Chapter 13
Cotton Diseases and Their Management

Sobia Chohan, Rashida Perveen, Muhammad Abid, Muhammad Nouman Tahir, and Muhammad Sajid

Abstract Cotton industry is an important sector of the economy in all agriculture-based countries. Nevertheless, cotton production is constantly endangered by pathogens that cause considerable economic losses. Worldwide, numerous different diseases have been identified in cotton. *Fusarium* and *Verticillium* wilt, *Alternaria* leaf spot and seedling diseases, boll rot, leaf curl disease, and bacterial blight are the major constraints to the cotton fiber production. Maintaining the disease incidence at low level is the ultimate preference of the researchers. Understanding the etiology is the main factor to estimate the economic impact of diseases, which eventually helps to develop the management strategies. Presently, cotton leaf curl has emerged as main risk to all cotton-growing areas because of the changes in viral disease complex. In this chapter, brief history of the major diseases, the host-pathogen interactions, the taxonomy of the recognized causal agents, and different control strategies applicable to each disease including some rising techniques such as genome modification for enhanced resistance are discussed.

Keywords Cotton viral diseases · Fungal diseases · Bacterial diseases · Wilt diseases

13.1 Introduction

Cotton belongs to Malvaceae family and is the fabric of our lives as it is the prime source of fiber worldwide. Cotton is a leading cash crop as it plays a key role in the lives of millions of people in Asia, Africa, Australia, South America, and North

S. Chohan (✉) · R. Perveen · M. N. Tahir · M. Sajid
Department of Plant Pathology, Bahauddin Zakariya University, Multan, Pakistan
e-mail: sobiachohan@bzu.edu.pk

M. Abid
Department of Plant Pathology, Bahauddin Zakariya University, Multan, Pakistan

Institute of Plant Protection and Agro-products Safety, Anhui Academy of Agricultural Sciences, Hefei, China

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America, and its fiber is a valuable product that provides income to farmers and industrialists (Ahmad et al. 2014, 2017, 2018; Abbas and Ahmad 2018; Ahmad and Raza 2014; Ali et al. 2011, 2013a, b, 2014a, b). Cotton has been cultivated for thousands of years across the world to make fabrics. It is a valuable component of farming systems in approximately 60 countries worldwide (Amin et al. 2017, 2018; Khan et al. 2004; Rahman et al. 2018; Tariq et al. 2017, 2018; Usman et al. 2009). The top three leading countries in cotton production are China, the United States, and India. Pakistan is the fourth largest cotton-producing country with ~2.17-million-ton annual production. Cotton grows well in tropical and subtropical regions with warm and humid climatic conditions. There are many biotic factors that are responsible for crop yield losses, plant vigor, and situations that eventually result in poor fiber quality. The reduced cotton production by biotic stresses is caused by plant fungal, bacterial, and viral pathogens.

To date, the struggle against cotton diseases continues for its proper management under sustainable agriculture. It has been revealed that cotton was affected by more than sixty diseases, which brought heavy yield losses. The work carried out so far shows that seedling rots, boll rot, different types of leaf spots, stunting, reduction in the size of leaves, premature opening of bolls, attack of nematodes, blights by bacteria, and leaf curling due to virus are found responsible for causing huge damage to cotton crop. The extent of disease damage depends on the environment and cultivar genotype.

Wilts, boll rots, and root rot are more important among other fungal diseases. The loss caused by certain fungal diseases is obvious, as in the case of root-rot plants that died, while in others, losses caused are not evident besides hard to quantify at numerous stages of development. The most significant bacterial disease is bacterial blight incited by *Xanthomonas campestris* pv. *malvacearum*, which occurs mainly in South Asia. The bacterium destroys the protoplasm of leaves that ultimately causes disruption in the process of photosynthesis. In case of severe attack incidence, the lesions appear on stem and bolls.

The crop record revealed that root and boll rots were considered as the most severe and destructive diseases of cotton, and since the last decade, cotton leaf curl disease (CLCuD) has been found to be a vital cotton disease. The CLCuD caused by whitefly-transmitted begomoviruses (family *Geminiviridae*) is predominant in South Asia and Africa and is a key constraint to productivity besides fiber quality.

This chapter includes some important pathogens of cotton crop and describes their control measures. Generally, single control strategy is not wholly effective to control diseases; thus, integrated approaches are more useful. The strategies which contribute to control one disease usually help in controlling others too. Besides chemical control, sowing disease-free seed and resistant varieties, employing crop rotation, and removing infected plant debris along with suitable practices should be part of integrated disease management strategy. Strict quarantine regulation should also be followed, as the exchange of diseased planting material is the key factor in the disease dispersion.

13.2 Fungal Diseases

13.2.1 Seedling Diseases

Seedling diseases are a worldwide problem in cotton-growing areas causing up to 5% estimated average yield loss annually. There are different fungal species involved in cotton seed rots and seedling diseases. The primary fungal genera associated to seed deterioration are *Fusarium* (Klich 1986), *Rhizoctonia* (Brown and McCarter 1976), *Pythium* (Devay et al. 1982), and *Thielaviopsis* (King and Barker 1934). These pathogens cause damping-off disease and colonize the weak cotton seedling plants.

13.2.1.1 Symptoms

The disease-causing organisms attack seeds and seedlings at pre-emergence and post-emergence stages. Symptoms include decay of seeds and young seedlings, partial or complete stem girdling, stunted growth, and seedling rot. The fungal pathogens invade the seedlings at soil level producing water-soaked, reddish-brown sunken lesions and girdling the hypocotyl, and the seedling may collapse. On examination of infected seedlings, dark lesions may expose on stem and roots. Seedling diseases not only kill the entire seedling population but also result in uneven stands with skips in rows. Surviving seedlings become pale and stunted and die soon. This condition of hypocotyl damage has been known as "sore shin" in the United States (Atkinson 1892).

13.2.1.2 Disease Cycle

Under favorable conditions, hyphae of *Rhizoctonia solani* grow rapidly in soil and convert into mycelia. The fungus may survive for years in soil/plant debris as sclerotia. *Pythium* spp. overwinter as oospores and infect host tissues through germ tube produced from an encysted zoospore. *Fusarium* spp. and *Thielaviopsis basicola* overwinter as chlamydospores in soil and plant residues for years.

13.2.1.3 Predisposing Factors

Seedling diseases are more prevalent under cool and wet climate. Sowing of cotton seeds in sandy soils with low organic matter increases the susceptibility of cotton to fungal pathogens. Some factors like deep planting, poor seed bed conditions, and compacted soil besides nematode or insect infestations may increase the problem.

13.2.1.4 Management

Cotton seedling disease control is based on preventive rather than curative treatments. Rotation with non-host monocotyledonous crops like wheat, corn, and sorghum can be useful in reducing the inoculum rate. Cultural practices like planting in raised beds can help in controlling seedling diseases by improving soil drainage. Cultivation of disease-resistant varieties and planting of good quality seeds are recommended. Eradication of the debris of the infected plant parts may help to control seedling diseases of cotton. Seed treatment with suitable fungicides like thiram, azoxystrobin, and metalaxyl is effective against these diseases. Nemli and Sayar (2002), while examining the effects of different fungicides, found that combinations of carboxin+thiram+metalaxyl besides fludioxonil+metalaxyl are more effective against seedling root rot. The use of bioagents to reduce fungal population and inoculum level in soil is also practiced by many researchers. Certain biofungicides are commercially available such as Kodiak, Subtilex, and Deny; these are suggested against seedling diseases (McSpadden Gardener and Fravel 2002). Moreover, Erdoğan et al. (2016), Wang et al. (2004), and Pleban et al. (1995) determined the effects of fluorescent *Pseudomonas* (FP) and *Bacillus subtilis* bacteria against *Rhizoctonia solani*, *Colletotrichum gossypii*, and *Fusarium* spp.

13.2.2 Foliar Diseases

13.2.2.1 *Alternaria* Leaf Spot

Leaf blight is a common foliar disease found almost in every cotton-growing area around the world (Fig. 13.1). This disease was firstly stated in the United States (Atkinson 1892; Paulwetter 1918). Later, similar leaf blight and spot were also found on upland cotton in Nigeria (Jones 1928), in Zimbabwe (Hopkins 1932), and in India (Rane and Patel 1956).

13.2.2.2 Symptoms

Initial symptoms appear as small, circular brown, and gray brown to tan lesions with purple margins on green leaves. These spots vary in size from 1 to 10 mm in diameter exhibiting concentric zonation on older leaves (Fig. 13.1). As the disease progresses, mature lesions may coalesce and become irregular and necrotic. The affected leaves become blighted and brittle and often crack exhibiting shot hole appearance. The disease is more severe on lower leaves than upper leaves, except leaves are affected by premature defoliation. Under humid weather conditions, prolific sporulation of the fungus may result in black sooty masses on necrotic lesions. Lesions may also appear on stem, bracts, and bolls. *Alternaria* leaf spot

Fig. 13.1 *Alternaria* leaf blight of cotton

may be mixed with angular spots of bacterial blight that is described later in this chapter.

13.2.2.3 Causal Organism

Taxonomy

Leaf spot is caused by *Alternaria macrospora* Zimm. It is an ascomycetous fungus belonging to class Dothideomycetes, order Pleosporales, and family Pleosporaceae. Earlier, it was recognized as *A. alternata* (Fr.) Keissler in Egypt and Russia (Kamel et al. 1971; Dzhamalov 1973), but recent reports from Zimbabwe described it as *A. macrospora* (Hillocks 1991).

Morphology

Alternaria macrospora comprises cylindrical to slightly tapering conidiophores which are formed solitarily or in clusters. They are septate, erect, flexuous, and pale brown in color. The conidia are produced singly or sometimes in chains of two, light to dark brown in color with 4–9 transverse septa and several longitudinal septa, ellipsoidal, melanized, and obclavate to obpyriform with narrow beak (Ellis 1971). There is substantial variation in conidial size, but most descriptions suggest as 70–180 × 15–22 μm (Ellis 1971; Sangeetha and Ashtaputre 2015; Venkatesh and Darvin 2016; Waghunde et al. 2018).

