Advances in Horticulture Vol. 3 – Fruit Crops : Part 3 (1993). Eds: *K.L. Chadha and O.P. Pareek*

Fungal Diseases of Subtropical Fruits

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1. INTRODUCTION

The subtropical fruits grow in wide range of agroclimatic conditions and are associated with the diversity of disease problems. A number of diseases, such as anthracnose, mildew, wilt, rust, die-back, canker, spots, blight, sooty mould and damping off are prevalent in one form or another throughout the country and attack almost every plant part, viz., root, stem, branch, twig, leaf, tendril, petiole, flower and fruits at different growth and developmental stages. Some of these diseases have become a limiting factor in the successful cultivation in some regions. Even minor disease like leaf spots can cause great losses. With the adoption of the modern methods of intensive management practices, a number of diseases have assumed greater severity during recent years and require proper identification and control to avoid serious losses. work done on the major fungal diseases on several subtropical fruits is reviewed.

2. FUNGAL DISEASES

2.1 Aonla

2.1.1 *Rust*

Rust is an important problem especially in Rajasthan (Tyagi, 1967). It has also been observed at Rehmankhera (Lucknow) and Rajgarh (Pratapgarh) (Anon., 1988a). Leaf rust is supposed to be caused by *Ravenelia embilicae* Syd.

On fruits, initially the pustules are black which later develop into ring. The pustules join together and cover a big area of the fruit (Fig. 1). The black spores get exposed by rupturing a papery covering. The fruit gives a dirty appearance. On leaves, pinkish brown pustules develop which may appear singly or in group. Tyagi (1967) stated that infection on fruit does not go on leaves and *vice versa*.

Three sprays of 0.5 per cent wettable sulphur (Elosal) at an interval of one month from July was found to be an effective control measure (Tyagi, 1967).

2.1.2 *Anthracnose*

Mishra and Shivpuri (1983) reported anthracnose on leaves and fruits during August-September at Udaipur.

The disease is caused by *Colletotrichum* state of *Glomerella cingulata* (Stonem) Spauld. and Schrenk. Initial symptom of the disease is in the form of minute, circular, brown to grey spots with yellowish margin on leaflets. The central area of spots remain greyish with dot-like fruiting bodies. On fruits, the depressed lesions, which later turn dark in the centre forming acervuli are often arranged in rings (Fig. 2). The lesion may vary in size and shape with spore masses appearing on fruiting bodies at high humidity. Consequently, the fruits become shrivelled and rot (Misra and Shivpuri, 1983).

Fig. 1 : Rust pustule on aonla fruit.

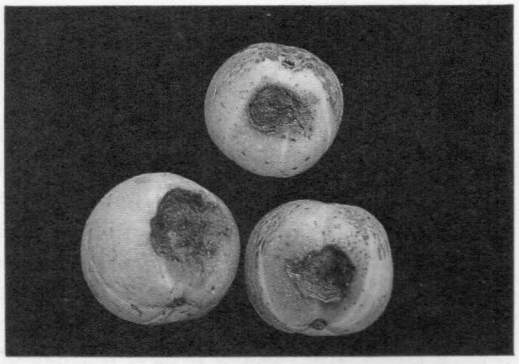

Fig. 2 : Anthracnose on aonla fruits.

2.1.3 *Fruit Rot*

Pandey *et al.* (1980) reported a fruit rot during December-January. The rot caused by *Phoma putaminum* Speg. starts as a small pinkish brown necrotic spot which extends towards both the ends of the fruit forming a eye-shaped appearance. In severe cases, lesions coalesce forming a bigger pustule. The mature lesions are dark brown and severely infected fruits show wrinkling. The underlying tissues in the rotten fruits become soft (Pandey *et al.*, 1980).

Jamaluddin *et al.* (1975) reported dry fruit rot of *aonla* caused by *Phoma emblicae.*

2.1.4 *Soft Rot*

During December to February, a severe fruit rot was observed in orchards and markets of Allahabad and Fatehpur (Lal *et al.*, 1982). This was caused by *Phomopsis phyllanthi* Punith.

Smoke brown to black, rounded lesions develop within 2-3 days of inoculation. The infected part later shows olive brown discolouration with water-soaked area and cover the whole fruit within 8 days. The shape of the fruit also gets deformed.

The disease is prevalent more during December to February. Although the fungus causes infection in young and mature fruits but, on inoculation, mature fruits are found to be more susceptible. Injury is essential for the development of the disease (Lal *et al.*, 1982).

The disease could also be inoculated on *Pyrus malus, Psidium guajava, Citrus sinensis, Musa paradisiaca, Citrus medica, Luffa cylindrica, Abelmoschus esculentus* and *Citrullus vulgaris.*

Difolatan (1500 ppm), Dithane M-45 and Bavistin (100 ppm) just after the harvest were found effective to control the disease (Lal *et al.*, 1982).

2.1.5 *Nigrospora Fruit Rot*

Fruit rot caused by *Nigrospora sphaerica* (Sacc.) Mason. was recorded during December at Kanpur. Loss due to this rot was estimated at 5-10 per cent annually (Kamthan *et al.*, 1981).

Rot initiation was from a small dot of 2 mm diameter extending to a black ring spot up to 7 mm. Several rings adjacent to each other coalesce resulting in complete rotting of the infected fruits (Kamthan *et al.*, 1981).

2.1.6 *Cladosporium Dry Fruit Rot*

Dry rot was recorded by Jamaluddin (1978) in Allahabad with an estimated loss of 2-5 per cent.

2.1.6.1 *Cladosporium tenuissimum* Cooke : Infection was noticed from November to February. The disease started as colourless area, slightly soft and subsequently progressed in a circular manner. Light brown mycelial growth of the fungus was also evident on infected areas. The diameter of the lesions increased from 1 to 1.5 cm in 7 days. Injury was necessary for manifesting the infection (Jamaluddin, 1978).

2.1.6.2 *C. cladosporioides* (Fries) de Vries : The rot shows dark brown necrotic lesions. Slight growth of the organism appears in the necrotic cavity. The severity of infection was recorded only in mature and ripe fruits. Some freshly harvested fruits also showed infection. One week old lesion measured 0.7 to 1.2 cm in diameter (Jamaluddin, 1978).

Tandon and Verma (1964) reported *C. herbarum* causing small spots of ochre-red colour on fruits.

2.1.7 *Pestalotia Fruit Rot*

A fruit rot was observed in the local market of Allahabad during November. It is caused by *Pestalotia cruenta* Syd.

The spots on the fruits are mostly irregular and brown. The disease usually starts as a brownish discolouration on the fruit surface which grows slowly. Later, the spots become mummy-brown and the skin around them develops light brown colouration. At a relatively later stage, the infected region becomes covered with white fluffy aerial growth of the fungus. The internal part of the diseased fruit shows a dry dark brown area (Tandon and Srivastava, 1964).

2.1.8 *Alternaria Fruit Rot*

During January and February, association of *Alternaria alternata* with dropped fruits was observed in Allahabad (Pandey *et al.,* 1984).

The rot started as a small, brownish, spherical necrotic spot which increased in circular fashion. In advanced stages, the spots became dark brown to black and neighbouring spots coalesced. The centre portion of the infected tissues became soft and pulpy.

2.2 ***Ber***

2.2.1 *Powdery Mildew*

It was reported from Allahabad (Mitter and Tandon, 1930), Kanpur (Mehta, 1950) and Bombay (Uppal *et al.,* 1935; Patel *et al.,* 1949). The disease is observed both on cultivated as well as wild *ber.*

2.2.1.1 *Symptomatology* : The disease is noticed generally at the end of October and prevails from November to April. Flowers and fruits are affected by the disease. Symptom starts in the form of white powdery spots on the surface of the fruits and later covers the whole fruit. The spots change into light brown to dark brown discolouration. Later, the infected area becomes slightly raised having cankers and the fruits crack. Finally, shedding of young fruits occurs.

2.2.1.2 *Causal organism* : It is incited by *Oidium erysiphoides* f. sp. *zizyphi* Yen and Wang. The fungal mycelium becomes external on the host, white conidiophores are upright, single, measuring 75.8 - 139.4 × 12.6 µm. Conidia are cylindrical, celled, hyaline, catenulate measuring 25.2 - 37.8 × 16.8 - 21.0 µm.

Kumar *et al.* (1978) reported *Oidiopsis* sp. on leaves, stem and fruits in Rajasthan. Gupta (1984) reported the germination of conidia of *O. erysiphoides* f. sp. *ziziphi* best on the host leaves. He further reported that on upper surface of leaves, germination and germ tube length were better than on lower surface or on agar and leaf extract.

It survives in budwood of the host plants or might have some alternate host to survive during the absence of flowers and fruits which may serve as the primary source of infection. The secondary spread of the disease takes place by the air-borne spores.

2.2.1.3 *Sources of resistance* : Cultivars Safeda Rohtak, Sua, Noki, Chonchal, Sanaur 5, Katha Phal, Sanaur 1, Illachi-Jhajjar, Kakrola Gola, Kala Gora, Pathani and Mirchia were found resistant (Gupta *et al.*, 1978; Kapur *et al.*, 1975; Jeyarajan and Cheema, 1972).

2.2.1.4 *Control* : Kapur *et al.* (1975) found that Benlate gave 92 per cent control followed by Afugan. Gupta *et al.* (1977, 1978) advocated Karathane WP (0.2%) or Karathane EC (0.1%) or Sulfex (0.2%) for control. The spraying should be done during first and 3rd weeks of November or when the fruits attain peanut size. This spray is important for effective control (Gupta *et al.*, 1978). Singh and Sandhu (1985) reported best control by two sprays of Bavistin (0.2%) at 25 days interval, starting from the time when fruits were of pea size followed by Karathane (Dinocap 0.1%) at 10-15 days, if needed. To ensure complete coverage, Triton or Teepol or Sandovit may be added to the fungicide solution.

2.2.2 *Alternaria Leaf Spot*

2.2.2.1 *Symptomatology* : The disease is characterised by the formation of small, irregular brown spots on the upper surface of the leaves. On the lower surface, dark brown to black spots are formed. The spots coalesce to form big patches. The leaves later drop (Gupta and Madaan, 1977).

2.2.2.2 *Causal organism* : Several species of *Alternaria* were reported from different parts of the country, such as *Alternaria* state of *Pleospora passeriniana* Berk. from Jodhpur (Panwar and Vyas, 1971), *Alternaria chartarum* Preuss. on *Z. jujuba* from Maharashtra (Rao, 1971), *Alternaria* sp. from Punjab (Jeyrajan and Cheema, 1972) and *Alternaria* state of *infectoria* Fuckel (Gupta and Madaan, 1977).

2.2.2.3 *Epidemiology* : The disease development is favoured at 20 to 30°C with an optimum at 25°C. Plant debris serve as potential source of primary infection. High humidity and frequent rainfall seem to be more important than temperature for the disease development. Frequent rainfall possibly helps in dissemination of spores which cause secondary infection (Madaan and Chand, 1985).

2.2.2.4 *Sources of resistance* : Jeyarajan and Cheema (1972) screened 35 cultivars of *Z. mauritiana* Lam. of which 25 were reported moderately susceptible. Gupta and Madaan (1980) found Bahadurgarhia, Govindagarh Special, Gola Gurgaon, Popular Gola, Seo Bahadurgarhia, ZG3, Safeda Rohtak, Jhajjar Special and Mirchia resistant to *Alternaria* leaf spot.

2.2.2.5 *Control* : The disease can be controlled effectively by spraying 0.2 per cent Difolatan or Dithane Z-78 (Gupta and Madaan, 1978).

2.2.3 *Black Leaf Spot*

Black leaf spot is also called *Isariopsis* mouldy leaf spot. It was reported to be caused by *Isariopsis indica* var. *ziziphi* in Haryana (Gupta and Madaan, 1977). The disease starts during October-November.

2.2.3.1 *Symptomatology :* Sooty tuft-like circular to irregular black spots develop on leaf surface. When infection advances, it covers a large area on the lower surface of the leaves and upper surface shows brownish discolouration.

2.3.3.2 *Epidemiology :* The fungus survives in plant debris and soil which serve as primary source of infection. The secondary infection occurs through spores present in air.

2.2.3.3 *Sources of resistance :* Cultivars ZG3, Safeda Rohtak, Mudia-Murhera, Sua, Sanaur 1, Pathani, Jhajjar Selection, Seo Bahadurgarhia and Jhajjar Special were found resistant to black leaf spot (Gupta *et al.,* 1980).

2.2.3.4 *Control :* The disease can be controlled by spraying 0.2 per cent Bavistin or Difolatan (Gupta and Madaan, 1985). A mycoparasite, *Hansfordia pulvinata* (Berk et Curt) Hughes, was found growing on the diseased spots which can keep the disease under control (Gupta and Madaan, 1979a).

2.2.4 *Cercospora Leaf Spot*

Two species of *Cercospora (C. zizyphi* Petch. and *C. jujubae* Chowdhuri) were reported to cause leaf spot. *C. jujubae* was reported from Punjab (Chona *et al.,* 1959), Bombay, Delhi (Vasudeva, 1960) and Jabalpur (Agarwal and Sahni, 1964). *C. zizyphi* was reported as the cause of the disease from Bangalore (Govindu and Thirumalachar, 1964), Hyderabad (Rao, 1962), Bihar (Yadav, 1963) and Haryana (Gupta and Madaan, 1975).

Circular to oval spots appear measuring up to 4 mm in diameter, epiphyllous, yellow at first and turn to brown surrounded by dark brown margin.

The cultivars Safeda Rohtak, ZG 3, Kakrola Gola, Bahadurgarhia, Reshmi, Jhajjar Selection, Popular Gola and Seo Bahadurgarhia were found resistant (Gupta and Madaan, 1985). Spray with Dithane M-45 (0.2%) gave satisfactory control.

2.2.5 *Cladosporium Leaf Spot*

The disease is caused by *Cladosporium zizyphi* Karst. and Roum. as reported by Uppal *et al.* (1935) from Bombay, Prasad and Verma (1970) from Muzaffarpur (Bihar), and Saini and Suppal (1981) from Patiala (Punjab). Gupta and Madaan (1975) reported *C. herbarum* as the causal organism of the leaf spot from Hisar (Haryana). The disease appears on lower surface of leaves as small, light brown to brown irregular spots.

The cultivars Banarasi, ZG 3, Villaity, Govindgarh Selection 3, Jhajjar Selection and Jogia were resistant (Gupta and Madaan, 1985). Spraying of Blitox-50 or Fytolan (0.2%) twice at two weeks interval at the appearance of symptoms is recommended.

2.2.6 *Rust*

The disease was first reported by Sydow and Sydow (1907) from Pusa (Bihar), Nagpur (Maharashtra) and Birbhum (WB). Yadav (1963) reported the disease from Patna (Bihar) and Gupta *et al.* (1984) from Hisar.

On the lower surface of the leaves, small irregular, reddish brown uredopustules appear which later advance to cover the whole area of the leaves. The infected leaves start drying and ultimately defoliation takes place. The disease is observed during January-February on both wild and cultivated varieties.

The cultivars Banarasi, Seo, Katha Gurgaon, Laddu, Dandan, Sanaur 1, Gola Gurgaon 2, Safeda Selection, Sanaur 3, Kishmish, Narma and Safeda Rohtak were resistant (Gupta and Madaan, 1985). Spray of Dithane M-45 (0.2%) or Dithane Z-78 (0.2%) or Sulfex (0.2%) or Vigil (0.1%) are recommended.

