Development of Bean Rust Epidemics in a Field Planted with Alternate Rows of a Resistant and a Susceptible Snap Bean Cultivar

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ABSTRACT

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In 1986 and 1987, half of a 0.4-ha field was planted with a cultivar of bean plants susceptible to Race 38 of the bean rust pathogen *Uromyces appendiculatus*. In the remaining half of the field, every other row was planted with the same susceptible cultivar alternating with a resistant cultivar on which no pustules were produced. The middle row of the susceptible cultivar in each half of the field was inoculated approximately 15 days after planting. On subsequent dates, counts of pustules on plants from rows equidistant from the inoculated row in the two halves of the field were compared. Generally, there were fewer pustules per plant in the half of the field with the two cultivars in alternating rows than in the half with only the susceptible cultivar. In 1986, the cumulative number of pustule days per plant in the alternating planting was about 30% less than in the half planted entirely to the susceptible cultivar. In 1987, this difference was about 6% less. The relatively small effect of the alternate row planting in reducing bean rust was probably a result of high rates of self-infection of plants. Dispersal gradients for urediniospores of *U. appendiculatus* are initially very steep and include a long, relatively flat tail, characteristic of a power law. Such a dispersal gradient not only allows plants far from a disease focus to become infected, but also strongly favors self-infection of susceptible plants. As bean plants grew and filled the space between rows, spores produced on lower leaves probably were even more likely to be deposited on the plant on which they were produced than to be carried to other plants. As a consequence, a small initial benefit of the alternate planting tended to decline with increasing number of generations of the pathogen.

Additional keywords: cultivar mixtures, spore dispersal.

Several studies have demonstrated the efficacy of mixtures of resistant and susceptible crop cultivars in reducing the rate of buildup of rust or mildew diseases of small grains (1,5–9,12,15,16). There have been few studies with mixtures of relatively large plants, such as snap beans, however. In one study (10), mixtures of susceptible and resistant cultivars of both beans and corn were shown to reduce the amount of disease compared with a planting of entirely susceptible plants, although the effect was smaller than generally found for mixtures of small grains. This reduced effectiveness for mixtures of corn or bean plants compared with small grains agrees with studies (11,12) that indicate that a mixture should become less effective in controlling disease as the size of a unit of the susceptible genotype area is increased.

The effectiveness of a host mixture for reducing the buildup of a foliar plant disease epidemic results mainly from its ability to reduce the amount of inoculum falling on susceptible plants (1). This effective dilution of the inoculum produced in a field can occur in two ways. First, dilution can occur simply because there are fewer susceptible plants per ground area, and the potential for multiplication of inoculum during each generation of the pathogen is less (8). This planting density effect (1) is largely independent of the presence of resistant plants in the mixture and can be achieved simply by increasing the space between the plants. Second, resistant plants can act as barriers to inoculum spread. Such barriers can directly intercept spores and keep them from reaching susceptible plants. In addition, they create a physical separation. This distance-barrier can keep susceptible plants from touching each other and can allow turbulent dispersion in the atmosphere to dilute the spores in the air traveling between potential source and receiver plants.

In mixtures of small grains, plants of the susceptible cultivar are grown close together, and diseased leaves from different plants of the susceptible variety can intermingle and touch. If this occurs, the barrier effect of the mixture is obviously reduced. For certain crops it may be desirable for purposes of harvesting and marketing to plant a single cultivar per row and achieve diversity in a field by alternating rows, or strips of several rows, of resistant and susceptible cultivars, or by intercropping different species of plants. For crops such as beans, which are usually grown in relatively wide rows, such a planting scheme would maintain an inter-row (but not within-row) barrier throughout the course of an

epidemic. This alternate-row type of planting might be advantageous, particularly if rows can be planted perpendicular to the prevailing direction of the wind. Unfortunately, the relatively large area of a unit of the susceptible genotype, due both to the size of an individual bean plant and to the length of a row, allows considerable potential for self-inoculation of susceptible plants, which would tend to reduce the effectiveness of such a planting for controlling disease (1,9–12). The purpose of the present study is to examine the effect of barrier rows of resistant bean plants planted between rows of susceptible bean plants on the development of a bean rust epidemic.

