Disease Resistance Breeding in Chickpea

Proceedings of the Consultative Meeting on Breeding for Disease Resistance in Kabuli Chickpea, 6-8 March 1989, Aleppo, Syria

K.B. Singh
M.C. Saxena

editors

International Center for Agricultural Research in the Dry Areas

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International Crops Research Institute for the Semi-arid Tropics

International Center for Agricultural Research in the Dry Areas

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Foreword

The International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), India has the world mandate for the improvement of chickpea, whereas the International Center for Agricultural Research in the Dry Areas (ICARDA), Syria has the regional mandate for the improvement of kabuli chickpea in West Asia and North Africa. Both centers conduct research with the common objectives of increasing and stabilizing chickpea production in developing countries. Among the different stress factors responsible for destabilizing chickpea production, diseases are the most important.

Although over 50 pathogens infest chickpea, ascochyta blight and fusarium wilt are the most damaging. ICRISAT has conducted in-depth studies on fusarium wilt, and ICARDA, in cooperation with ICRISAT, on ascochyta blight. To review the research on combating these and other diseases, the two centers held a consultancy meeting, in the period 6-8 March 1989, at ICARDA's headquarters in Aleppo. Specialists from the major chickpea-growing countries of the world discussed strategies for breeding disease resistance in chickpea. This book is a record of their deliberations.

We believe the information given here will motivate chickpea breeders and pathologists to undertake research to develop improved disease-resistant chickpea varieties.

Nasrat R. Fadda
Director General
ICARDA

Preface

Fifty years ago, in 1941, the first disease-resistant chickpea cultivar, F 8, was released for commercial production in British India. Since then many cultivars possessing disease resistance have been bred and released. However, because some factors such as epidemiology and variability in pathogen have not been properly understood, they have not been incorporated into breeding research. Consequently, cultivars initially resistant to a disease later succumb to it. For example, ascochyta blight-resistant cultivars suffer from prolonged conditions favorable to blight, and fusarium wilt-resistant cultivars suffer from other soil-borne diseases that are present with it in the soil. Because of these conditions, disease-resistant cultivars never have been very popular with farmers.

To understand the host-pathogen relationship, a consultancy meeting of breeders and pathologists was called and the problem was debated fully. The discussion focussed on understanding the pathogens, breeding strategies and other methods of disease control. Both strategic and applied research were deliberated and areas for future research were identified.

During this consultancy meeting, it became clear that the use of resistant cultivars is the best method of controlling the diseases of chickpea. Cultural practices seem less effective and the use of chemicals is uneconomical. Another important point emerging from this meeting is that the development of cultivars resistant to several diseases will be a more effective means of controlling the disease than the use of cultivars resistant to a single disease.

It is our belief that the recommendations made in this proceedings will motivate researchers to conduct experiments to solve some of the important issues. Following in-depth discussions on breeding strategies, future cultivars are expected to be more durable.

K.B. Singh
M.C. Saxena
Editors

Current Status and Prospects of Kabuli Chickpea Production

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Abstract

With an annual area of 9.5 million hectares and production of 7.0 million tonnes, chickpea is the third most important pulse crop in the world. It is estimated that about 20% of the total chickpea production is of the kabuli type. This type dominates in the West Asia and North Africa (WANA) region, the source of about 67% of the total global production of kabuli chickpea. Average yields of kabuli chickpea in the WANA region are higher than the global average yield, yet there is considerable scope for improving productivity and yield stability in this region. Winter sowing promises nearly 100% increases in yield if ascochyta blight-resistant and cold-tolerant cultivars are used. The joint ICARDA/ICRISAT Kabuli Chickpea Project has assisted national programs in the on-farm verification of winter sowing and in the release of cultivars for this purpose by national programs. Improved production techniques and cultivars also have been developed for spring sowing. A quick transfer of technology for winter and spring-sown crops would result in increased productivity at the farm level. Future work for improved productivity and yield stability of kabuli chickpea requires the development of more efficient screening and breeding techniques.

Introduction

With an annual area of 9.5 million hectares and an annual production of nearly 7.0 million tonnes (FAO 1988), chickpea (*Cicer arietinum* L.) is the third most important pulse crop in the world after dry beans (*Phaseolus vulgaris* L.) and dry peas (*Pisum sativum* L.). It probably originated in the area of present-day southeastern Turkey and adjoining northern Syria (van der Maesen 1987). Archaeological finds and historical records confirm the antiquity of its domestication in the Old World. From its Mediterranean origin, the crop has spread to other geographical regions in the world because of the versatility of its adaptation to environments and its wide acceptability, mainly as a human food and partly as animal feed.

Of the several intraspecific classifications proposed in chickpea the simplest, and perhaps the oldest, is based on seed color and to some extent on seed size. Thus, the two super cultivar groups kabuli and desi are commonly recognized; the former group has large cream-colored seeds and the latter group is small-seeded and has dark-colored

seeds (van der Maesen 1987). As noticed and reported early on by Vavilov (1926), kabuli cultivars dominate around the Mediterranean basin, whereas the desi type predominates in the Indian subcontinent. This paper mainly deals with the kabuli chickpea, a mandate crop for ICARDA and ICRISAT.

Importance and Uses of Chickpea

Chickpea is an important source of human food and animal feed and it plays a key role in the maintenance of soil fertility in the wheat-based systems of the dry rain-fed areas of the West Asia and North Africa (WANA) region. For human consumption, chickpea is used as a whole green or dry matured seed, dhal (decorticated dry-split cotyledons) and flour. Chickpea-based foods traditionally are prepared using methods such as soaking, sprouting, boiling, roasting, parching, frying and steaming. Chickpea blends well with vegetables, meat and sauces and can be used to make a variety of sweets and pastries. Nutritionally, the importance of chickpea in human food stems from the high protein content of its seeds, a better protein digestibility than several other pulses, no hemagglutinating activity and low antitrypsic activity. The amino acid composition of chickpea protein complements that of cereals and, therefore, chickpeas and cereals are an integral part of several traditional foods in the WANA and south Asia regions.

Dry chickpea seeds also are used for compound animal feed and can be an ideal substitute for imported soybean meal in the WANA region. The straw, although nutritionally inferior to that of lentil, serves as a valuable fodder for stall-feeding of animals and can be used for making pelleted feed.

The significance of chickpea in the cropping system of the dry rain-fed areas in WANA, and possibly elsewhere, is due to its ability to fix atmospheric nitrogen, which may exceed 70 kg/ha (Saxena 1988), and to leave the soil in a more productive state for a subsequent cereal crop (ICARDA 1981; Papastylianou 1987; Sharma and Jodha 1984; Suzuki and Konno 1982). It also serves as a 'break' in continuous cereal rotations.

Area, Production and Yield

Area, production and yield statistics in the FAO Production Year Books report for the whole crop with no distinction made for the kabuli and desi types. Changes in area, production and yield of chickpea in different countries of the world over a period from 1979/81 to 1985/87 are shown in Table 1. There is a clear trend for increases on a global basis. Area increased from 9.53 million hectares in 1979/81 to 10.03 million hectares in 1985/87, production from 5.97 million to 7.06 million tonnes and yield from 624 to 704 kg/ha.

Regionally, South Asia contributes most to the total chickpea production, followed by West Asia, Central and South America, East Africa and North Africa. Recent increases in area and production have been most conspicuous in West Asia and North Africa, with Turkey registering more than 100% increase, whereas in South Asia little change has occurred.

Table 1. Changes in area, production and yield of chickpea over a period of 9 years (1979/81 to 1985/87) in different countries†.

Country	Area (1000 ha)		Production (1000 Mt)		Yield (kg/ha)	
	1979/81‡	1985/87‡	1979/81	1985/87	1979/81	1985/87
North Africa	169	194	94	118	556	608
Algeria	42	63	16	17	392	274
Egypt	7	9	11	15	1538	1571
Morocco	53	81	38	55	623	676
Tunisia	67	41	29	31	441	748
East Africa	181	240	135	155	746	645
Ethiopia	154	180	128	135	829	750
Tanzania	27	60	7	20	275	333
South Asia	8341	8431	4989	5690	598	675
Bangladesh	57	48	40	37	701	778
Burma	127	177	79	154	607	870
India	7092	7163	4474	4935	627	689
Pakistan	1065	1043	396	564	372	541
West Asia	354	724	366	720	1034	994
Iran	48	101	52	73	1092	722
Iraq	20	16	12	12	633	771
Jordan	2	2	1	1	601	530
Lebanon	2	2	3	3	1150	1275
Palestine	4	4	4	6	1302	1636
Syria	65	67	49	40	722	602
Turkey	213	532	245	585	1149	1099
South Europe	158	131	98	90	614	689
Greece	15	5	15	5	1044	1160
Italy	14	9	16	10	1181	1148
Portugal	35	25	12	12	330	493
Spain	92	90	53	60	564	703
Central & South America	243	192	244	205	1000	1000
Argentina	5	3	4	3	814	1000
Chile	18	15	9	11	506	846
Mexico	195	150	218	177	1101	1178
Peru	3	3	2	2	778	828
Oceania	0	34	0	36	0	1058
Others	84	84	45	47	536	559
Total	9530	10030	5971	7061	624	704

† From FAO (1988).
‡ Average of three years.

The FAO statistics (Table 1) and a best guess of the proportion of the total area devoted to kabuli chickpea (Chandra 1985; Jodha and Subba Rao 1988), were used to provide an estimate of area and production of kabuli chickpea in different regions of the world (Table 2). It appears that about 16% of global chickpea area and about 19% of production are contributed by the kabuli type, with West Asia and North Africa together accounting for nearly 63% of the area and 67% of the production of the kabuli type chickpea. ICARDA, with its regional mandate to serve the dry rain-fed areas of WANA, is therefore well placed to undertake research on the improvement of kabuli chickpea.

Table 2. Estimate of area and production of kabuli chickpea in different regions based on the FAO statistics for all chickpeas, 1985/87.

	Area			Production (1000 Mt)	
		Kabuli			
Region	Total 1000 ha	%†	1000 ha‡	Total	Kabuli‡
North Africa	194	99	192	118	117
East Africa	240	10	24	155	15
North, Central America	150	50	75	177	89
South America	42	100	42	28	28
South Asia	8431	5	422	5690	285
West Asia	724	99	717	720	713
South Europe	131	100	131	90	90
Oceania	34	0	0	36	0
Others	84	10	8	47	5
Total	10030	16	1611	7061	1342

† Best guess, based on various reports and surveys.
‡ Estimate using best guess.

Yield Reducers

With the exception of Algeria, Jordan, Morocco and Syria, the average yields in the WANA countries are higher than that of the world average. There is apparently a large gap between the yield at farm level and the yield obtained in the well-managed research station and on-farm trials. The factors that constrain the productivity of kabuli chickpea

in WANA can be grouped in three major categories: environmental, agronomic and biotic.

Chickpea in WANA is traditionally grown as a spring-sown crop on the conserved soil moisture and is, therefore, exposed to terminal drought and heat stress during the reproductive phase (Fig. 1). Large fluctuations in the amount and distribution of winter rains are common in the region and often an inadequate moisture supply limits the productivity of rain-fed chickpea.

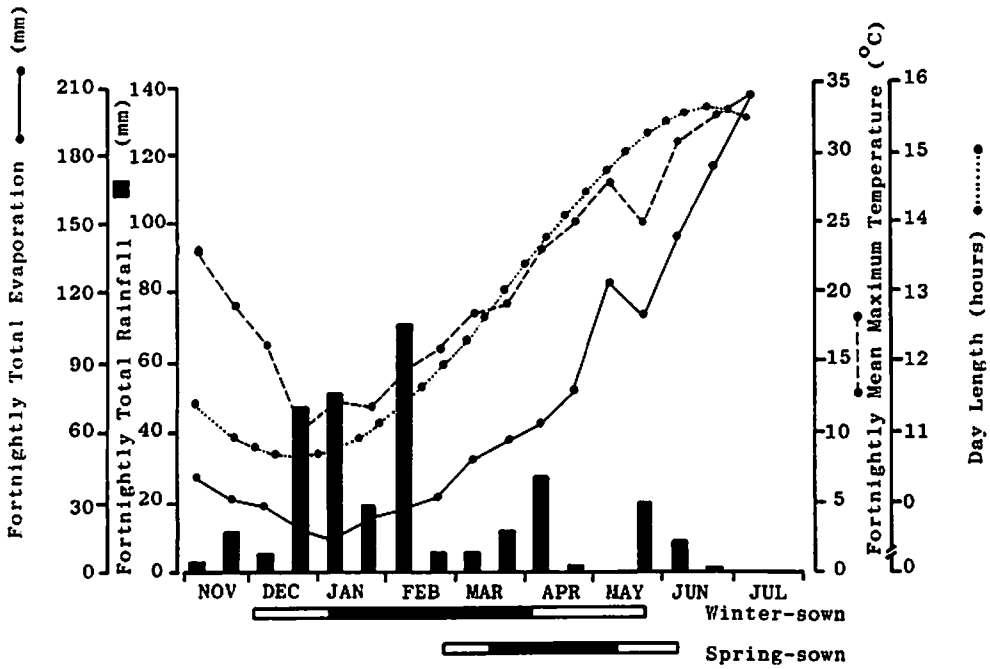

Fig. 1. Crop phenology of winter- and spring-sown ILC 482 chickpea at Tel Hadya, Syria, 1985/86, in relation to weather conditions during the period of crop growth. S = sowing, G = germination, F = flowering, M = maturity.

Use of inadequate amounts of production inputs (fertilizer, herbicide and pesticide) and traditional methods of land preparation and seeding are some of the major agronomic factors that retard the yield, but farmers tend to practise them to avert risk associated with the environmental uncertainty.

Biotic stresses imposed on the crop include depredation from diseases such as ascochyta blight [*Ascochyta rabiei* (Pass.) Labr.], dry root rot [*Rhizoctonia bataticola* (Taub) Butler], wilt [*Fusarium oxysporum* Schlecht. emend. Snyd. & Hans. f.sp. *ciceri* (Padwick) Snyd. & Hans.] and stunt virus, cyst (*Heterodera ciceri*) and root-knot

nematodes (*Meloidogyne* spp.), insect pests such as leafminer (*Liriomyza cicerina* Rondani) and pod borer (*Helicoverpa armigera* Hübner) and weeds including the parasitic species such as broomrape (*Orobanche crenata* L.). The incidence of these pathogens and pests varies greatly, depending on the environmental conditions and level of crop management, and therefore the chickpea yield in WANA shows large variation and instability.

The domestic demand of kabuli chickpea in many WANA countries falls short of the local production; imports are common to meet the requirement. A rise in the market price of chickpea has rendered this important food crop out of reach of the poorer sections of the population. There is, therefore, a need for speedy and substantial improvement in the productivity of the crop by developing economic and socially acceptable solutions for the problems constraining the yield in the region.

Prospects for Improved Production

The collaborative research done at ICARDA and in national programs in WANA, under the ICARDA/ICRISAT kabuli chickpea improvement, promises large productivity gains in the region.

Productivity gains of nearly 100% seem possible by adopting winter sowing of chickpea in place of the traditional spring sowing currently in vogue in WANA (Saxena 1980). Results obtained at experimental stations at ICARDA and in national programs have been confirmed in on-farm trials conducted throughout the regions by national programs. Winter sowing permits matching the chickpea crop phenology with the availability of optimum temperature and moisture regimes so that adverse effects of environmental conditions are minimized (Fig. 1). In contrast to spring-sown crops, winter-sown crops have a longer duration for vegetative and reproductive growth (Fig. 1), faster development of photosynthetic surfaces (Cooper 1983), larger crop canopy, more reproductive nodes and greater total dry matter production. As the reproductive growth occurs in more favorable thermal and moisture regimes, the number of filled pods increases and higher economic yield is obtained. All this improvement in growth and yield occurs without any significant increase in total evapotranspiration when compared with spring-sown crops; hence, the water-use efficiency is substantially increased (Keatinge and Cooper 1983).

The productivity of chickpea also can be improved by supplementary irrigation, which is commonly available in several chickpea-growing countries in WANA. Although both winter- and spring-sown crops showed improvement in their productivity at Tel Hadya in 1985/86 (total seasonal precipitation of 316 mm) when two supplementary irrigations of 55 mm each were given during the reproductive growth stage, the relative increase was higher in the spring- than in the winter-sown crop (Table 3).

Table 3. Effect of sowing date and supplementary irrigation on the mean seed yield (kg/ha) of five genotypes of kabuli chickpea, Tel Hadya, Syria, 1985/86.

	Sowing time (ST)		
Moisture supply (MS)	Winter	Spring	Mean yield
Rain-fed	1559	884	1221
Irrigated	2431	1522	1991
Mean	1995	1203	
SE ±	MS: 66	ST: 131	MSxST: 147

For successful winter sowing, cultivars must have a high level of cold tolerance and resistance to ascochyta blight. Also, weeds have to be effectively controlled. Research on all these aspects has been underway at ICARDA and in national programs, and several national programs have released cultivars for winter sowing (Table 4). Although some of the earlier releases combined a reasonable level of cold tolerance with an adequate level of ascochyta blight resistance, they required improvement with respect to seed size, earliness and durability of resistance. Hence, newer lines developed through hybridization have now reached the level of on-farm trials in several countries and a new generation of cultivars for winter sowing is now becoming available (Table 5). Solutions for "second generation" problems of winter-sown crops are being actively sought: increased tolerance to cold, durable resistance to ascochyta blight, tolerance to root rot, wilts and stunt virus, tolerance/resistance to *Orobanche*, better control of weeds, and improved biological nitrogen fixation.

Increases in production of the traditional spring crop also are possible, as our work on cultivar improvement and crop management has shown. Genotypes with higher yield potential and better tolerance to drought and high temperature, resistance to root rot and wilt diseases, resistance to leafminer and tall stature to facilitate mechanized harvest are being bred at ICARDA and in cooperative programs with the agriculture research national centers. Also, research on supplementary irrigation, chemical weed control, phosphate fertilization and inoculation with rhizobia has given valuable results (ICARDA 1988). Packages of production practices for different agroecological conditions have been developed and are in on-farm verification trials. Transfer of this technology would improve productivity of the traditional spring crop.

Table 4. Kabuli chickpea cultivars† released by national programs in West Asia, North Africa and South Europe.

Country	Cultivars	Year of release
Algeria	ILC 482	1988
	ILC 3279	1988
Cyprus	Yialousa (ILC 3279)	1984
	Kyrenia (ILC 464)	1987
France	TS 1009 (ILC 482)	1988
	TS 1502 (FLIP 81-293C)	1988
Italy	Califfo (ILC 72)	1987
	Sultano (ILC 3279)	1987
Morocco	ILC 195	1987
	ILC 482	1987
Spain	Fardan (ILC 72)	1985
	Zegri (ILC 200)	1985
	Almena (ILC 2548)	1985
	Alcazaba (ILC 2555)	1985
	Atalaya (ILC 200)	1985
Sudan	Shendi	1987
Syria	Ghab 1 (ILC 482)	1982
	Ghab 2 (ILC 3279)	1986
Tunisia	Chetoui (ILC 3279)	1986
	Kassab (FLIP 83-46C)	1986
	Amdoun 1 (Be-sel-81-48)	1986
Turkey	ILC 195-selection	1986
	ILC 482	1986

† All chickpeas are resistant to ascochyta blight and released for winter sowing except Amdoun 1, which is resistant to fusarium wilt and is for spring sowing. In Turkey, ILC 482 is released for spring sowing.

Table 5. Summary of the yield (kg/ha) of chickpea in on-farm trials for winter sowing in Syria, 1985/86 to 1987/88.

Entry	1985/86	1986/87	1987/88	Overall mean	Rank
FLIP 81-293C	2035	1826	1823	1895	3
FLIP 82-150C	1933	2002	2054	1996	1
FLIP 82-232C	1919	1869	1780	1856	4
Ghab 1	2007	1851	1893	1917	2
Ghab 2	1602	1705	1554	1620	5
Mean	1899	1851	182		
± SE	102.17	84.28	63.01		

Future Outlook

Research in future will have to be directed to further raising the yield potential of the crop through improved plant architecture and physiological functioning. Stability would be improved by increasing the level of tolerance/resistance to common biotic and abiotic stresses. While tackling various stress factors, we will have to review the procedures that we have adopted so far for our screening and breeding work and devise better strategies and procedures so that progress in the future is faster and more efficient. The start is being made with this consultancy meeting, which has been called to discuss a stress factor that in our assessment is the most important reducer of yield of kabuli chickpea in WANA, namely the diseases.

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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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Breeding for Resistance to Ascochyta Blight in Chickpea

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Abstract

The research work on disease resistance in the ICARDA/ICRISAT Kabuli Chickpea Project between 1978 and 1988 at ICARDA, Aleppo, Syria is reviewed in this paper. A major objective of the project was to develop high-yielding cultivars resistant to ascochyta blight (*Ascochyta rabiei*). To achieve this objective studies were carried out on inoculation technique, pathogenic variability, host-plant resistance and epidemiology of the disease. Six races of blight pathogen were identified from Syria and Lebanon. The pathogen was found to produce the perfect state on the overwintered infected chickpea debris in the field. The pathogen survived less than 4 months on diseased debris under field conditions, but survival in the infected seed in storage did not diminish much in 1 year. There are indications that the disease spreads over a long distance from the source of infection. The blight epidemic occurred in the field only when the weekly minimum and maximum temperatures were above 5 and 15°C, respectively.

An efficient field-screening technique using diseased chickpea debris and sprinkler or mist irrigation was standardized for ascochyta blight. Similar efficient greenhouse and laboratory techniques were standardized. Field and greenhouse disease rating scales were developed. The world collection of chickpea germplasm maintained at ICRISAT and ICARDA was screened and several lines were identified as resistant sources to ascochyta blight. Some of the wild *Cicer* species also were found resistant. Some lines resistant to as many as five races were identified. A number of lines showing location nonspecific resistance in the West Asia and North Africa (WANA) region were identified.

Two blight-resistant genes, one dominant and one recessive, were identified. Bulk-pedigree and backcross-pedigree methods of breeding were used effectively and several hundred high-yielding and ascochyta blight-resistant lines have been bred. International yield trials and on-farm tests helped in the release of 20 blight-resistant and high-yielding cultivars in nine countries of the Mediterranean region from the materials developed in the project.

One or two foliar applications of chlorothalonil (Bravo 500) to a blight-tolerant cultivar in the reproductive stage of the crop were found to provide good economic protection.

Introduction

Chickpea (*Cicer arietinum* L.) is an important food legume crop in the West Asia and North Africa (WANA) region, accounting for 27% of the total food legume production, compared with 26% for faba bean (*Vicia faba* L.) and 17% for lentil (*Lens culinaris* Medikus). It serves as a source of inexpensive high-quality protein in the diets of many people and provides a rich crop residue for animal feed. Like other legumes, chickpea has the ability to convert nitrogen from the air into the fixed nitrogen that plants need for growth, thus providing a renewable source of fertilizer. It is also an important component in cereal-legume rotations, where it helps to break cycles of diseases, weeds and insects affecting the cereal crops.

Despite the importance of a chickpea crop, most farmers in the WANA region are still growing unimproved landraces that give low yields and are susceptible to diseases and other stress factors. This situation is likely to change in the next few years. New cultivars that possess resistance to both ascochyta blight and cold are suitable for winter sowing at low to medium elevations in the Mediterranean region. Using traditional cultivars, farmers must plant in the spring season because these cultivars are susceptible to cold and ascochyta blight. Spring-sown chickpea faces the twin problems of diminishing moisture supply and rising temperatures. In contrast, cold-tolerant and ascochyta blight-resistant cultivars can be grown in the winter, making best use of winter rains and temperatures that favor a longer vegetative and reproductive period. This results in bigger plants, more flowers, more pods and hence a greater yield.

The yield differences between winter-sown and spring-sown chickpea can be spectacular. Winter sowing produces 50 to 100% higher grain yield than spring sowing. Other advantages of winter sowing include more protein harvest per hectare, more nitrogen fixation per hectare, better utilization of available moisture, a higher germination rate, less fusarium wilt disease and a better opportunity for mechanized harvest. The advantages of winter chickpea could be realized only with ascochyta blight- and cold-resistant cultivars. Such cultivars were developed in the project and extensively used by the national agricultural research systems (NARS). The problem of physiologic races arose, which necessitated the development of race-specific cultivars. In addition to ascochyta blight, wilts, root rots, viruses and nematodes were identified as problems in some countries of the WANA region.

The progress made on ascochyta blight in the world was reviewed in 1981 (Saxena and Singh 1984). Eight years later, it is considered desirable to hold a consultancy meeting to review the progress made on disease resistance breeding and to plan future

work. In this presentation, we review work that we conducted at ICARDA on ascochyta blight disease resistance breeding between 1978 and 1988.

Disease Survey

Limited systematic surveys have been conducted in the WANA region to identify the different disease problems of chickpea and determine their incidence, distribution and economic importance. Disease problems of chickpea in the WANA region have been dominated by ascochyta blight (Table 1). This disease not only reduces the yield drastically but also limits realization of the potential yield. Yields of chickpea in the WANA region could be greatly increased by advancing the sowing date if there were no risk of increased damage by ascochyta blight. Winter-sown chickpea is not possible without control of ascochyta blight.

Table 1. Relative importance of different diseases of chickpea in countries having a Mediterranean climate.

Country	Diseases	
	Major	Minor
	West Asia	
Afghanistan	Blight	Not known
Cyprus	Blight	Not known
Iran	Blight	Viruses, root rots
Iraq	Blight	Not known
Jordan	Blight	Stunt
Lebanon	Blight	Wilt, root rots
Syria	Blight	Root rot, wilt, viruses
Turkey	Blight	Viruses, stunt
	North Africa	
Algeria	Blight	Stunt
Egypt	Blight	Wilt, root rot
Morocco	Blight, wilt	Wilt, root rot
Tunisia	Blight, wilt	Sclerotinia stem rot
	South Europe	
France	Blight	Not known
Greece	Blight	Not known
Italy	Blight, wilt	Root rots
Portugal	Blight	Not known
Spain	Blight, wilt	Root rots
	Others	
Australia	Phytophthora root rot Blight	
	Botrytis gray mould Blight	
Chile	Wilt, root rots, stunt Not known	
USA	Wilt, virus, blight	Pythium seed, root rots

Other diseases in the region are of less importance at present. These include fusarium wilt [*Fusarium oxysporum* Schlecht. emend. Snyd. & Hans. f.sp. *ciceri* (Padwick) Snyd. & Hans.], verticillium wilt (*Verticillium albo-atrum* Reinke & Berth.), wet root rot (*Rhizoctonia solani* Kühn), dry root rot [*Rhizoctonia bataticola* (Taub) Butler], collar rot (*Sclerotium rolfsii* Sacc.) and stunt (bean leaf roll virus). Fusarium and verticillium wilts are important in Tunisia. Collar rot, root rots and stunt are prevalent throughout the WANA region.

Ascochyta Blight

The fungus causing blight in chickpea is *Ascochyta rabiei* (Pass.) Labr. and its sexual stage is found in *Mycosphaerella rabiei* (Pass.) Kovach. The disease has been known for more than 100 years since Passerini from France named it *Zythia rabiei* in 1867. It has now been reported from 26 countries (Nene 1984). Losses caused by this disease have been reported from many countries; in some countries yield losses have been up to 100%.

Pathogen

Pathogenic variability in *A. rabiei*

Pathogenic variability in *A. rabiei* is well recognized, but is not well understood. Loss of resistance in chickpea cultivars has been attributed to the appearance of new races of *Ascochyta*.

At ICARDA, six races of *A. rabiei* were identified based on their reactions on six chickpea lines (Table 2) (Reddy and Kabbabeh 1985). Further surveys and isolations from Syria and the blight nursery at ICARDA indicated the possible presence of several isolates differing in the aggressiveness of virulence. Studies to monitor the changes in pathogenic variability of *A. rabiei* are needed.

Table 2. Pathogenic variability in *Ascochyta rabiei* in Syria and Lebanon.

Race	Reactions† of chickpea cultivars						No. isolates representing the race
	ILC 1929	F 8	ICC 1903	ILC 249	ILC 3279	ICC 3996	
1	S	R	R	R	R	R	1
2	S	R	R	R	R	R	5
3	S	S	S	R	R	R	1
4	S	S	S	S	R	R	2
5	S	S	S	S	S	R	1
6	S	S	S	S	S	S	40

† S = susceptible, R = resistant.

Perfect state of *A. rabiei*

At Tel Hadya farm, *A. rabiei* was discovered on the overwintered chickpea debris during 1987, particularly on stem pieces in a field where chickpea was grown during the previous season and severely infected with ascochyta blight (Haware 1987).

It seems that conditions in eastern Europe and western Asia are favorable for the occurrence of the perfect state of *A. rabiei*. A severely cold winter might be a prerequisite for the production of perithecia on infected tissues. This stage may be playing an important role in the dissemination of the disease in this region. In sexual reproduction, a diploid phase is brought about by the fusion of two haploid nuclei, and thus new combinations of alleles can arise, creating new variations in the pathogen. The role of sexual reproduction in the life cycle of *A. rabiei* in the field needs study.

Epidemiology

Crop debris

Studies at ICARDA clearly indicated that *A. rabiei* survives for a limited period (<4 months) on infected host tissues in the field throughout summer and part of winter. Burial of the debris in the soil 10 cm or deeper hastened the decline of the fungus on the host tissue and also made it ineffective in transmitting the disease from the debris to seedlings.

Seed

The fungus survived in the infected seed in storage with little loss of viability for 1 year. The extent of transmission of the disease from the infected seed to seedlings was less than the extent of seed infection. This was due to the influence on disease transmission of the location of the lesion on the seed; the closer the lesion to the micropylar region, the greater the disease transmission. Deep sowing (10 cm) was found to drastically reduce seed transmission.

Long-distance spread

In January of 1987 and 1988, healthy seedlings of a blight-susceptible line (ILC 1929) grown in pots were placed at 17 locations at the Tel Hadya farm, two locations in Aleppo town (30 km east of Tel Hadya) and one location in a farmer's field near Tel Hadya. Each location was inspected after every 2 days for blight infection. Occurrence of ascochyta blight on chickpea seedlings located at several places (1 to 30 km from the possible source of infection) indicated that the inoculation could be disseminated over a long distance. Further studies are needed to confirm this type of disease spread.

Temperature and humidity

In a field trial conducted at Tel Hadya, the development of disease in susceptible, tolerant and resistant cultivars in relation to humidity and temperature was studied for three seasons. In the winter-sown chickpea, during the early part of crop growth (Nov-Feb), low temperatures limited disease development despite adequate humidity (Fig. 1). Disease development was virtually nil when the weekly night and day temperatures were below 5 and 15°C, respectively. The disease started building up rapidly

when temperatures started warming up. The relative humidity during this period was around 70%. Disease buildup continued until maximum temperatures reached 20°C.

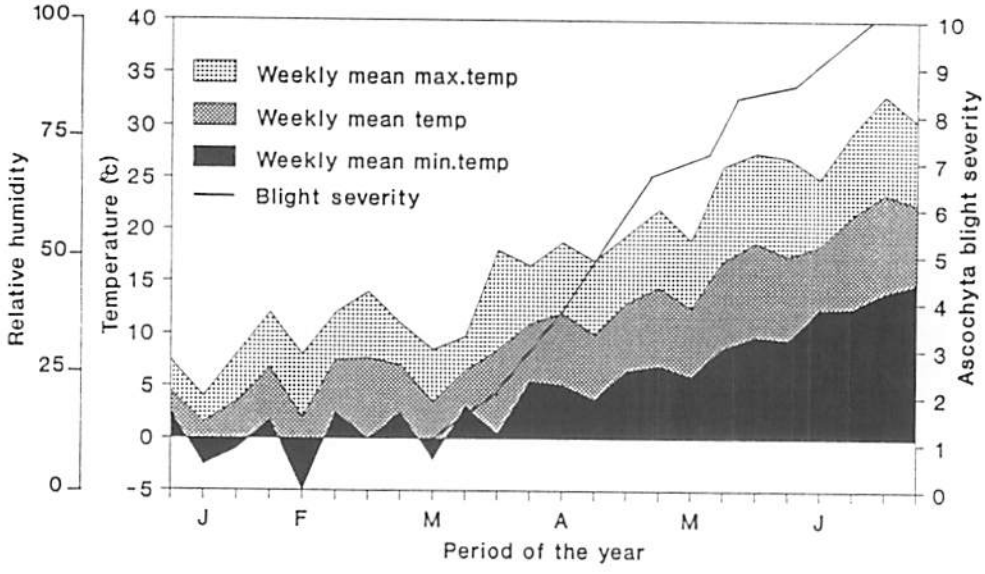

Fig. 1. Development of ascochyta blight-resistant chickpea in relation to temperature and relative humidity at Tel Hadya, Syria, 1983.

Yield Loss Study

The yield loss is total in susceptible cultivars under epiphytotic conditions. When conditions for disease development are optimum for only a short period, the diseased plants tend to recover. The extent of recovery depends on the stage at which the crop is infected, soil moisture and the prevailing air temperatures. If the disease occurs in the early stages of crop growth, recovery is better than in the later stages when the crop is growing in low soil moisture and high temperature conditions. Some genetic variability exists for recovery.

In a field trial conducted for three seasons at Tel Hadya (1982/83, 1983/84, 1985/86), the yield loss due to blight in 20 cultivars with different levels of infection in the vegetative and podding stage was estimated. There was a significant positive correlation between the vegetative infection and pod infection ($r = 0.8$) and negative

correlations between vegetative infection and yield (r = -0.5) and pod infection and yield (r = -0.4) (Table 3) (Reddy and Singh, unpublished data). While yield loss was total in susceptible cultivars (rating = 9), the loss in lines with a rating of 2 to 4 on a 1 to 9 rating scale (1 = free, 9 = killed) was less than 10%. With a similar disease rating, kabuli types in general showed less yield loss than desi types.

Table 3. Ascochyta blight severity and yield loss estimation in a set of chickpea germplasm lines with a range of ascochyta blight susceptibility at Tel Hadya, Syria, 1982/83, 1983/84 and 1985/86.

Line	Blight score on vegetative parts†	Pod infection (%)	Average yield (t/ha)		Yield‡ (%)
			Uninoculated	Inoculated	
ILC 183	2.6	26	2.0	2.0	0‡
ILC 194	3.0	42	2.1	2.0	-5
ILC 196	2.3	15	1.5	1.3	-13
ILC 201	2.2	10	1.6	1.8	+14
ILC 202	2.7	4	1.8	1.8	0
ILC 215	6.3	67	2.1	0.6	-71
ILC 236	2.6	44	2.0	1.7	-15
ILC 263	8.8	80	2.4	0.03	-99
ILC 482	3.9	52	2.3	1.7	-23
ILC 484	3.4	53	2.3	1.6	-31
ILC 1695	3.1	46	1.8	1.6	-19
ILC 1919	6.2	70	2.2	0.4	-81
ILC 1929	9.0	82	2.2	0.02	-99
ILC 2548	2.7	28	1.8	2.1	+14
ILC 3279	2.1	7	1.6	1.8	+9
ILC 3346	2.0	4	1.9	1.7	-9
ILC 3856	2.2	7	2.2	1.7	-24
G 543	2.8	29	2.3	1.7	-23
G 549	2.6	34	1.7	1.1	-40
ICC 4935	3.4	23	2.2	1.4	-35
SE	0.5	7.4	0.23	0.21	
CV (%)	23.0	35.4	20.4	26.2	

† Average of three seasons. Score: 1 = free; 5 = intermediate; 9 = killed.
‡ + = increase in yield; - = loss in yield.

Use of Fungicides in Disease Management

Limited studies were completed on the use of fungicides in the management of ascochyta blight at ICARDA. Priority was given to the exploitation of host-plant resistance for management of the disease, but in view of the seed-borne nature of disease and its importance in the initiation of disease epidemics and quarantine, a limited number of fungicides were evaluated for their efficacy in eradicating the fungus from infected seed. Calixin M (11% tridemorph + 36% maneb) and thiabendazole were found to be very effective in eradicating the fungus from infected seed (Reddy 1980; Reddy *et al.* 1982; Reddy and Singh 1984).

After encountering a lack of high levels of resistance in the germplasm, especially at the podding stage, and the presence of virulent races in the fungus, we evaluated a limited number of fungicides for their possible use in the production of disease-free seed for sowing purposes, for integrated management of the disease along with host-plant tolerance and for use as a stop-gap arrangement between the breakdowns of resistance and development of new resistant cultivars. Chlorothalonil (Bravo 500) was found to be a very effective foliar fungicide (Reddy and Singh 1983) (Table 4). Eleven sprays applied at 10-day intervals provided near complete protection to the highly susceptible cultivar ILC 1929 under very high disease pressure.

Table 4. Effect of foliar fungicidal application on blight severity and yield in chickpea at Tel Hadya, Syria, 1981/82.

Fungicide	Vegetative stage	Podding stage	Seed yield (kg/ha)
Bravo 500	2.0	1.0	2160
Calixin M	4.3	5.7	1184
Bravo 500/ Calixin M	2.0	1.0	2083
Rubigan	9.0	9.0	0
Control	9.0	9.0	0
SE			114.5
CV (%)			18.2

Screening Technique

Even in disease-endemic areas such as northwestern India, Pakistan, West Asia, North Africa and southern Europe, ascochyta blight severity is not uniform and high across the seasons. As the dependence on natural epidemics for screening of chickpea for blight resistance in the past hampered progress to a large extent, the emphasis at ICARDA in the beginning of the program was on the standardization of field and greenhouse

inoculation techniques. At present, we have simple and efficient inoculation techniques that have been adapted in several countries. Temperatures between 15 and 20°C and humidity between 70 and 100% are essential for blight infection and development. Leaf wetness for 6 to 8 hours is essential for infection.

Field

Inoculation of chickpea was achieved at flowering time or earlier by scattering the diseased debris or spraying with a spore suspension. The suspension (40,000 spores/ml) was prepared from the pure culture of isolates prevalent in an area, multiplied on chickpea seed meal dextrose broth (40 to 80 g chickpea seed meal, 20 g dextrose, 1 L water), or from the freshly infected plants. Mist or sprinkler irrigation was provided, if there were no rains after inoculation, until the successful establishment of the disease resulted in severe blight epidemics. Growing a susceptible cultivar around the screening nursery and at regular intervals in the field further helps in spreading and monitoring of the disease.

The advantage of inoculation with diseased debris is that it is not necessary to provide irrigation immediately after inoculation and the disease develops as and when conditions become favorable to the disease. The other feature of this method is that as the diseased debris used for inoculation is collected from the breeding nursery, which has a large amount of genetic variability, the inoculation is very broad-based and comprises new virulences that might arise from time to time. This means there is a selection for more virulent isolates, which is expected to occur when resistant cultivars are released for cultivation. Thus, this method is expected to help in developing lines resistant to several virulent isolates. It is necessary to inoculate and provide irrigation at podding time to encourage good pod infection as conditions generally become dry at that period.

Greenhouse

The technique to inoculate chickpeas grown in pots and trays in greenhouses also is standardized. The important point is to keep the foliage wet after inoculation for at least 8 hours, either by covering the inoculated plants with a polyethylene sheet or by providing mist irrigation to encourage development of infection. If the polyethylene covering or mist irrigation is prolonged, disease development is faster and a susceptible cultivar usually is killed within 10 days. An important point is that the polyethylene cage ceiling should be as close to the top of the foliage as possible.

Growth chamber

In this technique, chickpea seedlings are grown in plastic trays (14 x 11 x 8 cm) containing sterilized, fine riverbed sand. Five seedlings are kept for each line and five lines are grown per tray. Seven-day-old seedlings are inoculated with a spore suspension at a concentration of 200,000 spores/ml. Trays are covered with transparent lids (14 x 11 x 15 cm) and kept in plant growth chambers (Conviron) at 20°C with 12 hours light. Plastic lids are removed 5 days after inoculation and observations recorded 10 to 15 days after inoculation. Uniformity in disease development in the test lines is found and escapes from disease are not observed.

Rating Scale

During the course of work on screening for resistance to ascochyta blight, the difficulty in developing a detailed scale that is also suitable for use in large-scale field screening of materials was realized. Symptoms that were considered important for developing a rating scale were (i) extent of sporulation in leaf, stem and pod lesions, (ii) extent of stem breaking, (iii) stem lesion length and (iv) percent pods with lesions. Developing a scale for field scoring that included all these aspects was difficult. However, for detailed studies, such as race identification and inheritance of resistance, it was found necessary to consider all these aspects. Thus, two 9-point scales were developed, one for laboratory and greenhouse studies involving a limited number of materials, and the other for large-scale field use. The field scale takes into consideration mainly the extent of stem breaking and pod infection (Table 5), whereas the laboratory scale takes into account all four aspects mentioned above (Table 6). We fully realized the scope for improvement of these scales. The more difficult aspect of the 9-point rating scale was the delineation of lines with different scores into resistant, tolerant and susceptible groups. So far the lines with a 1 to 4 rating were arbitrarily considered resistant, 5 were tolerant and 6 to 9 were susceptible. In a field experiment conducted at ICARDA, the extent of yield loss for different scores was estimated (Table 7) (Reddy and Singh, unpublished data). Yield loss in lines with a 2 to 4 rating was about 10%, with 5 to 7 rating 25% and with 8 to 9 rating more than 80%. The yield data show that the above categorization of lines into resistant, tolerant and susceptible is objective.

Table 5. A 9-point rating scale for scoring ascochyta blight severity of chickpea under field conditions.

Disease rating		Blight reaction	Percent broken stems and infected pods
1	I	Apparently immune	0
2	HR	Highly resistant	1-5
3	R	Resistant	6-10
4	MR	Moderately resistant	11-15
5	T	Tolerant	16-40
6	MS	Moderately susceptible	41-50
7	S	Susceptible	51-75
8	HS	Highly susceptible	76-100
9	HS	Highly susceptible	Plants killed

Table 6. A 9-point rating scale for scoring ascochyta blight severity of chickpea in the laboratory/greenhouse.

Disease rating		Blight reaction	Sporulation on stem and pod lesions	Percent stems broken	Stem lesion type	Percent pods with lesions
1	HR	Highly resistant	None	0	None visible	0
2	HR-R	Highly resistant-resistant	None	1-5	0-2 mm long	1-5
3	R	Resistant	Low	6-10	2-6 mm long, girdling	6-10
4	R-T	Resistant-tolerant	Low	11-15	6 mm long, with girdling but no breakage	11-15
5	T	Tolerant	Moderate	16-40	6 mm long, with girdling and breakage	16-40
6	T-S	Tolerant-susceptible	High	41-50	6 mm long, with girdling and breakage	41-50
7	S	Susceptible	High	51-75	6 mm long, with girdling and breakage	51-75
8	S-HS	Susceptible-highly susceptible	High	76-100	100% girdling	76-100
9	HS	Highly susceptible	High	Plant completely killed		

Table 7. Relationships between ascochyta blight severity on a 1-9 disease severity rating scale and yield loss in chickpea germplasm lines at ICARDA, Tel Hadya, Syria, 1982/83, 1983/84 and 1985/86.

Line	Blight reaction†	Blight severity on vegetative parts‡	Percent pod infection	Average yield (t/ha)		Yield loss (%)
				Uninoculated	Inoculated	
ILC 3346	HR	2	1-5	1.9	1.7	9
ILC 202, ILC 3279, ILC 3856	R	3	6-10	1.9	1.8	7
ILC 196, ILC 201	MR	4	11-15	1.5	1.6	+3¶
ILC 183, ILC 2548, G 543, G 549, ICC 4935	T	5	16-40	2.0	1.7	16
ILC 194, ILC 215, ILC 236, ILC 1695	MS	6	41-50	2.0	1.4	26
ILC 482, ILC 484	S	7	51-75	2.3	1.6	27
ILC 1919	HS	8	76-100	2.2	0.4	81
ILC 263, ILC 1929	HS	9	NR§	2.3	0.06	98

† HR = Highly resistant; R = Resistant; MR = Moderately resistant; T = Tolerant; MS = Moderately susceptible; S = Susceptible; HS = Highly susceptible.
‡ Average of three seasons.
§ NR = Not recorded as there were no pods.
¶ + = increase in yield.

Screening of World Germplasm Collection

Cultivated species

A total of 15,310 germplasm accessions including 5107 kabuli and 10,203 desi maintained at ICARDA and ICRISAT have been evaluated since 1978. Our procedure was to evaluate lines in the first year at Tel Hadya under artificial epiphytotic conditions, followed by retesting the resistant lines in other hot-spot locations in Syria and Lebanon in the succeeding year. These accessions were evaluated against naturally occurring isolates of *A. rabiei* at Tel Hadya using infected chickpea debris collected in the previous year during the first 6 years, 1978 to 1983. Since 1984, the material was inoculated in the diseased debris against a mixture of the four most prevalent races in Syria (1, 2, 3, 4) after they were multiplied in the laboratory. Germplasm accessions were scored when the susceptible check was killed. Screening was successful in all 11 years with the exception of 1985 and 1987.

Screening helped in identification of 12 kabuli and 3 desi resistant lines (a score of 3 on a 1 to 9 scale). Also, 30 moderately resistant lines (a score of 4 on a 1 to 9 scale) were found (Table 8). Frequency of resistance was higher in the kabuli type than in the desi type.

Table 8. Reaction of chickpea germplasm to ascochyta blight at Tel Hadya, Syria, 1978 to 1988.

Blight reaction†	No. of accessions		Total no.	Percent of total
	Kabuli	Desi		
R	12	3	15	0.10
MR	17	13	30	0.20
T	55	76	131	0.86
MS	1453	1539	2992	19.54
S	3570	8572	12142	79.30
Total	5107	10203	15310	100.00

† R = resistant, MR = Moderately resistant, T = Tolerant, MS = Moderately susceptible, S = susceptible.

Some characteristics of ascochyta blight-resistant kabuli germplasm lines are listed in Table 9. Nine lines originated in the USSR and three in Bulgaria. They are late in maturity, poor in yield, have small seed size and are of mid-tall to tall stature. None of them were good for direct commercial exploitation (Reddy and Singh 1984; Singh *et al.* 1981).

