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Research Publications on Mango Malformation

1. Ghosal, S., D.K. Chakrabarti, K. Biswas and Y. Kumar. 1979. Toxic substances produced by *Fusarium*. X. concerning the malformation disease of mango, *Experientia*, **35** : 1633-1634.
2. Chakrabarti, D.K. and S. Ghosal. 1985. Effect of *Fusarium moniliforme* var. *subglutinans* infection on mangiferin production in the twigs of *Mangifera indica*, *Journal of Phytopathology*, **113** : 47-50.
3. Ghosal, S. and D.K. Chakrabarti. 1988. Differences in phenolic and steriodal constituents between healthy and infected florets of *Mangifera indica, Phytochemistry*, **27**(5) : 1339-1343.
4. Chakrabarti, D.K. and S. Ghosal. 1989. The disease cycle of mango malformation induced by *Fusarium moniliforme* var. *subglutinans* and the curative effects of mangiferin–metal chelates, *Journal of Phytopathology*, **125** : 238-246.
5. Chakrabarti, D.K., A. Singh and K. Singh. 1990. Physiological and biochemical changes induced by accumulated mangiferin in *Mangifera indica*, *Journal of Horticultural Sciences*, **65**(6) : 731-737.
6. Kumar, R. and D.K. Chakrabarti. 1992. Biochemical evidence of physiological specialization of *Fusarium moniliforme* Sheld., the incitant of malformation disease of *Mangifera indica* L., *Indian Journal of Experimental Biology*, **30**(5) : 448-450.
7. Chakrabarti, D.K. and R.C. Sharma. 1993. Mango malformation: relation of mangiferin concentration in differentiating buds to abnormal inflorescence of *Mangifera indica*, *Annals of Plant Protection Sciences*, **1** (1) : 51-53.
8. Chakrabarti, D.K., J. Prasad and K. Singh. 1994. Effect of mangiferin on vegetative growth of mango, *Indian Journal of Horticulture*, **51**(1) : 37-40.
9. Kumar, R. and D.K. Chakrabarti. 1995. Mango malformation: effect of mangiferin on morphology and parasitism in *Fusarium moniliforme*, *Proceedings of National Symposium On Sustainable Agriculture in Sub-humid Zone,* Sriniketan, India, March 3-5, 1995, pp.348-352.
10. Chakrabarti, D.K. 1996. Etiology and remedy of mango malformation. In: *Disease Scenario in Crop Plants*, Vol. 1 (Fruits and Vegetables), (eds. V.P.Agnihotri, Om Prakash, R. Kishun and A.K.Misra), International Books and Periodicals Supply Service, Delhi, India, pp. 49-59.
11. Chakrabarti, D.K., R. Kumar and S. Ali. 1997. Mango malformation: seasonal variation in *Fusarium moniliforme* population in relation to

environmental factors, mangiferin content and flushing in *Mangifera indica*, *Indian Journal of Plant Protection*, **25**(2) : 146-148.

12. Chakrabarti, D.K. and R. Kumar. 1997. Probability of break out of mango malformation epidemic in West Bengal, India, *Acta Horticulture*,,**455** : 609-611.
13. Kumar, R. and D.K. Chakrabarti. 1997. Spatial patterns of spread of floral malformation of mango, *Acta Horticulture*, **455** : 600-608.
14. Kumar, R. and D.K. Chakrabarti. 1997. Assessment of loss in yield of mango (*Mangifera indica*) caused by mango malformation, *Indian Journal of Agricultural Sci*ences, **67**(3) : 130-131.
15. Chakrabarti, D.K., R. Kumar, Kumud and S. Kumar. 1997. Interaction among *Fusarium moniliforme, Tyrolichus casei* and mangiferin as related to malformation of *Mangifera indica*, *Tropical Agriculture*, **74**(4) : 317-320.
16. Kumar, R. and D.K. Chakrabarti. 1998. Control of malformation of mango inflorescence, *Indian Journal of Plant Protection*, **26**(2) : 174-176.
17. Chakrabarti, D.K. and R. Kumar. 1998. Mango malformation : role of *Fusarium moniliforme* and mangiferin, *Agricultural Reviews*, **19**(2) : 126-136.
18. Chakrabarti, D.K. and R. Kumar. 1999. Effects of agro-climatic condition on floral malformation of mango and its pathogen, *Fusarium moniliforme* Sheld, *Science and Culture*, **65**(11-12) : 383-384.
19. Chakrabarti, D.K. and R. Kumar. 2000. Epidemiological principles of control of mango malformation – a review, *Agricultural Reviews*, **21**(2) : 129-132.
20. Chakrabarti, D.K., R.Kumar and S. Ali. 2001. An integrated disease management strategy for mango malformation. *Proceedings International Conference on Integrated Plant Disease Management for Sustainable Agriculture*, Vol. II, Indian Phytopathological Society, IARI, New Delhi, pp. 753-754 .
21. Kumar, R. and D.K.Chakrabarti. 2001.Techniques to reproduce floral malformation of mango. *Proceedings International Conference on Integrated Plant Disease Management for Sustainable Agriculture*, Vol. II, Indian Phytopathological Society, IARI, New Delhi, pp.1121-1122.
22. Kumar, R. and D.K. Chakrabarti. 2001. Anomalies in microsporogenesis in malformed mango flowers, *Indian Phytopathology*, **54**(1) : 126-127.
23. Chand, G. and D. K. Chakrabarti. 2002. Role of metal chelates in integrated management of mango (*Mangifera indica*) malformation, *Indian Journal of Agricultural Sciences*, **72**(10) : 613-615.

24. Chakrabarti, D. K. and R. Kumar. 2002. Mango malformation : present status and future strategy. In *IPM System in Agriculture*, Vol. 8, (eds. R.K.Upadhyay, D.K.Arora and O.P.Dubey), Aditya Books Pvt. Ltd., N. Delhi, pp. 237-255.