13.2.2.4 Disease Cycle

Crop residues and infected seeds are the main cause of inoculum and give rise to infected seedlings that support early stages of an epidemic. The conidia spread through air currents and water splashes onto healthy plants. Prolonged wet and humid weather conditions and temperature (about 27 °C) favor disease development initially from cotyledons to the lower leaves. Under favorable conditions, pathogen kills surrounding leaf tissues and produces abundant spores on the surface of the lesions within few days. Defoliation of leaves of susceptible varieties encourages maximum sporulation of *A. macrospora* (Bashi et al. 1983), and damaged bolls are responsible for the seed infection (Bashan 1984). Symptom development may have favored by nutritional or physiological stress to plants like premature senescence and heavy fruit loads. The disease cycle is completed with the shedding of infected leaves or the planting of infected seed.

13.2.2.5 Predisposing Factors

Extended periods of wet weather and high humidity favor the disease development. Minimum temperature for the disease to occur is 10 °C and maximum is 30 °C, whereas optimal temperature for disease development ranges between 20 and 30 °C. Soils deficient in potassium favor the disease development (Hillocks 1991).

13.2.2.6 Management

As fungus can survive on infested crop residues in soil, thus, residue management through tillage may reduce inoculum production in the field. Rotation of cotton with cereals helps in reducing seedling infection to a sufficient level. Application of fertilizers especially potassium is adequate to maintain soil fertility level (Hillocks 1991).

Planting healthy seeds and cultivation of resistant varieties are recommended. Bashan (1986) considered that higher phenol contents might be responsible for resistance to the disease.

Controlling the disease with foliar fungicides is usually considered economically useful. Foliar spray with copper fungicides, such as mancozeb at 2.5 g/L and difenoconazole at 1 ml/L, helps in reducing the primary inoculum. Similarly, fungicidal seed treatment with broad spectrum fungicides like strobilurins (trifloxystrobin) and sterol biosynthesis inhibitors (ipconazole) is found effective in protecting the cotyledons of emerging seedlings from the fungus.

Seed treatment with bioagent *Pseudomonas fluorescens* at 10 g/kg seeds after every 10 days of interval may reduce the disease intensity.

13.2.3 Grey Mildew Disease

The disease known as grey mildew or dahiya in India, false mildew/areolate mildew in the United States, and white mildew in South America (Hillocks 1991) was first reported in the United States (Atkinson 1891). The disease is of little importance in the United States; however, it is quite common in India, East Africa, and South America.

13.2.3.1 Symptoms

Initial symptoms appeared firstly on lower leaves as irregular-angular lesions measuring 1–10 mm in diameter after first boll set. They are light green to yellow green translucent spots bounded by veinlets (called areolate) on upper surface, but on under surface of the leaves, white mildew-like growth is observed as a result of abundant sporulation. Under high humidity, lesions may also become white on upper surface of the leaves. This is conidial stage of causal fungus. Later, the lesions turn dark brown in color and become necrotic. Symptoms also appear on cotyledons as circular water-soaked patches which turn reddish brown and chlorotic. Severe infection leads to defoliation and premature boll opening.

13.2.3.2 Causal Organism

Taxonomy

The causal agent of grey mildew is *Ramularia areola* (Atk.) (synonym: *Ramularia gossypii* Speg.). Its anamorph is *Cercospora gossypina* Cooke (Ehrlich and Wolf 1983). The teleomorph stage of fungus is known as *Mycosphaerella areola* Earle (Ehrlich and Wolf 1983; Gouws et al. 2001).

Morphology

Conidiophores of *R. areola* bear hyaline septate conidia measuring 14–30 × 4–5 μm. On lower surface of fallen leaves, spermogonia appear as black dots on the lesions in addition to conidial stage. They are 28–75 μm in diameter and on maturity liberate rod-shaped spermatia (2–4 × 0.4–2 μm) in a matrix through the ostiolum. Later, these conidia and spermogonia are replaced by dark brown-colored perithecia (70–80 μm diameter) with a slight papilla. These perithecia produce fusiform asci measuring 35–40 × 6–8 μm having eight elongated, biseriate ascospores which are 12.4–15.6 × 3.2–3.8 μm (Ehrlich and Wolf 1983).

13.2.3.3 Disease Cycle

The fungus passes through three separate phases during its whole life cycle. One stage is conidial stage that appears on the underside surface of the leaves. Next is spermagonial stage that develops on fallen leaves followed by the third stage, i.e., ascogenous, which produces on partially decayed leaves. Conidia and ascospores are the primary sources of inoculum. It is disseminated by wind and irrigation water. For germination of conidia and ascospores, free moisture is required with temperature range of 16–34 °C. The optimum temperature for growth is 25–30 °C.

13.2.3.4 Predisposing Factors

Humid conditions with sporadic rains are favorable for development of the disease.

13.2.3.5 Management

Destruction of crop residues, deep plowing and crop rotation are such cultural practices which reduce the multiplication and spread of primary inoculum.

Foliar application of benomyl at 200–300 g ha^{-1} is effective in controlling grey mildew of cotton.

Using resistant cultivars is the best approach to control the disease.

13.2.4 Boll Rot Disease

Boll rot occurs to some extent in most of cotton-growing regions in the world. However, yield losses occur only in areas of high humidity during late summer and fall. As a result of this disease, poor quality of lint is produced. Numerous microorganisms are associated with boll rots. Some of these organisms directly invade the cotton bolls, while others enter through insect wounds or as secondary invaders. Nearly hundred microorganisms have been isolated from rotted bolls (Hillocks 1991). Most commonly isolated fungi from rotted bolls of cotton is *Fusarium* spp. throughout the cotton-growing countries. In America, *F. oxysporum*, *F. roseum*, *F. solani*, and *F. moniliforme* are mainly isolated from rotted bolls (McCarter et al. 1970). *F. moniliforme*, *F. roseum*, *F. solani*, and *Colletotrichum* spp. are involved in boll rot in Africa (Follen and Goebel 1973), whereas in India, *F. equiseti* has been reported as a cause of boll rot (Sharma and Sandhu 1985). Recently, a species complex of *F. incarnatum-equiseti* is reported to cause boll rot in cotton (Chohan and Abid 2018).

Infection of boll first starts as appearance of water-soaked necrotic lesions on the margins of the bracts. During moist conditions, these lesions enlarge, and white to gray or salmon-colored/pale pinkish fungal growth covers the infected bolls. Severely infected bolls may drop from plants (Figs. 13.2 and 13.3).

Fig. 13.2 Cotton bolls infected with *Fusarium* spp

Fig. 13.3 Infected cotton bolls with *Fusarium* spp

To prevent boll rots, farmers should adopt agronomic practices such as to avoid excessive application of nitrogen, maintain low humidity by minimizing the size of crop canopy and burning of diseased crop residues. Acid delinting helps in eliminating seed-borne infections. Chemical approach may also be useful in controlling boll rot.

13.2.5 Wilt Diseases

Cotton plants are attacked by two destructive wilts; one is named as *Fusarium* wilt and the other as *Verticillium* wilt. In case of both wilts, the vascular system of plant is colonized by the pathogen. The microorganism invades the root of cotton plant, penetrates and proliferates within xylem tissues, and eventually spreads throughout the plant. Vascular wilt fungi are soil-inhabiting pathogens, but these may grow on crop residues in the absence of host for long periods in the form of thick-walled resting structures. On favorable conditions, the fungi sporulate and cause infection by blocking the vascular system of cotton plant.

13.2.5.1 Fusarium Wilt

Fusarium wilt of cotton was first reported by Atkinson (1892) from America. It is generally found wherever cotton is grown around the world. It has been originated from Mexico and spread to South America, the United States, Egypt, West Indies, Italy, Africa, Greece, Zimbabwe, China, France, Russia, and India (Menlikiev 1962; Cook 1981; Hillocks 1992).

Symptoms

Wilting symptoms may appear any time of plant growth, i.e., from seedling to maturity. If seedlings are infected, first symptoms appear as vein darkening, yellowing, and shriveling of young cotyledons. Later, the cotyledons become necrotic and shed, and eventually seedlings may wilt and die. In older plants, initial symptoms appear as marginal yellowing of lower leaves. The leaves become flaccid resulting in drooping and wilting of the whole plant. Sometimes, symptoms appeared on one side of the plant (Figs. 13.4 and 13.5). When conditions are conducive for disease expansion, the symptoms appear within 2 months after sowing resulting into wilting and death of plants with a few boll settings.

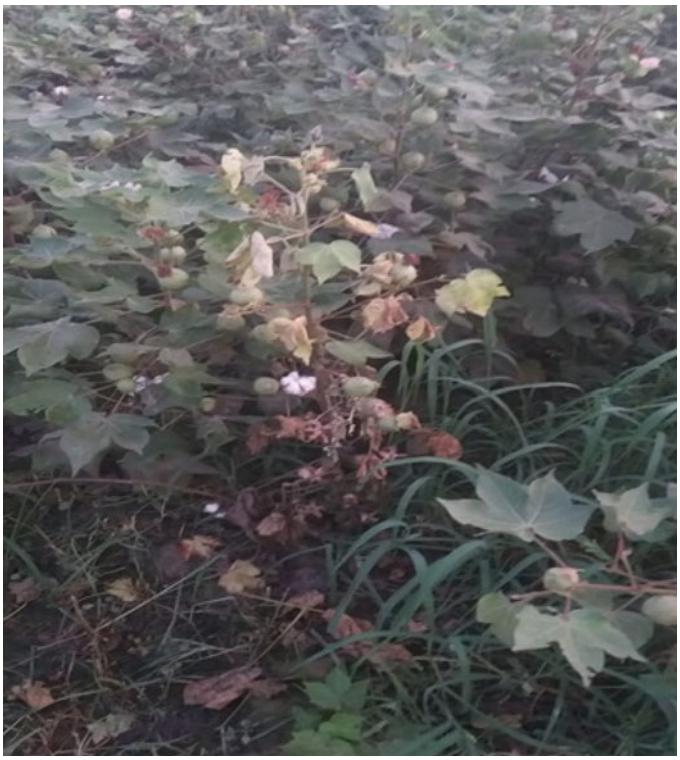

Fig. 13.4 Cotton plants showing symptoms of *Fusarium* wilt disease

Fig. 13.5 Cross section of cotton stem showing symptoms of *Fusarium* wilt disease

Causal Organism

Taxonomy

The causal microorganism is an asexual ascomycetous fungus belonging to the class Hyphomycetes. It is recognized as *Fusarium oxysporum* Schlecht f. sp. *vasinfectum* Atk. Syn. & Hans. Snyder and Hansen (1940) grouped the parasitic forms into *formae speciales* based on host specificity of the strains. There are more than 120 *formae speciales*.

