From these results, we conclude that ragweed probably does not serve as an inoculum source for Jerusalem artichokes or the other hosts.

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Use of Fungicide Treatments and Host Resistance to Control the Wilt and Root Rot Complex of Chickpeas

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ABSTRACT

Jiménez-Díaz, R. M., and Trapero-Casas, A. 1985. Use of fungicide treatments and host resistance to control the wilt and root rot complex of chickpeas. Plant Disease 69:591-595.

Efficacy of fungicide seed dressings, foliar sprays, and moderately resistant cultivars alone or in combination for control of the wilt and root rot (WRR) complex of chickpeas was studied in fields with a history of high incidence (≥80%) of the disease. The efficacy of treatments was assessed by their influence on the epidemic development of the disease. Most epidemics of the WRR complex in the treated and control plots could be linearized best by the logistic transformation. Seed dressings with each of captan, captafol, thiram, benomyl, triadimenol and the mixtures of benomyl with captan, captafol, or thiram; thiabendazole with captan or captafol; and captafol with carboxin, triforine, thiophanate-methyl + maneb, or triadimenol + TIM-5107 + fuberidazol significantly increased seedling emergence of the moderately resistant cultivars PV-24 and PV-25 but not of the very susceptible P-2245 in 1980–1981 trials. Some fungicide seed dressings significantly delayed early development of epidemics for cultivars PV-24 and PV-25, but none, except triadimenol for cultivar P-2245, significantly decreased either the rate of disease increase or the final incidence of dead plants.

Additional key words: *Cicer arietinum*, *Fusarium oxysporum* f. sp. *ciceri*, *F. solani*, integrated control, *Macrophomina phaseolina*

Chickpeas (*Cicer arietinum* L.) in Andalucía, southern Spain, are severely affected by a wilt and root rot (WRR) complex that involves mainly a vascular wilt or yellowing induced by biotypes of *Fusarium oxysporum* Schlecht. emend. Snyd. & Hans. f. sp. *ciceri* (Padwick) Snyd. & Hans., a nonvascular yellowing and black collar and root rot induced by *F. solani* (Mart.) Appl. & Wr. or *F. eumartii* Carpenter, and a dry collar and root rot induced by *Macrophomina phaseolina* (Tassi) Goid. (20). Preemergence damping-off associated with these and other fungi has been reported (11,22). Previous surveys in WRR-affected fields have not detected the occurrence of damping-off in Andalucía (20). When susceptible kabuli cultivars (seeds large, rounded, and pale cream) were inoculated with wilt isolates of *F. oxysporum* f. sp. *ciceri*, death of plants occurred 20–30 days after sowing (20). When the same cultivars were inoculated with isolates of *F. eumartii* or *F. solani*, some preemergence damping-off occurred and severe stunting, yellowing, and black collar and root rot were found on emerging plants 20–30 days after sowing (20). These reactions corresponded to the early plant death observed in chickpea crops affected by severe wilt and yellowing and differed from the delayed symptom development in plants infected by vascular yellowing isolates of *F. oxysporum* f. sp. *ciceri* or isolates of *M. phaseolina*. Both early and late symptoms result from the WRR complex (20).

No effective measures are available to chickpea farmers and Andalucía to control the WRR complex. Kabuli cultivars, the only ones grown in Andalucía, are very susceptible to the disease complex (9,20). Crop rotation is limited because few alternative crops are available for long rotations under the dry farming conditions of the area. Fungicide sprays applied by farmers to control Ascochyta blight, which rarely occurs, have been claimed to effect recovery of plants with symptoms of unknown etiology.

Fungicides and resistant cultivars may control the WRR complex. Treatment of chickpea seed with protectant or systemic fungicides has been reported to increase germination and seedling growth and to reduce preemergence damping-off (6,7,11,15,16,19,22). The objectives of this work were to determine the efficacy of fungicide seed dressings, foliar sprays, and moderately resistant cultivars alone or in combination for the control of the WRR complex of chickpeas in Andalucía.

MATERIALS AND METHODS

Trials were conducted in fields with clay soil, pH 7.8–8.1 and about 1% organic matter, at Montilla (Córdoba) in 1980–1981 and at Santaella (Córdoba) in 1982. The field at Montilla had been sown to chickpea for 3 yr and was highly infested with *F. oxysporum* f. sp. *ciceri* (vascular yellowing biotype), *F. solani*, and *M. phaseolina*. The field at Santaella was infested with *F. oxysporum* f. sp. *ciceri* (vascular wilt biotype) and to a

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lesser extent with *F. solani* and *M. phaseolina*; in that field, incidence of the WRR complex was 80% in 1981. Seedbed preparation and applications of insecticides and herbicides were done according to farmers' routine practices. Temperature and rainfall data were obtained from weather stations located 200 and 1,300 m, respectively, from the fields.