25. Pandey, M.K., D.K. Chakrabarti and S. Kumar. 2003. Analysing mango (*Mangifera indica*) malformation in relation to the host age, *Indian Journal of Agricultural Sciences*, **73**(7) : 395-396.

26. Chakrabarti, D. K., R. Kumar and S. Kumar. 2003. Predictors for forecasting mango (*Mangifera indica*) malformation. *Indian Journal of Agricultural Sciences* **73**(11) : 633-635

27. Chand G. and D.K. Chakrabarti. 2003. Techniques to reproduce malformation in mango (*Mangifera indica* L.). *Journal of Mycology and Plant Pathology* **33**(3): 431-438.

28. Pandey, Mukesh Kumar. 2003. Some Aspects of Epidemiology and Principles of Control of Mango Malformation, *Indian Society of Mycology and Plant Pathology News*, **9**(3) : 2 (Ph.D. Thesis, N.D.University of Agriculture and Technology, Faizabad, India ; Advisor: Dr. D.K. Chakrabarti).

29. Chakrabarti, D.K. and R. Kumar. 2004. Status of mango malformation disease in West Bengal. In *Plant Pathology : Problems & Perspectives* (Eds. S.K.Raj,S.K.Pan and S.B.Chattopadhyay), B.C.Krishi Viswavidyalay, Mohanpur, West Bengal, pp. 147-149.

30. Chand, G. and D.K. Chakrabarti. 2004. Mango malformation: reproduction of malformed shoots, *Journal of Mycology and Plant Pathology*, **34**(2) : 294-296.

31. Pandey Mukesh, K and D.K. Chakrabarti. 2004. Management of malformation of mango (*Mangifera indica*), *Journal of Mycology and Plant Pathology*, **34**(3) : 881-88.

32. Chakrabarti, D.K., Mukesh Pandey, R. Kumar and Sunil Kumar. 2005. Endemicity in malformation disease of mango (*Mangifera indica*), *Indian Journal of Agricultural Sciences*, **75**(3) : 172-174.

33. Pandey, Mukesh,K., D.K. Chakrabarti and Sunil Kumar. 2005. Production and germination of conidia of *Fusarium moniliforme* var. *subglutinans* incitant of malformation in mango (*Mangifera indica*), *Journal of Mycology and Plant Pathology*, **35**(1) : 163-166.

34. Chakrabarti, D.K., Ramesh C. Shakywar, Gireesh Chand and Sunil Kumar. 2006. Control of malformation and growth promotion inmango by amino acid based metal chelates, Aminocel Gold, *Pestology*, **30**(2) : 35-39.

35. Chakrabarti, D.K. and Pinaki Chakraborty. 2006. Expert system for management of malformation disease of mango, *ICAR News*, **12**(1) : 18.

Ghosal, S., D.K. Chakrabarti, K. Biswas and Y. Kumar. 1979. Toxic substances produced by *Fusarium*. X. concerning the malformation disease of mango, *Experientia*, **35** : 1633-1634.

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 was reported to arrest the secretion of fusaric acid by the *Fusarium*.

Chakrabarti, D.K. and S. Ghosal. 1985. Effect of *Fusarium moniliforme* var. *subglutinans* infection on mangiferin production in the twigs of *Mangifera indica*, *Journal of Phytopathology*, **113** : 47-50.

The effect of *Fusarium moniliforme* var. *subglutinans* on the concentration of mangiferin was studied. The role of temperature gradient on the severity of infection and mangiferin production was examined in light of the proliferation of the fungus. In healthy plants, during March to July, there was steady fall of concentration of mangiferin. From August onwards, its concentration increased steadily and reached a peak in December. The second peak was recorded in March. In the diseased plants, there was steep fall in the concentration of mangiferin during April- June. The minimum continued up to July followed by a staggering rise and fall between August to next April. The diseased plants were found to produce shootlets and rudimentary leaves continuously while in healthy plants main flushing took place in March. The disease severity was more in field under high range of temperature variation. But in plants kept in glass house at a constant 25^{o} C ambient temperature the severity was less.

Ghosal, S. and D.K. Chakrabarti. 1988. Differences in phenolic and steriodal constituents between healthy and infected florets of *Mangifera indica*, *Phytochemistry*, **27**(5) : 1339-1343.

Differences in the low- and medium M, phenolic and steriodal compounds in healthy and malformed florets of *Mangifera indica*, the latter infested with *Fusarium moniliforme* var. *subglutinans* are reported. Mangiferin, 1,3,6,7-tetrahydroxyxanthone , which are not normal constituents of healthy florets were found in substantial amounts in the diseased florets. Both mangiferin and 1,3,6,7-tetrahydroxyxanthone were found to be potent anti-fungal agents. The polymeric quinones caused collapse of the adjoining cells. The production and accumulation of zoosteroids, pregenolone and progesterone, instead of the normal phytosterols in the diseased flowers of mango, would seem to be associated with the impairment of floral sex expression resulting in staminate flowers with emaciated ovary. Amides of aromatic

hydrocinnamic acids which are found in hermaphrodite flowers were found to be completely absent in staminate flowers.

Chakrabarti, D.K. and S. Ghosal. 1989. The disease cycle of mango malformation induced by *Fusarium moniliforme* var. *subglutinans* and the curative effects of mangiferin–metal chelates, *Journal of Phytopathology*, **125** : 238-246.

