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Powdery Mildew of Mango: A Review

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Abstract: Powdery mildew is a serious disease of mango in the world caused by a fungus *Oidium mangiferee.* Young tissues of all parts of the inflorescence, leaves and attacked by the fungus. Crop losses up to 100 percent have been reported in oasis of blossom infection when disease spreads in epidemic form. Conidia are dispersed by wind and germinate best at 20.25°C with moderate humidity. Pathogen survive from one seauch to the next as mycelia in dormant buds and as haustoria en old infected leaves. Telemorph of the fungus. have not been reported in Pakistan or from the other mango growing regions of the world. No completely resistant cultivar of mango has been reported any where but they vary in their susceptibility to powdery mildew. Fungicides applied at 30-40 Percent flowering stage followed by two applications at 2-3 week intervals depending upon environmental conditions can effectively control the malady.

Key words: *Mangifara indica*, powdery mildew, review, mango

Introduction

Powdery mildew is an important disease in most of the mango *(Mangifera indica* L.) growing areas of the world (Palti *et al*., 1974). A fungus *Oidium mangiferae* Berthet is the causal organism, who's telemorph is still unknown (Johnson, 1994; Akhtar *et al*., 1999). This malady can cause upto 80 percent losses which remained underestimated when out breaks occurs during early flowering (Johnson, 1994). The fungus attacks the young tissues of all parts of the infloresences, Leaves and fruits (Goner, 1988; Johnson, 1994; Akhtar *et al*., 1999).

Powdery mildew of mango was present in Indo-Pak-Sub-Continent before 1874 (Johnson, 1994). This disease emerged as a limiting factor in Pakistan for the last few years, since then higher input costs have increased the economic importance of the disease. The basic concept of this review paper is to place the current situation in perspective and to draw the attention towards deserving areas.

Symptoms: The disease mostly appears in the month of February and March. It attacks flower scales, buds of tender flower heads, stalk of young leaves and fruits in the early stage (Singh, 1960). Fungus is a ectoparasite as it remained restricted to the epidermal layers of infected parts of mango plant. In the early stages, after infection small isolated patches of powdery white mycelium develops on the invaded tissues, which may coalesce in the later stages to cover large areas (Palti *et al*., 1974; Burchill, 1978). A velvety powdery deposit on a dark to smoky grey background is the characteristic symptoms (Kotze, 1985), fungal growth stops when infected tissues become necrotic (Palti *et al*., 1974).

The disease usually appears as small scattered water soaked leasions on the under surface of the young leaves, later, directly under watery spots white powdery growth of *Oidium mangiferae* appears to cause necrosis in advance stages. These irregular necrotic lesions may enlarge and coalesce to form large dead areas on the leaf, frequently resulting curling and distortion (Burchill, 1978; Anonymous, 1995).

Severe blossom infection can cause complete loss of fruit. Flower fails to open and then drop from the inflorescence (Ruehle and Ledin, 1956; Anonymous, 1996). Most inflorescence produces disease from tip to downwards and manifests itself by the appearance of wefts of white mycelium on the affected parts. More than 90 percent of the inflorescence are invaded after the opening of the flowers but in few cases young inflorescence with unopened flowers were also attacked (Anonymous, 1995). Sepals are particularly susceptible, while petals are much resistant. In severe cases, the inflorescence becomes completely covered by the wefts of mildew and eventually blacken (Burchill, 1978).

Powdery mildew can also effect fruits. Mycelium of the fungus entirely covers the newly borne fruits, with the expansion of the fruit epidermal tissues, infected areas becomes cracked and corky. This situation cause premature fruit fall (Palti *et al*., 1974; Burchill, 1978; Anonymous, 1995). On mature fruits white powdery growth appears which withers away to produce superficial irregular purplish brown blotches or corky surface (Gupta, 1989; Joubert *et al*., 1993).