MATERIALS AND METHODS

Crop and inoculum. Experiments were conducted in a 0.4-ha bean (*Phaseoli vulgaris*) field at Mt. Carmel, CT, from July to September of 1986 and 1987. Planting was on 23 July (day of year = 204) in 1986 and on 16 July (day of year = 197) in 1987. Beans were planted in 0.8-m-wide rows oriented east to west. The field was planted in two halves (Fig. 1), with each half of the field containing 78 rows. In both years, every row in the east half of the field was planted entirely to the susceptible cultivar Bush Blue Lake 47 (BBL 47). The west half of the field was planted in alternate rows to BBL 47 and to the resistant cultivar Bush Blue Lake 94 (BBL 94); BBL 47s were planted in even-numbered rows counting from the center inoculated row (Row 0). The two halves of the field were separated by four, 0.8-m-wide, north-south rows of the resistant BBL 94. Resistance and susceptibility refer to the host response to Race 38 (14) of *Uromyces appendiculatus* (Pers.) Unger (syn. *U. phaseoli* (Pers.) Winter). Race 38 induces susceptible infection-type pustules and urediniospores on BBL 47. On BBL 94, it induces necrotic spots but no urediniospores are produced.

The entire length of the center row (Row 0) of susceptible BBL 47s in each half of the field was inoculated on day 219 and 224 in 1986, and on day 210 and 215 in 1987, by spraying the plants with a water suspension of *U. appendiculatus* (Race 38) urediniospores. By day 230 in 1986 and by day 220 in 1987, pustules were erupting along the entire length (26 m) of the two inoculated line sources. An estimate of the uniformity of the inoculation along each source row was obtained in 1986 by comparing the number of pustules on three randomly chosen plants from comparable locations in the east and west half of the source row, and by comparing counts on five plants chosen from Row 2 in the southeast and southwest and in the northeast and northwest quadrants when disease spread was first assessed on day 240, before secondary infection occurred on these plants. In 1987, the uniformity of the inoculation was estimated by comparing the number of pustules on 10 plants randomly chosen on days 223, 225, 229, 231, and 236 from the source row in each half of the field, and by comparing counts in Row 2 at the first assessment of disease spread (day 237) in the same manner as in 1986.

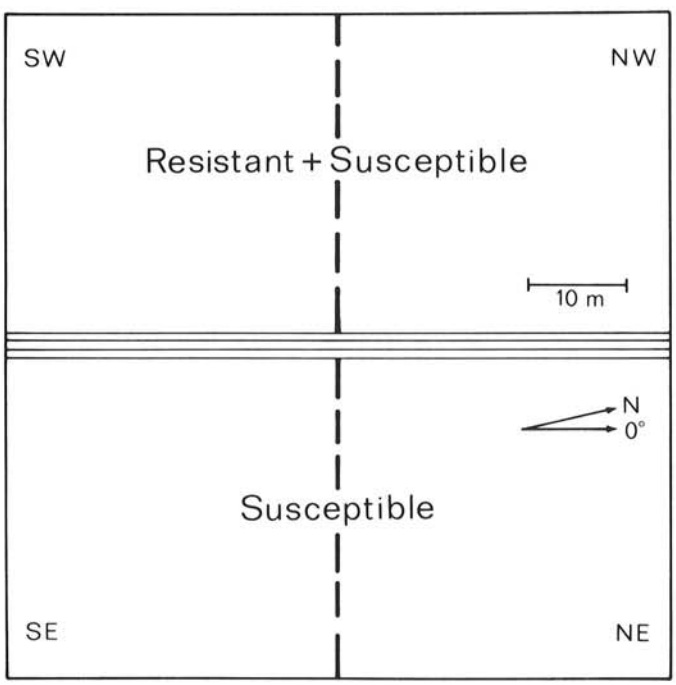

Fig. 1. Schematic of the bean field used to study bean rust increase. Heavy dashed line in the center of the figure represents the inoculated row. Polar angles for wind direction were measured in field coordinates, in which 0° was along a line perpendicular to the direction of the rows of susceptible bean plants and about 12° east of true north.

Disease assessment. The amount of disease present on a given date was determined by counting the number of pustules on the center leaflet of all trifoliolate leaves of entire (susceptible BBL 47) plants. Counts on the primary leaves were included in 1987. Counts from individual leaves were recorded in the order they were attached to the plant, starting at the bottom of the plant. From 220 to 520 leaflets were counted for each quadrant, depending on the growth stage of the crop at the time of sampling. For each sampling date, evaluations began with the rows farthest from the source and ended with rows nearest the source, in an attempt to reduce artificial spread of inoculum. Three to five plants were selected for counting from each sampled row, one each from distances of about 5, 10, 15, 20, and 25 m from the east or west edge of the field. The plants were cut at ground level, put in a plastic bag, and removed from the field for counting. The assayed plants were always obtained from corresponding even-numbered rows in the southeast and southwest quadrants and in the northeast and northwest quadrants.

For the final two determinations in 1987, the number of pustules per leaflet was estimated by counting the number of pustules within three-to-five standard areas (1.5 cm^2) on each leaflet. The sample areas were selected by placing a rectangular window cut from an opaque mask randomly on the leaf. In addition, the width of each leaflet was measured, and the area of a sampled leaflet was estimated using the regression equation between area and leaf width given in the next section. Finally, the number of pustules per leaflet was obtained by multiplying the mean pustule density by the area of the leaflet divided by the standard area.