Evidence is presented to suggest that the bunchy top, die-back, abnormal inflorescence and blossom blight disease symptoms of *Mangifera indica* are inter linked and can be expressed as a disease cycle in mango malformation. In the malformed shoots the concentration of mangiferin, which is normally produced in healthy shoots of *M. indica* in a low concentration was significantly increased. In healthy inflorescence mangiferin was either absent or was present in traces. In both abnormal and blighted inflorescence the concentration of mangiferin was appreciably increased. There was also a concomitant oxidative transformation of mangiferin. In the black rotten lump of abnormal inflorescence and in the die-back affected vegetative shoots, large amount of polymeric quinones were produced. In all the infected tissues the fusarial-borne sesquiterpene, resorcyclic acid and macrolactone toxins, which cause phytotoxicity, were present. The overt chemical changes in the bunchy top, die-back, abnormal inflorescence and blossom blight symptoms could be commonly expressed as due to accumulation of mangiferin and its subsequent transformation into polymeric quinones. The pathogen was not able to spread systemically through xylem vessels because of the barrier of the anti-*Fusarium* substance, mangiferin and remained localized in the outer cells of the affected parts. The conidia grown over the dead tissues after oxidation of mangiferin were main source of inoculum. The fungus invaded the host through soft organelles, viz. vegetative and floral buds. Partial control of mango malformation was accomplished by spraying the diseased parts with mangiferin Zn^{++} and mangiferin Cu^{++} chelates. The mangiferin metal chelates reduced the abnormally high concentration of mangiferin in the malformed tissues and restored biochemical function.

Chakrabarti, D.K., A. Singh and K. Singh. 1990. Physiological and biochemical changes induced by accumulated mangiferin in *Mangifera indica*, *Journal of Horticultural Sciences*, **65**(6) : 731-737.

Accumulation of mangiferin, a natural metabolite of *Mangifera indica* at the site of differentiating buds, influences the changes from reproductive to vegetative growth. Mangiferin-induced physiological changes in *M. indica* have been studied and a link established between the metabolic deviation and the observed effects of mangiferin accumulation in healthy and malformed mango plats. Mangiferin in high concentration suppressed the activity of peroxidase, catalase, α-amylase and IAA-oxidase. Polyphenoloxidase and invertase showed increased activity. Mangiferin accumulation increased the rate of photosynthesis but lowered those of transpiration and respiration. Mangiferin treatment increased the contents of chlorophyll, carbohydrates, total nitrogen, protein nitrogen, nucleic acids (RNA and DNA) and indole-3yl-acaetic acid (IAA).

Kumar, R. and D.K. Chakrabarti. 1992. Biochemical evidence of physiological specialization of *Fusarium moniliforme* Sheld., the incitant of malformation disease of *Mangifera indica* L., *Indian Journal of Experimental Biology*, **30**(5) : 448-450.

Mangiferin acts as a phytoalexin-like compound in *M. indica*. Non-pathogenic strains of *F. moniliforme* isolated from maize cob and banana fruits induced more mangiferin synthesis than the pathogenic isolate from malformed mango shoots *in vivo*. Activity of the mangiferin degrading enzymes, polyphenoloxidase of non-pathogenic strains of *F. moniliforme* was higher than that of the pathogenic strain .*In vitro* tests also confirmed rapid and massive degradation of mangiferin by non-pathogenic strains. Biochemical events associated with elicitation, degradation and accumulation of mangiferin determine the host specificity of *F. moniliforme* in *M. indica*.

Chakrabarti, D.K. and R.C. Sharma. 1993. Mango malformation: relation of mangiferin concentration in differentiating buds to abnormal inflorescence of *Mangifera indica*, *Annals of Plant Protection Sciences*, **1**(1) : 51-53.

Concentration of mangiferin in malformed and healthy mango floral buds and its adjoining leaves at different stages of differentiation was estimated. At the stages of bud pre-emergence and initial differentiation, mangiferin content in the leaves from the axils of which healthy and malformed buds emerged did not show significant differences although in the latter one mangiferin content was higher. But in the fully developed panicles at pre-blooming stage mangiferin content of the leaves attached to the malformed panicle was in traces and significantly lower than that of healthy one. Mangiferin content in malformed panicles also did not differ significantly at the initial stage. But the difference become highly significant after influx of mangiferin from axil leaves to the fully developed panicle at pre-blooming stage. Mangiferin due to its vegetative growth promoting property tilted the hormonal balance of malformed panicles in favour of vegetative growth resulting into transformation of malformed florets into green leafy structures.

Chakrabarti, D.K., J. Prasad and K. Singh. 1994. Effect of mangiferin on vegetative growth of mango, *Indian Journal of Horticulture*, **51**(1) : 37-40.

The vegetative growth promoting effect of mangiferin, a natural metabolite of *Mangifera indica* L. was investigated on both regular (Amrapali) and alternate (Banarasi Langra) bearing cultivars of mango. Mangiferin increased percentage of seed germination, number and size of roots and length of germinated seedlings. Mangiferin also increased the shoot length and leaf size. Banarasi Langra which had lower level of endogenous mangiferin content as compared with Amrapali responded better to the mangiferin treatment.

Kumar, R. and D.K. Chakrabarti. 1995. Mango malformation: effect of mangiferin on morphology and parasitism in *Fusarium moniliforme*,

Proceedings of National Symposium On Sustainable Agriculture in Sub-humid Zone, Sriniketan, India, March 3-5, 1995, pp. 348-352.

Mangiferin induced significant changes in the strains of *Fusarium moniliforme* Sheld. isolated from maize cob and banana fruits. The mangiferin treated strains produced more aerial hyphae but less pigment. Prolonged mangiferin treatment affected the saprophytic ability of the strains but improved its parasitism. The significance of mangiferin induced changes in evolving the host-specific strain of *F. moniliforme* of *M. indicia* lies in showing that an ecological disadvantage of survival in one niche (saprophytic) may prove advantageous in another (parasite). This finding contributes towards showing yet another evidence of a given way of evolutionary diversification of parasitism.

Chakrabarti, D.K. 1996. Etiology and remedy of mango malformation. In: *Disease Scenario in Crop Plants*, Vol. 1 (Fruits and Vegetables), (eds. V.P.Agnihotri, Om Prakash, R. Kishun and A.K.Misra), International Books and Periodicals Supply Service, Delhi, India, pp. 49-59.

The intrinsic discrepancies in different hypothesis on etiology of mango malformation have been critically discussed. Role of the toxic metabolites of *F. moniliforme* var.*subglutians* and aberrant host metabolites produced in response to the pathogenic invasion have been appraised. Environmental factors conducive to malformation, spatial aspects of the epidemiology and host specificity of the pathogen have been described. A disease cycle has been proposed. Plausible reason of failure of some much published control measures viz. systemic fungicide, naphthalene acetic acid and chemicals that were used to delay flowering in delivering expected results have been envisaged. Innovative approaches to manage the disease have been suggested.

