With compliments and
warmest regards to
Dr. Y.L. Nene.
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Disease Resistance Breeding in Chickpea

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Important Disease Problems of Kabuli Chickpea[1]

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Abstract

Both types of chickpeas, kabuli and desi, are affected by the same range of pathogens. Diseases that are more important in the kabuli chickpea-growing regions are: ascochyta blight (*Ascochyta rabiei*), botrytis gray mould (*Botrytis cinerea*), fusarium and verticillium wilts (*Fusarium oxysporum* and *Verticillium albo-atrum*), collar and root rots (*Sclerotium rolfsii*, *Rhizoctonia bataticola*, *Fusarium solani*, *Rhizoctonia solani* and *Pythium ultimum*), stunt (bean leaf roll virus) and nematodes. While foliar diseases such as ascochyta blight and botrytis gray mould have been responsible for devastating crops in different countries in certain years, soil-borne diseases such as fusarium wilt and collar and root rots have caused less spectacular but consistent damage. This paper gives a brief account of the important diseases of kabuli chickpea with special reference to economic importance, biology and epidemiology, and control. The present status on the availability of sources of genetic resistance to various diseases is discussed.

Introduction

Chickpea (*Cicer arietinum* L.) is the world's third most important grain legume after dry bean (*Phaseolus vulgaris* L.) and pea (*Pisum sativum* L.). Chickpeas are of two types, kabuli and desi. Since this paper deals mainly with the kabuli type, it is pertinent to mention a few characteristics of kabuli types that may have relevance to their susceptibility or resistance to different diseases. Compared with the desi type, plants of the kabuli type in general have fewer anthocyanins. Kabuli seeds have less seed coat mass, higher nitrogen and sugar content, less fiber in whole seed, less cellulose, more protein and sugar in cotyledons, and fewer polyphenols than desi seeds. Some of these characteristics probably make kabuli seeds more susceptible to seed and seedling rot fungi. We are not aware of any disease that affects only the kabuli genotypes and not the desi, although there may be differences in their relative susceptibilities.

Diseases are a major production constraint. More than 70 pathogens have been reported so far on chickpea from different parts of the world (Nene *et al.* 1984). Nene and Reddy (1987) have given a detailed review of the internationally important chickpea

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diseases. In this paper we discuss a few of the important diseases of chickpea that are prevalent in the geographical regions where kabuli chickpea is grown widely. Also, to limit the length of the paper, we have cited only selected references. For more details, the readers are referred to the review by Nene and Reddy (1987).

Ascochyta Blight

Ascochyta blight caused by *Ascochyta rabiei* (Pass.) Labr. is one of the most important diseases of chickpea. The disease has been reported from 31 countries: Afghanistan, Algeria, Australia, Bangladesh, Bulgaria, Canada, Cyprus, Egypt, Ethiopia, France, Greece, Hungary, India, Iran, Iraq, Italy, Jordan, Lebanon, Mexico, Morocco, Pakistan, Portugal, Romania, Sudan, Spain, Syria, Tanzania, Tunisia, Turkey, USA and USSR (Abdel Monem 1983; Nene *et al.* 1984). Severe outbreaks of the disease in many chickpea-growing countries have contributed to heavy crop losses (Table 1). The damage in Pakistan resulted in a severe shortage of pulses and necessitated their import to the extent of US $ 6.43 million in 1982/83 (B.A. Malik, pers. comm.). In the Mediterranean region, changing the time of sowing of chickpea from spring to winter increased ascochyta blight severity during 1976/77 (Hawtin and Singh 1984). Productivity of winter-sown chickpea was greatly increased when ascochyta blight was controlled.

Table 1. Reports of losses caused by ascochyta blight in different chickpea-growing countries†.

Country	Year	Extent of loss
Bulgaria	1936	20-50%
Greece	1957/58	10-20%
Morocco	1971	US $ 10 million
Pakistan	1920/30	50%
	1936	20-50%
	1978/79	17%
	1979/80	48%
	1980/81	Up to 15%
	1981/82	42%
Spain	Post-war period	20-100%
Syria	1974‡	0.5-1.0 t/ha
	1981	5-30%
	1982	30%
Tunisia	1981	40%
USA	1987§	US $ 1 million
USSR	1956¶	100%

† Based mainly on Nene and Reddy (1987).
‡ Hanounik (1980).
§ Kaiser and Muehlbauer (1988).
¶ Nemlienko and Lukashevich (1957).

