Yield Loss in Chickpeas in Relation to Development of Fusarium Wilt Epidemics

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ABSTRACT

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Development of 108 epidemics of Fusarium wilt of chickpea caused by *Fusarium oxysporum* f. sp. *ciceris* were studied on cvs. P-2245 and PV-61 in field microplots artificially infested with races 0 and 5 of *F. oxysporum* f. sp. *ciceris* in 1986 to 1989. Disease progression data were fitted to the Richards model using nonlinear regression. The shape parameter was influenced primarily by date of sowing and, to a lesser extent, by chickpea cultivars and races of *F. oxysporum* f. sp. *ciceris*. Fusarium wilt reduced chickpea yield by decreasing both seed yield and seed weight. These effects were related to sowing date, chickpea cultivar, and virulence of the prevalent *F. oxysporum* f. sp. *ciceris* race. Regression models were developed to relate chickpea yield to Fusarium wilt disease intensity with the following independent variables: time to initial symptoms (t_{is}), time to inflection point (t_{ip}) of the disease intensity index (*DII*) progress curve, final *DII* (DII_{final}), standardized area under *DII* progress curve (SAUDPC), and the Richards weighted mean absolute rate of disease progression (*rho*). Irrespective of the chickpea cultivar × pathogen race combination, the absolute and relative seed yields decreased primarily by delayed sowing. The relative seed yield increased with the delay in t_{is} and t_{ip} and decreased with increasing DII_{final}, SAUDPC, and *rho*. A response surface was developed in which seed yield loss decreased in a linear relationship with the delay in t_{is} and increased exponentially with the increase of *rho*.

Additional keywords: *Cicer arietinum*, crop loss models, quantitative epidemiology.

Chickpea (*Cicer arietinum* L.) is a major source of human and animal food and the world's third most important pulse crop after beans (*Phaseolus vulgaris* L.) and peas (*Pisum sativum* L.) (38). Chickpea production is severely curtailed by Fusarium wilt caused by *Fusarium oxysporum* (Schlechtend.:Fr.) f. sp. *ciceris* (Padwick) Matuo & K. Sato, in most chickpea growing areas of the world (17). In spite of its importance, there are few quantitative assessments of the impact of Fusarium wilt on chickpea yields. Annual chickpea yield losses from Fusarium wilt vary from 10 to 15% (17,43) but can result in total loss of the crop under specific conditions (14,15).

Fusarium wilt of chickpea has been managed primarily by the use of resistant cultivars (17), but virulent races of the pathogen have undermined their importance in recent years (16,19). Seven races, designated 0 to 6, of *F. oxysporum* f. sp. *ciceris* have been identified (16,19). Race 0, the least virulent of the seven races, induces progressive foliar yellowing compared with severe leaf chlorosis, flaccidity, and early wilt induced by races 1 through 6 (16,19). Races 1 through 4 were first described in India (19). Later, race 0 was reported in California, Spain, and Tunisia; races 1 and 6 were identified in California, Morocco, and Spain; and race 5 was found in California and Spain only (14,19).

Date of sowing is a key factor in determining yield of chickpea crops. In the Mediterranean region, chickpea is traditionally sown in the spring, but winter sowing enables matching of various crop growth stages with optimum environmental conditions and increases yield through better use of available water in soil (39). Choice of sowing time has been recommended for management of Fusarium wilt of chickpea (17,36), but those reports made no assessment as to how factors in the pathosystem would influence its efficiency. Navas-Cortés et al. (30) demonstrated that the effectiveness of sowing time as a management practice for Fusarium wilt of chickpea may be influenced both by virulence of the pathogen race prevalent in soil and susceptibility of the chickpea cultivar (30). However, the interactive effects of sowing date, chickpea cultivar, and race of *F. oxysporum* f. sp. *ciceris* on Fusarium wilt development and chickpea yield loss has not been determined. This information would provide a better understanding of the system and has the potential to be a valuable aid in the management of this disease.

Disease progress curves (25) can be described by growth curve models such as the monomolecular, Gompertz, logistic, Richards, and Weibull equations (8). Progression of diseases caused by soilborne pathogens can be determined by factors other than pathogen reproductive strategy (33). These can either be described by the logistic model (9,40) or they do not conform to either the monomolecular or the logistic model. In these cases, fits to disease progression data were improved with the nonsymmetrical Gompertz model (3) or the Weibull (32) and Richards models (8). Disease–yield loss relationship has been determined by single-point, multiple-point, and integral empirical models (8,27) that relate yield loss to disease intensity at: (i) a specific time or specific growth stage in the crop growing season; (ii) several points during this season; and (iii) a measurement of total disease derived by summing disease intensities over a specific period of crop growth (6,8,18). Increasing the understanding of crop yield losses in relation to development of plant disease epidemics may allow better predictions of crop losses and improved disease management practices (27). The objectives of this study were: (i) to determine the effects of sowing date, virulence of the pathogen race, and cultivar susceptibility on the development of Fusarium

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wilt epidemics in chickpeas; and (ii) to determine a relationship between seed yield losses and the development of disease epidemics.

MATERIALS AND METHODS

Experimental design. A microplot experiment was conducted in a field with sandy loam soil (pH 8.5, 1.4% organic matter) at the Alameda del Obispo Research Station near Córdoba (latitude 38° north, longitude 5° east) in three consecutive seasons (harvest years 1987, 1988, and 1989). This field had not been sown to chickpeas in the previous 10 years. The microplots (1.25 × 1.25 m, 50 cm deep) were established after fumigating the field with methyl bromide plus chloropicrin (80 g/m^2) on 30 October 1986 by burying thin pieces of concrete 50 cm deep into soil and raised 0.3 m above ground. Microplots were fertilized prior to sowing each season with 35 g of a 8-15-15 (N-P-K) commercial fertilizer Microplots were artificially infested with three inoculum rates of each of *F. oxysporum* f. sp. *ciceris* races 0 (Foc-0) and 5 (Foc-5) before sowing in the first year of the experiment on 14 December 1986 (early winter sowing date), 18 February 1987 (late winter sowing date), and 30 March 1987 (early spring sowing date) or maintained as uninfested controls. The experimental design consisted of a randomized, split-split plot design arranged in four replicated blocks. The treatments comprised three levels of sowing date (main factor), two chickpea cultivars (subplot), three initial inoculum rates (sub-subplot) of each of the two races of *F. oxysporum* f. sp. *ciceris*, and one noninfested control. For each sowing date, cultivars were randomly allocated to a set of microplots and pathogen races and inoculum rates were randomized within these microplots.

Microplots were sown on 16 December 1986 and 20 February and 2 April 1987 in year 1, 21 December 1987 and 3 February and 21 March 1988 in year 2, and 15 December 1988 and 31 January and 16 March 1989 in year 3. Cvs. P-2245 and PV-61 are small-seeded 'kabuli' (ram head shaped, beige seeds) chickpeas with the first highly susceptible to both Foc-0 and Foc-5 and the latter moderately susceptible to the two races (19). Seeds were treated with tridemorph (11% Calixin WP, 0.66 g a.i./kg of seed; BASF Española S.A., Barcelona) and captan (85% Captan WP, 3 g a.i./kg of seed; Argos, Valencia, Spain) fungicides to eradicate infections by *Didymella rabiei* and to control Pythium seed rot and preemergence damping-off, respectively (22,23). In each microplot there were three rows 0.4 m apart and 0.2 m from the closest microplot edge barrier (25 seeds per row). In year 3, an additional large-seeded 'kabuli' chickpea cv. PV-60, more susceptible to Foc-0 and Foc-5 than cv. PV-61 but less susceptible than cv. P-2245, was used. For the experiment in year 3, each microplot was sown to cvs. P-2245, PV-61, and PV-60, one row each. Weeds in the microplots were removed by hand and dimethoate (Romefos 40, 60 ml a.i./ha; Agrocrós S.A., Madrid) insecticide was applied for control of leaf miner (*Hylemiya* sp.) (12) as needed. Daily mean temperature and rainfall data were recorded at a weather station near the experimental site.