2.2.7 *Minor Diseases*

Disease	Pathogen/ host	Symptoms	Distribution	Authority
Septoria leaf spot	*Septoria capensis* Wint.	Dark brown spots measuring 2-8 mm in diameter, smaller spots coalesce to form bigger spots	Hisar	Madaan and Gupta (1976)
Pestalotia leaf spot	*Pestalotia subinae Fantrey*	Irregular dark brown measuring 1.5-4 mm in diameter. Black sub-epidermal, erumpent acervuli appear on the upper surface of the leaves	Hisar	Madaan and Gupta (1976)
Tandonella leaf spot	*Tandonella zizyphi* on *Z. jujuba* and *Z. mauritiana*		Bihar Punjab	Prasad and Verma (1970) Verma and Cheema(1983)
Sirosporium leaf spot	*Sirosporium carissae* on *Z. jujuba*		Rohilkhand	Pandey *et al.* (1986)

(Contd.)

(Contd.)

Disease	Pathogen/ host	Symptoms	Distribution	Authority
Botryodiplodia leaf spot	*Botryodiplodia theobromae* on *Z. jujuba*		Gwalior	Jain *et al.* (1983)
Wilt	*Fusarium equiseti* (Corda) Sacc. on *Z. mauritiana*	Wilting of the plants	Jodhpur	Lodha (1983)
	F. oxysporum Sachlacht		Hisar	Gupta and Madaan (1985)
	F. equiseti on *Z. mauritiana*		Jodhpur	Gupta (1984)

2.2.8 *Foliage Pathogens*

The following foliage pathogens have also been observed:

Pathogen	Host	Distribution	Authority
Elsinoe zizyphi	*Z. rotundifolia*	Maharashtra	Narashimhan *et al.* (1969)
Fusarium decemcellulare	on living galls of *Z. maurithiana*	Varanasi (UP)	Singh and Singh (1979)
Hypozylon hypomiltum	*Z. jujuba*	Pusa (Bihar)	Sydow *et al.* (1911)
Mitteriella zizyphina	*Z. jujuba* and *Z rotundifolia*	UP	Tandon (1935)
	Z. jujuba	J & K	Pandotra and Ganguli (1964)
	Z. nummularia	Rajasthan	Prasad *et al.* (1962)
Myriangium zizyphi	*Z. rotundifolia*	Maharashtra	Tendulkar (1970)
Othia zizyphus-jujuba	*Z. jujuba*	Aurangabad (Maharashtra)	Tilak (1965)

(Contd.)

(Contd.)

Pathogen	Host	Distribution	Authority
Poria sp.	Z. jujuba	Allahabad (UP)	Mitter and Tandon (1937)
Sclerotium rolfsii	Z. jujuba	Jabalpur (MP)	Mishra and Khare (1970)

2.3 Fig

2.3.1 *Rust*

The rust is cuased by *Cerotelium fici* (Cast.) Arth. Small round, brown black eruptive lesions develop on leaves. Pustules develop generally on lower surface. Later, it causes defoliation. Teliosori are less powdery in appearance than uredosori (Thirmulachar *et al.*, 1950). The disease appears on all species of *Ficus* and can be controlled by dusting sulphur or spraying Zineb (0.15 to 0.2 per cent). Padule *et al.* (1988) found all the 5 cultivars susceptible to rust under conditions of natural infection in Pune.

2.3.2 *Leaf Spot*

Mehta and Bose (1947) reported leaf spot of fig caused by *Cylindrocladium scoparium* Morg. from UP. In the beginning, minute brown spots appear which later enlarge into uniform, zonate, prominent reddish brown lesions with dark brown margins. These lesions afterwards coalesce and form irregular patches. The centre of the leaf becomes papery and drops off. Affected leaves shed earlier.

2.3.3 *Anthracnose*

The disease is caused by *Sphaceloma ficicaricae* Wani and Thirum and is common in fig growing areas. It can be controlled by spraying 40 ppm Aureofungin in soap solution + 20 ppm $CuSO_4$ (Wani and Thirmulachar, 1973).

2.3.4 *Ascochyta Leaf Spot*

Singh *et al.* (1984) recorded leaf spot of fig caused by *Ascochyta caricae* at Chaubattia (Ranikhet), Almora, UP in 1979 (HETC, 1902).

Symptoms appear on the upper surface of leaves after rains. Spots are irregular, grey measuring 0.5-2.0 cm in diameter. Pycnidia dark black, globose, septate, ostiolate, immersed in the host tissue, 75-210 µm in diameter. Conidiophores indistinct, conidia hyaline, ovoid to oblong, 2-celled, biguttulate, double walled, measure 3.5–10 x 2-6 µm in size (Singh *et al.*, 1984). Singh and Singh (1986) found that the disease can be controlled by Benlate (Benomyl) and Bavistin (carbendazim).

2.3.5 *Fruit Rot*

During February-March 1980, fruit rot caused by *Choanephora cucurbitarum* (Berk. & Rav.) Thaxt. was observed in Chandra Shekher Azad Park, Allahabad (Bhargava *et al.*, 1982). The disease was observed on green fruits. Initially, a light brown lesion develops on the surface of the fruit which gradually increases in size and the colour of the lesion changes to dark brown-black. As the infection progresses, the fruit surface shrinks and dries. Finally, the fruit gets detached from the twig and falls off. When the fruits are cut open, they are found to be infected from inside.

2.4 Grape

2.4.1 *Downy Mildew*

Downy mildew of grape is serious throughout south India, especially in Andhra Pradesh and Karnataka, and is a chief limiting factor in grape production (Sohi, 1983).

2.4.1.1 *Symptomatology :* The effects of downy mildew on grape vines are well documented (Srinivasan and Jeyarajan, 1976, 1977a, b). First symptoms of disease are yellow, translucent spots (oil spots) on the upper surface of leaves. On the underside of affected leaves, patches of white downy mildew appear soon afterwards. Severely affected vines are readily defoliated, preventing the ripening of fruit and maturation of canes and exposing fruit to damage by sun burn. Substantial crop loss also occurs when flower or young berries are killed (Srinivasan and Jeyarajan, 1983). Under favourable weather conditions (20-25°C, RH above 80%), the pathogen attacks young shoots, tendrils, inflorescences and berries resulting in flower/fruit shedding. Complete crop failure is usually encountered in south India whenever there is continuous rain during the early part of December (Sohi, 1983).

2.4.1.2 *Causal organism* : The disease is caused by *Plasmopara viticol* (Berk. and Curt.) Berl and de Toni. Detailed description of the pathogen is available (Butler and Jones, 1949). Srinivasan and Jeyarajan (1976) observed differences in mean sporangial germination between different times of collection. The maximum germinability of sporangia collected at 2 cm appears to serve a dual purpose in perpetuation of the fungus. It avoids loss of viability due to sun light and ensures infection by making use of a film of water on leaf surface.

2.4.1.3 *Sources of resistance* : Most of the commercial varieties except Bangalore Blue and Black Champa are highly susceptible. Fourteen varieties out of 233 were found resistant (Sohi and Sridhar, 1970).

2.4.1.4 *Control* : Bordeaux mixture is the most effective for the control of downy mildew. Besides, Zineb or Maneb (0.2 per cent), Captan (0.2-0.5 per cent) and Blitox-50 (0.3 per cent) are also effective. Cuman is another effective fungicide (Mathur *et al.*, 1973). Sohi and Sridhar (1972b) achieved best control with Fycol 8E (1.0 per cent). Kadkol

and Gopalakrishnan (1971) reported the efficacy of aureofungin (50 ppm). However, the time and number of fungicidal sprays depend on local conditions. Aluminium ethyl phosphide + Mancozeb mixture, Mancozeb and neutral Bordeaux mixture have been found to be most effective protectants (Srinivasan and Jeyarajan, 1974; Bhujbal *et al.,* 1981).

2.4.2 *Anthracnose*

In India, it was first recorded in 1903 near Pune (Butler, 1905). It is a serious disease prevalent almost throughout the country and is a limiting factor for grape production in Andhra Pradesh, Karnataka, Tamil Nadu, Rajasthan, Uttar Pradesh, Punjab and Haryana. Bedi *et al.* (1969) recorded 10-15 per cent loss due to the disease in Punjab and Haryana.

2.4.2.1 *Symptomatology* : Typical symptoms include grey-black spots with red brown margins which develop rapidly on actively growing leaves. With time, affected leaves become shot-holed and tattered. On stem, brown black spots develop into sunken cankers with slightly raised edges. As cankers coalesce, stems are girdled, weakened and killed. Slightly sunken brown spots, sometimes with grey centres, develop on immature berries. Crop loss results when flower or cluster stems and even entire shoots are girdled by cankers. Cankers also weaken canes and reduce their future productivity. Under north Indian conditions, the disease appears only during the monsoon season. The berries escape infection if the crop matures before the onset of rains. In south India, all the aerial young parts of the plant are attacked.

2.4.2.2 *Causal organism* : It is caused by *Elsinoe ampelina* Shear (Syn. *Manginia ampelina* V&P). The imperfect state of this fungus is *Sphaceloma ampelinum* de Bary (Syn. *Gloeosporium ampelophagum* (Pass) Sacc. Cheema *et al.* (1979) and Suhag *et al.* (1982) reported cultural and morphological variants and pathotypes of *E. ampelina.*

Mycelium survives in the cane and forms conidia and rarely the ascospores. The conidia can penetrate the unwounded shoots,leaves, petiole, tendrils and fruit stems. Fruits get infected when they are half grown. Anab-e-Shahi variety recorded heavy infection in 1962 when rainfall occurred immediately after October pruning at Hyderabad (Govinda Rao and Dakshinamurti, 1964). Prasad and Nirvan (1965) reported a positive correlation between heavy rains and the disease incidence. Suhag and Grover (1972) reported that the cankers are the main source of primary infection.

2.4.2.3 *Varietal resistance* : Cultivars of *Vitis vinifera* and other *Vitis* spp. differ widely in susceptibility to attack by *E. ampelina* (Mathur *et al.,* 1973; Mathur and Gupta, 1975; Kapur and Bindra, 1979; Yadav and Nirvan, 1981; Suhag and Kaushik, 1982). Beauty Seedless, Delight, Perlette and Thompson Seedless were susceptible to pathotypes of *E. ampelina* while Angur Kalan, Karachi Gulabi and Niagra were resistant

(Suhag *et al.*, 1982). Variety Delight proved the most tolerant at Hisar (Bakhshi *et al.*, 1970). Sridhar and Sohi (1970) reported resistance in 61 varieties. Goyal *et al.* (1971) reported Bharat Early and Hussaini to be completely free from infection whereas White Muscat was only slightly affected. Jeyarajan *et al.* (1970) noticed highest incidence on Bedana and the least on Khalili. Varieties Pusa Seedless, Chandigarh, Delight, Perlette, Romulus, Motia, Black Muscat, Fakdi, Hubshi, Hur and Thompson Seedless behaved as highly susceptible whereas Himrod, Schuyler White, Beauty Seedless, Niabel, Muscat, St. Valior, Large White, Golden Queen, Bangalore Blue, Isabella and Golden Muscat were resistant (Prasad and Nirvan, 1965).

2.4.2.4 *Control* : Prasad and Nirvan (1965) reported the effectiveness of an oil-based copper oxychloride formulation (Fycol 8E). Effective reduction of twig infection could be achieved by Blitox, Ziram and aureofungin (Bedi *et al.*, 1969). Effectiveness of Blitox plus Tenac (0.5 per cent each) and Miltox plus Tenac (0.3 per cent each) was reported by Ram *et al.* (1972). Sinha *et al.* observed that aureofungin (20ppm) was most effective followed by Forbam (0.3 per cent) and Bordeaux mixture. Removal of the diseased twigs, destruction of pruned materials, spraying of the vines immediately after pruning with Bordeaux mixture (5:5:50) and fortnightly sprays with Bavistin (0.1%) or Difolatan (0.3%) or Bordeaux mixture (3:3:50) during rainy season consistently gave good control at Bangalore (Sohi and Sridhar, 1972).

2.4.3 *Powdery Mildew*

Powdery mildew is prevalent almost throughout the country (Sohi, 1983) and is more problematic than downy mildew in relatively dry areas.

2.4.3.1 *Symptomatology :* The disease affects all the aerial parts. Powdery patches appear on leaves, cane, tendrils, flowers and young fruit bunches. On leaves, the powdery patches enlarge and upper leaf surface becomes dusty. The affected leaves may curl upward in dry weather. The powdery growth turns grey and finally becomes dark in colour. Diseased vines give a sickly look and show restricted growth. Affected berries do not grow, their skin becomes hard and ultimately cracks. In case of early infection, the fruits do not develop normally, become mis-shaped and crack. The disease is more prominent before the onset of rains (February-June) in south India, whereas it is more prominent during October-November under north Indian conditions. The disease infects the plants at any stage of growth (Sohi, 1983).

2.4.3.2 *Causal organism*: The disease is caused by the fungus, *Uncinula necator* (Schw.) Burr. (Syn. *Oidium tuckeri* Berk.) (Kapoor, 1967).

2.4.3.3 *Disease cycle* : Disease development is retarded by sunshine and is favoured by warm sultry weather (Sohi, 1983). Cleistothecia are believed to carry the mildew from one season to another in regions where they are formed. In majority of vineyards, the cleistothecial formation is unknown. It is considered that conidia survive the winter or the mycelium remains alive in sheltered areas on the shoot. The secondary infection

can take place by conidia. The release of conidia shows diurnal periodicity with mid-day peaks (Paddy and Subbayya, 1970). The fungus can grow over a wide range of atmospheric humidity. Its development is checked by low humidity and high temperature of summer months. Under Hyderabad conditions, warm winters favoured heavy incidence. When winter temperature was 20.1-33.5°C, the epidemic occurred (Govinda Rao and Dakshinamurti, 1964). The disease development is known to be adversely affected by precipitation.

2.4.3.4 *Source of resistance* : Cultivars of *V. vinifera* differ widely in susceptibility (Sohi and Sridhar, 1972a). Most of the cultivated varieties, especially Bangalore Blue, Bhokri and Anab-e-Shahi were highly susceptible (Sohi, 1983). Red Sultana, Saint George and 1613 were highly resistant (Sohi and Sridhar, 1970).

2.4.3.5 *Control* : Effective control can be achieved by use of sulphur (Uppal, 1927; 1930; Uppal *et al.*, 1931). Spraying the vines either with Sulfex (0.2%) or Bavistin (0.1%) or sulphur dusting at berry development gave effective control provided the first prophylactic treatment was given at proper time (Sohi, 1983). Overcrowding and dense growth of the vines should be avoided by proper pruning for disease control (Ramkrishnan and Sundaram, 1955).

2.4.4 *Minor Diseases*

Disease	Pathogen	Symptom	Distribution	References
Leaf spot	*Cercospora viticola* Ces. Sacc. *Mycosphaerella personata* Higgins	Dark brown, angular spots on leaves and young shoots	Widely prevalent in India	Munjal and Sethi (1966), Ramkrishnan and Sunderam (1955), Sinha *et al.* (1972a), Bose *et al.* (1975), Singh *et al.* (1976), Cheema *et al.* (1973)
Leaf blight	*Coniothyrium diplodiella* (Speg.) Petr. and Syd. perfect stage *Leptos-phaeria coniothyrium* (Fel.) Sacc.	Isolated red brown irregular spots appearing along the leaf margin. Leaves show reddening from the margin inwards. Lesions		Lele and Ram (1968)

(Contd.)

(Contd.)

