	30	433	1130	111	30	10	96	Rough
MM-D4	40	240	565	96	40	6	80	Rough
T7-D3	15	330	265	52	15	8	60	Smooth
T4-D4	58	420	405	93	58	6	116	Rough

D3 = 36 days after inoculation, D4 = 48 days after inoculation

Fig. 11. Transverse section of Moneymaker root showing rough epidermal surface and damaged parenchyma cells in the stele 36 days after inoculation with *Meloidogyne javanica*.

(x 3175.99)

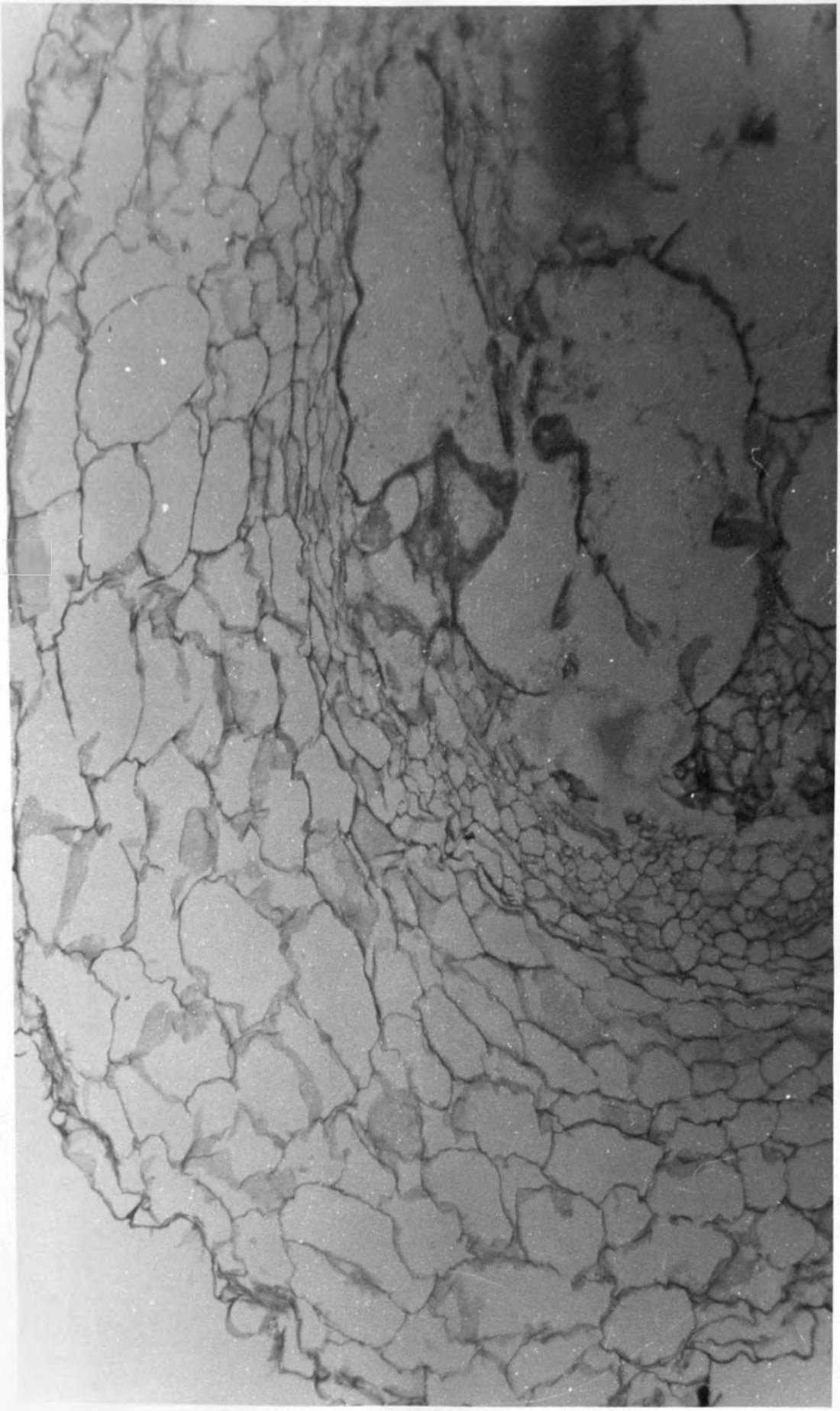
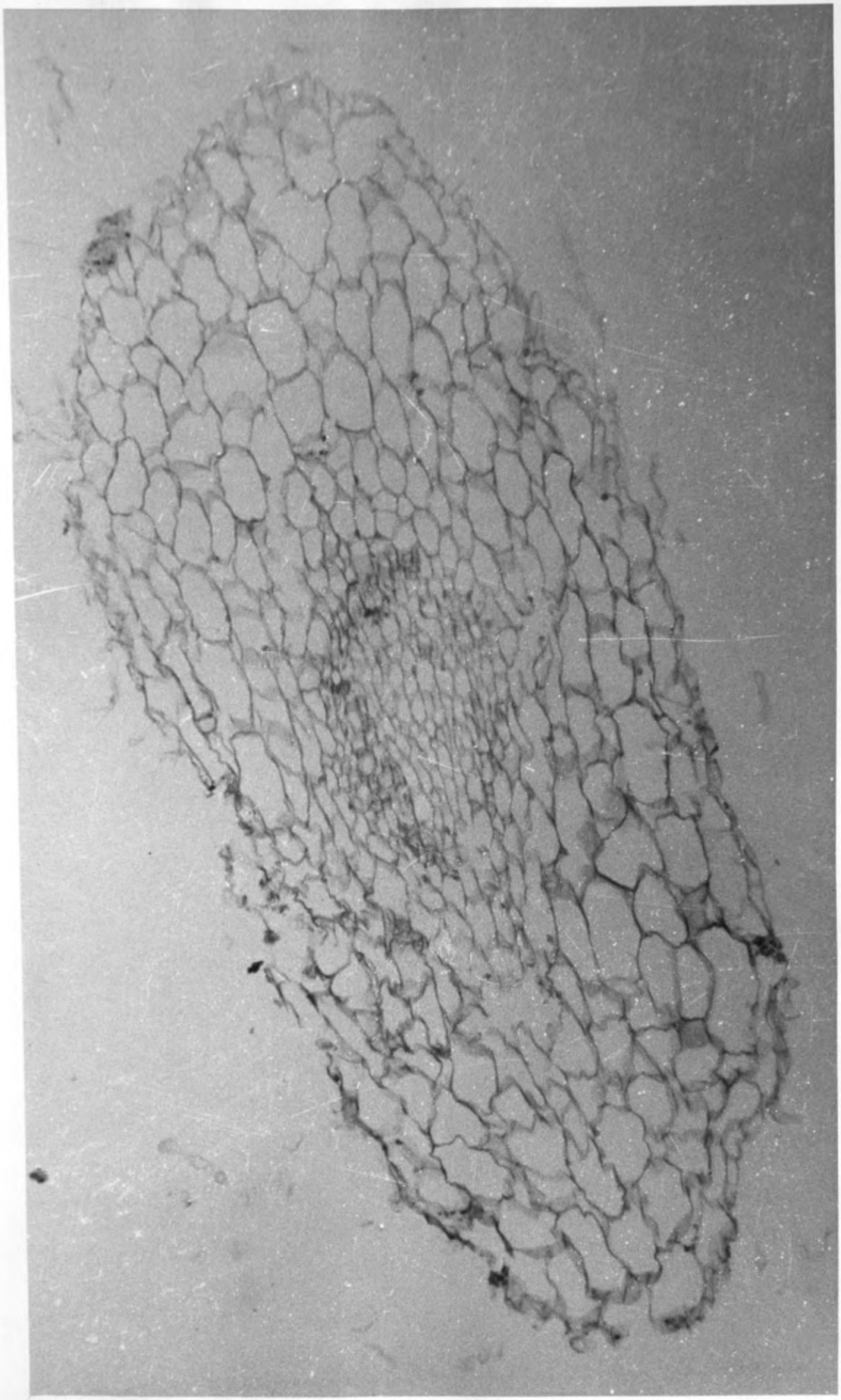
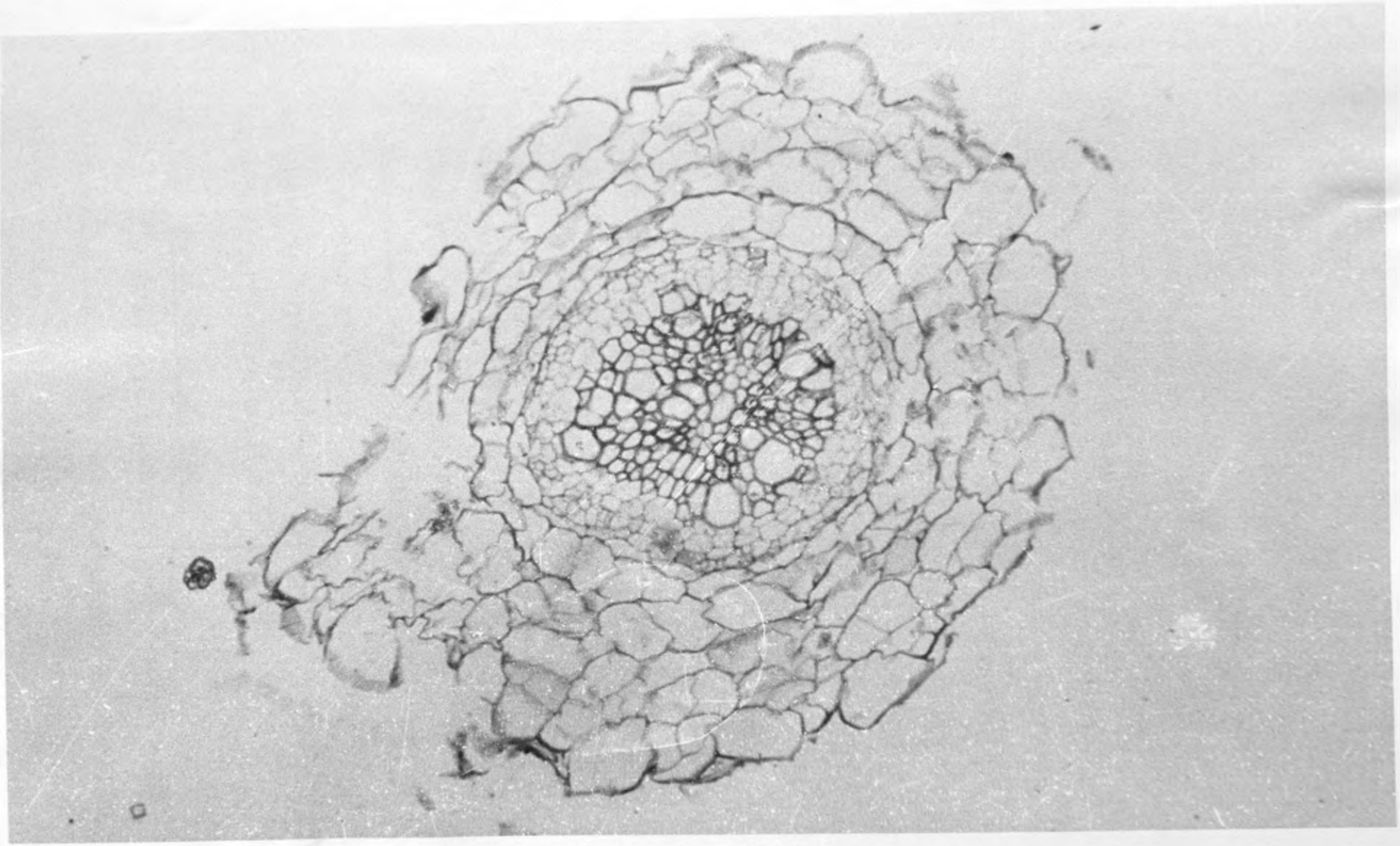