Inoculum of *F. oxysporum.* f. sp. *ciceris* isolates 7802 (race 0) and 8012 (race 5) was increased in a cornmeal-sand mixture (43) incubated at 25°C for 2 weeks. The upper 15-cm soil layer in a microplot was excavated and mixed thoroughly with desired volumes of infested cornmeal-sand with a cement mixer to establish three inoculum densities, and the soil was placed back in the corresponding microplots. The three rates of inoculum were 25.0 (low), 50.0 (intermediate), and 100.0 g (high) for Foc-0 and, 6.25 (low), 12.5 (intermediate), and 25.0 g (high) for Foc-5. Similar rates of noninfested cornmeal-sand were used for microplots that served as controls.

Assessment of disease and yield and data analyses. Disease incidence and severity were assessed at 7- to 10-day intervals. Severity of symptoms on individual plants were rated on a scale from 0 to 4 according to percentage of foliage with yellowing or necrosis in acropetal progression: 0 = 0%, 1 = 1 to 33%, 2 = 34 to 66%, 3 = 67 to 100%, and 4 = dead plant, as used previously (19,43). Incidence (*I*) and severity data (*S*) were used to calculate disease intensity index (*DII*) with $DII = (I \times S)/4$. Disease progress curves were obtained from the accumulated *DII* over time in days from the date of sowing.

Disease progress curves were characterized with the Richards model (8), which compared with monomolecular, Gompertz, and logistic models, gave the best fit to disease progress data from 3 years. The Richards model can be written as

$$DII(t) = K\,[1 - B\exp(-r \times t)]^{1/(1-m)} \text{ when } m < 1$$

and

$$DII(t) = K\,[1 + B\exp(-r \times t)]^{1/(1-m)} \text{ when } m > 1$$

in which *DII* = disease intensity index, K = asymptote parameter, B = constant of integration, r = rate parameter, m = shape parameter, and t = time of disease assessment in days after the date of sowing. For this model, the disease intensity at the inflection point ($DII(t_{ip})$) is given by $DII(t_{ip}) = Km^{1/(1-m)}$, and the time to reach this level (t_{ip}) is given by (26)

$$t_{ip} = [\ln(B) - \ln(1 - m)]r^{-1} \text{ when } m < 1$$

and

$$t_{ip} = [\ln(B) - \ln(m - 1)]r^{-1} \text{ when } m > 1$$

The least-squares program for nonlinear models (NLIN) procedure with Marquardt's compromise method was used to obtain estimates of parameters in the model (version 6.08, SAS Institute Inc., Cary, NC). For analyses, nonzero points for *DII* in experimental units and the average *DII* values of the four blocks were used. The coefficient of determination (R^2), the mean square error, the standard errors associated with the parameter estimates, confidence intervals of predicted values, and pattern of the standardized residuals plotted against either predicted values or the independent variable were used to evaluate the appropriateness of the model to describe the data (8,26). Because disease progress curves fitted to versions of the Richards model with different values of the asymptote and shape parameters cannot be compared directly, a common weighted mean rate parameter (*rho*) (8,26,31) was calculated for each epidemic in the study. This new rate parameter, named the weighted mean absolute rate of disease increase (8), is defined by $rho = Kr / (2m + 2)$, in which K is the asymptote parameter for *DII*, r is the rate parameter, and m is the shape parameter in the Richards function adjusted to a data set.

Disease development in a microplot was characterized by five variables associated to disease progress curves (30): (i) t_{is} = the time in days to initial symptoms, estimated as the number of days to reach a *DII* level of 0.05; (ii) t_{ip} = the time needed to reach the curve inflection point; (iii) final disease intensity (DII_{final}) = *DII* observed at the final date of disease assessment; (iv) the standardized area under disease progress curve (SAUDPC) calculated by trapezoidal integration method standardized by time in days (8); and (v) *rho* = weighted mean absolute rate of disease increase. The time needed to reach the inflection point and *rho* were obtained by the estimates of parameters of the Richards model fitted to *DII* progress data.

Chickpea yield in microplots was determined by the end of June every crop season, ≈28, 21, and 14 weeks after chickpea sowing in early winter, late winter, and early spring, respectively. Seed yield (total seed weight per row in microplot) and 100-seed weight were determined for each microplot. Actual loss of seed yield was determined as the difference between the total seed weight per row in a microplot and the average seed weight per row in the corresponding control microplots. Also, seed yield in a microplot for a treatment was expressed in relative units as relative yield (*RY*) by dividing seed weight per row (actual yield) by the average

seed weight per row in the corresponding, uninfested control microplots (attainable yield).

The effect of sowing date, host cultivar, and race of *F. oxysporum* f. sp. *ciceris* on chickpea yield was determined by analysis of variance. Yield data for each season were analysed with the general linear model (GLM) procedure of SAS. The three initial inoculum levels showed variance homogeneity as determined by the Barlett's test for equal variances. Therefore, initial inoculum rates and replications within them were considered random sources of variation. Separate analyses were performed for each year of experiment × chickpea cultivar combination. Mean comparisons among sowing dates within a cultivar were performed according to Fisher's protected least significant difference test at $P = 0.05$. The Dunnett's two-tailed t test was used to test significant differences between infested and noninfested control microplots at $P = 0.05$.

Yield loss–disease intensity relationships were determined by regression analyses with the five disease progress curves-associated variables as independent variables. For each year of experiments, regression analyses were performed by data pooled over sowing dates and initial inoculum rates. Therefore, a single model was produced for each chickpea cultivar and *F. oxysporum* f. sp. *ciceris* race combination, except for the 1986 to 1987 experiment. For this latter experiment, no disease developed in Foc-0-infested microplots, and available data for Foc-5 from both chickpea cultivars were included in a single model. Two models for the decrease in relative yield with disease development were fitted to data

$$RY = a + r_d X \quad (1)$$

$$RY = \exp[1 - \exp(r_d X)] \quad (2)$$

in which RY is the relative yield, a is an intercept, r_d is a rate of RY decrease, and X is either t_{is}, t_{ip}, DII_{final}, SAUDPC, or *rho*. A total of 60 regression analyses were performed by the least-squares procedure for linear (equation 1) and nonlinear models (equation 2). Coefficient of determination (R^2), the mean square error, the asymptotic standard error associated with the estimated parameter, and the pattern of the standardized residuals plotted against either predicted values or the independent variable were used to evaluate the appropriateness of a model to describe the data. The standard errors of parameters obtained from regression analyses were used to compare the effects of chickpea cultivars, as well as, the effect of pathogen races on relative yield (8).

RESULTS

Analysis of disease progression. Over all three seasons, a total of 108 Fusarium wilt epidemics were available for analysis in this study. The Richards model adequately described all epidemics in the study and improved fit of disease progression data to model. *DII* progress curves for 1988 to 1989 are illustrated in Figure 1.

Mean parameter values estimated either with the fixed-shape or the Richards models yielded similar values (data not shown). A range of values for the Richards model shape parameter (m) were obtained. Of the 108 *DII* progress curves in the study analyzed with the Richards model, 11 had no inflection point ($m = 0$) and 97 had an inflection point but were asymmetrical. Out of these 97 *DII* progress curves, 93 had a positive skewness ($0 < m < 2$, i.e., increase in the absolute rate was faster as the rate approached the inflection point) and only four had a negative skewness ($m > 2$) (Fig. 1).

