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Modeling of Yield Loss in Potato Early Dying Caused by *Pratylenchus penetrans* and *Verticillium dahliae*

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Abstract: Yield-loss models were developed for potato early dying, caused by an interaction between *Verticillium dahliae* and *Pratylenchus penetrans*. Yield data were collected over 5 years (1985–1989) from potato plants grown in microplots infested with *V. dahliae* and (or) *P. penetrans*. The model $y = b_0 + (1 - b_0)/(1 + [VD/36.7])$, where y was the relative yield (with uninfested controls = 1.0) and VD was the preplant density of *V. dahliae* microsclerotia per cm^3 soil, was fitted to the data set. When *P. penetrans* = 0, $b_0 = 0.55$ (SE = 0.099), and when *P. penetrans* > 0, $b_0 = 0.23$ (SE = 0.035). This model assumed that yield loss was proportional to the concentration of preplant microsclerotia of *V. dahliae*, and only qualitatively related to presence or absence of *P. penetrans*. This study contrasts with previous reports that predict yield loss being proportional to preplant population densities of both *P. penetrans* and *V. dahliae*.

Key words: nematode, nematode fungus interaction, potato early dying, *Pratylenchus penetrans*, *Solanum tuberosum*, *Verticillium dahliae*, yield loss.

Potato early dying (PED) results from infection of potato (*Solanum tuberosum* L.) roots by the soil-borne fungus *Verticillium dahliae* Kleb. and the root lesion nematode *Pratylenchus penetrans* Filipjev & Schuurmans Stekhoven. Potato early dying is a significant limiting factor in potato production (7,10). Disease symptoms include chlorosis and premature vine death during tuber development, often resulting in substantial yield reductions (4,6). Current control options for PED are limited to preplant decisions such as crop rotation, fumigation, or use of cultivars tolerant to *Verticillium*.

Although yield losses in potato can result from infection by *V. dahliae* alone, the combination of *V. dahliae* and *P. penetrans* has been associated with increased symptom development of PED and higher yield reductions than occurs with *V. dahliae* alone (4,6,7). Yield-loss models for PED based on a natural logarithmic transformation of sampled preplant densities of *V. dahliae* and *P. penetrans* added to microplots have been developed (4). Those models were based on the hypothesis that yield loss was proportional to the initial densities of both organisms. An alternative hypothesis is that yield loss is proportional only to population density of *V. dahliae;* however, presence of the nematode leads to a more severe yield loss function than in the absence of *P. penetrans*.

The mechanism of the interaction between *P. penetrans* and *V. dahliae* that results in the synergistic symptom and yield-loss response has been the subject of much speculation. A theory to explain the mechanism includes wounding from nematode feeding, which allows the fungus easier assess to the vascular system (5). This theory would lead to a yield-loss model where both nematode and fungus are treated as quantitative variables. A second theory involves a physiological mechanism where the plant becomes more susceptible to the fungus because of a translocated substance (5). Split root or double root techniques have been utilized to demonstrate that increased symptom development occurs when *V. dahliae* and *Pratylenchus* spp. are placed on separate halves of a root system, as compared to roots inoculated only with *Verticillium* (2,3). However, in both of these examples, the most severe symptom development occurred when *V. dahliae* and *Pratylenchus* spp. were placed on the same half of the root system (2,3). A model to predict yield losses based on a physiological mode of action could be qualitative with respect to the nematode population density or only require some threshold density before the increased sensitivity of the plant to

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Verticillium would occur. We hypothesized a model for a qualitative response of potato to *P. penetrans* in potato early dying. Our objective was to develop a model that predicts yield loss as a function of preplant population densities of *V. dahliae* (microsclerotia) and considers only the presence or absence of *P. penetrans*. A requirement for selecting an acceptable model (beyond normal regression modeling criteria) was that an interaction be found ($P < 0.05$) between the two organisms with respect to yield response.

Materials and Methods

The data set used for model development was collected over five growing seasons (1985–1989) from microplot experiments at two locations in Ohio. Only data from 1985 overlapped that used to develop previous models (4). Treatment yields were averaged over blocks or experiments, depending on experimental design. Inoculum for *P. penetrans* was produced on infested alfalfa (*Medicago sativa* L. cv. P545, Pioneer Hi-Bred International, Johnston, IA) callus in monoxenic cultures (9). Inoculum for *V. dahliae* was produced by growing the fungus in the dark at 20–24 C for 3–4 weeks on minimal medium overlaid with cellophane (4). Details on inoculum production and microplot technique have been published (4). Inoculum densities for *P. penetrans* typically ranged from 0 to 50 vermiform stages per 100 cm^3 soil, though densities up to 165 vermiforms per 100 cm^3 were included in the data set. The nematode density was determined by a pie-pan method (11) after infestation of soil with the nematode. Inoculum densities of *V. dahliae* typically ranged from 0 to 100 microsclerotia per cm^3 soil, though densities up to 139 microsclerotia per cm^3 soil were included in the data set. Density of microsclerotia was determined by direct plating the inoculum on a semi-selective streptomycin-alcohol medium (8) before the inoculum was added to soil. Inoculum was added to fumigated soil and a tuber seed piece of the potato cultivar 'Superior' was planted in each of 15 replicate microplots (10 liters soil/tile). Microplots were open ended, unglazed, clay drain tiles (25 cm inside diameter × 31 cm long). Yield (weight of all tubers in each microplot) was measured and expressed in relative units (y) by dividing each yield value by the mean yield for uninfested microplots. Variables considered for the models were preplant densities of *V. dahliae* (VD) microsclerotia per cm^3 soil, *P. penetrans* (PP) vermiform stages per 100 cm^3 soil, and the interaction between the two (VD × PP).

