

## Review Article

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## Malformation: Impending Danger in Mango Cultivation

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Mango (*Mangifera indica* L.) is an important fruit crop in India and honored as king of fruit in the country. Among the several abiotic and biotic factors, mango malformation is the most serious disease causes up to 60 per cent yield losses and confines the mango cultivation in tropical and subtropical countries of the worldwide. Mango malformation expresses two types of symptoms on plant i.e. vegetative and floral malformation, in which floral malformation is very virulent and causes the loss of entire crop than the vegetative one. Initially the epidemiology of mango malformation is not clearly understood and conflict reports regarding the causal agent existed. While, many studies have proven that fungus *Fusarium moniliforme* var. *subglutinans* is dominant pathogen responsible for mango malformation disease and their control yet to be resolved. Hence, the objective of present reviews at offering a clear and complete picture of the various aspects of development and management of mango malformation.

**Introduction**

Mango (*Mangifera indica* L) is an Indian sub continent originated fruit crop (Mukherjee and Litz, 2011) and belongs to family Anacardiaceae. It has been cultivated more than 4000 years ago and has a wide genetic diversity with great cultural and religious significance (Popenoe, 1932 and Purseglove, 1972). Mango occupies an eminent place amongst fruit crops and is acknowledged as 'King of fruits' in the country (Purseglove, 1972). India ranks the first among world's mango producing countries. It has contributed

to 57.18 per cent of the total world production with 19.22 million tones (Negi, 2000). Despite India's share of 57.18 % in world's mango production, its productivity is very low when compared to other mango producing countries.

Mango is a complicated tree and it is always confusing to understand its pattern of vegetative and reproductive growth and its various problems. These problems are largely associated with unavailability of quality scion and rootstock.

## Geographical distribution

The Mango malformation was first time reported by Maries (Watt, 1891) at Darbhanga in Bihar, India. Since then it has been reported from Israel and Maxico (Malo and Mc Millan, 1972; Verma *et al.*, 1974), Malaysia, Pakistan (Khan and Khan, 1960), Bangladesh (Meah and Khan, 1992) and United Arab Emirates (Burhan, 1991), South Africa (Schwartz, 1968), Sudan (Minessey *et al.*, 1971), Swaziland, Uganda, Egypt (Atiah, 1955), Brazil (Flechumann *et al.*, 1970), Cuba (Padron Soroa, 1983), United State of America (Malo and Mc Millan, 1972; Ploez *et al.*, 2002), Australia (Peterson, 1986) and Sultanate of Oman (Kvas *et al.*, 2008).

Incidence of mango malformation is recorded from all part of the country but it is more prevalent in north-western part than the southern part and north-eastern of the country (Varma, 1969 and Majumdar and Sharma, 1990). In Punjab, the incidence of malformation is the highest (Singh *et al.*, 1940). It is widespread in Uttar Pradesh (Singh and Chakrarvarthi, 1935) and negligible in Bihar (Mallik, 1959) and Maharashtra (Burns, 1910). In Gujarat (Burns and Prayag, 1920), the extent is quite higher in the northern districts. Recently it is also reported from Jammu and Kashmir (Chib *et al.*, 1984), Himachal Pradesh (Sharma and Badiyala, 1990) Madhya Pradesh (Mishra, 2004), Haryana (Mehta *et al.*, 1986) and Andhra Pradesh (Kulkarni, 1979).

## Losses

Globally, it has been identified that there are number of problems faced in mango production. These problems are largely associated with unavailability of quality scion and rootstock. Malformation is the most threatening malady that causes great economic loss ranged from 5-80%

(Srivastava, 1998 and Ginai, 1965) and limits the mango production in India (Kumar and Chakrabarti, 1997) and tropical and subtropical countries around the globe (Ploetz, 2001). In India, 86 per cent mango orchards are affected from malformation, however, in South Africa only 73 per cent with 1-70 per cent of disease severity (Kumar *et al.*, 1993). The malformations are characterized by a compact mass of hermaphrodite flowers, greenish in colour, stunted in growth and remain unproductive. The malformed panicles remain intact on the trees for a considerable time period (Hifney *et al.*, 1978). Floral malformation, in contrast to vegetative one, is very virulent and can cause the loss of the entire crop due to abort the fruits shortly after they have set; hence, the yield of mango crop reduced by as much as 50-80 per cent (Kumar *et al.*, 2011).

