AN ABSTRACT OF THE THESIS OF

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Experiments were performed to determine the nature of maize influence on bean disease in additive-type intercrops. Overall effects of intercrops on angular leaf spot (caused by Phaeoisariopsis griseola) in Kenya indicated >23% reductions ($P<0.05$) in area under the disease progress curve (AUDPC) in two of three season-site combinations. Fertilization tended to increase disease (135-205%, $P<0.10$), but changes in bean density or planting pattern had no effect. Intercrops reduced temperature and wind velocity, but increased relative humidity. A 27% AUDPC reduction ($P=0.07$) in bean rust (caused by Uromyces appendiculatus) due to intercropping was observed in Oregon in 1989 and 1990 in two of three locations.

Mechanisms of maize influence on rust were also assessed in Oregon. Intercropping, and competition of maize with beans alone, consistently steepened dispersal gradients ($P<0.10$). Interference of maize with dispersal alone tended to flatten gradients. Spore retention in

plots was increased in mid-season, then decreased late in the season, due to competition in both years ($P<0.05$). Intercropping reduced infection by 96% late in 1989 ($P<0.05$), probably due to microclimatic influence of maize.

The data from these experiments were used as inputs for computer simulation to evaluate effects of specific mechanisms on disease dynamics. Combination of all mechanisms (= intercrop) reduced AUDPC to 32% of monocrop, using 1989 data. Infection efficiency reductions, and to a lesser extent dispersal effects, were responsible for these changes. Intercrop effects declined as pathogen multiplication rate (DMFR) increased. No intercrop effect occurred at any DMFR using 1990 data, although interference and competition effects of maize alone both increased AUDPC at low DMFR. Partitioning dispersal effects into those due to gradient slope changes and spore retention indicate that the latter accounts almost entirely for disease alteration.

Effects of Intercropping Beans with Maize on Angular Leaf Spot and Rust of Beans

by

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Chapter I

INTRODUCTION

The long, regimented rows of genetically uniform single crops, a sight which we in the industrialized countries view as the natural state of things, is an anomaly for many of the world's farmers. Aside from plantations of bananas, coffee, and other export crops, and rice grown in flooded paddies, food for local consumption is generally grown in small (<5 ha), sometimes haphazard arrangements of species mixtures (9,14,27, personal observation). This simultaneous cultivation of multiple crops, or intercropping, is abundantly evident to casual observers travelling in developing countries, though good quantitative data are limited. Based on 1960 FAO census data, Kass (14) estimates that 70-80% of non-rice crops in India, 50% of maize in Jamaica, and 25% of millet and groundnuts in Senegal were intercropped at that time. Francis et al. (9) determined that 98% of the cowpeas in Africa, and 60% of the beans and maize in Latin America, are intercropped. Mixtures observed range from the ubiquitous sorghum-pigeonpea and maize-bean associations (27) to sugarcane-soybean mixtures (21), potatoes-

pyrethrum-pineapple mixtures (personal observation) and gardens of >10 species (8).

Farmers may grow multiple rather than single crops for a number of reasons, including overall yield advantages, increased yield stability, economic advantages (e.g. producing a food crop and cash crop simultaneously on limited land), pest control, efficient use of nutrients, and, of interest to us here, disease control (14). One must not overlook the strong influence of social tradition on most farming practices; our need to find a "reason" for intercropping reflects our own cultural bias as much as a scientific rationalism. Indeed, the transition from a hunter-gatherer to an agricultural society probably involved a conscious encouragement of desirable species in diverse natural plant communities (23), and it is the switch from this intercrop model to monoculture that requires a leap of faith for most of the world's farmers to this day.

The purpose of the research undertaken for my Ph.D. was to gain a better understanding of the relationship between intercropping and disease. I chose the common bean (Phaseolus vulgaris L.) and maize (Zea mays L.) combination as a model system, because of its common occurrence in the developing world (9), the importance of bean diseases to limiting production (2), logistic advantages of annual crops which can be cultivated in a variety of environments,

and the potential for such physiognomically disparate species to illustrate the full range of intercrop-disease interactions. Using the pathogens Uromyces appendiculatus (Pers. ex Pers.) Unger, cause of bean rust, and Phaeoisariopsis griseola (Sacc.) Ferraris, cause of angular leaf spot of beans, I sought to answer the following questions: (1) What is the magnitude of any intercropping effect on disease severity?; (2) What are the mechanisms by which intercrops affect disease?; and (3) What are the relative and combined effects of these mechanisms on overall disease?

The first question has been the inspiration for a good deal of speculation, and many scientists have suggested that intercropping generally should reduce disease (1,3). Empirical data are limited, however, and both reductions in disease (e.g. 6,10,22) and increases (11,14) relative to monocrops have been reported. In the bean-maize system, bean rust has decreased under intercropping in some studies (16,19,24) but has remained unaffected in others (16,22). ALS has been more variable, with maize intercrops lowering disease, increasing disease, or having no effect on disease (15,17,18,22). These results seem to depend on season and site (15,22). My own evaluation of intercrop effects on ALS in Kenya, including interactions with planting density, planting pattern, and fertility levels consistent with those used by local farmers, is described in Chapter II.

The influence of intercropping on rust in Corvallis, OR is reported in Chapter III.

The variable effects of intercropping indicate that a purely phenomenological research approach will not easily lead to generalizations or predictive power. An understanding of the mechanisms by which disease is influenced in crop associations is necessary. Several mechanisms have been suggested, including alteration of pathogen dispersal by the non-host through propagule interception or wind, rain, and vector disruption; reduced host density (in replacement-type intercrops); microclimatic change in the pathogen environment; and changes in infection due to induced resistance or non-host pollen (3,12,26). To this list may be added the competitive effects of the non-host, which might influence dispersal or susceptibility of the host. The small amount of research done on these topics has focussed on density effects (4,5,6,7), or microclimatic changes <u>per se</u> caused by addition of the second crop (15,25). I report on temperature and moisture alterations under maize intercrops in Chapter II and briefly in Chapter III. However, these studies have limited value; density effects do not apply to additive-type mixtures, and those measuring environmental effects cannot be directly related to pathogen response.

We therefore attempted to obtain new data by directly measuring the effects of maize on dispersal and non-

dispersal portions of the U. appendiculatus life cycle at various times during crop growth. Dispersal effects were further partitioned into those due to interference of maize with spore movement and competition by maize with beans. These experiments are reported in Chapter IV.

It is, unfortunately, impossible to devise a field experiment which will evaluate the effects of each of these mechanisms, alone and in combination, on disease development throughout the season. Computer simulation models allow at least a qualitative look at this question, but require input values which represent the isolated effects of each of the mechanisms. These data have previously been unavailable for intercrop systems, but the results from the experiments of Chapter IV provide the necessary inputs. Chapter V describes these simulated epidemics, run on a modified version of the EPIMUL program (13,20). A range of pathogen multiplication rates was employed, and it was possible to partition dispersal effects into those due to changes in gradient steepness and those due to changes in spore retention in plots.

The work reported herein has implications for future research directions and the value of various production approaches for intercrop systems. Although it has been conducted with subsistence agriculture in developing countries in mind, the findings are by no means limited to these circumstances, and may be used in nascent First-World

intercrop systems. I share John Vandermeer's (27, p. 13) view:

> There seems to be a prejudice among casual observers and intercropping researchers alike that intercropping is for peasant farming and has no place in modern agriculture. I am violently opposed to this idea. . . When 'modern' agriculture involves varieties specifically adapted for production in monoculture, machines specifically adapted for production in monoculture, and research methodology specifically adapted for improvement of monocultures, what might one expect? . . . until modern production technology is developed, including some sort of theoretical foundation for the agronomic aspects . . . it will be a *fait accompli* that intercropping will have no place in modern agriculture.

It is my hope that the research described here will become part of a larger "theoretical foundation," one which will at once help us understand the nuance of traditional crop husbandry, and allow us to predict and optimize the outcome of future intercrop production.

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Chapter II

CHANGES IN ANGULAR LEAF SPOT OF BEANS DUE TO INTERCROPPING BEANS WITH MAIZE IN KENYA

M.A. Boudreau

Abstract

Angular leaf spot severity was evaluated on common beans which had been planted alone (monocrop) and also simultaneously with maize (intercrop) at Kabete, Kenya in November 1986 (short rains), and at Kabete and Thika in April 1987 (long rains). Intercropping reduced the area under the disease progress curve (AUDPC) in the short rains and at Thika in the long rains by >23% at bean:maize proportions of 2:1 ($P<0.05$), but did not reduce AUDPC significantly at Kabete in the long rains. Additional treatments evaluating bean density and planting pattern (row vs. random) conducted in the short rains had no effect on AUDPC ($P<0.10$), although disease was reduced by 12-17% at higher bean densities when maize was not present. Fertilization increased AUDPC by 135-205% ($P<0.10$) in the long rains at both sites. Microclimatological observations made at Kabete in the long rains indicated average leaf temperature reductions of 0.6 C, air temperature reductions of 0.2 C, and wind velocity reductions of 55-63% in the intercrop relative to the monocrop. Relative humidity in

the intercrops averaged 1.8 percentage points above the monocrop values. The results corroborate other data indicating a significant but variable decrease in angular leaf spot due to maize intercrops.

Introduction

Intercropping, the simultaneous cultivation of more than one crop species in close association, is practiced extensively in much of the world, particularly in developing countries (15,29). Publications on intercropped systems to date have been focussed on overall yield effects under various species mixes (e.g. 11,15), though some limited work on insect pests has been reported (21,23). One additional outcome of a multiple-cropping strategy may be improved disease suppression, suggested both theoretically (2) and empirically (4,12,22) for a range of crop combinations. However, disease increase under mixed cropping has been reported (13,19). Workers have suggested probable mechanisms of disease alteration, such as microclimatic changes, spore trapping by non-host, and induced resistance, but most of their work is speculative (14,20,28). Furthermore, the research which has been undertaken on any aspect of intercropping has generally been done under idealized conditions (e.g. high fertility, irrigation, chemical pest controls) which may not represent the practices of small farmers.

One of the most common intercrop associations combines common bean (Phaseolus vulgaris L.) with maize (Zea mays L.) (9), and intercrops dominate bean/maize cultivation in Kenya (25). Bean production in the tropics is severely

constrained by angular leaf spot (ALS), caused by Phaeoisariopsis griseola (Sacc.) Ferraris, with yield losses estimated as high as 80% (8). Reductions in ALS severity due to intercropping have been reported (17,18,22), but in other experiments intercropping had no effect or even increased ALS severity (17,19).

This paper describes research conducted in Kenya over two growing seasons in 1986-87 to determine the effects of interplanting maize with beans on ALS, using varying plant arrangements, plant densities, and soil fertility regimes. The levels of these treatments, and cultural practices generally, were designed to reflect those of Kenyan farmers.

Materials and Methods

Experiments were conducted at the University of Nairobi Agriculture Field Station, Kabete, during October 1986 - January 1987 (the "short rains"); and at the National Horticulture Research Station, Thika, as well as Kabete, during April - July 1987 (the "long rains"). Thika is approximately 45 km northeast of Kabete, with generally warmer and drier conditions (16). Planting density and pattern, fertility, and weed and insect management regimes were chosen to approximate those employed by local farmers, based on personal observations, discussions with district crops officers, extension agents, and farmers in Central and Eastern Province; and survey data (25).

Short rains

The experiment was arranged in a multifactorial randomized complete block design with four blocks, using 5 x 5 m plots as the experimental unit. Three factors were evaluated: bean:maize proportion, bean density, and bean planting pattern. Three levels of the first factor (all beans, 4 bean plants:1 maize plant, 2 bean plants:1 maize plant), two of the second (9.2 and 5.6 bean plants/m^2), and two of the third (beans and maize in single alternating rows, or beans randomly placed among maize rows) were employed. Twelve treatments were therefore included in each block. The various bean:maize proportions were

achieved for each bean density by varying the within-row spacing of the maize. Inter-row spacing for maize, and beans when planted in rows, was 75 cm. Two border rows of maize were planted around the entire perimeter at 1.5 and 2.25 m from the edge of the plots.

Mwezi Moja NB 518 beans, produced by the Crop Science Dept. at Kabete, and Hybrid 5012 maize (Kenya Seed Co., Kitale, Kenya) were used. No pesticides were applied during the season. Planting occurred on 27, 28, and 30 October 1986; and plots were irrigated with overhead sprinklers for 3 h on 31 October to ensure germination. Emergence occurred by 10 November 1986. Plots were thinned and weeded manually immediately after emergence, and weeded again 3 weeks later. Maize was topdressed with 4 g diammonium phosphate per plant on 21 November when phosphorous deficiency symptoms appeared.

Disease assessment commenced with the onset of angular leaf spot symptoms on 25 November. Ten plants were chosen randomly in each plot from within the central 9 m^2 to avoid edge effects. Each leaflet of the first four trifoliate leaves on the main stem, hereafter known as T-1 (oldest, immediately above the primary leaves) through T-4 (youngest), was evaluated at 6-8 day intervals using the Horsfall-Barratt system (10). Although seven assessments were completed, defoliation of older leaves and late disease onset on younger leaves provided fewer than seven

assessments for leaves of any given age. Ultimately, three sequential estimates of disease severity for T-1 and T-2 and four for T-3 and T-4 were obtained, each representing different calendar dates. However, data for single sample dates were missing from one plot for T-1 and T-3, and three plots for T-2, out of the total of 48 plots.

Long rains

Experiments during the long rains, this time conducted at two sites, included some design modifications based on observations from the short rains. The main factor under study, proportion of maize, was included as before at the same levels. Density and planting pattern factors were abandoned in favor of a fertility treatment for beans (no fertilizer and 50 g diammonium phosphate per 5 m row), to reduce the high degree of heterogeneity in plant stature observed during the previous season. All maize was fertilized at planting with 4 g diammonium phosphate per planting hole. The alternating-row planting pattern and high bean density of the previous season were retained. Six treatments per block were therefore realized. In order to decrease between-plot interference, all plots were separated by 4 m with a swath of oats 1 m wide sown in the centre at 80 kg/ha at the time of bean/maize planting. A similar swath was planted as a continuous border around the site perimeter, 1.5 m from the edge of the plots. Irrigation was necessary at Thika only (see below).

Varieties and husbandry techniques were identical to the short rains experiment in all other respects. Planting was done on 8 April at Kabete and 20-21 April at Thika, with emergence by 20 April and 4 May, respectively. At Thika, animal damage necessitated some replanting followed by irrigation, and, unfortunately, the complete elimination of all 4:1 bean:maize treatments from the analysis. The high fertility, 2:1 bean:maize treatment in one block was also destroyed at Thika.

Disease assessment began on 14 May at Kabete and 22 May at Thika. Evaluations were done at five-day intervals, allowing four sequential disease severity estimates for T-1 and T-4, and five observations for T-2 and T-3. Again, these assessment periods represent different time frames during the epidemic. Data for a single sample date was missing from one plot for T-2.

To better understand potential mechanisms of disease alteration in mixed systems, some environmental factors were measured at the Kabete site. Abaxial leaf surface temperature, and air temperature and relative humidity immediately above the same leaf, were recorded for the middle leaflet of a mid-canopy trifoliate leaf (T-1, T-2, or T-3, depending on the date) on five randomly-selected plants in each plot. The three maize proportion levels for any given fertility level x block combination were done consecutively, allowing for the most precise comparison of

humidity and temperature effects due to intercropping with the equipment available. A complete sampling cycle of all plots was done once on 5 and 26 May, and twice on 11, 16, 31 May and 5 and 10 June, though no leaf temperatures were taken on the final date. A copper-constantan thermocouple leaf temperature probe built and kindly supplied by Dr. C.L. Coulson in the Crop Science Department, University of Nairobi; and an aspirated digital psychrometer were used for the measurements.

Wind velocities among the three maize-proportion treatments in one of the blocks were compared on the same days, except 5 May, using a portable miniature-cup anemometer (Rauchfuss Instruments & Staff Pty. Ltd., Burwood, Victoria, Australia). Four sets of readings were taken over a 1.5-hr period: two heights (approx. 25 cm above and at the top of the bean canopy) x two fertility treatments. In other words, an anemometer sensor was placed in the center of a high-fertility, all-bean plot; another in a high-fertility, 4:1 bean:maize plot; and a third in a high-fertility, 2:1 bean:maize plot; all at bean level, and all in the same block. These ran simultaneously and mean wind speeds recorded for 1.5 hr, then each rotor was raised and the readings repeated. Two more sets of readings were similarly taken in the low fertility plots of the same block. I was limited to three working sensors, precluding further simultaneous wind speed comparisons.

However, after the final disease assessment, wind measurements were taken in all plots of each of the four blocks to validate the consistency of patterns, until then only observed in one block.

Data analysis

Area under the disease progress curve (AUDPC) was calculated for each set of trifoliates using severity values, after the mid-point mean method of Shaner & Finney (24). Three-way and two-way analysis of variance with blocks were performed on the AUDPC data for the short rains and long rains experiments, respectively. Newman-Keuls multiple comparisons were used where appropriate. Plots with missing values for a single sample date were estimated iteratively as outlined by Shearer (26), and the estimates used for AUDPC calculations. A reduction in the error degrees of freedom was made in the ANOVA for each AUDPC value that was an estimate of a missing value.

