Mechanisms of Alteration in Bean Rust Epidemiology Due to Intercropping with Maize

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ABSTRACT

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We performed experiments to identify how maize influences bean rust (caused by *Uromyces appendiculatus*) in maize-bean intercrops. The effects of competition with maize and interference by maize on dispersal of rust urediniospores were evaluated in trials conducted three times during 1989 and 1990. Alterations in the nondispersal (infection) phase of the pathogen life cycle due to intercropping and competition with maize also were assessed. Overall effects of maize on rust severity were evaluated in another experiment. Competition consistently steepened dispersal gradients ($P < 0.10$) in trials conducted more than 50 days after planting alone or in combination with interference (intercrop). Interference had no clear effect on dispersal gradients. Estimated total spore deposition per plot was increased (second trial) and decreased (third trial) by competition in both years ($P < 0.05$). Intercropping only affected infection once, in late 1989, when rust severity was reduced by 96% ($P < 0.05$). Overall disease was reduced by intercropping at two plot locations in both years ($P = 0.07$), but not at a third location. Bean leaf area declined because of competition in 1989 but not in 1990. Steep gradients may be due to increased spore escape, and microclimatic changes created by maize are probably responsible for the nondispersal effect.

Additional keywords: *Phaseolus vulgaris*, *Zea mays*.

Several studies have related intercropping to changes in yield (25,38) and insect incidence (39,40), but few have evaluated the effects of intercropping on plant pathogens. Despite speculations that disease severity in intercrops generally is less than in monocrops (4,10), a wide range of results even within a single crop combination has been reported. For example, the severity of angular leaf spot of bean (caused by *Phaeoisariopsis griseola*) in bean-maize intercrops has been reported to be less than (9,29), equal to (9,29,41), or more than (29,34,36,41) the severity in bean monocrops. Bean rust (caused by *Uromyces appendiculatus* (Pers.:Pers.) Unger) generally has been either reduced (33,35,36, 41,43) or unaffected (49) by intercropping with maize.

The mechanisms of interaction among pathogen, host, and nonhost that determine disease levels in intercrops have not been clarified and remain speculative. Proposed mechanisms include amelioration of dispersal factors (e.g., wind or rain) by the nonhost, trapping of propagules by the nonhost, microclimate alteration of the pathogen environment, reduced density of host, and changes in infection elicited by microorganisms (induced resistance) or pollen associated with the nonhost (10,24,46). Empirical support for these suggestions is mostly indirect. For example, temperature reductions and increases in relative humidity have been measured for common beans grown with maize when compared to bean monocultures (9,45). High humidity and leaf wetness favor diseases such as bean rust and white mold (1,22,23) and, therefore, might increase disease intensity if these conditions were due to intercropping. Induced resistance to bean rust from inoculation with sunflower rust spores (*Puccinia helianthi*) or maize rust spores (a *P. sorghi-P. polysora* mix) has been demonstrated in the laboratory (2,50). In contrast, pollen will enhance infection and fungal growth by *Botrytis cinerea* on faba beans and by *Colletotrichum lindemuthianum* on cowpea, but will reduce cowpea yellow mosaic infection (3,16).

A few studies have provided direct evidence of mechanisms contributing to disease severity in intercrops. Burdon and Chilvers (11–13) experimentally determined that reductions in rates of damping-off of cress when mixed with ryegrass and rates of powdery mildew development on barley when intercropped with wheat were due mainly to reduced density of the host in mixtures. In all their experiments, morphologically similar species were mixed in replacement-type combinations (i.e., total plant density remained constant but host density decreased as nonhosts were added to the mixture). Thus, Burdon dismissed microclimatic influences as important in altering the epidemics (10). However, in a black walnut-autumn olive intercrop, microclimate-induced reductions in primary inoculum, as well as interference of autumn olive with inoculum dispersal, were cited as mechanisms for an observed 80% reduction in walnut anthracnose incidence (26). Chin and Wolfe (15) attempted to isolate density, induced resistance, and interference components of mildew reduction in barley cultivar mixtures and found density effects important early in the season; induced resistance was significant only later. Late season density effects could not be evaluated because of compensatory growth at low initial densities.

The objective of this study was to systematically evaluate, at different times during the growing season, the role of three factors that may influence bean rust in a common bean-maize intercrop. The factors were maize interference with dispersal of *U. appendiculatus* urediniospores; dispersal effects due solely to competition with maize; and maize effects on nondispersal components of the disease cycle.

MATERIALS AND METHODS

In overview, we evaluated the effects of maize on dispersal of bean rust by assessing the primary dispersal gradient away from a focal inoculum source over a 3-day period three times during the growing season. By using potted diseased beans as the inoculum source and potted healthy plants as spore traps,

it was possible to conduct these experiments in plots planted to a bean cultivar resistant to the rust race employed. This, in turn, allowed the same plots to be re-used for all three experiments, except for one destructive treatment (see below). Nondispersal effects of maize were evaluated in separate experiments three times during the season by spray-inoculating bean plants and assessing severity over approximately 14 days. Plots used for dispersal trials were also employed in these experiments; we utilized susceptible beans randomly planted among the resistant beans and covered them during the dispersal event to avoid contamination. Finally, the effect of intercropping on overall disease levels was evaluated in separate experiments conducted in monocrop-intercrop plot pairs of susceptible beans.

Other mechanisms of the intercropping-disease interaction were not evaluated. We eliminated induced resistance by ensuring that the maize was disease-free, and we eliminated host density effects by employing an additive rather than a replacement series design. It was not possible to eliminate the potential effects of maize pollen, which was present late in the season, or microorganisms not pathogenic on beans or maize.

Plant culture and inoculation in dispersal and nondispersal effects experiments. Snap beans (*Phaseolus vulgaris* L. 'OR91G', Rogers Brothers Seed Co., Twin Falls, ID) and hybrid sweet corn (*Zea mays* L. 'Jubilee,' supplied by H. J. Mack, Horticulture Dept., Oregon State University, Corvallis) were hand-planted on 22–25 June 1989 and 19–22 June 1990, in 20- × 20-m plots at the Oregon State University Botany and Plant Pathology Experimental Farm immediately east of Corvallis. Eighteen plots were arranged in three contiguous blocks of six plots each within an environment of heterogeneous vegetation as shown in Figure 1. The variable arrangement and spacing among plots was dictated by the limited land availability at the research farm. Annual ryegrass was planted between the plots and up to the site boundaries and mowed regularly to a height of approximately 15 cm. An alternating pattern of one maize row and two bean rows with 40 cm between rows and 45 and 15 cm within rows (maize and beans, respectively) was employed. In bean monocultures, maize rows were left unplanted, but the arrangement of beans was identical to that described above. In 15 plots, snap bean cultivar Pinto 111 (Independent Seed and Bean Co., Twin Falls, ID) was randomly planted in the bean rows (see below).

We applied overhead sprinkler irrigation after planting and 2 wk before each dispersal experiment. Irrigation of all plots required approximately 1 wk because of a limited supply of temporary irrigation pipe. Thirty-eight to 51 mm of water was applied to each plot at each irrigation. Fertility was maintained with broadcast 13-38-13 (N-P-K) fertilizer (544 kg/ha in 1989 and 874 kg/ha in 1990) applications before planting and a 34-0-0 (0.5 kilograms per row) sidedress approximately 3 wk after emergence. Before planting, alachlor at 3.4 kg/ha a.i. was applied for weed control, and insect protection was limited to three weekly applications of carbaryl (1.87 gm/ml a.i., sprayed to runoff) to Pinto 111 beans beginning on 13 July 1990 for controlling Mexican bean beetles.