## Symptoms

Mango malformation affects the leaf panicles and inflorescence leading to the massive reduction in yield and induces two types symptoms i.e., vegetative malformation and floral malformation (Verma, 1983).

## Vegetative Malformation

Vegetative malformation is more commonly seen on young seedlings in nursery (Ploetz *et al.*, 2002; Youssef *et al.*, 2007). Infected seedlings give rise to small shoot let's bearing small scaly leaves with a bunch like appearance on shoot apex. Many such shoots may be produced and form a bunch hence, it is also known as bunchy top (Bhatnagar and Beniwal, 1977; Kanwar and Nijjar, 1979). The seedlings, in the early stage which become malformed, remain stunted and die while the seedlings which are infected later attain normal growth above the malformed areas (Singh *et al.*, 1961; Kumar and Beniwal, 1992; Ploetz and Freeman, 2009; Chakrabarti,

2011; Freeman *et al.*, 2014). Symptoms of vegetative malformation include hypertrophied, tightly bunched young shoots with swollen apical and lateral buds. Vegetative growth occurs in several intermittent flushes, separated by resting periods with no apparent growth (Davenport, 2009; Hernandez Delgado *et al.*, 2011; Ramirez *et al.*, 2014).

### **Floral malformation**

Floral malformation, in contrast to vegetative one, is very virulent and can cause the loss of the entire crop. Affected panicles either do not set fruits or abort shortly after they have set, hence, could reduce the yield by 50-80 per cent (Kumar *et al.*, 2011). Floral malformation targets the panicles, is the most destructive as it affects trees at the bearing stage (Ploetz, 2001; Youssef *et al.*, 2007; Ploetz and Freeman, 2009; Chakrabarti, 2011). The malformed panicles are compact and overcrowded due to larger flowers and looks like a compact mass and are more green and sturdy and it bends down due to its own weight. These infected panicles do not set fruits (Ploetz, 2001; Youssef *et al.*, 2007; Ploetz and Freeman, 2009; Chakrabarti, 2011). They remain as black masses of dry tissue during summer but some of them continue to grow till the next season. It directly affects the productivity. Such panicles have numbers of flowers that remain unopened and are mostly staminate and rarely bisexual (Singh *et al.*, 1961; Schlosser, 1971; Hiffny *et al.*, 1978). The ovary of malformed bisexual flowers is exceptionally enlarged and non-functional with poor pollen viability (Mallik, 1963; Shawky *et al.*, 1980). Both healthy and malformed panicles may appear on same panicle or on the same shoot. Embryo abortion occurs at a faster rate in the malformed flowers. The severity of malformation may vary on the same shoot from light to medium or heavy malformation of panicles (Varma *et al.*, 1969a).

### **Causal agents**

The etiology of mango malformation was not well understood (Kumar *et al.*, 1993; and Ploetz *et al.*, 2001). However, many studies have proven that *Fusarium mangiferae* is the pathogen responsible for mango malformation disease and Koch's postulates have been completed successfully with this fungus in various countries worldwide (Kumar *et al.*, 1993). Although the cause of malformation has been controversial, the evidence of fungal pathogen, and eriophyid mite has been suggested by various workers (Hassan, 1944; Narasimhan, 1954). Besides these biotic factors the several abiotic factors viz., nutritional deficiency, physiological disorder and hormonal imbalance (Iyer *et al.*, 2009 and Kumar *et al.*, 1993) and Carbon / Nitrogen ratio of shoots for the causal agent of mango malformation (Tripathi, 1955; Mallik, 1963) have also been suggested from time to time.

### **Disease cycle**

Disease cycle of mango malformation was not well understood till date (Gamlieb-Atinsky *et al.*, 2009b; Kumar *et al.*, 1993; Ploetz *et al.*, 2001). The disease spread by grafting through which malformation is moved in new area ((Kumar *et al.*, 1993). Primary spread has also been clearly demonstrated nursery (Prakash and Srivastava, 1987), infected nursery stock (Haggag, 2010) and mango bud mite. The presence of *Aceria mangiferae*, the mango bud mite, within buds increased frequency and severity of bud colonization by the pathogen, indicating that the bud mites may enhance fungal infection Gamlieb-Atinsky *et al.*, (2009a). However, dissemination of pathogen within-tree and tree-to-tree in nurseries and orchards is yet to be understood (Ploetz, 2004). Number of reports indicates that the disease moves slowly in infected orchards (Kumar and Beniwal, 1992). The pathogen did not survive for extended periods

in soil under natural conditions and is not seed borne. Infected seedlings cultivated beneath trees were most likely affected by inoculums originating from mature panicles and not by systemic infections from soil borne inoculums since levels in the soil and lower stem sections were very low compared to levels in upper stem sections and apical meristems (Youssef, *et al.*, 2007).