Chakrabarti, D.K., R. Kumar and S. Ali. 1997. Mango malformation: seasonal variation in *Fusarium moniliforme* population in relation to environmental factors, mangiferin content and flushing in *Mangifera indica*, *Indian Journal of Plant Protection*, **25**(2) : 146-148.

The population density of *Fusarium moniliforme* was high in the months of February-March and July-September. During the spring flush (February) the fungal population (228.0 c.f.u. mg^{-1} d.w.) was at its peak, when the range of temperature, humidity and mangiferin content were 8-27^{0}C, 84.8% and 5.75% respectively. But in May, when mangiferin content was the maximum (9.38%), the relative humidity (65%) was next to the minimum point while the maximum temperature (42^o C) was close to the highest temperature of the year, the fungal population (7.0 c.f.u mg^{-1} d.w.) was dropped. In July-September the weather became mild (33.5-32.8^o C) and humid (87-84%), mangiferin content became low (4-6.75%)and consequently the inoculum density was again pushed up (106-126 c.f.u. mg^{-1} d.w.). The low fungal density in May coincided with increment of temperature, while that of December and January with the increment in mangiferin content and fall in ambient temperature respectively. Thus, the environmental parameters and mangiferin content affect the population of *F. moniliforme* and cause seasonal variation of the disease incidence.

Chakrabarti, D.K. and R. Kumar. 1997. Probability of break out of mango malformation epidemic in West Bengal, India, *Acta Horticulture*, **455** : 609-611.

Malformation disease (incitant *Fusarium moniliforme* Sheld.) of mango (*Mangifera indica* L) on being introduced to West Bengal, with diseased planting materials from the disease hot spot area (northern India) has registered its first appearance. Probability that one spore will succeed in infecting ("p"), "doublet test", and ease with which the pathogen has established after arrival and the presence of susceptible cultivars suggest the high probability of the disease becoming established in West Bengal where the disease was hitherto absent underlying the need of domestic quarantine barrier.

Kumar, R. and D.K. Chakrabarti. 1997. Spatial patterns of spread of floral malformation of mango, *Acta Horticulture*, **455** : 600-608.

Spatial patterns of spread of floral malformation among genetically diverse cultivars of mango (*Mangifera indica* L) was investigated. The disease progress curves of both regular and alternate bearing cultivars were sigmoid and bimodal respectively. The spread of the disease was faster in regular bearers than that of the alternate bearing cultivars. The rate of spread of the disease within the plant assumed logarithmic growth after the secondary source of inoculum was established in the plant in the form of dead necrotic malformed panicles of last year. Disease gradient curves in all the cultivars were hyperbolic, but that of regular bearing cultivars was flatten near the source soon after initiation of the disease. The disease reached long distance in step wise progression. The direction of disease gradient curves corroborated with the direction of rain drop drift in June-July.

Kumar, R. and D.K. Chakrabarti. 1997. Assessment of loss in yield of mango (*Mangifera indica*) caused by mango malformation, *Indian Journal of Agricultural Sciences*, **67**(3) : 130-131.

The direct and indirect losses incurred due to malformation in yield of alternate and regular bearing cultivars of mango both in 'on' and 'off' years were assessed. Loss in yield (number of fruits) corresponding to the number of malformed panicles may be assessed through linear regression analysis. In cultivars like Langra, Himsagar, Gilas and Neelum for every 1% increase in malformed panicles, the yield was reported to decline by 0.20, 0.60, 0.89 and 0.96 per cent respectively. In years when trees bloom profusely ('on year') the intensity of malformed panicles was greater but during 'off year' the incidence of floral malformation was less. The number of total panicles up to 50% of the disease intensity increased, with further increase in the disease intensity there was sharp reduction in flowering. The disease incidence of one year did not have much bearing on flowering in the next crop season.

Chakrabarti, D.K., R. Kumar, Kumud and S. Kumar. 1997. Interaction among *Fusarium moniliforme, Tyrolichus casei* and mangiferin as related to malformation of *Mangifera indica*, *Tropical Agriculture*, **74**(4) : 317-320.

Interaction among *Fusarium moniliforme* Sheld., *Tyrolichus casei* Oudemans colonizing malformed mango (*Mangifera indica* cv. Banarasi Langra) panicles, and mangiferin (1,3,6,7-tetrahydroxyxanthone C_2-ß-D glucoside), a defensive metabolite of the host plant in relation to floral malformation , was investigated. The fungal and mite populations were initially positively related to the mangiferin content and the disease incidence. Further increase in mangiferin content reduced the fungal and mite populations; however, the increase in infection rate was not affected until the *Fusarium* population was too low. The fungal conidia remained adhered to the body surface of the mites inhabiting malformed panicles, and its plating on potato dextrose agar showed a trail of fungal colonies along the pathway of its movement. *Tyrolicus casei* facilitated the ingress of the fungus into the host cells while *F. moniliforme* served as the feed of *T.casei* and increased its multiplication.

Kumar, R. and D.K. Chakrabarti. 1998. Control of malformation of mango inflorescence, *Indian Journal of Plant Protection*, **26**(2) : 174-176.

Attempt was made to manage floral malformation of mango (*Mangifera indica* cv. Amrapali) by an integrated disease management strategy which included removal of malformed shoots and panicles twice (May and October), spraying with acaricide (phosphomidon, 0.5%) in flushing period (May, October and February),mangiferin Cu^{++} chelate (40 ppm) in October, December and January and mangiferin Zn^{++} chelate(40 ppm) at the time of panicle elongation (January and February). It appears from the results that in 1998 in comparison with the previous year the treatments resulted in an overall increase in average number of total flowers (186.1/plant) and percentage of fruits (31.83%). But there was reduction in percentage of malformed panicles (26.01%). In untreated (control) plants, there was significant reduction in percentage of fruit yield (10.01%) during the same period. Percentage of malformed panicles was reduced (7.9%) but not to the significant level. Number of inflorescence was increased marginally (28.15/plant). In 1998, the treated plants produced considerably more flowers and fruits. The percentage of malformed panicles in the treated plants was almost half of the control.