Temperature and relative humidity data were ranked among the three maize proportion treatments at each fertility level x block combination for each sampling cycle. The number of sampling cycles for which the leaf and air temperature was higher in the monocrop than in both intercrop treatments, and for which the relative humidity was lower in the monocrop than in both intercrop treatments, were recorded for each fertility level x block combination. Chi-square analyses were done to determine if

these frequencies were greater than would be expected due to random variation.

Results

Disease

Effects of intercropping, plant density, and fertility on AUDPC shown in Figs. II.1-II.3 and Tables II.1-II.3 are presented separately for clarity, but means and P-values represent single analyses done for each of the three experiments (i.e. short rains Kabete, long rains Kabete, and long rains Thika). For example, only one ANOVA was done on all disease data from the Kabete short rains, and therefore the P-values given in Table II.1 for the maize proportion factor correspond to the AUDPC values of Fig. II.1.

In both the short rains at Kabete and the long rains at Thika, AUDPC was reduced for all bean leaves evaluated in the intercrops (Figs. II.1 & II.3). The high proportion of maize (2:1 bean:maize) significantly reduced disease levels from those in monocrops by 24% (T-2) and 33% (T-3) at P=0.045 and P=0.097, respectively, during the short rains. Intercropping with less maize (4:1 bean:maize) consistently resulted in a level of disease intermediate between the monoculture and high maize proportion treatment, though these differences were not significant below P=0.10 by the Newman-Keuls test. Only one maize proportion was available at Thika, but reduction of 25% (T-1) and 34% (T-3) were significant at P=0.010 and P=0.053,

respectively. At Kabete during the long rains, no pattern of disease alteration or effect significant below P=0.10 of the intercrop treatments was apparent (Fig. II.2).

Influence of bean density and planting pattern on ALS severity, and their interaction with intercropping, were not significant below P=0.10 for any of the leaves assessed during the short rains at Kabete. Although mean AUDPC for randomly planted beans was higher than that for row plantings on all four trifoliates, the greatest difference (28.45 for row planting and 31.01 for random planting on T-1) was negligible, and P>0.25 in all cases. Greater differences resulted from altering bean density (Table II.1), where an overall decrease in severity of 12-17% at higher densities was observed. P-values approach 0.10 in some cases and it is possible that the differences are not simply due to random variability. There is a suggestion that this effect was weakened or reversed under intercropping with a high proportion of maize.

Nutrient amendment resulted in the most pronounced effect on severity of ALS in the long rains experiments (Tables II.2 and II.3), more than doubling AUDPC for T-1 and T-4 at Kabete. Increases were smaller at Thika, and only here was any interaction with intercropping suggested, and then only for T-1 (P=0.054).

Microclimate

Total rainfall recorded at Kabete during the short rain months (Nov-Jan) was 373 mm, substantially less than for the long rains months (Apr-Jun) of 519 mm. The Thika station recorded only 386 mm during the long rains (Kenya Meteorological Dept., personal communication).

During the long rains at Kabete, relative humidity was measured in eight sets of plots each containing the three maize proportions. Because of variation in ambient conditions over the sampling cycle, absolute values for temperature and relative humidity are not so meaningful as the ranking of values among treatments. Relative humidity was lowest in the monocrop more often than would be expected due to random variation during the sampling cycles in all eight sets ($P<0.005$). Leaf and air temperature were highest in the monocrop in seven of eight sets ($P<0.05$). The changes were small, however. Relative humidity averaged only 1.69 (SD=2.78) and 1.98 (SD=3.03) percentage points lower than the 4:1 and 2:1 bean:maize mixtures, respectively. Leaf temperatures were 0.51 C (SD=1.60) and 0.64 C (SD=1.96) higher in the monocrop than in the 4:1 and 2:1 bean:maize proportions, with air temperatures increased by 0.14 C (SD=0.66) and 0.27 C (SD=0.82), respectively. The mean relative humidity over all monocrop plots during all the sampling cycles was 74.17%, with a range of 54.2-94.6% observed. Mean leaf temperature in monocrop plots

was 25.70 C (Range 18.9-34.2 C), and mean air temperature was 20.69 C (Range 16.6-25.9 C).

Intercropping reduced wind velocity as compared with the monocrops. Averaged over all sample dates for the unfertilized treatment, wind velocity in the 4:1 and 2:1 bean:maize mixtures were 70.0% (SD=13.6) and 55.8% (SD=13.4) of the monocrop velocity at bean canopy height, respectively, and 62.6% (SD=10.2) and 46.8% (SD=18.7) above the canopy (Fig. II.4). Wind speed observations were similar in the fertilized treatment: Mean wind velocities throughout the season for 4:1 and 2:1 bean:maize proportions, respectively, were 49.2% (SD=12.3) and 62.6% (SD=18.6) of the monocrop velocity at bean canopy height, and 56.5% (SD=10.6) and 52.6% (SD=8.3) above the bean canopy. Only one value, the negligible wind velocity reduction seen 55 days after planting in the unfertilized 2:1 mixture at canopy height (Fig. II.4), differs substantially from values seen at other fertility level/height combinations. Absolute wind speeds cannot be compared since they were measured at different times for these four sets of observations.

Discussion

The finding that intercropping beans with maize significantly lowered ALS severity in two out of three season-site combinations corroborates the work of Rheenen et al. (22) and Lanter (17), both of whom observed ALS severity reductions in bean-maize associations which were quite variable depending on location and/or season. Lanter (17), in particular, reported a decrease in AUDPC due to intercropping during two rainy seasons for one bean line, but an increase in AUDPC during the dry season. ALS was not affected by intercropping in a more susceptible line. My data also suggests that rainfall interacts with intercropping effects, since the drier conditions at Thika during the long rains were similar to those at Kabete during the short rains, and these are the two experiments in which significant disease reductions occurred due to intercropping. However, whereas I observed reductions in ALS severity owing to intercrops under the driest conditions which I experienced in Kenya, Lanter (17) reported her reductions in the rainy season, with significant disease increases in the dry season. This discrepancy is probably not due to the imprecision of the terms "rainy season" and "dry season." Though Lanter provides no rainfall data, average monthly precipitation values at her experimental site are approximately 150-175

mm/month during the rainy season and 130-135 mm/month during the dry season (30). These are similar to the levels we observed, 173 mm/month during the long rains at Kabete and 124-129 mm/month during the short rains and at Thika.

Despite this variability in the influence of rainfall on intercropping effects, it is not surprising that some interaction might occur between the two factors regarding ALS severity. Cardona-Alvarez and Walker (6) found that P. griseola required long periods of leaf wetness for infection and sporulation, and in Lanter's (17) work longer periods of leaf wetness corresponded to increased ALS severity. The increased relative humidity and decreased leaf temperatures in intercrops reported here for the long rains at Kabete would tend to favor dew formation and so ALS. One might expect intercropping to increase disease in this situation, or at least not reduce it, which is indeed what we observed for this particular experiment. Accordingly, microclimatological parameters in the short rains, and at Thika in the long rains, may have responded differently to intercropping and in turn might have caused the reduction in severity observed in these cases. It is perhaps more likely that the small changes in temperature and moisture recorded here and elsewhere for bean-maize intercrops (27) have a negligible effect on infection overall. A reduction of leaf temperature by 1-2 C from 26

C, due to any factor, may have little impact on P. griseola, which will infect successfully over a range of 16-28 C (6).

Reduction in wind velocity under intercrops, though similar to other work describing decreased velocity in response to reducing bean canopy density (7), is more difficult to relate to potential disease alteration. Reduced wind velocity may remove fewer spores from a lesion and lower their impaction efficiency, leading to lower severity; but also result in decreased air circulation and prolonged leaf wetness, favoring disease. The more important finding here is that even a low proportion of maize in an intercrop will reduce wind velocities substantially, so that whatever effects wind speed reduction has on disease may be realized with only a few maize plants added to a bean plot, a common occurrence in Kenya (personal observation). Instantaneous wind speeds and turbulence have not been considered here and may be of great importance, as well as weather conditions at night. Microclimatological profiles of intercrops and basic temperature-moisture relations of P. griseola have only begun to be understood, and much more data of this sort is needed.

Use of a random planting pattern or particularly low bean densities, commonly practiced by Kenyan farmers (25, personal observation), had little effect on disease. The

suggestion that higher density lowers disease (Table II.1) does not concur with other work for fungal pathogens (5), though the effect is small relative to those of intercropping and nutrient amendment. The marked increase in ALS severity due to soil nutrient amendment in the long rains may be a result of an increase in the canopy density, in turn providing more targets for spore interception and a more favorable microclimate for disease development (1,3,5). An informal assessment of leaf area taken 40 days after planting during the long rains at Kabete, based on leaf length and width in one block, indicated that beans in plots without nutrient amendments had approximately 33% of the leaf area of those in fertilized plots. An increased susceptibility of the fertilized beans might also have occurred (5).

Boudreau & Mundt (Ch. III), working with bean rust, found that plant competition in a bean-maize intercrop, with a concomitant reduction in bean leaf area index, was responsible for a steepened dispersal gradient but did not reduce the infection efficiency of the pathogen. Rust severity reduction due to intercropping was observed in a simultaneous experiment (Ch. IV), and it was suggested that spore escape created the steeper gradient and led to lower severity. This hypothesis might explain the similar disease reductions for ALS due to low fertility in the experiments reported here. The leaf area estimate at

Kabete also indicated that the high-proportion-maize intercrop reduced bean leaf area to approximately 75% of the monocrop value. Therefore, decreased ALS severity due to intercropping itself may result, at least in part, from a competition-induced reduction in bean leaf area.

TABLE II.1. Area under the disease progress curve for angular leaf spot of beans as influenced by bean density and proportion of maize intercropped with beans during the short rains at Kabete, Kenya

	T-1[a]	T-2	T-3	T-4
Treatment[b]				
All bean				
Low[c]	34.06	81.94	115.52	40.60
High	29.06	72.95	109.32	38.06
4:1 Bean:Maize				
Low	36.73	73.77	110.00	43.87
High	25.78	59.19	72.01	24.83
2:1 Bean:Maize				
Low	25.57	59.22	74.73	23.39
High	27.18	59.01	76.77	32.12
All proportions				
Low	32.12	71.64	100.09	35.95
High	27.34	63.72	86.04	31.67
Significance of factor[d]				
Proportion	0.264	0.045	0.097	0.284
Density	0.103	0.178	0.266	0.472
Proportion x density	0.214	0.594	0.390	0.166

Table II.1 Footnotes

[a]T-1 to T-4 = First (oldest) through fourth trifoliate leaf on main stem.

[b]Mean of eight plots corresponding to four blocks x two planting patterns, each plot calculated from percent leaf area diseased of 10 randomly-selected plants sampled 3-4 times at 6-8 day intervals.

[c]Low = 5.6 bean plants/m^2, High = 9.2 bean plants/m^2.

[d]Probability of falsely rejecting H_0: No difference among factor levels.

TABLE II.2. Area under the disease progress curve for angular leaf spot of beans as influenced by fertility and proportion of maize intercropped with beans during the long rains at Kabete, Kenya

	T-1[a]	T-2	T-3	T-4
Treatment[b]				
All Bean				
No amendment	88.10	130.35	130.94	45.71
Amendment[c]	165.49	187.47	135.03	84.01
4:1 Bean:Maize				
No Amendment	96.09	181.12	133.52	46.15
Amendment	175.61	202.36	148.06	97.49
2:1 Bean:Maize				
No amendment	80.17	124.22	108.47	38.20
Amendment	199.55	197.46	140.94	78.42
All proportions				
No amendment	88.12	145.23	124.31	43.35
Amendment	180.22	195.77	141.34	86.64
Significance of factor[d]				
Proportion	0.821	0.189	0.517	0.141
Fertility	<0.001	0.006	0.149	<0.001
Proportion x fertility	0.546	0.400	0.588	0.556

Table II.2 Footnotes

[a]T-1 to T-4 = First (oldest) through fourth trifoliate leaf on main stem.

[b]Mean of four replicate plots, each plot calculated from percent leaf area diseased of 10 randomly-selected plants sampled 4-5 times at 5-day intervals.

[c]Amendment = 50 g diammonium phosphate/5 m bean row at planting.

[d]Probability of falsely rejecting H_0: No difference among factor levels.

TABLE II.3. Area under the disease progress curve for angular leaf spot of beans as influenced by fertility and proportion of maize intercropped with beans during the long rains at Thika, Kenya

	T-1[a]	T-2	T-3	T-4
Treatment[b]				
All Bean				
No amendment[c]	57.81	110.54	141.18	36.38
Amendment	79.02	123.11	151.22	58.07
2:1 Bean:Maize				
No amendment	52.13	106.58	90.76	26.36
Amendment	50.01	103.43	102.66	41.42
All proportions				
No amendment	54.97	108.56	115.97	31.37
Amendment	64.51	113.27	126.94	49.75
Significance of factor[d]				
Proportion	0.010	0.378	0.053	0.136
Fertility	0.102	0.718	0.631	0.052
Proportion x fertility	0.054	0.555	0.969	0.691

Table II.3 Footnotes

[a]T-1 to T-4 = First (oldest) through fourth trifoliate leaf on main stem.

[b]Mean of four replicate plots (three for 2:1 Bean:Maize, amended treatment), each plot calculated from percent leaf area diseased of 10 randomly-selected plants sampled 4-5 times at 5-day intervals.

[c]Amendment = 50 g diammonium phosphate/5 m bean row at planting.

[d]Probability of falsely rejecting H_0: No difference among factor levels.

FIGURE II.1. Area under the disease progress curve for angular leaf spot of beans as influenced by proportion of maize intercropped with beans during the 1986 short rains at Kabete, Kenya. Values are the mean of 16 plots corresponding to four blocks x two densities x two planting patterns, each plot calculated from percent leaf area diseased of 10 randomly-selected plants sampled 3-4 times at 6-8 day intervals. Bars with different letters indicate significant differences ($P<0.10$) by the Newman-Keuls test; see Table 1 for all treatment probability values. T-1 to T-4 = First (oldest) through fourth trifoliate leaf on main stem; B:M = bean:maize proportion.

FIGURE II.1.

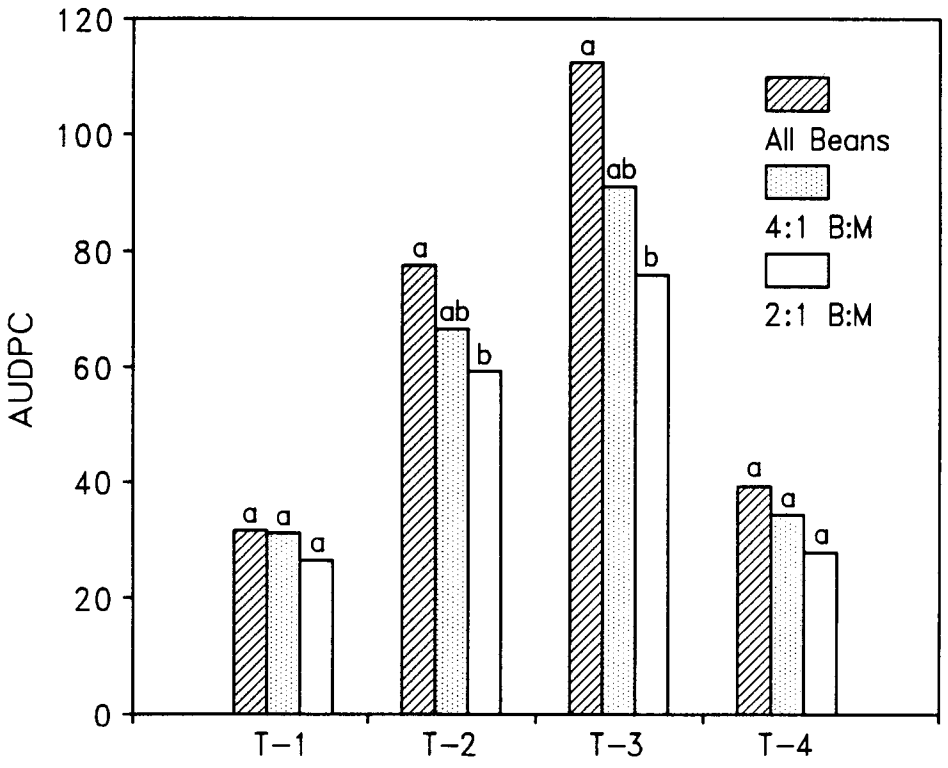

120
100
80
60
40
20
0
AUDPC
a a a
a ab b
a ab b
a a a
T-1
T-2
T-3
T-4
All Beans
4:1 B:M
2:1 B:M

FIGURE II.2. Area under the disease progress curve for angular leaf spot of beans as influenced by proportion of maize intercropped with beans during the 1987 long rains at Kabete, Kenya. Values are the mean of eight plots corresponding to four blocks x two fertility levels, each plot value calculated from percent leaf area diseased of 10 randomly-selected plants sampled 4-5 times at 5-day intervals. No treatment differences were significant by ANOVA ($P<0.10$); see Table 2 for all probability values. T-1 to T-4 = First (oldest) through fourth trifoliate leaf on main stem; B:M = bean:maize proportion.

FIGURE 11.2.

