Strategies for Management of Foliar Diseases of Chickpea

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Abstract

Ascochyta blight (AB), botrytis gray mold (BGM), alternaria blight (ALB), rust, and stemphylium blight (SB) are important foliar diseases of chickpea. Foliar diseases occur in areas that have the highest potential for chickpea production due to a long growing season and lack of drought stress. Thus good growing conditions for chickpea and occurrence of foliar diseases are linked and, unless the linkage is broken, there is very little chance of increasing chickpea production in the northern latitudes.

Among the foliar diseases, serious attempts have only been made to develop control measures for AB. High and stable genetic resistance, especially in the podding stage, is lacking in the available germplasm. Integration of host-plant resistance with foliar fungicidal sprays is effective and feasible, but needs wider testing and evaluation.

Limited screening for BGM resistance and observations on disease epidemics indicate that it may be difficult to obtain a sufficient level of genetic resistance for exploitation in the management of the disease. There appears to be some scope for manipulation of plant geometry (including intercropping) and crop maturity for the management of the disease. It is essential to integrate the control measures for AB, BGM, and other foliar diseases, as the incidence of these diseases can overlap in certain areas.

The epidemiology of the diseases is not fully understood and this information is essential for developing effective management practices. Germplasm enhancement for resistance and studies on genetics of resistance and pathogenic variability also should receive better attention.

Résumé

Stratégies de gestion des maladies foliaires du pois chiche : *La flétrissure ascochytique, la pourriture grise due à botrytis, la flétrissure causée par alternaria, la rouille et la pourriture due à stemphylium sont d'importantes maladies foliaires du pois chiche. Les maladies foliaires se produisent dans des régions qui ont le meilleur potentiel de production de pois chiche, en raison d'une saison de culture longue et de l'absence de stress hydrique. Il existe donc un lien entre de bonnes conditions de culture du pois chiche et l'apparition de maladies foliaires. Par conséquent, il ne semble guère possible d'accroître la production de pois chiche dans les latitudes nord à moins de briser ce lien.*

Parmi les maladies foliaires, seule la flétrissure ascochytique a fait l'objet d'efforts sérieux de mise au point de moyens de lutte. Il n'existe pas de sources de résistance élevée et stable, particulièrement au stade de formation des gousses, dans le matériel génétique disponible

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actuellement. L'intégration de la résistance de la plante-hôte avec la pulvérisation de fongicides sur les feuilles est efficace et faisable mais exige davantage d'essais et d'évaluations.

Un criblage restreint pour la résistance à la pourriture grise due à botrytis et des observations sur les épidémies de la maladie indiquent qu'il pourrait être difficile d'obtenir un niveau suffisant de résistance génétique pour servir à la lutte contre la maladie. Il semble possible de manipuler la géométrie des plantes (y compris la culture associée) et la maturité de la culture pour maîtriser la maladie. Il est essentiel d'intégrer des moyens de lutte contre la flétrissure ascochytique, la pourriture grise due à botrytis, et d'autres maladies foliaires, car l'incidence de ces dernières peut se chevaucher dans certaines régions.

L'épidémiologie des maladies n'est pas encore complètement comprise et cette information est essentielle pour mettre au point des pratiques de gestion efficaces. L'amélioration du matériel génétique pour la résistance ainsi que les études sur la génétique de la résistance et la variabilité pathogénique devraient également recevoir davantage d'attention.

Chickpeas suffer from some serious foliar diseases. In the order of importance worldwide, these are ascochyta blight (*Ascochyta rabiei* [Pass.] Labr.), botrytis gray mold (*Botrytis cinerea* Pers. ex Fr.), stemphylium blight (*Stemphylium sarciniforme* [Cav.] Wilts.), alternaria blight (*Alternaria alternata* [Fr.] Kiessler), and rust (*Uromyces ciceris-arietini* [Grogn.] Jacz. & Beyer). The incidence of these diseases is mainly confined to the chickpea-growing regions between latitudes 25° and 45°, where the weather is cooler and wetter than in growing regions at lower latitudes. As the higher latitude areas have the greater production potential, management of foliar diseases is important for increasing chickpea production.