Fig. 12. Transverse section of T7 root showing smooth epidermal surface and no internal damage 36 days after M. javanica inoculation.
(x 3175.99)





4.5.2. Histopathology

Root sections of all the three tomato cultivars observed 12, 24, 36 and 48 days after inoculation with egg masses of root-knot nematodes showed that the larvae penetrate near the root-tip (Fig 14) then they migrate to the stele where they feed and develop to maturity. Gall formation is induced by enlargement of cortical cells followed by hyperplasia. Larvae within the stele feed on vascular parenchyma cells, which initially enlarge and then divide to form groups of multinucleated giant cells (Fig. 15). Giant cells are necessary for development of M. javanica females. The nematodes developed to maturity in Moneymaker (Fig.16), T4 (Fig.17) and T7 (Fig. 18) but in T7 the shape of the nematode was deformed. It was not unusual to find more than one mature nematode in the stele of Moneymaker and susceptible T4 plants (Fig. 19, 20). A clear damage inside the stele of Moneymaker is shown in Fig. 21 where a lot of open spaces have been left after the breakdown of giant cells.

4.5.3. Nematode Development

The mean soil temperature (Table 11) were favourable for development of Meloidogyne javanica larvae during plant growth in pots. The nematode counts in the selected three heavily galled roots

Fig. 14. Longitudinal section of Moneymaker root tip showing 2nd stage larva of M. javanica 12 days after infection. (x 5041.25)

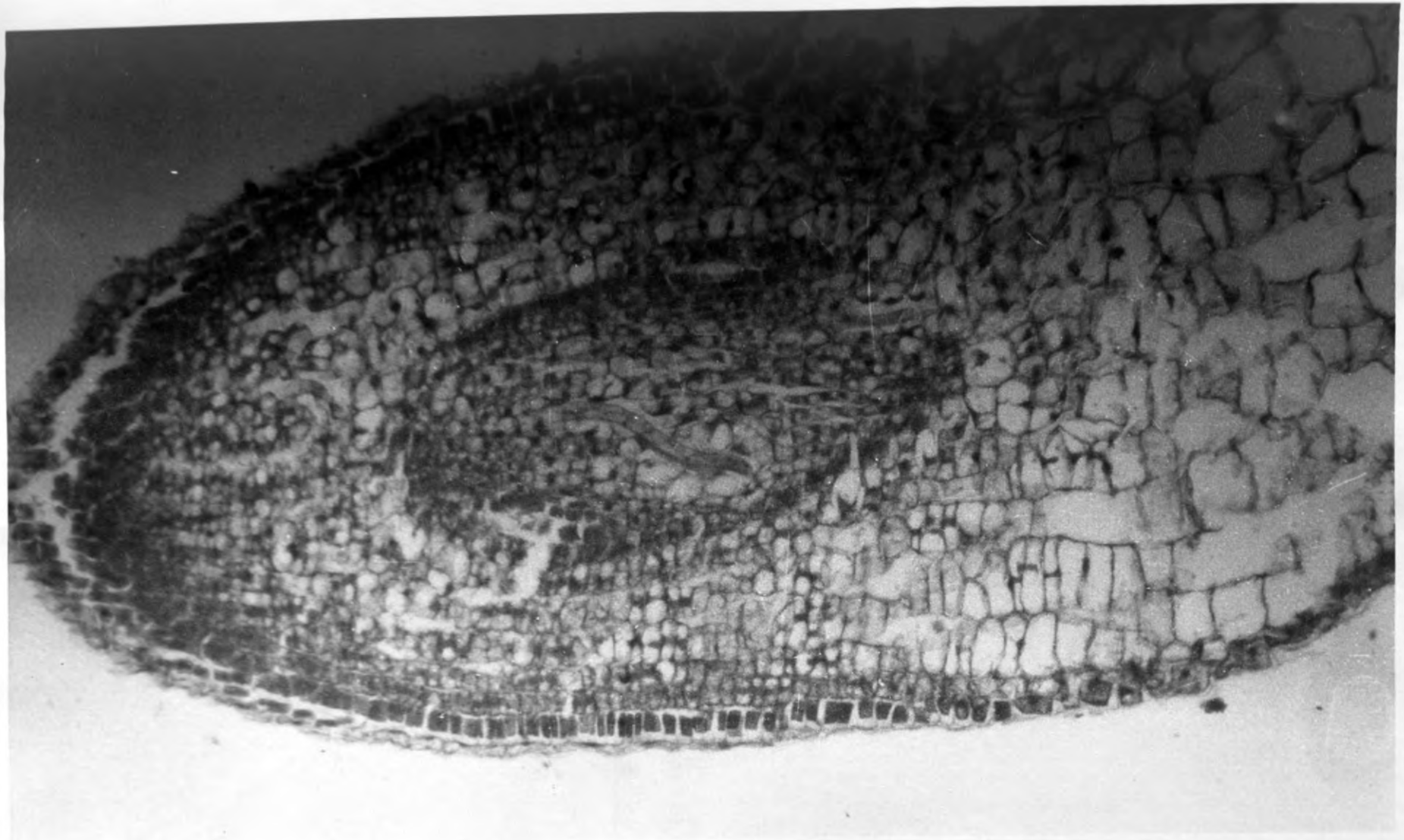


Fig. 15. Longitudinal section of Moneymaker root showing 2nd stage M. javanica larva and giant cells 24 days after infection.

(x 5041.25)