Seeds of two local kabuli cultivars, PV-24 and PV-25, and of cultivar P-2245 were used. Seeds of P-2245 were obtained from Y. L. Nene, ICRISAT, Hyderabad, India. The seeds were pale cream, large (>50 g/100), and rugose (PV-24 and PV-25) or small (20 g/100) and slightly rugose (P-2245). In previous trials at the Montilla plot, PV-24 and PV-25 were the least susceptible kabuli cultivars and P-2245 was the cultivar most susceptible to the WRR complex.

Fungicide treatments differed among trials. In 1980 and 1981, only fungicide seed dressings were tested. In 1982, fungicide seed dressings alone or in combination with foliar sprays were studied. Fungicides used in seed dressings were benomyl (Benlate 50WP), captafol (Difolatan 80WP), captan (Captazel 50WP), carbendazim (Sandomil 50WP), carboxin (Vitavax 75WP), copper 8-quinolinolate (Quinolate-400 40F), thiabendazole (Tecto 60WP), thiophanate-methyl 25WP + maneb 50WP (Peltar WP), thiram (Fernide 80WP), triadimenol (Baytan 15D), triadimenol 22D + TIM-5107 3.3D + fuberidazol 3D (unlabeled), and triforine (Funginex 19EC). Benomyl, carbendazim, copper 8-quinolinolate, thiabendazole, and triadimefon (Bayleton 25EC) were also used as foliar sprays in 1982. For seed dressings, all fungicides except triadimenol were applied as a slurry in distilled water by agitating the seeds in a glass jar until the seeds were covered with fungicide (8). For cultivars PV-24 and PV-25, 590 g of seeds (1,000 seeds) were treated with the fungicides (Tables 1–3) in 53 ml of water. For cultivar P-2245, 204 g of seeds (1,000 seeds) were treated at the same rates (Tables 1–3) in 20 ml of water. Triadimenol was applied similarly as a dry powder. When used with other fungicides, triadimenol was applied last just after the treated seeds were air-dried. Seeds were sown shortly after treatment. Untreated seeds were used as controls. Foliar applications were made with aqueous suspensions of the appropriate fungicides applied with a portable, single-nozzle sprayer that delivered 500 L/ha. Controls were sprayed with water.

Treatments were arranged in a split-plot design and replicated as randomized complete blocks. In the 1980–1981 trials, fungicide treatments and cultivars were assigned to main plots and subplots, respectively. Subplots consisted of two rows 0.75 m apart and 5 m long with 75 seeds per row; there were five replicates. Cultivars PV-25 and P-2245 were used in 1980, and PV-24 and P-2245, in 1981. In the 1982 trial, subplots consisted of five rows 0.75 m apart and 4.5 m long with 60

Table 1. Effect of fungicide seed dressing on epidemics of the wilt and root rot complex in two chickpea cultivars at Montilla, Córdoba, in 1980

Fungicide	Rate (g a.i./kg of seed)	Emergence[y] (%)		Final incidence of dead plants[w] (%)		Rate of increase[x]		Time (wk) to $y_i = 0.01$	
		PV-25	P-2245	PV-25	P-2245	PV-25	P-2245	PV-25	P-2245
None (control)	—	51	60	79	100	0.12	0.17	8.0	5.9
Benomyl	3.2	64*[z]	68	76	100	0.14	0.18	8.8	5.9
Captan	3.2	73*	71	81	99	0.14	0.16	8.4	6.5*
Thiabendazole	2.0	58	60	74	99	0.14	0.17	8.8	6.2
Thiram	2.0	67*	63	76	100	0.14	0.18	8.7	6.2
Triadimenol	2.0	65*	52	54*	98	0.14	0.14*	9.8*	—
Benomyl + captan	3.2 + 3.2	78*	70	80	100	0.14	0.18	8.4	6.1
Benomyl + thiram	3.6 + 3.6	67*	70	72	100	0.14	0.18	8.9*	6.2
Thiabendazole + captan	2.0 + 3.2	69*	66	65	100	0.13	0.19	9.3*	6.9*
Triadimenol + thiram	2.0 + 2.0	61	57	62	99	0.15	0.17	9.6*	6.4*
Mean[v]				72 a	99 b				

[y] Stand counts were made 3 wk after sowing. Average of five replicates.
[w] Total cumulative number of dead plants per total number of plants at stand count 13 wk after sowing. Average of five replicates.
[x] Slopes of linear regressions of logits of proportion of dead plants versus time in days (21).
[z] Numbers in same column followed by an asterisk are significantly different ($P = 0.05$) from the control according to Dunnett's test (18).
[v] Numbers followed by same letter are not significantly different ($P = 0.05$) according to Student's t test (18).