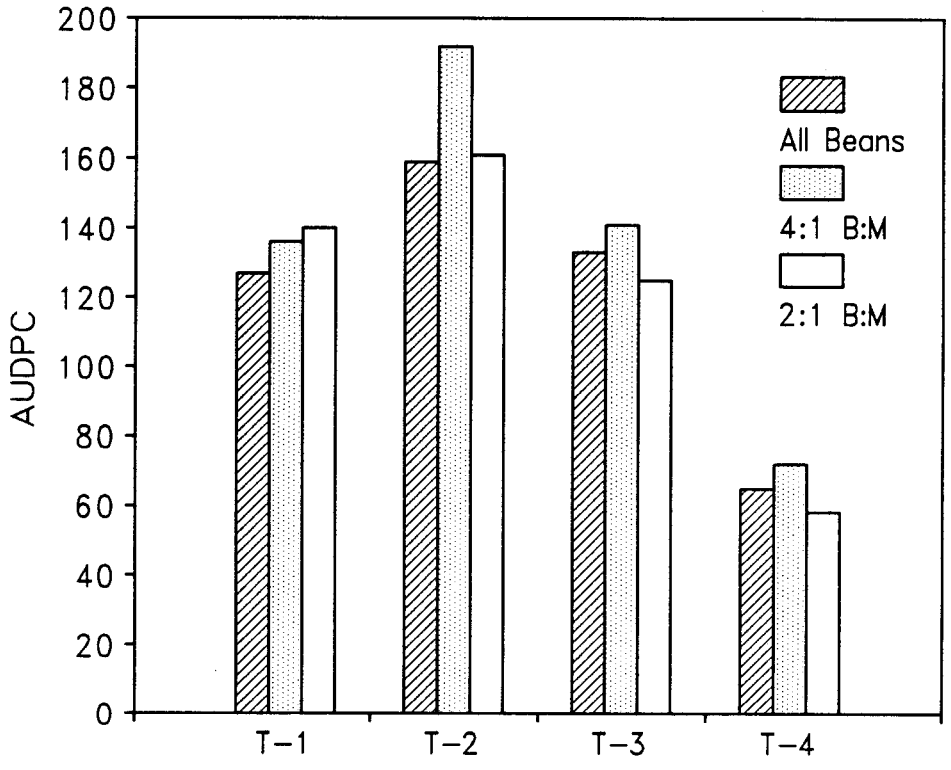

AUDPC
200
180
160
140
120
100
80
60
40
20
0
T-1
T-2
T-3
T-4
All Beans
4:1 B:M
2:1 B:M

FIGURE II.3. Area under the disease progress curve for angular leaf spot of beans as influenced by proportion of maize intercropped with beans during the 1987 long rains at Thika, Kenya. Values are the mean of eight plots corresponding to four blocks x two fertility levels (seven plots for 2:1 Bean:Maize proportion), each plot value calculated from percent leaf area diseased of 10 randomly-selected plants sampled 4-5 times at 5-day intervals. Bars with different letters indicate significant differences ($P<0.10$) by ANOVA; see Table 3 for all probability values. T-1 to T-4 = First (oldest) through fourth trifoliate leaf on main stem; B:M = bean:maize proportion.

FIGURE II.3.

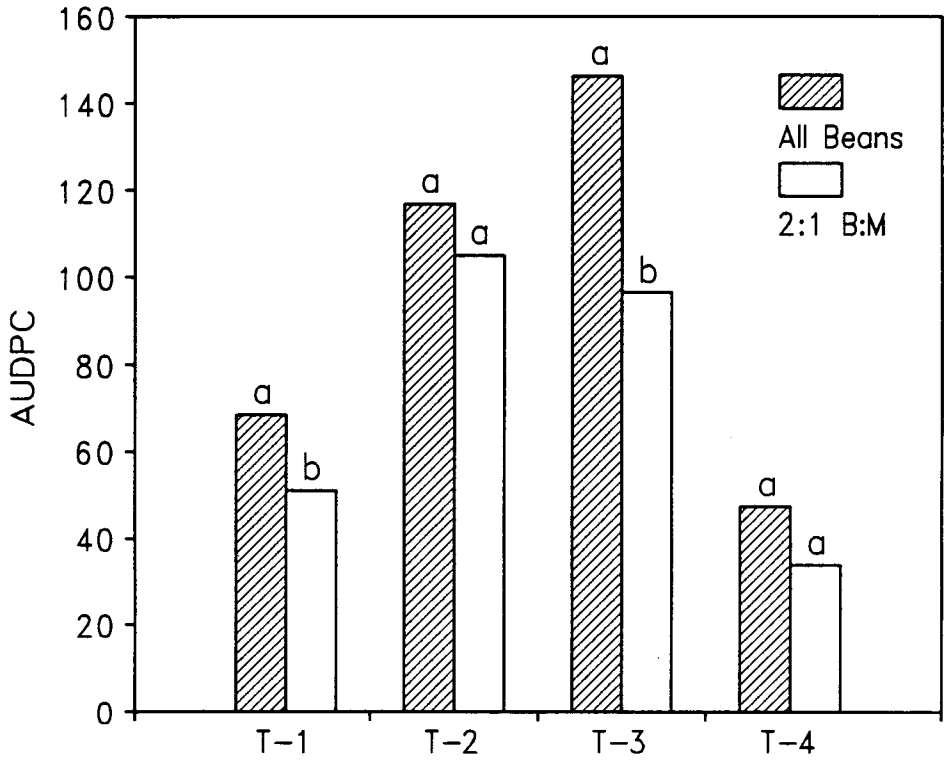
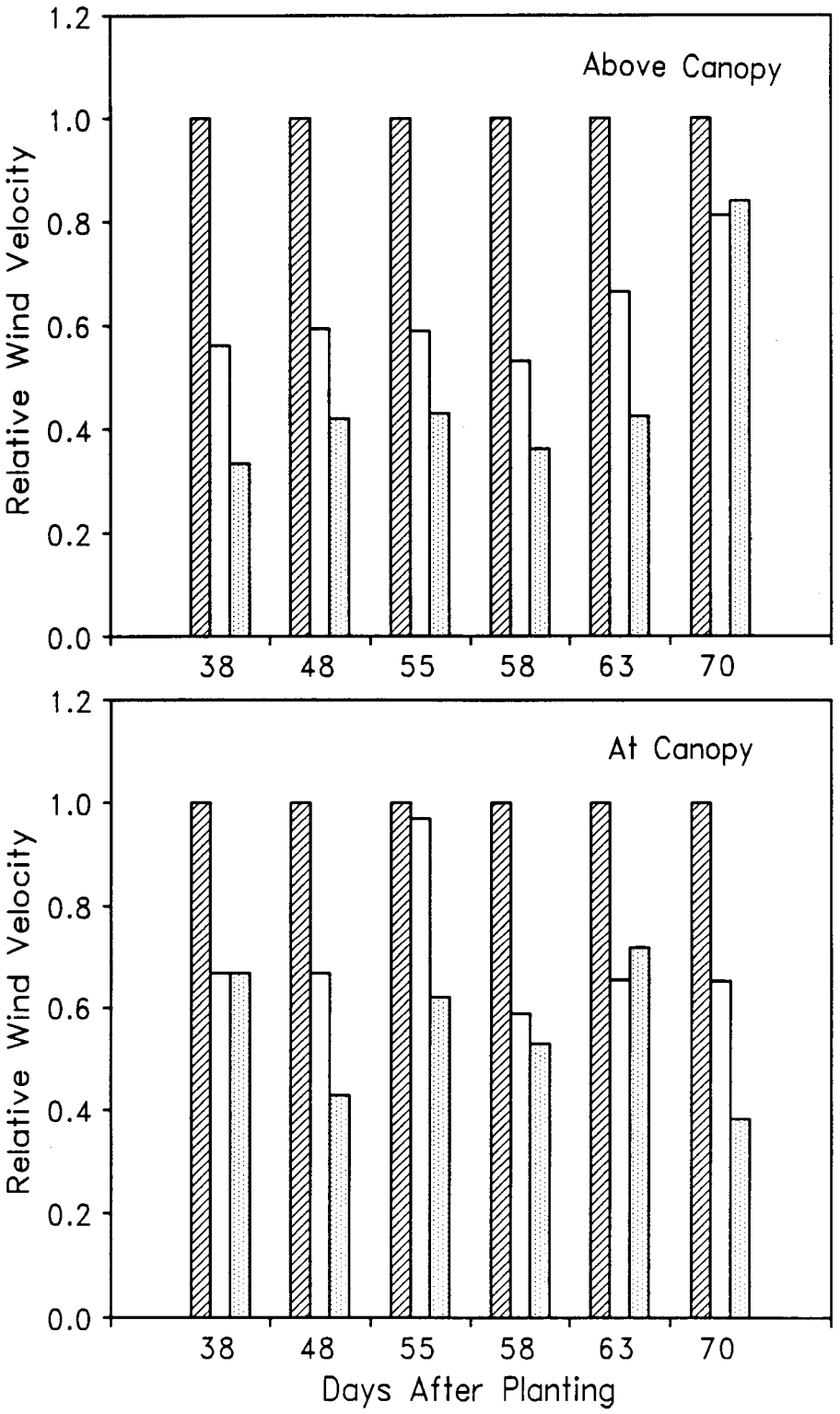

160
140
120
100
80
60
40
20
0
AUDPC
a
b
a
a
a
b
a
a
All Beans
2:1 B:M
T-1
T-2
T-3
T-4

FIGURE II.4. Relative wind velocity at and approximately 25 cm above bean canopies as influenced by proportion of maize intercropped with beans during the 1987 long rains at Kabete, Kenya. Values for each date and height represent the mean velocity over a 1.5-hr period in a single plot relative to the equivalent monocrop value. Hatched bar = bean monocrop; open bar = 4:1 bean:maize mixture; dotted bar = 2:1 bean:maize mixture.

FIGURE II.4.


Above Canopy
At Canopy
Relative Wind Velocity
1.2
1.0
0.8
0.6
0.4
0.2
0.0
38
48
55
58
63
70
Days After Planting

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Chapter III

CHANGES IN BEAN RUST DUE TO INTERCROPPING BEANS WITH MAIZE

Mark A. Boudreau and Christopher C. Mundt

Abstract

Focal inoculations and five subsequent weekly severity assessments were used to evaluate the influence of intercropping beans with maize on bean rust severity during 1989 and 1990 in three sets of paired monocrop and intercrop plots. In two of the plot pairs, intercropping reduced disease in both years (average 27% reduction, P=0.07). In the third pair, which was located in an area of the farm with a different pattern of surrounding vegetation, disease was greater in the intercrop than in the monocrop in both years (average 29% increase, P=0.32). Leaf wetness, measured in one pair of plots, may be important in accounting for the effects of intercropping on rust severity.

Introduction

Intercropping, the simultaneous cultivation of more than one crop species in close association, dominates non-mechanized production of food and fiber throughout the world (10,24). Publications on intercropped systems to date have been focussed on overall yield effects under various species mixes (e.g. 5, 10), though some limited work on insect pests has been reported (15,17). Reduction in plant disease due to intercropping is suggested theoretically (1,9,22), but rarely has it been evaluated. The existing reports indicate not only the expected decrease in disease (2,6,16), but also disease increases under mixed cropping (7,13). The mechanisms of disease alteration, important for an understanding of these variable results, have been studied even less. Mechanisms potentially important include microclimatic changes, spore trapping by the non-host, and induced resistance (1,9,22).

One of the most common intercrop associations combines common bean (Phaseolus vulgaris L.) with maize (Zea mays L.) (3). Bean rust, caused by Uromyces appendiculatus (Pers. ex Pers.) Unger var. appendiculatus, is a limiting factor in bean production throughout the world (25). Rust severity was reduced by intercropping with maize in studies conducted in Costa Rica (14,19) and in the wet season in Brazil (12), though no clear effect was observed in Brazil

during the dry season (12) nor in experiments done in Kenya (16).

No direct study of the mechanisms of these interactions has been undertaken previously (but see Ch. IV). Though small reductions in temperature and increases in relative humidity in bean canopies grown under maize have been measured (21; Ch. II), their effects on rust severity are not easily estimated. Leaf wetness is necessary for rust infection and may be a clearer predictor of disease severity (8). However, it has only been assessed under intercrop conditions in one study, and then with variable results (11).

This paper describes experiments conducted over two growing seasons to determine the effects of interplanting maize with beans on bean rust.

Materials and Methods

Cultivation

Three pairs of plots, each consisting of one bean monocrop and one bean-maize intercrop, were hand-planted on 24-25 June 1989 and 22 June 1990 at the Oregon State University Botany and Plant Pathology Experimental Farm, located east of Corvallis, OR. Plots were 18.3 x 18.3 m in 1989 and separated by 16.5, 7.5, and 23.0 m within the three plot pairs (hereafter known as pairs A, B, and C, respectively). In 1990, 20.0 x 20.0 m plots were used, with pairs A and C located in the same sites as those employed previously, but pair B moved to a nearby position to allow a greater distance between plots (19.8 m). All pairs but C were located within a mosaic of grapes, roses, and dwarf fruit trees 3.2-3.8 m in height. Pair C, however, was positioned at the corner of the farm with standard cherry trees 7.1-m-tall to the north, a golf course to the west, and dwarf trees 3.2-3.8 m in height to the east and south. Data on wind direction was not available at the research farm, but a daily resultant wind vector was recorded 57 km to the north at Salem, OR, also situated in the mid-Willamette Valley (23). 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).

For the intercrop, an alternating pattern of one maize row-two bean rows was employed, oriented north-south, with 40 cm between rows and 45 and 15 cm within rows (maize and beans, respectively). In bean monocultures, the maize rows were left unplanted but the arrangement of beans was identical to that in the intercrop. 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. Snap bean cultivar Pinto 111 (Independent Seed and Bean Co., Twin Falls, ID) and hybrid sweet corn 'Jubilee' (supplied by H.J. Mack, Horticulture Dept., Oregon State Univ., Corvallis, OR) were used, and all management practices were as described in Chapter IV.

Disease comparisons

An epidemic was initiated by placing potted source bean plants with sporulating lesions of U. appendiculatus race 40, grown and inoculated as described in Chapter IV, in the center of each plot on 31 July 1989 and 27 July 1990 (37 and 36 days after planting [DAP], respectively). The

beans growing in the central 1.2 m of these plots were removed from the two center bean rows, and 10 source plants were then placed in the resulting gap in each row. Four additional plants were placed between these rows, approximately 10 and 25 cm from each end, for a total of 24 source plants/plot. In 1989 the source plants were removed after 7 days. Because rain occurred frequently during the first 3 days in 1990, the plants were removed after only 5 days to 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). Two individuals each visually estimated percent leaf area infected for the north, center, and south one-third of five pairs of bean rows in each plot. This estimate integrated all leaves in the row pair. Row pairs sampled in 1989, numbering from one edge of 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 in 1990. The mean of these values was divided by 0.33 to correct for a maximum possible bean rust severity of 33% (20), then area under the disease progress curve (AUDPC) was determined for each plot after the mid-point mean method of Shaner and Finney (18).

Results

A strong interaction between intercropping effect and location is evident in severity values for 1989 and 1990. (Table III.1). AUDPC-values for individual plots indicate a consistent disease reduction due to intercropping in plot pairs A and B, located in the same section of the research farm; whereas pair C, in a different area, showed a severity increase in both seasons. This interaction requires a separate consideration and statistical analysis of the experiment at the two locations. When analysis of variance is performed on pairs A and B, with cropping system (monocrop/intercrop) as main effect and year and plot pair as independent blocks, the mean 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 due to intercropping was significant only at P=0.32. The power of this test, however, was lower than that for pairs A and B due to less replication.

Discussion

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 (14) and Soria et al (19). These studies, like our own, were of an additive design (bean density equal in monocrop and intercrop), thereby insuring that intercropping effects on disease are not due to changing host density, as may be the case in replacement-series experiments (1). However, Soria et al (19) presented their disease results qualitatively, and both they and Moreno and Mora (14) conducted their experiments during only one season at a single site. More representative may be the work of Monteiro et al (12) in Brazil, who observed that decreases in rust severity due to intercropping diminished depending on season or bean density (the two factors could not be separated). Rheenen et al (16), making a total of 1671 comparisons on intercrop/monocrop plot pairs at seven diverse sites in Kenya over several years, recorded that 459 pairs had more rust in the intercrop than the monocrop, 575 pairs had less rust in the intercrop, and 637 had equal levels of severity. Though the effect of intercropping was significant at $P=0.01$ by chi-squared analysis, the variability of the system is quite pronounced in their study.

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 (relative to pairs A and B) was not an artifact of some localized plot effect, because randomization reversed 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 >7 m in height to the north, the direction from which winds most often originated (23). This suggests that variable intercropping effects may be related to wind patterns. Alteration in wind velocity and turbulence could affect disease directly through changes in dispersal and indirectly through changes in microclimate, but the nature and magnitude of these changes, and the way in which they might interact with the presence of maize, is difficult to predict.

In addition to changes in air movement created by intercrops, microclimatic alteration due to shading and

transpiration by maize plants are likely to affect disease in a complex way. Leaf wetness sensors, placed in the C pair of plots in September-October of 1989, indicated that leaf wetness duration was generally less in the monocrop plot than in the intercrop plot. This relationship was reversed during two periods of long leaf wetness duration. 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.