The fungus attacks all the aerial parts of the plant. Dark brown lesions with pycnidia are produced on the blighted portions. The lesions on infected seeds are more prominent in kabuli than in desi types. Severely infected seeds are small and wrinkled (Haware *et al.* 1986). Infected-seed weight is less than healthy-seed weight (Luthra and Bedi 1932).

The fungus can survive for more than 2 years in naturally infected tissues at 10 to 35°C with 0 to 3% RH at the soil surface. However, the fungus loses its viability within 8 weeks at 65 to 100% RH or at soil depth of 10 to 40 cm (Kaiser 1973). In Syria, the viability of the fungus in diseased debris left in fields was lost within 8 months, and when buried 10 cm or deeper, it was lost within 4 months (ICARDA 1982, 1983). The pathogen also survives in seeds from infected plants.

Diseased-crop debris and infected seeds are the main sources of primary inoculum. The pycnidiospores produced at the foci of primary infection are the source of inoculum for further spread of the disease. They are wind disseminated and spores have been found in rainwater samples (ICARDA 1988). The disease spreads rapidly under cool, wet and windy conditions. High blight incidence was observed in years with high rainfall (>150 mm) during the chickpea-growing seasons. Rapid buildup and spread of the disease under field conditions occurred only when minimum and maximum temperatures were above 5 and 15°C, respectively (ICARDA 1983, 1984).

The perfect stage of the fungus, *Mycosphaerella rabiei* (Pass.) Kovach., was observed on overwintered chickpea in Bulgaria, USSR and Greece (Nene and Reddy 1987). Recently, it has been reported from Hungary (Kovics *et al.* 1986), the USA (Kaiser and Hannan 1987) and Syria (Haware 1987). Long-distance spread of blight in 1987 in the USA apparently was initiated by air-borne ascospores of *M. rabiei* (Kaiser and Muehlbauer 1988).

Disease incidence can be reduced by crop rotation, burial of crop debris deep in the soil, and removal and destruction of infected plant debris. Seed must be obtained from disease-free areas.

Seed dressing with fungicides, Calixin M (11% tridemorph + 36% maneb) (Reddy 1980) or thiabendazole (Reddy and Kabbabeh 1984a), can eradicate the seed-borne inoculum. Fungicidal foliar sprays are generally ineffective and uneconomical under epidemic situations, but were found useful in protecting a line (ILC 482) that is resistant in the vegetative stage and susceptible in the podding stage. One spray of chlorothalonil (Bravo 500) in the spring-sown crop and two sprays in the winter-sown crop during the reproductive stage were required to eliminate pod infection (ICRISAT 1987).

The best method of control should be the use of resistant cultivars. Extensive field and greenhouse screening and multilocation testing have resulted in the identification of a large number of resistant/tolerant kabuli and desi germplasm accessions. At ICARDA, 11 kabuli germplasm accessions were found to be resistant (Reddy and Singh 1984). Four

kabuli lines (ILC 72, ILC 191, ILC 3279, ILC 3856) were resistant in a total of eight of the 11 countries tested: Algeria, Greece, India, Jordan, Lebanon, Morocco, Pakistan, Spain, Syria, Tunisia and Turkey (Singh *et al.* 1984). Yield loss of resistant kabuli lines was minimal (2.8%), but in resistant desi lines it was substantial (44.9%) (ICRISAT 1985). Wild *Cicer* spp. were artificially inoculated and a few accessions of *C. bijugum* K.H. Rech., *C. judaicum* Boiss. and *C. pinnatifidum* Jaub. & Sp. were found resistant (ICRISAT 1988). A need to identify lines with stable resistance has been felt because of the existence of physiologic races. Reddy and Kabbabeh (1984b) reported six races from Syria and Lebanon; three lines were found resistant to five of these races (ICRISAT 1987). Gowen (1983), who studied pathogenic variation in a world collection of isolates, found one that was lethal to some resistant cultivars. Twenty isolates of *A. rabiei* collected from the northern belt of India were grouped into six pathotypes (M.S. Sangwan, pers. comm.). Singh and Reddy (1983) reported two genes for blight resistance, one recessive and one dominant.

In the Indian subcontinent, ascochyta blight epidemics have been observed only at latitudes north of 28°. Also, rapid disease spread usually occurs in March when thunderstorms are common. Cultivars that can mature by the end of February should therefore escape the severe effect of the disease.

