INTRODUCTION

Through the ages the mango has been known as an excellent fruit relished by adults and children alike and in cultivation in India since prehistoric times. The fruit appears to have a strong link with cultural history of India. It has been portrayed in the paintings and sculptures, Hindu folklore and mythology, legends and in the sacred Sanskrit scriptures dating back to 2000 B.C. Mango fruits have been an esteemed item of diet and the tree a subject of great veneration. Besides giving shade against the tropical sun, the tree provides timber

Mangifera indica L. is indigenous to North east India and North Burma in the foot hills of the Himalayas and is said to have originated to Indo-Burma region (Decandole 1904; Popenoe, 1920; Mukerjee, 1951). The mango was found throughout southeast Asia and the Malay Archipelago in the early days.

In India, the mango is distributed throughout the length and breadth of the country except in hilly regions above 915 m from mean sea level. According to the Crops Division, Union Ministry of Agriculture for 1978-79 mango occupies 42.6% of the total area under fruits The leading mango growing states in India are Uttar Pradesh, Andhra Pradesh, Bihar, Orissa and West Bengal. Commercial plantings of the most wanted variety 'Alphonso' for export are located in Maharashtra (Ratnagiri) and Gujarat (Bulsar).

A large number of diseases including mildew, Anthracnose, Diplodia-stem rot, leaf blight, bacterial canker and some pests including mangohopper, mango mealy bug etc cause damage to the plant. Among all the known diseases and insect pests the disease affecting flowers and fruits are the most serious and are of much concern to the mango industry. One of such serious disease is Mango malformation causing severe damage to vegetative and floral parts. The disease is well known in India and has also been confirmed in most mango-growing countries such as Pakistan, Egypt, South Africa, Brazil, Israel, Central America, Mexico and USA, Sudan, Cuba, Australia, Bangladesh and recently, the United Arab Emirates (c.f. Kumar *et al.*, 1993).

Mango malformation was first observed in India by Watt (1891). An increasing incidence of this malady since then has posed a threat to the mango industry of U.P.,

Bihar and Punjab in the past few decades (Jawanda, 1963; Mallik, 1963; Prasad *et al.*, 1965) Mango malformation has also been reported from Maharashtra, (Burns, 1910) and Gujarat (Desai *et al* , 1962).

The disease is endemic as a tree once infected never recovers (Mallik, 1963). Two distinct stages of malformation, vegetative and floral characterize the disease. Malformed inflorescence on a tree do not bear fruits, thus causing severe loses in yield. Tree loses upto 86% in one grove, have been recorded over a three-year period (Kumar, 1983) In Northern India in particular, over 50% of the trees are affected, with consequent heavy loses in yield (c.f. Singh and Dhillon, 1990).

1.0 Vegetative malformation (VM) : It is more pronounced on young seedlings, but also appears on mature trees. Typical symptoms on seedlings are loss of. apical dominance and swelling of axillary and terminal vegetative buds. These buds eventually form shootlets bearing small, scaly leaves with a bunch-like appearance, the so called bunchy-top stage.

In trees also shoots develop large number of vegetative buds leading to the bunchy growth of young shoots which may eventually dry and resume growth in the next season. Generally a branch showing vegetative malformation produces malformed inflorescence. Development of vegetative malformation on most branches of a tree lead to considerably reduced flowering or no flowering at all.

1.1 Floral or blossom malformation (FM) :

Floral malformation appears with the emergence of inflorescence. Any deviation of a part or entire panicle form the normal growth to abnormal is considered as a symptom of the disease The flowers in a malformed inflorescence are much enlarged and crowded around the hypertrophied axis of the panicle. These flowers usually fail to produce fruits However, in certain panicles a few fruits may develop but they fall off after reaching pea size. Increased and crowded branching in malformed panicles generally cause them to be heavier and these panicles are usually green in colour. A severely malformed inflorescence produces far more flowers, though most remain unopened.

The most conspicuous symptoms of malformation on panicles are phyllody and hypertrophy without necrosis (Kumar and Beniwal, 1992).

1.2 Causal Organism :

The cause of the disease has been attributed to various factors like physiological disorder, mites, as well as viral and fungal pathogens. There are many intrinsic discrepancies in these hypothesis which need indepth study.