Research on chickpea diseases has recently been reviewed by Nene and Reddy (1987). Here, we attempt to summarize progress made during the past 10 years, identify the gaps in knowledge as well as research constraints, and to suggest research strategies for the future.

Ascochyta Blight

Ascochyta blight (AB) is most serious between the latitudes 30° and 45°, where relatively low temperatures (15°-25°C) prevail during the crop season and favor its development. Appearance of the blight however is not regular. The disease develops whenever the winter-sown chickpeas in northwest India and Pakistan and spring-sown chickpeas in the Mediterranean region receive rains during the crop season. There is no AB problem if there are no rains but then drought reduces the yield. A good season for the chickpea crop is also favorable for AB and low yields result (Fig. 1). This relationship will have to be considered when we develop effective disease management strategies.

The average yield of spring-sown chickpeas in the Mediterranean region is low (about 0.75 t ha^{-1}) mainly due to drought and heat stress. Advancing the sowing date into autumn results in 50-100% yield increase provided AB is controlled (Hawtin and Singh 1984). Hence control of AB is essential for increasing chickpea production in the countries in the Mediterranean region but also in the major chickpea-producing regions in India and Pakistan.

Botrytis Gray Mold

Botrytis gray mold (BGM) causes concealed damage in chickpea, and its importance has only recently been realized. Without visible symptoms on foliage, the disease can cause flower drop resulting in poor pod setting, and extension of the crop duration. Some pod setting may occur late in the season when the day temperatures exceed 30°C and conditions are unfavorable for the disease. But the yields in such situations

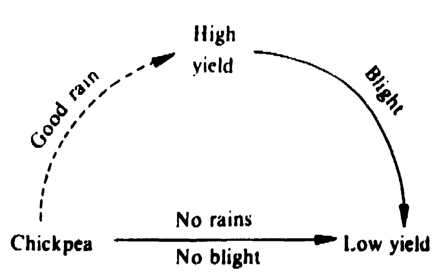

Figure 1. Relationship between chickpea yields and ascochyta blight (*Ascochyta rabiei*).

drastically reduced owing to drought and heat ..ss. The regions between 25° and 30° are most liable BGM. The disease has a slightly higher temperature uirement (25°C) than AB (20°C). It is a regular blem in parts of India, Nepal, and Bangladesh. ring 1979-1982 it caused heavy losses in the Indo-ngetic Plains of India (Grewal and Laha 1983). ldy et al. (1988) visually estimated about 40% yield s due to BGM in Nepal during the 1987/88 season. ough winter rains increase the disease problem they not seem to be essential for BGM development. This different from the situation for AB, for which rains essential. Heavy dew in the nights, irrigation, essive vegetative growth, early sowing, and dense nting predispose the crop to disease.

Other Diseases

Stemphylium blight, alternaria blight, and rust are at present of minor importance. Stemphylium blight causes considerable damage in northwestern parts of Bangladesh in some seasons. Alternaria blight occasionally assumes importance in northeastern India. Rust is more widespread and frequent, but as it occurs late in the season, it does not cause much loss in yield. Hardly any work has been carried out on the management of these diseases, probably because they have been somewhat over-shadowed by AB and BGM. Once these two major diseases are controlled, the potential damage caused by the minor diseases may be better realized.

ble 1. Mean ascochyta blight severity and yield loss estimation[1] under noninoculated and artificially inoculated nditions in a set of resistant chickpea germplasm lines at ICARDA, Tel Hadya, Syria, 1982/83–1985/86.

