European Journal of Plant Pathology **107:** 367–375, 2001.

Population dynamics of *Fusarium oxysporum* f. sp. *radicis-lycopersici* in relation to the onset of Fusarium crown and root rot of tomato

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Accepted 2 January 2001

Key words: dissemination, monocyclic, polycyclic, soilborne pathogens

Abstract

Fusarium oxysporum f. sp. *radicis-lycopersici* the causal agent of crown and root rot in tomato comprises two overlapping separate phases: monocyclic and polycyclic. Oversummering inoculum is the source of primary infection (the monocyclic phase) and the spread from plant to plant via root-to-root contact is the source of the secondary infection (the polycyclic phase). In the present work, relationships between initial inoculum density, population dynamics of the pathogen in the root zone of diseased plants, and disease onset were studied. For the monocyclic phase, 55.1% of the variance of disease onset was attributed to the rate of pathogen proliferation in the root zone of plants, and only 12.8% of the variance was attributed to the amount of initial inoculum density. For the polycyclic phase, disease onset was not related to either initial inoculum density or the rate of pathogen proliferation in the root zone. At disease onset, the inoculum density of the pathogen in the root zone of plants infected from oversummering inoculum reached an average of 4.08 log cfu g soil^{-1}. The inoculum density of the pathogen in the root zone of plants infected by their diseased neighbors was 3.23 log cfu g soil^{-1}. A large variation in pathogen proliferation rate in the root zone was found among individual plants, suggesting that differences in the level of soil suppressiveness may occur not only between fields, but even in the same field over short distances.

Introduction

Monocyclic and polycyclic diseases are differentiated in part by the effect of initial inoculum density on epidemic development. Monocyclic diseases are strongly governed by the density and distribution of the initial inoculum. Polycyclic diseases, on the other hand, are influenced mainly by the rates of reproduction and dissemination of the pathogens in space and to a less extent by the density and distribution of the initial inoculum (Campbell, 1986; Fry, 1982). It is generally accepted that many soilborne pathogens cause monocyclic diseases and many foliar pathogens cause polycyclic diseases. Nevertheless, some soilborne pathogens have short generation times and may migrate short distances from infected to healthy plants during a season, therefore causing polycyclic diseases (Fry, 1982).

Many studies have explored the relationship between initial inoculum density and disease incidence caused by soilborne pathogens. Frequently, a direct relationship is found between the two variables. This was demonstrated for various formae speciale of *Fusarium oxysporum* (Ben-Yephet et al., 1994; Harris and Ferris, 1991; Hartman and Fletcher, 1991; Marois and Mitchell, 1981; Marois et al., 1981; Rush and Karaft, 1986), *Pythium* spp. in rye (Mitchell, 1975), and *Phytophthora parasitica* in tobacco (Ferrin and Mitchell, 1986; Kannwischer and Mitchell, 1981) and processing tomatoes (Neher and Duniway, 1991). In some of these studies, plants were grown in containers where inoculum density was usually very high and did

not necessarily reflect field conditions. In a few cases, this relationship was also demonstrated in the field with natural inoculum. For example, for *Verticillium dahliae* in cotton (Ashworth et al., 1979; Paplomatas et al., 1992; Pullman and DeVay, 1982) and cauliflower (Xiao and Subbarao, 1998) and for *F. oxysporum* f. sp. *apii* in celery (Elmer and Lacy, 1987). In other cases, however, final disease incidence was not related to initial inoculum density. For example, for *Phytophthora capsici* (Ristaino, 1991; Ristaino et al., 1992) and *V. dahliae* in cotton (Bejarano-Alcazar et al., 1995).

Initial inoculum density may also affect the time of epidemic onset (Campbell, 1986), although only a few studies have demonstrated this effect. In general, as the amount of initial inoculum increases, disease onset occurs earlier and final disease incidence is higher. Such a relationship has been shown for *V. dahliae* in cotton (Bejarano-Alcazar et al., 1995) and cauliflower (Xiao and Subbarao, 1998) and for *F. solani* f. sp. *pisi* in peas (Rush and Kraft, 1986). Disease onset provides a good estimate of the effect of the pathogen on the host since the earlier the plant becomes infected and wilts, the greater the damage to yield (DeVay and Pullman, 1984; Hawere and Nene, 1980; Navas-Cortes et al., 1998).