Morphology

Mycelium of *F. oxysporum* f. sp. *vasinfectum* is initially white to grayish white or bluish purple in color and produces two types of conidia: microconidia and macroconidia. Microconidia are small, one to two-celled, and elliptical in shape measuring 5–20 × 2.2–3.5 μm. Macroconidia are multinucleate, usually three to five septate, fusiform, sickle-shaped, and light buff to salmon colored measuring 27–48 × 2.5–4.5 μm. Resting spores are called chlamydospores which are mostly spherical, single or in chains, terminal or intercalary measuring 7–13 μm in diameter. The distinguishing feature of *F. oxysporum* from other *Fusarium* species is formation of chlamydospores with short conidiophores.

Disease Cycle

Pathogens are soil borne and survive as chlamydospores in the absence of host for several years. Fungus also survives as saprophyte on plant debris. Chlamydospores are sources of primary infection. Infection starts as conidia germinate, 6 h after inoculation on root surface, and form a mycelial mat covering the root surface. Later, the penetrating hyphae become systemic and proliferate in the xylem vessels, and conidia are transported upward in transpiration stream and produce more mycelium. Wilt symptoms usually appear when cotton plants are about 5–6 weeks old.

Predisposing Factors

Optimum temperature for disease to develop is between 20 and 27 °C, and moisture contents of 80–90% capacity are favorable for disease.

Management

Wilt disease can be managed through using resistant varieties, implementation of cultural practices like mixed cropping, field sanitation, proper use of fertilizers and micronutrients, and crop rotation.

Biological control with non-pathogenic bacteria like *Pseudomonas fluorescens* is found effective in reducing incidence of wilt disease of cotton.

13.2.5.2 Verticillium Wilt

Verticillium wilt is the second vascular wilt disease affecting the cotton crop. Vascular pathogens have the ability to grow inside the vascular system, but these fungi grow outside the vascular tissues on advance stage of infection. *Verticillium* wilt may sporulate on plant residues after the death of host and specialized soil-invading pathogen with narrow host ranges. *Verticillium* wilt has the ability to survive for a long time in the absence of host plant in soil and is susceptible to toxic fungal combinations.

Verticillium wilt of cotton was first reported in 1974 after *Verticillium dahliae* was isolated from a few diseased plant of upland cotton growing in Arlington, Virginia. In 1932, Miles and Persons reported that *Verticillium* wilt of cotton occurred near Mississippi River heavy loam soil. In 1936, Barker and Sherbakoff confirmed the causal agent after the thorough survey of US cotton wilts and found that *Verticillium* wilt was common in Texas, Arizona, New Mexico, and Missouri as well as in California. After in the United States, it was also reported in Central Asia, Brazil, China, Turkey, and South Africa. Outbreak of cotton wilt occurred in Australia when cotton first grows on land (Evans 1967).

Symptoms

Initially, the young plants infected show yellowing, epinasty, and defoliation of the leaves, and in warm weather, they recover quickly and show stunted growth. Under high humid conditions, early infection causes little loss of yield and quality. The plant leaves show mosaic pattern with yellowing of tissues along margins besides between veins. Leaves and bolls of the plant remain defoliated and ultimately killed.

Taxonomy

The causal organism of *Verticillium* wilt of cotton was often given as *V. albo-atrum*. Five species of *Verticillium* are *V. albo-atrum* Reinke & Berthold 1879, *V. dahliae* Klebahn 1913, *V. nigrescens* Pethybridge 1919, *V. nubilum Pethybridge* 1919, and *V. tricorpus* Isaac 1953.

Morphology

Initially white or light cream, but later become black with formation of microsclerotia colonies on PDA of *Verticillium dahliae* grow moderately fast (2.0–3.5 mm at 20–25 °C). Microsclerotia are dark brown to black and then torulose. Conidia are ellipsoidal to short cylindrical with erect and prostrate conidiophores.

Disease Cycle

Verticillium wilt survives as dormant microsclerotia present in the soil debris and soil depths down to 40 cm. Microsclerotia produced colonies on cotton root surface in response to exudates. Hyphae from colonies penetrate the xylem vessels through wounds. Wounds are not required but increase wilt infection. Hyphae surrounding throughout the necrotic tissue of death leaves, stems, and roots, where microsclerotia are formed after several weeks and month and depending on moisture amount, which again dispersed in soil (Schnathorst 1981; Huisman and Gerik 1989; Bell 1992).

Predisposing Factors

Verticillium wilt grows well on simple sugars and amino acid that are normally found in root exudates and xylem sap. The optimal temperature for growth is 22–27 °C.

Management

Several methods are used for the control of wilt disease. A combination of cultural, chemical, and biological methods is also used to minimize the losses caused by wilt. Selection of resistant cultivars is another way to control the wilt disease because the cultivars have the capacity to resist against disease infection.

In cultural control, using different methods and resistant varieties is practiced to prevent the introduction and spread of the disease in soil and reduce the inoculum rate. The methods are rotation, use of fertilizer, control of soil moisture, planting time and tillage method, reduction of seed transmission, planting density, removal of weeds and crop residues, and solarization.

In chemical control, fungicides such as carbendazim and ethylene thiosulphonate can control seedling pathogens as well as prevent seed transmission of pathogen (Shen 1985). Benzimidazole fungicides are systematic and complete control of the *Verticillium* wilt in glasshouse and field with different concentration, for example, 100 ppm of Benlate in water drenchers and 10–20 kg of Benlate in field.

In biological control, more than 20 years *Trichoderma viride* (*T. lignorum*) has been used to control wilt in Russia (Fedorinchik 1964). In low organic matter soil, *Gliocladium roseum* may be better than *T. viride* as an antagonist of *V. dahliae* (Globus and Muromtsev 1990).

13.3 Viral Diseases

13.3.1 Cotton Leaf Curl Disease

CLCuD is the most noteworthy restraining factor in cotton productivity in Pakistan (Briddon and Markham 2000; Sattar et al. 2013, 2017). Whitefly-transmitted

CLCuD, prevalent from South Asia to China, is favored by high temperature conditions. The disease was initially stated in Nigeria in 1912 (Kirkpatrick 1931) and then also reported from other African countries such as Sudan, Tanzania, Egypt, and Malawi along with South Africa. In Pakistan, the first disease incidence was reported as a minor attack in 1967 near Multan. The disease came into noticed when cotton production of Pakistan suffered due to the epidemic of CLCuD in 1988. Later on, CLCuD was spread out across Pakistan as well as into northwest India. In India, the first incidence of CLCuD was reported in 1989 near Sri Ganganagar. In Pakistan, introduction of highly susceptible varieties in the 1990s resulted in high yield loss causing financial loss of about US$5 billion (Briddon and Markham 2000). During the late 1990s, cultivating locally developed tolerant varieties in Pakistan restored the cotton production. However, in 2001–2002, characteristic disease symptoms were observed on resistant cotton cultivars in Burewala region in Punjab, Pakistan, suggesting the second epidemic of CLCuD. Currently, this dominant resistance-breaking recombinant strain of CLCuD is spread throughout Punjab, Pakistan, and into northwest India.

The CLCuD-affected plants show distinctive symptoms like vein swelling, upward/downward leaf curling, and stunted plant growth along with the formation of cup-shaped leaf-like-outgrowth undersides of leaves known as enations (Fig. 13.6). Early infection results in severe stunting of plants with high yield loss, while late infection causes mild symptoms (Sattar et al. 2013).

Fig. 13.6 Cotton plant showing leaf curl symptoms with enations

13.3.1.1 Etiology of CLCuD

Causative agent of CLCuD from Africa besides Asia has been determined. In these continents, the disease is associated with begomovirus complexes, which consist of a monopartite begomovirus and a symptom-modulating satellite molecule called betasatellite, previously known as DNA β. CLCuD infection is also shown to be linked with a satellite-like molecule called alphasatellite, previously known as DNA 1.

13.3.1.2 Begomoviruses Associated with CLCuD

The most destructive whitefly-transmitted genus *Begomovirus* (family *Geminiviridae)* consists of small, single-stranded, circular DNA genomes encapsidated in twinned quasi-icosahedral particles. Excluding begomoviruses, all geminiviruses are monopartite, having a single genomic component that is capable of replication, systemic movement, and infections. The begomoviruses are either bipartite or monopartite (consisting of a component that is homologue of DNA-A). Begomoviruses from the New World have bipartite genome DNA A and DNA B, both of which are essential for successful infection (Stanley 1983). Although in the Old World there are bipartite begomoviruses causing disease in field crops, however, a large number of diseases are caused by monopartite begomoviruses.

In spite of early identification of CLCuD in Africa, causative agent was recognized too later. Single begomovirus *Cotton leaf curl Gezira virus* (CLCuGeV) has been identified with CLCuD in Africa (Idris and Brown 2002). CLCuGeV has been identified from numerous plant species as well as cotton, hollyhock, okra, and *Sida* spp. (Tahir et al. 2011). CLCuGeV is a geographically widespread begomovirus as was identified from different host plants including cotton from diverse areas in Asia (Tahir et al. 2011; Khan et al. 2012; Idris et al. 2014).

In Asia, the scenario regarding CLCuD is much more complex. Several begomoviruses have been associated with CLCuD. In Pakistan, during the first epidemic of CLCuD, three diverse species of begomoviruses were identified: *Cotton leaf curl Alabad virus* (CLCuAlV), *Cotton leaf curl Kokhran virus* (CLCuKoV), and *Cotton leaf curl Multan virus* (CLCuMuV) (Zhou et al. 1998). Later, analysis identified other species of begomoviruses: *Cotton leaf curl Rajasthan virus* (CLCuRaV), *Papaya leaf curl virus* (PaLCuV), and *Tomato leaf curl Bangalore virus* (ToLCBaV) (Mansoor et al. 2003; Kirthi et al. 2004).

However, during 2001, the change in the genetic makeup of begomoviruses in Pakistan results in the appearance of distinct resistance-breaking recombinant strain, named CLCuKoV-Burewala strain (CLCuKoV-Bu) (previously known as Cotton leaf curl Burewala virus) (Amrao et al. 2010a). Recombinant strain genome is derived from two begomovirus species linked with first CLCuD epidemic, CLCuMuV along with CLCuKoV. Following resistance breakdown, only CLCuKoV-Bu was found to be linked with CLCuD across Punjab, Pakistan.