Pinto 111 beans were grown in 10-cm plastic pots (one plant per pot) in the greenhouse for use as trap plants and inoculum source plants in dispersal experiments. Approximately 22 days after planting, when primary and two to three trifoliate leaves were present, those used as trap plants were brought to the field site and thereby acclimatized before experimental use 2–3 days later. We inoculated source plants with *U. a.* var. *appendiculatus* race 40 (provided by J. R. Stavely, USDA-ARS, Beltsville, MD) by spraying a urediniospore suspension (1×10^4 spores per milliliter in distilled water with 0.01% Tween 80) to runoff with a bulb atomizer (DeVilbiss Co., Somerset, PA). After plants had dried, they were placed in a closed tent at 100% RH for 16 h to maximize infection. Inoculations were done separately on primary, first trifoliate, and second trifoliate leaves corresponding to maximum susceptibility of each leaf (approximately 9, 18, and 24 days after planting, respectively). Source plants were maintained in a greenhouse separate from the greenhouse containing trap plants and were covered with polyethylene bags and transported to experimental plots immediately before use, approximately 33 days after planting. A source plant occasionally had relatively low numbers of uredinia, in which case a supplemental pot containing four 24-day-old beans with infected primary leaves was substituted to ensure homogeneity of inoculum supply. Unlike Pinto 111, cultivar OR91G used for field plantings is highly resistant (no sporulating pustules produced) to race 40 (*personal observation*).

Plant culture and inoculation in overall intercropping effects experiments. An additional experiment, designed to evaluate the combined effects of the mechanisms outlined above (i.e., the overall effects of intercropping on disease), was also conducted. Three pairs of plots, each consisting of one bean monocrop and one bean-maize intercrop, were planted at the locations indicated in Figure 1. These plots were planted and cultivated on the same dates and by the same methods as those given above for dispersal and nondispersal experiments. The following alterations were made. Pinto 111, susceptible to rust race 40, was used in all plots.

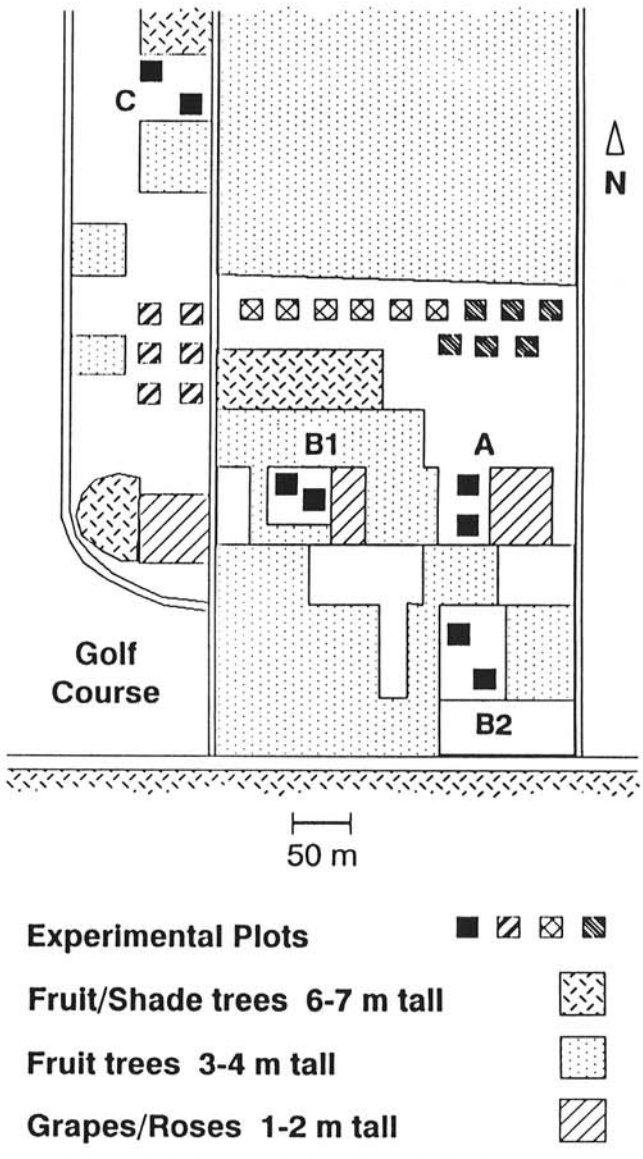

Fig. 1. Arrangement of experimental plots and nature of surroundings. Solid squares represent plots used for experiments comparing overall severity of bean rust in bean monocrops and bean-maize intercrops during 1989 and 1990. **A,** Pair A, both years; **B1,** pair B, 1989; **B2,** pair B, 1990; **C,** pair C, both years. Remaining squares represent plots used for assessing mechanisms of maize influence on dispersal and nondispersal aspects of *Uromyces appendiculatus*. Shading patterns in squares indicate three blocks of six plots each. All blank areas represent land planted to grass or annuals. Parallel lines represent roads.

Plots were 18.3 × 18.3 m in 1989 and 20.0 × 20.0 m in 1990. The size reduction in 1989 was due to space limitations for one plot pair (pair B in Fig. 1); this pair was moved in 1990 to allow for larger plots and more space between them. The plots included 16 maize rows during both years, but the smaller plots of 1989 had 15 rather than the 17 pairs of bean rows planted in 1990. This was achieved by eliminating the two outermost pairs of bean rows. Although pairs A and B were located within a mosaic of grapes, roses, and dwarf fruit trees near the plots described above for dispersal and nondispersal experiments, pair C was positioned at some distance away near the corner of the farm. Standard cherry trees were to the north of this site, and a golf course was to the west (Fig. 1). Data on wind direction, desirable in this situation, was not available at the research farm. A daily resultant wind vector was recorded 57 km to the north at Salem, OR, also situated in the mid-Willamette Valley (47).

Inoculum source plants for this experiment were produced as described above for dispersal experiments.

Dispersal effects. We evaluated the effects of maize on dispersal of *U. appendiculatus* by comparing primary dispersal gradients away from a focal inoculum source in individual plots (experimental units). Treatments were in a 2 × 2 factorial, randomized complete block design; the three physical blocks mentioned were used. The two factors under study were the presence of maize during bean growth (competition) and the presence of maize only during the dispersal event (interference). There were two levels of each factor (i.e., presence and absence), giving a total of four possible treatment combinations. These are illustrated in Figure 2. The no competition-no interference and competition with interference treatment combinations were represented by a bean monocrop (Fig. 2A) and a bean-maize intercrop (Fig. 2B), respectively. The competition without interference ("removal") treatment was accomplished by removing the maize from another bean-maize intercrop plot the day before the dispersal event began (Fig. 2C). Then, by artificially supporting these cut maize plants at corresponding locations in a bean monocrop for the duration of a dispersal event, the interference without competition ("addition") treatment was realized (Fig. 2D). Supports consisted of 10-cm nails projecting from boards buried in the plots, onto which maize stalks were impaled. Each time the experiment was repeated in a given year, the same plots were used for the intercrop, monocrop, and addition treatments. The removal treatment, because of its destructive nature, required a new plot for every experiment. Thus, 12 plots were used for one experiment (four treatments × three blocks), but a total of 18 plots were planted initially to supply a new removal treatment for each of three repetitions of the experiment.