Table 9. Some characteristics of kabuli chickpea germplasm lines resistant to a mixture of races 1, 2, 3 and 4 of ascochyta blight at Tel Hadya, Syria.

Line	Origin	Blight reaction†	Maturity	Seed type‡	100-seed wt (g)	Plant height (cm)	Yield
ILC 182	USSR	3	Late	I	19	50	Low
ILC 200	USSR	3	Late	I	19	57	Low
ILC 2506	USSR	3	Late	I	22	71	Low
ILC 2956	USSR	3	Late	I	27	76	Low
ILC 3274	USSR	3	Late	I	24	71	Low
ILC 3856	USSR§	3	Late	I	20	65	Low
ILC 3866	Bulgaria	3	Late	I	23	65	Low
ILC 3870	Bulgaria	3	Late	I	23	65	Low
ILC 4421	USSR	3	Late	I	19	57	Low
ILC 5586	USSR¶	3	Late	I	22	52	Low
ILC 5921	Bulgaria	3	Late	I	22	56	Low
ILC 6188	USSR¶	3	Late	I	24	77	Low

† 3 = Resistant.
‡ I = Intermediate type.
§ Via Morocco.
¶ Via France.

Wild species

Field screening. One hundred and fifteen accessions of eight *Cicer* species were evaluated in the field against a mixture of four races (1, 2, 3, 4) of *A. rabiei* during 1987/88 (Table 10). Some accessions of four species (*C. bijugum*, *C. cuneatum*, *C. judaicum* and *C. pinnatifidum*) showed resistant reactions to ascochyta blight. The remaining four species had no resistance to *A. rabiei*. Collections of a given species differed in disease reaction. Promising lines will be evaluated further.

Growth chamber screening. Collections of *Cicer* species also were screened in a controlled environment (Conviron) at the seedling stage. Seedlings of 8 to 10 cm height were inoculated and incubated for 7 days at 20°C (12 hours light) at 100% RH. The evaluation of accessions was done 15 days after inoculation (Table 11). Several sources of resistance were found in *C. judaicum* and *C. bijugum*. Three lines of *C. pinnatifidum* were found tolerant to each of two isolates of *A. rabiei*. One line of *C. echinospermum* was tolerant to one isolate. Resistance to ascochyta blight was not observed in accessions of *C. reticulatum*, *C. cuneatum* and *C. chorassanicum*.

Screening germplasm and breeding lines for resistance to six races of *A. rabiei*

Six races of *A. rabiei* from Lebanon and Syria have been identified (Reddy and Kabbabeh 1985). To identify sources of resistance to these races, 1069 germplasm accessions and breeding lines were screened in the greenhouse at Tel Hadya, Syria

Table 10. Evaluation of *Cicer* spp. for resistance to a mixture of four races of ascochyta blight in the field at Tel Hadya, 1987/88.

Cicer species	Number of lines with a 1-9 rating									
	1	2	3	4	5	6	7	8	9	Total
C. bijugum K.H. Rech.			15		2	2	2	1		22
C. chorassanicum (Bge) M.Pof.				1		2				3
C. cuneatum Hochst. ex Rich				2					1	3
C. echinospermum‡ P.H. Davis									1	1
C. judaicum Boiss.		2	31		3		2		3	41
C. pinnatifidum Jaub. & Sp.		1	23	1	3	1				29
C. reticulatum Ladiz.					1	4	5		3	13
C. yamashitae Kitamura							1		1	2
Total	0	3	71	2	9	9	10	3	7	114

† 1 = Free, 5 = intermediate, 9 = killed.
‡ No seed germinated in two lines sown.

Table 11. Evaluation of *Cicer* spp. for ascochyta blight resistance in the laboratory, Tel Hadya, Syria, 1987/88.

Cicer species	Race	No. lines tested	No. lines with disease reaction						
			1†	2	3	4	5	6	(7-9)
C. judaicum	3	39		3	3	8	8	8	9
	6	39			2	5	8	14	10
C. pinnatifidum	3	27					3	13	11
	6	27					3	9	15
C. bijugum	3	17		1	1	4	4	7	
	6	17		2	2	8	3	2	
C. reticulatum	3	13							13
	6	13						2	11
C. chorassanicum	3	2							2
	6	0							
C. echinospermum	3	3					1	1	1
	6	3							3
C. cuneatum	3	1							1
	6	1							1

† Severity rating on a 1-9 point scale.

during 1985/86. Preliminary screening of the germplasm was done by inoculating 10-day-old seedlings. Lines with little infection were retested in the seedling and podding stages. Of the 1069 lines, 47, 27, 29, 8, 13 and 4 lines were resistant to races 1, 2, 3, 4, 5 and 6, respectively (Table 12).

Table 12. Frequencies of disease scores of 1069 chickpea lines inoculated with six races of *Ascochyta rabiei* in the greenhouse at Tel Hadya, Syria, 1986.

Disease score†	Number of accessions with indicated mean disease score					
	Race 1	Race 2	Race 3	Race 4	Race 5	Race 6
1	0	0	0	0	0	0
2	0	0	1	0	1	0
3	20	9	2	1	6	2
4	27	18	26	7	6	2
5	25	2	15	2	1	0
6	154	74	199	37	75	37
7	76	75	298	51	84	95
8	31	16	173	19	15	52
9	736	875	355	952	881	881
Total	1069	1069	1069	1069	1069	1069

† 1 = Free, 5 = Intermediate, 9 = Killed.
Source: Singh and Reddy (1990).

Lines with resistance to three to five races of *A. rabiei* are listed in Table 13. Three lines (ILC 202, ILC 3856 and ILC 5928) had resistance to five races, six lines (ILC 72, ILC 201, ILC 2506, ILC 2956, ILC 3279 and FLIP 83-48C) had resistance to four races and three lines (ILC 190, ILC 482 and ICC 3996) had resistance to three races (Singh and Reddy 1990).

International testing of blight-resistant lines

One hundred and fifty-three lines were tested for resistance to ascochyta blight at 67 locations in 16 countries over a period of five seasons between 1982/83 and 1986/87. These lines included kabuli and desi germplasm accessions and breeding lines. Three lines (ILC 72, ILC 202 and ILC 3279) were tested at most locations and in all 5 years. Other lines were evaluated for 1 to 4 years. The evaluation was ignored if the susceptible check was not killed or was rated less than 7.

The number of lines found resistant in different locations is listed in Table 14. Two to 60 lines were found resistant in different countries. None of the lines was resistant across all locations and in all years. This differential reaction indicates the possible presence of a large number of physiologic races. On the one hand, no lines were resistant at Faisalabad and Chakwal (both in Pakistan), but on the other hand all lines were resistant at Khroub (Algeria), Basilicata (Italy) and Terbol (Lebanon), which suggests that Faisalabad and Chakwal races were highly virulent and Khroub, Basilicata

Table 13. Chickpea lines showing resistance to three to five races of *Ascochyta rabiei*.

Entry	Reaction to races†					
	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. resistant lines	12	9	8	7	8	4

† R = resistant (3 to 4 rating on a 1-9 scale), S = susceptible (6 to 9 rating on a 1-9 scale).
Source: Singh and Reddy (1990).

Table 14. Number of chickpea lines resistant to ascochyta blight in different locations when tested in Chickpea International Ascochyta Blight Nursery in different blight-prone countries from 1982/83 to 1986/87.

Country	Location	No. lines reported resistant	Country	Location	No. lines reported resistant
Algeria (1)†	Khroub	60‡	Portugal (1)	Elvas	36
Egypt (2)	Giza	2	Spain (2)	Cordoba	45‡
	Tahrir	22	Syria (4)	Jablch	44‡
France (2)	Montpellier	86		Jindiress	64
Greece (1)	Larissa	4		Lattakia	90‡
India (1)	Ludhiana	43			
Italy (3)	Basilicata	26	Tunisia (3)	Beja	63
	Tarquinia	40‡		Meliz	35
Jordan	Marow			Tunis	23‡
Lebanon (4)	Terbol	128‡	Turkey (3)	Ankara	23‡
Morocco (2)	Douyet	40		Eskisehir	36
	Marchouch	73‡		Izmir	65‡
Pakistan (5)	Tarnab	51‡	USA	Brookings	13
	Islamabad	46‡		Highmore	16

† Number of years.
‡ Entries not resistant every year.

and Terbol races were mild. Some lines had a resistant reaction in one year and a susceptible reaction in another year at the same location, suggesting an environmental role in disease development. Many lines had differential reactions within the same country, suggesting the presence of more than one race in the same country.

Five lines (ILC 202, ILC 3856), ILC 4421, FLIP 82-150C and FLIP 83-46C) were found resistant at more than 40% locations, indicating that they had location nonspecific resistance (Table 15). It is recommended that these lines be used extensively in breeding programs. Other promising lines were ILC 72, ILC 182, ILC 3279 and ILC 3856.

Table 15. Some chickpea lines with location nonspecific resistance to ascochyta blight identified through international testing, 1982/83 to 1986/87.

Entry	No. locations tested	Locations found resistant	
		No.	%
ILC 72	62	22	25.5
ILC 182	52	20	38.5
ILC 200	50	18	36.0
ILC 202	65	27	41.5
ILC 3279	67	24	35.8
ILC 3856	59	22	37.3
ILC 3866	58	26	44.8
ILC 3870	43	17	39.5
ILC 4421	57	24	41.1
FLIP 81-293C	52	16	30.8
FLIP 82-1C	45	14	31.1
FLIP 82-64C	45	16	35.6
FLIP 82-74C	45	18	40.0
FLIP 82-150C	45	19	42.2
FLIP 82-191C	45	15	33.3
FLIP 83-7C	45	14	31.1
FLIP 83-46C	45	20	44.4
FLIP 83-47C	45	12	26.7
FLIP 83-48C	45	17	37.8

Some of the major conclusions from the international testing of highly resistant lines in the last 5 years are as follows. First, NARS scientists identified sources of resistance to ascochyta blight in 16 countries. Second, existence of several physiologic races was evident. Third, a few lines with location nonspecific resistance were identified.

Genetics of Resistance

Inheritance of resistance has been studied in 23 lines and was found to be governed by a single gene, either dominant or recessive (Table 16). There is evidence that resistance is not only qualitative in nature, but also quantitative. Although pyramiding of resistance genes was suggested in 1981 (Singh *et al.* 1984), no progress has been made so far other than using all the resistant lines in the breeding program. Furthermore, the mechanism of resistance is not well understood and further studies are required.

Table 16. Reports on inheritance of resistance to ascochyta blight of chickpea.

Source	Gene	Cultivars
Hafiz and Ashraf (1953)	Monogenic dominant	F 8, F 10
Vir *et al.* (1975)	Monogenic dominant	IP 13
Eser (1976)	Monogenic dominant	Code No. 72-92
Singh and Reddy (1983)	Monogenic dominant	ILC 72, ILC 183, ILC 200, ILC 4935
	Monogenic recessive	ILC 191
Acikgoz (1983)	Monogenic dominant	ILC 200, ILC 201
	Monogenic recessive	72012, ILC 195, NEC 138-1
Tewari and Pandey (1986)	Monogenic dominant	EC 26446, PG 82-1 P 919, P1252-1, NEC 2451
	Monogenic recessive	BRG 8
Singh and Reddy (1989)	Monogenic dominant	ILC 72, ILC 202 ILC 2956, ILC 3279

Breeding Techniques

Selection

As a first step we began evaluation of the kabuli chickpea germplasm for resistance to ascochyta blight in 1977/78 and for cold tolerance in 1978/79. Lines found tolerant to both stresses were evaluated for yield and other agronomic traits. Lines combining tolerance to the two stresses and high yield were provided to NARS.

Hybridization

Bulk-pedigree method. The hybridization program was initiated in 1978 to develop ascochyta blight- and cold-tolerant genotypes. Several breeding schemes were tried, but the bulk-pedigree method was found to be best and is being used currently (Fig. 2). The scheme is operated using the off-season nursery facilities at Terbol, Lebanon, which reduces the time taken for cultivar development by 3 years. Between F_2 and F_6 generations, screening is conducted mainly for blight and cold tolerance, and photoperiod sensitivity. Material also is screened for resistance to leaf miner and cyst nematode. Selection is made for seed size, tall plant type, maturity and yield from F_4 generation onwards. All F_5 and F_6 progenies are grown during both winter and spring seasons. Tolerance to drought and heat is taken into consideration when selecting individual plants or bulking progenies in advanced generations sown during spring. Through hybridization, about 650 blight-resistant lines combining cold or drought tolerance with a range of seed size, plant height and maturity have been bred.

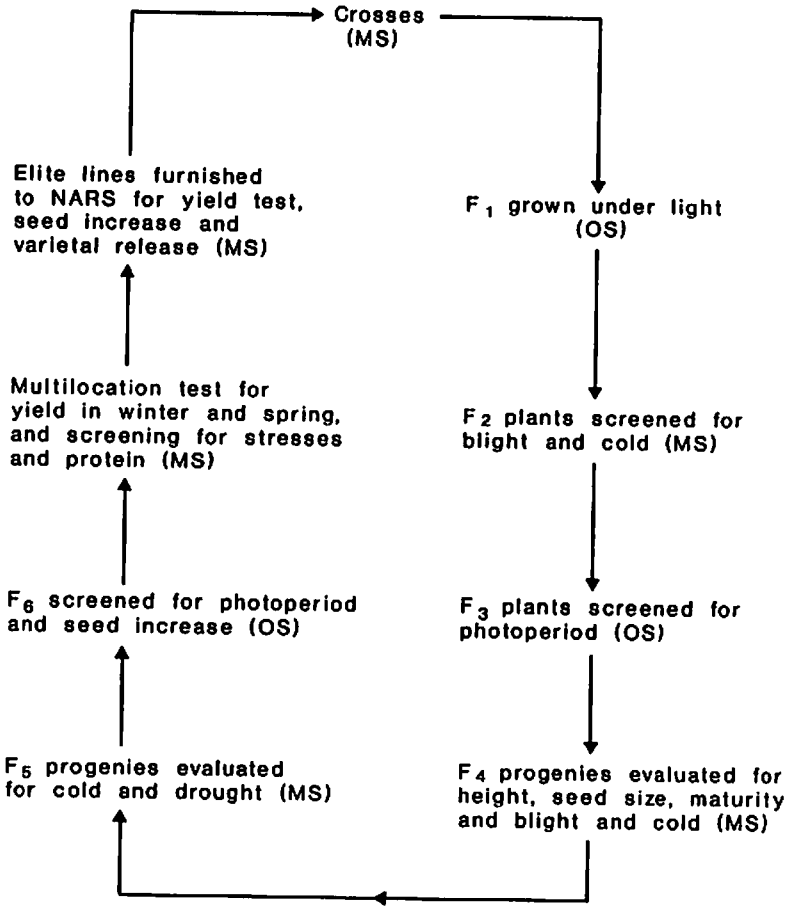

Fig. 2. Varietal development scheme in chickpea as implemented by ICARDA and National Programs. OS = off-season; MS = main season.

Backcross-pedigree method. Original sources of resistance to ascochyta blight were small-seeded, late maturing and poor yielders (Table 9). Success in developing large-seeded and early maturing lines was limited by using resistant sources in hybridization program. Later we discovered that both small seed size and late maturity were dominant over large seed size and early maturity (Singh and Malhotra, unpublished data). We also suspect that there is a linkage, although weak, between ascochyta blight resistance and small seed size and lateness. We observed that straight crosses between small-seeded and large-seeded lines never gave large-seeded segregants in F_2 generations, but when we gave one backcross to a large-seeded parent we were able to recover large-seeded plants in the F_2 generation. Following this observation, we adopted the backcross-pedigree method to develop large-seeded chickpea. A similar practice is being followed to develop an early type chickpea. In order to speed up the cultivar development process, we take three generations in one year: (i) June to September, backcross made in off-season, (ii) October to January, F_1 grown in greenhouse, and (iii) February to June, F_2 grown in the disease nursery.

A diagrammatic presentation of the backcross-pedigree method is given in Figure 3. The scheme is self-explanatory; however, a few clarifications follow. First, selection for less photoperiod sensitivity is practised during the off-season in F_3 and again in F_6 generations to eliminate photoperiod-sensitive lines that are not adapted to spring sowing in the Mediterranean region or winter sowing in the southern latitudes. Second, all F_5 progenies are grown during winter when selection for cold tolerance is practised and during spring when selection for heat tolerance is made. Third, analysis for protein is made mainly to maintain the level of protein content. Following this scheme, it has been possible to breed ascochyta blight-resistant chickpea lines with large seed or medium maturity. Lines with resistance and early maturity are yet to be bred.

Breeding for horizontal resistance

Singh *et al.* (1984) reviewed the published literature on horizontal resistance. As suggested by Robinson (1976), a program for horizontal resistance was designed during the 1980s at ICARDA and some work was done. Crosses were made between lines having tolerant reactions and material was screened in F_2 generations. Most of the segregating plants were found susceptible and a very few were tolerant in the vegetative stage only. Crosses were made between F_2 tolerant plants to produce the next cycle. It was soon realized that recovery of tolerant plants with pod resistance was nil. The whole effort appeared futile and hence the project was abandoned. However, we are in the process of reviving this type of breeding work in cooperation with the Department of Plant Breeding, University of Agriculture, Wageningen, Netherlands.

Multilines

Singh *et al.* (1984) reviewed the literature on multilines. Following Jensen's (1952) proposal, three multilines were composed and tested during 1980/81. The results were interesting (Table 17), but unfortunately the trial was discontinued. However, it would be worthwhile to try this approach again, especially with the new material that is known to differ in resistance to different races. We plan to do this work in collaboration with NARS.

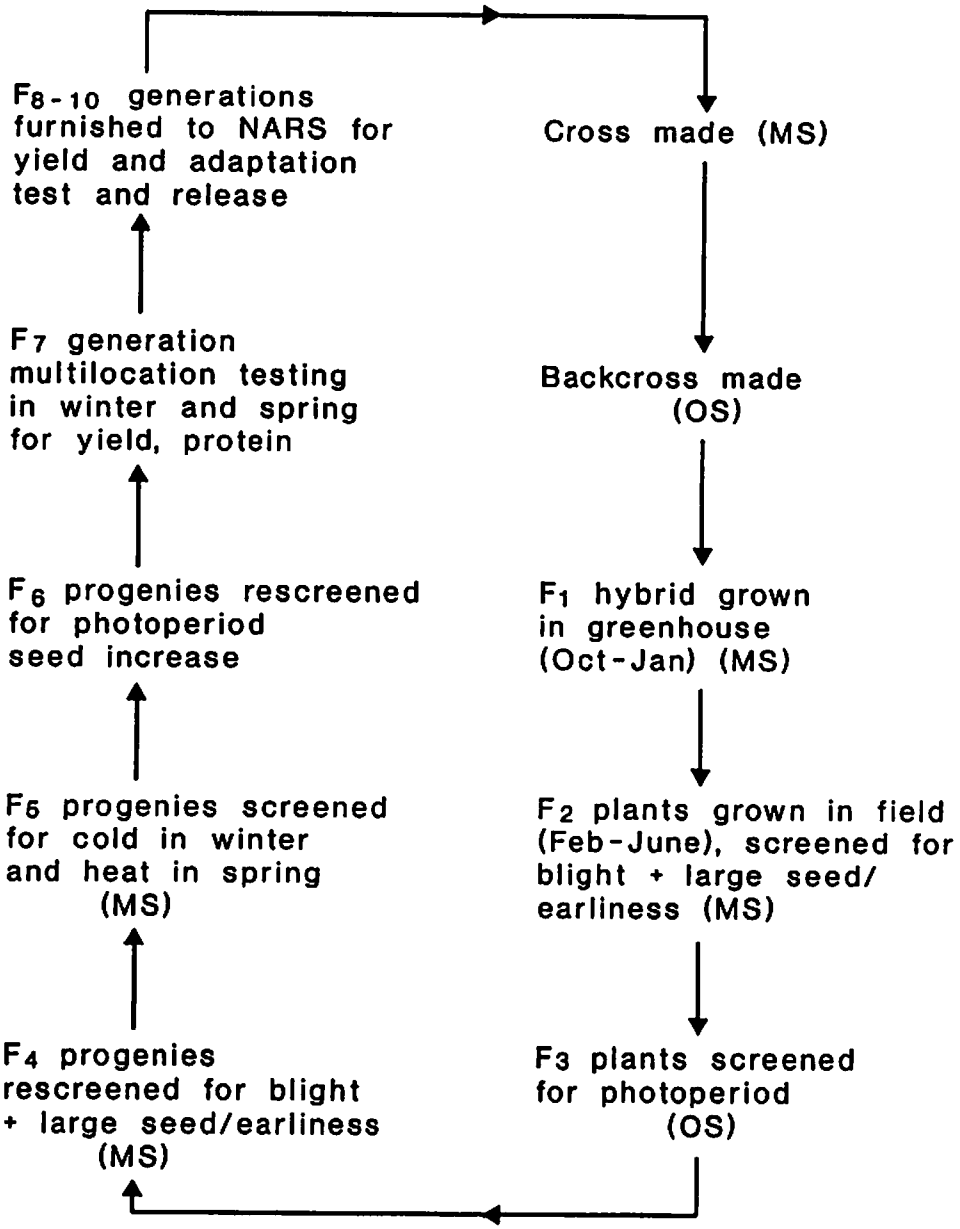

Fig. 3. Backcross-pedigree method used by ICARDA and NARS to develop ascochyta blight-resistant, large-seeded and/or early type kabuli chickpea.

Table 17. Average seed yield and ascochyta blight reaction of cultivars and mixture of cultivars (MC) in a yield trial at Tel Hadya, 1980-81.

Entry	Seed yield		Blight replication on 1-9 scale	
	(kg/ha)	Rank	Plant basis†	Plot basis‡
ILC 482	1935	8	4.07	3.00
ILC 484	2443	1	4.03	3.50
ILC 249	0	12	7.11	7.00
Mean	1459		5.07	4.50
MC1	1610	11	5.38	5.00
ILC 72	1623	10	3.76	3.25
ILC 202	1985	6	4.65	4.75
ILC 3279	1965	7	3.55	3.00
Mean	1858		3.99	3.67
MC2	2044	5	3.40	3.00
ILC 191	2052	4	4.05	4.25
ILC 194	2283	2	3.23	3.25
ILC 201	1702	9	4.43	5.00
Mean	2012		3.90	4.17
MC3	2069	3	3.75	4.25
SE	134.472			
CV (%)	16.838			

† Average reaction of four replications on 20 randomly selected plants per replication.
‡ Average reaction of four replications.

Evaluation of resistance of newly bred lines to ascochyta blight

Between 1978/79 and 1982/83, breeding materials were inoculated with diseased debris collected from the previous season. Since 1983/84 the materials have been inoculated with a spore suspension comprising a mixture of four races (1, 2, 3, 4) multiplied in the laboratory and disease debris. Therefore, all the 650 lines developed from 1981 through 1987 were screened together during 1987/88. Most of the lines had a resistant (1 to 4 rating) or tolerant (5 to 6 rating) reaction and only a small percentage (11.7%) was susceptible (7 to 9 rating) (Table 18).

Since the 1988/89 season, a new strategy has been adopted. We have begun to use a mixture of races 1 to 6 [identified by Reddy and Kabbabeh (1985)] for inoculating the breeding material. This practice has been adopted from the F_2 generation for two reasons. First, there is increasing evidence that race 6 is more widely distributed than was considered in 1983/84. Second, some F_2 populations have been developed using lines resistant to races 5 and 6, the two most virulent races identified from Syria.

Table 18. Evaluation of resistance of breeding lines to races 1-4 of ascochyta blight at Tel Hadya, 1987/88.

Scale†	Breeding lines	
	Number	% of total
1	0	0.0
2	0	0.0
3	129	19.85
4	111	17.07
5	215	33.07
6	119	18.31
7	62	9.54
8	10	1.54
9	4	0.62
Total	650	100.00

† 1 = Free, 5 = Intermediate, 9 = Killed.

Evaluation of Newly Bred Lines for Yield

Multiplication tests of newly bred lines

All newly bred lines are evaluated for yield and other agronomic characters during both the winter and spring seasons at three locations differing in altitude, temperature and annual precipitation each year. Results of the 1987/88 season are cited as an example (Table 19). Promising lines are then provided to NARS through the international nursery network.

Table 19. Performance of newly developed chickpea lines during winter and spring at Tel Hadya, Jindiress and Terbol, 1987/88.

Location and season	No. trials	No. entries			Range		
		Tested	Exceeding check†	Significantly exceed. check	Yield, best entries (kg/ha)	CV (%)	LSD (kg/ha)
Tel Hadya							
- Winter	17	387	44	3	2233-2881	7-17	306-674
- Spring	14	318	117	30	1351-1889	9-21	198-485
Jindiress							
- Winter	12	272	66	0	1915-2948	16-44	512-1394
- Spring	12	272	99	9	805-1905	14-29	240-458
Terbol							
- Winter	17	387	186	12	1810-3369	11-34	323-1112
- Spring	10	226	94	8	905-1926	13-24	218-508

† The check line was ILC 482.

International yield testing

Ascochyta blight- and cold-tolerant lines bred at ICARDA have been provided to NARS in the Mediterranean region in the Chickpea International Yield Trial-Winter-Mediterranean Region (CIYT-W-MR) since 1979/80. Results of 1 year (1986/87) are presented in Table 20. In all 12 countries, newly bred lines were superior to the local check. Second, very high yields were produced with yield potential exceeding 5 t/ha compared with the 0.8 t/ha average yield of the region. Third, yield variation among locations was larger, which was partly because of environment and partly due to management. Fourth, although not clear from the data, the yield of some lines was lower due to low levels of resistance to ascochyta blight and cold.

As well as finished materials, F_4 segregating populations are provided in the form of a CIF_4N for selection under local conditions. Many NARS have taken full advantage of this type of material and have selected and advanced material. Some crosses are made specifically for the needs of NARS.

The ICRISAT/ICARDA Kabuli Chickpea Project assists NARS in on-farm trials by supplying seed of promising lines and rendering advice on the choice of the material. NARS have released more than 20 cultivars from the material developed at ICARDA.

Integrated Control

It was realized that the use of foliar fungicides in the management of blight will not be practical and economical in the case of susceptible cultivars under epidemic conditions when, despite six sprays of Bravo 500, the yield loss in a Syrian local check was 100%. However, a field trial conducted at Tel Hadya during the 1981/82 season indicated that foliar fungicidal spray could be very useful for tolerant cultivars. One spray of Bravo 500 during the early podding stage considerably reduced pod infection in the tolerant cultivar ILC 482 and increased yield (Table 21) (Reddy and Singh 1983). The response to fungicidal spray of the resistant cultivar ILC 195 was low.

The field trials conducted later for three seasons (1982/83, 1983/84, 1985/86) at Tel Hadya with the tolerant cultivar ILC 482 and a limited number of sprays of Bravo 500 during the different stages of crop growth proved that two applications of Bravo 500, one each during the vegetative and reproductive stages or both during the reproductive stages, are highly economical in the management of blight (Table 22) (Reddy and Singh, unpublished data).

Other Diseases

Fusarium Wilt

Fusarium wilt [*Fusarium oxysporum* Schlecht. emend. Snyd. & Hans f.sp. *ciceri* (Padwick) Snyd. & Hans.] is a constraint to spring-sown chickpea in some areas of North Africa and southern Europe. As resistance sources in the kabuli chickpea were not available, 713 lines were screened in collaboration with the Universidad de Cordoba at Santaella,

Table 20. Seed yield (kg/ha) of entries at selected locations. Overall mean is based on all locations (N = 40) in CIYT-W-MR during 1986/87.

Entry	Guelma (Algeria)	Laxia (Cyprus)	Mont-pellier (France)	Tarqu-inia (Italy)	Marow (Jordan)	Terbol (Lebanon)	Zememra (Morocco)	Elvas (Portugal)	Cordoba (Spain)	Al-Ghab (Syria)	Beja (Tunisia)	Meneman (Turkey)	Mean
FLIP 82-101C	2579	1283	2551	4742	968	1583	917	1642	2691	2714	2769	1283	1793
FLIP 82-161C	1954	1242	2477	4071	885	1429	1142	1856	2688	2718	3125	1435	1737
FLIP 83-7C	1852	1369	2501	4226	958	1371	833	1627	2106	2238	2681	1694	1626
FLIP 83-41C	1238	1589	2390	4516	1529	1280	591	1976	1684	2214	2524	1194	1580
FLIP 83-47C	2207	1632	2457	5230	1338	1661	467	2158	1981	2718	2700	1200	1846
FLIP 83-48C	2931	1464	2908	3992	1412	1796	983	2056	2466	2702	3275	1711	1944
FLIP 83-49C	1875	1452	2468	4440	1382	1734	955	2005	2600	2857	2806	1300	1756
FLIP 83-71C	2598	1478	2539	5167	1046	1641	953	2010	2506	3171	3050	1756	1853
FLIP 83-97C	2148	1445	2680	5103	1480	1530	933	1910	2559	3044	2850	1300	1765
FLIP 83-98C	1704	1888	2314	5262	1354	1700	547	1913	2156	3119	2944	1603	1794
FLIP 84-79C	2941	1287	2480	4607	1525	1599	707	1855	2756	3175	3438	1689	1888
FLIP 84-80C	2556	1333	2442	4603	1098	1599	553	1799	2481	3151	2751	1000	1739
FLIP 84-85C	906	1047	2677	3421	1432	1540	908	2013	2275	2405	2519	1167	1704
FLIP 84-92C	2285	1422	2598	5333	1662	1631	1223	2273	2369	2996	3519	1406	1908
FLIP 84-93C	2117	1436	2518	5163	1387	1621	732	1934	2588	3044	3706	1303	1871
FLIP 84-99C	1783	1342	2791	3619	1613	1772	1009	2002	1784	2365	1744	1367	1821
FLIP 84-102C	1445	1523	2553	5040	1742	1591	715	2151	2506	3044	3169	110	1849
FLIP 84-109C	1729	1735	2906	4750	1615	1520	1027	1995	2350	2663	3531	1483	1878
FLIP 84-144C	803	1774	2864	5103	1250	1669	1150	2281	2609	2520	3550	1494	1915
FLIP 84-145C	1903	1872	3363	4853	1076	1841	1013	2269	2353	2587	3250	1494	1988
FLIP 84-158C	1441	1570	2966	4766	1419	1738	693	2066	2075	2611	3481	1317	1919
FLIP 81-293C	929	2596	3068	4353	1023	1651	805	2349	2791	2933	3488	2578	1742
ILC 482	1015	1633	2776	4270	1005	1774	1128	2606	2269	2290	3506	1222	1846
Local check	789	1438	3059	4024	505	1569	1218	1188	2244	2631	1950	1244	
Location mean	1822	1494	1685	4611	1279	1618	883	1997	2370	2746	3055	1389	
SE of mean	356	140	164	334	305	58	211	146	268	208	271	188	
CV (%)	39	19	12	15	48	7	48	15	23	15	18	27	

Table 21. Effect of foliar application of Bravo 500 on blight severity and yield of chickpea cultivars of differing degrees of susceptibility, Tel Hadya, 1981/82.

Cultivar	Time of application	Blight severity (1-9) Vegetative stage	Blight severity (1-9) Podding stage	Seed yield (kg/ha)
ILC 195	Flowering	3.0	3.3	909
	Early podding	3.0	2.8	1658
	Late podding	3.5	4.0	1124
	Control	3.0	3.3	1371
ILC 482	Flowering	5.0	8.0	717
	Early podding	5.0	5.3	1604
	Late podding	5.8	7.8	504
	Control	5.8	8.0	287
ILC 1929	Flowering	9.0		0
	Early podding	9.0		0
	Late podding	9.0		0
	Control	9.0		0
SE				160.2
CV (%)				51.7

Table 22. Cost benefit ratio of two foliar applications of chlorothalonil (Bravo 500) for control of ascochyta blight in tolerant chickpea cultivar ILC 482, Tel Hadya, Syria, 1982/83 to 1985/86.

Crop stage at spraying	Yield (kg/ha)	Value of additional yield ($)	Cost benefit ratio†
Seedling and early podding	2520	303.5	1:8
Seedling and late podding	2254	211.5	1:5
Midvegetative and early podding	2104	159.5	1:4
No spray control	1643		

†1 = Average of two seasons.
2 = Average of three seasons.
3 = At the rate of $ 0.35/kg of chickpea seed.
4 = Cost of two sprays of chlorothalonil at $ 38.50/ha.

Cordoba, Spain during 1987. Results are reported in detail by Prof. R.M. Jimenez-Diaz, a consultant to this meeting, but we present the summary here. Nine breeding lines were found to be resistant (Table 23). Four lines (FLIP 82-78C, FLIP 84-43C, FLIP 84-130C and FLIP 85-85C) showed 0 to 8.7% dead plants. These lines would be useful in multiple-resistance breeding programs.

Table 23. Evaluation of chickpea breeding lines resistant to fusarium wilt at Santaella, Cordoba, Spain, 1987.

Class†	No. of accessions	% of total
Resistant	9	1.3
Moderately resistant	22	3.1
Susceptible	682	95.6
Total	713	100.0

† Resistant = 0-20% plants killed from wilt, moderately resistant = 21-50% plants killed from wilt; susceptible = 51-100% plants killed.

Diseases other than Ascochyta Blight and Fusarium Wilt

Diseases other than ascochyta blight and fusarium wilt prevalent in the kabuli chickpea-growing regions are stunt (bean leaf-roll virus), root rots (*Rhizoctonia solani*, *Rhizoctonia bataticola*, *Fusarium solani*) and sclerotinia stem rot (*Sclerotinia sclerotiorum*). Screening for resistance or work on any other aspect has not been carried out at ICARDA.

Nematodes

Among the nematodes found attacking chickpea in Syria, cyst nematode (*Heterodera ciceri*) caused damage in parts of the country. Effective control of this nematode was observed through the use of nematicides, but these chemicals were uneconomical. Therefore, host-plant resistance is under investigation. No source of resistance has been found in 4600 germplasm accessions evaluated. However, tolerance has been seen in *Cicer bijugum* (Table 24) (Di Vito *et al.* 1988). Additional effort is continuing to locate resistance sources within the cultivated species because it has not been possible to cross *C. bijugum* with cultivated species.

Multiple Disease Resistance

In addition to ascochyta blight, other diseases such as wilt, root rots, seed rots and nematodes are problems in certain areas of kabuli chickpea-producing countries in North America, southern Europe and the Americas. In Tunisia, work on development of lines with combined resistance to fusarium and verticillium wilts and ascochyta blight has been in progress for the past several seasons. A breeding line (FLIP 83-60C) with tolerance

Table 24. Screening of *Cicer* species for resistance to cyst nematode at Tel Hadya, 1987/88.

Cicer species	Nematode reaction†
C. bijugum	T
C. chorassanicum	S
C. cuneatum	S
C. echinospermum	S
C. judaicum	S
C. pinnatifidium	S
C. reticulatum	S
C. yamashitae	S
C. arietinum	S

† T = tolerant, S = susceptible.

to wilt complex and blight has been identified (ICARDA 1986). There are no reports on identification of lines with multiple resistance to root rots, wilt, nematodes and blight and work on these lines needs to be strengthened.

Looking Ahead

The breeders/pathologists working on ascochyta blight resistance in chickpea should consider the following points:

1. How does the blight develop in epiphytotic form so suddenly in India and Pakistan where the sexual state of *A. rabiei* has not even been found? Blight causes severe damage from time to time in the Mediterranean region, but it never assumes epiphytotic form on a scale seen in India and Pakistan. Why?

2. There is a big debate about the existence of physiologic races of *A. rabiei*, although more evidence of their existence is being published. More studies need to be devoted to resolving this issue.

3. Inheritance of resistance to *A. rabiei* has been reported to be governed by a few genes. Some breeders believe that inheritance of resistance is under the control of polygenes due to continuous gradation of plants in different categories. Studies should be conducted to clarify the nature of inheritance to resistance of *A. rabiei*.

4. There seems to be little correlation between screening in field and greenhouse for resistance to ascochyta blight. It will be interesting to study this lack of correlation between the two screening methods.

5. It has been our experience in 11 years of breeding that ascochyta blight-resistant plants are late-maturing, even in crosses between early and late maturity lines. The cause for this needs to be examined. It is an important point because ascochyta blight-resistant, early maturity lines are required for spring sowing in the Mediterranean region. The progress in breeding early maturing resistant lines has been disappointing.

6. International testing of newly bred blight-resistant lines indicates that no line is resistant across the countries. One could infer from this result that resistance in new lines is location specific. At the international centers, effort should be made to develop location nonspecific resistance.

7. ILC 482 has partial resistance, being resistant at the vegetative but susceptible at the podding stage, and yet it has been released in seven countries. Does this mean that cultivars with partial resistance will be favored in future?

8. A little effort has been made in the past to develop cultivars with combined resistance to two or more diseases. Undoubtedly, cultivars with multiple-disease resistance will be more durable and acceptable to growers.

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Breeding for Resistance to Soil-borne Diseases in Chickpea

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Abstract

All soil-borne diseases of chickpea (*Cicer arietinum*) are mentioned, but only three of them are discussed in detail: fusarium wilt (*Fusarium oxysporum* f.sp. *ciceri*), dry root rot [*Rhizoctonia bataticola* (*Macrophomina phaseolina*)] and collar rot (*Sclerotium rolfsii*). This paper addresses techniques of screening for resistance, resistance sources, infection and resistance mechanisms, inheritance of resistance and proposed techniques for resistance breeding. So far only fusarium wilt can be handled with confidence in breeding programs.

Introduction

All diseases caused by pathogens that live in the soil and infect the root or lower part of the stem are called soil-borne diseases in this paper. They correspond with what Shipton (1984) calls foot and root rots for fungi, and with what Nene and Reddy (1987) grouped as fungal diseases infecting root and stem base, but comprise, additionally, foot and root diseases caused by nonfungal pathogens.

Nene and Reddy (1987) described nine fungal foot and root diseases, but only three are considered of global importance. These are: fusarium wilt, caused by *Fusarium oxysporum* Schlecht. emend. Snyd. & Hans. f.sp. *ciceri* (Padwick) Snyd. & Hans., dry root rot caused by *Rhizoctonia bataticola* Taub Butler (*Macrophomina phaseolina*) and collar rot, caused by *Sclerotium rolfsii* Sacc. None of the viral or bacterial diseases mentioned by the latter authors are soil-borne, but diseases caused by nematodes are, and in this group the root knot nematodes (*Meloidogyne* spp.) and cyst nematodes (*Heterodera* spp.) are probably the most important (Greco 1987; Sharma 1988). This paper deals only with resistance breeding for the three major soil-borne fungus diseases. We discuss resistance mechanisms, which could be important for screening and breeding procedures, summarize what is known about the inheritance of disease resistance, because inheritance pattern largely determines the breeding strategy to be followed. Finally, we propose techniques for resistance breeding that are either specific, where sufficient information is available on the resistance factors, or general, where sufficient information is lacking.

The terminology used in this paper follows the terms proposed by Robinson (1987) mainly for convenience and uniformity, although not all of these are commonly accepted, e.g., "horizontal" resistance (Nelson 1973).

Major Soil-Borne Diseases

Chickpea suffers from severe soil-borne diseases. In the order of importance, these are wilts (*F. oxysporum* f.sp. *ciceri* and *Verticillium albo-atrum* Reinke & Berth.), dry root rot (*R. bataticola*), collar rot (*S. rolfsii*), wet root rot (*Rhizoctonia solani* Kühn), black root rot [*Fusarium solani* (Mart.) Appel & Wr.], phytophthora root rot (*Phytophthora megasperma* Drechler), pythium root and seed rot (*Pythium ultimum* Trow.) and foot rot (*Operculella padwickii* Kheswalla) (Nene and Reddy 1987). The soil-borne diseases are most important in areas between latitudes 0 and 20°, where the chickpea-growing season is dry and warm. This is partly due to high temperature requirements of the fungi (Fig. 1) and accentuation of wilt and root rot symptoms under moisture stress conditions. The distribution of these diseases across the major chickpea-producing countries is known (Table 1), but precise information on the losses caused by them is not available. Wilt in India has been found to cause about 10% yield loss (Singh and Dahiya 1973).

Kabuli types are more susceptible to soil-borne diseases than desi types. More than one soil-borne disease commonly occurs in the same field at the same time, but most produce specific and characteristic symptoms and thus it is possible to diagnose them on the basis of symptoms, especially in the early stages of disease development (Nene *et al.* 1978). In addition to temperature and soil moisture, the age of the plant also determines its susceptibility to these diseases.

Fusarium wilt is the most important and widely distributed soil-borne disease (Table 1). Temperatures between 20 and 25°C are optimal for growth of the fungus (Fig. 1). The pathogen is both seed- and soil-borne (Haware *et al.* 1978). It can survive in the soil in the absence of chickpea for at least 6 years. Pigeonpea, lentil and pea can carry the wilt fungus as symptomless carriers. The pathogen exhibits physiologic specialization. Resistant and tolerant genotypes support the multiplication of the pathogen in the soil similarly to the susceptible types (J.N. Rao, M.V. Reddy and Y.L. Nene, unpublished data). An inoculum threshold of 67 to 483 propagules/g soil causes 100% mortality in susceptible cultivars.

Among the root and seed rots, dry root rot is the most important disease, causing the most severe losses in dry and hot conditions (35°C). The damage due to dry root rot is generally most extensive at the maturity stage, whereas other root and seed rots prevail in wet and warm (25 to 30°C) situations and the losses are more in the seedling stage.

Collar rot incidence is most common in the seedling stage under wet and warm soil conditions, especially when undecomposed organic matter is present in the soil.

Table 1. Distribution of major soil-borne diseases of chickpea†.

Disease	Country
Fusarium wilt	Algeria, Argentina, Australia, Bangladesh, Burma, Chile, Ethiopia, India, Italy, Malawi, Mexico, Morocco, Nepal, Pakistan, Peru, Spain, Syria, Tunisia, USA
Dry root rot	Australia, Ethiopia, India, Iran, Pakistan, Spain, USA
Collar rot	Bangladesh, Ethiopia, India, Pakistan, Syria
Wet root rot	Argentina, Australia, Chile, India, Iran, Mexico, Pakistan, Syria
Black root rot	Chile, India, Mexico, Spain, USA
Verticillium wilt	Pakistan, Tunisia‡
Phytophthora root rot	India, Australia
Pythium root and seed rot	Chile, India, Turkey, USA
Foot rot	India

† Nene *et al.* (1984); Nene and Reddy (1987).
‡ Halila and Harrabi (1987).

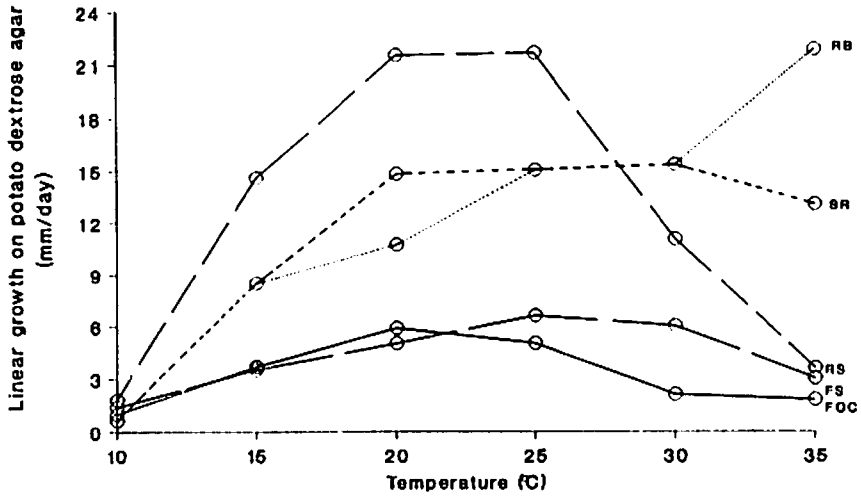


Fig. 1. The effect of temperature on growth of *Fusarium oxysporum* f. sp. *ciceri* (FOC), *F. solani* (FS), *Rhizoctonia bataticola* (RB) and *Sclerotium rolfsii* (SR).

Techniques for Screening for Resistance to Soil-borne Diseases

Fusarium wilt

Efficient field, greenhouse and laboratory inoculation techniques for rapid and large-scale screening of germplasm and segregating breeding materials are available (Nene *et al.* 1981). Experience at ICRISAT has shown that the development of a sick plot in slightly alkaline vertisol (pH 8.85) is relatively easy. Selecting a field that shows some incidence of wilt in patches and then growing a susceptible cultivar for two to three seasons and incorporating the dead plants in the field will result in a uniform sick plot. Thereafter, growing a susceptible cultivar after every two to four test rows helps in monitoring the wilt incidence and maintaining sickness in the field (Nene *et al.* 1981).

Dry root rot

As the disease development is highly influenced by environmental factors such as temperature and soil moisture, screening for dry root rot under field conditions is not as efficient as for fusarium wilt. High temperatures (30°C) and dry soil conditions, especially at flowering and podding stage, can considerably enhance disease development. Fields developed for fusarium wilt screening at ICRISAT Center were found to be infected with *R. bataticola* and were useful in eliminating the highly dry root rot-susceptible types. There are no reported cases of uniform and effective dry root rot-sick plots having been developed. Pot culture techniques for greenhouse screening and a paper towel technique for laboratory screening have been developed (Nene *et al.* 1981). There is, however, scope for further improvement in the techniques to make them correspond more reliably with field resistance. Further studies on the influence of inoculum levels, age of the plants, temperature and moisture on the host-plant resistance need to be done.

Collar rot

There are no reports on the development of field plots for screening of collar rot resistance in chickpea, but good infections can be achieved in pot culture. Multiplying the fungus on sorghum stem pieces, mixing them in the soil in pots or trays and incubating them at 25 to 30°C with a high level of moisture was found to produce about 80% mortality in susceptible cultivars.