Causal agent: Wagle (1928) declared that *Erysiphe cichoracearum* D.0 is the cause of powdery mildew of mango. Uppal (1937) studied the histological features of the fungus. He observed that the fungus produced globular haustoria, a characteristic of *E. polygoni* and suggested that *E. polygoni* is the cause of disease. Uppal *et al*. (1941) continued their investigations and reported that the fungus produced saccate or lobate haustoria which is different from the characteristic of *E. cichoracearum.* Berthet (1941) resolve this controversy, as he suggested that no description of the telemorph (perfect stage) of fungus has been reported, hence the name of conidial stage *Oidium mangiferae* should be preferred. No telemorph or sexual stage of the fungus is known or observed. However, the mode of conidial germination and production of globular haustoria suggests that the *E. polygoni* group is responsible for the disease development and mango is the only known host of *O. mangiferae* (Palti *et al*., 1974). Boesewinkel (1980) stated that the *Oidium* sp. attacking mango in New Zealand is identical to the fungus causing powdery mildew of Oak (*Microsphaera alphitoides* Griffon and Mauble). The same fungus was reported in South Africa causing European Oak mildew and also from various other hosts (Joubert *et al*., 1993). Goner (1988) stated that in South Africa *M. alphitoides* has been over taken by another powdery mildew identical to the North American Oak mildew caused by *M. extensa* Cook and Peek. No longer evident of *M. alphitoides* were reported from South Africa. Joubert *et al*. (1993) reported that conidiophores produced by the fungus on mango have two cells, while on Oak it produces 3-5 cells. So for the time being it is decided to suggest the anamorph name, *O. mangiferae* for the fungus causing powdery mildew disease of mango in South Africa.

The distinguishing morphological characters of *O. mangiferae* are 40-80 μm long hyphal cells and conidiogenous cells of moderate length (27, 4 - 40, 0 *pm).* Conidia are barrel shaped and produced singly (Uppal *et al*. 1941; Gorter, 1984, 1988).

Johnson (1994) described that no telemorph of the pathogen is

known. Conidial and haustorial traits suggests that *O. mangiferae* Berthet belongs to *E. polygoni* group. Conidia (33-43 × 18-23 µm) are septets, hyaline and elliptical to barrel shaped. Fibrosin bodies are absent and conidia are usually produced singly. Germ tubes are of variable length (depending upon humidity) and terminate in hook like appresoria. Globular haustoria can be seen in epidermal cells. Conidiophores are of the pseudoidium type and have 2-4 septa and a straight basal cell. The pathogen colonized Oak leaves in the laboratory. Histological studies performed in Pakistan shows that *O. mangiferae* (anamorph) has single, unicellular and hyaline conidium (17-28 × 40-49 m), while conidiophore are simple, short erect and haline (60-165 m) with superficial septate and haline (6 - 8 m) mycelium (Anonymous, 1996; Akhtar *et al*., 1999).

Distribution: Powdery mildew was present in Indo-Pak Subcontinent before 1874 (Johnson, 1994), while Kulkarni (1924) reported this disease in India for the first time, whereas McRae (1924) also reported it simultaneously from Hyderabad (India). Later on, Galloway (1935) and Uppal (1937) also reported this malady in India. Singh and Garg (1949) for the first time recorded the outbreak of disease in Luckhnow. Similar reports on the occurrence of this malady was received from Ceylon (Srilanka) by Haigh (1931) and Beaus (1946); from Jamica, West Indies, by Anonymous (1932); from Israel by Reichert and Palti (1951); from South Africa by Dyer (1947); from Southern Rhodesia by Hopkins (1941); in California (USA) by Fields (1945) and Mata Quesada (1950); from Latin America; by Gillman (1952); in Nayasaland (Africa), Rodriguez Landaeta and Figueroa (1963) from Venezuela and Boesewinkel (1980) from NewZealand.

Mode of damage: McRae (1924) reported that the *Oidium mangiferae* is responsible for the dropping off young mango fruits. Fungus attack flowers before fertilization which remained unfertilized and drop off the plant. Fruits may carry infection in their early stage of development and drop off prematurely to produce huge yield losses (Wagle, 1928). Pathogen was observed serious on the leaves of grown up trees, especially between 2000-4000 ft above sea level and was found all the year round, particularly during spring which cause premature falling of leaves and fruits (Bose, 1953).