Though intercrops have been found to increase humidity and lower wind speeds (Ch. II), both of which would tend to favor dew formation, monocrops may also encourage dew by allowing substantial radiative heat loss from bean leaves in the absence of a maize canopy. Increased periods of leaf wetness would be expected to favor disease (4,8), and indeed AUDPC was higher due to intercropping in the plots in which leaf wetness was measured. Leaf wetness patterns may have been different in the plot pairs showing the more typical reduction in rust severity under intercropping (pairs A and B), but other mechanisms, such as those affecting dispersal, may be responsible for disease alterations as well (Ch. IV). In addition, our leaf wetness data were from unreplicated plots, so it is unclear

if the measured differences were truly due to intercropping effects.

The variability in intercropping effects which we observed over a small spatial scale may be the result of complex interactions between these and other mechanisms. They should not be ignored, however, because these localized effects may influence disease a great deal in agriculture in developing countries. For example, subsistence farming in East Africa, where bean-maize intercrop fields <0.5 ha are surrounded by numerous obstructions of various heights (e.g. banana plants, sugar cane, low annual crops, huts) (personal observation), is well represented by the size and heterogeneous environment of the plots used in this study. Further consideration of the many parameters implicated in disease interactions with non-host species is essential to an understanding of the current and future value of intercropping.

TABLE III.1. Area under the disease progress curve for bean rust epidemics in bean monocrops and bean-maize intercrops

	Pair A[a]	Pair B	Pair C[c]	Mean Pairs A & B[d]
Treatment[b]				
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 & 1990				
Monocrop	367.79	330.60	205.68	349.20
Intercrop	239.73	267.28	266.24	253.51

Table III.1. Footnotes

[a]Pair = one bean monocrop plot and one bean-maize intercrop plot. Each plot was 18.3 x 18.3 m in 1989 and 20.0 x 20.0 m in 1990. All pairs located among mixtures of dwarf fruit trees (3.2-3.8 m in height), grapes, and roses; in addition, pair C had standard trees (7.1 m in height) to north and a golf course to the west.

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

[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.

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Chapter IV

MECHANISMS OF ALTERATION IN BEAN RUST EPIDEMIOLOGY DUE TO INTERCROPPING WITH MAIZE

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Abstract

Experiments were performed to identify components of maize influence on bean rust (caused by Uromyces appendiculatus) in maize-bean intercrops. Effects of maize on dispersal of rust urediniospores, due to both competition with maize for nutrients, light, etc., and the physical interference by maize with spore movement, were evaluated in trials conducted at three times after planting in 1989, and again in 1990. Alterations in the non-dispersal (infection) phase of the pathogen life cycle due to intercropping and competition with maize were assessed following each experiment. Competition consistently steepened the dispersal gradients ($P<0.10$), described well by the modified Gregory model, by 50 days after planting. Interference tended to flatten gradients in the absence of competition, but competition and interference in combination (intercrop) produced steeper gradients. Estimated total spore deposition per plot was both increased (second dispersal trial) and decreased (third trial) by competition in both years ($P<0.05$).

Intercropping had no effect on infection, except at one sampling point late in 1989 when rust severity was reduced by 96% ($P<0.05$). Growth measurements taken throughout both seasons illustrate a decline in bean leaf area due to competition. Steeper gradients may be due to increased spore escape associated with the reduced leaf area, and microclimatic changes created by maize are probably responsible for the non-dispersal effect. The interaction of these factors is related to reports of decreased rust severity in bean-maize associations and to management recommendations for intercrops.

Introduction

Several studies have related intercropping to changes in yield (24,35) and insect incidence (37,39), but few have evaluated the effects of intercropping on plant pathogens. Despite speculations that intercropping will generally reduce disease severity compared to levels in a monocrop (4,9), the limited data indicate a wide range of results, even for a single combination of crops. 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 (28; Ch. II), equal to (28,38; Ch. II) or more than (28,33) the severity in bean monocrops. Bean rust (caused by Uromyces appendiculatus (Pers. ex Pers.) Unger) has generally been reduced by intercropping with maize (32,34,40), but in some cases has not been affected (38). We report in Chapter III on bean rust severity in bean-maize intercrop experiments conducted at the same time and location as those described here. Intercropping reduced disease in two blocks, but did not affect or perhaps increased disease in a third block at another location, indicating an alteration of intercropping effects over even a small spatial scale.

The mechanisms of interaction among pathogen, host, and non-host which determine disease levels in intercrops have received still less attention, and much of this has

been speculative as well. Proposed mechanisms include amelioration of dispersal factors (e.g. wind, rain, or insect vectors) by the non-host, trapping of propagules by the non-host, microclimate alteration of the pathogen environment, reduced density of host, and changes in infection elicited by microorganisms (induced resistance) or pollen associated with the non-host (9,23,43). The empirical support for these suggestions is almost entirely indirect. For example, temperature reductions and increases in relative humidity have been measured for common beans grown with maize when compared to bean monocultures (42; Ch. II). High humidity and leaf wetness favor diseases such as bean rust and white mold (1,21,22), and might be expected to have similar effects if these conditions were due to intercropping. Induced resistance to bean rust elicited by inoculation with sunflower rust spores (Puccinia helianthi) or maize rust (a P. sorghi/P. polysora mix) has been demonstrated in the laboratory (2,44). On the other hand, pollen will enhance fungal growth and infection by Botrytis cinerea on faba beans and Colletotrichum lindemuthianum on cowpea, but reduce cowpea yellow mosaic infection (3,15).

A few studies have provided more direct evidence of mechanisms contributing to disease severity in intercrops. Burdon and Chilvers (10,11,12) experimentally determined that reductions in rates of damping off of cress when mixed

with ryegrass, and of powdery mildew on barley when intercropped with wheat, were mainly due to the reduced density of the host in the mixtures. In all of these experiments, morphologically similar species were mixed in replacement-type combinations, i.e., total plant density remained constant but host density decreased as non-hosts were added to the mixture. Thus, Burdon justifiably dismissed microclimatic influences as important in altering the epidemics (9). However, in a black walnut-autumn olive intercrop, both 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 (25). Chin and Wolfe (14) have attempted to isolate density, induced resistance, and interference components of mildew reductions in barley cultivar mixtures, and found density effects important early in the season with induced resistance significant only later. Late-season density effects could not be evaluated due to 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 spores;

dispersal effects due solely to competition with maize; and maize effects on non-dispersal components of the disease cycle.

Materials and Methods

Our general approach, described in detail below, was to evaluate 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, at three different stages of crop growth (i.e., at three times after planting). 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, excepting one destructive treatment (see below). Effects of maize on elements of the rust life cycle other than dispersal were evaluated by spray-inoculating plants and assessing severity in separate experiments lasting approximately 14 days, also conducted at three times during the season. The plots used for the dispersal trials also were employed in these experiments, by utilizing individuals of a susceptible bean cultivar randomly planted among the resistant beans, and covering them during the dispersal event to avoid premature inoculation.

Other mechanisms of intercropping-disease interaction were not evaluated. Induced resistance was eliminated by ensuring disease-free maize, and host density effects were

eliminated by employing an additive rather than replacement-series design.

Plant culture and inoculation

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 22-24 June 1989 and 19-22 June 1990, in 20 x 20 m plots at the Oregon State University Botany and Plant Pathology Experimental Farm immediately east of Corvallis. The plots were arranged in three contiguous blocks, with adjacent plots separated by 20 m, 14.5 m, and 10-12 m within their respective blocks. The plot site was bounded by dwarf trees (3 m tall) to the north and west at a distance of 15.5 m; grass pasture to the east; and a mosaic of grass, potatoes, dwarf trees, and bare soil to the south. Standard cherry trees (6 m tall) were located 30 m to the south of one of the blocks. 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-two bean rows with 40 cm between rows and 45 and 15 cm within rows (maize and beans, respectively) was employed for intercrops; in bean monocultures, the maize rows were left unplanted but the arrangement of beans was identical to that in the

intercrops. In some plots, snap bean cultivar Pinto 111 (Independent Seed and Bean Co., Twin Falls, ID) was randomly planted in the bean rows (see Non-dispersal effects section below).

Overhead sprinkler irrigation was applied as needed. 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 kg/row) sidedress approximately 3 wks after emergence. Alachlor at 3.4 kg/ha a.i. was applied before planting for weed control, and insect protection was limited to three weekly carbaryl applications (1.87 gm/l a.i., sprayed to runoff) to Pinto 111 beans beginning on 13 July 1990, to control Mexican bean beetles.

Pinto 111 beans were grown in square 10-cm wide plastic pots (one plant/pot) in the greenhouse for use as both trap plants and inoculum source plants in dispersal experiments. Approximately 22 days after planting (DAP), when primary and 2-3 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. Source plants were inoculated with U. a. appendiculatus race 40 (provided by J. R. Stavely, USDA-ARS, Beltsville, MD) by spraying a urediniospore suspension (1×10^4 spores/ml in distilled water with 0.01% Tween 80) to runoff with a bulb atomizer (DeVilbiss Co., Somerset,

PA). After the 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 DAP, respectively). Source plants were maintained in a greenhouse separate from that containing trap plants, and were covered with polyethylene bags and transported to their final destination in the experimental plots immediately before use, approximately 33 DAP. A source plant occasionally had relatively low numbers of sporulating lesions, in which case a supplemental pot containing four 24-day-old beans with infected primary leaves was substituted, prior to transport to the field, to ensure homogeneity of inoculum supply. Unlike Pinto 111, the OR91G cultivar used for field plantings is highly resistant (no sporulating pustules produced) to race 40 (personal observation).

Dispersal effects

Effects of maize on dispersal of U. appendiculatus were evaluated by comparing primary dispersal gradients away from a focal inoculum source in individual plots (experimental units). Treatments were in a 2 x 2 factorial, randomized complete block design using the three physical blocks mentioned. 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/absence, giving a total of four possible treatment combinations. These are illustrated in Fig. IV.1. The no competition/no interference and competition with interference treatment combinations were represented by a bean monocrop and a bean-maize intercrop, respectively. The competition without interference ("removal") treatment was accomplished by removing the maize from an additional bean-maize intercrop plot the day before the dispersal event began. The maize plants which were removed were then artificially supported at corresponding locations in a bean monocrop for the duration of a dispersal event. Thus, the interference without competition ("addition") treatment was realized. Supports consisted of 10 cm nails projecting from boards buried in the plots, onto which maize stalks were impaled.

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, with four additional plants 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. Holes were dug as needed to prevent the trap-plant leaves from extending over the existing bean canopy. When trap plants for all plots were in place, source plants were uncovered to initiate the dispersal event. Approximately 10 h later, just before sunset, all of the 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, giving a total exposure to U. appendiculatus spores of approximately 30 h. After day 3 the beans remained in the humidity tent for 16 h to maximize infection, then were returned to the greenhouse. Twelve days later we began to count 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 DAP, respectively), hereafter referred to as Release 1, 2, and 3 for both years. Experiments were repeated in the same plots in a

given year, except the removal treatment, which required a new plot for every release. Background contamination and non-primary gradients were minimized because U. appendiculatus is currently rare in western Oregon (27), 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 mentioned above), 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, the field next to the humidity tent (35 m from the nearest plot), and the greenhouse where the trap plants were grown.

Lesion count data were averaged over all directions in each plot and fit to both a negative exponential (26) and the modified Gregory inverse power function (36), the latter employing a truncation factor of 0.6 m, equal to the source radius. The gradient slopes were then taken as the experimental response variable and subjected to 2 x 2 multifactorial analysis of variance with blocks, using maize interference and competition as main effects. In order to compare number of spores retained in entire plots, estimates of total lesion number in an equivalent plot of

susceptible plants also were subjected to ANOVA. These estimates were obtained by multiplying the average absolute 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. Plot corners, which were beyond the outermost annulus containing trap plants, were estimated using values predicted by the modified Gregory model (36) for that distance.

All analyses were done using counts from the first trifoliate leaf only, and from the combined total of all leaves present on all plants for that release. Occasionally a datum would be absent due to leaf death. In this case, the gradient model was fit after eliminating all data at the same distance from the center in that plot in all other directions, 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 of 1990, however, 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. In this case, a gradient slope was calculated for each directional transect in each plot so that all available values could be used, resulting in fewer missing data. Second trifoliate values also were used because more of these survived than did first

trifoliate leaves. The slopes were analyzed by a hierarchical ANOVA with the four directions nested within the usual 2 x 2 treatment combinations with three blocks (48 values total). For all releases, appropriate reductions in the error term of the ANOVA were made where missing data points and poor fits to the regression models resulted in the elimination of values (4 out of 48 values for Rel. 2 1990; 1 out of 12 values for Rel. 3 1989-combined leaf data and Rel. 3 1990-first trifoliate and -combined leaf data).

Non-Dispersal Effects

Effects of maize on components of the U. appendiculatus life cycle other than dispersal were evaluated by uniformly inoculating Pinto 111 plants, which had been randomly planted in the monocrop and intercrop plots described above, then subsequently recording latent period and severity. An indication of the effects of competition were obtained by inoculating susceptible plants in the removal treatment plots as well. It was not logistically possible to evaluate non-dispersal effects due to the physical presence of maize during infection (i.e. microclimatic influences), because this would have entailed inoculating susceptible beans planted in the addition treatment plots and leaving the artificially-supported maize standing throughout the 2-3 wk experiment. The shading thus produced would have violated the requirement

for no competition in this treatment of the dispersal experiment, as well as in the non-dispersal experiment sought.

During dispersal trials, all Pinto 111 plants were covered with inverted greenhouse containers to avoid contamination, then uncovered after source plants had been removed. Twenty-cm diameter plastic containers (McConkey's Co., Sumner, WA) were used during Release 1 of 1989, resulting in a substantial loss of leaves due to contact with the sun-heated plastic. Twenty-five-cm diameter fiber containers (Western Pulp Products, Corvallis, OR) were used thereafter, with no apparent ill effects. After each dispersal experiment had been completed, five randomly selected Pinto 111 plants were sprayed at sunset to runoff with a 1 x 10^4 spores/ml ($7.5x10^3$ spores/ml 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 Inoc 1, 2, and 3 for both years. Plants were then observed daily for the occurrence of 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. Background contamination made an assessment of latent period impossible for Inoc 3 in 1989, however. The modified Cobb scale (41) was employed to estimate the per cent 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 of widely varying leaf demographics among plants in any one experiment, treatments were compared using severity values for 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 Inoc 1, 2, and 3, respectively; and 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 (3 treatments x 3 blocks) was performed on the $\log_{10}(x+1)$-transformed values of mean severity for each plot. The transformation was necessary because variation in severity per plot was proportional to mean severity. It successfully eliminated this heteroskedasticity. Latent period values were also subjected to ANOVA where appropriate. Multiple comparisons were performed using the Newman-Keuls method.

Crop growth

Leaf area and heights were observed after each dispersal experiment to assess the effects of intercropping on crop growth. Both 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 treatment plots on 1 August, 24 August, and 14 September (39, 62, and 83 DAP, 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. Due to 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 x 10 m sections in each plot. Plants for area measurement were taken on 29 July, 15 August, and 1 September (39, 56, and 73 DAP, respectively) and heights evaluated on 29 July, 15 August, and 3 September.

At the ends of both seasons, bean and maize density were estimated for all 18 plots used over the previous summer. Number of stems were counted in 7 m of row measured north from the center of the sixth and twenty-second bean row, and south from the center of the thirteenth and twenty-ninth bean row, numbering rows from the west edge of the plot. Maize density was taken from each adjacent row in the intercrop plots. Combining area/plant and density data allowed an estimate of leaf area index (LAI) for maize and leaf+pod area index (LPAI)

for beans in each plot. These represent the ratios of leaf area per unit area of ground (LAI), and combined leaf and bean pod area per unit area of ground (LPAI), and are therefore simple unitless values.

Mean height/plot and LPAI were compared for beans using two-way ANOVA (4 treatments x 3 blocks) for each of the assessment dates. ANOVAs also were performed after combining data from monocrop and addition plots, and intercrop and removal plots, since these pairs of treatments are almost identical with respect to bean growth, and the power of the test is thereby increased.

Results

Dispersal effects

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

Figure IV.2 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 IV.1. When subjected to ANOVA, the competition factor emerges as consistently (with one exception) steepening the dispersal gradient. This effect is 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 Release 2 and 3 of both years in all but one case (Release 2, 1989, combined leaves). Interference, though flattening the gradient when added to a monocrop (addition treatment), does not appear to have an influence when competition is present also (intercrop treatment). Only for Release 3 1990 single-leaf data does the significance

of the interference factor approach P=0.10. The competition x interference interaction effect that this pattern suggests is significant at P=0.093 in Release 2 of 1989, and P=0.046 in Release 2 of 1990, for single-leaf data. None of the factors had effects significant below P=0.10 during Release 1, when the stand was young. Lesion counts on control plants indicated that contaminating background inoculum, present at levels sometimes equivalent to those at the edge of experimental plots, originated both 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.

Estimates of total number of infections (Table IV.2) show that competition significantly alters 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; P=0.073 in 1990) (all P-values are for single-leaf data). Interference tended to reduce the number of spores deposited in both Release 2 and 3, although this effect is only significant below P=0.10 in the case of Release 2, 1990, single leaf. The interaction between interference and competition is significant below P=0.10 in several instances, but the effect is not consistent, even for

different leaf sets within a single release (see Release 3, 1990).