Sources of Resistance to Soil-borne Diseases

For fusarium wilt, several good sources of resistance at individual and multiple locations are available (Tables 1 and 2). These sources retain resistance under high levels of inoculum in the soil. Resistance is available in both desi and kabuli types. Also, good progress has been made in the development of wilt-resistant high-yielding cultivars (Singh

Table 2. Number of chickpea lines tested and resistant to wilt and root rots at different locations in the International Chickpea Root Rot and Wilt Nursery (1978-82)†.

Location	Total entries tested	Duration of resistance (years)‡		
		1	2	3
Bangladesh-Joydebpur	59	18	1 (ICC 11531)	NT
Egypt-Giza	55	28	NT§	NT
Ethiopia-Debre Zeit	141	53	10 (ICC 10,102,434, 1910,1913,6366,6455, 6926,8004,8970)	None
India				
Berhampore	141	11	1 (ICC 7248)	1(ICC 267)
Bijapur	56	14	NT	NT
Dahod	57	30	NT	NT
Delhi	120	13	None	None
Dholi	32	32	NT	NT
Durgapur	59	17	None	NT
Faizabad	120	9	None	None
Gurdaspur	141	62	None	None
Hisar	141	62	3 (ICC 1443,1611,6671)	1 (ICC 858)
Jabalpur	120	89	37	4 (ICC 267, 2083,3103, 3439)
Kanpur	141	18	None	None
Ludhiana	141	29	1 (ICC 4552)	1 (ICC 858)
Patancheru (ICRISAT)	141	55	26	3 (ICC 858, 2083, 3103)
Varanasi	126	61	1 (ICC 4552)	None
Mexico-Culiacan	176	77	12	1 (ICC 8933)
Nepal-Parwanipur	81	3	2 (ICC 267,10104)	None
Peru-Chiclayo	63	56	4 (ICC 858,8003,8004, 11531)	None
Spain-Cordoba	6	5 (ICC 31,41, 858,11088, ICCL 8011)	NT	NT
Sudan-Hudeiba	80	18	None	None
USA-San Luis Obispo	141	119	45	7 (ICC 267, 519,858, 2566,2660, 3439,8933)
Yemen Arab Republic-Ibb	85	4 (ICC 190,435, 5864,8976)	None	NT

† Unpublished data provided by Y.L. Nene.

‡ Any entry was considered resistant when it showed resistance (< 10% mortality) in each year at a particular location. ICC 858 was resistant at Patancheru for 4 years. ICC 858, 3439 and 8933 were resistant for 4 years in the USA and ICC 8933 was resistant for 5 years.

§ NT = not tested.

1987). Compared with fusarium wilt, studies on the resistance to dry root rot at different locations are limited. Experience at ICRISAT shows that some lines have reasonable field tolerance to dry root rot (Tables 2 and 3). Although these show some root rot, they do not dry prematurely. In the multilocation trials, they showed less than 10% mortality. Lines such as ICC 12441 and ICC 12450 showed tolerance to dry root rot in pot culture, paper towel screening and sick plots at ICRISAT. At present, there are no reports on resistance sources for sclerotium collar rot in chickpea.

Table 3. Chickpea lines with broad-based resistance to wilt and root rots identified through International Chickpea Root Rot and Wilt Nursery†.

	Reaction of line‡					
	ICC 2862	ICC 9023	ICC 9032	ICC 10803	ICC 11550	ICC 11551
Bangladesh-Joydebpur	S	S	S	R	R	R
Ethiopia-Debre Zeit	R	S	R	R	R	R
India						
Berhampore	S	S	S	S	R	S
Dahod	R	R	R	R	R	S
Delhi	R	R	S	S	R	R
Dholi	R	R	R	R	R	R
Durgapur	S	R	R	S	S	S
Faizabad	S	R	S	S	S	S
Gurdaspur	S	R	S	S	S	S
Hisar	R	R	R	R	S	S
Jabalpur	R	R	R	S	R	R
Kanpur	S	R	R	S	R	R
Ludhiana	S	S	R	R	R	R
Patancheru (ICRISAT)	R	S	R	R	S	S
Varanasi	S	S	S	S	S	R
Mexico-Culiacan	R	R	R	R	S	R
Peru-Chiclayo	R	R	R	R	R	R
USA-San Luis Obispo	R	R	R	R	R	R
No. locations tested	18	18	18	18	18	18
No. locations resistant	10	12	12	10	11	11

† Unpublished data provided by Y.L. Nene.
‡ R = <10% mortality, S = >10% mortality.

Infection and Resistance Mechanisms of the Major Soil-borne Diseases

The vascular wilt diseases are distinct from those that produce local lesions in that the infection becomes systemic. The pathogen resides principally in the xylem vessels. Its propagules and the polluting products of its action, or of its interaction by the host, may be moved throughout the stems and leaves by the transpiration system.

The precise mode of entry of *F. oxysporum* into the vascular system remains unknown. Penetration of the root surface is occasionally prevented by physical or chemical barriers. It has been suggested that the root exudates from susceptible and resistant cultivars may stimulate or inhibit the process of pathogenesis in diseases caused by soil-borne pathogens, including the vascular wilt pathogen (Schroth and Hildebrand 1964). The studies at ICRISAT (Haware and Nene 1984) suggested that the resistance of CPS 1 to race 1 of *F. oxysporum* f.sp. *ciceri* was caused by the production of a root exudate that inhibited spore germination and retarded mycelial growth. The roots of JG 62 produced an exudate capable of stimulating spore germination, which might account for the extreme susceptibility of this cultivar.

It is unlikely that disease resistance in vascular wilt is dependent upon a single mechanism. Coordinated chemical defense mechanisms, with different metabolic sites of action at different stages in the host-parasite interaction, may explain the long-term stability of wilt-resistant chickpea cultivars.

Once the infection has occurred in the roots, the pathogen tries to invade vascular tissues. Vascular colonization by the wilt pathogen is extensive in wilt-susceptible plants, but is limited to the basal part of late wilters and restricted to the root of resistant chickpea plants (M.P. Haware, unpublished). Wilting of the plant depends upon the capacity of the pathogen to invade and establish in the conductive xylem system.

Vascular gelation, the first visible structural change in the sequence of vessel-occluding processes, begins during the first day after vascular infection (Beckman and Halmos 1962). The gels, which are highly resistant to physical and chemical degradation, serve to cut off the transpiration stream and to embed and immobilize the secondary spores of the parasite at the sites of their formulation.

Soil and air temperatures play a determining role during disease development and in the expression of wilt symptoms.

Infection by *R. bataticola* may occur through cotyledons during emergence, through small rootlets, or through small wounds on the root surface. The fungus grows inter- and intracellularly and invades the cortical cells. Hyphae colonize the vascular system and sclerotia develop in the xylem vessels. Dry root rot of chickpea is considered to be a high-temperature pathogen. The severity of the disease increases with increasing temperature with a maximum between 28 and 35°C.

Infection of chickpea and other crop plants with *S. rolfsii* occurs in wet soil. This fungus is very active near the soil surface. It has a low competitive saprophytic ability in the soil and relies on saprophytic growth in the dead host remains. Therefore, removal of plant debris from the field reduces plant mortality caused by *S. rolfsii*.

Inheritance of Resistance to the Major Soil-borne Diseases

The inheritance of resistance to soil-borne diseases has been reported only for *F. oxysporum* and *R. bataticola*. These are described here and suggestions for future studies are given.

Wilt

Studies conducted under field conditions indicate that resistance to fusarium wilt is governed by a single recessive gene (Haware *et al.* 1980; Kumar and Haware 1982; Sindhu *et al.* 1983). Haware and Nene (1982) reported the existence of physiological races in *F. oxysporum* f.sp. *ciceri*. Systematic work on the inheritance of race 1 of this pathogen at the ICRISAT center was initiated in 1978. Kumar and Haware (1982) found that resistance in each of the crosses of resistant parents WR 315 and CPS 1 with susceptible cultivar C 104 (kabuli) segregated for one recessive gene. No segregation for wilt susceptibility occurred in the WR 315 and CPS 1 cross. However, when the crosses of the same parents with susceptible cultivar JG 62 were tested, the proportion of the susceptible segregant was much more than could be explained by a 3 (susceptible) to 1 (resistant) ratio.

Continuing these studies, Upadhyaya *et al.* (1983a) observed a difference in the number of days taken to wilting by the two susceptible cultivars JG 62 (early wilting) and C 104 (late wilting). This difference in early and late wilting was governed by a single gene with early wilting partially dominant to late wilting (Upadhyaya *et al.* 1983b). Singh *et al.* (1987) studied a cross of two late-wilting parents (C 104 and K 850) and recovered a resistance segregant in the F_2 generation. The two genes for late wilting appeared to complement each other to impart complete resistance to an individual carrying these in homozygous recessive form. The F_2 generation segregated in a 9 (early) to 6 (late) to 1 resistant ratio. In a later study of crosses of another late-wilting parent (H 208) with C 104 and K 850, resistant segregants were recovered in each cross (Singh *et al.* 1987). The resistance gene in H 208 was partially dominant. The genotypes and reactions of the parents studied are listed below:

JG 62	H_1H_1 H_2H_2 h_3h_3	early wilting
K 850	h_1h_1 H_2H_2 h_3h_3	late wilting
C 104	H_1H_1 h_2h_2 h_3h_3	late wilting
H 208	H_1H_1 H_2H_2 H_3H_3	late wilting
WR 315	h_1h_1 h_2h_2 h_3h_3	resistant
CPS 1	h_1h_1 h_2h_2 h_3h_3	resistant

Thus it appears that resistance can be recovered through hybridization of any two of the abovementioned late-wilting parents. We have unpublished data to show that there are several late-wilting genotypes in the available germplasm collection at ICRISAT and some of these complement the known genes for late wilting to impart complete resistance. Our studies indicate that other mechanisms of resistance exist. Recently, JG 62 was shown to be resistant to a new race in Tunisia and Spain. Studies with other races have not been done. It is possible that some of the genes effective to race 1 may operate against other races as well. Sources of resistance to more than one race exist and have been utilized in developing resistant cultivars.

To understand the mechanisms of resistance, studies of other late-wilting and resistant parents with race 1 and other races are necessary.

Dry root rot

A single report is available on the inheritance of resistance to dry root rot (Ananda Rao and Haware 1987). Crosses of two resistant (H 208 and C 104) parents with two susceptible parents (K 850 and C 104) segregated for a single dominant gene for resistance. Recently it has been observed that even the resistant parents develop symptoms of the disease if plants are grown for a longer period in infected soil (S.K. Singh, pers. comm.). However, lines with higher levels of resistance are not known. Inheritance studies of resistance to *F. solani* and *S. rolfsii* have not been conducted.

Techniques of Breeding for Soil-borne Disease Resistance

Soil-borne diseases are less mobile than air-borne diseases and therefore may seem less dangerous, more static and more predictable, but this is not necessarily the case. If a field is infected with a soil-borne pathogen, it poses a continuous threat to a susceptible crop. This is different from air-borne diseases, where often a period of absence or reduced presence of the pathogen occurs. Possibilities of change due to mutation, heterokaryosis, or sexual propagation are similar for air-borne and soil-borne diseases, or possibly even greater for the latter because of their sustained population size over seasons. Only the spread of new pathotypes will be slower. It is suggested that in principle the breeding for resistance to soil-borne disease may not be different from breeding for resistance to other diseases, or to other stress factors for that matter.

Fusarium wilt has been studied more extensively than the other major chickpea soil-borne fungal and nematode diseases. This will have a bearing on the techniques to be applied in resistance breeding, as is discussed below.

Fusarium wilt

The inheritance of resistance to wilt is relatively simple and the situation for chickpea at present is a typical example of vertical resistance breeding with differential reactions between pathodemes and pathotypes (Haware and Nene 1982; ICRISAT 1989; Smithson

1985). The oligogenic nature of the control of fusarium wilt gives no reason to be alarmed. ICRISAT reports and personal communications confirm that over a period of more than 10 years and in different environments, the resistance has been durable. No cases of genetic defeat of pathodemes by pathotypes that have acquired adjusted pathogenicity or virulence have been reported and the fusarium wilt case resembles those of many other diseases where simply inherited resistances have been durable (Parlevliet 1983). The techniques for resistance breeding are therefore relatively easy, as experienced at the ICRISAT Center and other institutions, where uniform wilt-sick fields are available. Conventional breeding methods for self-pollinating crops can be applied: bulk population breeding, pedigree breeding and backcross breeding (Fehr 1987). Combinations of these methods also are possible. We will look at the first and the last method in more detail.

Pedigree breeding is similar to bulk population breeding, but it starts with a single plant selection in the F_2 rather than in F ≥4. Figure 2 shows a possible procedure for bulk-population breeding in which the numbers given are arbitrary. The scheme starts with the selection of parents: an agronomically good but susceptible cultivar as female, P_1 and a good source of resistance, P_2. The number of F_1 seeds required depends on the desired size of the F_2 population. If we want to assemble the two wilt-resistant genes and eight independent field genes (2n = 16 chromosomes for chickpea) from the good cultivar, which are absent in the donor, then the smallest complete F_2 population must consist of 1.05×10^6 plants (Sneep 1977). Only plants having the alleles $h_1h_1h_2h_2$, or one-sixteenth of the population, will survive, leaving us with 6.6×10^4 plants. Such numbers cannot be realized, but in the F_2, one in every 10 plants may still carry all the eight desired yield genes and therefore a random sample of 40 F_2 surviving plants may not yet have suffered an irreparable loss of desired alleles. In later generations these numbers increase considerably. The conclusion is that the larger the population, the better it will be, but for practical reasons and also in order to accommodate more crosses, the numbers are reduced.

The effective F_2 population in scheme 1 (Fig. 2) corresponds with the minimum number of F_2 plants mentioned by Allard (1960) for pedigree breeding. The F_3 population may be exposed to a second stress factor, for example, ascochyta blight. The F_4 population is then ready for single plant selection and the scheme finally ends with the three-location replicated testing of the $F_{4:6}$ progeny bulks. Through all generations from F_2 to F_6, selection for highly heritable characters like flowering initiation and seed size can be done effectively.

Figure 3 shows a possible procedure for backcross breeding. The number of backcrosses needed depends on the difference between a donor and recurrent parent. Wehrmann *et al.* (1988) observed that one backcross was sufficient for soybeans if the donor yielded ≤10% less than the recurrent parent. For kabuli chickpea this may be possible if the BC_0F_2 population is large and is grown in a wilt-sick field, with superimposed artificial ascochyta blight infection. It may, however, be advisable to continue the backcross program for several more generations and then test differences between BC_0 and BC_5 at the end of the program.

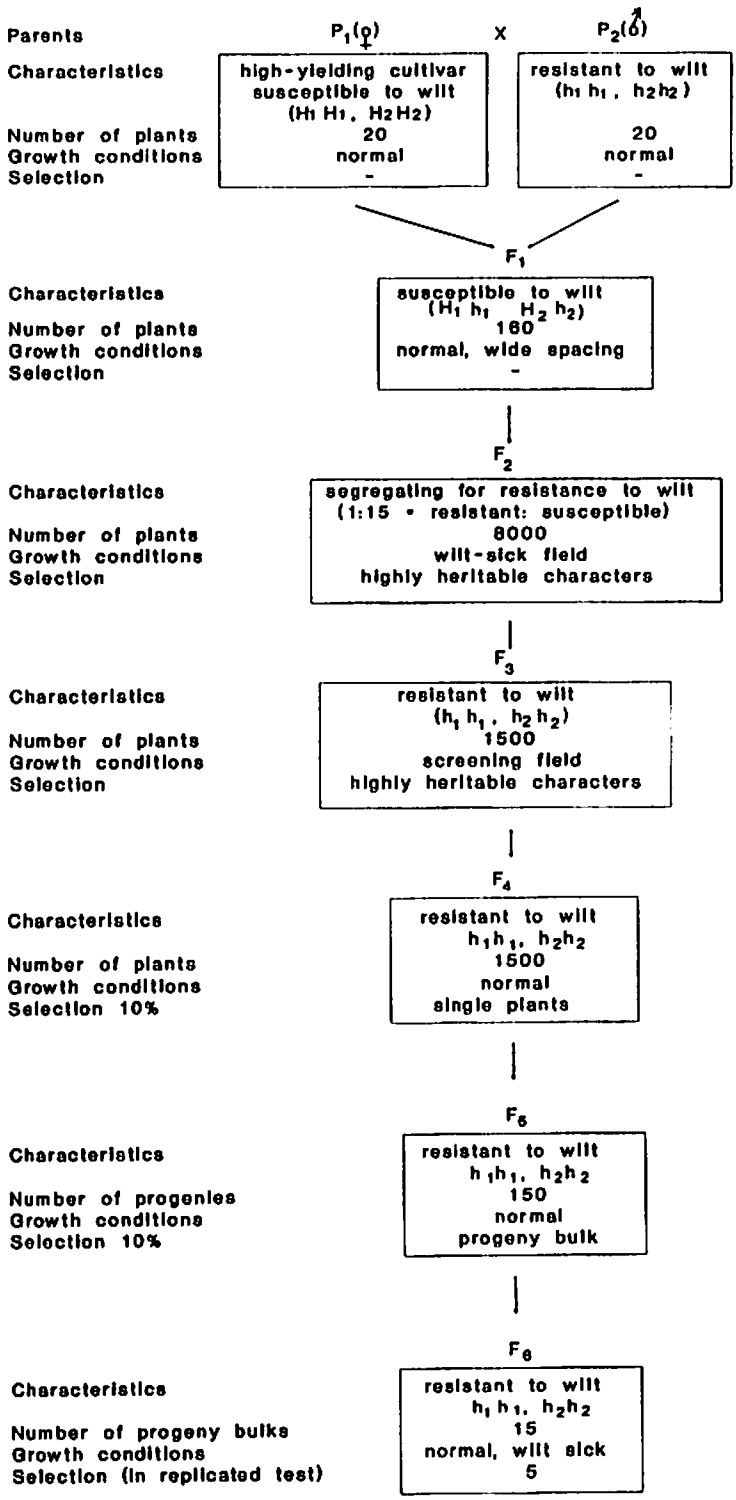


Fig. 2. Bulk-population breeding for the development of fusarium wilt-resistant chickpeas; h1 and h2 are resistance genes for fusarium wilt.

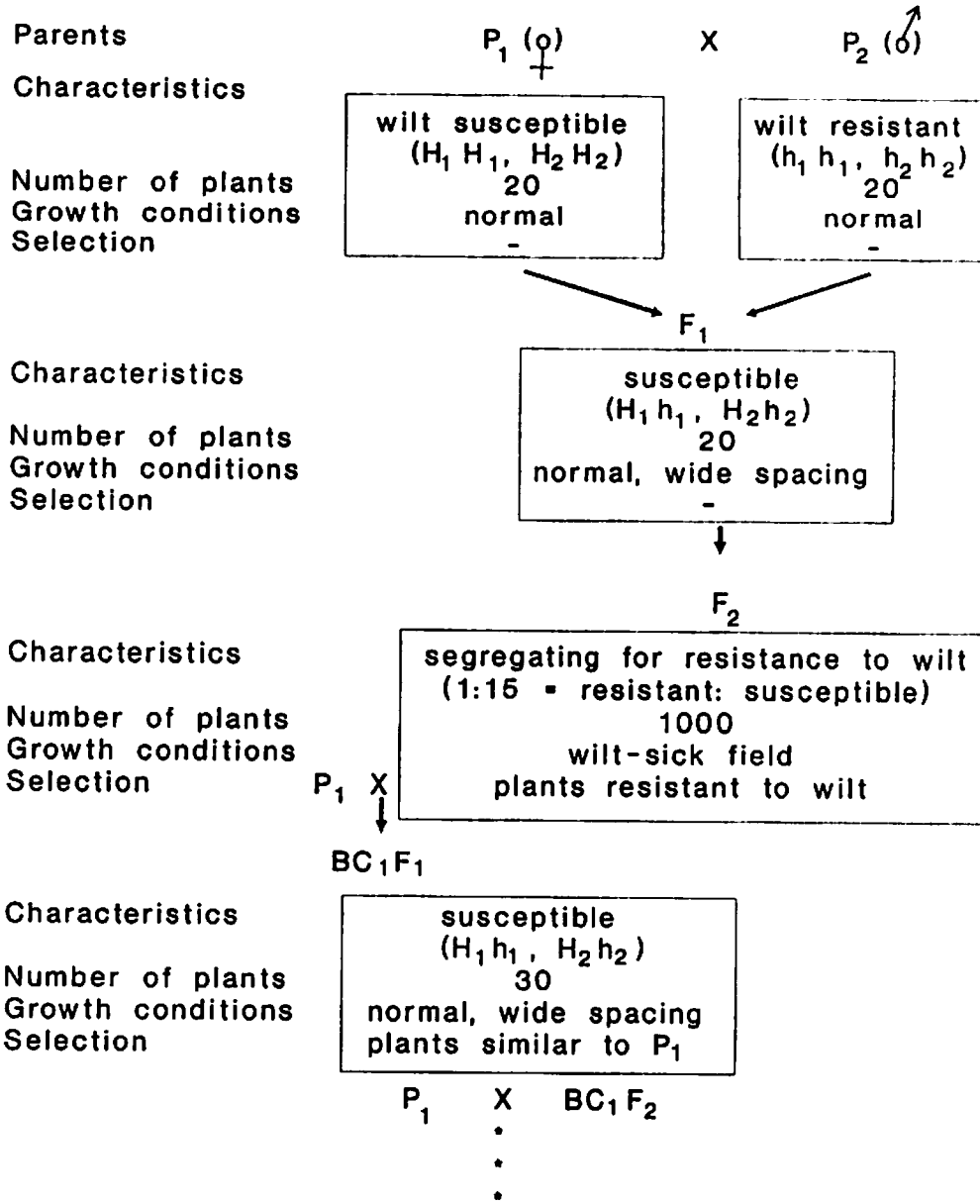


Fig. 3. Backcross-breeding for the development of fusarium wilt-resistant chickpea; h1 and h2 are resistance genes for fusarium wilt.

Other soil-borne diseases

From the sections on techniques of screening for resistance and inheritance of resistance to the major soil-borne diseases, it is clear that the screening techniques for dry root rot, collar rot and nematodes need further improvement; that the sources of resistance need further confirmation and study; that the inheritance of the resistance is not yet known, and that we have no documented cases of a breakdown in resistance to any of the three pathogen species. This puts the breeding for resistance in a state of uncertainty. Therefore, we can make only some general suggestions.

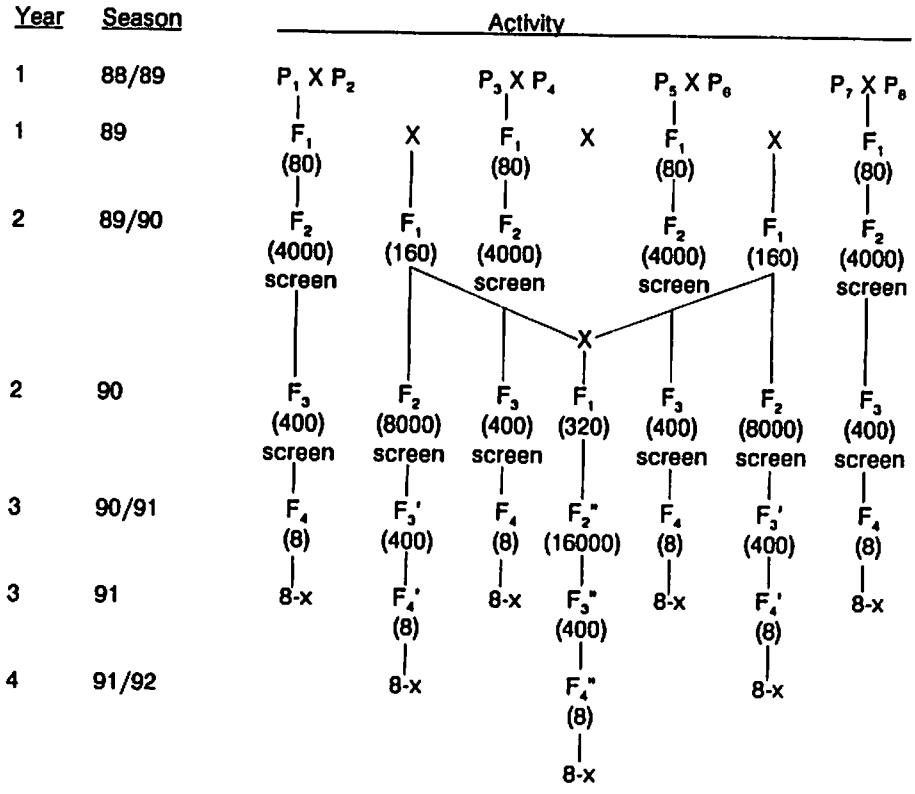
As there is no reason to assume that the resistances so far reported are unstable or not durable, we suggest the following methodology for use, assuming that screening facilities and reported sources of resistance are available.

A selected cultivar is crossed with a resistant line. The F_1 is grown under optimal conditions. The F_2 population is sown in the disease nursery for screening purposes. If the plants show different infection types, as for instance described by van Dyk *et al.* (1988) for wheat cultivars in The Netherlands, and we can distinguish between highly resistant, moderately resistant, moderately susceptible and highly susceptible plants, then two groups can be selected, keeping the plants separate: highly resistant; moderately resistant and moderately susceptible. The most susceptible plants can be discarded.

The F_3 progenies are grown again in the disease nursery and the best progenies of A are used for further single plant selection, while the best lines of B are for intercrossing to start a recurrent selection program.

The F_4 progenies of A are grown again in the disease nursery and the best progenies are bulked for replicated testing. The B crosses also are grown in the disease nursery and single plant selections are made. This recurrent process as described above continues as long as it increases the resistance. This dual approach has two advantages. In case the resistance is simply inherited and durable, the A group will yield satisfactory results. This will become apparent in the F ≥3 generations. But in case the resistance is partial, polygenically controlled, either with or without additional race-specific major genes, the B group will show better results as it did for leaf rust and powdery mildew resistance breeding in barley (Parlevliet 1983; Parlevliet and van Ommeren 1988).

Finally, some remarks are made about pyramiding of resistance genes in a germplasm enhancement program. Responding to suggestions made by K.J. Frey, Iowa State University, Ames, USA, the chickpea group at ICRISAT started a germplasm enhancement program for resistance to *Helicoverpa* spp. and ascochyta blight. Figure 4 shows a provisional procedure we have proposed, but some changes still may be incorporated. The recurrent selection approach is based on similar principles, but for dry root rot, collar rot and nematode resistance there is a need to consider such a scheme only if the polygenic nature of the resistance is established.



F_1 = single cross, F_1' = double cross, F_1'' = 8-way cross
8-x = 8-way cross; handle as in main scheme: F_1'' etc.

* Scheme results in 7 main streams: 4 single crosses
  2 double crosses
  1 8-way cross

* At any time after the first screening, parents can be selected from the still-segregating populations for use in a conventional breeding program.
* At any time a new, similar scheme can be initiated as resources permit. The parents for a new scheme can be drawn from single crosses, double crosses, 8-way crosses or new germplasm.

Fig. 4. Enhancement of germplasm for resistance to stress factors.

Acknowledgement

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Breeding for Ascochyta Blight-resistant Kabuli Chickpea in Turkey

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Abstract

Ascochyta blight (*Ascochyta rabiei*) is the most serious disease affecting chickpea (*Cicer arietinum*) in Turkey. Developing resistant cultivars is the best way of reducing the damage from this disease. Research on ascochyta blight resistance began in 1976 at several agricultural research institutes in Turkey with selection from indigenous and introduced germplasm. A hybridization program was initiated in 1983 for the development of large-seeded, blight-resistant types. Selected lines are now in the yield trials.

Introduction

Diseases are the main problems in chickpea-growing countries. Ascochyta blight, caused by *Ascochyta rabiei* (Pass.) Labr., is the most serious disease affecting chickpea in Turkey, causing damage in all chickpea-growing areas. In some years, depending on climatic conditions, heavy yield losses occur. Turkey experienced its worst ascochyta blight epidemic in 1983 (Table 1).

Table 1. Area, production and yield of chickpea in Turkey from 1978 to 1987†.

Year	Area sown (ha)	Production (tonnes)	Yield (kg/ha)
1978	168,000	205,000	1220
1979	200,000	225,000	1125
1980	240,000	275,000	1146
1981	200,000	235,000	1175
1982	245,000	280,000	1143
1983	334,000	290,000	867
1984	345,000	335,000	971
1985	399,000	400,000	1003
1986	534,000	630,000	1180
1987	655,324	725,000	1106

† Data from Anonymous (1987).

Although the severity of blight disease can be reduced by crop rotation, late sowing, sowing of disease-free seeds and application of fungicides, the development of resistant cultivars is the most effective way of controlling this disease.

There were few studies on chickpea in Turkey until 1976, except some improvement studies conducted by the Faculty of Agriculture, Ankara University and some research institutes. In 1976, chickpea cooperative research activities were started by the National Food Legume Project for developing the large-seeded ascochyta blight-resistant cultivars. Activities are conducted mainly by the Aegean Agricultural Research Institute, Field Crops Central Research Institute, South-Eastern Anatolia Agricultural Research Institute and the Transitional Region Agricultural Research Institute. Some other research institutes and the Faculty of Agriculture, Ankara University also are conducting research on chickpea. All the abovementioned institutions have their own breeding and agronomy programs and they are cooperating with each other and with ICARDA in Syria and ICRISAT in India.

Studies on Ascochyta Blight in Turkey

Although it was listed as one of the important diseases of cultivated crops in Turkey in 1947 (Bremmer 1948), only a few studies had been done on ascochyta blight. Most of these studies were on the application of seed dressing and fungicides (Anonymous 1978; Karahan 1968; Senyurek *et al.* 1977).

The first information on the seed-borne nature of the pathogen was reported by Gobelez (1956). Later, Maden *et al.* (1975) found that 70% of the seed samples collected from six provinces in Central Anatolia were infected by *A. rabiei*, and in each sample the pathogen was present in varying amounts from 1 to 16%. According to a similar study conducted in the Aegean region during 1976/77, the percentage of seed samples infected by *A. rabiei* was between 48 and 65% and the average presence of the fungus in each seed sample was 1.6% (Yildiz and Delen 1978).

There have been almost no studies of the races of *A. rabiei*. Reactions of host cultivars to the isolates collected from the Aegean region suggested that seven different virulence patterns were present among the isolates used (Acikgoz 1983). Evaluation of lines in different regions revealed a susceptible reaction in some regions but a resistant reaction in another region, indicating the existence of races in *A. rabiei*. For instance, ILC 482 showed a resistant reaction in southeastern Anatolia, but in other regions was susceptible. Kinaci and Dalkiran (1985) also have pointed out the existence of different races in their study conducted in Ankara and Cankiri in 1983 and 1984.

Breeding for Ascochyta Blight Resistance

Mechanism of Resistance

Only one study has reported on the mechanism of the resistance (Onogur and Gocmen 1985). It pointed out that leaf hair exudates and total amount of phenoles did not affect the infection, but phytoalexins were considered to be one of the resistance factors.

Inheritance of Resistance

The first study dealing with the inheritance of resistance, conducted by Eser (1976), showed that a single dominant gene was responsible for resistance in line 72012. Later, Acikgoz (1983) reported a single recessive gene conferring resistance in lines 72012, ILC 195 and NEC 138-1, and a single dominant gene controlling resistance in lines ILC 201 and ILC 202.

Sources of Resistance

Eser (1976) was the first to report sources of resistance to blight in Turkey. A black-seeded chickpea line was found to be highly resistant in her study. In another study conducted by Acikgoz (1980), 36 out of 5000 lines were found resistant to varying degrees. Sources of resistance to ascochyta blight have been identified in different research institutes since 1980. Some resistant lines (Table 2) are being used in hybridization programs.

Table 2. Ascochyta blight-resistant lines of chickpea suggested for hybridization programs in Syria.

ILC 72	ILC 5928	FLIP 83-72C
ILC 182	NEC 138-1	FLIP 83-97C
ILC 191	FLIP 82-150C	FLIP 84-22C
ILC 193	FLIP 82-169C	FLIP 84-48C
ILC 194	FLIP 82-172C	FLIP 84-78C
ILC 195	FLIP 82-186C	FLIP 84-79C
ILC 196	FLIP 83-7C	FLIP 84-80C
ILC 200	FLIP 83-13C	FLIP 84-81C
ILC 201	FLIP 83-15C	FLIP 84-83C
ILC 202	FLIP 83-21C	FLIP 84-85C
ILC 2506	FLIP 83-22C	FLIP 84-86C
ILC 2956	FLIP 83-28C	FLIP 84-91C
ILC 3279	FLIP 83-31C	FLIP 84-92C
ILC 3856	FLIP 83-46C	FLIP 84-93C
ILC 3868	FLIP 83-48C	FLIP 84-182C
ILC 4421	FLIP 83-60C	FLIP 85-13C

Breeding Methods: Plant Introduction and Selection

Collections made by different institutions were shared with other institutions. Introductions also were made from the ICARDA/ICRISAT project in Syria. These materials were evaluated for resistance to ascochyta blight and resistant lines were selected for further use in the breeding program. Some well-adapted lines have been released as commercial cultivars. These include lines ILC 195/2 and ILC 482. Line ILC 195/2 is a medium-seeded cultivar with resistance to ascochyta blight in all regions. It can be grown in coastal areas in winter and harvested by combine. Furthermore, it has a good cooking quality. This line was licensed for seed production and is expected to be sown on approximately 500 ha in 1989. Line ILC 482, a high-yielding cultivar with acceptable seed characteristics, proved resistant to ascochyta blight in southeastern Anatolia. As shown in Table 3, 14 of the lines tested in variety registration yield trials were developed by selection.

Table 3. Chickpea lines tested in variety registration yield trials, 1989.

Line†	Institute	Method of breeding
FLIP 81-70W	Aegean Agric. Res. Inst.	Introduction, selection
FLIP 82-64C	Aegean Agric. Res. Inst.	Introduction, selection
FLIP 82-259C	Aegean Agric. Res. Inst.	Introduction, selection
FLIP 83-31C	Aegean Agric. Res. Inst.	Introduction, selection
FLIP 83-41C	Aegean Agric. Res. Inst.	Introduction, selection
FLIP 83-77C	Aegean Agric. Res. Inst.	Introduction, selection
FLIP 85-13C	Aegean Agric. Res. Inst.	Introduction, selection
FLIP 85-14C	Aegean Agric. Res. Inst.	Introduction, selection
FLIP 85-15C	Aegean Agric. Res. Inst.	Introduction, selection
FLIP 85-60C	Aegean Agric. Res. Inst.	Introduction, selection
87 AK 71 112	Field Crops Central Res. Inst.	Selection from segregating
87 AK 71 114	Field Crops Central Res. Inst.	material originating
87 AK 71 115	Field Crops Central Res. Inst.	from ICARDA
ILC 482	South-Eastern Anatolia Agric. Res. Inst.	Introduction, selection

† All lines are selections from the ICRISAT/ICARDA international nurseries.

Segregating materials also have been introduced from ICARDA. Two lines developed from segregating material by the Field Crops Central Research Institute are being tested in variety registration yield trials. Many of the selected lines are now in the preliminary and regional yield trials at the Aegean Agricultural Research Institute.

Hybridization Program

A hybridization program was initiated in 1983 for the development of large-seeded,

blight-resistant types with high yield at the Aegean Agricultural Research Institute, Field Crops Central Research Institutes and Transitional Region Agricultural Research Institute. The scheme of the hybridization program is given in Figure 1. Plants of F_1 generation are sown in the spring to escape blight damage. In general, segregating material is grown under artificial blight epidemics. Single-plant selection sometimes begins in the F_3 generation but mostly in F_4. Rows of F_6 progeny are grown under the maximum disease pressure both in the field and the greenhouse, along with susceptible and resistant check lines. In this generation, progenies with uniform high yield and disease resistance are bulked for replicated yield tests.

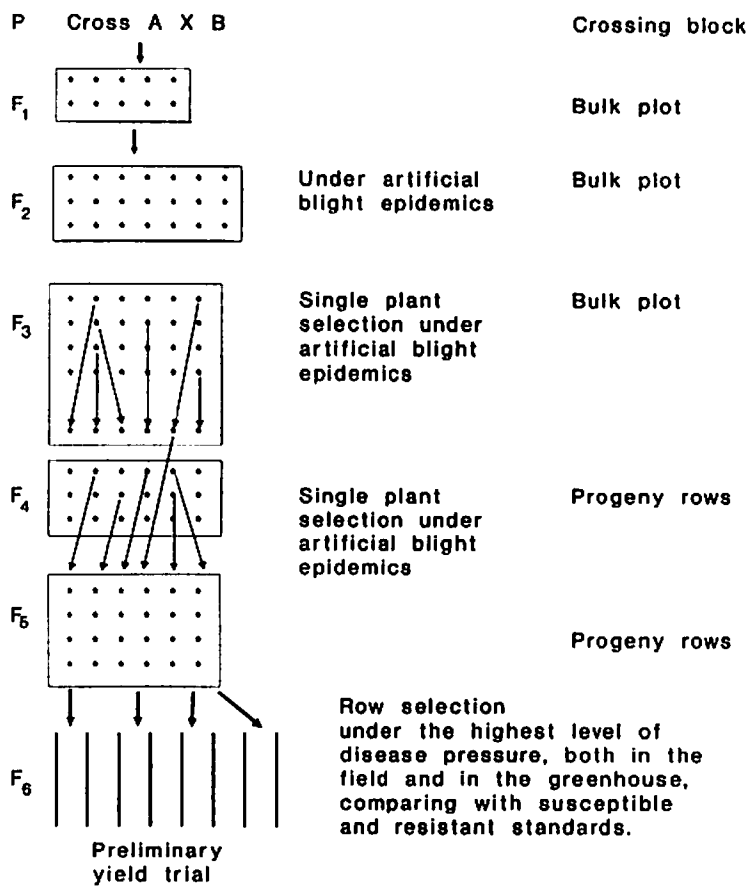

Fig. 1. Hybridization program for chickpea in Turkey.

Promising lines developed by different institutes will be tested together as a uniform nursery in different agroecological conditions throughout Turkey.

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Breeding for Ascochyta Blight-resistant Desi Chickpea in Pakistan

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Abstract

Chickpea (*Cicer arietinum*) is the premier pulse crop in Pakistan where it is grown on about 1 million hectares. Ascochyta blight, caused by *Ascochyta rabiei*, devastated this crop in three successive years (1980-82) in Zones I and III, which comprise 89% of the total area under chickpea. In these two zones, predominantly the desi type of chickpea is grown. Concentrated efforts began in the 1980s to stabilize yield through the development of blight-resistant cultivars. Three methods of breeding, introduction and selection, hybridization and mutation, are employed. Epidemiology, race vs aggressiveness and the genetics of resistance vs vertical and horizontal resistance are discussed.

Introduction

Agriculture is the cornerstone of Pakistan's national economy. According to the 1984 Agricultural Statistics of Pakistan, out of 79.61 million hectares of total geographical area, 20.34 million hectares or only 25% are currently under cultivation. Chickpea occupies just over 1 million hectares (Table 1).

Table 1. Area, production and yield of chickpea in Pakistan†.

Year	Area (1000 ha)	Production (1000 t)	Yield (kg/ha)
1976-77	1095	649	593
1979-80	1129	313	278
1980-81	843	337	400
1981-82	902	294	326
1988-89‡	1060	583	539

† Sources: Yearbook of Agriculture Statistics (1971-72); Agriculture Statistics of Pakistan (1982).
‡ Estimates only.

[1]Resident ICRISAT scientist.

In Pakistan, chickpea is the primary pulse crop. Table 1 compares the area, production and yield in 1976/77 with those of some other years including the projected figures for 1988/89. It can be seen that area, production and yields have actually declined over the past 12 years, an unacceptable situation for the premier pulse. There are various reasons for such decline, but the most important one is the damage from ascochyta blight caused by *Ascochyta rabiei* (Pass.) Labr.

Following careful and thoughtful analysis of climatic and edaphic factors and production problems of the major chickpea-growing areas, Pakistan has recently defined three zones with respect to chickpea production, namely Zone 1: Thal desert or region; Zone 2: Sind and Baluchistan rice-based system; Zone 3: northern and eastern regions outside the Thal, in the Punjab and the North West Frontier Province. The major characteristics of the zones are presented in Table 2. Details of the zones can be found in the 1988 Annual Report on the Project (Rahman 1989).

Table 2. Major characteristics of zones and constraints to chickpea production in Pakistan.

Zone	Constraints		Planting method	Main chickpea type	Soil type	Ambient temp. (°C)†	Annual rainfall (mm)	Average productivity (kg/ha)
	Major	Minor						
I	Blight, drought, weeds	Wilt + root rot, virus	Drilling in 30-cm-wide rows	Desi	Sandy-loamy sand	15.7	200-250	500
II	Pod borer, lack of suitable genotype for late sowing, weeds	Wilt + root rot, virus	Broadcast	Kabuli	Silty clay-clay	18.6	100-125‡	800
III	Blight, pod borer, weeds	Wilt + root rot, virus	Drilling in 30-cm-wide rows	Desi	Sandy-loamy loam	14.3	>500	550

† Average of mean monthly temperature during the growing season (Nov-Mar).
‡ Area receives supplemental irrigation.

Out of some 50 pathogens reported so far that affect chickpea around the world, only a few have the capacity to cause distress and ascochyta blight appears to be the most important one (Nene and Reddy 1987). This disease brought severe epidemics in Zones I and III, which comprise 89% of the total area under chickpea in Pakistan. About

75 to 80% of the total production in these two zones is from the desi type. This prompted the Government of Pakistan (GOP) to strengthen chickpea research through implementation of a Cooperative Research Program across the provinces in the 1980s. To accelerate the research efforts for overcoming the chickpea blight menace, GOP also sought technical assistance from the Asian Development Bank and technical assistance from ICRISAT. Thus, the project came into operation in November 1984 when the junior author joined as Resident Scientist. The major objective of this project was to help in stabilizing the yield through the development of disease-resistant cultivars.

Epidemics of a serious nature in three successive years, 1980 to 1982, strongly suggest that there must be an efficient mechanism by which the fungus survives and spreads so quickly during the cropping season. It is now known that diseased chickpea debris and infected seeds are major sources of inoculum and that the disease spreads rapidly when ambient temperatures are around 9 to 24°C and wet conditions prevail for 10 hours or more (Weltzien and Kaack 1984).

Over the years, a number of ways have been proposed to effectively reduce or control this disease. They include crop rotation, field sanitation, seed dressing, fungicide spray and use of blight-free seeds and resistant cultivars. In the Workshop on Ascochyta Blight and Winter Sowing of Chickpeas at ICARDA in 1981, Kaiser (1984) emphasized the use of blight-free seeds. Use of resistant cultivars has been emphasized by many as the best possible means of effectively controlling this disease (Hawtin and Singh 1984; Nene and Reddy 1987).

Screening of Germplasm

We evaluated about 8000 germplasm accessions obtained mainly from ICRISAT and ICARDA. They were grown in single 4-m rows at the National Agricultural Research Centre in Islamabad (33° 42'N) and screened against blight following the method developed by Singh *et al.* (1981) with some modifications such as the use of isolates obtained from all areas of Pakistan that suffer from blight during epidemic years. Thus our inoculum contained a mixture of both aggressive and less aggressive isolates. We have identified materials that maintained their resistance in Pakistan even when tested in different blight-prone areas against very aggressive isolates from Attack. Some of these lines even showed good tolerance when tested against six ICARDA races in a growth chamber and the plastic house. Following this procedure, we were able to select 20 lines that showed ratings of 3 to 5 on the 1 to 9 scale of Reddy *et al.* (1984). In our studies, we designated those lines having ratings of 1 to 3 as resistant, 4 to 5 as moderately resistant and 6 to 9 as susceptible. The lines that we selected had mainly moderate levels of resistance to blight.

Epidemiology

Buddenhagen and Kaiser, in their 1988 Consultancy Report, hypothesized that the fungus

was now completely absent in the Thal and that the non-Thal Northern and Eastern areas (Zone III) are the only places where the fungus could survive in the off-season. Fungi persist in the seed and straw stacks and produce ascospores that are dispersed by winds. They believed that the development of the teleomorph (sexual, perfect, or ascospore) stage of the fungus was a precondition for spores to reach the new leaves from the straw stacks. One of the major reasons for our failure to properly understand the epidemiology is that we still do not know whether the teleomorph is the perfect stage of the fungus. Obviously, a better understanding of the epidemiology of blight disease is necessary in view of the fact that the reappearance of the devastating epidemics after a lapse of some years is not explained by the current knowledge.

Race vs Aggressiveness

There is great controversy on whether many races of *A. rabiei* exist or if there is only a single race with the ability to be aggressive under proper environmental conditions. A race is a group of plants or animals having common characteristics that distinguish them from other members of the same species by physical characteristics (The New Collins Concise Dictionary of the English Language, 1982). Aggressiveness may be defined as vigorous attack or highly harmful action by organisms against plants or by humans against their own kind.

Species of pathogenic fungi usually are made up of many physiological races with varying pathogenicity, but by the preceding definition, they cannot be morphologically identical, contrary to the belief of Allard (1960). Physiological races are highly stable but a race may produce numerous other races by sexual reproduction and recombination of genes governing virulence or avirulence (Allard 1960). Buddenhagen and Kaiser (unpublished), by contrast, believe in a highly variable nature of the pathogen in aggressiveness and in adaptation potential to different host genotypes. Van der Plank (1984) considered the main effect of isolates as a significant difference in aggressiveness. The great variation in the effect of isolates of *A. rabiei* from India, Iran, Pakistan and Turkey on growth rate, sporulation and colony appearance observed by Kaiser (1973) may then be described as variation in aggressiveness.

