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Botrytis grey mould of chickpea: a review of biology, epidemiology, and disease management*

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Abstract. Botrytis grey mould (BGM), caused by *Botrytis cinerea* Pers. ex. Fr., is an economically important disease of chickpea (*Cicer arietinum* L.), especially in areas where cool, cloudy, and humid weather persists. Several epidemics of BGM causing complete crop loss in the major chickpea-producing countries have been reported. The pathogen *B. cinerea* mainly survives between seasons on infected crop debris and seeds. Despite extensive investigations on pathological, physiological, and molecular characteristics of *B. cinerea* causing grey mould type diseases on chickpea and several other hosts, the nature of infection processes and genetic basis of pathogen variability have not been clearly established. This lack of information coupled with the need for repeated application of chemical fungicides forced the deployment of host plant resistance (HPR) as a major option for BGM management. Effective and repeatable controlled-environment and field-screening techniques have been developed for identification of HPR. Of the selected portion of chickpea germplasm evaluated for BGM resistance, only few accessions belonging to both cultivated and wild *Cicer* spp. were tolerant to BGM, and the search for higher levels of disease resistance continues. Fungicide application based on disease predictive models is helpful in precision-based fungicide application. Integrated disease management (IDM) of BGM has proved more effective than any of the individual disease management components in large-scale, on-farm studies conducted in India, Nepal, and Bangladesh. Further information on the biology of *B. cinerea* and epidemiology of the disease is needed to strengthen the IDM programs. In this paper the biology of *B. cinerea* including its variability, epidemiology of BGM, identified sources of resistance, and other management options, and available information on biochemical and genetic basis of disease resistance have been reviewed with a mention of future research priorities.

Additional keywords: *Botryotinia fuckeliana*, biochemical, histopathological, variability.

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Introduction

Chickpea (*Cicer arietinum* L.) is the third most important grain-legume crop in the world. It provides a high-quality diet for human consumption as a main source of protein, especially for the vegetarian population of the Indian subcontinent. It is also used in stock feed rations. Chickpea grown in rotation with cereals increases the yield of cereals by enhancing the soil nitrogen and breaking the disease cycle of important cereal pathogens. Because of its tolerance to heat and drought, it is suitable for low-fertility soils. Globally, chickpea is cultivated on about 11.12 million ha, adding 8.62 million tonnes of grain to the global food basket (FAO 2005). Despite the large acreage under chickpea cultivation, the total production and productivity are quite low in most of the chickpea-growing areas and a wide gap exists between the yield of chickpea achieved in experimental plots, frontline demonstrations, and farmers' fields. Susceptibility of chickpea to a number of fungal pathogens from seedling stage till harvest is the primary cause for low yields. Botrytis grey mould (BGM), caused by *Botrytis cinerea* Pers. ex. Fr., is the second most potentially important disease of chickpea after Ascochyta blight caused by *Ascochyta rabiei* [Pass] Labour. BGM can devastate chickpea, resulting in complete yield loss in years of extensive winter rains and high humidity (Reddy *et al*. 1988; Pande *et al*. 2002).

Geographical distribution and ecological occurrence

The first occurrence of BGM on chickpea was reported from India by Shaw and Ajrekar (1915) and later by Butler and Bisby (1931). The first epidemic of BGM was reported by Carranza (1965) in Argentina, which resulted in a crop loss of 95%. Subsequently, several BGM epidemics with almost complete yield loss have been reported from many chickpea-growing countries. This disease is of serious concern in India, Bangladesh, Nepal, Pakistan, Australia, and Argentina (Haware and McDonald 1992, 1993; Bakr *et al*. 1993; Dhar *et al.* 1993; Karki *et al*. 1993; Malik *et al*. 1993; Haware 1998; Pande *et al*. 2002; Davidson *et al*. 2004) where yield losses of up to 100% were reported under conducive conditions. BGM has also been reported from Canada, Chile, Colombia, Hungary, Mexico, Myanmar, Spain, Turkey, the USA, and Vietnam (Fig. 1) (Nene *et al*. 1984; Pande *et al*. 2002). The disease reached epidemic proportions in India during the 1978–79 crop season, destroying about 20 000 ha of chickpeas (Grewal and Laha 1983; Grewal *et al*. 1992; Haware 1998). In Nepal, the disease occurs almost every year, with average yield losses of 15% (Joshi 1992). The disease was first documented in Bangladesh during 1981 and reached devastating proportions in 1988, destroying almost all the crop (Bakr and Ahmed 1992). Currently, it is considered the most damaging foliar disease of chickpeas in Bangladesh (Bakr *et al*. 2002). The effects of BGM on pod yield depend on the onset of the disease in relation to crop growth, and disease severity, both of which depend largely on weather conditions and inoculum levels of the pathogen.

Causal organism

The asexual stage of the necrotrophic fungus *B. cinerea* (Moniliaceae, Hyphales) is dominant in chickpea crops. *B. cinerea* grown on potato dextrose agar (PDA) has a white, cottony appearance, which turns light grey with age. The young hyphae are thin, hyaline, and 8–16 µm wide, and they become brown and septate with age. The conidiophores are light brown, septate, and erect, ramified pseudodichotomically with slightly enlarged tips bearing small pointed sterigmata bearing 1–2-celled, hyaline, oval, or globose conidia forming clusters. Conidia from infected chickpea plants and on PDA measure 4–25 × 4–18 µm and 4–16 × 4–10 µm, respectively (Jarvis 1980; Nene and Reddy 1987; Pande *et al*. 2002). Sporodochia formed on the host

