Verticillium Wilt on Resistant Tomato Cultivars in California: Virulence of Isolates from Plants and Soil and Relationship of Inoculum Density to Disease Incidence

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ABSTRACT

GROGAN, R. G., N. IOANNOU, R. W. SCHNEIDER, M. A. SALL, and K. A. KIMBLE. 1979. Verticillium wilt on resistant tomato cultivars in California: Virulence of isolates from plants and soil and relationship of inoculum density to disease incidence. Phytopathology 69:1176-1180.

Verticillium wilt caused by race 2 of *Verticillium dahliae* is common in California on tomato cultivars with the Ve gene for resistance to race 1. About 47% of 124 isolates of *V. dahliae* taken directly from tomato field soils were race 2; the remaining isolates were either race 1 (43%) or nonpathogenic on tomato (about 10%). In contrast, isolates from diseased tomato plants (race 1 resistant cultivars) from the same fields were predominantly race 2 (about 86% of 153 tested). Race 1 was more virulent than race 2 on cultivars lacking the Ve gene for resistance (susceptible). Average virulence of race 2 isolates was lower on cultivars with the Ve gene than on susceptible cultivars. Incidence of Verticillium wilt (DI) on race 1 resistant cultivars was essentially 100% in 46 fields where soil inoculum density (ID) of total microsclerotia (ms) (race 1 and 2 in undetermined proportions) was about 5.7 ms per gram of soil. In five other fields, however, in which the numbers of race 2 ms were determined, a linear correlation was observed between numbers of race 2 ms (0.0 to 2.0 ms/g of soil) and DI (0 to 100%) when data were plotted arithmetically after conversion of DI % to $\log_e (1/1-DI)$ (slope = 2.0 and r = 0.877). The line for the same data plotted on a $\log_{10}$-$\log_e$-$\log_{10}$ scale had a slope of 1.0 (or 1.57 if assumed to be nonlinear and transformed to $\log_{10}$ before regression analysis) instead of 0.66, as predicted by Baker et al (10) for abstract mathematical Model II. Thus, the models and equivalent interpretations for slopes of lines in arithmetic, $\log_{10}$-$\log_{10}$ and $\log_{10}$-$\log_e$-$\log_{10}$ plots appear to be of questionable validity.

Additional key words: Lycopersicon esculentum.

Wilt of tomato (*Lycopersicon esculentum* L.) caused by *Verticillium dahliae* Kleb. (microsclerotial form of *V. albo-atrum* Reinke and Berth.) was reported from California in 1926 (22). In 1951, Schaible et al (23) reported a high level of resistance to Verticillium wilt in a small-fruited wild tomato from Peru. This resistance, conferred by a single dominant gene (Ve) was incorporated into most tomato cultivars grown in California and for several years was effective in preventing losses from Verticillium wilt. Alexander (1) reported a new race of *V. dahliae* in Ohio pathogenic on resistant tomato cultivars. Subsequently, a similar new race (race 2) was reported in several European countries (21) and, more recently, in North Carolina (11). In California, isolated instances of infection by *V. dahliae* in race 1 resistant cultivars of tomato were reported in 1972 by Hall and Kimble (15).

In this study, we determined the inoculum density (total microsclerotia [ms]) of *V. dahliae* in field soils and the relationship of inoculum density (ID) to the incidence of Verticillium wilt (DI) on race 1 resistant cultivars. The populations of *V. dahliae* in five other fields were analyzed quantitatively for races and strains with different virulence on resistant and susceptible cultivars to determine the relationship between virulence and ID of race 2 ms in soil and disease incidence.

MATERIALS AND METHODS

Field surveys. In 1973 an extensive field survey of the major areas of California producing processing tomato was undertaken to determine the extent of Verticillium wilt caused by race 2 of *V. dahliae* on race 1 resistant cultivars. Fields in which the disease was found were recorded, but no attempt was made to determine the percentage of diseased plants in each field. The disease was detected by examination of plants for foliar symptoms and vascular discoloration of the stem and by isolation of the pathogen from samples of discolored stems. Cultures of *V. dahliae* obtained from these isolations were maintained on potato-dextrose agar (PDA) slants and tested in the greenhouse for pathogenicity on susceptible and race 1 resistant cultivars.