The soilborne pathogen *F. oxysporum* f. sp. *radicis-lycopersici* is common in many countries. It causes Fusarium crown and root rot disease in tomato which is characterized by cortical rot at the soil level, vascular discoloration of the lower stem, wilt at fruit ripening, and conspicuous pinkish masses of conidia along the stem (Jarvis, 1988). The disease caused by this pathogen comprised of two separate phases: monocyclic and polycyclic (Rekah et al., 1999b). In the field, first diseased plants are distributed randomly and are infected by oversummering inoculum which is the source of primary infection (the monocyclic phase). The pathogen's ability to spread from plant to plant via root-to-root contact (Rekah et al., 1999b) is the cause of secondary infection (the polycyclic phase). Plants infected from oversummering inoculum (the monocyclic phase) (Van der Plank, 1963), are referred to hereafter as 'focus plants'. Plants infected from their neighbors, via root-to-root contact (the polycyclic phase), are referred to hereafter as 'nonfocus plants' (Rekah et al., 1999b). The two phases may overlap during the growing season. Differentiation between primary and secondary infection cycles of pathogens was also suggested recently for *Gaeumannomyces graminis* var. *tritici* in wheat (Bailey and Gilligan, 1999), *Polymyxa betae* in sugar beet (Webb et al., 1999) and *Ustilago scitaminea* in sugar cane (Amorim et al., 1993). Consequently it is hypothesized that initial inoculum density will govern disease onset in the focus plants as they are part of the monocyclic phase of the disease cycle, but disease onset in nonfocus plants will not be related to initial inoculum density as they are part of the polycyclic phase of the disease cycle.

In this work, we studied the role of initial inoculum density on the onset of Fusarium crown and root rot disease in tomato under field conditions, in relation to the monocyclic and polycyclic phases of the disease. The relationship between the rate of pathogen proliferation in the root zone of plants and disease onset was also studied. A preliminary report has been published (Rekah et al., 1998).

Materials and methods

Field experiments

Experiments were conducted in the Kikar Sedom Experimental Station at 'En Tamar, near the Dead Sea, in the southeastern Israeli desert. Tomato (*Lycopersicon esculentum* Miller cv. 5656) transplants, susceptible to the pathogen, were planted in the first week of October in 1995 and 1996 for the corresponding 1996 and 1997 seasons. Two experimental plots were designated for the 1996 season and four plots for the 1997 season. Each plot consisted of six 11-m long rows; the distance between rows was 1.93 m and plants were spaced 0.5 m apart within rows. The direction of the rows was north–south. The plots were prepared, cultivated and treated with pesticides according to the recommendations of the Extension Service in that region excluding treatments against *F. oxysporum* f. sp. *radicis-lycopersici*. Irrigation was applied via a drip system. The plots were naturally infested with the pathogen and used to monitor the initial inoculum density at planting, changes over time in pathogen population size in the root zone of the plants, disease onset and disease incidence during the growing season.

Relationship between initial inoculum density and disease onset

Initial inoculum density at planting was assessed for a total of 24 randomly chosen plants in two experimental plots in 1996 and for all plants (552 in total) in

the four experimental plots in 1997. Soil samples (ca. 100 g soil each) were taken from the transplant planting cores from a depth of 10 cm. Inoculum density was determined separately for each sample by spreading 0.2-ml aliquots of a 1 : 10 soil dilution with 0.1% water agar or 0.2 g soil on each of 5 petri dishes, containing a modified peptone-pentachloronitrobenzene *Fusarium*-selective medium (FSM) (Gamliel and Katan, 1991). Soil samples from the root zone of infected plants and samples of roots and crowns of diseased plants were taken to confirm the presence of *F. oxysporum* f. sp. *radicis-lycopersici*. The samples were plated on FSM and ca. 150 *F. oxysporum*-like isolates were tested for pathogenicity to confirm the identification of *F. oxysporum* f. sp. *radicis-lycopersici.* Tomato seedlings (cv. Rehovot 13) were inoculated with a spore suspension of each isolate, as previously described (Katan et al., 1991). Most (98%) of the tested isolates were pathogenic to tomato seedlings and therefore considered to be *F. oxysporum* f. sp. *radicis-lycopersici*. Inoculum size was expressed as colony forming unit (cfu) per gram soil.

