DISCUSSION

The mango, an important of food crop, is adversely affected by malformation disease which is dispersed worldwide. The disease appears in two distinct forms; vegetative and floral Vegetative malformation is characterised by the bunch - life appearance of shoots with many short and stunted shootlets. In floral malformation, the panicles appears stunted with many unopened flowers crowded on the thick axis.

4.0 Morphological Study :

The malformed panicles appear green and continue to bear flowers even after fruit setting is completed in normal panicles (Singh *et al.*, 1961). Similar symptoms of the disease has also been noticed in the cultivar Rajapuri of *Mangifera indica* L. The emergence of malformed and normal panicles is simultaneous but in the former further growth of panicles is very slow and blooming is delayed compared to the healthy ones. Our study indicates that the incidence of floral malformation varies from 10-50% in different orchards in and around Baroda city (the study area) is in the western part of the country. The incidence of this disease is variable in different climatic conditions of India. Earlier reports reveal that the disease incidence is most severe in north and western parts of India and less in other regions (Pandey *et al.*, 1977). It is also reported that more than 50% of trees are affected, causing heavy loses in yield, particularly in northern parts of India (Kumar and Beniwal, 1992). However, Kumar (1983) observed that the incidence of disease is of quite scarce in the southern parts of India, where the temperatures remain relatively high during major part of the year.

Among malformed panicles usually the blooming of bisexual flowers is delayed while staminate flowers bloom simultaneously with the healthy ones. The number and arrangement of sepals and petals are variable among the flower of diseased panicles. They show either tetra or hexamerous condition differing with the variety. The number of petals may reach upto seven in the flower of cultivar Rajapuri. The aestivation of petals and sepals is imbricate and quincuncial compared to the imbricate arrangement in the normal flower (Rao *et al* . 1996)

There is a distinct variation in sex expression of flowers between malformed and healthy panicles The percentage occurrence of bisexual flowers is highly reduced in

malformed panicles. However, the percentage of staminate flowers is enhanced compared to healthy except in variety Kesar where there is a slight increase in number of bisexual flowers. These observations coincide with the earlier reports of Hifny *et al.*, (1978); Jagirdar *et al.*, (1966) and Kumar *et al.*, (1993). This impairment in floral sex expression might be contributed by the accumulation of zoosteroids (Pregenolone and progesterone) instead of normal phytosterols in diseased flowers of mango (Ghosal and Chakrabarti, 1988). Accumulation of high concentration of GA_3 like substances in the malformed panicles may induce maleness (Abou-Hussein *et al.*, 1975, Mishra and Dhillon, 1980). The diseased panicles are often characterized by the presence of lesions on the floral organs. These lesions on peduncle, pedicel, sepals, petals, glandular disc, anthers and style (stylar transmitting tissue) may be caused by the degradation of cells by the pathogen. The increase in length of sepals, petals, anthers and size of ovary glandular disc in flowers of malformed panicle might be due to harmonal imbalance in floral buds following infection. Thus the dimensional changes in essential and non-essential organs mentioned above could be the result of imbalance between growth promoters and growth inhibitors which are inter-linked in some way producing malformed panicles (Pandey *et al.*, 1974).

Staminate flowers mature and dehisce pollen at the time when bisexual flowers are not yet bloom in the malformed panicles. Thus the pollen dehisced from flowers of malformed panicles go in vain, disturbing the normal fertilization process. However, self fertilization in bisexual flowers of malformed panicles could be responsible for the production of pea-sized fruits from the available fertile pollen. It has been suggested that delay in blooming of flowers and arrest of the fruit development at pea stage in the malformed panicles would be a result of iso-penetenyladine (produced by the pathogen) and its occurrence only in the infected tissues which can also metabolize trans-zeatine resulting in reduced production of dihydrozeatin (DHZ) like compounds that are necessary for the normal flower and fruit development (Kumar and Beniwal, 1992). Malformed panicles produced during late bearing season (February-March) produce leaf-lets resembling normal leaves. These leaflets are known to be produced due to influx of mangiferin in the shoots bearing such panicles (Chakrabarti and Ghosal, 1985).

