Disease Incidence Gradient and the Effect of Seedbed Infection on Potato Virus Y Outbreaks in Tobacco

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ABSTRACT

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Veinbanding caused by potato virus Y (PVY) has become widespread in Chilean tobacco, affecting flue-cured, burley, and oriental types. Severe losses have resulted from a necrotic strain of PVY. A disease incidence gradient was determined by regression analysis ($\log_e I = \log_e a - b \log_e X$) of disease incidence over distance in meters from a line-focus source of weeds growing along irrigation ditches. Results indicated that weeds may be important sources of PVY and/or breeding sites for aphid vectors. Incidence of PVY in tobacco seedlings just before transplanting varied from 1 to 19%; however, final incidence seemed to be highly dependent on other factors, presumably aphid vector activity.

Additional key words: epidemiology, *Nicotiana tabacum*

Veinbanding, also called "necrosis severa," caused by potato virus Y (PVY) has become widespread and is currently considered the most damaging disease affecting flue-cured, burley, and oriental tobaccos in Chile (7). Severe outbreaks in the 1978–1979 and 1979–1980 growing seasons were associated with a necrotic strain of PVY (PVY^N) that causes chlorotic mottling and veinbanding followed by vein necrosis and necrotic lesions on midribs of leaves and stems. Leaf dwarfing, distortion, and rapid dieback were commonly found among dieased plants affected by PVY^N.

The overwintering sources of PVY for tobacco have not yet been established under Chilean conditions. Nevertheless, it has been assumed from the literature that the primary sources of inoculum may be other solanaceous crops, primarily potatoes, tomatoes, or peppers (2,10,11). Weeds may also play an important role as virus reservoirs or as overwintering sites for insect vectors (1,11). For instance, a common solanaceous weed, nightshade (*Solanum gracile* Link), was found to be an important host of PVY in Florida, and disease gradients were established in pepper fields away from a line-focus source of infected nightshades (8,9). In Chile, a higher disease incidence was observed near irrigation ditches in 1980–1981 where numerous annual, biennial, or perennial weeds grew year-round. This study was undertaken to establish the spread patterns of PVY and evaluate the significance of natural seedbed infection on overall disease incidence.

MATERIALS AND METHODS

Field observations on the distribution of tobacco veinbanding were obtained from five fields of tobacco: *Nicotiana tabacum* L. 'Coker 86' (field E), '347'

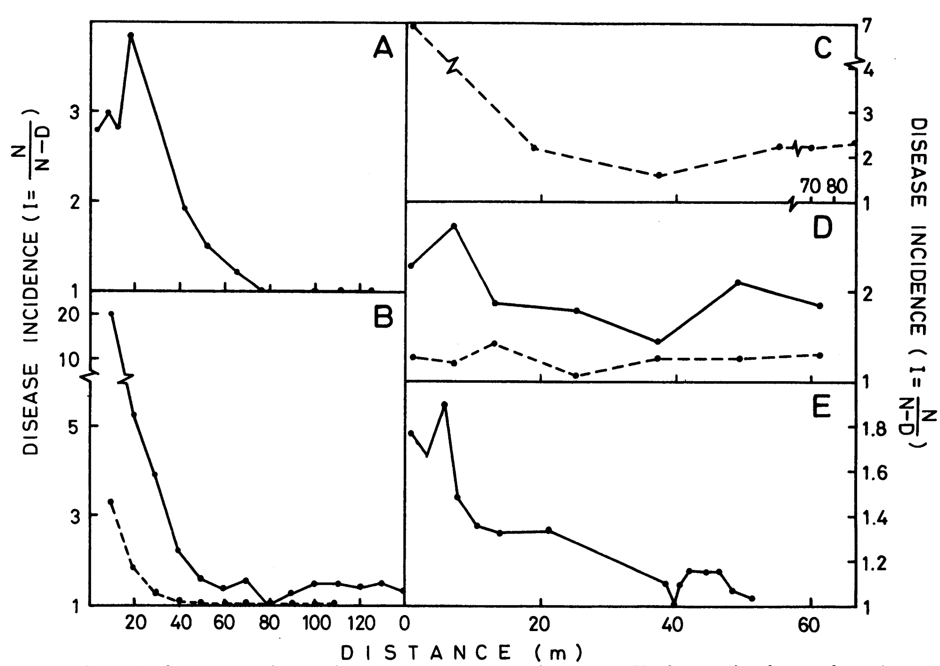

Fig. 1. Spread of tobacco veinbanding, caused by potato virus Y (PVY), from a line focus of weeds growing along irrigation ditches: **(A and B)** tobacco cultivar Coker 347 plots with a single line-focus source of weeds, **(C)** tobacco cultivar NC-744 with two line-focus sources of weeds, **(D)** tobacco cultivar Coker 258, and **(E)** tobacco cultivar Coker 86 with single line-focus sources of weeds. I = disease incidence; D = number of diseased plants in a row out of a total of N plants. Solid lines are the total number of diseased plants and broken lines are plants infected by the necrotic PVY-strain.