ickpea mplasm es	Blight severity on vegetative parts[2]	Pod infection (%)	Average yield (t ha⁻¹)		Yield loss/ increase (%)
			Noninoculated	Inoculated	
C 72	2.3	8	2.0	2.3	+ 15
C 182	2.3	9	2.5	2.6	+ 4
C 187	2.4	5	2.2	2.4	+ 9
C 191	2.5	12	2.1	2.1	0
C 195	2.4	8	2.4	2.4	0
C 200	2.4	2	2.5	2.2	- 12
C 1757	3.3	36	2.6	1.4	- 46
C 2300	2.2	3	2.5	2.5	0
C 2506	2.0	9	2.3	2.7	+ 17
C 2956	2.7	3	2.0	2.3	+ 15
C 3001	2.7	30	1.3	1.8	+ 38
C 3274	2.1	4	2.0	2.1	+ 5
C 3400	2.7	20	2.2	2.1	- 5
C 3634	2.0	16	2.0	2.2	+ 10
C 4200	2.9	29	2.3	1.9	- 19
C 4248	2.9	32	2.3	1.9	- 17
C 5124	2.9	16	2.1	2.2	+ 5
C 6262	2.2	2	2.3	2.6	+ 13
C 6981	2.0	18	2.3	2.6	+ 13
Mean	2.47	13.8	2.2	2.2	0
C 1929 usceptible control)	9.0	94	2.6	0.03	99
SE mean	±0.27	±5.3	±0.21	±0.17	
CV (%)	16.5	53.7	16.0	14.0	

[1] Average of 1982/83, 1984/85, and 1985/86 seasons.
[2] Scored on a scale of 1-9, where 1 = free and 9 = killed.

urce: M.V. Reddy and K.B. Singh, unpublished.

Progress to Date

Ascochyta blight

Though work on ascochyta blight has been going on for over 80 years, progress towards managing the disease has not been satisfactory. Several effective seed dressing and foliar fungicides have been identified, but their application under field conditions has been neither feasible nor economical. As many as 12 foliar sprays were insufficient to control the disease in a susceptible variety under epiphytotic conditions at the International Center for Agricultural Research in the Dry Areas (ICARDA) in Syria. Fungicides are often needed when it is raining or drizzling and it is not practicable to spray. The resistant varieties released from time to time in India and Pakistan eventually became susceptible due to the appearance of new races of the pathogen (FAO 1963). The extensive resistance breeding work undertaken in the ICARDA-ICRISAT joint program over the past 10 years has helped in the identifica and development of several blight-resistant, hi yielding kabuli varieties for the Mediterranean re (Tables 1 and 2). However, none was sufficie resistant in India and Pakistan, the two major chick producing countries (Table 3). It is now well-establis that the fungus *Ascochyta rabiei* is highly variable, that the races present in Pakistan and India are m aggressive than those prevalent in the Mediterran region (Singh et al. 1984). Lines with resistance in vegetative stage to isolates of *A. rabiei* prevalent in In (Singh et al. 1988) and Pakistan are available ne has resistance in both the vegetative and podding sta PK 51863 x NEC 138-2 the only line resistant un field conditions in Pakistan (Iqbal et al. 1989), show resistance in the vegetative stage to Indian isolates of *rabiei* but not in the podding stage (Table 3).

Reliable inoculation techniques and disease rati scales have been developed and standardized (Reddy al. 1984). A system for multilocational evaluation h

Table 2. Ascochyta blight resistant and high-yielding kabuli chickpea cultivars released in the Mediterranean regio

Country	Cultivars released	Year of release	Specific features
Algeria	ILC 482	1988	Wide adaptation
	ILC 3279	1988	Tall
Cyprus	Yialousa (ILC 3279)	1984	Tall
	Kyrenia (ILC 464)	1987	Large seeds
France	TS1009 (ILC 482)	1988	Wide adaptation
	TS1502 (FLIP-81-293)	1988	Cold tolerance
Italy	Califfo (ILC 72)	1987	Tall
	Sultano (ILC 3279)	1987	Tall
Morocco	ILC 195	1987	Mid-tall
	ILC 482	1987	Wide adaptation
Spain	Fardan (ILC 72)	1985	Tall
	Zegri (ILC 200)	1985	Mid-tall
	Almena (ILC 2548)	1985	Tall
	Alcazaba (ILC 2555)	1985	Tall
	Atalaya (ILC 200)	1985	Mid-tall
Syria	Ghab 1 (ILC 482)	1986	Wide adaptation
	Ghab 2 (ILC 3279)	1986	Tall
Tunisia	Chetoui (ILC 3279)	1986	Tall
	Kassab (FLIP 83-46C)	1986	Large seeds
Turkey	ILC 195	1986	Mid-tall
	Gunej Sarisi (ILC 482)	1986	Wide adaptation

Source: K.B. Singh, personal communication.