These measurements gave estimates of the number of pustules (on the center leaflet of trifoliolate leaves) on plants within specified rows in each quadrant on several different dates during the course of the epidemic. The effect of the alternate rows of resistant plants on slowing the epidemic was determined by comparing counts of pustules in corresponding rows in the southeast (entirely susceptible) and southwest (alternate resistant-susceptible) quadrants and likewise for the northeast and northwest quadrants. By analogy to the area under the disease progress curve (11,13), I calculated cumulative pustule days (*PD*) for corresponding rows in the east and west quadrants using:

$$PD = (1/2) \sum_{i=1}^{N-1} (P_i + P_{i+1}) (D_{i+1} - D_i)/2 \qquad (1)$$

where P is the number of pustules per plant and D is the date at the i^{th} and $i+1$th sampling. The variability in the PD was determined from the subsamples in each row.

Host growth assessment. The area of leaves on entire plants, representative of those selected for counting, was determined at various times during the growth of the crop either by direct measurement using a portable area meter (Model LI-3000, LI-COR, Lincoln, NE) or by using relationships between leaf dimensions and leaf area derived as follows. On a sample of 40 leaves, the width (at the widest point) of the center leaflet of a trifoliolate leaf, the area of this leaflet, and the area of the remaining two leaflets of the trifoliolate were measured. Regression analysis between the width (W) of the center leaflet of a trifoliolate and the area of that leaflet (A_{CL}) and between W and the area of the entire trifoliolate (A_T) yielded: $A_{CL} = 0.78 \cdot W^{1.98}$, $r^2 = 0.94$, and $A_T = 1.65 \cdot W^{2.15}$, $r^2 = 0.85$, where the values of r^2 are for the nontransformed coordinates. The leaf area per ground area of

the crop (leaf area index, LAI) was estimated on several dates during the season by multiplying the average area per plant by the number of plants per ground area, obtained by counting the number of plants in randomly selected 1-m lengths of row throughout the field.

Meteorological conditions. Wind speed and direction, solar irradiance, air temperature, and relative humidity were continuously monitored at the center of the field using a Campbell Model CR-21 datalogger (Campbell Scientific, Inc., Logan, UT). The wind speed sensor (Met-One Model 014A cup anemometer), Met-One, Inc., Grants Pass, OR, the vane (Met-One Model 024A), and the pyranometer (LI-COR Model LI200S) were located at about 3 m above the top of the crop canopy. The wind-ventilated temperature and relative humidity probe (Campbell Model 210) was shielded from the sun and located 1.5 m above the ground. Leaf wetness was estimated using two leaf wetness sensors (Campbell Model 731), one placed in each half of the field at about mid-canopy height. The surface of the leaf wetness grid was coated thinly with light-green latex paint (3). Measurement of rainfall was obtained from a standard weather station about 200 m from the field. The Campbell data were missing for 8 days of record in 1987. For these days, values of T_{avg} were obtained from a maximum and a minimum thermometer, and hours of leaf wetness were estimated from periods of saturated humidity indicated by a recording hygrothermograph, all located in a standard weather shelter 1.5 m above the ground and 200 m from the field.

RESULTS

Weather. In general, the time during which disease developed in 1986 was somewhat cooler and wetter than in 1987 (Fig. 2). Over these same periods, the cumulative daily solar irradiance was about 610 MJ m^{-2} in 1986 and 659 MJ m^{-2} in 1987 (data not shown).