Chakrabarti, D.K. and R. Kumar. 1998. Mango malformation : role of *Fusarium moniliforme* and mangiferin, *Agricultural Reviews*, **19**(2) : 126-136.

The malformation disease of mango (*Mangifera indica* L.) is characterized by the excessive vegetative growth and transformation of floral structure into vegetative one. The uniqueness of the disease symptoms and lack of technique to reproduce the disease artificially caused the ambiguity in the etiology of the disorder. It has now been increasingly accepted that *Fusarium moniliforme* Sheld. induces the malformation and is vectored by the mites. The symptoms are the combined effects of aberrant host metabolites produced in response to infection and phytotoxins secreted by the pathogen. The pathogen has been identified as a physiological race of *F. moniliforme* (*F. moniliforme* f. sp. *mangifera*) developed due to interaction with the host metabolite, mangiferin for a prolonged period. The disease cycle is greatly influenced with the biochemical changes in the host tissues. Host metabolites also effect the seasonal variation of population of the pathogen vis-a -vis disease

incidence. Proper balance of mangiferin and the *Fusarium* population is required for disease manifestation. Either by suppressing or avoiding elicitation of hypersensitive reaction of the host at the initial stage of infection, colonization by the pathogen and subsequent symptom production could be effected. The disease incidence could be minimized by removing stress factor (the pathogen) through removal of malformed plant parts and supplying the malformed plants necessary micronutrients by spraying with mangiferin metal chelates.

Chakrabarti, D.K. and R. Kumar. 1999. Effects of agro-climatic condition on floral malformation of mango and its pathogen, *Fusarium moniliforme* Sheld., *Science and Culture*, **65**(11-12) : 383-384.

The variation in symptoms of floral malformation of mango (*Mangifera indica* L.)) in the distinctly different new agroclimatic condition and the morphological and biochemical changes of the pathogen (*Fusarium moniliforme* Sheld.) were investigated. The infected planting materials (saplings of cv Amrapali) were introduced from Uttar Pradesh, the hot spot area for mango malformation, to West Bengal. In Uttar Pradesh both summer and winter are extreme with highly variable temperature. But West Bengal, being at coastal area, is hot and humid. It has mild cold in winter and temperature fluctuation between summer and winter is minimal. Floral malformation in West Bengal was mostly of light type and did not survive long (up to March). On the other hand, malformed panicles of Uttar Pradesh were of compact type and survived up to June –July. The growth rate of the strain from Uttar Pradesh was higher. The strain from West Bengal unlike that from Uttar Pradesh showed difficulty to grow at very low (15^{o} C) or high (30^{o} C) temperature The colonies of the strain of Uttar Pradesh produced more aerial hyphae and lesser pigments. Biochemical analysis revealed that pH and C/N ratio of the strain from Uttar Pradesh were higher but had lesser amount of zinc and iron content. Abundant aerial hyphae, less pigmentation, high C/N ratio and low catabolic activity (as indicated by high pH and low iron content) helped the strain to establish better compatibility with the host. Thus, the physiologically specialized strain of *F. moniliforme* being introduced in the agroclimatic condition of West Bengal lost to some extent its virulence which was reflected in the less severe manifestation of the disease symptoms.

Chakrabarti, D.K. and R. Kumar. 2000. Epidemiological principles of control of mango malformation – a review, *Agricultural Reviews*, **21**(2) : 129-132.

An epidemiological descriptor of mango malformation has been proposed and accordingly a control strategy has been envisaged. The disease is polycyclic, the pathogen, *Fusarium moniliforme* Sheld., is polyetic and host specific. Maximum fungal population was recorded during February-March while the highest disease incidence in July-November. Latent period extended from late November to early February. New crop of conidia (propagules) on host surface was formed during July-September. The disease was transmitted by vector (mites) and infected scions. The gradient of spread was steep. The plant to plant infection was slow. Logarithmic phase started at 1.34-5.01% disease incidence. Mean maximum disease incidence in

regular and alternate bearers were 40-48 and 72-73% respectively. Pattern of epidemic in former one was sigmoid while in the latter it was bimodal. Duration of the epidemic was year round. Thus rate of plant to plant spread was slow, propagules for dissemination were available for short period. But tissues remained infectious for long and small amount of initial inoculum could start the epidemic. Therefore, rate of increase of the disease could be minimized through sanitation (pruning of infected plant parts, killing of the propagules with fungicides).

Chakrabarti, D.K., R.Kumar and S. Ali. 2001. An integrated disease management strategy for mango malformation, *Proceedings International Conference on Integrated Plant Disease Management for Sustainable Agriculture*, Vol. II, Indian Phytopathological Society, IARI, New Delhi, pp. 753-754 .

The results of an integrated disease management trial under field condition and the associated biochemical changes in the host (*Mangifera indica* L.) were reported. The maximum yield and disease reduction were obtained with pruning followed by spraying with mangiferin metal chelates. Copper chelates stopped influx of mangiferin into developing buds, decreased iron and carbohydrate content of the host, pathogen population over flower buds and its C/N ratio. Zinc chelates increased auxin and carbohydrate content but induced both flowering and malformation. Pruning stimulated flowering and decreased disease incidence but to a lesser extent. Phosphamidon seemed to affect vector (mites) population.

Kumar, R. and D.K. Chakrabarti. 2001. Techniques to reproduce floral malformation of mango. *Proceedings International Conference on Integrated Plant Disease Management for Sustainable Agriculture*, Vol. II, Indian Phytopathological Society, IARI, New Delhi, pp.1121-1122.