Soil sampling and assay. Soil samples were collected from processing-tomato fields planted to race 1 resistant cultivars in the Sacramento and San Joaquin valleys of California. Soil collections were made near the end of the growing season (usually within the last month before harvest) from three random rows per field. Ten 25-cm deep soil cores were taken from each row (at 10–15 m intervals) with a 2.5-cm diameter soil tube, and the 30 cores from each field were bulked into a composite sample. The samples were returned to the laboratory, air-dried for 15 days at 20–27 C, pulverized in a revolving jar mill (13) for about 25 min in a cold room, and assayed for microsclerotia (ms) of *V. dahliae* using a modification (18,19) of the wet-sieving technique (4,16). Triplicate 15-g samples from each composite sample were wet-sieved through 125- and 38-μm sieves, and the soil residues retained by the second sieve were treated with 0.5% NaOCl (2) and cultured in 15 plates containing modified pectate agar (18,19). After incubation for 12 days at 22–24 C, the soil was washed from the surface of the medium and the microsclerotial colonies were counted with a dissecting microscope. The number of propagules per gram of soil was calculated by assuming that each microsclerotial colony originated from a single propagule. To obtain cultures, agar blocks were taken from selected microsclerotial colonies and ms were separated from the medium by grinding the agar block with a pestle and mortar. The sucrose flotation technique of Huisman and Ashworth (17) also was used occasionally to isolate ms directly from soil samples. The separated ms were suspended in sterile distilled water and collected on a 14-μm Millipore filter by vacuum filtration. The ms then were treated with 0.5% NaOCl for 5 sec, rinsed with sterile distilled water, and transferred individually into culture plates containing water agar (WA). Single spores from selected colonies were transferred onto PDA slants.

Determination of disease incidence. The incidence of Verticillium wilt in tomato fields was determined at the same time that soil samples were collected for determination of ID. Three

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hundred plants in each sampled field, ie, 10 plants around each site from which a soil sample was taken, were examined for foliar symptoms of Verticillium wilt. Plants with foliar symptoms were examined further for vascular discoloration at the base of the stem. Only plants with both foliar symptoms and vascular discoloration were recorded as "diseased." Infection by *V. dahliae* was confirmed in the laboratory by isolation of the pathogen from samples of discolored stems plated on WA. Cultures from these isolations (referred to as tissue isolates) were single-spored and maintained on PDA slants for use in pathogenicity tests.

Virulence of isolates on race 1 resistant and susceptible tomato cultivars. Tomato cultivars Earlypak 7 (susceptible) and Pakmor (race 1 resistant) were used as indicators in greenhouse inoculation tests. Other cultivars also were used occasionally. Seeds of each cultivar were sown in flats containing sand. After 8–10 days, seedlings with fully expanded cotyledons (before true leaves had developed) were uprooted and inoculated by the root-dip technique; roots were washed with running water, trimmed to about 2 cm, and placed for 10 min into a suspension containing about 10^5 conidia per milliliter. The inoculated seedlings were transplanted into plastic trays containing U.C. soil mix (5). Each of five replicated trays contained five seedlings for each isolate to be tested for pathogenicity. Noninoculated controls (dipped in water or autoclaved conidial suspension) and controls inoculated with known race 1 or race 2 isolates were included in each test.

Trays were arranged in a randomized complete block design on greenhouse benches maintained at 18–25 C, and plants were observed daily for symptoms. For routine race differentiation of isolates, the primary criterion for pathogenicity was wilting of cotyledons within 1–2 wk after inoculation. In tests to determine a comparative virulence index (VI), plants were maintained in the greenhouse and observed for symtoms for 5 wk. The primary criterion for virulence was wilting and/or yellowing of leaves accompanied by various degrees of stunting. Typically, seedlings with wilted cotyledons after 1–2 wk developed leaf symptoms 2–4 wk after inoculation. Seedlings inoculated with a few isolates, however, developed temporary wilting of cotyledons; the plants later recovered, with no leaf symptoms or apparent effect on growth after 5 wk. Such isolates were considered nonpathogenic. In a few other instances, inoculated plants developed leaf symptoms without previous cotyledon wilting at the seedling stage; these isolates were considered pathogenic.

As used here, pathogenicity is defined as the ability of an isolate to induce symptoms, regardless of severity. Isolates pathogenic on the susceptible cultivar but nonpathogenic on the race 1 resistant cultivar were classified as race 1; isolates pathogenic on both the susceptible and the race 1 resistant cultivars were classified as race 2.