In recent years from studies, it is evident that the malformed inflorescences serve as the primary source of inoculums which disseminate passively in the air as conidia are blown or fall from dry malformed inflorescences as dry debris. Conidia land on the foliage and reach the infection site, namely, the apical bud. Systemic colonization and infection by the fungus was not evident for this host-pathogen interaction, since there was no infection via roots or survival in soil, there was no continuum of colonization via the vascular tissues, and, the pathogen is concentrated within apical and lateral buds, only at but not between the nodes (Freeman *et al.*, 2015). It was also reported that *F. mangiferae* remained viable in various parts of the trees for up to seven years, indicating that the pathogen survives for long periods in woody portions of the tree, however, only where lateral buds were present (Lahav *et al.*, 2001). This further indicates that the pathogen infects locally, at the buds, and is not transferred systemically via the vascular system.

### **Abiotic factors**

#### **Malformation with respect to season**

Seasonal variations in the occurrence and severity of malformation correlate with ambient temperature at flowering (Majumdar and Sinha, 1972). In Egypt panicles appearing on spring shoots are most severely affected (Shawky *et al.*, 1980). In Florida the heaviest

infection occurs under unusually wet conditions (Campbell and Marlatt, 1986). In India, the direction of disease gradient curves corroborated with the direction of rain drop drift in June-July (Kumar and Chakrabarti, 1997). The incidence of mango malformation has been found to vary from season to season. It is more prevalent in main flowering season the off flowering season (Majumdar and Sinha, 1972). This seasonal variation of disease incidence in mango is due to the environmental parameters, host metabolites and mangiferin content (Chakrabarti *et al.*, 1997; Chakrabarti and Kumar, 1998). The disease development was prompted by high rainfall but affected by high temperature and bright sunlight. As regard the height, the malformed seedlings initially were taller than the healthy ones.

#### **Malformation with respect to temperature**

The temperature apparently has a key role in disease development. In India, the mango malformation is present in all mango-producing areas (Verma *et al.*, 1971), with a lower incidence in the southern and eastern than in the northern region where mean temperature during flowering remains between 10-15°C. It is mild where corresponding temperature is 15-20°C, sporadic at 20-25°C and nil over the 25°C. This trend is also reflected in the world distribution of the disorder and its incidence is mainly recorded where mean temperature during winter is < 16°C. However, the temperature does not appear to have a significant role when symptoms have already developed on the panicles, because affected panicles continue to grow and producing excessive growth in dry hot summer (Verma *et al.*, 1969a-c). Earlier emerging floral buds are the most severely damaged, whereas later ones escape the disease (Kumar *et al.*, 1993). Escape was attributed to the occurrence of relatively high temperature during panicle

development. Coincidentally, a study of seasonal variation of the population density of *F. moniliforme* on mango shoots indicated that spore density reached a maximum in February, when temperature ranged from 8 to 27°C and the humidity was 85%, and that a decline of spore density coincided with hot, dry conditions (Kumar *et al.*, 1993). Majumdar and Sinha (1972) reported that the incidence of mango malformation has been found to vary from season to season, but the causes of such variation are not well defined. However, it was observed that the variety Neelum, which had 59 per cent floral malformation in the normal flowering season (March), had only 4-5 per cent floral malformation in June flowering. Incidence of Mango malformation was observed to be 20 per cent at 400 meter altitude, while almost all plants were found to be free from floral malformation at an altitude of 1250 meter 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). Puttarudrih and Basavana (1961), Singh *et al.*, (1992) and Chadha *et al.*, (1979a) reported that the occurrence of malformation differed according to the age of plants. They recorded more disease in young plants than in older ones. Singh *et al.*, (1961) concluded that about 91 per cent disease incidence was recorded in 4-8 years old plants and only 9.6 per cent in older plants. Age of flowering shoots also influence the incidence of floral malformation (Verma *et al.*, 1983a)