Symptoms of floral mango malformation were reproduced consistently by treating flower buds with cycloheximide (translation inhibitor) before inoculation or by employing mites (*Tyrolichus casei*) for inoculation. The treatments induced less accumulation of mangiferin (a host defense phenolic compound) and permitted invasion and colonization by the pathogen without much resistance from the host at the inoculation site.

Kumar, R. and D.K. Chakrabarti. 2001. Anomalies in microsporogenesis in malformed mango flowers, *Indian Phytopathology*, **54**(1) : 126-127.

Flowers of malformed mango inflorescence were mainly staminate; pollens were scanty and mostly sterile. Hence, the anomalies during microsporogenesis were investigated. Various types of meiotic anomalies were noticed. In malformed buds 2-4 chromosomes were found lying away from the metaphase plate. At anaphase bridges and laggards were of common occurrence. Gradual reduction in chromosome number was recorded from anaphase to telophase. At telophase degeneration of other organelles like cell wall of pollen mother cells was seen. Spindle anomalies like development of 3 separate parallel spindles were recorded. And finally polyads were formed.

Chand, G. and D.K. Chakrabarti. 2002. Role of metal chelates in integrated management of mango (*Mangifera indica*) malformation, *Indian Journal of Agricultural Sciences*, **72**(10) : 613-615.

Three chelating agents, viz. mangiferin ethylene diamine tetrahydroxy acetic acid (EDTA) and amino acid (aminocel), were tested for their micronutrient (Cu^{++} and Zn^{++}) mobilizing capacity in mango shoots and concomitant effects on the development floral malformation of mango incited by *Fusarium moniliforme* Sheldon var. *subglutinans* Wolleneeb and Reinking. Mangiferin, a natural metabolite of mango, served as the best chelating agent followed by amino acid and EDTA. Amount of Cu^{++} and Zn^{++} in buds of trees from which malformed shoots and panicles were not eradicated was more after micronutrient treatment but its consumption in comparison with buds of pruned trees was less. The Cu^{++} reduced the fungal population and increased Zn^{++} concentration. While the latter one helped the normal development of the buds and increase in flowering. The minimum disease incidence and maximum flowering were recorded with treatment of mangiferin Cu^{++} chelate and aminocel (containing both Cu^{++} and Zn^{++} in amino acid) respectively. Use of a mixture of Cu^{++} and Zn^{++} ions with mangiferin as chelating agent after removal of malformed shoots and panicles was suggested to manage mango malformation.

Chakrabarti, D.K. and R. Kumar. 2002. Mango malformation : present status and future strategy. In *IPM System in Agriculture*, Vol. 8, (eds. R.K.Upadhyay, D.K.Arora and O.P.Dubey), Aditya Books Pvt. Ltd., N. Delhi, pp. 237-255.

Malformed mango plants developed dwarf shoots in bunches (bunchy top) and compact ever green panicles (floral malformation). The disease is induced by *Fusarium moniliforme* Sheld. The malformed disease symptoms are the combined effects of the alternate host metabolites and the fusarial toxins. The disease is severe in juvenile stage and loss is more in "on year". The loss is linear with the disease grade. The progress curve is typical for polyetic disease. The spread of the disease is faster on regular bearing cultivars. Disease gradient is hyperbolic. The disease spread over the short distance. The mites helps the pathogen in transmission and invading host cells while it uses the fungus as feed. A proper balance among population of fungus, mite and quantity of mangiferin (host defense compound) is necessary for the disease development. The fungal density reached a maximum in February when minimum and maximum temperature ranges from 8-28^{o}C and humidity is high (85%). In hotter period the fungus population declines. The fungus is transmitted through planting materials to a new agroclimatic zone. The implication of the results in evolving disease management strategy has been discussed.

Pandey, M.K., D.K. Chakrabarti and S. Kumar. 2003. Analysing mango (*Mangifera indica*) malformation in relation to the host age, *Indian Journal of Agricultural Sciences*, **73**(7) : 395-396.

The pattern of changes in incidence of vegetative and floral malformation of 'Dashehari' mango (*Mangifera indica* L.) with host age was expressed mathematically. The disease progress curve was modeled using a set of experimental

data at Faizabad, India in 2000-2001. The model ($Y=111.8925-9.895X+ 0.2517X^2$, R^2 =0.9818, where Y number of vegetative malformation and X, hostage) was tested with a set of data obtained in Lucknow, India in 1979 and the structure of the model was validated. The close fit between the modeled and experimental curves showed the main trend of the epidemic was represented. The incidence of vegetative malformation was maximum when the plants were of 5 years old, thereafter, the disease declined. Similarly, based on the results of regression analysis equation ($Y= 17.605+7.7754X-0.3294X^2$, R^2 = 0.9989, where Y, number of malformed panicles and X, host age) for floral malformation was developed. The floral malformation up to initial 11 years showed increment and then a curvilinear decrease.

Chakrabarti, D. K., R. Kumar and S. Kumar. 2003. Predictors for forecasting mango (*Mangifera indica*) malformation, *Indian Journal of Agricultural Sciences*, **73**(11) : 633-635

An attempt was made during 1996-98 to identify predictors for forecasting incidence of floral malformation induced by *Fusarium moniliforme* Sheld. var. *subglutinans* Wollenewb and Reinking on 'Chausa' mango (*Mangifera indica* L.) under agro - climatic condition of Rudauli-Sohawal mango belt of Uttar Pradesh, India by regression analysis. In 23 plants content of micronutrients (zinc, copper, iron and manganes), C/N ratio, mangiferin and auxin content and population of *F. moniliforme* var. *subglutinans* prior to flowering were estimated in addition to vegetative malformation and total panicles. Nature of correlation of mangiferin, zinc, copper and *F. moniliforme* with the incidence of malformation varied. Auxin, manganese, C/N ratio and total panicles consistently showed negative, while iron, showed positive correlation with the disease incidence. But values of correlation coefficients were not always at significant level. Consistent significant correlation was obtained only with the vegetative malformation parameter. A linear equation for prediction ($Y= 2.1364+ 0.6968$, where , Y and X were number of malformed shoots and panicles respectively) were developed and validated by chi-square test (χ 2= 3.0434).

