INTRODUCTION

Plant-parasitic nematodes cause morphological and physiological changes of the affected tissues in plants. These changes involve damage or death of the cells by removal of their contents, or the host cells adapt to nematodes by enlarging and increasing their metabolic activities, or the cells undergo growth and multiplication. These effects of nematode parasitism of plants have been termed as destructive, adaptive and neoplastic, respectively (Dropkin, 1980). Development of galls induced by some nematodes especially root-knot nematodes (Meloidogyne species) results from neoplastic changes. Root-knot nematodes are endoparasites. The female nematodes remain wholly embedded in the roots of the host plant. They show distinct sexual dimorphism.

Second-stage juveniles penetrate roots at or near the root tip. The movement of juveniles within the roots is primarily intercellular (Endo and Wergin, 1973). Infection with root-knot nematode stimulates the formation of variable number of discrete giant cells (usually about 6, but reported to vary from 2 to 14) in host tissue. Hyperplastied and hypertrophied tissues often surround the region of infection and galls
are formed terminally or subterminally on the infected roots. The term giant cell refers to the multinucleate transfer cells usually induced by root-knot nematodes in which the multinuclear condition of each giant cell results from repeated mitoses without cytokinesis (Endo, 1987).

Nemec (1910) noted that the cells in the plerome close to the nematode head immediately commence to enlarge, their plasma contents increase and their nuclei divide without formation of separating walls. Kostoff and Kendall (1930) observed multinucleate cells formed by nuclear division without accompanying cell division in early stages of root-knot nematode invasion on the roots of *Nicotiana*. But they believed that giant cells were formed by dissolution of cell walls followed by coalescence of cell contents. Christie (1936) observed progressive dissolution of cell walls followed by coalescence of their protoplasms during expansion of the giant cell in tomato roots.

Huang and Maggenti (1969a) in their detailed study on giant cell development in *Vicia faba* roots found no evidence of cell wall dissolution or break-down. They noted that multinucleate condition of giant cells arose from repeated mitoses without cytokinesis of a single diploid cell. Jones and Payne (1978) provided support for the concept of single-cell induction through a study on early stages of giant cell formation in
balsam roots induced by *Meloidogyne incognita* (Kofoid and White) Chitwood. They found no evidence of cell wall breakdown and dissolution in the stimulated cell and concluded that the giant cell originated from a single cell and the cell wall intrusions reported along the giant cell wall were not wall fragments but infolded walls of the giant cell.

The number of nuclei within the developing giant cell could be correlated with the number of host cells that would normally occupy the volume of the syncytium (Owens and Specht, 1964). Huang and Maggenti (1969a) suggested that chromosome count can be predicted in a giant cell. While studying giant cells in roots of *Vicia faba* infected with *Meloidogyne javanica* (Treub) Chitwood, they proposed an equation: \( N = 2^d \times 12 \), where \( N \) represents total number of metaphase chromosomes, \( d \) the number of mitotic cycles, and 12 the diploid chromosome number of the host plant.

In giant cells nuclear changes range from a nucleus having a hypertrophied nucleolus to nuclei with various stages of membrane deterioration and a lobulated periphery. Other nuclear aberrations include nucleolar fragmentation so that small granules, stained like nucleoli remain scattered throughout the nucleus. Irregularly shaped, dumbbell or sickle shaped nuclei have been reported by many workers (Krusberg and Nielsen, 1958; Owens and Specht 1964; Rubinstein and Owens, 1964; Huang
and Maggenti, 1969a). Nuclear enlargement results from swelling and in some cases from nuclear fusion where some of the nuclei attain a diameter of 35 μm as compared to the normal cell nucleus of 6 μm. The nuclear volume may increase to 10 – 12 times in tomato roots infected with root-knot nematodes (Rubinstein and Owens, 1964). Siddiqui and Taylor (1970) observed one or more vacuoles in the hypertrophied or fragmented nucleoli.

The cytoplasm of the young giant cells (within 48 h) becomes dense and encloses a large vacuole. The vacuole diminishes and the cytoplasm becomes more dense as well as granular with the giant cell development. Christie (1936) found more dense and hyperchromatic cytoplasm near the head region of the nematode than remainder of the giant cell cytoplasm. As the nematode matures and nutrient demand from the giant cells increases, the giant cell cytoplasm shows signs of intense metabolic activity. Nuclei become highly lobed and heterochromatic with prominent and numerous nuclear pores. The cytoplasm becomes vacuolated and vacuolation increases when nematode reaches its maturity. The giant cell cytoplasm is disintegrated after egg laying of the nematode. The cytoplasm finally becomes extracted as the giant cells senesce leaving a little or no ground cytoplasm.

Galling is one of the earliest host responses of root-knot
nematode infection in the roots of the host plant. Christie (1936) reported that the nematode stimulated the cells of pericycle to divide which resulted in a layer of small cell parenchyma. While working with *Meloidogyne incognita*, *Meloidogyne incognita acrita* and *Meloidogyne hapla* Chitwood on Gardenia, Davis and Jenkins (1960) found cortical and stelar proliferation in all infections. *Meloidogyne javanica* infection on soybean roots caused hypertrophy, hyperplasia and giant cell formation in the tissue surrounding the nematode head and consequently led to gall formation (Ibrahim and Massoud, 1974).

According to Siddiqui and Taylor (1970) gall formation is attributable to hypertrophy of the cortical cells, xylem parenchyma and metaxylem, hyperplasia of pericycle and xylem parenchyma, formation of giant cells, nematode development and egg mass production.

