

# Chapter 10

## The Vital Foliar Diseases of *Cicer arietinum* L. (Chickpea): Science, Epidemiology, and Management



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### 10.1 Introduction

Chickpea production in the world has increased over the past two decades, ranking third after dry bean (*Phaseolus vulgaris* L.) and field pea (*Pisum sativum* L.) (Hirdyani 2014). It dominates other legumes in the international market, and its trade traffic is more than 8 billion dollars annually (Stagnari et al. 2017). This crop contributes to agricultural sustainability through N<sub>2</sub> fixation and allows agricultural production by diversification. India is also the largest chickpea-producing country with 9.33 million tonnes production in 9.48 million ha of cultivated areas (Pande et al. 2005). The productivity in India is lesser in comparison to other chickpea-producing countries because of the biotic and abiotic stresses and also due to fungal foliar diseases. Chickpea is grown commercially in soils having residual moisture and with or without minimum irrigation in RRFL (rainfed rice fallow lands) (Pande et al. 2012). The optimal conditions needed for growth and development of chickpea include temperature around 18–26 °C during the night and 21–29 °C during the day and a total of 560–660 mm of annual rainfall. Chickpea is broadly classified into two types: desi type and kabuli type. Desi-type chickpea has seeds that are small and have sharp angular edges, and the color of the seed varies from black to almost cream color or yellow. The desi-type flowers are pink in color and produce about 80–90% of the chickpea throughout the world. *Dal* (the splits) and *besan* (flour) are made up of desi type (Purushothaman et al. 2014; Toker et al. 2007). The kabuli type has large, rounded seeds that are head-shaped having cream beige seed color and white seed coats (Pande et al. 2012). Production of chickpea is constrained by foliar diseases as well as insect pests. In general, fungal foliar diseases like *Ascochyta* blight, *Botrytis* gray mold, etc. are spread in northern, northern-western, and eastern India (Bretag et al. 2008).

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## 10.2 History and Origin: Chickpea

Chickpea is a historical crop of the modern age; it was cultivated since 9500 years ago in the Fertile Crescent, through Turkey to Iran (Harlan 1971). Chickpea is cultivated in association with other crops like wheat, pea, barley, lentils, flax, and vetch as a part of agricultural evolution in the Fertile Crescent (Abbo et al. 2003a). The large area spreading over Israel to Western Iran, from southeast Turkey to Jordan and Iraq, ascertained a balanced collection of basic needs like carbohydrate, protein, oil, and fiber (Diamond 1997). Wild plants were cultivated primarily in this region and were observed archaeologically, and information from 7500 BC and recent years remain feasible (Fuller and Harvey 2006). Chickpea is used as a food in the eighth millennium BC (Tanno and Willcox 2006). Even though, archeological records in chickpea are scarce because the seed is almost crushed down in the carbonization of seed Neolithic chickpea supported the distribution which restricted during the Fertile Crescent, especially at Anatolia and the eastern Mediterranean (Van der Maesen 1972). Later, the Neolithic Period, chickpea expanded westward to modern Greece. During the Bronze Age, chickpea has been spread widely to the west of Crete, south of Upper Egypt, eastward through recent Iraq toward the Indian subcontinent, where the other was found in Harappan community in Pakistan and various sites in Maharashtra and Uttar Pradesh (Colledge et al. 2004). During the Iron Age, chickpea was spread in South and West Asia and in Ethiopia. The crop expanded with the group of originator crops from the Fertile Crescent toward West Central Asia and also Europe from 5500 BC (Moreno and Cubero 1978). In the sixteenth century AD, chickpea was produced by the Spanish region and Portugal; and in the eighteenth century, kabuli type spread in the Indian region from the Mediterranean region (van der Maessen 1972). Indian immigrants in the later nineteenth century imported the desi chickpea to Kenya (van der Maessen 1972). At present in the USA, Canada, and Australia, chickpea breeding programs have started. The related species of chickpea is *Cicer reticulatum*, which is the only related species in the gene pool and spread in southeast Turkey. Numerous additional *Cicer* species of almanac and perennial are hereditarily found in the genetic makeup as per AFLP (amplified fragment length polymorphism) analysis (Kumar et al. 2016). The actual difference among the wild relatives and the native chickpea is the loss due to vernalization which is a polygenetic attribute (Abbo et al. 2003a). The most widespread production of chickpea occurs in North America and the Middle East and un-moistured winter regions of India (Abbo et al. 2003b).

## 10.3 Center of the Diversity of Chickpea

The spread of old and wild type occurs in the main three areas from 8° N to 56° N latitude and 8° W to 85° E longitude especially Ethiopia, Crete, Western Mediterranean, Greece, the Caucasus Iran, Asia Minor, Central Asia, Himalayan

region, and Afghanistan. Domestic chickpea is presently highly nurtured in Australia, southern South America, African Mediterranean regions, Ethiopia, the European Mediterranean region, southern Asia toward Iran to Myanmar, and the Middle East encompassing Turkey, Iraq, and Israel (Van der Maesen 1972). International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), India, is the largest GenBank for chickpea, which consists of 17,250 accessions and 6390 of Indian diversity, followed by 4850 of Iran, 930 of Ethiopia, 700 of Afghanistan, 480 of Pakistan, 470 of Turkey, 390 of Mexico, 220 of Syria, 139 of Chile, 133 of Soviet Union, and many additional countries from Northern Africa, Southern Europe, East Africa, North America, and South America (Abbo et al. 2003a). International center for Agricultural Research in the Dry Areas (ICARDA), Aleppo, Syria, for chickpea of kabuli type, the genebank consists of 12,070 accessions from 1780 of Iran; 970 of Turkey; 410 of India; 340 of Chile; 300 of Uzbekistan; 280 of Spain; 270 of Tunisia; 230 of Morocco; 210 of Bulgaria; 170 of Portugal; 160 of Russian Federation; 160 of Mexico; 150 of Jordan; 120 of the USA; 110 of Bangladesh, Tajikistan, and Azerbaijan; and some further provinces lesser (100) like Italy, Ethiopia, Palestine, South America, Algeria, North Europe, tropical Africa, and Egypt (Diamond 1997).