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(fields A and B), '258' (field D), and 'NC-744' (field C). The latter has been considered resistant to the necrotic strain in North Carolina (4). Disease incidence was recorded when plants were near flowering about 8–10 wk after transplanting. Observations were made on each plant in a row, varying between 106 and 187 plants per row. Rows were about 57–92 m long and ran parallel to the line focus of weeds. At least 10 rows, 110 m wide were considered in each field. Rows in field A (Fig. 1A) were perpendicular to the focus of weeds; therefore, blocks every 10 m away from the weeds were considered. Identification of PVY was based on the characteristic symptoms caused by the mild and necrotic strains. The mild strain causes mild mottling and veinbanding. Necrotic lesions on veins, midribs, or stems and leaf distortion and stunting were symptomatic of PVY^N. The presence of PVY on five to 10 leaf samples from each field was confirmed by immunodiffusion tests (3) in plates containing 0.8% (w/v) agarose, 0.3% sodium dodecyl sulfate, 1% sodium azide, and 0.85% sodium chloride.

The relationship between disease incidence and distance from the line focus of weeds was examined by regression analysis according to the following model: $\log_e I = \log_e a - b \log_e X$ (5,6), where X = distance in meters from the line focus of weeds and I = disease incidence estimated by the multiple-infection transformation according to Gregory (5,6):

$I = \frac{N}{N - D}$, where D is the number of diseased plants out of a total of N plants in each row.

To evaluate the possible effect of an early seedbed infection, 180 plants in 1980–1981 and 100 plants in 1981–1982 were taken at random from commercial seedbeds of Coker 86, 258, and 347 tobacco established on soils fumigated with methyl bromide (453.6 g/m^2). Plants were potted individually and maintained for 2 mo in an aphidproof greenhouse. Final veinbanding incidence was estimated from more than 1,000 plants about 1 wk after symptoms appeared. The presence of PVY was determined serologically (3).

RESULTS

Spread. The patterns of spread of tobacco veinbanding are given in Figure 1 as disease incidence (I) at a distance from a line focus of weeds considered potential sources of PVY. Fields A, B, C, and E had a single line focus of weeds about 1–3 m wide, primarily composed of *Amaranthus* spp., *Apium* spp., *Brassica nigra* (L.) Koch, *Cestrum parqui* L'her., *Chenopodium glaucum* L., *Cichorium intybus* L., *Conium maculatum* L., *Convolvulus arvensis* L., *Datura stramonium* L., *Digitaria sanguinalis* (L.) Scop., *Foeniculum vulgare* Mill., *Lactuca serriola* L., *Malva* sp., *Melilotus officinalis* (L.) Lam., *Mentha* sp., *Picris echioides* L., *Plantago lanceolata* L., *Portulaca oleracea* L., *Rubus ulmifolius* Schott, *Sorghum halepense* (L.) Pers., *Taraxacum officinale* Weber, and *Vicia* spp. Field D had weeds along two opposite edges of the field, parallel to the planting rows and of a similar composition to the other fields.

PVY^N was found in fields B, C, and D, and the spread patterns are shown by a broken line in Figure 1B, C, and D, respectively. In these fields, the solid line represents the total number of plants showing veinbanding, either mild or necrotic. In field C (NC-744), incidence was 100% by the time this evaluation was conducted. We were only able to determine the spread of PVY^N in this field.

Results of regression analysis of disease incidence over distance are given in Table 1. The calculated values of $\log_e a$ varied from 0.71 to 4.49 for the tobacco planting with the lowest and the highest incidence of veinbanding (mild + necrotic strains), respectively. The scaling factor *a* ($\log_e a$) for the necrotic strain varied from 0.24 to 2.0 ($\log_e a$ values may vary from $\log_e 0 = 0.0$ to $\log_e 100 = 4.6$). Disease incidence gradient rates (*b*) were all negative values that varied from −0.002 to −0.08. Values of *b* approaching zero were recorded in field D, −0.002 to −0.08, where more than one line-focus source of weeds was found, and consequently there was a more uniform level of disease throughout the plot.

Highly significant correlations were obtained for data in fields A, B, and E, where disease incidence decreased rapidly with distance (Table 1; Fig. 1A,B,E).

Seedbed infection. None of the 900 Coker 86 plants were infected with PVY at the time of transplanting in 1980–1981; however, 65 of 700 plants, with incidence varying from 1 to 19%, were infected before transplanting in the seedbed in the 1981–1982 growing season (Table 2). Symptoms developed about 2 wk after moving the transplants to the greenhouse, primarily characterized by veinbanding and chlorotic mottling of the leaves. No necrotic symptoms were observed at this stage. Plants were kept for 40 days before they were discarded.

