CHAPTER- II

REVIEW OF LITERATURE
Mango (Mangifera indica L.), a native to the Indo-Burma region, is an important fruit crop of the tropical and subtropical areas of the world (Gangolly et al., 1957; Singh, 1960; Majumdar and Sharma, 1990, Singh, 1990; Crane and Campbell, 1994). In India, it grows from the sea level to an altitude 1500 m i.e., from Cape of Camorin to Himalayas. It is grown commercially in areas upto 600m altitudes where temperature rarely goes below 0 °C (Negi, 2000). Mango thrives best at an annual mean temperature of 26.7 °C (Woodrow, 1910). There are about 1500 varieties of mango in the world of which about 1200 are found in India (Pandey, 1998). Among the known diseases of mango, mango malformation is a serious threat/malady to mango cultivation in India and other mango growing countries causing a great concern and heavy economic losses to orchards of mango (Mallik, 1963; Varma et al., 1974; Hifny et al., 1978). There is a lot of confusion in the literature about the etiology of the malady. Inspite of several decades of incessant research since its recognition in 1891, the etiology of this "century-old" disease has not yet been discovered due to paucity of information and thus no effective control measure is known (Ram, 1991; Kumar
and Beniwal, 1992; Kumar et al., 1993; Singh and Dhillon, 1993; Srivastava, 1997; Ram and Yadav, 1999; Pant, 2000; Ram 2001; Bains and Pant, 2003; Kvas et al., 2008; Lopez et al., 2005; Marasas et al., 2006; Shah et al., 2009; Sirohi et al., 2009).

**Distribution of mango malformation:**

Mango malformation on age-old disease, was reported for the first time by Maries, a progressive farmer of Darbhanga district in Bihar (India) in 1891 (Watt, 1891). Later, this disease was recorded from Bombay (Burns, 1910), Uttar Pradesh (Singh and Chakrabarti, 1935), Punjab (Singh et al., 1940; Sattar, 1946, Iqubal et al., 2004, Krishna et al., 2009), Maharashtra (Narasimhan, 1954), Bihar (Mallik, 1961) and West Bengal (Chakrabarti and Kumar, 2002). Now it is widely distributed all over India, But its incidence is more in the north-west than in the north-east and south India (Malik, 1963). The incidence of malformation is sporadic in southern parts (Summanwar, 1967). However, a few cases have also been reported in Southern India (Kulkarni, 1979). The disease has also been recorded from other mango-growing countries like Middle East (Hassan, 1944; Sayed, 1946; Attiah, 1955), Pakistan (Khan and Khan 1960; Khan et al., 1963), South Africa (Schwartz, 1968), Brazil (Flechtmann et al., 1970) Sudan (Minessy et al., 1971), Central America, Mexico and U.S.A. (Malo and Mcmillan, 1972), Egypt (Hifny et al., 1978), Cuba (Padron, 1983), Malaysia (Lim and Khoo, 1985), Australia (Peterson, 1986), Israel (Sourial et al., 1986), U.A.E. (Burhan, 1991) and Bangladesh
Natural occurrence:

Malik (1963) reported that the disease was endemic as a tree once infected never recovers. Prasad et al., (1965) found greater incidence of disease in the seedlings than the grafted trees. Some of the trees showed infection year after year but the adjacent trees belonging to the same or the different varieties did not show infection (Schlosser, 1971). Jagirdar and Jafri (1966) observed that seeding mangoes were the first to exhibit malformation and the neighbouring grafted stocks were affected later. They did not find the disease in very young bearing trees. Majumdar and Sinha (1972) recorded the seasonal variation of a high degree in the occurrence of disease depending upon the climatic conditions. This disease also varied according to the age of tree as reported by Puttarudriah and Channa Basavanna (1961). They found high degree of malformation in case of younger plants than older ones. Heavy infection (91%) in 4-8 year old plants was observed as compared to 9.6% infection in older trees (Singh et al., 1961). However, Putto et al., (1975) reported less incidence in young trees (2.8%) and more incidence in 100 years old trees (100%). The flower buds emerging early in the seasons had heavy infection of malformation than those emerging latter (Singh et al., 1977), Singh et al., (1992; Haggag, 2010) found that younger plants were more affected by the disease than older trees.
Extent of damage/economic Importance of the disease:

Malformation causes heavy damage to trees as the inflorescence fails to produce fruits. The extent of damage varies from 50 to 60 percent in some cases but in severe cases the loss may be 100 percent (Summanwar, 1967). Prasad et al., (1965) reported that the intensity of disease were higher in western districts of Uttar Pradesh than eastern. Varieties in Lucknow has 30 to 60 percent incidence of the floral malformation, whereas, in Saharanpur 47.03 percent panicles were affected (Nirvan, 1953). It was found that 22% trees were affected with the malformation and 0.1% to 100% panicles were affected on a tree (Singh and Chakrabarty, 1935). Verma et al., (1969) recorded highest incidence of malformation from Punjab, Delhi and Western U.P., Where almost 50% plants were affected, whereas in Eastern U.P., Maharastra, Andhra Pradesh and Tamil Nadu, the incidence was hardly 10% while in Kerela and Kanyakumari, this malady was not found at all. Varma et al., (1971) reported that 50 to 70% trees were attacked in north-western, north-eastern and north-southern India. Kulkarni (1979) found that there was an increase in incidence of malformation in Andhra Pradesh. Kumar (1983) observed the losses as high as 86% in U.P. terrain. More than 50% of the trees were found to be affected by floral malformation, particularly in North India (anon, 1983). Chib et al., (1984) recorded 21-45% floral malformation in Jammu. According to Majumdar and Sharma (1990) the region beyond Hyderabad was free from this malady.
Symptoms:

External Symptoms: Depending on the plant parts affected, mango malformation has been broadly classified as vegetative and floral malformation (Kumar and Beniwal, 1987). Both vegetative and floral malformation have been assumed to be symptoms of the same disease, since hypertrophy of tissues is involved in both cases, and vegetative malformation appears at times on trees bearing malformed inflorescences (Tripathi, 1954; Schlosser, 1971; Kumar and Beniwal, 1987). An experimental proof was achieved by grafting diseased scion on to healthy rootstocks. The diseased scion that would have produced a malformed inflorescence in on-year (flowering year) produced symptoms typical of vegetative malformation, clearly demonstrating that vegetative and floral malformation are symptoms of the same disease (Kumar and Beniwal, 1987; Gamliel 2009a, 2009b; Ploetz, 2006; Joshi et al., 2014; Singh et al., 2015; Ansari et al., 2013; Asari et al., 2015; Singh et al., 2014; Lima et al., 2012).

Vegetative malformation:

Vegetative malformation, first described in 1953 (Nirvan, 1953), is more pronounced on young seedlings but may also appear on mature trees. More than one vegetative bud at one point gets activated, and numerous small shootlets arise bearing very small scale leaves at the short internodes. These leaves get so crowded that shootlets and their branches are not distinguishable and the whole mass of rudimentary leaves gives a
bunch-like appearance. Such symptoms when present at the apex of seedling are referred to as bunchy-top (BT) stage. Sometimes, thick shootlets arise from the swollen axillary buds which ultimately have secondary branches that elongate further and bear small rudimentary leaves at the internodes. Collectively, the whole structure gives a Witches broom-like appearance and also called as "bunchy top stage" (Nirvan, 1953; Tripathi, 1954; Flechtmann et al., 1970; Abou-Hussein et al., 1975; Bhatnagar and Beniwal, 1977; Kanwar and Nijjar, 1979; Kumar, 1983; Singh and Dhillon, 1993). These bunches have been recorded to be of varying sizes (Paracer and Chahal, 1963). The seedlings affected at an early stage remain stunted and may dry up while those getting infected later resume normal growth above the malformed areas (Singh et al., 1961; Kumar and Beniwal, 1992). The malformed branches dry up after a few months and remain attached to the shoots as dry masses but the branches or twigs continue to grow in spite of the disease. Vegetative malformation results in reduced or no flowering or fruiting (Nirvan, 1953; Singh et al., 1961; Malik, 1963; Paracer and Chahal, 1963).

The malformed seedlings have shallower root system with few tertiary roots and tap roots often have necrotic appearance (Varma et al., 1969; Schlosser, 1971a; Singh et al., 1992).

**Floral malformation:**

Symptoms of floral malformation appear with the emergence of
inflorescences. The primary, secondary and tertiary rachises are short, thickened and are much enlarged or hypertrophied. Such panicles are greener and heavier with increased crowded branching (Hassan, 1994). Flowers of malformed panicles are bigger than normal. Frequently, but not always, the sex ratio (hermaphrodite Vs. male) of flowers in a malformed inflorescence decreases (Singh et al., 1961; Khan and Khan, 1962; Schlosser, 1971; Hifny et al., 1978; Kumar 1983; Rana, 1992). Pistils in malformed hermaphrodite flowers are usually non-functional (Mallik, 1963) and pollen exhibits poor viability (Shawky et al., 1980). In most acute cases, the flower heads are very compact, green and sturdy which bend with their own weight (Mallik, 1963). Malformed flowers show high degree of embryo degeneration (Singh and Dhillon, 1990 C). The malformed panicles amy sometimes bear a few fruits but they do not grow beyond the pea stage (Jawanda, 1963; Mallik, 1963; Hifny et al., 1978). Off-season fruiting is also veru common in malformed panicles (Jawanda, 1963). Both healthy and malformed flowers appear on the same panicle or on the same shoot (Pandey et al., 1977). Many a time both healthy and malformed panicles appear at a single growing point and the healthy ones bear normal fruits (Kumar et al., 1992).