## 10.4 Chickpea Production

Chickpea is also known by different local names: hamas (in the Arab world), zimbra (in Ethiopia), nohud or lablabi (in Turkey), chana (in India), and garbanzo (in Latin America). Chickpea crop production spread from 6.6 million tonnes in the year 1998–1999 to 9.5 million tonnes by 2000–2001 (Moreno and Cubero 1978).

## 10.5 Ecology of Chickpea

The chickpea evolution is different from the other wild type of the West Asian Neolithic crops, and it shows a part in regulating the crop habitat. The chickpea habitat can be characterized easily with the advanced high-resolution information of the climate and geographical information system (GIS) software freely present in the public databases (Hijmans et al. 2001). The areas like Egyptian Nile Valley, Iraq, Pakistan, and central Iran retain the lowermost annual report precipitation in cold winter and mix the midsummer heats with adequate winter temperatures (Rousta et al. 2018).

- *Temperature and altitude of chickpea*

Altitude and rainfall variableness remain less in Europe than in West Asia and North. South Asia's yearly temperatures remain higher, earlier to the beginning of the monsoon. It was observed minor dissimilarity between the mean

temperature of the warmest quarter in between northern and southern halves of the subcontinental distribution of chickpea which ranges from 30.8 to 31.9 °C, and the mean winter temperatures vary from the North (16.8 °C) to the South (22.1 °C). In Central Asia chickpea is cultivated in areas with a high series of temperature variation and rainfall unevenness, which leads to a hasty change in the altitude region (Bhat et al. 2017).

- *Summer-dominant rainfall region environments of chickpea*

Chickpea-growing regions like South Asia, Peru, and East Africa are summer-dominant rainfall environments (Ahmed et al. 2016). There is a strong decrease in rainfall in the Indian subcontinent from the southeast to northwest; Madhya Pradesh, a central state with higher rainfall and summer-dominant rainfall region in the subcontinent, produces 50% of the chickpea (Bhat et al. 2017). Chickpea growing region in Mexico is arid from 119 to 284 mm/year and is a summer-dominant rainfall region, where summer and winter rainfall proportion increases from 36% to 43% and 33% to 46%, respectively (Nicholson 2014).

## **10.6 Adaptation of Chickpea: Stresses, Cropping Systems, and Habitats**

### **10.6.1 Stresses in Chickpea**

Stresses in chickpea can be classified as biotic stress and abiotic stress based on coarse agro-climate divisions. In the Mediterranean rainfall region and summer-dominant rainfall region, drought is dangerous and is intensified by heat pressure (Saxena et al. 1996). In India, for most summer-dominant rainfall region, *Fusarium* wilt-root rot complex, *Ascochyta* blight, and *Botrytis* gray mold are the biotic stresses which contribute to disease distribution and are estimated to cause 10% of the annual yield loss (Singh 1990).

### **10.6.2 Cropping Methods of Chickpea**

Seeding methods for chickpea vary in various environments. The highest range of seeding approaches was found to be in the Mediterranean region, because of the comparative strength of the biotic and abiotic stress (Rasool et al. 2015).

#### **Maturation of Chickpea in Late Spring or Early Summer of the Autumn-Sown Rainy Season**

It is a regular chickpea cultivation system for regions with relatively warm winter and less biotic stress or pressure because it works on intra-seasonal rainfall and decreases the disclosure to drought. In West Asia and North Africa (WANA), especially in warmer regions of Iran and in the Nile Valley, both countries use this

system to grow chickpea with supplementary irrigation in the areas to decrease drought stress (Saccardo and Calcagno 1990). At present-day winter, sowing and drill irrigation have been used by approximately 90% of Israeli farmers. Australia is biotic stress-free until the mid-1990s, and later production of chickpea declined, but it is recovered by the release of resistant variety and by adopting good management practices (Hughes et al. 1987). In Mediterranean Australia, winter temperature is moderate, and autumn sowing of chickpea is exposed to suboptimal temperature on flowering and can delay pod set by 30 days more. Prompt flowering expands yield constancy and attains alteration to water deficiency but expands the threat of encompassing less temperature (Saccardo and Calcagno 1990).

### **Spring-Sown Chickpea in Post-Rainy Season Maturation in Summer**

It is a regular chickpea cultivation system of Mediterranean climates in WANA and minimizes the risk factor of winter frosts, disease stresses, and the farmers to take a decision for planting based on soil moisture profile (Hamwiah and Imtiaz 2015). In Tunisia, winter stress is lowered geographically, as the crops are grown in low elevation of <600 m deep clay loams in areas of semiarid, avoids heavy rainfall of >1000 mm/year, along with areas and frost-prone areas. Chickpea is sown in the middle of May to escape high temperatures which will occur post-October in north-eastern Australia (Saccardo and Calcagno 1990). Cultivation can be done in dry-sown if sufficient soil moisture is present, or as farmers delay for opening rain, which leads postponement of sowing till August in a few of the regions (Hamwiah and Imtiaz 2015). The chickpea crop can tolerate heat stress at phases of maturation, normally in November (27–30 °C). Although rainwater tends to rise from October in many regions, chickpea crop cannot enter the similar terminal drought stress in South Asian environments (Kumar 2017).