Although during the 1990s, southern parts in Pakistan remained unaffected. However, disease appeared in Sindh province since 2004 (Mansoor et al. 2006) and extended to northwest Indian states during 2005 (Rajagopalan et al. 2012). Additionally, another species of begomovirus associated with CLCuD was identified in southern India, named *Cotton leaf curl Banglore virus* (CLCuBaV) (Reddy et al. 2005). A new recombinant species was characterized which has been shown to be associated with CLCuD in Sindh province, named CLCuKoV-Shahdadpur strain (CLCuKoV-Sha), formerly known as Cotton leaf curl Shahdadpur virus (Amrao et al. 2010b). In Sindh province, in addition to CLCuKoV-Sha and CLCuKoV-Bu, CLCuD-associated African begomovirus, CLCuGeV has been also reported in cotton (Tahir et al. 2011).

During both epidemics, from Indo-Pak subcontinent region, only monopartite begomoviruses with DNA satellites are associated with CLCuD until 2013–2014. However, in recent times, bipartite begomoviruses *Tomato leaf curl Gujarat virus* (Zaidi et al. 2015) and *Tomato leaf curl New Delhi virus* (ToLCNDV) (Zaidi et al. 2016) have been found from infected cotton plants in Pakistan. Furthermore, *Okra enation leaf curl virus* and a *Mastrevirus Chickpea chlorotic dwarf virus* have also been identified from cotton plants. Recent studies show that ToLCNDV has not only established in Asia but also spread out to Africa and Europe (Mnari-Hattab et al. 2015; Zammouri et al. 2017; Ruiz et al. 2015; Panno et al. 2016; Juárez et al. 2014). Under experimental conditions, DNA A of ToLCNDV trans-replicates CLCuD-associated betasatellite resulting in accumulation of symptom-modulating satellite components (Saeed et al. 2007). The presence of bipartite ToLCNDV along with monopartite begomoviruses suggests a possible future epidemic in areas under cotton cultivation by CLCuD complexes in South Asia (Sattar et al. 2017).

Similarly, in Africa, like Asia, a bipartite begomovirus *Cotton yellow mosaic virus* (CYMV) has been reported first time in cultivated cotton (Leke et al. 2016), suggesting accumulation of distinct begomoviruses from their hosts to susceptible cotton plants in the OW.

13.3.1.3 DNA Satellites Associated with CLCuD

Satellites are termed as viruses/nucleic acids which depend on a helper virus for their replication but lack extensive nucleotide-sequence homology to the helper virus and are dispensable for proliferation. In OW, majority of the monopartite begomoviruses are associated with more frequently found ssDNA satellite components recognized as betasatellites (Briddon and Stanley 2006), satellite-like components alphasatellites, and newly characterized deltasatellites (Zhou 2013; Fiallo-Olivé et al. 2012).

Betasatellites are around half of the size of helper begomoviruses (~1350 nt) having no sequence homology with helper begomoviruses, excluding non-nucleotide sequence TAATATTAC (Briddon et al. 2003). Betasatellites genome consists of single coding sequence in complimentary sense known as

βC1. Pathogenicity determinant gene βC1 has been involved in upregulating viral titer in host and overcoming the host defenses (Qazi et al. 2007; Briddon et al. 2001; Amin et al. 2011). All betasatellites have sequence rich in adenine (A-rich region) along with highly conserved region recognized as satellite conserved region (SCR).

In OW, both Asian and African CLCuD complexes require their cognate symptom-modulating betasatellites. In Africa, a distinct betasatellite, Cotton leaf curl Gezira betasatellite (CLCuGeB), is found to be linked with CLCuGeV (Idris and Brown 2002). In Asia, different strains of the most important betasatellite CLCuMuB are linked with CLCuD (Akhtar et al. 2014).

In the 1990s, during the first epidemic of CLCuD, although six distinct monopartite begomoviruses were identified, only a single betasatellite Cotton leaf curl Multan betasatellite-Multan strain ($CLCuMuB^{Mul}$) was found to be linked with CLCuD (Mansoor et al. 2003). In the second epidemic of CLCuD, with resistance-breaking begomovirus CLCuKoV-Bur, a distinct "Burewala strain" of CLCuMuB ($CLCuMuB^{Bur}$) became prominent. $CLCuMuB^{Bur}$was also found to be a recombinant, containing some sequence within the SCR from Tomato leaf curl betasatellite (ToLCB) (Amin et al. 2006). Similarly, CLCuD in Sindh, which was found to be linked with CLCuKoV-Sha, a new recombinant betasatellite of CLCuMuB called "Shahdadpur" strain ($CLCuMuB^{Sha}$), was characterized (Amrao et al. 2010b). As compared to $CLCuMuB^{Bur}$, $CLCuMuB^{Sha}$ contains a smaller fragment from ToLCB in SCR. Along with malvaceous betasatellites, $CLCuMuB^{Bur}$and $CLCuMuB^{Sha}$, a non-malvaceous betasatellite Chili leaf curl betasatellite has also been reported from cotton in Sindh (Tahir et al. 2011). The presence of a distinct biotype of *B. tabaci* may be the reason for the association of different begomoviruses with distinct strains of betasatellites in Sindh, Pakistan.

Alphasatellite is the third component of CLCuD complex. Alphasatellites are able to replicate autonomously in host; thus, they are known as satellite-like molecules. These molecules depend on their helper virus for transmission along with encapsidation (Mansoor et al. 1999; Briddon et al. 2004).

Alphasatellites are circular ssDNA molecules (~1400 nt) having highly conserved genome with a hairpin structure with a non-nucleotide sequence (TAGTATTAC), an A-rich region, and a single large virion-sense gene, encoding a rolling-circle replication initiator protein (the replication-associated protein [Rep]) (Briddon et al. 2004). Alphasatellite Rep shows high level of sequence homology with the Rep encoded by another family of DNA viruses, *Nanoviridae* (Saunders and Stanley 1999). Thus, the origin of alphasatellites is supposed to be nanoviruses (Briddon et al. 2004).

In Africa, Cotton leaf curl Gezira alphasatellite (CLCuGeA) has been involved in CLCuD complexes in association with CLCuGeV and CLCuGeB. In Asia, Cotton leaf curl Multan alphasatellite (CLCuMuA) is found to be linked with CLCuD complexes.

Although alphasatellites were first identified in CLCuD-affected cotton in 1999 in Pakistan (Mansoor et al. 1999), however, following resistance-breaking epidemic of CLCuD, alphasatellites were not detected with CLCuD until 2009. In recent studies, alphasatellites are not only identified with CLCuD complexes but also found to be associated with other begomovirus and betasatellite complexes. Another alphasatellite

Gossypium darwinii symptomless alphasatellite has been reported from wild cotton in Pakistan (Nawaz-ul-Rehman et al. 2012).

From OW, alphasatellites have only been described in relationship with monopartite begomoviruses and betasatellite complexes. However, recently alphasatellites are reported in NW in association with bipartite begomoviruses but in the absence of betasatellites (Paprotka et al. 2010; Romay et al. 2010; Ferro et al. 2017). Due to the rising number of alphasatellites, scientists create a new system of nomenclature and classification of alphasatellites. Alphasatellites are assigned to a new family *Alphasatellitidae*, which is divided into two subfamilies: *Geminialphasatellitinae* that includes geminivirus-associated alphasatellites and *Nanoalphasatellitinae* consisting of nanovirus-associated alphasatellites (Briddon et al. 2018). According to the current classification, subfamily *Geminialphasatellitinae* consists of four genera, and CLCuD-associated alphasatellites are grouped in the genus *Colecusatellite* (Briddon et al. 2018).

The precise reason for the presence of alphasatellites with begomovirus-betasatellite complexes yet needs to be cleared. However, evidence regarding the function of Rep encoded by alphasatellites in suppression of posttranscriptional gene silencing shows their involvement in overcoming host defense mechanism (Nawaz-ul-Rehman et al. 2010).

13.3.2 Cotton Leaf Crumple Disease

A bipartite begomovirus *Cotton leaf crumple virus* (CLCrV), transmitted by whitefly in persistent manner, is found to be associated with cotton leaf crumple disease (CLCrD) in the NW. CLCrV infection produced characteristic foliar discoloration and veinal hypertrophy resulting in puckering or crumpling, downward leaf curling, and shortening of internodes along with stunting of host (Brown et al. 1987). CLCrD losses depend on plant age at infection time. Disease severity is more pronounced if plants become infected during early stage (Brown et al. 1987).

Cook made the first report of CLCrD in 1924 and named it as crazy top at that time (Cook 1924). The disease became prominent due to an outbreak in the 1950s when it was reported in California (Dickson et al. 1954) and in Arizona later (Allen et al. 1960). CLCrD mainly occurs in California, Arizona, Texas, and Mexico. In California in 1958, 71–85% yield losses due to CLCrD were reported (Van Schaik et al. 1962). In 1983, CLCrD resulted in 23–55% yield reduction in Arizona (United States). Although the seed index was reduced, however, the lint weight or lint index was not affected (Allen et al. 1960; Van Schaik et al. 1962; Brown et al. 1987). Ratooning cotton with early increase in whitefly populations was the contributing factor of CLCrD. Elimination of stub cotton in southwestern United States helps in controlling CLCrD since 1982. However, CLCrD tolerant lines have also been designated for developing resistant cotton cultivars Idris and Brown 2002.

13.3.3 Perspectives for the Viral Disease Management

Begomoviral disease can be managed by controlling the insect vectors using pesticides, as a single viruliferous whitefly is capable of transmitting virus. On the other hand, removal of viral inoculum sources (alternate hosts) is also efficient in reducing the diseases spread by whitefly feeding on infected plants. However, in cotton-vegetable cropping systems when there is no host-free period, it is difficult to attain significant control of the whitefly populations. In Pakistan, such conditions prevail due to prevalence of distinct viruses with wide host range. Thus, cultural control of whiteflies is not very effective because of broad host range of whitefly.

Resistance to CLCuD is the effective approach to control the disease, when disease incidence occurs regularly in the production season. The genetic makeup of the host and whitefly populations with the concentration of inoculum at a particular area influences the natural incidence of CLCuD.

In the late 1990s, using conventional breeding approaches, a number of CLCuD-resistant varieties were developed. Cultivation of these varieties resulted in alleviating losses due to CLCuD in Pakistan till the appearance of resistance-breaking CLCuKoV-Bur in 2001–2002. There is no single variety that is genetically resistant to the prevalent CLCuD complex. Breeding efforts are again underway in South Asia for the identification of resistant germplasm.