Severity of symptoms, determined by reduction in dry weight of inoculated plants compared with noninoculated controls, was used to determine the VI of an isolate on a cultivar. At the end of the 5-wk observation period, the plants were cut at ground level and weighed after drying for 5 days at 100 C. The VI of isolates was calculated with the formula:

$$VI = 1 - \frac{\text{dry wt of inoculated plants}}{\text{dry wt of controls}}$$

Isolates that caused no reduction in dry weight of inoculated plants received a virulence rating of V = 0; the highest rating was V = 1.0 (all plants killed).

RESULTS

Relationship between inoculum density and disease incidence. ID and DI were determined during the 1974 and 1975 growing seasons in 46 commercial tomato fields in the San Joaquin and Sacramento valleys of California. All fields were planted to race 1 resistant cultivars; cultivar VF 145-B7879 was the most common. Verticillium wilt was found in all sampled fields.

In 1974, ID of *V. dahliae* in 24 sampled fields ranged from 0.05 to 41.2 ms per gram of soil, with an average of 5.9 ms per gram. In 1975, the average ID in 22 sampled fields was 7.5 ms per gram of soil, with a range from 0.13 to 46.6 ms per gram. The incidence of Verticillium wilt was essentially 100% in all fields where the ID of race 1 and 2 in undetermined proportions was 4.5 and 7.2 ms per gram of soil in 1974 and 1975, respectively.

In five other fields, however, in which the proportion of race 2 isolates was determined, the numbers of race 2 ms within the range of 0.0 to 2.0 ms per gram of soil was correlated (r = 0.816) with DI (%) within the 0–100% range (Table 1 and Fig. 1A). An arithmetic plot of ms numbers vs. DI transformed to $\log_e(1/1-DI)$ showed good fit to a straight line with a slope of 2.2 (r = 0.877) that passed very close to the origin (Fig. 1B). (An analysis of the same data with a computer program designed to force the straight line of Fig. 1B through the origin [0,0] showed that the slope of the line is 1.998.) There was no apparent correlation, however, between DI and numbers of total ms (r = 0.101) or race 1 ms (r = −0.314). The percentage of race 2 ms was highest in the three fields with the highest DI (ie, fields 30, 7, and 26), and the percentage of race 2 ms also was correlated with DI (r = 0.899) (Table 2). The straight line of the same data plotted $\log_{10}$-$\log_e$-$\log_{10}$ had a slope of 1.0 (or 1.57 if assumed to be nonlinear and transformed to $\log_{10}$-$\log_{10}$ before regression analysis) instead of 0.66, as predicted by Baker's (6–10) abstract mathematical Model II (Fig. 1D).

Comparative pathogenicity of soil and tissue isolates. A total of 124 soil isolates and 153 tissue isolates were tested in the greenhouse on the two indicator cultivars, and their pathogenicity was determined. Most isolates were collected from six processing-tomato fields with various ID-DI levels.

TABLE 1. Pathogenicity of isolates of *Verticillium dahliae* on two indicator cultivars of tomato

Pathogenicity[a]			Number and source of isolates in each group[b]	
Earlypak 7	Pakmor	Designation	From soil	From plant tissue
−	−	Nonpathogenic	12	1
+	−	Race 1	53	21
+	+	Race 2	59	131
−	+	...	0	0

[a] Based on symptom development during a 5-wk observation period. Earlypak 7 was used as the susceptible and Pakmor as the resistant indicator.

[b] Tissue isolates were collected from stems of field-grown tomato with typical Verticillium wilt symptoms, and soil isolates were obtained by screening microsclerotia directly from soil samples collected from the same fields.

TABLE 2. Correlation between disease incidence and numbers of race 2, race 1, and total microsclerotia per gram of soil

	Microsclerotia per gram of soil				Disease incidence	
Field[a]	Total	Race 1	Race 2[b]	% Race 2	(%)	$\log_e(1/1-DI)$[c]
30	2.8	1.1	1.7	60	98	3.912
7	4.2	3.2	1.0	25	57	0.844
26	0.7	0.1	0.6	83	86	1.966
15	2.7	2.3	0.4	14	32	0.386
10	0.8	0.7	0.1	11	4	0.041
r for DI-ID						
DI (%)	0.188	−0.180	0.816	0.872		
DI($\log_e[1/1-DI]$)	0.101	−0.314	0.877	0.899		

[a] Six fields were sampled, but one with 41.2 ms/g of soil (race 2) and a DI of 100% was eliminated because the ID was superfluous.