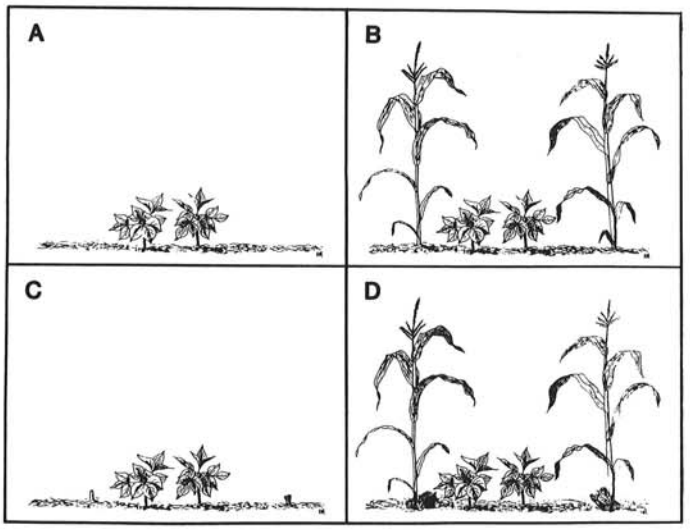

Fig. 2. Diagrammatic representation of treatments used to evaluate dispersal gradients of *Uromyces appendiculatus* on beans, as influenced by competition and interference effects of intercropping with maize. **A,** Beans grown alone; interference and competition are absent (monocrop treatment). **B,** Beans and maize grown together; interference and competition are present (intercrop treatment). **C,** Beans and maize grown together, with maize removed from plot immediately before spore dispersal event. Interference absent but competition present (removal treatment). **D,** Beans grown alone, with cut maize from removal treatment artificially supported on nails protruding through buried boards, immediately before spore dispersal event. Interference present but competition absent (addition treatment).

Primary dispersal gradients were estimated in each plot over a 3-day period in the following way. On the morning of day 1, beans were removed from the central 1.2 m of each of the two center bean rows. Ten covered source plants were placed in the resulting gap in each row, and four additional source plants were placed between these rows approximately 10 and 25 cm from each end of the gap (24 plants total). Trap plants were then placed at the center and at 1.2-m intervals along the four cardinal directions away from the center to the plot edge, resulting in eight plants in each direction positioned midway between adjacent bean rows. Because trap plants were of the same age regardless of the plot they were placed in or the date of the particular experiment, their size relative to the field plants varied. This, coupled with the height of the pots themselves, made it necessary to dig holes in some cases to prevent the leaves of the trap plant from extending over the existing bean canopy. When trap plants for all plots were in place, source plants were uncovered to initiate dispersal. Approximately 10 h later, just before sunset, all trap plants were collected and placed together under ideal infection conditions in a humidity tent at the farm site. In this way, treatment differences due to factors other than dispersal (e.g., effects of maize on spore germination) were made negligible. On days 2 and 3, trap plants were returned to their original field positions during the day, followed by nights in the humidity tent; the total exposure to *U. appendiculatus* spores was approximately 30 h. After day 3, the beans remained in the humidity tent for 16 h to maximize infection; then, they were returned to the greenhouse. Twelve days later, we commenced counting of all pustules on all leaves present. Heavily infected leaves were removed and stored at 4 C until counting, if leaf death appeared imminent.

The experiment was repeated on 28 July, 18 August, and 8 September in 1989 and 25 July, 11 August, and 29 August in 1990 (35, 56, 77, and 35, 52, 70 days after planting, respectively), hereafter referred to as releases 1, 2, and 3 for both years. Background contamination and nonprimary gradients were minimal because *U. appendiculatus* is rare in western Oregon (28), and source plants were removed from the farm immediately after each experiment. Control trap plants were placed in unused plots during each release (the two removal plots in each block reserved for release experiments at other dates), one plant in the center and one at the edge downwind to the nearest plot being used. These were treated like the other trap plants in all other respects. Three additional sets of controls, each with six plants, were added in 1990 to assess *U. appendiculatus* spore deposition in the humidity tent, in the field next to the humidity tent (35 m from the nearest plot), and in the greenhouse where trap plants were grown.

Lesion counts were averaged over all directions in each plot and fit to a negative exponential (27) and the modified Gregory inverse power function (37); the latter employed a truncation factor of 0.6 m, equal to the source radius. Gradient slopes were then taken as the experimental response variable and subjected to 2 × 2 multifactorial analysis of variance with blocks; maize interference and competition were used as main effects. To compare spore numbers retained in entire plots, we estimated the total number of uredinia that would have been produced had the plots been planted to susceptible plants. We then subjected these values to analysis of variance (ANOVA). The estimates were obtained by multiplying the average lesion number at a given distance from the center by the plot area estimated by that number (1.2-m-wide annuli) and summing these values. Lesion numbers in plot corners beyond the outermost annulus containing trap plants were estimated with values predicted by the modified

Gregory model for that distance.

We did analyses by using counts from the first trifoliate leaf only and from the combined total of all leaves present on all plants for that release, except for release 2, 1990, as noted below. Occasionally a datum would be absent because of leaf death. In such a case, the gradient model was fit after all data at the same distance from the center in that plot in all other directions were eliminated; this was done to avoid possible inaccuracies due to directional effects. For total lesion number estimates, no data were eliminated, but missing points were estimated from the modified Gregory model for that distance.

For release 2, 1990, extremely hot weather on the first day eventually killed many of the leaves and resulted in an excessive number of missing points when values were averaged over all directions. Therefore, we calculated a gradient slope for each directional transect in each plot to use all available values and thereby give fewer missing data. In this case, second trifoliate values were used because more of these survived than did first trifoliate leaves. Gradient slopes were analyzed by hierarchical ANOVA with the four directions nested within the usual 2 × 2 treatment combinations with three blocks (48 values total).

For all release experiments, appropriate reductions in the error term of the ANOVA were made where missing data points and poor fit to the regression models resulted in the elimination of values (four out of 48 values for release 2, 1990; one out of 12 values for release 3, 1989, combined leaf data and release 3, 1990, first trifoliate and combined leaf data).

Nondispersal effects. We evaluated the effects of maize on components of the *U. appendiculatus* life cycle other than dispersal by uniformly inoculating healthy Pinto 111 plants randomly planted in the monocrop and intercrop plots used for the dispersal experiments, then we recorded latent period and severity. Ideally, a 2 × 2 factorial design with maize presence before infection (assessing competition) and maize presence during infection (assessing microclimatic influences) as main effects would have been employed. The first factor was evaluated by inoculating susceptible plants in the removal treatment plots after maize removal. However, the second factor could only be assessed by inoculating susceptible beans planted in the addition plots. This treatment was logistically impossible, because it would have required leaving the artificially supported maize standing throughout the 2- to 3-wk experiment. Shading, thus produced, would have violated the requirement for no competition in this treatment of the dispersal and nondispersal experiments. Therefore, the final design includes only three combinations of the factors given above: no maize present before or during infection (monocrop), maize present before but not during infection (removal), and maize present before and during infection (intercrop).

During dispersal experiments, all Pinto 111 plants were covered with inverted greenhouse containers to avoid contamination; they were uncovered after source plants had been removed. Twenty-centimeter-diameter plastic containers (McConkey's Co., Sumner, WA) were used during release 1, 1989; substantial leaf loss resulted from contact with the sun-heated plastic. Twenty-five-centimeter-diameter fiber containers (Western Pulp Products, Corvallis, OR) were used thereafter; no apparent ill effects were observed. After completion of each dispersal experiment, five randomly selected Pinto 111 plants previously covered were sprayed at sunset to runoff with a 1×10^4 spores per milliliter (7.5×10^3 spores per milliliter on 27 Aug 1989) suspension in distilled water with 0.01% Tween 80. These inoculations took place on 3 August, 27 August, and 13 September 1989 and 28 July, 14 August, and 1 September 1990, hereafter known as inocs 1, 2, and 3 for both years. Plants were then observed daily for the first sporulating lesion (a measure of latent period) and removed to storage at 4 C after 11–12 days for severity assessment within the next several days. Inoculating beans in removal plots was not possible for inoc 1, 1990, because of poor growth of the maize in these plots, and background contamination made an assessment of latent period impossible for inoc 3 in 1989. The modified Cobb scale (44) was employed for estimating the percentage of leaf area infected on all leaves of the main stem in 1989 and all leaves of the main stem and primary and first trifoliate axillary leaves in 1990. Some leaves on axillary stems were evaluated in 1989 as substitutes for missing main stem leaves of similar age (based on leaf size).