The malformed panicles are classified into two groups, namely, spreading (loose) type and compact type. The spreading type of malformed panicles bear lateral axes ending in an aggregate mass of flower buds without bracts, whereas, compact type of malformation can be characterized by mass of clustered green bracteated flower buds (Sharma, 1953). Subsequently,
the compact type of malformed panicles were further categorized into light, medium and heavy types (Varma et al., 1969).

**Internal symptoms or historical changes:**

Very little information is available on anatomical changes due to malformation. Internal symptoms include the development of hyperplastic and hypertrophied cells in malformed vegetative and floral parts (Narsimhan, 1954; Ibrahim and Foad, 1981; Kumar, 1983). Vegetative malformation does not differ much in the internal structures whether born on seedlings or trees. Deformation is confined to poorly developed leaves and vascular tissues, and absence of the palisade (Ram, 1991).

Deformation in floral malformation is confined to lower buds only. The surface of the buds becomes warty, puckered and contains dense growth of epidermal hairs. The cortex and stele of panicles may develop hyperplastic cells (Narsimhan, 1954). However, the pith cells of malformed panicles are deformed and packed with starch (Mallik, 1963). There is no deformation in pith cells of vegetative shoots and pedicels of malformed (Prasad et al., 1965). The pith cells of vegetative shoots have large number of spaces lined up with some small elliptical cells and contain large amount of brownish fluid (Ram and Yadav, 1999). Similar fluid was also found in the hypodermal cells of shoots and panicles without any difference in their conducting tissues. The affected branch contains leaves having poorly developed tissues. Marked anatomical differences have been reported in the rachis of healthy
and malformed panicles. The Cortex, Xylem, Vessels and Pith of the affected panicles contain 50% less number of cells per unit area than the healthy ones. The thickness of rachis and smaller number of cells per unit area of the rachises of malformed panicles are due to enlarged cell size (Pandey et al., 1977).

Varma et al., (1974) were able to locate the fungal hyphae in the Cortex (Chakrabarti and Ghosal, 1989). Phloem and parenchymatous pith cells (Bhatnagar and Beniwal, 1977) of malformed shoots and panicles. The hyphae were mostly intercellular but occasionally formed intracellular agglomerates. Ibrahim et al., (1975) observed discoloration of Xylem tissues and also the presence of fungus in the cortex cells. Hyphae of the fungus were observed in the inter and intracellular spaces. The fungus formed globose structure similarly to chlamydospores in the cortex after artificial inoculation with spore suspension in naturally infected tissues. The formation of globose structures were observed within cells particularly in pith cells. However, some workers did not find microorganism in the healthy and malformed sections of vegetative shoots (Prasad et al., 1965) and malformed inflorescences (El-Barkauki et al., 1978; Ibrahim and Foad, 1981).

Ultrastructure studies in malformed tissues of mango were done to determine the presence of mycoplasma like organisms. These studies did not indicate the involvement of any virus or MLO in the causation of the disease (Kishtah et al., 1985). Fungal mycelium has also been detected at the juncture of shoot tip and malformed inflorescences, in malformed
auxiliary buds, axes of petals and sepals on malformed buds, and in the degenerating embryos of mustard-stage malformed fruit (Kumar, 1983). Studies with scanning electron microscope in mango variety Amrapali revealed the presence of many hair-like cracks, pin-sized to large holes, disorganized cells and fungal mycelial infection at the base of the malformed bud during bud inception stages (Usha et al., 1997). Koti-Babu and Rao (1998) noticed a close association of the fungus *Fusarium moniliforme* with the floral and vegetative apices, resulting in high incidence of malformation disease due to pathogenic interaction. Deposition of starch in the pith cells of shoot was relatively more in the floral season.

**Gummosis/Resinosis:**

Gummosis is known to be a common response of many plants to wounding (Talboys, 1968), stress inducing high temperature (Wilde and Edgerton, 1975) and injury by insects and pathogens (Narsimhudu and Reddy, 1992).

Development of resin ducts in mango: A character feature of the 'Anacardiaceae' family is the presence of resin canals, which are associated in the shoot with the primary and secondary phloem. In many genera, including *Mangifera*, resin ducts are also found in the pith and develop lysigenously (Fahn and Joel, 1976).

The process of duct development in the primary mango shoot can be divided into five main stages i.e., Initiation differentiation, secretion,
darkening of the disintegrated epithelial cells and quiescence (Joel and Fahn, 1980).

Resin secretion in mango (resinosis): The stem exudate of mango contains mangiferin, a polyphenolic compound important for its pharmaceutical uses (Anon, 1983). The resin of mango shoot mainly consists of terpenes which are produced by plastids known as secretoplasts. Phenols, proteins and carbohydrates have also been detected in this resin (Corsano and Mincione, 1965, 1967 a, b; Bhatia et al., 1967; Joel and Fahn, 1980).

Plastids, ER, Golgi apparatus and mitochondria are all involved in the process of resin secretion in Anacardiaceae (Fahn and Evert, 1974). The process of resin secretion in mango shoot is mainly in association with the type of plastid termed as secretoplast and in periplastidal ER. In the next stage it is seen in Golgibodies and occasionally in association with mitochondria. The third stage is marked by filling up of the inner space of ER with secretory material, and at the end of the process of secretion the osmiophilic material is limited to the periphery of the secretoplasts occurs on the outside surface of the plastid envelope or between its membranes (Joel and Fahn, 1980).

Cultural and morphological characters of the pathogen:

The genus *Fusarium* was erected by Link (1809) for species bearing fusiform to falcate conidia, mostly with a differentiated foot cell. Earlier the disease situation was confused due to frequent misidentification of the
fungus as *F. moniliforme*. The three species and several varieties placed by Woolenweber and Reinking (1935) in section-Liseola have in recent years generally been regarded as representing a single species with one telomorph, *Gibberella fujikuroi* (Table-1). The cultural and morphological characters of *Fusarium moniliforme* Var. *subglutinans* (teloporph : *Gibberella fujikuroi* Var. *subglutinans*) have been described by many workers (Wollenweber and Reinking, 1935; Gordon, 1960; Booth, 1971; Joffe *et al*., 1973; Gerlach and Nirenberg, 1982; Windels, 1991, Summerell *et al*., 2003). Several investigators compared the identical morphology of *Fusarium moniliforme* isolates from different host plants collected from various places (Wollenweber and Reinking, 1935; Joffe, 1974; Booth, 1977; Mitra and Lele, 1981). Kuhlman (1982) described/differentiated four varieties i.e. *G. fujikuroi, F. subglutinans, F. moniliforme* and *F. intermedium*, on the basis of mating groups, variation in ascospore and perithecial size, phialide type and microconidial formation.

The mycelium is aerial, floccose, white to greyish-whiste at the tips and buff or in most isolates vinaceous purple below, becoming purple to dark purple. Microconidia begin to form 2-3 days after inoculation of a plate. They are formed initially from simple lateral phialides but are soon replaced by branched conidiophores terminating in polyphialides. Microconidia are aseptate, hyaline, oval to obclavate, 8-12x2.5-3μm (Varma *et al*., 1971; 1974. Booth, 1985). Short chains of microconidia were observed
in a culture of *F. subglutinans* (*Gibberella fujikuroi* Var. *subglutinans*). TEM showed this to be aberrant conidiogenesis, where phialides produced microconidia which in turn acted as phialides to give the impression of

Table 1: Summary of synonymy within the Liseola section

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catenate conidium production (Tiedt and Jooste, 1988). The majority of conidia and hyphal cells are uninucleate, but up to 5 nuclei have been observed (Bilai and Gorbik, 1972).

Macroconidia develop initially from simple phialides formed as termination of short lateral conidiophores but as the cultures develop they tend to aggregate into sporodochia. The phialides measure 15-22 x 2.5-3 µ. Macroconidia are thin walled, falcate. 3-5 septate, 32-35 x 3-4.5 µ (Booth, 1985).

Perithecia as in *F. moniliforme* belong to *Gibberella*, they are heterothallic and appear to be rare in nature. Perithecia will develop in wheat or rice straw cultures if suitable mating strains are introduced under suitable conditions (high humidity and 25°C temperature). Perithecia have eight narrower ascospores which measure 12-15 x 4.5-5 µ (Booth, 1985).

