Cultural practices can be used in conjunction with fungicides to reduce the development and spread of *A. lentis*. Deep plowing reduces inoculum carryover between cropping seasons. A minimum rotation of 2 years of a nonhost crop is required between lentil crops. Other cultural practices include desiccating crops before harvest, harvesting early to prevent seed infection, altering sowing time, and planting a nonhost barrier between lentil crops and lentil residues from a previous year.

In the long term, the use of resistance genes as well as suitable crop rotations and other cultural practices offer the most economical and environmentally acceptable means of controlling *A. lentis*. Several sources of resistance have been characterized, and resistant cultivars have been developed and used in production in several countries. Planting either cultivar mixtures or multilines (mixtures of near-isogenic lines differing only in their resistance genes) produces a heterogeneous host population within a field, which may limit the potential for pathogen adaptation through direct host-induced selection and hence reduce epidemics of the disease. There are several sources of moderate to high resistance to *A. lentis* in lentil, including cultivars Laird, Indianhead, and Northfield and recently the more robust resistant line ILL7537. Wild *Lens* species closely related to the cultivated form may also contain useful resistance sources because of their long period of coevolution with the pathogen. Breeding for resistance also needs to take into account pathogenic variability. There have been reports of up to six pathotypes of *A. lentis* based on quantitative differences in pathogenicity.

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(Prepared by R. Ford, P. Inder, P. W. J. Taylor, and K. Lindbeck)

Botrytis Gray Mold of Chickpea

Botrytis gray mold of chickpea was first reported by F. J. F. Shaw and S. L. Ajrekar in 1915. The pathogen was initially identified as *Rhizoctonia napi* but was eventually changed to *Botrytis cinerea*. Botrytis gray mold is an important disease of chickpea in Argentina, Australia, Bangladesh, India, Nepal, and Pakistan, where yield losses of up to 100% have been reported under conducive conditions. The disease has also been reported from Canada, Chile, Colombia, Hungary, Myanmar, Spain, Turkey, the United States, and Vietnam (Fig. 75).

Symptoms

The pathogen attacks all aerial parts of the plant, but the growing tips and flowers are most vulnerable. Symptoms often appear first as water-soaked lesions on stems, branches, leaves, flowers, and pods. The lesions then turn gray or dark brown and are covered with erect, hairy sporophores and masses of hyaline conidia (Fig. 76). Stem lesions are 10–30 mm long and may girdle the stem completely. Affected leaves and flowers turn into a rotting mass. On thick and hard stems, the mold growth gradually transforms into a dirty gray mass containing dark green to black sporodochia (Fig. 77). Sometimes tiny, black sclerotia are formed on the dead tissues. When the disease affects pods, no seed or only shriveled seeds are formed. Occasionally, grayish white mycelia can be seen on immature seeds. *Botrytis* spp. readily grow in culture (Fig. 78).

Under natural conditions when the relative humidity is high (≥95%) and temperatures are low (20–25°C), the disease first appears in isolated patches where the crop canopy is dense (Fig. 79). Flower drop is common and leads to poor pod formation and low grain yields. The pathogen is also associated with seedling disorders of chickpea involving soft rot. Although foliar infection is considered more important in most chickpea-growing

