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MANGO MALFORMATION: A REVIEW

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ABSTRACT:

Mango malformation is a serious malady in mango production, occurring worldwide and causing significant economic loss due to the general incapacity of malformed vegetative and inflorescences bearing fruits. It has so far eluded a proper diagnosis of the causative agent until recently where Fusarium mangiferae and it association are revealed as the dominant causal agent of this disease. However, the control is still unresolved. Hence, this review aims at offering a lucid and complete view of the various aspects of development in mango malformation.

KEY WORD: Mango, Economic loss and malformation.

INTRODUCTION:

The malady is one of the most serious and destructive diseases of mango in nature (Prakash and Srivastava, 1987; Kumar and Beniwal, 1992; Ploetz, 2001) because of economic losses faced every year vary between 5-30% (Srivastava, 1998) or as high as 80% (Ginai, 1965). Maximum loss in India due to this deformation is 86%, in South Africa 73% of the mango farms are affected and severity varies from 1-70% (Kumar *et al*., 1993*)*.

Distribution of mango malformation disease in the world: Mango malformation was first reported in India in 1891 by Kumar and Beninwal (1991). Since then, it has also been reported from several countries in Israel, Malaysia, Pakistan, Bangladesh and UAE of Asia, South Africa, Sudan, Swaziland, Ugandad Egypt of Africa, Brazil, Central America, Cuba, Mexico, USA of America and Australia (Ibrahim *et al.,* 1975; Manicom, 1989; Ploetz and Gregory, 1993; Freeman *et al.,* 1999; Ploetz, 2001; Iqbal *et.al.,* 2004), which caused a significant impediment in increasing mango production in these countries (Ploetz *et al.* 1999).

Pattern of Occurring of the disease: The severity of the disease varies from variety to variety, tree to tree of the same variety and cycle to cycle (Azzous *et al*., 1978; Nath *et al*., 1987). Seasonal

variations in the occurrence and severity of problem correlate with ambient temperature at flowering (Majumdar & 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 & 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). This seasonal variation of disease incidence in mango is due to the environmental parameters, host metabolites and mangiferin content (Chakrabarti *et al*., 1997; Chakrabarti & Kumar, 1998).

Spread and distribution of the diseases in plant parts: Malformation is spread by grafting, by which the disease is moved to new areas (Kumar *et al.,* 1993). Spread has also been clearly demonstrated in nurseries (Prakash and Srivastava, 1987), infected nursery stock (Haggag, 2010) and mango bud mite. In Egypt, non-grafted seedlings used for production fields, are commonly cultivated directly beneath mature trees bearing malformed tissues (Ploetz *et al*., 2002). However, within-tree and tree-to-tree dissemination of the pathogen in nurseries and orchards is not well understood (Ploetz, 2004).

Most reports indicate that the disease moves slowly in infected orchards (Kumar and Beniwal, 1992). Macro- and microconidia of *F. mangiferae* are most likely the infective propagules since they are the only propagules that are produced by the fungus and form profusely on the different malformed tissues (Freeman *et al*., 2004). It appears that the pathogen does not behave as a typical soilborne fungus since conidia of the pathogen declined rapidly in soil under controlled and outdoor conditions (Freeman *et al.,* 2004). Prakash and Srivastava, (1987) in India did not detect conidia of *F. Subglutinans* (probably *F. mangiferae*) in rotary traps that were placed in an affected mango orchard. Thus, aerial dissemination of conidia of this pathogen may be uncommon. Freeman et al. (1999) transformed isolates of *F. mangiferae* from mango with the GUS reporter gene (β-glucoronidase), and used them to artificially inoculate mango. Their results verified that bud and flower tissues of the host are primary infection sites, and that wounds provide points of entry for the pathogen. The fungus *F. mangiferae* was also detedted by Lahav *et al.*, (2001) using PCR analysis in the infected sample toward the length of the branches with majority of the pathogen was observed in the grafted scions with least instance of fungal movement below the graft union. The fungus *F. mangiferae* was widely distributed in symptomatic tissues of mango obtained from diverse origins showing up to 97.0% infection (Iqbal *et al*., 2003).