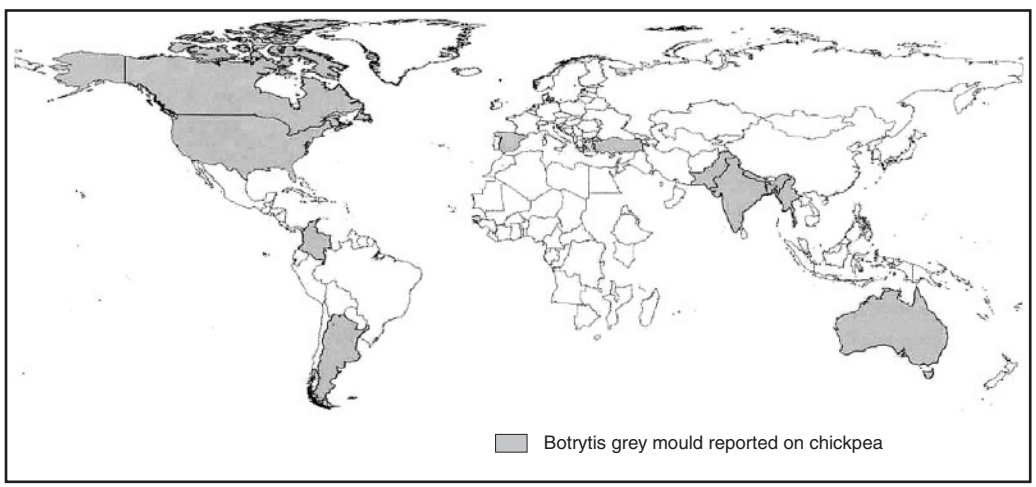

Fig. 1. Global occurrence of Botrytis grey mould in chickpea.

surface measure 0.5–5.0 µm in diameter (Joshi and Singh 1969) and may turn into hard sclerotial masses. However, cultural characteristics and sporulation of *B. cinerea* largely depend on and vary with nutrient medium, temperature, and other ecological factors.

Sclerotia, which germinate asexually by producing conidiophores bearing conidia, can form on crop stubble. The teleomorphic stage of *B. cinerea*, *Botryotinia fuckeliana* (de Bary) Whetzel (Family: Pezizales, Ascomycotina) is formed following fertilisation of sclerotia with uninucleate microconidia followed by their exposure to cold temperatures. The sexual stage germinates from fertilised sclerotia by the emergence of apothecia that release sexually produced ascospores (Faretra and Grindle 1992). Apothecia formation requires either 2 sexually compatible isolates (MAT 1-1 and MAT 1-2) or a pseudo homothallic isolate (MAT-1/2) (Faretra and Grindle 1992). There are no reports of the sexual state of *B. cinerea* occurring naturally on chickpea stubbles. However, it has been produced under laboratory conditions in India (Singh *et al*. 1997).

Disease diagnosis

Characteristic symptoms

All the aerial parts of chickpea are susceptible to the disease with growing tips and flowers being the most vulnerable (Fig. 2*a*; Bakr and Ahmed 1992; Grewal *et al*. 1992; Haware and McDonald 1992; Haware 1998; Bakr *et al*. 2002). Symptoms of BGM usually become apparent following crop canopy closure (Knights and Siddique 2002). BGM often appears first as water-soaked lesions on the stem, near ground level, that extend along the stem, and lead to infection of other stems (Knights and Siddique 2002). These lesions may be 10–30 mm long and completely girdle the stem. Branches break off at the rotting point and the affected leaves and flowers turn into a rotting mass (Bakr *et al*. 2002; Pande *et al*. 2002). Initially, the disease is randomly distributed within a crop, with infected plants being scattered, with yellowing or dying branches, or if the lesions are at ground level, as scattered dead plants (Fig. 2*b*). Drooping of the affected terminal branches is a common field symptom (Haware and McDonald 1992) and branches may break off at the point of infection (Grewal *et al*. 1992). The fungus can form grey or brown to light brown lesions on leaflets, branches, and pods, covered with hairy sporophores and masses of single-celled, hyaline spores (Haware and McDonald 1992; Haware 1998). Lesions on pods are water-soaked and irregular. Sometimes tiny black sclerotia are formed on dead tissue (Nene and Reddy 1987; Nene *et al*. 1991; Haware and McDonald 1992; Haware 1998). Grey fungal growth and profuse sporulation will occur if conditions within the canopy are moist or humid and rapidly spread through the canopy resulting in patches of dead plants (Knights and Siddique 2002). Flower drop is common leading to poor pod formation and low grain yields (Haware 1998; Knights and Siddique 2002; Pande *et al*. 2002) and this is often undetected unless the crop is closely monitored. Depending on the site of infection, mature seeds from diseased plants may be shrunken, dark coloured or, when the fungus has invaded the pod, the seeds are covered in a white/grey fungal mat (Fig. 2*c*; Bakr and Ahmed 1992; Tripathi and Rathi 1992; Haware 1998; Bakr *et al*. 2002; Knights and Siddique 2002).

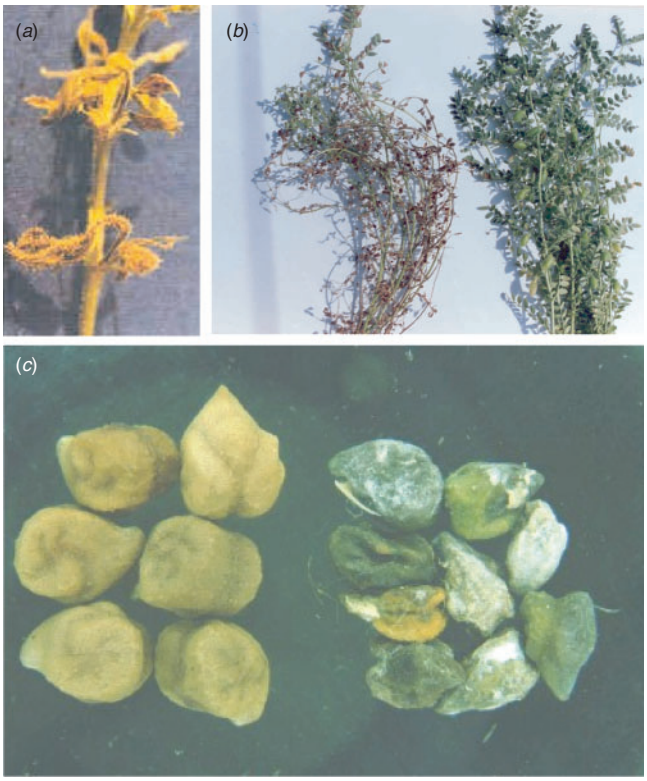

Fig. 2. Symptoms of Botrytis grey mould (BGM) infection in chickpea: (*a*) infected flowers, (*b*) BGM infected and dead plant (left side) in comparison to healthy plant (right side), and (*c*) seeds harvested from BGM infected pods (left side) and severely infected seed (right side).