Yield loss–disease intensity relationships. Chickpea seed yield (gram per microplot row) and 100-seed weight in uninfested control microplots were influenced by sowing date and varied among cultivars and years of experiment (Table 1). Overall, there was a trend for seed yield to decrease as sowing date was delayed from early winter to early spring in the 1986 to 1987 and 1988 to 1989 experiments ($P < 0.05$). Seed yield was highest in the 1987 to 1988 experiment and was lowest in 1988 to 1989. The year also influenced 100-seed weight but to a lesser degree than for seed yield to sowing date (Table 1).

Both the seed yield and 100-seed weight were affected by the time of disease onset and development, but the effect on seed yield was larger than that on 100-seed weight in all treatments (Table 1). For each sowing date, seed yield loss was determined primarily by virulence of the *F. oxysporum* f. sp. *ciceris* race and to a lesser extent by susceptibility of the chickpea cultivar. Seed yield loss was higher with the highly virulent Foc-5 than with the less virulent Foc-0. This loss in seed yield was greater in cv. P-2245 than in cvs. PV-60 and PV-61 (Table 1). Seed yield loss caused by Foc-5 averaged over sowing dates in the 3 years of study was highest (99.7%) in the most susceptible cv. P-2245 and lowest (81.9%) in the least susceptible cv. PV-61. Similarly, seed yield loss caused by Foc-0 ranged from 65.6 to 30.6% for cvs. P-2245 and PV-61, respectively. Yield reduction by Fusarium wilt was also associated with poor seed size and quality, as indicated by the correlation between seed yield loss and 100-seed weight ($r = -0.697$, $P < 0.001$).

The quantitative relationship between the decrease in relative chickpea seed yield and Fusarium wilt development was explained by a linear regression model that includes t_{is}, t_{ip}, or DII_{final} as an independent variable and by an exponential regression model that includes SAUDPC or *rho* as an independent variable. Because yield was 0 in microplots infested with Foc-5, regression analyses could not be performed for treatments involving this race. Regression models were compared by the rate of relative seed yield decrease (r_d). This rate was influenced primarily by the race of the pathogen in soil and secondly by the chickpea cultivar used (Table 2).

The relative seed yield was significantly ($P < 0.05$) correlated with t_{is} and t_{ip} in the 1987 to 1988 and 1988 to 1989 experiments, but not in 1986 to 1987. In 1987 to 1988 and 1988 to 1989, the RY increased when disease onset (t_{is}) and subsequent development (t_{ip}) were delayed. Comparisons of r_d estimates and parallelism analyses (to test if the same r_d value could be used for each chickpea cultivar × pathogen race combination) indicated that r_d was not influenced by chickpea cultivar or pathogen race, but a common r_d for all cultivar × race combinations was detected (Table 2; Fig. 2). However, for similar t_{is} or t_{ip} values and for Foc-0 and Foc-5 the reduction in RY was greater in cv. P-2245 than in cvs. PV-60 and PV-61 (Fig. 2). The parallel-lines model predicted a RY increase of 0.60 and 0.37% per each 1-day delay in t_{is} and of 0.55 and 0.33% per each 1-day delay in t_{ip} in 1987 to 1988 and 1988 to 1989, respectively (Fig. 2).

Relative yield decreased significantly ($P < 0.05$) with the increase in the DII_{final} (Table 2; Fig. 3). The selected model explained 72 and 96% of the total RY variation in 1986 to 1987 and 1988 to 1989, respectively, and predicted 1.0% RY reduction for each 1% increase in the DII_{final} (Fig. 3). In the 1988 to 1989 experiment, the r_d in cv. P-2245 × Foc-0 interaction was significantly ($P < 0.05$) greater than that in other cultivar × race combinations (Table 2), and the data set from this interaction showed a strong deviation from those of other interactions. Parallelism analyses of regression lines for interactions between cvs. PV-60 and PV-61 and Foc-0 or Foc-5 indicated a common r_d among them. The parallel-lines model explained 98% of the total RY variation and predicted a RY reduction of 1.2% for each 1% increase in the DII_{final} (Fig. 3).

Relative yield decreased according to a negative exponential model with both increase in the weighted mean absolute rate of *DII* increase (*rho*) and the SAUDPC. The rate of reduction in RY (r_d) was highest with low SAUDPC or *rho* and decreased as SAUDPC or *rho* increased (Figs. 4 and 5). In 1987 to 1988 and 1988 to 1989, r_d was significantly higher ($P < 0.05$) with Foc-5 than with Foc-0, irrespective of chickpea cultivar and independent variables (Table 2). Comparisons of data fit to separate and

parallel-curve models indicated that the fitted curves had no r_d parameter in common (data not shown), except for regressions using SAUDPC data from 1987 to 1988 for which data fitted best to the single-curve model (Fig. 4). For the remaining models, r_d was higher ($P < 0.05$) for cv. P-2245 than for cvs. PV-60 and PV-61, irrespective of the *F. oxysporum* f. sp. *ciceris* race. These models explained 62 to 97% of the total *RY* variation (Table 2; Figs. 4 and 5).