Eriophyid mites mainly *Aceria mangifera* are known as vectors for spreading malformation disease in mango (Hassan, 1944; Narsimhan, 1954; Puttarudriah and Channa Basavanna, 1961; Summanvar, 1967). In our study except for few thrisps, eriophyid mites or other groups of Acarina are not found in the panicles. However, these mites are found associated with vegetative malformed twigs and vegetative buds. Besides eriophid mites (*Aceria mangifera*), other members of phytoseiid, cryptostigmatids, *Chelatogenes* sp. and few other mites are also found associated with shoots and young vegetative buds. Phytoseiid mites are associated with phytophagous activity and *Typlodromus husaini* is known to make puntures close to each other on bases of leaves for feeding (Khan, 1970). Similar feeding injuries and lesions on the bud scales and leaf bases are noticed in the present study. *Aceria mangifera* isolated from vegetative malformed buds and shoots, are known to cause partial or complete death of mango bud (Khan, 1970). In the present study, however, completely perished buds are not encountered but necrotic portions on the bud scales or shoots are prominant. Further *Chelatogenes* sp. is predacious upon mites and could be useful in controlling the main vectors (mites) of phytoseiid category which in the present study are evidently carriers of the pathogen (*Fusarium moniliforme*). Moreover many members of cryptostigmatids though are known to responsible for degradation or bring about scenescence of the plant organs. These might also be responsible for lodging the inoculum of the pathogen in the scenescing tissues which in the floral season regain the vigour from the meristems and give rise to malformed panicles. Macro and microconidia of *F moniliforme* attached to the body of phytoseiid and cryptostigmatid mites suggest that these are the effective vectors of the pathogen and spread the disease feasibly to the healthy branches of the same tree and healthy trees in the surrounding area.

4.1 Histological and Histochemical Study :

4.1.0 Terminal buds · Several studies pertaining to mango malformation have been carried out in various aspects in cultivars Hindy and Zebda (Raafat *et al.*, 1995b); Dashehari, Langra, Himsagar and Gilas (Kumar and Chakrabarti, 1997); Banasari Langra (Ghosal *et al.*, 1978), Amrapali and Banasari Langra (Ghosal and Chakrabarti, 1988, Chakrabarti *et al* , 1993). Further, nearly thirty different cultivars of mango not cited above were also studied (Kumar and Beniwal, 1992) with reference to disease

resistance and susceptibility of their germplasms. As Rajapuri cultivar has not been studied earlier and it shows relatively high disease incidence, it has been studied in detail.

Humid and cold climatic conditions prevailing in February-March resulted in more malformed inflorescences (Majumdar and Sinha 1972). The colonization of the pathogen close to the bud meristem noticed in the present investigation in January-March and November is in agreement with previous findings.

Phenolic catechins and proteins are known to polymerise and form a physical barrier (melanin) to the invading pathogen (Vansumere *et al.*, 1975). In the present study the gum-resin in duct lumen of pith shoot during severely infected months stained positive for catechin derivatives and proteins. This occlusion might polymerise to form melanin and perform a similar function against the pathogen. Howell *et al.*, (1976) also inferred that catechin derivatives could inhibit mycelial growth in the host. Similarly catechin derivatives localised in tissues like leaf primordia and the meristematic cells in the severely infected months viz. March-April and November in mango may act as inhibitors of mycelial growth

Despite the induction of physical barrier and mycelial growth inhibitors by the host, it is evident that there is profuse growth of the pathogen in the apices (March-April) and the degradation of meristematic cells which are the result of virulent pathogen that passifies the resistant factors. Mangiferin is known to accumulate in the differentiating buds which would develop into abnormal panicles (Chakrabarti and Sharma, 1993). Further mangiferin is known to oxidise itself into polymeric quinones that do not have perceptable anti-fusarium activity (Ghosal *et al.*, 1979). Thus in the present study mangiferin accumulated in the floral buds during March (late floral season) may lose its anti-fusarial activity but responsible for the development of intermingled leaf-lets in the panicles. Vegetative growth promoting activity of mangiferin is already well documented (Ghosal *et al.*, 1978 and 1979). Infection by the parasites alters the metabolism of tissues, so that respiratory substrates move towards the site of infection from elsewhere in the plant. Alternatively, more of these substrates are produced by cells at the infection site As a result more substrates are available for respiration and release of energy by

diseased cells (Wood, 1967). Similarly, gradual increase of starch accumulation following the disease severity in the shoots of mango from September to February indicates the mobilization of starch from leaves of the terminal twigs.