Non-dispersal effects

Mean severity levels for spray-inoculated leaves are summarized in Fig. IV.3, presented relative to the monocrop treatment. Actual values varied considerably among inoculations, but this may be due to several factors which changed between experiments, e.g., different-aged leaves compared; small change in inoculation rate for Inoc 2, 1989; effect of covering plants during dispersal experiments. Thus, comparisons of absolute severity levels are inappropriate. Although the relative values suggest that intercropping may increase the level of disease early in crop growth (Inoc 1 and 2, 1989, and Inoc 1, 1990), only the pronounced reduction in disease in late 1989 (Inoc 3) was significant ($P=0.023$). The influence of competition alone on severity (removal treatment) did not appear significant in any case at the $P<0.10$ level. The intermediate values for this treatment in 1989 might suggest an additive relationship for competitive and non-competitive (i.e. microclimatic) influences of maize, but the 1990 data indicate a more complex interaction.

All plants began showing sporulating lesions 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 in days, are as follows for monocrop,

removal, and intercrop treatments, respectively: 8.7, 9.1, and 8.3 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.

Crop growth

Beans experiencing competition (intercrop and removal treatments) had consistently lower LPAI than those without competition (monocrop and addition) in 1989 (Fig. IV.4). Although this difference is not significant when analyzed as four treatments, an ANOVA combining pairs of treatments as above increases the power of the test sufficiently to yield differences significant below P=0.05 for the second and third assessments in 1989. No significant differences are 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 are also illustrated in Fig. IV.4. In 1989, the reduction in bean leaf area late in the season quantifies an observed senescence and defoliation by Release 3/Inoc 3. The experiments were conducted at slightly shorter intervals in 1990, partly to avoid this phenomenon, and the

data in Fig. IV.4 bear this out. Overall, LPAI-values were roughly equivalent at approximately 60 DAP for the two years, and though it is likely that growth occurred after this date in 1989, defoliation was observable by the time of Release 3, 77 DAP.

Bean height (Fig. IV.4) was not significantly influenced by treatment in either year below P=0.10. Although the maize quickly exceeded the beans in height, the contribution of maize to total leaf area was relatively small due to the low number of maize plants relative to bean plants.

Discussion

Dispersal effects

The purpose of these experiments was to identify factors which may be important in altering bean rust epidemiology when beans are intercropped with maize. The factor most clearly influential is 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 (10,11,12) in that interference was not a factor in intercrop disease reductions for either a soilborne or airborne foliar pathogen (they did not evaluate dispersal gradients). The most obvious outcome of competition that would affect dispersal would be a reduction in the size of the bean plants in the plot canopy, and such a reduction is evident in the LPAI data of Fig. IV.4. Steeper gradients parallel reductions in LPAI in 1989. In 1990, where intercrop but not removal LPAI-values were high, competition effects were still present, though at lower significance levels.

It is difficult to predict what effect reduced LPAI would have on a dispersal gradient (30). Workers have failed to demonstrate consistent relationships between these factors (19,29), despite assumptions that increased plant 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 (6,16) 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 (6,16,17), as was the case with our data. Our estimates of spore deposition for Release 3 (Table IV.2) support the view that spore escape created the steepened gradients in plots with maize competition, because fewer spores were retained in these plots. The plots showing the same effect of competition on gradient slope in Release 2 appeared however to retain more spores, suggesting that increased deposition rather than turbulent diffusion may have dominated at that point in time. Mechanisms of gradient alteration may themselves have changed during crop growth, though 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, which was not the case. The

mechanisms accounting for spore retention data are therefore unclear.

The evidence for steeper gradients coupled with reduced spore retention and reduced LPAI due to 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 due to greater surface roughness in a low-density bean stand made more sparse through competition, or increased convective turbulence due to more exposed ground, both of which could vary spatially in the plot.

This effect is complicated, however, 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 be diminished by the relatively low density of the maize used in this study and indicated by the maize LAI data; 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 three-day dispersal event. Nevertheless, we cannot completely rule out the possibility that the treatment may not mimic the interference effects of living maize, so that the intercrop treatment is not a true combination of competition + interference treatments. Even so, the importance of competition in these studies is not diminished. Other factors might conceivably affect dispersal via competition, such as changes in bean architecture, though data is not available on these effects as yet.

Non-dispersal effects

Non-dispersal influences of intercropping were less pronounced than dispersal effects in this study, though in late 1989 disease severity was significantly reduced by non-dispersal effects which were, furthermore, not due to competition. Microclimatic changes produced by maize are most likely responsible, since other influences of maize on beans (e.g. nitrogen levels of leaves, water status of soil) would have been apparent in the results from the competition treatment. Our results are not consistent with assumptions that the higher daytime humidity measured in bean canopies grown under maize (42; Ch. II) would favor rust, though Lanter (28) did measure a lower vapor pressure/higher saturation deficit in maize intercrops compared to monocrop beans. Leaf wetness and low light

levels also have been shown to favor infections (6,22). Growing beans under maize has resulted in both prolonged and decreased periods of leaf wetness, depending on season (28; Ch. III), and the duration of direct illumination on upper bean leaves is reduced under intercropping (personal observation).

The mechanism by which the observed disease reduction in the intercrop occurred is therefore unclear. It is important to recognize that this phenomenon was only seen once in these experiments, at a time when significant defoliation had taken place. The unique result may involve an interaction between bean leaf loss, which did not affect disease alone (Removal treatment, Fig. IV.3), and the presence of maize. Under the more typical conditions of the other trials, the comparatively small microclimatic effects of intercropping measured thus far (e.g. 2-3% increase in relative humidity [42; Ch. II]), coupled with the high degree of spatial and temporal variability in the bean canopy of a large field, may in sum encompass the range of conditions under which U. appendiculatus will infect successfully in either the monocrop or intercrop conditions.

Combined effects and disease

The outcome of the intercrop-induced alterations described above on overall disease, alone and in combination, is not easily estimated in detail. A

reduction in infection after inoculation would both reduce disease severity and provide less inoculum for dispersal, though this may only occur late in the season. Dispersal gradients were steeper due to intercropping, which may slow the velocity of disease spread (8,31), 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, described in Chapter V, 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, indicating 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 as host density declines (13).

The qualitative sum of these factors suggests that (a) rust severity may be reduced, as has been observed in most bean-maize intercrops (32,34,40); and (b) there also should be an expectation for more variable results (38). The data from the experiments reported here, when used as inputs for computer-simulated epidemics (Ch. V), led to overall disease reductions due to intercropping in 1989, but no

disease alterations in 1990. Also particularly relevant are data from a simultaneous experiment at the same site, reported in Chapter III, in which rust was reduced by intercropping in two sets of plots but not in a third over both years. This variability may have been created by the unknown interactions of the mechanisms studied here with localized environmental differences (the plots showing disease increase had 6-m-tall trees immediately to the north, the direction of prevailing winds). It should also be noted that it was necessary to plant a different (susceptible) bean genotype in those experiments, and that the beans generally grew more vigorously (personal observation) than in the present study. 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 all contribute variability in other settings.

The main value of the findings reported here is in suggesting which factors are most important in altering bean rust due to intercropping with maize, and 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 a disease reduction in intercrops, conditions creating more shallow gradients such as larger, more abundant foci or strong background inoculum levels (20) 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 (18), might also eliminate any advantages of intercropping for disease suppression. Additional research on the mechanisms of intercrop-disease interactions will provide a framework for methodically evaluating the efficacy of a particular cropping system.

TABLE IV.1. Slopes of dispersal gradients of Uromyces appendiculatus on beans, as influenced by competition with maize present prior to 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	
	Rel 1[c]	Rel 2	Rel 3	Rel 1[d]	Rel 2	Rel 3	Rel 1	Rel 2	Rel 3	Rel 1	Rel 3
Main effects[e]											
Competition											
Absent	-0.939	-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[f]	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[g]											
-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[h]	-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

Table IV.1 Footnotes

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

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

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

[d]Poor plant growth eliminated Addition & 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. Based on modified Gregory model applied to mean lesion count on leaves of four trap plants equidistant from 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 modified Gregory model applied to mean lesion count on leaves of four trap plants equidistant from inoculum source. See text for explanation of treatments.

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

TABLE IV.2. Estimated total number of infections of Uromyces appendiculatus on beans in 20 x 20 m plots, as influenced by competition with maize present prior to 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	
	Rel 1[c]	Rel 2	Rel 3	Rel 1[d]	Rel 2	Rel 3	Rel 1	Rel 2	Rel 3	Rel 1	Rel 3
Main effects[e]											
Competition											
Absent	15892	23875	17480	--	1091	32165	40128	40048	36151	--	57527
Present	12870	28333	10330	--	2147	23040	38350	46158	25315	--	37297
Significance[f]	0.327	0.070	0.001	--	0.014	0.073	0.843	0.095	0.004	--	0.093
Interference											
Absent	14047	26853	14560	--	1827	29755	36286	45022	36676	--	47036
Present	14714	25356	13250	--	1458	25931	42193	41184	26684	--	49887
Significance	0.822	0.488	0.294	--	0.079	0.373	0.518	0.260	0.132	--	0.706
Interaction[g]											
-Cmp/-Int (Monocrop)	14767	25745	20326	50190	669	33583	32974	45271	43484	103957	53814
+Cmp/-Int (Removal)	13327	27961	8793	--	2986	25927	39597	44774	26458	--	40258
-Cmp/+Int (Addition)	17017	22005	14633	--	1556	30746	47281	34825	28815	--	61241
+Cmp/+Int (Intercrop)	12412	28706	11867	43159	1377	18708	37104	47543	24553	90372	32856
+Cmp/-Int + -Cmp/+Int[h]	15172	24983	11713	--	2271	28337	43439	39800	25505	--	50750
Significance	0.596	0.311	0.008	--	0.015	0.834	0.367	0.076	0.018	--	0.655

Table IV.2 Footnotes

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

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

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

[d]Poor plant growth eliminated Addition & 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 lesion 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 lesion 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.

FIGURE IV.1. 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 both absent (Monocrop). **B**, Beans and maize grown together. Interference and competition both present (Intercrop). **C**, Beans and maize grown together, with maize removed from plot immediately prior to spore dispersal event. Interference absent but competition present (Removal). **D**, Beans grown alone, with cut maize from Removal treatment artificially supported on nails, protruding through buried boards, immediately prior to spore dispersal event. Interference present but competition absent (Addition).

FIGURE IV.1.

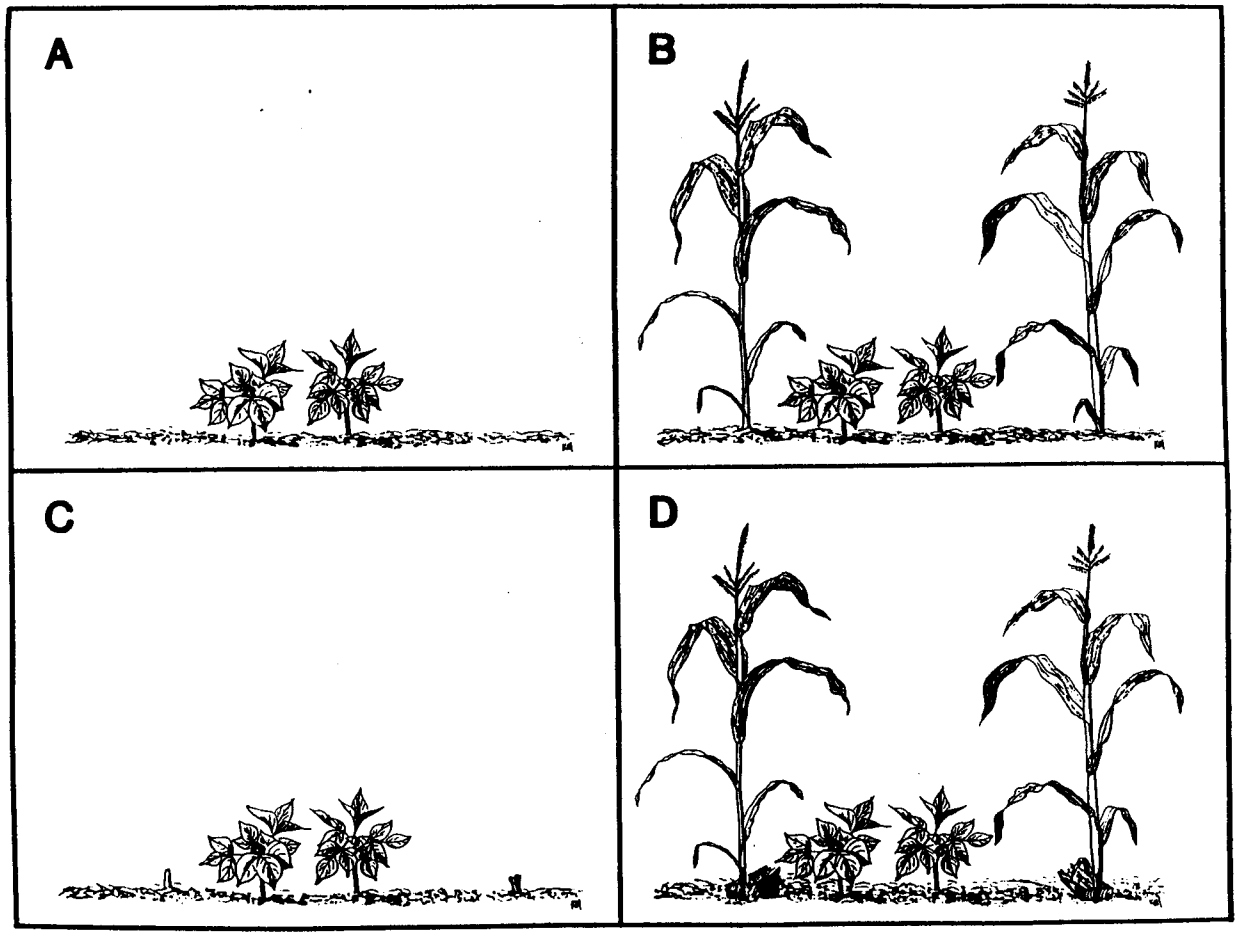


FIGURE IV.2. Dispersal gradients of Uromyces appendiculatus on beans as influenced by competition and interference effects of intercropping with maize, for experiments done 77-79 days after planting in 1989 and 70-73 days after planting in 1990. Points represent mean lesion counts on first (oldest) trifoliate 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 regression for the three plots by the modified Gregory model (truncation factor = 0.6 m), except for Intercrop-1990, in which one replicate (represented by solid rectangles) was not included due to missing data. Monocrop = no competition or interference; Addition = Interference w/o competition; Removal = Competition w/o interference; Intercrop = Competition with interference. See text for explanation of treatments.

FIGURE IV.2.

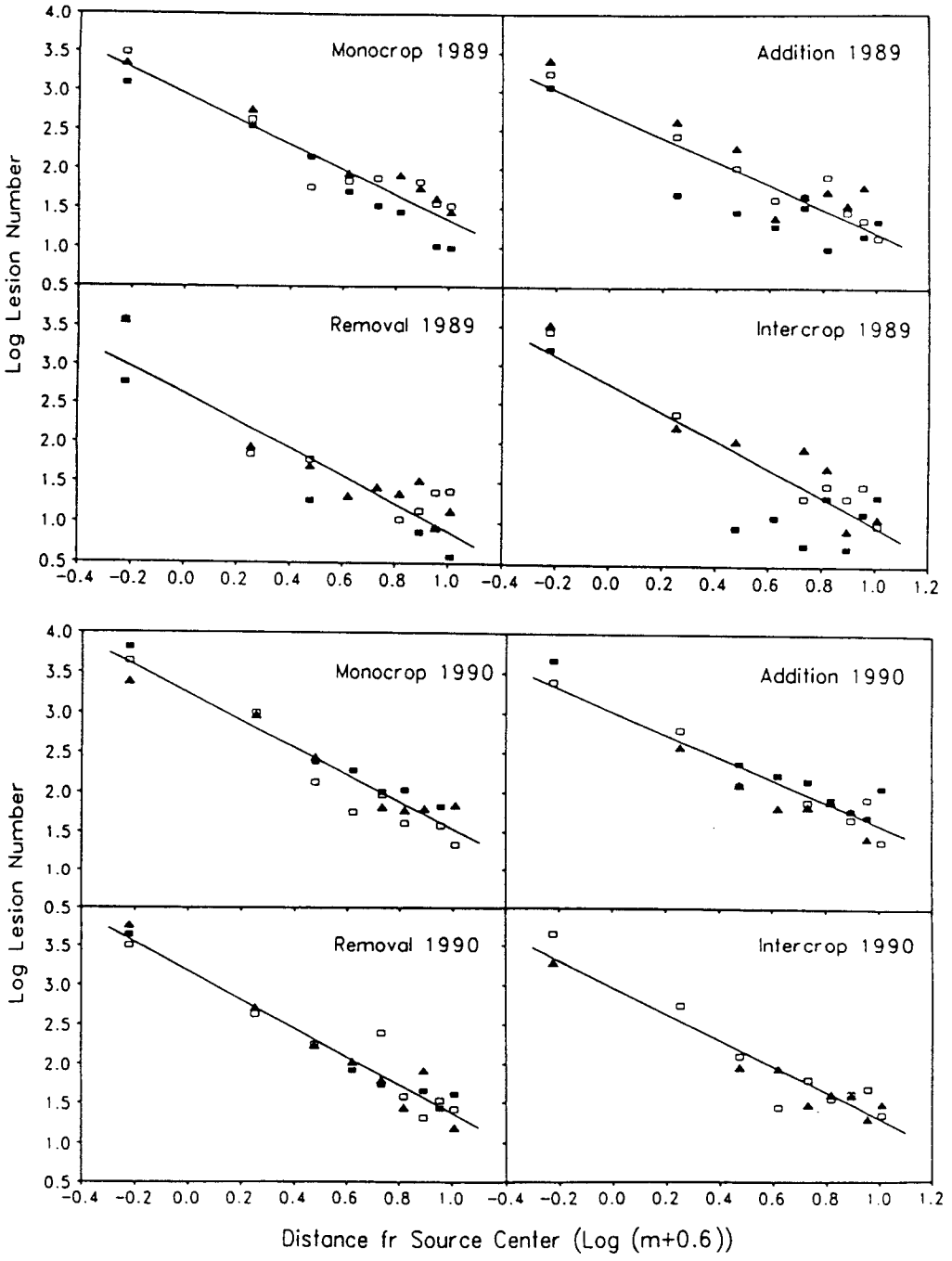

Monocrop 1989
Addition 1989
Removal 1989
Intercrop 1989
Monocrop 1990
Addition 1990
Removal 1990
Intercrop 1990
Log Lesion Number
Distance fr Source Center (Log (m+0.6))

FIGURE IV.3. Relative severity levels, approximately 12 days after inoculation with Uromyces appendiculatus urediniospores, on same-aged single leaves of bean plants growing within canopies of rust-resistant beans in monocrop, intercrop with maize, and intercrop with maize until immediately prior to inoculation (Removal). 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 (DAP). Each date was a separate inoculation experiment and utilized different plants. Actual severity values for monocrops were 26.3, 3.2, and 4.5% in 1989; and 0.2, 11.0, and 7.4% in 1990, respectively. Bars with different letters indicate significant differences ($P<0.05$) by the Newman-Keuls test performed on Log(x+1)-transformed data.