Chand G. and D.K. Chakrabarti. 2003. Techniques to reproduce malformation in mango (*Mangifera indica* L.), *Journal of Mycology and Plant Pathology*, **33**(3) : 431-438.

To reproduce consistently the symptoms of floral and vegetative malformation in *Mangifera indica* L. with *Fusarium moniliforme* Sheld. an inoculation technique was developed. Floral buds of cv Amrapali were inflicted with microneedle injuries during inception stage (November) and inoculated with the inoculum grown on the host tissues (inoculum stripes). Prior to inoculation buds were treated with hydrogen peroxide solution (20%) and after inoculation with cycloheximide solution (2 ppm). Hydrogen peroxide solution detoxified mangiferin (the host defense anti-fusarial compound) while cycloheximide affected is biosynthesis. Inoculated buds produced 60-67.5% malformed panicles in next March and 65-70% vegetative shoots in November. The disease developed only when the temperature was mild (8-19^o C) and RH was high (> 85%).

Pandey, Mukesh Kumar. 2003. Some Aspects of Epidemiology and Principles of Control of Mango Malformation, *Indian Society of Mycology and Plant Pathology News*, **9**(3) : 2 (Ph.D. Thesis, N.D. University of Agriculture and Technology, Faizabad, India ; Advisor: Dr. D.K. Chakrabarti).

The objectives of the study were characterization of temporal progress of malformation disease of mango and selection of epidemiology oriented control principles. A schematic diagram depicting the dynamics of the disease was envisaged. Every phase of the disease cycle viz. sporulation, invasion, colonization, symptom development was modeled separately (analytical model). The changes of the disease incidence with season and some important host factors (age, bearing habit, flowering time) that either slow down or accelerate changes from one to another state of epidemic were characterized. These observations were used to explain host-patho (*Mangifera indica* L. and *Fusarium moniliforme* Sheld. var. *subglutinans)* system and to develop predicting equations. The field experiments were conducted in the university as well as farmers orchards at Rudauli-Sohawal mango belt, Faizabad, India during 1999-2002. A brief perspective of the pathogen, climatic and host factors in epidemic frame work are presented.

Pathogen factors**.**

Dead necrotic malformed panicles or shoots were the site of conidia production. Maximum production was recorded (1,89,960 g^{-1} f.w.) in July (temp. 25-30^{0}C, RH 78-92 %). Conidia showed maximum viability in the morning hours and germinated within 5-6 h at 30^{0}C. The infection hyphae entered the host through wounds mainly at the basal part of buds and colonized in the epidermal and cortical cells. The intensity of the vegetative malformation progressed steadily from mid June to end of July (temp. 25-30^{0}C, RH > 85%) while floral malformation in February -March (temp. 9.8-19^{0}C, RH > 87%). Development of floral malformation compared with vegetative one was more sensitive to higher temperature. Once the active growth phase was over neither conductive weather nor presence of inoculum could initiate or continue the disease development. Conidia production and its germination were greatly influenced by climatic variables but invasion, colonization and symptom manifestation were largely governed by physiological conditions of host cells.

Host factors**.**

Optimum age for maximum incidence of floral and vegetative malformation in cv. Dashehari were 10 and 5 yrs. respectively. Floral malformation in regular bearing cultivars compared with alternate bearers was more. But early flowering varieties irrespective of regular or alternate bearing habit suffered more than those flowered later in warmer period.

Climatic factors.

The highest percentage of infected buds and development of malformed shoots were recorded during spring flush followed by that in autumn while minimum in summer.

Additionally, during spring and autumn the fungal population was at its peak but it was scanty in summer. Mild temperature (8-19^{0}C) and high humidity (87%) were conducive both for invasion and symptom manifestation.

Control principles**.**

Management by increasing Δt. The primary inoculum level was dropped with mangiferin Cu^{++} chelate and *Aspergillus niger* treatment Consequently lesser was floral malformation. Sanitation also reduced the disease incidence and increased yield, But sanitation increased the rate of progress of the disease, thus its effects did not sustain long.

Management by reducing 'r'. Bavistin affected growth of germ tube and conidia production, thus reduced the infection rate. But it required longer period to bring down the disease level.

Recommendations. In epidemic prone areas alternate bearing and late flowering varieties should be grown. Buds emerged from spring and autumn flushes should get prophylactic spraying with Cu^{++} chelate and a curative spray with bavistin in October during flower bud initiation. Young buds should be protected from insect injuries with acaricides. Systemic and regular removal of malformed panicles and shoots may be practiced. Special care must be taken for 5-10 year-old plants.

Chakrabarti, D.K. and R. Kumar. 2004. Status of mango malformation disease in West Bengal. In *Plant Pathology : Problems & Perspectives* (Eds. S.K.Raj,S.K.Pan and S.B.Chattopadhyay), B.C.Krishi Viswavidyalay, Mohanpur, West Bengal, pp. 147-149.

Malformation disease of mango (*Mangifera indica* L) induced by a physiological strain of *Fusarium moniliforme* Sheld. var. *subglutinans* Wollenewb and Reinking is assuming a serious form in West Bengal, India. The pathogen seems to be introduced in the state with large scale planting materials from northern India where the disease is endemic. The pathogen has succeeded to survive and initiate fresh infection in new agro-climatic conditions. From the source plants the disease is spreading to the local cultivars. Due to high mutable nature of the pathogen and high susceptibility of local cultivars and large scale introduction of infected planting materials, there is a probability of beak out of malformation in epidemic form. Internal quarantine and an integrated management strategy have been suggested to prevent the disease build up.

Chand, G. and D.K. Chakrabarti. 2004. Mango malformation: reproduction of malformed shoots, *Journal of Mycology and Plant Pathology*, **34**(2) : 294-296.