Xylem and phloem tissues become abnormal with reference to structure and orientation. Because of abnormalities, the translocation of water and mineral nutrients from the root towards the shoot, and of photosynthates towards roots, is disrupted. Formation of giant cells, proliferation of parenchyma cells and multiplication of pericycle and the cells around the nematode head causes the conducting xylem to scatter from its normal path. Vessel elements are also hypertrophied and become irregular in shape. Small as well as hypertrophied parenchyma cells
are transformed into vessel like elements and constitute the abnormal xylem. Production of enormous amount of abnormal xylem elements is thought to carry water and nutrients in greater amounts to compensate the loss caused by root-knot nematode infection (Pasha et al., 1987).

Wallace (1971) found that when initial population level of root-knot nematode was low, the plant growth was stimulated, but when the population level was high, the plant growth and the yield were suppressed. At higher population levels when there was a competition for a limited food supply and the space, the size of the mature female was reduced. Triantaphyllou (1960) found that percentage of males tended to increase in the total population as the population density of the nematode increased. The above-ground symptoms on the plants of heavily infected roots include stunting, loss of yield, reduction in quality of produce, severe deficiency symptoms of some elements, particularly nitrogen, incipient wilting during hot periods of the day, and even loss of resistance to foliage diseases and vascular wilts and to certain other pathogens (Jenkins and Taylor, 1967). Plants infected with root-knot nematodes often display potassium deficiency symptoms. Application of high dose of potassium results in improved top growth of plants and improved nematode reproduction (Dropkin, 1980). The host nutrition generally affects the severity of the disease and
growth of the parasite, but not in the same way in all host-parasite combinations.

Attractiveness of *Meloidogyne* species juveniles was observed toward the root and excised shoot tissues of several host plants (Linford, 1939). The juveniles of *Meloidogyne hapla* were equally attracted toward the resistant and the susceptible alfalfa seedlings when an egg mass was placed midway between germinating seedlings (Griffin and Waite, 1971). In resistant varieties the juveniles of root-knot nematode either do not enter or if entered they do not develop properly (Sasser and Taylor, 1952). Resistance in plants toward root-knot nematode may develop with age (Griffin and Himt, 1972).

Absorption and translocation is perhaps markedly affected by *Meloidogyne* infection as there are conflicting reports about accumulation or deficiency of N, P, and K in above-ground plant parts. Hunter (1958) did not find any alteration in N, P, and K contents in leaves although shoot growth was suppressed and foliage became chlorotic in *M. incognita* infected tomato plants. Oteifa and Elgindi (1962) found an impaired translocation of 32P in *M. incognita* infected tomato plants, increasing in roots and decreasing in shoots. *Meloidogyne incognita* infected lima bean plants showed decrease in mineral elements, but with increase in K contents in the nutrient medium, the mineral
contents in the leaves increased (Oteifa, 1952). Khan (1969) found an increase in N, P and K contents in roots of okra infected with *M. incognita*. However, when K was in excess in nutrient medium, the infected plants exhibited an increase in K content in roots, stems and leaves.

When the plants are deficient in a single essential element, they show deficiency symptoms on stems and leaves. The plants deficient in an essential element, if infected with *M. incognita*, show more pronounced symptoms. Rate of development of the nematode is also influenced with increase or decrease in N, P and K concentrations in rooting medium of infected plants. Excess or deficiency of these elements may also influence egg production and giant cell size (Oteifa, 1951, Bird, 1960, Bird, 1970).

*Luffa* (*Luffa cylindrica* L.) is a climber grown throughout India for its smooth, cylindrical, tender fruits, which are used as vegetable. Apart from fungal and viral diseases, it is also attacked by root-knot nematodes resulting in relatively large sized galls.

In response to root-knot nematode infection giant cells are formed which are supposed to be developed from undifferentiated vascular tissue. After egg laying mature female dies and the giant cells collapse. Gall formation results due to
hyperplastic and hypertrophic reactions in affected tissue. Due to these two reactions vascular strands become abnormal. Number of nematodes attacking a particular site may correspond to gall size. Gall size may also depend on the type of the host plant. Plant growth and nematode development are greatly influenced by altering N, P or K concentrations in the rooting medium. Their deficiency or excess may also affect structure and function of vascular elements.

The present studies were carried out:

(i) to compare anatomical changes leading to the formation of giant cells and galls from earlier findings,
(ii) to investigate the origin of giant cells and their fate after death of the nematode,
(iii) to find out any link of giant cells with phloem,
(iv) to observe abnormalities in xylem and phloem,
(v) to examine response of plant to different inoculum levels,
(vi) to study any resistance towards root-knot nematode infection in different varieties, and
(vii) to compare any effect of deficiency or excess of N, P, or K on plant growth; nematode development; N, P and K contents in leaves, stems and roots, separately; and anatomy of the galled tissues.
The experiments were performed to compare and confirm earlier reports and to provide some new information. For this purpose following experiments were conducted.

SECTION I

Experiment 1. Formation of giant cell in roots of *Luffa cylindrica* infected with *Meloidogyne incognita*.

Experiment 2. Histopathological changes in luffa roots leading to gall formation.

Experiment 3. Abnormalities in vascular tissue as a result of root-knot nematode infection.

SECTION II

Experiment 1. The effect of different inoculum levels of *Meloidogyne incognita* on *Luffa cylindrica*.

Experiment 2. Histopathological responses of some varieties of *Luffa cylindrica* to *Meloidogyne incognita*.

SECTION III

Experiment 1. The effect of deficiency or excess of nitrogen, phosphorous, and potassium on luffa infected with root-knot nematode.