Because leaf demographics varied widely among plants within experiments, we compared treatments by using severity values from the single most common leaf for that experiment, in terms of stem and position on stem. Thus, in 1989, the first trifoliate, fifth trifoliate, and seventh trifoliate leaves on the main stem were utilized for inocs 1, 2, and 3, respectively. In 1990, the first trifoliate leaf on the main stem and the second and fourth trifoliates on an axillary stem of the primary leaves were used. Two-way ANOVA (three treatments × three blocks) was performed on $\log_{10}(x + 1)$ transformed values of mean severity for each plot. The transformation was necessary to eliminate heteroscedasticity. Latent period values also were subjected to ANOVA when appropriate. Multiple comparisons were performed with the Newman-Keuls method.

Overall effects. In the plot pairs used to evaluate overall intercropping effects on disease, an epidemic was initiated by placing source bean plants in the center of each plot, as described for dispersal experiments above, on 31 July 1989 and 27 July 1990 (37 and 36 days after planting, respectively). In 1989, the source plants were removed after 7 days. Because rain occurred frequently

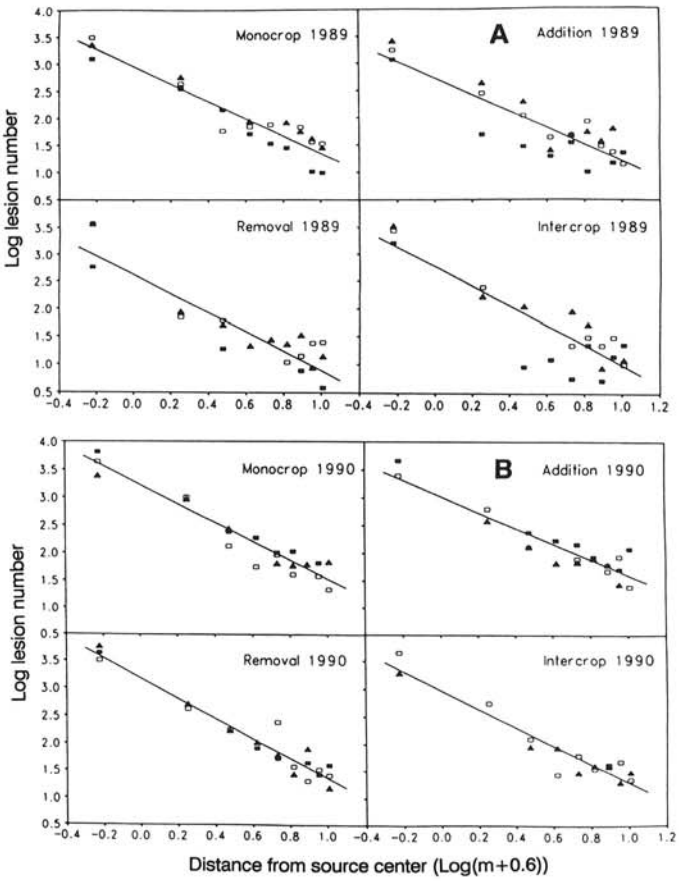

Fig. 3. Dispersal gradients of *Uromyces appendiculatus* on beans as influenced by competition and interference effects of intercropping with maize, for experiments done **A,** 77–79 days after planting in 1989; and **B,** 70–73 days after planting in 1990. Points represent mean lesion counts on first (oldest) trifoliate leaves of four trap plants equidistant from focal inoculum source. For each distance from the source in each graph, different symbols represent each of the three replicates (plots) used. The line represents the mean least-squares regression for the three plots by the modified Gregory model, $\log(y) = \log(a) - b[\log(x + c)]$. In these experiments, y = number of uredinia at distance x from source center, and $c = 0.6$ m, the source radius. For intercrop-1990, one replicate (represented by solid rectangles) was not included because of missing data. Monocrop = no competition or interference; addition = interference without competition; removal = competition without interference; intercrop = competition with interference. See text for explanation of treatments.

during the first 3 of the 7 days, in 1990 the plants were removed after only 5 days in an attempt to achieve an inoculum level similar to that of 1989.

Disease severity estimates were made for each plot on 14, 18, and 26 August and 1 and 8 September 1989 (14, 18, 26, 32, and 39 days after inoculation, respectively); and 14, 21, 28 August and 4 and 11 September 1990 (18, 25, 32, 39, and 46 days after inoculation). Percentage of leaf area infected was visually estimated by two people simultaneously for the north, center, and south one-third of five pairs of bean rows in each plot. Row pairs sampled in 1989, numbering from one edge of the plot, were 2, 5, 8 (the center pair), 11, and 14. Row pairs sampled in 1990 were 3, 6, 9 (the center pair), 12, and 15. The mean of these values was divided by 0.33 to correct for a maximum possible bean rust severity of 33% (44). Area under the disease progress curve (AUDPC) was determined for each plot, from date of inoculation to final assessment date, by use of the midpoint mean method of Shaner and Finney (42).

Crop growth. We observed leaf area and heights after each dispersal experiment in plots used for that experiment to assess the effects of intercropping on crop growth. Leaf and pod areas were included in the bean measurements. In 1989, seven bean and seven maize plants (where present) were randomly sampled from each of the four dispersal treatment plots on 1 August, 24 August, and 14 September (39, 62, and 83 days after planting, respectively) and stored at 4 C for subsequent measurement on an electronic leaf area meter (Model LI-300, Li-Cor Co., Lincoln, NE). Maximum height of foliage was recorded for 15 randomly selected bean and maize plants in the same plots on 2 August, 28 August, and 14 September. Because of greater within-plot heterogeneity in 1990, a stratified random sampling scheme was employed in which one area and two height observations were randomly taken from each of eight 5- × 10-m sections in each plot. Plants for area measurement were taken on 29 July, 15 August, and 1 September (39, 56, and 73 days after planting, respectively), and heights were evaluated on 29 July, 15 August, and 3 September.

At the ends of both seasons, bean density was estimated for all 18 plots used the previous summer for the dispersal and nondispersal experiments. The number of bean main stems was counted in 7 m of row measured north from the center of the sixth and 22nd bean rows and south from the center of the 13th and 29th bean rows; rows were numbered from the west edge of the plot. Maize density was taken in the same way from each adjacent row in the intercrop plots. Combining area per plant and density data allowed an estimate of total bean leaf + pod area and total maize leaf area for each plot. Dividing these values by the plot area (400 m^2) provided the leaf area index (LAI) for maize and leaf + pod area index (LPAI) for beans.