Further, sporulation of the fungus utilises the starch accumulated in the leaves (Mac Donald and Strobel, 1970). Although sporulation of the pathogen is not noticed in the mango shoot buds but profuse growth of mycelium in form of mat in March-April months could be responsible for starch depletion.

Higher disease incidence and starch depletion in these two months is followed by total protein augmentation. This might be in response to the more demand of respiratory substrates (in the shoots) in the absence of starch. These proteins may combine with phenolic catechins and polymerise to polymeric quinones and act as physical barrier against the pathogen attack.

Flavonoids with a catechol-B-ring have a sparing effect on IAA by inhibiting oxidase activity and hence theoretically they have a stimulating effect on plant growth (Stenlid, 1968 and 1976) Growth malfunctions as a result of pathogen interference are numerous. Plants become dwarf or giant, plant parts assume new and exotic shapes, buds that ordinarily are held in check are suddenly released, individual cells escape from organism control and grow as cancerous mass (Wood, 1967). In such diseases, excess or imbalance in plant growth substances have often been found and the pathogen involved has been shown to be capable of producing growth regulating substances *in vitro* Thus it is tempting to speculate that a direct contribution of pathogen to growth substance imbalance in the host cell which is very important in redirection of the development The above mentioned symptoms seem to be fit to those of vegetative malformation (Kumar and Beniwal, 1992). Thus catechols either combine with proteins to form melanin and act as phytoalexin or act as a harmone. It is likely that the latter function could be attributed to catechol derivatives which would result in development of bunchy top symptoms as opined by Kumar and Beniwal (1992).

Decline in fungal density during summer is due to the rise in temperature (39.7%), low humid (47%) weather. On the other hand, favourable climatic conditions

during monsoon i.e. high humidity (86%) and fall in temperature (August 32.4°C) may play an additive role in enhanced growth of fungus. These results are corroborative with earlier studies (Kumar *et al.*, 1993).

It is noticed that increased lumen diameter of gum-resin duct of pith in the terminal buds goes hand in hand with high disease incidence. In February, the exponential rise in lumen diameter (LD) might compensate the lower values of percentage area (PA) and frequency (FR) of gum-resin ducts in accounting for more accumulation of phytoalexin. Sinclair *et al.*, (1972) assigned the susceptibility and resistance of dutch elm trees based on decrease and increase in vessel lumen diameter respectively after inoculating the fungus, *Cerotocystis ulmi*. It is noteworthy that mango apices with higher LD, PA and FR values of resin ducts show positive correlation to susceptibility to malformation and those with relatively lower values are resistant. However, these findings are justifiable in view of the key role played by climatic conditions

4.1.1 Floral malformation :

Fungus (*Fusarium moniliforme*) occurs intra and intercellularly in the infected tissues (vegetative and floral), particularly in the cortex and has ability to penetrate the host mechanically (Ibrahim *et al.*, 1975). It forms globose structures, similar to chlamydospores, particularly in the cortex. Further, histopathological studies revealed the increase in cell size and their number in the diseased tissues. The number of cortical and pith cells increased tremendously in the main axis and to a lesser extent in the secondary axis of malformed panicles. Fungal mycelium has not been found in any section of main and secondary axes (Kumar and Beniwal, 1992). Recently, Raafat *et al.*, (1995b) also could not observe inter or intracellular fungal or bacterial growth in different tissues of stem of floral malformation. Moreover, other studies reveal that pistils in malformed hermaphrodite flowers are usually non-functional (Mallik, 1963) and pollen exhibit poor viability (Shawky *et al.*, 1980). Histological studies in the present investigation reveals the rare association of fungus with vegetative malformed tissues. Intra or intercellular mycelium are rarely noticed in the primary and secondary axes of malformed panicles. Of all the organs of malformed panicles, flowers are more susceptible to fungal invasion. Both the light and electron microscopic observations

reveal the intra or intercellular association of hyphae in the anthers and ovular tissues. The increase in size of cells in individual organs results the enlargement of flowers in malformed panicles.