**Physiological studies:**

A little attention has been given on understanding the effect of different physiological factors such as culture media, temperature, relative humidity, light etc., particularly on the growth and sporulation characteristics of *Fusarium moniliforme* Var. *subglutinans* perhaps due to its fast and vigorous growth in culture. The subsequent record of information concerning the use of different synthetic and non-synthetic media for culturing different species of *Fusarium* has been ascribed (Singh and Nene, 1965; Sharma and Singh, 1973; Jaurihar and Mehta, 1979; Mitra and Lele, 1981; et
Cultivation of *Fusarium* sp. on potato-dextrose agar medium favored both mycelial growth and sporulation, while Joffe's medium and potato sucrose agar induced sporulation in most species of *Fusarium*. Potassium chloride medium was useful for identification of *Fusarium* in the 'Liseola' section (Burgess and Liddell, 1983). Best identification was carried out on carnation leaf agar media that favored sporulation rather than mycelial growth. Sporulation and pigmentation were favored by fluctuating temperature (25°C day/20°C night). Perithecial formation enhanced with the use of natural substrates (Synder and Hansen, 1947). Jaurihar and Mehta (1973) found a pH of 6.0 as most suitable for sporulation of *F. moniliforme*.

Zachariah *et al.*, (1956) found that the optimal temperature for growth of *Fusarium oxysporum* Schlecht lies between 25-30°C. The maximum and minimum temperature for growth of fungus was 37°C and 5°C, respectively. For optimal sporulation, temperature 20-25°C with 12:12 h alternating light and dark was effective. Korobeinikova (1960) reported that the fungus grew and sporulated in a wide range of pH between 2.2-9.0 with an optimum of 7.7. Chi and Hanson (1964) observed the increase in mixed...
inoculum of *Fusarium* in liquid shake culture or on media Czapek's-dox agar, V-8 juice agar, potato sucrose agar or potato dextrose agar. *Fusarium* change morphological characteristics or lose pathogenecity or sporulation when transferred repeatedly. The source of N or C:N ratio in media influences sporulation, spore size, morphology, viability and infectivity potential. Optimum sporulation on agar medium was obtained when incubated under NUV or cool white fluorescent lights (12-16 hr/day) for 2 to 3 weeks.

A medium containing oxgall and PCNB in V-8 juice agar proved most efficient for the selective isolation of *Fusarium* sp. (Singh and Nene, 1968). Sporulation in *F. moniliforme* under submerged conditions occurred on sucrose containing media (Vezina et al., 1965). It has been reported that little growth of *F. moniliforme* Sjeld. occurred at 6°C and none at >30°C (Joffe et al., 1973). Sharma and Singh (1973) described a selective isolation technique for *F. moniliforme* based on culture media with malachite green, PCNB and dicyclicin. Nirenberg (1976) reported that the optimal temperature for growth of *Fusarium* sp. was 22.5-27.5°C, the maximum 2.5-5.0°C. Sporulation was promoted by light (12:12 hr. light: dark at 20-23°C) and the production of macro-conidia was induced by black light.

Isolates of *F. moniliforme* Var. *subglutinans* from perithecia on moist wheat chaff and potato-dextrose agar (Tio et al., 1977). Growth of *F. moniliforme* Var. *subglutinans* was best on pectin, followed in decreasing order by mannitol, starch, Xylose and fructose. Maximum sporulation was obtained on mannitol, followed by starch and pectin. Among the N sources,
peptone was best for both growth and sporulation (Chattopadhyay and Nandi, 1981). In studies with three isolates of *F. moniliforme* Var. *subglutinans* from pineapple, radial growth was maximum on soluble starch, lactose and maltose, while microconidia were most numerous on sucrose, starch and fructose. The fungus grew well over a wide range of temperatures, optimum for growth and sporulation being 25 °C. Light was not required for either process. Maximum production of microconidia was at 0.1-0.2 m KCl, macroconidia were relatively few only at 0.1 M (Bolkan et al., 1982). Pimental et al., (1985) observed the effect of carbon sources and temperature on the radial growth, pigmentation, colony characters and spore production of *F. oxysporum* fsp. *lycopersici*, *F. graminearum* (G. Zeae), *F. sambucinum* and *F. solani* fsp. dianthi.

Werner (1990) investigated that maximum growth and sporulation of *F. oxysporum* isolates occurred on potato-dextrose and malt extract agar at 25-30 °C and on plain potato agar at 18, 15 and 30 °C. Medium type and temperature had the greatest effect on spore production. Osman et al., (1992) reported the optimum culture conditions for maximum growth and biomass of *F. oxysporum*. Potato-dextrose agar was the best culture media for the fungus. Optimum conditions were recorded by incubating cultures for 8 days at 30 °C introducing sucrose as the carbon source in the medium.

**Induction of malformation:**

Singh et al., (1961) reported 80% vegetative malformation and 5% floral malformation by transferring *Aceria mangiferae, Tyrophagus Castellani,*
Typhalodromus asiaticus from malformed plant to healthy seedlings and flowering shoots of mango. It was confirmed by Puttarudriah and Channa Basavanna (1961), but Bindra and Bakhetia (1971) could not reproduce malformation with the inoculation of only one kind of mite.

Summanwar et al., (1966) induced malformation by inoculation of F. moniliforme. Varma et al., (1974) also claimed to induce 100% vagatative malformation in seedlings and 43% floral malformation in the grafted plants of cvs. Neelam and Muvandan from the isolates taken from malformed vegetative shoots or malformed panicles. Bhatnagar and Beniwal (1977) produced the typical bunchy top symptoms in mango seedlings by inoculating the fungus Fusarium oxysporum schlecht, through soil on the other hand, Salma et al., (1979) inoculated three Fusarium species viz., F. moniliforme, F. oxysporum and F. solani in mango, out of these only F. moniliforme produced symptoms especially in the month of February, but Kumar (1983) failed to induce malformation by inoculation of the same fungus.

Chadha et al., (1979) induced malformation like symptoms on mango within 15 days of spraying of morphactins with increased activity of IAA oxidase. Ghosal et al., (1978, 1979) also claimed to produce symptoms similar to vegetative malformation by the treatment with mangiferin (1 × 10⁻⁴m) in one year old seedlings.

Varietal susceptibility:

Susceptibility to malformation in mango varieties vary from one
percent to cent percent depending upon the cultivar, age of the plant and agroclimatic conditions of the area. Numerous poly and mono embryonic mango cultivars have been examined in India and Egypt for the varietal susceptibility (Sharma, 1953; Tripathi, 1954; Khan and Khan, 1960; Singh and Jawanda, 1961; Jawanda, 1963; Prasad et al., 1965; Jagirdar and Shaik, 1968; Dhar et al., 1978; Azzous et al., 1978; Kumar and Beniwal, 1987; Om Prakash and Raoof, 1987; Ram et al., 1990). In general, late blooming varieties are less susceptible to malformation that the early blooming ones (Khurana and Gupta, 1973). Early and mid-season cultivars showed moderate to high incidence of malformation while late season cultivars showed low incidence of the disease (Singh et al., 1961, 1977). However, this could not be confirmed by Prasad et al., (1965). Younger trees possess higher incidence and intensity of malformation than ones (Puttarudriah and Channa Basavanna, 1961.; Putto et al., 1975; Ram et al., 1990); Khan and Khan (1960) opined that no variety is completely resistant against the disease (Schlosser, 1971; Om Prakash and Raoof, 1987). Varieties from southern India where the disease incidence is sporadic were found to be more severely infected when introduced in northern India (Singh and Jawanda, 1961; Mallik, 1963).

Prasad et al., (1965) observed that seedling trees were comparatively less infected that grafted ones, there being no correlation between the intensity of the disease and the variety. Among the grafted varieties, collector, Langra, and Neelam (2-8%) were found tolerant while Answer, Rataul (45-50%), Alphanso (70-90%) Dashehari (15-69%), Malda (50-90),
Samarbahist (20-98%) were classified as susceptible (Khan and Khan, 1960). Prasad et al., (1965) claimed Bhadauran- a monoembryonic variety, to be the only one which is free from this disease (Rām et al., 1990) while Dashehari, Langra, Malda, Safeda and Chausa had 40-100% infection. Jawanda (1963) supported by Singh and Jawanda (1965) found Durre Bahisht and Kishan Bhog to be fairly tolerant (1%) and Mohammadwala to be highly susceptible (96%). The former author also reported Khasulkhas, Alphanso, Bombay yellow and Fajri to be heavily infected while Dashehari, Langra and Samar Bahisht-Chausa had fair degree of incidence. Prasad and Singh (1972) reported from Bihar that Gulab Khas, Himsagar, Bombai, Poona, Alphanso, Krukane, Mylipilean to be susceptible. Studies conducted at CISH, Lucknow during 1978-1979 under field conditions on 122 cultivars revealed that cultivar Ilaichi was free from this malady.

In Egypt, Azzous et al., (1978) observed that the cultivar Glyore, Mabrauka, Tamour and Company show severe symptoms, whereas Zebda (Wabha et al., 1986), Hindi and Anshas were rarely affected. Dang and Daulta (1982) found that only cultivar Langra was tolerant and varieties Bombay-green, Sipia, Samar-Bahist-Chausa, Khas-Ul-Khas and Malda were susceptible under Hissar conditions. The studies conducted by Majumdar and Diware (1985) revealed that cultivars Chausa and Bombay Green indicated 90% infection while the cultivars Bhadauran had negligible infection. During a survey, Chib et al., (1984) reported that cultivar Chausa had highest susceptibility (45.21%), whereas, cultivar Langra had lowest susceptibility (21.42%)
to malformation in mango growing of Jammu and Kashmir. In addition to *Magnifera indica* L., disease has also been recorded on *M. zeylanica* (Prasad *et al.*, 1965).