The concept of physiological races is based on differences in the types of infection produced on "differentials". The infection type is the visible expression of the interaction of the host with the pathogen in a particular set of environmental conditions (Allard 1960). Using this concept and the reaction of a differential set of 18 chickpea lines, Reddy and Singh (1985) classified the isolates into four distinct races and four biotypes. Buddenhagen and Kaiser (unpublished) believe that the concept of aggressiveness is not properly understood, that it is usually confused with virulence/avirulence and that races are based on virulence/avirulence reactions. It appears that the various discrepancies about race and aggressiveness lie with the various concepts of different scientists. Much more definitive work is needed to resolve the issue.

Genetics of Resistance to Blight vs Vertical/Horizontal Resistance

There is considerable debate on questions of whether resistance in the host plant is controlled by a single or many genes, whether the resistance is vertical or horizontal and what breeding strategy should be followed. Basically, two approaches have been proposed: one is to conduct a breeding program in which different vertical resistance (VR) or major genes from different parents are identified and pyramided into a single cultivar or cultivars. This approach is based on the assumption that resistance is controlled by a single dominant gene (see Singh *et al.* 1984). Jensen (1952), Borlaug (1959), Nelson (1978), Byth *et al.* (1980) and Singh *et al.* (1984) suggest the development of multiline or composite varieties that would be composed of compatible lines having similar heights, maturities, etc., but carrying different major genes for resistance. Such lines, if developed, could provide a long-term solution to the problem. However, very few different major genes have been identified to date and therefore development of such lines is still in its infancy.

Another approach is to develop cultivars with horizontal resistance (HR) that emphasizes minor genes in a massive crossing program using recurrent selection. Buddenhagen (1983), Van der Plank (1984), Simmonds (1988) and Robinson (1976) believe that HR is the long-term solution to the blight problem. Buddenhagen and Kaiser (unpublished) argued for HR, as Buddenhagen (1983) did elsewhere, in order to reduce the risk of quick collapse of new cultivars with VR. Van der Plank (1968) developed the concept of broad-based HR, which involved crossing cultivars with moderate levels of resistance and wide adaptation and bulking the F_2s equally to form a composite population. Simmonds (1988) mentioned four types of resistance: (i) major gene (VR), (ii) polygene (HR) (iii) pathogen-nonspecific major gene resistance (NR), and (iv) interaction or mixture resistance (IR). The use of all these may have merit for breeding resistant chickpea materials.

Robinson's (1976) view of HR theory is to utilize all fully susceptible genotypes in a mass recurrent polycrossing system. The theory behind the development of HR requires massive recombination and therefore massive crossing and recurrent selection. This procedure is highly labor-intensive and thus is not likely to be adopted by time-limited programs. Meanwhile, parents having location-specific resistance may be used in a crossing program to provide diversity to the materials generated, as suggested by Singh *et al.* (1984). Some in-depth discussions are in order to settle these issues of crucial importance.

Screening in Pakistan

The following is a brief discussion of the mechanics of the project we are involved with. One of the objectives of screening germplasm accessions and fixed lines introduced from ICRISAT, ICARDA and other places is to determine if any of them can be released as cultivars following rigorous testing in the adaptation trial. Over the years we selected 10

lines, which are currently being tested in Zones I and III. These lines are: ICC 11514, NEC 138-2, E 101, HG 202-6-1, ILC 72, ILC 202, ILC 3279, ICC 820039, ICC 820049 and ICC 820317.

To develop cultivars with moderate resistance to blight, we use all three methods that are available following hybridization in a self-pollinated crop like chickpea, namely bulk method, pedigree method and backcross method.

Bulk Method

In the coordinated research program on chickpea at the National Agricultural Research Center (NARC), the bulk method was used from 1980 until 1985. Because of many constraints, we made only a few crosses. From F_2 through F_4 some promising plants were selected and bulk harvested. The selected plants in F_5 were put in the preliminary yield trial, followed by an adaptation trial and finally the national uniform yield trial.

Following this method we were able to select four lines with moderate resistance to blight: Pk 51825 X CM 72, Pk 51832 X CM 72, Pk 51835 X CM 72 and Pk 51863 X NEC 138-2.

Pedigree Method

Most disease-resistant cultivars have been developed by this method (Allard 1960). It has been used extensively at ICARDA (Singh *et al.* 1984) and at NARC since 1985. The NARC scheme is shown in Figure 1.

Crosses usually are made at NARC during March; limited crosses are made at the high-altitude Research Station at Kaghan (34° 43'N, 2103 m elevation) during late August to early September. One of the parents used is usually an adapted cultivar and the other has a high or moderate level of resistance to blight. In a few crosses, one of the parents used is early maturing, since development of early maturing cultivars has recently been suggested to escape blight (see below). The F_1s are grown at Kaghan. Seeds of each F_1 plants are collected individually, hand threshed and thoroughly examined to rogue out the selfed ones. Then the F_2 seeds are bulked within crosses. The F_2 populations are grown in the pathology block to screen against blight. Those plants having ratings of 1 to 5 on the 1 to 9 scale are selected and harvested individually. The F_3 seeds are again sown in the same block as progeny rows for reconfirmation against blight. Those plants having ratings of 1 to 5 are selected and harvested individually. The F_4 plants are grown in the breeding block at the NARC to screen for desirable agronomic characteristics. Following selections, the plants are harvested and bulked within progeny rows. After threshing, seeds are cleaned and thoroughly examined in the store/laboratory to screen against mixture or segregation. If the amount of F_5 seed is considered sufficient, it is included in a preliminary yield trial (PYT). Otherwise, seed is increased at Kaghan and F_6 seeds are used to form the PYT. The Adaptation Trial (ADT) includes those selections (F_6 or F_7) that show promise in the PYT. The ADT is usually planted at a number of locations in Zones I and III. Based on the performance in the ADT, the selected ones, if any, are included in the Chickpea National Uniform Yield Trial at 15 to 20 locations throughout the country.

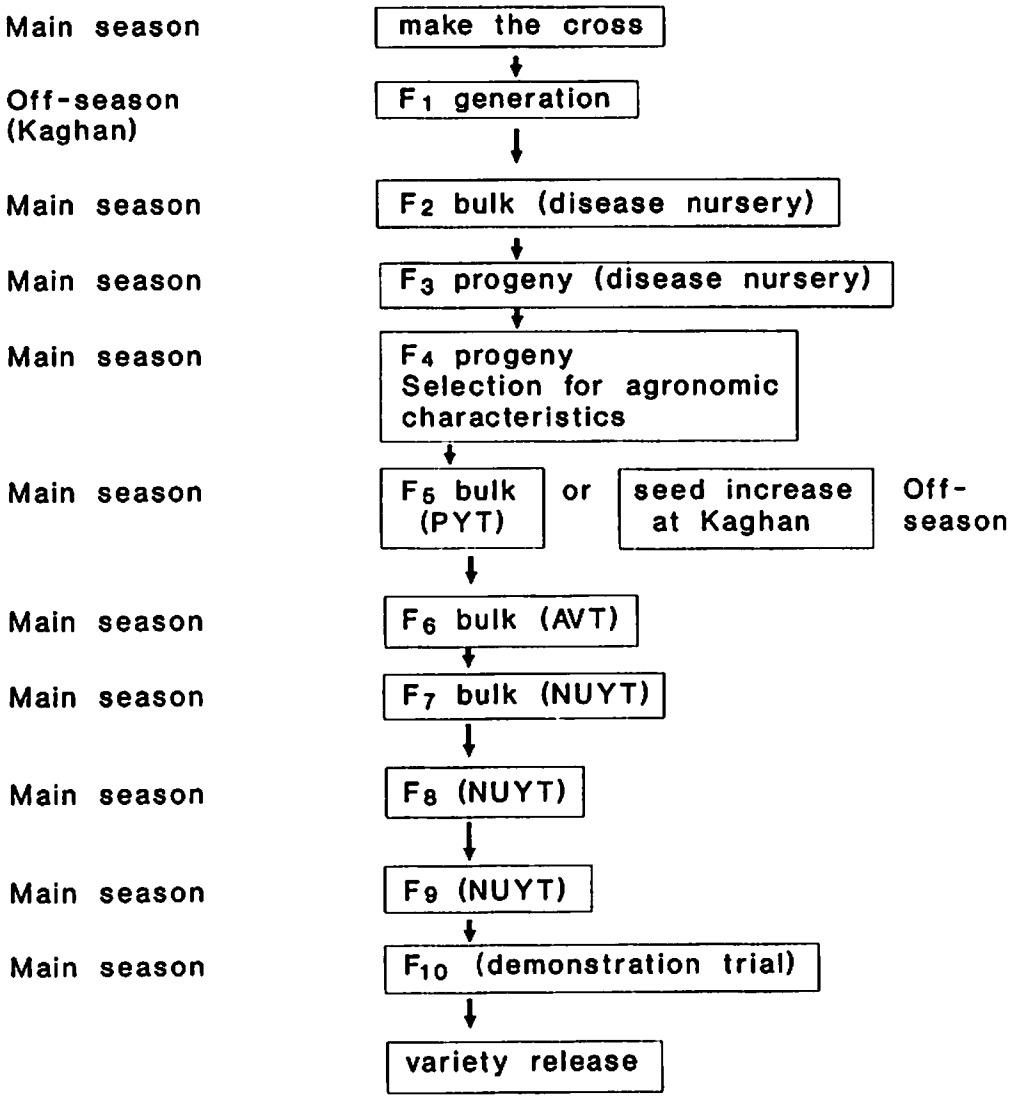

Fig. 1. Development of blight-resistant cultivars at NARC, Islamabad, Pakistan.

Following the pedigree method and this scheme, we have been able to develop a number of advanced (F_4 to F_6) lines that proved moderately resistant to induced blight over 2 years. We have included a few of these lines (F_5 to F_6) in this year in PYT at NARC and adaptation trials at four places (Rahman 1988).

Backcross Method

We have used this method only occasionally to improve blight resistance in the two widely used cultivars CM 72 and C 44.

Mutation Breeding

Mutation breeding is a valuable tool for improving plant type and yield. It is particularly useful for changing qualitative characters such as plant height, plant type, etc., in many crops including chickpea (Micke 1988) and also for improving landraces that lack particular characters.

In Pakistan, the most widely used cultivar (C 44) initially had some resistance to blight, but since 1987 resistance appears to have broken down. This cultivar also suffers from iron-deficiency chlorosis. It has moderate yield potential but its plant type and seed characteristics are superior. We have used it extensively in our crossing program over several years.

Because of its sensitivity to iron chlorosis and blight we irradiated 3 kg seeds with 15 kr gamma rays from a ^{60}Co source to select mutants that are resistant to chlorosis and blight.

Acknowledgement

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Recent Advances in Chickpea Breeding for Ascochyta Blight Resistance in India

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Abstract

India has the distinction of being the first both in acreage and production of chickpea (*Cicer arietinum*) in the world. However, the productivity of this legume is low. Among several factors limiting the production of chickpea, diseases rank first. Although many diseases infest chickpea, fusarium wilt (*Fusarium oxysporum*) f.sp. *ciceri*, ascochyta blight (*Ascochyta rabiei*), botrytis gray mould (*Botrytis cinerea*) and dry root rot (*Rhizoctonia bataticola*) are the most destructive. The occurrence of ascochyta blight is limited to the region falling between 28 and 36°N latitude, comprising Jammu and Kashmir, Himachal Pradesh, Punjab, the northwestern parts of Rajasthan and Haryana, Delhi and western Uttar Pradesh. Since this is the most productive zone for chickpea, the loss due to ascochyta blight is much more than in other areas. Crop residue and seeds are the source of inoculum and the cool and wet seasons are conducive for the spread of the disease.

Epidemics of ascochyta blight from 1981 to 1983 compelled breeders and pathologists to take up the ambitious resistance breeding program against this disease. Laboratory and field screening techniques have been developed. Under the All India Coordinated Pulses Improvement Programme, a special trial on ascochyta blight-resistant lines is conducted. As a result of this program, a large number of genotypes having resistance/tolerance to this disease have been identified. Since 1984 seven cultivars of desi chickpea (GNG 146, Gaurav, BG 244, BG 256, BG 261, BGM 408 and BGM 413) having moderate to high levels of resistance have been released for general cultivation. So far, no cultivar of kabuli chickpea resistant to ascochyta blight has been released. The intensive breeding program has now begun to develop kabuli chickpea cultivars.

Introduction

Chickpea (*Cicer arietinum* L.) is the most important pulse crop grown in India. The area and production of this pulse in 1987 were, respectively, 7.78 million ha and 4.45 million tonnes (Table 1). Chickpea contributes 29.3 and 37.9% to the total acreage and production of pulses, respectively. India has the distinction of ranking first both in acreage and production of chickpea in the world. Both desi and kabuli types are grown, the former having the maximum acreage.

Reasons for Low Production

Although India is the global leader in chickpea production, productivity is low (657 kg/ha). The area cropped to chickpea is declining rapidly, although production is not decreasing proportionately owing to increases in the productivity (Table 1). The following

Table 1. Area, production and productivity of chickpea in India, 1966-1987.

Year	Area (million ha)	Production (million tonnes)	Yield (kg/ha)
1966-67	8.00	3.62	453
1967-68	8.26	5.97	723
1968-69	7.11	4.31	607
1969-70	7.75	5.55	715
1970-71	7.84	5.20	663
1971-72	7.91	5.08	642
1972-73	6.97	4.54	651
1973-74	7.76	4.10	528
1974-75	7.04	4.02	570
1975-76	8.32	5.88	707
1976-77	7.97	5.42	680
1977-78	7.97	5.41	678
1978-79	7.71	5.74	745
1979-80	6.99	3.36	481
1980-81	6.58	4.33	657
1981-82	7.87	4.64	590
1982-83	7.40	5.29	715
1983-84	7.16	4.75	663
1984-85	6.91	4.56	661
1985-86	7.66	6.68	743
1986-87	7.78	4.45	657

are the major reasons for a reduction in the chickpea area:

(i) In the irrigated belt of the country, it does not compete with cereals such as wheat because the latter responds to input application better than chickpea. Therefore, as soon as irrigation facilities expand, the chickpea area is diverted to wheat.

(ii) Part of the rain-fed chickpea area has gone to rapeseed and mustard because of better economic returns.

(iii) In the northern belt, owing to the lack of suitable chickpea cultivars for late planting, rice is followed by wheat.

(iv) There is no attractive incentive to the farmers in terms of procurement of the produce and better market price.

(v) Chickpea is a more risky and unstable crop than wheat and other cereals for two reasons: its cultivation in marginal lands and rain-fed situations imposes several stresses, the scarcity of the moisture being the major one, and the insect pests and diseases cause tremendous loss to the crop.

Pathogens on Chickpea

More than 50 pathogens that attack chickpea have been reported from different parts of the world (Nene 1980). In India serious diseases are ascochyta blight, fusarium wilt, botrytis gray mould and dry root rot. Fusarium wilt is widespread, whereas ascochyta blight is limited to the northern belt only. However, the loss due to ascochyta blight is much more than from other diseases. In this paper the status of this disease in India and efforts to breed resistant cultivars are discussed.

Distribution of Ascochyta Blight

The first report of the prevalence of this disease came from the North West Frontier Province of British India (now part of Pakistan) in 1911 (Butler 1911). Since then, repeated epidemics of this disease have occurred in northwestern parts of the country. Until 1981, the disease was confined to submontane regions of Punjab, but from 1981 to 1983 it assumed alarming incidences in Punjab, Haryana, Himachal Pradesh, the northern hills of Uttar Pradesh, Jammu, Kashmir and Western Rajasthan. The epidemics and the area of incidence are increasing owing to changes in the microclimate induced by high inputs such as irrigation and fertilizer.

In India, the natural occurrence of ascochyta blight is in the region between 28 and 36°N latitude. This encompasses only 10% of the chickpea area in the country, but productivity of the region is the highest because of its favorable climate (Amin 1988).

Causal Organism

Ascochyta blight is caused by *Ascochyta rabiei* (Pass.) Labr. The asexual reproduction is through the formation of pycnidia, which produce pycnidiospores. Pycnidia are visible as minute dots arranged in concentric rings on aboveground plant parts. The pycnidiospores are oval to oblong, straight or slightly bent at one or both ends, hyaline, occasionally bicelled, but usually one-celled (Nene and Reddy 1987). The fruiting bodies (perithecia), found on crop residue, are dark brown or black and globose with a hardly perceptible beak and ostiole. The asci are cylindrical clavate, more or less curved, pedicellate and the ascospores are monostichous, rarely distichous ovoid, divided into two unequal cells. The sexual stage of the fungus is known as *Mycosphaerella rabiei* (Pass.) Kovach. However, this stage has not been reported from India.

Physiological Races

The knowledge of occurrence of physiological races is essential for successful screening and breeding material. Bedi and Aujla (1969) reported 11 races of the fungus from Punjab after studying a large number of isolates in terms of symptomology, manner of pycnidial formation on the host and the pathogenetic behavior. Grewal (1984) reported two races and one biotype of race 2 using six chickpea lines. Reddy *et al.* (1983) reported that some chickpea lines developed in Syria were found susceptible in Pakistan, which indicated the existence of different virulent races of *A. rabiei* in Pakistan and Syria. Further, Reddy and Kabbabeh (1985) differentiated 50 isolates into six races of this fungus in Syria and Lebanon. In India, a large number of isolates collected from Punjab, Haryana, Himachal Pradesh, Rajasthan, Uttar Pradesh, Jammu and Kashmir, and Delhi were classified in 23 groups on the basis of cultural characters and reactions on 12 differentials; 12 races were reported (Singh 1985).

Host Range

Many workers reported chickpea as the only host of *A. rabiei* (Nene 1980). However, Kaiser (1973) could artificially infect cowpea and bean in Iran. He had observed small reddish brown spots on the stems, petioles and leaves of cowpea and beans, but these lesions did not increase in size. Tripathi *et al.* (1987) inoculated as many as 44 crops and weed species from the 'tarai' area of Nainital district in India under glasshouse conditions, but could not infect any crop or weed species and thus concluded that the Pantnagar culture of *A. rabiei* was specific only to chickpea. On the other hand, fungus infected peas, beans and cowpea in the greenhouse experiments at ICARDA, Syria and it has been isolated successfully from these hosts (ICARDA 1982).

Epidemiology

The severe epidemics of the disease in the northwestern parts of the country suggest a strong mechanism of survival and perennation of the fungus. However, the crop residue and seed are considered to be the source of inoculum. Temperatures between 20 and 25°C, rainfall of more than 150 mm during the growing season of chickpea and wet and windy days are conducive for disease infection, development and spread of ascochyta blight.

In India, Luthra and Bedi (1932) first demonstrated the seed-borne nature of the disease and showed that the seed coat and cotyledons of the diseased seeds contained the mycelium. Later, the seed-borne nature of the disease was reported from other countries by several workers (Gobelez 1956; Halfon-Meiri 1970; Khachatryan 1961; Zachos 1952).

Luthra *et al.* (1935) also considered infected crop residue to be a source of primary infection for the succeeding season and they reported that the fungus could survive in crop debris for 2 years. They also reported that the fungus lost viability when it was buried 4 cm deep. Survival of the fungus in debris from season to season also has been reported by Khachatryan (1962), Kaiser (1973) and ICARDA (1982, 1983). However, the

soil depth at which the debris is buried, temperature and relative humidity are the guiding factors for perpetuation of the fungus.

Screening Procedure

Various techniques are employed for identifying the lines of chickpea resistant to *A. rabiei*. However, the incorporation of chopped, infected debris of chickpea in the crop has been found to be an efficient technique for multiplication of the disease and consequent screening of genotypes for resistance (Labrousse 1931; Luthra *et al.* 1938; Sattar and Hafiz, 1951; Vedysheva 1966). G. Singh (pers. comm., Punjab Agricultural University, Ludhiana, India) developed a technique for large-scale screening of the material in the laboratory. Under this technique, two to three seedlings per pot are raised in an open space. At the 5 to 10-cm stage, seedlings are sprayed with the aqueous suspension of spores and transferred to polyethylene-covered cages provided with artificial light. In these cages the humidity is automatically increased because of the polyethylene cover.

The technique for large-scale field screening of germplasm and the segregating material also have been developed and are being used at the Punjab Agricultural University, Ludhiana, Haryana Agricultural University, Hissar and other centers located in the disease-prone areas. With this technique, an infector row (a genotype susceptible to the disease) is planted after every two to four rows of test material. Seedlings of the test material are sprayed with the inoculum of the fungus. The humidity is increased with a sprinkler system of irrigation. This technique has proven to be efficient in creating a high intensity of infection.

Source of Resistance

The first known source of resistance to this disease from India was 4F 32, later renamed F 8 (Luthra *et al.* 1938). This line was used in hybridization as a source of resistance and the first cultivar (C 1234) was developed by incorporating resistance in the local cultivar Pb 7. Later, another resistant variety (C 235) was developed from Punjab. This variety is still popular among farmers, although its resistance has decreased.

To regularly provide the breeding program with resistant lines as donors for disease resistance, an ambitious screening program of germplasm and the segregating material was launched both at ICRISAT and under the national program of the All India Coordinated Pulses Improvement Project (Table 2). Leading centers working in India are Ludhiana, Hissar, Sriganganagar, Delhi and Pantnagar, which have favorable climatic conditions for the development of disease and infrastructures available for screening.

As well as germplasm and segregating material, the screening of entries included in various trials—Initial Evaluation Trial (IET), Coordinated Varietal Trial (CVT) and Kabuli Coordinated Trial Varietal Trial (KCVT)—was started in 1984/85 under the All India Coordinated Pulses Improvement Project. A separate trial named Ascochyta Blight Coordinated Varietal Trial (ABCVT) also was started to evaluate the yield potential of resistant lines at the field stage. As a result of this effort 12 entries (BG 257, BG 261, BG 276, BG 300, H 75-35, ICC 41, WB 17-3, H 83-11, H 83-16, H 81-73, GG 575 and GL 83007) were found resistant to this disease (Table 3). These lines are being used as sources of resistance in breeding program.

Table 2. Sources of resistance to ascochyta blight in chickpea from various locations in India.

Location	No. lines screened	No. lines resistant	Source
ICRISAT,	3500	18	Haware *et al.* (1980)
Patancheru	91	8	ICRISAT (1984)
Pantnagar	1000	5	Shukla *et al.* (1984)
Ludhiana	406	8	Singh *et al.* (1984)
	1000	12	Singh *et al.* (1987a)
	NA†	28	Singh *et al.* (1987a)
Palampur	125	3	Katyar and Sood (1985)
Hissar	625	11	Jalali and Khirbat (1984)
	766	27	Jalali and Khirbat (1985)
	1674	58	Jalali *et al.* (1986)
	638	27	Jalali and Harichand (1987)
	1938	6	Jalali and Harichand (1988)

† NA = not available.

Table 3. Sources of resistance to ascochyta blight in chickpea through multilocation testing under All India Coordinated Pulses Improvement Project, India.

Year of testing	No. testing locations	No. entries tested	Trial†	Resistant entries
1984-85	3	47	CVT	BG 261, H 75-35
	3	52	IET	BG 257, BG 276, BG 300
1985-86	3	63	CVT	ICC 41, WB 17-3, BG 300
	3	19	ABCVT	BG 261, BG 300, H 83-11, H 83-16 GG 575
	3	13	KCVT	H 81-73, GL 83007
1986-87	5	34	CVT	-
	5	54	IET	-
1987-88	6	57	CVT	BG 261, GG 575
	6	60	IET	-
	6	25	KCVT	-
	6	17	ABCVT	BG 261, BG 300, H 83-11, H 83-16, GG 575

† CVT, Coordinated Varietal Trial; IET, Initial Evaluation Trial; KCVT, Kabuli Coordinated Varietal Trial; ABCVT, Ascochyta Blight Coordinated Varietal Trial.

Inheritance of Resistance to Ascochyta Blight

Inheritance of resistance to ascochyta blight has been reported by several workers (Table 4). A single dominant gene responsible for resistance has been reported by Hafiz and Ashraf (1953), Vir *et al.* (1975) and Eser (1976). Singh and Reddy (1983) reported a single dominant gene conditioning resistance in ILC 72, ILC 183, ILC 200 and ICC 4935, whereas the resistance in ILC 191 was conferred by a single recessive gene. Verma *et al.* (1987) found resistance to be monogenic recessive in H 75-35, G 543, GNG 146 and BG 257; digenic recessive in NEC 138-2; and monogenic dominant in E 100Y (M). Tewari and Pandey (1985) reported the resistance to be governed by a single dominant gene in EC 26448, PG 82-1 and P 1252-1 and by a single recessive gene in BRG 8 against *A. rabiei* culture of Pantnagar, India.

Table 4. Inheritance of resistance to ascochyta blight in chickpea.

Mode of inheritance	Varieties/cultures used	References
Monogenic dominant	F 8, F 10	Hafiz and Ashraf (1953)
	IP 13	Vir *et al.* (1975)
	72-92	Eser (1976)
	ILC 72,ILC 183,ILC 200, ILC 5935	Singh and Reddy (1983)
	EC 26448 (kabuli), P 1252-1,P 482-1 (kabuli)	Tewari and Pandey (1985)
	E 100 Y (M)	Verma *et al.* (1987)
Monogenic recessive	ILC 191	Singh and Reddy (1983)
	H 75-35, G 543, GNG 146, BG 257	Verma *et al.* (1987)
	BRG 8	Tewari and Pandey (1985)
Digenic recessive	NEC 138-2	Verma *et al.* (1987)

Breeding for Ascochyta Blight Resistance

Breeding for ascochyta blight resistance in chickpea was initiated as early as 1941. Lines F 8, F 9 and F 10 resistant to this disease were developed (Khan 1980). Line F 8 was high yielding and did well in a blight-endemic area and therefore was released for cultivation in Punjab. However, it was not successful as it proved highly susceptible to wilt. Therefore, it was used as a resistance source in the breeding program for ascochyta

blight resistance. Cultivar C 1234 was developed from a cross (Pb 7 x F 8) and released for general cultivation in 1944 (Ahmed *et al.* 1949). This cultivar also was used as a donor parent for resistance and another resistant cultivar C 235 was evolved from a cross (IP 58 x C 1234) and released for cultivation in 1960 (Bedi and Athwal 1962).

Cultivar C 235 maintained resistance for a long period and has been a very popular cultivar in northern India. It has now become susceptible to possibly new races of the pathogen. During the period 1981-83 incidence of the disease was heavy, which compelled us to intensify our breeding efforts. As a result, a large number of genotypes having resistance to this disease were developed. Since 1984 as many as seven cultivars having resistance/tolerance have been released for general cultivation in the country (Table 5). Another variety (GNG 146) developed through selection has been released for Rajasthan in 1984. This variety is suitable for the irrigated and blight-endemic areas of Rajasthan, southwestern Haryana and western Punjab. Gaurav (H 75-35) evolved from a cross (C 235 x E 100 Y) at the Haryana Agricultural University, Hissar and was released in 1984 for the irrigated northern belt of the country. It has broad foliage and seeds. Three varieties—BG 261 from a simple cross (P 827 x P 9847), BG 244 from a three-way cross (850-3/27 x P 922) P 9847 and BG 256 from a double cross (JG 62 x 850-3/27) x (L 550 x H 208)—were developed from the Indian Agricultural Research Institute (IARI), Delhi and released for general cultivation in 1985. Variety BG 261 is tall with an upright growth habit. Also from IARI are two varieties (BGM 408 and BGM 413) developed through mutation breeding; they possess resistance to ascochyta blight.

So far no cultivar of the kabuli type having resistance to ascochyta blight has been released for cultivation. However, HK 81-69, a kabuli genotype developed at the Haryana Agricultural University, Hissar has given promising performance in terms of resistance to this disease and yield potential (Singh *et al.* 1987b).

The segregating material and elite lines of desi and kabuli chickpea derived from crosses between genotypes resistant to ascochyta blight and long-duration types at ICRISAT are being tested extensively in the National Program (ICRISAT 1984, 1985, 1986).

In order to diversify the source of resistance to this disease, an extensive crossing program has been launched and facilities for screening have been developed. This will help in the development of resistant cultivars of both desi and kabuli types for different agroclimatic situations in the country.

Table 5. Ascochyta blight-resistant varieties of chickpea released in India.

Cultivar	Pedigree	Release year	Salient features
C 1234	Pb 7 x F 8	1944	-
C 235	IP 58 x C 1234	1960	Medium tall, bushy; seeds small, smooth and yellowish brown, suitable for humid areas of northern India.
G 543	C 235 x C 168	1977	Semi-spreading; seeds small, yellowish brown, wrinkled; suitable for Punjab.
GNG 146	Selection	1984	Tall; foliage broad; seeds medium bold, grayish yellow; suitable for irrigated parts of Rajasthan and adjoining states.
Gaurav	C 235 x E 100 Y	1984	Tall, erect; foliage broad; seeds bold, brown, round and smooth; suitable for northern irrigated belt of India.
BG 244	(850-3/27 x P 922) x P 9847	1985	Medium tall, semi-spreading; foliage dark green; seeds medium bold, light brown; suitable for Madhya Pradesh, Rajasthan and Gujarat.
BG 256	(JG 62 x 850-3/27) x (L 550 x H 208)	1985	Tall, semi-spreading; foliage broad; seeds bold, light brown; suitable for Uttar Pradesh, Bihar and WestBengal.
BG 261	P 827 x P 9847	1985	Tall, upright, compact; seeds medium bold, golden yellow; suitable for Haryana, Rajasthan and Uttar Pradesh.
BGM 408	Mutant of G 130	1985	Medium tall, semi-erect; seeds medium bold, yellowish brown; suitable for Punjab, Haryana, Rajasthan, Uttar Pradesh and Delhi.
BGM 413	Mutant of G 130	1985	Tall, semi-erect; seeds medium bold, smooth, golden yellow; suitable for Uttar Pradesh, Bihar and West Bengal.

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Breeding for Combined Resistance to Ascochyta Blight and Fusarium Wilt in Kabuli Chickpea in Tunisia

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Abstract

Most studies on the genetics of resistance to ascochyta blight (*Ascochyta rabiei*) and fusarium wilt (*Fusarium oxysporum* f.sp. *ciceri*) in chickpea have assumed a gene-for-gene interaction. Recent information indicated that the genetic system governing resistance to *A. rabiei* is more complex. We used quantitative models to elucidate the nature of resistance to *A. rabiei* in kabuli chickpea and found additive and epistatic gene effects responsible for most variations in expressions of symptoms. Using this information and what is presently known on the genetics of resistance to fusarium wilt, a breeding strategy to combine resistance to *A. rabiei* and *F. oxysporum* is discussed.

Introduction

Wilts caused by *Fusarium* spp. and *Verticillium albo-atrum* Reinke & Berth. and ascochyta blight caused by *Ascochyta rabiei* (Pass.) Labr. have been reported from several countries (Halila and Harrabi 1987; Pathak *et al.* 1975). Both of these diseases can cause significant yield losses, reaching 100% when conditions are favorable. These diseases can be best controlled by host plant resistance.

Recently, researchers (Bravo *et al.* 1969; Haddad *et al.* 1988; Lopez 1974; Singh and Reddy 1983; Tewari and Pandey 1985; Vir and Grewal 1975) have proposed genetic models for inheritance of resistance to wilt and ascochyta blight in chickpea. We will review this information and relate it to selection schemes that may combine resistance of these two diseases in selected chickpea genotypes.

A few reports on the genetics of resistance to *Fusarium oxysporum* Schlecht. emend. Snyd. & Hans. f.sp. *ciceri* (Padwick) Snyd. & Hans. are available (Lopez 1974; Pathak *et al.* 1975; Sindhu *et al.* 1983). All of them show a qualitative type of resistance conditioned by one or two genes. However, Pathak *et al.* (1975) and Upadhyaya *et al.* (1983a) could not explain the inheritance of resistance in some crosses on the basis of

a simple Mendelian model. This necessitates the investigation of further interactions between pathogen and host. No reports are available on the inheritance of resistance to verticillium wilt in chickpea. Preliminary information indicates that genes resistant to both fusarium and verticillium wilts may be conditioned by independent loci.

The wilting process may be complex in nature because chickpea genotypes have been shown to differ in time of wilting (Upadhyaya *et al.* 1983b). This may be due to many factors. A plausible explanation could be the interaction of a virulence gene product/dominant (recessive) gene product complex that promotes a cascade effect in the host and subsequently causes retardation of symptom expression. The single recessive locus that was reported by early workers (e.g., Sindhu *et al.* 1983) may, in fact, contain multiple alleles or may be a result of an interaction between minor genes or modifiers as indicated by variation in late wilters such as C 104 (Upadhyaya *et al.* 1983b). These authors have observed a high variance in C 104 (σ^2 = 39.8) and a continuous variation in the cross between this genotype and the early wilter JG 62. Lack of discrete classes in F_2 is a good indication of a more complex genetic system governing the late wilting phenotype. If an analogy can be drawn between late wilting in chickpea and slow rusting in cereals, then one might expect a gene action that includes dominance, epistasis and additive effects and interactions among them that condition late wilting.

As with wilt, limited research data on the genetics of resistance to ascochyta blight are available. There is an urgent need to further investigate the inheritance of resistance to this disease. This aspect is further emphasized by recent recommendations to shift from the traditional spring sowing to winter sowing. Winter sowing could lead to a substantial increase in yield, provided resistant cultivars are used (Saxena 1980).

Hafiz and Ashraf (1953) were the first to report on genetics of resistance to *A. rabiei*. They concluded that a single gene pair controlled resistance in two French genotypes. The same conclusion was reached by Vir and Grewal (1974) in a desi type chickpea. Singh and Reddy (1983) studied the genetics of resistance in a set of resistant lines. They concluded that resistance follows a simple genetic model and proposed the terms rar1 and rar2 to designate resistance in ILC 191 and ILC 200, respectively. Recently, Halila *et al.* (1989) investigated genotypes from ICARDA germplasm and found that resistance is monogenically controlled, but they encountered difficulty in adopting the 1 to 9 scale. Thus, their classification of plants in segregating generations was highly subjective. Further observations indicated that resistance to *A. rabiei* in chickpea is not as simple as widely reported. These observations stemmed mainly from the lack of complete resistant reaction in chickpea genotypes and also from the controversy that exists on the race situation. All the previous studies on the genetics of ascochyta blight resistance assumed a gene-for-gene interaction. This appears to be a simplistic approach to a rather complex nature.

We propose that resistance to *A. rabiei* depends on, but cannot be attributable to in total, a single gene product. We have recently approached the problem differently and adopted a quantitative model to elucidate the nature of ascochyta blight resistance.

Four parameters—number of lesions per plant, average lesion length, total lesion and linear infection index defined as the ratio of total lesion length to stem length—were used to evaluate resistance in crosses between resistant and susceptible genotypes. As expected, the additive, dominance and epistatic gene effects were significantly predominant in the crosses investigated (Table 1). Therefore, the qualitative approach used by previous workers can be quite misleading in terms of development of a breeding strategy to improve ascochyta blight resistance.

Breeding for Multiple Resistance to Ascochyta and Wilt Diseases

There are a few prerequisites for the development of any breeding strategy for disease resistance:

(i) a clear understanding of the genetics of resistance to the disease,
(ii) a reliable method for screening plant material, and
(iii) the development of an appropriate selection scheme leading to the identification of resistant genotypes.

Genetics of Resistance to Both Diseases

Compared with cereals, chickpea has been neglected in terms of research. The bibliography on the genetics of resistance to ascochyta blight and fusarium wilt in kabuli chickpea is not abundant. This lack of essential information has hampered the development of germplasm resistant to these diseases. We propose that international centers with a chickpea mandate should invest more in terms of:

(i) training young scientists or directly funding research projects to systematically screen the chickpea collection for as many races as possible of *A. rabiei* and *F. oxysporum* f.sp. *ciceri*,
(ii) following up the screening phase with in-depth studies on the genetic systems regulating resistance to these diseases,
(iii) investigating resistance at the molecular level.

The literature indicates that there are possibly three to four genes for resistance to ascochyta blight in kabuli chickpea. Lines ILC 191 and ILC 195 have been reported to have rar1 and rar3, respectively; ILC 182, ILC 191, ILC 195 and ILC 482 may each have one dominant gene. ILC 200 has the dominant Rar2 gene (Haddad 1985; Singh and Reddy 1983). We have investigated the allelic relationship among some of the resistant sources. Lines ILC 72 and ILC 482 were found to have either one gene in common or genes that were closely linked. Other genotypes appeared to have independent genes for resistance to ascochyta blight. Further allelic investigations need to be undertaken to better evaluate the gene pool in chickpea.

Table 1. Estimated values of gene effect.

Cross	LA x ILC 182	LA x ILC 191	LA x ILC 482
Lesion number/plant			
m	1.53	1.37	2.04
a	0.69 ± 0.30*	-	0.04 ± 0.18*
d	-	-	-0.50 ± 0.31
aa	-	0.93 ± 0.28***	-
ad	0.31 ± 0.36	-0.59 ± 0.19***	-
dd	2.49 ± 0.72***	1.13 ± 0.23	-
R^2	0.1297	0.2160	0.0580
Average lesion length			
m	4.49	5.03	3.94
a	0.95 ± 0.74	-	-
d	-1.66 ± 0.78*	-7.95 ± 1.94***	-2.23 ± 0.98**
aa	-	-6.81 ± 1.75***	-1.29 ± 0.74
ad	-1.38 ± 0.89	-2.45 ± 0.49***	-2.18 ± 0.38
dd	-2.62 ± 1.82	9.75 ± 3.45***	-
R^2	0.180	0.216	0.058
Total lesion length			
m	6.66	7.81	7.92
a	4.87 ± 1.25***	2.90 ± 1.72	2.08 ± 1.87
d	-3.21 ± 2.23	-14.62 ± 4.69***	-4.64 ± 1.53***
aa	1.51 ± 1.64	-6.59 ± 4.24	-
ad	-1.79 ± 1.51	-6.59 ± 2.09***	-4.44 ± 2.14**
dd	-	17.32 ± 8.34	-5.09 ± 3.72
R^2	0.405	0.378	0.306
Linear infection index			
m	3.73	3.80	4.49
a	2.08 ± 0.77***	1.98 ± 0.99*	2.02 ± 1.25
d	-3.01 ± 0.80***	-8.57 ± 2.70***	-5.38 ± 1.74***
aa	-	-2.76 ± 2.44	-2.20 ± 1.32
ad	-1.73 ± 0.93*	-4.50 ± 1.20***	-1.94 ± 1.42
dd	-	10.38 ± 4.81*	-
R^2	0.353	0.469	0.290

LA = Local Amdoun.
R^2 = Determination coefficient.
*, ***: significant at P ≤0.05, 0.01.

For wilt also, only a few publications have dealt with the subject. There are probably two to three recessive genes conditioning resistance to *F. oxysporum* f.sp. *ciceri*. These are found separately in C 104, a late-wilting genotype, and in CPS 1 and WR 315, both of which were resistant to race 1 (Hyderabad) and may have one gene in common. Recently, Singh *et al.* (1987) reported a recessive allele for resistance in K 850 independent of that found in C 104. The presence of both alleles in the same genotype confers complete resistance. Other genotypes reported to have the recessive gene for these include 1231, 32/35-8/7 and 32/35-32/2. No allelic relationship was reported. Genetic studies on the inheritance of resistance to *V. albo-atrum* are completely lacking.

A Reliable Method for Screening Plant Material

The above reports assumed simple genetic models to explain the variation observed in the segregating populations. They also assumed a discrete classification of plants in the segregating generation, a situation rarely observed, especially when using different subjective methods to rate plants for their disease reaction to *A. rabiei* (Riahi 1988). These include:

(i) Number of lesions/plant. Figure 1 shows the distribution frequency of different plants representing a wide range of number of lesions/plant. Classification of resistant and susceptible plants based on this parameter gave a significant overlapping region. This area represents about 26% risk of misclassification.

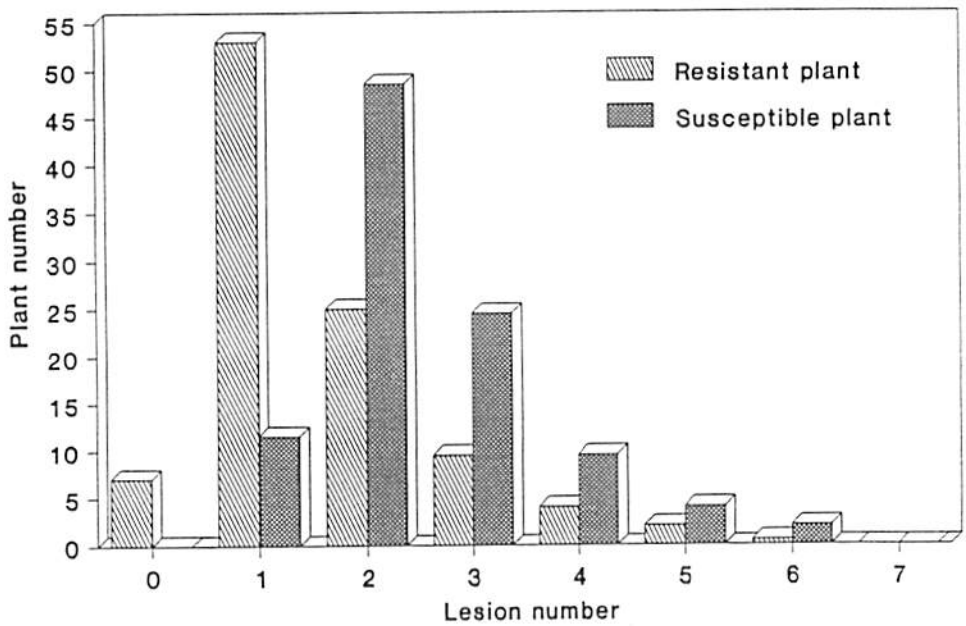

Fig. 1. Frequency distribution of lesion number per plant.

(ii) Average lesion length. There is about 23% overlapping region between the resistant and susceptible classes when using average lesion length (Fig. 2). The chances of misclassifying plants are still too high.

(iii) Total lesion length. This parameter was defined as the product of number of lesions and their average length. There is about 11% chance of misclassification of resistant and susceptible plants (Fig. 3). This probability, although small, is still not acceptable, especially in genetic studies where precision is highly desirable.

(iv) Linear infection index (LII). This index is defined as:

$$LII\ \% = (NL \times ALL)/SL$$

where: NL = Number of lesions
ALL = Average lesion length (mm)
SL = Stem length (mm)

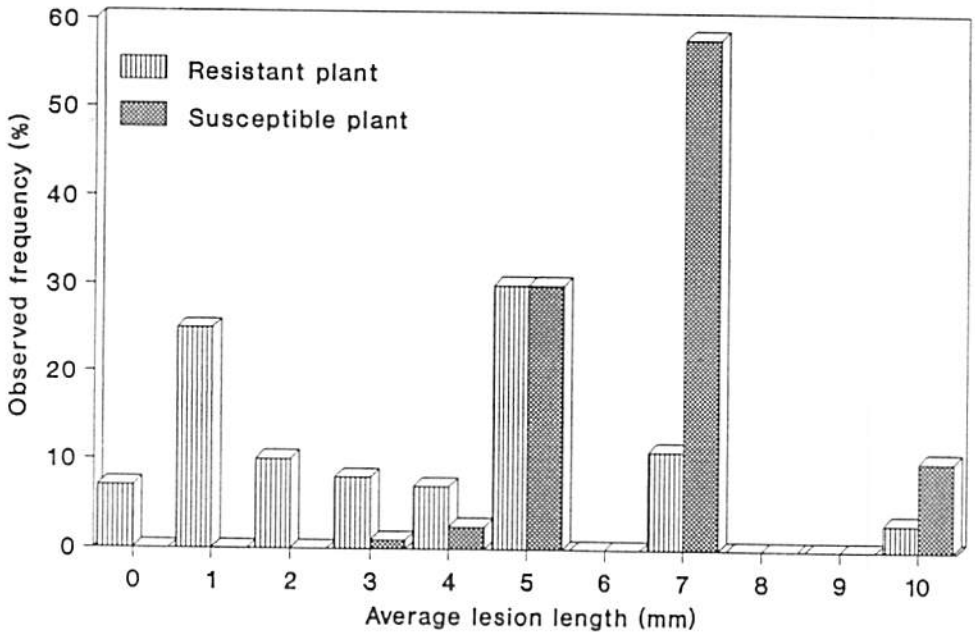

Fig. 2. Frequency distribution of average lesion length.

This index was developed mainly for the following reasons:

(a) infected plants become stunted, especially when lesions develop close to the apical region,

(b) percentage of infected area is difficult to estimate visually, and

(c) there is a high variation among chickpea genotypes in terms of stem length and diameter.

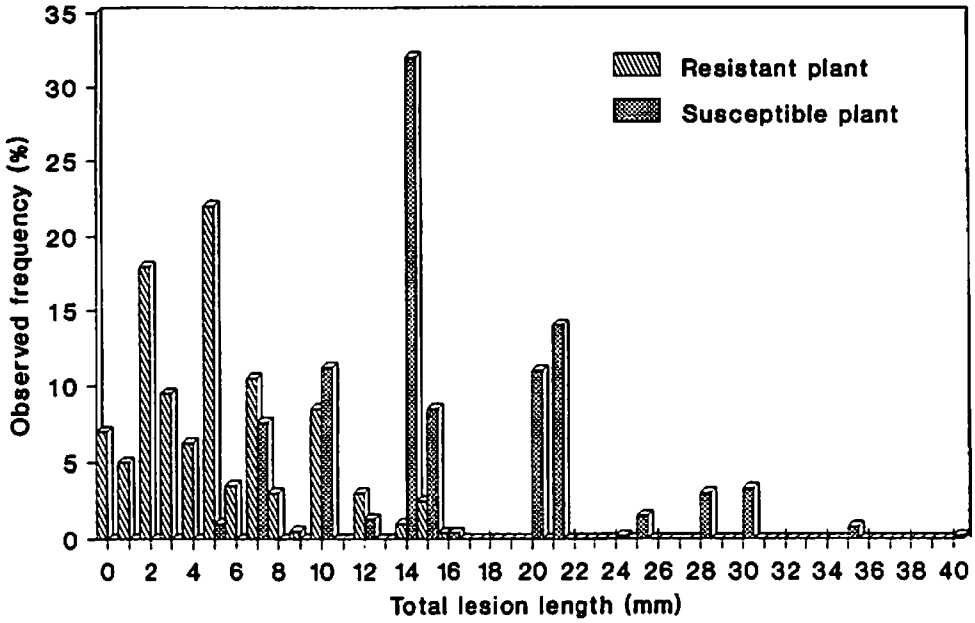

Fig. 3. Frequency distribution of total lesion length.