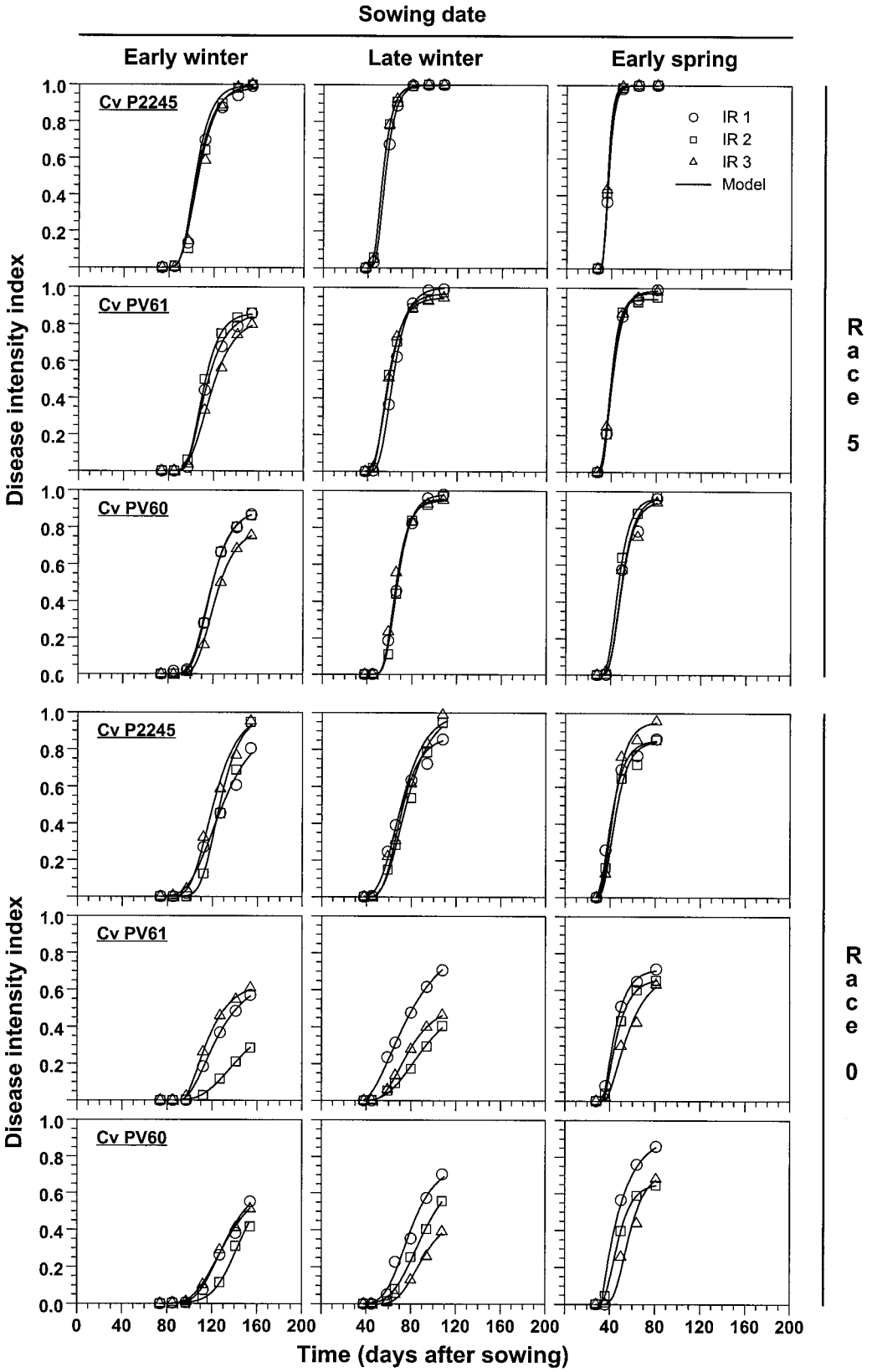

Fig. 1. Fusarium wilt progress on chickpea cvs. P-2245, PV-60, and PV-61 sown at three dates in microplots infested with three initial inoculum rates (IR) of 6.25 g (IR1), 12.5 g (IR2), and 25.0 g (IR3) per kg of soil for race 5, and 25.0 g (IR1), 50.0 g (IR2), and 100.0 g (IR3) per kg of soil for race 0 of *Fusarium oxysporum* f. sp. *ciceris* races 0 and 5 for 1988 to 1989. Each point represents the mean disease intensity index of four replications. Solid lines represent the predicted disease progress curves calculated by the Richards equation. Chickpeas were sown on: 15 December 1988 (early winter), 31 January 1989 (late winter), and 16 March 1989 (early spring).

TABLE 1. Effect of combinations of races of *Fusarium oxysporum* f. sp. *ciceris*, chickpea cultivars, and sowing dates on seed yield loss (YL) and yield components[z]

		Seed yield (g/microplot row)					100-Seed weight (g)				
			Race 0		Race 5			Race 0		Race 5	
Cultivar	Sowing date	Control	Infected	YL (%)	Infected	YL (%)	Control	Infected	YL (%)	Infected	YL (%)
1986–1987											
P-2245	Early winter	154.2 b	…	…	9.5 b*	94.7 ± 5.5	18.9 a	…	…	10.5 a*	45.5 ± 32.7
	Late winter	184.2 a	…	…	39.9 a*	78.3 ± 11.0	18.2 a	…	…	15.0 a	17.1 ± 10.5
	Early spring	112.7 c	…	…	30.1 a*	73.4 ± 15.1	18.0 a	…	…	16.2 a*	10.8 ± 2.8
PV-61	Early winter	133.3 a	…	…	78.7 b*	43.0 ± 15.1	27.9 a	…	…	25.0 b*	10.5 ± 2.7
	Late winter	140.9 a	…	…	150.3 a	–7.1 ± 6.5	25.4 b	…	…	27.8 a	–9.2 ± 2.7
	Early spring	103.0 b	…	…	91.7 b	11.1 ± 6.7	23.8 b	…	…	24.0 b	–0.6 ± 6.5
1987–1988											
P-2245	Early winter	279.2 a	233.1 a*	15.5 ± 19.5	2.4 a*	99.2 ± 1.0	18.3 b	18.5 a	–1.1 ± 3.0	8.0 a*	56.2 ± 16.6
	Late winter	210.3 b	172.1 b*	22.3 ± 12.8	0.0 b*	100 ± 0.0	18.5 b	17.5 a	7.3 ± 4.4	0.0 b*	100 ± 0.0
	Early spring	180.1 c	25.5 c*	86.0 ± 5.2	0.9 a*	99.5 ± 0.8	22.6 a	11.0 b*	51.0 ± 19.0	18.3 a*	76.3 ± 23.2
PV-61	Early winter	349.7 a	319.0 a	8.8 ± 10.8	207.9 a*	40.4 ± 23.5	26.0 b	25.6 a	1.3 ± 0.2	21.5 a	17.5 ± 14.5
	Late winter	248.4 b	232.3 b	6.6 ± 2.0	36.2 b*	85.5 ± 7.7	27.4 ab	25.7 a	5.7 ± 6.0	14.4 a*	47.4 ± 14.9
	Early spring	177.2 c	93.8 c*	47.1 ± 20.6	16.2 b*	90.9 ± 11.8	29.4 a	23.5 a	20.2 ± 27.7	15.3 a*	47.8 ± 18.5
1988–1989											
P-2245	Early winter	40.0 a	7.1 a*	83.8 ± 14.4	0.4 a*	99.1 ± 1.3	15.1 a	7.4 a*	50.3 ± 23.7	1.1 a*	92.3 ± 7.3
	Late winter	37.9 a	2.4 a*	94.5 ± 9.3	0.0 b*	100 ± 0.0	19.5 a	1.9 a*	90.3 ± 12.5	0.0 b*	100 ± 0.0
	Early spring	40.5 a	9.0 a*	77.4 ± 15.0	0.0 b*	100 ± 0.0	15.2 a	4.6 a*	68.1 ± 20.9	0.0 b*	100 ± 0.0
PV-61	Early winter	185.8 a	130.4 a	32.3 ± 19.4	39.4 a*	78.8 ± 1.7	23.4 a	22.6 a	1.7 ± 23.8	7.3 a*	68.5 ± 4.3
	Late winter	149.2 ab	100.5 a	34.2 ± 29.8	3.8 b*	97.3 ± 2.5	28.3 a	21.5 a	23.6 ± 21.4	6.9 a*	74.6 ± 19.1
	Early spring	107.3 b	48.0 a*	54.6 ± 8.9	1.7 b*	98.4 ± 1.5	22.4 a	13.2 b*	41.2 ± 17.7	5.2 a*	75.2 ± 15.6
PV-60	Early winter	158.0 a	114.4 a	26.1 ± 18.9	33.2 a*	79.0 ± 7.6	56.9 a	48.6 a	14.0 ± 15.5	23.8 a*	58.7 ± 15.9
	Late winter	158.7 a	82.2 a*	46.4 ± 15.4	2.5 b*	98.5 ± 1.3	62.0 a	53.2 a	13.3 ± 14.3	10.3 a*	83.9 ± 8.1
	Early spring	99.3 a	34.8 b*	64.3 ± 16.3	1.4 b*	98.5 ± 0.5	51.1 a	25.5 b*	50.2 ± 26.4	8.9 a*	83.3 ± 10.5

[z] Fumigated field soil in microplots were artificially infested with *F. oxysporum* f. sp. *ciceris* races 0 and 5 grown in cornmeal-sand or mixed with noninfested substrate (Control). The infested substrate was mixed thoroughly with the upper 15-cm layer of soil. Microplots were sown on 16 December 1986 and 20 February and 2 April 1987 for 1986 to 1987; 21 December 1987 and 3 February and 21 March 1988 for 1987 to 1988; and 15 December 1988 and 31 January and 16 March 1989 for 1988 to 1989. Data are the average of four blocks (microplots, 75 plants each) with three initial inoculum rates per block. Seed yield and 100-seed weight were determined for each microplot row. YL was determined as the difference between the total seed weight per row in a microplot and the average seed weight per row in the corresponding control microplots. Means in a column followed by the same letter for each cultivar within an experimental period are not significantly different according to Fisher's protected LSD ($P = 0.05$). Means in a row followed by an asterisk are significantly smaller ($P < 0.05$) than the mean for the corresponding uninfested control according to Dunnett's test.