Currently, a number of efforts have been deployed to develop CLCuD-resistant plants by using genetic engineering approach. Such strategies are mainly based on interference with the begomovirus replication or movement within the cotton plants to reduce symptom expression in the host, which is the prime objective for plant protection without using pesticides.

Anti-sense RNA strategy has been conducted against CLCuD complex to inhibit the target mRNA expression in transgenic cotton (Amudha et al. 2011; Hashmi et al. 2011). Diversity between CLCuD-associated begomoviruses urges scientists for broad-spectrum resistance against all the prevailing viruses in the field. Successful results using RNA-interference (RNAi) technique against *African cassava mosaic virus* (Chellappan et al. 2004) and *Mungbean yellow mosaic virus* (Pooggin et al. 2003) have been reported. In recent times, RNAi-based construct targeting V2 gene of CLCuKoV-Bur limits virus replication in cotton plants (Yasmeen et al. 2016).

Site-specific genome editing using the clustered regularly interspaced short palindromic repeats/CRISPR-associated protein 9 (CRISPR/Cas9) system has recently emerged as a groundbreaking tool to introduce required traits in eukaryotic species, including plants. Scientists have used CRISPR/Cas9 system to engineer resistance in plants against geminiviruses. Recent studies demonstrated the efficient control of geminiviruses in plants using CRISPR/Cas9 system (Baltes et al. 2015; Ji et al. 2015; Ali et al. 2015, 2016). Targeting the conserved non-nucleotide sequence of CLCuKoV-Bur via CRISPR/Cas9 can be used for broad-spectrum geminivirus resistance (Ali et al. 2016).

Moreover, CRISPR/Cas9-based multiplexing technique to target CLCuD begomoviruses and associated satellites has been proposed (Iqbal et al. 2016).

Recently, the expression of insecticidal proteins and RNAi against whitefly has been demonstrated (Shukla et al. 2016; Raza et al. 2016; Javaid et al. 2016). Thus, the ability of begomoviruses and associated satellites to cause mixed infections could be obstructed by engineering dual *Begomovirus-Bemisia tabaci* resistance in plants (Zaidi et al. 2017).

13.4 Bacterial Diseases

13.4.1 Bacterial Blight of Cotton

13.4.1.1 Introduction

Bacterial blight of cotton is the most destructive disease that causes significant losses in yield in the rainy season (Delannoy et al. 2005). It was first reported during 1891 in Alabama in the United States (Atkinson 1891). In Pakistan, this disease appeared in Burewala near Multan. The world area cultivated with cotton is 33.4 M ha with a production of 121.4 M bales, while in Pakistan, it is cultivated on an area of ≈3.0 M ha during 2013–2014 with a production of 9.5 M bales. This disease causes 30% yield losses in diverse cotton-growing areas of the world (Ramapandu et al. 1979; Chidambaram and Kannan 1989). It was assumed that the yield damages generally reached between 10% and 30% in Asian countries, and up to 50% were registered in African countries (Bayles and Verhalen 2007). Approximately 37–40% of yield losses were observed in the Faisalabad area (Bhutta and Bhatti 1983; Khan et al. 1999).

13.4.1.2 Pathogen and Disease Spread

The bacterium can survive in the field on debris from previously harvested crops, and its initial inoculum was seed born (Mohan 1983). The bacterium sticks to the leaf surface, enters the leaf via the stomata or open wounds, and then produces symptoms in susceptible plants. It causes defoliation, swelling of the stem, black arm, breakage of the weakened trunk, and detachment of the bolls. The enhancement of disease reduces the quality of the cotton from lint staining with the consequent loss of yield (Verma 1986). Damage to the tissues of the stem and bolls has occurred, which gradually produce necrotic, angular, waxy, and marshy lesions on the leaf surface; these lesions are called bacterial blight, angular leaf spot, lesion of the black arm, and boll rot (Hillocks 1992). The first symptom appeared as tiny lesions impregnated with water (dark green flaccid lesions) and then spread to the bottom of young leaves (Verma 1986). As the disease progresses, diseased leaves defoliate early (Ridgway et al. 1984), and the disease spreads along the veins of the host plant known as the bacterial veins (Verma 1986). Bacterial ooze stained cotton fiber in diseased bolls (Brown and Ware 1958). Fruit positions become vulnerable to lesions

of the black arm due to the delicate nature of the infected stems (Innes 1983; Akello and Hillocks 2002). The bacterium has overwintered on infected seeds and on plant residues and could survive at least 22 months upon seed (Kirkpatrick and Rothrock 2001). Wind-driven rain and running water along with rain were main ways of spreading this bacterium (Brown and Ware 1958). The blowing sand was a general source of spreading pathogen. Dust along with storms first produced wounds in the plant tissues and then later caused infections in plants. Furthermore, seeds, machines, insects, and animals are also responsible for the transmission of this pathogen (Thaxton and Zik 2001).

13.4.1.3 Epidemiology

This disease was more severe in subhumid than semiarid regions with wind, rainfall ranging from 25.4 to 76.2 mm, and dust events in growing season (Kirkpatrick and Rothrock 2001). Disease infestation was higher in high-humidity areas that favored growth besides the spread of pathogen (Voloudakis et al. 2006). In natural environmental conditions, black cotton arm infections damage 35% of bolls.

13.4.1.4 Symptoms

Bacterial blight begins as small water soaked lesions (spots) on the leaves and seedlings and mature plants. The lesions progress into characteristic angular shapes when leaf veins limit bacterial movement (Fig. 13.7a). Unlike many other lesions on more or less circular cotton leaves, those associated with bacterial blight are more triangular or rectangular, although the shape may be more difficult to distinguish with leaf aging. Bacterial lesions may appear on the upper surface of the leaf; however, the wet or "greasy" appearance of the lesions is often observed more clearly on the underside of the leaf (Fig. 13.7b).

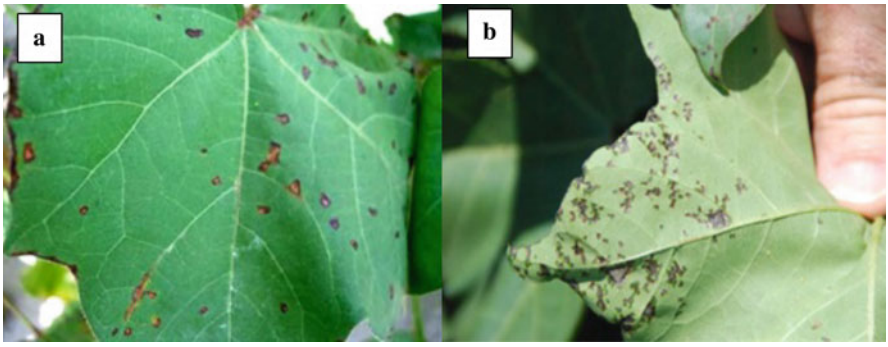

Fig. 13.7 Bacterial blight of cotton (**a**) and bottom of an infected leaf (**b**)

13.4.1.5 Management

- Resistant varieties against pathogen should be sown.
- Remove the infected stems and cotton debris present in the fields as soon as possible.
- Avoid applying agronomic practices in the field during wet conditions.
- Use growth regulators to activate the defense mechanism of the plant against pathogen.
- Use an alternate crop in the fields and apply irrigation timely.
- Manage seed sanitation to overcome the diseases by using acids, copper compounds, or chlorine derivatives and heat treatments.
- Disease forecasting models are very important to the farmers for disease prediction.

13.4.2 Bacterial Seed Rot and Boll Rot of Cotton

13.4.2.1 Introduction

The bacterial seed and boll rot of cotton represent a new threat for cotton growers. This disease was first discovered in South Carolina, United States (Hudson 2000), with a yield loss of 10–15%. In 2006, it was reported in the Chinese province of Xinjiang (Ren et al. 2008) causing 20% of stimulated performance losses. In Pakistan, firstly, it was reported in Faisalabad District (Ehetisham-ul-Haq et al. 2014). Usually, the disease is observed when the bolls open, and diseased locules are recognized as "hard lock" having dense lint with rotted seeds (Fig. 13.8a).

At the time of collection, partially open infected bolls fall sooner or are not harvested efficiently (Hudson 2000). Despite the reduction in cotton, even rot of

Fig. 13.8 Symptoms of boll rot of cotton (**a**) and hard lock (**b**)

seeds deteriorates the quality of the cotton fiber. The infected seeds do not support the development of cotton fibers. The quality of the cotton became poor when immature fiber was mixed with the healthy one, which eventually decreases the yield. The affected bolls do not open normally and remain attached to plants or fall to ground. Yield besides quality of cotton decreases when infected/immature fiber is mixed with the healthy one (Hudson 2000).

13.4.2.2 Pathogen and Disease Cycle

According to Stewart (2007), who made a comprehensive note on boll rot in cotton, the pathogen enters the plant by penetrating sucking insects. If the bolls are attacked in the first 10 days of their formation by the insect, they are unable to develop further due to wound, hormonal imbalance, or digestive juice of the insect. After flowering, the pathogen invades the wounds produced by insect feeding. Bolls remain susceptible to invasion of pathogens till the initial 3 weeks. After that, no damages occur when the bolls go even under secondary deposition, and insects cannot pierce into their stilettos. Symptoms did not appear on the external side of the carpel. Immature fiber and discoloration of brown seeds on diseased bolls are important symptoms.

13.4.2.3 Epidemiology

Boll rot of cotton is favored by wet and humid conditions and especially by high humidity. Rainfall that splashes soil up onto lower bolls enables infection. Immature bolls due to disease attack and lodged plants are major sources, and there is a high risk of disease spread in rainy season. In wet weather conditions, the chances of boll rot and hard lock development are high at boll opening time; high humid conditions delay boll opening and provide enough time for pathogen development.

13.4.2.4 Symptoms

Hudson (2000) describes the symptoms of the disease. According to him, discolored and semi-dead seed is observed when the infected immature boll is open or dissected. In particular, the immature locule has been attributed to the development of "hard locks" (Fig. 13.8b). In contrast to typically white and downy fiber, lint from infected locules (normally 4 locules/boll) is brown and dense. Simply, the disease pattern for seed besides rotting of SC cotton bolls includes (1) the asymptomatic external carpels of the infected bolls, (2) incomplete maturity of fibers and seeds, and (3) necrotic brown color of fibers besides the seed tissues. Mauney et al. (2003) describe structural differences and development between embryos dissected from typical cotton seeds and SC capsules that exhibit "semi-empty," an idiom adopted while awaiting identification of one or more random agents. They discovered that

normal fertilization took place in both situations; however, a discrepancy in successive growth was evident (Mauney et al. 2004).