Mean height per plot values were compared, and LPAI values were compared for beans by using two-way ANOVA (four treatments × three blocks) for each assessment date. ANOVAs also were performed after data from monocrop and addition plots and from intercrop and removal plots were combined, because

TABLE 1. Slopes of dispersal gradients of *Uromyces appendiculatus* on beans, as influenced by competition with maize present before the dispersal event and/or interference by maize present during the dispersal event, during three releases from focal inoculum sources

	Single leaf[a]						Leaf combination[b]				
	1989			1990			1989			1990	
Factor	Release 1[c]	Release 2	Release 3	Release 1[d]	Release 2	Release 3	Release 1	Release 2	Release 3	Release 1	Release 3
Main effects[e]											
Competition											
Absent	−0.939[f]	−1.572	−1.560	...	−1.674	−1.563	−0.807	−1.633	−1.543	...	−1.575
Present	−0.942	−1.749	−1.758	...	−1.891	−1.758	−0.603	−1.729	−1.884	...	−1.826
Significance[g]	0.987	0.105	0.045	...	0.090	0.127	0.245	0.486	0.033	...	0.096
Interference											
Absent	−0.950	−1.706	−1.671	...	−1.717	−1.757	−0.822	−1.737	−1.717	...	−1.775
Present	−0.932	−1.615	−1.647	...	−1.857	−1.526	−0.588	−1.625	−1.683	...	−1.586
Significance	0.926	0.366	0.762	...	0.789	0.104	0.188	0.416	0.703	...	0.177
Interaction[h]											
−Cmp/−Int (Monocrop)	−1.058	−1.710	−1.606	−1.504	−1.512	−1.701	−1.024	−1.803	−1.618	−1.589	−1.697
+Cmp/−Int (Removal)	−0.842	−1.701	−1.736	...	−1.923	−1.812	−0.620	−1.671	−1.866	...	−1.853
−Cmp/+Int (Addition)	−0.820	−1.433	−1.514	...	−1.853	−1.425	−0.590	−1.463	−1.469	...	−1.452
+Cmp/+Int (Intercrop)	−1.043	−1.796	−1.779	−1.718	−1.861	−1.678	−0.586	−1.786	−1.896	−1.674	−1.787
−Cmp/+Int + +Cmp/−Int[i]	−0.831	−1.559	−1.625	...	−1.888	−1.619	−0.605	−1.567	−1.668	...	−1.652
Significance	0.296	0.093	0.422	...	0.046	0.495	0.252	0.128	0.393	...	0.549

[a] Single leaf for release 2, 1990, second trifoliate; all others, first trifoliate.
[b] Combinations for 1989 are release 1, primaries + first trifoliate; release 2, primaries + first + second trifoliates; release 3, first + second trifoliates. For 1990, release 1, primaries + first + second trifoliate; release 2, only single leaf available; release 3, primaries + first trifoliate.
[c] Each release conducted over a 3-day period of approximately 30 daylight hours beginning 35, 56, and 77 days after planting in 1989, and 35, 52, and 70 days after planting in 1990 for releases 1, 2, and 3, respectively.
[d] Poor plant growth eliminated addition and removal treatments. Significance levels for one-way comparison of monocrop and intercrop in release 1 were 0.293 for single leaf and 0.582 for leaf combination.
[e] Mean of all plots with level of factor given (i.e., competition absent = mean of monocrop and addition treatment plots; competition present = mean of intercrop and removal plots; interference absent = mean of monocrop and removal plots; interference present = mean of intercrop and addition plots). See text for explanation of treatments.
[f] Slope for each plot is parameter b of modified Gregory model, $\log(y) = \log(a) - b[\log(x+c)]$. In these experiments, y = number of uredinia at distance x from source center, and c = 0.6 m, the source radius. Parameters a and b were determined by least-squares regression applied to mean lesion counts on leaves of four trap plants equidistant from inoculum source.
[g] Probability of falsely rejecting H_0, no difference among factor levels.
[h] Mean of all plots with level combinations given (e.g., −Cmp/+Int = mean of addition treatment plots in which competition [Cmp] was absent but interference [Int] was present). See text for explanation of treatments.
[i] Mean of addition and removal treatments for comparison to intercrop treatment.

these pairs of treatments are almost identical in bean growth. The power of the test is thereby increased.

RESULTS

Dispersal effects. The modified Gregory model, with no apparent pattern in residual values and relatively high coefficients of determination, consistently explained the data better than the negative exponential. The first quartile r^2 ranged from 0.782 to 0.918 for modified Gregory and 0.448–0.863 for negative exponential for single-leaf data from each experiment. Modified Gregory slopes were, therefore, used for further analysis.

Figure 3 illustrates the general fit and steepness of the gradient for single-leaf assessments from release 3 in both years. Gradient slopes for other releases and leaf combination assessments are summarized in Table 1. When subjected to ANOVA, the competition factor emerged as consistently steepening the dispersal gradient. This effect was significant at $P = 0.045$ and $P = 0.033$ (single leaf and combined leaves, respectively) in release 3, 1989, and significant at approximately $P = 0.10$ for releases 2 and 3 of both years in all but one case (release 2, 1989, combined leaves). Interference generally did not have an effect on dispersal gradients, although a more shallow slope may have occurred in release 3, 1990, for single-leaf data ($P = 0.104$). The competition × interference interaction effect was significant at $P = 0.093$ in release 2 of 1989 and $P = 0.046$ in release 2 of 1990 for single-leaf data. In the former case, the gradient was flattened again by interference (addition treatment) but steepened when combined with competition. Interference alone steepened the gradient in the latter case, however. None of the factors had significant effects during release 1, when the stand was young. On the basis of lesion counts on control plants, background inoculum contamination was present at levels sometimes equivalent to those at the edge of experimental plots and originated in the field (due to long gradient tails or our earlier inoculations at the same location) and in the humidity tent during the overnight periods of residence.

It appears from estimates of the total number of uredinia (Table 2) that competition significantly altered deposition, but that the effect is not consistent. In release 2, deposition was increased by competition in 1989 ($P = 0.070$) and 1990 ($P = 0.014$), but by release 3 a pronounced reduction in deposition occurred in both years ($P = 0.001$ in 1989 and $P = 0.073$ in 1990; all P values are for single-leaf data). Interference tended to reduce the number of spores deposited in releases 2 and 3, although this effect was significant below $P = 0.10$ only in the case of release 2, 1990, single leaf. The interaction between interference and competition was significant below $P = 0.10$ in several instances, but the effect was not consistent, even for different leaf sets within a single release (see release 3, 1990).

Nondispersal effects. Mean severity levels for inoculated leaves relative to the monocrop treatment are summarized in Figure 4. Actual values varied considerably among inoculations, due possibly to several factors that changed between experiments (e.g., leaves of different ages compared; small change in inoculation rate for inoc 2, 1989; different weather conditions). Thus, com-

TABLE 2. Estimated total number of uredinia of *Uromyces appendiculatus* on beans in 20- × 20-m plots, as influenced by competition with maize present before the dispersal event and/or interference by maize present during the dispersal event, during three releases from focal inoculum sources

	Single leaf[a]						Leaf combination[b]				
	1989			1990			1989			1990	
Factor	Release 1[c]	Release 2	Release 3	Release 1[d]	Release 2	Release 3	Release 1	Release 2	Release 3	Release 1	Release 3
Main effects[e]											
Competition											
Absent	15,892	23,875	17,480	...	1,091	32,165	40,128	40,048	36,151	...	57,527
Present	12,870	28,333	10,330	...	2,147	23,040	38,350	46,158	25,315	...	37,297
Significance[f]	0.327	0.070	0.001	...	0.014	0.073	0.843	0.095	0.004	...	0.093
Interference											
Absent	14,047	26,853	14,560	...	1,827	29,755	36,286	45,022	36,676	...	47,036
Present	14,714	25,356	13,250	...	1,458	25,931	42,193	41,184	26,684	...	49,887
Significance	0.822	0.488	0.294	...	0.079	0.373	0.518	0.260	0.132	...	0.706
Interaction[g]											
−Cmp/−Int (Monocrop)	14,767	25,745	20,326	50,190	669	33,583	32,974	45,271	43,484	103,957	53,814
+Cmp/−Int (Removal)	13,327	27,961	8,793	...	2,986	25,927	39,597	44,774	26,458	...	40,258
−Cmp/+Int (Addition)	17,017	22,005	14,633	...	1,556	30,746	47,281	34,825	28,815	...	61,241
+Cmp/+Int (Intercrop)	12,412	28,706	11,867	43,159	1,377	18,708	37,104	47,543	24,553	90,372	32,856
+Cmp/−Int + −Cmp/+Int[h]	15,172	24,983	11,713	...	2,271	28,337	43,439	39,800	25,505	...	50,750
Significance	0.596	0.311	0.008	...	0.015	0.834	0.367	0.076	0.018	...	0.655

[a] Single leaf for release 2, 1990, second trifoliate; all others, first trifoliate.