Conidia of fungus are disseminated by wind, these wind borne conidia cause infection after 5-7 hours of germination and in about 2 days, produce mycelium to complete life cycle on vegetative shoot in 9 days (Wagle, 1928). Palti *et al*. (1974) observed that wind borne conidia reaching on new growth flushes and young flowers, germinate to produce germ tubes which gives rise to appreassoria on the host surface. Appressoria penetrates into the cuticle and cell wall of the host with the help of penetration pegs which give rise to the tube like haustoria then swell inside the epidermal cells to form globular structures. Fugue produced conidia with in five days of infection (Cook, 1975). Under unfavourable conditions or when susceptible tissue is not available the fungus will presumably survives as mycelium on older leaves (infected) and malformed panicles (Palti *et al*., 1974; Anonymous, 1996). Powdery mildew cause sever losses during flowering and when growth of flushes occurs during dry cool conditions. Disease outbreaks may be started either from inoculum harbored on the tree or by air borne conidia from other infection sites. Air borne conidia subsequently cause secondary spread with in the trees (Johnson, 1994).

Epidemiology: Cloudy weather with heavy morning mist favoured the disease and was particularly destructive in the coastal areas of Bombay (India) during cold and wet season (Kulkarni, 1924), while Wager (1937) stated that hot dry spring with heavy dews at night are conducive for severe out-break in South Africa. Conidia germinates best at 20-25°C under moderately high relative humidity (Uppal *et al*., 1941). Bose (1953) added that rains during flowering encourages disease.

Palti *et al*. (1974) performed *in vitro* studies on the temperature and humidity requirement of *O. mangiferae* using glass slides. They observed that 70 percent germination was observed at low humidities (20% RH) while it decreased to 33 percent RH. So they concluded that low and intermediate humidities (20-65 % RH) are excellent for maximum germination of spores rather than high humidities (81-100% RH). They also observed that 22°C is optimum while 9°C and 30-32°C are respectively minimum and maximum temperature ranges for the spore germination. Free water generally causes damage to the superficial mycelium of fungus and the spore numbers remain low for several days after germination. Rain is not always harmful as showers can stimulate disease by raising the atmospheric humidity (Butt, 1978). Kotze (1985) reported that disease do not preceded by rain or dews. Same results were reported by Joubert *et al*. (1993). Epidemics were frequent at 20-25°C with intermittent rains during flowering (Palti *et al*., 1974; Cook, 1975). Johnson (1994) described that conidia of the fungus can germinate in the absence of water. He added that germination takes place with in 5-7 hr at 23°C with 20 percent relative humidity. Optimal disease development was observed in the diurnal range of 10-31°C at 60-90 percent RH. Conidia of *O. mangiferae* exhibit a diurnal pattern of dispersal and they mainly liberates from 12.00 to 16.00 hours. Rains reduced the dispersal of conidia. Fungus took 3-4 days to reach pre-rain levels when dry weather followed rainy period (Gupta, 1988). Conidia attached to the conidiophores on mildewed leaves and infloriscences can retain their viability for up to 40 days in comparison with detached conidia which lasted 20-35 days on glass slides and host leaf surfaces. At room temperature (22-30°C) fungus can survive for 15 days instead of 21 days on host surface and 7 days instead of 10 days on glass slide (Gupta, 1989). Schoman and Manicom (1995) monitored the epidemiology of powdery mildew on the blossoms. Conidia of fugus were trapped during 1989-91. Hourly aerial conidial concentrations were correlated positively with hourly RH, vapor pressure deficit and leaf wetness. Number of trapped airborne conidia were characterized by a distinct diurnal periodicity. Greatest number were trapped between 11.00 and 16.00 hours. They further reported that susceptibility of inflorescence begins when the main axes changed their colour and ended at fruit set. Akhtar *et al*. (1999) reported that wind-born conidia are released from the tissues harboring the dormant fungal hyphae under favourable weather conditions, to produce disease. There was positive correlation between rising temperature, lowering RH and no. of spores in the air after a low temperature, high RH and cloudy spell of weather. Maximum no. of conidia were released around the temperature 25°C and 40-60 percent RH. It tooks 5-8 days for the emergence of disease symptoms after the detection of first air-borne conidia. Susceptibility of inflorescence varied with its developmental stages.