[b] Number of race 2 ms in soil as calculated (total ms/g × % race 2 ms in soil × 100) for each field.

[c] Multiple infection correction transformation of DI (%) to $\log_e(1/1-DI)$.

The isolates were divided into three groups based on pathogenicity on the two indicator cultivars (Table 1). Group I isolates were nonpathogenic on both indicators; about 10% of the soil isolates were in this group, but only one of the 153 tissue isolates was nonpathogenic on both indicators. These isolates also were nonpathogenic on susceptible cultivars Bonny Best and Pearson and on the race 1 resistant cultivars VF 145-B7879 and Roma VF. Group II isolates were pathogenic on the susceptible cultivar Earlypak 7 but nonpathogenic on the race 1 resistant cultivar Pakmor and were classified as race 1; about 43% of the soil isolates and 14% of the tissue isolates were in this group. Group III isolates were pathogenic on both the susceptible and the resistant cultivar and were classified as race 2. About 47% of the soil isolates were in this group, but the percentage of tissue isolates was much higher, about 86%. No soil or tissue isolates were pathogenic on Pakmor and nonpathogenic on Earlypak 7.

Comparative virulence of isolates. The soil and tissue isolates in each pathogenicity group were tested to determine VI on each of the two indicator cultivars based on reduction in dry weight of inoculated plants.

All isolates nonpathogenic (based on failure to induce symptoms) on either the susceptible or the race 1 resistant indicator also failed to reduce significantly the dry weight of inoculated plants. Likewise, none of the race 1 isolates reduced significantly the dry weight of the race 1 resistant cultivar Pakmor. The same race 1 isolates, however, reduced the dry weight of the susceptible cultivar Earlypak 7. Essentially no difference was noted in the virulence of race 1 isolates from soil and from plant tissue, the average virulence being 0.648 and 0.626, respectively. Both groups of isolates showed a wide range of virulence, but most isolates were within the two middle virulence ranges, ie, 0.41–0.80 (Tables 3 and 4).

Isolates of race 2 from soil and plant tissue reduced the dry weight of both the susceptible and the race 1 resistant cultivar, and there were essentially no differences in VI between soil and tissue isolates. Both groups of isolates had similar patterns of distribution into virulence groups (Tables 3 and 4). The average VI on the susceptible cultivar Earlypak 7 was 0.544 for soil and 0.552 for tissue isolates. The average VI on the resistant cultivar Pakmor was 0.492 for soil and 0.484 for tissue isolates.