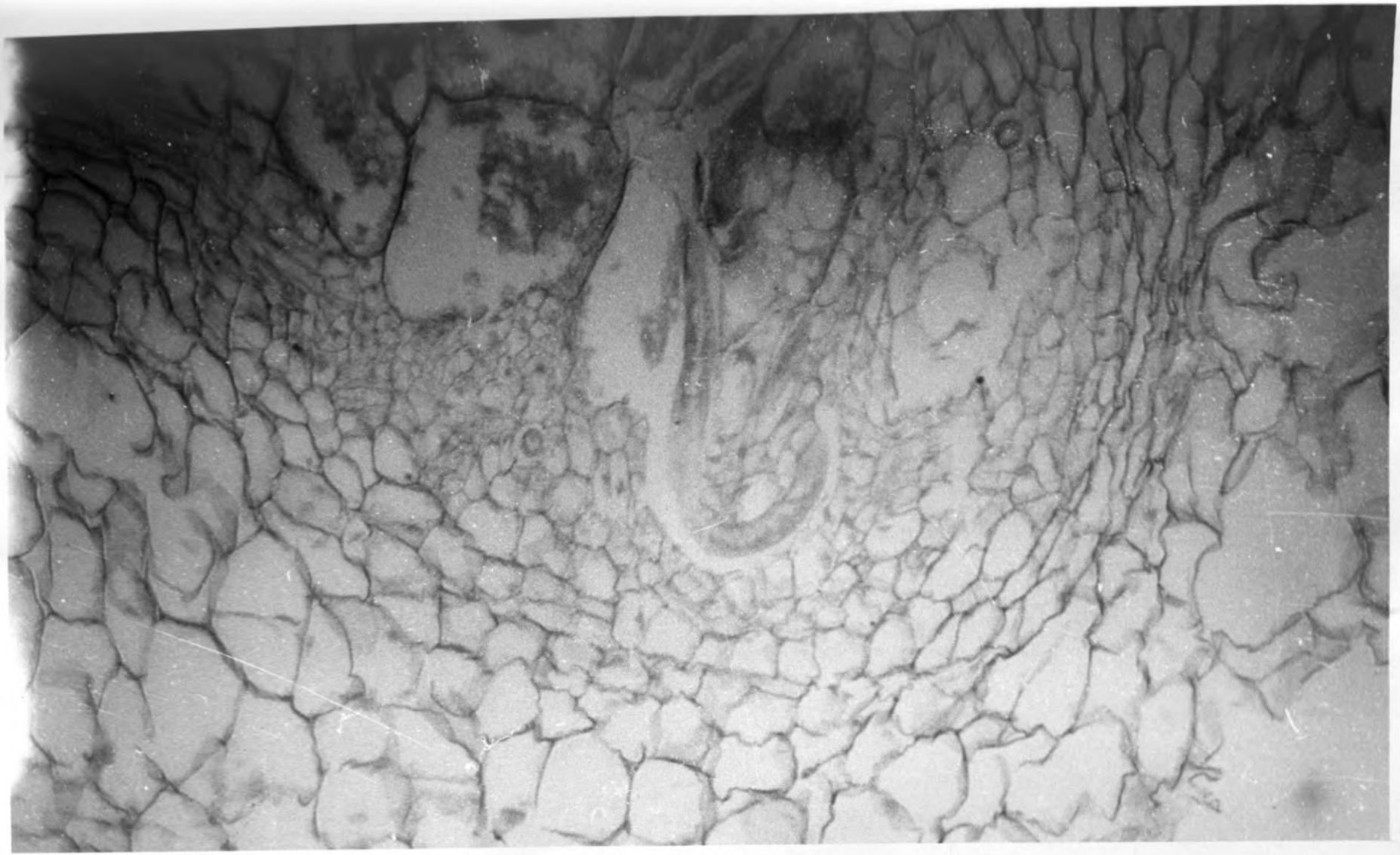
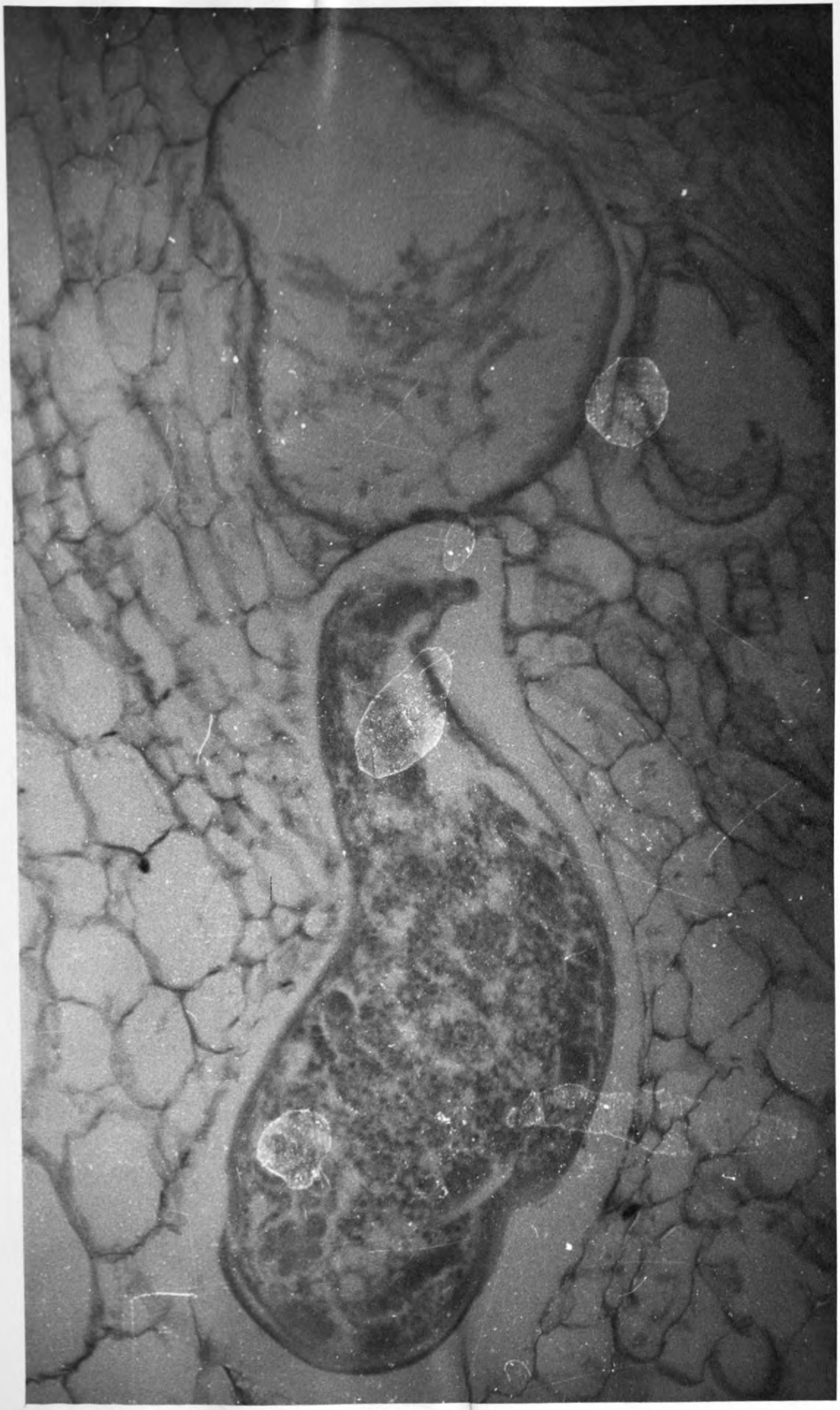


Fig. 16. Longitudinal section of Moneymaker root showing a mature M. javanica female and giant cells 48 days after infection.

(x 4065.26)



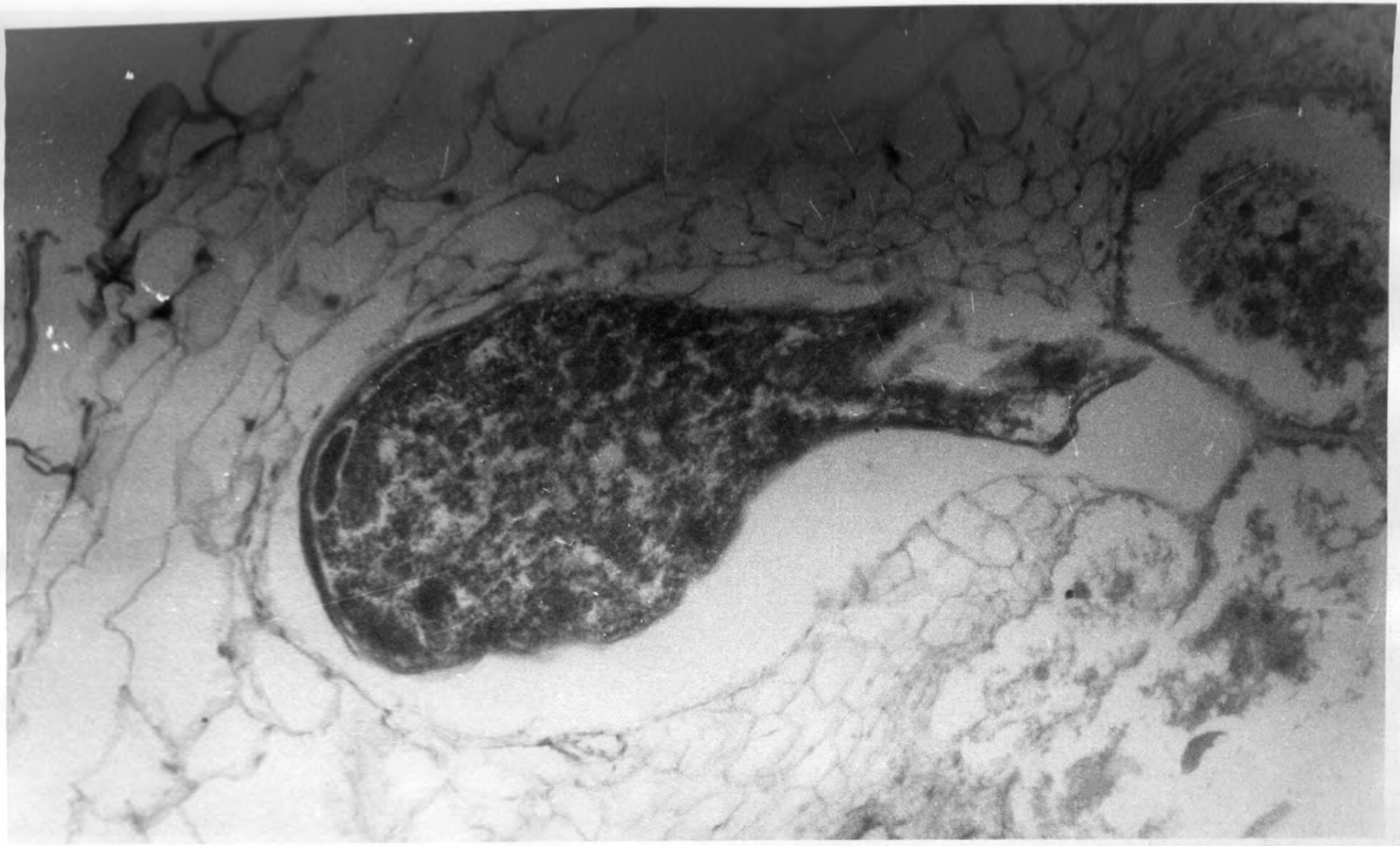


Fig. 18. Longitudinal section of T7 root showing a deformed mature M. javanica female 48 days after infection. Giant cells have broken down.