### **10.6.3 Chickpea Habitat Range**

Chickpea is grown in diverse habitat which consists of altered climate, cropping system, and stress. Chickpea is essentially separated into definite ecotypes, showing local selection pressures in the region of millennia. From the past 30 years, there has been an evaluation of germplasm ranging from characterization and resistance screening by many international centers and physiological studies based on accession number (Upadhyaya 2003). Chickpea physiological and habitat understanding is a must, and major stresses can be avoided by combinations of sowing strategies and appropriate phenology (Berger et al. 2006). Chickpea phenology is increased by drought stress as it decreases the thermal time for flowering, maturity, and pod fill; it also lowers the water potential, photosynthesis pod number, and yield (Berger et al. 2006). Chickpea is also having dehydration postponement and consistency of tolerance, like deep rooting, extraction of high soil water, and adjustment of osmosis (Summerfield et al. 1985). Chickpea is highly tolerable to heat stress than the other cold grain legumes like field pea, lentils, and faba bean, and it also absorbs less

incident radiation approximately <50% photosynthetically available radiation (PAR) than other seasonal legumes. Kabuli-type characters were demonstrated in East Asia, Europe, and the Mediterranean, while the desi character was common in Africa and also in Southeast Asia (El-Amier et al. 2015). The vegetative phase, when extended under long-season conditions, increased biomass accumulation and reproduction and delayed flowering till the temperature becomes sufficiently warm to aid the pod set. The difference among provinces has been detected in the assessment of Ethiopian drought-tolerant germplasm (Berger et al. 2006).

## 10.7 Uses, Consumption, and Utilization

From the beginning of agricultural time, legume crops have several uses depending on the utilization of different plant parts. Dry or green seeds are applicable for animal feed, fodder, and organic manure. It is also used as a whole and mixed with other cereals (Kumar 2017). Legumes are eaten as a main course in the dish either singly or with meat, fish, and snacks, green or dried. One of the examples of legume is chickpea. Chickpea can be packaged, ice-covered, canned, and precooked. It is a source of oil which is used in baking protein-rich cake (Venn and Mann 2004). It contains protein and carbohydrates and has nutritive value. It can also fix nitrogen from the atmosphere which is secreted into the soil. The cultivation is decreasing in recent years due to the cause of their marginalization of a late entry in the market (Rimal et al. 2015). The legume crop is the essential food of the vegetarian dietary system, so it is directly linked with Indian civilization (Agbola et al. 2002). The pulses or legumes can be dried properly and conserved to consume throughout the year. Consumption per capita of pulses of 80 g/day is advised by the World Health Organization (WHO) and consumption of 47 g/day by the Indian Council of Medical Research (ICMR) (Misra et al. 2011). Consumption in India is less than 30–34 g/day/person because of the unavailability and price rise of pulses (Akinjayeju and Ajayi 2011).

## 10.8 Nutritional Value of Chickpea

Nutrition through food is necessary for human life. Nutrition provides energy, macronutrients, micronutrients, etc. for growth, tissue maintenance, regulation of metabolites, and physiological functions. Chickpea in many countries is a staple food and plays an important element in the diet of vegetarians around the world. Chickpea is a valuable source of minerals, vitamins, energy, fibers, and also health-beneficial phytochemicals (Brenes et al. 2008).

### Nutritional Composition

The nutritional composition can vary due to the environment, climate, soil biology, soil nutrient, stress factors, and agronomic factors (McCleary 2003).

- *Energy*  
Energy is defined as gross energy (MJ/kg) or as a caloric value (kcal/100 g). Chickpea has an energy value of 14–18 MJ/kg or 334–437 kcal/100 g for desi types, and for kabuli type, it is 15–19 MJ/kg or 357–446 kcal/100 g. It showed that the kabuli type has higher energy than the desi type due to the presence of the seed coat component (Perttilä et al. 2005).
- *Protein and amino acid*  
The protein concentration of desi type ranges from 16.7% to 30.6% and for kabuli type 12.6% to 29.0%. Chickpea is used for the treatment of malnutrition and kwashiorkor in children because of its high protein content (Greenfield and Southgate 2003). The body is also provided with amino acids to synthesize new proteins for repairing and replacing damaged tissue and to synthesize enzymes, hormones, and growth factors. Chickpea has a high amount of sulfur amino acid than the lysine (Sotelo et al. 1987).
- *Lipid and fatty acid*  
Chickpea consists of 2.9–7.4% lipid content for desi and 3.4–8.8% content for kabuli (Jukanti et al. 2012). The total lipid content consists of 62–67% of polyunsaturated, 19–26% of monounsaturated, and 12–14% of saturated fatty acids. Essential fatty acids like linolenic and linoleic acid are supplied through the diet (Trumbo et al. 2002).
- *Carbohydrates*  
Carbohydrates are the most important component in chickpea, having 54–71% for desi type and 54–71% for kabuli type (Greenfield and Southgate 2003). The key types of carbohydrates present are oligosaccharides (like raffinose (2.2%), stachyose (6.5%), ciceritol (3.1%), and verbascose (0.4%)), polysaccharides (like starch (30–57%)), monosaccharides (like glucose (0.7%), ribose (0.1%), fructose (0.25%), and galactose (0.05%)), and disaccharides (like maltose (0.6%) and sucrose (1–2%)) (Joint FAO/WHO 1998).
- *Minerals*  
Chickpea plants absorb the minerals (like, B, Fe, Mn, Zn, Cu, Ni, Ca, Mg, K, P, S, Cl, and Mo) from the soil and transfer it to the seed performs a metabolic activity like photosynthesis, respiration, chlorophyll synthesis, and cell division (Sujak et al. 2006).
- *Vitamins*  
Chickpea comprises of a high source of water-soluble vitamins like the B-complex vitamins (B1, B2, B3, and pantothenic acid) and vitamin C and lipid-soluble vitamins like vitamin A (provitamin A carotenoids), vitamin E (tocopherols and tocotrienols), and vitamin K (Who and Consultation 2003).