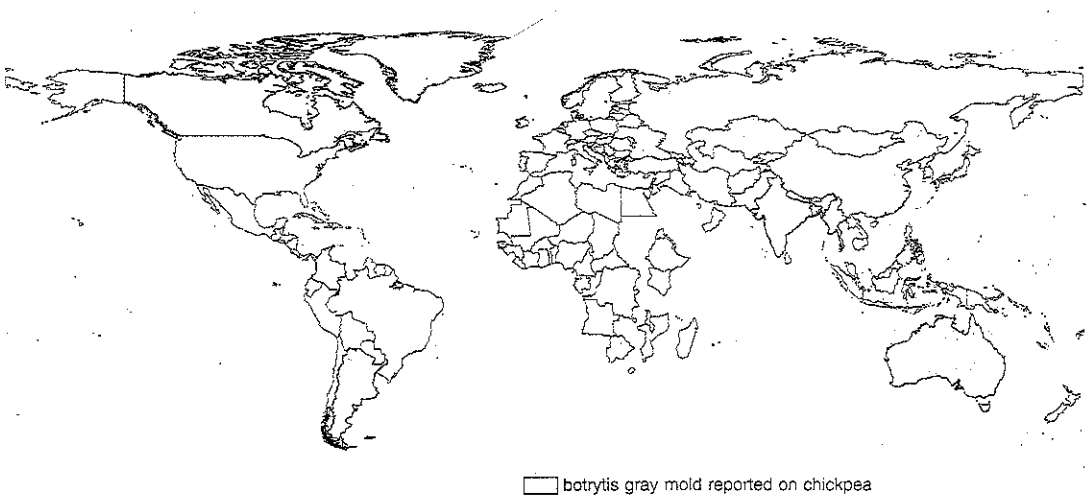

Fig. 75. Countries where *Botrytis cinerea* occurs on chickpea. (Courtesy S. Pande)

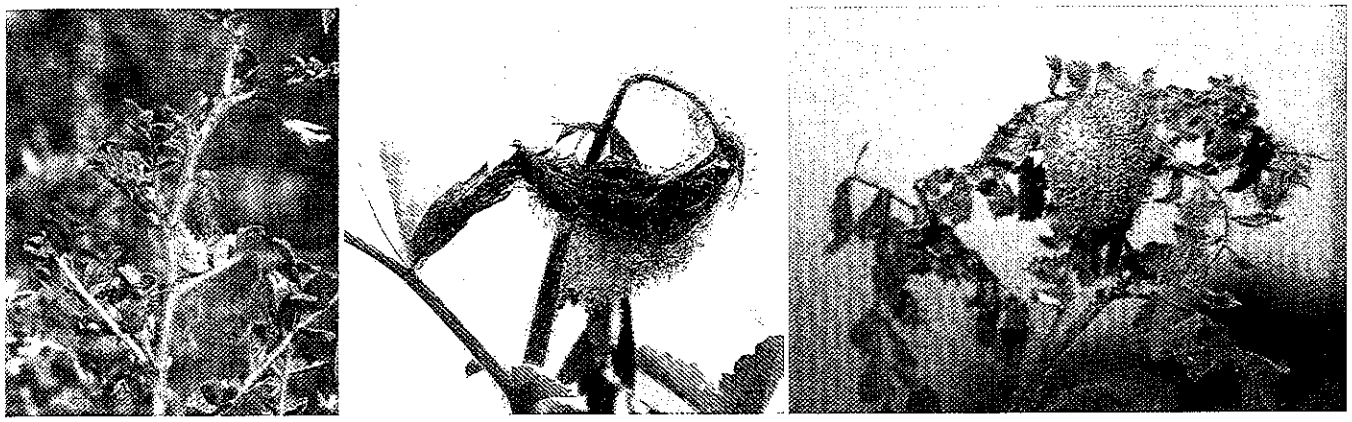

Fig. 76. Symptoms of Botrytis gray mold on chickpea. Left, infected branches, leaves, and flowers; center, fungal sporulation on infected stem, branches, and flowers; and right, fungal sporulation on pods. (Courtesy S. Pande)

Fig. 77. Mycelial growth and grayish sporulation of *Botrytis cinerea* on chickpea stem. (Courtesy J. Davidson)

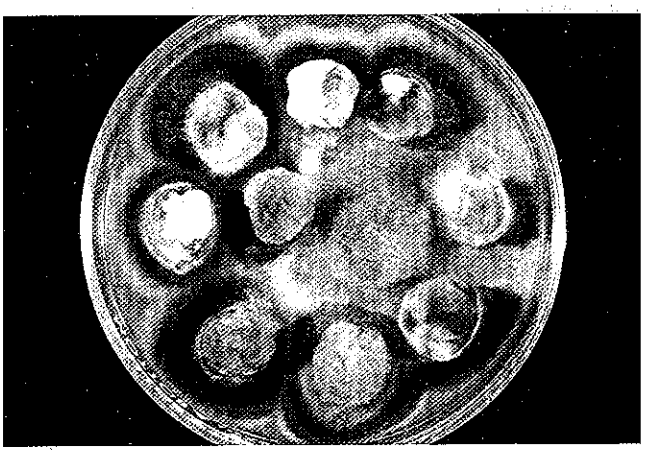

Fig. 78. *Botrytis cinerea* growing from infected chickpea seeds plated on potato dextrose agar. (Courtesy W. J. Kaiser)

regions, in Australia soft rot of young seedlings resulting from seedborne inoculum can result in total crop failure. Symptoms include poor emergence, yellowing, wilting, and death of seedlings and pale yellow to light tan discoloration of the taproot.