Disease	Pathogen	Symptom	Distribution	References
		turn cinnamon brown and bear pycnidia		
Rust	*Phakospora vitis* Syd. *P. ampelophagum*	Production of orange coloured sori on the lower surface of leaves	Salem, Nilgiris, Coimbatore	Ramkrishnan and Sundaram (1955a)
Foot rot	Rhi*zoctoria solani* *Fusarium* sp., *Alternaria* sp.			Sinha *et al.* (1969)
Grapevine drying	*Hendersonula toruloidea* Natt.	Leaf with ring, cracking shedding and peeling of bark and formation of black crust-like cankerous deposition on stem	Akola	Wangikar *et al.* (1969)
Bitter rot	G*reeneria fuligenea* Seribner and Viala	Ripe and unripe berries just near the peduncle are affected. It causes light brown to dark, fuliginous, full of dense acervuli. On young berries, superficial circular brown spots develop	Dholi (Bihar)	Prakash *et al.* (1974)
Blight	A*lternaria vitis* Cavara	Appearance of patches mostly along the margins of the leaves		Vidyasekaran *et al.* (1969)

(Contd.)

(Contd.)

Disease	Pathogen	Symptom	Distribution	References
Leaf spot	*Drechslera rostrata*	Profuse marginal lesions of light brown colour which gruadually change to reddish brown	Rajasthan	Reddy (1973)
Anthracnose	*Botryodiplodia palmarum*			Patil and Moniz (1969)
Berries rot	*Pestalotia menezesiana* Bres. and Torr	Disease also appears on twigs and leaves. Infection starts just near the peduncle or tip of the fruit and rapidly cover the upper portion of the fruit. Lesions appear as water-soaked spots which turn seness'a colour with numerous acervuli of the fungus. In severe case, lesions become depressed and are associated with raised acervuli	Dholi (Bihar)	Mishra *et al.* (1974)
Wilt	Basidiomycetes	Some branches of the affected vine suddenly dry up and die eventually. In		Govinda Rao *et al.* (1964)

(Contd.)

(Contd.)

Disease	Pathogen	Symptom	Distribution	References
		severe case, whole vine dies		
Dead arm	*Phomopsis viticola* Sacc. and *Pestalotiopsis viticola*	Small angular spots on the leaves, stems and flower clusters with yellowish margins and dark centre. Pathogen also kills the arms which do not sprout after pruning and result in death of the plant	Punjab, Maharashtra, Andhra Pradesh, Karnataka	Chohan (1965), Gopalkrishna *et al.* (1978), Lali and Arya (1982)
Berry rot	Gr*eeneria uvicola*	Brown, hard, depressed patches on the berries which subsequently bear numerous dot-like black fructification		

2.5 Guava

2.5.1 *Wilt*

In India, the disease was first reported in 1935 from Babakkarpur area of Allahabad. Das Gupta and Rai (1947) recorded the disease in a severe form in orchards of Lucknow. During 1949-50, guava trees suffered serious losses in 11 districts of UP (Anon., 1949). Prasad *et al.* (1952) reported that guava wilt rapidly spread to cover about 20000 m^2 area in UP. Mathur (1956) reported that 15-30 per cent of trees in Allahabad, Farrukhabad and Unnao districts, 5-15 per cent in Kanpur and Jaunpur, and less than 5 per cent in Gorakhpur, Ballia, Hardoi, Barabanki and Varanasi districts were affected by the disease. The disease took severe form in Varanasi district (Pandey and Dwivedi, 1985). It was also reported from western districts of UP (Singh and Lal, 1953) and Delhi (Anon., 1953). Occurrence of serious wilt was reported from guava orchards

in West Bengal (Chattopadhyay and Sen Gupta, 1955) where it was confined mainly in the Gangetic alluvium of Baruipur area in the district of 24 Parganas and in the lateritic zone of Jhargram and Midnapur in the districts of Midnapur and Kashakul in the district of Bankura (Chattopadhyay and Bhattacharya, 1968). Its occurrence was reported in Karimganj, Bithoor (Kanpur), Ganga Ghat (Unnao), Abbubakarpur (Allahabad), Lucknow, Bichpuri (Agra), Sasni (Aligarh) (Anon., 1986, 1987) and found that the cultivars Safeda and Purba were badly affected. The disease has also been reported to occur in Haryana, Punjab and Rajasthan.

Singh and Lal (1953) observed loss at about 5-10 per cent every year in 12 districts of UP which was estimated around Rs. 0.8 to 1 million. In West Bengal, it reduced the yields by 80 per cent, i.e., from 113.5 q/ha in healthy plantations to about 18.2-22.7 q/ha in affected plantations (Chattopadhyay and Sen Gupta, 1955). The attempts to regenerate the affected trees failed and new seedlings or grafts planted in affected areas showed stunted growth, rarely flowered and succumbed to wilt within a very short time (Chattopadhyay and Bhattacharya, 1968).

2.5.1.1 *Symptomatology* : First external symptom of the disease is yellow colouration with slight curling of the leaves on the terminal branch. Plants, at a later stage, show drowsiness with yellow to reddish discolouration of leaves. Subsequently, there is premature shedding of leaves and fruit size become smaller. Some of the twigs become bare and fail to bring forth new leaves or flowers and eventually dry up. Fruits of all the affected branches remain underdeveloped, hard and stony. Later, the entire plant becomes defoliated and eventually dies. Usually, it takes fifteen days for complete wilting of the tree but may sometimes take even up to one year. The finer roots show black streaks which are prominent on removing the bark (Das Gupta and Rai, 1947). Chattopadhyay and Bhattacharya (1968) reported that the roots also show rotting at the basal region. The bark is easily detachable from the cortex. The cortical regions of the stem and root show distinct discolouration and damage. Light brown discolouration is also noticed in vascular tissues. According to Singh and Lal (1953) and Edward (1960), it attacks young as well as old bearing trees. New seedlings and grafts also show disease symptoms.

2.5.1.2 *Causal organism* : The exact cause of the disease is still not fully understood but the pathogens, viz.,*Fusarium oxysporum* f. sp. *psidii, F. solani, Macrophomina phaseoli, Rhizoctonia bataticola* and *Cepahlosporium* sp. may incite the disease.

Both *M. phaseoli* and *F. solani* were found to incite wilt either individually or jointly. In either case, the fungus first colonises the surface of the roots and then enters into the epidermal cells. Thereafter, intercellular mycelium establishes first in epidermal cells and then spreads into cortical cells which are considerably damaged and filled with the mycelium. *F. solani* enters the xylem vessels, grows inside and blocks them. *M. phaseoli* first invades the phloem and destroys it. The xylem vessels are also attacked in few cases (Chattopadhyay and Bhattacharya, 1968).

The disease is caused by *Gloeosporium psidii* Delacroix = *Glomerella psidii* (Del.) Sheld. which is now called *Colletotrichum psidii* Curzi.

The mycelium becomes intercellular, branched and light brown in colour. Brown to dark brown acervuli are formed on the affected parts of the plants. Setae and conidia are formed in the acervuli. Conidiophores are hyaline and small, setae are long, tapering at the end, dark brown to black in colour. Conidia are formed at the tip of the conidiophore and are sickle-shaped, unicellular, hyaline measuring 11.24 × 4.5 - 5 mm. They germinate by germ tube. In moist weather, acervuli appear as black dots on twigs or fruits which later produce pinkish spore mass. Spores are disseminated by wind or rain and initiate fresh infection.

2.5.2.1 *Die-back phase :* In Minto Park, Allahabad, in 1952, the intensity of the disease varied from complete dead to half dead plants. Ripe fruits showed pinkish masses of acervuli.

a) *Symptomatology :* The plant begins to die backwards from the top of a branch. Young shoots, leaves and fruits, while still tender, are readily attacked. The greenish colour of the growing tip is changed to dark brown and later to black necrotic areas extending backwards causing the die-back. The disease is noticeable more after a period of incubation in the infected buds and twigs. The brown spots, formed previously, change into silvery grey and ultimately develop at the junction of the diseased and healthy part. The fungus develops from the infected twigs and then petiole and young leaves are attacked. These may droop down or fall leaving the dried leafless twigs. In moist condition, acervuli of the fungus may be seen as black dots scattered throughout the dead parts of the twigs (Tandon and Agarwala, 1954).

b) *Disease development :* After slight rain, old twigs show symptoms of die-back. In the first fortnight of August, the disease causes death of small, tender growing twigs. From August to October, the infected twigs wither and shrivel at their tips. The disease appears in epidemic form during August-September. It is also noticed in December, after which the older leaves and twigs remain immune from attack. During extreme and less humid weather conditions from January to June, further progress of the disease is greatly inhibited (Tandon and Agarwal, 1954).

c) *Pathogenicity :* Tandon and Agarwal (1954), from pathogenicity test on twigs and stalks, observed that in rainy season, symptoms develop after 15-20 days of inoculation. Inoculation without injury gives negative result. In summer, even after injury symptom development fails. Thus, humidity is necessary for the growth of the organism. Old branches resist infection. The young leaves show dead areas on the margin and the tips which generally appear after one week of inoculation. When flowers were inoculated, unopened buds showed infection within two or three days and failed to develop, petals turned brown and buds

fell without opening. On inoculation of fruits, rot developed in the ripened fruits but they never developed mummy.

d) *Control* : Although complete control was not possible, the application of 3:3:50 Bordeaux mixture and 0.22 or 0.33 per cent Perenox gave encouraging results in reducing the development of die-back and mummies (Tandon and Agarwal, 1954).

2.5.2.2 *Fruit and leaf infection phase* : Fruit and leaf infection was reported from Saharanpur (Tandon and Singh, 1969). Appearance of fruit spots was seen specially in rainy season.

a) *Symptomatology* : Pin-head spots were first seen on the unripe fruits which gradually enlarged measuring 5-6 mm in diameter. They were dark brown to black in colour, sunken, circular and had minute, black stromata in the centre of the lesions which produced creamy spore masses in moist weather. Several spots coalesce to form bigger lesions. The infected area in the unripe fruits become corky and hard, and often develop cracks in case of severe infection. On ripe fruit, the infection causes softening of tissue and lesions attain a diameter of 10 to 20 mm (Tandon and Singh, 1969) Unopened buds and flowers are also attacked and cause their shedding. Spread of infection is very rapid on fully mature green fruits, whereas young fruits do not normally take infection (Midha and Chohan, 1968), perhaps owing to the differences in the concentration of K-ions in the tissues.

On leaves, the fungus causes necrotic lesions at the tip or on the margin. These lesions are usually ashy grey and bear fruiting bodies. The tender twigs are also infected which wither and die from the tip downwards giving it a wither-tip appearance (Tandon and Singh, 1969).

b) *Pathogenicity* : The leaves and stems of seedlings, new leaves, buds, flowers and fruits are infected readily. Tandon and Agarwala (1954) were unable to infect the green fruits under artificial conditions and were of the opinion that the fungus remains latent for $2^1/_2$ to 3 months and spots appear when fruits ripen. According to them, the hard tissues of young fruits prevent the fungus from growing but Tandon and Singh (1969) were able to infect the young, hard and ripe fruits.

The disease was found to develop more rapidly at 30°C and 96.1 per cent relative humidity both in ripe and unripe fruits.

c) *Varietal resistance* : Apple Guava (deep red fleshed), Apple Shaped Seedling, Behat Coconut, Red Chittidar, Muzaffarnagar, Bulandshahar, Lucknow-49 and *Psidium chinense* Lodd., *P. cattleianum* var. *lucidum* Sab., *P. guinense* and *P. molle* Bertol were susceptible to anthracnose. Apple Guava (light fleshed) had moderate resistance (Tandon and Singh, 1969). *P. chinense* resisted leaf

infection whereas *P. molle* and Beumont were highly susceptible and Allahabad Safeda developed heavy infection on fruits (Anon., 1974).

d) *Control* : Effective control of anthracnose can be achieved by sprays of Bordeaux mixture (3:3:50) at 7 days interval. Copper oxychloride and cuprous oxide also controlled the disease (Tandon and Singh, 1969). Monthly sprays of Difolatan (0.3%) closely followed by Dithane Z-78 (0.2%) were found effective (Anon., 1974). Since Bordeaux mixture and other copper fungicides caused russetting of fruits especially in Allahabad Safeda variety, it reduced their market value (Sohi and Sridhar, 1969).

Gupta *et al.* (1973) found that 20 minutes dip treatment in 500 ppm tetracycline is effective but none of the five fumigants could inhibit spore germination. Pre-treatment of sodium metabisulphite and stable bleaching powder proved cent per cent effective under *in vivo* conditions (Singh and Sharma, 1982). Sharma *et al.* (1983) found that pre- and post- treatment with five chemicals could not completely protect the fruits from rotting although thiabendazole and aureofungin proved better.

2.5.3 *Canker*

Fruit canker caused by *Pestalotia psidii* was recorded from Bombay (Chibber, 1911) and later from Mysore (Narsimhan, 1939; Venkatkrishniah, 1952), Thane, Dharwar, Pune (Patel *et al.,* 1951) and Ponta valley (Himachal Pradesh) (Verma and Sharma, 1976). Since then no information is available from other parts of the country except Lucknow (Anon., 1986).

2.5.3.1 *Symptomatology* : The disease generally occurs on green fruits and rarely on leaves. The first evidence of infection on fruit is the appearance of minute, brown or rust coloured, unbroken, circular, necrotic areas which, in advanced stage of infection, tears open the epidermis in a circinate manner. The margin of lesion is elevated and a depressed area is noticeable inside. The crater-like appearance is more noticeable on fruits than on leaves. The canker is confined to a very shallow area and does not penetrate deep into the flesh of the fruit. In older cankers, fruits of white mycelium consisting of numerous spores are noticeable. Canker on the green fruits of different varieties exhibit considerable differences in their appearance (Patel *et al.,* 1951). In severe cases, raised, cankerous spots develop in great numbers and the fruits break open to expose seeds. The infected fruits remain underdeveloped, become hard, malformed and mummified and drop in great numbers. Sometimes small rusty brown angular spots appear on the leaves (Venkatakrishniah, 1952). Verma and Sharma (1976) studied the seasonal variation in symptoms caused by *Pestalotia psidi* in Ponta valley. In winter, the cankerous spots are common but in rainy season, minute red specks are formed.

2.5.3.2 *Causal organism :* Fruit canker is caused by *Pestalotia psidii* (Chibber, 1911). Narsimhan (1940) and Venkatakrishniah (1952) found *Colletotrichum psidii* Curzi

(*Glomerella psidii* (Del) Sheld) and *Pestalotia psidii* Pat. associated with a canker. Venkatakrishniah (1952) advocated that *Colletotrichum psidii* is a general parasite and *Pestalotia psidii* is specialized to guava. Both species were present on young, green and mature fruits/leaves.

Patel *et al.* (1951) found that the canker of guava is caused by *Pestalotia psidii.* The dark black and circular pycnidia on the culture media and fruits contain conidiophores and conidia. The conidia are typically 5-celled, oblong, clavate or elliptic-fusoid, erect, hardly constricted at septa, measuring 13-31 × 5-10 m; 3 medium cells are guttulate, highly brownish, the central cell being the thickest and greatly bulged, and cells are hyaline; the apical, conical or cylindrical cell grows out into 3 hyaline, slender, elongated appendages, the basal cell is obtuse, erect with a small pedicel. The mycelium of young culture is sub-aerial, serrate, thin, septate, cottony white to pinkish, irregularly branched and measuring up to 3 μ in diameter. In old cultures, the hyphae are more or less thickened (Patel *et al.,* 1951).