Botrytis Gray Mould

Botrytis gray mould caused by *Botrytis cinerea* Pers. ex Fr. is the second most important foliar disease of chickpea. It has been reported from Argentina, Australia, Bangladesh, Canada, Colombia, India, Nepal, Pakistan, Spain, Turkey and the USA (Nene *et al.* 1984). Field incidence was first reported from Argentina where the disease caused 95% loss in yield (Carranza 1965). The first record in India of gray mould on chickpea was during the 1967/68 season from Pantnagar. The disease was responsible for heavy losses in the Indo-Gangetic plains of India during 1979/82. This disease can be as important as ascochyta blight in some countries, for example Bangladesh and Nepal. In 1988, the disease caused up to 100% yield loss in research farms in Nepal (Reddy *et al.* 1988).

The fungus attacks all aerial parts of the plant. At times, no seeds or only small, shrivelled seeds are formed in affected pods. The fungus survives as black, sclerotial bodies on infected seed and plant debris. The conidia are responsible for spread of the disease. The fungus has a wide host range and the inoculum is almost always present in the environment; it develops when weather conditions are favorable. In chickpea, *B. cinerea* is seed-borne, but the viability of seed-borne inoculum is greatly reduced during room-storage periods (Laha and Grewal 1983). The role of seed-borne inoculum relative to the inoculum present in the environment in the epidemiology of botrytis gray mould of chickpea is not clearly established.

Seed-borne inoculum can be eradicated through dry-seed dressing with vinclozolin (Ronilan), a combination of methyl benzemedazol carbamate (MBC) + thiram, or MBC alone (Grewal and Laha 1983). In pot studies, foliar sprays of these fungicides were found effective in controlling aerial infection. ICRISAT pathologists have not been able to identify high levels of resistance. The kabuli type is generally less susceptible than the desi types. Three accessions of *C. bijugum* evaluated under greenhouse conditions have resistance to gray mould (ICRISAT 1988).

Fusarium and Verticillium Wilts

Fusarium wilt caused by *Fusarium oxysporum* Schlecht. emend. Snyd. & Hans. f.sp. *ciceri* (Padwick) Snyd. & Hans. is a serious disease of chickpea in Burma, India, Iran, Nepal, Pakistan, Spain and Tunisia. It also has been reported from Bangladesh, Chile, Ethiopia, Malawi, Mexico, Peru, Sudan, Syria and the USA (Nene *et al.* 1984). No precise information on losses caused by this disease is available from any country. According to a rough estimate, about a 10% loss in yield due to wilt was considered to be a regular feature in chickpea-growing states of India (Singh and Dahiya 1973). Likewise, an estimated annual loss of 12 million rupees (approx. US $ 1 million) was reported from Pakistan (Sattar *et al.* 1953). The production of kabuli chickpeas in California has declined in recent years, mainly because of fusarium wilt (Buddenhagen *et al.* 1988).

The pathogen systemically invades all tissues of chickpea. It is seed-borne and survives through chlamydospores in seed and infected plant debris. It can survive in soil in the absence of the host for more than 6 years (ICRISAT 1985). Lentil, pigeon pea and pea were identified as symptomless carriers of the fungus (Haware and Nene 1982a).

The seed-borne inoculum can be eradicated by seed dressing with Benlate T (30% benomyl + 30% thiram) at the rate of 0.15% (Haware *et al.* 1978). Since the fungus can survive in the soil for up to 6 years and also in symptomless carriers, it is not possible to control the disease through normal crop rotations.

The use of resistant cultivars is the obvious answer. Efforts have been made at various institutes to identify and develop lines resistant to the pathogen. A massive screening program for wilt resistance is underway at ICRISAT. Both laboratory and field screening procedures have been developed and standardized (Nene *et al.* 1981). So far, 160 germplasm accessions have been identified as resistant to wilt; some of them have additional resistance to dry root rot, black root rot, botrytis gray mould, ascochyta blight and/or sclerotinia blight. Ten of the wilt-resistant lines are kabuli types. Three accessions of *C. bijugum* have resistance to both wilt and root rots (ICRISAT 1988). The first kabuli line that carried wilt resistance was ICC 32, developed at ICRISAT. Two kabuli wilt-resistant varieties, UC 15 and UC 27, have been released in the USA (Buddenhagen *et al.* 1988).