Although, it was considered that the root is actually an infection court (Abdel-Sattar, 1973). Haggag *et.al*., (2010) also observed *Fusarium* isolate colonized seedling root systems and became systemic, spreading to above-ground plant tissues include apical and lateral buds. In contrast, Darvas (1987) could not detect the pathogen in roots of malformed trees. Freeman *et. al.,* (2004)

also detected no infection on the seed and seed coat of the fruit harvested from infected trees suggesting that the pathogen is not seed-borne. However, inoculum of the pathogen was isolated from the surface of these fruit, indicating that there is a possibility of survival and transmission of the pathogen on the surface of fruits picked from infected orchards but not through seedling. This was further confirmed by Youssef et. al (2007) through PCR-specific primer amplification, the *Fusarium mangiferae* was detected in 97% of seedling apical meristems, declining gradually to 5% colonization in roots and concluded that inoculum of the pathogen originates from infected panicles and affects seedlings from the meristem, with infections descending to lower stem sections and roots. Minor infections of roots may occur from inoculum originating from infected panicles, but the pathogen is not seed borne.

Causes: Mango malformation has been intriguing scientists as to its cause and control for more than 100 years (Haggag *et.al*. 2010). Studies have not yet clearly revealed either the cause or possible control measures for mango malformation (Chadha *et al.,* 1979 and Dang and Daulta, 1982). However, the following might be the causes:

Fungus and mites: Although the cause of malformation has been controversial, but fungus is one of the major possibility causes. Summanwar *et al.* (1966) and Varma *et al*. (1969) in India were the first to report that the floral and vegetative malform in mango was caused by *Fusarium moniliforme* (recognized later as *F. subglutinans*). Since then, this fungus has been shown to cause malformation in Egypt (Ibrahim et al., 1975), South Africa (Manicom, 1989), Florida (Ploetz and Gregory, 1993), Israel (Freeman *et al.,* 1999) and Sultan of Oman (Haggag *et al.,* 2010). Malformations including both floral and vegetative were reported to be reproducible by simply spraying spore suspension of *Fusarium* spp. (Chakrabarty and Ghosal, 1989; Ploetz and Gregory, 1993). In contrast to this, symptoms could not be produced unless the tissue was wounded prior to inoculation (Manicom, 1989) and artificial induction of the disease by others always relied upon wounding prior to inoculation (Summanwar *et al*., 1966; Ploetz and Gregory, 1993).

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). This fungus was subsequently referred to as *F. subglutinans*. However, *F. subglutinans sensu lato* is a very large and polyphyletic species complex that contains several host-specific taxa that cause a number of different plant diseases including ear rot of maize, pokkah boeng disease of sugarcane, pitch canker of pines, fusariosis of pineapple and malformation disease of mango (Steenkamp *et al.,* 2000). Total confusion resulted for many years because the fungi that cause this array of different plant diseases, including mango malformation disease, were all called "*F.*

subglutinans". In 2002, a new species, *F. mangiferae*, was established based on nuclear and mitochondrial DNA sequences; it included strains of *F. subglutinans* from Egypt, Oman, Florida, Israel, Malaysia, and South Africa, some of which had been shown to cause mango malformation disease by artificial inoculation (Britz *et al.*, 2002; Ploetz *et al.,* 2002, Freeman, *et al*., 2004 and Kvas *et al*., 2008). Subsequently another new group of fungus causing malformation isolates was described, which shown to be phylogenetically distinct from the *F. mangiferae* in South Africa (Britz *et al.* 2002) and was subsequently also reported to occur in Brazil (Zheng and Ploetz, 2002) . Although pathogenicity tests have not been performed with the latter isolates, their clonal relatedness and recovery from only malformed mango trees suggest strongly that *F. sterilihyphosum* also causes this disease. *F. mangiferae* and *F. sterilihyphosum* are members of the *Gibberella fujikuroi* species complex, but do not form a *G. fujikuroi* teleomorph (Leslie, 1995; Steenkamp *et al.,* 2000; Ploetz *et al.,* 2002). Morphologically *F. sterilihyphosum* can be differentiated from *F. mangiferae* because of the shorter 3-5- Based on DNA sequences for several nuclear and mitochondrial regions, *F. sterilihyphosum* forms part of the so-called "American Clade" of the *G. fujikuroi* complex (O'Donnell *et al.,* 1998) together with *F. guttiforme, F. subglutinans* Mating Population E and *F. circinatum*. A third *Fusarium* taxon was reported by Britz et al. (2002), but not formally described and its phylogenetic relatedness to other *Fusarium* species is unknown. All three of the available isolates of this species originated from Malaysian malformed mango tissue. Iqbal, *et. al.,* (2010) studied the assay of malformed parts of mango varieties in Pakistan revealed the association of four fungi viz., *F. mangiferae*, *F. pallidoroseum*, *F. equiseti* and *Alternaria alternata* while *F. mangiferae* proved to be the major infecting and dominant in association with malformed tissues of diverse origins.