[b] Combinations for 1989 are release 1, primaries + first trifoliate; release 2, primaries + first + second trifoliates; release 3, first + second trifoliates. For 1990, release 1, primaries + first + second trifoliate; release 2, only single leaf available; release 3, primaries + first trifoliate.

[c] Each release conducted over a 3-day period of approximately 30 daylight hours beginning 35, 56, and 77 days after planting in 1989, and 35, 52, and 70 days after planting in 1990 for releases 1, 2, and 3, respectively.

[d] Poor plant growth eliminated addition and removal treatments. Significance levels for one-way comparison of monocrop and intercrop in release 1 were 0.739 for single leaf and 0.905 for leaf combination.

[e] Mean of all plots with level of factor given (i.e., competition absent = mean of monocrop and addition treatment plots; competition present = mean of intercrop and removal plots; interference absent = mean of monocrop and removal plots; interference present = mean of intercrop and addition plots). Based on uredinia counts on 33 trap plants located along two transects intersecting at the inoculum source. See text for explanation of treatments.

[f] Probability of falsely rejecting H_0, no difference among factor levels.

[g] Mean of all plots with level combinations given (e.g., −Cmp/+Int = mean of addition treatment plots in which competition [Cmp] was absent but interference [Int] was present). Based on uredinia counts on 33 trap plants located along two transects intersecting at the inoculum source. See text for explanation of treatments.

[h] Mean of addition and removal treatments for comparison to intercrop treatment.

parisons of absolute severity levels are inappropriate. Although intercropping increased the level of disease early in crop growth (inocs 1 and 2, 1989, and inoc 1, 1990), only the pronounced reduction in disease in late 1989 (inoc 3) had a high level of significance ($P = 0.023$). The influence of competition alone on severity (removal treatment) was not significant in any case at the $P < 0.10$ level (Fig. 4).

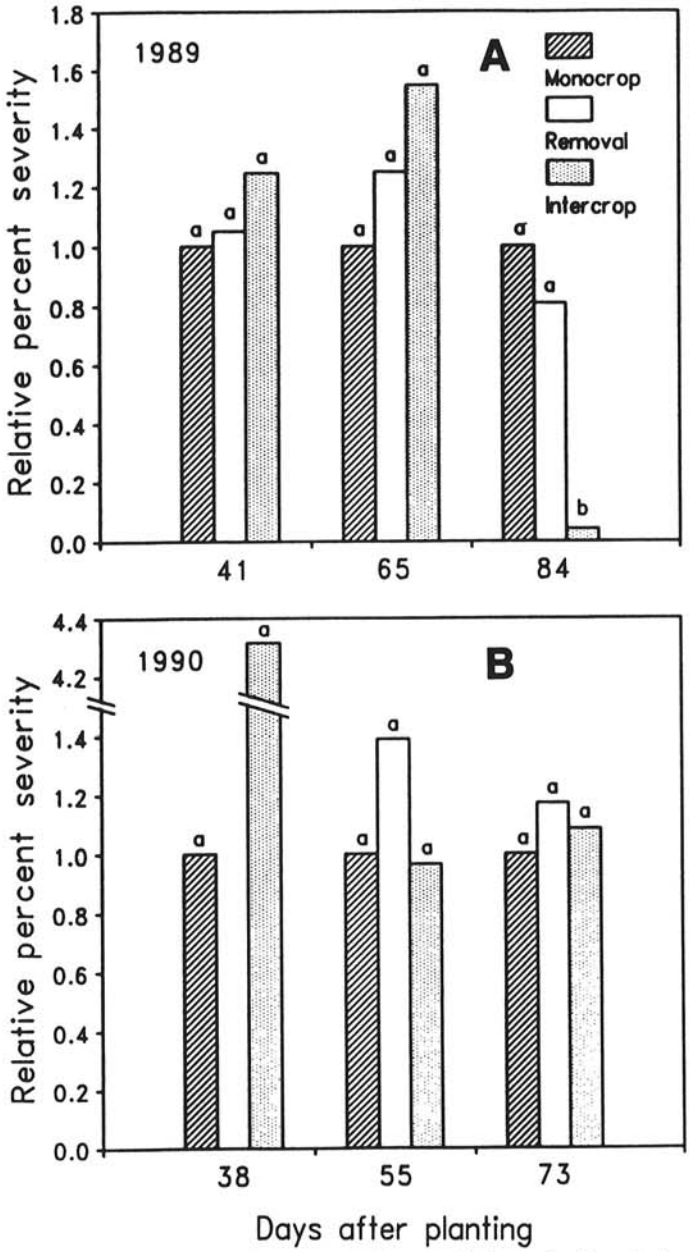

Fig. 4. Relative severity levels, approximately 12 days after inoculation with *Uromyces appendiculatus* urediniospores, on single leaves of the same age of bean plants growing within canopies of rust-resistant beans in monocrop, intercrop with maize, and intercrop with maize until immediately before inoculation (removal). **A,** 1989; **B,** 1990. Bars represent means of three plots each containing five inoculated plants. Data for each year are presented relative to the monocrop at three different dates after planting. Each date was a separate inoculation experiment and utilized different plants. The removal treatment was not possible for inoc 1, 1990, because of poor maize growth. Actual severity values for monocrops were 26.3, 3.2, and 4.5% for successive inoculations in 1989; and 0.2, 11.0, and 7.4% for 1990. Treatment significance levels, based on one-way ANOVA (with blocking) of $\log(x + 1)$ transformed severity values, are $P = 0.431$, 0.941, and 0.023 for successive inoculations in 1989, and $P = 0.504$, 0.613, and 0.945 for 1990. Bars with different letters indicate significant differences ($P < 0.05$) by the Newman-Keuls test performed on $\log(x + 1)$ transformed data.

Uredinia appeared on all plants 9 days after inoculation for inoc 1, 1989, and latent period could not be assessed for inoc 3, 1989. For other inoculations, mean latent period (days) for monocrop, removal, and intercrop treatments were 8.7, 9.1, and 8.3, respectively, for inoc 2, 1989; 13.0, 13.3, and 11.5 for inoc 1, 1990; 11.2, 11.3, and 10.9 for inoc 2, 1990; and 9.1, 8.9, and 10.0 for inoc 3, 1990. None of these differences was significant below $P = 0.10$ for any inoculation.

Overall effects. An interaction between intercropping effect and location was evident in severity values for 1989 and 1990 (Table 3). The interaction of cropping system (monocrop/intercrop) × plot pair in an ANOVA performed on all plot pairs, blocked by year, had a significance level of $P = 0.112$. AUDPC values for individual plots showed a consistent disease reduction because of intercropping in pairs A and B, located in the same section of the research farm, whereas pair C, in a different area (Fig. 1), showed a severity increase in both seasons. This interaction warrants a separate consideration and statistical analysis of the experiment at the two locations. When ANOVA is performed on pairs A and B, with cropping system as main effect and year and plot pair regarded as blocks, the severity reduction of 27% was significant at $P = 0.07$. ANOVA of pair C, with cropping system again as main effect and year regarded as a block, indicated that the 29% severity increase because of intercropping had only $P = 0.32$. However, the power of this test was lower than that for pairs A and B because of less replication.