### **Malformation with respect to soil moisture**

Mango malformation is caused by excessive soil moisture. Because of vegetative activity during the fruiting period, inflorescences produced in April or later were changed into leafy structures (Burns, 1910). The disease was therefore held to be a physiological disorder (Burns and Prayag, 1920), not

transmitted by fungi, insects, or through sap. In tropical conditions, flowering occurs after a period of drought. Water stress advances floral bud break by nearly two weeks in about 40% buds of mango. It stimulated growth of floral buds and delayed the vegetative buds. Low temperature after a period of drought has been shown to be beneficial for flower initiation (Schole field *et al.*, 1986). Lu and Chacko (2000) and Tahir *et al.*, (2003) reported that the controlled soil water deficit lasting five weeks was demonstrated to promote earlier and more intense flowering in mango trees. Further, they reported that flowering was found dependent on drought stress. Sharma *et al.*, (2015) reported that increase in soil moisture content delayed the bud initiation and panicle emergence. The minimum intensity ( $2.7 \text{ m}^2$ ) and severity (24.7%) of malformation was recorded under restricted soil moisture conditions. The higher moisture content in soil increased the intensity ( $4.5 \text{ m}^2$ ) and severity (42.7%) of malformation. Planting direction of mango plant may also affect the malformation incidence. The panicles of north direction were most severely infested with malformation whereas, the canopy facing south direction was least infested with this malady.

### **Malformation with respect to Plant nutrients**

Several attempts were made to apply nutrients to diseased trees to affect a cure and at the same time establish nutritional imbalance as a cause of the disorder, have had mixed results. In some cases, nutrient application has improved the nutritional status of the trees and led to increased fruit yield. However, inability to reproduce these results and the unconvincing rationale supporting the disease etiology detracted from the initial successes. It is found that very little differences in the mineral constituent of healthy and malformed

tissue, however, micronutrient deficiency, especially iron and zinc have been reported to be associated with the causation of malformation (Abou El dahab, 1975; Martin – Preveli *et al.*, 1975; Minessey *et al.*, 1971; Singh and Rajput, 1976). However, zinc sulphate alone or in combination with growth regulator (NAA) could not reduce the diseases incidence in Amrapali variety of Mango (Pandey and Pandey, 1997b). El-Beltagy *et al.*, (1980) concluded that soil treatment with Bayfolan (containing, N, P, K, Mn, Fe, Cu, Mg, B, Zn, Ca and Mo) @ 100ml per tree in 22 years old mango tree cv. Taimour also did not affect the coverage of malformation. However, Singh *et al.*, (1991) suggested that the vegetative malformation was found to be associated significantly with higher amounts of all nutrients except Ca which was significantly higher in healthy seedlings. Hence, point to lower Ca could be act as one of the pre-disposing factors causing malformation in mango (Singh *et al.*, 1991). Singh *et al.*, (1994) analyzed the contents of Fe, Zn, Mn, Cu and Co in malformed and healthy panicles of mango leaves and shoots bearing fully swollen bud, bud inception, fully grown panicles prior to full bloom and at full bloom. They found that the concentrations of Fe, Zn, Cu, Mn and Co, in both malformed and healthy part of plant are not significantly differed to each other; hence, malformation in mango is not caused by the deficiency of these micronutrients.

Number of reports indicates that the shoots of malformed panicles had lower (Pandey *et al.*, 1973, 1977) as well as higher (Mishra, 1976) levels of nitrogen than the healthy tissues. This might be due to varietal response and varying soil conditions. Enhanced nitrogen application was found to reduce malformation incidence, whereas addition of Phosphorus and Potash increased the disease incidence significantly (Kanwar and Kahlon, 1987). The minimum number of malformed panicles and

higher number of healthy panicles was observed in NPK treated plant. In addition, N (1000 g/ plant) P (750 g / plant) K (750 g / plant) treated plant exhibited higher length of healthy panicles. Moreover, NPK treatment showed lowest malformation intensity percentage as compared to other fertilizer treatments (Muhammad Azam *et al.*, 2020). Application of NPK in 9:3:3 ratio is not curtail the disease severity and concluded that the malformation is not directly associated with nutritional imbalance (Bindra and Bakhetia, 1971) these may influence the incidence of the malady.

### **Malformation with respect to Plant growth regulators**

The malformation was related to hormonal imbalance at flower bud differentiation (Jagirdar and Jafri. 1966). More recent reports (Kumar and Beniwal, 1992) indicate that hormonal imbalances in the malformed tissues are attributable to metabolic changes stemming from host-parasite interactions.