A recent study indicated that genetic diversity is limited in *F. mangiferae* in Florida, Egypt, India, Israel, and South Africa (Zheng and Ploetz, 2002). Six VCGs and three random amplified polymorphic DNA (RAPD) profiles were identified among 71 isolates of *F. mangiferae* that were tested, but four of the six VCGs were characterized by a single RAPD profile (Marasas *et al.,* 2006). Thus, populations of this pathogen probably reproduce clonally. Britz et al. (2002) used a polymerase chain reaction (PCR)- based method to determine mating type (MAT-1 and MAT-2). In *F. mangiferae*, 27 isolates from Egypt, Israel, Florida, and South Africa were MAT-2 and two from Malaysia were MAT-1, whereas in *F. sterilihyphosum*, 14 isolates were MAT-1 and three were MAT-2. When isolates of opposite mating type were crossed, sexual compatibility was not observed within and between the two species.

The mechanism of fungal pathogens causing malformation may be via root, which completely colonized the seedling root systems and became systemic, spreading to apical plant tissues

including apical buds (Haggag, 2010). Apart from competition for nutrients, the fungus may release secondary metabolites, which could create further hormonal imbalance and inhibit the normal growth of the meristematic tissue of the buds (Tapan, *et al*., 2006). Fungus, which is closer to vascular channels of the mother plant, competes for the nutrients by acting as a more powerful sink than the buds of the malformed inflorescence and could be a reason for the low uptake of assimilates by the malformed buds as observed in tracer studies (Freeman, *et al*., 2004).

Mango bud mite, *Eriophyes mangiferae*, may also play an important role in the natural development of malformation, and is often observed in high numbers on malformed trees (Ploetz, 2004). It has been shown that contaminated mites act as a vector of *F. subglutinans* (*F. mangiferae*) on its body (Abdel-Sattar, 1973; Manicom, 1989), could play a role in disseminating the fungus and enabling it to infect its mango host (Singh *et al*., 1961, Doreste, 1984; Crookes and Rijkenberg, 1985) by wounding host tissues while feeding on epidermal cells of floral and vegetative buds of mango (Haggag, 2010). However, this hypothesis that mites caused the disorder did not last long as acaricides failed to control the problem (Yadav, 1999).