Disease incidence was determined visually for all plants. A plant was considered diseased when it exhibited typical irreversible wilt symptoms accompanied by brown necrosis on the crown and vascular discoloration on the lower stem (Jarvis, 1988). All plants were monitored every 10–14 days from disease onset, during 170 days of the growing season and the time at which each plant became diseased was recorded. Data for individual plants were used to plot the date of disease onset (in days from planting, y-axis) versus the density of initial inoculum (log cfu g soil^{-1}, x-axis) and to calculate the correlation coefficients for that relationship.

Population dynamics of the pathogen in the root zone

Changes over time in inoculum density of *F. oxysporum* f.sp. *radicis-lycopersici* in the root zone of individual plants were used to calculate the proliferation rate of the pathogen. Calculations were made for 12 plants in 1996 and for 48 plants in 1997. The plants were randomly chosen at the beginning of the season and marked with colored bands. Soil samples were taken from these plants at planting, after 42 days, and thereafter at 10- to14-day intervals until the end of the season, 170 days after planting. At each assessment, soil samples (ca. 100 g soil each) were taken from a different site around the plant at a distance of 10 cm from the stem and from a depth of 10 cm. Soil samples were dug out from 5-cm diameter cores with a trowel. After taking the samples, the sampling cores were recovered. The inoculum density of *F. oxysporum* f. sp. *radicis-lycopersici* was determined for each sample by dilution on FSM as described earlier. Proliferation rate of the pathogen was calculated only for plants that eventually exhibited disease symptoms. The rate was calculated for 49 individual plants in total (9 in 1996 and 40 in 1997), among which 29 were focus plants and 20 were nonfocus plants. Final inoculum density was also recorded for 11 nondiseased plants (controls). Proliferation rate was estimated as follows: Inoculum density in the root zone (log cfu g soil^{-1}, y-axis) was plotted over the time that had passed since planting (days, x-axis). Then, a linear regression was performed for the data points recorded until the plant had exhibited disease symptoms. The slope of the regression equation (log cfu g soil^{-1} day^{-1}) was considered as an estimate of the pathogen's proliferation rate. Of the 49 equations, 34 were significant at $P < 0.05$ (r^2 values ranges from 0.76 to 0.97) and used for further calculating the relationship between the date of disease onset and proliferation rate. This was done for 20 focus plants and 14 nonfocus plants. The remaining equations were not significant, thus, could not be used to estimate proliferation rate. Finally, the relationship between the date of disease onset (days, y-axis) and proliferation rate (log cfu g soil^{-1} day^{-1}, x-axis) was established. Calculations were made separately for the focus plants and for the nonfocus plants.

Results

Relationship between initial inoculum density and disease onset

Initial inoculum density of the pathogen in the soil was determined for samples taken from the planting cores at planting in 1996 and 1997. An example of the spatial distribution of initial inoculum density in two experimental plots in 1997 is presented in Figure 1. Initial inoculum density in the soil varied markedly between planting cores and ranged from undetectable (<1 cfu g soil^{-1}) to 262 cfu g soil^{-1}. The mean number ($\pm$ SE) of *F. oxysporum* f. sp. *radicis-lycopersici* propagules was 14.6 ± 2.6 cfu g soil^{-1}. In the other two plots in 1997, and in the 24 samples taken in 1996, the

