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).

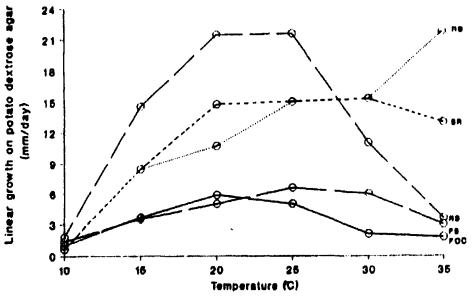


Fig. 1. The effect of temperature on growth of *Fusarium oxysporum* f. sp. *ciceri* (FOC), *F. solani* (FS), *Rhizoctonia bataticola* (RB) and *Sclerotium rolfsii* (SR).

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 $F \geq 4$. 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 1.05×10^8 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 $F_{4:8}$ progeny bulks. Through all generations from F_2 to F_8, 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.

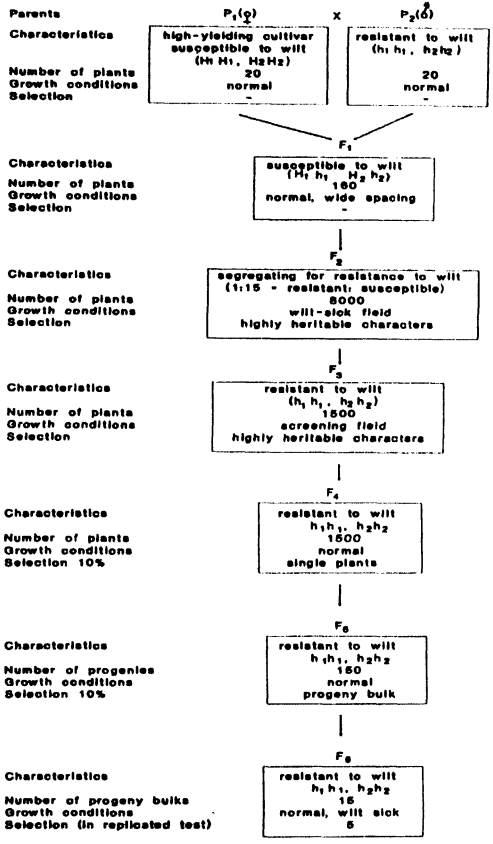


Fig. 2. Bulk-population breeding for the development of fusarium wilt-resistant chickpeas; h1 and h2 are resistance genes for fusarium wilt.

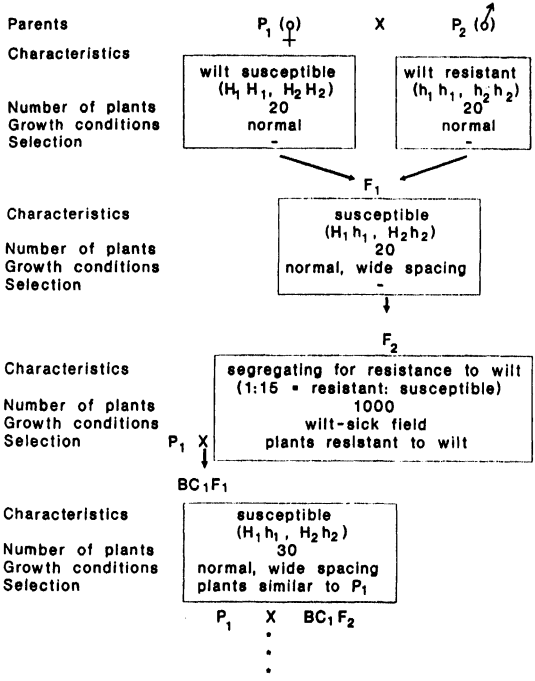


Fig. 3. Backcross-breeding for the development of fusarium wilt-resistant chickpea; h1 and h2 are resistance genes for fusarium wilt.

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.

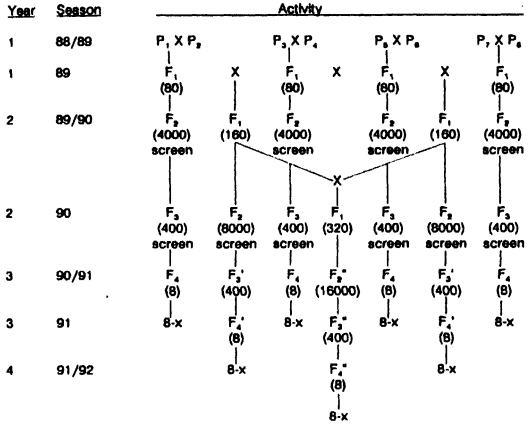
As there is no reason to assume that the resistances so far reported are unstable or not durable, we suggest the following methodology for use, assuming that screening facilities and reported sources of resistance are available.

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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