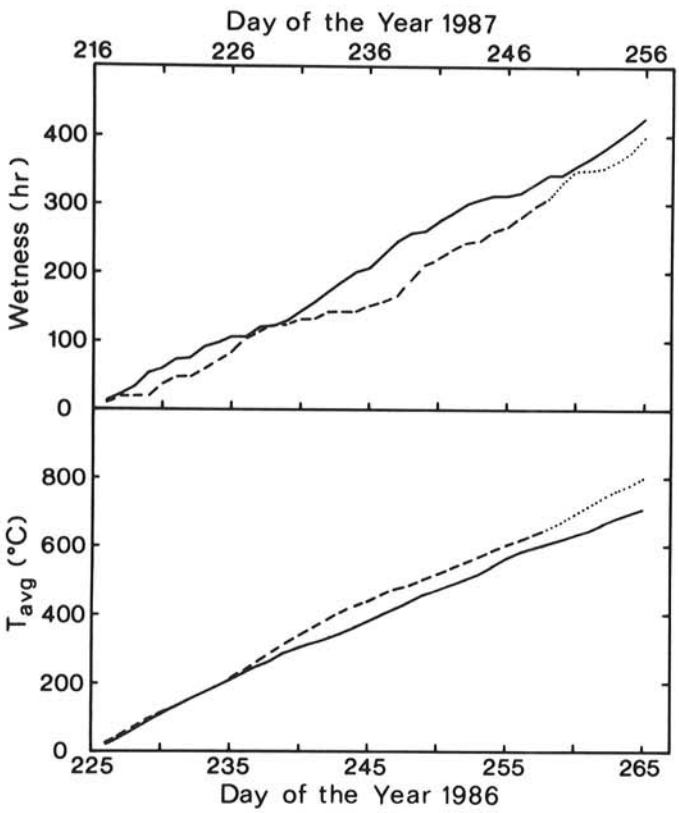

Fig. 2. The variation with time (day of the year), starting with the day that symptoms of infection were first seen, of the cumulative daily hours of leaf wetness, and the cumulative average daily temperature $(T_{max} + T_{min})/2$. Inoculations were done on day 219 and 224 in 1986 (————) and on day 210 and 215 in 1987 (---). The data for the last 8 days of the record in 1987 (. . . . .) were obtained from instruments located in a standard weather shelter 200 m from the field.

There was little difference in either the direction or the speed of the wind in the two years (Fig. 3). In both years, the most frequent directions of the wind were perpendicular to the rows of susceptible bean plants. However, the highest wind speeds tended to be associated with winds from the northwest and the lowest speeds tended to be associated with winds from the east.

Development of the crop. In both years, the plants emerged 5–6 days after planting and the beans were of prime harvest size and quality at about 55 days after planting. The relative increase in leaf area per plant (compared with the maximum) tended to lag behind in 1987 compared to 1986 (Fig. 4). Plants were somewhat larger in 1987 than in 1986, owing, in part, to the smaller stand density in 1987. The average number of plants per meter of row was about 23 in 1986 and 12 in 1987. The maximum crop LAI was about 6 in 1986 and about 4 in 1987. The rate of increase in height of the crop was very similar in both years: the maximum crop height (59 cm in 1986 and 51 cm in 1987) was obtained at 49 days after planting in both years. The average number of trifoliolate leaves per plant was similar during the first 40 days after planting in both years; however, at the time of maximum leaf area, there were about three more leaves per plant in 1987 (avg = 12.8) than in 1986 (avg = 10.1).

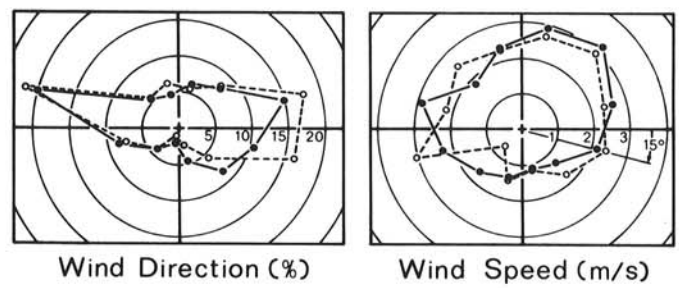

Fig. 3. Polar histogram diagrams of the wind direction and wind speed during the 1986 (————) and the 1987 (–o–) bean rust epidemics. The diagrams have the same orientation as Figure 1. The dots are plotted at the center of the 30° sectors used as the class intervals. Distance from the center (+) to successive concentric circles are in increments of 5% for the direction frequency histograms and increments of 1 m sec^{-1} for the speed histograms.

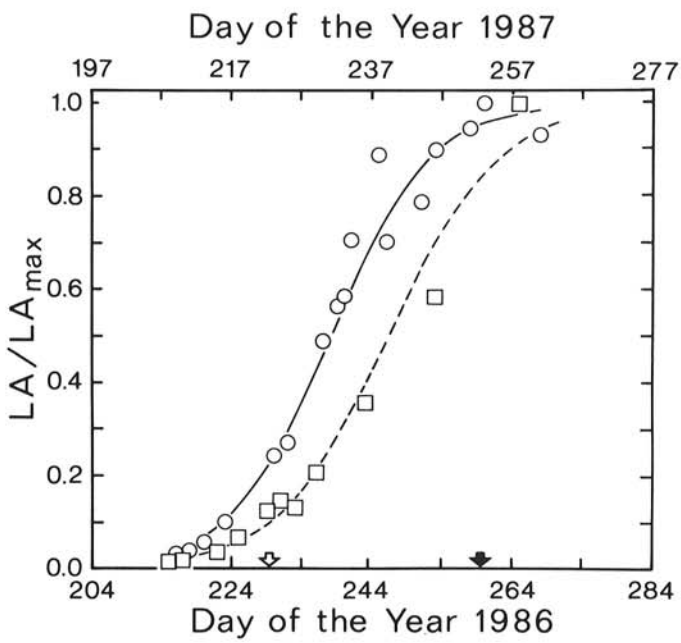

Fig. 4. The variation with time (day of the year), starting with the planting date, of the normalized average leaf area per plant (1986, o; 1987, □). The leaf area has been normalized by dividing measured values by the maximum average area per plant, which was 20.8 dm^2 in 1986 and 26.7 dm^2 in 1987. The arrows indicate the day when pustules were first noticed on inoculated plants (open arrow) and when beans were ready for harvest (solid arrow).

