# Rate-Reducing Resistance to Ascochyta Blight in Chickpeas

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**ABSTRACT**

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In a field experiment conducted at the International Center for Agricultural Research in the Dry Areas in Syria during 1982–83, 1983–84, and 1985–86 crop years, disease progress of Ascochyta blight was studied in chickpea cultivars ILC 464, ILC 482, and ILC 3279. On the basis of apparent infection rate ($r$), ILC 482 ($r$ = 0.16–0.29) and ILC 3279 ($r$ = 0.03–0.23) were identified as cultivars with rate-reducing resistance in comparison with the susceptible cultivar ILC 464 ($r$ = 0.24–0.68). Average grain yield for three seasons was nearly 1,800 kg·ha$^{-1}$ for ILC 482 and ILC 3279 but only 100 kg for ILC 464.

Ascochyta blight (caused by *Ascochyta rabiei* (Pass.) Labrousse)—an important disease of chickpea (*Cicer arietinum* L.) in India, Pakistan, western Asia, North Africa, and southern Europe—assumes epidemic proportions in seasons with rainfall of 150 mm or more (4). Without blight, the rain would enhance chickpea yields substantially. During dry seasons, blight is not severe, but the grain yield is reduced by drought stress in rainfed production. Thus, management of Ascochyta blight is essential for increasing and stabilizing chickpea yields.

In the Mediterranean region, chickpeas are traditionally sown at the beginning of spring (March–April), but the yield of chickpea can be increased by 50% by sowing in winter (early December) provided that Ascochyta blight is controlled (2). Ascochyta blight is severe in the winter season because of cool and wet weather (10).

Management of Ascochyta blight can be achieved with fungicides and resistant cultivars. Several protective fungicides have been identified, but chemical control is neither practical nor economical, because more than six fungicide applications per season are needed to control the disease in susceptible cultivars (8). Development of cultivars with resistance to Ascochyta blight has not been successful due to lack of higher and stable resistance (5,11). With the appearance of new races of the pathogen, there is a continuous need for new disease-resistant cultivars (11). There are several reports of resistant chickpea genotypes, but the type of resistance is unknown (11). While evaluating the world collection of chickpea germ plasm for resistance to Ascochyta blight at the International Center for Agricultural Research in the Dry Areas (ICARDA), we observed a few accessions with low levels of disease during epidemics that killed susceptible cultivars (7).

Because of the variability of *A. rabiei* and ineffectiveness of host resistance, we decided to study disease progress and yield in cultivars with low blight severity. Chickpea cultivars with reduced rates of disease progress may improve the management of Ascochyta blight.

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## MATERIALS AND METHODS

Two ICARDA cultivars (ILC 482 and ILC 3279) with a low final severity of Ascochyta blight and the susceptible cultivar ILC 464 were studied (7). ILC 464 is a highly susceptible, large-seeded (40 g per 100 seeds) kabuli cultivar. ILC 482 is a moderately suceptible kabuli cultivar with medium seed size (30 g per 100 seeds). ILC 3279 is a moderately resistant intermediate-type cultivar with small pea-shaped, orange seed (28 g per 100 seeds). The cultivars were sown in field plots (10 × 100 m each) in the first week of December of 1982, 1983, 1984, and 1985. The experimental design was a randomized block with three replications. Spacing between rows was 30 cm and within rows 10 cm. One month after sowing, plants in the plots were inoculated by scattering infested chickpea debris (7). During the 1982–83 season, the debris collected from susceptible cultivar ILC 1929 was used. In the subsequent three seasons, debris from a mixture of test entries was used. The experimental crops were grown under rainfed conditions. Temperature and relative humidity were recorded using a hygrothermograph (10).

Starting from the onset of symptoms, incidence and severity of Ascochyta blight were recorded at weekly intervals throughout the crop season. The interval between some observations during 1982–83 was longer (19 days), and only eight observations were made. During the 1983–84 and 1985–86 crop seasons, 15 and 9 observations were recorded. For evaluation of incidence and severity, during the 1982–83 season 33 plants were selected randomly for each cultivar once at the beginning of each replication. During the 1983–84 and 1985–86 seasons, 26 plants were selected. Disease severity was recorded on a scale of 1 to 9 (7) taking into consideration the extent of stem breaking, defoliation, and pod infection. The percentage of affected tissues was scored as follows: 1 = no infection, 2 = 1–5%, 3 = 6–10%, 4 = 11–15%, 5 = 16–40%, 6 = 41–50%, 7 = 51–75%, 8 = 76–100%, and 9 = plants killed. First, average disease incidence and severity for each plot were calculated and then the average of each cultivar for three replications.