FIGURE IV.3.

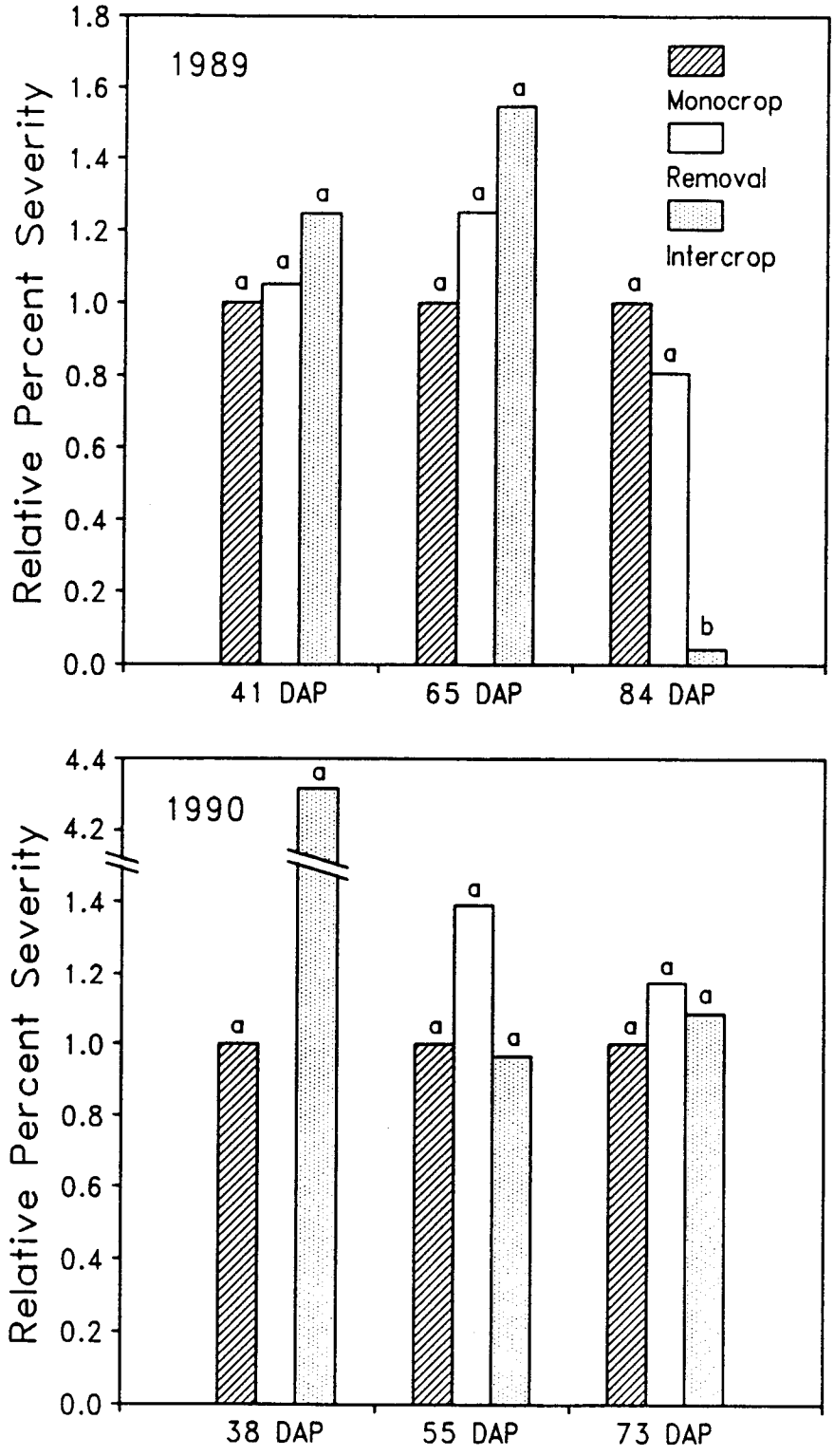
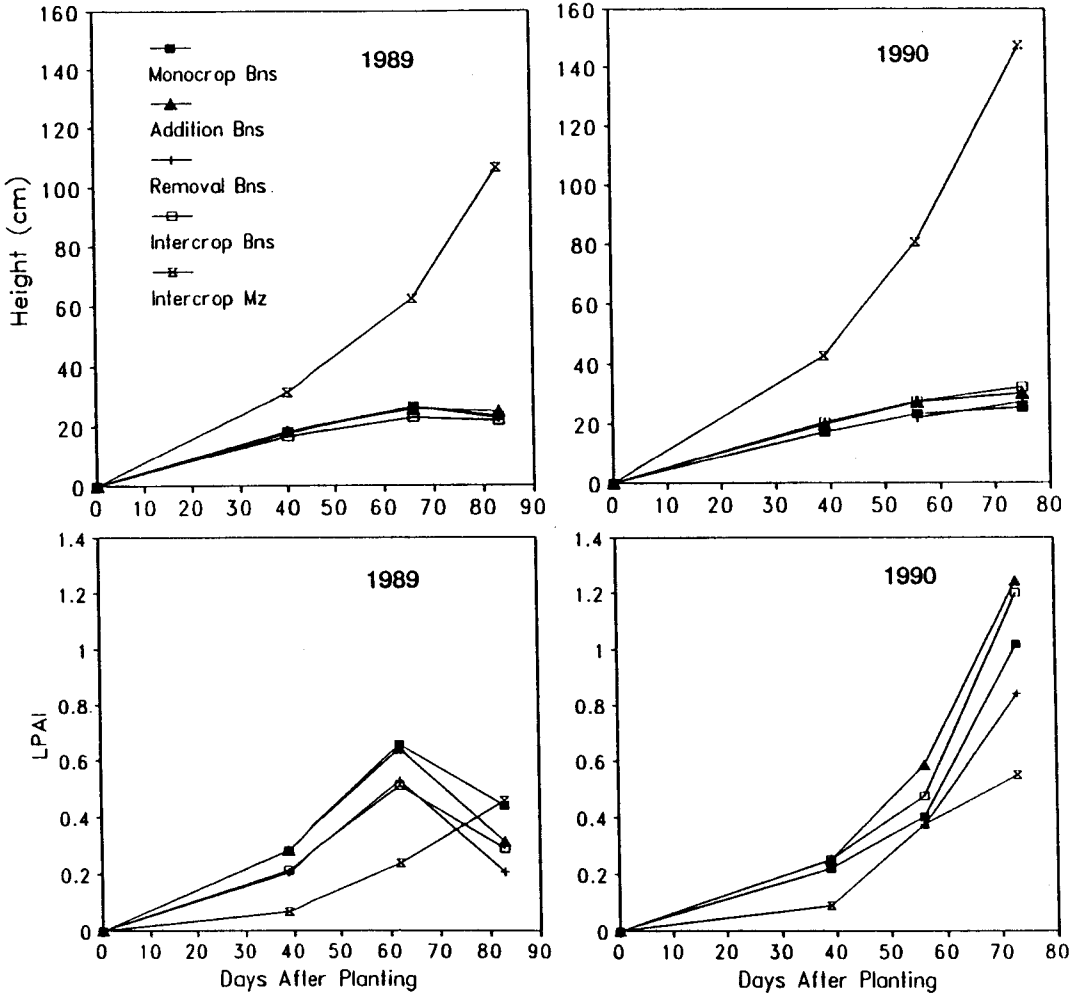

1989
Monocrop
Removal
Intercrop
Relative Percent Severity
1.8
1.6
1.4
1.2
1.0
0.8
0.6
0.4
0.2
0.0
a
a
a
a
a
a
a
a
b
41 DAP
65 DAP
84 DAP
1990
Relative Percent Severity
4.4
4.2
1.4
1.2
1.0
0.8
0.6
0.4
0.2
0.0
a
a
a
a
a
a
a
a
a
38 DAP
55 DAP
73 DAP

FIGURE IV.4. 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 7 and 15 plants sampled randomly for leaf + pod area and height, respectively, in 1989, and 8 and 16 plants in 1990. Bean data for the Removal treatment in 1990 is shown between the second and third sample dates only, because Removal treatment plots were not available at the first sample date due to poor maize growth. See Materials and Methods section for explanations of treatments, sampling method, and area index calculations. LPAI = (Leaf+Pod) Area Index; Bns = Beans; Mz = Maize.

FIGURE IV.4.


1989
1990
Monocrop Bns
Addition Bns
Removal Bns
Intercrop Bns
Intercrop Mz
Height (cm)
LPAI
Days After Planting
Days After Planting

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Chapter V

MECHANISMS OF DEVELOPMENT OF COMPUTER-SIMULATED BEAN RUST EPIDEMICS AS INFLUENCED BY INTERCROPPING WITH MAIZE

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Abstract

Computer simulations were performed to determine the dynamics of bean rust epidemics as influenced by components of maize-pathogen interaction in intercrops of beans and maize. Simulations were based on field data from 1989 and 1990, which allowed independent input of maize interference effects on spore dispersal, competition of maize with beans as it affected dispersal, and effects of maize on infection after spore deposition. A range of daily multiplication factors (DMFRs) were used. It was found that when all effects were combined, simulating an intercrop, area under the disease progress curve (AUDPC) was reduced to 32% of the monocrop value at low DMFR when using 1989 data, but that this difference declined as DMFR increased. Isolation of maize effects indicated reductions of infection efficiency as largely responsible for the lowered disease in 1989 simulations, although dispersal effects reduced disease to 81% of monocrop AUDPC at low DMFR. No infection effects were seen in 1990 field data, but simulations indicate AUDPC increases of 52% and 212%, respectively, for

interference and competition effects on dispersal at low DMFR. When the 1990 dispersal effects are combined (intercrop), the disease progress curve is almost identical to that of the monocrop. All dispersal effects on disease progression diminished as DMFR increased. Partitioning dispersal effects into those due to gradient slope changes and spore retention in the plot indicate that the latter accounts almost entirely for disease alteration. We have thus identified sources of variability seen in field studies on disease in bean-maize association: Effects of intercropping may be very dependent on rate of disease progress; microclimatic effects which may be inconsistent from year to year can have a strong impact on disease; and dispersal effects which may be qualitatively consistent between years can produce very different epidemics, largely due to the magnitude of spore escape.

Introduction

It has been suggested that intercropping, the simultaneous cultivation of more than one crop in close association, will generally reduce disease severity as compared with monocrops of the constituent species (1,5). However, of the few studies which have been done on this subject, some indicate no effects of intercropping on disease (18,21), while some indicate an increase in disease (11,16). This kind of variability can occur even within a single intercrop-pathogen system in different seasons or at different sites (14,15,21; Ch. II,III). A more detailed look at specific interactions among pathogen, host, and non-host at different stages in the disease cycle is required before generalizations can be attempted.

Mechanisms by which intercrops might affect disease have been proposed, including amelioration of dispersal factors (e.g. wind or rain) by the non-host, trapping of propagules by the non-host, microclimate alteration of the pathogen environment, reduced density of host, and changes in infection elicited by microorganisms (induced resistance) or pollen associated with the non-host (5,12,26). In addition, competition by the non-host may alter the host in such a way as to influence dispersal, distinct from dispersal effects due to the physical interference of the non-host with spore movement.

Competition alone could potentially alter the susceptibility of the host to the pathogen as well. Unfortunately, it is very difficult to evaluate the impact of these factors on disease, alone or in combination, on a field scale.

Burdon and Chilvers (6,7,8) were able to isolate plant density as the factor primarily responsible for disease reductions in barley-wheat mixtures infected with barley powdery mildew, and ryegrass-cress mixtures with damping off, but these studies were done under controlled-environment conditions. Chin and Wolfe (9) found density to be important in the field for mildew reductions in barley cultivar mixtures early in the season. Compensatory growth, however, which altered density within treatment levels of initially equivalent densities, invalidated comparison of density effects later in the experiment. Furthermore, these replacement-type experiments fail to explain disease alterations seen in additive-type mixtures (14,17,23; Ch. II,III), in which host planting density remains constant.

We have conducted experiments over two seasons evaluating the influence of maize (Zea mays L.) intercropped with beans (Phaseolus vulgaris L.) on Uromyces appendiculatus (Pers. ex Pers.) Unger, cause of bean rust (Ch. IV). By using an additive design and maintaining disease-free maize, we were able to consider the mechanisms

outlined above in three broad categories: The physical interference of maize with spore dispersal, the competition of maize with beans as it affects spore dispersal, and the influence of maize on infection after spore deposition. In one set of experiments, we found that the physical interference of maize with spore dispersal in isolation may produce more shallow dispersal gradients, but competition by maize with beans during growth, alone or in combination with interference, produces a steeper gradient ($P<0.05$). Besides gradient steepness, another measure of maize effects on dispersal was spore retention in plots, as opposed to spore escape from plots. These data indicated that competition had a strong influence ($P<0.05$), but the direction of the effect was reversed at different times in the season (i.e. more spores retained due to competition in mid-season, but fewer retained late in the season). In a second set of trials, intercropping greatly decreased infection efficiency of U. appendiculatus, but only late in one season. This was most likely due to microclimatic alterations created by maize, since competition with maize during growth alone did not effect infection efficiency. Although we were able to identify and quantify specific mechanisms of intercrop-disease interaction, we could not evaluate their combined impact on disease throughout the season.

One approach for methodically assessing the contributions of various intercropping effects on overall disease is through simulation modelling. Such a strategy requires knowledge of these individual effects, which can be translated into input variables for the model. The experiments described above provide the needed data.

The objectives of this study are to employ a simulation model, EPIMUL (13), to determine the disease resulting from intercropping effects on disease cycle components, in isolation and combination, based on empirical data for the bean-maize-bean rust system. These effects include interference of maize with spore dispersal, maize competition with beans as it affects spore dispersal, and the alteration of infection efficiency due to maize.

Materials and Methods

Simulator Operation

The disease simulator EPIMUL (13) divides the study area into a matrix of square compartments. In general, disease develops in each compartment according to a specified latent period, infectious period, leaf area index (LAI), and a daily multiplication factor (DMFR) of progeny lesions produced by each currently-sporulating lesion, a value analogous to the basic infection rate of Vanderplank (27). In the version of EPIMUL used for this study (20), spores that produce these progeny lesions are dispersed daily to new compartments based on the slope and truncation factor of the modified Gregory model (19). A further modification of EPIMUL (K. Johnsrude, unpublished) allowed alterations in gradient dispersal and lesion multiplication parameters at any time during the course of the epidemic. This version was run on an AT&T mini-computer at the Department of Botany and Plant Pathology, Oregon State University.

Constant Inputs. All input variables were based on bean-maize intercropping experiments conducted in 1989 and 1990 and described in Chapter IV. Although EPIMUL was originally designed to model epidemics in mixtures of immune and susceptible plants, we performed our simulations specifying only one susceptible crop component (i.e.,

beans) and no immune components. Any effects of maize were implicit in the particular input parameters used. This approach was possible because the intercropping system of interest represents an addition, rather than a replacement, design.

A 17 x 17 matrix of 1.2 x 1.2 m compartments was employed, corresponding to the field intercropping arrangement of 16 maize rows each 1.2-m apart, with two bean rows between each pair of maize rows. Bean monocrops were arranged in the same way in the field, but with maize absent. EPIMUL compartments of this size also were chosen to mimic the area of source inoculum and the area estimated by spore trap plants in field dispersal experiments. The truncation factor (c of the modified Gregory model) was the radius of the spore source (0.6 m), as used for all dispersal gradient estimates from the field. All epidemics were initiated with 135,000 spores deposited in the central compartment, based on an estimate of source intensity in the field experiments. Lesion size was assumed to be 1.5 mm^2 (15), but multiplied by 3 to accommodate the estimate that 33% severity corresponds to maximum area infected for U. appendiculatus (24). Infectious periods were taken as 25 days (personal observation) and epidemics simulated for a total of 75 days from inoculation.