(x 5041.25)

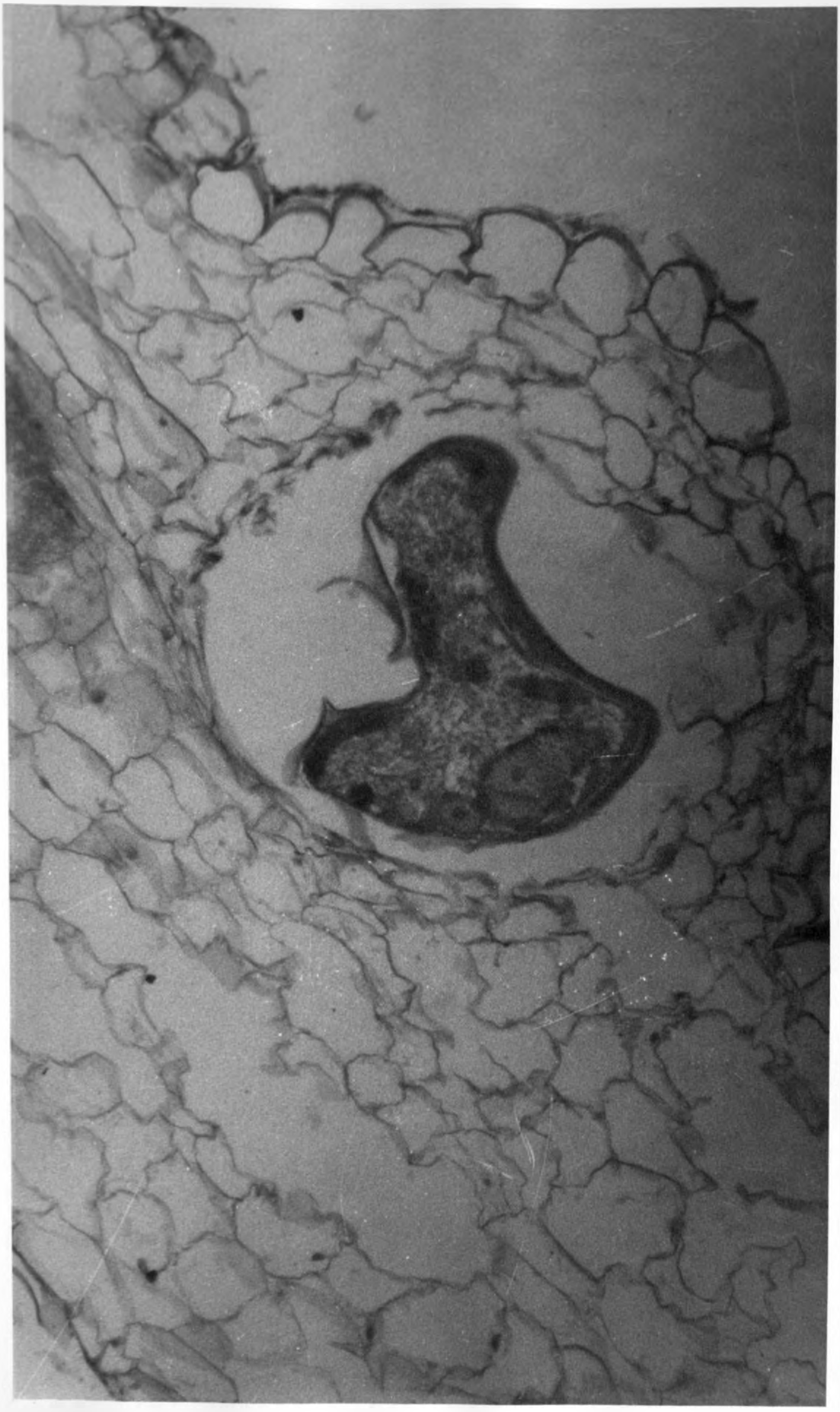
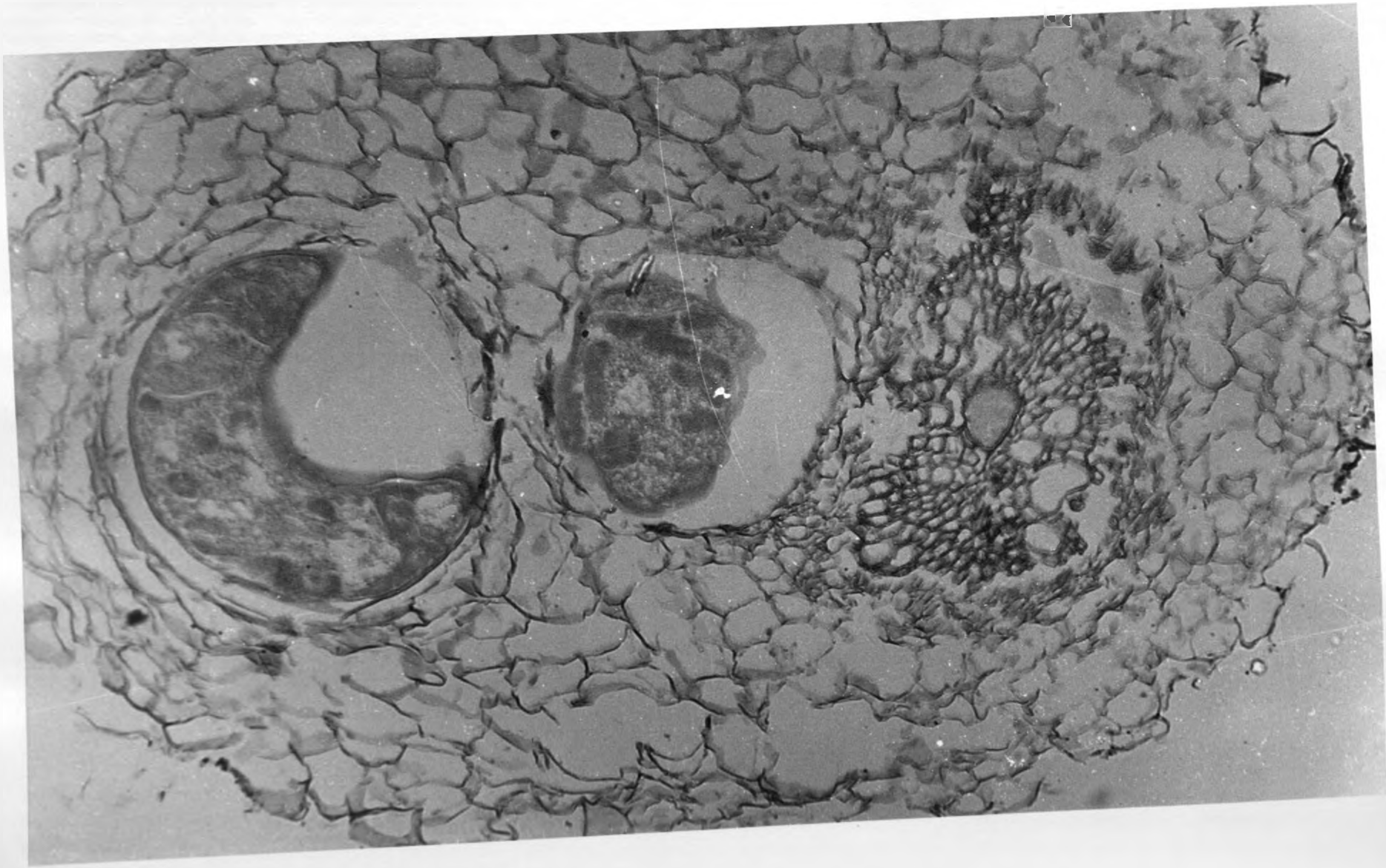


Fig. 19. Transverse section of Moneymaker root showing infection by two rook-knot nematodes (M. javanica) 48 days after infection.

(x 3175.99)



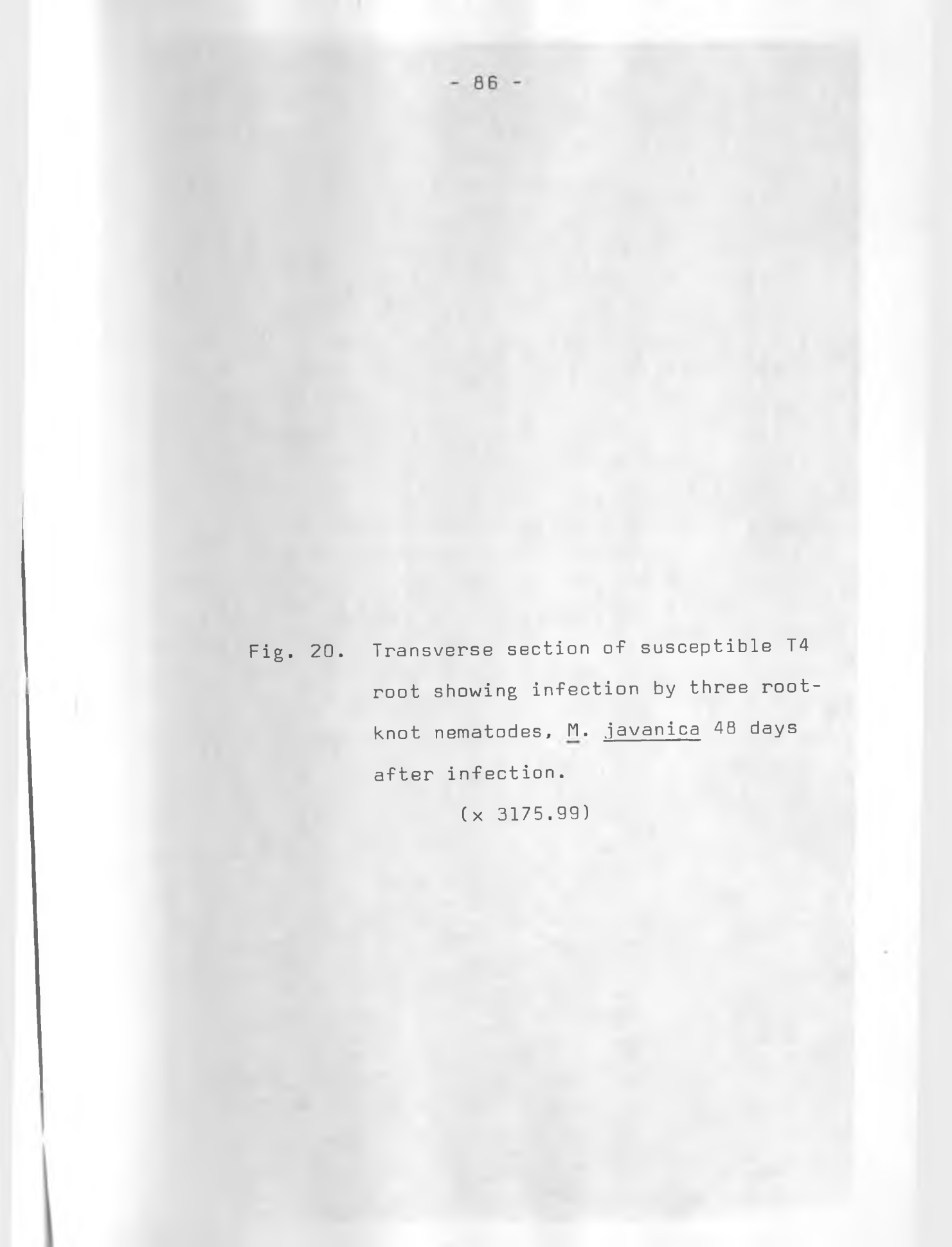


Fig. 20. Transverse section of susceptible T4 root showing infection by three root-knot nematodes, M. javanica 48 days after infection.