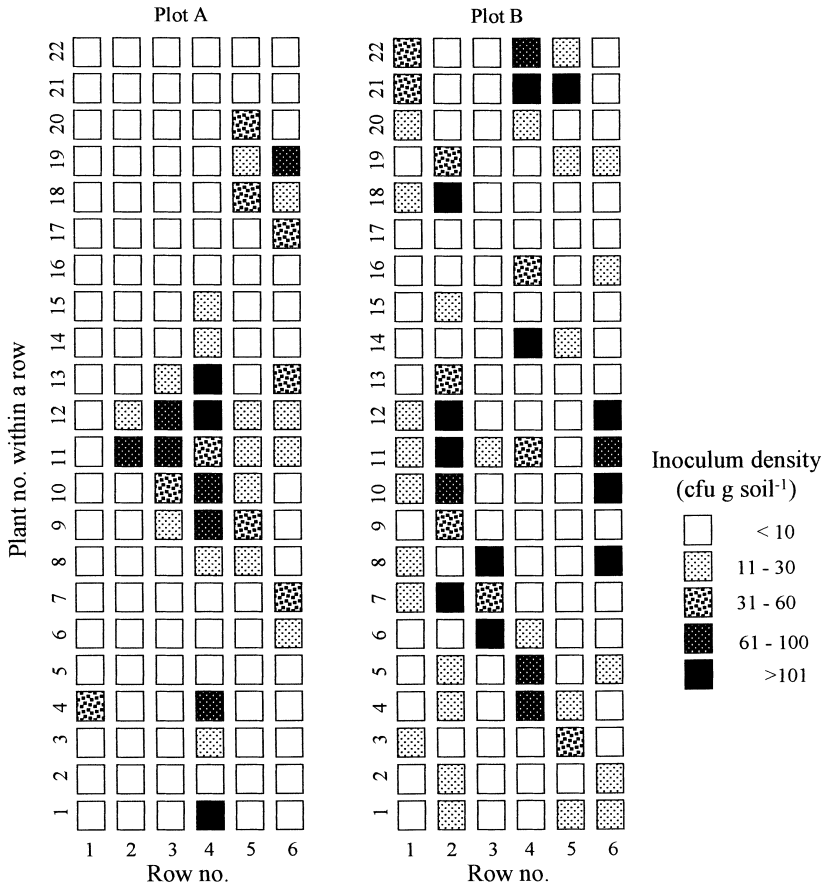

Figure 1. Spatial distribution of initial inoculum density of *Fusarium oxysporum* f. sp. *radicis-lycopersici* on planting day in representative experimental plots in 1997. The plots consisted of six rows (1.93 m apart) with 22 planting cores in each row (spaced 0.5 m apart). Detectable level: 1 cfu g soil^{-1}.

ranges and the mean (± SE) were: <1–345 (24.5 ± 4.5) cfu g soil^{-1}, <1–201 (13.3 ± 2.4) cfu g soil^{-1} and <1–1470 (149.6 ± 71.5) cfu g soil^{-1}, respectively. The relationship between initial inoculum density and disease onset was insignificant in both years (Figure 2). Final disease incidence was 97% and 93% for the 1996 and 1997 seasons, respectively. Similar results were observed when calculations were made separately for each plot or separately for the focus and nonfocus plants (results not shown).

Population dynamics of the pathogen in the root zone

Changes in pathogen inoculum density over time in the root zone were determined for 29 focus and 20 nonfocus plants. At disease onset, the inoculum density of *F. oxysporum* f. sp. *radicis-lycopersici* in the root zone in focus plants reached an average level of 4.08 (±0.08) log cfu g soil^{-1} (values ranged from 2.9 to 4.7) and the frequency distribution of the logarithmic transformed inoculum density resemble normal distribution (Figure 3A). In nonfocus plants, the average inoculum density of *F. oxysporum* f. sp. *radicis-lycopersici* in the root zone at disease onset was 3.23 (± 0.13) log cfu g soil^{-1} (values ranged from 2.3 to 4.3) and the frequency distribution of the logarithmic transformed inoculum density resembled uniform distribution (Figure 3B). Inoculum density in the root zone of plants that did not exhibit visual disease symptoms was lower, 2.73 (± 0.34) log cfu g soil^{-1} (values ranges from 0 to 3.53).