Table 2. Effect of fungicide seed dressing on epidemics of the wilt and root rot complex in two chickpea cultivars at Montilla, Córdoba, in 1981

Fungicide	Rate (g a.i./kg of seed)	Emergence[y] (%)		Final incidence of dead plants[w] (%)		Rate of increase[x]		Time (wk) to $y_i = 0.01$	
		PV-24	P-2245	PV-24	P-2245	PV-24	P-2245	PV-24	P-2245
None (control)	—	67	72	68	100	0.09	0.20	6.4	5.5
Captafol	3.2	89*[z]	76	73	99	0.09	0.19	6.5	5.6
Thiram	3.2	78*	69	67	100	0.09	0.19	7.5*	5.8
Benomyl + captafol	3.5 + 3.2	87*	74	66	100	0.09	0.19	7.1	5.7
Benomyl + thiram	3.5 + 3.2	78*	70	60	100	0.09	0.19	7.7*	5.7
Carboxin + captafol	0.9 + 3.2	86*	74	79	100	0.09	0.19	5.9	5.6
Thiabendazole + captafol	2.4 + 3.2	86*	78	67	99	0.09	0.19	6.9	5.7
Thiophanate-methyl + captafol + maneb	1.0 + 2.0 + 3.2	79*	72	50	99	0.09	0.17	7.8*	5.6
Triadimenol + TIM-5107 + fuberidazol + captafol	0.44 + 0.07 + 0.06 + 3.2	83*	73	60	100	0.10	0.21	8.1*	6.1
Triforine + captafol	1.14 + 3.2	84*	76	65	99	0.10	0.19	7.2	5.7
Mean[v]				66 a	99 b				

[y] Stand counts were made 3 wk after sowing. Average of five replicates.
[w] Total cumulative number of dead plants per total number of plants at stand count 12 wk after sowing. Average of five replicates.
[x] Slopes of linear regressions of logits of proportion of dead plants versus time in days (21).
[z] Numbers in same column followed by an asterisk are significantly different ($P = 0.05$) from the control according to Dunnett's test (18).
[v] Numbers followed by same letter are not significantly different ($P = 0.05$) according to Student's t test (18).

seeds per row of PV-25; there were four replicates. Treatments consisted of fungicide seed dressings alone or in combination with foliar sprays. Sprays were applied twice, 6 and 8 wk after sowing. Active ingredients and mode of application were assigned to main plot and subplots, respectively.

The efficacy of treatments for control of the WRR complex was assessed by their influence on epidemic development. Stand counts were made 3 wk after sowing. The increase in incidence of dead plants (total cumulative number of dead plants per total plant number at stand count) over time was obtained by recording and removing dead plants in each plot at weekly intervals from 3 to 13 wk (1980) or from 3 to 12 wk (1981) after sowing. In 1982, the incidence of dead plants was determined in three central rows per subplot at 7- to 12-day intervals from 6 to 13 wk after sowing. At each recording date, a sample of dead plants was used for isolations. Roots and collars were washed in running tap water, cut into pieces 5–10 mm long, surface-disinfested in 1% sodium hypochlorite for 2 min, plated on potato-dextrose agar or water agar, and incubated at 23–27 C for 7 days. Fungi growing from the tissues were identified. Analyses of variance were performed with arc sine transformation of percentages. Mean comparisons were done by the Dunnett's or Student's *t* tests (18).

In the 1980–1981 trials, incidences of dead plants over time for each treatment were transformed by the simple-interest disease (SID, monomolecular) and compound-interest disease (CID, logistic) transformations of Vanderplank (21). Incidence values of 0 or 100% disease incidence were not used in these transformations. Linear regression analyses then were performed with transformed data. Correlation coefficients and distribution patterns of residuals plotted against expected values were used to indicate the appropriateness of a model to describe the data (3). Comparisons of linear regressions were made to detect the influence of fungicide treatments on the increase of incidence of dead plants.