Disease progress curves were analyzed by a logistic model (1) with the linearized equation $\ln[y/(1-y)] = \ln[y_0/(1-y_0)] + r_L t$, in which $r_L$ is a rate parameter per unit of time, $t$ (apparent infection rate $r$). Disease progress curves for each of the three cultivars for the three seasons were analyzed statistically (Table 1). Weighted mean analyses of variance of intercepts and slopes ($r$) of the disease progress curves were calculated for comparison of the cultivars within each season and over three seasons (Table 2).

## RESULTS AND DISCUSSION

Ascochyta blight was severe in three (1982–83, 1983–84, 1985–86) of the four seasons of studies, as indicated by the high disease severity score for the susceptible cultivar ILC 464 (7.4–8.8, on a 1–9 scale). No other disease was observed in the experimental plots. Because of extremely low temperatures (−10 C) in the 1984–85 trial, the plants died, and data were not analyzed. In the 1982–83, 1983–84, and 1985–86 trials, the onset of the disease was identical for ILC 464 and ILC 482. During the 1982–83 season, blight was observed on 3 March in all three cultivars. During the 1983–84 season, blight was observed in ILC 464 and ILC 482 on 24 January and in ILC 3279 on 31 January. During the 1985–86 season, blight appeared in ILC 464 and ILC 482 on 3 April and in ILC 3279 on 10

April. Except during 1982–83, when final blight incidence differed among the cultivars (ILC 464 = 100%, ILC 482 = 97%, ILC 3279 = 11%), there were no differences in the other two seasons. The weighted mean analysis of variance on intercepts of the disease progress curves showed significant differences between the genotypes, and the interaction between genotype and season was highly significant (Table 2).

Progress of blight in the three seasons differed significantly between cultivars (Table 1). In each season the blight epidemic progressed rapidly in the susceptible cultivar ILC 464 ($r_L$ = 0.69, 0.24, 0.30) compared with ILC 482 ($r_L$ = 0.17, 0.16, 0.23) and ILC 3279 ($r_L$ = 0.01, 0.01, 0.02). The progress was much more rapid in 1982–83 and 1985–86 than in 1983–84. Plants were nearly dead in mid-May (Fig. 1). The 3-yr average yield of the susceptible cultivar ILC 464 was 107 kg·ha$^{-1}$. No grain yield was produced by ILC 464 in 1982–83 or 1983–84.

In ILC 482, the disease progress in all the three seasons was slow, and the final disease severity score (4, on a 1–9 scale) was less than half of ILC 464 (8.3). The $r_L$ for 1985–86 was higher (0.29) than for 1982–83 (0.17) and 1983–84 (0.15). The 3-yr average yield of ILC 482 was 1,742 kg·ha$^{-1}$. In ILC 3279, during the 1982–83 season, the disease severity was almost negligible (1.2) and $r_L$ was very low ($r_L$ = 0.031). During the 1983–84 and 1985–86 seasons, disease severity was greater (2.4–3.9) and apparent infection rate $r_L$ was higher (0.08–0.23) but it was much lower than in ILC 482. ILC 3279 yielded 1,816 kg·ha$^{-1}$ on the average of the 3 yr.

The rate of disease progress for the same cultivar was significantly different in different seasons, and the interaction between cultivar and season was also significant (Table 2). Temperatures that prevailed during different seasons and type of inoculum used in the experiments were responsible for this variation (1). During 1982–83 and 1985–86, the minimum temperature was lower than 5 C until mid-April, whereas during 1984–85 minimum temperature rose above 5 C by mid-March. Because minimum temperatures below 5 C are not compatible with blight infection, disease epidemics were delayed during 1982–83 and 1984–85 (10). During 1983–84, although disease appeared early because of warm night temperatures, the blight epidemic was slowed due to a sudden increase in day temperatures (>20 C) by mid-March.