Population dynamics of *F. oxysporum* f. sp. *radicis-lycopersici* in the root zone of three representative

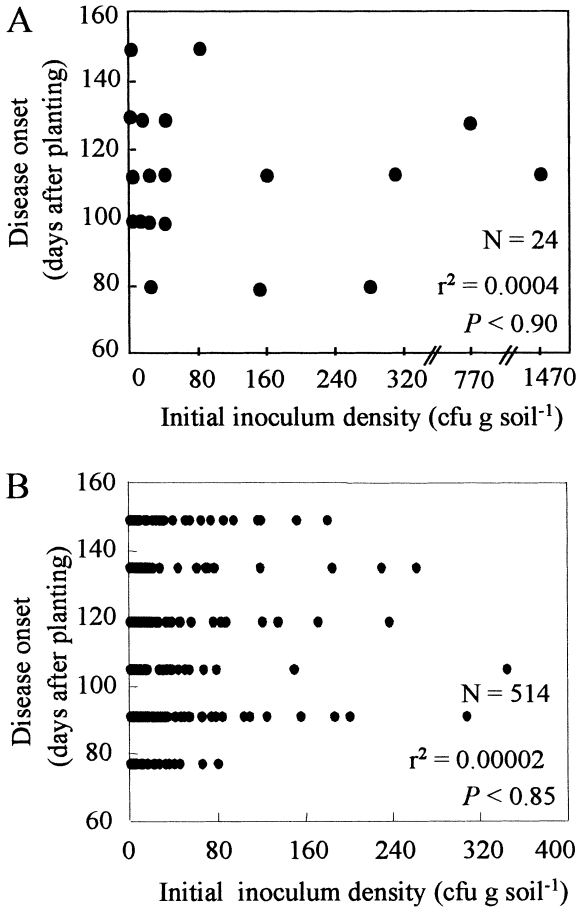

Figure 2. Effect of initial inoculum density of *Fusarium oxysporum* f. sp. *radicis-lycopersici* on the onset of Fusarium crown and root rot in tomato plants in 1996 (A) and 1997 (B). Coincidence between the two variables was insignificant.

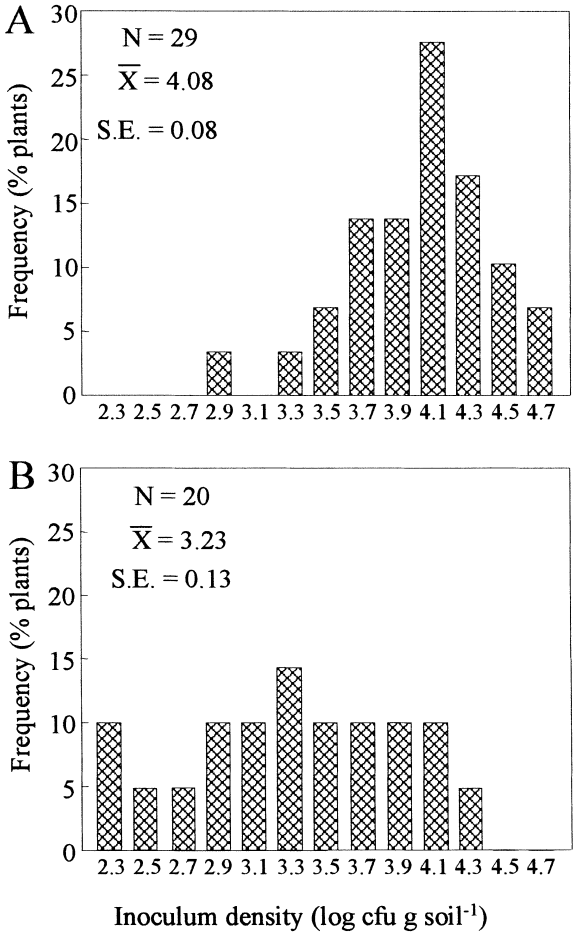

Figure 3. Frequency distribution of inoculum density of *Fusarium oxysporum* f. sp. *radicis-lycopersici* in the root zone of tomato plants at the date of disease onset. (A) focus plants; (B) nonfocus plants.