Wind data were available from Salem, OR (47). Winds were from the north octant 45 and 40% of the days during which this experiment was conducted (inoculation to final assessment) in 1989 and 1990, respectively. Winds were from the south octant 10 and 17% of the days, the southwest 13 and 13% of the days, the west 5 and 17% of the days, and the northwest 25 and 13% of the days in 1989 and 1990, respectively. The resultant daily vector was never from the east or southeast, and only in 1989 was it from the northeast (3% of the days).

Crop growth. Beans experiencing competition (intercrop and removal treatments) had consistently lower LPAI than those without competition (monocrop and addition) in 1989 (Fig. 5). Although this difference is not significant when analyzed as four treatments, an ANOVA combining pairs of treatments as above increased the power of the test sufficient to yield differences significant below $P = 0.05$ for the first two assessments in 1989. No significant differences were evident by either analysis for 1990 data, and, indeed, the beans in the intercrop were larger than those in the monocrop. This concurs with visual observation, particularly in two of the blocks, and may have been due to soil and irrigation factors early in the establishment of the plots rather than the presence or absence of maize.

The differences in crop growth patterns between 1989 and 1990 also are illustrated in Figure 5. In 1989, the reduction in bean leaf area late in the season (Fig. 5C) was due to senescence and defoliation that could be observed visually by release 3 and inoc 3. Experiments were conducted at shorter intervals in 1990, partly to avoid this phenomenon, and this approach was successful (Fig. 5D). Overall, LPAI values were roughly equivalent at approximately 60 days after planting for the 2 yr, and although it is likely that growth occurred after this date in 1989, defoliation was observed by the time of release 3, 77 days after planting.

Bean height (Fig. 5A,B) was not significantly ($P < 0.10$) influenced by treatment in either year. Although maize quickly exceeded beans in height (Fig. 5A,B), the contribution of maize to total leaf area was relatively small at these planting densities (Fig. 5C,D).

DISCUSSION

Dispersal effects. Among the factors that may be important in altering bean rust epidemiology when beans are intercropped with maize, the one most clearly influential was that of competition as it affects spore dispersal. Any contribution of the physical presence of maize to dispersal alterations was apparently overwhelmed by competition effects. These findings agree with those

of Burdon and Chilvers (11–13), in that interference was not a factor in intercrop disease reductions for either a soilborne or an airborne foliar pathogen (they did not evaluate dispersal gradients). The most obvious outcome of competition that would affect dispersal is a reduction in the size of the bean plant, and this was the case as measured by LPAI data in 1989 (Fig. 5C). Steeper dispersal gradients paralleled reductions in LPAI during this season (Table 1). However, in 1990 the effect of competition on LPAI was more ambiguous; intercrop but not removal LPAI values were relatively high (Fig. 5D). Competition effects on dispersal were still present during 1990, but at lower significance levels (Table 1).

It is difficult to predict what effect reduced LPAI would have on a dispersal gradient (31). Workers have failed to demonstrate consistent relationships between these factors (20,30), despite assumptions that increased density would increase deposition and lead to steeper gradients. However, as Barrett (7) demonstrated for disease reductions in multilines, a fractional decrease in spore removal from a spore cloud at each distance from a source, due to any factor, will not steepen a gradient described by an inverse power law. Aylor and Ferrandino (6,17) argued that turbulent mixing and rapid spore escape rather than deposition dominate *U. appendiculatus* dispersal in a sparse bean canopy (LAI < 1.6) when winds are not calm, conditions similar to those experienced here. Turbulent removal is theoretically expected to be described by an inverse power law model rather than a negative exponential model (6,17,18), as was the case with our data. The view that spore escape creates the steepened gradients in plots with maize competition may be inferred from consideration of spore deposition estimates for release 3 (Table 2), because fewer spores were retained in these plots. However, plots with the same effect of competition on gradient slope in release 2 appeared to retain more spores, suggesting that increased deposition rather than turbulent diffusion may have dominated at that point in time. The mechanisms of gradient alteration may themselves change during crop growth, although if this were the case one would expect the shallow gradients seen in interference treatments without competition to result in greater spore retention in release 2; this was not the case. The mechanisms accounting for spore retention are, therefore, unclear.

The evidence for steeper gradients coupled with reduced spore retention and reduced LPAI from increased competition under intercropping is nonetheless compelling, in terms of magnitude and significance level, in release 3 of both years. If a change in LPAI produced a change in turbulent mixing that altered spore removal nonuniformly (i.e., greater vertical movement away from the center of a plot), then a steeper gradient may be observed within the plot. One might expect increased mechanical turbulence because of greater surface roughness in a sparse bean stand or increased convective turbulence because of more exposed ground, both of which could vary spatially in the plot.

Interpreting this effect of competition on dispersal, perhaps because of reductions in bean leaf area, is complicated by the relatively benign influence of the presence of maize during dispersal. The expectation that a layer of maize foliage would prevent spore escape may have been greatly influenced by the low density of the maize; the "breaks" observable in the maize canopy could have permitted substantial spore escapes. Furthermore, reductions in convection due to shading may be offset by an increase in surface roughness at this density. The artificially supported maize plants in the addition treatment maintained much of their physical integrity and were sometimes virtually indistinguishable from the intact maize plants even after the end of the 3-day dispersal event. Nevertheless, we cannot completely rule out the possibility that the treatment may not have mimicked the interference effects of living maize, so that the intercrop treatment was not a true combination of competition and interference treatments. Even so, the importance of competition in these studies is not diminished. Other factors conceivably might have affected dispersal via competition, such as changes in bean architecture.

Nondispersal effects. Nondispersal influences of intercropping were less pronounced than dispersal effects in this study, although in late 1989 disease severity was significantly reduced by nondispersal effects that were, furthermore, not due to competition. Microclimatic changes produced by maize were most likely responsible. These results are not consistent with assumptions that higher daytime humidity, measured in bean canopies grown under maize (9,45), would favor rust. Low light levels and leaf wetness also have been shown to favor infections (5,22,23). The duration of direct illumination on upper bean leaves is reduced under

TABLE 3. Area under the disease curve for bean rust epidemics in bean monocrops and bean-maize intercrops

Treatment[a]	Pair A[b]	Pair B	Pair C[c]	Mean Pairs A and B[d]
1989				
Monocrop	387.26	323.33	256.52	355.30
Intercrop	325.46	315.95	349.76	320.71
1990				
Monocrop	348.32	337.86	154.84	343.09
Intercrop	154.00	218.61	182.70	186.31
Mean 1989 and 1990				
Monocrop	367.79	330.60	205.68	349.20
Intercrop	239.73	267.28	266.24	253.51

[a] Area under the disease progress curve based on five weekly severity assessments of five pairs of bean rows in each plot, after inoculation with heavily diseased beans placed in plot center for 5–7 days.

[b] Pair = one bean monocrop plot and one bean-maize intercrop plot. Each plot was 18.3 × 18.3 m in 1989 and 20.0 × 20.0 m in 1990. All pairs located among mixtures of dwarf fruit trees (3–4 m in height), grapes, and roses; also, pair C had standard trees (7 m in height) to north and a golf course to the west.

[c] Probability of falsely rejecting H_0, no difference between monocrop and intercrop by ANOVA for individual plot values in pair C is 0.315.

[d] Probability of falsely rejecting H_0, no difference between monocrop and intercrop by ANOVA for individual plot values in pairs A and B is 0.066.