Table 3. Evaluation of *Cicer* spp accessions for resistance against five isolates of *Ascochyta rabiei* in a growth room at ICRISAT Center, 1988.

	Blight score[1]					
icer accession	CPK 2 isolate	C 235 isolate GH	E 100Y isolate	Gurdaspur isolate	IARI isolate	Mixture of isolates
CC 202 (USSR)	4 (100)[2]	5 (100)	9 (100)	9 (100)	6 (50)	8 (100)
CC 3996 (Iran)	4 (33)	5 (100)	9 (100)	6 (-)[3]	6 (33)	5 (100)
LC 72 (USSR)	5 (45)	3 (93)	5 (100)	6 (50)	6 (77)	6 (69)
LC 3279 (USSR)	4 (0)	4 (59)	4 (89)	6 (85)	6 (100)	5 (75)
LC 249 (India)	4 (0)	4 (100)	7 (100)	5 (100)	6 (100)	7 (100)
CC 1903 (Morocco)	4 (33)	5 (100)	8 (90)	6 (80)	8 (100)	7 (100)
13 (ISRAD)	3 (25)	5 (100)	6 (100)	7 (100)	7 (89)	6 (91)
C 26435 (Morocco)	6 (0)	6 (80)	7 (86)	9 (100)	9 (57)	7 (25)
CC 51276 (India)	4 (44)	3 (94)	6 (100)	4 (75)	7 (91)	5 (100)
235 (India)	3 (67)	5 (100)	5 (100)	7 (100)	7 (100)	6 (71)
138 (Mexico)	5 (0)	8 (100)	9 (100)	9 (100)	9 (100)	9 (71)
LC 191 (USSR)	5 (0)	7 (75)	7 (67)	7 (86)	6 (64)	6 (80)
LC 2380 (USSR)	3 (13)	4 (94)	6 (100)	4 (43)	6 (93)	6 (100)
CH 128 (Morocco)	4 (9)	6 (73)	6 (58)	5 (85)	8 (-)	7 (100)
M 72 (Pakistan)	4 (0)	7 (100)	8 (100)	8 (50)	8 (40)	8 (100)
100YM (India)	5 (-)	4 (83)	6 (-)	8 (-)	6 (100)	6 (-)
CC 607 (India)	3 (0)	6 (100)	5 (90)	9 (100)	8 (100)	5 (100)
G 261 (India)	5 (13)	5 (70)	6 (100)	9 (78)	8 (55)	7 (100)
G 575 (India)	6 (33)	6 (100)	4 (100)	4 (100)	5 (100)	4 (100)
B 7 (India)	5 (38)	7 (100)	7 (100)	9 (100)	6 (92)	7 (100)
ANT-G-82-1 (India)	5 (67)	5 (92)	4 (69)	6 (44)	7 (100)	3 (89)
EC-138-2 (India)	3 (33)	6 (82)	5 (94)	6 (83)	7 (75)	5 (83)
LC 195 (USSR)	5 (0)	5 (67)	5 (78)	6 (91)	6 (-)	6 (52)
LC 482 (Turkey)	4 (0)	5 (100)	6 (94)	6 (86)	4 (54)	5 (75)
judaicum	4 (-)	4 (100)	6 (100)	4 (0)	7 (100)	6 (75)
reticulatum	4 (50)	7 (100)	8 (100)	6 (86)	7 (100)	7 (-)
-100Y (HAU)	6 (0)	3 (88)	6 (100)	5 (67)	5 (100)	6 (50)
K51825xCM 72 (Pakistan)	3 (9)	3 (91)	6 (100)	3 (50)	7 (67)	5 (70)
K51832xCM 72 (Pakistan)	5 (0)	5 (100)	5 (100)	5 (90)	7 (100)	6 (90)
K51835xCM 72 (Pakistan)	6 (0)	5 (69)	4 (100)	5 (75)	5 (91)	5 (89)
K51863xNEC-138-2 (Pakistan)	4 (0)	3 (65)	4 (100)	3 (44)	4 (67)	4 (73)

1. Scored on a scale of 1-9, where 1 = free and 9 = killed.
2. Figures in parentheses are % pod infection.
3. (-) = no podset.