focus plants is shown in Figure 4. In general, inoculum density increased exponentially over time until the plants began to exhibit disease symptoms; inoculum density did not change markedly thereafter (Figure 4). The slope of the regression equation during the period of population growth was considered as an estimate of the proliferation rate of the pathogen. For the focus plants, a significant nonlinear relationship ($P < 0.01$, $r^2 = 0.54$) was obtained between the time of disease onset and the rate of pathogen proliferation in the root zone (Figure 5A). This relationship was not significant ($P < 0.80$, $r^2 = 0.0054$) for the nonfocus plants (Figure 5B). Multiple regression analysis was employed to quantify the effects of both initial inoculum density (X_1) and rate of pathogen proliferation (X_2) on the date of disease onset (Y) in the focus plants. A power relationship between proliferation rate and the date of disease onset was assumed because this was the type of relationship observed for these parameters (Figure 5A). The resultant equation $Y = \beta_0 + \beta_1 X_1 + \beta_2 X_2^{-0.4} + \varepsilon$ was highly significant ($P < 0.0001$) and accounted for 67.9% of the variance in disease onset. A substantial part of the variability of disease onset (81.1% of the explained variance, 55.1% of the total variance) was attributed to the rate of pathogen proliferation in the root zone. The amount of initial inoculum accounted for only 18.9% of the explained variance (12.8% of the total variance) (Table 1).

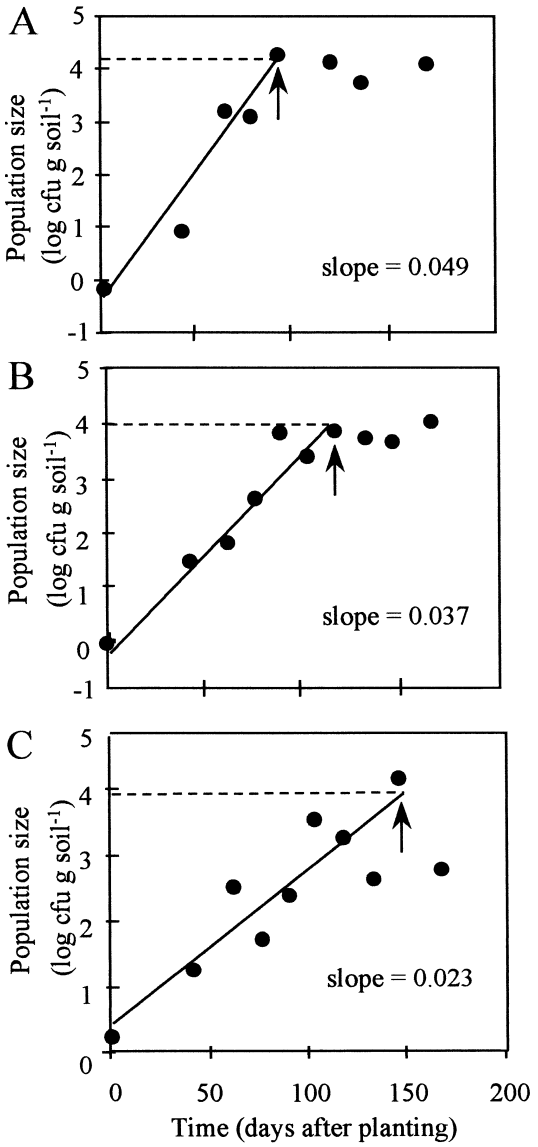

Figure 4. Changes over time in the size of *Fusarium oxysporum* f. sp. *radicis-lycopersici* populations in the root zones of three representative tomato plants. Arrows indicate the time of disease onset. The solid lines represent the regression equations that were fit to the data; slopes represent the rate of pathogen proliferation (log cfu g soil^{-1} day^{-1}) in the root zone of each plant. The dashed lines represent the inoculum density in the root zone of each plant at the date of disease onset.