Seedling rot

The pathogen *Botrytis cinerea* is one of the many fungi associated with seedling disorders of chickpea (Cother 1977*a*; Bretag and Mebalds 1987), creating a soft rot (Burgess *et al*. 1997*a*). In most chickpea-growing regions of the world, foliar infection is considered most important, whereas in Australia, soft rot of young seedlings resulting from seed-borne infection is also important and can result in total crop failure (Burgess *et al*. 1997*a*). Symptoms include poor emergence, yellowing, wilting, and death of seedlings and pale yellow to light tan discoloration of the taproot. Most plants that develop soft rot become flaccid and then die within a few days. Plants seldom recover from the disease.

Epidemiology

Pathogen survival

Seed infection

Botrytis cinerea survives on chickpea seed (Laha and Grewal 1983; Haware *et al*. 1986; Meeta *et al.* 1986) without any visible symptoms for at least 5 years and may be internally or externally seed borne (Grewal and Laha 1983), although Burgess *et al*. (1997*a*) found it to be largely external. The survival period on seed is affected by the storage temperature (Tripathi and Rathi 1992), the longest being up to 5 years at 5–10°C (Pande *et al*. 2002), and relative humidity (Laha and Grewal 1983). Burgess *et al*. (1997*a*) found that survival of the pathogen on chickpea seed was reduced from 95 to 2% after 12 months storage at 20°C. Heating moist seed to 50°C for 5 min resulted in significant reduction in viable seed infection. Seed from diseased plants may not show external symptoms and a laboratory seed-testing procedure is required for detection of the fungus (Haware *et al*. 1986). Seed infection levels up to 95% have been recorded from diseased crops (Burgess *et al*. 1997*a*). Seed-borne inoculum appears to be most important under Australian conditions (Lenne and Haware 1997) and seeds with infection level greater than 5% are considered unsuitable for planting (Wright 2000).

Seed-borne infection has been thought to be the most important means of transmitting Botrytis seedling rot as first reported by Cother (1977*b*). Burgess *et al*. (1997*a*) established the importance of seed-borne inoculum as a source of primary infection for Botrytis seedling rot. Seedling rot resulting from infected seed caused failure of several commercial chickpea crops in the Wimmera region of Victoria in Australia. The fungus *B. cinerea* is not systemic in infected chickpea seedlings. Occurrence of foliar phase of BGM requires spread of conidia from infected seedlings or from other inoculum sources in the surrounding area (Burgess *et al*. 1997*a*).

Plant debris and soil

Studies conducted at ICRISAT, Patancheru, showed that infected chickpea leaves decomposed within a few months but the stems took considerably longer (Haware and McDonald 1992). In India, the pathogen survives on plant debris on the soil surface for up to eight months (Meeta *et al.* 1986; Singh 1989; Tripathi and Rathi 1992; Singh and Tripathi 1993) and is considered the main source of primary inoculum. The pathogen also survives in the soil as mycelia and sclerotia (Mahmood and Sinha 1990). In Western Australia, *B. cinerea* remained viable for 9–11 months in the previous season's chickpea stubbles and survived over the hot (>35°C), dry conditions of summer and through the following growing season (Galloway *et al*. 2004). Asexual sporulation of the fungus occurs on this stubble under warm (>20°C), moist conditions associated with prolonged periods of high relative humidity (Galloway *et al.* 2004). Spores can be blown several hundred metres from their source (MacLeod and Sweetingham 2000), indicating that plant debris could also be a major source of primary inoculum for BGM in Australia, similar to India.

The fungus *B. cinerea* has been reported to survive in the soil in India even at a depth of 0.10–0.25 m at 40°C, from one crop season to the next, both in the form of mycelium and sclerotia (Singh and Tripathi 1992), whereas in Australia, *B. cinerea* did not survive on chickpea stubble buried at a depth of 0.5 m (Galloway and MacLeod 2003).

Sclerotia and chlamydospores

The fungus *B. cinerea* is known to produce sclerotia on crop stubbles of many host species. The sclerotia are thought to be the main means of the fungal long-term survival (Coley-Smith 1980). In Europe, apothecia emerge from the fertilised sclerotia and wind-dispersed ascospores are released mainly in the spring after chilling and periods of high rainfall on *Vicia* beans (Harrison 1988). Sclerotia develop on the previous season's chickpea stubble in Australia after exposure to cold (>10°C) winter temperatures. As day-time temperatures increase in spring, sclerotia germinate asexually, forming conidia on conidiophores. The sclerotia remain viable for the rest of the growing season but do not survive the following hot, dry summer conditions (Galloway and MacLeod 2003); hence, sclerotia are not considered to be a means of long-term survival in Australia.

Chlamydospores of *B. cinerea* are formed in response to drought, nutrient and oxygen deficiency, attack by bacteria, and pH alterations. Chlamydospores serve as structures of survival and infection. The chlamydospores germinate to produce mycelium, which either directly or after production of macroconidia, serves as secondary inoculum (Urbasch 1986).

Alternative hosts

Due to the wide host range of this pathogen, the role of alternative hosts is likely to play an important part in survival from one chickpea crop to another (Coley-Smith 1980; Haware 1998; Knights and Siddique 2002; Pande *et al*. 2002). However, further studies are required to understand the host-specific pathogenicity of *Botrytis* isolates of chickpea.

Disease development

There is a wealth of literature available on the temperature and relative humidity requirements of *B. cinerea* on many crops of importance. It should, however, be noted that the temperature and relative humidity requirements for *B. cinerea* appear to be influenced by the host plant and even by the plant part being infected (Elad *et al*. 1992).

On chickpeas, the optimum temperature for sporulation and conidial germination is 25°C (Mahmood and Sinha 1990; Singh 1997) and 20°C (Rewal and Grewal 1989*a*),

respectively, with 5°C and 30°C being the minimum and maximum extremes for conidial germination. However, different isolates were found to require differential light intensities and relative humidity for conidial germination (Rewal and Grewal 1989*a*).