Common mango cultivars in India have been categorized on 1 to 9 rating scale: resistant, moderately resistant, tolerant, moderately susceptible and susceptible (Kumar and Beniwal, 1987). In northern India, the choicest cultivars which include Langra, Dashehari, Chausa, Mallika and Amrapali are most susceptible (>20% disease index). Resistant sources were further identified for inclusion in breeding programs (Nath *et al.*, 1987).

Badilya and Lakhan Pal (1990) studied 12 cultivars for the relative susceptibility and found that malformation was highest in Amrapali (57%), Bombay Green (56.2%) and Mallika (55.0%) and the percentage was lowest in Langra (4.37%), Totapari (16.5%) and Alphanso (17.25%). Later, Ram *et al.*, (1990) observed the relative susceptibility in 8 cultivars and found that Bombay Green, Dashahari, Lucknow Safeda and Chausa bear high percentage of malformation (10.8-24.2%), while in Baramasi it was 0.32-1.92%. Ram (1993) stated that most resistant cultivars to malformation viz. Alib, Cherumani, Malda Handle and Dudhia Langra provided help in the development of new mango hybrids resistant malformation.

Yadav and Singh (1995) found that the intensity of malformation was more in Mallika (12.3%) followed by Amrapali (8.3%) in comparison to Dashehari (4.6%) mango. Exotic cultivar Kensington (19.2%) was most susceptible to floral malformation. Singh and Yadav (1999) examined forty
two mango germplasma and found the maximum incidence of moderate type of malformation in Ahra Baramasia whereas least incidence was recorded in Rataul and none were found to be free from floral malformation.

Wada et al., (2001) classified 24 mango cultivars into five groups on the basis of polyphenol oxidase activity, phenolic content and panicle formation (Table-2).

<table>
<thead>
<tr>
<th>Resistance/susceptibility to panicle malformation</th>
<th>Varieties</th>
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<tbody>
<tr>
<td>Highly resistant</td>
<td>Bhadauran and H-8-1</td>
</tr>
<tr>
<td>Moderately resistant</td>
<td>Dashehari, Langra</td>
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<td></td>
<td>Kurukkan and Fazil.</td>
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<td>Susceptible</td>
<td>Sensation, Eldon, Rataul,</td>
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<td></td>
<td>Mallika and Alphanso</td>
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<tr>
<td>Moderately susceptible</td>
<td>H-31-1, Lalsundri, Totapari</td>
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<tr>
<td></td>
<td>Red smell, Himsagar,</td>
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<td></td>
<td>Neelum, Exterme, Zill,</td>
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<td></td>
<td>Edward and Amrapali</td>
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<tr>
<td>Highly susceptible</td>
<td>Tommy, Atkins, Chause,</td>
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<td></td>
<td>Zardalu and Ratna.</td>
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</table>
Spread and Transmission:

The annual recurrence of mango malformation in new seedlings and trees increases gradually and unpredictably. The disease spreads slowly from infected to healthy seedlings/trees in a nursery/orchard (Nirvan, 1953; Singh et al., 1961; Schlosser, 1971), which indicates its natural spread. A healthy tree adjacent to diseased one may remain healthy for many years (Mallik, 1963, Prasad et al., 1965). During one three year study (Kumar, 1983), only one new infection was recorded in a grove although disease severity (percent malformed inflorescences) in the infected trees gradually increased. Zones representing high, medium, low or no disease (floral malformation) identified in the first year remained unchanged throughout the study. In another study, the incidence of vegetative malformation in the same nursery revealed that plot showing maximum infection during the first year also showed maximum disease incidence during the first year also showed maximum disease incidence during subsequent years when new crops were raised from seeds (Kumar, 1963). These observations, together with the pattern of disease development in a tree where only a few branches continue to bear malformed inflorescences year after year, point to systemic infection and a slow-moving, possibly vectored, soil-borne source of contagion. Efforts made by early workers to transmit the disease by grafting or budding (Singh et al., 1961; Mallik, 1963; Prasad et al., 1965) could not be proved later on (Kishtah et al., 1985); Kumar, 1983; Mohan and Ram, 1992; Tripathi and Ram, 1994). The disease could also not be transmitted mechanically
or through the seed and dodder (Kishtah *et al.*, 1985; Kumar and Beniwal, 1992).

The aerial movement of the spores of *F. moniliforme* was studies by Varma *et al.*, (1971) by using rotary traps for six months in an orchard with high incidence of malformation, but they failed to trap any fungal spores. The role of the mango bud mite *Aceria mangiferae* has been involved in spread of the disease (Puttarudriah and Chana Basavanna, 1961; Singh *et al.*, 1961; Nariani and Seth, 1962). The mites were observed carrying *F. moniliforme* spores on their body surface. However, sporadic incidence of disease in southern and eastern India where plentiful of mites were observed, could not be explained on this basis (Summanwar and Raychaudhary, 1968; Varma *et al.*, 1971). Schlosser (1971 a) also emphasized the role of insects in disease spread by the absence of disease in nurseries sprayed with insecticides. Thus, the disease production experiments by several ways such as sap inoculation, fungal and mite infection etc. also did not yield convincing results.

*Fusarium subglutinans* appears to be the systemic and endemic in mango trees and seedlings (Bhatnagar and Beniwal, 1977; Andotra *et al.*, 1984; Kumar and Beniwal, 1992; Ploetz, 1994). Strong correlation existed between the presence of the fungus in the soil and symptomless healthy tissues lies in the degree of the infection by a pathogen (Darvas, 1987; Ploetz, 1994). Presence of the *Fusarium subglutinans* and related species throughout the plant (root, shoot, panicle) is indicative of an endemic and systemic nature of the fungus. Therefore, the disease may be soil-borne,
but its air-borne nature can not be completely ruled out, because malformation symptoms have been produced by the inoculation of the fungus through slit (Summanwar et al., 1966, Varma et al., 1971), soil (Bhatnagar and Beniwal, 1977) and spore suspension (Chakrabarti and Ghosal, 1989; Ploetz, 1994).

Epidemiology:

The epidemiology of mango malformation is poorly understood because of the lack of uniformity in its occurrence and variations in the severity of disease from season to season. However, some workers have tried to correlate the seasonal disease variation with ambient temperatures at the time of flowering (Varma et al., 1969; Majumdar and Sinha, 1972 a). They found lesser incidence of malformation when temperature was raised around the trees artificially during flowering period. Singh et al., (1977) observed that the flower buds which emerged earlier were heavily affected with malformation whereas those emerging later escaped the disease. This trend was particularly observed in the cultivars which had early burst. Singh et al., (1979) found that malformation was maximum in the floral buds emerging during first 10 days in all the cultivars and was reduced in the floral buds emerging later. Such a reduction was attributed to a relatively high temperature prevailing during panicle development, particularly before the balloon stage (Rajan, 1886). In India, the effect of climate on the incidence of disease was reflected as one moves from the warmer southern region, where the incidence of infection was low to the month, where cooler conditions
preceded flowering and the disease was more severe (Varma et al., 1971, Kumar, 1983).

In Egypt, panicles appeared on spring shoots were most severely affected, followed by summer and early autumn shoots (Shawky et al., 1980). In Florida, the heaviest infection occurs under unusually wet conditions (Campbell and Marlatt, 1986). In North Guerrero, Mexico, temporal progress of mango malformation and the dynamics of epidemics was studied by Noriega-cantu et al., (1999). They also found the greatest spore density of *Fusarium* sp. trapped in the canopy.

**Etiology/Causes**:

The etiology of mango malformation is still a matter of controversy. Several workers have been reviewed the work done on the causes of this malady which indicates that the results are still inconclusive. Various biotic and abiotic factors have been reported to be associated with the causation of disease and are listed as follows:

**Biotic factors**:

**Mites as the causal agent**: Presence of on eriophid mite, Aceria (Eriophyes) mangiferae was attributed to be cause of mango malformation (Hassan, 1944; Narasimhan, 1954; Singh et al., 1961; Narayanan and Ghai, 1963; Srivastava and Bhutani, 1993; Sternlicht and Goldenberg, 1976). Several other species of mites, including predatory species namely, *Cheletogenes*
ornatus, Typhalodromus rhenanus, T. roshanlati, T. nesbitti (Narayanan and Ghai, 1963), T. asiaticus and Tyrophagus castellanii (Singh, 1957; Singh et al., 1961) were reported to be associated with the disease. The mites were associated with vegetative and floral malformation (Sayed Taher, 1946). It was also suggested that high humidity was a pre-requisite for the propagation of the disease, and it is induced in two growth periods i.e., March to April and July to October (Nariani and Seth, 1962). On transferring A. mangifera, T. castellani and T. asiaticus from malformed to healthy seedling, there was 80% vegetative malformation and 50% floral malformation (Singh et al., 1961).