Causal Organism

The causal organism of Botrytis gray mold is *Botrytis cinerea* Pers.:Fr., a necrotrophic fungus. *B. cinerea* grown on potato dextrose agar has a white, cottony appearance and turns light gray with age (Fig. 80). The mycelium is septate and brown, and young hyphae are hyaline and 8–16 µm wide. Conidiophores are light brown, septate, and erect with slightly enlarged tips bearing small, pointed sterigmata. One-celled, hyaline, oval or globose conidia form in clusters on short sterigmata. Conidia from lesions on chickpea plants measure 4–18 × 4–25 µm and from potato dextrose agar 4–10 × 4–16 µm. Sporodochia formed on the host surface measure 0.5–5.0 µm in diameter and may turn into hard sclerotial masses. However, the cultural characteristics and sporulation of *B. cinerea* vary greatly with nutrient medium, temperature, and other environmental factors.

The teleomorph, *Botryotinia fuckeliana* (de Bary) Whetzel, is formed after fertilization of sclerotia with uninucleate microconidia followed by exposure to low temperatures. Fertilized sclerotia germinate by producing apothecia that release ascospores. Two sexually compatible isolates (MAT 1-1 and MAT 1-2) or a pseudohomothallic isolate (MAT-1/2) are required for apothecium formation. The teleomorph has not been observed on chickpea stubble in nature. However, it has been produced under laboratory conditions in India.

Fig. 79. Patches of chickpea plants infected with *Botrytis cinerea* in the field. (Courtesy S. Pande)

Host Range

B. cinerea is a nonspecialized pathogen and has a wide host range of more than 100 plant species, including vegetables, fruits, ornamentals, field crops, several weeds, and postharvest produce.

Disease Cycle and Epidemiology

Infested soil and infected plant debris are the main sources of primary inoculum of *B. cinerea*. The fungus can survive on chickpea seed without causing any visible symptoms for at least

5 years and may be internally or externally seedborne. Seed-infection levels of up to 95% have been recorded from diseased samples. Seedborne inoculum is most important in Australia, and seeds with an infection level greater than 5% are considered unsuitable for planting. In Western Australia, *B. cinerea* has been shown to remain viable for 9–11 months in chickpea stubble, having survived temperatures above 35°C and the dry conditions of summer through to the following growing season. In India, the pathogen also survives on plant debris and in the soil as mycelia and sclerotia. The sclerotia are thought to be the main means of long-term survival. Chlamydospores of *B. cinerea* also serve as survival and infection structures. Chlamydospores germinate to produce mycelium and conidia that serve as secondary inoculum. Chlamydospores are formed in response to adverse conditions such as drought, oxygen and nutrient deficiency, changes in pH levels, and attack by bacteria.

Because of the wide host range of this pathogen, alternative hosts can play an important part in its survival from one chickpea crop to another. Relative humidity, leaf wetness, and temperature are the most important factors for disease initiation and development. Consequently, elaborate misting systems may be required for screening chickpea germplasm lines for resistance to Botrytis gray mold (Figs. 81 and 82). The disease can spread rapidly when the relative humidity is 95% or above and the temperature is a maximum of 25°C. In a dense crop canopy, the infection cycle can be completed in 7 days and the disease can assume epidemic proportions. On chickpea, the optimal temperature for formation and germination of *B. cinerea* conidia is 15–20°C; 5 and 30°C are the minimum and maxi-