Four physiologic races (1, 2, 3, 4) have been reported from India (Haware and Nene 1982b) and three races (0, 1, 5) from Spain (Cabrera de la Colina *et al.* 1985). Wilt reaction of several cultivars suggests that race 1 is prevalent also in Mexico and the USA. Another race (race 6) was reported from California, USA (Phillips 1988). At ICRISAT Center, efforts were made to identify lines with multiple-race resistance. In pot-screening tests, 52 lines were found resistant to races 1, 2 and 4 (ICRISAT 1988).

The resistance in the host is governed by a single recessive gene (Kumar and Haware 1982; Sindhu *et al.* 1983), although recent studies at ICRISAT revealed that additional genes may be involved and early or late wilting depends upon genetic composition of a cultivar (Upadhyaya *et al.* 1983).

Soil solarization reduced wilt incidence and the pathogen population.

Verticillium wilt (*Verticillium albo-atrum* Reinke & Berth.) has been observed in Pakistan and the USA. The xylem tissue of affected plants shows lighter discoloration than that caused by *F. oxysporum* f.sp. *ciceri* (Nene and Reddy 1987).

Collar and Root Rots

These are reported from many countries and can become serious under favorable conditions (Chauhan *et al.* 1988).

Collar rot (*Sclerotium rolfsii* Sacc.) has been reported from Bangladesh, Ethiopia, India, Pakistan and Syria (Nene *et al.* 1984). The disease is seen most often in the seedling stage (up to 6 weeks after sowing), particularly if the soil is wet. Affected seedlings may collapse or dry prematurely. Incidence of collar rot is associated with high soil moisture content, the presence of undecomposed organic matter near the soil surface, low soil pH and temperatures of 28 to 30°C. It is normally a problem in the seedling stage, but in irrigated crops the disease can occur at any stage if temperatures are not low. Fungal sclerotia and colonized organic matter serve as the primary inoculum. The fungus has a wide host range.

Destroying the stubble of previous crops should help in reducing the incidence of collar rot. Date of planting influences the mortality due to collar rot. Host resistance is difficult to obtain, but lines showing tolerant reaction have been reported. Seed treatment with Bavistin + thiram + pentachloronitrobenzene (1:1:1) at the rate of 3 g/kg of seed has been recommended (Kotasthane and Agrawal 1978). At ICRISAT Center, seed dressing with 0.2% Rizolex (tolclofos-methyl) gave good protection against the pathogen.

Dry root rot [*Rhizoctonia bataticola* (Taub.) Butler; *Macrophomina phaseolina* (Maubl.) Ashby] appears suddenly when the ambient temperature range is between 20 and 30°C and when the crop is in pre-maturity stages. The disease has been reported

from Australia, Ethiopia, India, Iran, Lebanon, Pakistan, Spain, Syria and the USA (Nene *et al.* 1984). In central and southern India, the disease is more frequent in deep black vertisols. It is significantly more severe in sandy loam soils than in clay loams. Dry root rot is a minor disease in West Asia and the Mediterranean basin.

The fungus *R. bataticola* survives as sclerotia in the soil and the primary infection is by sclerotia (Nene 1980). The best control is by the use of resistant cultivars. ICRISAT scientists have identified several chickpea lines with a high degree of tolerance, singly or in combination with resistance to other diseases such as fusarium wilt and black root rot. Five kabuli lines have been found to be resistant under laboratory conditions. Resistance in the host is governed by a single dominant gene (ICRISAT 1987).

Black root rot [*Fusarium solani* (Mart.) Appel & Wr.] incidence under field conditions was first reported in 1974 from India and the USA. It also has been reported from Chile, Mexico and Spain (Nene *et al.* 1984). The disease may appear at any stage and affected plants dry prematurely. The fungus survives as chlamydospores in colonized organic matter and these propagules are responsible for primary infection. The disease occurs more commonly when soil moisture content is high and soils are light. ICRISAT pathologists have identified 18 desi lines that are resistant to black root rot as well as to fusarium wilt. These can be used in resistance breeding programs.

Wet root rot (*Rhizoctonia solani* Kühn) has been reported from Argentina, Chile, India, Iran, Mexico, Morocco, Pakistan, Syria and the USA (Nene *et al.* 1984). The disease mostly occurs in the seedling stage when soil moisture content is high. In irrigated chickpea, the disease can occur at any time. In India, it is more frequent in chickpea planted after the rice harvest when the soil is wet. In Syria, the disease occurs in spring-sown chickpeas but not in the winter-sown crop because the temperature is less than 15°C (Reddy 1983).