Source: M.V. Reddy et al., unpublished.

also been built up (Singh et al. 1984). The potential for integration of host-plant resistance and a limited number of foliar sprays in the management of the disease is shown in Table 4. Two foliar sprays of chlorothalonil at the seedling and early podding stage in a moderately resistant cultivar were most cost-effective (Reddy and Singh, 1990). Limited information on the genetics of resistance to blight exists (Singh and Reddy 1983, 1989). Some information on the development of disease in relation to humidity and temperature has been obtained (Fig. 2). Temperature is a more critical factor than humidity for the epidemic build-up of AB in winter-sown chickpeas in the Mediterranean region where usually the required humidity exists.

Botrytis Gray Mold

Compared to AB, very little work has been carried out on BGM. During the past 5 years, there have been some

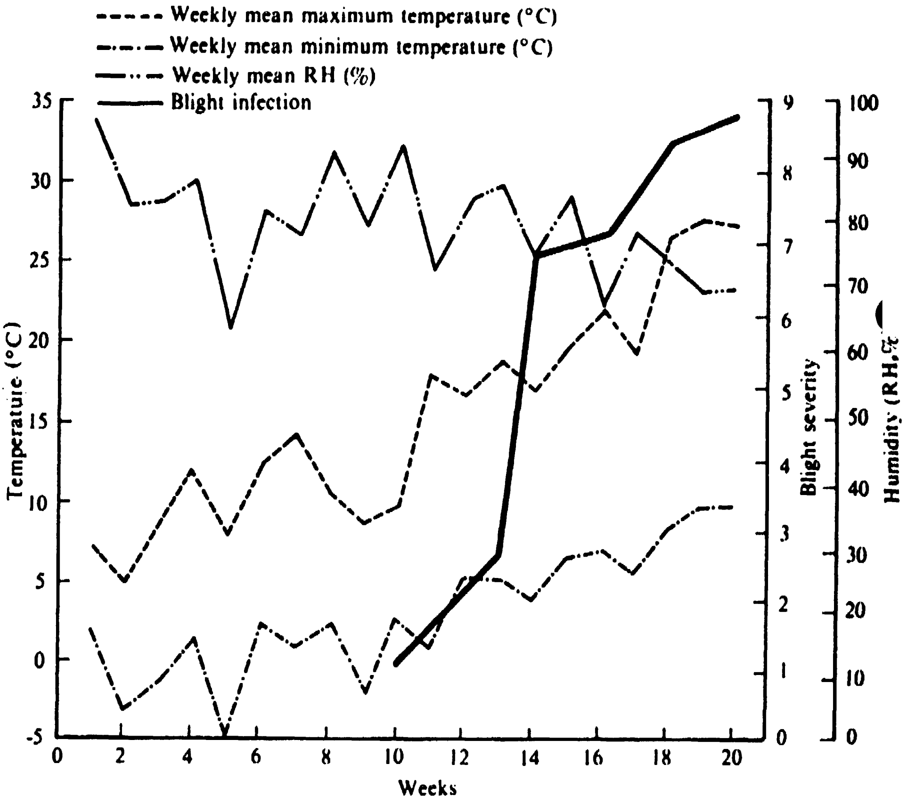

Figure 2. Relationship between temperature, humidity, and development of ascochyta blight infection in chickpea genotype ILC 464, Tel Hadya, Syria, 1982/83.

Table 4. Cost-benefit ratio of two foliar applications of chlorothalonil (Bravo 500®) for control of ascochyta blight in moderately resistant chickpea cultivar ILC 482, Tel Hadya, Syria, 1982/83–1985/86.

Stages of the crop when sprayed with chlorothalonil	Yield ($t\ ha^{-1}$)	Value of additional produce (U.S.$)[3]	Cost-benefit ratio[4]
Seedling and early podding	2.52[1]	209.0	1:5
Mid vegetative and early podding	2.10[2]	159.5	1:4
Seedling and late podding	2.25[1]	115.9	1:3

1. Average of two seasons.
2. Average of three seasons.
3. At the rate of U.S.$ 0.35 kg^{-1} of chickpea seed.
4. Cost of two sprays of chlorothalonil at U.S.$ 38.5 ha^{-1}.