BGM may develop rapidly over time and space, depending on the environmental conditions. Relative humidity, leaf wetness, and temperature are the most important factors (Tripathi and Rathi 1992; Butler 1993; Pande *et al*. 2002). Bakr and Ahmed (1992) found that disease increased at temperatures of 17–28°C and 70–97% relative humidity. In Bangladesh, maximum disease severity was recorded at a temperature range of 20–28°C (Bakr *et al*. 1997) and 25–30°C in India (Reddy *et al*. 1990; Tripathi and Rathi 1992). In the Indian sub-continent, BGM epidemics have occurred in years with high rainfall and a high number of rainy days (Bakr and Ahmed 1992; Joshi 1992; Tripathi and Rathi 1992; Davidson *et al*. 2004). The duration of leaf wetness appears to have some influence on the development of BGM on chickpeas. Disease severity increased with leaf wetness periods greater than 12 h/day (Singh and Kapoor 1984). The epidemics can spread rapidly at 95% or above relative humidity and up to a maximum temperature of approximately 25°C in a dense crop canopy. Under such conditions the disease cycle can be completed in 7 days (Haware 1998).

Initial work from India has shown that the lower leaves of the chickpea plant are infected by *B. cinerea* at a very early growth stage, and subsequently the disease spreads to upper leaves under favourable conditions (Haware and McDonald 1992). Epidemics occur once the crop canopy closes, due to an increase of relative humidity in the microclimate of the crop canopy. Singh *et al*. (1997) showed that the disease increased significantly at high plant populations under a dense crop canopy. Chickpea appears to be most susceptible to infection at the flowering stage (Saxena and Johansen 1997). In Australia, foliar epidemics of BGM are sporadic. The disease symptoms can appear at any time during plant growth, but maximum development of the disease is observed at the reproductive phase.

Host range

Botrytis cinerea is a non-specialised pathogen well known for its global distribution and extensive host range of more than 100 plant species from different genera including ornamental plants, vegetables, fruit, field and glasshouse crops, several weeds, and post-harvest produce. The host range includes species such as black gram, strawberry, grapevine, apple, cabbage, carrot, cucumber, eggplant, lettuce, lentil, mungbean, mustard, paddy, pea, pepper, pigeonpea, squash, tomato, chrysanthemum, dahlia, lily, rose, gladiolus, and tulip (Chand 1997). *B. cinerea* isolated from chickpea, infected 8 crops and 21 weed species under artificial inoculation conditions (Rathi and Tripathi 1991). Meeta *et al*. (1988) tested *B. cinerea* from chickpea on 20 plant species from 17 families under greenhouse conditions and found it infecting peas and 7 weeds, none of which was a previously reported host.

Pathogen variability

The pathogen *B. cinerea* is reported to have extreme variability and adaptability to a wide range of environmental conditions. Joshi and Singh (1969) and Singh (1970) observed the formation of sclerotial and/or sporodochial bodies on *B. cinerea*-infected chickpea plants in the Tarai region of Nainital, India, which were not found later from the same area (Pandey 1988). Singh and Bhan (1986) and Rewal and Grewal (1989*b*) identified 4 and 5 pathotypes, respectively, among the *B. cinerea* isolates collected from northern India. Kishore (2005) differentiated 8 chickpea isolates of *B. cinerea* collected from India and Nepal into distinct pathotypes based on their morpho-cultural characters and reaction on 39 differential lines and RAPD markers.

Molecular markers such as microsatellites are powerful tools for accurate detection of genetic diversity because they are highly polymorphic across numerous loci and are reproducible. In chickpea, microsatellites have revealed genetic variation among isolates of *Ascochyta rabiei* (Phan *et al*. 2003). A recent study that used microsatellite DNA markers developed specifically for the *B. cinerea* genome (Fournier *et al*. 2002), revealed genetic variation in *B. cinerea* isolates of chickpea from 4 regions in Bangladesh, India, and Nepal (Isenegger *et al*. 2005). Furthermore, hierarchical sampling of field sites in Bangladesh elucidated the level of genetic variation at various spatial scales. Consequently, high genetic diversity was determined within and among subpopulations and was detected in the smallest spatial scale sampled within field sites (1–2 m). Multilocus microsatellite profiles showed considerable genotypic diversity and discriminated up to 50% of isolates examined within a field. Evidence for a mixed reproductive system and gene flow was revealed within and among subpopulations (Isenegger *et al*. 2005).

A UPGMA tree revealed that isolates from Bangladesh are quite diverse and several were closely related to isolates from India and Nepal (Isenegger *et al*. 2005). Isolates from all subpopulations from Bangladesh showed potential for a highly adapted pathogenic group to chickpea, which can threaten (or break down) long-term control with fungicides and durable host resistance. Ardley and Weichel (2005) differentiated *B. cinerea* isolates from different hosts based on the presence of transposable elements. Transposable element *Flipper* was found in lettuce and grapevine isolates, whereas, transposable element *Boty* was present in grapevine, chickpea, and lentil isolates. Genetic similarity of internal transcribed spacer (ITS) regions indicated that lentil and chickpea isolates were closely related.

Previously, molecular evidence revealed the role of genetic recombination in *B. cinerea* from grapevine in France (Giraud *et al*. 1997). This is important, as genetic recombination can generate new genotypes, hence genetic diversity can spread quickly via asexual conidia. In other studies in *B. cinerea*, molecular markers have revealed high genetic diversity and high gene flow among populations from vegetable crops in Europe (Alfonso *et al*. 2000; Moyano *et al*. 2003). Elucidating the genetic structure by measures of genetic diversity within and among fungal pathogen populations is of major importance as it infers adaptive potential that can hamper disease management based on fungicide and host resistance. Indeed, fungal populations with high genetic diversity, mixed reproductive systems, and gene flow are considered to be highly adaptable and therefore pose a risk of rapid breakdown of host resistance (McDonald and Linde 2002). Furthermore, the durability of resistance will depend on genetic control such as single gene or quantitative resistance, and deployment strategies that would require consideration on a regional to multi-regional scale (McDonald and Linde 2002).