A positive correlation between mite population and disease incidence was noted, and the application of acaricides controlled the disease to some extent (Giani, 1965; Dickman et al., 1982).

The acarological theory of mite as a causal agent of malformation was, however, refuted because no correlation between the mite population and disease incidence was established (Latif et al., 1961; Bindra and Nakhetia, 1969, 1971; Kumar, 1992; Usha et al., 1997) and the acaricidal spray did not check malformation (Prasad et al., 1965). It was also suggested that the mite may serve as a vector for viruses (Puttarudriah and Channa Basavanna. 1961; Giani, 1965; Schlosser, 1971). and for fungus Fusarium moniliforme (Summanwar and Raychaudhary, 1968; Varma et al., 1971). However, Aceria mangiferae does not cause the disease, but may play a role in the overall malformation process (Wabha et al., 1986; Labuschagne et al., 1993). Further involvement of F. subglutinans as the causal agent and the
bud mite *A. mangiferae* as the transfer and wounding agent has been suggested (Pinkas and Gazit, 1992).

**Virus and mycoplasma-like organisms (MLO):** The occurrence of malformation in both grafted and seedling plants of mango and a gradual increase in the incidence year after suggested that the malformation could be of viral origin (Sattar, 1946; Ahmad and Sattar, 1950; Kauser, 1959; Khan and Khan, 1960; Latiff *et al.*, 1961). Viral nature of the malady was also suspected because of greater incidence of malformation in areas severely infested with mango hoppers, the vector of viral disease (Singh and Jawanda, 1961). However, the disease could not be transmitted by mango hoppers, mealy bugs, thrips and aphids. Subsequently, they could transfer the disease through mites but failed to confirm viral nature of the disease (Singh *et al.*, 1961). Workers also failed to transmit the disease by inoculating the sap of malformed panicles and shoots in healthy ones (Mallik, 1961; Singh *et al.*, 1979; Salma *et al.*, 1979).

Successful transmission of malformation by grafting or budding has been reported (Ahmad and Sattar, 1950; Vasudeva, 1957; Kauser, 1959; Mallik, 1963; Bindra and Bakhetia, 1971), although no measures were taken to prevent the movement of mites, but a few workers could not transmit it from malformed stock to healthy scion or Vice-Versa (Singh *et al.*, 1961; Prasad *et al.*, 1965; Kumar, 1983; Mohan, 1991). Presence of mycoplasma-like organisms (MLO) was also reported (Khan and Hussain, 1985). A new evidence was provided by demonstrating that the *Fusarium moniliforme* var.
*Fusarium subglutinans* infected with a mycovirus caused the shoot malformation (Gupta, 1991). However, results from electron microscopy, transmission, cultural and serological studies in India and Egypt have convincingly disproved the involvement of virus or mycoplasma-like organisms (Kishtah *et al.*, 1985; Kumar and beniwal, 1992).

**Fungi as the causal agent:** The early workers not isolate any of the pathogenic organisms form disease tissues (Singh *et al.*, 1961; Nariani and Seth, 1962). However, the fungus *Fusarium subglutinans* was isolated from malformed panicles of mango and after inoculation induced vagatative malformation in young seedlings grown in glasshouse, thus establishing its pathogenicity (Summanwar *et al.*, 1966). It was further observed that mites carried the fungus on their bodies and the injury caused by these mites provides a way for the entry of pathogen into the tissues of the host plant and thus assisted in the dissemination of disease (Summanwar and Raychaudhauri, 1968). This view was supported by other workers (Fletchmann *et al.*, 1970; Sharma and Tiwari, 1975). The fungus could not be isolated from the healthy area adjoining the infected tissues and it was concluded that the fungus was not systemic but localized in nature (Summanwar, 1970). The malady was, however, found to be systemic in branches (Varma *et al.*, 1969).

Extensive isolation and identification of the fungus *F. moniliforme* from infected parts of different cultivars from all over India was conducted by Varma *et al.*, (1969, 1971, 1972, 1974). *F. moniliforme* was isolated from
infected twigs or rachises and flowers of malformed panicles and the fungus was reported as *F. moniliforme* Var. *subglutinans* (Fletchmann *et al.*, 1970; Varma *et al.*, 1974). Vegetative malformation has been reproduced by slit inoculation, soil inoculation and by spraying the plants with spore suspension of *Fusarium* sp. (Varma *et al.*, 1974). They were able to induce 100% vegetative malformation and 43% floral malformation by the isolates of malformed shoots or panicles (Varma *et al.*, 1974). Only 20% malformation could, however, be induced in healthy seedlings by the fungus isolated from malformed plant parts (Prasad *et al.*, 1972). Similar results were obtained by other workers (Malo and Mcmillan, 1972; Chadha *et al.*, 1979). Other fungal species associated with the malformed tissues were *F. oxysporum* and *F. solani* (Ibrahim *et al.*, 1975; Bhatnagar and Beniwal, 1977; Salma *et al.*, 1979). The involvement of *F. oxysporum* schlecht in causation of the malformation disease was reported and its pathogenicity was proved (Bhatnagar and Beniwal, 1977). In central Brazil, *F. sacchari* was isolated from malformed shoots and panicles of mango variety Tommy Atkins. Pathogenicity tests were carried out by slit inoculation of seedlings using *F. sacchari* mycelia and conidia. Symptoms of vegetative malformation appeared in 63.6% inoculated plants (Anjos *et al.*, 1998).

Mango malformation probably involves two principles (a) the malformation inducing principle (MIP) which works through imbalance in the growth substances and in the conditioning of cells, and (b) the toxic principle (TP) which causes growth retardation and toxicity symptoms (Kumar
and Beniwal, 1992); it is suspected that *Fusarium* sp. is the likely source of MIP and T.P. and is the causal agent of the disease (Kumar and Beniwal, 1992). However, it is reported that *Fusarium suoglutinans* produces secondary metabolites, including trichothecene derivatives that are also detected in malformed tissues (Ghosal et al., 1979), naphthazarines and malformation (Stoessal, 1980) which could constitute TP (Kumar et al., 1993). On the other hand, *F. moniliforme* var. intermedium (Pandey and Ram, 1994), *F. moniliforme* var. *subglutinans* and *F. moniliforme* sheld (Kumar and Ram, 1998) secreted malformation into their cultural filtrates which were similar to mango malformation. Malformin treated mango seeds and branches of bearing trees produced malformed seedlings and shoots (Tripathi and Ram, 1998). It was suggested that the malformation in mango was caused by malformation which could act as the MIP or one of its constituents (Singh and Dhillon, 1987, 1989; Kumar and Beniwal, 1992). Experiments have suggested that a certain pathogen level of *F. moniliforme* determines the disease, and the propagules of the fungus are drawn up in the seedlings by the damaged roots (Kumar and Beniwal, 1992).

The identical morphology and etiology of vegetative and floral isolates of *F. moniliforme* (*Gibberella fujikuroi*) var. *subglutinans* was also studied (Mitra and Lele, 1981). The positive role of plant growth regulators was suggested in the growth and sporulation of *F. moniliforme* var. *subglutinans*. Maleic hydrazide proved to be one of the best source for growth and sporulation of fungus followed by GA, IBA and NAA (Chattopadhyay and
Nandi, 1981). The involvement of the *F. subglutinans* is getting more acceptance than the other fungi because both the vegetative and floral malformed tissues invariably contain the fungal hyphae in a higher density than the healthy ones (Summanwar, 1970; Varma *et al*., 1971, 1974; Kumar, 1983; Darvas, 1987; Burhan, 1991, Ploetz, 1994; Ram, 1997).

Malformed tissues also show inter and intracellular distribution of the fungi in the cortex, phloem and parenchymatous pith cells (Varma *et al*., 1974; Chakrabarti and Ghosal, 1989). *F. moniliforme* var. *subglutinans* is now widely accepted fungus responsible for this disease from several countries namely India, USA; South Africa and Israel (Summanwar *et al*., 1966; Andotra *et al*., 1984; Campbell and Marlatt, 1986; Manicom, 1989; Ploetz, 1994; Usha *et al*., 1997; Koti Babu and Rao, 1998; Noriega-Cantu *et al*., 1999; Freeman *et al*., 1999, 2000; Bains and Pant, 2003). The role of fungus in causing mango malformation has recently been emphasized through transformation studies using GUS (β-glucuronidase) transformants of *F. subglutinans* (Freeman *et al*., 1999, 2000). *Fusarium subglutinans* was isolated consistently from diseased (86%) and asymptomatic (5%) vegetative and flowering shoots (Noriega-Cantu *et al*., 1999).

**Abiotic factors associated with malformation:**

**Excessive soil moisture:** It was first reported by Maries that mango malformation could be due to excessive soil moisture (Wass, 1891). Later investigations, however, could not conclusively prove the theory (Abdel
Nutritional imbalance: The association of nutrient deficiency or excess with the malformation has been contradictory. However, leaves of vegetatively malformed seedlings were reported to contain more ash, silica, calcium and water but lesser potassium as compared with healthy seedlings (Tripathi, 1955). Injecting (solid or liquid) or spraying with solution of boron, calcium, copper, iron, magnesium, manganese, potassium, sodium and zinc did not control the malady (Sharma, 1953; Tripathi, 1955; Singh et al., 1961; Saeed and Schlosser, 1972). Later on, it was found that higher nitrogen dose decreased the malformation (Khan and Khan, 1958; Prasad et al., 1965). Application of N, NK or NP reduced malformation while PK or K increased it (Jagirdar and Saikh, 1969). However, malformation could not be reduced by nitrogen (Bindra and Bakhetia, 1971).