2.5.3.3 *Epidemiology* : The fungus grows profusely on different media but scanty vegetative growth occurs on green guava decoction. Sporulation is quick and abundant on Richards' and PDA. The pathogen grows vigorously at temperatures between 15 and 30°C. Best growth and sporulation is seen at 26°C. Kaushik *et al.* (1972) stated that the maximum disease occurs at 25-30°C and at high RH. The fungus grows profusely on media containing mannitol, dextrine and sucrose, fairly on maltose and salicin, poorly on ripe guava decoction, glycerol and Richards' modified agar. Best sporulation is recorded on mannitol, dextrine and sucrose. With different sources of nitrogen, the growth characters of *P. psidii* remained practically constant but the sporulation was found profuse in case of potassium nitrate and asparagin. The fungus grows in a wide range of H-ion concentration, but the optimum pH was 3.9-4.9 with maximum growth at pH 4.9. The spores of the fungus germinated at 10°C and increased with rise in temperature up to 32°C. Maximum growth was recorded on sulphates or cystein (Tandon, 1950). On thiourea, growth was moderate but sulphide, dithionate and persulphate were only feebly utilized.

Addition of green fruit tissue and its decoction gave greater stimulation to germination than the ripe fruit tissue. The fungus remained viable in conidial stage up to 38.8°C (Patel *et al.,* 1951). Ramaswami *et al.* (1984) observed that germination of spores of *Pestalotia psidii* was maximum at 30°C and it did not germinate below 15°C or above 40°C. The best germination medium was guava fruit extract. High RH (98%) was required for germination.

2.5.3.4 *Pathogenicity* : Detached fruits, both ripe and unripe, failed to produce symptoms. Fresh, young, green guava fruits when inoculated after wounding gave typical severe symptoms while in unwounded fruits, symptoms developed in 8 days. Artificial infection on leaves was generally unsuccessful. The pathogen is primarily a wound parasite (Patel *et al.,* 1951).

2.5.3.5 *Varietal resistance* : In Lucknow 49, development of canker pustule was large, more elevated and numerous. On Dholka, it was not well developed. On Sind, the development of pustule was insignificant and inconspicous while cultivar Nasik was almost immune (Patel *et al.,* 1951). Edward *et al.* (1964) found Safeda and Apple Colour as highly resistant cultivars to canker.

2.5.3.6 *Control* : The spread of disease (in early stages of infection) was controlled by 3 or 4 sprayings of 1 per cent Bordeaux mixture or lime sulphur (1 in 25) at 15 days interval (Venkatakrishniah, 1952).

Khanna and Chandra (1977) observed that the homeopathic drugs, Kali (potassium) iodide at potencies of 1,20,24,61 and 67, and Arsenicum album (arsenic oxide) at potencies of 60,65 and 82 completely inhibited spore germination *in vitro.* Potassium iodide at potencies of 1,20,24 and 61, and arsenic oxide at potency of 60 inhibited growth of the pathogen. Fruits treated with some of the effective potencies before inoculation did not develop rotting.

Leaf extracts of *Azadirachta indica* and *Ocimum sanctum* were found to inhibit the germination of spores *in vitro.* Dipping of guava fruits in these extracts before or after inoculation was effective. Use of *O. sanctum* extract was recommended as it did not affect fruit flavour (Pandey *et al.,* 1983).

2.5.4 *Phytophthora Fruit Rot*

It was first reported by Mitra (1929) from Pusa, Bihar. The fallen fruits were especially affected as also those hanging near the ground level. It caused considerable losses in Mysore during rainy season on Allahabad Safeda and is now known to occur in Tamil Nadu, Andhra Pradesh, Punjab and Maharashtra (Sohi and Sridhar, 1971).

The incidence of disease varied from 5 to 20 per cent on varieties Allahabad Safeda, Apple guava, Red Fleshed and Pink Fleshed.

2.5.4.1 *Symptomatology* : *Phytophthora nicotianae* (Van Breda de Haan) var. *parasitica* (Dastur) Waterh. attacks unripe fruits at the styler end. The whitish cottony growth develops very fast as the fruit ripens and covers almost entire surface within 3-4 days in humid weather. The fruits near the soil level, covered with dense foliage, under high relative humidity are most severely affected. The skin of the fruit below the whitish cottony growth becomes a little soft, turns light brown to dark brown and emits a characteristic unpleasant smell. The diseased fruits generally retain their normal shape unless they are invaded by saprophytes which cause rotting. These fruits either remain intact or drop off. When the disease appears on young and half grown fruits, they shrink, turn dirty brown to dark brown, hard in texture and either remain intact as mummified fruit or drop off (Singh *et al.,* 1976a).

2.5.4.2 *Control* : Kothari (1968) recommended weekly sprays of Bordeaux mixture (2:2:50) and copper oxychloride (0.2 per cent). According to Sohi and Sridhar (1969),

it is not advisable to use copper fungicides on fruits of cultivar Allahabad Safeda since toxicity appear in the form of numerous, small dark brown specks which later coalesce and form extended russetted area. Excellent control of the disease was achieved by Dithane Z-78 (0.2 per cent) and aureofungin (10 ppm).

2.5.5 *Dry Rot*

The dry rot of guava fruits is caused by *Diplodia natalensis.* In Vellayani, it was observed during 1969. In some of the infected trees, more than 40 per cent of the fruits showed symptoms which appeared initially as light brown spots mostly at the stalk end or at the calyx-end of the fruit. In few cases, infection originated from other parts of the fruits. The infection spreads quickly and within 3 to 4 days the entire fruit is affected. Completely infected young and mature fruits become dark brown to almost black and ultimately dry up. A number of dry fruits can be seen on infected trees. Numerous pycnidia of the pathogen appear as pin-head structures on the rind of the dried fruits.

The pycnidia of the fungus produced on guava fruits are erumpent, more or less globose, dark coloured and measure 175-475 µ × 90-185 µm. Pycnidiospores are initially hyaline, oblong and unicellular. On maturity, they become oblong to elliptical, two-celled and dark brown having longitudinal striations on the wall. They are 21.5 to 32.2 µ long and 12.2 to 16.5 µm wide (Rajgopalan and Wilson, 1972a).

Ghosh *et al.* (1964) observed that sucrose, glucose and fructose concentration increased with age in non-inoculated fruits. Sucrose of guava fruit was slowly hydrolysed by *D. natalensis.*

2.5.6 *Minor Diseases*

Disease	Causal organism	Symptoms	Control	Reference
Twig blight	*Phomopsis psidii* Nagraj & Pannapa	Light brown water-soaked lesions formed on the nodes and internodes of young shoots and twigs, which develop into dark brown spots. The spots later extend upward and downward.	—	Singh and Verma (1985)

(Contd.)

(Contd.)

Disease	Causal organism	Symptoms	Control	Reference
		In severely attacked plants, the twigs defoliate and dry		
Stem canker	*Diplodia natalensis* Pole-Evans/ *Physalospora psidii* Uppal	Longitudinal cracks in the bark of stem or branches are visible on scraping, dark brown to black streaks or bands are present in the subcutical region. The affected bark turns dark brown to greyish with vertical cracks and ultimately passes on to the main trunk and upper roots. Leaves on the engirdled portions lose their colour slowly and become purplish bronze	—	Rana (1981), Uppal (1936)
Drying/ defoliation and die-back	*Torula* stage of *Hendersonula toruloidea* Nattrass.	Die-back, drying of twigs and defoliation of leaves from the tree	—	Mathur and Singh, (1959, 1964)
Leaf blight	*Phoma jolyana* Pirozy & Morg.	Both young and old leaves are susceptible to	—	Sridhar and Ullasa, (1978)

(Contd.)

(Contd.)

Disease	Causal organism	Symptoms	Control	Reference
		disease. Small circular spots with dark brown centre surrounded by a reddish margin, gradually enlarge and coalesce resulting in large necrotic patches. Several affected plants are completely defoliated		
Leaf blight	*Alternaria alternata* (Fr.) Keissler	Irregular brown to dark brown spots appear on the leaves. The smaller spots coalesce to form blighted patches. Later defoliation take place	Spray Difolatan or copper oxychloride (0.2%) twice at 15 days interval	Gupta and Madaan (1979)
Leaf spot/ white spot	*Cercospora sawadae* Yamamoto *C. psidii* Rengel	Disease appears as water-soaked, brown, irregular patches on the lower surface and yellowish on the upper surface of the leaf. Old leaves are mostly affected. The central portion of the spot turns white, surrounded by brownish margin	—	Ragunathan and Prasad (1969), Bose and Muller (1967), Bose (1969)

(Contd.)

(Contd.)

Disease	Causal organism	Symptoms	Control	Reference
Leaf spot	*Pestalotia jodhpurensis* Bilgrami & Purohit, *Pestalatiopsis psidii* (Pat.) Mordue	Spots originate from the tips or margins, gradually advance towards the base, assuming dark brown-grey colour. Fruiting bodies develop profusely on the upper surface of leaves	—	Bilgrami and Purohit (1971), Anon. (1987)
Leaf spot	*Curvularia siddiquii* Ahmad & Quraishi	Dark brown spots appear on the leaves, infection is restricted only on the tips/ margins but subsequently spot covers the whole leaf lamina	—	Srivastava (1964)
Damping-off	*Rhizoctonia solani* Kuhn.	Both phases of the disease are observed. In pre-emergence phase, the infected seeds and seedlings show water-soaked discolouration. The seed becomes soft and ultimately rots. Affected young seedlings	Seed treatment with Bavistin and Brassicol at 3 and 5 g/kg seed respectively	Gupta (1978)

(Contd.)

(Contd.)

Disease	Causal organism	Symptoms	Control	Reference
		are killed before they reach the soil surface. In post-emergence phase, the hypocotyl is discoloured into yellowish to brown and later turns soft, rots and constricts. Such seedlings ultimately topple down and die		
Dry rot	*Diplodia natalensis* Evans	Symptoms appear initially as light brown spots mostly at the stalk end or at the calyx end of the fruit. The infection spreads quickly and within 3-4 days, the entire fruit is affected. Completely infected young and mature fruits become dark brown to almost black in colour and ultimately dry up. A number of dry fruits can be seen on infected trees	Ziride (3000 ppm) at 15 days interval	Rajgopalan and Wilson (1972a, b)

(Contd.)

(Contd.)

Disease	Causal organism	Symptoms	Control	Reference
Phomopsis rot	*Phomopsis destructum*	The infected fruit shows disease symptom near the stalk. Lesions are dark brown, at first, small and increase in size to 2 cm diameter. The tissue soften and the entire fruit rots within 8-12 days. The rotten fruits fall causing a heavy loss in yield	Griseofulvin (22 ppm) followed by aureofungin and Nystatin (100 ppm), Blitane, Blitox and Cuman 0.1% *in vitro*	Rao *et al.* (1976)
Guignardia fruit rot	*Guignardia psidii* Ullasa & Rawal	Minute depressed or flattened spots develop on ripening fruits in a concentric manner. Several spots later coalesce and form bigger lesions	—	Ullasa and Rawal (1984) Anon. (1986)

2.6 Litchi

A number of fungal diseases have been reported on litchi but none of them are of economic importance. Some post-harvest rots commonly occur after the injury on fruits. These diseases are summarised below :

Disease	Pathogen	Symptoms	Distribution	Reference
Leaf spot	*Pestalotia pauciseta* Sacc.	Show light discolouration on both surfaces of leaf. Size	Muzaffarpur	Prasad (1962, 1967)

(Contd.)

(Contd.)

Disease	Pathogen	Symptoms	Distribution	Reference
		of the spot varies from 0.5-2 x 0.2-2 cm. Lesions coalesce at later stage. The colour of the spot changes from brown to russet or mars brown		
Leaf spot	*Cephaleuros virescens* Kunze	Orange yellow out-growth surrounded by dark yellow halo with a velvety coating on the lower surface of the leaf. Spots are circular to semi-circular	Dholi (Bihar)	Mishra *et al.* (1973)
Leaf spot	*Microdiplodia litchi*	The diseased leaves show yellowish brown to brick red areas mostly around the margin. The coloured areas gradually become light brown and show black dot-like pycnidia	Udaipur (Rajasthan)	Pathak and Desai (1971)
Leaf spot	*Colletotrichum gloeospo-rioides* Penz.	Spots usually start from the tip of lamina and extend towards the base. Spots are irregular in outline, black to brown in colour with a prominent brown margin encircling them	Muzaffarpur (Bihar)	Prasad (1962, 1967)
Leaf spot	*Botryodiplodia theobromae* Pat.	Spots usually start from tip or the margin of lamina. These are deep chocolate in colour. The	Muzaffarpur (Bihar)	Prasad (1962, 1967)

(Contd.)

(Contd.)

Disease	Pathogen	Symptoms	Distribution	Reference
		limiting margins of the spot with irregular outline are vandyke brown. Black pycnidia appear on both surfaces of leaves		

Some of the minor pathogens of litchi are given below :

Source	Pathogen	Reference
Leaf spot	*Arthrinum phaeospermum*	Billgrami (1970)
	Drechslera hawaiiensis	Misra *et al.* (1973)
	Alternaria tenuis	Tandon (1967)
	Cladosporium herbarum	-do-
	Memmoniella echinata	-do-
	Nigrospora sphaeriea	-do-
Twig blight	*Curvularia ovoidea*	Bilgrami (1970)
	C. verruculosa	-do-
	Nigrospora sphaerica	Tandon (1967)
	Dictyoarthrinium sp.	Bilgrami (1970)
Flower blight	*Botryodiplodia theobromae*	Tandon (1967)
	Pestalotia pauciseta	-do-
	Nigrospora sphaetica	-do-
	Chaetonium globosum	Bilgrami (1970)
	Chlamydomyces sp.	Tandon (1967)
	Fusarium sp.	-do-

2.7 Loquat

2.7.1 *Wither Tip*

The disease is reported from Delhi and western UP (Lele and Ram, 1969). It causes shedding of leaves and withering of tips of branches. On leaves, first light green spots appear which later turn brown. Generally, the spots develop at the tip or margin of the leaves. Acervuli develop as black dots on twigs and leaves from which

pinkish masses of spores ooz out. For its control, branches should be first pruned and then treated with Chevastelon solution (6 per cent cold solution of potassium dichromate mixed with 6 per cent cold solution of copper sulphate) (Lele and Butani, 1975).

2.7.2 *Collar Rot*

It is reported from UP (Tandon, 1965) and is caused by *Sclerotium* sp. which results in complete girdling. *Diplodia* sp. infects invariably as secondary pathogen. Earlier, Tandon (1961) described the disease due to *Diplodia natalensis.* For control of the disease, scraping of infected bark and then application of copper oxychloride or Bordeaux paste is suggested.

2.7.3 *Twig Blight and Canker*

Twig blight and canker reported by Lele and Ram (1969) from Delhi and Western UP, was caused by *Cytospora chyrysosperma* Pers. ex. Fr. The symptom of the disease is similar to die-back. The affected plant shows sick look, less foliage and blighted appearance. On diseased twigs, embossed, globose and cankerous pimples appear. The bark becomes loose and give amber-yellow exudate which becomes horny on drying. Lele and Butani (1975) suggested application of Chevastelon solution after pruning.

2.7.4 *Die-back*

It is caused by *Cytospora eriobotryae* Curzi and Barbaini (Singh *et al.,* 1969). The plant dries from top downwards. The affected twigs dry and the bark becomes soft and shrunken. Later, orange coloured spore tendrils (cirrhi) are produced. The bark cracks and twigs dry.

2.7.5 *Leaf Blight*

Dar and Zargar (1986) isolated *Fabraea maculata (Diplocarpon mespili)* in imperfect state from loquat with severe leaf blight in Srinagar.

2.7.6 *Soft Rot*

Lesions on fruit are caused by *Pestalotiopsis versicolor* and *Fusarium semitectum* (Joshi *et al.,* 1983).

2.7.6.1 *Pestalitiopsis versicolor* (Speg.) Steyart. : The young spots are small, dark brown and water-soaked. In advanced stages, the infected portions of fruits become very soft and tufts of spores are found over the infected surface.