Biochemical and histopathological basis of host-plant resistance

Unlike other major plant–pathogen systems of crop plants, detailed investigations have not been undertaken on the infection process of *B. cinerea* on the chickpea plant and the biochemical basis of BGM resistance in chickpea has not been determined. Preliminary investigations on the infection process recorded that inoculated spores of the fungus germinate within 6–8 h and the germ tube proliferates saprophytically and forms a mycelial mat on the leaf surface. During the period of proliferation and formation of a mycelial mat, the hyphal tips, in direct contact with the host surface, swell to form appressoria and form the infection hyphae, which penetrate directly through the cuticle and form the subcuticular and subepidermal mycelium. In some cases the hyphae penetrate directly through the host surface, although penetration through stomata has also been observed (Pandey 1988). After penetration the infection hyphae grow and ramify in the leaf tissue subcuticularly and subepidermically. Mycelium grows within the mesophyll cells, which thickens and branches after penetration. The pathogen causes extensive damage to the leaf tissue by destroying epidermal and mesophyll cells, most probably by degrading the cell walls even in advance of invading hyphae. It indicates the involvement of cell-wall degrading enzymes such as pectinases, polygalacturonases, and cellulases. Only lignified xylem and tracheary elements remain unaffected probably due to the inability of the pathogen to degrade lignin.

It was observed that palisade and spongy parenchyma cells in the resistant genotype (ICC 10302) were more compact than in the moderately resistant (GG 588) and susceptible genotype (H 355). No significant anatomical alterations were observed in any of the genotypes up to 48 h of germination. Degradation of mesophyll cells was quite evident in most parts of the susceptible cultivar 72 h after inoculation, which became more pronounced after 96 h, resulting in complete necrosis of the leaf after 120 h. In moderately resistant and resistant parents, the breakdown of mesophyll cells was first recorded 96 h after inoculation. Consequently, yellowing was observed after 120 h and complete degradation of mesophyll cells was quite pronounced after 120 or 144 h (Pandey 1988).

It was observed that under high humidity, even the field resistant genotypes were infected by *B. cinerea*. However, the infection and colonisation in resistant cultivars were delayed by 24–48 h as compared with the susceptible cultivars. Mohhamadi (1987) reported that leaf surface inhibitors, probably phenolic in nature, are important for resistance in chickpea under field conditions and a high correlation was observed between total phenolic content of the leaf washings and degree of resistance of the genotypes. In the presence of inhibitors, spore germination and germ tube growth were delayed for 6–8 h and this time is sufficient for desiccation of spores under tropical conditions. However, under humid conditions there was no desiccation of the spores and germ tubes on the leaf surface, hence, even the field tolerant varieties became susceptible.

Total phenolic content, sugars, antifungal peptides, and phytoalexins are observed to be associated with BGM resistance. *Botrytis* spp. are known to be the 'high sugar' pathogens that usually attack plant tissues with more sugar content (Horsfall and Dimond 1957). These results are supported by the studies of Mitter *et al*. (1997) who observed that healthy chickpea plants of a BGM-resistant genotype ICC 1069 had significantly lower total soluble sugars and free amino acids and higher total phenol levels than the susceptible var. BGM 408. The amount of sulfur-containing amino acids, methionine and cystine, was almost double in genotype ICC 1069 compared with BGM 408. Further, shoot tips of both the cultivars had higher quantities of sugars and free amino acids and low content of phenols compared with the middle and lower leaves.

Two antifungal peptides with novel N-terminal sequences, designated cicerin and arietin with molecular weights of 8.2 and 5.6 kDa, respectively, were found in chickpea seeds. Arietin exhibited a higher translation-inhibiting activity in a rabbit reticulocyte lysate system and was found to be highly antifungal to *B. cinerea*, *Mycosphaerella arachidicola*, and *Fusarium oxysporum* (Ye *et al*. 2002).

A pterocarbon phytoalexin, maackiain, was found associated with BGM resistance in *C. bijugum* Rech. f., a wild relative of chickpea (Stevenson and Haware 1999). The concentration of maackiain in *C. bijugum* foliage was 200–300 μg/g, compared with <70 μg/g in susceptible species. After inoculation with *B. cinerea*, maackiain concentration increased to >400 μg/g in *C. bijugum*, whereas

no significant increase was recorded in the susceptible species. Maackiain inhibited the germination of *B. cinerea* conidia in a dose-dependent manner, and <10% of spores germinated when treated with 500 µg/mL (Stevenson and Haware 1999).

Genetic basis of host–pathogen interactions

The limited reports available on genetics of BGM resistance in chickpea suggest that a few major genes control resistance in the host to BGM. The first report was by Tewari *et al*. (1985), who studied inheritance of BGM resistance in a cross between ICC 1069 (resistant) and Pant G 114 (susceptible). The parents, F_1, F_2, and backcross generations (BC_1 and BC_2) were screened for BGM resistance in the field under artificial epiphytotic conditions. The study revealed that resistance in ICC 1069 was controlled by a single dominant gene, designated Bor_1. Rewal and Grewal (1989*c*) also identified a single dominant gene for BGM resistance in crosses of ICC 1069 with the susceptible cultivars BGM 413 and BGM 256. However, in crosses of ICC 1069 with 2 other susceptible cultivars, BGM 419 and BGM 408, they observed a ratio of 13 resistant : 1 susceptible plant, which indicated that 2 genes with epistatic interaction controlled resistance. Chickpea line ICC 1069 has been widely used in disease-resistant breeding programs in India (Haware *et al*. 1992) and Australia (Ted Knight, pers. comm.). Chaturvedi *et al*. (1995) used crosses involving 3 resistant (ICC 1069, P 349-2, NEC 2451) and 2 susceptible (JG 62, T 3) genotypes to study inheritance of BGM resistance. They found that resistance was controlled by a single dominant gene in all 3 resistant parents. The F_2 from the cross ICC 1069 × P 349-2 (both resistant) segregated in a ratio of 15 resistant : 1 susceptible plant, indicating that the dominant resistance genes in these 2 parents were different (non-allelic). These reports suggest that introgression of BGM resistance in elite material should not be difficult provided donor parents with a high level of resistance are identified in the cultivated or cross-compatible wild species.

