EPIDEMIOLOGICAL PRINCIPLES OF CONTROL OF MANGO MALFORMATION - A REVIEW

D.K. Chakrabarti and R. Kumar
Department of Horticulture,
N. D. University of Agriculture and Technology, Faizabad-224 229, India

ABSTRACT

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* Sheldon, 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 increase of the disease could be minimised through sanitation (pruning of infected plant parts, killing of the propagules with fungicides, using cultivars with vertical resistance and growing plants in dry weather condition).

Malformation disease of mango (*Mangifera indica* L.) incited by a physiological race of the fungus *Fusarium moniliforme* Sheldon (basionym: *F. moniliforme* var. *subglutinans*) (Summanwar *et al.*, 1966; Kumar and Chakrabarti, 1992, 1992a, 1995; Ploetz and Prakash, 1997) is considered as a plant disease of international importance. Lack of information on epidemiology of the disease had been a limiting factor to confirm the etiology of the disease and also to find out rational management strategy (Kumar *et al.*, 1993). Hence, an intensive study on the disease dynamics in natural state was undertaken by the present authors. On the basis of the findings of these experiments, an epidemiological descriptor (Table 1), as suggested by Kranz (1998) has been proposed here. Such descriptors are known to assist the improvement of systems of control and systems of design.

To determine the proportion of disease at any time Van der Plank (1963) proposed the following equation: \( X = X_0 e^{rt} \), where \( X \) is proportion of disease, \( X_0 \) is initial inoculum and \( r \) and \( t \) are respectively average infection rate and time during which infection has occurred.

It appears from the descriptor that the propagules (conidia) are produced and available for dissemination for a short period. Hence, \( t \) does not seem to be a controlling factor of the epidemic. Besides the disease particularly in northern India has attained the endemic stage. And when the disease is endemic, time \( t \) becomes unimportant (Van der Plank, 1975). So far \( r \) is concerned, the pathogen for dissemination depends mainly on slow moving mites or propagation with infected scions; thus, not easily dispersable. Although rate of infection within the plant is fast but plant to plant spread is slow. Thus, \( r \) is also not an important factor in breaking the epidemic. Additively the latent period is long (Noriega-Cantu *et al.*, 1998). All these render the pathogen epidemiologically incompetent (Kranz, 1988). However, the tissues remain infectious for long and a small amount of initial inoculum can start the epidemic. Therefore, initial inoculum level, \( X_0 \), appears to be most crucial in causing malformation epidemic. Hence, increase of \( X \) presumably could be minimised by minimising primary inoculum level, \( X_0 \). And this could be achieved through sanitation (Van der Plank, 1963; Kranz, 1988; Chakrabarti, 1996). Destruction of initially infected tissues through pruning have been reported to reduce the disease incidence (Chakrabarti, 1996; Chakrabarti and Kumar, 1997a). The effect of pruning can be achieved by destroying the propagules through...
Table 1. Epidemiological descriptor for malformation diseases of mango.

| Life cycle | Polycyclic, pathogen is polyetlc in nature (Kumar and Chakrabarti, 1997) |
| Host range | Forma specialis (Kumar and Chakrabarti, 1992, 1992a, 1995) |

**Range of environmental conditions required:**

I) **Higher fungal population**
   a) February-March: range of temperature, relative humidity and mangiferin (anti-fusarial compound produced by the host plant in defence) content were 8 - 30°C, 77 - 85% and 4 - 6% respectively (Chakrabarti et al., 1997; Chakrabarti and Kumar, 1998).
   b) July-September: range of temperature, relative humidity and mangiferin content were 25 - 33°C, 84 - 87% and 4 - 6% respectively (Chakrabarti et al., 1997; Chakrabarti and Kumar, 1998a; Noriega-Cantu et al., 1998).

II) **Maximum disease incidence**
   July-November: range of temperature, relative humidity and mangiferin content were 15 - 33°C, 82 - 87% and 4 - 6% respectively (Chakrabarti and Kumar, 2000a; Noriega-Cantu et al., 1998).

III) **Latent period**
   Last week of November - first week of February: range of temperature, relative humidity and mangiferin content were 8 - 27°C, 82.3 - 84.8% and 3.98 - 5.75% respectively and long dew period (Kumar and Chakrabarti, 1997b).

IV) **Conidial production in vivo**
   July-September: on dead rotten malformed plant parts; temperature and relative humidity were 25.1-30.5°C and 84 - 87% respectively and mangiferin was totally absent (Chakrabarti et al., 1997; Chakrabarti and Kumar, 1998, 2000a; Noriega-Cantu et al., 1998).

Dispersal and spread:

I) **Means**
   Vector (mites), infected scions (Chakrabarti et al., 1997a; Chakrabarti and Kumar, 1999, 2000).

II) **Gradient of spread**
   Hyperbolic (Kumar and Chakrabarti, 1997).