The localisation of peroxidase enzyme at the infection sites of the peduncle and pedicel tissues along with catechol derivatives might result in imbalance of harmones like auxin supplied to the flowers. This observation is in agreement with suggestion made by Stenlid (1968 and 1976) that the effect of phenolic compounds on Indole acetic acid oxidase, a peroxidase type enzyme which is capable of axin destruction. It is well established that, *in vitro*, flavonoids with catechol B-ring have a sparing effect on IAA by inhibiting IAA oxidase activity and hence theoretically they have a stimulating effect on plant growth. Thus there could be an imbalance of auxin production between inhibitors and precursors of IAA production. Thus leading to the malformation syndrome.

Harmonal imbalance has been found to occur in bunchy top and malformed inflorescence tissues. Levels of auxins and gibberellins were found to be decreased in the diseased tissues as a result of infection, while those of inhibitors and IAA oxidase were much higher (Kumar and Beniwal, 1992). Hormonal imbalance between growth promoters and growth inhibitors is also suggested in the development of malformed panicles (Pandey *et al.*, 1974).

Fusarium moniliforme J. Sheld var. subglutinans is now known to be the causal agent of the mango malformation (Summanvar *et al.*, 1966; Varma *et al.*, 1972 and 1974; Manicom, 1989). The present study reveals the entry of pathogen into host terminal buds occur as early as in November i.e. few months before floral bud inception. Accordingly the close association of *Fusarium moniliforme* with youngest unopen buds is evident. The infection sites of the host at the basal portion of anthers, the tip of anthers filament, anther wall and the stylar transmitting tissue are accumulated with phenolic contents. Such accumulation is a result of host - species hypersensitive reaction producing antagonistic substances (e.g. phytoalexins) to delimit the growth of the pathogen (Ghosal and Chakrabarti, 1988)

The phenolic contents accumulated at the infection sites may represent mangiferin, a phenolic metabolite, which accumulates at high concentrations in the malformed shoots of *M. indica*. Mangiferin is known to inhibit the ingress of *Fusarium moniliforme* into the host and also immobilize itself from the site of infection (Chakrabarti *et al* , 1990) Further the thick cell wall of the anther cell wall might certainly offer itself a physical barrier against the attacking pathogen. But the thick mycelial mat (instead of single filament) of *Fusarium moniliforme* perhaps would produce sufficient concentration of enzymes which would dissolve the anther walls at the basal groove. Thus the fungus seems to enter into anther locule from the anatomically weak barrier of the anther i.e. base of the anther lobes where the filament is attached.

Moreover, the phenolic compounds accumulated in the malformed essential organs might be insufficient to combat the invading pathogen and the necrotic cells in these organs enhance the immobilization to developing organs becomes disrupted and affects the normal growth of the malformed panicles (Chakrabarti *et al.*, 1990). At light microscope level the anthers, the style seems to be anatomically weak barriers for the attacking pathogen. However, ultrathin sections under electron microscope reveal that at the basal groove of the anther, the anther wall is quite thick and grooved but hyphal fragments are also noticed in close association. The stigma of mango is also known to possess uncommon cuticle which is thick walled (Philip *et al.*, 1987). Thus to combat such thick cuticle which forms a strong physical barrier for the fungus to attack and hence the fungus forms a thick mat which would enable to dissolve the host cell walls. The basal groove of the anthers and the stylar transmitting tissue appear to be the entry zones of the pathogen into the essential organs of *M. indica*. On the other hand, the fungus can easily derive nutrients from the transmitting tissue which otherwise allows rapid growth of pollen tubes.