Disease management

Cultural methods

Using pathogen-free seed can reduce seed transmission of the disease. Practices such as manipulating sowing dates, using erect cultivars, and lower plant densities are helpful in reducing the level of BGM in chickpeas (Haware 1998). Late sowing reduces the vegetative growth and hence lowers disease incidence; however, this can also lead to reduced grain yield (Haware and McDonald 1992, 1993; Karki *et al*. 1993). Wider row spacings allow for more aeration of the crop canopy and reduced periods of leaf wetness and relative humidity, which in turn reduce the disease incidence (Haware and McDonald 1992, 1993; Bakr *et al*. 1993; Knights and Siddique 2002; Pande *et al*. 2002). Increased plant spacing in paired rows and intercropping with linseed (Reddy *et al*. 1990; Bakr *et al*. 2002) or wheat (Tripathi and Rathi 2000) have been reported to reduce the disease and increase grain yield. Foliage detopping increases the duration and intensity of light to the lower canopy and makes the microclimate unfavourable for disease development (Rathi and Tripathi 1995).

Similarly, plants with erect and compact growth habits show lower disease incidence than bushy spreading genotypes due to improved aeration (Haware and McDonald 1992, 1993; Sethi *et al*. 1993). Erect chickpea types may escape the disease as the open canopy allows air movement and an early drying of the foliage after rainfall (Haware 1998). Crop lodging exacerbates disease through poor canopy ventilation and genotypes with different lodging susceptibilities suffer different levels of BGM (Knights and Siddique 2002). In addition, crop rotation, burning infected debris, and deep ploughing reduce inoculum levels.

Fungicides

Seed treatments with fungicides, *viz*. iprodione, mancozeb, thiabendazole, triadimefon, triadimenol, vinclozolin, thiram, benomyl, carbendazim, or captan are effective in reducing seed infection (Cother 1977*a*; Grewal and Laha 1983; Laha and Grewal 1983; Singh and Bhan 1986; Singh and Kaur 1990; Bakr *et al*. 1993; Haware 1998; Pande *et al*. 2002; Davidson *et al*. 2004). Seed treatment with fungicides has effectively controlled Botrytis seedling rot in Australia (Knights and Siddique 2002).

Foliar sprays, used at regular intervals with the first appearance of the disease, can control an epidemic in the crop (Pande *et al*. 2002), particularly when used in combination with a seed-dressing fungicide (Grewal and Laha 1983). Effective fungicides used as a foliar spray 50 days after sowing or with the first sign of the disease include captan, carbendazim, chlorothalonil, mancozeb, thiabendazole, thiophanate-methyl, thiram, triadimefon, triadimenol, or vinclozolin (Singh and Kaur 1990; Haware and McDonald 1992; Bakr *et al*. 1993; Haware 1998; Knights and Siddique 2002; Pande *et al*. 2002; Davidson *et al*. 2004). Sometimes multiple sprays are recommended, although generally one spray at flowering followed by another 10 days later on a moderately resistant chickpea cultivar provides the best protection against BGM on chickpea (Pande *et al*. 2002).

Disease prediction models facilitate the timely application of fungicides for effective and economical disease control. Weather variables and BGM severity data over an 18-year period have been used to develop disease prediction models in Nepal and Bangladesh (Pande *et al.* 2005*b*). Temperature and relative humidity were characterised as the key variables to determine BGM development. Based on coefficient of correlation analysis, maximum temperature (tmx) and afternoon relative humidity (rha)

during the period corresponding to standard meteorological weeks 9–12 (26 February–25 March) were identified as important components of the disease prediction model. An early appearance of disease preceding this period helped in initial inoculum build up and rapid disease take-off, if the subsequent conditions were favourable. A function of these 2 variables is used as a basis for a predictive scheme to schedule fungicide sprays for managing BGM (Pande *et al.* 2005*b*).

However, the use of fungicides has not been widely adopted by resource poor farmers in Asia and hence integrated management of BGM is encouraged using agronomic practices, erect cultivars, biological control agents, and targetted fungicidal sprays (Haware and McDonald 1992, 1993; Bakr *et al*. 2002; Pande *et al*. 2002, 2005*a*).

Biological control

Greenhouse experiments with *Trichoderma harzianum* Rifai (Haware 1998) and *T. viride* (Mukherjee and Haware 1993) have controlled BGM and field experiments have given encouraging results. Five isolates of *T. viride* and *T. harzianun* were highly antagonistic to *B. cinerea* and completely parasitised the pathogen on PDA. *Trichoderma viride* inhibited the growth of *B. cinerea* and resulted in swelling of the hyphal tips. Seed treatment with *T. viride* increased percentage seed germination, root and shoot length, and plant vigour. It had no adverse effect on nodulation and resulted in significantly reduced BGM incidence (Agarwal and Tripathi 1999). *Trichoderma* sp. strain T15 isolated from the chickpea rhizosphere was an effective biocontrol agent of chickpea BGM when used as a prophylactic spray at a concentration of 10^7 conidia/mL (Mukherjee *et al*. 1995). Benomyl-tolerant isolates of T15 and vinclozolin-tolerant isolates of *T. viride* were equally effective as the wild type isolates in their biocontrol efficacy. An integrated application of fungicide-tolerant *T. viride* and vinclozolin was more effective in combating BGM than vinclozolin alone (Mukherjee *et al*. 1995, 1997). Seed treatment with *Gliocladium roseum* and *T. virens* resulted in the establishment of seed that had been artificially infected with *B. cinerea* and the disease protection was equivalent to that of thiram (Burgess *et al*. 1997*b*).