Development of disease. The rate of development of disease was similar in both years (Fig. 5). The increase in the average number of pustules per plant, P, with time, t, was described well by the exponential model ($P = a \cdot \exp(b \cdot t)$). Values of the regression coefficient b for the southeast and southwest quadrants, respectively, were 0.14 and 0.18 in 1986 and 0.17 and 0.17 in 1987. The value of the coefficient of determination r^2 (in nontransformed coordinates) was 0.99 in all four cases. The average number of pustules per dm^2 of foliage at the last assessment date was very similar in the two years (accounting for differences in plant size) (Fig. 4). With one exception, differences in pustule counts between the east and west halves of the inoculated row (Row 0) or Row 2 at the first assessment date (Table 1) were not significant.

In 1986, there were significantly more pustules in the even-numbered rows of the southeast (all susceptible) quadrant than in corresponding rows of the southwest (alternate resistant and susceptible rows) quadrant, whereas in 1987 there was relatively little difference between the southeast and southwest quadrants. Cumulative pustule days were obtained for each sampled row by numerically integrating the area under the disease progress curves using equation 1. There were generally fewer pustule days in corresponding rows of the southwest quadrant compared to the southeast quadrant, and of the northwest quadrant compared to the northeast quadrant (1986, Table 2; 1987, Table 3). In both years, the differences between these quadrant comparisons tended to decrease as the epidemic progressed and tended to be smaller for rows closer to the inoculated row. On the last two assessment dates in 1987, most evaluations were done in rows that had not been entered previously for sampling. For most of these rows, differences between east and west quadrants were small and none were significant (Table 3), although the differences for the last two distances in the south end of the field were relatively large at the final determination. Averaging over all sampled rows, the number of pustule days in the southwest quadrant was about 30% less than in the southeast quadrant during 1986 and about 6% less during 1987.

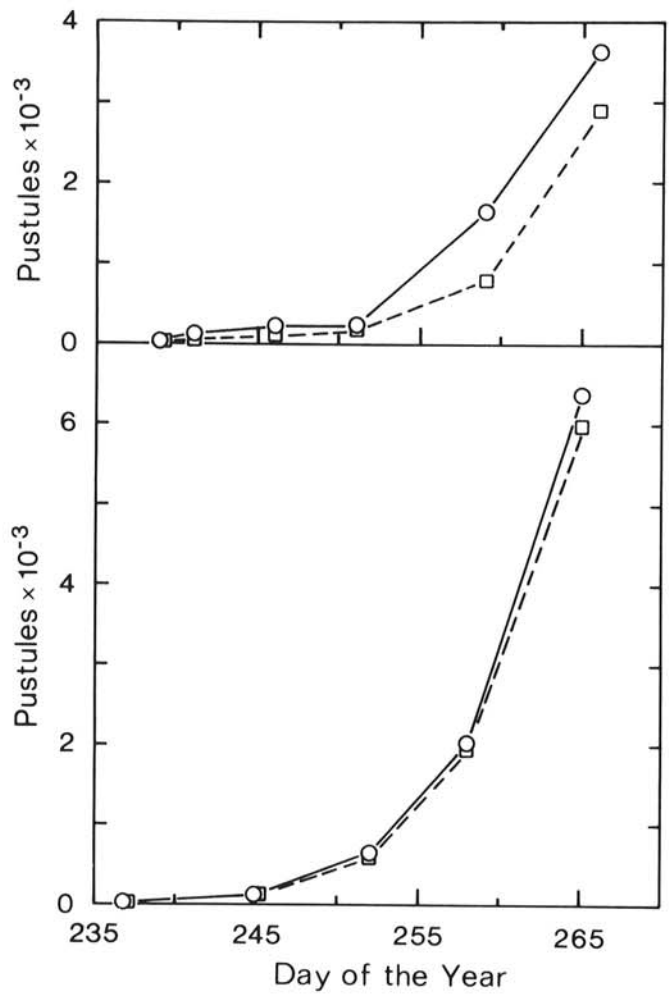

Fig. 5. The variation with time (day of the year) of the average number of pustules per plant in the southeast quadrant (o) and southwest quadrant (□) during the course of the epidemics in 1986 (top panel) and 1987 (bottom panel).