## 10.9 Foliar Fungal Disease

Chickpea is the most essential cool season pulse crop grown in dry regions. The chickpea plant agonizes commencing fungal foliar diseases that distress the growth stage of chickpea. The pathogens that infect the plant include bacteria, nematodes,

viruses, fungi, and mycoplasma, which lead to severe crop yield loss. Among this, fungi are the most threatening group that affect the roots, stems, flowers, leaves, and pods of chickpea (Nene et al. 2012).

### **10.9.1 *Ascochyta Blight (Ascochyta rabiei (Pass.) Labr.)***

#### **Distribution**

*Ascochyta blight* (AB) is a viral disease found in West Asia, Southern Europe, and Northern Africa. In Pakistan, it occurs in February and March and disease will develop accordingly; and in Northern India, it happens when the crop canopy is very dense. In West Asia, Northern Africa, and Southern Europe, such situations usually occur from March to May. In winter chickpea is sown toward the Mediterranean region, and the blight symptoms are found when the climate is wet and warm in November and December. The disease has been found to develop among 35 countries along 6 continents and presently seen in Canada and Australia; it can expand swiftly to different areas of chickpea production (Nene et al. 2012).

#### **Economic Importance**

The fungal foliar disease causes crop yield loss and quality loss of up to 100% (Nene et al. 2012).

#### **Epidemiology**

*Ascochyta blight* occurs through seed transmission of *Ascochyta rabiei*. Airborne spores of *A. rabiei* are found to play a major vital role in epidemics of the disease (Kaiser et al. 2000). *A. rabiei* either lives on the seed or inside it or can be found in the plant debris of diseased left over in the fields as a mycelium and pycnidia or at its teleomorph stages and can serve as an agent of the disease (Santra et al. 2001). The secondary spread of this fungus occurs through conidia and ascospores. Development of teleomorph, the stage of the sexual reproductive, appears due to the mating of compatible new types in new areas spread through the air (Guarro et al. 1999). The teleomorph stage assists the pathogen in a longer duration of survival in its host, though it has never been seen in the newly infected host. In many regions, though, pseudothecia are found in infected plant wastes. Seed transmission in a field causes pathogen distribution randomly, giving the cause of many initial infections. Wet, cloudy, and cool weather is favorable for the disease development. In a cool climatic condition, the density of asci and ascospore production per pseudothecium are much higher than the warm condition (Daehler et al. 2004). Ascospores are also necessary for dispersal of the pathogen to long distances. The ascospore gets discharged to the air from pseudothecium during the wet condition. Production of ascospore on largely infected crop residues can reach up to  $1.5 \times 10^4$  ascospores/mm<sup>2</sup> on the tissue surface (Manstretta and Rossi 2015). The productions of conidia per pycnidium are much more in cool regions compared to warmed counterparts. Strong wind and rain can scatter conidia grown on diseased plant parts, provided if conidia are present in water droplets or rain splash. Relative humidity compared to temperature plays a more vital role as a critical factor in the determination of the



development of pseudothecia and pycnidia on crop debris (Vidal et al. 2017). The disease best develops at low temperature, optimum being at 20 °C. The moist environment also acts as a vital factor to produce severe infection. Dry periods after immediate inoculation may sometime induce disease severity though dry period exceeding 12 hours after 6 hours of wet treatment may reduce the disease development. In tropical countries, *A. rabiei* by crop debris get influenced by the low rainfall and high temperature during the out of season summer months, which is detrimental for the survival of the pathogen *A. rabiei*. Impacts of light in in vitro conditions reportedly have insignificant influence on pseudothecial development and discharge of ascospores (Sehulster and Chinn 2003).

### Symptoms

AB is typically seen during the flowering and podding stage as patches (Gurjar et al. 2012). The disease can be observed at an early stage of growth. When the pathogen is seed-borne, the germination time is favorable for the development of disease at the stem base with dark brown lesions (Lammerts van Bueren et al. 2004). The seedlings which are affected can be collapsed and die due to the formation of pycnidia. The disease spread from the seedling to the flowering and podding which results in patches of diseased plants. The disease appears in the form of spots of small water-soaked in the young leaves in the branches when the origin is airborne and conidia or ascospores (Nene et al. 2012). These spots enlarge and integrate which blights the leaves and the buds that lead to disease development under favorable conditions and also pycnidia presence on blighted leaves and buds. Because of susceptible cultivation, the necrosis spread through the buds, which kill the plant. In severe infection of the foliar disease, the entire plant gets dry and falls off. If the temperature is hot, the condition is unfavorable for a disease formation, and the infection remains in the leaves, stems, pods, and petioles as discrete lesions. The symptoms appear like round spots that have brown margins where pycnidia are presently showing a gray center that appears like concentric rings. Lesion size varies from 3 to 4 cm long on stems. If the disease arises during the pre-flowering stage when conditions are unfavorable, the crop grows with the symptoms that are visible on the older branches. Pods with fully developed lesions are round having 0.5 cm diameter along with pycnidia arranged in concentric rings. The pod becomes blighted and fails to grow any seed if infection occurs in the early developmental stages of the pod growth. Shriveled seed and infected seed have resulted from late infection. The seed shows symptoms of brown discoloration and visible pycnidia which can be seen by the naked eye (Pande et al. 2012).