### **Biotic factor**

Recently three major biotic factors are reported to be pathogen of mango malformation disease which is directly or indirectly responsible to spread disease incidence. These factors include eriophyid mites, viruses and fungi.

### **Eriophyid mites**

Theory of mites as a causal agent of mango malformation came into light in 1944 when Hassan (1944) and Sayed (1946) reported the mite *Aceria mangiferae* from Egypt. Similar findings were also recorded in India by Narasimhan (1959) who, claimed eriophyid mite could be causal organism and found it inter and intracellular in the meristem and tender regions. Several species of mites,

including predatory species, were reported to be associated with the disease (Singh, 1955, 1975; Narayana and Ghai, 1961, 1963). However, several other researchers suggested that there is no correlation of mite population and bud malformation incidence (Prasad *et al.*, 1965, 1972; Labuschang *et al.*, 1993). The role of mite in wounding and as an vector of fungus (*Fusarium moniliforme* var. *subglutinans*) was admitted by Summanwar (1967), Summanwar and Raychoudhury (1968) and Pinkas and Gazet (1992), Kumar *et al.*, (1993), Ploetz *et al.*, (1994) Gamliel-Atinsky *et al.*, (2009).

### **Viruses**

The Mango malformation occurs on both grafted and seedlings plants and incidence increases slowly, hence Sattar (1946) considered the mango malformation of viral nature. Mallik (1963) transmitted the disease successfully from branch to seedlings, seedlings to scions and seedlings to seedlings by grafting, budding, or by dodder and considered virus was the main cause of the diseases just after failure of physiological theory. Though, the findings of Singh *et al.*, (1961), Kausar (1959), Prasad *et al.*, (1965), Bindra and Bakhetia (1971), Beniwal and Bhatnagar (1975) were fail to transmit disease through grafting, budding, mechanical inoculation or through insect from branches to seedlings, seedlings to seedlings, seedlings to scions. The results thus obtained from these studies do not suggest that mango malformation is of viral etiology (Verma, 1983b).

### **Fungus**

In India Summanwar *et al.*, (1966) and Varma *et al.*, (1969) were first time to report that the floral and vegetative malformation in mango was caused by *Fusarium moniliforme* (recognized later as *F. subglutinans*). Verma

*et al.*, (1969a, b, 1971, 1972, 1974a) consistently isolated fungus the *Fusarium moniliformae* from various parts of affected malformed plants and Koch's postulates have been completed successfully with this fungus in various countries worldwide (Crookes and Rijkenberg, 1985; Freeman *et al.*, 1999; Kumar *et al.*, 1993; Varma *et al.*, 1974).

Today, it is well cited and confirmed that a fungus *Fusarium moniliforme* (*Gibberella fujikuroi*) var. *subglutinans* is the dominant causal agent of mango malformation (Campbell and Marlatt, 1986; Salazar- Garcia, 1995; and Kumar *et al.*, 1997, Ploetz and Gregory, 1993 and Britz *et al.*, 2002). *Fusarium mangiferae* has been identified in China, Egypt, India, Israel, Malaysia, Oman, South Africa, Spain, Sri Lanka and the USA, and appears to be the most common causal agent of MMD worldwide (Freeman *et al.*, 2014c). A second mango malformation disease causal agent, *F. sterilihyphosum*, was described from South Africa (Britz *et al.*, 2002) and Brazil (Lima *et al.*, 2009), while another causal agent, *F. mexicanum* was described exclusively from Mexico (Otero Colina *et al.*, 2010).

A fourth recently described species, *F. tuiense* sp. Nov. (Resembling *F. sterilihyphosum*), has been shown to cause malformation in Brazil (Lima *et al.*, 2012), Senegal (Senghor *et al.*, 2012) and Spain (Crespo *et al.*, 2016). Most recently, *F. pseudocircinatum* has been described as an additional mango malformation disease causal agent in Mexico and the Dominican Republic (Freeman *et al.*, 2014; Garcia Lopez *et al.*, 2016). In addition, *F. mangiferae*, *F. proliferatum*, *F. pseudocircinatum* and other *Fusarium* species have been isolated from affected mango in Australia (Liew *et al.*, 2016). All *Fusarium* species responsible for mango malformation disease cause similar disease symptoms.