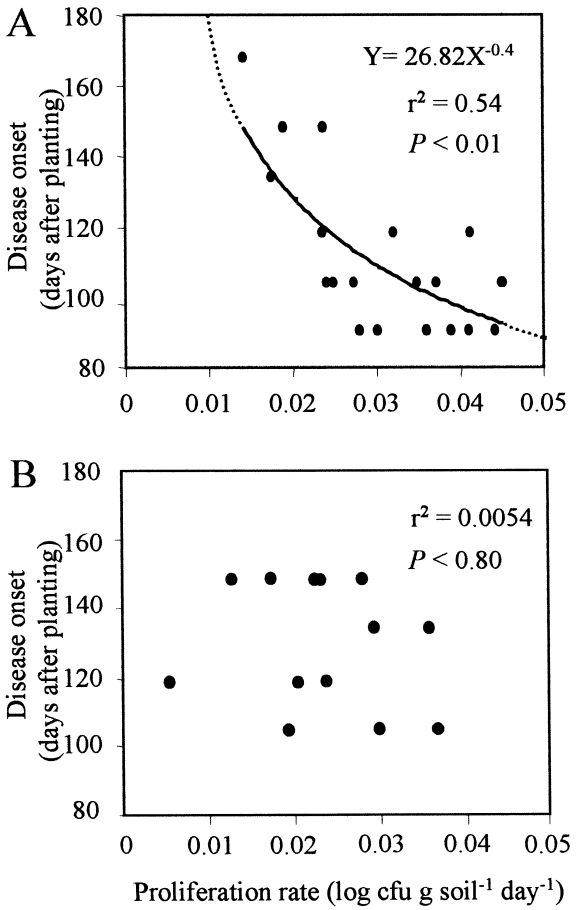

Figure 5. Relationship between the proliferation rates of *Fusarium oxysporum* f. sp. *radicis-lycopersici* in the root zone of tomato plants and the time of Fusarium crown and root rot onset. (A) focus plants; (B) nonfocus plants. The solid line represents the regression equation that was fit to the data; the dashed line is an extrapolation of that relationship.

Discussion

Fusarium oxysporum f. sp. *radicis-lycopersici* is a unique soilborne pathogen with respect to disease development in time and space. Tomato plants may be infected either from oversummering inoculum (primary infection), or from their neighbors via root-to-root contact (secondary infection). The focus plants which appear randomly distributed in the field were most likely infected from the oversummering inoculum, while the nonfocus plants were infected by inoculum originated from adjacent diseased plants (Rekah et al., 1999b). The two phases of the disease overlap later on during the growing season.

Disease onset was not related to the amount of initial inoculum (Figure 2). This finding differs from

Table 1. Analysis of variance for the multiple regression describing the coincidence between initial inoculum density and the rate of *Fusarium oxysporum* f. sp. *radicis-lycopersici* proliferation and the onset of Fusarium crown and root rot disease in tomato*

Source	df	SS	F	Prob > F	R^2
Model	2	7799	17.96	< 0.0001	0.679
Error	17	3689			
Total	19	11488			

Term	Estimate	Std error	t Ratio	Prob > |t|
Intercept	−26.37	24.13	−1.09	0.289
Initial inoculum	−0.25	0.09	−2.75	0.014
Proliferation rate	33.14	5.83	5.69	< 0.0001

*The regression equation was: $Y = \beta_0 + \beta_1 X_1 + \beta_2 X_2^{-0.4} + \varepsilon$, where Y = date of disease onset (days); X_1 = initial inoculum density (cfu g soil^{-1}); X_2 = proliferation rate (cfu g soil^{-1} day^{-1}); $\beta_0, \beta_1, \beta_2$ = regression coefficients. A power relationship between proliferation rate and the date of disease onset was assumed because this was the type of relationship observed for these parameters as illustrated in Figure 5A.