Efficacy of 7 essential oils was evaluated for antifungal activity against *B. cinerea*, using the paper disc method. Clove oil, cinnamon oil, and geraniol were found to be the most effective. These compounds, at a concentration of 1000 ppm, inhibited *in vitro* conidial germination and reduced the germ tube length of *B. cinerea* by >90 and 80%, respectively (S. Pande, unpublished data). A rapid assay procedure using an automatic microtitre plate reader was developed to determine the antifungal activity of plant extracts and essential oils against *B. cinerea*. Using this method, 13 plant extracts of *Allium* and *Capsicum* spp., and essential oils of *Cymbopogon martini*, *Thymus zygis*, *Cinnamomum zeylanicum*, and *Syzygium aromaticum* were found highly antagonistic to *B. cinerea* (Wilson *et al*. 1997). Hamilton-Kemp *et al*. (1992) reported that aldehydes including C6 and C9 compounds formed by the lipoxygenase enzyme pathway on wounding of leaves inhibited growth of fungi. Inhibitory activity of elecampane (*Inula helenium* L.) against *B. cinerea* was evaluated by Bourrel *et al*. (1993). The oil contains sesquiterpinoid lactones and isoalantolactones. Daferena *et al.* (2003) found complete growth inhibition of *B. cinerea* by oregano, thyme, dictamnus, and marjoram essential oils at relatively low concentrations (85–300 µg/mL). Three sprays of garlic extract have also been reported to reduce BGM under greenhouse and field conditions (Tripathi and Rathi 1999). However, no attempts have been made in the published literature to test abiotic elicitors to induce systemic resistance against BGM infection in chickpea.

Host plant resistance

Screening for disease resistance

Different screening techniques have been used for screening the germplasm for BGM resistance under *in vitro*, greenhouse, and field conditions (Rewal and Grewal 1989*a*; Pande *et al*. 2002; Gurah *et al.* 2003). The cut-twig technique developed by Singh *et al*. (1998) offers a non-destructive sampling of the plants and is particularly useful in wide hybridisation programs.

At the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), Patancheru, a unique facility has been established for chickpea BGM screening under controlled-environment conditions (CEC) in a growth room. Ten-day-old seedlings of the test genotypes grown in plastic trays (45 by 30 by 5 cm), filled with sterilised sand and vermiculite (4 : 1) and placed in a greenhouse at 25 ± 2°C, along with susceptible checks H 208/JG 62, are used for artificial inoculation. *B. cinerea* was multiplied on autoclaved flowers of *Tagetus erecta* (marigold) for 8 days at 25°C and 12-h photoperiod. Conidia from the profusely sporulating culture are harvested into sterile distilled water and a conidial suspension at the concentration of 3×10^5 conidia/mL is used as inoculum. Greenhouse-grown seedlings of the test genotypes and susceptible check are transferred to CEC in a growth room 24 h before inoculation. These are uniformly sprayed with the inoculum. The growth room is maintained at 15 ± 2°C and 95–100% RH with a 12-h photoperiod of 2500–3000 lux intensity. The severity of the disease in all the test genotypes is recorded on a 1–9 rating scale (Table 1) after 14 days of inoculations or when the disease severity in the susceptible check reaches 9.0.

Identification of HPR

Out of 17 258 chickpea germplasm accessions available at ICRISAT around 2800 have been screened. A limited screening of chickpea germplasm has found no genotypes

Table 1. Rating scale for Botrytis grey mould of chickpea

Rating scale	Description
1	No infection on any part of the plant
2	Minute water-soaked lesions on emerging tender leaves, usually not seen
3	Minute water-soaked lesions on 1–5% emerging and upper-most tender leaves, usually seen after careful examination
4	Water-soaked lesions on 6–10% upper-most tender leaves and tender shoots
5	Water-soaked lesions; soft rotting of 11–25% of tender leaves and shoots
6	Water-soaked lesions and soft rotting of 26–40% of top leaves and shoots
7	Soft rotting and fungal growth on 41–55% of the leaves and branches
8	Soft rotting, fungal growth on 56–70% of the leaves, branches, and stems
9	Extensive soft rotting, fungal growth on above 70% of the leaves, branches, and stems

with high levels of resistance (Haware and Nene 1982; Haware and McDonald 1992, 1993; Dhar *et al*. 1993; Karki *et al*. 1993; Rathi and Tripathi 1993; Sethi *et al*. 1993; Bakr *et al*. 2002; Pande *et al*. 2002; Gurah *et al.* 2003; Davidson *et al*. 2004). During 2005, 428 Australian advanced chickpea breeding lines were evaluated for BGM resistance. Out of these, 99 genotypes were moderately resistant (disease reaction 4–5 on 1–9 rating scale). These lines were also highly resistant to Ascochyta blight. In comparison with cultivated *Cicer* species, higher levels of resistance have been found in wild *Cicer* species, including *C. judaicum*, *C. bijugum*, *C. echinospermum*, and *C. pinnnatifidum* (Singh *et al*. 1991; Sethi *et al*. 1993; Haware 1998; Pande *et al*. 2002). Several wide and intraspecific hybridisations have been carried out to transfer the identified disease resistance in wild types and land races to commonly adopted and widely grown chickpea cultivars (Table 2). Through these breeding programs a few interspecific hybrids with moderate levels of resistance to BGM and desirable agronomic traits have been identified (Singh *et al*. 1998). Further details of other chickpea lines derived from the wide hybridisation and their resistant parents were provided by Pande *et al*. (2002).

Integrated disease management

An adequate level of genetic resistance to BGM is not available in the cultivated genotypes and fungicides become ineffective during conditions of high disease pressure. Hence, integrated disease management (IDM) using the available management options is essential to successfully manage the disease and mitigate yield losses. Chemical control of BGM combined with wider row spacing (Reddy *et al*. 1993) or the use of *T. viride* (Agarwal *et al*. 1999; Haware *et al*. 1999) as a biocontrol agent has been attempted. Ahmed *et al*. (2002) reported that use of tolerant genotype ICCL 87322 in combination with wider row spacing and spraying with bavistin was the best combination followed by the use of the tolerant genotype ICCL 87322 in combination with wider row spacing and intercropping with linseed. Judicious use of fungicides as a seed treatment and/or foliar spray in an IDM system could be economical and affordable to the resource-poor farmer.