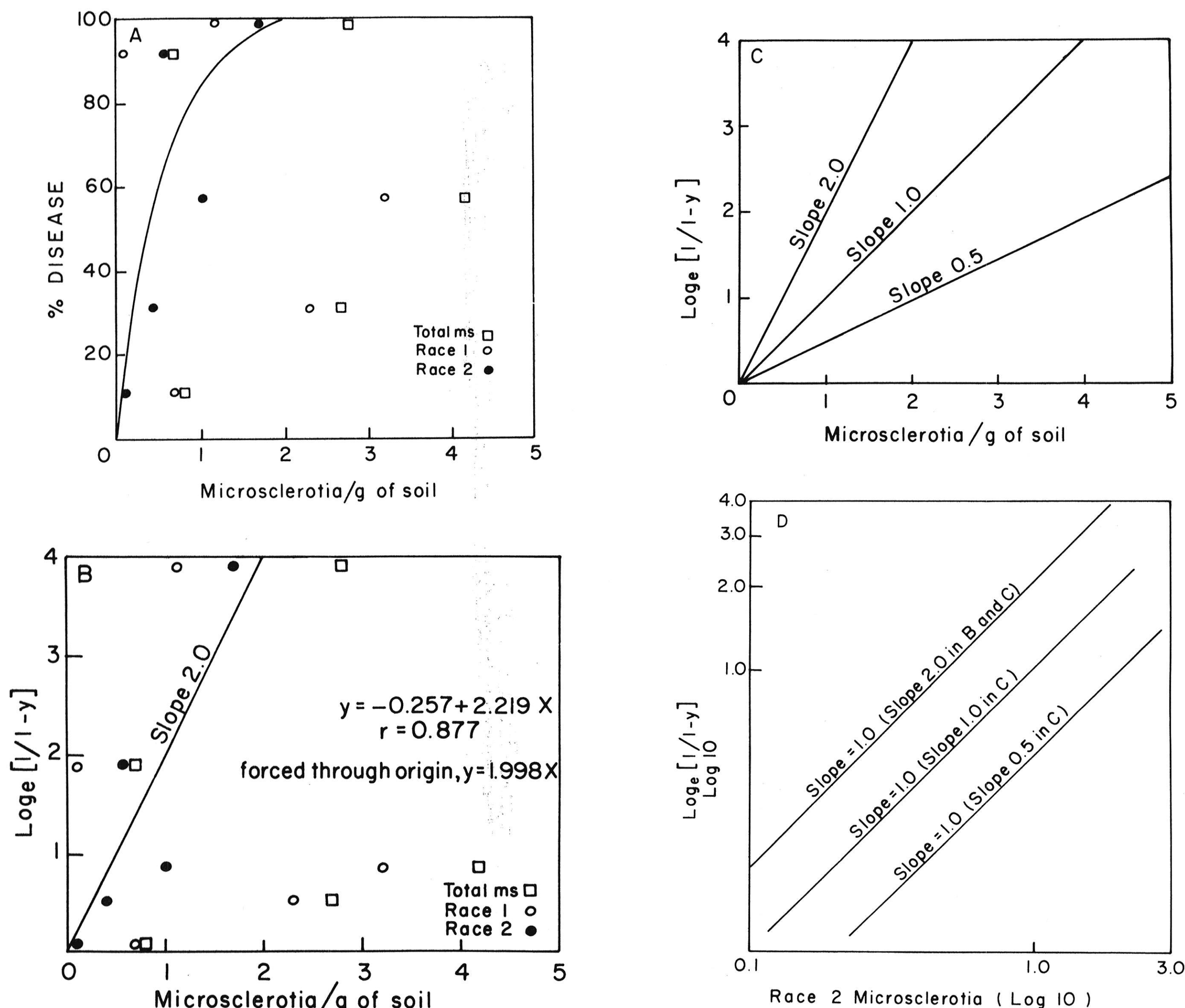

Fig. 1. Relation of inoculum density (ID) of Verticillium microsclerotia to disease incidence (DI): **A**, arithmetic plot; **B**, arithmetic plot after transformation of DI (%) to DI ($\log_e$ [1/1 −DI]) to correct for multiple infections; **C**, same type of plot as in **B** but with slopes of 2.0, 1.0, and 0.5; **D**, plot with the $\log_{10}$-$\log_e$-$\log_{10}$ transformation of same data for the three slopes of lines in **C**. The ID-DI relationship indicated in this type of plot is the same as in **B** and **C**, but the **D** plot is more difficult to evaluate visually because the lines do not pass through the origin. Furthermore, all three lines have a slope of 1.0 if the ID-DI relationship in plot **C** is linear. The formula for the slope 1.0 line in plot **C** as plotted in **D** is $\log_{10}$-$\log_e$-$\log_{10} = 0 + 1.0 \log_{10}X$; the formulas for the other lines are the same except for the different y-intercept values.

DISCUSSION

The widespread distribution of race 2 of *V. dahliae* in California became apparent only a few years after the first isolated instances were observed (15). Contamination of clean fields with infested debris or soil from a few infested fields seems an unlikely cause of such rapid and extensive spread. Furthermore, we have been unable to show transmission of either race 1 or race 2 with tomato seed (authors, *unpublished*). *V. dahliae* was reported to be seedborne in safflower (20), and both tomato races of *V. dahliae* can infect safflower and are transmitted in the seed as microsclerotia (authors, *unpublished*). This finding, however, cannot explain the apparently rapid spread of race 2 throughout the tomato production areas of the state, because safflower is used only to a limited extent as a rotation crop with tomatoes.

Race 2 is much more prevalent among tissue isolates from race 1 resistant cultivars. Inasmuch as the infected plant tissues are the source of ms, which are later released into the soil (14,18,19), the use of race 1 resistant cultivars exerts a selection pressure on the soil population of *V. dahliae* that favors an increase of race 2 in the soil. This suggests that low levels of race 2 have existed within the population of *V. dahliae* for some years and that the population of race 2 has increased because of the selection pressure exerted by extensive use of race 1 resistant cultivars.