**Table 1.** Summary of logistic model statistics used in evaluation of Ascochyta blight progress in three chickpea cultivars[a]

| Year<br>Cultivar | Intercept ± SD | Rate parameter ± SD (slope) | $R^2$ (%) | MSE | Residual df |
|---|---|---|---|---|---|
| 1982–83 | | | | | |
| ILC 464 | −2.822 ± 0.432 | 0.6781 ± 0.0855 | 89.8 | 0.3071 | 6 |
| ILC 482 | −2.426 ± 0.0482 | 0.1730 ± 0.00955 | 97.9 | 0.003834 | 6 |
| ILC 3279 | −2.196 ± 0.0361 | 0.0293 ± 0.00927 | 64.2 | 0.001503 | 4 |
| 1983–84 | | | | | |
| ILC 464 | −1.66 ± 0.2660 | 0.2359 ± 0.02900 | 84.5 | 0.2343 | 11 |
| ILC 482 | −1.827 ± 0.1580 | 0.1450 ± 0.01730 | 84.1 | 0.08381 | 12 |
| ILC 3279 | −2.391 ± 0.0408 | 0.0798 ± 0.00445 | 96.4 | 0.005454 | 11 |
| 1985–86 | | | | | |
| ILC 464 | −1.713 ± 0.5270 | 0.3879 ± 0.09360 | 66.9 | 0.5261 | 7 |
| ILC 482 | −2.377 ± 0.52090 | 0.2869 ± 0.03720 | 88.0 | 0.08315 | 7 |
| ILC 3279 | −2.313 ± 0.1810 | 0.2269 ± 0.03180 | 87.7 | 0.06063 | 6 |

[a] Determined as described in Campbell and Madden (1). $R^2$ = Coefficient of determination for agreement between observed and predicted $y$ (incidence or severity of disease); MSE = mean square error. All residuals were OK.

**Table 2.** Weighted analysis of variance on intercepts and slopes of Ascochyta blight disease progress curves in three chickpea cultivars in 1983, 1984, and 1986

| | | Intercept | | Slope | |
|---|---|---|---|---|---|
| Source of variation | df | SS[a] | Prob.[b] | SS | Prob. |
| Year (unadjusted) | 2 | 1.570 | 0.4561 | 54.82 | 0.000 |
| Genotype (adjusted for year) | 2 | 8.906 | 0.0116 | 164.81 | 0.000 |
| Genotype (unadjusted) | 2 | 6.258 | 0.0438 | 175.58 | 0.000 |
| Year (adjusted for genotype) | 2 | 4.219 | 0.1213 | 44.05 | 0.000 |
| Genotype × year | 4 | 27.429 | 0.000 | 41.11 | 0.000 |
| Total | 8 | 37.905 | ... | 260.74 | ... |

[a] SS = The sum of squares is a weighted sum, using as weights the inverse of the variance of the estimates, and approximately follows a chi-square distribution with the indicated degrees of freedom.

[b] Prob. is the probability of obtaining a greater $F$ value.

The lower disease incidence in ILC 3279 during 1982–83 in comparison with the other seasons resulted from the type of infested debris used for inoculation. The diseased debris used in the 1982–83 season was from a single susceptible cultivar, ILC 1929, inoculated with a single isolate (Tel Hadya isolate, later designated as race 3) (6). In the 1983–84 and 1985–86 seasons, the infested debris used for inoculation was from a blight-screening nursery in which several germ plasm accessions and breeding lines were inoculated with a mixture of isolates of *A. rabiei*. The increased blight incidence in ILC 3279 during the 1983–84 and 1985–86 seasons thus could have resulted from the increased frequency of isolates of *A. rabiei* in the inoculum that were able to infect it. The same factor may have been responsible for the increased severity of blight in the later two seasons compared with the first season.

If the time of blight appearance is considered the beginning of the epidemic (13), during all three seasons the epidemic started in ILC 482 at the same time as in susceptible cultivar ILC 464, but it progressed more slowly because of the low infection rate. By mid-May, when the crop reached maturity, the amount of disease in ILC 482 was less than half that in the susceptible line, and consequently yield loss was lower. This result is in agreement with the horizontal resistance concept of Vanderplank (13), in which horizontal resistance does not delay the start of an epidemic but only reduces the rate of progress.