Soil application of chelated iron (containing 6-10% Fe) at the rate of 50-100 g per young tree and 554 g per mature tree are reported to control malformation (Minessy et al., 1974; Abou-El-Dahab (1977). Combined application of potassium sulphate as soil application and monocrotophos as trunk injection cured the melady (Peswani et al., 1979). The intensity of malformation by spraying Bayfolan (containing N, P, K, Fe, Cu, Mn, B, Zn, Co and Mo) also failed to reduce (El-Beltagy et al., 1980). In another study, malformed panicles were found to contain higher nitrogen and potassium and lower zinc levels as compared to healthy panicles (Mishra, 1976). Higher levels of soluble and total nitrogen have been reported in healthy panicles...
than malformed ones (Pandey et al., 1977). Furthermore, enhanced nitrogen application curtailed malformation whereas addition of phosphorus and potassium significantly increased its incidence (Kanwar and Kahlon, 1987). In South Africa, floral malformation was effectively controlled by two years of trunk injection with phosetyl-Al+Zn+B mixture (Darvas, 1987). Thus, nutrient deficiency was not considered to be primary cause of mango malformation (Singh and Rathore, 1983 a; Singh, 1986).

The lower amount of Ca in malformed tissues was suggested to be a predisposing factor causing malformation in mango (Singh et al., 1991). Analysis of micronutrient levels in malformed and healthy tissues of mango revealed that most of the micronutrients (Fe, Zn, Mn, Cu) accumulate in panicles, leaves and shoots as a result of malformation. It was proposed that malformed tissues might be acting as an effective sink for these micronutrients (Singh et al., 1994). Reduction in the disease by the mangiferin metal chelates (Chakrabarti and Ghosal, 1989) and sulphates of Co, Cd and Ni were also reported, but none of these applications could be proved beneficial elsewhere (Tripathi and Ram, 1994). However, replicated trials with cheated Zn and Fe could not control the malformation (Tripathi, 1994). It was reported that significant differences were observed in the levels of cations and anions (Cl\(^{−}\), SO\(_{4}^{2−}\) and PO\(_{4}^{3−}\)) in healthy and malformed tissues at, prior to full bloom and full bloom stage, but a consistent pattern was not seen. The exception was that of phosphate ion which was present in higher concentration in the malformed floral tissues at full bloom stage (Kaushik, 2002).
Temperature: The incidence of mango malformation has been found to vary from season to season, but the causes of such variation are not well understood. However, studies have revealed that temperature is closely related with the variation in the incidence of mango malformation (Malik, 1963; Jagirdar and Shaik, 1968). In India, the effect of climate on the incidence of disease was reflected as one moves from the warmer southern region, where the incidence of infection was low, to the north, where cooler conditions preceded flowering and the disease was most severe (Varma et al., 1971). Seasonal variation in the occurrence and severity of disease have been correlated with ambient temperature at flowering (Majumdar and Sinha, 1972a). In general, late blooming varieties have been found to be less susceptible to malformation than early blooming ones (Khurana and Gupta, 1973). Lower percentage of malformation was attributed to relatively higher temperature which prevailed during the period of panicles development (Singh et al., 1979; Ram, 1991).

A study of seasonal variation of the population of *Fusarium moniliforme* on mango shoots in India indicated that fungal density reaches a maximum in February, when minimum/maximum temperature ranges from 8-27 °C and humidity is high (85%). Hotter and drier periods coincide with a decline in the fungal population (Kumar et al., 1993).

Various experiments employing the use of polythene bags to raise the temperature were successful in controlling malformation in various cultivars. Panicles covered with polythene bags had higher rates of growth
and length and were also free from malformation (Tripathi, 1992; Tripathi and Ram, 1995). Similar results were obtained by other workers (Singh and Saini, 1966; Singh et al., 1998).

Incidence of malformation was observed to be 20% at 400 m altitude, while almost all plants were found to be free from floral malformation at an altitude of 1250 m and above. Night temperature below 10 °C for long duration was found to be responsible for suppressing the incidence of floral malformation (Singh et al., 1999). It may be visualized that the occurrence of mango malformation in Malaysia (Lini and Khoo, 1985) may be due to factor(s) other than the chilling temperature (Pant, 2000; Bains and Pant, 2003).

Effect of biotic and stresses on the metabolism of mango:

Metabolic imbalance: The malformed tissues contain more carbohydrate and nitrogen than normal tissue (Tripathi, 1955; Mallik, 1963) and higher C/N ratio was reported in malformed tissues (Khan and Khan, 1963; Pandey et al., 1973; Mishra, 1976). It was postulated that high C/N ratio could be responsible for greater percentage of staminate flowers on malformed panicles (Khan and Khan, 1963; Majumdar and Sinha, 1972) and suppression of development of flower and fruit set (Pandey et al., 1973). Contrary to these findings lower levels of starch as well as lower C/N ratio in shoots bearing malformed panicles were also reported (Singh, 1986). This decline in carbohydrate titer and C/N ratio was attributed to enhanced
utilization of these compounds and therefore, excessive growth of malformed
panicles (Singh and Dhillon, 1993).

Accumulation of excessive amount of acid hydrousable
polysaccharides in malformed panicles was reported (Pandey et al., 1977).
Malformation was also found to be associated with degradation of cellu­
lose and lignin (Chattopadhyay and Nandi, 1977 a). Lower levels of reduc­
ing, non-reducing and total sugars, starch and carbohydrates in malformed
tissues than in healthy ones have been observed (Singh and Rathore, 1983b).
Lower rate of photosynthesis and lower leaf saccharides were on served in
malformed mango cultivars (Yadava and Singh, 1995). It was also observed
that chlorophyll content was low in leaves of shoots bearing clustered
fruits that the bearing normal fruits. Hoever, the chlorophyll content, re­
ducing sugars, rate of respiration and activities of enzymes such as
polyphenol-oxidase and amylase were higher in clustered fruits (Singh and
Yadava, 1977).

Higher levels of total phenols (El-Ghandour et al., 1976; Rajan,
1986) and ortho-dihydroxyphenols in malformed shoots than the healthy
ones (Singh, 1986). The increased peroxidase and polyphenoloxidese ac­
tivity in malformed panicles was reported (Chattopadhyay and Nandi, 1976)
but a lower nitrate reductase activity in malformed buds and panicles was
observed (Singh et al., 1992).

Higher amounts of free and lower amounts of bound aminoacids
were reported in the leaves of malformed shoots than in those of healthy
shoots (Sandhu, 1975). High RNA content in malformed tissues has been attributed to increased phosphorus content at the site of malady when *F. moniliforme* var. *subglutinans* was artificially inoculated into susceptible cultivars (Chattopadhyay and Nandi, 1976). However, lower level RNS, DNA, soluble proteins, total amino acids and amides were reported in malformed panicles compared to healthy panicles (Pandey *et al.*, 1975, 1977). Further, the content of DNA and RNA was reported to be higher in malformed seedlings than in healthy ones (Singh, 1986). The malformed seedlings has higher protein and total amino acids that the healthy seedlings (Singh and Dhillon, 1989c). Thus significant differences were not observed in total protein content of healthy and malformed panicles of mango cultivars (Kaushik, 2002).

**Hormonal imbalance**: Hormonal imbalance in malformed tissues is considered to be one of the important reasons for creating the malformation (Majumdar *et al.*, 1970; Pandey *et al.*, 1974; Kumar *et al.*, 1993).

**Auxins**: It was observed that floral malformation may be related to the imbalance of auxins and antiauxins caused by vectors, diseases or nutrient deficiencies (Jagirdar and Jafri, 1966). Low levels of auxins have been reported in malformed tissues as compared to healthy ones (Pandey *et al.*, 1974; Beniwal, 1976; Mishra and Dhillon, 1978; Singh and Dhillon, 1989b; Pandey and Ram 1995). It was shown that the levels of indole acetic acid (IAA) acetonitrile (IAN) were 98.4 and 92.6 per cent lower in bunchy top affect tissues which showed increase activity of IAA oxidase, peroxidase and polyphenoloxidase (PRO) (Kumar *et al.*, 1980). However, levels of acidic,
non-acidic and total auxin were found to be higher in healthy than in malformed panicles (Abou-Hussein et al., 1975; Pal et al., 1983; Dahshan, 1987; Pandey, 1988).