RESULTS

Emergence of seedlings of cultivars PV-24 and PV-25 increased when most fungicides and fungicide combinations were applied as seed dressings in the 1980–1981 trials compared with the controls (Tables 1 and 2). Only thiabendazole and triadimenol + thiram had insignificant effects. No comparisons were made among fungicide treatments themselves, but differences in effectiveness appeared to exist among them. Emergence of cultivar P-2245, which is highly susceptible to the WRR complex, was not increased significantly by any fungicide (Tables 1 and 2). Triadimenol + thiram was phytotoxic and reduced emergence. In the 1980 trial, seedlings of cultivars PV-25 and P-2245 from seed treated with triadimenol or thiram developed chlorotic and narrow leaflets, and these symptoms were more severe when both fungicides were applied together. Similar but milder symptoms were observed in 1981 associated with dressings involving thiram, but no symptoms were observed in the treatment involving a lower dose of triadimenol (Table 2). In both trials, affected seedlings recovered from symptoms by flowering time.

Severe epidemics of the WRR complex developed in the 1980–1981 trials regardless of treatments. Symptoms characteristic of WRR (20) were observed on affected plants. *F. oxysporum*, *F. solani*, and *M. phaseolina* were recovered with similar frequencies from dead plants for both treated and control plots. The logistic transformation gave a somewhat better linear fit for most curves of incidence increase in 1980–1981 than did the monomolecular transformation (Tables 4 and 5) based on the higher correlation coefficients obtained for the regressions. The CID transformation was also determined to be more appropriate based on the pattern of residuals. The overall linear fit by the logistic transformation was better for the most susceptible cultivar, P-2245, than for PV-24 and PV-25. Also, a better fit was obtained for disease incidences from the 1980 trial than for those from the 1981 trial (Tables 1 and 2). Seed dressing with triadimenol was the only treatment that significantly reduced the rate of increase of proportion of dead plants in the 1980–1981 trials, and that effect was shown only for the most susceptible cultivar (Tables 1 and 2). In the 1980 trial, significant delays in the onset of the epidemic were obtained with triadimenol, thiram combined with benomyl or triadimenol, and thiabendazole + captan for cultivar PV-25 and with captan alone or combined with thiabendazole and triadimenol + thiram for cultivar P-2245 (Table 1). In the 1981 trial, however, similar significant delays were obtained only for cultivar PV-24 with thiram alone or combined with benomyl and with the mixtures thiophanate-methyl + captafol + maneb and triadimenol + TIM-5107 + fuberidazol + captafol (Table 2). Nevertheless, only triadimenol in the 1980 trial significantly reduced the final incidence of dead plants of cultivar PV-25 compared with untreated controls by 12–13 wk after sowing (Tables 1 and 2). Average final incidence of dead plants for cultivars PV-25 and PV-24 were significantly lower than for cultivar P-2245 (Tables 1 and 2).

Seedling emergence was not increased nor was the final incidence of dead plants reduced by any fungicide treatment in 1982 (Table 3). Some 1982 epidemics were linearized best by the SID

Table 3. Effect of fungicide treatments on epidemics of the wilt and root rot complex in chickpea cultivar PV-25 at Santaella, Córdoba, in 1982

Treatment					
Seed dressing (g a.i./kg of seed)	Foliar spray[a] (g a.i./100 L)	Emergence[b] (%)	Final incidence of dead plants[c] (%)	Time (wk)[d] to y_t	
				0.1	0.5
None (control)	...	54	89	7.7	10.3
None	None (control)	67	90	7.0	10.0
Benomyl (3.5) + captafol (3.2)	...	70	87	7.7	10.4
Benomyl (3.5) + captafol (3.2)	Benomyl (35)	78	84	7.6	10.6
Carbendazim (2.5) + captafol (3.2)	...	62	84	7.9	10.7
Carbendazim (2.5) + captafol (3.2)	Carbendazim (35)	74	77	7.6	10.7
Copper 8-quinolinolate (0.5) + captafol (3.2)	...	76	81	7.6	10.6
Copper 8-quinolinolate (0.5) + captafol (3.2)	Copper 8-quinolinolate (48)	58	85	7.7	10.6
Thiabendazole (2.4) + captafol (3.2)	...	78	84	7.4	10.3
Thiabendazole (2.4) + captafol (3.2)	Thiabendazole (120)	67	77	7.7	10.9
Triadimenol (0.44) + TIM-5107 (0.07) + fuberidazol (0.06) + captafol (3.2)	...	67	82	7.3	10.4
Triadimenol (0.44) + TIM-5107 (0.07) + fuberidazol (0.06) + captafol (3.2)	Triadimefon (12.5)	74	84	7.6	10.6