13.4.2.5 Management

- Sowing of resistant or tolerant varieties, crop rotation, and seed treatment with chemicals and insecticides (for pests and disease vectors).
- Avoid very low plant populations that give rise to expose the soil that can be splashed on low bolls at the end of the season.
- Avoid dense vegetation.
- Evaluate the incidence before and after defoliation by counting all bolls on 10 plants of 10 designated sites in the field.
- Completely remove the crop residues.

13.5 Conclusion

In order to increase cotton yield, it is very much important for cotton growers to have knowledge about cotton diseases, causal organisms, and disease attack time along with their control measures through integrated disease management options including cultural, biological, and chemical means.

References

Abbas Q, Ahmad S (2018) Effect of different sowing times and cultivars on cotton fiber quality under stable cotton-wheat cropping system in southern Punjab, Pakistan. Pak J Life Soc Sci 16:77–84

Ahmad S, Abbas Q, Abbas G, Fatima Z, Atique-ur-Rehman NS, Younis H, Khan RJ, Nasim W, Habib ur Rehman M, Ahmad A, Rasul G, Khan MA, Hasanuzzaman M (2017) Quantification of climate warming and crop management impacts on cotton phenology. Plants 6(7):1–16

Ahmad S, Iqbal M, Muhammad T, Mehmood A, Ahmad S, Hasanuzzaman M (2018) Cotton productivity enhanced through transplanting and early sowing. Acta Sci Biol Sci 40:e34610

Ahmad S, Raza I (2014) Optimization of management practices to improve cotton fiber quality under irrigated arid environment. J Food Agric Environ 2(2):609–613

Ahmad S, Raza I, Ali H, Shahzad AN, Atiq-ur-Rehman SN (2014) Response of cotton crop to exogenous application of glycinebetaine under sufficient and scarce water conditions. Braz J Bot 37(4):407–415

Akello B, Hillocks RJ (2002) Distribution and races of Xanthomonas axonopodis pv. Malvacearum on cotton (Gossypium hirsutum) in Uganda. J Phytopathol 150:65–69

Akhtar S, Tahir MN, Baloch GR, Javaid S, Khan AQ, Amin I, Briddon RW, Mansoor S (2014) Regional changes in the sequence of cotton leaf curl Multan betasatellite. Viruses 6:2186–2203

Ali H, Abid SA, Ahmad S, Sarwar N, Arooj M, Mahmood A, Shahzad AN (2013a) Integrated weed management in cotton cultivated in the alternate-furrow planting system. J Food Agric Environ 11(3&4):1664–1669

Ali H, Abid SA, Ahmad S, Sarwar N, Arooj M, Mahmood A, Shahzad AN (2013b) Impact of integrated weed management on flat-sown cotton (*Gossypium hirsutum* L.). J Anim Plant Sci 23 (4):1185–1192

Ali H, Afzal MN, Ahmad F, Ahmad S, Akhtar M, Atif R (2011) Effect of sowing dates, plant spacing and nitrogen application on growth and productivity on cotton crop. Int J Sci Eng Res 2 (9):1–6

Ali H, Hameed RA, Ahmad S, Shahzad AN, Sarwar N (2014a) Efficacy of different techniques of nitrogen application on American cotton under semi-arid conditions. J Food Agric Environ 12 (1):157–160

Ali H, Hussain GS, Hussain S, Shahzad AN, Ahmad S, Javeed HMR, Sarwar N (2014b) Early sowing reduces cotton leaf curl virus occurrence and improves cotton productivity. Cercetări Agronomice în Moldova XLVII(4):71–81

Ali Z, Abulfaraj A, Idris A, Ali S, Tashkandi M, Mahfouz MM (2015) CRISPR/Cas9-mediated viral interference in plants. Genome Biol 16:238

Ali Z, Ali S, Tashkandi M, Zaidi SS, Mahfouz MM (2016) CRISPR/Cas9-mediated immunity to geminiviruses: differential interference and evasion. Sci Rep 6:26912

Allen RM, Tucker H, Nelson RA (1960) Leaf crumple disease of cotton in Arizona. Plant Dis Rep 44:246–250

Amin A, Nasim W, Mubeen M, Ahmad A, Nadeem M, Urich P, Fahad S, Ahmad S, Wajid A, Tabassum F, Hammad HM, Sultana SR, Anwar S, Baloch SK, Wahid A, Wilkerson CJ, Hoogenboom G (2018) Simulated CSM-CROPGRO-cotton yield under projected future climate by SimCLIM for southern Punjab, Pakistan. Agric Syst 167:213–222

Amin A, Nasim W, Mubeen M, Nadeem M, Ali L, Hammad HM, Sultana SR, Jabran K, Habib ur Rehman M, Ahmad S, Awais M, Rasool A, Fahad S, Saud S, Shah AN, Ihsan Z, Ali S, Bajwa AA, Hakeem KR, Ameen A, Amanullah, Rehman HU, Alghabar F, Jatoi GH, Akram M, Khan A, Islam F, Ata-Ul-Karim ST, Rehmani MIA, Hussain S, Razaq M, Fathi A (2017) Optimizing the phosphorus use in cotton by using CSM-CROPGRO-cotton model for semi-arid climate of Vehari-Punjab, Pakistan. Environ Sci Pollut Res 24(6):5811–5823

Amin I, Hussain K, Akbergenov R, Yadav JS, Qazi J, Mansoor S, Hohn T, Fauquet CM, Briddon RW (2011) Suppressors of RNA silencing encoded by the components of the cotton leaf curl begomovirus-betasatellite complex. Mol Plant-Microbe Interact 24:973–983

Amin I, Mansoor S, Amrao L, Hussain M, Irum S, Zafar Y, Bull SE, Briddon RW (2006) Mobilisation into cotton and spread of a recombinant cotton leaf curl disease satellite. Arch Virol 151:2055–2065

Amrao L, Akhter S, Tahir MN, Amin I, Briddon RW, Mansoor S (2010b) Cotton leaf curl disease in Sindh province of Pakistan is associated with recombinant begomovirus components. Virus Res 153:161–165

Amrao L, Amin I, Shahid SM, Briddon RW, Mansoor S (2010a) Cotton leaf curl disease in resistant cotton is associated with a single begomovirus that lacks an intact transcriptional activator protein. Virus Res 152:153–163

Amudha J, Balasubramani G, Malathi VG, Monga D, Kranthi KR (2011) Cotton leaf curl virus resistance transgenics with antisense coat protein gene (AV1). Curr Sci 101:300–307

Atkinson CF (1892) Some disease of cotton: 3. Frenching. Bull Alabama Agric Exp Station 41:19–29

Atkinson GF (1891) Black rust of cotton: a preliminary note. Bot Gaz 16:61–65

Baltes NJ, Hummel AW, Konecna E, Cegan R, Bruns AN, Bisaro DM, Voytas DF (2015) Conferring resistance to geminiviruses with the CRISPR-Cas prokaryotic immune system. Nat Plants 1:15145

Bashan Y (1984) Transmission of *Alternaria macrospora* in the cotton seeds. J Phytopathol 110:110–118

Bashan Y (1986) Phenols in cotton resistant seedling and susceptible to *Alternaria macrospora*. J Phytopathol 116:1–10

Bashi E, Rotem J, Hans P, Kranz J (1983) Influence of controlled environment and age on development of *Alternaria macrospora* and on shedding of leaves in cotton. Phytopathology 73:1145–1147

Bayles MB, Verhalen LM (2007) Bacterial blight reactions of sixty-one upland cotton cultivars. J Cotton Sci 11:40–51

Bell AA (1992) Biology and ecology of *Verticillium dahliae*. In: Lyda SD (ed) Comparative pathology of Sclerotial-forming plant pathogens: a Phymatotrichum Omnivorum symposium. Texas A & M University Press, College Station

Bhutta AR, Bhatti MAR (1983) Incidence of bacterial blight of cotton and reaction of different cultivars to Xanthomonas pv. Malvacearum. Pak Cottons 27(I):75–78

Briddon RW, Bull SE, Amin I, Idris AM, Mansoor S, Bedford ID, Dhawan P, Rishi N, Siwatch SS, Abdel-Salam AM, Brown JK, Zafar Y, Markham PG (2003) Diversity of DNA ß, a satellite molecule associated with some monopartite begomoviruses. Virology 312:106–121

Briddon RW, Bull SE, Amin I, Mansoor S, Bedford ID, Rishi N, Siwatch SS, Zafar Y, Abdel-Salam AM, Markham PG (2004) Diversity of DNA 1: a satellite-like molecule associated with monopartite begomovirus-DNA ß complexes. Virology 324:462–474

Briddon RW, Mansoor S, Bedford ID, Pinner MS, Saunders K, Stanley J, Zafar Y, Malik KA, Markham PG (2001) Identification of DNA components required for induction of cotton leaf curl disease. Virology 285:234–243

Briddon RW, Markham PG (2000) Cotton leaf curl virus disease. Virus Res 71:151–159

Briddon RW, Martin DP, Roumagnac P, Navas-Castillo J, Fiallo-Olivé E, Moriones E, Lett JM, Zerbini FM, Varsani A (2018) Alphasatellitidae: a new family with two subfamilies for the classification of geminivirus- and nanovirus-associated alphasatellites. Arch Virol 163:2587–2600

Briddon RW, Stanley J (2006) Subviral agents associated with plant single-stranded DNA viruses. Virology 344:198–210

Brown EA, McCarter SM (1976) Effect of seedling disease caused by the *Rhizoctonia solani* on subsequent growth and yield of cotton. Phytopathology 66:111–115

Brown HB, Ware JO (1958) Cotton, 3rd edn. McGraw-Hill Book Company, Inc, New York, p 411

Brown JK, Mihail JD, Nelson MR (1987) Effects of cotton leaf crumple virus on cotton inoculated at different growth stages. Plant Dis 71:699–703

Chellappan P, Masona MV, Vanitharani R, Taylor NJ, Fauquet CM (2004) Broad spectrum resistance to ssDNA viruses associated with transgene- induced gene silencing in cassava. Plant Mol Biol 56:601–611