Stress ethylene: It is proposed that mango malformation may be due to stress ethylene (Krishnan *et.al*., 2009). The phenomenon of increasing ethylene production in response to stress is commonly called 'stress ethylene'. Production of stress ethylene in malformed trees initiate various physiological responses, which include leaf epinasty, abscission, formation of aerenchyma etc. (Abeles and Abeles, 1973), suppression of apical dominance, hypertrophy of lenticels and increased gummosis (Pant, 2000). Furthermore, the putative causal agent of mango malformation, such as excessive soil moisture, insect infestation, fungal pathogens, virus, chemical stimuli such as metal ions, herbicides and gases like SO2 etc., seem to add to the production of stress ethylene. In the light of these facts, it was suggested that the disorder may be due to the production of 'stress ethylene' by mango plants (Pant, 2000). An increased in temperature at 12 noon to 2 pm caused a heat stress which increases ethylene production and cyanide in malformed as well as healthy tissues of mango cultivars Amrapali, Khas-ul-Khas, Dashehari (Krishnan, 2003, Nailwal *et al*., 2006). The disease severity was more in field under high range of temperature variation while, less in plants kept in glass house at a constant 25^{o} C ambient temperature (Chakrabarti and Ghosal, 1985). Stress ethylene' also produced cyanide, which may result in the accumulation of toxic levels of cyanide, effect on respiration and the possibility of the development of cyanide insensitive respiration in the malformed tissue resulting in the necrosis and death of malformed tissues of mango (Rychter *et al.,* 1988 and Kukreja and Pant, 2000). Besides, ethylene (9.28 to 13.66 n mol /g dry wt/ day) was also produced by *Fusarium* sp. from mango (Ansari, 2004).

Mangiferin: Ghosal *et al*., (1979) reported that accumulation of mangiferin (1,3,6,7-tetrahydroxyxanthone-C_2-ß-D glucoside, a phenolic metabolite of mango), degraded carotenoids and toxic metabolites of *Fusarium moniliforme* has been suggested to be responsible for the malformation disease of mango (*Mangifera indica* L.). Mangiferin, a non-toxic polyphenol and a normal metabolite was reported to arrest the secretion of fusaric acid by the *Fusarium*. There is also an increase in the activity of polyphenol oxidase in infected tissues, which was considered as mangiferin degrading enzyme (Kumar and Chakraborty, 1992). Symptoms of mango malformation induced by accumulated mangiferin are presented in Table 1.

Table 1.: Symptoms of mango malformation induced by accumulated mangiferin

Effect of Mangiferin	Symptoms
1. Increased IAA content 2. Increased chlorophyll content 3. Increased photosynthesis 4. Reduced respiration and amylase activity 5. Reduced catabolism 6. Reduced transpiration	1. More vegetative growth 2. Malformed shoots/panicles look greener. 3. More carbohydrate synthesis 4. Carbohydrate accumulation disturbed C/N ratio 5. More longevity 6. High moisture content

Reference: Chakarabarti and Kumar, (2002)

Though, it was suggested that higher concentration of mangiferin in diseased tissues may lower the level of *Fusarium* sp. infection inside the diseased tissue (Chakrabarti et al., 1990). However, it does not clearly reveal the fact that *Fusarium* sp. infection prevents the translocation of mangiferin which results into its accumulation at the site of synthesis and do not predict authentic correlation between *Fusarium* sp. infection level and higher concentration of mangiferin with respect to disease incidence (Ansari, 2004).

Cultivars responses: Mango cultivars showed considerable difference among themselves in susceptiblity to malformation ; the governing factors being temperature, age of the tree, time, etc. In general, the most early and mid-season cultivars exhibit a lower incidence of the disorder than late blooming varieties (Nirvan, 1953; Singh *et al*., 1961; Khurana and Gupta, 1973). All the commercial monoembryonic 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 2, which indicated that none of the cultivar are completely resistant to malformation. The only cultivar known to be completely free of malformation is Bhadauran (Prasad *et. al*., 1965).

Table 2. Mango cultivars in relation to malformation

Sl.No	Cultivars and Severity of incidence of diseases	References
1	Collector, Langra and Neelum (2-8%), Anwar Rata	Khan and Khan (1960)

	(45-50%), Alphanso (70-95%), Dusehri (15-69%), Mal (50-90%), Samar Bahisht (20- 98%).	
2	Bombay Green, Dashehari, Lucknow safeda and Chou showed 10.8-24.2% and Baramasi (0.32-1.92%)	Ram *et. al.,(*1990)
3	Kensington (19.2 %), Mallika (12.3%), and Dasheh (4.6%)	Yadav and Singh (1995)
4	Amrapali (57.12%), Bombay Green (56.25%), Malli (55.0%), Langra (9.37%), Totapuri (16.53%) a Alphonso (17.25%)	Badliya and Lakhanpal (1990)
5	Tomy Atkin (54 - 17%)	Sao-Jose *et al*., (2000)
6	Sindhri (36.24%), Anwar Rataul (31.02 %) and Dusel (26.83% %)	Iqbal, *et. al.,* (2004)