[a] Sprays were applied 6 and 8 wk after sowing.
[b] Stand counts were made 3 wk after sowing. Average of four replicates.
[c] Total cumulative number of dead plants/total number of plants at stand count 13 wk after sowing. Average of four replicates.
[d] Determined by interpolation.

transformation, but most epidemics were linearized best by the CID transformation. In 1982, the influence of fungicide treatments on the disease was examined by comparing the time required for the incidence (y) to reach y_t = 0.1 or 0.5 values. Those times were determined by interpolating between consecutive non-transformed values including the given incidence of dead plants. The time to y_t = 0.1 or 0.5 was not delayed by any of the fungicide treatments (Table 3).

DISCUSSION

Seed dressing with several protectant or systemic fungicides used singly, or as mixtures at recommended rates (2,4–7,12,15,22), significantly increased emergence of chickpea seedlings in a naturally infested plot (Tables 1 and 2). However, the effects of fungicide seed dressings on seedling emergence varied with cultivars and years of trial. The emergence of WRR-susceptible P-2245 was not increased by any of the fungicides; however, emergence was increased for the more WRR-resistant PV-24 (20). Some improvement in 1981 over 1980 in the performance of fungicide treatments on seedling emergence could also be due to environmental conditions. Conditions for seed germination and emergence appeared to be better in 1981 than in 1980. Average mean temperatures during the crop season (April to June) were similar in both years, but 115 mm of precipitation occurred after sowing in 1981 and none occurred in 1980. Some of the fungicide treatments tested in the 1982 trial improved seedling emergence compared with the controls but not significantly (Table 3). The experimental plot used in 1982 was infested mainly with the vascular wilt biotype of *F. oxysporum* f. sp. *ciceri* and to a lesser extent with *F. solani* and *M. phaseolina*. In contrast, the plot used in 1980–1981 was highly infested by the latter two pathogens and the vascular yellowing biotype of *F. oxysporum* f. sp. *ciceri*.

Linearization of curves for increase in incidence of dead plants allowed a comparison of effects of fungicide seed dressings on epidemics of the WRR complex in two of the three years of trials. The logistic transformation provided the best linear fit for most of the curves (Tables 4 and 5), as has been found for some other diseases induced by soilborne fungi (1,14,17). However, we do not imply that the logistic growth observed is related to a polycyclic nature of infections but rather that other plant or environmental factors might be involved (1,13).

None of the fungicide treatments provided adequate control of epidemics

Table 4. Regression analysis of the wilt and root rot complex increase in two chickpea cultivars treated with fungicide seed dressings at Montilla, Córdoba, in 1980

		Appropriateness of model for data description							
		PV-25				P-2245			
		SID transformation[x]		CID transformation[x]		SID transformation		CID transformation	
Fungicide	Rate (g a.i./kg of seed)	R^2[y]	Distr. of resid.[z]	R^2	Distr. of resid.	R^2	Distr. of resid.	R^2	Distr. of resid.
None (control)	...	0.74	NA	0.87***[y]	A?	0.87	NA	0.95***	A
Benomyl	3.2	0.58	NA	0.79***	A?	0.81	NA	0.92***	A
Captan	3.2	0.66	NA	0.81***	A?	0.74	NA	0.92***	A
Thiabendazole	2.0	0.74	NA	0.87***	A?	0.76	NA	0.92***	A
Thiram	2.0	0.76	NA	0.98***	A?	0.77	NA	0.95***	A
Triadimenol	2.0	0.76	NA	0.87***	A?	0.66	NA	0.90***	A
Benomyl + captan	3.2 + 3.2	0.74	NA	0.83*	A	0.83	NA	0.95***	A
Benomyl + thiram	3.6 + 3.6	0.52	NA	0.81***	A?	0.77	NA	0.92***	A
Thiabendazole + captan	2.0 + 3.2	0.62	NA	0.81***	A?	0.72	NA	0.92***	A
Triadimenol + thiram	2.0 + 2.0	0.58	NA	0.81***	A?	0.79	NA	0.95***	A

[x] Proportions of dead plants (y) were transformed to $\ln(1/(1-y))$ or $\ln(y/(1-y))$ for SID and CID transformations, respectively (21).
[y] All coefficients of determination (R^2) were significant at $P = 0.01$. Significant differences between coefficients of determination for SID and CID transformations are indicated by * ($P = 0.05$) and *** ($P = 0.001$).
[z] Residuals were plotted against expected values (Distr. of resid.) and observed for discernible pattern (3). Models were considered not acceptable (NA) if a readily discernible pattern was apparent. Acceptable models showed no discernible (A) or some scarcely discernible (A?) pattern.