2.7.6.2 *Fusarium semitectum* Berk and Rav. : The lesions are water-soaked and smaller but completely destroy the fruit within 15 days. The entire rotten surface of fruit is covered by pinkish white cottony growth of pathogen.

2.8 Mango

2.8.1 *Powdery Mildew*

Powdery mildew is one of the devastating diseases of mango affecting almost every cultivar. In India, the disease is widespread (Mc Rae, 1924; Kulkarni, 1924; Anon., 1930; Galloway, 1935; Uppal, 1937; Singh and Garg, 1949; Vaheeduddin, 1953; Venkataraman, 1956) including in the hill valleys (*terai* region) and plains of UP (Singh and Garg, 1949; Bose, 1953; D.I.J., 1964) and is a serious threat to mango production. Its severity mainly depends on climatic conditions (Prakash and Srivastava, 1987). In Maharashtra, the losses have been estimated up to 20 per cent (Anon., 1930; Cheema *et al.,* 1954). At Lucknow, loss due to this disease varied from 30 to 90 per cent (Prakash and Srivastava, 1987).

2.8.1.1 *Symptomatology* : The disease usually manifests during January to March (flowering time) but at elevations of 600 to 1200 metres, it is known to persist for longer periods. The characteristic symptom is the white superficial, powdery appearance of fungal growth on inflorescences, leaves, stalks of the inflorescences and young fruits (Fig. 3). The mildew attacks mango flowers before fertilization and results in dropping of infected unfertilized flowers. Young fruits are covered entirely by the mildew. As the fruit grows, its epidermis in the infected area cracks and corky tissue is formed and then it drops prematurely normally at pea size stage. The disease is largely responsible for dropping of young fruits in India (Kulkarni, 1924). Dropping of unfertilised infected flowers and immature fruits leads to serious losses (Wagle, 1928). New leaves are attacked mostly on the underside, but in advanced cases, both sides of the leaf are attacked. The symptoms are restricted to the area of the central rib and such leaves curl and become distorted (Prakash and Srivastava, 1987).

2.8.1.2 *Causal organism* : Powdery mildew was attributed to *Erysiphe cichoracearum* (Wagle, 1928). Histological studies revealed that mildew fungus has globular haustoria and the type of its conidial germination should be referred to *Erysiphe polygoni* (Uppal, 1937). It was later observed that the fungus produced seccate or lobate haustoria which is not characteristic of *E. cichoracearum* (Uppal *et al.,* 1941). As no description of the perfect stage of mango mildew fungus was given, the name of the conidial stage, *Oidium mangiferae* Berthet is preferred (Uppal *et al.,* 1941). Wagle (1928) studied the causal fungus which is disseminated by wind from infected leaves or inflorescences to the healthy ones. The wind borne conidia cause infection after germination which takes 5-7 hours and mycelium is produced on the fourth day which gives rise to conidia in 5 days. The life cycle of the mildew is completed in 9 days on the vegetative shoots (Wagle, 1928). The morphology of *O. mangiferae* has been described in detail (Uppal *et al.,* 1941). Conidia germinate by protrusion of a germ tube. Hyphae in contact with the host form appressoria and from the latter and elsewhere on the hyphae, tube like haustoria are formed. These pierce the cuticle and cell walls and then swell inside the epidermal cells to form globular structures.

2.8.1.3 *Morphology* : The mycelium is septate, composed of hyphae with 4.1-8.2 µm. Conidiophores emerging from the superficial mycelium are unbranched, 64-163 µm long and bear at their ends unicellular, hyaline, elliptical conidia. These vary greatly in size, but are most frequently 33-43 µm long and 18-22 µm wide. Conidia are borne singly, sometimes in pairs or in chains of 20-40 on detached leaves kept in closed containers.

2.8.1.4 *Epidemiology* : The fungus has been stated to be favoured by cloudy weather and heavy morning mists (Kulkarni, 1924). The disease is particularly destructive in the coastal area of Maharashtra during cold and wet season. Minimum, optimum and maximum temperature for germination are respectively 9°, 22° and 30-32°C (Uppal *et al.,* 1941). Gupta (1979, 1985) found that the atmospheric temperature affected the appearance and development of the disease. Minimum temperature of 10-13°C and maximum of 27-31°C alongwith high humidity were found to be most suitable for the development of the disease. Increased humidity owing to rainfall helped in maintaining high level of infection (Gupta, 1979; Prakash and Srivastava, 1987). Misra and Prakash (1988) stated that predominance of susceptible variety (Dashehari), high wind velcocity for 3-4 days with maximum temperature above 30°C, minimum temperature around 15°C, and maximum relative humidity of 73.3-83.9 per cent and minimum of 23.4-25.5 per cent were found conducive for the rapid spread of powdery mildew during 1986-87 in Malihabad and Kakori in UP.

2.8.1.5 *Perpetuation of fungus* : The fungus is not known to attack hosts other than mango and its sexual form has not been recorded (Prakash and Srivastava, 1987). During off season, the pathogen has been reported in intact green malformed panicles mostly hidden under dense foliage (Singh, 1979). Studies conducted at Lucknow revealed that mildew pathogen persists on infected leaves of the previous year's flush which are retained on the plant in the succeeding year. During flowering period (January-March), the conducive environmental conditions activate the dormant mycelium in necrotic leaves. Abundant conidia are produced and blown over to the new flushes on young panicles which provide sufficient spore load for initiating the disease. Fresh infection of mildew on young leaves in the first week of December (when flowers are not present) further confirms that the fungus perpetuates in asexual form on leaves of mango (Prakash and Raoof, 1985c). Datar (1985), Gupta (1985) and Munshi *et al.* (1988) studied the perpetuation of *O. mangiferae* on leaves and malformed inflorescence. The conidia could not be located after August in malformed bunches, germination was highest in June, decreased in July and no germination was observed in August (Datar, 1985).

2.8.1.6 *Biochemical studies* : There was a continuous decrease in the percentage of moisture and acidity while pH increased with rise of powdery mildew infection. In infected panicles, the protein content was higher and reducing sugars were low. The total sugars and tannins decreased with the severity of the disease but due to excessive moisture loss in the panicles at 3rd stage, a slight increase was registered than in healthy panicles (Prakash *et al.,* 1989).

2.8.1.7 *Varietal susceptibility* : At Basti (UP), out of 90 mango varieties, only Neelum, Zardalu, Bangalora, Totapari-Khurd and Janardan Pasand were found resistant (Gupta, 1976). Datar (1983) found Totapari to have some degree of resistance. During 1986 and 1987, incidence of powdery mildew was recorded in 106 and 275 mango cultivars respectively. Out of these, 85 showed low, 19 medium and 2 high incidence of powdery mildew (Anon., 1986). In 1987, 250 cultivars showed low, 18 medium and 7 high incidence of the disease (Anon., 1987).

2.8.1.8 *Control* : Three applications of sulphur dust (250-300 mesh) were recommended in the State of Bombay (Anon., 1930). Spray of 1 per cent Bordeaux mixture followed by Fernasol (1 gallon in 60 gallons of water) controlled mildew in Mysore (Venkataraman, 1956). Bose (1953) found that Sandolin (0.3%) was quite effective besides three other sulphur preparations, namely, Thiovit, lime sulphur and Spersul. Cheema *et al.* (1954) recommended the use of 1-3 lb of sulphur in the coastal areas. Bordeaux mixture (3:3:50) was also reported to be very effective (Naik, 1949). The disease can be controlled by dusting sulphur (200-300 mesh) spraying Hexaferb (0.2 per cent) or Thiovit (Pathak, 1970), thiophanate methyl followed by Dinocap, Bavistin, Oxythioquinone (Datar, 1981). Sridhar and Sohi (1973) achieved good control by spraying Cosan. Effective control of mildew has been achieved by spraying Bavistin, Benomyl, Calixin, Microsul and Nimrod at Lucknow (Prakash, 1979); Prakash and Srivastava, 1980; Prakash and Singh, 1982). At Lucknow, Bavistin was quite effective followed by Benomyl when sprayed at 10 days interval. Bavistin even when sprayed at 20 days interval was quite effective in reducing the disease incidence (Prakash and Raoof, 1982a, 1985d). Three sprays of Microsul (0.2%) or Bavistin (0.1%) were the most effective in controlling disease (Prakash and Raoof, 1982).

In certain regions, both powdery mildew and mango hopper occur together in which case a combined treatment of fungicides and insecticides was recommended (Pal and Prakash, 1984). Spray with fungicides Karathane, Wettasul (Gupta and Yadav, 1984), Baycor, Bayleton, Calixin (Rawal and Ullasa, 1985), Sulfex-O and Sulfex-A *in vitro* 2,000 ppm (Jadeja *et al.*, 1985), Tridemorph and Dinocap (Joshi and Chauhan, 1985), Karathane followed by Bavistin, Calixin, Jkstein, Thiovit, Devisulf and Saprol (Anon., 1986) have been found effective against the disease.

All the wettable sulphur fungicides significantly checked the disease without any phytotoxic effect. Use of wettable sulphur reduced the cost of application. Fungicides other than carbendazim and sulphur like Baycor (0.1%), Systhane (12% EC) 0.1% SAN 619 F (0.15%), Bayleton (0.1%) and Saprol (0.1%) also proved effective (Anon., 1987).

2.8.2 *Anthracnose*

Anthracnose, also known as blossom blight or leaf spot or fruit rot, is a common, destructive and widespread disease in all mango growing states of India. Various manifestations of the disease on mango include blossom blight, peduncle blight, leaf

spot, twig blight, wither tip, fruit russeting or staining and fruit rot (Singh, 1960; Prakash, and Raoof, 1985a; Prakash and Srivastava, 1987; Mc Rae, 1924; Uppal *et al.* 1935; Stevens and Pierce, 1933; Sattar and Mallik, 1939; Hayes, 1953; Gandhi, 1955; Sohi *et al.,* 1973; D.I.J., 1964; Paracer and Chahal, 1963). The disease is severe both in field and storage. Bose *et al.* (1973) reported that young plantations of Bombay Green variety were completely wiped out from *terai* region of UP as a result of severe wither tip.

2.8.2.1 *Symptomatology* : Numerous oval or irregular vinaceous brown or deep brownish spots of variable size, round or angular in shape scattered over the leaf surface are the characteristic symptoms (Fig. 4). The spot may begin at the tip or from any other portion on the margin or it may develop in the centre of the leaf. Under damp conditions, the spot grows rapidly forming elongated mars brown or mummy brown necrotic areas measuring 20-25 mm in diameter which later rupture and become blighted. They do not become much larger as the leaf grows, but often dry and fall out, giving the older leaves a shot hole appearance. Young leaves are more prone to attack than old ones. Leaves and young panicles infected with gall midge insect activate the fungus resulting in heavy incidence of the disease in certain areas (Prakash and Srivastava, 1987). The midge larvae mine the leaves and form small galls on them. After the larvae have left the leaves to pupate in the soil, the injuries caused by them on the leaves develop into spots and subsequently into shot holes. The anthracnose fungus may frequently be isolated from such injuries.

The petiole, when affected, turn grey or black and the leaves droop down, become dry and ultimately fall off, bearing the black scar on the twig (Sattar and Mallik, 1939). The disease produces elongated black necrotic areas on the twigs. The tip of very young branches are attacked first and the twigs go on drying from the tip downwards. Under humid conditions, all the branches as well as the main stem of young plants may dry out completely. Young thin branches are also similarly affected on older plants, but the big branches are not attacked (Sattar and Mallik, 1939). When the graft union is affected, the plant usually dies. Gummosis is usually the after-effect of the disease (Bose *et al.,* 1973).

The attack is observed at two stages, at the time of flowering causing blossom blight and at fruit ripening causing typical anthracnose.

The blossom blight and the accompanying peduncle blight are by far the most destructive phases of this disease as these prevent fruit setting and reduce production. The earliest recognizable symptoms of the disease are the production of blackish brown specks on the peduncle and flowers. Small black spots appear in the open flower panicle, which gradually enlarge and often coalesce to cause death of flowers either directly or indirectly by drying up of flower stalks. The loss may be small but under favourable conditions, the whole flower stalk may become blackened, blighted and set no fruit. The infected flowers fall off, leaving the more persistent spikes on

the peduncles. The severity of the disease may vary depending on the prevailing weather conditions (Prakash and Raoof, 1985a).

The fruit is attacked during any stage of development. Young fruits, a week or two old, often become severely infected and fall in large numbers. On older fruits, black spots are produced which not only make them unsightly but spoil their keeping quality. Initially the spots are usually round but often become so numerous that they run together and form large irregular blotches or even cover the entire fruit. The spots frequently coalesce over large areas, have large, deep cracks, penetrate deeply into the fruit causing extensive rotting. Under moist conditions, the blackened areas become covered with minute pinkish pustules or reproductive bodies and produce large number of minute spores, each of which is capable of causing fresh infection (Prakash and Srivastava, 1987). On the ripening fruit, the disease occurs as sunken, blackish brown blotches upon which salmon buff masses of spores develop. In storage, it causes rotting of the fruit.

2.8.2.2 *Latent infection* : The latent infection is carried from the field. Healthy fruits develop infection after coming in contact with diseased ones (Sohi *et al.,* 1973). The latent infection of the fungus does not begin to spread until it reaches eating maturity so the loss of fruit intended for local consumption is not very serious unless infection is severe. The fruit which ripens late in July-August, when the rains have commenced, may also be seriously damaged (Prakash and Srivastava, 1987).

2.8.2.3 *Causal organism* : The disease is caused by *Colletotrichum gloeosporioides* which has been considered a conidial stage of *Glomerella cingulata.*

The twig blight phase of disease reported by Hayes (1953) from UP is caused by *Gloeosporium mangiferae* P. Henn but it is identical to *C. gloeosporioides* (Sattar and Mallik, 1939). Kelkar and Rao (1962) reported six new species of *Colletotrichum* which include *C. mangiferae.*

2.8.2.4 *Morphology* : The morphology of the fungus was described by Sattar and Mallik (1939) and Bose *et al.* (1973). The acervuli of the fungus *(Colletotrichum gloeosporioides)* develop profusely on diseased parts of the plant. They are irregular and appear as brown to black dots. On the leaves, these occur on both the surfaces. Setae are common on twigs but not on fruits. The acervuli when mature exude pink masses of conidia under moist conditions (Sattar and Mallik, 1939). The acervuli are sub-epidermal but later the epidermis ruptures and exposes them. The acervuli are reported to measure 115-467 × 15-22 μm (Bose *et al.,* 1973) and 80-250 μm (Sattar and Mallik, 1939). The marginal setae are rare. When present they are dark fuliginous, cylindrical, continuous (rarely one septate), 40-90 × 4-6 μm (mean 60 × 5 μm). The conidia are borne on distinct, well developed hyaline conidiophores. The conidia are straight, cylindrical or oval, 8-20 × 5-7 μm hyaline usually with two, rarely one, oil drops (Sattar and Mallik, 1939). The size of conidia varies from 11-16 × 4-6 μm (Bose *et al.,* 1973).

2.8.2.5 *Physiology of the causal organism* : The fungus produces good aerial mycelium in Richard's and Brown's agar, and profusely sporulates on oat meal agar alongwith abundant development of acervuli in rings and few setae. Optimum pH was between 5.8 and 6.5. The growth was recorded to be optimum at 25°C and ceased beyond 35°C. The fungus grows on starch and peptone. Glutamic acid and alanine supported best growth and sporulation. The fungus was found to be autotrophic for vitamin synthesis. It does not grow below 95 per cent RH (Anon., 1970). Ghose *et al.* (1965) inoculated the ripe Dashehari fruits with induced oligosaccharide which reached maximum concentration 4-6 days after inoculation. Singh and Prasad (1967) studied the nutritional aspects of *C. gloeosporioides* var. *alatae,* especially the response to trace elements and vitamins, on growth and sporulation. The role of auxin-phenol complex in shot hole syndrome has been studied by Vidhyasekaran and Durairaj (1973).