TABLE 2. Relationship between relative seed yield of chickpea cvs. P-2245, PV-60, and PV-61 and disease progress curve-associated variables of Fusarium wilt epidemics developed in microplots artificially infested with races 0 and 5 of *Fusarium oxysporum* f. sp. *ciceris*[y]

		Disease progress curve-associated variable[z]									
		t_{is}		t_{ip}		DII_{final}		SAUDPC		*rho*	
Race	Cultivar	$r_d \pm$ SE	R^2	$r_d \pm$ SE	R^2	$r_d \pm$ SE	R^2	$r_d \pm$ SE	R^2	$r_d \pm$ SE	R^2
1986–1987											
Foc-5	P-2245, PV-61	NC	…	NC	…	–1.04 ± 0.16	0.72	2.15 ± 0.28	0.71	87.44 ± 18.88	0.36
1987–1988											
Foc-5	P-2245	…	…	…	…	…	…	…	…	…	…
	PV-61	0.0074 ± 0.0011	0.86	0.0055 ± 0.0009	0.84	–1.22 ± 0.15	0.90	2.06 ± 0.27**	0.63	53.57 ± 3.30**	0.94
Foc-0	P-2245	0.0069 ± 0.0008	0.93	0.0066 ± 0.0009	0.90	–0.99 ± 0.09	0.96	1.99 ± 0.30	0.90	34.95 ± 5.38	0.90
	PV-61	0.0042 ± 0.0010	0.73	0.0041 ± 0.0010	0.72	–0.95 ± 0.04	0.99	1.46 ± 0.07	0.97	29.96 ± 2.26	0.93
1988–1989											
Foc-5	P-2245	…	…	…	…	…	…	…	…	…	…
	PV-60	0.0034 ± 0.0006	0.82	0.0031 ± 0.0006	0.82	–1.37 ± 0.12	0.95	2.28 ± 0.09**	0.85	53.84 ± 1.19**	0.96
	PV-61	0.0032 ± 0.0003	0.94	0.0030 ± 0.0003	0.92	–1.33 ± 0.14	0.93	2.00 ± 0.09**	0.78	52.97 ± 3.10**	0.70
Foc-0	P-2245	0.0031 ± 0.0012	0.48	0.0029 ± 0.0010	0.53	–2.14 ± 0.30*	0.88	2.19 ± 0.15*	0.63	58.38 ± 4.21*	0.62
	PV-60	0.0047 ± 0.0011	0.71	0.0039 ± 0.0009	0.72	–1.15 ± 0.13	0.92	1.66 ± 0.11	0.81	39.36 ± 3.45	0.65
	PV-61	0.0038 ± 0.0015	0.48	0.0037 ± 0.0013	0.55	–1.12 ± 0.15	0.89	1.22 ± 0.11	0.68	31.51 ± 2.95	0.67

[y] Fumigated field soil in microplots artificially infested with *F. oxysporum* f. sp. *ciceris* races 0 (Foc-0) and 5 (Foc-5) grown in cornmeal-sand or mixed with noninfested substrate (Control). The infested substrate was mixed thoroughly with the upper 15-cm layer of soil. Microplots were sown on 16 December 1986 and 20 February and 2 April 1987 for 1986 to 1987; 21 December 1987 and 3 February and 21 March 1988 for 1987 to 1988; and 15 December 1988 and 31 January and 16 March 1989 for 1988 to 1989.

[z] t_{is} = Time in days to initial symptoms, estimated as the number of days to reach a disease intensity index (*DII*) level of 0.05; t_{ip} = the time needed to reach the curve inflection point; DII_{final} = disease intensity index observed at the final date of disease assessment; SAUDPC = area under disease progress curve standardized by time in days; *rho* = weighted mean absolute rate of disease progression. The time needed to reach the inflection point and *rho* were obtained by the estimates of parameters of the Richards model fitted to *DII* progress data. For each year of experiments, regression models were performed by pooling data over sowing dates and initial inoculum rates. r_d = relative rate of seed yield decrease; SE = standard error; and R^2 = coefficient of determination. The standard errors of the r_d obtained from regression analyses were used to compare the effects of experimental treatments. NC indicates the relative seed yield was not significantly correlated with t_{is} and t_{ip} ($P = 0.05$). No regression analyses were performed on Foc-5 cv. P-2245 because severe disease in microplots resulted in no seed yield for most cases. For each experimental period and *F. oxysporum* f. sp. *ciceris* race, the r_d value for a cultivar in a column followed by an asterisk is significantly higher ($P < 0.05$) than the corresponding values for other cultivars. For each experimental period and chickpea cultivar, ** indicates the r_d value for a pathogen race in a column is significantly higher ($P < 0.05$) than the corresponding r_d at the other pathogen race.

Based on the single regression analyses, a response surface for *RY* as a function of both t_{is} and *rho* was developed with original data from 1987 to 1988 and 1988 to 1989 and the equation

$$RY\,(t_{is}, rho) = (c_1 + c_2 t_{is}) \times \{\exp[1 - \exp(c_3 rho)]\} \quad (3)$$

In this equation, *RY* is the relative seed yield, c_1 is an intercept, and c_2 and c_3 are rates of *RY* decrease. Parameters ($c_1 - c_3$) in that equation were simultaneously estimated for each chickpea cultivar × *F. oxysporum* f. sp. *ciceris* race combination. Because the single model analysis indicated no effects of t_{is} on r_d estimates (Table 2), regression analyses were carried out with common c_2 parameter for each year of the study, and the effects on c_1 and c_3 were separated. The reduction in *RY* was influenced both by the nature of the *F. oxysporum* f. sp. *ciceris* race and susceptibility of the chickpea cultivar (Fig. 6). In the cultivar × race combination most conducive for disease development (cv. P-2245 × Foc-5), 100% seed yield loss (maximum *RY* value) occurred even with a very late epidemic onset, i.e., 90 days after sowing or very low *rho* of 0.012. On the contrary, reduction in *RY* of cvs. PV-60 and PV-61 caused by the less virulent Foc-0 was small when disease onset was at least 120 days after sowing or *rho* < 0.002. Conversely, *RY* reduction of the same cultivars caused by the highly virulent Foc-5 ranged from 75 to 100% for epidemics with early onset, i.e., t_{is} < 40 days after sowing, because *rho* was >0.016 (Fig. 6).

DISCUSSION

Fusarium wilt of chickpeas can be managed by adjusting the sowing date (17,36) but the efficiency of this disease management practice may be influenced by factors in the pathosystem determining the disease development (30). No prior quantitative studies have been carried out on the relationship between Fusarium wilt development and chickpea yield loss. The aim of this work was primarily to relate seed yield losses to Fusarium wilt development, as influenced by several components in the pathosystem, i.e., virulence of the pathogen race, susceptibility of the host cultivar and environment as determined by sowing date.