(x 3175.99)

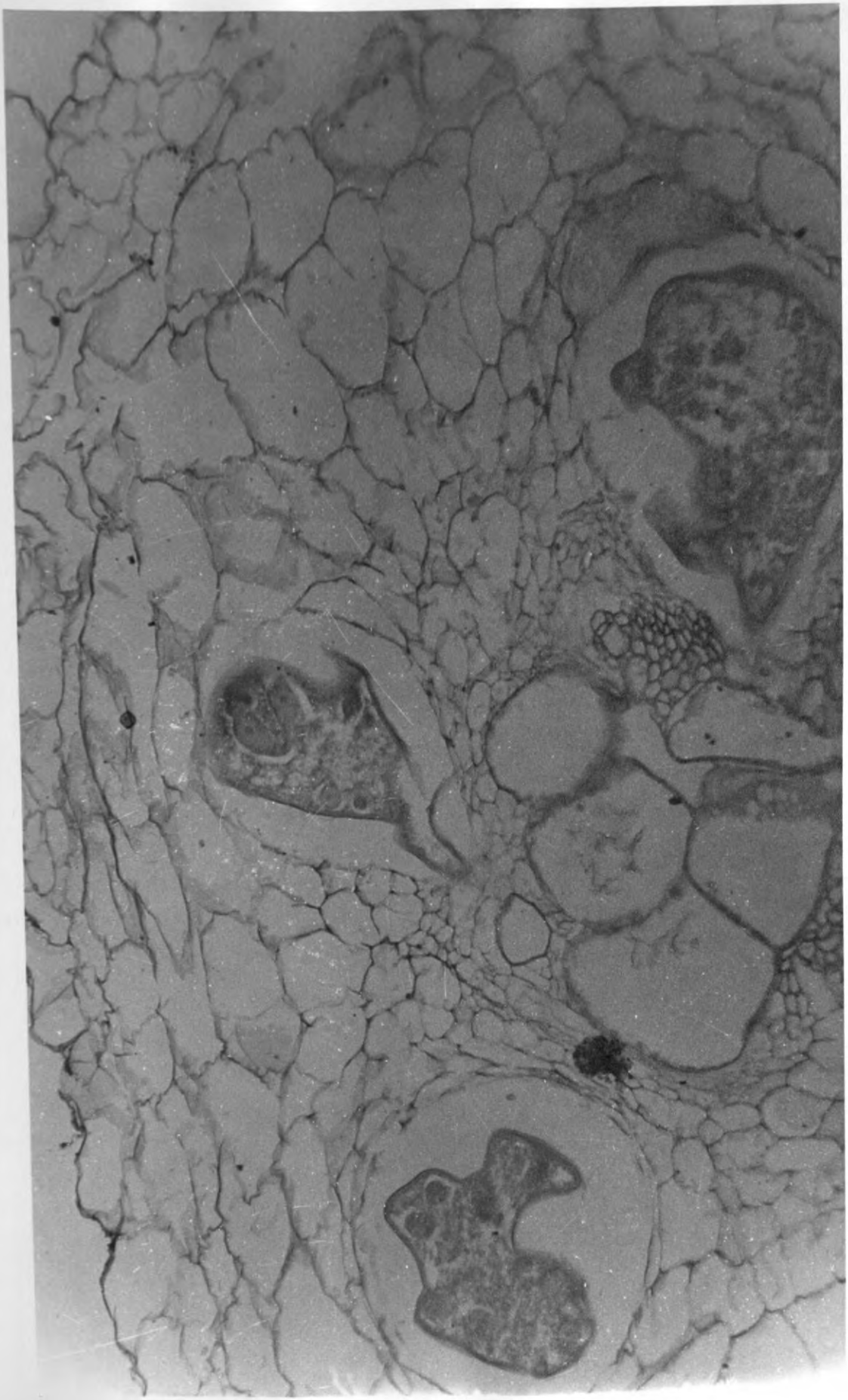


Fig. 21. Transverse section of Moneymaker root showing internal damage in the stele 48 days after infection.

(x 1613.20)

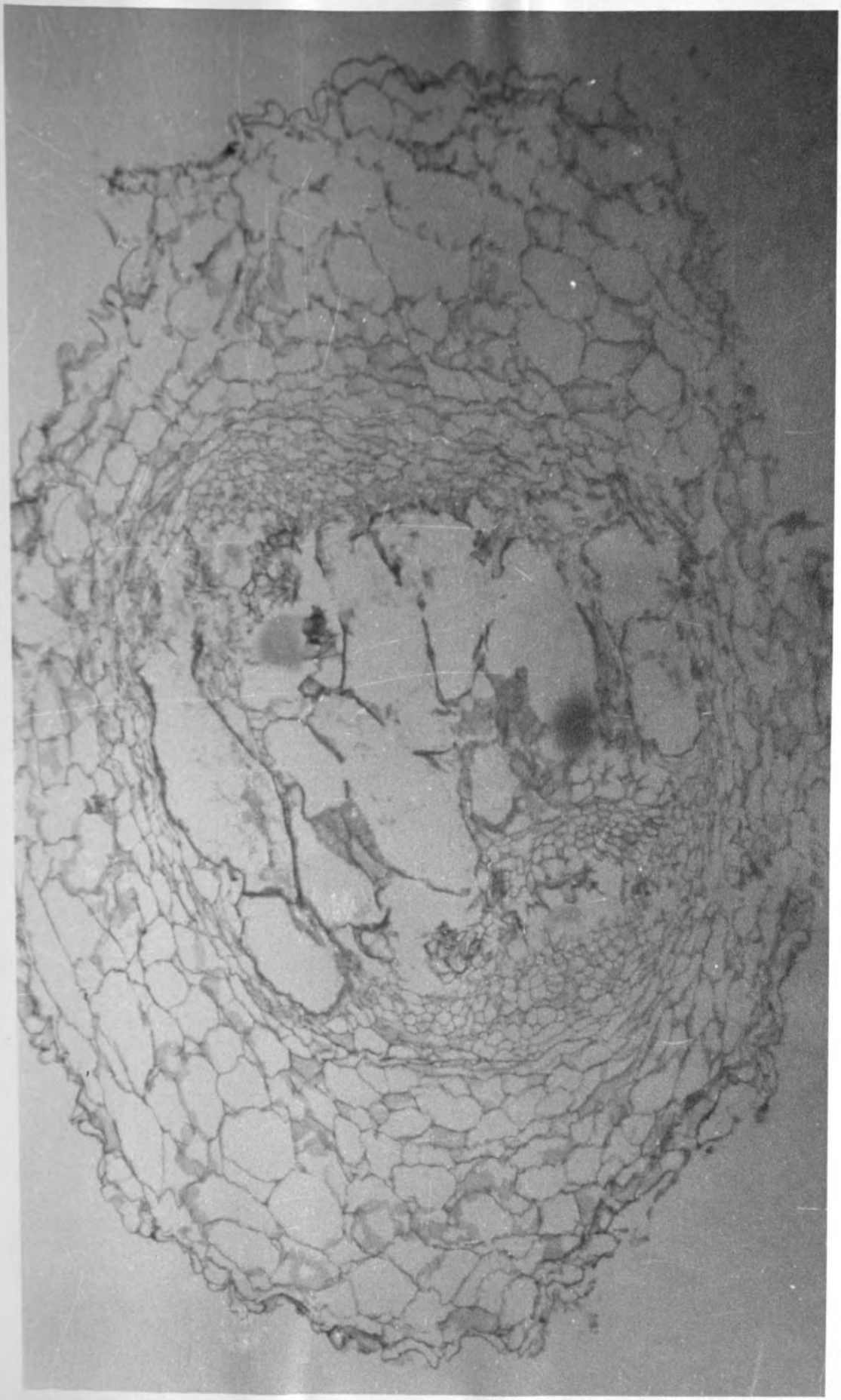


Table 11. Mean Weekly Soil Temperatures ^{o}C.