Chidambaram P, Kannan A (1989) Grey mildew of cotton. Tech Bull Central Inst Cotton Res Regional Station Coimbatore, India

Chohan S, Abid M (2018) First report of *Fusarium incarnatum-equiseti* species complex associated with boll rot of cotton in Pakistan. Plant Dis 103:151

Cook OF (1924) Acromania or 'crazy top', a growth disorder of cotton. J Agric Res 28:803–828

Cook RJ (1981) Fusarium disease in the People's republic of China. In: Nelson PE, Toussoun TA, Cook RJ (eds) Fusarium diseases: biology and control. Pennsylvania State University Press, University Park and London, pp 53–55

Delannoy E, Lyon BR, Marmey P, Jalloul A, Daniel JF, Montillet JL, Essenberg M, Nicole M (2005) Resistance of cotton towards Xanthomonas axonopodis pv. Malvacearum. Annu Rev Phytopathol 43:63–82

Devay JE, Garber RH, Matherson D (1982) Role of Pythium species in the seedling disease complex of cotton in California. Plant Dis 66:151–154

Dickson RC, Johnson MM, Laird EF (1954) Leaf crumple, a virus disease of cotton. Phytopathology 44:479–480

Dzhamalov A (1973) Irrigation and Alternaria leaf spot of cotton. Zaschita Rastenii 12:48

Ehetisham-ul-Haq M, Khan MA, Javed MT, Atiq M, Rashid A (2014) Management of newly emerging bacterial seed and boll rot of cotton with antibiotics, homeopathic products and plant extracts. J Tropic Agric 92:196–206

Ehrlich J, Wolf FA (1983) Areolate mildew of cotton. Phytopathology 22:229–240
Ellis MB (1971) Dematiaceous Hyphomycetes. CAB International, Wallingford, p 608
Erdoğan O, Bölek Y, Göre ME (2016) Biological control of cotton seedling diseases by fluorescent *Pseudomonas spp*. Tar Bil Der 22(3):398–407
Evans G (1967) Verticillium wilt of cotton the situation in the Namoi Valley. Agric Gaz N S W 78:581–583
Fedorinchik NS (1964) Biological method of controlling plant diseases. Vses Naucb -Issled Inst Zashch Rast Tr 23:201–210
Ferro CG, Silva JP, Xavier CAD, Godinho MT, Lima ATM, Mar TB, Lau D, Zerbini FM (2017) The ever increasing diversity of begomoviruses infecting non-cultivated hosts: new species from Sida spp. and Leonurus sibiricus, plus two New World alphasatellites. Ann Appl Biol 170:204–218
Fiallo-Olivé E, Martínez-Zubiaur Y, Moriones E, Navas-Castillo J (2012) A novel class of DNA satellites associated with New World begomoviruses. Virology 426:1–6
Follen JC, Goebel S (1973) Rots of cotton capsules in irrigated crops in Côte d'Ivoire. Relationship with varietal characteristics, irrigation method and date of sowing. Cott Trop Fibers 28 (3):401–407
Globus GA, Muromtsev GS (1990) The use of *Gliocladium roseum* as antagonist for defense of cotton from phytopathogenic fungi. In: Proceedings of the fifth international verticillium symposium, Leningrad, USSR, p. 90
Gouws MA, Prinsloo GC, Van der Linde EJ (2001) First report of *Mycosphaerella areola*, teleomorph of *Ramulariopsis gossypii*, on cotton in South Africa. Afr Plant Prot 7(2):115–116
Hashmi JA, Zafar Y, Arshad M, Mansoor S, Asad S (2011) Engineering cotton (*Gossypium hirsutum* L.) for resistance to cotton leaf curl disease using viral truncated AC1 DNA sequences. Virus Genes 42:286–296
Hillocks RJ (1991) *Alternaria* leaf spot of cotton with special reference to Zimbabwe. Trop Pest Manag 37(2):124–128
Hillocks RJ (1992) Cotton diseases. Melksham UK, pp. 39–86
Hopkins JCE (1932) Some disease of cotton in southern Rhodesia. Empire Cotton Growing Rev 9:109–118
Hudson J (2000) Seed rot hits South Carolina cotton. Southeast Farm Press. Available via http://southeastfarmpress.com/mag/farming_seed_rot_hits/. Accessed June 2018
Huisman OC, Gerik JS (1989) Dynamic of colonization of plant roots by *Verticillium dahlia* and other fungi. In: Tjamos EC, Beckman CH (eds) Vascular wilt disease of plants, NATO ASI (series H: cell biology), vol 28. Springer, Berlin, Heidelberg, pp 1–17
Idris A, Al-Saleh M, Amer M, Abdalla O, Brown JK (2014) Introduction of cotton leaf curl Gezira virus into the United Arab Emirates. Plant Dis 98:1593
Idris AM, Brown JK (2002) Molecular analysis of cotton leaf curl virus-Sudan reveals an evolutionary history of recombination. Virus Genes 24:249–256
Innes NL (1983) Bacterial blight of cotton. Biol Rev 58:157–176
Iqbal Z, Sattar MN, Shafiq M (2016) CRISPR/Cas9: a tool to circumscribe cotton leaf curl disease. Front Plant Sci 7:475
Javaid S, Amin I, Jander G, Mukhtar Z, Saeed NA, Mansoor S (2016) A transgenic approach to control hemipteran insects by expressing insecticidal genes under phloem-specific promoters. Sci Rep 6:34706
Ji X, Zhang H, Zhang Y, Wang Y, Gao C (2015) Establishing a CRISPR-Cas-like immune system conferring DNA virus resistance in plants. Nat Plants 1:15144
Jones GH (1928) An Alternaria disease of the cotton plant. Ann Bot 42:935–947
Juárez M, Tovar R, Fiallo-Olivé E, Aranda MA, Gosálvez B, Castillo P, Moriones E, Navas-Castillo J (2014) First detection of tomato leaf curl New Delhi virus infecting zucchini in Spain. Plant Dis 98:857
Kamel M, Ibrahim AN, Kamal SA, El-fahl AM (1971) Spore germination of Alternaria leaf spot disease. U A R J Bot 14:245–254

Khan AJ, Akhtar S, Al-Shihi AA, Al-Hinai FM, Briddon RW (2012) Identification of cotton leaf curl Gezira virus in papaya in Oman. Plant Dis 96:1704

Khan MA, Khan HA, Ilyas MB, Rashid A (1999) Correlation of environmental conditions with bacterial blight disease on six commercially grown cotton cultivars in five districts of the Punjab. Pak J Agric Sci 36(1–2):1–5

Khan MB, Khaliq A, Ahmad S (2004) Performance of mashbean intercropped in cotton planted in different planting patterns. J Res (Sci) 15(2):191–197

King CJ, Barker HD (1934) An interval collar rot on cotton. Phytopathology 29:75

Kirkpatrick TL, Rothrock CS (2001) Compendium of cotton diseases, 2nd edn. APS Press, St. Paul, MN, p 77

Kirkpatrick TW (1931) Further studies on leaf-curl of cotton in the Sudan. Bull Entomol Res 22:323–363

Kirthi N, Priyadarshini CG, Sharma P, Maiya SP, Hemalatha V, Sivaraman P, Dhawan P, Rishi N, Savithri HS (2004) Genetic variability of begomoviruses associated with cotton leaf curl disease originating from India. Arch Virol 149:2047–2057

Klich MA (1986) Mycoflora of cotton seeds from southern USA. A three-year study of distribution and frequency. Mycologia 78:706–712

Leke WN, Khatabi B, Mignouna DB, Brown JK, Fondong VN (2016) Complete genome sequence of a new bipartite begomovirus infecting cotton in the Republic of Benin in West Africa. Arch Virol 161:2329–2333

Mansoor S, Amrao L, Amin I, Briddon RW, Malik KA, Zafar Y (2006) First report of cotton leaf curl disease in central and southern Sindh province in Pakistan. Plant Dis 90:826

Mansoor S, Briddon RW, Bull SE, Bedford ID, Bashir A, Hussain M, Saeed M, Zafar Y, Malik KA, Fauquet C, Markham PG (2003) Cotton leaf curl disease is associated with multiple monopartite begomoviruses supported by single DNA β. Arch Virol 148:1969–1986

Mansoor S, Khan SH, Bashir A, Saeed M, Zafar Y, Malik KA, Briddon RW, Stanley J, Markham PG (1999) Identification of a novel circular single-stranded DNA associated with cotton leaf curl disease in Pakistan. Virology 259:190–199

Mauney JR, Scottsdale AZ, Stewart JMcD (2003) Embryo development in bolls exhibiting the "Hollow Seed" syndrome in South Carolina. In: 2003 Beltwide Cotton Conference, Nashville, TN January 6–10, pp. 1653–1655

Mauney JR, Scottsdale AZ, Stewart JMcD, Fayetteville AR, Jones M (2004) Onset and progression of the "Hollow Seed" (seed rot) Malady of South Carolina. In: 2004 Beltwide Cotton Conference, San Antonio, TX, January 5–9, pp. 1967–1969

Mccarter SM, Roncardi RW, Crawford JL (1970) Microorganisms associated with *Aspergillus flavus* boll rot in Georgia. Plant Dis Resporter 54:586–590

McSpadden Gardener BB, Fravel DR (2002) Biological control of plant pathogens: research, commercialization, and application in the USA. Plant Health Prog. https://doi.org/10.1094/PHP-2002-0510-01-RV

Menlikiev NY (1962) Fusarium wilt of fine-staple cotton and a study of *Fusarium oxysporum* f. sp. *vasinfectum* strains as the causal agent of the disease in conditions of the Vakash Valley. Rev Appl Mycol 43:3381

Mnari-Hattab M, Zammouri S, Belkadhi MS, Doña DB, ben Nahia E, Hajlaoui MR (2015) First report of tomato leaf curl New Delhi virus infecting cucurbits in Tunisia. New Dis Rep 31:21

Mohan SK (1983) Seed transmission and epidemiology of Xanthamonas campestris pv. Malvacearum. Seed Sci Technol 11:569–571

Nawaz-ul-Rehman MS, Briddon RW, Fauquet CM (2012) A melting pot of Old World begomoviruses and their satellites infecting a collection of Gossypium species in Pakistan. PLoS One 7:e40050

Nawaz-ul-Rehman MS, Nahid N, Mansoor S, Briddon RW, Fauquet CM (2010) Post-transcriptional gene silencing suppressor activity of two non-pathogenic alphasatellites associated with a begomovirus. Virology 405:300–308