In our study, the amount of Fusarium wilt varied considerably among the 3 years of the study. The differences in the levels of disease intensity may be due in part to differences in weather

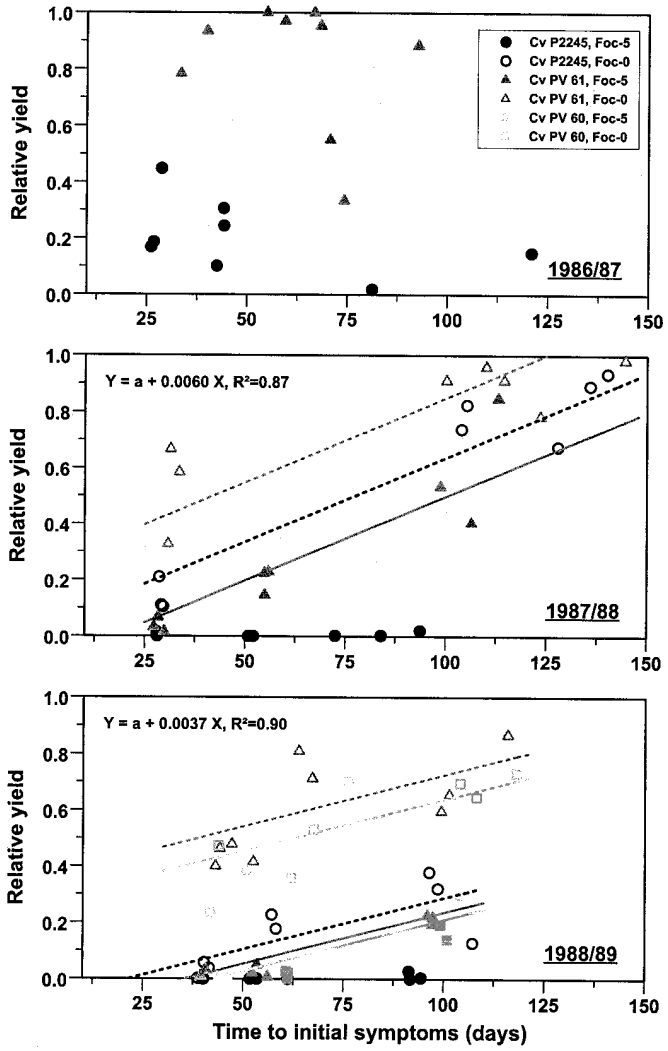

Fig. 2. Relationship between time in days to initial symptoms and the relative yield of chickpea cvs. P-2245 (black circles), PV-60 (light gray squares), and PV-61 (dark gray triangles) grown in microplots artificially infested with *Fusarium oxysporum* f. sp. *ciceris* races 0 (Foc-0, dashed line and open symbols) and 5 (Foc-5, solid line and solid symbols) for three experimental periods. Each point is the mean of data from four microplots (75 plants each). For each year of experiments, regression analyses were performed by pooling data over sowing dates and initial inoculum rates. Solid and dashed lines represent the predicted models with common rate of relative yield decrease according to parallel-lines analyses.

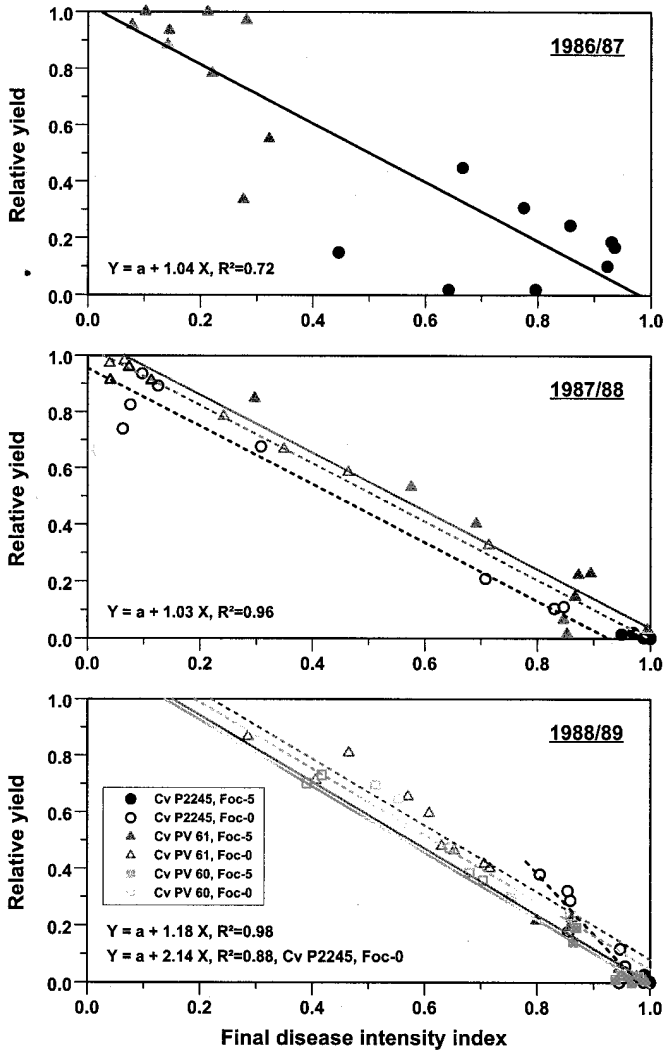

Fig. 3. Relationship between final disease intensity index and relative seed yield of chickpea cvs. P-2245 (black circles), PV-60 (light gray squares), and PV-61 (dark gray triangles) grown in microplots artificially infested with *Fusarium oxysporum* f. sp. *ciceris* races 0 (Foc-0, dashed line and open symbols) and 5 (Foc-5, solid line and solid symbols) for three experimental periods. Each point is the mean of data from four microplots (75 plants each). For each year of experiments, regression analyses were performed by pooling data over sowing dates and initial inoculum rates. Solid and dashed lines represent the predicted model with common rate of relative yield decrease estimated by parallel-lines regression analyses.

conditions and the increase in the pathogen population in soil as a result of successive sowings of susceptible chickpeas in microplots. The annual variation in severity of Fusarium wilt of chickpeas is often attributed to differences in temperature and inoculum density (13,29). For each cropping season, changes in Fusarium wilt development are related mainly to date of sowing, cultivar susceptibility, pathogen race virulence, and their interactions. For each chickpea cultivar × pathogen race combination, the differences in epidemics that developed in the three sowing dates can be explained by differences in rainfall and temperature. Delaying sowing from the middle of December to the middle of March results in decreased soil moisture and increased temperature that favour development of Fusarium wilt (4,41).

Progression of Fusarium wilt intensity in chickpea cultivars sown at different times in soil infested with races 0 or 5 of *F. oxysporum* f. sp. *ciceris* could not be described by either the fixed-shape models or the Richards model. On the contrary, a wide range of values for the shape parameter was estimated by the Richards model. Overall, disease progression was better described by a sigmoid-shape model, asymmetric or in a few cases symmetric. The monomolecular shape (i.e., the Richards shape parameter close to 0) was appropriate only for epidemics on susceptible cv. P-2245, caused by the highly virulent race (Foc-5) and the presence of conducive weather (spring sowing date). The Richards model offers a continuous range of inflection points and thus provides an alternative to choosing different models for different epidemics (31). However, this model has been used infrequently to describe plant disease progression. In this study, it enabled analysis of all shapes of Fusarium wilt progress curves produced. The consistently good fit provided by the Richards model and the well defined epidemiological concepts corresponding to the parameters, make it ideal for describing disease progress of Fusarium wilt epidemics in chickpeas.

Several epidemics caused by soilborne plant pathogens have been appropriately described by a sigmoidal disease progress model (2,10,40). Deviations of Fusarium wilt of chickpea from