Invasion of the pathogen into essential organs and its growth in them is not reported. The mode of infection of *F. moniliforme* in panicles of *M. indica* via terminal buds, young floral buds, anthers and ovary as suggested in the present study gives a possible evidence of epidemiology and supports etiology of the mango malformation disease as fungal. The entry of *F. moniliforme* directly into the host crossing the physical and chemical barriers such as anther and ovary wall and the phenolic accumulation

respectively and gaining intimate association with the pollen and pistil suggests the virulent activity of the fungus. Such virulent nature of this strain of fungus is attributed to the toxic substances like carotenoid entities (Violaxanthin and Zeaxanthin) produced by it (Ghosal *et al.*, 1979).

Similarly the thick mat of mycelium in the anthers as well as ovary might release such toxic substances which result in non-functional or abortive nature of pollen and pistil in the affected flowers. The close and direct association of fungus with male and female reproductive organs of flowers might lead to the development of non-viable gametes, thus disturbing the fertilization process whereby fruit setting is hindered in malformed panicles. The localization of peroxidase at the infection sites i.e., basal groove in the anther and the stylar transmitting tissue and the ovule cells might aid in biosynthesis of cell wall components including lignin, suberin and cross-linked extension (Grisebach, 1981; Greppin *et al.*, 1986. Peroxidase localised in the cell walls of anther and stylar transmitting tissue in the flowers of malformed panicles may induce further cell wall material which would form a physical barrier for the pathogen.

At the initial stages of infection the accumulation of starch increment is noticed at infection sites mainly i e , anther filament and pollen grains. There after as infection severity increases there is a decline in accumulation of starch in the peduncle and pedicel tissues of malformed panicles which are a source of starch reserves for anther filament or the pollen grains. Thus it appears the invading thick mat of *Fusarium moniliforme* utilises the starch at these infection sites which is reflected in the source tissues of the host as infection severity increases. This observation is in agreement with Mac Donald and Strobel (1970) that sporulation of the fungus in the host tissues utilises the starch. Epithelial cells of gum ducts and the infection sites of anthers (basal groove) accumulate more proteins which might combine with phenolic contents and oxidise to polymeric quinones that acts as a physical barrier and as a phytoalexin (Ghosal and Chakrabarti, 1988).

The intense reaction of succinic dehydrogenase (SDH) and higher amounts of proteins in epidermal, cortical and vascular parenchyma of diseased peduncle and pedicel indicates high energy utilisation. Intense SDH activity in the loci of traumatic duct

development indicates as elevated rate of metobolism in such cells (Subramaniam, 1981) Similarly in the present investigation the SDH activity in the peduncle and pedicel indicates high rate of metabolism as a result more of total proteins are localised due to infection by *F. moniliforme* Interestingly the anthers epidermal cells showing faint reaction indicates the reduced metabolic rate in anthers as a result of colonization of the pathogen.

Lipid containing components appear dark blue to black while cuticle and suberized walls are black when stained with sudan black 'B' (O'Brien and Mc Cully, 1981). The cuticle stained black with sudan black 'B' and the cortical cells along with vascular parenchyma localised both lipids and phenolic contents. It is correlated that lipids and phenols may be coupled with peroxidase through aldehyde coupling in developing suberic walls of endodermis in plants (Van Fleet,1970). Thus the formation of suberin in the cuticle might act as a physical as well as chemical barrier for the attacking pathogen and would delimit its proliferation into the host tissues. The similar distribution (localisation) of total lipids in both malformed and healthy panicle tissues like the vascular parenchyma but reduced accumulation in healthy cuticle and paratracheal parenchyma compared to those of malformed panicles is in agreement with the above suggestion.

4.1.2 Vegetative malformation :

As stated earlier the pathogen invasion into the mango plant starts at a very stages of shoot buds and evident pathogenic interaction with the vegetative buds of March-April, August-November/ December either in the form of fusarial mat and insect remains. Further, mites prominently found associated with vegetative malformed shoots might essentially produce vegetative malformation symptoms, although the pathogen is not endophytic in these tissues.