## Management

Management measures of mango malformation shown inconsistency since a reduction in disease incidence was observed in some orchards but not in others (Chakrabarti, 1996). The scientists have been

very keen to tackle the mysterious malformation through different methods viz., physical alteration, chemical spray, plant growth regulators, botanicals, and biocontrol agents. Some of the recent findings are targeted to decrease the incidence and increase the yield.

**Table.1** Response of different mango cultivars against Mango malformation

S.No.	Varieties with incidence (%) of malformation	References
01	Collector, Langra and Neelum (2-8%), Anwar Rataul (45-50%), Alphanso (70-95%), Dusehri (15-69%), MaIda (50-90%), Samar Bahisht (20- 98%).	Khan and Khan (1960)
	Amrapali (57.12%), Bombay Green (56.25%), Mallika (55.0%), Langra (9.37%), Totapuri (16.53%) and Alphonso (17.25%)	Badliya and Lakhanpal (1990)
02	Bombay Green, Dashehari, Lucknow safeda and Chousa showed 10.8-24.2% and Baramasi (0.32-1.92%)	Ram <i>et al.</i> , (1990)
03	Neelum (17.39-29.24), Bangalora ( 18.13 - 19.45%) Kesar (15.39- 18.49%) and Krishnabhog (9.6 - 10.23 %),	Singh <i>et al.</i> , (1994)
04	Kensington (19.2 %), Mallika (12.3%), and Dashehari (4.6%)	Yadav and Singh (1995)
05	Resistant cultivars- Bhadauran, H-8-1, Ellaichi, Rataul	Singh <i>et al.</i> , (2012)
07	Tomy Atkin (54 - 17%)	Sao-Jose <i>et al.</i> , (2000)
08	Anwar Rataul (56.63%), Chaunsa ( 44.05% ), Malda (43.05 %), Dashehari (36.73%), Langra( 34.48%) and Sensation (16.51%).	Ahmad <i>et al.</i> , (2002)
09	Sindhri (36.24%), Anwar Rataul (31.02 %) and Dusehri (26.83% )	Iqbal, <i>et al.</i> , (2004)
10	Lab-e-Mashooq' (68%), G.M. Wala (64.28%), 'Sensation' (7.2%) and 'GulabKhas' (7.8%).	Ishfaq <i>et al.</i> , (2008)
11	Desi (46%), Dashehari (38%), Almas (34%), Chaunsa (30.4%) and Sindhri (26%) Neelum (22.0%), Langra (20.8%), Swarnarika (18%), Siroli (16%) and Fajri (14%).	Khaskheli <i>et al.</i> , (2008)

## Host Resistance

Severity of Mango malformation was varied from variety to variety and tree to tree of the same variety (Azzous *et al.*, 1978; Nath *et al.*, 1987, Soni *et al.*, 2013). The early and mid season mango varieties showed lower incidence of the diseases than late blooming varieties (Nirvan, 1953; Singh *et al.*, 1961; Khurana and Gupta, 1973). Monoembryonic

mango cultivars like Dashaheri, langra, Chausa, Malda and Safeda and Polyembryonic like Carabao, Peach, Cecil and Turpentine were affected by malformation (Prasad, *et. al.*, 1972). The degree of incidence of disease in various cultivars is presented in table 1, which indicated that none of the cultivar is completely resistant to malformation. Elaichi, Alib, and Bhadauran are completely free from

this disorder but their fruit quality is not good hence their use is restricted to only for resistant (Singh *et al.*, 2017).

### **Selection of planting materials**

New Orchard should be established with pathogen-free nursery stock. Scion material should never be taken from an affected orchard, and affected plants that are observed in the nursery should be removed and destroyed. Nurseries should also not be established in orchards, especially where affected by malformation. This practice is common in Egypt and India, two of the most severely affected areas (Ploetz, 2001).

### **Pruning**

Moderate pruning of 20 cm shoot bearing malformed panicles in the month of January at panicle emergence stage can be effective in suppressing the incidence of malformation in cv. Dashehari (Sirohi, *et.al.*, 2009), which is usually very high in early emerging flower buds and panicles, (Singh, *et al.*, 1974).