Nemli T, Sayar I (2002) Aydin Söke region the prevalence of cotton-depleting disease the factors and prevention of the possibility of investigation. Scientific and Technical Research Council of Turkey the TARP-2535 Ankara, p. 57

Panno S, Iacono G, Davino M, Marchione S, Zappardo V, Bella P, Tomassoli L, Accotto GP, Davino S (2016) First report of tomato leaf curl New Delhi virus affecting zucchini squash in an important horticultural area of southern Italy. New Dis Rep 33:6

Paprotka T, Metzler V, Jeske H (2010) The first DNA 1-like alpha satellites in association with New World begomoviruses in natural infections. Virology 404:148–157

Paulwetter RC (1918) The Alternaria leaf spot of cotton. Phytopathology 8:98–115

Pleban S, Ingel F, Chet I (1995) Control of *R. solani* and *S. rolfsii* in the greenhouse using Endophytic bacillus spp. Eur J Plant Pathol 101(6):665–672

Pooggin M, Shivaprasad PV, Veluthambi K, Hohn T (2003) RNAi targeting of DNA virus in plants. Nat Biotechnol 21:131–132

Qazi J, Amin I, Mansoor S, Iqbal MJ, Briddon RW (2007) Contribution of the satellite encoded gene betaC1 to cotton leaf curl disease symptoms. Virus Res 128:135–139

Rahman MH, Ahmad A, Wang X, Wajid A, Nasim W, Hussain M, Ahmad B, Ahmad I, Ali Z, Ishaque W, Awais M, Shelia V, Ahmad S, Fahad S, Alam M, Ullah H, Hoogenboom G (2018) Multi-model projections of future climate and climate change impacts uncertainty assessment for cotton production in Pakistan. Agric For Meteorol 253-254:94–113

Rajagopalan PA, Naik A, Katturi P, Kurulekar M, Kankanallu RS, Anandalakshmi R (2012) Dominance of resistance-breaking cotton leaf curl Burewala virus (CLCuBuV) in northwestern India. Arch Virol 157:855–868

Ramapandu S, Sitaramaiah K, Subbarao K, Prasada Rao MP (1979) Screening of cotton germplasm against bacterial blight caused by Xanthomonas axonopodis pv. Malvacearum. Indian Phytopathol 32:486–487

Rane MS, Patel MK (1956) Diseases of cotton in Bombay 1. Alternaria leaf spot. Indian Phytopathol 9:106–113

Raza A, Malik HJ, Shafiq M, Amin I, Scheffler JA, Scheffler BE, Mansoor S (2016) RNA interference based approach to down regulate osmoregulators of whitefly (*Bemisia tabaci*): potential technology for the control of whitefly. PLoS One 11:e0153883

Reddy RVC, Muniyappa V, Colvin J, Seal S (2005) A new begomovirus isolated from *Gossypium barbadense* in southern India. Plant Pathol 54:570–570

Ren YZ, Liu YQ, Ding SL, Li GY, Zhang H (2008) First report of boll rot of cotton caused by *Pantoea agglomerans* in China. Plant Dis 92:1364–1364

Ridgway RL, Bell AA, Veech JA, Chandler JM (1984) Cotton protection practices in the USA and the world, pp. 265–365. In: Kohel RJ, Lewis CF (eds) Cotton. American Society of Agronomy/ Crop Science Society of America/Soil Science Society of America, Madison, pp 226–365

Romay G, Chirinos D, Geraud-Pouey F, Desbiez C (2010) Association of an atypical alphasatellite with a bipartite New World begomovirus. Arch Virol 155:1843–1847

Ruiz ML, Simón A, Velasco L, García MC, Janssen D (2015) First report of tomato leaf curl New Delhi virus infecting tomato in Spain. Plant Dis 99:894

Saeed M, Zafar Y, Randles JW, Rezaian MA (2007) A monopartite begomovirus-associated DNA ß satellite substitutes for the DNA B of a bipartite begomovirus to permit systemic infection. J Gen Virol 88:2881–2889

Sangeetha KD, Ashtaputre SA (2015) Morphological and cultural variability in isolates of Alternaria sp. causing leaf blight of cotton. Karnataka J Agric Sci 28(2):214–219

Sattar MN, Iqbal Z, Tahir MN, Ullah S (2017) The prediction of a new CLCuD epidemic in the Old World. Front Microbiol 8:631

Sattar MN, Kvarnheden A, Saeed M, Briddon RW (2013) Cotton leaf curl disease–an emerging threat to cotton production worldwide. J Gen Virol 94:695–710

Saunders K, Stanley J (1999) A nanovirus-like component associated with yellow vein disease of Ageratum conyzoides: evidence for interfamilial recombination between plant DNA viruses. Virology 264:142–152

Schnathorst WC (1981) Life cycle and epidemiology of verticillium. In: Mace ME, Bell AA, Beckman CH (eds) Fungal wilt disease of plants. Academic Press, New York, pp 81–111

Sharma YR, Sandhu BS (1985) A new fungus associated with boll rot of *G. arboreum* cotton. Curr Sci 54:936

Shen CY (1985) Integrated management of *Fusarium* and *Verticillium* wilt of cotton in China. Crop Prot 4:337–345

Shukla AK, Upadhyay SK, Mishra M, Saurabh S, Singh R, Singh H, Thakur N, Rai P, Pandey P, Hans AL, Srivastava S, Rajapure V, Yadav SK, Singh MK, Kumar J, Chandrashekar K, Verma PC, Singh AP, Nair KN, Bhadauria S, Wahajuddin M, Singh S, Sharma S, Omkar URS, Ranade SA, Tuli PK, Singh PK (2016) Expression of an insecticidal fern protein in cotton protects against whitefly. Nat Biotechnol 34:1046–1051

Snyder WC, Hansen HN (1940) The species concept in Fusarium. Am J Bot 27:64–67

Stanley J (1983) Infectivity of the cloned geminivirus genome requires sequences from both DNAs. Nature 305:643–645

Stewart J (2007) Boll development and seed rot. In: World Cotton Research Conference-4, Lubbock, TX, USA, 10–14 September, 2007. International Cotton Advisory Committee (ICAC)

Tahir MN, Amin I, Briddon RW, Mansoor S (2011) The merging of two dynasties - identification of an African cotton leaf curl disease-associated begomovirus with cotton in Pakistan. PLoS One 6: e20366

Tariq M, Afzal MN, Muhammad D, Ahmad S, Shahzad AN, Kiran A, Wakeel A (2018) Relationship of tissue potassium content with yield and fiber quality components of Bt cotton as influenced by potassium application methods. Field Crop Res 229:37–43

Tariq M, Yasmeen A, Ahmad S, Hussain N, Afzal MN, Hasanuzzaman M (2017) Shedding of fruiting structures in cotton: factors, compensation and prevention. Trop Subtrop Agroecosyst 20(2):251–262

Thaxton PM, Zik KME (2001) Bacterial blight. In: Kirkpatrick TL, Rothrock CS (eds) Compendium of cotton diseases, 2nd edn. American Phytopathological Society, St. Paul, MN, pp 34–35

Usman M, Ahmad A, Ahmad S, Irshad M, Khaliq T, Wajid A, Hussain K, Nasim W, Chattha TM, Trethowan R, Hoogenboom G (2009) Development and application of crop water stress index for scheduling irrigation in cotton (*Gossypium hirsutum* L.) under semiarid environment. J Food Agric Environ 7(3&4):386–391

van Schaik PH, Erwin DC, Garber MJ (1962) Effects of time on symptom expression of the leaf-crumple virus on yield and quality of fiber cotton. Crop Sci 2:275–277

Venkatesh I, Darvin G (2016) An overview on cotton Alternaria leaf spot and its management. Int J Appl Bio Pharm Tech 7(2):135

Verma JP (1986) Bacterial blight of cotton. CRC Press, Boca Raton, FL, pp 278–279

Voloudakis AE, Marmey P, Delannoy E, Jalloul A, Martinez C, Nicole M (2006) Molecular cloning and characterization of (Gossypium hirsutum) superoxide dismutase genes during cotton Xanthomonas axonopodis pv. Malvacearum interaction. Physiol Mol Plant Pathol 68:119–127

Waghunde RR, Patel UT, Vahunia B (2018) Morphological and cultural variability of *Alternaria Macrospora* causing leaf blight in cotton. J Pharmacogn Phytochem 7(3):3096–3099

Wang C, Wang D, Zhou Q (2004) Colonization and persistence of a plant growth-promoting bacterium *Pseudomonas fluorescens* strain CS85 on roots of cotton seedlings. Can J Microbiol 50(7):475–481

Yasmeen A, Kiani S, Butt A, Rao AQ, Akram F, Ahmad A, Nasir IA, Husnain T, Mansoor S, Amin I, Aftab S, Zubair M, Tahir MN, Akhtar S, Scheffler J, Scheffler B (2016) Amplicon-based RNA interference targeting V2 gene of cotton leaf curl Kokhran virus-Burewala strain can provide resistance in transgenic cotton plants. Mol Biotechnol 58:807–820

Zaidi SS, Briddon RW, Mansoor S (2017) Engineering dual begomovirus-Bemisia tabaci resistance in plants. Trends Plant Sci 22:6–8

Zaidi SSA, Iqbal Z, Amin I, Mansoor S (2015) First report of tomato leaf curl Gujarat virus, a bipartite begomovirus on cotton showing leaf curl symptoms in Pakistan. Plant Dis 99:1655

Zaidi SSA, Shafiq M, Amin I, Scheffler BE, Scheffler JA, Briddon RW, Mansoor S (2016) Frequent occurrence of tomato leaf curl New Delhi virus in cotton leaf curl disease affected cotton in Pakistan. PLoS One 11:e0155520

Zammouri S, Zaagueri T, Eddouzi J, Belkhadhi MS, Hajlaoui MR, Mnari-Hattab M (2017) First report of tomato leaf curl New Delhi virus on tomato crop in Tunisia. J Plant Pathol 99:799–818

Zhou X (2013) Advances in understanding begomovirus satellites. Annu Rev Phytopathol 51:357–381

Zhou X, Liu Y, Robinson DJ, Harrison BD (1998) Four DNA-A variants among Pakistani isolates of cotton leaf curl virus and their affinities to DNA-A of geminivirus isolates from okra. J Gen Virol 79:915–923