The development of the vegetative malformation symptoms of the twigs could be attributed to the production of mangiferin by *F. moniliforme* in the inoculated shoot tips (Kumar and Chakrabarti, 1992; Ghosal *et al.*, 1979). The culture fluid of the fungus is known to cause complete abscission of the tender mango leaves when the shoots assumed the shape of a Witche's broom (Ghosal *et al.*, 1979), which is a typical vegetative malformation symptom Moreover it is reported that accumulation of IAA in

the healthy shoots brings about a new flush, but in malformed shoots accumulation of mangiferin and IAA allow development of new shootlets throughout the year (Chakrabarti *et al.*, 1990). Recurrent production of vegetative malformed shoots in the present investigation corroborates with earlier studies of Chakrabarti *et al.*, (1990).

The association of the mites with vegetative malformed twigs in the present study strongly supports the observations that eriophid mites can induce vegetative malformation symptoms (Puttarudraiah and Channa Basavanna, 1961). Thus the fusarial toxins through the feeding activities of the mites may result in production of vegetative malformation symptoms. Contrastingly the mites noted carrying the fusarial spores could not successfully invade the malformed shoots. This may be a consequence of physical and chemical barriers offered by the host. The physical barriers may include extensively thick cuticle of the shoots and leaves. The key role of chemical barrier would be attributed to increase phenolic contents which might represent mangiferin. This mangiferin translocated into the malformed shoots after its symthesis in tender leaves is known to offer resistance to the fungal attack in mango twigs (Ghosal *et al.*, 1978).

Some types of plant secondary metabolites offer effective resistant mechanism against both fungal infection and insect herbivory. The hypersensitive response (including lignification) in the host mainly falls under three classes of induced chemicals viz. phytoalexins, protienase inhibitors and Lipoxygenase (Hatcher, 1995). Similarly the presence of thick cuticle of malformed shoots and leaves and a layer of relatively thick sclerides in the cortical cells of shoots might be due to a hypersensitive reaction inducing lignification in the host (shoots) as result of insects (mites) herbivory. The accumulation of phenolic contents in ray cells of malformed shoots may be result of phytoalexin response. The lumen diameter of vessels become reduced in both malformed shoots as well as leaves. Similar observations along with decrease in the number of vascular elements has been reported by Raafat *et al.*, 1995a. However, in the present study the frequency of these elements in shoots and leaves of mature trees is found to be increased. Harmonal imbalance may lead to the development of bunchy top and malformed inflorescences. The levels of auxins and gibberellins are found to be decreased in the diseased tissues as a result of infection (Kumar and Beniwal, 1992). The above mentioned histological disorders are accompanied by lower gibberellins and

higher cytokinin activities in the growth of bunchy-top shoots (Raafat *et al.*, 1995a). The comparatively longer xylem fibres in malformed shoots also be due to the same reason as suggested by Raafat *et al.*, (1995a). Thus these histological disorders could correspond to the lowered levels of gibberellins, auxins and cytokinins collectively.

The reduced accumulation of starch and enhanced deposition of lipids and proteins in the resin ducts might be due to the conversion of the former into the latter in order to combat the feeding of mites in the shoots or leaves. The resin secreations form a protective layer of hardened resin on wounds in mango. An enzyme laccase is responsible for such formation of protective layer (Joel *et al.*, 1978). Further, resin of the mango shoot consists mainly of terpenes, phenols, proteins, carbohydrates cited by many authors along with lipophilic substances (Joel and Fahn, 1980). Phenolic contents in the ray cells of the shoots might be induced due to demand for protection of vascular tissues. Thus presence of lipids and proteins in the malformed shoot ducts might form a protective layer and phenolic content provide a phytoalexin effect against feeding activity of mites. Simple phenolic acids, precursors of isoflavonoid phytoalexins are found feeding deterrents in *Epilachna varivestis* (Fisher *et al.*, 1990). The precursors of lignin formation are generally thought to be the cinnamyl alcohols (Mader and Fussl, 1982) The polymerization of these phenols is catalysed by peroxidase in the presence of H_2O_2 (Elstner and Haupel, 1976; Gross *et al.*, 1977). Similarly the localisation of peroxidase in the cortex of malformed shoots and the vascular tissues of malformed leaves suggests that the enzyme might play a key role in lignification. The relatively more number of lignified sclerid layers in cortex of malformed shoots and the relatively thick walled (lignified) vessels in malformed leaves could be attributed to the lignification role of peroxidase enzyme. Feeding by the mites induces the formation of small necrotic lesions, pathogenesis related proteins, Chitinase and β-1-3-glucanase along with peroxidase (Bronner *et al.*, 1991a,b). Thus lignification may make cell walls more resistant to mechanical penetrations of the mites in mango tissues. Further peroxidase localised in either epidermal layers of malformed leaves also might be related to the lignification of the cell walls and function of the thick cuticle to offer resistance to the invading mites. The more activity of succinic dehydrogenase in malformed shoots and leaves might be correlated with the enhancement of respiration as the enzyme is known as mitochondrial enzyme Localised accumulation of relatively more SDH in epidermal,