Source: M.V. Reddy and K.B. Singh (1990).

reports on identification of lines with field-resistance to the disease (Table 5) (Rathi et al. 1984; Shukla et al. 1987; Sahu and Sah 1988). However, it appears that the reactions of the lines depend very much on disease pressure. Almost all lines that showed resistance to the disease at Pantnagar (latitude 29°N) in northern India, where the disease pressure is usually moderate, showed high susceptibility, when tested at Rampur (latitude 27°N) in Nepal, where the incidence is much higher. However, several showed some promise at Nepalganj (latitude 28°N) in Nepal, where compared to Rampur the disease pressure is moderate. Whether this variation is also due to different races, or only to environmental factors needs to be investigated. Several seed dressing and foliar fungicides have been found effective (Table 6) (Grewal and Laha 1983; Singh and Bhan 1986a; Singh and Kaur, unpublished), but, the economics of the use of foliar fungicides for management of the disease needs to be worked out. Singh and Bhan (1986b) reported four physiologic races from states in northern India. Observations made at ICRISAT Center also indicate variability in the pathogen. Field observations have shown that kabuli types are comparatively less susceptible than desi types. Also the tall and compact types suffer less than the traditional bushy and spreading types.

Table 5. Chickpea lines resistant to gray mold at Ludhiana, Punjab, India (1982/83–1988/89).

Variety	Average[1] disease score[2]
GL 635	3
GL 699	3
GL 907	3
GL 926	3
GL 930	3
GL 84133	5
GL 84038	4.5
GL 84065	4.5
GL 84212	5
GL 86094	4
ILC 200	5
ICC 1903	4
ICC 1905	4.5
ICC 4000	5
ICC 4018	5
ICC 4065	5
ICC 4950	5
ICC 5033	5
P 1528-1	5
C 8	5
Cicer pinnatifidum	1

1. Average of 6 seasons.
2. Scored on a scale of 1-9, where 1 = free and 9 = killed.

Source: Gurdip Singh, unpublished.

Gaps in Knowledge and Constraints

Ascochyta Blight

The major gap in our knowledge of the disease is in the AB epidemiology. The fungus is known to survive in the seeds and infected debris, and it is also known that its ability to survive in debris under field conditions is less than 2 years. No alternative hosts have been found. The recurrence of the disease in severe form, over extensive areas, after gaps of up to 10 years is quite puzzling. The likelihood of diseased debris or infected seed serving as sources of inoculation in such cases is remote. The logical explanation for such epidemics is that long-distance dispersal of spores has occurred. Hard evidence for this possibility is lacking. The role of the teleomorph in the epidemiology of blight in the Palouse region of USA by long-distance (>8 km) dissemination of airborne ascospores has been recently reported by Kaiser and Muehlbauer (1988). Lack of information on the nature of pathogenic variability in *A. rabiei* is yet another major gap in our knowledge. The major constraint in the management of AB is the lack of strong and stable genetic resistance, also in wild *Cicer* spp.

Botrytis Gray Mold

The epidemiology of the disease, especially the relationship between temperature, humidity, and disease development under field conditions is not understood. High levels of genetic resistance are not available. The pathogenic variability in *B. cinerea* and the distribution of races needs further investigation. The influence of sowing date, plant population, irrigation, crop duration, intercropping, and crop rotation on disease buildup is also not fully comprehended.

Future Research Strategies

Ascochyta Blight

The suggested future research strategies for the management of ascochyta blight are listed below:

1. Concerted efforts should be made to fully under-

Table 6. Effect of seed treatment and one foliar spray on the severity of botrytis gray mold in chickpea, Ludhiana, India.