### Pathogenesis

*Ascochyta rabiei* germinates after 12–48 hours of inoculation. Through leaflets, the pathogen reaches to petiole and then attacks the stem. Following its germination, the pathogen forms its germ tube and appressorium-like structure, which is a specialized hyphal cell that occurs at the tip toward the germ tube required for penetrating the plant cell. The appressorium is kept apart from the germ tube through a septum and surrounded by mucilaginous exudates. The fungus at first penetrates its hyphae through the cuticle and traversing the subcuticular region reaches the forefront of epidermal cells. Penetration in the epidermal cells occurs through the wall,

keeping the protoplasmic structures intact, reaches to the intercellular space, and resides and grows between epidermal and palisade parenchymal cells (Pande et al. 2005). The diameter of hyphal cells varies in and out of the cell as 3.5  $\mu\text{m}$  and 2  $\mu\text{m}$ , respectively. Meanwhile, dark aggregates of mycelia start to grow at the subepidermal portion. Subsequently, the structure of epidermal, palisade, and spongy parenchyma starts to deteriorate and eventually gets disorganized. Infection near the stoma occurs through penetration of hypha through the juncture of guard cells and subsidiary cells regardless of whether the stoma is open or close. After the disorganization of leaf cells, pycnidium emerges from the damaged tissues. From pycnidium, conidiophores arise and subsequently conidium gets dispersed into the surrounding environment and through which new chickpea crops get infected (Galloway and MacLeod 2003). The pycnidia originate after the fifth day of inoculation. By the seventh day, non-lignified cells almost get deteriorated particularly through necrosis, but lignified cells like xylem and tracheary elements remain mostly unharmed. The pathogen while spreading from leaflet to stem through petiole infects the phloem vessels with less or no harm to xylem vessels, and consequently in some instances, the leaf breaks off from petiole. However, the fungal hyphae colonize both the xylem vessels and phloem vessels in the stem, and the walls of xylem and phloem vessels remain intact, while extensive damage happens to parenchymatous tissues (Smith et al. 2017). Although pathogen infects stems directly through its cuticle evading the usual route from the leaf, during pycnidia formation, parenchymatous cortical degradation and tissues of the pith degradation suggest that involvement of toxins and enzymes for cell wall digestion is inevitable (van den Brink and de Vries 2011). Reportedly, in the process of the pathogenesis of *A. rabiei*, solanapyrone A, solanapyrone B, and solanapyrone C are required. Though under in vivo condition only solanapyrone C has been found and nonappearance of other toxins in experimentation probably due to their low concentration. The application of solanapyrone in combination or independently results in prominent symptoms followed by an epidermal, palisade, and spongy parenchymal tissue contraction due to the effect of toxins in the protoplasm. Solanapyrone A is said to be the most toxic, resulting in shriveling, loss of turgor, broken stem, and chlorotic leaves (Kim et al. 2015). Phytoalexins like pterocarpan get degraded by *A. rabiei* through its conversion to 2-OH isoflavones and 1a-OH pterocarpan due to the activity of reductase and hydroxylase enzymes. The two kinds of enzymes particularly act upon two isomeric forms of phytoalexins, namely, maackiain and medicarpin. Apart from these enzymes, cutinase and polygalacturonase are also found to act upon the host system (Uchida et al. 2017).

### **10.9.2 *Botrytis Gray Mold (Botrytis cinerea Pers. ex Fr.)***

#### **Distribution**

*Botrytis gray mold* (BGM) is a foliar disease found in Bangladesh, Nepal, India, Pakistan, Argentina, and Australia. BGM has also been observed in Canada, Chile, Mexico, Hungary, Spain, Turkey, Vietnam, and the USA (Jain 2011).

## Economic Importance

BGM fungal foliar disease causes yield losses of about 10% (Tivoli et al. 2006).

## Epidemiology

Botrytis gray mold is the most detrimental crop disease after Ascochyta blight (Shafique et al. 2014). The pathogen of this foliar disease has a very high host range and can live on other crops as well as weeds, and hence the disease is widespread. Damages mostly occur during higher temperatures and humidity. The temperature required is greater than that of the optimum temperature needed for Ascochyta blight development. BGM originates from seed, and the fungus has a large range of hosts. The disease is generally observed during floral growth when the canopy of the crop is fully matured. Excessive vegetation, too much irrigation, rain, and close spacing are causes that favor disease growth and development. Temperature ranges between 20 and 25 °C and high humidity during podding and flowering period also favor disease growth. The disease may also occur subsequently after the appearance of Ascochyta blight (Malhi et al. 1994). *Botrytis cinerea* can inhabit on chickpea seed without showing any symptoms for more than 5 years. The period of survival gets largely affected by the storage temperature, particularly between 5 and 10 °C being optimum for survival for up to 5 years. The temperature at 20 °C has been observed to have reduced growth of the pathogen from 95% to 2% at the duration of 12 months. Heating the infected seed at the moist condition at a temperature of 50 °C resulted in a significant reduction of the infection (Williamson et al. 2007). Studies showed that chickpea leaves infected with the fungus get decomposed within a couple of days to months, but the deterioration of stems through infection requires longer duration. In India, the pathogen is observed to survive for approximately 8 months in leftover infected crops on the soil and is the principal source of the initial inoculum. Asexual sporulation of the pathogen occurs on the stubble during higher temperatures and high humidity. Spores get blown to the air from the debris of the infected crop and spread to other places. The pathogen inhabits the soil in the form of mycelia and sclerotia (Bhaskar et al. 2009). In crop stubbles, sclerotia occur in many host species, as the disease has long-term survival on the host. However, in Australia, sclerotium does not show long-term survival. In Europe, apothecia originate from fertilized sclerotia (Cannon and Kirk 2007). Chlamydospore occurs during extreme conditions like drought, nutrient deficiency, bacterial attack, and change of pH. Mycelium can be produced through the germination of chlamydospores, which serves as secondary inoculum (Stevens 2002).