Disease incidence under field conditions varied from 1 to 32.8% in 1980–1981 and from 12% to 67% in 1981–1982 (Table 2). No necrotic symptoms were observed in 1980–1981. The presence of PVY both in

Table 1. Regression analyses of the spread of tobacco veinbanding from a line focus of weeds, considered a potential source of potato virus Y (PVY)

Field	Cultivar	PVY-strain[a]	*a*[b]	*b*[b]	*r*[c]
A	Coker 347	Mild	1.96	−0.39	−0.86*
B	Coker 347	Mild + necrotic	4.49	−0.91	−0.92*
		Necrotic	2.00	−0.46	−0.91*
C	NC-744	Necrotic	1.75	−0.22	−0.70
D	Coker 258	Mild + necrotic	0.90	−0.08	−0.52
		Necrotic	0.24	−0.002	−0.03
E	Coker 86	Mild	0.71	−0.17	−0.93*

[a] Based on symptom expression in the field; plants infected with the mild strain developed chlorotic mottle, veinbanding, or veinclearing but not vein necrosis or stem necrosis, as did plants infected with the necrotic strain.
[b] Regression coefficient *a* is the $\log_e a$, according to the regression model $\log_e a - b \log_e X$, where X = distance in meters from the line focus of weeds; *b* is the disease gradient rate; *a* is considered a scaling factor, and I is the disease incidence calculated from $I = \frac{N}{N-D}$, where D = diseased plants out of N (total number).
[c] Correlation coefficient; * = significant at $P = 0.01$.

Table 2. Evaluation of seedbed infection on the final incidence of potato virus Y (PVY) in tobacco

			Disease incidence (%)	
Field	Location	Cultivar	Seedbed[a]	Field[b]
1980–1981				
1	San Esteban	Coker 86	0	32.8
2	Cerrillos	Coker 86	0	3.7
3	Catemu	Coker 86	0	1.4
4	Rinconada	Coker 86	0	1.0
5	Nogales	Coker 86	0	2.4
1981–1982				
6	Catemu	Coker 258	10	58.0
7	Catemu	Coker 86	19	67.0
8	San Esteban	Coker 347	6	42.0
9	Catemu	Coker 86	1	31.0
10	Romeral	Coker 86	13	14.0
11	Romeral	Coker 86	12	12.0
12	Rinconada	Coker 86	4	28.0

[a] Calculated from a random sample of 180 plants in 1980–1981 and 100 plants in 1981–1982. Each plant was potted and kept under aphidproof greenhouse conditions for 2 mo.
[b] Estimated from more than 1,000 plants in each field; includes mild + necrotic PVY-strains.

seedbed plants held in the greenhouse and diseased plants in the field was confirmed serologically.

DISCUSSION

Results indicated a gradient of tobacco veinbanding from the line focus of weeds, which indicates that weeds play an important role in the epidemiology of the disease. Weeds may act as virus reservoirs and/or overwintering sites for aphid vectors. Similar results have been reported for veinbanding of peppers and potatoes (1,8,9). Several of the plant species found along the irrigation ditches are known hosts of PVY, and aphids, primarily *Myzus persicae* Sulzer, were found associated with biennial or perennial plants, eg, *C. parqui* and *F. vulgare*. Additional research, however, is needed for a better understanding of the significance of weeds in the epidemiology of tobacco veinbanding.

The disease incidence gradients were relatively steep and fit well with Gregory's model (5,6) for line focus source of inoculum, but the b values were relatively larger than the theoretical disease gradient rates described by Gregory (5). No evidence of contamination by inoculum from outside sources other than the hypothetical were found after analyzing our data for fields A, B, and E. This reinforces our belief that tobacco veinbanding spreads gradually from the edges into the field. There were no signs of a random distribution. The lack of a significant correlation and relatively low disease gradient rates in fields C and D (Table 1) could be attributed to the presence of more than one source of inoculum and/or to a saturation effect caused by a relatively high disease incidence. The distribution patterns of PVY^N in field C and the total PVY-infected plants observed on field D (Fig. 1) approached a "U" shape, characteristic for a double source of inoculum according to Gregory (5).

The presence of PVY in tobacco seedbeds before transplanting was demonstrated symptomatically and confirmed by the serological assays in 1981–1982. In the 1980–1981 growing season, final disease incidence depended exclusively on outside sources of inoculum. The substantial difference between field 1 and the other fields (Table 2) can be partially attributed to the presence of tomato fields next to field 1. Fields 2, 3, 4, and 5 were surrounded by nonhost crops like oats, wheat, corn or alfalfa.

Although some plants were infected at transplanting in 1981–1982, the final disease incidence was dependent on other factors, presumably aphid activity. This was particularly significant in fields 10 and 11 (Table 2), where veinbanding incidence did not increase in the field, although 13 and 12% of the plants were infected before transplanting, respectively. No significant correlation was obtained between the initial seedbed infection and the final incidence of veinbanding observed in the field. Based on these results and also considering that the spread pattern of PVY in these fields followed the trends described previously, seedbed infection seems to be secondary in importance to the development of tobacco veinbanding epidemics.

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