2.8.2.6 *Disease cycle :* Under field conditions, the disease can be found on the fallen leaves and the blighted peduncles frequently remain *in situ* for many weeks. These produce spores under favourable moisture conditions and serve as foci of infection for the succeeding bloom. Even after they have fallen to the ground, they may continue to be a source of infection for few weeks. Under tropical conditioins, fresh supplies of spores are continuously produced throughout the year. Sattar and Mallik (1939) studied the viability of the fungus in detached diseased twigs and leaves. The fungus could be isolated from the diseased twigs even after two year's exposure to open weather conditions. Seventy per cent of the spores of the fungus borne in acervuli on the twigs were viable. In the diseased leaves, the fungus remained viable for 14 months. They successfully reproduced the disease symptoms by inoculating the twigs and leaves on young mango plants. The viability of the fungus in diseased twigs persisted on the tree. The acervuli were abundant on the marked dead twigs and 80 per cent of the spores in them were viable. Fresh acervuli go on appearing on dead twigs persisting on the tree. These caused typical anthracnose disease when inoculated on young plants. Thus the disease perenates in the detached diseased twigs and leaves which remain lying on the surface of soil and in the diseased twigs which may remain attached to the trees. The optimum temperature for infection was found to be 25°C.

There is no evidence to show that the fungus perpetuates through ascospores. The perithecial stage of the fungus is not very common. However, if this stage does occur, then there is no doubt that this may be the major means of survival in decaying leaves/twigs/debris, and for sudden and rapid development of new races of the fungus (Prakash and Srivastava, 1987).

2.8.2.7 *Control* : Control measures for anthracnose are generally taken along with those recommended for fruit anthracnose. In order to avoid fresh infection, the sources of infection should be eliminated. Diseased twigs, leaves and fruits which fall on the ground in the orchard should be collected and all infected twigs from the tree should be pruned away and such refuse should be burnt because the fungus has a long saprophytic survival ability on dead twigs. Plant vigour plays an important role in

keeping them free from twig infection. Therefore, proper irrigation and fertilization are essential to maintain the tree vigour. Plants may be sprayed regularly with suitable fungicides in places where the disease exists. Although total suppression of infection by these practices is not possible, the disease can be reduced considerably. This will also minimise the incidence of latent infection on fruits (Prakash and Srivastava, 1987).

Sattar and Mallik (1939) recommended spraying of young plants in the nursery with Bordeaux mixture (3:3:50) during February, April and September. This treatment could also control the disease on grown up trees (Cheema *et al.*, 1954; Mallik and Hasan, 1959; Sattar, 1946; Singh and Singh, 1955). Sprays of Zineb or Bordeaux mixture (4:4:50), twice at flowering and then at 14 days interval until harvest (Tandon and Singh, 1968) and of Benlate (0.2%) and Dithane Z-78 (0.2%) were extremely toxic to the fungus in culture (Bose *et al.*, 1973). Micop, Fycol, Blitox, Dithane or Blizene were found effective (Lingaraj, 1969). Gadre (1979, 1982) achieved good control by spraying fungicides like Bordeaux mixture, Captan, Blitox and Difolatan. Spraying of copper oxychloride+ Zineb after completion of heavy showers followed by Wettable sulphur (0.2 per cent) before flowering, carbendazim (0.3 per cent) at pea stage and Zineb (0.2 per cent) before maturation of stone (Jadeja and Vaishnav, 1984) and Bavistin (0.1%) at 15 days interval proved effective (Prakash and Misra, 1988).

Control of anthracnose through the development of resistant varieties is being investigated in many countries. Available germplasm need thorough screening against anthracnose for selecting suitable resistant clones for cultivation and breeding programme.

2.8.3 *Mango Malformation*

Mango malformation, also known as bunchy top, is a very serious threat to the mango industry, particularly in northern India. The etiology of the disease still remains obscure and diverse claims have been made about its causes, e.g., physiological, viral, fungal, acarological and nutritional.

2.8.3.1 *Symptomatology* : Three distinct types of symptoms are bunchy top of seedlings (BT), malformation vegetative (MV) and floral malformation (MF). Intermediate stages of these types have also been observed (Singh and Chakravarti, 1935). These symptoms can be grouped into two broad categories, vegetative or floral (Varma, 1983).

Bunchy top phase of the disease is reported by Nirvan (1953). The disease appears on young plants in the nursery beds when they are 4-5 months old. The characteristic symptom of the disease is the formation of a bunch of thickened small shootlets bearing small rudimentary leaves or occasionally several bunches arising from a leaf axil at the top or lower down the main shoot. These shootlets are much thicker than main axis from which they arise. The disease symptoms are shown by

the swelling of several buds in the axil of a leaf or the production of several small shoots at the apical end. The shoot remains short and stunted and growth of the plant is stopped giving an appearance of bunchy top.

Vegetative malformation (MV) induces in young seedlings excessive vegetative branches of limited growth and swollen and very short internodes forming bunches of various sizes often at the top of the seedlings (Varma, 1983) giving a bunchy top appearance. Such formations are also frequently found on seedling trees but rarely on grafted trees. The axillary buds of dwarf and even normal looking branches are usually enlarged indicating disturbance in apical dominance. Bunchy top and malformation are considered to be the expression of the same disease as they show similar symptoms (Tripathi, 1954).

Malformation of inflorescence (MF) shows variations in malformed panicles (Singh *et al.,* 1961). In the early stages of panicle formation, no differentiation between healthy and diseased panicles can be made. The abnormal inflorescences may persist long after the normal one has fallen off and may finally become vegetative. The malformed heads dry up in black masses and persist on the tree for a long time even until the next flowering season (Mallik, 1959 b). The most characteristic symptom of MF is a reduction in the length of the primary axis and the secondary branches of the panicle which make the flowers to appear in clusters. Frequently, the flower buds are transformed into vegetative buds and large number of small leaves and stems, which are characterised by appreciably reduced internodes and are compacted together giving a witches' broom appearance. In other cases, the flower buds seldom open and remain dull green. In still other cases, the main axis is shortened but the flowers are transformed into small leaves. Branches with the diseased inflorescence can produce both malformed as well as healthy panicles in the following bearing season. The affected inflorescences are of three types, heavy, medium and light (Varma *et al.,* 1969a,b). The heavy panicles are very compact due to extreme crowding of flower and unlike normal panicles, keep growing to form large hanging masses of flowers. The medium panicles are slightly less compact, their growth is not continuous but may start after sometime, and like the heavy panicles, persist on plants longer than the normal panicles (Singh and Chakravarti, 1935). The light panicles are difficult to distinguish from the normal ones as these are only slightly compact and do not persist on plants. Due to larger bracts, malformed panicles give leafy appearance unlike in normal panicles which is not due to phyllody. Variation in malformed panicles have been illustrated by Singh *et al.* (1961). Sharma (1953) reported the compact and the spreading types of malformation. There is a change in the sex ratio owing to shift from hermaphroditic to staminate flowers (Khan, 1943; Khan and Khan, 1960).

Malformed panicles normally do not bear as the fruits set in the season do not grow more than pea size. These have been found to yield normal fruit in off season (Mallik, 1963; Jawanda, 1963). Bombay Green also retains fruits even in normal season (Majumdar and Sinha, 1972). Mallik (1963) could not get success in hand pollination of hermaphrodite flowers in malformed panicles with normal pollen but

Varma *et al.* (1974a,b) succeeded when they used normal pollen of Totapari on hermaphrodite flowers of malformed panicles of Dashehari. Infection appears to be localised. However, possibility of systemic infection in branches cannot be ruled out. All the branches produced on a malformed branch may not bear malformed panicles and even in a malformed panicle some secondary rachis may be normal (Mallik, 1963). Conversely, on a healthy panicle, some parts may be malformed and such expressions are not very common (Varma, 1983).

2.8.3.2 *Causal fungi* : Summanwar *et al.* (1966) reported the fungus *Fusarium moniliformae* Sheld, associated with mango malformation (MF and MV) and proved its pathogenicity. Summanwar and Raychaudhuri (1968) investigated that mite carried the fungus on the bodies and irritation caused by the mites provided pathway to the fungus. The fungus *F. moniliforme,* has been consistently isolated from various parts of malformed plants. Isolations were made from malformed panicles of 392 isolates from 130 trees of which 336 were *F. moniliforme* and 36 *Cylindrocarpon mangiferum* (Chowdhary and Varma, 1986; Prasad *et al.,* 1972; Summanwar *et al.,* 1966; Varma *et al.,* 1969 a, b, 1971, 1972 a, b; Chadha *et al.,* 1979b).

The fungus was further identified as *F. moniliforme* var. *subglutinans* (Varma *et al.,* 1974 b; Chadha *et al.,* 1979 b). Aerial mycelium appearing powdery due to microconidia 0-1 septate, oval to fusiform, produced from polyphialides, macroconidia lacking or rarely produced 1-2 septate, falcate, no chlamydospores, pigmentation typical violet. The fungus does not have any special nutritional requirement (Chattopadhyay and Nandi, 1981; Mitra and Lele, 1981). Varma *et al.* (1971) reported that the growth of the fungus is inhibited during summer months even at room temperature. Vegetative malformation (Prasad *et al.,* 1972; Summanwar *et al.,* 1966; Varma *et al.,* 1969 a,b; Chadha *et al.,* 1979b) and floral malformation (Varma *et al.,* 1974 a, b) can be initiated in the healthy test plant by artificial inoculation of aerial branches as it is mostly intercellular and occasionally forms intracellular agglomerates in the cortex and phloem regions and the fungus formed globose bodies similar to chlamydospores, particularly in the cortex when inoculated with spore suspension (Varma *et al.* , 1972, 1974b). Cross inoculation studies with strain of *F. moniliforme* further confirmed the host specificity and the mango strains, only caused typical disease symptoms and infection (Varma *et al.,* 1974 a,b; Chadha *et al.,* 1979b). A definite evidence revealed that the disease is caused by *F. moniliforme* var. *subglutinans* (Ghosal *et al.,* 1977 a,b). Fusarial pathogens in contact with the host species produce the common toxic compounds, namely, fusaric acid, lycomarasmin and 12-13 epoxytrichothecences.

F. oxysporum was shown to be involved in causing malformation of mango and the fungus was isolated from all parts of plants. Typical BT symptoms could be produced in the seedling by inoculating the fungus through soil. The fungus was systemically present in parenchymatous cells of the pith region of malformed tissues (Bhatnagar and Beniwal, 1977). It is possible that the disease is caused by more than one species of *Fusarium.*

Fungal etiology and behaviour of mango malformation was studied by Andotra *et al.* (1984). A. *Fusarium* sp. was isolated from infected tissue. Inoculation of mango seedlings showed that the disease is neither systemic nor completely localized but behaves erratically. Internal spread is always acropetal and is supposed to be facilitated through active cell division of terminal growth under environmental conditions favourable to both host and pathogen.

The mango twigs infected with *F. moniliforme* var. *subglutinans* contained less mangiferin than those of healthy plants. In both cases, mangiferin concentration was high during cooler months and low during hot months (Chakrabarti and Ghosal, 1985).

2.8.3.3 *Disease spread* : The annual recurrence of malformation in new seedlings has been observed. Increase in infection has been reported by Nirvan (1953), Singh *et al.* (1961) and Mallik (1963). Although fungus *F. moniliforme* does not sporulate *in situ* but it does so on drying malformed panicles (Varma *et al.,* 1974a). Rotary traps for six months in the places of high incidence could not yield the spores of *Fusarium* (Varma *et al.,* 1971) which indicated the role of some other agency in transmission of the disease. Puttarudriah and Channabasavanna (1961), Singh *et al.* (1961) and Nariani and Seth (1962) successfully reproduced the disease by transferring the mites which were later reported to carry the fungus *F. moniliforme* (Summanwar and Raychaudhari, 1968). Varma *et al.* (1971) explained the possibility of mites being the carrier of malformation. The feeding behaviour of the mites also supported this view (Varma *et al.,* 1983). The small percentage of mites carrying the fungus and their presence in southern and eastern parts of India (Varma *et al.,* 1971), where the disease is sporadic in nature, indicated possibility of involvement of some additional factors in the movement of the disease (Varma *et al.,* 1974a,b). The propagation and distribution of diseased plant material may cause wide and erratic distribution of the disease (Varma *et al.,* 1971).

The severity of the disease varies considerably from year to year. A tree once affected cannot escape the disease in subsequent years (Mallik, 1963). Majumder and Sinha (1972) and Varma *et al.* (1969a) observed 60 per cent diseased panicles in cultivar Neelum during February-March flowering, whereas the same plant had only 4.5 per cent malformation during off season flowering in June when the average minimum and maximum temperatures were higher. Fluctuation in the incidence of malformation in varieties Neelum, Alphonso and Seedling trees were investigated by Jagirdar and Shaik (1968). The disease is serious in the north-west region where temperature is between 10 and 15°C during December-January (winter) before flowering. The disease is mild in the areas where temperature is between 15 and 20°C, sporadic between 20 and 25°C and nil beyond 25°C (Puttarudriah and Channabasavanna,1961). Singh *et al.* (1961) and Chadha *et al.* (1979a) reported that the occurrence of malformation differed according to the age of the plants. They observed more disease in young than old plants. About 91 per cent incidence in 4-8 years old plants and

9.6 per cent in older plants was reported (Singh *et al.,* 1961). Age of flowering shoot influenced the incidence of MF (Varma, 1983).

2.8.3.4 *Control* : Some recovery in plants treated with Phorate and Captan after pruning has been reported by Bindra and Bakhetia (1971). Varma *et al.* (1971) reported that Benlate and aphidan inactivated the fungus *in vivo* when cut malformed branches were partially dipped in solutions. These were also effective when sprayed on malformed plants. However, none of the treatments including pruning male panicle and deblossoming with or without fungicidal and acaricidal applications was successful in reducing floral malformation in Dashehari cultivar (Chib *et al.,* 1986).

Varma *et al.* (1971) realised the need of systemic fungicides as the causative fungus was located in the cortex-phloem portion. Spraying of Benlate alongwith thorough pruning could reduce the disease from 69 per cent to less than 1 per cent. Using disease-free plant material (Varma *et al.,* 1971) and prophylactic spray with fungicides (Varma *et al.,* 1971; Chattopadhayay and Nandi, 1977) were considered effective to keep the plants healthy and check further spread of malformation.

Sharma and Tewari (1975) found that Bavistin did not appear to work systemically against mango malformations. However, spraying this fungicide could reduce the disease. Varma *et al.* (1971) screened 34 fungicides, 17 insecticides and 4 growth regulators for their fungicidal and fungistatic action, and found that Benlate, Brestan, Busan, Captan, Dithane M-45, Banogan and Thiram were the most effective. Among the insecticides, aphidan and Phosphamidon were fungicidal at 1000 ppm but not at lower concentrations. Other insecticides and growth hormones were only fungistatic. Benlate and aphidan could inactivate the fungus *in vivo.* Dwarf shoots developed into normal vegetative shoots when these chemicals were sprayed on diseased plants and fungus could not be isolated from fresh growth, whereas the fungus was present in the dwarf shoot parts, indicating their concentration in sprayed shoot to be below fungicidal level.