1.2.1 Physiological disorder . According to this hypothesis, a physiological imbalance is created due to the deficiency of macro- (Prasad *et al.*, 1965) and micro-nutrients (Lynch and Runchle, 1940) disturbed C/N ratio (Khan and Khan, 1960), accumulation of gibberellins (Mishra and Dhillon, 1980; Campbell and Marlatt, 1986) and cytokinins (Bist and Ram, 1986; Nicholson and Staden 1988) in high amounts. The disease may also be caused due to drop in auxin level (Pandey *et al.*, 1977) which finally results in the formation of malformed shoot or abnormal inflorescence. On the other hand malformation disease spreads very fast in nature. The incidence of vegetative malformation becomes doubled in five months (Nirvan, 1953). Had a pathogen not been the cause, the disorder could have never spread so rapidly.

1.2 2 Eriophyes mite The idea of mite as the causal organism was mooted by the Egyptian scientist, Hassan (1944). There are number of papers (Narasimhan, 1954; Puttarudraiah and Basavanna, 1961) supporting this hypothesis. However, Latif *et al.*, (1961) ruled out the role of mites in causing malformation in mango. Finally, role of mite has been accepted as the carrier of the fungal pathogen Summanwar (1967), (Kumar *et al.*, (1995).

1.2.3 Virus : In 1946, scientists working at the Layalpur Research Station (Pakistan) failed to isolate any pathogen from the malformed shoots and panicles. They concluded that the disease "may be due to a virus" (Sattar, 1946). Later, Latif *et al.*, (1961) who shared this view could not establish the viral nature of the disease. However, Kistah *et al* , (1985) proved conclusively that neither virus or mycoplasma is the causal organism of the disease

1.2.4 Fungus : The hypothesis that the disease is caused by *Fusarium moniliforme* was based on the evidence that it satisfied Koch's postulates (Summanwar *et al.*, 1966). But neither physiology of the pathogenisis was investigated nor any attempt was made to correlate the manifestation of the disease syndrome with internal metabolic changes (Chakrabarti, 1996).

Isolates from vegetative and floral malformed tissues consistently show a much higher association of *Fusarium* species with diseased tissues than in corresponding healthy tissues (Darvas, 1987). Furthermore, it is not uncommon to detect more than one species of *Fusarium* in malformed tissues (Kumar and Beniwal, 1992). However, subsequent detection of *Fusarium* sp. in healthy tissues (up to 70.2%) has led some investigators (Rajan, 1986; Rana, 1992) to characterize it as a non-pathogenic parasite associated with mango tissues.

1.3 Etiology :

Finally, role of eriophid mite has been accepted as the carrier of the fungal pathogen *F moniliforme* (Summnawar, 1966, 1967; Kumar *et al.*, 1995) The disease symptoms are the combined effects of the aberrant host metabolites including mangiferin produced in response to the pathogenic invasion and the phytotoxic compounds secreted by the pathogen with in the host (Chakrabarti and Kumar, 1997). Thus a proper balance of mangiferin, the fungal pathogen and the mites (Vector) is essential for development of the disease (Chakrabarti, 1996).

Nevertheless, earlier histological findings (Varma *et al.*, 1974; Chakrabarti and Ghosal, 1989) of malformed tissues revealed occasional inter-and intracellular distribution of fungal hyphae in cortex, phloem (Varma *et al.*, 1974) and parenchymatous pith cells (Bindra *et al*, 1971, Varma *et al.*, 1974). Fungal mycelium has also been detected at the juncture of the shoot tip and malformed inflorescence, in malformed axillary buds, axes of petals and sepals of malformed buds (Kumar, 1983).

Only recently Raafat *et al.*, (1995) studied endogenous activity of gibberellins and cytokinins of malformed shoots in vegetative and floral malformation giving emphasis to histological disorders. Electron microscopy study of petals, leaf midribs and fine roots revealed no association of the pathogen (Kistah *et al.*, 1985).

Though the etiology of the mango malformation is understood to some extent little is known about cellular and subcellular details of malformed parts and the structure and functional interrelationship between the plant tissues and disease causing organism. Hence, the present study was initiated to investigate into histological, histochemical and ultrastructural changes of malformed organs of mango with a focus on the following objectives:

1.4 Objectives :

1) Morphological changes associated with the floral and vegetative parts
2) Anatomical changes in shoots following vegetative and floral malformation.
3) Structural changes associated with anthers and ovary of malformed flowers.
4) Association of fungus with terminal buds
5) Histochemical localisation of phenolics, starch, proteins, lipids and enzymes like succinic dehydrogenase and peroxidase in shoots and panicles of vegetative and floral malformed twigs.
6) Ultrastructural cytology of anthers and ovary from flowers of malformed panicle.

The above objectives have been studied comparing between healthy and malformed organs and tissues.