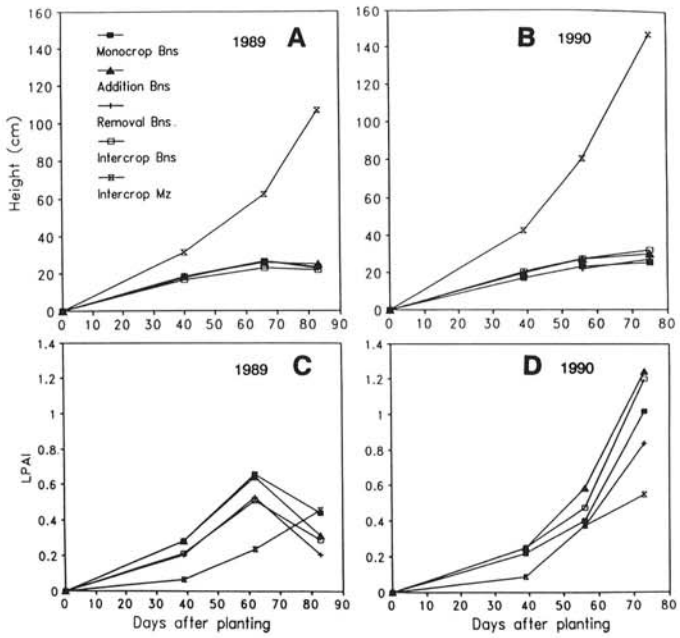

Fig. 5. Growth of beans and maize under the cropping treatments used to test competition and interference effects of maize on bean rust epidemiology. Points represent means of three replicates (plots) with randomly sampled plants in each plot. **A,** Height, 1989 (15 plants sampled per plot); **B,** height, 1990 (16 plants sampled per plot); **C,** leaf + pod area, 1989 (seven plants sampled per plot); **D,** leaf + pod area, 1990 (eight plants sampled per plot). Bean data for removal in 1990 are shown between the second and third sample dates only; removal treatment plots were not available at the first sample date because of poor maize growth. See text for explanations of treatments, sampling method, and area index calculations. LPAI = leaf + pod area index; Bns = beans; Mz = maize.

intercropping (*personal observation*), and growing beans under maize has resulted in prolonged and decreased periods of leaf wetness, depending on the season (29).

In September–October 1989, leaf wetness sensors were placed in one pair of plots used to evaluate overall effects of intercropping on disease (pair C). Leaf wetness duration was 14% greater in the intercrop plot than in the monocrop plot on 33 of the 45 days sampled, or a mean increase of 65 min (SD = 55 min). This relationship was reversed during the 12 remaining days, all of which had long absolute leaf wetness periods (leaf wetness duration 9% less in the intercrop or 79 min, SD = 72 min). In all cases, leaf wetness differences between the monocrop and intercrop occurred during dew formation and not during leaf drying or rainfall. Resource constraints prevented further sampling in other plot pairs or in the subsequent year, so it is unclear if the measured differences were truly due to intercropping effects. Wind speeds have been reduced in maize-bean intercrops (9), and this, along with increased humidity, would tend to favor dew formation under intercropping. Monocrops also may encourage dew by allowing substantial radiative heat loss from bean leaves in the absence of a maize canopy.

The mechanism by which disease reduction occurred in the intercrop in late 1989 is, therefore, unclear. It is important to recognize that this reduction was seen only once in these experiments, at a time when significant defoliation had taken place. That unique result may involve an interaction between bean leaf loss, which alone did not affect disease (removal, Fig. 4A), and the presence of maize. Under the more typical conditions of the other trials, comparatively small microclimatic effects of intercropping measured thus far (e.g., 2–3% increase in relative humidity [9,45]), coupled with the high degree of spatial and temporal variability in the bean canopy of a large field, may encompass the range of conditions under which *U. appendiculatus* will infect in either the monocrop or intercrop conditions.

Combined effects. In the experiments on overall disease effects of intercropping, the observation that bean rust was consistently reduced by interplanting with maize in two pairs of plots corroborates similar reductions reported earlier by Moreno and Mora (35) and Soria et al (43). More comprehensive studies conducted in Brazil (33) and Kenya (49) have shown decreases in rust severity due to intercropping that diminished or reversed depending on site and season. In our experiments, such variability was reflected in plot pair C, where intercropping did not affect or perhaps increased disease in both seasons. This contrasted with the disease reductions observed in pairs A and B, also during both seasons. The anomalous outcome in pair C was not an artifact of some localized plot effect, because randomization happened to reverse the positions of the monocrop and intercrop treatments in the two plots of pair C between 1989 and 1990. It was, therefore, the small change in location from pairs A and B to pair C that determined the effects of intercropping and not the variations between the two seasons.

The most notable distinction between the location of pairs A and B and that of pair C was the open exposure of the latter to the west (a golf course) and the presence of trees greater than 7 m in height to the north (Fig. 1), the direction from which winds most often originated (47). We suggest that variable intercropping effects may have been related to wind patterns. These may influence disease through dispersal directly or influence infection through changes in microclimate. As mentioned in the preceding section, an increase in leaf wetness duration was observed under maize in pair C, and this would be expected to favor disease (22,23). Indeed, AUDPC was higher in the pair C intercrop. The more typical reduction in rust severity under intercropping, seen in pairs A and B, may have been due in part to different patterns of leaf wetness.

Mechanism interaction. We conducted the experiments on the mechanisms of intercropping effects on disease to better understand the spatial and temporal variability observed by ourselves and others. However, the relative role of each of these mechanisms and their potentially complex interactions are not easily estimated. A reduction in infection after inoculation, as seen in one case in the nondispersal experiments, would reduce disease severity and provide less inoculum for dispersal, although this may only occur late in the season. Steeper dispersal gradients due to intercropping may slow the velocity of disease spread (48,32) but increase severity near the source. However, if increased spore escape is responsible for the steeper gradient, as discussed earlier, then less inoculum would be available for infection, and overall disease would be reduced. Computer simulations based on data from these experiments (8) showed that changes in total spore deposition rather than gradient slope were primarily responsible for dispersal-mediated effects on disease when each factor was varied independently; this indicates the importance of spore escape in this system. The observed reductions in LPAI due to competition would also remove effective targets for deposition and limit inoculum production later, as suggested for disease reductions when host density declines (14).

The qualitative sum of these factors may cause the reduction in rust severity commonly observed (33,35,41,43) and the potential for variable results (49). The experiment on overall disease reported here represents the true combination of all mechanisms at work, and strong variability was apparent between different sites on the research farm. Comparison between the experiment on overall disease and those on mechanisms should be made with some caution, because different bean genotypes were used and the beans generally grew more vigorously in the overall disease experiment (*personal observation*).

If these results are applied to other settings, additional mechanisms (e.g., induced resistance) and interactions of all mechanisms with other cropping systems and environments, different inoculation levels and patterns, and different spatial and temporal scales could contribute to variability. The main value of our findings is suggesting which factors are most important in altering bean rust due to intercropping with maize and the implications for intercropping as a disease management tool. The small plots used and their heterogeneous surroundings make the results relevant to small farmers in developing countries, particularly considering the consistent effects on epidemic components that were observed in this environment. If steepened dispersal gradients are primarily responsible for disease reduction in intercrops, conditions creating more shallow gradients such as larger, more abundant foci or strong background inoculum levels (21) may render intercrops ineffective. Furthermore, if gradient changes are due to competition, then efforts to improve bean yield in intercrops through competition reduction (increased fertility, wider maize row spacing, planting maize after beans), as has been suggested (19), might also eliminate any advantages of intercropping for disease control. Additional research on the mechanisms of intercrop-disease interactions will provide a framework for methodically evaluating the efficacy of a particular cropping system.

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