Foliar spray of 200 ppm Bavistin (carbendazim) gave maximum disease reduction (95%) in Dashehari and 91.3 per cent in S B Chausa. Potassium metabisulphite gave maximum disease control in S B Chausa (92.5%) and NAA in Dashehari (94%) (Mehta *et al.,* 1986; Siddiqui *et al.,* 1987).

Thus, although definite control schedule for the control of mango malformation can not be advocated, the following measures may reduce its incidence :

i) Scion sticks from infected trees should not be used and indexing of healthy mango trees may be done to serve as material for propagation.

ii) As soon as the disease appears, the affected terminals alongwith the basal, apparently healthy 15-20 cm portion should be removed and burnt.

iii) In areas having more than 25 per cent affected plants, deblossoming at bud burst stage should be done to delay the flowering (Varma, 1983).

iv) In areas having 5-25 per cent affected plants, the branches should be pruned thoroughly and when flowers emrege, malformed panicles should be removed to prevent further drain of the nutrients (Varma, 1983).

v) In areas having 5-10 per cent affected plants, all the diseased plants should be destroyed or at least the infected branches should be removed (Varma, 1983).

vi) Healthy orchards located in disease prone pockets should be sprayed as a prophylactic measure to avoid recurrence of the disease.

2.8.4 *Die-back*

Die-back, death of plant from top downwards, is prevalent in mango growing States of the country. It was described by Das Gupta and Zachariah (1939, 1945) from UP. Edward (1954) isolated the fungus from dead roots of mango seedlings from Allahabad, Verma and Singh (1970) from Jaipur, Rath and Mohanan (1977) and Rath *et al.* (1979) from Orissa. Since 1975, the disease has become severe in western UP and created an alarming situation in Amroha (Prakash and Singh, 1976a; Prakash and Srivastava, 1987). In Moradabad, 30-40 per cent roadside and other plantations were affected. Besides, UP and Rajasthan, the disease is also prevalent in Orissa, Maharashtra, Bihar, West Bengal, Haryana and Tamil Nadu.

2.8.4.1 *Symptomatology* : The effect of disease on the general appearance of tree is noticeable any time in the year but is conspicuous after monsoon during October and November (Prakash and Raoof, 1985). The disease is characterised by dying back of twigs from top downwards, particularly in the older trees, followed by complete defoliation giving an appearance of scorching by fire (Fig. 5). Discolouration and darkening of bark at a certain distance from the tip is the external evidence of disease. Such dark patches are generally seen on young green twigs and are hardly distinguishable in older branches. The bark is discoloured at several places. When the dark lesions increase in size, dying of young twigs begin at the base affecting the leaf midribs extending outwards along the veins. The upper leaves lose their healthy green colour and gradually turn brown accompanied by upward rolling of leaf margin. In advanced stage, such leaves shrivel, fall off in a month or more, leaving the shrivelled twigs altogether bare. Internal browning in the wood tissue is observed on slitting along the long axis. Cracks appear on branches which exude gum before they die. When the graft union of young plant is affected, it usually dies. It has been found that infection occurs at node at variable distance below the growing point and part of the twig above and below this point dies.

2.8.4.2 *Causal organism* : The cause of the disease is found to be *Botryodiplodia theobromae* Pat. but perfect stage of the fungus is not reported (Prakash and Srivastava, 1987).

2.8.4.3 *Epidemiology* : High summer temperatures predispose the mango plants to the attack of the disease through reducing the vitality of the plant (Dasgupta and

Zachariah, 1945). Relative humidity of about 80 per cent, maximum and minimum temperature of 31.5°C and 25.9°C, respectively, and rains favour disease development. The growth of germ tube of single celled spores was best at 30°C. On exposure for 10 minutes, loose spores lost their viability at 54°C (Verma and Singh, 1970).

The organism is a wound parasite and is capable of causing great damage when mango grafts are kept in a humid propagation shed.

Bhatnagar and Singh (1979) found that 10 ppm 2,4-D resulted in maximum growth of the fungus. According to Srivastava (1969), sugar contents decreased greatly while many amino acids decreased, some occasionally increased due to the proteolysis of host proteins. Srivastava and Tandon (1970) reported that the mango isolate grew better on a mixture of amino acids.

2.8.4.4 *Histopathology* : According to Das Gupta and Zachariah (1945), the infected twig shows an internal discolouration which, at an earlier stage, extended about an inch on either side towards the tip and the base of the twig beyond the external darkened bark. The diseased twigs, after slant cutting along the long axis through the infected region, revealed a brown discolouration of cambium and phloem. The internal discolouration is diffused and uniform and appears as a dark streak between the stele and the cortex. At very early stage, the twig appeared healthy about four inches below the growing point except for a short discoloured area on the stem showing shrivelled epidermal and sub-epidermal cells. The internal discolouration was manifested by browning of certain regions of the cambium and phloem, where some of the cells were found to be plugged with a yellow gum-like substance. A few hyphae were seen in the xylem vessels. At advanced stage, the cells of different tissues of stem were badly shrivelled. The xylem vessels were plugged with fungal mycelium. The stele and the other layers got separated from each other along the discoloured band at the cambial region where the cells had disintegrated. Numerous hyphae were found in this region. A few hyphae were also found in the cells of the cortex. The mycelium was found not only in the bundles of the stem, but also in the petioles and midribs of leaves of the infected twigs.

2.8.4.5 *Varietal susceptibility* : Variety Mohan Bhog appeared to be susceptible while Siroli was resistant (Verma and Singh, 1973). In Saharanpur (UP), the incidence was noticeable on Bombay Green (88.8%), Dashehari (70%), Sadabahar (63.6%) and Sadaphal (63.2%). At Pinjore, the disease incidence varied between 23 and 60 per cent. At Durgapura (Jaipur), maximum incidence was recorded in seedling trees (96.5%), Langra (95.7%), Siroli (85.7%) , Chausa (74%), Dashehari (40.6%) and Fajli (55.5%) varieties (Prakash and Srivastava, 1987).

2.8.4.6 *Control* : Preventive measures, such as selection of scion from healthy trees, sterilization of the budding knife, keeping the grafted tree in a relatively dry environment and gradual exposure to full sunlight are effective.

Captan has been found effective *in vitro* against the fungus (Srivastava and Tandon, 1971). Pruning of the diseased twigs 7-8 cm below the infection site followed by spray of Bordeaux mixture (5:5:50) or copper oxychloride (0.3%) were effective in controlling the disease (Prakash and Raoof, 1985, 1989).

2.8.5 *Sooty Mould*

Sooty mould or sooty blotch is very common wherever honey dew or sugary substance secreting insects, such as mango hopper, scales, coccids and mealy bugs are found (Hansford and Thirumalachar, 1948; Vaheeduddin, 1953; Kulkarni and Kulkarni,1978; Peethambharan and Aravindakshan, 1975; Singh and Singh, 1972; Prakash and Srivastava, 1987).

The disease appeared in epidemic form in Bulandshahar district of UP during August 1985 and in certain areas, even thick branches died (Prakash and Srivastava, 1987).

2.8.5.1 *Symptomatology* : The disease is characterized by the presence of a black velvety thin membranous covering on the leaf lamina. The entire lamina is covered or it may be only as flakes on the leaf. In severe cases, the tree completely turns black with mould on entire surface of twigs and leaves. The affected leaves curl and shrivel under dry conditions. The fungus multiplies on the 'honey dew' secreted by the insects and spreads on the plant surface making it black and ugly owing to the masses of black spores on the leaf surface. The severity of incidence is dependent upon the sugary secretion by the insects. During flowering time, its attack results in reduced fruit set and sometimes causes fruit fall. It is also noticed on fruits of late mango varieties (Prakash, 1988).

2.8.5.2 *Causal organism* : Sooty mould is caused by the fungi, *Meliola mangiferae* Earle (Butler and Bisby, 1931; Uppal *et al.,* 1935; Hansfored and Thirumalachar, 1948); *Capnodium mangiferae* Cke. & Brown (Vaheeduddin, 1953); *Capnodium ramosum* Cke. (Butler and Bisby, 1931; Uppal *et al.,* 1935) and *Tricospermum acerinum* (Syd.) Speg (Das and Mohanty, 1972). In UP, the disease is caused by *Microxyphium columnatum, Leptoxyphium fumago* and *Triopospermum myrti* (Prakash, 1988). The fungus is non-pathogenic because its mycelium does not enter the host tissue to absorb nutrients but draws from the sweet 'honey dew'. Thus, the damage by the fungus is not direct as it thrives saprophytically and interferes with the normal functioning of plant by cutting off the effective photosynthesising leaf area.

2.8.5.3 *Predisposing factors* : Disease is severe in old and dense orchards where light intensity is low. Trees exposed to eastern side (sunlight) had less incidence while the trees in centre of the orchard had 95 per cent incidence. High incidence of insects and the sugary substance secreted by them favour development of sooty mould. Continuous and heavy rainfall washes down this substance but high humidity proved congenial for growth of the fungus (Singh and Singh, 1972).

2.8.5.4 *Physiological effects* : Kulkarni and Kulkarni (1978) found that there was an increase in the amount of iron and potassium, and decrease in sodium, manganese and calcium in infected leaves by *Capnodium mangiferae* mould as compared with those of healthy leaves. Singh and Singh (1972) reported that the infection of fruits reduced the total soluble solids content and induced early deterioration and rotting.

2.8.5.5 *Varietal susceptibility* : Screening of 29 cultivars showed that Alphonso was resistant (Peethambharan and Aravindakshan, 1975).

2.8.5.6 *Control* : Since the mould dies out for want of a suitable growth medium, if honey dew secreting insects are killed by suitable insecticides, covering both the surfaces of leaves, checks fungal growth. Singh and Singh (1972) reported that spraying of Elosal (900 g/450 litres) at 10-15 days interval proved quite effective. Spraying of Wettasul + Metacid + gum acacia (0.2 + 0.1 + 3.0%) at 15 days interval could control sooty mould (Prakash, 1988). Spraying insecticides and then removing the sooty mould growth in dry flakes by subsequent application of soluble starch was very effective method of control.

The incidence on fruits can be controlled by dipping them for two minutes in a bleaching solution (1/4 lb each of chloride of lime and boric acid/gallon of water) and subsequently washing them.

2.8.6 *Black Banded*

The occurrence of the disease on mango was recorded at Pune (Saccardo, 1906) and then in Goa, West Bengal, Karnataka, Maharashtra, Tamil Nadu (Prakash and Srivastava, 1987), Andhra Pradesh (Subramanian, 1956; Reddy *et al.,* 1961) and Gujarat (Vala *et al.,* 1985). The disease does not occur in severe form.

2.8.6.1 *Symptomatology* : Black velvety fungal growth is noticed on the midribs and veins of leaves, and bark of twigs and branches of mango. The incidence is very low on main branches. It presents a characteristic and conspicuous black banded appearance. The mycelial growth and clusters of conidiophores present a velvety appearance during rainy season which drop off in summer months leaving light black bands on the affected portions. The fungus remains confined to the upper layer of the bark (Reddy *et al.,* 1961).

2.8.6.2 *Causal organism* : The disease is caused by *Rhinocladium corticolum* Massee. Subramanian (1956) considered this fungus as imperfect stage of *Pexiotrichum corticolum* (Massee) Subramanian.

2.8.6.3 *Control* : Application of Bordeaux paste or Bordeaux mixture may control the disease followed by gunny rubbing or *Ganji* treatment (Reddy *et al.,* 1961).

Neelum, Alampur Baneshan, Nawab Pasand, Kovaji Patel and Sambandham were highly susceptible. About 40 varieties were found free from the disease including *Mangifera odorata* (Reddy *et al.,* 1961).

2.8.7 *Phoma Blight*

The disease was reported to be widespread in Lucknow (Prakash and Singh, 1977). It is also prevalent in West Bengal, Bihar, Maharashtra, Karnataka, Madhya Pradesh, Rajasthan, Gujarat, Uttar Pradesh and Goa (Prakash and Srivastava, 1987).

2.8.7.1 *Symptomatology* : Symptoms of this disease are noticeable on old leaves only. Initially, the lesions are minute, irregular, yellow to light brown and scattered over the leaf lamina (Fig. 6). As the lesions enlarge, their colour changes from brown to cinnamon and these become irregular. Fully developed spots are characterised by dark margin and dull grey necrotic centres. When infection is severe, the spots coalesce to form 3.5-13 cm patches which result in withering and defoliation of infected twigs.

2.8.7.2 *Causal organism* : The disease is caused by fungus *Phoma glomerata* (Corda) Woll. and Hochapf. The morphology of the fungus has been described by Prakash and Singh (1977).

2.8.7.3 *Control* : Out of seven fungicides tried for two consecutive years, Benomyl (0.2%) followed by copper oxychloride (0.3%) were found effective (Prakash, 1978, 1979a).

During 1986, 110 cultivars were evaluated out of which 51 had low, 43 medium and 16 high incidence of phoma blight (Anon., 1986). During 1987, out of 279 cultivars, 30 had medium and 12 high incidence (Anon., 1987).

2.8.8 *Pink Disease*

The disease is widespread in tropical and subtropical regions of the country, and has been reported from south India by Subba Rao (1936) and Mc Dougall (1940). It is also known as thread blight, rubellosis and cobweb.

2.8.8.1 *Symptomatology* : The disease is noticed as a pinkish powdery coating on the twigs and branches. By then, the fungus would have invaded the bark to get established in the internal tissues and interfere with the transport of nutrients. The fungal growth often girdles the stem. Severely infected bark shreds and the wood gets exposed. Leaves turn yellow and dry, shoot and branches of the affected plants wilt and dry. Roots are not infected. The pink colour on the tissues is owing to the profuse conidial production by fungus. In advanced cases, the fungus may produce pustular or nectar stage. These pustules are orange red and arranged systematically in rows along the stem.

The disease is seen during or just after rainy season and persists from one season to another through dormant mycelium inside the bark. The cankerous tissues serve as a potential source of infection in wet season.

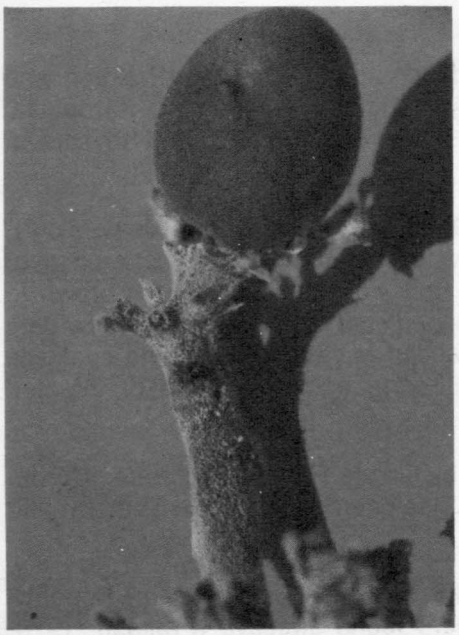

Fig. 3

Fig. 4

Fig. 3 : Powdery mildew of mango.

Fig. 4 : Anthracnose on mango fruits.

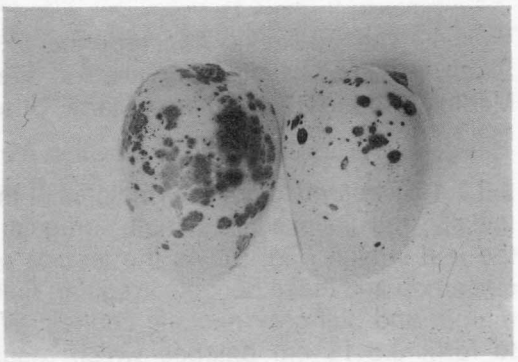

Fig. 5

Fig. 6

Fig. 5 : Die-back of mango.