**Gibberellins**: A reduction in malformation of panicles was observed by the use of GA₃ at the flower bud differentiation stage (Kachru et al., 1971; Shawky et al., 1978). Low levels of gibberellins and higher levels of inhibitors were reported in malformed tissues (Fl-Ghándour et al., 1976; Kumar and Beniwal, 1979; Singh and Dhillon, 1989; Singh et al., 1992). However, higher levels of gibberellins were reported from malformed panicle, which may account for production of male flower and continuous growth of malformed panicles (Abou-Hussein et al., 1975; Mishra and Dhillon, 1980; Bist and Ram, 1986; Dahshan, 1987; Mishra et al., 2009; Ansari et al., 2012, 2013). Exogenous application of 100 or 250 ppm GA₃ was found to increase malformation in panicles (El-Beltagy et al., 1980). Further, it was reported that 200 or 500 ppm chloromequat (CCC) mixed with 100 ml Bayfolan treatment reduced percentage of malformed panicles, but these results were not reproducible (Rana, 1992).

**Cytokinins**: Higher levels of cytokinins were observed in malformed panicles than healthy ones (Bisht and Ram, 1986; Dahshan and Abdul-Raheem, 1987). A comparison of cytokinins complement of healthy and malformed inflorescences indicated the absence of transzeatin (Tz). Dihydrozeatin (DHZ) and ribosyldihydrozeatin (DHRZ) in malformed flowers (Nicholson and Van Staden, 1988). Further, studies using (8 ¹⁴C)-acenine
showed that *Fusarium* sp. produced and released a biologically active compound similar to iso-pentenyladenine (Van Staden and Nicholson, 1989).

**Abscisic acid**: Malformed panicles showed 85% more inhibitory activity than healthy (Mishra and Dhillon, 1978). Higher levels of abscisic acid (ABA) were observed in both, floral and vegetative malformed tissues (Singh, 1986; Singh and Dhillon, 1990; Singh *et al*., 1992; Pandey and Ram, 1994, 1995; Hatzfeld *et al*., 2000; Kukveja *et al*., 2000; Nailwal *et al*., 2006). ABA levels in vegetative tissues known to be elevated in response to various environment stresses (Zeevart, 1999). However, no significant difference was observed between the abscisic acid content of healthy and malformed tissues of mango cultivars (Kaushik, 2002).

**Ethylene**: As some of the symptoms malformation resemble with that of ethylene effects, several workers have implicated a role of ethylene in mango malformation. Levels of ethylene were found to be higher in malformed vegetative and floral tissues as compared with that of healthy tissues at various stages of development (Singh and Dhillon, 1990; Pant, 2000; Bains and pant, 2003; Ansari *et al*., 2008; Bist *et al*., 2000; Tapan *et al*., 2006; Youssef *et al*., 2009). The incidence of floral malformation was reduces by 300-500 ppm ethaphon spray (Singh and Dhillon, 1990). However, other workers failed to reduced malformation by application of ethaphon (Ram, 1991).
Malformation factors:

Malformin: Malformin is a regulator, a cyclic pentapeptide (Cyclo D-cystenyl-D-Cysteiny1-L-Valyl-D-Leucyl-L-lsoleucyl) and affects cellular orientation (Takahashi and Curtis; 1961). Malformin-like substances have been reported in malformed panicles but absent in healthy ones (Ram and Bist, 1984). Application of antimalformins like glutathione, ascorbic acid and silver nitrate caused disappearance of malformin from panicles, which fruited like healthy controls (Bist and Ram, 1985). Malformins may cause imbalance of growth substances and conditioning of host cells to produce malformed growth (Bist and Ram, 1986; Singh and Dhillon, 1987, 1989d; Raina and Ram, 1991; Tripathi, 1992).

Fusarium moniliforme var. intermedium (Pandey and Ram, 1994) and F. moniliforme var. subglutinans and F. moniliforme sheld (Kumar and Ram, 1998) secreted malformins into their culture filters which were similar to mango malformins. Mango malformin treated mango seeds and branches trees produced malformed seedlings and shoots (Tripathi and Ram, 1998). Recent studies revealed that malformin-like substances are involved in the causation of mango malformation, with malformin-stimulated ethylene production (Singh, 1998).

Mangiferin: Mangiferin (1, 3, 6, 7-tetra hydroxyxanthone-C2-β-D-glucoside), a non-toxic polyphenolic metabolite in mango was reported to play a significant role in the malformation of mango (Ghosal et al., 1978, 1979). The concentration of mangiferin was high in malformed panicles while
it was present in traces in healthy panicles (Chakrabarti et al., 1990). An increase in the activity of polyphenol oxidase (a mangiferin degrading enzyme) was observed in the infected tissue (Kumar and Chakrabarti, 1992). Symptoms of mango malformation were included by accumulation of mangiferin (Chakrabarti and Kumar, 2002).

**Control:**

As there is no dearth of opinions regarding the cause of the controversial disease, attempts have been made by several workers to control malformation but the results are neither successful nor reproducible. Various approaches to control the disease are given below.

**Pruning of malformed parts:**

Pruning of malformed parts reduced malformation (Narasimham, 1960; Desai et al., 1962; Malik, 1963; Doval et al., 1976; Singh et al., 1983; Mishra and Om Prakash, 2000). Pruning, however, had no effect on malformation (Bindra and Bakhetia, 1971) spray of 1% Folidol or Etakin in July combined with pruning of the diseased shoots substantially reduced malformation (Desai et al., 1962). Pruning followed by spraying with the mixture of fungicide (Captan 0.1%), miticide (Akar 338-0.1%) and sticker (Tenae) helped considerably in controlling at least the spread of the disease (Summanwar, 1967). Pruning of diseased parts and spraying with diazinon were also reported to control the malady (Rai and Singh, 1967; Yadava, 1972).
Elimination of affected seedlings from nurseries was found effective in reducing the malformation (Schlosser, 1971a). However, cutting of scion sticks for grafting during August and September increased the incidence of malformation (Khäder et al., 1986). It has also been observed that pruning with or without fungicidal or acaricidal applications was not successful in reducing floral malformation in cv. Dashehari (Chib et al., 1984). Pruning of young malformed panicles or shoots reduced the fungal mycelium and the metabolite like malforrin and thus, make them free from the malformation (Tripathi and Ram, 1998).

Application of nutrients:

A direct inhibitory effect of chemicals against pathogen is inferred together with secondary control through improved nutritional status of trees. The effects of exogenously applied macro-and micro-nutrients on reduction of malformation have been discussed in etiology and summarized (Table-3) as below:
Table-3  Effect of exogenous application of essential nutrients on malformation

<table>
<thead>
<tr>
<th>Cultivar (S)</th>
<th>Nutrients</th>
<th>Inference</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bombay Green</td>
<td>Manganese 4: 2: 100, Zinc sulphate 4: 2: 100, Copper sulphate 4: 2: 100,</td>
<td>Did Not reduce malformation</td>
<td>Tripathi (1955)</td>
</tr>
<tr>
<td>Fazrijafrani</td>
<td>Borax 2: 1: 100 (nutrient : CaO: water), 3 foliar spray in April,</td>
<td></td>
<td></td>
</tr>
<tr>
<td>S B Chausa</td>
<td>September and February</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dusheri</td>
<td>N: P₂O₅: K₂O (9:3:3) soil application</td>
<td>High level of N, low level of P and K reduced</td>
<td>Prasad et al.,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>floral malformation</td>
<td>(1965)</td>
</tr>
<tr>
<td></td>
<td>N: P₂O₅: K₂O (9:3:3) soil application in January</td>
<td>No reduction in incidence if floral malformation</td>
<td>Bindra and Bakhetia (1971)</td>
</tr>
<tr>
<td></td>
<td>April and October</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dusheri</td>
<td>Nitrogen (200 f/tree). soil application, half in, February and half in</td>
<td>Nitrogen reduced floral Malformation from 80%</td>
<td>Cheema and Malhi (1986)</td>
</tr>
<tr>
<td></td>
<td>September P₂O₅ (200g/tree) half in February and half in September</td>
<td>to 45%.</td>
<td></td>
</tr>
<tr>
<td>Dusheri</td>
<td>Foliar application of ZnSO₄, FeSO₄, MnSO₄, and CUSO₄, (0.2 to 0.4%)</td>
<td>Did not substantially reduced floral</td>
<td>Singh (1980)</td>
</tr>
<tr>
<td></td>
<td>during first week of October (prior to FBD)</td>
<td>malformation</td>
<td></td>
</tr>
<tr>
<td>Dusheri</td>
<td>N (300 g/tree) soil application half in March and half in October</td>
<td>Reduced floral malformation</td>
<td>Kanwar and Kahlon (9187)</td>
</tr>
<tr>
<td>Dusheri</td>
<td>Cobalt sulphate (1000 ppm) Foliar spray in first week of October</td>
<td>Reduced floral malformation</td>
<td>Singh and Singh (1990)</td>
</tr>
</tbody>
</table>
Deblossoming:

Deblossoming at the bud burst stage alone or in combination with spraying of 200 ppm NAA prior to FBD was reported to be very effective in controlling malformation (Singh et al., 1974; Majumdar et al., 1976; Anon, 1977, 1983; Singh, 1978; Chadha et al., 1979a, Singh et al., 1979; Pandey and Sharma, 1981; Majumdar and Diware, 1985, 1985; Singh, 1986; Singh and Dhillon, 1981, 1986, 1988b; Mishra and Om Prakash, 2000). Deblossoming between 20th January and 25th February regenerated new panicles in the same season. Regenerated panicles bore fruits similar to healthy ones. Deblossoming after February failed to regenerate panicles (Tripathi and Ram, 1998). However, others reported that there was no effect of deblossoming in reducing the intensity of malformation (Singh and Dhillon, 1986b, 1988b, 1990b; Bains, 1989).