Week	Time of day		Mean
	Morning (10 a.m.)	Evening (4.30 p.m.)	
First	20.4	23.6	22.0
Second	20.2	32.9	26.6
Third	18.2	31.8	25.0
Fourth	20.3	32.4	26.4
Fifth	19.5	24.6	22.1
Sixth	21.2	27.3	24.3

of each parent are shown in Table 12. This table shows that very few nematodes entered T7 roots compared to Moneymaker and susceptible T4 (T4 plant 48 days from inoculation). At 12 days from inoculation 33 second stage larvae were found in the Moneymaker roots compared to 10 in T4 and 1 in T7.. At 24 days from inoculation 55 root-knot larvae had entered the roots of Moneymaker compared to 22 in T4 and 11 in T7. At 36 days from inoculation 82 root-knot nematodes had entered the Moneymaker roots compared to 19 in T4 and 7 in T7 while at 48 days from inoculation 74 root-knot nematodes had entered Moneymaker roots, 110 had entered susceptible T4 and only 20 had entered T7 roots. It was apparent that reduced larval penetration in the roots is one way of ensuring resistance as found in resistant plants of T7 and T4. In some T4 plants, apparently susceptible ones, a high number of fifth stage nematodes were found 48 days after inoculation.

The development of the root-knot nematode followed an interesting pattern. At 12 days from inoculation all the larvae in Moneymaker, T4 and T7 were at the 2nd stage. At 24 days one 3rd stage larva was noticed in Moneymaker roots while in T4 and T7 there were only 2nd stage larvae. At

Table 12. Number of *Meloidogyne javanica* in different stages of development in the roots of Moneymaker, T7, and T4 at 12, 24, 36 and 48 days after inoculation

Development Stage	MM				T4				T7			
	12	24	36	48	12	24	36	48	12	24	36	48
2nd Stage	33	54	49	4	10	22	10	1	1	11	2	4
3rd Stage	-	1	10	1	-	-	-	-	-	-	-	-
4th Stage	-	-	8	-	-	-	-	1	-	-	-	-
5th Stage female without eggs.	-	-	11	59	-	-	9	87	-	-	5	9
Female adult with eggs	-	-	4	10	-	-	-	21	-	-	-	7
Male Adult	-	-	-	-	-	-	-	-	-	-	-	-
Total	33	55	82	74	10	22	19	110	1	11	7	20

36 days from inoculation there were 4 female adults with eggs in Moneymaker while in T4 and T7 there were only young females. It was not until 48 days from inoculation that mature females with eggs in T4 and T7 were found. It seems therefore that the Meloidogyne javanica life cycle takes less number of days (24 to 36) in susceptible parent Moneymaker than in the resistant parents T4 and T7 (36 to 48).

4.6. Inheritance of Yield and Component Characters

4.6.1. Population Means

The mean performance of the population for each cross for the seven characters are given in Table 13. The F_1 population mean performance was greater than the top parent performance for vitamin C content, yield (except the cross MM x T7), and titratable acidity (except the cross T7 x T4). The F_1 population mean performance was less than the top parent in fruit diameter, fruit length, locule number and sugar content (except the cross MM x T7). The F_2 mean performance was better than the top parent in vitamin C (except the cross MM x T7) and titratable acidity (except the cross T7 x T4). The backcrosses performed better than the top

Table 13. Mean performance of the parents and crosses for the seven attributes in tomato.

Crosses	Populations					
	P1	P2	F_1	F_2	$P_1F_1(B_1)$	$P_2F_1(B_2)$
			Yield/kg/plant)			
MM x T7	1.420	1.415	1.375	1.367	1.242	1.608
T7 x T4	1.415	1.223	1.462	1.330	1.418	1.335
T4 x MM	1.223	1.420	1.492	1.447	0.968	1.271
			Vitamin C(mg/100ml)			
MM x T7	19.73	14.73	20.93	17.77	21.00	19.23
T7 x T4	14.73	11.73	19.37	21.10	21.73	22.20
T4 x MM	11.73	19.73	22.97	21.50	23.30	23.60
			Sugar (gm/100ml)			
MM x T7	2.43	2.50	2.70	2.50	2.67	2.53
T7 x T4	2.50	3.07	2.53	2.60	2.70	2.57
T4 x MM	3.07	2.43	2.70	2.57	2.57	2.77

Table 13 (Contd..)

Crosses	Populations					
	P1	P2	F_1	F_2	$P_1F_1(B_1)$	$P_2F_1(B_2)$
			Titratable acidity (% as citric)			
MM x T7	0.54	0.50	0.69	0.64	0.65	0.58
T7 x T4	0.50	0.64	0.61	0.57	0.62	0.65
T4 x MM	0.64	0.54	0.67	0.68	0.67	0.73
			Fruit diameter(cm/fruit)			
MM x T7	5.42	6.31	5.00	5.50	5.06	5.39
T7 x T4	6.31	6.39	5.05	5.81	5.50	5.18
T4 x MM	6.39	5.42	4.85	5.26	4.89	4.82
			Fruit length (cm/fruit)			
MM x T7	4.27	4.69	4.07	4.25	4.09	4.22
T7 x T4	4.69	4.54	4.05	4.51	4.35	4.10
T4 x MM	4.54	4.27	3.83	4.25	3.89	4.03
			Locule number/fruit			
MM x T7	2.250	6.075	2.450	3.075	2.575	2.575
T7 x T4	6.075	6.650	3.100	3.375	2.325	2.525
T4 x MM	6.650	2.250	2.725	2.550	2.400	2.250

parent in vitamin C content in all the three crosses. The backcrosses did not increase the fruit diameter, fruit length and locule number.

4.6.2. Estimates of the Gene Effects

The gene effects for yield, locule number, fruit diameter and fruit length were estimated and are given in Table 14.

Yield The additive gene effect was significant in two out of the three crosses showing that it is important in determining the yield of tomato plants. However, the additive gene effects were negative and less in magnitude compared to the mean effects indicating preponderance of genes with negative effects for yield. In the cross T4 x MM additive, dominance, additive x additive and dominance x dominance gene effects were all significant. The dominance x dominance gene effect was positive and greater in magnitude than the mean effect. The other significant gene effects were negative and less than the mean effect in magnitude. The dominance x dominance gene effects had the greatest influence in yield of T4 x MM.

Table 14. Mean estimates of the six gene effects for the four attributes in tomato

Cross	Gene effects					
	m	a	d	aa	ad	dd
			Yield.(kg/plant)			
MM x T7	1.367**	-0.366*	0.189	0.232	-0.368	-0.347
T7 x T4	1.330**	0.083	0.328	0.186	-0.013	-0.130
T4 x MM	1.447**	-0.303**	-1.140**	-1.310**	-0.205	2.459**
			Diameter (cm).			
MM x T7	5.50**	-0.33*	-1.97**	-1.10*	0.12	1.93**
T7 x T4	5.81**	0.32*	-3.19**	-1.88**	0.36*	3.32**
T4 x MM	5.26**	0.07	-2.68**	-1.62**	-0.42**	3.71**
			Length (cm)			
MM x T7	4.25**	-0.13	-0.80*	-0.38	0.08	0.86*
T7 x T4	4.51**	0.25**	-1.71**	-1.14**	0.17	1.57**
T4 x MM	4.25**	-0.14	-1.74**	-1.16**	-0.27*	1.79**
			Locule Number			
MM x T7	3.075**	0	-3.713**	-2.000*	1.913**	4.925**
T7 x T4	3.375**	-0.200	-7.063**	-3.800**	0.087	13.025**
T4 x MM	2.550**	0.150	-2.625**	-0.900*	-2.050	5.950**

* 5% level of significance
** 1% level of significance

Fruit Diameter. The additive, dominance and the epistatic gene effects were all significant. The dominance and additive x additive gene effects were all negative and less in magnitude compared to the mean effects. The dominance x dominance gene effect was significant in all the three crosses and was positive. It was also less than the mean effect. The additive gene effect was negative in the cross MM x T7 and positive in the crosses T4 x MM and T7 x T4. The additive x dominance gene effect was positive in the cross T7 x T4 and negative in T4 x MM. Dominance and dominance x dominance and additive x additive gene interations had the greater influence in fruit diameter than those of additive (a) and ad gene effects.

Fruit Length. The dominance and dominance x dominance gene effects were significant in all the three crosses. The dominance x dominance gene effect was positive while the dominance gene effect was negative. Both were less in magnitude when compared to the mean effect. The aa interation was the third important gene action. The additive and ad

gene effects were found least important. In the determination of fruit length both the dominance x dominance and dominance gene effects had the greatest influence.

Locule Number. The dominance, additive x additive, and dominance x dominance gene effects were significant in all the three crosses. The dominance and additive x additive gene components were negative. The dominance gene effects were greater in magnitude than the mean effects in all the three crosses. The dominance x dominance gene effects were positive and greater than the mean and dominance effects. The additive x dominance gene effect was positive and significant in the cross MM x T7 but it was also less than the mean effect. The dominance x dominance gene effect had the greatest influence in the inheritance of locule number and was followed closely by the dominance and aa gene effect.

CHAPTER 5

DISCUSSION

5.1. Inheritance of resistance to root-knot nematode (*Meloidogyne javanica*)

Based on galling scores two resistance genes, one dominant and one recessive were found to confer resistance to *M. javanica* in the materials studied. In case of parent T7 the resistance to *M. javanica* was governed by a single recessive gene, designated as $LMjr_1$ (i.e. *Lycopersicon esculentum* host, *Meloidogyne javanica* parasite, recessive gene for resistance number one), while a single dominant gene $LMjR_2$ was identified as confering resistance in T4. In earlier studies the resistance to *M. incognita* was reported in most cases to be governed by one or two dominant genes. From the original cross of *L. esculentum* x *L. peruvianum* made by Smith (1944), Watts (1947) suggested two dominant genes for resistance to *M. incognita*. Subsequently Frazier and Dennett (1949) postulated that one or two dominant genes were responsible for the host resistance. Later, Gilbert and McGuire (1956) identified a single dominant gene for resistance in the linkage group VI which they designated as *Mi* gene. Barham and Sassaer (1956) reported that the resistance to *M. incognita*, *M. incognita acrita*, *M. javanica* and *M. arenaria* was controlled by one or more

dominant genes. But later results suggested that resistance to those four root-knot nematode species was conferred by a single incompletely dominant gene (Barham and Winstead, 1957; Winstead and Barham, 1957). Using material from the L. esculentum x L. peruvianum cross, Thomas and Smith (1957) reported a single dominant gene for the resistance against M. incognita acrita and, possibly, M. javanica. Sidhu and Webster (1973) reported two dominant genes and one recessive gene for resistance against M. incognita, which they designated as $LMiR_1$, $LMiR_2$ and $LMir_3$, found in cultivars Nematex, Small Fry and Cold Set - 1, respectively. Recently, Kalloo et al. (1978) showed the resistance to M. javanica and M. incognita in tomato cultivars Nematex and 'R-2' to be controlled by a single dominant gene. Studies on linkage and allelic relationships among $LMiR_1$, $LMiR_2$ and $LMir_3$ indicated that two of the resistant genes, $LMiR_1$ and $LMiR_2$ are closely linked and are approximately 5.65 morgan units apart, while the resistant genes $LMiR_1$ and Mi were found to be either indentical or allelic (Sidhu et al. 1975). The resistance gene $LMjR_2$ found in T4, appears to be allelic or identical to other dominant resistant genes at Mi locus reported by previous workers.