Fig. 6 : Phoma blight.

2.8.8.2 *Causal organism* : The disease is caused by *Pellicularia salmonicolor* (Berk and Br.) Dastur (Syn. *Corticium salmonicolor* Berk and Br.). The new name of the fungus is *Botryobasidium salmonicolor* (Berk and Br.) Venkataraman.

2.8.8.3 *Control* : The disease can be kept under control by cutting and removing the affected branches and painting Bordeaux paste on cut ends. Disease can also be controlled by use of lime sulphur and oil based copper compounds.

2.8.9 *Grey Blight or Pestalotia Leaf Spot*

The disease is reported in India by Tandon *et al.* (1955), Sarkar (1960), Pandey and Mohammad (1974-75), Prakash and Srivastava (1987) and Vala *et al.* (1985).

2.8.9.1 *Symptomatology* : The disease is characterized by the presence of brown spots on the lamina, more during winter. They may develop at the margins or tips but, in some cases, are distributed irregularly on the entire leaf. Initially, the spots are brown and minute which gradually increase in size and become dark brown. Some of the spots often enlarge considerably to form large lesions, in which the central region turns greyish white or light olive grey encircled by a ring of tan colour. At this stage, black spots of acervuli may become visible to the naked eye in the central region, more on upper surface, and never extend beyond the midrib. If the infection starts from the tip, it advances regularly on either side of the midrib. Finally, after 3-4 months, the infected portions get detached from the leaf. In severe infection, defoliation follows. The fungus is capable of attacking healthy full grown mango leaves (Sarkar, 1960).

On mature green fruit, brown small spots appear which gradually increase in size, become dark brown with greyish white centre having numerous black dots of acervuli. The fruits drop off when the disease advances to the stalk end. Wounding results in quick attack by the fungus and in early spread of lesions. Moist atmosphere helps in increasing and hastening infection (Sarkar, 1960).

The infection does not kill the plant entirely but photosynthetic activity is undoubtedly reduced. It is a weak parasite capable of infecting injured tissues and healthy fruits in contact with the diseased ones (Tandon *et al.,* 1955). However, the pathogenicity of the *Pestalotia* has been established on leaves, stem and fruits of mango (Tandon *et al.,* 1955; Pandey and Mohammad, 1974-75).

2.8.9.2 *Causal organism* : The disease is caused by *Pestalotiopsis mangiferae* (P. Henn) Stey. *Pestalotiopsis* sp. and *P. versicolor* (Speg.) Stey (Prakash and Srivastava, 1987), *P. glandicola* (Cast). Stey. (Ullasa and Rawal, 1985), *Pestalotia* sp. (Jadeja & Vaishnav, 1984) and *P. funerae* var. *mangiferae* (Uppal *et al.,* 1935) have been reported from various parts of the country.

2.8.9.3 *Varietal resistance* : Of the varieties, Bombai, Himsagar and Langra, the former is the most susceptible, Langra is the least, while Himasagar is moderately susceptible (Sarkar, 1960). Pandey and Mohammad (1974-75) also reported that Bombai variety was the most susceptible whereas Chausa was resistant to brown spot.

2.8.9.4 *Epidemiology* : The fungus is capable of growing at temperatures between 10 and 35°C, and maximum growth occurs at 20-25°C. The temperature range for spore germination is from 10 to 34°C with an optimum of 30°C. Best mycelial growth with intensive sporulation takes place pH 5.5 to 6 and grows well on Potato

Dextrose Agar, Czapex Dox Agar, Richards Agar, Oat Meal Agar and Decoction Agar with abundant conidial growth in the first three media and moderate in the others (Sarkar, 1960). Growth studies have also been conducted in culture media containing various carbohydrates and nitrogen compounds (Sarkar, 1960; Pandey and Mohammad, 1974-75). The maximum growth and sporulation was observed on Host Extract and Richard's media, and optimum temperature for growth and sporulation was 30°C (Pandey and Mohammad, 1974-75).

2.8.9.5 *Control* : Dusting the leaves with zinc sulphate controlled the disease but it failed to control fruit rot (Tandon *et al.*, 1955). Jadeja and Vaishnav (1984) recommended wettable sulplhur (0.2%) + Zineb (0.2%) after heavy rains followed by wettable sulphur (0.2%) before flowering, Carbandezim (0.3%) at pea stage and Zineb (0.2%) before maturation of stone to reduce the incidence.

2.8.10 *Minor Diseases*

Disease	Causal organism	Symptoms	Control measures	Reference
Cercospora Leaf spot	*Stigmina (Cercospora mangiferae* (Koorders)Ellis, *C. mangiferae indicae* Munjal, Lal & Chona	Irregular or round lesions on both sides of the leaf blade, measuring 3.5-10 mm in diameter, light brown but later become brown to black, surrounded by yellow halo centre. Somewhat dull in colour, spot may coalesce to form longer irregular patches, soon dry and wither resulting in a scorched appearance of foliage	-	Munjal *et al.* (1961) Prakash and Srivastava (1987)
Alternaria spot	*Alternaria tenuissima* (Fr.)	Disease is noti-ced on leaf, twig	Spray of Merculin	Mukherji and Bhattacharya

(Contd.)

(Contd.)

Disease	Causal organism	Symptoms	Control measures	Reference
	Willtsh and *A. alternata* (Fr.) Keissler)	and fruits, spots first appear as small brownish circular and black patches on twigs, initially apparent at lower surface, shows light brown discolouration. Spots gradually enlarge and form irregular black patches. The tender leaves are more susceptible than mature ones. On fruit, spots gradually enlarge and become water soaked and the fruit rots	followed by mercurised copper oxychloride, Kirti Copper	(1965), Yadav and Udai Narain (1970), Singh and Tandon (1967), Prakash and Raof (1985b)
Sclerotium rot	*Sclerotium rolfsii* Sacc. *S. delphinii* Welch.	Presence of mycelial weft on the base of stem at ground level in patches, beneath the growth a dark brown spot develops, gradually encircles the base of stem. At this stage, the succulent top droops, bends and tissues	Sanitation, crop rotation cultural practices are recommended besides chemical control. Two minutes dip of stones in agallol/ brassicol/ Captan/Thiram	Baruah and Baruah (1952) Prakash and Singh (1976) Gadre (1979a)

(Contd.)

(Contd.)

Disease	Causal organism	Symptoms	Control measures	Reference
		lose turgidity, seedling dies within a week, sclerotial bodies are seen around the stem and root zone portions. Such roots are soft and black and the sclerotia of the fungus adhere to the cotyledons	and subse-quent soil drenching at 10-15 days interval	
Root rot and damping-off	*Rhizoctonia solani* Kuhn.	Disease is characterised by dropping of leaves after emergence of plants from soil, until the stem has hardened sufficiently to resist invasion during prolonged rainy weather, infection generally occurs at or below the ground level with circular to irregular water-soaked patches. On account of rotting, the	Two soil treatment, with Ceresan is recom-mended, first ten days before sowing and second two days after turning over the soil up to a depth of 20 cm. Spraying of Bordeaux mixture (1.5%) at weekly interval is recom-mended	Prakash and Singh (1980), Gupta and Srivastava (1975)

(Contd.)

(Contd.)

Disease	Causal organism	Symptoms	Control measures	Reference
		tissues become soft, dark brown or black and entire seedling collapses and dies		
Twig blight	*Phoma* sp.	Produce water-soaked areas on twigs during rainy season, which rapidly enlarge, invariably there is a rapid upward extension of invaded region, but the lateral spread is quite limited and does not girdle the twig. The affected bark turns dark brown and the shoot dries		Kantikar and Uppal (1939)
Leaf spot	*Phoma sorghina* (Sacc.) Baerema. Doren & Vankest	Irregular, oval to roughly circular water-soaked spots on young leaves measuring pin-head to 2.5 mm in size, brown in colour, later differentiated into brown		Prakash and Singh (1976a) Prakash and Raoof (1985b)

(Contd.)

(Contd.)

Disease	Causal organism	Symptoms	Control measures	Reference
		margin with straw colour and yellow halo. Such leaves become brown and dry. Symptoms produced by this fungus are somewhat similar to early stages of anthracnose		
Leaf blight	*Macrophomina mangiferae* Hingorani & Sharma	The disease is characterised by presence of yellowish pin-head spots on leaves, twigs and rarely on stems. Soon after, spots enlarge which first become light brown to dark brown and then broad dark purplish and are slightly raised. Later, tissues become ash coloured due to appearance of pycnidia on the surface of lamina. Spots are initially round, but later become oval or	Since the pathogen survives in the leaves, sanitation by way of removal and destruction of infected parts is essential to reduce the inoculum load. Burgundy mixture, Perenox, lime-sulphur and Dithane can be tried under field conditions	Hingorani *et al.* (1960), Prakash and Srivastava (1987)

(Contd.)

(Contd.)

Disease	Causal organism	Symptoms	Control measures	Reference
		irregular depending upon the environmental conditions. The bark of the infected stem/twig later turns grey, girdle the tissues at the point of infection		

2.9 Pomegranate

2.9.1 *Colletotrichum Leaf Spot*

Leaf spot caused by *Collectotrichum gloeosporioides* is reported from UP by Chandra and Tandon (1965). Minute, dull voilet black or black spots appear on leaf which are surrounded by yellow region. Later, the spots coalesce and become bigger causing defoliation. Tillex proved effective in controlling leaf spot.

2.9.2 *Fusarium Leaf Spot*

During July 1977, a new leaf spot of pomegranate caused by *Fusarium fusarioides* (Frag. and Cif.) Booth. was recorded from orchard of Sangli district of Maharashtra (Sherkar and Utikar, 1982a). The disease was characterized by appearance of minute specks towards the leaf margin which were brownish and circular to irregular in outline. With the advancement of the disease, the spots coalesce to form big dark brown necrotic blotch. The fungus *F. fusarioides* made good growth in Richard's agar and PDA. Minimum, optimum and maximum temperature requirements for the fungus are 5°, 27-30° and 45°C. In bioassay, Difolatan and Miltox were found effective in inhibiting the growth of fungus.

2.9.3 *Phomopsis Leaf Spot*

Shreemali (1972) recorded *Phomopsis aucubicola* from Choupasani at Jodhpur, Rajasthan during October 1967. The lesions were mostly marginal or sometimes scattered having buff brown colour. Black pycnidia were unsparingly present on the upper surface of the leaf.

Hyphae are colourless, poorly branched, closely septate, 1.4-3.8 µm wide, pycnidia densely gregarious, black, immersed than erumpent, globose to sub-globose,

201.6-489 μ (av. 326.8 μ) in diameter, dark brown to black, ostiolate, conidiophores simple, short, unbranched, hyaline 4.6 × 8.2 μ long, alpha spores hyaline, ovoid to oblong or rarely fusoid to subfusoid, often biguttulate, 7.4-11.8×2.4-3.1 μ (av. 9.8 × 2.9 μ), beta spores filiform, curved or unicinate, 19.8-34.4 × 0.54-1.4 μm (av. 27.4 × 0.98 μm).

Verma *et al.* (1982) screened 16 varieties infected by *Phomopsis aucubicola.* Variety Jeolicola Local was the least susceptible to the fruit rot, whereas Country Large Red and Muscat White were most susceptible.

2.9.4 *Fruit Spot*

During July 1977, a new fruit spot caused by *Beltaraniella humicola* was recorded by Sherkar and Utikar (1982b). The disease is characterized by appearance of black circular spots which gradually enlarge and coalesce to form big dark black spots leading to necrosis. The margin of spots vary from reddish to brown in colour. Infection is restricted to the rind of the fruit and, to some extent, to underside of the pulp.

Among the carbon and nitrogen source, glucose and ammonium tartrate supported good growth of *B. humicola. In vitro* studies showed inhibiting effect of Miltox, Benlate and copper oxinate (Sherkar and Utikar, 1982b).

2.9.5 *Drechslera Fruit Spot*

Fruit spot caused by *Drechslera rostrata* on pomegranate was first reported by Utikar *et al.* (1976), being first observed in 1972 on Muscat variety. Fruits at all stages are attacked. The disease is characterized by the appearance of numerous, small black spots scattered all over the fruit. The margin of the spots varies from dark green to orange in colour. In advanced stages, few spots gradually enlarge and coalesce to form big dark spots of various sizes. Mild infection is confined to rind of the fruit but severe infection extends to the inner tissues and even to the seeds, showing ashy discolouration.

The conidia of *Drechslera rostrata* are pale to dark olivaceous brown, cylindrical to rostrate, somewhat less curved, measuring 43.06-124.94 × 9.24-17.63 μ in size with 5-13 transverse septa and a minute hilum protruding from the base. The other end is bluntly tapered. The septa from basal and apical cells are prominent being thicker and darker than the intermediate septa.

Lande and Utikar (1978) further reported that *D. rostrata* showed abundant mycelial growth and sporulation on PDA, M-2 agar, Czapek's agar and Coon's agar medium. Fungicides, Dithane M-45 and Captan were found best for checking the growth and sporulation of the fungus completely.

2.9.6 *Flower and Fruit Lesion*

Thankamma (1983) reported lesions on the male and female flowers, and fruits caused by *Phytophthora nicotianae.* The infected flowers are shed prematurely.

The fruit remains on the plant but carry infection. Scrapings from the fruit surface show plently of *Phytophthora* sporangia. Inoculated flowers on twig produce typical lesions.

The fungus in culture is uniformly fluffy with no zones. Hyphae are fairly uniform in diameter measuring 4.8 mm with hyphal swelling. Chlamydospores measuring 36 mm are produced in plenty and are both intercalary and terminal. Chlamydospores readily germinate in water. Sporangia are broadly turbinate with spherical basal portions and apical part prolonged into a beak, papillate and measure 63 × 44 µm. Sporangia do not usually shed. No oospore is produced in single culture but readily produce with rubber isolate.

2.9.7 *Cladosporium Fruit Rot*

Cladosporium oxysporum Berk and Curt. cause fruit rots in *Punica granatum* (Panwar and Vyas, 1974). Initially, the diseased fruits develop orange-red to dull-brown circular spots and the infected grains become olive-brown. In advanced stage, the entire fruit rots.

C. oxysporum grows well on PDA at 25°C. The mycelial colony is dark olive green, hyphae are septate, light olive green, 2.5-30 µm in width, conidiophores are light brown, simple and conidia are light brown to olive-green, 1-celled, fusoid, 1-20 × 3.5-4.5 µm.

2.9.8 *Aspergillus Fruit Rot*

Srivastava and Tandon (1971) reported fruit rot caused by *Aspergillus flavus* Link at Kanpur and Jodhpur. Philip (1979) recorded *Aspergillus* rot at Trivandrum in 1977. Two other species, *A. nivens* and *A. versicolor,* were also found to cause rot (Sharma *et al.,* 1981).

2.9.8.1 *Aspergillus flavus* : It causes brownish discolouration, which gradually becomes blackish and a little slimy. Subsequently, it gets slightly depressed and is later covered by green conidial heads of the incitant. The disease causes soft rot and emits fermented odour (Srivastava and Tandon, 1971).

2.9.8.2 *Aspergillus nivens* : It produces almost circular and light yellow patch. The infected tissue turns soft, darker in colour and soon gets covered by mycelial growth. Secretion of yellow watery substance and emittance of foul smell were associated with it (Sharma *et al.,* 1981).

2.9.8.3 *Aspergillus versicolor* : It produces small brownish patch which increases in diameter and turns darker with blackish tinge in the centre. The diseased tissue shrinks and ultimately disintegrates especially in the central region, followed by irregular depression and exudation of slimy mass emitting foul odour (Sharma *et al.,* 1981).

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