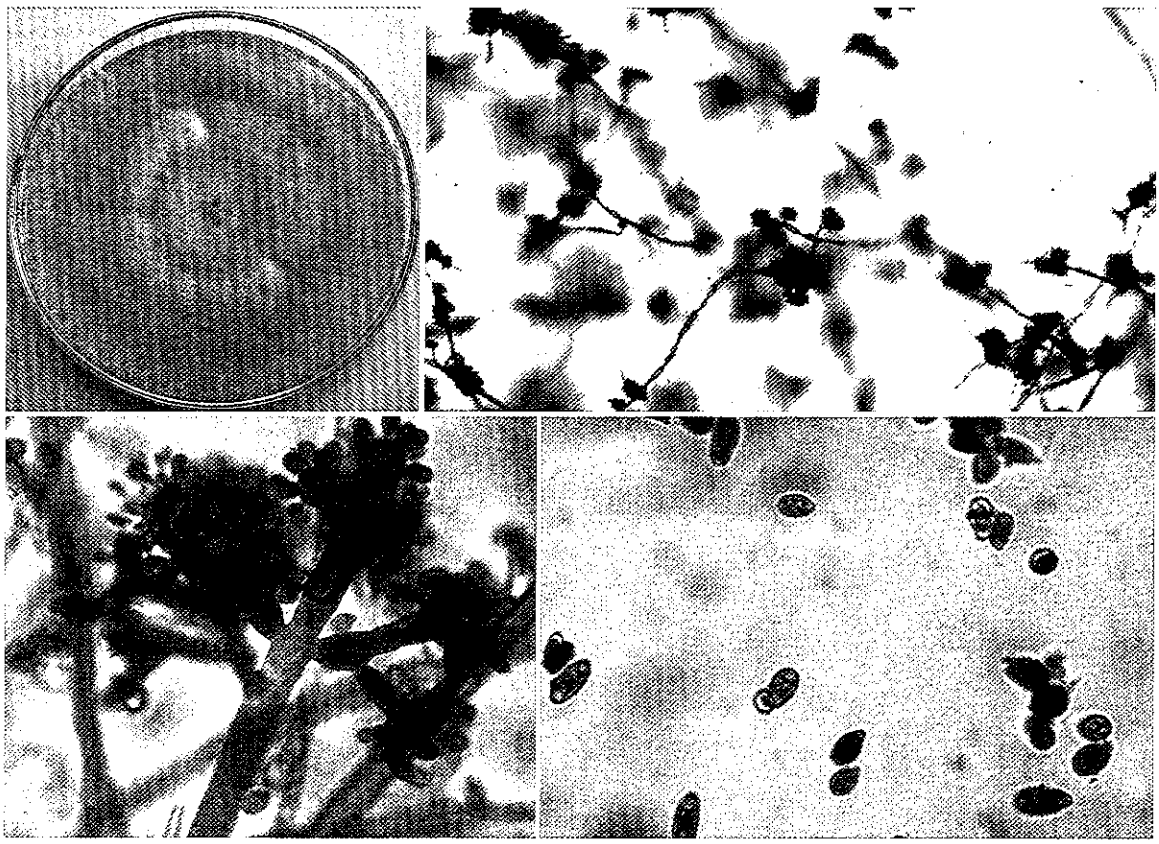

Fig. 80. Cultural characteristics of *Botrytis cinerea*. Upper left, growth on potato dextrose agar; upper right, mycelium with clusters of conidia; lower left, conidiophore bearing sterigmata and conidia; and lower right, conidia. (Courtesy ICRISAT)

Fig. 81. Growth room set up for screening resistance to Botrytis gray mold at ICRISAT. (Courtesy ICRISAT)

Fig. 82. Misting system in field plots used for screening for resistance to Botrytis gray mold. (Courtesy S. Pande)

mum, respectively. The effects of the disease on yield depend on the onset of the disease and its severity at different growth stages of the crop. In the Indian subcontinent, Botrytis gray mold epidemics have occurred in years with high rainfall and a high number of rainy days. Disease severity increases with periods of leaf wetness greater than 12 h per day.