previous studies involving soilborne pathogens where disease onset was related to initial inoculum density (Bejarano-Alcazar et al., 1995; Rush and Kraft, 1986; Xiao and Subbarao, 1998). This difference may be attributed to the various factors that govern the time of disease onset. Wilt symptoms became apparent in the focus plants when inoculum density in the root zone reached a lethal threshold level of 4 log cfu g soil^{-1} (Figure 3A). However, the rate of pathogen proliferation in the root zone varied among individual plants (Figures 4 and 5A). Thus, both the initial inoculum density and the proliferation rate in the root zone of focus plants determined the time at which the inoculum density had reached the lethal threshold level. Of these two factors, proliferation rate accounted for a large portion of the variability in time of disease onset of focus plants (81.1% of the explained variance, 55.1% of the total variance) (Table 1). The amount of initial inoculum density accounted for a small portion of the variability in our study. However, it may be the predominant factor when inoculum density is exceptionally high, or in cases where variation in proliferation rate of the pathogen among plants is very low (e.g., in artificially inoculated plants under controlled conditions). Only two-thirds of the variability of disease onset was explained by the two parameters (Table 1), suggesting that additional factors govern the expression of wilt in an individual plant. Airborne conidia produced by this pathogen (Rowe et al., 1977), which may infect the plants, are one such potential factor (Rekah et al., 1999a).

For the polycyclic phase of the epidemic, disease onset was not related to the density of initial inoculum or to the rate of pathogen proliferation in the root zone (Figures 2 and 5B). Moreover, the inoculum density of the pathogen in the root zone of the nonfocus plants at the time of disease onset was lower than in the focus plants and its frequency distribution resembled uniform distribution (Figure 3B). These findings can be attributed to the fact that disease in nonfocus plants had originated from their infected neighbors via root-to-root contact. Similarly, final disease incidence of polycyclic epidemic caused by zoosporic soilborne pathogens, such as *P. capsici* (Ristaino, 1991; Ristaino et al., 1992) and *P. parasitica* var. *nicotianae* (Kannwischer and Mitchell, 1981), was not related to initial inoculum density. In polycyclic diseases, small amounts of inoculum early in the season may increase rapidly and reach high levels (if conditions are conducive to an epidemic), ultimately causing severe damage (Elmer and Lacy, 1987; Hartman and Fletcher, 1991; Neher and Duniway, 1991; Ristaino, 1991; Ristaino et al., 1992).

The nonlinear relationship between the rate of pathogen proliferation in the root zone of focus plants and disease onset (Figure 5A) indicates that when the proliferation rate is very low (<0.005 log cfu g soil^{-1} day^{-1}), disease will not appear. Moreover, extrapolation of the regression equation suggests that even at very high proliferation rates (>0.07 log cfu g soil^{-1} day^{-1}), disease onset will not become apparent sooner than 66 days after planting. Indeed, our field observations in the last six years confirm this conclusion.

The large variation in pathogen proliferation rate in the root zone among individual plants grown in the same field (Figures 4 and 5) is surprising. This variation suggests differences in the level of soil suppressiveness not only between fields, but even in the same field within the short distances separating adjacent plants. Variations in the level of soil suppressiveness may result from different biotic and abiotic microenvironments or microhabitats in the root zone. These factors are partly or totally excluded or underestimated in experiments carried out under controlled conditions in the greenhouse, especially when the

density of inoculum is high, the inoculum is uniformly distributed in pots, when young seedlings are used, or when the duration of the experiment is short.

Since *F. oxysporum* f. sp. *radicis-lycopersici* behaves as a polycyclic soilborne pathogen under field conditions (Rekah et al., 1999b), data on initial inoculum density cannot serve exclusively in the decision-making process concerning the need for disease management. The separation of the epidemic into monocyclic and polycyclic phases of infection can be used to identify critical stages for intervention. Understanding the role of primary and secondary infections in the field is crucial for planning a holistic approach which includes both preplanting control measures, such as sanitation, disinfestation and biocontrol to suppress the primary inoculum, and postplanting control measures, such as fungicides and biocontrol to suppress the secondary inoculum. These are needed to ensure an economic level of plant health in the entire field.

Acknowledgements

We thank Hillary Voet for her help with the statistics, Sara Erez, Suzan Lourie and Sarit Ben-David for their technical assistance, Abraham Gamliel for valuable suggestions, Talma Katan for providing *F. oxysporum* f. sp. *radicis-lycopersici* isolates and helpful suggestions, and Ami Maduel for technical help in the field experiments. This work was partially funded by the Chief Scientist of the Ministry of Agriculture, Israel.

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