When this index was used, there was a significant decrease in the overlapping region between resistant and susceptible classes (Fig. 4). The probability of misclassification becomes 4%. Therefore we suggest that this parameter be used in controlled experiments.

For wilt we have tried an aquaculture medium where a spore suspension is added directly to a tube containing a chickpea plant growing in sterilized water (ICRISAT 1981). This technique was good in identifying early wilters but was not suitable for detecting late wilters. The best screening technique available is the development of a wilt-sick plot. It has the advantage of monitoring genotype reaction until maturity, provided an ample water supply in the soil is available (drought effects can mimic wilt symptoms and thus confuse selection).

The following steps could help establishment of a wilt-sick plot:

(i) conduct a race survey of the country where chickpea is grown,
(ii) grow a wilt-susceptible cultivar in each area that contains specific races,
(iii) harvest chickpea plant debris from each area,
(iv) mix debris in equal proportions,
(v) plow the debris mixture under the soil of the wilt-sick plot,
(vi) grow a susceptible chickpea cultivar the following 2 years to allow inoculum buildup and at the same time monitor increases in the number of propagules per gram of soil.

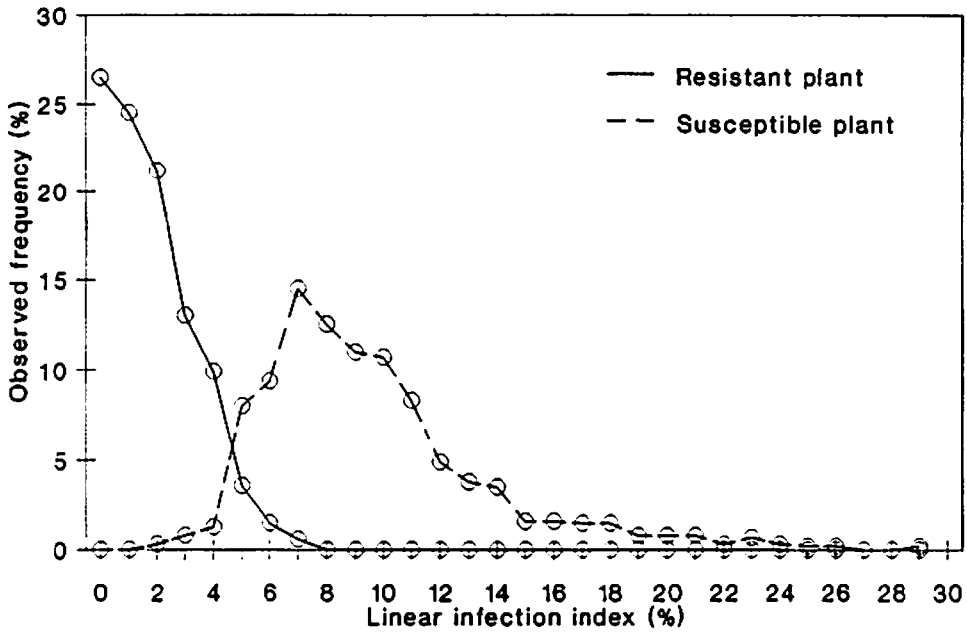

Fig. 4. Frequency distribution of linear infection index.

Selection Strategies

We will relate the above information to the development of selection schemes that may lead to the development of genotypes with dual resistance to *A. rabiei* and wilt pathogens.

The shuttle breeding approach (Halila and Harrabi 1988) is efficient if the genetics of the sources of resistance used in the breeding program are not well established (Fig. 5). A modification of shuttle breeding can be justified based on the recent findings that additive gene action is predominantly present in most characters investigated (Riahi 1988). These include total lesion length, number of lesions, average lesion size and linear infection index. Genetic variations in these traits are important to the chickpea breeder. Of these heritable variations, the additive variance reflects the magnitude of resemblance between parents and offspring. This type of variance is the most important to the breeder of self-pollinated crops because it is correctable in later generations. Based on this information, we propose pedigree selection, where selection for *A. rabiei* resistance is done at the F_4 and F_5 generations, but selection for wilt resistance will begin at the F_2 and F_3 in the wilt-sick plot. Negative selection for *A. rabiei* is performed at the F_6 generation. The resistant F_6 rows are bulked and tested for yield potential and later for multilocation testing (Fig. 6).

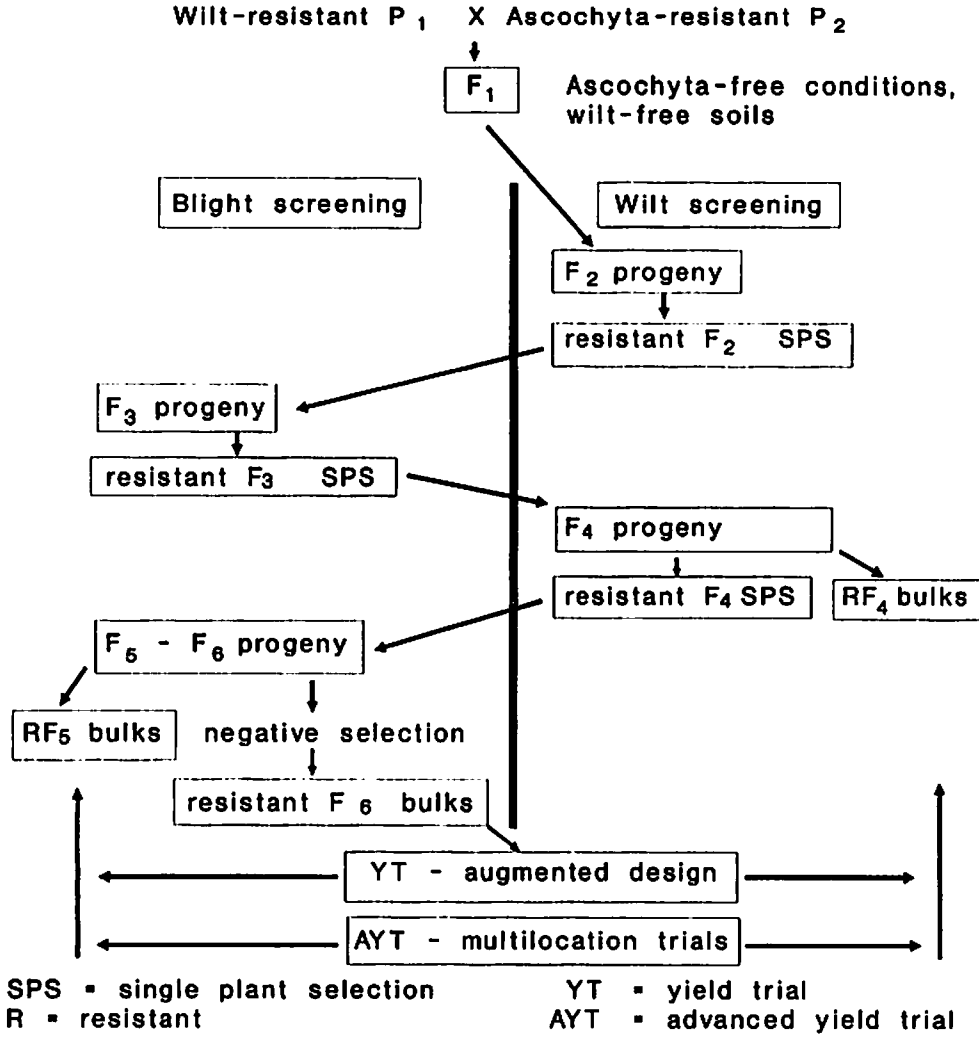

Fig. 5. Shuttle breeding method for combined resistance to ascochyta blight and fusarium wilt.

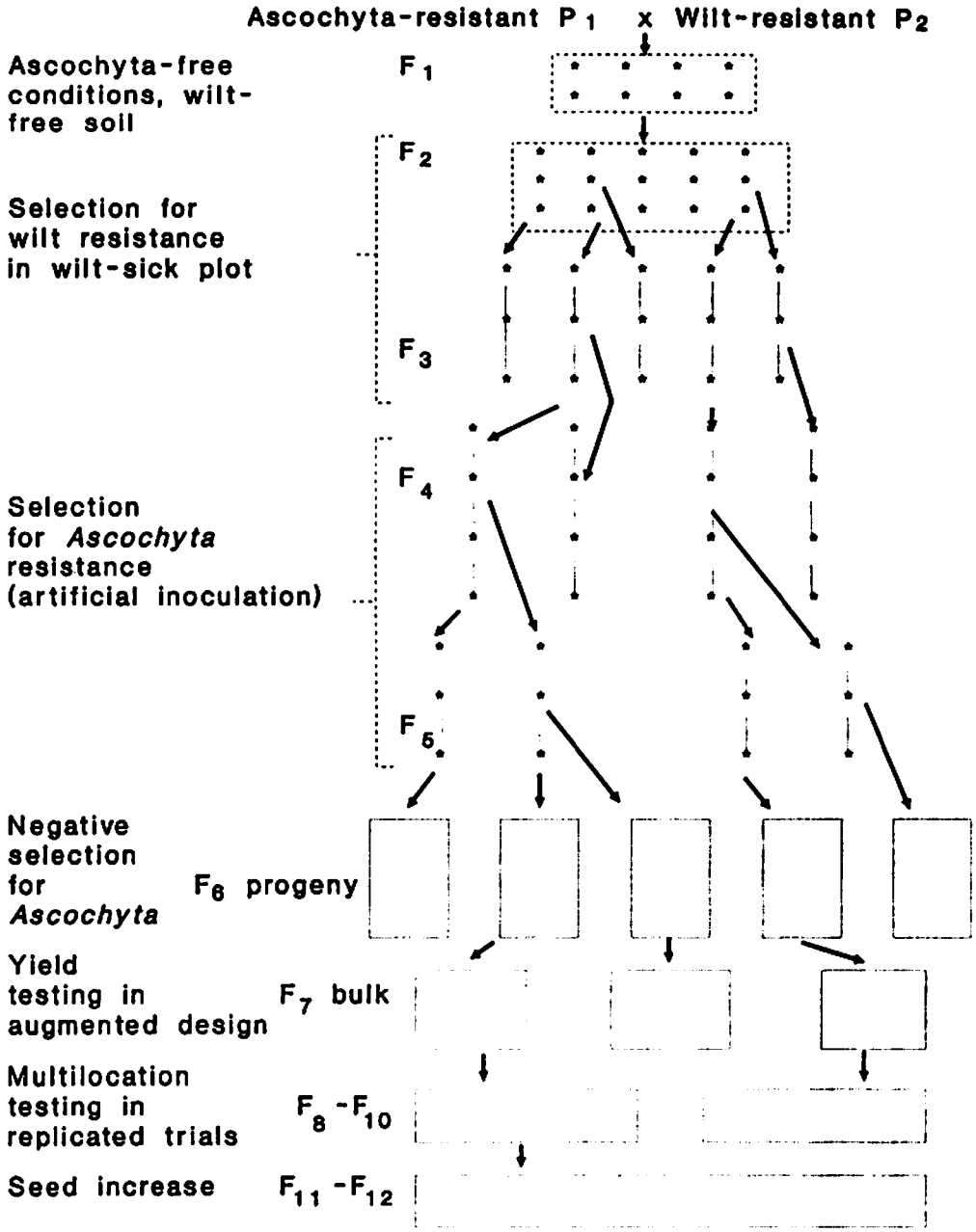

Fig. 6. Pedigree selection scheme for dual resistance to ascochyta blight and wilt diseases.

Although some of the explanations discussed above depart from the classical approach, they encompass some recent information on the genetics of disease resistance to *A. rabiei*. It is hoped that by drawing attention to the need for genetic studies to elucidate the regulation of disease resistance in chickpea we will motivate international centers to strengthen national programs and fund fundamental research on breeding for disease resistance in chickpea.

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Breeding for Ascochyta Blight Resistance in Food Legumes

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Abstract

Ascochyta blight on pea (*Pisum sativum*) and faba bean (*Vicia faba*) and artificial infection methods for resistance screening are described for greenhouse and field conditions. The greenhouse test was as effective as the field test in screening genotypes of faba bean against *Ascochyta fabae*. Resistance in pea and faba bean against different races of *A. pisi* and *A. fabae* was detected. A pea line (Rondo) is resistant to race C of *A. pisi* but is susceptible to race 4. Gullivert is resistant to race 4. Resistance of Rondo to race C is controlled by one recessive major gene. Only a few lines (L 2199, P 1259, Pis 405, Pis 802) seem to be moderately susceptible to *A. pinodes*. The behavior of 20 genotypes from ICARDA and 9 French lines was studied under artificial infection. The respective resistances of lines 29H, 29E, Asco 16 and Asco 17 were compared. Resistance in 29H seems to be controlled by one dominant major gene.

Ascochyta Blight in Food Legumes

Several legume species are affected by *Ascochyta* fungi: pea (*Ascochyta pisi*, *A. pinodes*, *A. pinodella*); faba bean (*A. fabae*); lupin (*A. caulicola*); bean (*A. bolsthauseri*, *Ascochyta* sp.) and chickpea (*A. rabiei*).

Blight lesions produced by *Ascochyta* spp. are spots on the leaves, stems and pods. The size, shape and color of the spots depend upon species. Under favorable environmental conditions, many tissues are killed and productivity is greatly affected. The perithecial stage usually is not known. The pycnidial form is responsible for dissemination of the disease.

On lupin *A. caulicola* causes no serious losses. In France, *A. caulicola* has been detected only once in 14 years on 600 samples from different crops.

Ascochyta bolsthauseri on bean seems not very severe, although serious losses have been reported occasionally in France, where this disease appears on late crops, inducing some red spots on pods. However, its importance can increase in tropical areas, especially in Columbia above 1700 m. It seems that the Columbian strain is different from *A. bolsthauseri* and all genotypes of *Phaseolus polyanthus* are resistant to it. This resistance could be incorporated into *Phaseolus vulgaris* since a cross between the two species is possible.

This paper will focus on *Ascochyta* spp. on pea and faba bean.

Ascochyta Diseases of Pea

The three *Ascochyta* species of pea (*A. pisi*, *A. pinodes* and *A. pinodella*) can be distinguished by their symptoms on pea, the size and the shape of their spores and their cultural characters on Mathur medium (Fig. 1) (Cousin 1973, 1974).

Ascochyta pisi shows pink spore masses and 10 to 13 µ-long bicellular spores. *Ascochyta pinodes* shows a mixture of light and dark pycnidia and 12 to 14 µ-long bicellular spores. *Ascochyta pinodella* shows black pycnidia distributed along the branches of mycelia, 3 to 7 µ-long unicellular spores and black chlamydospores.

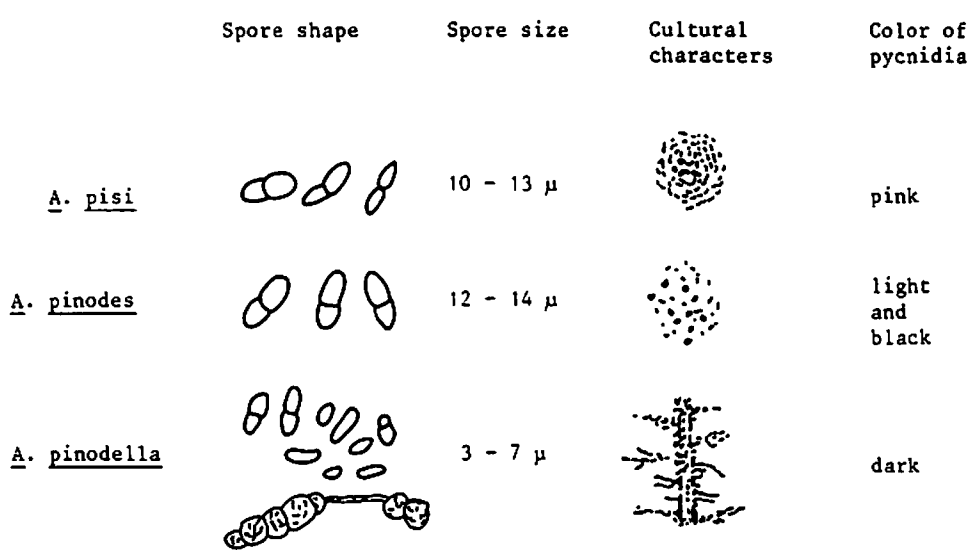

Fig. 1. Differences between the three *Ascochyta* species observed in culture on Mathur medium.

Ascochyta pisi

This species has been reported from every pea-growing area of the world but nowhere is it very important at present. *Ascochyta pisi* is only known in pycnidial form. It produces circular gray spots with reddish brown margins on leaves and pods. More elongated spots or circular spots appear on the stems. The many pycnidia that appear in the center of the spots release bicellular spores in a mucilaginous carrot-red exudate. Wind-blown rain and high humidity increase dissemination of the spores.

Ascochyta pinodes

This is the only species for which the perithecial stage *Mycosphaerella pinodes* is known. *Mycosphaerella* occurs worldwide and is economically significant when cropping techniques and environmental conditions favor its development. It is the most important of the three ascochyta diseases of pea. The spreading lesions of *A. pinodes* are more severe than the limited lesions of *A. pisi*. The disease is more prevalent on winter crops than on spring crops where spots are more numerous, their color is darker and their shape less regular. Yield decreases in plants with a bushy growth habit. High moisture conditions increase the incidence of the disease. Aerial discharge of ascospores is responsible for much wider dissemination than that effected by pycnidiospores.

Ascochyta pinodella

Ascochyta pinodella attacks chiefly the lower part of the stem. Brown purple spots appear from this disease and it causes foot rot. The best artificial infection method consists of spraying spores on young seedlings grown in the greenhouse at 20°C and high humidity. Spores must be collected 3 or 4 days after subculturing when the culture becomes pink.

The studies concerning plant and pathogen interactions show that the young tissues of the plant are the only susceptible organs.

The behavior of more than 400 lines has been studied. Some of them, namely Kelvedon Wonder, Aldot and Petit Provencal, are very susceptible. Most of them are susceptible. Only a few lines developed from Rondo show a good level of resistance (Cousin 1974). Presently a few cultivars, especially in the group of proteaginous pea, show resistance: Amino, Calypso, Finale, Frilene, Maxi, Miranda and Solara.

Genetic study reveals that the resistance of Rondo is practical dominance and is controlled by one recessive major gene. It gives plants a good level of resistance against the most prevalent strains of *A. pisi*. The studies undertaken at Versailles have led to the identification of lines resistant to different physiological races of *A. pisi*. The resistance found in the cultivar Rondo is effective against nearly all physiological strains of *A. pisi* and shows high heritability. Some strains can infect Rondo, but new resistant lines can be found. Thus we can list the pathotypes that can be distinguished by differential hosts (Table 1) (Cousin *et al.* 1985).

Table 1. Physiological races of *Ascochyta pisi* and differential hosts.

Host cultivar	Races						
	D No.1	Several strains	- No.4	- No.14	C Tezier	B -	E -
Gullivert	R†	R	R	R	S	R	R
Rondo	R	R	S	VLS	R	R	S
Finale	R	R	S	LS	R		
Kelvedon Wonder	R	S	S	S	S	R	R
Dark-skinned Perfection	S	S	S	S	S	R	S
Arabal, Cobri, Starcovert, Supcovert, Vitalis	S	S	S	S	S	S	S

† R = resistant, S = susceptible, VLS = very little susceptibility, LS = little susceptibility.

Rondo also has a good level of resistance against *A. pinodella*, but a rather high susceptibility to *A. pinodes*, so we have started to look for genes of resistance against this disease.

In Australia, Ali *et al.* (1978) selected some lines resistant to certain strains, but these lines appear to be susceptible to the Versailles *M. pinodes* isolates.

A group of French plant breeders, G.I.E.S.P.P., is screening a large collection of pea lines. No line free of damage from disease has been found, but some lines seem to be moderately susceptible such as L 2199, P 1259, Pis 405 and Pis 802.

Ascochyta Blight on Faba Bean

Ascochyta fabae is the most prevalent *Ascochyta* sp. in France, especially in winter faba bean. During winter *A. fabae* infects the leaves and gives ash gray colored spots. They have an anthocyanic margin and often elongate between the veins. In the center of these spots, black pycnidia can be seen. They release pycnidiospores which are responsible for secondary contaminations: lozenge-shaped, depressed, margined spots that appear on the stems, then on the pods and maturing seeds.

The best incubation results are obtained when the initial temperature is low, 5 to 10°C, then increases to 10 to 20°C at the end of the test.

Two methods have been compared under greenhouse and field conditions.

Greenhouse

Plants were grown in pots after a 5-week period of vernalization at 3 to 5°C. Plants at four to seven leaves and flowering stages were sprayed with a spore suspension containing 0.5 million spores/ml. The growth media were malt extract agar and barley grain.

Inoculated plants were kept under polyethylene bags for 5 days with alternate day and night temperatures of 25 to 15°C, respectively. After the bags were removed, plants were sprayed with water twice a day.

Field

A suspension of inoculum was sprayed when the plants had reached the 3-leaf stage. Dissemination occurred by splashing. Scores were calculated following Kharbanda and Bernier's (1980) assessment method. The results show that the greenhouse test is as effective as the field evaluation (Table 2) (Tivoli *et al.* 1987). This method revealed a genetic variability for resistance against *A. fabae*. Line 48 B is very susceptible and lines 29 H and 29 E appear resistant.

Table 2. Average disease scores per plant of nine genotypes of *V. faba* and one of *V. narbonensis* (Vn) after spraying with *A. fabae* spores at the young plant and flowering stages and in the field, 1983/1984†.

	Young plant stage		Flowering stage		Field	
Genotype	Leaves	Pods	Leaves	Pods	Leaves	Pods
Vn	1.0	1.0	1.0	1.0	1.0	1.0
29H	1.1	1.0	2.0	1.5	1.0	1.0
29E	1.1	1.0	3.3	1.9	1.0	1.0
Bourdon	2.1	1.1	2.9	2.0	2.6	1.9
S45	2.9	1.5	3.0	1.8	2.6	2.1
Soravi	3.2	1.4	3.7	2.1	2.9	2.2
245.17	3.3	1.4	3.2	1.9	3.9	2.9
48B	3.5	1.9	4.0	2.5	3.7	3.1
3.33	4.0	1.7	3.3	1.7	3.5	2.4
LCF	4.3	1.7	2.5	1.9	3.1	2.0
F Value	180.4	22.3	32.5	7.0	64.5	81.5
F at P≤0.05	1.9	1.9	1.9	1.9	2.2	2.2
LSD	0.4	0.2	0.4	0.4	0.4	0.2

† FABIS Newsletter No. 17 (1986).

Laboratories in Syria and France screened a wide collection to find different sources of resistance. ICARDA found 20 resistant lines, Asco 1...Asco 20. These lines, tested in France at Rennes under field conditions, showed a genetic variability for this resistance (Table 3) (Tivoli *et al.* 1988).

Like 29 H, Asco 17 is quite resistant on leaves and pods; Asco 18 is resistant on leaves and moderately susceptible on pods; Asco 16 is moderately susceptible on leaves and resistant on pods. Other lines were susceptible.

Table 3. Average disease reaction of certain ICARDA genotypes to *Ascochyta fabae* under field conditions in France during the 1985/86 season†.

Genotype	Percent of damaged leaves	Genotype	Percent of damaged leaves
Asco 18	5.5	Asco 17	0.0
Asco 17	6.8	Asco 16	0.3
Asco 16	18.1	Asco 9	9.8
Asco 9	19.1	Asco 2	10.0
Asco 2	23.9	Asco 19	10.1
Asco 3	24.3	Asco 13	10.3
Asco 19	24.9	Asco 6	12.2
Asco 20	26.4	Asco 12	14.1
Asco 8	26.7	Asco 18	16.3
Asco 15	27.1	Asco 4	19.2
Asco 5	28.1	Asco 5	19.7
Asco 12	31.3	Asco 8	20.4
Asco 6	34.9	Asco 3	22.1
Asco 14	36.9	Asco 10	24.9
Asco 10	37.2	Asco 7	26.0
Asco 4	38.8	Asco 1	26.0
Asco 13	38.9	Asco 20	30.9
Asco 7	49.2	Asco 11	32.7
Asco 11	50.5	Asco 15	38.3
Asco 1	53.5	Asco 14	44.5
29 H	4.3	29 H	0.6
48 B	59.5	48 H	25.0
LSD (5%)	23.7		

† FABIS Newsletter No. 21 (1988).

These results suggest that different races occur in France and Syria. Several distinct sources of resistance against these races were found .

The resistance of 29 H seems to be controlled by one dominant major gene. However, the resistance found in Syrian lines seems to be more complex as the behavior is different on leaves and pods.

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Epidemiology of *Ascochyta rabiei*

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Abstract

Ascochyta blight of chickpea (*Cicer arietinum*) caused by *Ascochyta rabiei* is a devastating disease in many countries of South Asia, the Middle East and Mediterranean regions and North America. Epidemics of chickpea blight have occurred since the early 1900s in many countries, yet we still lack answers as to why epidemics occur when and where they do. For an epidemic of blight to occur several factors are required, including the presence of numerous susceptible plants, abundant inoculum of virulent isolates of the pathogen and an environment favorable for disease development over a period of time. Although a great deal is known about ascochyta blight and its control by various means, there are serious gaps in our knowledge of the different factors that affect epidemiology. More information is needed on the importance of infected seed and infected debris in the disease cycle. Questions concerning the role of the teleomorph (sexual stage) and alternate hosts in survival and spread of blight in different countries need to be addressed. This paper will review what is known about the epidemiology of ascochyta blight and highlight areas where additional research is required.

Introduction

Ascochyta blight of chickpea (*Cicer arietinum* L.), caused by *Ascochyta rabiei* (Pass.) Labr., is one of the most important diseases of chickpea in many countries of South Asia, the Middle East, the Mediterranean region and North America (Kaiser and Muehlbauer 1988; Nene 1982; Nene and Reddy 1987; Punithalingam and Holliday 1972). Since the disease was first described on chickpea by Butler in 1911 (Butler 1918) in the North West Frontier Province of Pakistan, research has been conducted out by many investigators on the pathogen, its biology, spread, survival and control.

Serious gaps still exist, however, in our knowledge of the disease, particularly factors related to its epidemiology. In this paper, I will review the information that is known about the epidemiology of ascochyta blight of chickpea and emphasize areas where additional research is required.

To understand how and why epidemics of ascochyta blight occur at various times in different regions of the world, we need to review information on the pathogen, the

source, survival and spread of inoculum, host range and environmental conditions required for infection and disease development. A disease cycle of ascochyta blight of chickpea in the Pacific Northwest, USA, is presented in Figure 1.

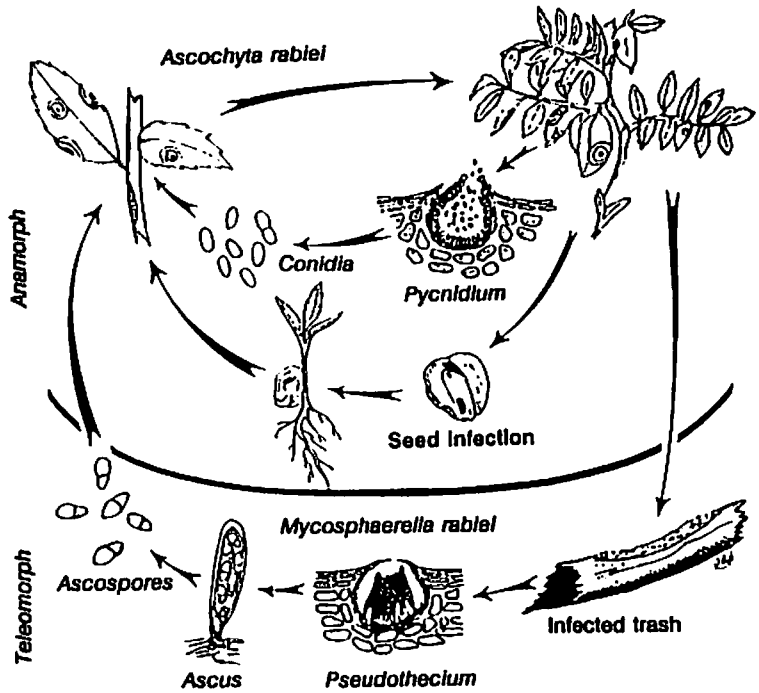

Fig. 1. Disease cycle of ascochyta blight of chickpea caused by *Ascochyta rabiei* in the Pacific Northwest, USA. Pycnidia also can exist on chickpea debris. (Drawing by R.M. Hannan.)

The Pathogen

Ascochyta rabiei is the anamorph (asexual or imperfect stage) of the causal organism of ascochyta blight of chickpea; *Mycosphaerella rabiei* (Pass.) Kovach. [= *Didymella rabiei* (Kovach.) v. Arx] is the teleomorph (sexual or perfect stage) (Kaiser 1987; Nene 1982; Punithalingam and Holliday 1972).

Anamorph

The asexual reproductive stage (*A. rabiei*) infects all foliar tissues of chickpea during the growing season. In different culture media and on susceptible chickpea plants, *A. rabiei* produces pycnidia containing masses of hyaline spores (conidia), most of which are unicellular.

Isolates of *A. rabiei* differ in growth rate, sporulation and colony appearance (Kaiser 1973; Luthra *et al.* 1939). The fungus grows and sporulates on different culture media (Bedi and Aujla 1969, 1970; Kaiser 1973; Luthra and Bedi 1932; Luthra *et al.* 1939; Sattar 1933a). The optimum temperature for mycelial growth, pycnidial formation and spore germination of different isolates of *A. rabiei* is about 20°C, with the lower and upper limits around 5 and 30°C, respectively (Bedi and Aujla 1970; Kaiser 1973; Luthra *et al.* 1939; Zachos *et al.* 1963).

At temperatures of 20 to 25°C, conidia germinate in 2 to 3 hours on glass microscope slides in water or water containing nutrients (Kovachevski 1936; Zachos *et al.* 1963). Conidia are reported to survive adverse environmental conditions for various time intervals (Sattar 1933b; Zachos *et al.* 1963). For example, when chickpea plants were incubated in a dry atmosphere (<50% RH) at 20°C for 96 hours immediately after inoculation, conidia remained viable and were still capable of infecting the tissues when the plants subsequently were placed in a saturated atmosphere at 20°C (Trapero-Casas and Kaiser 1987b, and unpublished data).

Disease symptoms may appear within 3 to 6 days of inoculation, depending on the temperature and humidity (Khachatryan 1962; Zachos *et al.* 1963). When chickpea plants were inoculated with conidia of *A. rabiei* and incubated at 20°C and 100% RH, symptoms appeared in 4 to 6 days and pycnidia containing viable spores were present in 5 to 7 days (Chauhan and Sinha 1973; Trapero-Casas and Kaiser 1987b).

In greenhouse inoculation studies, Chauhan and Sinha (1973) observed no disease development at 10° and 30°C, apparently because these temperatures were unfavorable for spore germination, mycelial growth and infection. Weltzien and Kaack (1984) reported no infection of inoculated chickpea plants below 6°C or above 30°C. Relative humidity is important in infection, symptom expression and disease development. Symptoms occurred in 84 hours at 20°C in inoculated plants incubated at 85 to 100% RH, but not at 52 to 70% RH (Chauhan and Sinha 1973).

Teleomorph

Mycosphaerella rabiei, the sexual reproductive stage, develops on dead, infected chickpea residue after harvest. In 1936, Kovachevski (1936) described *M. rabiei* on chickpea debris that had overwintered on the soil surface in southern Bulgaria. Subsequently, *M. rabiei* has been reported on overwintered chickpea residue in the USSR (Gorlenko and Bushkova 1958), Greece (Zachos *et al.* 1963), Hungary (Kovics *et al.* 1986), the USA (Kaiser and Hannan 1987), Spain (Jimenez Diaz *et al.* 1987) and Syria (Haware 1987). The teleomorph has been found only on chickpea refuse. It has not been observed on culture media or on infected chickpea plants during the growing season (Kovachevski 1936; Trapero-Casas and Kaiser 1987a). Pseudothecia did not form on stems of sterile sweet clover (*Melilotus alba* Medik.) (Kovachevski 1936).

The pseudothecia on infected chickpea debris are dark brown to black and similar in size, shape and color to pycnidia. Pseudothecia usually are scattered over the dead tissues rather than localized in clusters or rings within lesions.

Moisture is essential for initiation and development of pseudothecia and discharge of ascospores. Pseudothecial development ceased when the infected debris was air dried (Trapero-Casas and Kaiser 1987a).

Pseudothecial development on naturally infected chickpea debris was studied at temperatures of 5 to 30°C. When tissue was placed between moist paper towels in plastic bags, pseudothecial initials formed at 5 to 25°C. Mature pseudothecia developed in about 50 days at 5 to 10°C. Only a few mature pseudothecia formed at 15°C and none developed at 20 to 25°C. Light was not required for development of mature pseudothecia (Trapero-Casas and Kaiser 1987a).

Ascospores germinate at temperatures of 5 to 30°C (W.J. Kaiser, unpublished data). At 20 to 25°C, ascospores germinated in 3 to 4 hours (Kovachevski 1936). The ascospores are tolerant to adverse environmental conditions. They remained viable for 15 days at 10°, 22-25° and 30°C and <40% RH in petri dishes (W.J. Kaiser, unpublished data) and for 96 hours at 20°C on chickpea foliage that was dried immediately after inoculation (A. Trapero-Casas and W.J. Kaiser, unpublished data). When ascospores were discharged onto chickpea foliage maintained at 20°C and 100% RH, symptoms developed in 4 to 5 days (W.J. Kaiser, unpublished data). Once chickpeas are infected by ascospores, the anamorph stage begins. Cultures that develop from ascospores on culture media vary greatly in growth rate, sporulation and colony appearance (Jimenez Diaz *et al.* 1987; W.J. Kaiser, unpublished data). The teleomorph does not develop on the living host.

Little is known of the genetics of pseudothecial development. However, it appears that the fungus is heterothallic, requiring at least two compatible thalli (mating types) for sexual reproduction (before pseudothecia develop on infected chickpea debris) (Trapero-Casas and Kaiser 1987a).

Sources of Blight Infection

Infected Seed

Infected seed is an efficient method of transporting the pathogen, from season to season and from one area to another. It is also important in the epidemiology of the disease (Kaiser 1972; Luthra and Bedi 1932; Luthra *et al.* 1935; Sattar 1933b; Weltzien and Kaack 1984; Zachos *et al.* 1963). Butler (1918) was perhaps the first to report infection of chickpea seed by the blight pathogen. He also described transmission of the pathogen from infected seed to seedlings during germination, but Luthra and Bedi (1932) were the first to conduct detailed studies on the seed-borne nature of the blight disease.

Infected chickpea seed has been responsible for the introduction of the pathogen into several countries or regions that were previously free of ascochyta blight, including Australia (Cother 1977), Canada (Morrall and McKenzie 1974; Tu and Hall 1984), Iran (Kaiser 1972) and the USA (Kaiser and Muehlbauer 1984). In several instances, the introduction of *A. rabiei* with seed for research trials or commercial plantings resulted in extensive damage to the crop in the same year that the seed was introduced or soon thereafter. For example, ascochyta blight of chickpea was first detected in the Pacific Northwest, USA, in 1983 (Kaiser and Muehlbauer 1984). In 1984, it was found in 23 of 30 commercial chickpea plantings in northern Idaho, USA (Derie *et al.* 1985). The teleomorph was discovered on overwintered chickpea debris in northern Idaho in March 1986 (Kaiser and Hannan 1987) and in 1987 blight was widespread in the region, causing severe damage to the crop that resulted in drastically reduced yields and lower seed quality (Kaiser and Muehlbauer 1988).

The incidence of *A. rabiei* in commercially produced seed may be more widespread than previously realized. Maden *et al.* (1975) detected *A. rabiei* in 70% of the chickpea seed samples from central Anatolia, Turkey, with seed infection ranging from 1 to 16%. Halfon-Meiri (1970) found seed infection exceeding 5% in many local seed lots. In the Pacific Northwest, USA, seed infection of commercial seed lots varied with the year and ranged from 0.5 to 31% (W.J. Kaiser, unpublished data) (Table 1).

Table 1. Incidence of *Ascochyta rabiei* in commercial chickpea seed lots from the Palouse region of eastern Washington and northern Idaho, USA.

Year	Seed samples		Range in seed infection (%)
	Total	Infected	
1984†	50	4	0.5-1.5
1985	23	3	0.5
1986	16	3	1-2
1987	38	34	1-31

† The number of seeds plated on potato-dextrose agar per sample in 1984, 1985, 1986 and 1987 were 200, 400, 200 and 100, respectively. Seeds were surface disinfected in 0.25% NaOCl for 5 minutes.

When harvesting of the blight-infected chickpea crop was delayed with rain before harvest, Lukashevich (1958b) in the USSR found that seed infection by *A. rabiei* increased 1.5 to 2 times.

Seed infection does not necessarily result in transfer of the pathogen to the germinating seedling. In chickpea, Maden (1983) found that seed transmission of *A. rabiei* varied from 12% (of 131 infected seeds) under field conditions to 25% (of 30 infected

seeds) in greenhouse experiments. Weltzien and Kaack (1984) observed seed transmission of the blight pathogen in 30 of 95 infected chickpea seeds planted in a field trial in Syria. In chickpea seed with >40% seed-borne *A. rabiei*, transmission of the pathogen to seedlings under greenhouse conditions was as high as 20% in untreated seed (Kaiser and Hannan 1988). However, when these heavily infected seeds were treated with benomyl or thiabendazole plus captan and planted in sterile potting medium or field soil in the greenhouse, transmission of *A. rabiei* to emerging seedlings was usually 1% or less (Kaiser and Hannan 1988). At times, emergence and subsequent growth of untreated seeds infected with *A. rabiei* was significantly reduced (Halfon-Meiri 1970; Kaiser and Hannan 1988; Maden 1983).

Under some conditions (temperature, moisture, antagonistic seed and soil microflora), the frequency of seed transmission can be very low, perhaps only one seed among thousands. However, the rarity of seed transmission is offset by rapid increase of *A. rabiei* on infected tissues when exposed to environmental conditions that favor blight development.

Seed transmission ensures a random distribution of the blight pathogen in a field, providing many primary infection foci from which the pathogen can spread, depending on weather conditions and a susceptible chickpea crop. In an area with a climate highly favorable to blight development, the pathogen can spread rapidly from only a few infected seeds. Very low levels of infection (in the range of 0.01 to 0.1% infected seed) combined with cool, wet weather appear to have been responsible for outbreaks of ascochyta blight in many commercial plantings in northern Idaho in 1984 (Derie *et al.* 1985; W.J. Kaiser, unpublished data).

The pathogen survives adverse environmental conditions for long periods both internally and externally on infected chickpea seed. Sattar (1933b) demonstrated that conidia smeared on the surface of chickpea seed survived 5 months at 25 to 30°C, at which time germination of conidia exposed to 25 and 30°C was 50%, while that at 35°C was 5%. When inoculated seeds were planted, *A. rabiei* was transmitted to many of the emerging seedlings.

Maden *et al.* (1975) found that one-third of the spores washed from heavily infected seeds stored at 2 to 5°C for 14 months germinated. Kaiser (1972) showed that the blight fungus survived in infected chickpea seeds for over 117 weeks in a weather station shelter in southwestern Iran where summer temperatures exceeded 45°C. Survival studies in Iran and the USA demonstrated that the fungus was still viable in infected chickpea seed stored over 5 years at 2 to 5°C and 30 to 40% RH (W.J. Kaiser, unpublished data). It is conceivable that *A. rabiei* will survive in infected chickpea seed in germplasm collections at temperatures of 4 to 18°C and <50% RH for as long as the seed remains viable. Great care must be taken by germplasm centers in different countries to prevent the inadvertent spread of *A. rabiei* with chickpea seed into areas where the disease or more virulent isolates of the pathogen do not occur.

Infected Debris

Researchers in different countries have emphasized the importance of infected chickpea residue in survival of the pathogen from one growing season to the next and as a source of infection for subsequent chickpea plantings (Askerov 1968; Kaiser 1973; Khachatryan 1962; Kovachevski 1936; Lukashevich 1958b; Luthra *et al.* 1935; Zachos *et al.* 1963).

After death of the host, Lukashevich (1958b) in the USSR found that *A. rabiei* developed as a saprophyte on the chickpea debris and by the following spring the tissues were heavily infected by the pathogen. Colonization of chickpea refuse after harvest by *A. rabiei* also was noted by Luthra *et al.* (1935), Trapero-Casas *et al.* (1988) and Zachos *et al.* (1963). In Greece, Zachos *et al.* (1963) observed that infected debris left in the field for 2 years became covered with pycnidia and pseudothecia containing viable spores. They found the first immature and mature pseudothecia on infected refuse in January and February, respectively.

In northern Idaho, USA pseudothecia developed in August or September in large numbers on naturally infected chickpea refuse that overwintered on the soil surface after harvest. Maturity of pseudothecia, determined by ascospore discharge, usually began in February or March and increased to a maximum in April or May (Trapero-Casas and Kaiser 1987a; Trapero-Casas *et al.* 1988). Maximum discharge of ascospores varied from year to year and ranged from 400 to 1,600/mm^2 of infected chickpea tissue (Fig. 2). The discharge of ascospores in the spring coincided with the early vegetative stage of chickpea. Ascospore discharge usually decreased drastically in June to October (Trapero-Casas and Kaiser 1987a). Repeated wetting and drying of naturally infected chickpea debris at weekly intervals revealed that ascospores were discharged for up to 6 weeks, with the maximum discharge usually occurring within the first 24 hours (W.J. Kaiser, unpublished data).

In laboratory studies, pycnidia containing viable conidia developed on chickpea stem pieces at temperatures of 10 to 30°C, but formed quickest at 20°C. Mature pycnidia developed on stem pieces at 20°C in 46 hours in continuous light, 50 hours in alternating light and dark and 68 hours in continuous dark (Kaiser, 1973).

Luthra *et al.* (1935) were probably the first to report the importance of infected debris in survival of the pathogen. The pathogen was still viable in infected chickpea residue on the soil surface for more than 2 years and it lost its viability on refuse buried at least 5 cm in moist soil during the summer. In Iran, Kaiser (1973) found that the pathogen survived more than 2 years in naturally infected chickpea refuse in the laboratory at 10 to 35°C, 30% RH or less and outdoors on the soil surface. At the end of the experiment the fungus was still highly pathogenic to chickpea. In Syria, the pathogen survived in infected debris on the soil surface for 8 months, but only for 4 months when buried at least 10 cm deep (Nene and Reddy 1987). Weltzien and Kaack (1984) did not consider infected chickpea refuse an important source of inoculum in field studies in Syria.

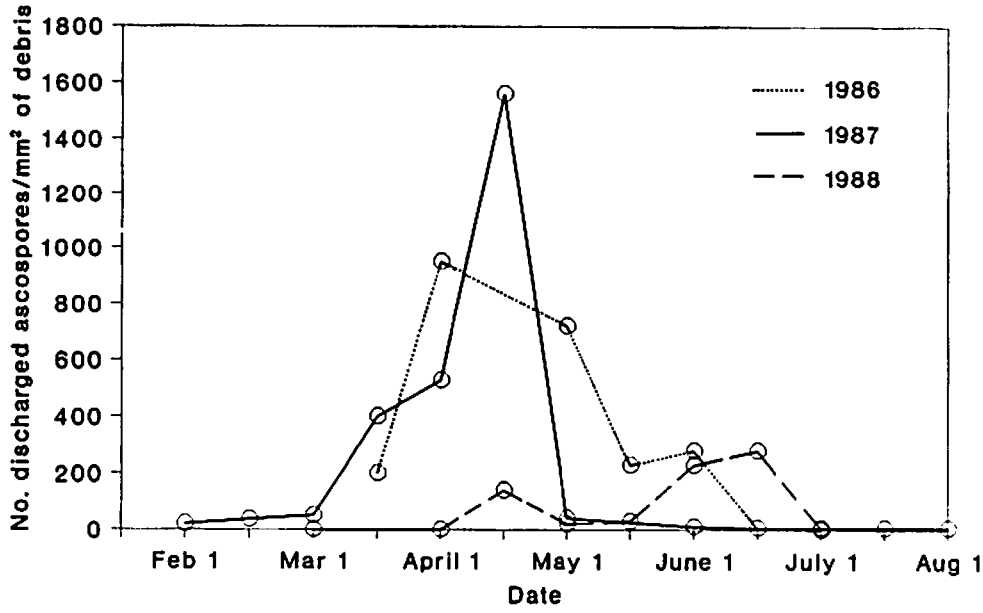

Fig. 2. Number of ascospores of *Mycosphaerella rabiei* discharged from naturally infected chickpea debris collected at periodic intervals from 1986 to 1988 from a farm in Genesee, Idaho, USA.

Survival of the anamorph and teleomorph of the blight pathogen in naturally infected chickpea debris was studied in field soil at Pullman, Washington, USA. Conidia remained viable on infected pods and stems on the soil surface for 57 and 81 weeks, respectively, but when buried 16 cm deep conidia lost their viability in 10 and 15 weeks on stems and pods, respectively. In a weather station shelter or at 4°C and 30 to 40% RH, conidia still germinated after 120 weeks (Kaiser *et al.* 1987) (Fig. 3). Discharge of ascospores from pseudothecia in chickpea debris ceased after 21 weeks on the soil surface and 8 weeks when buried 16 cm deep. Viable ascospores were discharged from infected tissue after 50 weeks in a weather station shelter or at 4°C and 30 to 40% RH (Kaiser *et al.* 1987).