Blight incidence in ILC 3279 during 1982–83 was very low, which indicated reduction of initial inoculum. In two out of three seasons (1982–83 and 1983–84), blight epidemic in ILC 3279 was delayed by 7 days. However, if the start of the epidemic is considered in relation to the logarithmic phase of the disease (13), there was no delay in the start of epidemic in ILC 3279 compared with the other two cultivars. During the 1982–83 season, the epidemic did not progress. During the 1983–84 and 1985–86 seasons, the epidemic was drastically slowed down. Thus, except for the reduction in initial inoculum during the 1982–83 season, which is a characteristic of vertical resistance, resistance in ILC 3279 was in conformity with the horizontal resistance concept of Vanderplank (13).

ILC 482 and ILC 3279 were widely tested for Ascochyta blight resistance and yield in the Mediterranean region in the winter season. ILC 3279 was resistant to Ascochyta blight at most locations (12). In ILC 482, which was resistant to blight at ICARDA in earlier years, patches of dead plants were observed during 1981 (3). The *A. rabiei* isolate from these ILC 482 dead plants was later found to be of a different race than the one found earlier (6). Under field conditions, ILC 482 usually does not

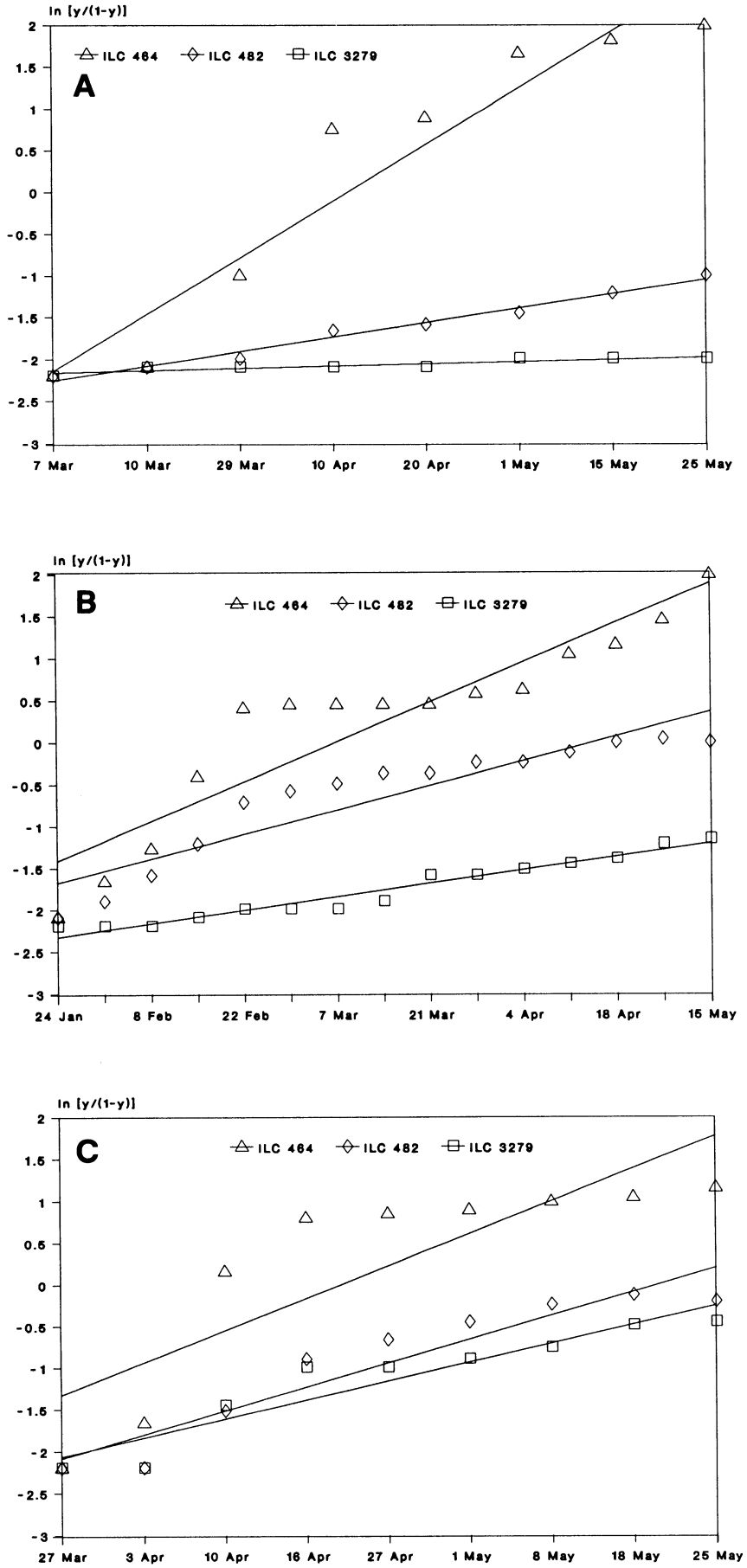


**Fig. 1.** Progress of Ascochyta blight in three chickpea cultivars at the International Center for Agricultural Research in the Dry Areas (ICARDA), Aleppo, Syria, in **(A)** 1982–83, **(B)** 1983–84, and **(C)** 1985–86.

show complete susceptibility to the new race, but it has disease severity scores of 5–6 on a 1–9 scale compared with scores of 3–4 against the old race. In a situation where the susceptible cultivar was killed, the yield of ILC 482 averaged 1,700 kg·ha$^{-1}$ over three seasons in comparison with 2,300 kg·ha$^{-1}$ in plots completely protected against blight (9).

ILC 482 has been released in Algeria, France, Jordan, Lebanon, Morocco, Syria, and Turkey and ILC 3279 in Algeria, Cyprus, Italy, Jordan, Syria, and Tunisia for cultivation in the winter season; both are maintaining their resistance. In Syria alone, the estimated area of winter cultivation of ILC 482 is 40,000 ha. Thus the rate-reducing resistance in these lines is useful over a wider geographic area. These are the first chickpea cultivars resistant to Ascochyta blight that have been released in more than one country. Because these cultivars demonstrate rate-reducing resistance against a mixture of isolates and an ability to perform well in a large number of countries against the pathogen *A. rabiei*, their resistance may be more stable than the resistance of previous cultivars.