Since the process of deblossoming is cumbersome and it is advisable to develop a chemical for deblossoming. Application of 200 and 500 ppm ethereal completely controlled malformation (Chadha et al., 1979a). 250 ppm of cycloheximide was also found very effective in deblossoming the panicles (Pal and Chadha, 1982). Application of 750-6000 ppm Dikegulac and 500 ppm ethereal at bud burst stage were ineffective (Singh and Dhillon, 1986b).

Use of plant Hormones and other growth regulators; Spray of NAA (100-200 ppm) during October has been suggested to reduce floral malformation (Majumdar et al., 1976; Shant, 1975; Mishra, 1976; Anon 1977;
Singh et al., 1977, 1979, 1983; Bajpai and Shukla 1978; Chaddha et al., 1979; Mehta et al., 1986; Singh and Dhillon, 1986; Mishra et al., 1998). Later, four sprays of 250 ppm NAA at weekly intervals from 20th October onwards have been resulted in maximum reduction of malformation (Mishra and Dhillon, 1978). Spraying 200 ppm NAA in first week of October followed by spraying of 500 ppm etheral at bud inception stage during February was highly effective in reducing floral malformation (Singh and Dhillon, 1986). Application of NAA (200 ppm) in the first week of October followed by deblossoming in late-December to January reduced the incidence of malformation (Mishra and Om Prakash, 2000).

Spray of gibberellic acid (50 ppm) at flower bud differentiation stage delayed pannicle emergence, increased number of perfect flowers, increased pollen viability and thus, reduced malformation considerably (Kachru et al., 1971; Shawky et al., 1978; El-Beltagy et al., 1980; Das et al., 1989). Application of 400 ppm Ethaphon at bud inception stage significantly checked the floral malformation. However, attempts to control or reduce the disease by spraying NAA (200-400 ppm), GA3, chloremquat-bayfolan mixture and ethaphon failed (Rana, 1992).

Treatment with antimalformation like glutathione (560 ppm), ascorbic acid (1055 ppm), K2 S2 O3 (560 ppm), silver nitrate (2400 ppm) and NAA (200 ppm) resulted in 87, 93, 80 and 40% conversions to healthy; panicles respectively (Ram and Bist, 1984; Bist and Ram, 1989). Substantial reduction in floral malformation by antimalformin spray at flower bud inception
stage was observed (Singh and Dhillon, 1989, 1990). Spraying of 1000 ppm paclobutrazo (10-16 g/tree), prior of FBD, during the first week of October, reduced malformation, increased number of healthy flowers and increased yield (Dhillon and Singh, 1989).

**Application of insecticides:**

Control of mango malformation has been attempted by using various insecticides and acaricides. Claims have been made that the application of insecticides, viz., 0.04 percent Diazinon and chlorobenzilate (Singh, 1956), two sprays with 0.32 percent Diazinon (Sirigh 1957; Prasad et al., 1965), methylbromide (30-40 mg/litre) fumigation for 1.30 hour (Seth and Nariani, 1966), 0.03 percent Diazinon (Rai et al., 1966; Rai and Singh, 1967; Yadav, 1969; Yadav and Varma, 1969), adicarb, apocarb, diamethoate and phorate (0.05 g/plant) as soil application (Varma and Yadav, 1970), 0.15 percent trithion (Prasad and Singh, 1972), 0.1 per cent diazinon, monocrotophos, phosphamidon (Srivastava, 1974) and 0.3% phosphamidon, 0.1 per cent parathion, methyl demeton, 0.25 per cent WP. sulphur (Doval et al., 1976) minimised malformation. Pruning followed by a spray of insecticides viz., follidol and/or maetasystox as a control measure was recommended (Giani, 1965). Aceria manifera was effectively controlled by a spray of 0.15% phosphon or Formothion (Wafa and Osman, 1972). Significant reduction in the malady was reported by applying various insecticides (Giani, 1965; Dickman et al., 1982). However, application of insecticides/acaricides was
not helpful in checking the appearance of malformation (Khan and Khan, 1960; latif et al., 1961). Spray application of dichrotophos (0.24%) and phorate (0.2%) failed to reduce malformation (Bindra and Bakhetia, 1971). Likewise, consistent reduction in panicle malformation by the spray of dimethoate (0.06%), dicofol (0.04%), formathin (0.05%), diazinon (0.4%) and phosphamidon (0.04%) was not observed (Chadha et al., 1979a).

**Application of fungicides:**

The association of *Fusarium* sp. with the malformed tissues necessitated the use of systemic fungicides for controlling the disease (Varma et al., 1971). Pruning in combination with spray of Bavistin was recommended by same group of workers. It was also concluded that copper fungicides were superior to organic fungicides due to excellent tenacity under monsoon conditions. The studies with screening of 34 fungicides for their fungicidal and fungistatic action revealed that Benlate, Brestan, Busan, Captan, Dithane M-45, Banogan and Thiram were the most effective (Varma et al., 1971). Captan at 0.1% concentration was found to be the most effective in inhibiting spore germination of the fungus (Summanwar and Raychaudhury, 1969).

There was some recovery seen in the plants treated with captan and phorate after pruning (Bindra and Bakhetia, 1971). Khurana and Gupta (1973) reported that treatments of fungicides in combination with pruning of malformed shoots gave beneficial results. Spray of captan, dithane (3 g/lit.) and benlate (1 g/lit.) was not found effective in reducing the malady (Ibrahim et al., 1975)
while spray of 0.1% Bavistin on the appearance of symptoms reduced the malformation considerably (Sharma and Tiwari, 1975). Control of the disease by benomyl and sensitivity of the isolated *Fusarium* sp. to benomyl were also reported (Sharma and Tiwari, 1975). The inhibitory effect of different fungicides viz., Fytolan, Hexaferb and Captan on mango plants artificially inoculated with *F. moniliforme* var. *subglutinans* was studied (Chattopadhyay and Nandi, 1977). Fytolan was maximally effective at all concentration followed by Hexaferb and Captan, effective to an extent at 0.1% concentration. In view of experimental evidences and economy of Fytolan, it may be recommended for controlling malformations of mango in areas where the disease is a serious problem (Chattopadhyay and Nandi, 1977). Foliar spray with Bavistin (200 ppm) provided maximum (95%) disease reduction in Dashehari followed by 91.3% in Cahusa (Mehta et al., 1986).

However, spraying of benlate (0.02%, captan (0.3%), difolatan (0.3%), demosan (0.1%), dithance M-45 (0.3%), Karathane (0.1%) aureofungin (100 ppm), fytolan (0.3%), bavistin (0.1%) and methyl-3- benzimidazole carbamate (0.2%) failed to check malformation (Chadha et al., 1979b). Benomyl failed to control the problem in South Africa (Dickman et al., 1982) but some success in reducing disease severity by spray application of benomyl has been reported in India (Siddique et al., 1987) and in Israel (Pinkas and Gazit, 1992). Soil drenching with carbendazim decreased the population of *Fusarium* in the rhizosphere soil; by 99% and in bunchy top tissues by 56%. The disease
was not controlled in terms either of recovery of diseased seedlings or of suppressing new infections in the nursery (Kumar and Beniwal, 1992). Carbendazim through trunk injection (35 g/tree) or soil application (100 g/tree), either alone or in combination with cultural practices such as root pruning showed no improvement (Kumar and Beniwal, 1992) in tree health.

Out of three systemic fungicides, benlate (benomyl), folicur (tebuconazole) and topsin-M (thiophenate methyl) applied twice through trunk injection, the first application in September and the second in January, benlate and folicur proved highly effective giving 72.50 and 71.10% reduction in floral malformation of mango, respectively (Iqbal et al., 1998).

Four fungicidal sprays (Copper oxychloride @ 2.6 g a.i./litre) applied at monthly intervals during the vegetative period and three sprays (Captan @ 1.5g ai/litre, benomyl @ 0.25 g ai/litre and mancozeb @ 4g ai/litre) applied at fortnightly intervals from before flowering until fruit set, resulted in lower levels of initial and final disease and hence, reduced malformation considerably (Noreiga-Cantu et al., 1999).

Mehta and Vala (2001) tested 13 chemicals at various concentrations against \textit{F. moniliforme} var. \textit{subglutinans}. Out of these, carbendazim and thiophenate methyl each at 0.1% and chlorothalonil 0.5% @ 301/tree applied five times as soil drenching was found quite effective for control of the disease. However, three sprays of these fungicides at 10 days interval on disease appearance showed carbendazim 0.25% as most effective.
Other measures:

Venêer grafting is an effective control measure by way of propagation. Higher incidence of the malady was recorded in seedling trees than grafted ones (Prasad et al., 1965). The seedling trees exhibited malformation earlier than grafted ones (Jagirdar and Jafri, 1966).

Thus, many control measures have been tried but results have not been substantiated and the efficacy of these are still being questioned.