DISCUSSION

Theoretically, the maximum effect of a mixture in reducing a foliar plant disease should occur if the spores produced in a field are dispersed uniformly over all plants in the field. As long as infection sites are not limiting, the advantage of a mixture should increase with each generation, and potentially, the amount of disease (relative to a completely susceptible planting) could be reduced in a 1:1 mixture of resistant and susceptible plants by about $(1/2)^n$, where n is the number of generations of the pathogen (8). Bean rust has a latent period for pustule development of about 7 to 8 days (4), which should have allowed about three to four generations of the pathogen to develop during the course of the epidemics studied here, theoretically yielding a maximum disease reduction of 88–94%. This theoretical maximum was not

TABLE 1. Average number of rust pustules per bean plant in the inoculated row (Row 0) and in Row 2 of a field with susceptible plants in every row (pure planting, east [E] half) and with resistant and susceptible plants in alternate rows (mixture planting, west [W] half)[a]

	Day[b]	Pure (E) pustules[c]	Mixture (W) pustules	n[d]
Row 0				
1986	240	4119 ± 987	2877 ± 877	3
1987	223	43 ± 5	68 ± 9	10
	225	87 ± 11	117 ± 9	10
	229	118 ± 24	198 ± 42	10
	231	697 ± 145	756 ± 97	10
	236	1435 ± 228	2520 ± 465	10
Row 2				
1986	239	118 ± 48	52 ± 9	5
	240	26 ± 8	17 ± 4	5
1987	237	77 ± 19	63 ± 9	5
	237	129 ± 26	94 ± 11	5

[a] See Figure 1.
[b] Day of the year from January 1.
[c] Mean ± standard error of the mean for the number of pustules on the primary leaves and the center leaflet of the trifoliolate leaves of plants randomly chosen from locations equally spaced along the row.
[d] Number of plants counted on each side (east and west) of the row.

TABLE 2. Cumulative pustule days, *PD*, during the course of the bean rust epidemic in 1986 in corresponding rows of a bean field with susceptible plants in every row in the east (E) half and with resistant and susceptible plants in alternate rows in the west (W) half[a]

	South of source row			North of source row		
Row	Pure (E) PD_{SE}[b]	Mixture (W) PD_{SW}	$DIFF_S$[c] (%)	Pure (E) PD_{NE}	Mixture (W) PD_{NW}	$DIFF_N$ (%)
2	88.6 ± 6.5	55.4 ± 3.4	38	18.7 ± 2.0	11.9 ± 1.6	36
4	46.9 ± 4.0	31.8 ± 7.4	32	7.2 ± 0.8	6.5 ± 1.0	9
6	34.7 ± 1.9	21.4 ± 3.2	38	7.6 ± 0.6	5.0 ± 0.4	34
10	27.1 ± 2.4	19.1 ± 2.7	30	3.8 ± 0.7	2.2 ± 0.4	42
16	14.6 ± 2.1	10.8 ± 1.0	26	1.7 ± 0.6	1.1 ± 0.3	32
20	14.1 ± 2.2	5.9 ± 0.4	58	1.0 ± 0.2	0.7 ± 0.1	27
28	6.9 ± 0.5	5.5 ± 0.7	20	...	...	...
38	3.0 ± 0.5	2.0 ± 0.4	33	0.3 ± 0.05	0.2 ± 0.1	10

[a] Row 0 was inoculated. See Figure 1.
[b] Pustule days (in thousands) calculated from equation 1 using counts made on day (of the year) 239, 246, 251, 258, and 266 in the southeast and southwest quadrants and on day 240, 245, 253, and 261 in the northeast and northwest quadrants.
[c] The percent differences in *PD* between the east and west quadrants (e.g., $[(PD_{SE} - PD_{SW})/PD_{SE} \cdot 100]$).

achieved (and is never achieved in practice) because of two effects: a limitation of infection sites, and nonuniform spore dispersal, which allows a disproportionate number of the spores produced on a plant to be deposited back on the same plant, potentially leading to autoinfection (1,10,12).

The number of pustules per plant (P) increased steadily with time with no discernible slowing (Fig. 5), even though, by the time of the last evaluations, some of the lower trifoliolate leaves had fallen, directly resulting in a reduction in P. The crop continued to grow vigorously and put out new leaves throughout the course of the epidemic, and disease severity on a whole-plant basis (assuming a pustule area of 2 mm^2) did not exceed about 15% at the final evaluations. Thus, the potential number of infection sites on a whole plant was not a limiting factor. On the other hand, as the epidemic progressed, disease severities approached 100% on some older, lower leaves, where available infection sites clearly did become limiting. Because the values of P were heavily weighted by counts on these older leaves, this may have reduced somewhat the differences between the pure and the alternate planting.