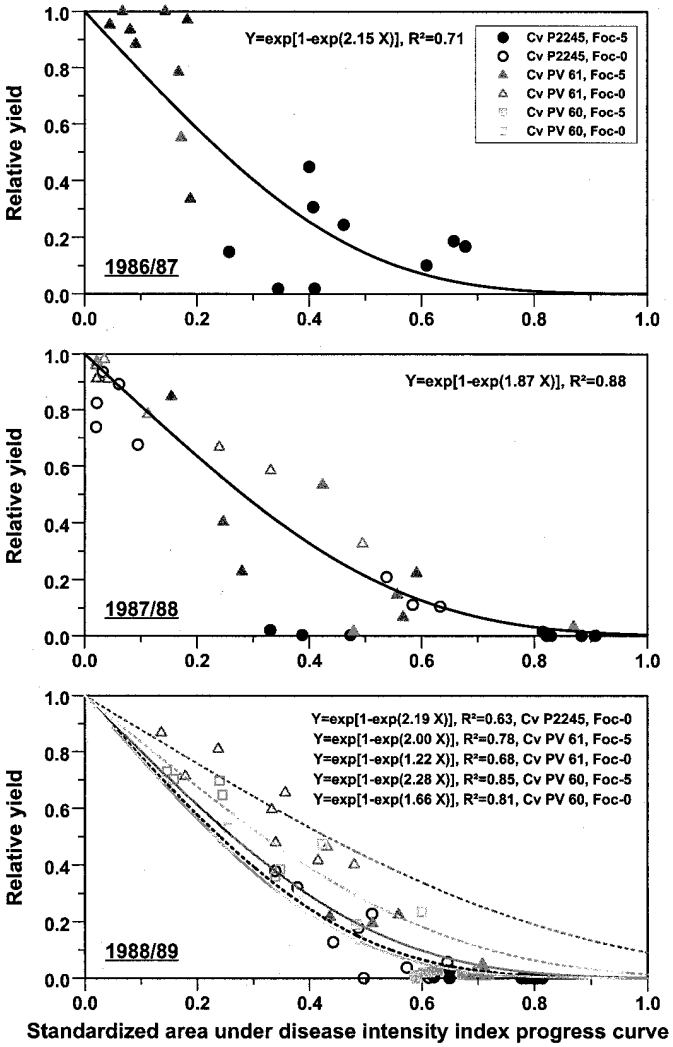

Fig. 4. Relationship between the standardized area under Fusarium wilt intensity progress curve and the relative seed yield of chickpea cvs. P-2245 (black circles), PV-60 (light gray squares), and PV-61 (dark gray triangles) grown in microplots artificially infested with *Fusarium oxysporum* f. sp. *ciceris* races 0 (Foc-0, dashed line and open symbols) and 5 (Foc-5, solid line and solid symbols) for three experimental periods. Each point is the mean of data from four microplots 75 plants each). For each year of experiments, regression analyses were performed by pooling data over sowing dates and initial inoculum rates. The solid and dashed lines represent the predicted model with common (1987 to 1988) or separate (1988 to 1989) rate of relative yield decrease estimated by parallel-curves regression analyses.

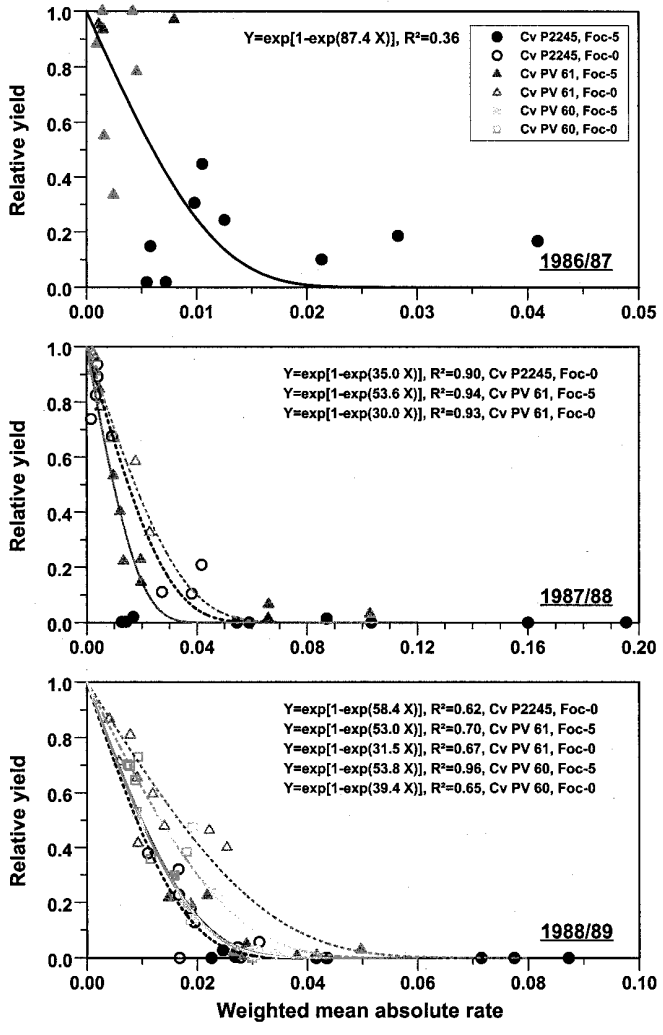

Fig. 5. Relationship between the Richards weighted mean absolute rate of Fusarium wilt intensity index progress curve and the relative seed yield of chickpea cvs. P-2245 (black circles), PV-60 (light gray squares), and PV-61 (dark gray triangles) grown in microplots artificially infested with *Fusarium oxysporum* f. sp. *ciceris* races 0 (Foc-0, dashed line and open symbols) and 5 (Foc-5, solid line and solid symbols) for three experimental periods. Each point is the mean of data from four microplots (75 plants each). For each year of experiments, regression analyses were performed by pooling data over sowing dates and initial inoculum rates. Solid and dashed lines represent the predicted model with separate rate of relative yield decrease estimated by parallel-curves regression analyses.

the monomolecular shape, which involve an acceleration of disease during early stages of the epidemic, could be due to modifications of disease progression by weather, cultivar susceptibility, or pathogen virulence. Also, the assessment of disease by the severity of foliar symptoms compared with disease incidence, might have an influence on these modifications. Disease progress of a theoretical 'monocyclic disease' in the field can be modified by environment (5), host susceptibility (34), environment–host genotype interactions (40), and consequently, is poorly described by the monomolecular function (33). Sporulation of *F. oxysporum*