cortical cells and vascular tissues of vegetatively malformed shoots and leaves along with enhanced peroxidase activity reflects greater metabolic activity in these tissues as a result of mite feeding. The relatively more SDH and peroxidase in the vascular tissues of malformed shoots seems to be closely associated with secretion of respiratory substrates (lipids and proteins). While peroxidase is associated with cell wall and lignin and suberin deposition. As opined earlier, that enhanced peroxidase activity could induce lignin and suberic walls act as a physico-chemical barrier against invading pathogen in floral malformed tissues. Similar correlation can be inferred to the vegetative malformed shoots keeping in view that either insect feeding or fungal attack (biological stress) induced common responses in the host (Hatcher, 1995). Further increased substrates (lipids and proteins) in diseased shoots and leaves is reflected by showing high SDH activity in these organs. Accumulation of substrates is a consequence of augmented synthesis by diseased cells and it is this synthesis which is the primary cause of the rise in respiration (Wood, 1967). High respiration in diseased tissues of mango shoots and leaves hence indicates a vital role of mitochondria in supplying energy to encounter the ingressing biological stress resulted by mite (insect) feeding.

4.2 Ultrastructural study :

While much work has been carried out on the physiological and biochemical aspects, there is little information on the ultrastructural details of malformation disease. Examination of thin sections of petals, leaf midribs, and fine roots from malformed mango trees under electromicroscope (EM) revealed that phloem and xylem tissues were apparently free from any pathogen (Kistah *et al.*, 1985).

Our study using SEM indicates close association of the pathogen (*F. moniliforme*) with sepals, and ovary from mature flowers. The pathogen is often noticed at basal portion of the anther lobe where filament is attached and between the two anther lobes. These results support the foregoing light microscopic observations on the association of *Fusarium* with the essential organs of malformed flowers. Absence of distinct grooves on the surface of diseased anthers might be attributed to the enzymatic degradation of the cell walls by pathogen (in the thick mat form). Few disorganised pollen in the anther locule also could be a result of such enzymatic activity.

Earlier reports on sweet corn seed and plants infected with *F. moniliforme* and *F. oxysporum* suggest the production of cell wall degrading enzymes by the fungus (Lawrence *et al.*, 1981)

The fragments of *F. moniliforme* close to anther in the ultrathin sections of young buds under TEM reveal changes in the host cells such as electron dense cytoplasm and smooth vesciculate ER in the tapetal cells and electron dense cytoplasm in the cells of mature anther and ovary However, other cellular organelles are not distinctly seen in the diseased tissues. This may be due to masking of the organelles following the accumulation of electron dense contents in the cells. These electron dense substances could be a result of hypersensitive reaction of the host and might resemble the phenolic contents which accumulate at the infection sites observed at light microscope level.

The association of bicelled or three celled microconidia like structures on the surface of ovule, the inter and intracellular hyphae in the anther and ovary confirms the intimate association of the pathogen with the essential organs of flowers in malformed panicles.

Intracellular hyphae, highly granulated, electron dense cytoplasm in host cells and highly lipophilic and electron dense hyphae are characterised by dead maize tissue in response to endophytic colonization by *Fusarium moniliforme* (Bacon and Hinton, 1996). Similarly, in the present study although the cytoplasm of anther cells or the ovule cells are not granular but the cytoplasm invariably was dense in both hyphal fragments and host cells. The cellular changes noticed at ultrastructural level in both the anther and ovary may be brought about by the mycotoxins produced by *Fusarium moniliforme*.