Spore deposition gradients, resulting in enhanced autoinfection, were probably the main mechanism responsible for reducing the difference in the amount of disease between the two plantings. Urediniospores of *U. appendiculatus* are deposited along gradients that are relatively steep near the source and relatively flat far from a source, which is characterized by a power law (2). Although, on the one hand, a steep spore deposition gradient near a source should allow the alternate rows of resistant plants to potentially intercept a disproportionately greater share of the inoculum pool compared to a shallow gradient, and thus give an advantage to the mixed planting, a steep gradient also allows a disproportionately greater amount of autoinfection, which tends to counteract the effect of a mixture (7,9–12). The potential for autoinfection generally increases as the size of the plant increases and as the spore dispersal gradient becomes steeper (10,12).

Early in the growth of the crop, the plants were relatively small and much of the area in the field was bare ground. The openness between the rows should have allowed greater ventilation in the canopy and encouraged the dispersal of spores. This, together with the small plant size, should have favored the spread of spores to other plants, or loss of spores on the ground, over autoinfection. Thus, the difference in disease between the two plantings would be expected to be greatest early in the season (12). Later in the season, when the plants had grown large enough to fill the space between the rows, the ability for the resistant rows to filter spores from the air should have been at its maximum. Coincidentally, however, closure of the canopy probably also reduced air movement in the lower canopy, making it less likely for spores to be spread to other plants and more likely for spores to cause autoinfection. Of course, there also is a tendency toward increased autoinfection as plants grow larger, independent of the dispersal gradient. The present findings were in qualitative agreement with this expectation, in that disease reduction, although small, appeared to be greatest early in the course of the epidemic and to decline as the epidemic progressed. Thus, it seems likely that both autoinfection and a limited number of infection sites combined to keep small the relative difference in P beween the pure and the alternate row planting.

One difference between the epidemics in the two years was that the disease developed earlier in the growth of the crop in 1987 than in 1986 (Fig. 4, open arrow). The larger plants in 1987 did not entirely compensate for the smaller planting density; consequently the LAI in 1987 was only about two-thirds of that in 1986. Row closure, resulting in complete ground cover, did not occur in 1987 until the last sampling date, whereas in 1986 nearly complete row closure occurred by the fourth sampling date. Thus, in 1986 more of the spores produced on the lower leaves were probably confined inside the canopy, where they could have been filtered from the air by neighboring resistant plants. In 1987, the canopy remained partly open almost to the end of the epidemic, which should have allowed spores to more easily escape from the canopy and be spread to other plants. Although both of these effects can potentially enhance the effect of the mixture, they were apparently outweighed by the effects of autoinfection. For diseases with initially steep spore deposition gradients, mixture effectiveness is generally decreased to a greater extent for a general inoculation than for a focal inoculation when the size of genotype units is increased (11). It is possible that the spore dispersal that occurred when the canopy was more open allowed for a more general inoculation of plants in the field. Mundt and Leonard (10) also found that mixture effectiveness was greater when focal inoculation occurred relatively later in the development of the crop canopy.

An effective crop mixture should reduce the rate of increase of disease (1,8). The rate of disease development found in 1987 was nearly the same in both halves of the field, and disease actually appeared to develop a little more slowly in the pure planting than in the alternate planting in 1986 (Fig. 5). The greater numbers of pustules per plant found in the half of the field with all susceptible plants may have been partly due to a higher initial level of infection (Table 1). The first infections noted in Row 2 were primary infections on these plants and, taken together with the results for the inoculated row, indicate that there may have been slightly more inoculum on the east than on the west side of the field at the start of the epidemic in 1986 and vice-versa in 1987. If this were true, then the relative reductions in disease due to interplanting cultivars found in 1986 (Table 2) were probably somewhat greater than would have occurred had the initial infection on both sides of the field been completely uniform. This effect would have been reversed in 1987 (Table 3).