RAPD analysis for establishing genetic variability showed that the amplified DNA fell in the range of 1400 to 350 kb. The pattern differed with each primer. Unique bands of different sizes specific to malformation were obtained with all the primers (Krishnan, 2003). The UPGMA (Unweighted pair-group method with arithmetical averages) dendrogram revealed that healthy and malformed inflorescence of each of the varieties studied were quiet distantly placed in the dendrogram further confirming genetically diverse nature of healthy and malformed inflorescence (Nailwal, 2004).

The disease has also been found to be associated with higher concentration of phosphate ion (Kaushik, 2002), physiologic disorders and hormonal imbalances (Sattar, 1946, Tapan *et al*., 2006), nutrient deficiency or toxicity (Shah *et al*., 2009), reduced nitrate reductase activity, nitrogen and soluble sugar content an increase content of starch, auxin, gibberellins, abscisic acid and some unknown type of hormone like substances were involved in mango malformation in the malformed tissues of mango (Singh, et al.,1992). Viral as causal agent has also been reported (Kausar, 1959; Das *et al*., 1989) but in contrast, Prasad, *et. al.,* (1972) attempted to transmit the disease during 1958-1964 to test the virus nature if any. They also isolated *Fusurium Moniliformae* from affected portion and inoculated in the healthy young seedling and on shoot of bearing plants. However, in both the cases, they found that none of the inoculated plants could develop a disease. Thus, this several claimed has been rejected due to lack of etiological association (Iqbal, *et. al*., 2010) and concluded that fungal association is the main causal agent of mango malformation (Freeman *et al.,* 2004; Haggag, 2010).

Symptoms: Malformation is noticed on seedlings, saplings and floral organs (Iqbal, 2004). Malformation causes gross distortions of vegetative and floral tissues in mango (Ploetz 2001).

a. Vegetatives: The disease infected young seedling in nurseries and are the most vulnerable was reported from Saharanpur (Nirvan, 1953; Kumar and Beniwal, 1992). On young seedling the disease appears at quite an early stage. Even 3-4 months old plants have been found to be affected. The malformed bunch may be at the apex or lower down at leaf axil . The seedlings produce small shootlets bearing small scaly leaves with a bunch like appearance on the shoot apices. Apical

dominance is lost in these seedlings and numerous vegetative buds sprout producing hypertrophied growth, which constitutes vegetative malformation. The multi-branching of shoot apex with scaly leaves is misshapen and have dramatically shortened internodes known as "Bunchy Top", also referred to as 'Witch's Broom" (Bhatnagar and Beniwal, 1977; Kanwar and Nijjar, 1979, Ploetz, 2004). Leaves are dwarfed, and are narrow, brittle and bend back towards the supporting stem. Shoots do not expand fully, resulting in a tightly bunched appearance of these portions of the plant. If all buds on a plant are affected, it remains stunted (Ploetz, 2004). The seedlings, which become malformed early, remain stunted and die young while those getting infected later resume normal growth above the malformed areas (Singh *et al.*, 1961; Kumar and Beniwal, 1992). **T**rees of ages, 4 to 8 years suffer the most (90.9%) from vegetative malformation (Singh *et al.,* 1961). Furthermore, the disease seriously debilitates seedlings used as rootstock and complicates the safe national and international movement of germplasm (Ploetz 2001).