It seems rather curious that all the tomato cultivars tested to date for resistance to M. incognita and M. javanica apparently carry only one Mi locus for resistance. Parasitism is a dynamic association evolving progressively under changing environmental conditions and although there are hardly any genetic studies on Meloidogyne, it is reasonable to assume that there are strains or biotypes within the species. Sturhan (1971) reported that M. incognita may be composed of many biotypes or strains and populations with variable pathogenicity to various hosts. If this is the case it is only reasonable to suppose that fine differences in resistance (= pathogenicity) would be expected to exist in different cultivars of the same host species. Based on the linkage studies Sidhu et al. (1975) presumed that the Mi gene may eventually be found to have different but very closely linked resistance genes or pseudoalleles depending upon their duration of association with the parasite and breeding background.

The recessive gene LMjr$_1$ found in T7 appears to be of rare occurrence in tomato. The only other report of a partial recessive resistant gene LMir$_3$ is that of Sidhu and Webster (1973) and was found in Cold Set -1 when used as female parent. But,

even the partially resistant plants were not recovered when cultivar Cold Set -1 was used as a pollen parent. Such resistance of Cold Set -1 was assumed by them to be a cytoplasmic effect. Therefore, the recessive resistance gene LMjr$_1$ of T7 appears to be a new record. In both cases, either used as pollen or female parent the T7 gene segregated into 3 susceptible: 1 resistant progenies in F_2 and 1 resistant: 1 susceptible in test cross. The recessive resistance gene LMjr$_1$ could be located in chromosome VI or in a separate chromosome. In absence of linkage test its location could not be ascertained in the present studies.

5.2. Histopathology and Development

The Meloidogyne javanica larvae entered near the root tips in Moneymaker and susceptible T4 and migrated to its feeding site in the stele where it induced giant cells. Such changes have been reported and studied in detail by Myuge (1956), Owens et al. (1960), Bird (1962), Owens and Specht (1964, 1966) and Owens and Bottino (1966). It is a known fact that resistant and susceptible plants have different chemical make up and especially an increase in phenolics increases the degree of resistance as reported by Pi and Rohde (1967), Chia-Ling Hung

et al. (1971), Singh et al. (1974) and Alam et al. (1976). Although the chemical make up of T7 and resistant T4 was not looked at it is apparent that reduced larval penetration plays a part in the resistance of these two tomato lines. Reduced larval penetration has also been reported by Dean and Struble (1953), Peacock (1959) and Gowen et al. (1969) in L. peruvianum and resistant Nemared tomato.

The first mature female M. javanica with eggs were noticed 36 days after inoculation in susceptible Moneymaker tomato. In T4 and T7 the mature females with eggs were seen 48 days after inoculation hence it seems the life cycle is slightly longer in T4 and T7 compared to Moneymaker. Peacock (1959) noticed a slower rate of development of the larvae in resistant L. peruvianum compared to susceptible tomato, L. esculentum. Slower rate of development also plays a part in the resistance of non-hosts to root-knot nematodes. The deformation of the mature female nematode in the roots of T7 (Fig. 18) may have been due to a toxic compound in the roots.

5.3. Tomato Yield Loss

The results on tomato yield loss assessment trial showed that root-knot nematodes reduced

both fresh fruit weight and fruit numbers. Similar results were found by Chitwood (1951), Sayre et al. (1964), Barker et al. (1976), Olthof et al. (1977) and Wisnuwardana (1978). Although no actual known larval populations were inoculated per kg. of soil it seems that the 156 gms. of the chopped galled roots contained enough nematodes beyond the economic threshold value (> 500 larvae/kg. of soil as shown by Wisnuwardana, 1978). The D-D soil fumigant was as effective as the genetic resistance in T7 in lowering the nematode galling and hence the effect of the nematodes in the host (Table 8). There was a significant reduction in both fruit numbers and fresh fruit weight in the inoculated plots of T7 as compared to the fumigated plots. This, coupled by the fact that some female nematodes reached maturity in T7 and actually laid some eggs would suggest that the recessive resistance gene in T7 confers resistance only to galling but not to the development of the nematode. It could be reasonable to suppose that host resistance and galling response are controlled by separate genetic mechanisms in the present case. A similar situation was encountered in breeding root-knot nematode resistant snap beans (Fassuliotis et al., 1970). In this case the resistance gene $\underline{LMjR_2}$ in T4 will be more useful

in a breeding programme than the recessive gene in T7 where galling and host resistance appear to be governed by the same gene.

The percentage loss of 31.9% in Moneymaker would have been much higher if there was no blossom end rot as the average weight of a Moneymaker fruit attacked by the disease was 27 gms. compared to 50 gms of a marketable disease free fruit. Without the disease the percentage loss is postulated to have been around 40%.

The root-knot nematodes did not affect the sugar and vitamin C content of the fruits but they increased the titratable acidity of Moneymaker fruits. This higher acidity in the fruits from the inoculated plots might have been due to the earlier ripening as compared to the fruits from fumigated plots (Wisnuwardana, 1978). The root-knot nematodes did not affect the fruit diameter, fruit length and locule number of the fruits. These are mainly governed by the genotype of the tomato cultivar (Haughtaling, 1953; Rick and Butler,1956 as quoted by Mital et al.)

5.4. Inheritance of Other Quality Characters

For yield the additive gene action was significant in two out of the three crosses. It was the major gene action in the cross MM x T7. This major contribution is expected yield being mainly a quantitative character. In T4 x MM additive, dominant and epistatic gene effects were found. The dominance effect was greater than the additive effect in magnitude. Among the epistatic effects the dominant x dominant effects were more important than the additive x additive gene effect. The dominant x dominant epistatic gene effect was more important thant the mean effect.

In the fruit size characters (fruit diameter, length and locule number) in all the crosses with Moneymaker as one of the parents the fruit shape and size tended towards Moneymaker, the one with the smaller fruits, hence it is not surprising to find the dominance and dominance x dominance gene effects contributing more than the additive and additive x additive gene effects. The significant additive gene effects for the fruit diameter and length in the crosses MM x T7 and T7 x T4 support the finding of Young and MacArthur, 1947 as quoted by Mital *et al*. that the fruit size depends on as many as 9 to 15 genes.

5.5. Breeding root-knot nematode resistant varieties of tomato

Rohde (1965) indicated that plants are resistant to nematodes for one or more of the following reasons:

(1) The plant is not attractive. The roots may lack an attractant or may produce a toxic substance.

(2) The host tissues are not suitable for continued nematode feeding. In hypersensitive reactions, the plant tissues die so rapidly that the nematode is isolated in necrotic tissue and its development is hindered.

(3) The host fails to respond to the presence of parasite. For example, galls or giant cells may not form, or they may not form sufficiently for nematode reproduction.

(4) The plant responds physiologically, morphologically, or in other ways that react adversely on the parasite.

(5) Genetic (and other) reactions of the plant are modified by its environment.

Such effects include temperature, mineral nutrition, soil textures, and age and vigor of plant.

In resistant cultivars T7 and T4 fewer Meloidogyne javanica larvae entered the roots compared to susceptible Moneymaker tomato. This suggests that the plants are not attractive for nematode entrance which may be due to toxic substances in the roots. It seems that the thickness of the epidermis and the smoothness of the epidermal surface are not a factor in the resistance as resistant T4 as well as susceptible Moneymaker had thick epidermis and rough epidermal surfaces.

In Kenya, Moneymaker, Roma and Mecheast are the popular commercial varieties. If they are improved in their resistance to root-knot nematode damage they would be more attractive from production view point. With the available resistance source and breeding techniques the appropriate method of transferring resistance is the backcross pedigree (Allard, 1960). It is easier to transfer recessive than dominant genes as in the former case all resistant plants will be in a homozygous condition while in the latter case it is not easy to identify homozygous dominant and heterozygous

plants. This would suggest that recessive resistance of T7 would be preferred but its use could be avoided in view of the fact that this resistance was not effective to control the yield loss, although there was no galling. The more reliable resistance is the dominant gene as found in T4. It is therefore suggested that screening for root-knot nematode resistance should be directed not only on the low galling incidence but also on the number of nematode eggs produced and actual yield loss assessments.

CONCLUSION

Meloidogyne javanica was found to be the predominant root-knot nematode species in the soils of Thika area. Based on galling scores two resistance genes, one recessive, $LMjr_1$ in cultivar T7 and the other dominant, $LMjR_2$ in T4 were found to confer resistance to *M. javanica*. There was reduced larval penetration in resistant cultivars T4 and T7 compared to that of the susceptible cultivar Monemaker. However, in case of T7 some larvae entered the roots and matured and reduced the fruit number and fruit weight. This suggests that the recessive gene $LMjr_1$ of T7 was effective only in suppressing the galling but not the nematode development. The dominant gene $LMjR_2$ of T4 on the other hand was effective in both suppressing the gall formation as well as nematode development.