Dissemination of Inoculum

Conidia

In moist weather, conidia of *A. rabiei* ooze from pycnidia in a gelatinous matrix. Moisture is necessary to dissolve this matrix and free the spores which then may be washed or splashed from plant to plant or scattered in the water droplets of driving rain.

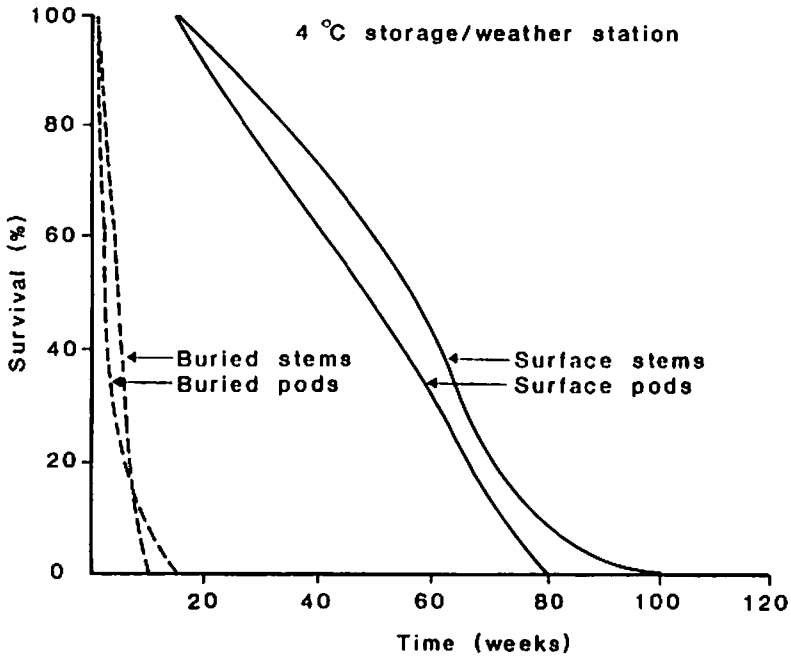

Fig. 3. Survival of *Ascochyta rabiei* in naturally infected chickpea stems and pods on the soil surface, buried 16 cm deep, or incubated at 4°C or outdoors in a weather station shelter at Pullman, Washington, USA.

In dry weather the extruded conidia dry into hard masses on the lesions, subject to dispersal by subsequent wetting. This method of spore production is adapted to dispersal by water. Splash dispersal of conidia of fungi like *A. rabiei* usually occurs over short distances. However, the combination of splashing rain with strong wind may spread spores over considerable distances, particularly if the conidia are contained in droplets <20 µm (Ingold 1978). The drops that are most effective in splash dispersal of conidia are those of heavy showers and others dripping from foliage (Ingold 1978). Zachos *et al.* (1963) observed that when rainfall was not accompanied by wind, the disease spread in circles, but that in the presence of wind-driven rain, the disease spread in the direction of the wind. Sattar (1933b) and Luthra *et al.* (1935) observed that infected tissues from diseased plants could be blown in the wind for hundreds of meters and lodge among healthy chickpeas where they acted as a source of infection in wet weather. Since conidia are produced in enormous quantities, it is evident why blight spreads so rapidly in a field once it gets started and as long as wet weather prevails. Rapid spread of blight over large areas was described by Luthra and Bedi (1932) and Sattar (1933b) in Pakistan over 50 years ago and occurred in many commercial chickpea fields of northern Idaho in June and July, 1987 (Kaiser and Muehlbauer 1988).

Ascospores

Pseudothecia contain two-celled, hyaline ascospores that develop in asci, which protrude

through the opening of the pseudothecium during wet weather and forcibly discharge the ascospores into the air (Kaiser 1987). At 15 to 25°C, over 70% of the ascospores were discharged from mature pseudothecia within 2 hours of wetting the infected chickpea debris (Trapero-Casas and Kaiser 1987a) (Fig. 4). Ascospores are carried by wind and may be blown some distance. Ascospores, therefore, spread the fungus faster and over greater distances than conidia. Although ascospores are designed for dissemination by air currents, they depend on free moisture for discharge from the pseudothecia and for infection of the chickpea foliage. In the Palouse region of eastern Washington and northern Idaho, pseudothecia usually mature in the spring during the vegetative stage of chickpea. The crop may be subject to a shower of ascospores that effects a blighting of the field within a few days. The author has observed this phenomenon on several occasions in northern Idaho in 1985-87, where chickpea fields are separated by several kilometers (W.J. Kaiser, unpublished data). Infection in a field new to chickpeas and planted with clean seed may occur from ascospores liberated by pseudothecia-bearing chickpea debris that has remained on the soil surface of a neighboring field cropped to chickpeas the preceding season.

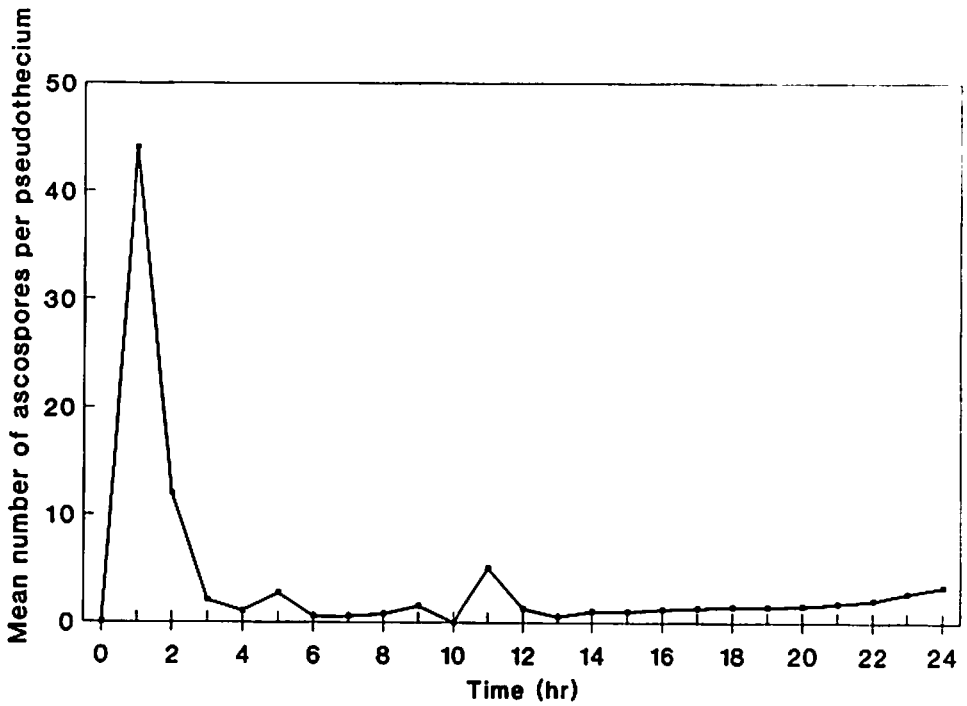

Fig. 4. Discharge of ascospores of *Mycosphaerella rabiei* from naturally infected chickpea debris over a 24-hour period. Debris was wetted at the start of the experiment. Rewetting after drying started another discharge.

Host Range

The host range of *A. rabiei* usually is thought to be restricted to the genus *Cicer* (Gorlenko and Bushkova 1958; Khachatryan 1962; Sprague 1930). Zachos *et al.* (1963) were unsuccessful in their attempts to infect lentil (*Lens culinaris* Medik.), pea (*Pisum sativum* L.) and vetch (*Vicia* sp.) with *A. rabiei*. Sprague failed to infect several plant species, including pea, lentil and bean (*Phaseolus vulgaris* L.). In Iran, Kaiser (1973) inoculated different locally grown food legumes with an Iranian isolate of *A. rabiei*. Cowpea (*Vigna unguiculata* Walp.) and bean were the only plant species infected, other than chickpea. Small reddish-brown spots formed on the inoculated leaves, petioles and stems of cowpea and leaves of bean. The lesions did not increase in size and no pycnidia formed in the lesions. The fungus could be re-isolated from the lesions on cowpea and bean and it was pathogenic to chickpea. At ICARDA in Aleppo, Syria, *A. rabiei* infected cowpea, bean and pea in greenhouse inoculation tests and the fungus was re-isolated from these hosts (Nene and Reddy 1987).

In eastern Washington and northern Idaho, USA, the author (unpublished data) made numerous isolations in May and June, 1988 from different plant species growing in fields where chickpea debris from the 1987 blight-infected chickpea crop remained on the soil surface. The chickpea refuse was heavily covered with pseudothecia of *M. rabiei*. The blight fungus was isolated from the foliage of nine plant species, including alfalfa (*Medicago sativa* L.), henbit (*Lamium amplexicaule* L.) and pea. Some of the plant species from which *A. rabiei* was isolated are listed in Table 2. Studies continue on the role that these naturally infected alternate hosts might play in the survival and spread of the blight fungus and on acquiring information about the teleomorph.

Table 2. Isolation of *Ascochyta rabiei* from plant species growing in an ascochyta blight-screening trial at Pullman, Washington, USA, where chickpea debris infected with the blight pathogen was on the soil surface.

		Tissue pieces†	
Plant species	Common name	Total	Infected
Amaranthus albus	Tumble pigweed	16	0
Capsella bursa-pastoris	Shepherdspurse	32	0
Chenopodium album	Common lambsquarters	64	0
Cirsium arvense	Canada thistle	32	0
Lamium amplexicaule	Henbit	48	1
Medicago sativa	Alfalfa	48	4
Pisum sativum	Pea	64	1
Solanum nigrum	Black nightshade	112	1

† Tissue surface sterilized in 0.25% NaOCl for 5 minutes and plated on 2% water agar.

Environmental Conditions

Although much research has been done on certain aspects of ascochyta blight and its control by various means, there is a scarcity of information on the effects of different environmental conditions on the epidemiology of the disease. The environmental conditions that appear to have the greatest influence on disease development and spread are temperature, rainfall and wind.

In Greece, Zachos *et al.* (1963) observed that temperatures usually were favorable for infection of chickpeas during much of the growing season. Rainfall was the critical weather factor. If the crop was planted during late winter or early spring, the disease often developed and spread rapidly. If chickpeas were seeded at a later date and there was little or no rainfall, blight was of no consequence. Zachos *et al.* (1963) recognized the importance of wind, particularly during rain, in the spread of inoculum, as disease spread was in the direction of the wind.

In the USSR, the disease was favored by frequent rainfall during warm weather (Lukashevich 1958a). Blight often developed later in the growing season with the onset of wet weather (Nemlienko and Lukashevich 1957). Askerov (1968) found that temperatures of 20 to 25°C were favorable for infection. Khachatryan (1962) observed that an average daily temperature of not lower than 15°C with a relative humidity of over 60% and rainfall of 350 to 400 mm during the summer favored the incidence and spread of the disease.

Excellent epidemiological studies on ascochyta blight were done more than 50 years ago by Luthra, Sattar and Bedi in British India (present-day Pakistan). Luthra *et al.* (1935) and Sattar (1933b) reported that if wet weather occurred during the flowering and fruiting periods (February to April), the crop could be destroyed by blight in a few days. However, if the weather remained dry during this period, disease spread was slow and restricted. They studied the incidence and severity of blight in relation to rainfall in the Attock Tehsil of the Punjab for the period of 1920-1930 during which several blight epidemics occurred. In all years but one in which blight caused severe and widespread damage, rainfall was above average and most fell during February and March. Sattar (1933b) also noted that the incidence of blight in different districts of the Punjab was highly correlated with the amount of rainfall during the flowering and fruiting periods. Blight often was serious in chickpea-growing districts where rainfall during the months of October to April was 15 cm or more. In areas with less than 9 cm of rain, blight occurred sporadically and damage was insignificant. Temperature generally had less of an effect on infection and disease development, except in areas where the mean daily temperatures during March and April were close to the maximum temperature growth of the pathogen. Wind was considered to play an important role in spread of the disease in both wet and dry weather (Luthra *et al.* 1935; Sattar 1933b).

Kausar (1965) noted that severe blight epidemics had occurred in Pakistan since 1915. He studied the influence of winter rainfall during the growing season (October to April) and summer rainfall, which preceded the next chickpea crop (May to September), on the occurrence of blight epidemics from 1906 to 1941 and observed a positive correlation ($r = +0.36$) between high winter rainfall and the high incidence of blight. For 15 years the crop area destroyed by blight was >50% when winter rainfall averaged more than 15 cm. During the 35-year period, >15 cm of rainfall was received in 26 years and the area destroyed by blight was >10% during 27 of 35 years. There appeared to be a negative correlation (although not statistically significant) between high summer rainfall and a low incidence of blight in the winter crop that followed.

Weltzien and Kaack (1984) studied epidemiology of chickpea blight near Aleppo, Syria. In a date-of-planting experiment using a blight-susceptible cultivar and a blight-resistant cultivar, disease was not observed before mid-February. However, beginning in mid-March, blight developed rapidly when weather conditions were favorable. Although a high percentage of plants in both cultivars was infected, disease intensity (blight damage to the foliage) was much higher in the susceptible cultivar. In growth chamber studies, they found that disease intensity increased with longer leaf wetness periods over a wide range of temperatures. Severe disease did not develop when the average temperature was <6°C and leaf wetness periods were fewer than 6/hour. No infection occurred below 6°C or above 30°C. At temperatures between 9 and 24°C and leaf wetness periods of at least 10 hours, disease development was very fast. Under Syrian conditions, Weltzien and Kaack (1984) observed that infected debris was a poor source of fungal inoculum, but that *A. rabiei*-infected seeds were a very efficient source of primary inoculum.

Ketelaer *et al.* (1988) analyzed climatological data from several countries in relation to ascochyta blight. From their data and that of Weltzien and Kaack (1984), they found that there had to be monthly average temperatures of 8°C or higher and a monthly rainfall of 40 cm or more before an epidemic could occur.

In field studies at Genesee, Idaho, USA in 1987 and 1988, severity of blight on chickpea trap plants was correlated with rainfall, particularly that which fell during the seedling to flowering stages (Fig. 5). In the absence of rainfall, the disease was less evident. However, when the average daily temperature increased (weeks 9-11 in Fig. 5), blight severity ratings decreased dramatically, even in the presence of rain. The adverse effects of high temperatures on disease development and spread also were observed in field trials at Pullman, Washington, USA, where plots were sprinkler irrigated to promote disease development when maximum daily temperatures were over 30°C (W.J. Kaiser, unpublished data). Little or no infection of chickpea trap plants occurred during these periods of high temperatures, even though over 15 mm of water was applied to the plots on two successive days in late July 1988.

The air above the chickpea trials at Genesee, Idaho and Pullman, Washington was sampled during the growing season for ascospores of *M. rabiei* with a Kramer-Collins spore trap. Ascospore discharge usually was associated with rainfall and began shortly

after the infected chickpea debris was wetted (W.J. Kaiser, unpublished data). At Genesee in 1987, ascospores still were detected on 15 August after a period of rainfall and cool weather. At Pullman in 1988, the last ascospores were collected on 14 July, after which date the weather turned hot and dry (only 0.5 mm of rain until harvest in early September) (W. J. Kaiser, unpublished data).

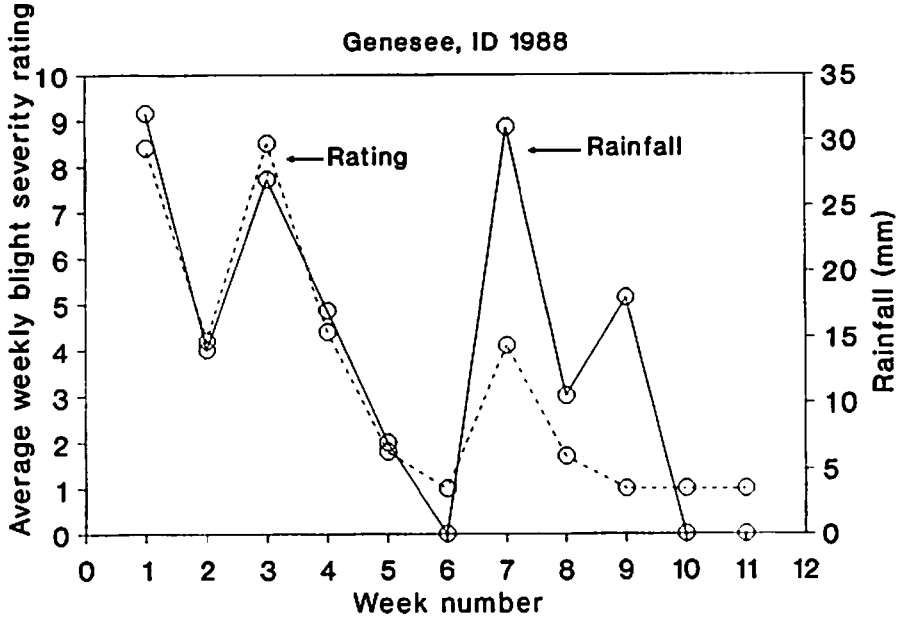

Fig. 5. Effect of rainfall on the ascochyta blight severity rating of 10 chickpea trap plants placed at weekly intervals in a field at Genesee, Idaho, USA. Blight ratings were from 1 to 10, where 1 = no disease and 10 = all plants dead. Week 1 = week beginning with 24 April 1988.

Future Research Requirements

Information on various factors that affect the epidemiology of ascochyta blight of chickpea is incomplete, misleading, or lacking. Some of the information published on the epidemiology of the disease is based on assumptions and not on experimental evidence. Additional research in the laboratory, greenhouse and field is needed if we are to have a better understanding of how and why epidemics occur when and where they do. If and when we have answers to some of these pertinent questions, it might be possible to develop a forecasting system for different blight-prone areas of the world that will enable us to predict when an epidemic is most likely to occur and then to implement appropriate control measures. Research is needed in the following areas:

(i) The teleomorph (sexual stage) has been described from some countries. It is important to establish whether the teleomorph is present and with what regularity and abundance in chickpea-growing countries where ascochyta blight occurs. The author believes that the teleomorph will be found in most countries after a careful search is made. It can be overlooked very easily.

(ii) What role does the teleomorph play in the epidemiology of the disease? How far can ascospores be carried by the wind and still infect a susceptible chickpea crop? Can it be tens or hundreds of kilometers? The pathogen is highly variable in aggressiveness. Is the teleomorph responsible for production of more aggressive isolates of the fungus?

(iii) In countries like India and Pakistan where blight epidemics have occurred periodically for many years, presence of the teleomorph would help to explain how blight epidemics occur every few years. Is it possible that the widespread appearance of the disease in a short period of time over vast areas is due to infection by air-borne ascospores? If not, are infected seeds or chickpea debris the primary source of inoculum that initiates the disease? The appearance of widespread epidemics after a cycle of nonblight years is not explained by existing knowledge. Also, how widely scattered must the primary infection foci be for secondary spread to be so rapid and devastating? If primary inoculum is present at only a few sites, can an epidemic occur over vast areas and, if so, how?

(iv) It is well established that infected seed is important in survival and spread of the blight pathogen. However, not all infected seeds transmit the pathogen to germinating seedlings. What is the minimum number of infected seeds required to initiate an epidemic under favorable environmental conditions?

(v) Improved techniques are needed to detect low levels of infected seed (0.1 to 0.01%) since it may only take a few infected seedlings per hectare to serve as primary inoculum for initiation of an epidemic.

(vi) There are conflicting reports in the literature on the importance of infected chickpea debris as a source of primary inoculum. In areas where it seems to be important (Kaiser and Muehlbauer 1988), the infected debris is exposed to a cold, wet period (winter) before the next crop is planted. However, in some countries such as India and Pakistan, the infected debris remains exposed to the hot, wet monsoon that follows harvest and precedes the next crop. Over 50 years ago, Sattar (1933b) reported that the blight pathogen survived more than 2 years in infected chickpea debris in Pakistan and that wind-driven rain and dry winds may disperse this inoculum. Follow-up research on this matter is definitely warranted.

(vii) How important are other species in the spread and survival of the blight pathogen? At least nine plant species were naturally infected by the blight fungus in the Pacific Northwest, USA (W.J. Kaiser, unpublished).

(viii) More research is needed like that of Weltzien and Kaack (1984) to study epiphytotic spread of blight on growing plants in the field and the effect of climatic conditions on infection. Analysis of climatological data (Ketelaer *et al.* 1988) from different chickpea-growing areas should prove useful in assessing the risk of epidemics of *A. rabiei*.

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Variability in *Ascochyta rabiei*

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Abstract

Ascochyta rabiei, the causal agent of ascochyta blight of chickpea (*Cicer arietinum*), shows a large morphologic variability in cultural appearance and the dimensions of pycnidia and conidia. The loss of resistance of C-12/34 reported in 1963 in India first suggested that different races of the fungus could exist. Since then, observations in several countries have provided evidence of a large pathogenic variability of *A. rabiei*. Studies in Syria and Lebanon and subsequently in Italy on isolates of the fungus artificially inoculated sets of chickpea lines under controlled environmental conditions. The results, obtained by somewhat different techniques, in all cases made possible the division of isolates into six groups according to pathogenic behavior. Other research recently completed at ICARDA seems to indicate a variability in aggressiveness rather than in virulence.

International cooperation will contribute to the understanding of pathogenic variability of *A. rabiei* through comparison of germplasm reaction in different geographic areas and through several cooperative projects. The standardization of methods should be a major concern of future cooperation.

The genetics of the host-pathogen interactions is not well known. Further research is needed to provide a better understanding of this aspect, which is of paramount importance to define strategies to be used in breeding programs for disease resistance.

Introduction

Ascochyta blight is one of the most important diseases of chickpea (*Cicer arietinum* L.) in the major growing areas of the world. It has been reported in a continuous belt from Portugal across most Mediterranean countries eastward to South Asia, in North America and in scattered areas elsewhere, including Australia (CMI 1986; Kaiser and Muehlbauer 1988; Nene and Reddy 1987). Within the Mediterranean region, ascochyta blight is the main factor hindering the shift from spring to winter sowing, despite the potential yield increase expected with the latter. The use of resistant cultivars, taking into account important economic, ecologic and epidemiologic aspects, is thought to be the most promising way to control disease. A knowledge of the pathogenic variability of the fungus is a prerequisite for any sound breeding program for resistance.

The Pathogen and the Disease

The causal agent of the disease, *Ascochyta rabiei* (Pass.) Labr., perfect state *Mycosphaerella rabiei* (Pass.) Kovach., overwinters in plant debris at the soil surface or in the first soil layers, as well as on the seeds. It attacks all aerial parts of the plant, causing leaf lesions conducive to more or less severe defoliations. However, the most important damage is caused to stem and branches. The girdling lesions often observed on these organs cause wilting of the upper part and breakages. Damping-off of seedlings can be observed in early infections, mostly from infected seeds. Pod infection is frequent under suitable climatic conditions, resulting in infection or contamination of seed. The seed also could be contaminated during threshing. The pathogen has been detected in several seed samples (Haware *et al.* 1986) and it is assumed to have been introduced into North America by this means (Morall and McKenzie 1974). Pycnidia, often in concentric rings, appear on the blight lesions. They can produce large amounts of conidia, which are easily dispersed by the independent or joint action of water and wind.

The perfect state, first recorded by Kovachevski (1936) in Bulgaria on overwintered chickpea straw, was observed in the Soviet Union (Gorlenko and Bushkova 1958), Hungary (Kovics *et al.* 1986), Greece (Zachos *et al.* 1963), Syria (Haware 1987b), Spain (Jimenez-Diaz *et al.* 1987) and the USA (Kaiser and Hannan 1987). Its occurrence seems to be widespread. It would be of interest to know whether it is related to the increased research on chickpea pathology or to changes in the population of the fungus. *Mycosphaerella rabiei* appears to be heterothallic, thus one possible explanation for this sudden occurrence could be the diffusion of compatible mating types in regions where they were not previously present. Temperatures between 9 and 24°C and wetness periods of 10 hours or more resulted in the best conditions for disease development in a controlled environment (Weltzien and Kaack 1984). Detailed information on the epidemiology of ascochyta blight is reported in the paper by Kaiser elsewhere in these proceedings.

Host Range

Until recently the host range of *A. rabiei* under field conditions was considered restricted to the genus *Cicer*. Kaiser (this volume) reports the isolation of *A. rabiei* from the leaves of nine plant species, including *Medicago sativa* L., *Lamium amplexicaule* L. and *Solanum nigrum* L. These results cast a new light on the capability of the blight fungus to colonize several species and to survive on them. Further investigations on the possible role of other hosts in the epidemiology of the disease are needed.

The pathogenicity of *A. rabiei* on species outside the genus *Cicer* under artificial conditions was already known. In fact, Kaiser (1973) obtained mild symptoms on cowpea (*Vigna sinensis* Endl.) and bean (*Phaseolus vulgaris* L.). At ICARDA, Syria, infection was obtained on cowpea, bean and pea (*Pisum sativum* L.) in the greenhouse (Nene and

Reddy 1987). As well as *Cicer arietinum* L., germplasm accessions of wild species were tested for their reaction to the blight agent. Differences between species and among different accessions of the same species were observed (Singh *et al.* 1981). These investigations were continued on more accessions both at ICARDA and in Italy. The first results confirm the different reaction of accessions within the same species (K.B. Singh, ICARDA, Syria and P. Crino, ENEA, Italy, pers. comm.).

Variability of the Fungus

Morphologic Variability

Ascochyta rabiei shows a large morphologic variability on culture media for rate of growth, colony appearance, size of pycnidia, sporulation capability and conidial dimensions (Grewal 1984; Kaiser 1973, Porta-Puglia *et al.* 1987). No positive evidence of correlation between morphology and pathogenicity seems to exist.

Pathogenic Variability

The existence of differences in pathogenic behavior of *A. rabiei* was taken into consideration for the first time when the loss of resistance in C-12/34 was reported in India (Nene and Reddy 1987). Since then, several observations provided evidence of pathogenic variability of the fungus in many countries.

Aujla (1964) artificially inoculated 15 lines with 11 isolates in the field in Punjab, India. One of the isolates attacked all the lines tested except F 8 which, on the contrary, was attacked by all the other isolates. Moreover, these isolates differed from one another in the type of reaction induced on several lines. The author concluded that races could be responsible for breaking down the resistance in lines.

The existence of physiologic races of the fungus was suggested in a later paper by Bedi and Aujla (1969). Pathogenicity studies by Kaiser (1973) on isolates from four countries showed differences in the reaction of different chickpea lines, indicating that there may be different strains of the pathogen. Vir and Grewal (1974) divided 268 isolates into 13 groups on the basis of morphology, then tested one isolate from each group on 14 chickpea lines. The reactions of five lines, evaluated according to a 5-point scale, differentiated the isolates into five groups. Because of minor differences in reaction caused by some of the groups of isolates, the authors concluded that it was appropriate to consider only two races. According to Nene and Reddy (1987), five pathogenic groups and several strains were found respectively by Qureshi in Pakistan and by Nevin in Turkey. Reddy and Kabbabeh (1985) tested 50 isolates of *A. rabiei* by artificial inoculation on a set of six lines of chickpea in a plastic house. They were able to distinguish six races. Crino *et al.* (1985) observed divergent behavior of some chickpea lines in field trials at two Italian locations. For instance, ILC 3279 was rated 1 in Latium, compared with the local check rated 9, while in Sicily both the check and ILC 3279 were rated 4 in a 1 to 9 rating scale. The pathogenic variability of the fungus in Italy was

confirmed in greenhouse trials (Porta-Puglia *et al.* 1985). Further experiments, in which isolates from different Italian chickpea-growing areas were tested on 14 lines and two landraces under controlled environmental conditions, confirmed the wide pathogenic variability of *A. rabiei* (Porta-Puglia *et al.*, 1986, 1987). From these observations Porta-Puglia and Crino (unpublished) selected a set of five lines and one highly susceptible landrace that appeared most suitable to characterize the pathogenic traits of the blight fungus. Artificial inoculation performed on them with 50 Italian isolates permitted the division of the isolates into six groups of similar pathogenic behavior. The reactions of the lines to each group are shown in Table 1.

Table 1. Pathogenic variability of *Ascochyta rabiei* in Italy.

	Pathogenic groups of *A. rabiei*					
Line	1	2	3	4	5	6
Calia	S†	S	S	S	S	S
ICC 5127	R	S	S	S	S	S
ILC 484	R	R	S	S	S	S
ILC 72	R	R	R	S	S	S
ILC 3279	R	R	R	R	S	S
ICC 3996	R	R	R	R	R	S

† S = Susceptible, R = Resistant.

The methods used by different workers to study the variability of *A. rabiei* largely differ from one another. The host lines, inoculum concentration, inoculation technique, environmental conditions and age of the plants were different. Different rating scales were used (Nene 1984; Reddy *et al.* 1984). The methods adopted by Porta-Puglia and Crino (unpublished) and Reddy and Kabbabeh (1985) are compared in Table 2. Some parameters are similar, such as inoculum concentration and plant age at inoculation, as are some differentials. Moreover, despite the different rating scales, both groups of workers attached a basic importance to girdling lesions on stems to discriminate between resistance and susceptibility. Both sets of experiments detected a high frequency (80% in Syria and Lebanon and 30% in Italy) of isolates able to attack all the tested lines.

Recent research at ICARDA on over 100 isolates of *A. rabiei* from Syria indicates a variability in aggressiveness rather than in virulence. Disease intensity within the same isolate line combination could be variable according to the environmental conditions. Therefore, Haware (1987a) considers the differences in host reaction not clear enough to classify the isolates into races.

Table 2. Comparison between methods for studying pathogenic variability of *Ascochyta rabiei*.

	Reddy and Kabbabeh (1985) ICARDA	Porta-Puglia and Crino (unpublished) PPRI and ENEA
Medium for isolation and conidial production	Chickpea seed meal dextrose agar	Potato dextrose agar
Inoculum concentration	1.2×10^5	1.8×10^5
Chickpea varieties	ILC 1929, F 8, ICC 1903, ILC 249, ILC 3279, ICC 3996	Calia, ICC 5127, ILC 484, ILC 72, ILC 3279, ICC 3996
Plant age at inoculation (days)	15	15
Days of high RH after inoculation	5	3
Temperature (°C)	15-20	19-25
Rating, days after inoculation	30	12-14
Disease rating	Resistant/susceptible	0-5 point scale
Criterion for discriminating between resistant and susceptible	40% breakage of branches	2.5 average (3 = girdling stem lesions)

Whether or not all these results indicate that the different pathogenic variants of *A. rabiei* are races provides grounds for intriguing discussion. Robinson (1976) writes that "terms having a linguistic meaning can be employed in a system of logic. The less precise their meaning, the less useful they are. Names do not require a linguistic meaning, being only labels. This is why an imprecise word such as race is acceptable as a name (e.g., Race 1234 of *P. infestans*), but not as a term." If we go to the very core of the problem, we must admit that we do not have enough information on the genetics of host-pathogen relationships. Therefore we still are in a phase in which precise 'terms' cannot be used. We are obliged to rely on 'names' for the practical purpose of distinguishing the differences in pathogenicity that we actually observe among the isolates of the fungus. Whichever name we use—race, pathotype, variant or other—we still lack basic information about what is really behind this label. Some information on single dominant or recessive genes governing resistance has been reported (Nene and Reddy 1987; Singh 1987). The experience with other fungal diseases suggests that a gene-for-gene pattern should be expected in most host-pathogen interactions. This pattern is not necessarily obvious, but upon detailed analyses it has been demonstrated in most systems of interactions accurately investigated (Ellingboe 1986). In the case of ascochyta blight, we observe variability in the behavior of individuals of the same line of chickpea when inoculated with the same single-conidium isolate under equal experimental conditions.

Moreover, relatively small differences in temperature and humidity greatly affect the reaction of the host. The latter phenomenon, in the experience of our Italian working group, seems to involve some lines more than others. These and other observations seem to indicate a rather complex genetic background in the relationship between pathogen (*A. rabiei*) and host (chickpea). Complicating factors such as modifier genes cannot be excluded.

International Cooperation

International cooperation is playing an important role in the improvement of chickpea for disease resistance. The huge ICARDA and ICRISAT germplasm collections and new lines tested in the Chickpea International Ascochyta Blight Nurseries during more than a decade in different environments have supplied invaluable information on pathogenic variability of *A. rabiei*.

Cooperation also includes joint research projects for resistance of chickpea to the main diseases, including ascochyta blight. The research program on chickpea resistance to blight and wilt, involving ICARDA and some Italian institutions—the University of Naples, the Plant Pathology Research Institute (PPRI), the National Committee for Research and Development of Nuclear and Alternative Energy (ENEA) and the Cereal Research Station for Sicily—is a recent example in the field. Within the framework of this program, surveys on variability of populations of *A. rabiei* in Syria and in Italy, including cross-comparison of the results obtained in the two countries, are conducted. The isolates of race 6 from Syria and pathogenic group 6 from Italy were tested last month in Rome. They caused very similar reactions on the host set used by PPRI and ENEA.

As mentioned above, workers dealing with the variability of *A. rabiei* are still using different methods. International cooperation can promote actions for standardization of sets of lines, inoculation methods and environmental conditions during testing. Rating scales also need to be compared and discussed among workers of different countries. In fact, more effort should be given to establishing an objective rating system to be universally accepted. The Linear Infection Index proposed in these proceedings by Harrabi and Halila is an example of the endeavor toward objectivity in disease measurement worthy of submission to comparative tests.

Conclusions

The morphologic and pathogenic variability of *A. rabiei* is large. Although some results have been achieved in the comprehension of the host-pathogen relationship, avenues are still open to investigation. The presence of toxin, dealt with in these proceedings by Strange and Alam, provides another challenging approach. New methods for the identification of pathotypes should be investigated to discover substitutes for the conventional time-consuming techniques.

A better understanding of the genetics both of the fungus and of the basis for plant resistance is a prerequisite for further theoretical and practical progress. The recent advances concerning the requirements of the pathogen for the production of a teleomorph (Trapero-Casas and Kaiser 1987) provide a promising tool for future genetic analysis.

More research is needed to define the extension of the host range of the pathogen. Testing of a large number of accessions of wild *Cicer* species collected by international and national institutions can supply useful information both on *A. rabiei* specialization and on sources of resistance.

In the strategy of breeding programs for resistance, two apparently conflicting facts should be taken into account. The high frequency of race 6, able to severely attack all lines currently used as differentials, was ascertained among the fungus populations in some areas. On the other hand, a satisfying field resistance was constantly demonstrated in the same areas by lines derived from some of the abovementioned lines. A consistent explanation for these phenomena is still lacking. If found, it could greatly contribute to both theoretical knowledge and practical results.

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Role of Host-plant Resistance in Integrated Disease Management

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Abstract

Elements of integrated disease management relevant for resistance breeding are discussed, using the chickpea ascochyta blight, *Ascochyta rabiei*, as the main example. Emphasis is laid on studies of geographic distribution for host and pathogen. Breeding strategies can be improved if ecological factors controlling disease intensity are known. Seed transmission proved to be significant for the onset of epidemics. Maintenance breeding and seed multiplication are tools for improved disease management. Variability in pathogen virulence calls for continued monitoring and experimental research and should be seen within the context of pathotype distribution studies. This will allow the development of breeding strategies with regional relevance and better management of available genetic resources.

Introduction

> "Integrated control is a procedure using all methods economically, ecologically and toxicologically acceptable, to keep harmful organisms below the economical threshold level, with special emphasis on natural limiting factors."

Accepting this definition (IOBC 1981) of integrated control, we have to look at as many as possible of the natural factors influencing the disease process. Understanding them allows us to evaluate them and consider their priority in a disease management system, which may then serve as a strategy for an integrated production system. The role of plant resistance within such a complex system cannot be seen in isolation but must be considered in its interrelationships with all other factors. In the following I will try to use mainly examples from ascochyta blight [*Ascochyta rabiei* (Pass.) Labr.] of chickpea (*Cicer arietinum* L.), but other relevant diseases will be discussed as well. If no other reference is cited, results have been drawn from research manuscripts prepared under the author's guidance (Kaack 1983; Ketelaer 1986; Volk, 1987).

Elements of Integrated Disease Management

Host-plant Distribution

The first factor to be considered by an international agricultural research center is the host-plant distribution. The map developed by Ketelaer for chickpea presents an overview (Fig. 1) without differentiating properly between climatic zones within the individual countries. The map developed by Volk (Fig. 2) for faba bean (*Vicia faba* L.) is clearly more specific and also indicates winter or summer sowing. There is certainly a need for detailed regional or country maps, which would require continuous updating. Solid agrogeography is clearly an important element of any international crop improvement strategy and it is probably often neglected.

Pathogen Distribution

Pathogen occurrence, its frequency and disease intensity are parameters of high significance for control strategies, but often our data base is incomplete. Ketelaer used the information collected by Nene (1980) for his map (Fig. 3). Some countries have host plants but no disease records (compare Figs. 2 and 3). The lack of introduction of the pathogen and/or unsuitability of environmental conditions are two possible reasons for the nonexistence of the disease. These conditions should be well understood to strengthen or avoid plant quarantine within the control strategy. In case of unsuitability of environment, pathogen introduction may well lead to established disease pockets, but no economic threat will result to the crop. In the concept of geophytopathology (Weltzien 1978), three disease intensity areas are commonly differentiated (Table 1).

Table 1. Disease intensity areas in geophytopathology†.

Area	Disease intensity
Main damage areas	Regular epidemics of economic significance
Marginal damage areas	Irregular epidemics of economic significance, depending on annual weather fluctuations
Areas of scattered attack	No epidemics of economic significance

† From Weltzien (1978).

In the map developed by Volk (Fig. 4), the principle of geophytopathology was used. We can clearly recognize the three different areas, although some of them may be debatable and need updating. Changing host resistance or susceptibility as well as pathogen virulence also may change the distribution pattern.

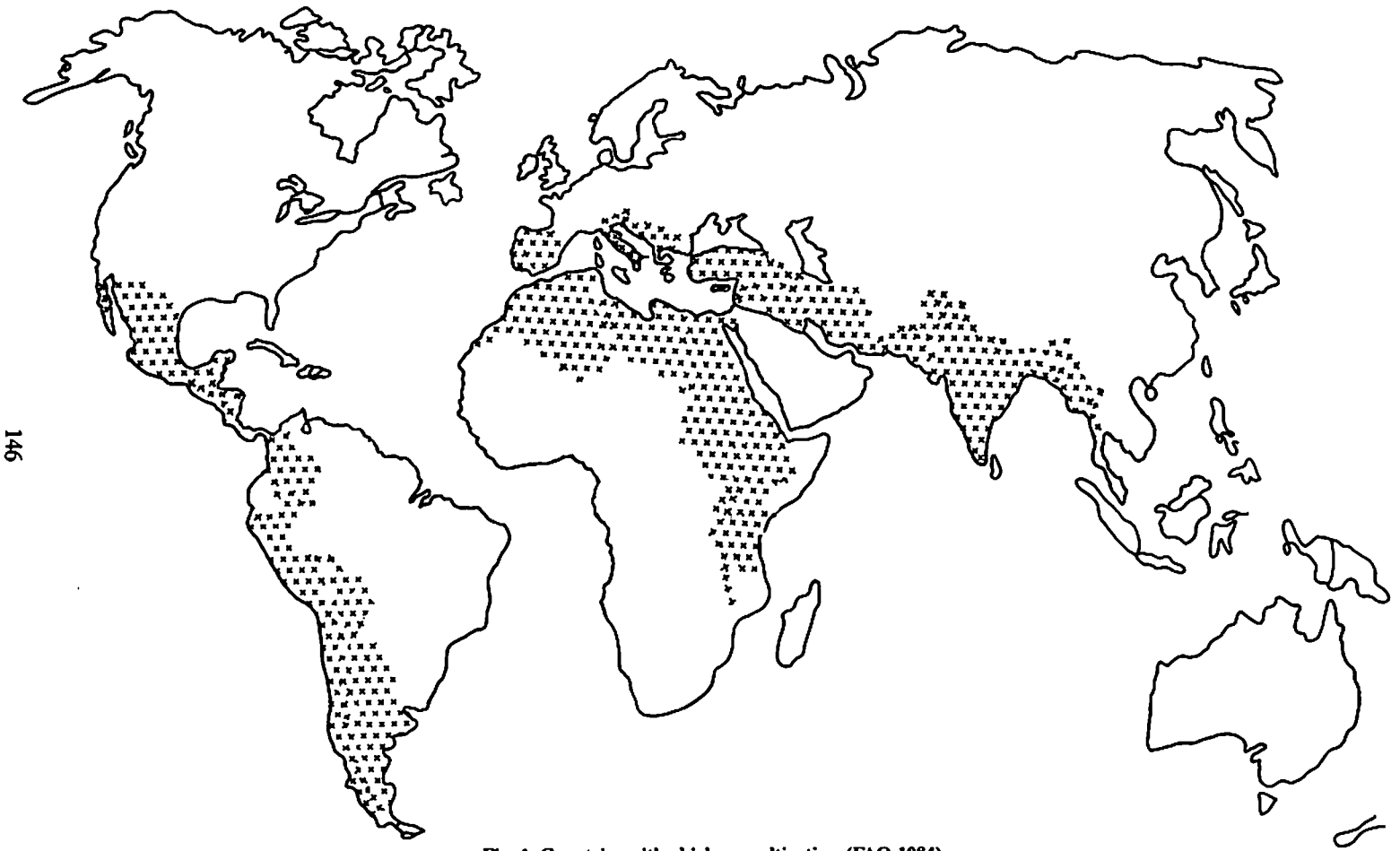

Fig. 1. Countries with chickpea cultivation (FAO 1984).

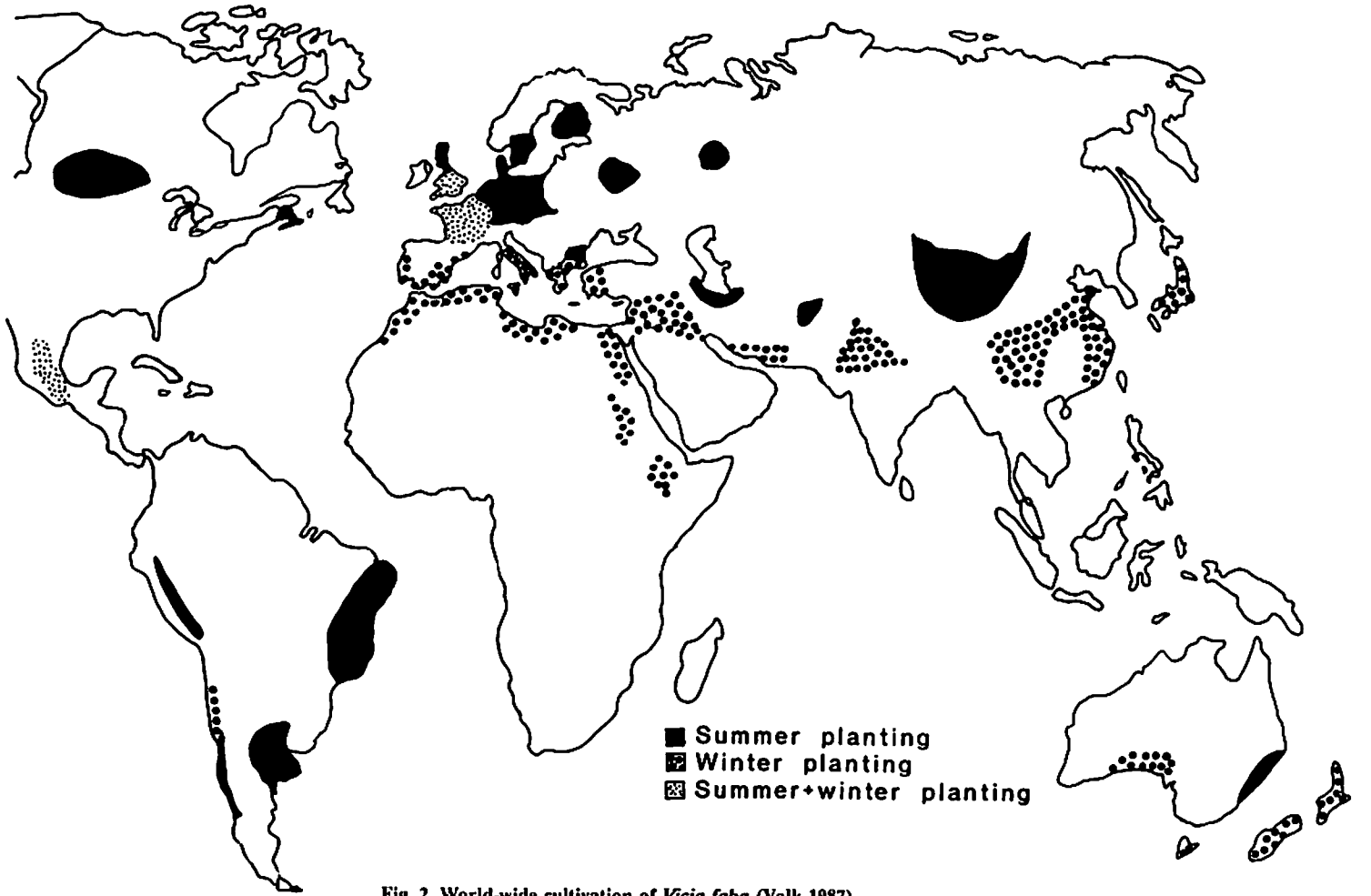

Fig. 2. World-wide cultivation of *Vicia faba* (Volk 1987).