## Symptoms

The absence of pod setting is the primary symptom of the disease where leaves and stems do not show symptoms. The disease shows symptoms under highly favorable conditions and forms patches in the plant which often dies. The symptoms are visible on stems, pods, leaves, and flowers as a dark brown or gray lesions layered with sporophores under high humidity. 10 mm- to 30-mm-long lesions are present on the stems which grid the stem fully. The branches break at the place of the gray mold where it has caused rotting. The leaves and flowers which are affected become a rotting mass. Lesions become water-soaked and shaped irregular on the pod.

The pod consists of small and shriveled seeds or a lack of seed in the infected plants. In the infected seeds, grayish-white mycelium is observed (Narayanasamy 2011).

### **Pathogenesis**

The spore of *Botrytis cinerea* germinates after 6–8 hours of inoculation. The fungus *B. cinerea* being a necrotrophic organism grows saprophytically on the leaf. The germ tube develops and forms a mycelial connection on the leaf. The tip of the germ tube forms appressorium, necessary for penetrating the plant cells. The pathogen penetrates the host system through the cuticle of leaf and resides and formation of mycelium at subcuticular or subepidermal layer. The penetration through stomata has been observed in the spore of *Botrytis cinerea* which germinate after 6–8 hours of inoculation. The germ tube develops and forms a mycelial connection on the leaf. After establishing itself at subcuticular or subepidermal position, the hyphae grow and reach to mesophyll cells. The hyphae thicken and start branching at the mesophyll layer, consequently damaging mesophyll and epidermal cells. The degradation of the two layers requires cell wall enzymes such as pectinases, cutinases, cellulases, and polygalacturonases. As the pathogen cannot degrade lignin, it does not affect the lignified cells like xylem and tracheary elements. The degradations of mesophyll cells occur after 72–96 hours of inoculation. The total necrosis of the leaf takes place after 120 hours of inoculation, and characteristic yellowing of the leaf is observed (Arranz et al. 2000). The reactive oxygen species (ROS) can be generated by *B. cinerea* during its metabolic processes or with the help of NADPH oxidases (NOX). The NOX is a protein of multi-subunit and can reduce superoxide anion from oxygen. The BcNoxA and BcNoxB are catalytic subunits of NOX; BcNoxA helps pathogens to colonize on host tissues, whereas BcNoxB is necessary for primary infection. Apart from these two subunits, another regulatory subunit BcNoxR is responsible for the growth, sporulation, and increased virulence of the pathogen (Hua et al. 2018). Cell wall enzymes are necessary for degrading the structural polysaccharides of the host cells. Cutinases are responsible for degrading cuticles and cellulases for cellulose. Endo- $\beta$ -1,4-xylanases and pectin methylesterases found in the cell wall are necessary for degrading xylan and dimethyl esterification of cell wall components like polygalacturonase, respectively, and therefore endorse the pathogen into its entry to host environment. Two endo-polygalacturonases, BcPG1 and BcPG2, are required for virulence of the pathogen. Both BcPG1 and BcPG2 are necessary for primary infection, while BcPG2 is also involved in lesion expansion (Ten Have et al. 2010).

## **10.10 Management**

### **10.10.1 Host-Plant Resistance**

Host-plant resistance can be termed as the adaptation taken from different herbivores or pathogens for improvement in reproduction and sensitivity. Plants are sensitive; they produce several allelochemicals (secondary metabolites) which have

been used by the plant to inhibit the growth, behavior, and survival of different pathogens (Pande et al. 2006). Pathogen inhibition can be also triggered by hypersensitivity (HR), reinforcement of cell wall by deposition of lignin, callose glycoprotein which is rich in hydroxyproline, polyphenols or cinnamic acid, etc. against leaf cuticle thickening parasite by epithelium thickening, which provides a mechanical barrier. In the case of a disease like *Ascochyta* blight, resistance is also induced by increasing the respiration rate and carbohydrate content of second days after inoculation (DAI). It has resulted in a hypersensitivity response. Second DAI gives resistance to ILC 32792 genotype by hypersensitivity response. Rather than hypersensitivity response, metabolic compounds like phytoalexin are involved in the exertion of defense mechanisms toward photogenic fungi. It had been found that when the crude culture filtrate (CCF) of the strain *A. rabiei* was applied, accumulation of medicarpin (phytoalexin) is increased in the culture. Accumulation of phenolic compounds like formononetin and biochanin A also helps in inducing plant defense. Studies show that defense-related enzyme like hydrolytic enzymes and phenylpropanoid pathway's enzymes also has their role in plant defense. Accumulation of  $\beta$ -1,3-glucanase and peroxidase in the cell wall causes the hydrolyzing of the cell of fungi. *Ascochyta* blight disease can be controlled by inducing HPR (host-plant resistance) (Waliyar et al. 2016). In the case of *Ascochyta* blight, there are several screening methods used in field and greenhouse conditions. Screening in chickpea germplasm by HPR shows a high level of resistance against BGM, by using this HPR, advanced chickpea breeding lines Australia evaluates BGM resistance germ lines. These lines equally give resistance against *Ascochyta* blight (AB) (Kumar et al. 2018).