III) **Distribution of loci**
   Nonrandom aggregation (Chakrabarti and Kumar, 1997).

IV) **Ease of spread**
   Plant to plant is slow, but within plant is very fast (Kumar and Chakrabarti, 1997).

Epidemic competence:

I) **Infection period**
   Late November-March and July-September (Chakrabarti and Ghosal, 1989; Chakrabarti and Kumar, 1998, 2000; Chakrabarti et al., 1997).

II) **Mean latency (p)**
   5-6 weeks (Chakrabarti and Ghosal, 1989; Kumar and Chakrabarti, 1997b).

III) **Mean infection rate (within the plant)**
   \( r_1: 0.5 - 0.8 \) per unit per year (Kumar and Chakrabarti, 1997).
   \( r: 153 - 323 \) per unit per year (Kumar and Chakrabarti, 1997).

IV) **Disease incidence before logarithmic phase starts**
   1.34 - 5.01% (Kumar and Chakrabarti, 1997).

V) **Disease incidence before reaching second peak** (in case of alternate bearing cultivar where the disease progress curve is bimodal) 0.72 - 1.84% (Kumar and Chakrabarti, 1997).

VI) **Mean maximum disease incidence**
   In alternate bearing cultivar: 72 - 73% (Kumar and Chakrabarti, 1997; Noriega-Cantu et al., 1998). In regular bearing cultivar: 40-48% (Kumar and Chakrabarti, 1997).

VII) **Longevity and infectiousness of inoculum**
   a) In malformed shoots and panicles the pathogen remains infectious condition round the year (Chakrabarti and Ghosal, 1989).
   b) The pathogen multiplies on the host surface and spreads mainly during July - October (Chakrabarti and Ghosal, 1989; Chakrabarti and Kumar, 1998, 2000a).

VIII) **Yield loss mean**
   Cumulative loss 1399 - 3579% per year (Kumar and Chakrabarti, 1997a).
IX) Efficiency of infection value
For cultivar Mallika: 0.951 (Chakrabarti and Kumar, 1997).
For cultivar Amrapali: 0.894 (Chakrabarti and Kumar, 1997).

X) Mean sporulation capacity
7.050 conidia/g. necrotic malformed panicle/day, when range of temperature and RH were 25.8 - 32.4°C and 85 - 92%, respectively (M.K. Pandey and D.K. Chakrabarti, unpublished).

Epidemic pattern:
I) Types of disease progress curves
In alternate bearing cultivars: bimodal (Kumar and Chakrabarti, 1997; Chakrabarti and Kumar, 2000),
In regular bearing cultivars: sigmoid (Kumar and Chakrabarti, 1997; Chakrabarti and Kumar, 2000).

II) Mean delay between crop and disease appearance
The disease appears from seedling (even 3-4 months old) stage (Nirvan, 1953).

III) Mean duration of the epidemic
Round the year, the disease incidence is low during summer months (Chakrabarti and Ghosal, 1989; Chakrabarti, 1996).

IV) Mean duration of progressive phase
Time required reaching the first peak of the bimodal disease progress curves is 3 - 4 years from the first appearance of the disease symptoms (Kumar and Chakrabarti, 1997).

V) AUDPC

Spraying of fungicides like captan (Summanwar, 1967), mangiferin copper chelates (Chakrabarti and Ghosal, 1982, 1989; Chakrabarti and Kumar, 1997a). But the sprayings were effective when sprayed before arrival of inoculum (Summanwar, 1967; Ibrahim et al., 1975; Chakrabarti, 1996; Chakrabarti and Kumar, 2000). Because if spraying starts before the inoculum first arrives the rate of the disease is curbed from the beginning (Van der Plank, 1963). In nature amount of X₀ is controlled by vertical resistance (VR) while r is determined by horizontal resistance (HR) of the host plants (Van der Plank, 1963). In case of VR, the pathogen fails to produce symptoms and to reproduce itself over the host. HR affects spore germination and penetration of the pathogen and prolongs latent period (Van der Plank, 1963). Since no species of mango is known to possess VR against the pathogen, the effects of VR can be achieved by sanitation. But some mango cultivars seem to possess HR. For example, in Banarasi Langra both r and AUDPC values are considerably smaller. The r value may also be reduced by prophylactic sprayings. Fungicides form a protective layer over the host surface and affect the infection process. The performance of prophylactic sprayings gets a synergistic boost if the treated plants possess HR. Drier weather also behaves like fungicides. During summer months the amount of inoculum is reduced to the minimum (Chakrabarti et al., 1997; Chakrabarti and Kumar, 1998), consequently shoots developed during this period are disease free (Varma et al., 1971). However, method of control becomes more effective if it reduces both X₀ and r. Recently an integrated disease management strategy (IDM) consisting of pruning and prophylactic spraying with copper fungicides successfully managed the disease on cv. Banarasi Langra (Chakrabarti and Kumar, 1997a).

REFERENCES