Despite the extensive use of race 1 resistant cultivars for about 20 yr, race 1 of *V. dahliae* is still prevalent in tomato fields of California (Table 1). Microsclerotia of *V. dahliae* survive passively in soil for long periods (24). Weeds (25) and rotation crops may also perpetuate race 1. The factor that perhaps contributes most to the long-term survival of race 1, however, is its ability to infect race 1 resistant cultivars of tomato without causing symptoms (12). In our study, about 15% of the isolates of *V. dahliae* from race 1 resistant cultivars were race 1 (Table 1). This percentage may not represent the actual potential of race 1 to infect resistant cultivars, as all the tissue isolates were taken from plants with wilt symptoms; race 1 isolates infecting apparently healthy plants were thus excluded.

Some of the soil isolates were nonpathogenic on both the susceptible and the resistant tomato cultivars (Table 1). These isolates may have been perpetuated in the soil by the same means as that proposed for race 1.

Race 2, on the average, was less virulent than race 1 on cultivars that do not have the Ve gene for resistance (Tables 3 and 4). Race 2, by definition, can attack both susceptible and race 1 resistant cultivars, but its average virulence on resistant cultivars was somewhat less than that on susceptible cultivars, suggesting that the ability of race 2 to overcome the Ve gene is associated with a loss of virulence on susceptible cultivars. We wish to emphasize, however, that the differences refer only to average virulence values. Individual isolates within each race differed widely in virulence on both susceptible and resistant cultivars, as shown by the distribution of isolates into virulence groups (Tables 3 and 4). Because the limits set for the virulence groups are arbitrary, there is a continuum of virulence among the isolates from nonpathogenic on susceptible cultivars to highly virulent on race 1 resistant cultivars. Apparently, each of the two tomato races of *V. dahliae* is comprised of many strains (groups of isolates), with different degrees of virulence on cultivars with or without the Ve gene for resistance.

A 25% reduction in the yield of race 1 resistant processing-tomato cultivars was observed under conditions of 100% incidence of Verticillium wilt caused by race 2 of *V. dahliae* (authors, *unpublished*). Because tomato fields with essentially 100% disease incidence are common in California (more than half the 46 fields surveyed in 1974 and 1975), the overall yield loss suffered by the processing-tomato industry is considerable. The effort to find a reliable source of resistance to race 2 must be continued and intensified. Perhaps, screening programs should attempt to find satisfactory levels of tolerance based on dry weight or yield reduction instead of lack of symptoms in the seedling root-dip test.

A significant positive correlation was noted between total numbers of soilborne ms and DI in the 46 fields surveyed, and fields with more than about 6 ms per gram of soil were essentially 100% diseased by the end of the growing season. A similar relationship in cotton was reported by Ashworth et al (3) and by Butterfield (13). The correlation between total ms and DI of race 1 resistant cultivars is fortuitous, however, and probably results from the inclusion of a number of fields with 100% DI and a large ID of race 2 ms (superfluous inoculum) in the analyses. It does show that, if inoculum is not limiting, environmental factors usually are not sufficiently limiting to prevent the common occurrence of 100% Verticillium-diseased plants in California fields.

Baker (6,7,9) cited the Verticillium wilts of mint, cotton, and tomato as typical examples of abstract mathematical Model II (10), in which inoculum is fixed and root tips moving through the soil activate a larger portion of the inoculum than do the nonmoving infection courts of Model I. Baker thus assumes that "inoculum density would not be as significant a factor in disease severity for motile infection courts as for those that are fixed" (10). The data in Table 2, plotted in Figs. 1A and 1B, show, however, that inoculum levels of race 2 between 0 and 2 ms per gram of soil did indeed limit DI, and interpolation from Fig. 1B indicates that DI 50% was about 0.5 ms per gram of soil. Also, Fig. 1B shows that the ID-DI relationship, when inoculum is within a limiting range, is arithmetically linear, indicating that a single ms can be infectious and cause systemic symptoms. Inasmuch as ID was estimated as ms per gram of soil and DI as percentage of diseased plants, we cannot determine the numbers of ms or infections per plant that resulted in DI 50%. Thus, the slope 2.0 in Fig. 1B can be interpreted only with regard to relative efficiency of inoculum. As shown in Fig. 1C, each increment of inoculum for slope 2.0 influences DI more than do comparable increments of inoculum for slopes 1.0 and 0.5. No other biologic interpretations for these slopes can be made, however. If this is so, will a $\log_{10}$-$\log_e$-$\log_{10}$ type of plot provide additional information and allow other interpretations of the ID-