The fungus survives as sclerotia and mycelia in colonized organic matter and the propagules are responsible for primary infection. The disease occurs within a temperature range of 18 to 30°C in a soil moisture range of 30 to 80% and at high nitrogen levels (Nene and Reddy 1987). Avoiding high soil fertility should reduce the disease. No specific source of resistance is known.

Pythium root and seed rot (*Pythium ultimum* Trow.) is more common in the kabuli type than in the desi type chickpeas. The disease reduced plant stands in the USA. It also has been reported from India, Iran and Turkey (Nene *et al.* 1984).

The seed rot can occur at temperatures ranging from 10 to 25°C. The incidence of pre-emergence damping-off is significantly less as radicle length increases and shoot growth begins (Kaiser and Hannan 1983). When cultured on natural media, three fungi (*P. ultimum*, *M. phaseolina*, *R. solani*) grew from soil clumps found in chickpea seed lots (Raabe 1985). No pathogens were isolated from the seed, but the soil particles containing these fungi served as inoculum.

Metalaxyl (Ridomil) and captan are very effective seed treatment fungicides for preventing seed rot and pre-emergence damping-off. Metalaxyl at a rate as low as 0.075 g a.i./kg seed is effective and rates as high as 0.6 g a.i./kg seed are nonphytotoxic. Metalaxyl-treated seeds stored at 4°C for up to 464 days before sowing were protected (Kaiser and Hannan 1983). Seed treatment with conidia of *Penicillium oxalicum* was found to significantly reduce seed rot and pre-emergence damping-off caused by *P. ultimum* (Kaiser and Hannan 1984).

Stunt

Stunt caused by the bean leaf roll virus (BLRV) is the most important viral disease of chickpea. The disease has been observed in Algeria, Bangladesh, Ethiopia, India, Iran, Lebanon, Morocco, New Zealand, Pakistan, Spain, Sudan, Tunisia, Turkey and the USA (Nene *et al.* 1984). It is particularly serious in northern India, Pakistan, Ethiopia and Tunisia. Infection during the early stages of plant growth leads to a total loss. Affected plants are stunted with discolored foliage, which is more pronounced in desi (reddish) than in kabuli (yellow) type because of the presence of anthocyanin pigments. If plants survive up to the podding stage, very few pods are produced. Many plants dry prematurely (Nene *et al.* 1978).

The BLRV is a phloem-specific virus and it is not mechanically transmissible. It is not seed-borne. It has a wide host range, mainly leguminous plants such as beans (*Phaseolus vulgaris* L.), faba beans (*Vicia faba* L.), lentils (*Lens culinaris* Medikus), peas (*Pisum sativum* L.), chickpea and clovers. Alfalfa, which is usually symptomless, is a reservoir of the virus. The virus is vectored by *Acyrthosiphon pisum* Harris and *Aphis craccivora* Koch. The latter was found to be responsible for the spread of the stunt in chickpea in Iran and India (Kaiser and Danesh 1971; Nene and Reddy 1976). *Aphis craccivora* extensively colonized *Tribulus terrestris*, a common weed that also harbors the virus (ICRISAT 1988).

ICRISAT scientists have identified five kabuli lines that possess a moderate resistance to stunt. Delay in the date of sowing reduces the disease in northern India.

Nematodes

Many species of parasitic nematodes of chickpea have been reported (Nene *et al.* 1984), but only a few cause severe damage to the crop. Among them, the root-knot nematodes *Meloidogyne incognita* (Kofoid and White) and *M. javanica* (Treub) Chitwood are of economic importance in India and Nepal (S.B. Sharma, pers. comm.) and *M. artiellia* Franklin in Syria, Spain and Italy (Greco 1987). A cyst nematode (*Heterodera* sp.) population that differed from other cyst nematodes reported on chickpea was observed in chickpea fields (ICRISAT 1986). The cyst nematode *Heterodera ciceri* and the lesion

nematode, *Pratylenchus thornei* Sher and Allen, have caused marked yield losses in Syria (Greco 1987). The reniform nematode, *Rotylenchus reniformis* Linford and Oliveira, is also a common pathogen of chickpea.

Soil solarization helps to reduce nematode populations in the soil (Chauhan *et al.* 1988) and crop rotation also may reduce them. Chemical control is uneconomical. The use of resistant cultivars will help in reducing the damage caused by nematodes. No chickpea line was found resistant to *P. thornei* in Syria (Greco *et al.* 1988). Few lines resistant to *H. ciceri*, *M. incognita*, *M. javanica* and *R. reniformis* have been identified.

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