Weather variables and records of Botrytis gray mold severity over 18 years have been used to develop disease-prediction models in Nepal and Bangladesh. Temperature and relative humidity are the key variables in predicting Botrytis gray mold development. Based on coefficient of correlation analysis, maximum temperature and afternoon relative humidity during the period corresponding to standard meteorological weeks 9 to 12 (26 February to 25 March) have been identified as important components of the disease-prediction model. An early appearance of disease preceding this period helps in initial inoculum buildup and the rapid spread of disease if subsequent conditions are favorable. A function of maximum temperature and afternoon relative humidity is used as a basis for a predictive scheme to schedule fungicide sprays for managing the disease.

Management

Cultural practices such as late sowing, wide row spacing, choice of plant type (use of erect and compact cultivars), intercropping with linseed or wheat, and avoiding excessive vegetative growth and irrigation reduce disease severity. In addition, crop rotation, burning infected crop debris, and deep plowing reduce inoculum levels. The use of pathogen-free seed can reduce seed transmission of the disease.

Seed treatment with fungicides such as carbendazim plus thiram (1:1), vinclozolin, carbendazim, triadimefon, mancozeb, triadimenol, thiabendazole, iprodione, and thiram can reduce seedborne inoculum. In Australia, this practice has effectively eliminated Botrytis seedling rot in chickpea. Foliar sprays with vinclozolin, carbendazim plus thiram, or carbendazim at regular intervals beginning at the appearance of the first symptoms, particularly in combination with seed treatment, can provide considerable control of the disease. Other fungicides effective as foliar sprays include captan, chlorothalonil, mancozeb, thiabendazole, thiophanate methyl, thiram, triadimefon, and triadimenol. They should be applied at 50 days after sowing or at the first sign of symptoms.

Biological control agents such as *Trichoderma viride*, *T. harzianum*, and *Gliocladium roseum* have been found to be highly antagonistic to *B. cinerea*. Seed treatments with these biocontrol agents increase the percentage of seed germination, root and shoot length, and plant vigor and significantly reduce Botrytis gray mold incidence. *Trichoderma* sp. isolated from the chickpea rhizosphere has also been shown to be effective against *B. cinerea* when used as a prophylactic spray at a concentration of 10^7 conidia per milliliter.

Sowing disease-resistant cultivars is the most economical and effective method to combat Botrytis gray mold. No genotypes with high levels of resistance have been found in extensive screening of chickpea germplasm and breeding lines, but many chickpea lines with moderate resistance have been identified. High levels of resistance are available in wild *Cicer* species, namely, *C. judaicum, C. bijugum, C. echinospermum,* and *C. pinnatifidum*. A list of chickpea lines derived from wide hybridization and their resistant parents is available.

An adequate level of genetic resistance to Botrytis gray mold is not available in cultivated genotypes, and the general use of fungicides has not been widely adopted by resource-poor farmers in Asia. Hence, integrated disease management, including the use of agronomic practices, erect cultivars, biological control agents, and targeted fungicidal sprays, is encouraged. An integrated management technology has been devised for Nepal consisting of the gray mold-tolerant genotype ICC 14344, soil application of diammonium hydrogen orthophosphate, wide row spacing (60 cm), seed treatment with carbendazim plus thiram, and foliar application of carbendazim strategically timed on the basis of weather conditions. Similarly, disease management in Bangladesh consists of planting a Botrytis gray mold-tolerant cultivar such as Barichola 5 or ICCL 87322, a low seeding rate (37.5 kg/ha), fungicide seed treatment, delayed sowing, and need-based foliar application of fungicides.

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(Prepared by S. Pande, M. Sharma, and J. Narayana Rao)

Botrytis Gray Mold of Lentil

Botrytis gray mold of lentil, also known as Botrytis stem and blossom blight, can affect any of the aboveground parts of the plant, including leaves, stems, flowers, pods, and seeds. The disease has worldwide distribution. Severe epidemics have been reported in Australia, Bangladesh, Canada, Colombia, Nepal, New Zealand, and Pakistan. In India, Morocco, Syria, and the United States, it is considered of minor importance. When conditions are conducive to disease, particularly high humidity, yield losses can exceed 50%.

Symptoms

Infection may first develop on flowers and pods or on the lower foliage of established plants as dark green lesions that become pale tan spots (Fig. 83). Senescent or injured plant parts