In nature logarithmic increase in malformed shoots of mango (*Mangifera indica* L) was observed during later part of July. Mild temperature (25-30^{o}C), high relative humidity (>85%) and number of rainy days during the period influenced the disease development positively. Various inoculation techniques were attempted to reproduce vegetative malformation in *M. indca* cv Amrapali by inoculating emerging buds in

July. Inoculum strip was used as infectious entity. The highest disease incidence was recorded when the buds were inflicted with needle injuries, sprayed with cycloheximide (2 ppm) and then inoculated. In these buds the fungal population and host metabolite, mangiferin (that induces vegetative growth and imparts protection against the *Fusarium*) were high and in a state of balance. In buds where hydrogen peroxide was used in place of cycloheximide, the fungal population was high but its mangiferin was transformed in to polymeric quinine which neither could stimulate vegetative growth nor arrest the fungal multiplication. In these buds, higher number of *F. moniliforme* var. *subglutinans* population was recorded but the buds were in quiescent state. Cycloheximide and hydrogen peroxide showed incompatibility in producing the disease symptoms. In the buds only injured before inoculation produced malformed shoots. In these buds both mangiferin content and the fungal population were high. In uninjured buds symptom production was less.

Pandey Mukesh, K. and D.K. Chakrabarti. 2004. Management of malformation of mango (*Mangifera indica*), *Journal of Mycology and Plant Pathology*, **34**(3) : 881-88.

Efficacy of carbendazim and mangiferin Cu $^{++}$ chelate and an antagonist *Aspergillus niger* van Tiegh isolated from dead malformed mango panicle, in reducing population of the pathogen, *Fusarium moniliforme* Sheld var. *subglutinans* Wollenewb and Reinking was studied *in vitro*. Mangiferin Cu^{++} chelate killed the conidia of *F. moniliforme* var. *subglutinans* and caused lysis of its mycelia. *A. niger* acted antagonistically through overgrowth and parasitization. Carbendazim arrested the growth of the germ tubes produced by conidia and reduced the infection rate. In field, application of *A. niger* brought down the inoculum level on dead panicles. Removal of malformed panicles successfully reduced the disease level over control in the next crop season. But in the second year due to an increased rate of disease progress in the pruned plants effects of sanitation was not sustained. Though the application of carbendazim reduced the rate of disease progress, its effect became pronounced only in the second year.

Chakrabarti, D.K., Mukesh Pandey, R. Kumar and Sunil Kumar. 2005. Endemicity in malformation disease of mango (*Mangifera indica*), *Indian Journal of Agricultural Sciences*, **75**(3) : 172-174.

Endemicity (State of balance in host-patho system) in malformation disease of mango (*Mangifera indica* L.) induced by *Fusarium moniliforme* Sheldon var. *subglutinans* Wollenewb & Reinking was investigated during 1999-2002. The disease incidence after continuous increase for 4-5 years (epidemic stage), a state of host-pathogen equilibrium was attained, i e endemic stage. In epidemic phase production of daughter infection per parent infection (iR) was slightly exceeding 1 but at endemic stage declined below 1. With increase in the disease percentage, the epidemic approached faster towards being leveled off (endemicity). In 'off year' production of smaller number of flowers limited the infection sites; thus the inoculum potential. In presence of more mother malformed panicles the infection plants attempted to produce profuse flowers to reduce the disease proportion. In alternate bearing cultivars of 'Dashehari'

and 'Langra' endemicity was attained earlier and asymptote L, at which the disease would level off, was higher indicating that fitness of these cultivars was less affected even in constant presence of the disease. At endemic phase the catastrophic initial epidemic of mango malformation was abated without intervention of fungicide.

Pandey, Mukesh,K., D.K. Chakrabarti and Sunil Kumar. 2005. Production and germination of conidia of *Fusarium moniliforme* var. *subglutinans* incitant of malformation in mango (*Mangifera indica*), *Journal of Mycology and Plant Pathology*, **35**(1) : 163-166.

The production and germination of conidia of *Fusarium moniliforme* Sheldon var. *subglutinans* Wollenwb and Reinking, the incitant of malformation disease of mango (*Mangifera indica* L.) were investigated both in growth chamber and natural condition in relation to environmental parameters. In nature, the peak period of conidia production was July-August. For conidia production, 35 ^{0}C ambient temperature and 90% relative humidity (RH) were highly favorable. A hyphae took about 24 h to start conidia production. But once production started, subsequent conidia from the same hyphal tip were produced at about 1 h interval. Conidia germinated within 5-6h. A temperature regime of 30 ^{0}C and high RH (100%) favoured germination. Conidia showed maximum viability in the early morning hours; later it was reduced.

Chakrabarti, D.K., Ramesh C. Shakywar, Gireesh Chand and Sunil Kumar. 2006. Control of malformation and growth promotion in mango by amino acid based metal chelates (Aminocel Gold), *Pestology*, **30**(2) : 35-39.

Effects of amino acid based metal chelates (Aminocel Gold) on flowering, vegetative growth, incidence of malformation disease and population of its pathogen, *Fusarium moniliforme* Sheld. var. *subglutinans* Wollenewb and Reinking in mango (*Mangifera indica* L) were tested in a large-scale field trial. The biochemical changes brought about by the test compound in the host and their effects on increased productivity and the diseased resistance were investigated. The amino acid metal chelates increased contents of chlorophyll, carbohydrates and indoleacetic acid, which subsequently increased flowering and vegetative growth. The test compound could not inhibit the mycelium growth of the pathogen *in vitro*. But in the treated host there were increase in phenol (mangiferin) content and activity of poly phenol oxidase with suppressed activity of catalase and a concomitant reduction in population of *F. moniliforme* var. *subglutinans*.

Chakrabarti, D.K. and Pinaki Chakraborty. 2006. Expert system for management of malformation disease of mango, *ICAR News*, **12**(1) : 18.

The Expert System for Management of Malformation of Disease of Mango (ESMMDM) helps to predict the disease incidence and suggests appropriate integrated management strategy. The expert system is based on the information generated from long term research on the etiology, epidemiology and management under both laboratory and field conditions.