Fungicide	Dose (gL^{-1} or $g\ kg^{-1}$)	Disease score[1]		
		1986/87	1987/88	Mean
Bavistin®	2	1.3	2.5	1.9
Hexacap®	3	1.0	1.6	1.3
Dithane M.45®	3	1.0	1.5	1.2
Rovral®	3	2.0	2.5	2.2
Thiabendazole	2	1.6	1.3	1.4
Baytan®	2	1.3	1.6	1.4
Thiram®	3	1.6	1.5	1.[illegible]
Ronilan®	3	2.3	1.7	2.0
Bayleton®	2	1.3	1.7	1.5
Bavistin® + Thiram® (1:1)	3	1.6	2.0	2.1
Tópsin-M®	3	1.6	2.4	2.0
Seed treated (Bavistin® + Thiram®)	3	1.3	2.2	1.7
Control (Seed untreated)	-	6.3	6.3	6.4
CD at 5%		1.4	0.2	0.7

1. Scored on a scale of 1-9, where 1 = free and 9 = killed.

Source: Gurdip Singh and Livinder Kaur, unpublished.

stand the epidemiology of the disease. The primary sources of inoculum for epidemic buildup should be identified. The role of alternative hosts and resting forms of the fungus, if any, in addition to the infected seed, diseased debris, and teleomorph, in the perpetuation of the disease needs to be investigated. The means by which the disease spreads rapidly over very large areas need to be understood.

2. The extent of variability in *A. rabiei*, and its distribution, and the means by which the variability occurs, need to be further investigated. Temperature appears to play some role in the development of variability. Sangwan (1989) showed that certain isolates of *A. rabiei* produce more sectoring at 25°C than at 20°C in petri dishes.
3. Further studies on the genetics of resistance to blight are needed for mapping of the resistance genes, which will help resistance breeding programs.
4. Germplasm enhancement for blight resistance is important. In the absence of complete information on the different genes available for blight resistance in chickpea, intercrossing lines with different types of resistance, and lines resistant to different races of *A. rabiei* can be undertaken. A single dominant gene was found to govern resistance in ILC 72, ILC 183, ILC 200, ILC 202, ILC 2956, ILC 3279, and ICC 4935, whereas resistance in ILC 191 was conferred by a single recessive gene (Singh and Reddy 1983, 1989). In all the former lines the same gene was found to confer resistance, but they did not show the same resistance pattern against a set of races (Singh and Reddy 1990). Although there are no lines resistant to all races, in both the vegetative and podding stage, lines resistant to individual races are available (Table 7) (Singh and Reddy 1989). Crossing and selection among such lines may result in enhanced resistance.
5. In the past there has been hardly any work on the integrated management of the disease. Most efforts have been on the development of resistant varieties and fungicidal control. Effective seed dressing fungicides such as thiabendazole and Calixin M ® are now available. One or two foliar sprays of chlorothalonil during the podding stage in moderately resistant cultivars have been found very effective in the control of the disease. More effective systemic foliar fungicides with longer residual action will make their application more economical and practical. Taller and more compact genotypes with stronger stems that would not easily break at the site of lesions, and with terminal, exposed pods

Table 7. Chickpea lines showing resistance to several races of *Ascochyta rabiei* in a greenhouse study in Syria.

Genotype	Reaction to races[1]					
	Race 1	Race 2	Race 3	Race 4	Race 5	Race 6
ILC 72	R	R	R	R	S	S
ILC 190	R	S	R	S	R	S
ILC 201	R	R	S	R	R	S
ILC 202	R	R	R	R	R	S
ILC 482	R	R	S	S	R	S
ILC 2506	R	R	S	R	S	R
ILC 2956	R	S	R	S	R	R
ILC 3279	R	S	R	R	R	S
ILC 3856	R	R	R	R	S	R
ILC 5928	R	R	S	R	R	R
FLIP 83-48C	R	R	R	S	R	S
ICC 3996	R	R	R	S	S	S
No. of resistant lines	12	9	8	7	8	4

1. R = resistant (3 to 4 rating);
S = susceptible (6 to 9 rating) on a 1-9 scale, where
1 = free and 9 = killed.

Source: Singh and Reddy 1989.

may also help in minimizing pod infection. Work carried out at ICARDA has shown, that when tall genotypes are mechanically bent to the ground during the podding stage, they develop increased pod infection. Thus, combined use of tall and compact varieties with resistance to blight, use of seed-dressings, and limited foliar sprays of effective systemic fungicides in the podding stage should be helpful in the successful management of blight.