### 10.10.2 Seed Treatment

In countries like Australia, Canada, Iran, the USA, etc., *Ascochyta* blight in chickpea had been reported due to infected seed which results in the low seed weight and discoloration. In the case of chickpea, blight-free seed productions are widely used in disease management (Sharma and Ghosh 2016). The selection of larger-sized seeds against smaller ones reduces the chances of blight disease as small-size chickpea seeds have a higher level of *Ascochyta* infections. Seed immersion in the hot water and chemicals like  $\text{CuSO}_4$  solution, thiram, malachite green, etc. are used to treat chickpea. Again fungicide dressing in the seeds of chickpea improves the resistance as it halts the spore germinations and mycelial growth on the surface of the seed (Singh and Reddy 1996). But due to several factors like soil characteristics, weather condition, and plant growth inhibition, it is found that blight disease is not prevented against the phytotoxicity of fungicides which give adverse effect on seed germination. It has been reported that treating chickpea using thiram, tridemorph, imazalil, etc. causes the loss of vigor and hence is not practiced widely (Mohammed et al. 2017).

### ***10.10.3 Culture Control Method***

The main concept of disease management is to produce pathogen-free seed. Different practices like erect cultivars, manipulating in sowing dates, etc. help in reducing different foliar diseases. Late sowing lowers the vegetative growth and thus reduces the disease incidence. To allow more aeration, wider row spacing is practiced in the crop field, and it reduces leaf wetness, relative humidity, etc. Thus it helps in the reduction of disease occurrence in plants. Another practice in the plants with compact and erect growth also helps in reducing diseases than that incuse of bushy spreading. Bushy spreading happened because of low aeration. By practicing all the above, we can reduce the disease incidence in chickpea (Heydari and Pessaraki 2010).

### ***10.10.4 Cut-Twig Method***

In the cut-twig method, test genotypes are grown in a plastic bag (45/30/5 cm) which is filled with vermiculites (4:1) and sterilized sand and placed in a glasshouse at  $25 \pm 2$  °C with susceptible check H208/JG 62 used for artificial inoculums. 10–15-cm-long tender shoot of chickpea plant was cut with a sharp edge blade in the evening. It is transferred to the test tube by wrapping the course portion with a cotton plug containing fresh tap water. It inoculates in a test tube by the susceptible check (G543 or H208 OR L3.0). The symptoms start to appear 24 hours, and after 6 days, 100% mortality of susceptible lines can be seen (Udall and Wendel 2006).

### ***10.10.5 Resistance Sources and Studies on Disease Management***

In reducing the control of Ascochyta blight, foliar spray of chlorothalonil and benomyl was used for increasing seed height and yield (Bretag et al. 2008). In Australia, they used thiabendazole and thiram for treating the chickpea seed which increases the yield by up to 20%. Complete resistance was seen in using inoculation of pre-germinated seed and in the seed coat (1995). Benomyl or sulfur is used for spraying the foliages (Hagedorn 1996).

In Australia, the host plants which are resistant used in the industries are the best for various conditions or option for controlling these diseases. Some of them use the pathogen-free seed to break off at least 3 years between chickpea crops in the same field. They keep it at least 500 m away from last year's crop in delaying sowing to applying fungicide sprayed many times. Crop management practices where emphasized to decrease or to reduce the damage occurred due to diseases. Pathogenicity is the step of pyramiding resistance genes into genetic makeup. The key component of disease management is host resistance. Fungicide dressings help to prevent the

spore germination and to eradicate the fungus from the seed coat. Another method used is the crop rotation which helps in controlling the diseases (Salam et al. 2011).

### **10.10.6 Breeding for Disease Resistance**

In single plant progenies and advanced breeding lines, they use field screening techniques and growth room for segregation. The deoxyribonucleic acid marker will encourage using an exotic source of disease resistance. ICRISAT has seen the growth of AB resistance lines in desi-type chickpea. From the diverse source, multiple crosses are produced to accumulate resistance gene (Serraj et al. 2003). Conventional breeding method NIFA-88 has been developed with the application of propineb, zineb, ferbam, etc. This method helps to reduce the secondary spread of AB in crops (Sarmah et al. 2012).

### **10.10.7 Biological Control**

Studies show that the strains like *Trichoderma harzianum* Rifai and *Trichoderma viride* give antagonistic effect on the *B. cinerea*. The growth of *B. cinerea* on the hyphal tips is inhabited by *T. viride* species. Spraying of *T. viride* on the seeds helps in the germination of the seeds. The T15 strain of *Trichoderma* species is used as an effective biocontrol agent. *T. viride* and vinclozolin are found to be more effective with the application of fungicides. To produce artificial resistance, it is treated with *T. viride* and *Gliocladium roseum* (Monte 2001). This application is equivalent to that of seed treated with thiram. Compounds like essential oil production in the plants also reduce the infection of *B. cinerea* from 90% to 80%. These essential oils include cinnamon oil, clove oil, etc. The essential oil effect is studied by an automatic microtiter plate. Bacterial species like *Thymus zygis* and *Cymbopogon martini* help in the production of essential oil which is antagonistic against *B. cinerea* (Wilson et al. 1997). Different techniques are involved in the study of growth inhibition of fungi, and this includes the production of glyoxalate which helps to combat different diseases. The biological control of foliar disease also helps in disease management without applying chemicals to the crop field (Shamsi and Khatun 2016).