b. Inflorescence: Floral malformation appeared in the panicles significantly impacts fruit production since affected inflorescences usually do not set fruit. Thus, it is more serious problem than vegetative malformation (Mahrous, 2004). The malformation of mango inflorescence has been known since 1891 (Watt, 1891). The symptoms appeared in the primary, secondary and tertiary rachises are short, thickened and are much enlarged or hypertrophied and highly branched (Kumar and Beniwal, 1992; Ploetz and Prakash, 1997). Such panicles are greener and heavier with increased crowded branching, possess numerous flowers that remain unopened, are male and rarely bisexual (Singh *et al*., 1961; Schlosser, 1971; Hiffny *et al.,* 1978). Malformation increases the number of male flowers in an inflorescence and the ovary of malformed bisexual flowers is exceptionally enlarged and non-functional with poor pollen viability or either sterile or, if fertilized, eventually abort (Mallik, 1963; Shawky *et al*., 1980; Ploetz, 2004). Both healthy and malformed flowers appear on the same panicle or on the same shoot. The severity of malformation may vary on the same shoot from light to medium or heavy malformation of panicles (Varma *et al*., 1969). The heavily malformed panicles are compact and overcrowded due to larger flowers. They continue to grow and remain as black masses of dry tissue during summer while some of them continue to grow till the next season. They bear flowers even after fruit set has taken place in normal panicles (Singh *et al*., 1961; Varma *et al*., 1969; Hiffny *et al*., 1978; Shawky *et al*., 1980) and contain brownish fluid (Prasad *et al*., 1965; Ram and Yadav, 1999). As malformed inflorescence fails to produce fruits, the damage of individual tree may vary from 50-80% and in severe cases the loss may be almost total (Summanwar, 1967). Affected panicles either do not set fruit or abort fruit shortly after they have set; yields can be reduced by as much as 90% (Ploetz 2001).

Management: The control measures of mango malformation have shown inconsistent results because a reduction in the incidence of the disease was observed in some orchards and not in others (Chakrabarti, 1996). However, a combination of some of these individual measures resulted in a better control of the disease.

- New plantings 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).
- Breed resistant cultivars to malformation and in epidemic prone areas alternate bearing and late flowering varieties should be grown (Pandey, 2003).
- 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 accumulate 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).
- Combination of pruning because it reduces the levels of inoculum in an orchard (Ploetz, 2001) and the use of insecticides, fungicides and growth regulators may control the mango malformation disease (Varma *et al.,* 1974).
- The use of chemical substances as foliar application proved to be effective in reducing Mango malformation disease, because they may delay or advance 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 Benomyl control of the disease (Sharma and Tiwari, 1975), foliar sprays 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. However, foliar applications with different fungicides (Chakrabarti and Ghosal, 1985) and acaricides like Phosphamidon (Yadav, 1999; Chakrabarti, et. al., 2001) failed in checking malformation probably due to the fungi is systemic and less role of mites.

- Partial control of mango malformation can be accomplished by spraying the diseased parts with mangiferin Zn^{++} and mangiferin Cu^{++} chelates since, mangiferin metal chelates reduced the abnormally high concentration of mangiferin in the malformed tissues and restored biochemical function (Chakrabarti and Ghosal 1989). Mangiferin treatment also increased the contents of chlorophyll, carbohydrates, total nitrogen, protein nitrogen, nucleic acids (RNA and DNA) and indole-3yl-acaetic acid (IAA) in treated plants (Chakrabarti and Ghosal, 1989)
- Integrated management package 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.
- Use of PCR-based method (species-specific primers) for accurate detection of *F. mangiferae* in plants, could prove useful in preventing the introduction of this pathogen into new germplasm (Zheng and Ploetz, 2002; Youssef, et.al, 2009).

CONCLUSION AND PROSPECTS:

More than 100 year research has gone into fact finding for the cause and control of mango malformation since 1891 and important information about the nature of disorder with regarding to it symptoms, cultivars susceptibility etc., has been revealed but the cause of these mystery disease was still not clearly understood, until recently where fungus is revealed as the dominant cause of mango malformation disease. However, the controlling strategies are still an enigma. In the future, molecular characterization by means of gene sequencing will be essential for the identification of these *Fusarium* spp. associated with mango malformation disease that are morphologically very similar to each other and to other *F. subglutinans sensu lato* lineages so that a conclusive control method could be developed and to investigate whether mycotoxin they produced possess a potential threat to human health or not. However, more work also remains to be done with respect to epidemiology and horticultural control of the disease.

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