In general, the severity of disease varies with height in the crop canopy, and this bears on the assessment of crop mixtures in reducing the spread of disease. Mixtures of resistant and susceptible maize plants appeared to be more effective in reducing common maize rust when evaluations were only on upper leaves rather than on a whole-plant basis (10). This is to be expected because, after several generations of the pathogen, autoinfection should contribute a relatively greater share of pustules on an entire plant, whereas allo-infection should contribute a relatively greater share of the infections found on the upper leaves. The whole-plant evaluations used in the present study should account fully for autoinfection and thus evaluate conservatively the effect of the

TABLE 3. Cumulative pustule days, PD, during the course of the bean rust epidemic in 1987 in corresponding rows of a bean field with susceptible plants in every row in the east (E) half and with resistant and susceptible plants in alternate rows in the west (W) half[a,b]

	South of source row			North of source row		
Row	Pure (E) PD_{SE}[c]	Mixture (W) PD_{SW}	$DIFF_S$[d] (%)	Pure (E) PD_{NE}	Mixture (W) PD_{NW}	$DIFF_N$ (%)
2	10.7 ± 1.3	11.2 ± 1.4	−4	1.59 ± 0.15	1.24 ± 0.19	22
4	4.5 ± 0.2	4.5 ± 0.3	0	0.87 ± 0.07	0.58 ± 0.10	33
6	3.4 ± 0.6	4.2 ± 0.5	−22	0.54 ± 0.06	0.28 ± 0.04	48
10	2.5 ± 0.2	1.3 ± 0.2	47	0.24 ± 0.05	0.18 ± 0.03	25
16	1.8 ± 0.2	0.9 ± 0.1	49	0.13 ± 0.02	0.09 ± 0.01	32
20	1.4 ± 0.5	0.7 ± 0.1	45	0.11 ± 0.03	0.07 ± 0.01	38
28	0.6 ± 0.1	0.4 ± 0.0	44	0.05 ± 0.01	0.02 ± 0.01	56
36	0.03 ± 0.1	0.2 ± 0.0	26	0.03 ± 0.00	0.02 ± 0.01	31
2	61.8 ± 1.6	69.1 ± 17.3	−12	95.7 ± 21.5	94.4 ± 12.8	1
4	53.8 ± 11.7	42.4 ± 3.9	21	77.7 ± 4.0	73.5 ± 9.9	5
8	28.9 ± 2.2	33.0 ± 3.2	−14	93.8 ± 22.1	78.7 ± 19.5	16
12	22.3 ± 4.0	21.6 ± 3.1	3	53.3 ± 8.7	42.8 ± 7.0	20
18	17.2 ± 1.8	15.5 ± 1.5	10	39.3 ± 5.8	39.3 ± 2.0	−0.2
26	13.0 ± 3.9	7.7 ± 2.0	40	24.5 ± 7.6	25.1 ± 5.5	−3
34	9.1 ± 1.1	4.3 ± 0.6	52	10.2 ± 2.8	9.2 ± 2.8	10

[a] Row 0 was inoculated. See Figure 1.

[b] Top half of table: Pustule counts made on day 237, 245, and 252 in the southeast and southwest quadrants and on day 237 and 243 in the northeast and northwest quadrants. Bottom half of table: Pustule counts made on day 258 and 265 in the southeast and southwest quadrants and on day 259 and 267 in the northeast and northwest quadrants.

[c] Pustule days (in thousands) calculated from equation 1.

[d] The percent differences in PD between the east and west quadrants (e.g., $[(PD_{SE} - PD_{SW})/ PD_{SE} \cdot 100]$).

mixture on the progress of disease in the crop.

Mundt and Leonard (10) also studied the effect of mixtures of the same two cultivars of snap beans used in the present study. In their study, various numbers of plants of the susceptible cultivar were planted in groups to create different-sized areas of the two genotypes. The relative reduction in disease that they found for the two smallest (0.023 and 0.093 m^2) genotype areas was similar to that found in 1986 for the alternate-row planting of resistant and susceptible bean plants in the present study (approximately 30%). When they grouped susceptible plants into larger (0.372 and 0.836 m^2) areas, however, they found only about 1–10% relative reduction in disease. For 1:3 mixtures, they found generally larger reductions in disease than for 1:1 mixtures, but for the two larger genotype units the results were inconsistent between 2 yr. The authors suggested that this difference may have been due to the difference in timing of initial infection relative to the development of the crop canopy as already noted above.

In the present study, there was generally less disease on susceptible plants in the alternate-row plantings of resistant and susceptible snap bean cultivars than on comparable plants in a pure planting of the susceptible cultivar in the first year of the study. In the second year of the study, there was little or no reduction of disease attributable to the mixed planting. Apparently, autoinfection of the bean plants dominated the development of disease, and the isolation of susceptible plants from touching afforded by the wide row spacing and interplanted resistant variety was insignificant. Although it may be possible that alternating two or more rows of a resistant cultivar would show a greater benefit, the characteristic shape of the spore dispersal gradients of the bean rust pathogen mitigates against this. The dispersal gradient exhibits at the same time a steep gradient close to the source and a relatively flat gradient far from a source, which is characteristic of a power law dispersal function. This kind of dispersal function encourages autoinfection but also allows infection of distant plants. Thus, it appears that this host pathogen system may not be well suited for the mixture approach to disease control.

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