Table 5. Regression analysis of the wilt and root rot complex increase in two chickpea cultivars treated with fungicide seed dressings at Montilla, Córdoba, in 1981

		Appropriateness of model for data description							
		PV-24				P-2245			
		SID transformation[x]		CID transformation[x]		SID transformation		CID transformation	
Fungicide	Rate (g a.i./kg of seed)	R^2[y]	Distr. of resid.[z]	R^2	Distr. of resid.	R^2	Distr. of resid.	R^2	Distr. of resid.
None (control)	...	0.56	A?	0.66*[y]	A?	0.74	NA	0.92***	A?
Captafol	3.2	0.56	A?	0.64*	A?	0.67	NA	0.88***	A
Thiram	3.2	0.48	NA	0.79***	A	0.69	NA	0.88***	A?
Benomyl + captafol	3.5 + 3.2	0.52	A?	0.69**	A	0.64	NA	0.87***	A
Benomyl + thiram	3.5 + 3.2	0.61	A?	0.71*	A	0.71	NA	0.88***	A
Carboxin + captafol	0.9 + 3.2	0.72	A?	0.72	A?	0.69	NA	0.88***	A
Thiabendazole + captafol	2.4 + 3.2	0.74	A?	0.77	A?	0.72	NA	0.88***	A
Thiophanate-methyl + maneb + captafol	1.0 + 2.0 + 3.2	0.46	A?	0.72***	A?	0.69	NA	0.90***	A?
Triadimenol + TIM-5107 + fuberidazol + captafol	0.44 + 0.07 + 0.06 + 3.2	0.56	A?	0.85***	A	0.66	NA	0.88***	A
Triforine + captafol	1.14 + 3.2	0.59	A?	0.77***	A	0.71	NA	0.88***	A

[x] Proportions of dead plants (y) were transformed to $\ln(1/(1-y))$ or $\ln(y/(1-y))$ for SID and CID transformations, respectively (21).
[y] All coefficients of determination (R^2) were significant at $P = 0.01$. Significant differences between coefficients of determination for SID and CID transformations are indicated by * ($P = 0.05$), ** ($P = 0.01$), and *** ($P = 0.001$).
[z] Residuals were plotted against expected values (Distr. of resid.) and observed for discernible pattern (3). Models were considered not acceptable (NA) if a readily discernible pattern was apparent. Acceptable models showed no discernible (A) or some scarcely discernible (A?) pattern.

of the WRR complex in our trials. Seed dressings with triadimenol at a phytotoxic dosage decreased the rate of increase in incidence of dead plants but was ineffective at a lower dosage. Also, in two of the three years, some fungicide seed dressings appeared to delay the early development of the epidemic but had no effect on final incidence of dead plants (Tables 1 and 2). This result might derive from reduction in the efficacy of the fungicide, changes in plant susceptibility, or occurrence of new infections because of growth of roots into fungicide-free soil (10). A delay in epidemic development would be of interest if proven to be correlated with yield loss (14); however, yield comparisons were not made in this study.

The efficacy of fungicide seed dressings in delaying the early phase of an epidemic was increased with less WRR-susceptible cultivars (PV-24 compared with PV-25) and less severe epidemic development (1980 trial compared with 1981 trial) (Tables 1 and 2). A lack of a significant delay occurred in the 1982 trial. That trial was carried out with the more susceptible PV-25 in a plot heavily infested with a highly virulent vascular wilt isolate of *F. oxysporum* f. sp. *ciceri* (20) and resulted in an average 84% final incidence of dead plants.

Preemergence damping-off and WRR complex were severe, and fungicide seed dressings together with moderate host resistance to WRR increased seedling emergence. Other workers have also found that fungicide seed dressings may increase seedling emergence (6,11,15,16,19) and protect the plant up to 30 days after artificial inoculation (22). However, in agreement with other workers (7,11), we found that these fungicide treatments do not provide control for the WRR complex. Therefore, other measures must be considered, including modification of cultural practices and development of cultivars with a higher level of resistance, to control the WRR complex of chickpea.

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