## LITERATURE CITED


1. Campbell, C. L., and Madden, L. V. 1990. Pages 161-202 in: Introduction to Plant Disease Epidemiology. John Wiley & Sons, New York.
2. Hawtin, G. C., and Singh, K. B. 1984. Prospects and potential of winter sowing of chickpeas in the Mediterranean region. Pages 7-16 in: Ascochyta Blight and Winter Sowing of Chickpeas. M. C. Saxena and K. B. Singh, eds. Martinus Nijhoff/Dr. W. Junk Publishers, The Hague.
3. ICARDA. 1981. Report of work on Ascochyta blight of chickpea, 1980–81. ICARDA, Aleppo, Syria. 71 pp.
4. Kausar, A. G. 1965. Epiphytology of recent epiphytotics of gram blight in West Pakistan. Pakistan J. Agric. Sci. 2:185-195.
5. Nene, Y. L., and Reddy, M. V. 1987. Chickpea diseases and their control. Pages 233-270 in: The Chickpea. M. C. Saxena and K. B. Singh, eds. C.A.B. International, Wallingford, U.K.
6. Reddy, M. V., and Kabbabeh, S. 1985. Pathogen variability in *Ascochyta rabiei* in Syria and Lebanon. Phytopathol. Mediterr. 24:265-266.
7. Reddy, M. V., and Singh, K. B. 1984. Evaluation of a world collection of chickpea germ plasm accessions for resistance to Ascochyta blight. Plant Dis. 68:900-901.
8. Reddy, M. V., and Singh, K. B. 1990. Management of Ascochyta blight of chickpea through integration of host plant tolerance and foliar spraying of chlorothalonil. Indian J. Plant Prot. 18:65-69.
9. Reddy, M. V., and Singh, K. B. 1990. Relationship between Ascochyta blight severity and yield loss in chickpea and identification of resistant lines. Phytopathol. Mediterr. 29:32-38.
10. Reddy, M. V., and Singh, K. B. 1991. Relationship between temperature, relative humidity and Ascochyta blight development in winter-sown chickpea in Syria. Phytopathol. Mediterr. 29:159-160.
11. Singh, K. B., and Reddy, M. V. 1991. Advances in disease-resistance breeding in chickpea. Adv. Agron. 45:191-222.
12. Singh, K. B., Reddy, M. V., and Nene, Y. L. 1984. International testing of chickpeas for resistance to Ascochyta blight. Plant Dis. 68:782-784.
13. Vanderplank, J. E. 1963. Plant Diseases: Epidemics and Control. Academic Press, New York. 349 pp.