Pruning of shoot probably removes malformation inducing principle (Kumar *et al.*, 1993) which accumulates at the shoot tip. Conventionally, affected terminals and the subtending three nodes are cut from trees, removed from the field and burned. If these measures are practiced for 2 or 3 consecutive years, the disease can be reduced to insignificant levels. Thereafter, the disease can be kept in check by removing symptomatic tissues every other year (Muhammad *et.al.*, 1999; Ploetz, 2001). In south Africa, (Darvas, 1987) and United States (Campbell and Marlatt, 1986) the only control method recommended commercially is the pruning of infected branches while in Mexico, pruning after harvest at 80 and 30 cm from the affected zone maintained the lowest bud deformation(Lopez-Estrada *et al.*, 2005).

### **Nutrient management**

The nutrient application has improved the nutritional status of the trees and led to increased fruit yield. Partial control of malformation has recently been claimed in India by Chakrabarti, and Ghosal (1989), while these claims have also been refuted (Rajan, 1986), by spraying the diseased plant parts with mangiferin-Zn<sup>2+</sup> and mangiferin-Cu<sup>2+</sup> chelates. The normal balance of mangiferin and micronutrients revived in the diseased tissues, and the population of Fusarium declined (Chakrabarti, and Ghosal (1989). In South Africa (Darvas, 1987) FM was effectively controlled and significantly higher fruit yields recorded after two years of trunk injection with phosetyl-AI + Zn + B mixture.

The treatment significantly raised Zn levels in the leaves without effectively reducing Fs population in the MIs. A direct inhibitory effect of chemicals against the pathogen is inferred, together with secondary control through the improved nutritional status of trees.

The incidence of floral malformation was lowest (20%) in trees treated with Rogor + Multiplex + urea and highest (55.37%) in the non-treated trees. Similarly, the incidence of vegetative malformation was lowest (6.5%) in trees treated with Rogor + Multiplex + urea compared with 28.12% in non-treated trees (Thakur *et al.*, 2000).

The effect of different combinations of N, P and K on incidence of malformation of mango (*Mangifera Indica*. L), Cv. Dusher was studied with each treatment of N (1000 g), P (750 g) and K (750 g) was applied in soil during February and August, results indicates that the minimum number of malformed panicles and higher number of healthy panicles was observed in NPK treated plant.

### Hormonal management

The use of chemical substances as foliar application proved to be effective in reducing Mango malformation disease, because they may delayed or advanced the beginning of flowering (Shawky *et al.*, 1978 and Nunez *et al.*, 1986). In addition, the application of GA3 at 50 ppm reduced flower malformation of Taimour mango trees (Shawky *et al.*, 1978 and Azzouz *et al.*, 1980 and 1984).

Application of Naphthalene acetic acid at 100ppm, or at 200 ppm in October reduced the incidence of malformation in the following season particularly at the higher rate (Majumder *et al.*, 1970, 1976 and Majumder and Diware, 1989; Mahrous, 2004). The incidence of floral malformation was reduced most by using NAA at 100 ppm and also by IBA at 200 ppm (Singh and Dhillon, 1986) prior to flower bud differentiation.

### Fungicides

To date, no effective chemicals are available for disease control or for that matter for ‘curing’ infected trees. Among various fungicides, Prochloraz is an imidazole group of fungicide was the most efficient fungicide to decrease the disease incidence when applied at the interval of three weeks along with the removal of the malformed plant parts at regular intervals results in a notable decrease of incidence (Magaritha *et al.*, 2018, Freeman *et al.*, 2014)). The Spray of Benomyl 50 WP @ 2g L<sup>-1</sup> water results in 70.37% reduction of disease incidence as compared to previous year (Zafar Iqbal *et al.*, 2011).

### Antibiotics

Isolated *Streptomyces aureofaciens* was chosen as antagonists to *Fusarium*

*moniliforme var. subglutinans*, the causative agent of MMD. Bioactive metabolites secreted by *S. aureofaciens* were measured as growth reductions of *F. moniliforme var. subglutinans*.

The effectiveness of the bioactive metabolite produced by of *S. aureofaciens* at 1:5 concentrations against vegetative buds malformation disease of mango seedlings under artificial infested conditions were determined (Haggag *et al.*, 2014)

### Integration of Management Measures

Integrated management practices includes sanitary pruning, incorporation of organic matter to the soil, control of vectors, irrigation management, balanced chemical fertilization, protection of new buds, weed control and promoting anticipated blooming (GIIM, 1998; Noriega *et al.*, 1999) may keep the disease severity below those economic loss level.

### References

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