An IDM program involving cultivation of a BGM-tolerant genotype Avarodhi, soil application of diammonium phosphate, wider row spacing (0.60 m), seed treatment with carbendazim + thiram (2 g/kg seed), and need-based

Table 2. Host-plant resistance against BGM infection in chickpea as determined by screening programs conducted in various countries

Resistance	Genotypes	References
Wild species	ILWC 35/S-1 (*C. echinospermum*) and ILWC 9/S-1 (*C. pinnatifidum*)	Singh *et al.* (1991)
	C. judaicum 182, *C. judaicum* ILWC 19–2, *C. pinnatifidum* 188, *C. pinnatifidum* 189, *C. pinnatifidum* 199, *C. pinnatifidum* ILWC 9/S-1, *C. bijugum* ILWC 9/S-1, *C. bijugum* ILWC 7/S-1, *C. echinospermum* ILWC 35/S-1, and *C. echinospermum* ILWC 39	Singh *et al.* (1998)
Land races	GPC 14, HIMA, and P 6223	Singh and Kant (1999)
	ICC 1069, 6250, 7574, and 10302	Rathi *et al.* (1984)
	ICC 466, ICC 478, ICC 662, ICC 755, ICC 756, ICC 799, ICC 800, ICC 1069, ICC 1591, ICC 7574, ICC 10302, ICCL 87322	Tripathi and Rathi (2000)
	GL 84212 and ICC 1905	Singh and Kaur (1989)
	GNG-3, C-235, and BG-249	Pandey *et al.* (1982)
	P 919, CPI 56566, JM 995, and E 100 Y	Singh and Kapoor (1985)

foliar application of carbendazim has been devised. This IDM program was evaluated in farmers' participatory research in 2 districts of Nepal during 1998–99 crop season as a collaborative research activity between ICRISAT, Nepal Agricultural Research Council (NARC), and Natural Resources Institute (NRI), UK, and has resulted in a 400% increase in grain yields and 300% increase in net income. As a consequence, during the 2002–03 crop season, there was a 100-fold increase in the number of farmers voluntarily adopting IDM programs compared with the 110 experimental trials in 1998–99. By the 2004–05 crop season, the program was firmly adopted by >20 000 farmers. This IDM program re-established chickpea production in Nepal, as the crop was hardly sown after the severe epidemic of BGM in 1997–98, when no grain could be harvested and no seed was available for sowing in the next season (Pande *et al*. 2005*b*).

Integrated disease management (IDM) of BGM in Bangladesh consisted of a BGM-tolerent cultivar such as Barichola 5 or ICCL 87322, lower seed rate (37.5 kg/ha), fungicide seed treatment, delayed sowing, and need-based foliar application of fungicides. Mean grain yield in IDM plots was 678–1610 kg/ha compared with 450–1373 kg/ha in non-IDM plots. Growing chickpea was found to be economically more viable than any other crop grown after rice, especially in rainfed rice fallows (Bakr *et al*. 2005; Pande *et al*. 2005*b*).

Gene plant technology for BGM resistance

For gene technology to be effective in delivering new traits such as BGM resistance in chickpea, the development of reliable and efficient regeneration and transformation systems is essential. In addition, cloned and characterised genes that confer antifungal activity on *B. cinerea* are of particular importance. Thus, the expression of genes with antifungal metabolites is a feasible approach for BGM resistance in advanced breeders' lines or cultivars of chickpea. A range of antifungal proteins, such as the fungal cell-wall degrading hydrolytic enzyme chitinase, have been demonstrated to suppress fungal growth of *B. cinerea* within leaf tissue in transgenic plants such as tobacco (Kishimoto *et al*. 2002), cucumber (Tabei *et al*. 1998), and *Dendranthema morofolium* (Takatsu *et al*. 1999). Similarly, expression of soybean β-1,3-glucanase in kiwifruit reduced symptoms of *B. cinerea* infection (Nakamura *et al*. 1999). Interestingly, the expression of an iron-binding protein, ferritin, from alfalfa showed improved protection from the oxidative damage that was caused during necrosis by *B. cinerea* infection (Deak *et al*. 1999). Indeed, pathogenesis or defence related proteins are potential gene candidates; however, further research is required to assess newly characterised genes and elucidate the mechanisms that suppress BGM infection processes in chickpea.

Polygalacturonase inhibiting proteins (PGIPs) are extracellular plant proteins present in dicotyledonous plants that degrade the fungal endopolygalacturonases (PGs). Most fungal pathogens, such as *B. cinerea*, secrete PGs to degrade the plant cell wall before penetration into the host tissue. Thus, transgenic plants containing PGIP would provide a potential approach in developing resistance. PGIP genes from various plant species have been cloned (De Lorenzo *et al*. 2001), with expression of foreign PGIPs having been shown to confer resistance against *B. cinerea* in few of the plant species tested (Powell *et al*. 2000). Consequently, 2 PGIP genes isolated from raspberry (rPGIP) and kiwifruit (kPGIP) have been transferred and expressed in the chickpea cultivar H 208 (Senthil *et al*. 2004). In controlled-environment resistance screening at ICRISAT, these transgenic chickpea lines were observed to be moderately resistant (disease rating of 4–5 on a 1–9 rating scale) to *B. cinerea*. With advances in proteomic and metabolic characterisations of plant development, there is the potential to determine if the introduced PGIP genes could have affected other cellular functions that are involved in disease resistance and plant growth. Advances in understanding the infection process and expression of pathogenicity genes at the molecular and physiological level of *B. cinerea* on chickpea will lead to more candidate genes and an improved approach for disease resistance.

Future outlook

In chickpea, BGM is a devastating disease and extensive studies on the biology of the pathogen and screening programs to identify host-plant resistance have failed. Despite the extensive investigations in other hosts, the infection process of *B. cinerea* on chickpea has not been studied. Also, very little is known about the resistance mechanisms of chickpea against *B. cinerea*. Knowledge of the infection process and host defence mechanisms will help in devising management strategies for BGM. Resistance to BGM identified in wild *Cicer* spp. should be transferred to land races through wide hybridisation programs. BGM management should not completely rely on the use of fungicides, as development of fungicide resistance in *B. cinerea* has been commonly observed. Hence, IDM programs suitable for adoption by resource-poor farmers should be emphasised. It is advised that BGM management in chickpea should be based on the location-specific disease predictive models. Farmers' participatory on-farm validation of the IDM programs, extension, and seed distribution systems should be the tools for promotion of IDM programs developed at research centres. Biological control options for BGM management should be further exploited. Nutrient supplementation of the foliar spray of biocontrol agents, and also using a combination of biocontrol agents each with several mechanisms of disease suppression may enhance the performance of biocontrol agents against

BGM. Transgenic plant technology using PGIPs and other antifungal proteins could be the possible approach for imparting disease resistance to commonly adapted cultivars in the future.

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