Botrytis Gray Mold

Extensive screening in India and Nepal has failed to identify high levels of genetic resistance in the chickpea germplasm. Lines such as ICC 1069, ICC 1913, ICC 3640, ICC 4954, ICC 6299, and ICC 7111, that showed good promise of resistance at Pantnagar over a period of 5 years were almost completely killed at Rampur in Nepal. Though some lines do not show much damage on vegetative parts, they suffer severe flower infection resulting in no pod formation. In areas where the disease pressure is moderate, there appears to be good scope for integrated management of the disease. A field experiment at Pantnagar during the 1988/89 season indicated that the disease incidence was much lower in a tall and compact genotype (ICCL 87322) than in a bushy and spreading type (H 208) (Table 8). In both genotypes, there was a large increase in yield when interrow spacing was increased from the normal 30 cm to 60 cm, keeping the plant population constant. Intercropping experiments at Nepalganj in Nepal also indicated that when chickpea was intercropped with linseed there was a marginal increase in chickpea yield, and the linseed yield was a bonus (Onkar Singh 1988). Plant architecture changes suggested for reducing susceptibility of AB should also be useful in the management of BGM. Thus combined use of genetic resistance in the tall and compact plant type background, increased interrow spacing and intercropping with crops such as linseed, and a limited number of foliar fungicide sprays during the flowering and podding period may prove very effective in the management of this disease.

Germplasm enhancement and utilization of related wild species are also suggested as promising avenues to attain resistance that could be used in the management of the disease.

Other Diseases

Some of the strategies adopted for management of AB and BGM such as wider row spacing and use of tall and

Table 8. Influence of growth habit of chickpea genotypes and inter and intra-row spacing on botrytis gray mold severity and grain yield, Pantnagar, 1988/89.

Treatments[1]	Disease score on 1-9 scale[2]		Yield ($t\ ha^{-1}$)
Sprayed with Ronilan®			
ICCL 87322 (30×10cm)	4.3	(1.5)[3]	3.5
ICCL 87322 (60×5cm)	3.3	(1.2)	5.2
H 208 (30×10cm)	6.7	(1.9)	2.8
H 208 (60×5cm)	5.0	(1.6)	3.1
Mean	4.8	(1.6)	3.7
No spray			
ICCL 87322 (30×10cm)	5.0	(1.6)	3.4
ICCL 87322 (60×5cm)	4.1	(1.4)	4.5
H 208 (30×10cm)	8.0	(2.1)	1.0
H 208 (60×5cm)	7.1	(1.9)	1.9
Mean	6.1	(1.8)	2.7
SE	±0.9	(0.2)	±0.14
CV%	15.8	(10.0)	4.3

1. ICCL 87322 is tall and compact and H 208 is bushy and spreading.
2. Scored on a scale of 1-9, where 1 = free and 9 = killed.
3. Figures in parentheses are $\log_e$ values.

Source: M.V. Reddy et al. unpublished.

compact genotypes may also help in minimizing other diseases. For example, in Bangladesh, tall, compact genotypes suffer much less from stemphylium blight than do the traditional spreading genotypes. Search for sources of genetic resistance may prove fruitful. Kabuli types were found to suffer less from stemphylium blight in Bangladesh than did desi types.

Multiple Disease Management

It is essential to develop management practices effective against combinations of foliar diseases. Though the two major diseases have specific zones of occurrence, there are certain regions such as northwestern Uttar Pradesh and Punjab in India, and Punjab in Pakistan where these overlap in certain seasons. Stemphylium and ascochyta blights generally occur along with botrytis gray mold. Unless the research efforts on foliar diseases are considerably increased, it will not be easy to develop management practices for all the foliar diseases in chickpea. Development of cold-tolerant chickpea cultivars that can mature by the end of t winter (end of February) in India, Pakistan, Bangl desh, and Nepal when the temperatures are lower th 15°-25°C may help in avoiding most of the foli disease problems as the low temperatures will not fav epiphytotics.

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