Losses: The flowers remain unfertilized due to powdery mildew and diseased young fruits of mango drop off the plant which lead to serious yield losses (Wagle, 1928). In the state of Bombay up to 20 percent losses have been reported in certain years (Anonymous, 1930). Hopkins (1941) reported substantial losses of mango fruit from Southern Rhodesia.

The damage caused by powdery mildew to the inflorescence of mango is often underestimated as disease outbreaks occur early during the flowering stage (Joubert *et al*., 1993). Crop losses of 80-90 percent have been reported in South Africa

(Brodrick, 1971; Kotze, 1985). While losses of up to 20 percent have been recorded in Florida during some seasons by Cook (1975).

Ruehle and Ledin (1956) reported that losses caused by the disease are mainly due to blossom infections, although young tissues are also susceptible. Severe infection effect entire panicle to exhibit complete failure of mango fruit formation. Tahir (1984) reported more than 86 percent fruit losses in infected mango fields while 37 percent in protected fields. Blossom infection cause poor fruit setting and if infection is severe disease cause huge losses usually very high upto 100 percent (Lonsdale and Kotze, 1993a,b).

Host resistance: Resistance to powdery mildew has been investigated by researchers in several countries. No resistance has been found through out the Punjab province but the cultivars were found varying in susceptibility to powdery mildew, depending on climatological factors (Anonymous, 1995; Akhtar *et al.*, 1999).

Chemical control: Powdery mildew of mango is a cureable disease. It could be controlled effectively with the proper use of effective fungicides for good 'twit production. In Pakistan the current recommendation for the first fungicide application are before or at 20-30 percent flowering. In preliminary studies Akhtar *et al.* (1999) efficiently control the disease with spray of proper fungicides at 30-40 percent blooming or when first conidia trapped followed by two further sprays with 15 days interval of the first spray depending on the weather and disease conditions. They added that disease could be reduced by the use of proper (Curative) fungicides when it occurs in high infestation form, but is too late and cause more than 50 percent reduction in yield. Chemical control is not always effective because the fungus can develop resistance against certain chemicals. So, alternative sprays are necessary for the efficient control of disease. Number of fungicides are present in the market against the disease, Some most effective are listed in Table 1.

Table 1: Important fungicides used for the control of Powdery mildew of mango in Pakistan

Name of Fungicides	Dose/100 lit. of H_2O
Topsin-M	100-150 gm
Thiovit	250 gm
Topes	50 ml
Afugan	50-100 cc
Derosal	50-70 ml
Daconil	250 ml
Rubigan	50 ml
Calixin	50 ml
Bayleton	50 ml
Bordeaux mixture	4:4:50

Powdery mildew is a serious disease of mangoes in Pakistan and most of the other mango growing regions of the world with significant economical losses. Very little is known about the pathogen e.g. texonomy, alternate hosts which could have an Influence on its epidemiology. So cross inoculation studies are therefore required. The epidemiology of disease also needs extensive studies to determine the climatic requirements for disease development. These studies will help to decide the best time to begin sprays.

Commercial varieties of mango are generally susceptible to the disease. Genetic resistance does exist in the land races and undescribed germplasm of mango which could be used in selection and breeding programs for developing resistant varieties.

A wide range of fungicide have been registered in Pakistan for the control of this malady but serious losses still occur. These fungicides are not applied properly which ultimately increase economic losses. So extensive Extension studies are required to educate peoples for the proper use of fungicides (i.e. use of proper fungicides at proper time with proper equipment) to obtain maximum disease control.

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