### **10.10.8 Resistance Sources and Disease Management**

In reducing the control of Ascochyta blight, foliar spray of chlorothalonil and benomyl was used for increasing seed height and yield (Bretag et al. 2008). In Australia, they use thiabendazole and thiram for treating the chickpea seed which increases the yield by up to 20%. Complete resistance was seen in using inoculation of pre-germinated seed and in the seed coat. Benomyl or sulfur is used for spraying the

foliages. In Australia, the industries use host-plant resistance as the best long-term administration for diseases. Some of them use the pathogen-free seed to break off a minimum of 3 years before sowing chickpea crops in the same field. They keep a distance of 500 m from last year's crop in delaying the sowing to applying fungicide spray for several times (Pande et al. 2005). Crop management practices were emphasized to minimize the damage caused by these diseases. Pathogenicity is also the method of pyramiding resistance genes into genetic materials. The key component of disease management is host resistance. Fungicide dressings help to prevent the spore germination and to remove the fungal infections from the seed coat. Another method used is the crop rotation which helps in controlling the diseases (Johansen et al. 2008).

### ***10.10.9 The Genetic Basis of Host-Pathogen Interaction***

In the case of BGM, the gene control resistance was reported in 1985. In this, parents F1 and F2 and their backcross generation BC1 and BC2 screening for resistance against BGM under epiphytotic condition. A single dominant gene *Bor1* gives resistance to ICC 1069. The cross of ICC 1069 with BGM 413 and BGM 256 gives the ratio like 13 resistances is to 1 susceptible plant. It shows that the two epistatic interaction genes control resistance. Different studies on resistant varieties like ICC 1069, P 349, NEC 2451 and 2 susceptible genotypes JG 62 and T3 in India and Australia produced BGM resistance cross. The resistance in the entire three parents is controlled only by one single dominant gene. The F2 produces 15 resistances in 1 susceptible plant (Leroux et al. 2002).

### ***10.10.10 Gene Plant Technology***

Gene technology nowadays is used for crop/plant improvement. In the case of chickpea, gene plant technology is used to treat diseases infected by both AB and BGM. Production of antifungal metabolites by expressing different genes is one such kind of gene plant technology. Different antifungal proteins and hydrolytic enzymes like chitinase are also accumulated by gene plant technology which degrades the cell wall of fungi. In the case of kiwi fruit, the production of  $\beta$ -1,3-glucanase reduced symptoms of *B. cinerea* infection. In the case of alfalfa ferritin, an iron-binding protein is also produced which gives protection against oxidative damage of necrotic pathogen. The transgenic plant which consists of polygalacturonase-inhibiting protein (PGIP) gives resistance against *B. cinerea*. The PGIP works against the PG that is secreted by the pathogen against the plant cell wall. This PGIP is isolated from raspberry and kiwi fruit which is introduced in different plants by gene plant technology. QTL mapping is used to study *Ascochyta* blight disease in pea plants (Sagi et al. 2017).



### ***10.10.11 Integrated Disease Management (IDM)***

Integrated disease management is the technique that manages the disease and mitigates yield at the same time. It involves the cultivation of pathogen-tolerant genotype, application of diammonium phosphate in soil and of Carbendazim or Thiram in seeds, and wider row spacing (0.6 m) against foliar diseases like *Ascochyta* blight and BGM. It is reported that ICCL873 22 genotypes were controlled by chemicals of BGM, wider row spacing is used, and *T. viride* is sprayed on the genotype (Pande et al. 2006). The Nepal Agricultural Research Council (NARC) and Natural Resources Institute (NRI), UK, reported the increase of health by 400% after the IDM program (Pande et al. 2006).

### ***10.10.12 Field and Control Environment Screening for Disease Resistance***

Different techniques for screening are developed at different research centers for chickpea, and it gives artificial resistance against foliar diseases like *Ascochyta* blight. The field screening and control environment screening are two major screening methods standardized by the ICRISAT (International Crops Research Institute for the Semi-Arid Tropics) and ICAR (Bidinger et al. 2009) against AB. This involves the planting of test material in a 40 cm row space. It also involves independent cultivation that serves as the indicator or spirit line. In a cloudy day, the spores are incubated in the plants at flowering time, and infected debris are spread between rows. Again these inoculates are integrated during the dry weather for approximately 15 days. In these plants, no visible lesions are found. Again, in the environmental screening, air temperature is maintained at  $20 \pm 1$  °C, 12 hours of photoperiod, etc. (Landa et al. 2001).

## **10.11 Conclusion**

Chickpea is a quantitative source of carbohydrates, proteins, minerals, vitamins, and fibers. Chickpea also fixes atmospheric nitrogen and reduces the need for nitrogen fertilizers. The crops are affected by serious foliar diseases, which affect the development stages. *Botrytis* gray mold and *Ascochyta* blight are among the most prominent diseases of chickpea. New and suitable understanding of the science, ecology, distribution, symptoms, epidemiology, pathogenesis, economic importance, and integrated management or control measures of the major foliar fungal diseases of chickpea is studied or focused on this chapter. The foliar disease has restricted chickpea production in many countries; therefore integrated management or control strategies are needed to be adopted to prevent loss of crop and pulses. Investigation of the pathogen's genetic basis of host-pathogen interaction and

identification of the host-plant resistance will help in improving or breeding a resistant variety of chickpea and will be useful to farmers and researchers. Damage caused by fungal foliar diseases can be reduced by using moderate integrated resistant cultivars with the strategies of agronomic management practices. The management practice will result in a better resistance for the host plant and will lead to greater opportunities for sustainable agriculture and maximum productivity. Agronomic options are added to management to decrease the damage which is caused by the pathogen.

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