Variable Inputs. In field experiments, effects of intercropping components on U. appendiculatus dispersal

(i.e. physical interference of maize with spore movement, and competition with beans) were quantified. This was done by estimating the slope of the dispersal gradient (b of the modified Gregory model) and total lesion number (an estimate of spore retention vs. escape from the plot) from a focal inoculum source, with non-dispersal factors held constant across all treatments. Effects of intercropping on non-dispersal aspects of the rust disease cycle were quantified by estimating latent period and infection efficiency. Leaf + pod area index (LPAI) was also estimated, and all assessments were done at three times during both 1989 and 1990. Measured values of gradient slope and latent period were used directly as EPIMUL inputs. LPAI was input for LAI, since pods as well as leaves became infected in simultaneous field experiments described in Ch. III (personal observation). Infection efficiency relative to the monocrop (treatment infection efficiency/monocrop infection efficiency), was multiplied by the monocrop DMFR to account for this effect in simulations.

EPIMUL provides no option for altering spore retention in a plot; in fact, it assumes that all spores are lost from the spore cloud and deposited in an area four times that of the study area. Ignoring this assumption could result in inaccurate relative disease levels among treatments. This is particularly problematic in the case

of U. appendiculatus, for which the gradient in the vicinity of the plot may largely be created by spore escape from the plot rather than deposition within the plot boundaries (3,10). The DMFR can be changed for a given treatment in a way which mimics the change in spore retention relative to the monocrop, or relative spore retention (RSR, calculated as treatment retention/monocrop retention). This adjustment must take into account any change in dispersal gradient as well. For example, if two treatments were shown to have equal spore retention in the field but unequal gradient slopes, the EPIMUL version we used will intrinsically retain more spores inside the plot with the steeper gradient. Therefore, the DMFR needs to be lowered in this case to simulate equal spore retention while preserving the different gradient slopes.

To derive appropriate DMFR values for each retention-slope combination, calibration simulations were run to determine the disease severity, at two latent periods after inoculation by a single infectious spore, for each slope used in the simulations at a range of DMFR values. By holding other input parameters constant, the severity becomes a constant multiple of the number of spores dispersed from a single lesion in one day and retained in the plot, i.e., an estimate of relative spore retention among slopes. Spore retention values have an exact linear relationship to DMFR, with a line unique to each gradient

slope. In other words, for a given dispersal gradient, each spore retention value has a corresponding DMFR value which can be calculated from a simple linear model. We used this relationship to accurately alter spore retention relative to monocrop retention using DMFR. The lesion number observed for a given DMFR in a monocrop calibration simulation was multiplied by RSR, then this value used in the calibration simulation for a different gradient slope to calculate a new DMFR. This DMFR is the value necessary to achieve the level of retention desired for the treatment, relative to the monocrop, at the new gradient slope.

Although dispersal parameters (gradient slope and spore retention) were determined in the field using data from both single leaf (generally first trifoliate) and multiple leaf combinations (Ch. IV), the single leaf data are used in these simulations because they are more comparable through time. The single-leaf and multiple-leaf data indicate the same pattern of responses to treatments (Ch. IV).

Treatments

Simulations were performed separately for the 1989 and 1990 field data (Ch. IV). The simulation treatments were a 2 x 2 x 2 factorial, the factors being: maize interference with spore dispersal (present or absent); the effect of maize competition with beans on spore dispersal (present or

absent); and the effect of maize on U. appendiculatus infection (present or absent). Eight treatment combinations were therefore possible. The treatment combination in which all three factors are absent represents the monocrop; when all are present, it represents an intercrop.

For each treatment, variable input values were reset on 11 and 32 days after inoculation (DAI) in 1989, and 9 and 26 DAI in 1990, corresponding to the midpoints of the time periods covered by the values estimated in field experiments (e.g., 1989 experiments were conducted 0, 21, and 42 DAI, so that the first experiment is appropriate to the period before 10.5 DAI, the second the period 10.5-31.5 DAI, and the third 31.5 DAI and later). Because EPIMUL does not at present allow daily changes in LAI, the value at the second assessment period was used for the entire simulation. This was thought to be the most appropriate value, because most of the disease increase observed in an experiment inoculated simultaneously (Ch. III) could be attributed to spores dispersed during the time period represented by this second assessment. Furthermore, values from the first leaf area assessment represent the period less than one latent period after the initial inoculation, before any spores were dispersed. The third assessment in 1989 occurred shortly after substantial defoliation (see Fig. III.3) and would not indicate leaf area in the early

part of the third time period. Although defoliation did not occur before the third assessment in 1990, comparison of epidemics between years warranted use of LAI values from the same assessment date.

Among the simulation treatments, input variables (LAI, gradient slope, DMFR, latent period) were changed as described in the preceding paragraphs. However, a change due to a particular factor in a particular time interval is only made if it had a significant effect at $P<0.10$ in the field experiments for that time interval, either for single- or multiple-leaf assessments. If it did not, the mean value of both levels of that factor was used in the simulator. If the interaction term was significant for any combination of factors in a particular time interval (at $P<0.10$), then values for all treatment combinations were employed regardless of main effects. Because maize had no significant effect on rust infection efficiency at any time in 1990, only the four treatment combinations that related to spore dispersal were simulated for that year (i.e., no interference or competition present, interference present without competition, competition present without interference, and both interference and competition present). Also, latent period was not affected by maize in any case, and is therefore set at the mean value (rounded to the nearest whole day) of 9 d for 1989 and 11 d for 1990. LAI was only changed in 1989, when competition

reduced leaf area at $P<0.10$ for the second and third field assessments. No significant changes in LAI occurred in 1990.

Input values for a base (monocrop) DMFR of 1.00 are given in Table 1. Each simulation was run at base DMFR values of 0.25, 1.00, 3.25, 5.50, 7.75, and 10.00. These values represent the range of basic infection rates (0.7-13.8), derived from apparent infection rates of 0.17-0.34, observed in monocrop bean plots from an adjacent experiment (Ch. III). Calculations of basic infection rates were done after Vanderplank (27), using the latent periods given above. Published values of apparent infection rate for bean rust are similar to the lower values in the range we observed (2). Although these calculations assume a logistic disease increase, and EPIMUL does not, they at least provide a rough guide to the range of disease progress rates which might be encountered.

Dispersal Partition

The dispersal components of gradient slope and spore retention are closely linked in the field, but simulation modelling allowed us to isolate the two factors to determine their relative importance to disease alteration in intercrops. Simulations were performed in which maize interference effects on gradient slope but not spore retention were incorporated, and in which interference effects on spore retention but not gradient slope were used. The same was done for competition effects of maize; combined effects of interference and competition; and combined effects of interference, competition, and maize influence on infection (i.e., the intercrop).

The simulations were done for both 1989 and 1990 at base DMFR values of 1.00, 3.25, and 7.75. Where slopes were altered, they were as indicated in Table 1. Where spore retention was altered, the RSR values were as in Table 1 but DMFR adjustments were different to accommodate the corresponding slope as described above.

Results

Disease progress curves for simulations with DMFR=1.00, 3.25, and 7.75 are presented in Figs. V.1-V.3. The relative effects of the treatments follow the same pattern for other DMFR values, with absolute severity levels increasing as DMFR is raised. For example, monocrop and intercrop severities 75 DAI were only 2.4% and 1.1%, respectively, in 1989; and 2.8% and 2.9% in 1990 at DMFR=0.25. At DMFR=10.00, monocrops and intercrops reached >95% severity at 43 and 42 DAI, respectively, in 1989; and at 49 and 48 DAI in 1990. The dispersal and infection effects of intercropping in 1989 are divided into two graphs for clarity, with the monocrop and intercrop curves provided on both for reference.

Overall Effects of Intercropping

In comparing only monocrop and intercrop disease progress curves, it is clear that intercropping tended to reduce disease in simulations based on 1989 data, but have virtually no effect in 1990 simulations. The 1989 intercrop simulation resulted in an area under the disease progress curve (AUDPC, calculated to 75 DAI after the mid-point mean method of Shaner and Finney (22)) of 272.6 percent-days relative to 844.2 percent-days for the monocrop at DMFR=1.00. This effect declined as DMFR increased, however, so that at DMFR=3.25, AUDPC for the

monocrop simulation was 2921.6 percent-days and for the intercrop 1777.0 percent-days. At a DMFR value of 7.75, both treatments become >95% diseased by the end of the epidemic, with AUDPC values of 3779.1 (monocrop) and 3749.2 percent-days (intercrop). Increases in DMFR accelerated both monocrop and intercrop epidemics in 1990 as well, but differences between the treatments remain negligible at all points in the epidemic; AUDPC values for monocrop and intercrop, respectively, are 653.7 and 710.2 percent-days at DMFR=1.00 and 3323.8 and 3540.9 percent-days at DMFR=7.75.

Intercropping Effects on Infection

Maize only influenced infection significantly in the field in 1989, and then only later in the season, but the effect in simulations is profound (Fig. V.1). Much of the intercrop disease reduction is attributable to this component, which gave rise to AUDPC values of 226.9, 1712.2, and 3596.6 percent-days at DMFR=1.00, 3.25, 7.75, respectively, in all cases lower than the corresponding intercrop values of 272.6, 1777.0, and 3749.2 percent-days. Disease progress curves indicate that comparing AUDPC alone may be misleading. The rate of disease progress after effects on infection are realized is dampened when dispersal effects are included, as indicated by the shallow slopes of the "+Infec/+Int" and "+Infec/+Cmp" lines compared to the "+Infec" line beyond 41 DAI. The severity

resulting from the effects of infection alone ("+Infec") tended to surpass all non-monocrop treatments by the end of the epidemic in these simulations. Only at low DMFR values are the severities due to intercropping (DMFR=0.25 or 1.00) or infection + interference effects (DMFR=0.25) not exceeded by those due to infection effects only.

Intercropping Effects on Dispersal

The effect of maize interference and competition on dispersal produced different epidemic patterns between years. At DMFR=1.00 using 1989 data (Fig. V.2), interference and competition both reduced disease severity (AUDPC=544.4 and 542.0 percent-days, respectively) relative to the monocrop (AUDPC=844.2 and 653.7 percent-days). Their combined effects (AUDPC=679.7 percent-days) resulted in more disease than a purely additive effect would predict. In 1990 simulations (Fig. V.3), however, both interference and competition increased disease (AUDPC=1078.8 for interference and 2215.5 percent-days for competition), with a particularly steep increase 30-37 DAI in the competition treatment. The combined treatments interacted to produce a disease progress curve and AUDPC (710.22 percent-days) very similar to that of the monocrop.

For both 1989 and 1990 simulations, treatment differences became much less pronounced as DMFR increased, although the hierarchy of treatment effects remained constant. At a DMFR of only 3.25, monocrop and all

dispersal treatments achieved >95% severity between 57 and 63 DAI in 1989, and 56 and 62 DAI in 1990.

The simulations partitioning dispersal effects into gradient slope and spore retention are given for DMFR values of 1.00 and 7.75 (Figs. V.4-V.6). A similar pattern emerges in both 1989 and 1990 simulations at all DMFR values and for either competition, interference, or their combined effects: Changes in gradient slope produced almost no change from monocrop disease progress curves, whereas changes in spore retention produced curves very similar to the effect of both components combined. This pattern also occurs when infection effects of maize are included (not shown). The disease progress curves representing monocrop and gradient slope alterations due to competition are indistinguishable at DMFR=1.00 in 1989 (Fig. V.5). Only when the effects of competition are partitioned at DMFR=1.00 in 1990 (Fig. V.5) is the total effect of competition on dispersal (+Ret/+Slp line) much greater than the effect of retention alone (+Ret line).

Discussion

Our simulated epidemics indicate a variability in overall effects of intercropping consistent with that seen in field experiments. Published reports indicate reductions in rust due to intercropping maize with beans in some cases (15,17,23; Ch. III), similar to simulations from 1989 data, and no effect of intercropping on rust in other cases (15,21; Ch. III), more similar to the simulations based on 1990 data. We did not observe increased severity due to intercropping in simulations, and increases have not been reported in the literature. Our own data from simultaneous experiments, conducted at the same location as those used to derive data for the simulations, resulted in severity reductions due to intercropping in both 1989 and 1990 in two sets of plots. However, there was no effect of intercropping in either year for a third set located in a different area of the farm (Ch. III). The site-to-site variability may have been attributable to the unique environment of this third set of plots (e.g. 6-m-tall trees immediately to the north, the direction of prevailing summer winds). The plots from which data were generated for our simulations (Ch. IV) were in an environment more akin to those showing disease decrease due to intercropping. Caution must be exercised in comparing these experiments, however, due to the use of bean

cultivars with different growth habits, and a generally more vigorous growth of the beans and maize in the intercropping trials of Ch. III (personal observation).

Overall effects of intercropping and their components became less pronounced as the rate of disease progress increased, another factor potentially accounting for variability in field studies. During early periods of crop growth, input variables are the same for all treatments (see Period 1, Table 1), presumably due to the small stature of the maize at this time. Since more of the epidemic occurs during this period as rate of disease progress increases, intercropping effects later in the season would be expected to influence overall disease less. This result is reinforced by the fact that events early in an epidemic are compounded and have a disproportionately large impact on disease relative to later events. Apparently, a sufficiently slow epidemic, or perhaps a delayed disease onset, is necessary to manifest the influence of a mature intercrop.

Disease reductions in 1989 simulations are largely attributable to maize influence on infection efficiency, particularly at high DMFR values (Fig. V.1). These effects were not related to maize competition with beans, and so were most probably due to microclimatic alterations in the intercrop, as argued in Chapter IV. Changes in microclimate have often been mentioned in the literature as

a likely mechanism of disease alteration (5,18,21,26), and these simulations indicate that its impact can indeed overwhelm other effects, even when it occurs late in the season. The crucial question is: How common is this magnitude of microclimatic influence on disease? It did not occur to any degree in our 1990 field experiments (Ch. IV), and the limited data available indicate only small changes in temperature, leaf wetness, and humidity parameters in bean canopies under maize (14,25; Ch. II). Nonetheless, the microclimatic effect is another potential mechanism for disease variability in intercrops. Microclimatic effects may be related to reductions or reversals of maize influence on bean disease between wet and dry seasons, as have been reported for rust (15) and angular leaf spot (14; Ch. II).

Even in the absence of the strong effects on infection, the influence of maize on dispersal in 1989 simulations resulted in substantial disease reductions at low DMFR (Fig. V.2). This did not occur using data from 1990 (Fig. V.3), suggesting that dispersal mechanisms may also be implicated in year-to-year variability in field results. In addition, interference and competition exhibited a strong interaction in 1990 simulations only, which ultimately maintained disease at monocrop levels. The high level of spore retention in the second interval of 1990, seen in RSR values and their corresponding slope-

corrected DMFR values (Table 1), are responsible for the rapid elevation of severity levels due to competition (30-37 DAI, Figs. V.3-V.4).

The mechanisms which may have generated these dispersal data are complex and discussed in Chapter IV. One new aspect of the dispersal phenomena suggested by these simulations is that spore retention, but not gradient slope, appeared to impact disease. Figs. V.4-V.6 clearly indicate that the increased steepness in gradient slope due to competition demonstrated in field experiments (Ch. IV) was never large enough to appreciably affect disease in simulations. Changes in spore retention, on the other hand, seem to drive any dispersal effects of intercropping on disease. These factors are not necessarily independent in the field. A uniform increase in deposition in a plot could increase retention (raise a in the Gregory model) without changing the gradient slope (4), but a non-uniform spore escape through turbulent diffusion, as suggested in Chapter IV, could result in a steeper slope and lower retention. The partitioning of these components in simulations nonetheless helps isolate the source of disease alterations seen in intercrops.

A second result of note in this study is that quite different effects of intercrops on overall disease can be produced by qualitatively similar effects on dispersal parameters between two years. Not only were effects on

gradient steepness consistent for the second and third periods in 1989 and 1990, but a reversal in intercropping effects on retention (more retention in Period 2; less in Period 3) occurred in both years (Ch. IV; also see Table V.1). At DMFR>1.00, however, differences between years due to dispersal are less apparent.

One implication of these simulations, then, is that a qualitative understanding of the constituent effects of intercropping on disease cycle components, even if they are consistent from year to year, may not have a great deal of predictive value. Knowing what disease level will result from intercropping in a particular environment may require more detailed quantitative knowledge of each factor of importance. We have shown that, in the bean-maize-bean rust system, this might best be pursued by gaining a more precise understanding of microclimatic influences of maize on infection; and of the influence of maize on spore retention, particularly due to competition. Rates of disease progress also need to be taken into account. The complex interactions we have observed could quite clearly lead to the kind of spatial and temporal variability seen in the field, a situation by no means unique to intercrop pathosystems.