The nematode infestation had no effect on sugar and vitamin -C contents of both Moneymaker and T7 but it increased the acidity of Moneymaker fruits. It had no effect on fruit diameter, length and locule number of both Moneymaker and T7.

The additive and dominance x dominance gene effects had the greatest influence on yield of two

out of three crosses studied. The dominance and dominance x dominance gene effects played a major role in determining the fruit size (diameter x length x locule number) in all the three crosses.

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Appendix 1

Export of Processed Horticultural Produce from Kenya, by value in 1973 and 1974.

	1973 K.Shs.	1974 K.Shs.
Dried fruit	219,274	629,242
Jams, marmalade etc.	1,073,224	1,945,265
Marmalades	.....	115,249
Jellies	.....	30,211
Fruit and veg. juices incl. passion fruit	1,871,375	1,998,881
Pineapple juice	700,252	975,153
Tomato juice	52,732	168,227
Tomato sauce	.....	344,677
Other fruit and veg. juices	229,403	244,891
Pineapple canned	29,603,168	28,232,280
Nuts and fruit preserved	254,175	1,345,300
Beans, peas, lentils, dried	23,464,905	30,891,230
Vegetables dehydrated	3,183,507	3,777,937
Mowers of beans, peas, lentils	4,191,443	7,363,478
Yellow gram flour	.....	385,006
Vegetables and Fruit preserved by vinegar	25,929	186,409
Vegetables otherwise preserved	4,523,744	4,053,980
	69,393,131	82,687,416

Source: Annual Trade Report, E.A. Customs and Excise Department 1973/74.

Appendix 2

Production estimate (hectares) in Kenya

1975-77

Province	1975	1976	1977
Central	1491	1059	-
Eastern	-	942	1358
Rift Valley	-	343	240
Western	325	425	-
Coast	-	135*	-
Nyanza	150.3	280.6	382.1
North Eastern	-	32	-

Source: Provincial Director of Agriculture Annual reports.

*Estimate from Taita Hills only.

Note: A dash denotes no figures available.

Appendix 3

Tomato production estimates in the top 2 provinces 1976/77

Province	District	1976		1977	
		Ha.	Tons	Ha.	Tons
Central	Kiambu	527	8324	659	11,028
	Murang'a	196	3400	274	1,538
	Nyeri	127	1266	-	344,027
	Kirinyaga	209	3129	191	1,260
	Nyandarua	-	-	-	-
	Sub-total	1059	16119	-	357,853
Eastern	Meru	500	30000	550	31000
	Machakos	385	66	513.3	1184
	Embu	42	457	44	396
	Marsabit	4	3	6.5	5.5
	Kitui	15	450	18	72
	Isiolo	-	-	4	12
	Sub-total	946	30976	1135.8	32669.5
	Total	2005	47095		

Source: Provincial Director of Agriculture annual reports.

Appendix 4 — Nematode Parasites of Plants

Lycopersicon esculentum Mill.	Aph. ritzemabosi	Junges, 1938
	Bel. gracilis	Holdeman & Graham, 1953
	Dit. dipsaci	Williams, 1936
	Dolichodorus heterocephalus	Christie, 1952b
	Helicotylenchus microlobus	Taylor, 1960
	Helicotylenchus sp.	Martin & Birchfield, 1955
	Hemicycliophora arenaria	van Gundy, 1959
	Hemicycliophora similis	Khera & Zuckerman, 1963
	Het. schachtii	Golden & Shafer, 1959a
	Het. schachtii =rostochiensis	Morgan, 1925
	Het. tabacum	Lownsbery & Lownsbery, 1954
	Het. trifolii	Holtzmann & Aragaki, 1963
	Longidorus elongatus	Jensen, 1961
	Longidorus maximus	Sturhan, 1963

Appendix 4 (contd..)

Mel. acronea	Coetzee, 1956
Mel. arenaria	Tarjan, 1952C
Mel. arenaria thamesi	v.d. Linde, 1956
Mel. hapla	Chitwood, 1949b
Mel. incognita	Tarjan, 1952c
Mel. incognita acrita	Chitwood, 1949b
Mel. javanica	Tarjan, 1952c
Mel. sp.	Neal, 1889
Nacobbus batatiformis	Thorne & Schuster, 1956
Nacobbus sp. C.W.Graham, 1958=N.	serendipiticus see Franklin, 1959
Paratylenchus projectus	Coursen et al., 1958
Pra. brachyurus	Martin & Birchfield, 1955
Pra. neglectus	Mountain & Fisher, 1954
Pra. penetrans	Mountain & Fisher, 1954

Appendix 4 (contd..)

Pra. ?pratensis	Godfrey, 1929
Pra. pratensis Hastings & Bosher, 1938=P. penetrans see	Mountain, 1961
Pra. scribneri	Thomason & O'Melia 1962
Pra. zeae	McBride et al., 1961
Rad. similis	Feder & Feldmesser, 1957
Rotylenchulus reniformis	Linford & Yap, 1940
Rotylenchus buxophilus	Golden, 1956b
Tetylenchus joctus	Khera & Zuckerman, 1963
Trich. christiei	Coursen et al., 1958
Trich. sp.	Christie & Perry, 1951
Tylenchorhynchus capitatus	Mountain & McKeen, 1962
Tylenchorhynchus claytoni	Krusberg, 1959
Xiph. diversicaudatum	Schindler, 1954

Appendix 4 (contd..)

Lycopersicon esculentum Mill. v. Aureum	Het. rostochiensis	R.D. Winslow, 1954
	Het. schachtii	Raski, 1952
Lycopersicon esculentum Mill. v. Cerasiformis Alef.	Mel. javanica	G.C.M. 1958
Lycopersicon esculentum Mill. v. Commune Bailey	Trich. christiei	Rohde & Jenkins, 1957

KEY: Aph. = Aphelenchoides
Bel. = Belonolaimus
Dit. = Ditylenchus
Het. = Heterodera
Mel. = Meloidogyne
Pra. = Pratylenchus
Rad. = Radopholus
Trich. = Trichodorus
Xiph. = Xiphinema

Appendix 5

Chemical	Other names
Basamid	Dazomet (DMTT)
Methomyl	Lannate
Dimethoate	Rogor E, Perfekthion etc
Parathion	Parathion, Ekatox 50 etc
Malathion	Maladrex, Kilpest etc
Fenitrothion	Sumithion, Folithion
VC - 13	-
Chloropicrin	Picfume, Acquimite
BAY - 25141	-

Active Ingredient

Tetrahydro-3, 5 dimethyl-2H-1, 3, 5-thiadiazine-2 thione.

1-(Methylthio) ethylideneamino N-methylcarbomate.

Dimethyl S-(N-Methylcarbamoylmethyl) Phosphorothiolothionate.

Diethyl-4-nitrophenyl Phosphorothionate.

S-(1,2-di(ethoxycarbonyl)ethyl) dimethyl Phosphorothiolothionate.

Dimethyl 3-methyl-4-nitrophenyl phosphorothionate.

2,4-dichlorophenyl diethyl phosphorothionate.

Trichloronitromethane

O,O-diethyl 1-O-P (Methylsulphinyl) Phenyl phosphorothionate.

Appendix 5 (contd..)

Chemical	Other names
Gardona	Rabon, Rabond, Appex
Thionazin	Nematos, Zinophos
Disulfoton	Disystox, Solvivex
Formothion	Anthio
Aldicarb	Temik, carbamoyl oximes, oxamyl
Carbofuran	Furadan
Di-Trapex	Vorlex, MENCS, MIC, MITC, Trapex
Ethoprop	Ethoprophos, Mocap, Prophos
Maleic hydrazide	Maleic hydrazine, sucker stuff, Retard, Sprout stop, Slo-Gro

Active Ingredient

2-Chloro-1-(2,4,5-trichlorophenyl) vinyl dimethyl phosphate.

Diethyl O-2-Pyrazinyl phosphorothionate.

Diethyl S-(2-(ethylthio)ethyl) phosphorothiolothionate.

S-(N-formyl -N-Methylcarbamoylmethyl) dimethyl phosphorothiolothionate.

2-(Methyl-(Methylthio) propionaldehyde O-methyl-carbamoyl oxime.

2,3-dihydro-2, 2-dimethyl-7-benzofuranyl-N-methyl carbamate.

Methyl isothiocyanate.

O-Ethyl S, S-dipropyl phosphorodithionate

1,2-Dihydro-3, 6-pyridazinedione

Appendix 5 (Contd)

Chemical	Other names
Phenamiphos	BAY 68138,Fenamiphos,Nemacur
DD	Vidden-D, Nemafene
DBCP	Nemagon, Fumazone 86 Fumagon, Fumazone 86E Nemafume
EDB	Ethylene Dibromide, Ethylene bromide, Fumo-gas, E-D-BEE, Bromo-fume, Soilfume, Dowfume W-85, Urifume, Soilbrome -85. Kopfume, Nephis, Clemide, Pestmaster, Soilbrom-40, Soilbrom-90EC.
Methamsodium	Vapam, VPM, S.M.D.C., Monam, Metam, Herbatim, Karbation, Maposol,Vaporooter, Trimaton,Sodium N-methyl dithiocarbamate.
Oryzalin	-
BAS 083	BAS-08300W

Active Ingredient

Ethyl-3-methyl-4-(methyl thio) phenyl (1-methylethyl) phosphoramidate.

1,3-Dichloropropene 1,2-Dichloropropane.

1,2-Dibromo-3-chloropropane

1,2-Dibromoethane

Methyl isothiocyanate

3,5 - dinitro-N^4,N^4-dipropyl-sulfanilamide

1,1- dimethylpiperidinium chloride