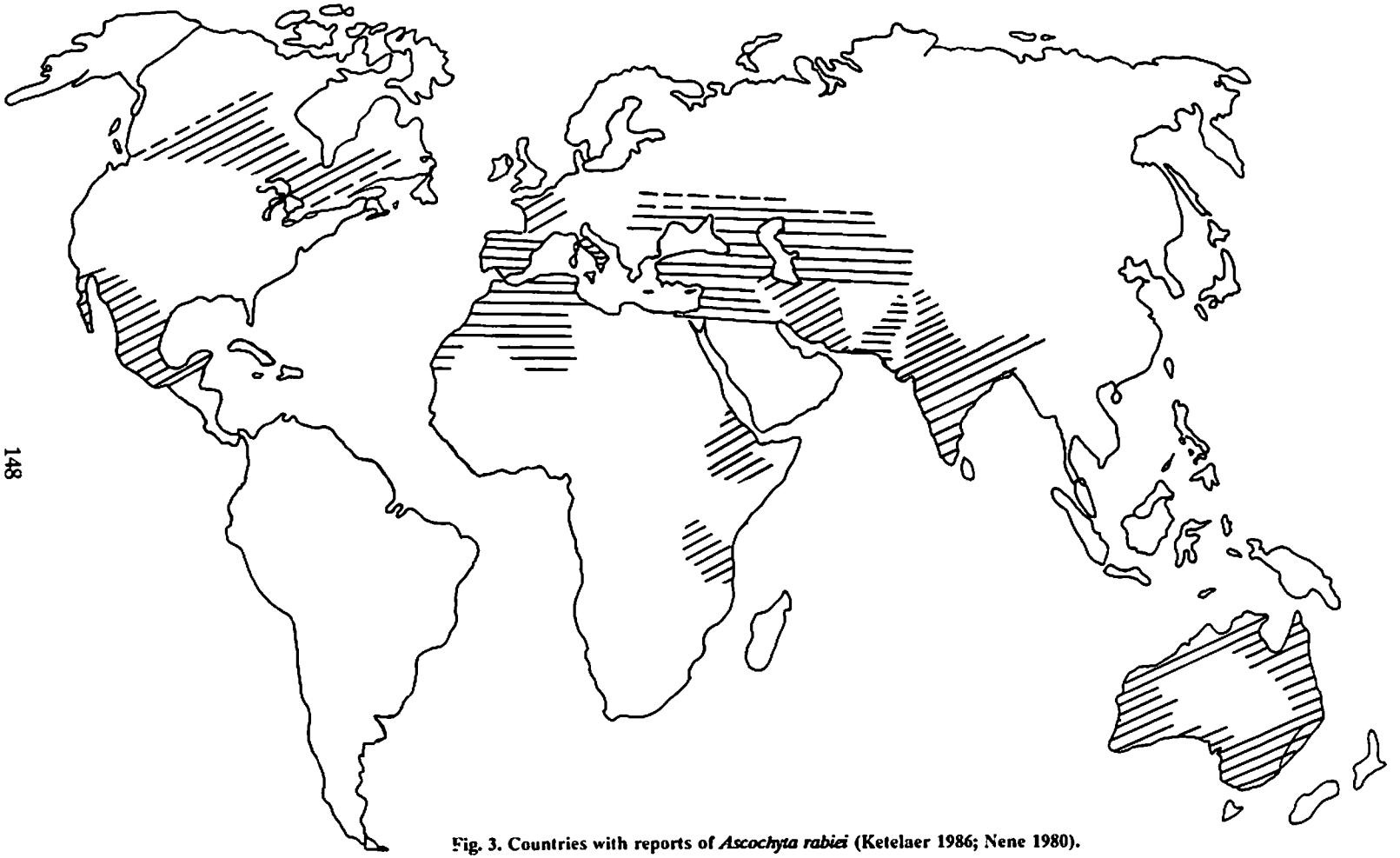

Fig. 3. Countries with reports of *Ascochyta rabiei* (Ketelaer 1986; Nene 1980).

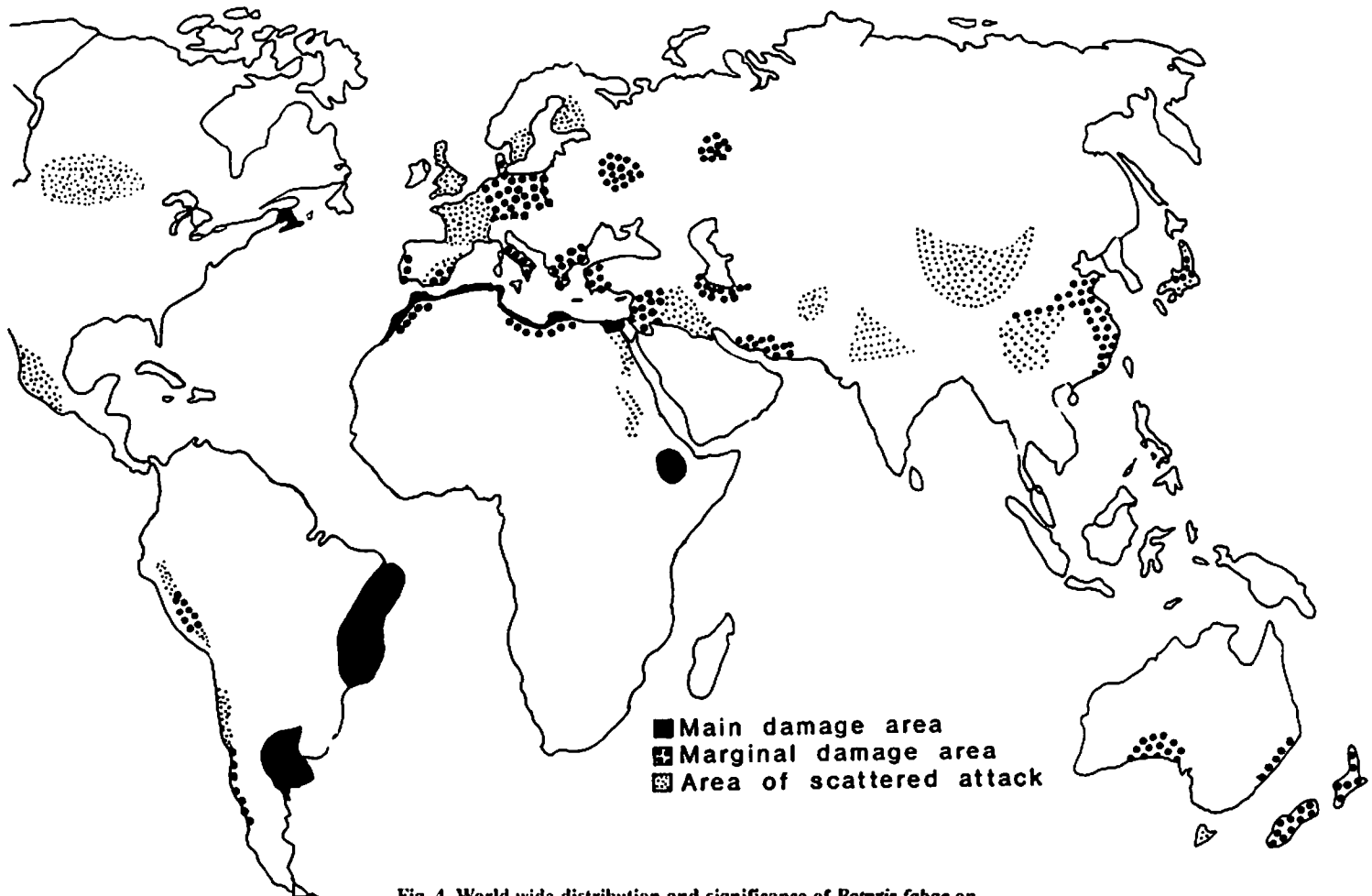

Fig. 4. World-wide distribution and significance of *Botrytis fabae* on *Vicia faba* (Volk 1987).

Forecasting of Pathogen Distribution

If the main environmental parameters determining disease intensity are known and can be correlated with macroclimatic data, the disease intensity areas (Table 1) can be forecast with some probability. The principle is shown in Figure 5 (Weltzien 1978) where actual and potential distribution patterns are indicated. It was successfully applied for a world map of expected or observed disease intensity areas of sugar beet powdery mildew, *Erysiphe betae* (Weltzien 1978). Application of the same principle to forecast distribution patterns of other diseases may prove a useful tool for breeding strategies.

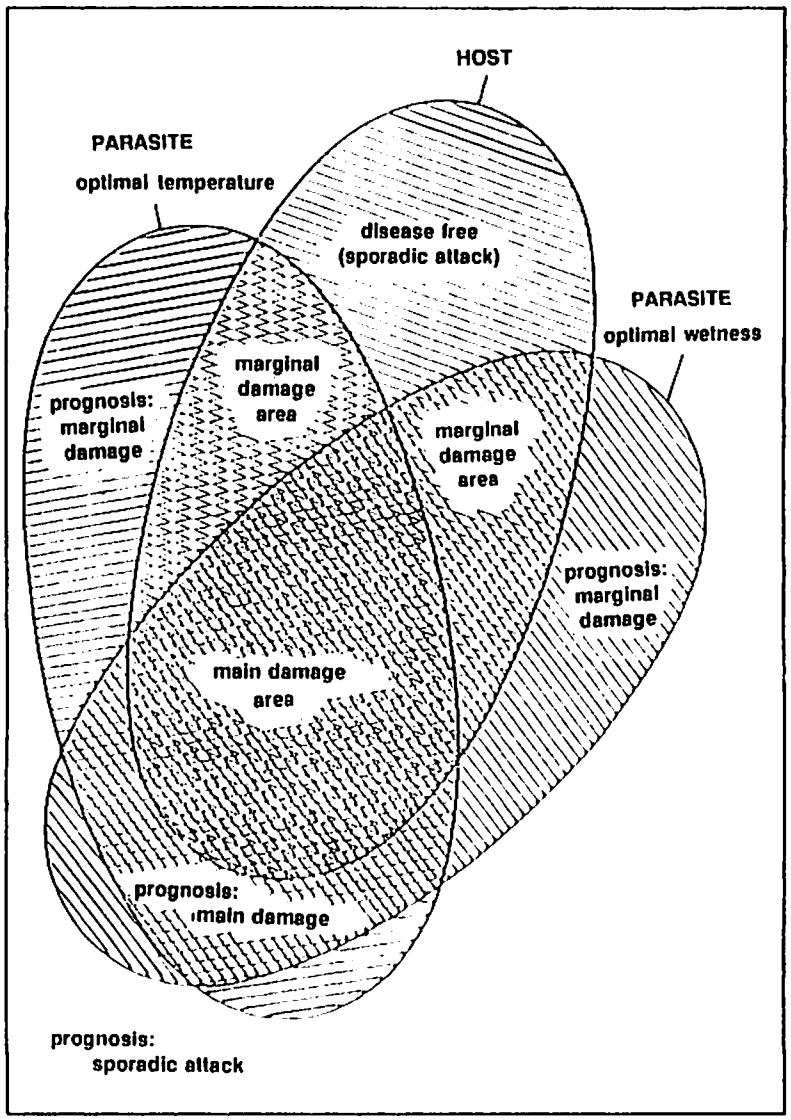

Fig. 5. Model for disease intensity forecasting (Weltzien 1978).

Seed Transmission

International breeding networks must be well aware of pathogen spread through seed transmission. Kaack (1983) tested 52 chickpea seed samples of different origin for *A. rabiei*. Visible and invisible infection had to be distinguished. Table 2 summarizes some results. There seem to be clear differences between regions of origins. While Pakistani seeds were heavily infected, Algerian samples were clean. The Americas, although inhabited by the fungus, are excellent sources for healthy seeds. Experimental proof for the importance of seeds as carriers of *A. rabiei* was obtained by sowing seeds at the end of November. Severe spread of disease from primary infected plants started only after 2 February. Between 14 March and 26 March all plants in the test plots were infected. Maintenance breeding and seed multiplication schemes must try to use disease-free areas or seasons to produce healthy seeds. It is worth exploring, if breeding for lower seed infection rates is possible. Chemical seed treatment can eliminate remaining infections (Table 3). Phytotoxic effects such as delayed emergence may occur, but screening for suitable fungicides should continue.

Table 2. Number of *A. rabiei*-infected seed samples of chickpea from 10 countries and percentage of seeds with visible and invisible infections†.

	Samples examined		Mean % infected seeds	
Country	Total	Infected	Visible	Invisible
Pakistan	22	22	3.3 (12.4-0.4)	12.5
India	1	1	4.5	9.0
Turkey	2	2	3.0	1.2
Greece	5	3	3.1	1.8
Ethiopia	10	2	0.9	1.7
Syria	3	3	2.8	0.5
Chile	2	0	-	-
USA	3	0	-	-
Algeria	3	0	-	-
Iraq	1	0	-	-
Total	52	33		

† From Kaack (1983).

Variability in Pathogen Virulence

Genetic variability of pathogens is a major threat to all efforts in resistance breeding. For *A. rabiei* we have clear evidence that pathotypes exist. Assuming that all plants with symptoms classified at or below class 4 on a 1 to 9 scale (where 1 = free and 9 = plants

killed) were considered resistant, Kaack (1983) developed a matrix for 10 fungus strains vs 11 breeding lines (Table 4).

Table 3. *Ascochyta rabiei* infection of chickpea plants (ILC 1933) 68 days after planting (27 Nov 1980) with and without seed treatment†.

Seed treatment	Infected seeds (%)
Check, healthy seeds	0.3
Check, infected seeds	33.7
Infected seeds + triadimenol + imazalil + fuberidazol	0.0‡
Infected seeds + benomyl + tridemorph	0.3

† From Kaack (1983).
‡ Emergence delayed.

Breeding for resistance can respond to the pathogen variability through recording multiple race resistance and carefully analyzing pathotype distribution. Data collected for *Erysiphe graminis* in Europe after Limpert (1987) may serve as an example. The well-known variability of host susceptibility will not be discussed here, as others will deal with this subject.

Conclusions

After trying to judge the role of host-plant resistance in the integrated disease management for chickpea ascochyta blight we can draw some conclusions.

Studies of the geographic distribution of the host plant provide the necessary basic information for a breeding strategy with world-wide implications. Agroecological factors determine major breeding goals such as the need for *A. rabiei* resistance in main damage areas or the use of susceptible germplasm in areas or seasons without infection pressure. A world-wide prognosis of such areas is possible. The methodologies developed for geophytopathology can be applied.

As *A. rabiei* pathotypes do exist, their distribution must be analyzed and studied to determine if pathotypes are spread within or across regional boundaries. There is an urgent need for more information on genetic variability of existing pathogen populations. These studies should include fitness studies for survival of different pathotypes in natural environments.

Table 4. Different virulence of 10 *Ascochyta rabiei* strains against 11 chickpea lines.

A. rabiei strain	Line ILC										
	191	202	482	248	249	194	210	484	215	618	1929
Eskisehir (Turkey)	R†	S	S	S	S	R	S	S	S	S	S
Haymana (Turkey)	R	R	R	S	S	S	R	S	S	S	S
Tel Hadya (Syria)	R	R	R	S	S	S	R	S	S	S	S
Saida (Algeria)	R	R	R	R	R	S	S	S	R	S	S
Beja (Tunisia)	R	R	R	R	R	R	R	S	S	S	S
Sidi-Bel-Abbes (Algeria)	R	R	R	R	R	R	S	R	S	S	S
Lattakia (Syria)	R	R	R	R	R	R	R	R	R	S	S
Terbol (Lebanon)	R	R	R	R	R	R	R	R	R	S	S
Zair Plateau (Morocco)	R	R	R	R	R	R	R	R	R	S	S
Rabat (Morocco)	R	R	R	R	R	R	R	R	R	S	S

† R = resistant, S = susceptible. R = < class 4 symptoms of ICARDA scale (no stem-penetrating infections) (Kaack 1983).

Seed transmission is a major factor in starting epidemics in the field. Chemical seed treatment is a promising element in integrated disease management and must be explored further. No information on selection of fungicide-resistant pathotypes is available. Laboratory studies on this subject are recommended. They also may prove useful if fungicide applications for disease control become more common. Little information seems to be available on genetic variability of seed infection. Breeding to lower seed infection rates can be a major contribution toward disease control.

Seed multiplication itself must be concentrated in regions and seasons of very low to no infection pressure, even at the expense of lower total yields. Seed certification must include seed health testing, both in the field and after harvest. Strict plant quarantine also should be observed.

Both international centers concerned are encouraged to develop their disease management strategy for *A. rabiei* together with national programs. The full complex of threatening pathogens and pests of each chickpea-growing area must be kept in mind. The ultimate goal is certainly an economically and ecologically sustainable production system, which has to be area specific. I have no doubt that this can be achieved.

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Importance of Chickpea Soil-borne Diseases in the Mediterranean Basin

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Abstract

Diseases induced by soil-borne fungi are the major constraint to kabuli chickpea (*Cicer arietinum*) cultivation in the Mediterranean basin. However, precise information is lacking on incidence, prevalence and distribution of soil-borne diseases, which occur as a disease complex. Work in Spain indicates that fusarium wilt and root rots are the main components of the complex. Disease surveys would help to understand the etiology, distribution and importance of those complexes affecting chickpea in the Mediterranean basin. Control of fusarium wilt and root rots is achieved best by use of resistant cultivars. In southern Spain, three races of *Fusarium oxysporum* f.sp. *ciceri* (races 0, 5 and 6) have been identified. *Fusarium oxysporum*, *F. eumartii* and *F. solani* are associated with the fusarium wilt and root rot complex. The diversity and pathogenic variability of agents involved in the disease complex require improvement of multiple disease-resistant kabuli chickpea cultivars. Breeding for multiple-disease resistance should consider the different genetic systems that may regulate reaction to different diseases. Partial disease resistance could be complemented by other control measures such as fungicide seed treatments and modifications of cultural practices in the integrated disease management programs. However, research on epidemiology of diseases is needed to develop such programs.

Introduction

Chickpea (*Cicer arietinum* L.) can be affected by more than 50 diseases. Some diseases induced by soil-borne fungi are among the most important internationally (Nene and Reddy 1987). Most research on these soil-borne diseases has been on the desi type chickpea (small, angular and colored seeds) by plant pathologists in the Indian subcontinent and at ICRISAT in India.

In the Mediterranean basin, the kabuli type chickpea (large, ram-head-shaped and pale-cream colored seeds) is grown as a spring-sown crop. The Mediterranean climate (hot, dry summers alternating with cool, wet winters) exposes the crop to abiotic stresses that limit its productivity (Saxena 1988). This type of environment includes soil-borne diseases, which are of considerable importance to the chickpea crop (Cubero 1975; Haware 1990; Saxena 1988). However, limited research was done on soil-borne diseases

of chickpea in the Mediterranean basin until recently (Cabrera de la Colina *et al.* 1985; Halila *et al.* 1984; Trapero-Casas and Jimenez-Diaz 1981, 1985).

Several diseases induced by soil-borne fungi have been reported from countries in the Mediterranean basin. These include black collar and root rot, dry root rot, fusarium wilt, rhizoctonia damping-off, rot necrosis, sclerotinia stem rot and verticillium wilt (Halila and Harrabi 1987, 1990; Haware 1990; Maataoui and Hassani 1984; Reddy 1983; Reddy *et al.* 1980; Trapero-Casas and Jimenez-Diaz 1981, 1985). In most cases, the information was obtained from limited disease surveys in farmers' fields. Thus, precise quantitative data are not available on disease incidence and prevalence, or on yield losses caused by them in the region (Haware 1988).

Because information is limited, we do not intend to review the subject in detail. Rather, we present an overview of the research that we have completed on soil-borne diseases of kabuli chickpea in southern Spain during the past 10 years.

The Disease Complex

The Seca or Fusariosis disease, reportedly associated with *Fusarium* sp. (Benlloch 1942), was the main problem affecting chickpea in southwest Spain until 1975 (Cubero 1975). Systematic disease surveys conducted during the 1979-81 period and the research conducted on the etiology of the Seca disease in southern Spain indicated that a disease complex occurred with a range of overlapping symptoms (Trapero-Casas and Jimenez-Diaz 1981, 1985).

Symptoms in the Field

Symptoms included in the complex were: (i) vascular wilt or yellowing induced by *Fusarium oxysporum* Schlecht. emend. Snyd. & Hans. f.sp. *ciceri* (Padwick) Snyd. & Hans., (ii) foliar yellowing and black collar and root rot induced either by *F. eumartii* Carpenter or *F. solani* (Mart.) Sacc., (iii) foliar yellowing and cortical collar and root necrosis induced by nonvascular *F. oxysporum*, (iv) dry collar and root rot induced by *Macrophomina phaseolina* (Tassi) Goid., (v) yellow stunt (BLRV?) and (vi) iron deficiency chlorosis. These different symptoms were discernible in early stages of development but became difficult to distinguish in later stages.

Wilt symptoms occasionally were observed on 20-day-old plants, but they were most conspicuous at the onset of flowering, 50 days after sowing during the spring season. Individual leaves showed flaccidity followed by a dull-green discoloration, desiccation and plant death. Necrotic leaflets remained attached to the petioles. Vascular and pith tissues were colored dark-brown and roots showed no external symptoms.

Foliar yellowing was most conspicuous from onset of flowering. Yellowing gradually progressed up the plant and necrotic leaflets fell off. Affected plants had either

dark-brown vascular and pith tissues, black collar and root rot, or a cortical collar and root necrosis. Dry collar and root rot occurred most frequently at the seed development stage. Stems and leaves dried up and became straw colored and roots and collars became dry and brittle. Affected plants produced pods that were either empty or had small, poor-quality seed.

Prevalence, Incidence and Severity of Symptoms

One hundred and eight fields were inspected in the 3 years of surveys and all but one of them were affected by at least one symptom. In 55 and 21 fields, respectively, two or three symptoms were present in the same field (Table 1).

Table 1. Prevalence, incidence and severity of symptoms associated with chickpea in southern Spain†.

		Number of fields						
			Incidence‡					
Symptom	Year	Sampled	Affected	Trace to 4%	5-20%	21-50%	>50%	Severity range¶
Foliar yellowing	1979	40	35	4	6	0	25	2-5
	1980	30	29	1	0	0	28	2-5
	1981	38	22	0	0	0	22	2-5
Total		108	86	5	6	0	75	
Severe wilt and yellowing	1979	40	12	10	2	0	0	4-5
	1980	30	28	13	10	5	0	4-5
	1981	38	14	7	2	0	5	4-5
Total		108	54	30	14	5	5	
Dry collar and root rot	1979	40	3	3	0	0	0	4-5
	1980	30	3	3	0	0	0	4-5
	1981	38	26	0	3	5	18	4-5
Total		108	32	6	3	5	18	
Yellow stunt	1979	40	16	13	3	0	0	4-5
	1980	30	4	3	1	0	0	4-5
	1981	38	6	5	1	0	0	4-5
Total		108	26	21	5	0	0	
Chlorosis	1979	40	5	4	1	0	0	2-4
	1980	30	4	4	0	0	0	2-4
	1981	38	0	0	0	0	-	
Total		108	9	8	1	0	0	

† Fields were randomly chosen along predetermined itineraries in Cordoba and Sevilla provinces.
‡ Based on counts of plants with symptoms in 10-m lengths of rows for at least three sites per field.
¶ Based on a 1-5 scale according to percentage of foliage with yellowing or necrosis in an acropetal progression (1 = 0%), 2 = 33%, 3 = 66%, 4 = 100%, 5 = dead plant).

Foliar yellowing occurred in about 80% of all fields sampled, of which over 87% had a disease incidence (DI) higher than 50%, and 60% had an average disease severity lower than 3 within a range 2 to 5. In fields with a higher average severity, foliar yellowing was associated with other symptoms, particularly fusarium wilt.

Severe wilt and yellowing, with most affected plants dying or dead, occurred in 50% of the 108 fields sampled. The DI was lower than 5% in 56% of the affected fields and ranged from 5 to 20% in 26% of them. Dry collar and root rot were most prevalent in 1981, when severe drought and high temperatures prevailed. That year about 68% of the fields were affected and 69% of them had a DI higher than 50%. In 6 of 70 fields sampled in 1979 and 1980, DI was lower than 5%. Stunt virus and iron deficiency chlorosis were the least prevalent symptoms.

Although we did not make measurements of yield losses in fields affected by the Seca disease complex, we estimated an annual loss of 12 to 15% based upon incidence and severity of attacks in the surveyed fields. That loss amounts to 8 to 10 million US dollars in Spain. We concluded that fusarium wilt was the most prevalent and damaging disease in the complex and that black collar and root rot were second in importance. Fusarium wilt is also a major disease of chickpea in Tunisia (Halila *et al.* 1984; Halila and Harrabi 1990) and it has been observed in Algeria, Morocco and Syria (Haware 1990).

Neither black collar and root rot nor dry root rot have been reported from other countries in the Mediterranean basin. However, root rot associated with *Fusarium* sp. and *R. solani*, respectively, have been found in Tunisia (Reddy *et al.* 1980) and Morocco (Maataoui and Hassani 1984). In our disease surveys, we observed neither verticillium wilt nor sclerotinia stem rot. Verticillium wilt, induced by *Verticillium albo-atrum* Reinke & Berth., is a major disease of chickpea in Tunisia where it is spreading (Halila and Harrabi 1987, 1990). Similarly, attacks by *Sclerotinia sclerotiorum* (Lib.) de Bary have been observed in Syria and Tunisia and the pathogen is widespread in chickpea crops in Algeria and Morocco (Haware 1990; Reddy *et al.* 1980).

Control of Fusarium Wilt and Root Rots

Most workers agree that control of fusarium wilt and root rots is achieved best by the use of resistant cultivars (Haware and Nene 1980; Nene and Haware 1980; Nene and Reddy 1987). Control of these diseases by means of crop rotation is not possible because of the ability of the pathogens both to survive in soil for a long time and to infect other hosts or nonhosts (Cabrera de la Colina *et al.* 1987; Haware and Nene 1982a; Trapero-Casas and Jimenez-Diaz 1985).

Progress in improving resistance to a single disease may be influenced by factors such as the genetics of resistance in the host and the pathogenic variability in the pathogen. Despite this, significant chickpea improvement has been made in breeding for resistance or tolerance to a number of single diseases (Singh 1988). However, germplasm

with resistance to a single disease has shown a highly susceptible reaction to other important diseases in a number of cases. Because of that and the complex nature of the disease situation that the crop has to face as illustrated by our studies (Trapero-Casas and Jimenez-Diaz 1981, 1985), strategies such as breeding for multiple disease resistance and/or integrated management should be considered to control fusarium wilt and root rots.

Pathogenic Variability in *F. oxysporum* f.sp. *ciceri*

Haware and Nene (1982b) were the first to identify pathogenic races of *F. oxysporum* f.sp. *ciceri*, namely races 1, 2, 3 and 4, based on differential interactions among 10 chickpea lines and isolates of the pathogen from India. Recent work indicates that resistance to race 1 is conferred by recessive alleles from at least two independent loci (Singh *et al.* 1987; Upadhyaya *et al.* 1983a, 1983b). Lines WR 315, CPS 1, BG 212 and JG 74 carrying recessive alleles at both loci are resistant, while lines C 104 and K 850, which are homozygous recessive at one of the loci, show a delayed wilting. Line JG 62, which is susceptible to all four pathogenic races in India, carries neither of the recessive alleles and shows wilt symptoms within 20 days after sowing.

Early in our research, we found that some isolates of *F. oxysporum* f.sp. *ciceri* from southern Spain are not pathogenic to line JG 62 (Trapero-Casas and Jimenez-Diaz 1981). These isolates induce a vascular yellowing syndrome in local kabuli cultivars, which is characterized by a progressive foliar yellowing that develops 30 to 40 days after inoculation and death of the plant at a late stage (Trapero-Casas and Jimenez-Diaz 1985). Other isolates of the pathogen from the same region are highly virulent to most chickpea cultivars and induce a vascular wilt syndrome. The wilt syndrome is characterized by severe leaf chlorosis, flaccidity and plant death, which develops 15 to 20 days after inoculation (Trapero-Casas and Jimenez-Diaz 1985). This suggested that pathogenic races of *F. oxysporum* f.sp. *ciceri* might occur in southern Spain.

Further research in our laboratory indicated that of 61 isolates of the pathogen, 55 (90%) induce the vascular yellowing syndrome and 6 (10%) induce the vascular wilt syndrome. When the 6 wilt-inducing isolates and 11 selected yellowing-inducing isolates were single-spored and together with races 1, 2, 3 and 4 from India were inoculated to race-differential cultivars, three new races of the pathogen—races 0, 5 and 6—were identified (Cabrera de la Colina *et al.* 1985; Jimenez-Diaz *et al.* 1989b). All three appear to differ from the description of races 1, 2, 3 and 4 in India (Table 2).

Race 0 was proposed to designate all 11 yellowing-inducing isolates that are not pathogenic to desi cultivar JG 62 (Cabrera de la Colina *et al.* 1985), which is susceptible to all other races of the pathogen (Table 2). Race 0 is mildly to highly virulent to kabuli lines C 104, PV 24, P 2245 and to desi line 12-071/10054.

Race 5 was proposed to designate five wilt-inducing isolates that are pathogenic to desi lines CPS 1 and JG 74 and not pathogenic to desi lines BG 212 (Cabrera de la

Colina *et al.* 1985) (Table 2). Lines CPS 1 and JG 74 are highly resistant to race 2 (Haware and Nene 1982b) (Table 2). Furthermore, race 5 is highly virulent to all kabuli lines inoculated but not pathogenic to desi 12-071/10054 (Table 2). Isolates of race 5 induce vascular wilt to all susceptible cultivars except CPS 1 and JG 74. In these cultivars, symptoms consisted of a delayed foliar yellowing that is distinct from that induced by race 0.

Table 2. Disease reaction of race-differential chickpea cultivars to races of *Fusarium oxysporum* f.sp. *ciceri*†.

	Cultivar										
Race	12-071 10054	JG 62	C 104	JG 74	CPS 1	BG 212	WR 315	ICCV 2	ICCV 4	PV 24	P 2245
Spain											
0	S‡	R	M	R	R	R	R	R	R	M	S
5	R	S	S	M	M	R	R	S	S	S	S
6	R	S	M	R	R	R	R	M	M	S	S
India											
1	M	S	M	R	R	R	R	R	R	S	S
2	R	S	S	S	S	S	R	M	S	S	S
3	R	S	S	R	M	M	S	M	S	S	S
4	R	S	S	R	M	M	R	M	S	S	S

† Pooled results of six experiments that shared in common several race-cultivar combinations. Disease reactions for these combinations were consistent over the experiments. Plants were inoculated by the pot-culture method (Nene and Haware 1980).

‡ Assessed on a 0-4 scale according to percentage of foliage with yellowing or necrosis in acropetal progression (0 = 0%, 1 = 1-33%, 2 = 34-66%, 3 = 67-100%, 4 = dead plant) 40-50 days after inoculation. Scores <1 and >3 were considered as resistant (R) and susceptible (S) reactions, respectively. Scores in between were considered as moderately susceptible reaction (M).

Race 6 was proposed to designate a wilt-inducing isolate that is pathogenic to desi line JG 62 and to kabuli lines ICCV 2 and ICCV 4 but not pathogenic to desi lines 12-071/10054, JG 74, CPS 1 and BG 212 (Jimenez-Diaz *et al.* 1989a) (Table 2). Previously in our studies the race 6 isolate had been identified as race 1 because tests were carried out in the absence of lines 12-071/10054, ICCV 2 and ICCV 4 (Cabrera de la Colina *et al.* 1985). Race 6 is distinguished from race 1 by lines 12-071/10054 (susceptible), ICCV 2 and ICCV 4 (resistant, Kumar *et al.* 1985), from race 2 by lines JG 74, CPS 1 and BG 212 (susceptible), from race 3 by lines CPS1, BG 212 and WR 315 (susceptible) and from race 4 by lines CPS 1 and BG 212 (susceptible) (Haware and Nene 1982b) (Table 2).

Recently, Phillips (1988) designated a race 6 in California, based on the disease reaction of the 10 differential cultivars of Haware and Nene (1982b) in infected soils at San Luis Obispo. This race differs from race 1 by the resistant reaction of cultivars Annigeri, Chafa and 850-3/27. We do not know how that race 6 relates to race 6 in southern Spain, since in the absence of reactions of lines 12071-0054, ICCV 2 and ICCV 4 it would be indistinguishable from reaction to race 1 in our experiments. In our laboratory, results of artificial inoculations with isolates of *F. oxysporum* f.sp. *ciceri* from San Luis Obispo suggest that race 0 also might exist in California. Those isolates were recovered from samples of infected lines Mexico 2117 and 4012 provided by Dr. I.W. Buddenhagen and they proved not pathogenic to JG 62 but pathogenic to P 2245 and PV 24.

Race 0, the least virulent of the Spanish races, is widespread in southern Spain, whereas races 5 and 6 hitherto have been found only within a restricted area. Isolates of *F. oxysporum* f.sp. *ciceri* nonpathogenic to JG 62 also have been found in Tunisia (Nene and Sheila 1986), which suggests that race 0 might occur in other countries in the Mediterranean basin.

Resistance to *F. oxysporum* f.sp. *ciceri*

Resistance to *F. oxysporum* f.sp. *ciceri* appeared to be restricted mainly to the desi germplasm (Haware *et al.* 1980; Haware and Nene 1980; Haware *et al.* 1981) and much of the effort in breeding for resistance has been on the desi type.

Recently, however, plant breeders have been successful in developing kabuli germplasm with resistance to *F. oxysporum* f.sp. *ciceri*. The earliest example is that of plant breeders at Culiacan, Mexico. They developed wilt-resistant cultivars Surutato 77 and Sonora 80 by transferring genes for resistance from desi lines L 41 and L 1186 to kabuli lines Macarena and Breve Blanco (Singh 1987) by screening for resistance in an infected field plot. However, no indication of race status was given. Fusarium wilt-resistant kabuli chickpeas also have been developed at Hermosillo, Sonora, Mexico, where resistant cultivars Gavilan, Liz and Tubutama have been developed and in Tunisia where resistant kabuli germplasm has been identified (Halila *et al.* 1984). Plant breeders at ICRISAT have developed four short-duration kabuli lines, namely ICCV 2, ICCV 3, ICCV 4 and ICCV 5, carrying resistance to race 1 of the pathogen (Kumar *et al.* 1985).

Our work on screening chickpea germplasm for resistance to *F. oxysporum* f.sp. *ciceri* started in 1979. During 1979-1983, some 600 entries including collections of Spanish landraces as well as germplasm from Mexico, Morocco, Turkey, the USA and disease nurseries from ICARDA, Syria and ICRISAT, India were tested in field plots heavily infected with *F. oxysporum* f.sp. *ciceri*. No resistance was found in kabuli germplasm, which represented more than 80% of the entries, although some small-seeded kabuli showed less susceptibility than large-seeded kabuli. During 1983 and 1985, germplasm from the ascochyta blight nursery of ICARDA was screened for resistance to the pathogen in our infected plot at Santaella with similar results (Tables 3 and 4). All kabuli

germplasm showed a highly susceptible reaction to *F. oxysporum* f.sp. *ciceri*, except for the small-seeded local landrace PV 61. Cultivar JG 62 showed a resistant reaction, which indicates that race 0 of the pathogen was prevalent in the plot at that time.

Table 3. Reaction of some chickpea lines in two fields heavily infected with *Fusarium oxysporum* f.sp. *ciceri* in 1983†.

Line‡	Incidence of dead plants (%) in nursery plots¶	
	Montilla	Santaella
ILC 72	89.3	100.0
ILC 182	95.7	100.0
ILC 191	98.2	100.0
ILC 194	97.8	100.0
ILC 195	93.1	97.4
ILC 200	97.1	100.0
ILC 202	90.2	100.0
ILC 249	96.2	100.0
ILC 482	99.0	100.0
ILC 484	97.2	100.0
ILC 1407	100.0	100.0
ILC 2548	95.3	100.0
ILC 2555	85.4	77.7
ILC 2912	100.0	100.0
ILC 3279	96.9	100.0
P-2245	99.9	100.0
PV-25	76.9	76.4

† Mean of four replications. Stand counts were made 3 weeks after sowing.

‡ ILC lines are kabuli chickpea obtained from ICARDA. P-2245 is a kabuli cultivar (20 g/100 seeds) obtained from ICRISAT. PV-25 is a local kabuli cultivar (56 g/100 seeds) commonly grown in Andalucia.

¶ Total cumulative of dead plants/total cumulative number of plants at stand count 3 months after sowing.

In 1987, a screening trial in an infected field at Santaella showed that valuable fusarium wilt resistance is present in some kabuli germplasm bred for tolerance to *Ascochyta rabiei* and cold at ICARDA (Jimenez-Diaz *et al.* unpublished). Of the 713 lines tested, more than 95% showed a highly susceptible reaction to fusarium wilt (Table 5), with a final incidence of dead plants higher than 85%. However, nine lines (1.3%) showed resistance, of which four (FLIP 85-85C, FLIP 84-43C, FLIP 82-78C and FLIP 84-130C) had an incidence of dead plants between 0 and 8.7% and a 100-seed weight of 27 to 29 g. Nevertheless, we must keep in mind that the efforts by breeders to develop

chickpea cultivars resistant to *F. oxysporum* f.sp. *ciceri* may be curtailed by the occurrence of races of the pathogen. As an example, recent work in our laboratory indicates that cultivar Surutato is resistant to races 0, 5 and 6 but moderately susceptible to race 1. On the contrary, cultivars Gavilan, Liz and Tubutama (kindly provided by Dr. M. Jimenez Leon, Hermosillo, Sonora, Mexico) showed a highly resistant reaction to all four races.

Table 4. Disease reaction of chickpea lines in a field heavily infected with *Fusarium oxysporum* f.sp. *ciceri* at Santaella, Cordoba, Spain, in 1985.

Line	Number of entries	Emergence (range, %)†	Incidence of dead plants (range, %)‡	
			2 mo	3 mo
ILC 72	1	86.0	0.0	76.7
ILC 182	1	88.0	0.0	100.0
ILC 215	1	28.0	0.0	92.9
ILC 482	1	80.0	65.0	100.0
ILC 3279	1	76.0	0.0	100.0
ILC 3856	1	60.0	33.3	100.0
ILC 3868	1	60.0	13.3	100.0
ILC 4421	1	90.0	6.7	100.0
FLIP 81-293C	2	70.0-78.0	2.6-5.7	100.0
FLIP 82-150C	15	46.0-98.0	0.0-2.2	91.4-100.0
FLIP 83-46C	12	52.0-92.0	0.0-25.0	65.1-100.0
JG 62	1	86.0	0.0	15.6
WR 315	1	82.0	0.0	1.1
P 2245	1	62.9	47.4	100.0
PV 25	1	59.1	4.9	80.8
PV 61	1	59.3	0.0	10.0

† Single-row plots 4 m long. Stand counts were made 6 weeks after sowing.

‡ Total cumulative number of dead plants/total cumulative number of plants at stand count 2 and 3 months after sowing.

Fusarium Root Rots

Although root rot of chickpea has been reported to be associated with *Fusarium* spp. from several countries (ICRISAT 1980), *F. solani* appears to be the major species inducing black collar and root rot of chickpea (Grewal *et al.* 1984; Nene and Reddy 1987; Westerlund *et al.* 1974). In southern Spain, a foliar yellowing associated with collar and root necrosis of varying severity is induced either by *F. eumartii* or *F. solani* and occasionally by nonvascular isolates of *F. oxysporum* (Trapero-Casas and Jimenez-Diaz 1985).

Table 5. Reaction of chickpea breeding lines to fusarium wilt in a field infected with *Fusarium oxysporum* f.sp. *ciceri* at Santaella, Cordoba, Spain in 1987.

Breeding line†	No. entries	Entries (%) with incidence of dead plants‡ after 2 months 0-20%	2 months 21-50%	2 months 51-100%	3 months 0-20%	3 months 21-50%	3 months 51-100%
FLIP lines	713	45.4	30.5	24.1	2.2	2.7	95.1
ILC 1929	83	13.2	66.3	20.5	0.0	1.2	98.8
PV 60	82	74.4	21.9	3.7	1.2	0.0	98.8
JG 62	82	65.8	14.6	19.6	7.3	25.6	67.1

† ILC 1929, PV 60 and JG 62 are susceptible cultivars and sown every 10 test entries.

‡ Total cumulative number of dead plants/total number of plants at stand count 2 and 3 months after sowing. Resistant = 0-20% plants dead, moderately resistant = 21-50% plants dead; susceptible = 51-100% plants dead (Haware and Nene 1982b).

Distinction between *F. eumartii* and *F. solani* is not widely accepted by fusarium taxonomists. However, our isolates of *F. eumartii* differ from those of *F. solani* (sensu stricto) in morphology and virulence to chickpea and other legumes. Isolates of both species can infect a number of legumes, but chickpea, broad bean and pea, in that order, are the most susceptible to them. In general, isolates of *F. eumartii* are more virulent than isolates of *F. solani* to chickpea lines. In pot-culture inoculations, isolates of *F. eumartii* induce foliar yellowing associated with extensive collar and root rot necrosis, stunting and plant death by 3 to 4 weeks after inoculation. Under similar conditions, most isolates of *F. solani* induce a delayed yellowing associated with restricted collar and root necrosis and rarely they cause stunting and death by 6 to 8 weeks after inoculation (Cabrera de la Colina 1986; Trapero-Casas and Jimenez Diaz 1985). Nonvascular isolates of *F. oxysporum* are the least virulent of the isolates of *Fusarium* inducing collar and root necrosis.

Desi lines JG 62 and WR 315 were resistant to *F. eumartii* and *F. solani* in pot-culture inoculation experiments. Disease reactions consisted of shallow root necrosis without foliar yellowing. On the contrary, all kabuli lines inoculated were susceptible to both pathogens. Large-seeded kabulis such as PV 24 and PV 25 (>50 g/100-seed weight) were more susceptible than small-seeded kabuli such as P 2245 and ICC 81001 (<30 g/100-seed weight). Lines PV 2139 and PV 39 showed an intermediate reaction.

Integrated Management of Fusarium Wilt and Root Rots

Results to date indicate that large-seeded kabuli cultivars grown in the Mediterranean basin lack resistance to fusarium wilt and root rots. This emphasizes the need to develop

control measures that might complement the available resistance level. Use of fungicide as seed dressings may increase seedling emergence (Shukla *et al.* 1981; Verma and Vyas 1977) and delay the development of epidemics (Jimenez-Diaz and Trapero-Casas 1985). However, those treatments did not provide satisfactory control of the disease complex (Jimenez-Diaz and Trapero-Casas 1985).

Modification of sowing dates that allow early development of the crop with low temperature and high moisture in soil may reduce the incidence of fusarium wilt (Padwick and Bhagawagar 1947; Raheja and Das 1957). In the Mediterranean basin, these conditions are obtained by advancing sowing from spring to fall or early winter, which greatly increases plant growth and yield (Saxena and Singh 1984). Early results (Trapero-Casas and Jimenez-Diaz 1986) and current experiments in Cordoba indicate that advancing sowing to December or early February significantly reduces incidence and severity of fusarium wilt for moderately susceptible kabulis but not for highly susceptible ones.

Saxena and Singh (1984) pointed out the drawbacks of winter sowing in the Mediterranean basin, which include increased severity of ascochyta blight epidemics, damage from cold, broomrape (*Orobanche crenata* L.) attacks and weed infestations. In addition, new soil-borne diseases may appear or those already occurring may increase in severity. Recently, we have observed unusually poor stands, post-emergence damping-off and severe root rots in winter-sown kabulis in southern Spain. Pre-emergence damping-off was associated with infections by *Pythium ultimum* Trow, while post-emergence damping-off and severe root rots were associated with infections by *Phytophthora megasperma* Drechler and *F. solani*. Kabuli chickpeas seem to be highly susceptible to *P. ultimum* and *Phytophthora megasperma* f.sp. *medicaginis* (Brinsmead *et al.* 1985; Kaiser and Hannan 1983). Thus, the occurrence of those diseases might be of importance to winter sowing in the Mediterranean basin and they may compound the difficulty of controlling the disease complex as well as the breeders' task of developing germplasm with multiple resistance.

Conclusions

Diseases induced by soil-borne fungi are one of the major constraints to chickpea production. Limited information from countries in the Mediterranean basin indicates that disease complexes occur, of which fusarium wilt and root rots might be major components. However, disease surveys should be carried out in the Mediterranean countries to understand the etiology, distribution and importance of disease complexes in the region.

Control of fusarium wilt and root rots is achieved best by use of resistant cultivars. Some progress has been made in developing kabuli germplasm resistance to a single disease such as fusarium wilt. However, the existence of races of the pathogen and the

occurrence of other diseases with distinct pathogenesis present an additional difficulty to the task of plant breeders.

The occurrence of disease complexes requires improvement of germplasm with multiple disease resistance. Resistance to races of the fusarium wilt pathogen appears to be oligogenic and the resistance mechanism seems to restrain the pathogen from getting into the vascular tissues of the plant (Jimenez-Diaz *et al.* 1989a). Some desi cultivars resistant to races of *F. oxysporum* f.sp. *ciceri* show resistance to root necrosis-inducing *Fusarium*. This does not necessarily mean that the same genes are involved in resistance to those pathogens. The type of pathogenesis involved in root-rot diseases would suggest that resistance to them might be controlled by a polygene. In such a case, selection for wilt resistance conferred by major genes may erode existing levels of resistance to root rot diseases.

Large-seeded kabulis lack resistance to fusarium wilt and root rots. Therefore, efforts should be made to develop resistant germplasm. In addition, research on the epidemiology of diseases will help to develop alternative control measures. Those measures should be combined with the limited resistance available in the integrated disease management programs.

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Toxins of *Ascochyta rabiei* and their Putative Role in Screening Chickpea for Blight Resistance

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Abstract

An isolate of *Ascochyta rabiei* from Pakistan synthesized two phytotoxic compounds when grown on a liquid medium consisting of Czapek Dox ingredients and an aqueous extract of chickpea seed. The toxins were decalins with a pyrone side-chain which, in one of the compounds, was substituted with an ethanolamine group. The compounds are identical with solanapyrone A and solanapyrone C, two toxins isolated from culture filtrates of *Alternaria solani*, the causal agent of early blight of potato. Solanapyrone A caused 50% death of cells isolated from chickpea leaflets at 4-22 μM, depending on the cultivar, and was about four times as toxic as solanapyrone C.