Results and Discussion

The variable PP alone did not contribute to yield losses in any regression analysis (Fig. 1A), and was excluded as a separate term from the model. The model selected to represent the data was of the general form: $y(VD) = b_0 + (1 - b_0)p(VD)$, where y(VD) is relative yield in relation to VD microsclerotia density level, b_0 represents the minimal relative yield, and p(VD) is a function that describes the form of the yield-VD curve (1). The curve for the mass action law: $p(VD) = 1/(1 + [VD/b_1])$, where b_1 represents VD level at which 50% of the maximum yield loss occurs (1), was chosen based on plots of y versus VD.

The model was fitted to data for cases where PP = 0 (Fig. 1B), and PP > 0 (Fig. 1C) using nonlinear regression. For PP = 0, (108 data points) (Fig. 1B), estimates of b_0 and b_1 were 0.55 (SE = 0.099) and 36.7 microsclerotia per cm^3 soil (SE = 20.8), respectively, with $R^2 = 0.40$. The model was fitted to the data with PP > 0 (194 data points; range of PP was 10–165 vermiform stages per 100 cm^3 soil). When b_1 was fixed at 36.7, then b_0 was 0.23 (SE = 0.035), with an $R^2 = 0.53$ (Fig. 1C). The error mean squares (EMS) did not change substantially from when b_1 was fixed (EMS = 0.028), to the case where both b_0 and b_1 were estimated by nonlinear regression for PP > 0 (EMS = 0.027). The interaction was expressed in terms of b_0, because b_1 was set at 36.7. The minimum yield pa-

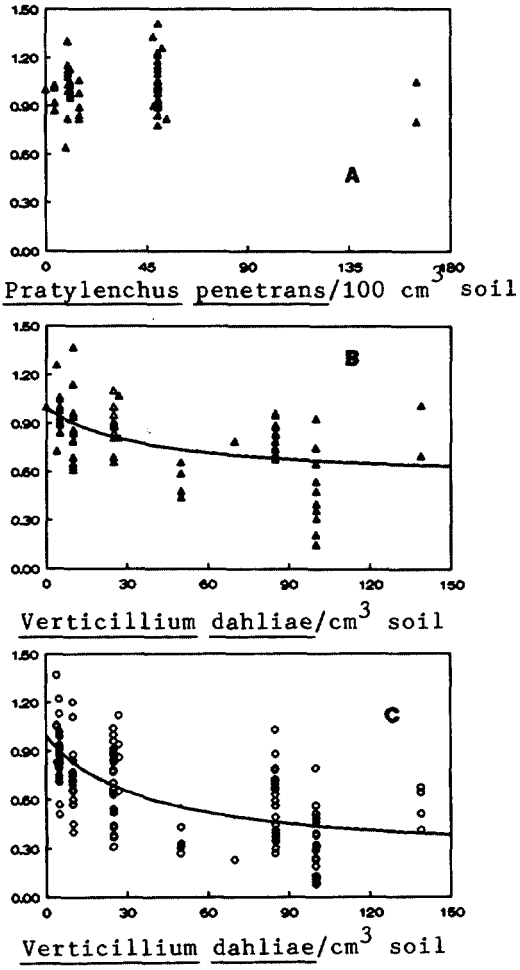

FIG. 1. Relative yields of potato grown in microplots infested with various densities of *Verticillium dahliae* and (or) *Pratylenchus penetrans*. *Pratylenchus penetrans* population density is based on vermiform stages per 100 cm^3 soil at planting. *Verticillium dahliae* population density is based on microsclerotia per cm^3 soil at planting. A) *Pratylenchus penetrans* alone. B) *Verticillium dahliae* alone where the solid line is the predicted yield according to the equation: $y = 0.55 + (1 - 0.55)/(1 + [VD/36.7])$, where y is relative yield and VD is the initial population density of *V. dahliae* per cm^3 soil. C) *Verticillium dahliae* and *P. penetrans* where preplant density of both organisms was greater than 0. The solid line is the predicted yield according to the equation: $y = 0.23 + (1 - 0.23)/(1 + [VD/36.7])$. All densities of *P. penetrans* > 0 result in this yield-loss curve.

rameter (b_0) was significantly lower ($P < 0.05$) when soil was infested with both VD and PP than with VD alone. A combined analysis of variance for the proposed model had an $R^2 = 0.52$, and a lack-of-fit test did not reject the model at $P = 0.05$.

The best linear model for the same data was: $y = 1.02 - 0.037(VD^{1/2}) - 0.056(\log_{10}[\{VD \times PP\} + 1])$, which had an $R^2 = 0.52$, identical to the nonlinear model. This model is the same form as that of previous models (4), except that in this model the square root of VD was taken instead of the logarithm, and preplant levels of *V. dahliae* were represented as microsclerotia added to the soil, not microsclerotia recovered from the soil by wet sieving (4). This was done because of the imprecise recovery of *V. dahliae* microsclerotia by wet sieving. The high variability seen in Figure 1 is typical for this disease.

When establishing the validity of models, biological relationships should be considered. The nonlinear model is consistent with a change in host susceptibility to *V. dahliae* initiated as a result of *P. penetrans* in the roots. Increased susceptibility resulted if *P. penetrans* was present but did not change further as the concentration of the nematode increased. The model proposed previously (4) could be explained based on a wounding phenomenon, where infection by the fungus was facilitated by wounds from *P. penetrans*. The mechanism of the interaction between *P. penetrans* and *V. dahliae* has not yet been determined, but it will be difficult to reject either hypothesis based on microplot studies. The variability associated with PED and with environmental conditions does not allow for the necessary precision to reject either hypothesis without ancillary information. Furthermore, there may be more than one mechanism responsible for the synergistic yield loss response.

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