TABLE 3. Distribution into virulence groups of isolates of *Verticillium dahliae* (race 2) from soil[a]

Virulence on Pakmor	Virulence on Earlypak 7: 0–0.20	0.21–0.40	0.41–0.60	0.61–0.80	0.81–1.0	Total
0–0.20	0[b]	0	2	2	0	4
0.21–0.40	1	2	7	3	3	16
0.41–0.60	0	3	8	7	4	22
0.61–0.80	2	1	5	2	1	11
0.81–1.0	0	2	3	1	0	6
Total	3	8	25	15	8	59
Average virulence on Pakmor						0.492
Average virulence on Earlypak 7						0.544

[a]Virulence was rated on a scale from 0 to 1.0, where 0.0 represents no reduction of the dry weight of the two indicator cultivars Earlypak 7 (susceptible) and Pakmor (resistant) and 1.0 represents death of all plants.
[b]Number of isolates in each virulence group.

TABLE 4. Distribution into virulence groups of isolates of *Verticillium dahliae* (race 2) from plant tissue[a]

Virulence on Pakmor	Virulence on Earlypak 7: 0–0.20	0.21–0.40	0.41–0.60	0.61–0.80	0.81–1.0	Total
0–0.20	1[b]	4	2	2	0	9
0.21–0.40	2	10	15	16	1	44
0.41–0.60	1	5	19	12	6	43
0.61–0.80	3	2	5	11	4	25
0.81–1.0	0	2	2	4	2	10
Total	7	23	43	45	13	131
Average virulence on Pakmor						0.484
Average virulence on Earlypak 7						0.552

[a]Virulence was rated on a scale from 0 to 1.0, where 0.0 represents no reduction of the dry weight of inoculated plants of the two indicator cultivars Earlypak 7 (susceptible) and Pakmor (resistant) and 1.0 represents death of all plants.
[b]Number of isolates in each virulence group.

DI relationship? According to Baker et al (10), abstract mathematical Model II predicts that slop values of $\log_{10}$-$\log_e$-$\log_{10}$ plots of ID-DI data (Fig. 1D) should be 0.66 and that deviation from this value results from the effects of environment on symptom expression. A slope of 0.66 in the Fig. 1D type of plot indicates that the ID-DI relationship in Fig. 1B is nonlinear as per the formula:

$$\log_{10}[\log_e(1/1-DI)] = 0.66 \log_{10}X$$

We contend, however, that the ID-DI relationship in Fig. 1B is linear as per the formula:

$$\log_{10}[\log_e(1/1-DI)] = 1.0 \log_{10}X$$

and, therefore, have plotted in Fig. 1D three lines all with slope 1.0 that correspond with the slopes 0.5, 1.0, and 2.0 in Fig. 1C. Regression analysis after $\log_{10}$-$\log_{10}$ transformation of the ID-DI data (Table 2) for race 2 ms used in Fig. 1B resulted in the formula:

$$\log_{10}[\log_e(1/1-DI)] = -0.034 + 1.57 \log_{10}X$$

which indicates that the slope of the upper line in Fig. 1D should be 1.57. Whether the slope in Fig. 1D is 1.0 or 1.57, however, no additional information can be obtained or other interpretations of the ID-DI relationship made. The slopes have the same limitations as those noted for the slopes of the lines in Figs. 1B and 1C, and relative efficiency of inoculum is more difficult to visualize because the lines cannot be plotted through the origin.

Furthermore, slopes of lines in straight arithmetic, $\log_{10}$-$\log_{10}$, and $\log_{10}$-$\log_e$-$\log_{10}$ plots apparently have been considered equivalent, inasmuch as the same interpretations (0.66, 1.0, and $>$ 1.0 = rhizoplane, rhizosphere, and synergism, respectively) have been applied to all three types of plots (6–10). Obviously, they cannot be equivalent because each different mathematical transformation of a set of data results in a different slope of line in each of the plots. We believe there is ample reason to raise serious questions concerning the biologic relevance of the abstract mathematical models and interpretations of slopes of data curves from plots of transformed data.

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