Isolates differed in their ability to synthesize the compounds. None were detected in 12-day-old cultures of one isolate, whereas cultures of another isolate grown under the same conditions synthesized as much as 95.4 μM solanapyrone A and 19.5 μM solanapyrone C. Three other isolates synthesized intermediate amounts. Toxin was produced if chickpea extract was omitted from the medium, and production varied with incubation time. The cultivar from which the extract was obtained influenced the final titres, suggesting variation among cultivars in a toxin-inducing substance(s).

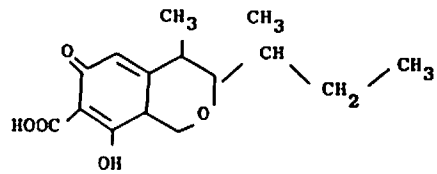
These experiments indicate that production of toxins by *A. rabiei* may be an important virulence or pathogenicity factor and that their synthesis is influenced by a substance(s) from the host. Both phenomena may be susceptible to exploitation in terms of screening for resistance; chickpea germplasm could be selected for toxin insensitivity and also for low concentrations of toxin-inducing substance(s).

Introduction

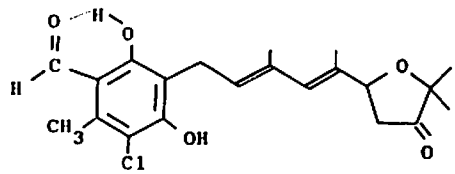
Several species of *Ascochyta* produce toxins. *Ascochyta fabae* and *A. pisi* synthesize ascochitine (Oku and Nakanishi 1963; Bertini 1957) and *Ascochyta viciae* synthesizes ascochlorin (Tamura *et al.* 1968) and ascofuranone (Sasaki *et al.* 1972). Decumbin is produced by *A. imperfecta* (Suzuki *et al.* 1970) and epoxydon and its monoacetate are produced by *A. chrysanthemi* (Fig. 1; Assante *et al.* 1981). Ascochitine has been

implicated as a virulence factor in blight of pea since cultivars that are sensitive to the toxin are susceptible to the fungus and isolates that produce high titres of toxin are more virulent than those that produce low titres (Le Poivre 1982).

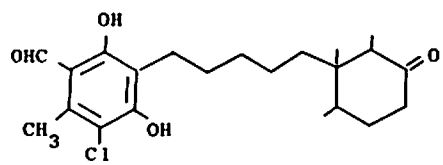
Ascochitine
A. pisi
A. fabae



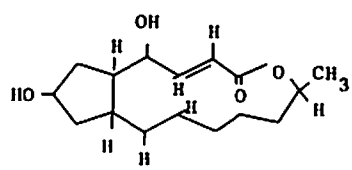
Ascofuranone
A. viciae



Ascochlorin
A. viciae



Decumbin
A. imperfecta



R = H (+)-Epoxydon
R = Ac and its monoacetate
A. chrysanthemi

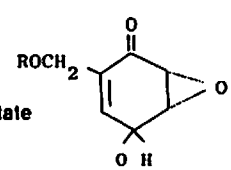


Fig. 1. The toxins identified from species of *Ascochyta*.

Ascochyta rabiei causes symptoms in chickpea that are consistent with toxin production. Lesions are initially water-soaked and infected plants show epinasty. Later, the whole of the aerial part of the plant may dry out, blacken and die. Such symptoms could be explained if the parasite produced a toxin that destroyed the selective permeability of the plasmamembrane. Accordingly, this possibility was investigated using culture filtrates of the fungus and initially monitoring toxin production by a bioassay involving cells from chickpea leaflets isolated by enzyme digestion. Once the characteristics of the toxins were known, they were analyzed quantitatively by means of high performance liquid chromatography (HPLC).

Materials and Methods

Plant and Fungal Material

Chickpea lines and five isolates of *A. rabiei* were kindly provided by the National Agricultural Research Centre, Islamabad, Pakistan. The isolates were single spored and stored in 10% glycerol under liquid nitrogen. When inoculum was required, an ampoule containing 1 ml of spore suspension (10 spores/ml) was used to inoculate about 20 sterile chickpeas in a 100-ml flask. After incubation at 20°C for 7 to 10 days, the contents of the flask were agitated with sterile distilled water and the resulting spore suspension was adjusted to 10/ml (Alam *et al.* 1987).

Toxin Production

The medium for toxin production consisted of Czapek Dox ingredients supplemented with an extract of chickpea seed. The extract was made by boiling chickpea seeds in water for 30 minutes and adding the extract to the other ingredients at a rate of 60 g of extracted seed/L. Normally, line ILC 200 was used for making the seed extract. The medium was distributed among 1-L Roux bottles (100 ml/bottle) and autoclaved. After cooling, each bottle was inoculated with spore suspension of *A. rabiei* (1 ml; 10 spores/ml) and incubated flat side down at 20°C. The cultural filtrate was harvested by straining the contents of the bottles through muslin and centrifuging before finally passing through a glass fiber filter (Whatman GF/A). The product was freeze dried.

Bioassay of the Toxins

Culture filtrates and other toxin preparations were bioassayed with isolated cells from chickpea leaflets. The cells were prepared by digesting leaflets in an enzyme cocktail of Macerozyme R 10 (0.30 g), Cellulase R 10 (2.00 g), pectolyase Y 23 (0.07 g) and BSA (0.05 g) dissolved in holding buffer (100 ml; final pH 5.55). The holding buffer consisted of citric acid monohydrate (10.50 g), glucose (100.00 g), $MgSO_4 \cdot 7H_2O$ (1 mM), K_2HPO_4 (1 mM), $CaCl_2 \cdot 2H_2O$ (5 mM), NaOH (6.20 g) and water (1 L). Leaflets were agitated in a 10-ml beaker by means of a magnetic stirrer set at 60 rpm until disintegration had occurred (15 to 30 minutes). The resulting cell suspension was filtered through two layers of muslin cloth and centrifuged at 85 *g* for approximately 5 minutes. The pelleted cells were washed three times on the centrifuge in holding buffer adjusted to pH 6.1 and made up to 10^5 spores/ml.

Serial two-fold dilutions of toxin preparations in duplicate were placed in wells of a microtest plate (50 µl/well) and cell suspension (105 cells/ml; 50 µl/well) added. After incubation for 3 hours at 25°C, fluorescein diacetate was dispensed to each well (100 µg/ml; 50 µl/well). The microtest plate was viewed under an inverted microscope with epifluorescence optics. Cells with intact plasmamembranes fluoresce a yellow-green color under these conditions whereas dead cells remain unstained. The LD_{50} values were extracted from graphs of probit percent cell death (corrected for control values) plotted against $\log_2$ dilution factor. For convenience the LD_{50} value was termed a unit of activity and the activity of all preparations expressed in these terms.

Toxin preparations also were tested for their effect on chickpea cuttings from 3-week-old seedlings.

Toxin Purification

Cultural filtrates were adjusted to pH 3.0 with HCl and extracted three times with ethyl acetate. The ethyl acetate fractions were combined and dried over anhydrous sodium sulphate and film evaporated at <40°C to a small volume. The concentrated extract was separated by flash chromatography on column of silica gel 60 (400 mesh, E. Merck No. 9385). The column was first eluted with 600 ml hexane/ethyl acetate (3:1) containing 0.1% acetic acid and then 600 ml of a 1:1 mixture of the same solvent also containing 0.1% acetic acid. Fractions (30 ml) were cut and assayed for toxicity to chickpea cells derived from leaflets. Active fractions were checked for purity by thin layer chromatography and HPLC.

Thin Layer Chromatography

Samples were chromatographed on Merck silica gel plates (250 µm) containing a fluorescent indicator and viewed under short wave UV irradiation. Compounds showed as dark spots on fluorescent background. The toxins also were revealed by staining in iodine vapor and by spraying with phosphomolybdic acid (10% in ethanol).

HPLC

The HPLC was manufactured by Philips and consisted of a pump that could be programmed to deliver varying mixtures of up to four solvents, an autojector that could accommodate up to 64 samples, a column (250 x 4.6 mm i.d.) of ODS silica (10 µm) and a diode array detector that could be set to monitor absorption either in the UV (190-390 nm) or the visible (390-590 nm) range. Data were acquired from the detector by a computer and could be displayed in many ways such as a three-dimensional chromascan, a single wavelength chromatogram or a spectrum from a compound eluting after a specific retention time. In addition, the computer could be programmed to measure the concentration of any compound with a UV or visible wavelength chromophore.

Toxin samples for analysis by HPLC were prepared by solid phase extraction. A 1-ml Gilson pipette tip was plugged with glass wool and 400 mg of ODS silica added (10-40 µm). A second plug of glass wool was tamped on top of the ODS silica and the 'mini column' was conditioned by elution with 10 ml methanol and 10 ml water. Culture

filtrates (10 ml) were passed through the column and the column washed with 5 ml water. The toxins were eluted in 1 ml acetonitrile.

Samples (10 µl) of toxin solutions in acetonitrile were chromatographed isocratically on the HPLC in a solvent consisting of acetonitrile/water 1:1 and the eluent was monitored with the instrument set in the UV mode.

Results

When *A. rabiei* was grown on Czapek Dox liquid medium no toxic activity against isolated cells of chickpea was found in the culture filtrates. In contrast, when the medium was supplemented with an aqueous extract of chickpea at the rate of 60 g extracted seed/L, culture filtrates killed the cells. Reduced activity was recorded when the chickpea extract was added at the rate of 120 g extracted seed/L and none when this was reduced to 30 g/L or less. Toxin titres were influenced by the cultivar from which the extract had been obtained and the line from which the isolated cells for the assay were prepared (Table 1).

Toxin activity, fungal mass and pH were maximal in fungal cultures incubated for 12 days (Fig. 2).

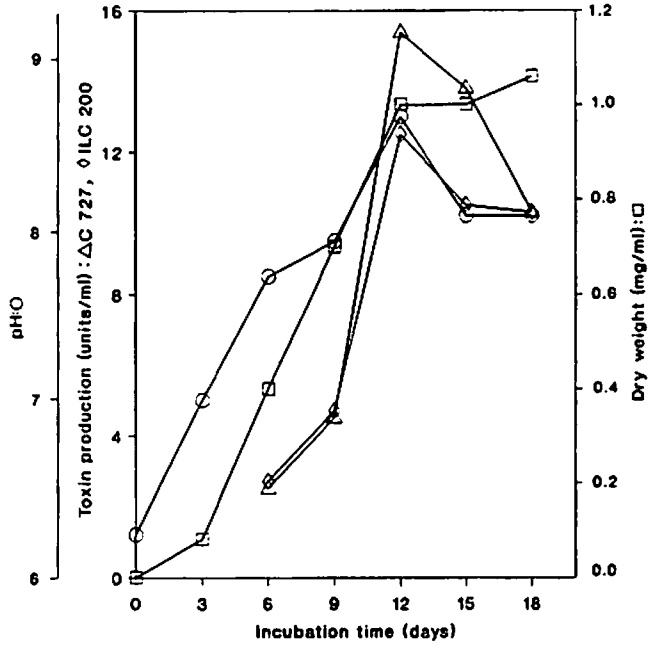


Fig. 2. Toxin production, pH changes and dry weight accumulation of the fungus during incubation on Czapek Dox liquid medium supplemented with chickpea extract. Toxin values are given as units/ml, where 1 unit is the amount of toxin that kills 50% of the isolated cells in the assay.

Table 1. Toxin induction by seed extracts of six lines of chickpea and their effect on toxin titres assayed with cells of these lines.

Extract cultivar	Assay cultivar	Toxin activity (units/ml)†
ILC-200	C-727	119.5 ± 46.1‡
Cypressa		73.5 ± 6.3
Turkey (local)		73.2 ± 1.0
CM-72		21.4 ± 0.5
C-727		23.2 ± 4.4
ILC-202		21.4 ± 0.0
ILC-200	ILC-202	70.5 ± 9.9
Cypressa		56.4 ± 8.5
Turkey (local)		97.5 ± 18.0
CM-72		27.0 ± 3.8
C-727		27.2 ± 4.0
ILC-202		0.0 ± 0.0
ILC-200	CM-72	66.2 ± 26.7
Cypressa		29.6 ± 1.8
Turkey (local)		33.5 ± 5.4
CM-72		24.9 ± 1.6
C-727		Trace
ILC 202		0.0 ± 0.0
ILC-200	Cypressa	21.8 ± 1.5
Cypressa		21.6 ± 0.3
Turkey (local)		22.6 ± 3.3
CM-72		43.8 ± 15.7
C-727		0.0 ± 0.0
ILC-202		0.0 ± 0.0
ILC-200	ILC-200	37.8 ± 1.8
Cypressa		Trace
Turkey (local)		30.7 ± 3.7
CM-72		Trace
C-727		Trace
ILC-202		0.0 ± 0.0
ILC-200	Turkey (local)	56.5 ± 15.5
Cypressa		24.6 ± 3.3
Turkey (local)		28.9 ± 4.4
CM-72		0.0 ± 0.0
C-727		0.0 ± 0.0
ILC-202		0.0 ± 0.0

† Units/ml of a ten-fold concentration of culture filtrate.
‡ ± Standard deviation.

Toxin activity partitioned virtually quantitatively into ethyl acetate (EtOAc) when the pH of the culture filtrate was adjusted to 3. Flash chromatography of the ethyl acetate solution yielded two compounds that were active in the isolated cell assay. When chromatographed on TLC silica gel in 1:1 hexane + EtOAc containing 0.1% acetic acid, toxin 1 and toxin 2 migrated with Rf values of 0.26 and 0.18. They were visualized by observation under short wavelength UV and in white light after exposure to iodine vapor or spraying with phosphomolybdic acid in which they stained yellow and gray, respectively.

On reserve phase HPLC, toxins 1 and 2 had retention times of about 500 and 400 seconds, respectively. The chromoscan mode of the HPLC revealed that each compound had a distinctive UV spectrum (Fig. 3), shown more clearly in Figure 4.

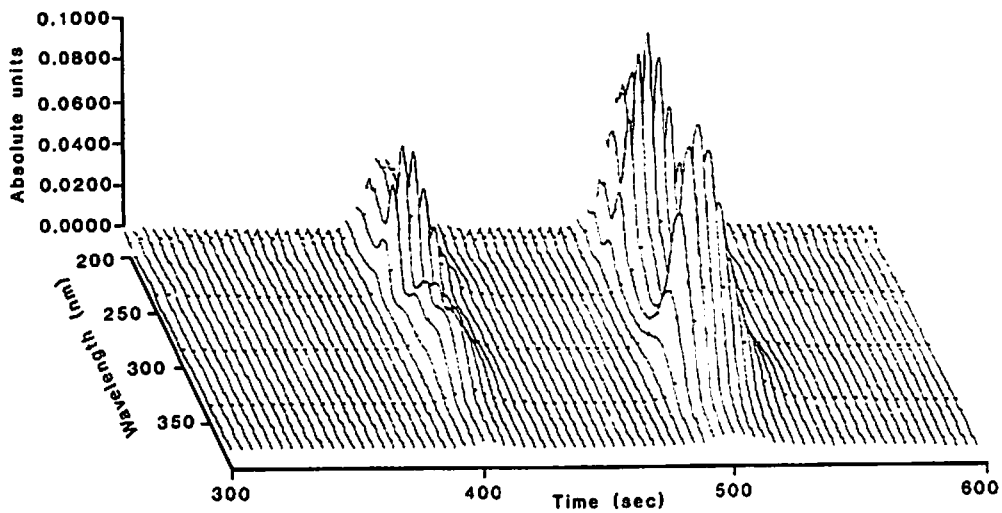


Fig. 3. Chromoscan of the toxins of *A. rabiei*.

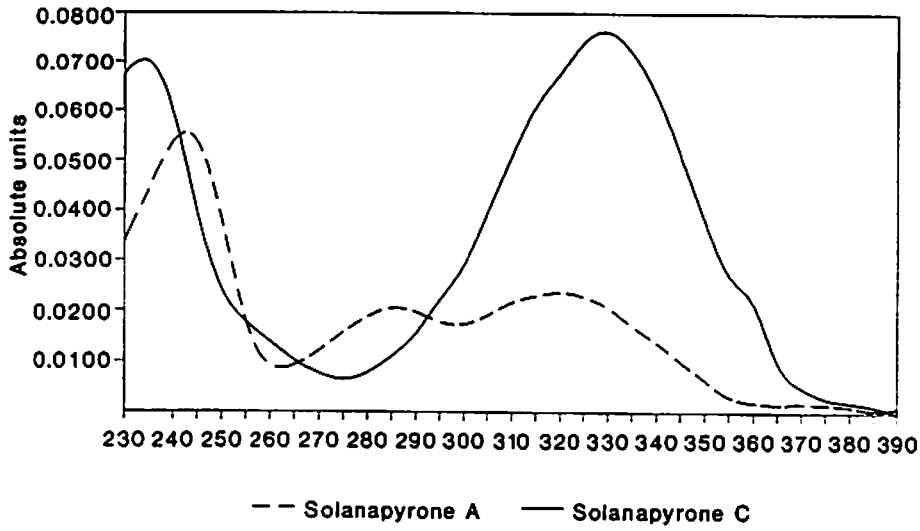


Fig. 4. UV spectra of solanapyrone A and solanapyrone C.

The structure of the compound was elucidated by a combination of mass spectrometry and both proton and ^{13}C NMR spectroscopy. Details of this work will be published elsewhere (Alam 1989). Toxin 1 was identified as solanapyrone A and toxin 2 as solanapyrone C (Fig. 5).

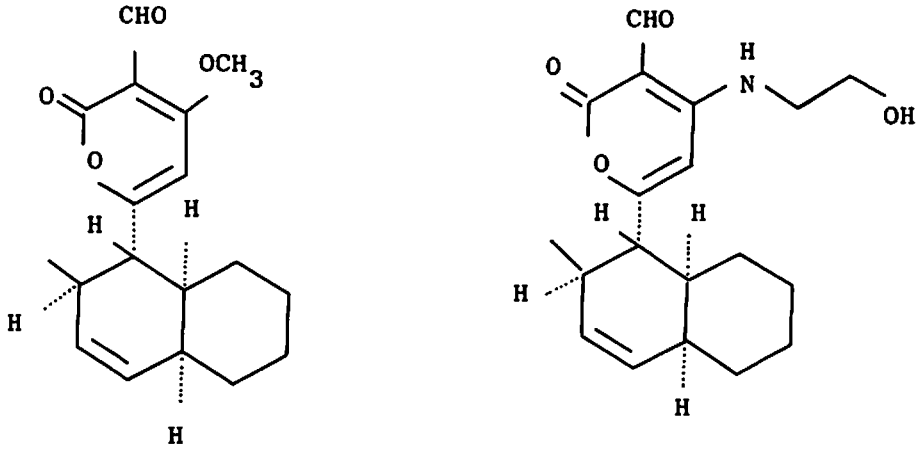


Fig. 5. The structure of solanapyrone A and solanapyrone C.

Five isolates of *A. rabiei* grown in liquid culture for 12 days under the standard conditions described in the methods differed markedly in toxin production. One isolate produced 95.4 μM solanapyrone A and 19.5 μM solanapyrone C but the compounds were not detectable in the culture filtrates of a second isolate. Three further isolates synthesized intermediate amounts (Table 2).

Table 2. Variation in toxin production by six isolates of *A. rabiei*.

Isolates	Replicates 1	2	3	Mean ±SD
		Solanapyrone A		
26	0.0†	0.0	0.0	0.0 ± 0.0
28	63.6	24.9	55.6	48.0 ± 20.4
29	16.3	6.6	8.8	10.6 ± 5.1
39	2.9	0.0	0.0	1.0 ± 1.7
6K	67.7	36.0	95.4	66.4 ± 29.7
		Solanapyrone C		
26	0.0*	0.0	0.0	0.0 ± 0.0
28	12.2	8.3	15.7	12.1 ± 3.7
29	8.0	7.5	7.5	7.7 ± 0.3
39	3.2	0.0	0.0	1.1 ± 1.8
6K	16.1	14.4	19.5	16.7 ± 2.6

† Concentration in μM.

Solanapyrone A was about four times as active as solanapyrone C on a selection of 16 chickpea lines, with concentration ranging from 4 to 22 μM of the more active compound required to kill 50% of the cells (Fig. 6).

Discussion

The finding that the *A. rabiei* toxins were indicated with solanapyrone A and solanapyrone C was unexpected. These two compounds are synthesized by *Alternaria solani* and are toxic to potatoes, in which the fungus causes early blight (Ichihara *et al.* 1983; Materon *et al.* 1978). Preliminary data (not shown) have demonstrated that isolated potato cells are about 80% as sensitive to the compounds as the most sensitive chickpea cultivar (C-727) but that other species including cabbage, lentil, tomato, marrow, pigeonpea, groundnut, pea, broad beans and barley are less sensitive or insensitive at the

maximum concentrations tested. Chickpea cultivars also varied in sensitivity to the toxins (Fig. 5).

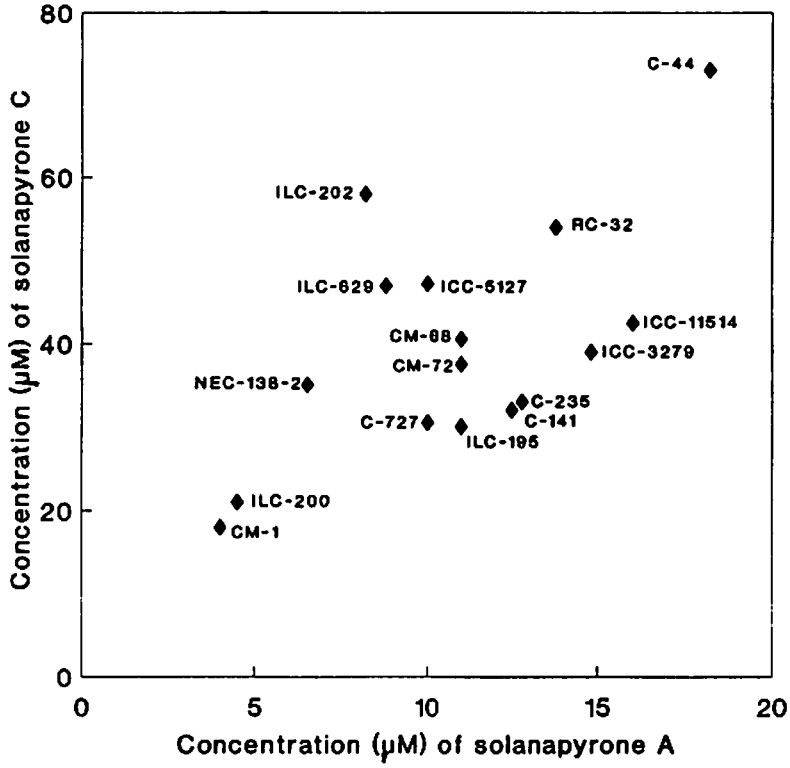


Fig. 6. The activity of solanapyrone A and solanapyrone C tested with 16 lines of chickpea.

The solanapyrones also were toxic to cuttings of chickpea seedlings in the µMolar range, causing chlorosis and epinasty, symptoms characteristic of early infection of the pathogen.

A further noteworthy observation is that chickpea extract is required for toxin production by the fungus *in vitro* and that when the fungus was grown on extracts of various cultivars, toxin titres differed. This suggests that the plant contains a toxin-inducing substance(s).

It is possible that the results described in this paper could be exploited in terms of producing more resistant plants by two methods: plants may be selected for toxin insensitivity and also for reduced concentrations of the toxin-inducing substance(s).

Acknowledgements

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The Use of Fungicides for Disease Management in Chickpea

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Abstract

Some of the important fungal diseases of chickpea are fusarium wilt, various root and stem rots caused by soil-borne fungi, ascochyta blight and botrytis gray mould. In chickpea, diseases are managed through cultural practices, host-plant resistance and application of fungicides. Seed-borne inoculum of pathogens causing fusarium wilt, ascochyta blight and botrytis gray mould can be eradicated by fungicidal seed treatments. Seed treatment in such cases would not only control the disease but also work as a safeguard against inadvertent introduction of the pathogens along with the seed. Most chickpea diseases can be managed by using clean seed from a healthy crop, selecting the proper time and depth of sowing, using a resistant/tolerant cultivar suitable for the area and by treating seed with a suitable fungicide. Additionally, for ascochyta blight and botrytis gray mould, where a good level of resistance is not available, one or at the most two field applications of effective fungicides (chlorothalonil for ascochyta blight and vinclozolin for botrytis gray mould) may be required. Seed treatment with a broad-spectrum protectant fungicide (e.g. thiram, captan, captafol) would improve emergence in general. Efforts must continue to search for more effective fungicides and to devise more efficient application methods.

Introduction

Chickpea (*Cicer arietinum* L.) is an important grain legume crop of dryland agriculture in Asia, Europe, North and Central America, South America and Africa. Although the overall yield potential of the present-day chickpea cultivars is >3000 kg/ha, the average productivity is only about 755 kg/ha. Some of the reasons for low productivity are that chickpea is grown by small farmers on marginal lands, usually on the residual moisture with negligible amounts of inputs. Chickpea is neglected in favor of cereals or other more remunerative crops. Farmers consider chickpea as a more risky crop, mainly owing to the uncertainty of an assured level of production. Diseases are the most important factors responsible for poor and uncertain yields of chickpea.

The general methods of plant disease management are cultural and biological, including host plant resistance and chemical treatments. Maximum emphasis has rightfully been put on exploitation of the host-plant resistance for disease management. The

dynamic nature of the plant pathogens makes breeding for disease resistance a continuing process. The most effective and economical management of a disease can be achieved through a suitable combination of different methods of control. Use of fungicides is popular for disease control in high-value crops and for diseases causing great economic losses. Fungicides as a component of the effective disease management strategy have a place even in a low-input, low-income crop like chickpea under certain situations. Combined use of tolerant cultivars and effective fungicides can result in superior disease control and may prolong the effective life of both the tolerant host and the specific fungicide. Where host resistance fails or where a high level of host resistance is not available, fungicide use may be required for better management of the disease. Fungicide seed treatment, owing to its highly effective and favorable cost-benefit ratio, has great potential for use. There is a need for more research efforts in the area of fungicidal control of chickpea disease, so that more efficient methods of use and effective chemicals become available for use, at least in emergencies. This paper summarizes the available information on the use of fungicides in the management of important fungal diseases of chickpea.

Ascochyta Blight

Ascochyta blight caused by *Ascochyta rabiei* (Pass.) Labr. is a widespread and destructive disease. It has been reported from 31 chickpea-growing countries. Several epiphytotics have been reported from several countries in the recent past. The disease symptoms are produced on all the aboveground parts of the plant (Nene and Reddy 1987).

The primary source of the inoculum is infected seed and diseased crop debris left in the field (Luthra *et al.* 1935). For rapid blight development, temperatures between 9 and 24°C and a leaf wetness period of 10 hours or more are required (Weltzien and Kaack 1984). Cool, wet conditions favor the disease and its spread in the field is very rapid under rainy, windy and cloudy conditions. No infection occurs below 6°C or above 30°C. There would be no blight problem if the average air temperature were ≤6°C and wetness periods were less than 6 hours.

Disease Management

Use of resistant cultivars

Resistance sources both in kabuli and desi germplasm have been identified. Blight-resistant cultivars (like ILC 482 in Syria and C 235, G 543 and G 688 in India) have been released in different chickpea-growing countries (Reddy and Singh 1985; Singh and Kapoor 1986). The fungus is known to have physiologic races, and breakdown of resistance also has been reported in some cultivars.

Cultural practices

Management of inoculum borne in crop debris. The pathogen can survive in diseased crop debris on the soil surface for 2 years but it cannot survive if the infected debris is buried in moist soil (Luthra *et al.* 1935; Reddy 1983).

Crop rotation can eliminate this source of the primary inoculum if dead plant debris is removed or ploughed deep. This eliminates the chances of production of the perfect stage of the pathogen in overwintered crop debris. Viable ascospores can be carried long distances in air currents, which introduces the pathogen to new and distant places (Trapero-Casas and Kaiser 1987). Sexual reproduction could be responsible for the creation of new variability in the pathogen (Kaiser and Muehlbauer 1988).

Management of microclimate. Date of sowing, through its effect on plant growth and canopy development, affects both ascochyta blight incidence and crop yield. In Pantnagar (North India), early sowing (October) resulted in higher disease incidence and hence lower yield. Although growth was slow in late sowings, vegetative growth was restricted so that both disease incidence and yield were low. Mid-November sowings had the lowest disease incidence and highest yields (Tripathi *et al.* 1988). However, in the Mediterranean region, changing the date of sowing from spring to early winter resulted in an ascochyta blight epidemic on susceptible lines (Hawtin and Singh 1984). Intercropping chickpea with crops like wheat, barley and mustard could reduce the spread of disease in the crop season (Luthra *et al.* 1935).

Management of seed-borne inoculum. Seed-borne inoculum is the most important primary source. The pathogen can be present as mycelia and pycnidia in seed (Luthra and Bedi 1932). There also may be surface contamination of seed by fungus spores, which can survive up to 5 months at 25 to 30°C. Pre-threshing storage of the harvested material increases the seed infection. Both superficial and deep infections are equally effective in disease transmission. In infected seeds the pathogen can survive for more than 2 years. Thus, as far as possible, healthy seed from a disease-free crop must be used. Deep sowing (10 to 15 cm) also provides control by not allowing the infected seed to emerge. The seed-borne inoculum could be significantly reduced by sun drying of the chickpea seed. In 15 days, sun drying for 8 hours/day reduced seed infection from 31.5 to 16.0% with no adverse effect on seed germination (Tripathi *et al.* 1987b). Dry heat treatment of seed also is effective in inhibiting the seed-borne inoculum. However, the effect on the inoculum and on seed germination is dependent upon the temperature and duration of treatment. Treatment for 6 hours at 55°C completely eradicated the seed-borne inoculum, but seed germination also was reduced, to less than 50%. Exposure at 45°C reduced the inoculum by 50% and seed germination by 20% (Tripathi *et al.* 1987a). Soaking seed in water for 6 hours at 20°C followed by a 15-minute dip in hot water (53°C) gives good control of the inoculum but seed germination is reduced (Sattar 1933).

Use of fungicide

Seed treatment. Immersion of seeds for 2 hours in malachite green (0.0005%), for 4 hours in formalin or for 12 hours in 150 µg/ml Pimaracin eradicates the seed-borne inoculum. Granosan, phenthiuram, thiram, benomyl and Calixin M have been reported to be effective for seed treatment (Bashir and Ilyas 1983b; Kaiser and Hannan 1985a; Nene and Reddy 1987; Reddy *et al.* 1982). Calixin M (11% tridemorph + 36% maneb) as seed treatment was reported to eradicate the seed-borne inoculum (Reddy *et al.* 1982). The slurry seed treatment at 2.5 g/kg with Calixin M, Calixin M + thiram (1:1), Calixin

M + Bavistin, and Bavistin + thiram (1:3) when tested by the blotter method completely eliminated seed-borne inoculum, but on oatmeal agar 4 to 6% of treated seed yielded the fungus (*A. rabiei*) colonies compared with 30% recovery from untreated seed. However, Calixin M, Calixin M + thiram and Calixin M + Bavistin significantly reduced the seedling vigor. Bavistin + thiram seed treatment was best for eradication of the seed-borne inoculum, significantly increasing the seed germination and seedling vigor (Tripathi *et al.* 1986). Thiabendazole seed treatment (3 g/kg seed) has been reported to be more effective and safer than Calixin M (Reddy and Kabbabeh 1984). Treatment of seed with an effective fungicide would control the disease most economically and allow the free movement of seed internationally without danger of introduction of the pathogen to new areas (Kaiser and Muehlbauer 1988).

Foliar application. Once the disease has appeared in the field, the only option available for disease management is the use of an effective fungicide. Foliar applications of fungicides such as Bordeaux mixture, wettable sulphur, Zineb, ferbam, maneb, Captan, Daconil, Captafol and dithianon have been reported to significantly reduce the disease spread (Bashir and Ilyas 1983a; Bhatti *et al.* 1984; Gaur and Singh 1985; Kaiser and Hannan 1985a; Kaiser *et al.* 1973; Maden 1983; Nene and Reddy 1987; Vir and Grewal 1974). Under favorable weather (environmental) conditions on susceptible cultivars, disease development is very fast and usually fungicidal applications are ineffective. Otherwise, at least four to eight spray applications are required for significant reduction in disease (Bashir *et al.* 1987). Although copper oxychloride, mancozeb and benomyl did not control the disease effectively, they reduced the yield losses appreciably, indicating differential interaction between cultivar and chemical and also between genotype and the pathogen (Singh and Gaur 1988). For the most effective and economical disease control, a combination of host-plant resistance with fungicide sprays is recommended (Hanounik 1980; Reddy and Singh 1983, 1985).

Botrytis Gray Mould

Botrytis gray mould (*Botrytis cinerea* Pers. ex Fr.) of chickpea has been reported from many countries (Nene *et al.* 1984). This disease also has the potential to cause very severe damage under favorable environmental conditions. It caused heavy losses in India from 1979 to 1982 (Grewal and Laha 1983). All aboveground plant parts are affected by the disease, and at times no or only small, shrivelled seed is produced in affected pods.

The fungus has a wide host range. In chickpea the pathogen is seed-borne. The viability of the inoculum in seed is greatly reduced during storage under room temperature (Laha and Grewal 1983). Little work has been done on the epidemiology of the disease, but it is known that moist conditions with moderate temperatures favor disease development. Rains and cloudiness favor rapid spread of the disease.

Disease Management

The relative importance of seed-borne inoculum, compared with the inoculum present in the environment, in the epidemiology of gray mould disease of chickpea is not clearly understood. Use of healthy seed from a disease-free crop may be desirable. A good level of resistance in the world germplasm is not available. As far as possible, an adapted, moderately resistant/tolerant chickpea cultivar may be used. In a blotter test, seed treatment at 2 g/kg seed rate with carbendazim + thiram (Bavistin 25% + thiram 50%, 1:1), vinclozolin, or Carbendazim was reported to completely eradicate the pathogen from the seed (Grewal and Laha 1983). Recently, Singh and Bhan (1986) reported the maximum eradication of only 95% of seed-borne inoculum by seed treatment with triadimefon at 1 g/kg seed rate followed by Dithane M-45 (3 g/kg), triadimenol (1 g/kg), thiabendazole (1 g/kg), iprodione (3 g/kg), thiram (3 g/kg) and carbendazim + thiram (3 g/kg) in the order listed. Foliar sprays with vinclozolin (0.1%), carbendazim + thiram (1:1, 0.1%), or carbendazim alone at 0.2% can be given as soon as conditions become favorable or just as the first symptoms appear (Grewal and Laha 1983).

Fusarium Wilt

Fusarium wilt caused by *Fusarium oxysporum* Schlecht. emend. Snyd. & Hans, f.sp. *ciceri* (Padwick) Snyd. & Hans. is a serious disease (Nene *et al.* 1984), causing about 10% loss in yield in India (Singh and Dahiya 1973).

The fungus is soil-borne and is present internally in seed and in dead plant debris as chlamydospores (Haware *et al.* 1978). The fungus can survive in soil for more than 6 years. Lentil, pigeonpea and pea are symptomless carriers of the fungus. The optimum temperature for fungus and for infection is about 25°C. Alkaline soils favor the wilt incidence.

Disease Management

Host resistance

A high degree of resistance against the wilt disease is available. Some of these materials have resistance against other diseases such as dry root rot, black root rot, botrytis gray mould, ascochyta blight, or sclerotinia blight. Cultivars like Avrodhi, JG 315, BG 2344, H 355 and ICCV 32 are resistant to wilt disease.

Cultural practices

In India, the disease decreased when planting was delayed until the cool weather of October or November compared with September sowing (Dahiya *et al.* 1988; Padwick 1948; Singh and Singh 1984). Deep sowing also reduced the wilt incidence (Dahiya *et al.* 1988). Soil solarization is also effective in reducing the incidence of wilt. Soil amendments with oilseed meal reduced the fungus population in soil and the disease incidence (Zakaria and Lockwood 1980). Because the pathogen survives in soil for more than 6 years, crop rotation may not be very effective in disease control. Intercropping, plant

population, layout and fertilizer application had no effect on wilt incidence (Zote *et al.* 1986). In seeds from plants that wilt after pod formation, seed-borne fungus infection is observed. Hence, roguing of such wilted plants (at least for seed purposes) at harvest would reduce the number of infected seeds. Production and use of inoculum-free seed are important from the point of view of guarding against the chances of introduction of the pathogen to new areas.

Fungicides

Several reports of using chemicals for seed dressing have appeared in the literature. A 5-minute seed soak in 0.1% ceresan wet solution followed by seed treatment with 0.2% thiram or 0.2% PCNB was reported to check the wilt considerably. Bavistin Seed Treatment at a rate of 2.5 g/kg seed was effective in pot experiments (Shukla *et al.* 1981; Verma and Vyas 1977). Seed dressing with Benlate T (30% benomyl + 30% thiram) at a rate of 1.5 g/kg seed has been reported to eradicate the seed-borne inoculum (Haware *et al.* 1978).

Root and Stem Rots

Dry Root Rot

Dry root rot is caused by *Rhizoctonia bataticola* (Taub) Butler (Nene and Reddy 1987). It usually appears late in the season when the crop is flowering or podding and soil moisture is low and temperature is high (30°C). The disease is more severe in deep black vertisols. It is more severe in sandy loam soils than in clay loams (Singh and Mehrotra 1981). The fungus survives as sclerotia in soil (Nene 1980).

Disease management

Several tolerant lines have been identified at ICRISAT Center, India. Some of these lines also have resistance against wilt (e.g., H 355).

Early sowing, use of early maturing lines and irrigation can help in minimizing the disease. Soil amendment with mature crop residue of wheat or oat is reported to significantly reduce the pathogen population and also the infection in pot experiments (Singh and Nema 1987).

Seed treatment with captan, thiram and PCNB at a rate of 2.5 g/kg seed may reduce the pre-emergence damping-off phase. Combined use of host-plant resistance with fungicide seed treatment gave better seedling emergence and delayed the onset of the wilt and root rot complex (Jimenez-Diaz and Trapero-Casas 1985).

Wet Root Rot

Wet root rot is caused by *Rhizoctonia solani* Kühn (Nene *et al.* 1984). Most of the incidence is in the seedling stage when soil moisture content is high, but it may appear any time in irrigated chickpea. The fungus survives as sclerotia and mycelia in colonized

organic matter. Disease appears when temperatures are 18 to 30°C, the soil moisture range is 30 to 80% and the nitrogen levels are high. Disease is more prevalent when chickpea is sown in the field after a rice harvest (Nene 1980).

Disease management

Avoid sowing chickpea under wet soil conditions and in soil with high fertility. Grow local, tolerant cultivars. Treat the seed either with captan, thiram, Benlate or PCNB at 2.5 g/kg seed (Viswakarma and Basuchaudhary 1982).

Black Root Rot

Black root rot is caused by *Fusarium solani* (Mart.) Appel & Wr. (Nene *et al.* 1984). Disease can appear at any stage. Light soils with a high soil moisture content show more disease.

Disease management

Use a resistant/tolerant cultivar (e.g., ICC 32). At ICRISAT, a number of lines resistant to black root rot and fusarium wilt have been identified. Crop rotation may be useful. Application of $ZnSO_4$ at 3 kg/ha + P_2O_5 at 40 kg/ha resulted in the lowest root rot incidence (Gaur and Vaidya 1983). Soil amendments with compost, sawdust or neem cake were effective in pot experiments (Chandrasekaran and Sanmugam 1984). Oilseed meal amendments reduced the *Fusarium* soil population (Zakaria and Lockwood 1980). Seed treatment with thiram + benomyl or thiram + captan protects the seedlings (Cother 1977).

Pythium Rot

Pythium root and seed rot caused by *Pythium ultimum* Trow. is more common in the kabuli type than in the desi type chickpea (Nene and Reddy 1987). Seeds rot between 10 and 25°C. Both pre- and post-emergence damping-off and root rot occur. The pathogen is a soil-borne fungus.

Disease management

Metalaxyl (0.3 g a.i./kg) and captan (3 g a.i./kg) are very effective seed treatment fungicides for preventing seed rot and pre-emergence damping-off. Metalaxyl up to 0.6 g a.i./kg seed is not phytotoxic. Storage of treated seed up to 464 days at 4°C did not reduce the effectiveness of metalaxyl (Kaiser and Hannan 1983, 1985b).

Collar Rot

Collar rot is caused by *Sclerotium rolfsii* Sacc. The disease is seen up to 6 weeks after sowing, especially in wet soils. High soil moisture content, presence of undecomposed organic matter near the soil surface, low pH and temperatures of 28 to 30°C are associated with collar rot incidence. Chickpea following paddy suffers from more collar rot infection.

Sclerotia and colonized organic matter are the primary sources of inoculum (Nene 1980).

Disease management

Clean cultivation that destroys the stubble of the previous crop helps reduce the incidence of collar rot. Seed treatment with Rizolex at 2 g/kg seed resulted in 73% emergence and only 4.1% collar rot incidence in seedlings compared with 23% emergence and 73.9% collar rot in seedlings from untreated seeds. Seed treatments with Agrosan GN or Vitavax-200 at 4 g/kg seed also are effective (Viswakarma and Basuchaudhary 1982).

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Recommendations on Resistance Breeding Strategies

Ascochyta Blight

1. Variability in *Ascochyta rabiei*: Considerable variability exists in the blight pathogen *A. rabiei*. In order to understand the existing variability, standardization of the methodology in relation to single spore isolation (ascospores and pycnidiospores), preservation of isolates, identification of differential varieties, inoculum level, age of plants, incubation, temperature and humidity, duration of incubation and the disease rating scale are needed. Simultaneous monitoring of the variability through trap nurseries in the WANA region, Pakistan and India is recommended. Dr M.C. Saxena is requested to form a committee of three members with a coordinator to work out the details and invite suggestions from ascochyta blight workers before formally instituting the committee.

2. Perfect stage of *A. rabiei*: *Mycosphaerella rabiei* is reported from some countries in the WANA region, Europe, USSR and the USA. A search for its presence is recommended in countries where it has not yet been observed. Detailed guidelines to help in locating the perfect stage may be requested from Dr W.J. Kaiser. Pathologists from NARS may be supported to visit laboratories where work on the perfect stage is in progress. Experts on locating *M. rabiei* may be supported to visit countries where the perfect stage has not yet been reported to help NARS scientists, if required.

3. Chickpea and ascochyta blight distribution maps: A 'chickpea atlas' should be prepared in collaboration with NARS. ICARDA is requested to collect this information and publish it.

4. Plant architecture: It may be desirable to change the chickpea plant architecture to help minimize ascochyta blight development. Basic information on the interactions between ascochyta blight and plant height, stem strength, seed size, leaf size and pod position needs to be generated.

5. Epidemiology: ICRISAT and ICARDA may consider hiring consultants to work on ascochyta blight epidemiology in collaboration with NARS pathologists. Studies on ascospore release and dissemination and their possible role in blight epidemiology, as well as field experiments to study the relationship between relative humidity, temperature and blight development need to be undertaken in Pakistan and India.

6. Inoculation with individual or mixtures of races/isolates: The identification of parents, using individual races/isolates, and inoculation of the breeding materials with a mixture of the most prevalent ones in a region or country is suggested.

7. Seed production should be taken up in blight-free areas to produce inoculum-free seed.

8. Fungicide seed dressings are strongly suggested to reduce primary sources of inoculum and prevent the spread of races/isolates.

9. Disease escape strategies, wherever possible and meaningful, need to be adopted in breeding programs.

10. The work on germplasm enhancement and breeding for horizontal resistance to ascochyta blight needs to be strengthened.

11. Work on multiple-disease resistance needs to be strengthened. The multiple-disease resistance needed by region is:

WANA	ascochyta, cyst nematode, wilt
India and Pakistan	
Northern latitudes	ascochyta, botrytis, wilt, stunt
Southern latitudes	wilt, dry rot, stunt
Ethiopia	wilt, dry rot, stunt.

Soil-borne Diseases

1. It is realized that fusarium wilt is the most important disease of chickpea after ascochyta blight in the WANA region. Fortunately, ICRISAT Center at Hyderabad has contributed in disease resistance and other aspects of chickpea wilt. There is a need to strengthen the work on breeding for resistance to soil-borne diseases in the WANA region.

2. We propose that systematic surveys should be conducted in the WANA region to characterize wilts and root rots in different agroecological zones. To achieve this goal, ICARDA and ICRISAT should conduct frequent training programs in diagnosis of these diseases for scientists working in national programs.

3. Information is needed on distribution of the races of fusarium wilt pathogen in the WANA region. This should be obtained by using common standardized techniques and differential cultivars.

4. More information should be generated on the epidemiology of the soil-borne diseases. This may be achieved through research projects addressed to specific questions by researchers at universities and research institutions.

5. We need to understand the mechanisms of resistance to fusarium wilt and root-rot diseases as well as to increase our understanding of the genetics of resistance.

6. We encourage the use of wilt-sick plots for identification of resistance to the soil-borne pathogens prevalent in different agroecological regions of a country. This will strengthen the breeding for resistance.

7. Control of the soil-borne pathogen is successfully achieved through integrated disease management. This includes chemicals, biological agents, cultural practices and resistant cultivars.

8. We recommend that a "Working Group on Chickpea Soil-Borne Diseases" be formed to (i) plan research work, (ii) standardize methodology, (iii) exchange information, and (iv) participate in ICARDA/ICRISAT training programs.

Other Diseases

1. Extensive surveys for the geographic distribution, relative incidences and importance of various chickpea diseases in different countries should be organized. This information will be of great importance in initiating and/or strengthening the research work on several aspects of these diseases in different areas/institutions. These surveys may be organized with the active participation of the national scientists working on chickpea diseases.

2. Once the disease problems have been identified in different areas, there may be a need to organize training to improve research capabilities in the national programs.

3. The group felt that more information on the following diseases should be obtained:

botrytis gray mould	phoma blight
rust	powdery mildew
colletotrichum blight	nematodes
alternaria blight	stunt and other virus diseases
stemphylium blight	*Orobanche* spp.

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