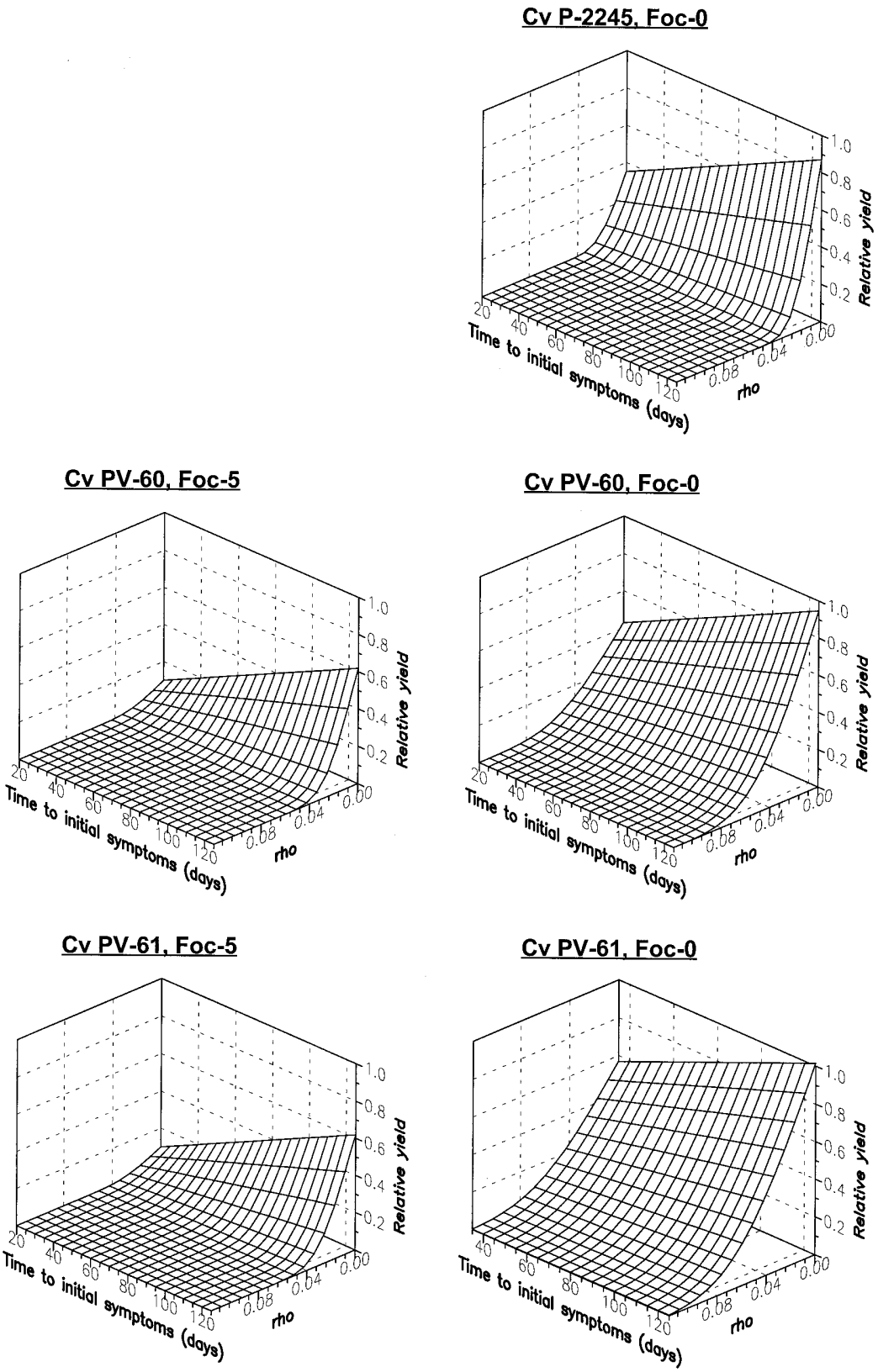

Fig. 6. Response surfaces as a function of time to initial symptoms and the Richards weighted mean absolute rate (*rho*) of Fusarium wilt intensity index progress curve for the decrease in relative seed yield of chickpea cvs. P-2245 and PV-61 grown in microplots artificially infested with *Fusarium oxysporum* f. sp. *ciceris* races 0 (Foc-0) and 5 (Foc-5) for 1988 to 1989. Regression analyses were performed by pooling data over sowing dates and initial inoculum rates.

f. sp. *ciceris* on chickpea stem surfaces under our field conditions has not been observed unlike *F. oxysporum* f. sp. *lycopersici* (24), which could play a role as secondary inoculum. Furthermore, if soilborne or airborne inoculum of *F. oxysporum* f. sp. *ciceris* were produced, it is unlikely that they might play a role in disease progression because early infection of chickpea seedlings by the pathogen must take place for severe symptoms to develop (20). Thus, a number of factors involved in the appearance of foliar symptoms of the disease (i.e., conduciveness of weather, cultivar susceptibility, virulence of race prevalent in soil, and their interactions) may be responsible for the observed deviations in the disease progress curves.

Results in uninfested microplots indicate that the chickpea yield is determined by the time of planting, with decreasing seed yield as planting is delayed from early winter to early spring. Reductions in seed yield by the disease was larger than reductions in 100-seed weight. Therefore, the overall yield loss caused by the disease may be attributed mainly to a significant decrease in the number of seeds per plant and to a lesser extent to reduced mean seed weight. Similar effects were reported for field beans due to infections by *Botrytis fabae* (11). Irrespective of sowing date, seed yield in infested microplots was determined by virulence of the *F. oxysporum* f. sp. *ciceris* race and susceptibility of the chickpea cultivar.

Our results show a significant relationship between chickpea seed yield and Fusarium wilt disease progress curve-associated variables, by single-point models, (i.e., the time of disease onset (t_{is}) and development (t_{ip}), or the disease intensity index at the end of the season (DII_{final})) integral models, (i.e., SAUDPC and the weighted mean absolute rate of disease increase (*rho*)), or surface-response models. Seed yield increased linearly with increasing t_{is} and t_{ip}, and decreased linearly with increasing DII_{final}. The rate of seed yield loss (r_d) attributable to either chickpea cultivar or pathogen race was not observed. However, the intercepts of the seed yield loss linear models developed varied for cultivar × race combinations. Therefore, for similar t_{is}, t_{ip}, or DII_{final} values and for the two *F. oxysporum* f. sp. *ciceris* races included in the study, yield loss was higher in cv. P-2245 than in cvs. PV-60 and PV-61. Work by Haware and Nene (15) in India showed that yield loss caused by Fusarium wilt in 'desi' cultivars planted in early October was influenced by the growth stage at which the disease developed. However, no quantitative relationship was demonstrated between yield loss and disease onset or development, and no assessment was made concerning the influence of pathogen race virulence on that effect. In that study (15), early wilting reduced the seed number per plant and caused more yield loss than late wilting, whereas the latter produced less seed and caused substantial yield loss. One hundred-seed weight was also adversely affected by wilt in all cultivars in the study even when plants showed symptoms at the preharvest stage. In Verticillium wilt of cotton, a similar pathosystem, the effect of epidemics on cotton yield was related to the phenological stage of plants when foliar symptoms first developed and to virulence of the *Verticillium dahliae* pathotype prevalent in soil (1,35).

Reduction of chickpea seed yield by Fusarium wilt development followed a negative exponential model with the increase in SAUDPC or *rho*. In contrast to linear models related to t_{is}, t_{ip}, and DII_{final}, the rate of yield loss for models including SAUDPC or *rho* depended on virulence of the *F. oxysporum* f. sp. *ciceris* race and susceptibility of the chickpea cultivar. In all cases, the greatest absolute rate of decrease in seed yield occurred at low SAUDPC or *rho*, and the absolute rate of decrease declined in absolute value as SAUDPC or *rho* increased. This type of model, a type I curve (28), suggests that the remaining, apparently healthy green tissue of the host is affected by the pathogen (21). SAUDPC and AUDPC have been used as a predictor of yield losses for several pathosystems (8,26,45).

Results from the present study allowed the development of a response-surface function for relative seed yield decrease as influenced by t_{is} and *rho*. This response surface confirmed that in the chickpea–*F. oxysporum* f. sp. *ciceris* pathosystem the relative seed yield loss increases in a linear relationship with increasing t_{is} and decreases in a negative exponential relationship with increases in *rho*. A similar model was developed for stem rust of wheat caused by *Puccinia graminis* f. sp. *tritici*, where yield loss was estimated as a function of the rate of disease increase and growth stage of the host at time of epidemic onset (7). Models represented by a response surface provide a conceptual framework based on a knowledge of disease epidemiology and crop physiology for modelling disease-loss systems (42). Our model incorporates the effect of chickpea cultivar susceptibility and pathogen virulence, thereby accounting for the effects of different disease progress curve-associated variables on seed yield and providing a better understanding of the chickpea–*F. oxysporum* f. sp. *ciceris* interaction. However, this model does not include the influence of other variables such as plant stand, weather, soil type, or plant density, all of which relate to healthy chickpea yield potential. This would also make the model unwieldy.

In the Mediterranean region, chickpeas are traditionally planted in the spring and the crop develops on the residual moisture in soil from winter rains. As the season proceeds, the crop experiences rising temperatures and increasing soil moisture stress that shorten the vegetative and reproductive periods and decrease yields (37,39). Fusarium wilt incidence and severity are enhanced by warm, dry soils occurring in spring-sown crops (13,43,44). Planting in winter corresponds with optimum environmental conditions for chickpea growth, ensures better use of available water, and increases yield (39). Also, under conditions in southern Spain, planting in early winter instead of early spring significantly delays epidemic onset, slows the rate of development, and reduces the final amount of disease (30). Experiments conducted in India showed that Fusarium wilt intensity decreased and chickpea seed yield increased in plantings delayed until the middle of October (17,36). However, no assessments were made in these studies of the influence of pathogen virulence and cultivar susceptibility. Our results confirm that planting time determines the severity of Fusarium wilt and the quantum of chickpea seed yield.

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