TABLE V.1. Input values for EPIMUL simulations of bean rust epidemics with DMFR=1.000[a], as affected by components of maize intercropping, based on field studies from 1989 and 1990

Treatment[b]			LAI[a]	Slope[a]			RSR[a]			DMFR[a]		
Int	Cmp	Inf		Per 1[c]	Per 2	Per 3	Per 1	Per 2	Per 3	Per 1	Per 2	Per 3
1989												
-	-	-	0.650	-0.941	-1.710	-1.560	1.000	1.000	1.000	1.000	1.000	1.000
+	-	-	0.650	-0.941	-1.433	-1.560	1.000	0.855	0.720	1.000	1.291	0.720
-	+	-	0.519	-0.941	-1.701	-1.758	1.000	1.086	0.433	1.000	1.100	0.326
+	+	-	0.519	-0.941	-1.796	-1.758	1.000	1.115	0.584	1.000	0.993	0.440
-	-	+	0.650	-0.941	-1.710	-1.560	1.000	1.000	1.000	1.000	1.000	0.130
+	-	+	0.650	-0.941	-1.433	-1.560	1.000	0.855	0.720	1.000	1.291	0.094
-	+	+	0.519	-0.941	-1.701	-1.758	1.000	1.086	0.433	1.000	1.100	0.042
+	+	+	0.519	-0.941	-1.796	-1.758	1.000	1.115	0.584	1.000	0.993	0.057
1990												
-	-	NA	0.459	-1.611	-1.512	-1.701	1.000	1.000	1.000	1.000	1.000	1.000
+	-	NA	0.459	-1.611	-1.853	-1.425	1.000	2.326	1.000	1.000	1.443	1.511
-	+	NA	0.459	-1.611	-1.923	-1.812	1.000	4.463	0.716	1.000	2.545	0.617
+	+	NA	0.459	-1.611	-1.861	-1.678	1.000	2.058	0.716	1.000	1.264	0.740

Table V.1 Footnotes

[a]DMFR = Daily multiplication factor (progeny lesions produced/infectious lesion/day), adjusted from base monocrop value of 1.00 to reflect maize effects on infection efficiency and spore retention. LAI = leaf area index, using leaf + pod area index from field experiments. Slope = slope of primary dispersal gradient, based on modified Gregory model with truncation factor of 0.6 m applied to field data. RSR = relative spore retention, calculated as (estimated total lesion number in treatment plot/estimated total lesion number in monocrop plot) from field data. See Chapter IV for determination of empirical values in field trials, and Chapter V text for use of these values as inputs.

[b]Int = interference of maize with spore dispersal; Cmp = effect of maize on spore dispersal due to competition with beans; Inf = effect of maize on infection efficiency. "-" indicates the factor is absent in treatment; "+" indicates it is present; "NA" indicates the factor had no statistically significant effect in field data that year and was not included.

[c]Period of time over which input variable holds value given. For 1989, Per 1 = 0-10 days after inoculation (DAI); Per 2 = 11-31 DAI; Per 3 = 32-75 DAI. For 1990, Per 1 = 0-8 DAI; Per 2 = 9-25 DAI; Per 3 = 26-75 DAI.

FIGURE V.1. Disease progress curves for computer-simulated bean rust epidemics in bean monocrops, bean-maize intercrops, and as affected by components of maize influence on rust infection and spore dispersal, based on 1989 field data. +Infec = influence of maize on infection efficiency of pathogen included; +Int = interference of maize with spore dispersal included; +Cmp = competition of maize with beans as it affects spore dispersal included. DMFR = base daily multiplication factor (progeny lesions produced/infectious lesion/day in monocrop treatment), a determinant of the rate of disease progress. See text for explanation of input variable determination.

FIGURE V.1.

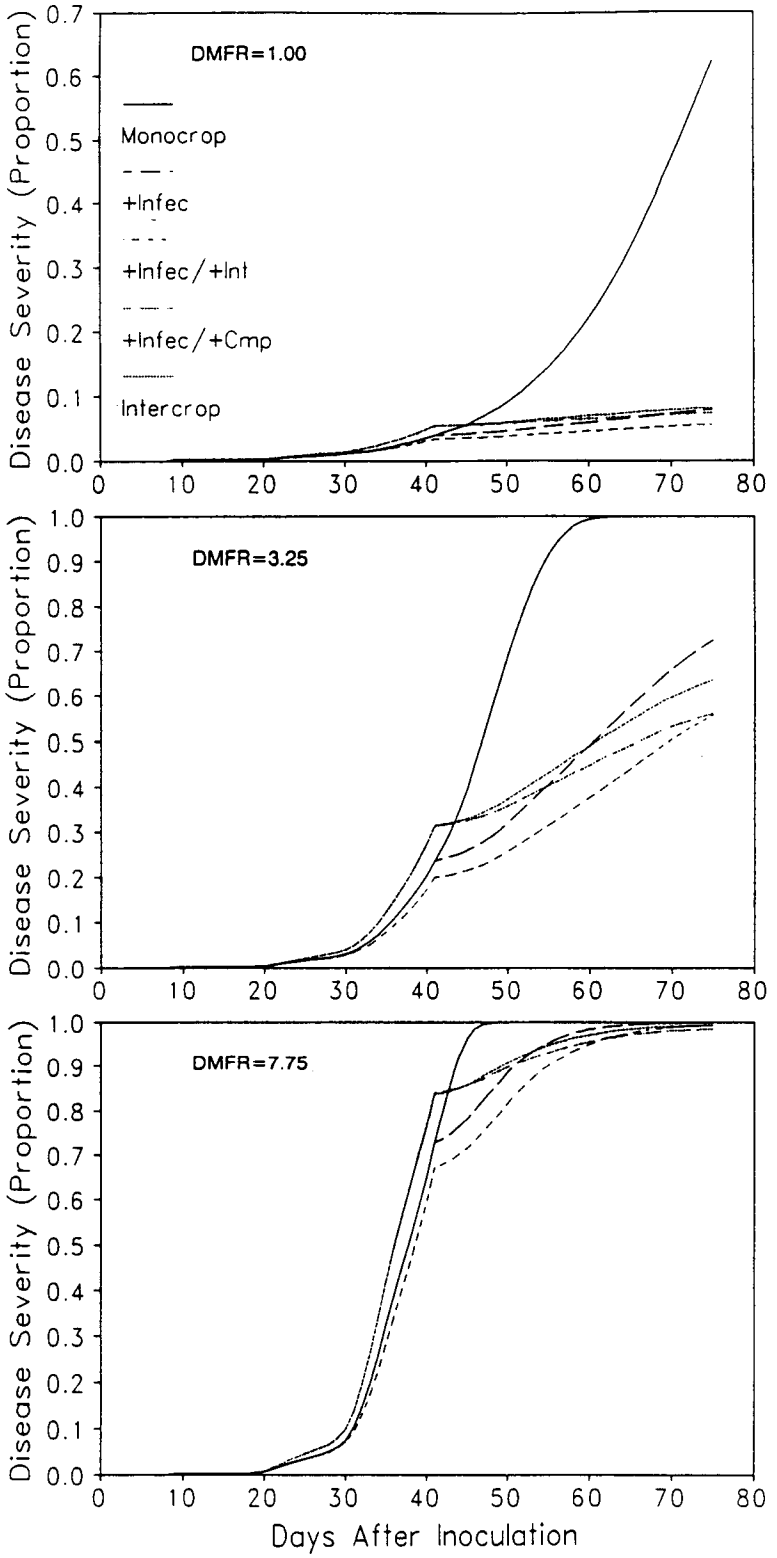

DMFR=1.00
Monocrop
+Infec
+Infec/+Int
+Infec/+Cmp
Intercrop
DMFR=3.25
DMFR=7.75
Disease Severity (Proportion)
Days After Inoculation

FIGURE V.2. Disease progress curves for computer-simulated bean rust epidemics in bean monocrops, bean-maize intercrops, and as affected by components of maize influence on spore dispersal, based on 1989 field data. +Int = interference of maize with spore dispersal included; +Cmp = competition on maize with beans as it affects spore dispersal included. DMFR = base daily multiplication factor (progeny lesions produced/infectious lesion/day in monocrop treatment), a determinant of the rate of disease progress. See text for explanation of input variable determination.

FIGURE V.2.

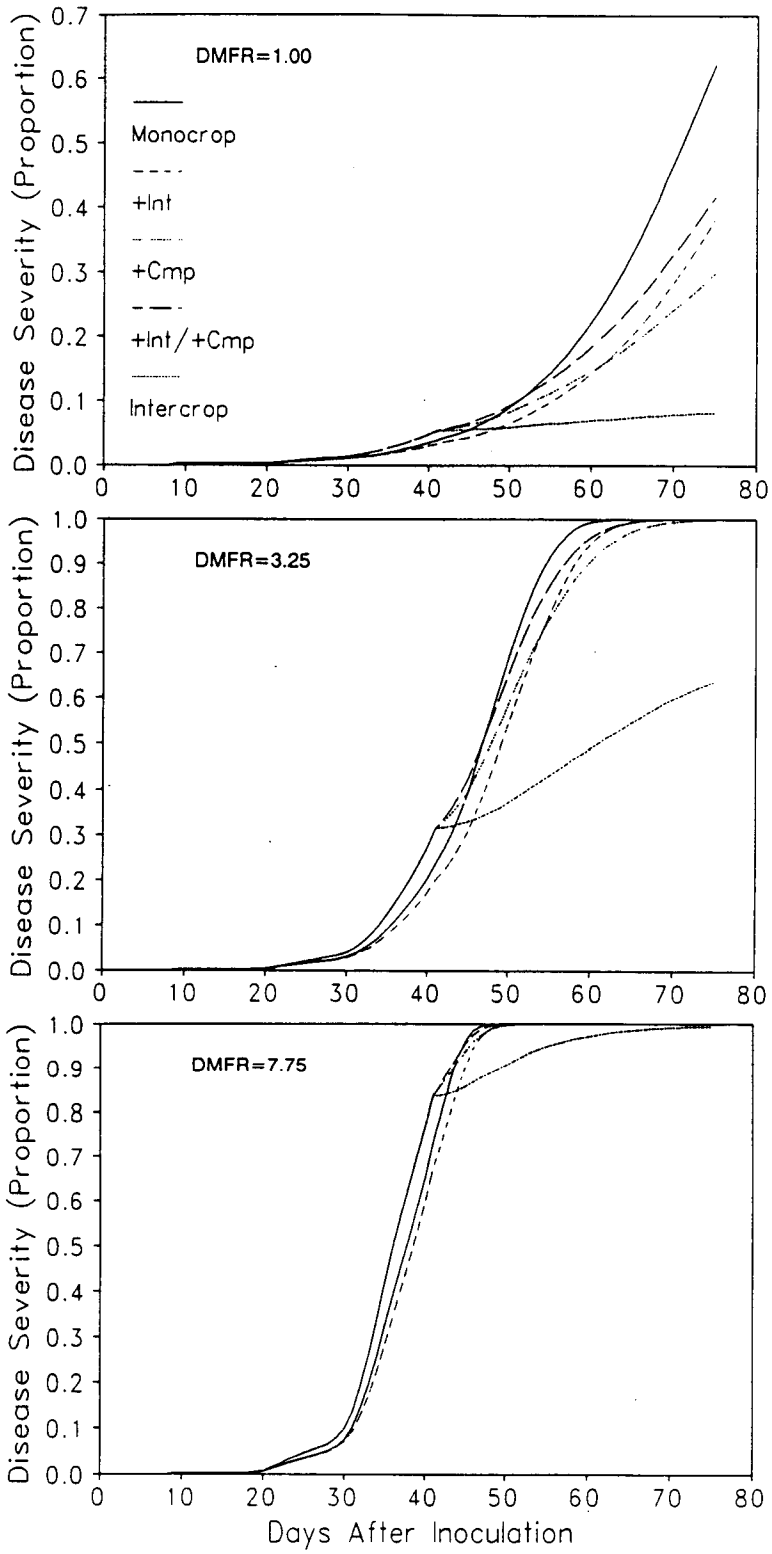

DMFR=1.00
Monocrop
+Int
+Cmp
+Int/+Cmp
Intercrop
DMFR=3.25
DMFR=7.75
Disease Severity (Proportion)
Days After Inoculation
0.0
0.1
0.2
0.3
0.4
0.5
0.6
0.7
0.8
0.9
1.0
0
10
20
30
40
50
60
70
80

FIGURE V.3. Disease progress curves for computer-simulated bean rust epidemics in bean monocrops, bean-maize intercrops, and as affected by components of maize influence on spore dispersal, based on 1990 field data. +Int = interference of maize with spore dispersal included; +Cmp = competition on maize with beans as it affects spore dispersal included. DMFR = base daily multiplication factor (progeny lesions produced/infectious lesion/day in monocrop treatment), a determinant of the rate of disease progress. See text for explanation of input variable determination.

FIGURE V.3.

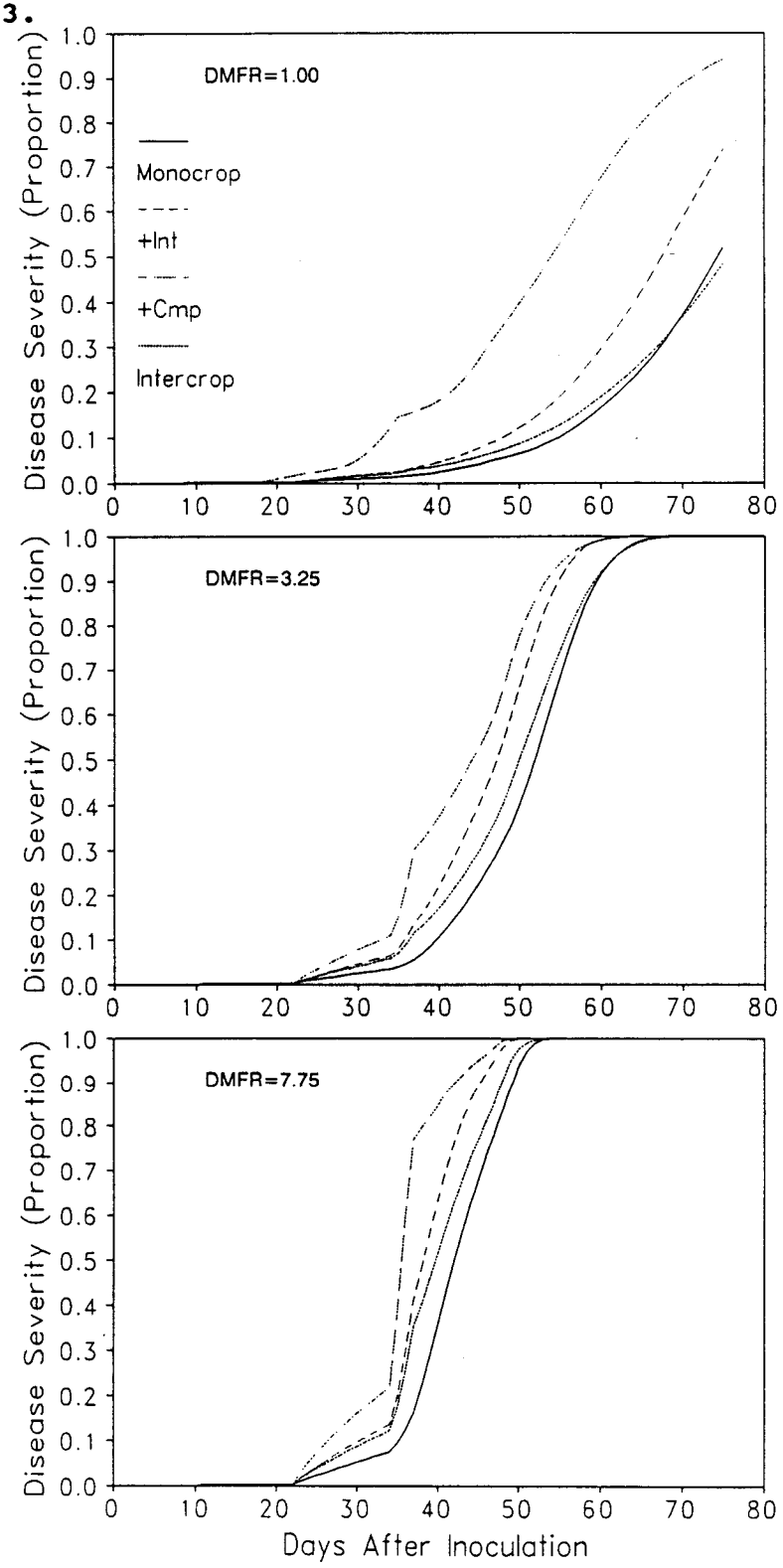

DMFR=1.00
Monocrop
+Int
+Cmp
Intercrop
DMFR=3.25
DMFR=7.75
Disease Severity (Proportion)
Days After Inoculation
0.0
0.1
0.2
0.3
0.4
0.5
0.6
0.7
0.8
0.9
1.0
0
10
20
30
40
50
60
70
80

FIGURE V.4. Disease progress curves for computer-simulated bean rust epidemics in bean monocrops, and as affected by components of maize interference with spore dispersal, based on field data from 1989 and 1990. +Ret = maize interference effect on spore retention in bean plot included; +Slp = maize interference effect on gradient slope steepness included. Combined effects (+Ret/+Slp) equivalent to total dispersal effects of maize due to interference (+Int in Figs. V.2 and V.3). DMFR = base daily multiplication factor (progeny lesions produced/infectious lesion/day in monocrop treatment), a determinant of the rate of disease progress. See text for explanation of input variable determination.

FIGURE V.4.

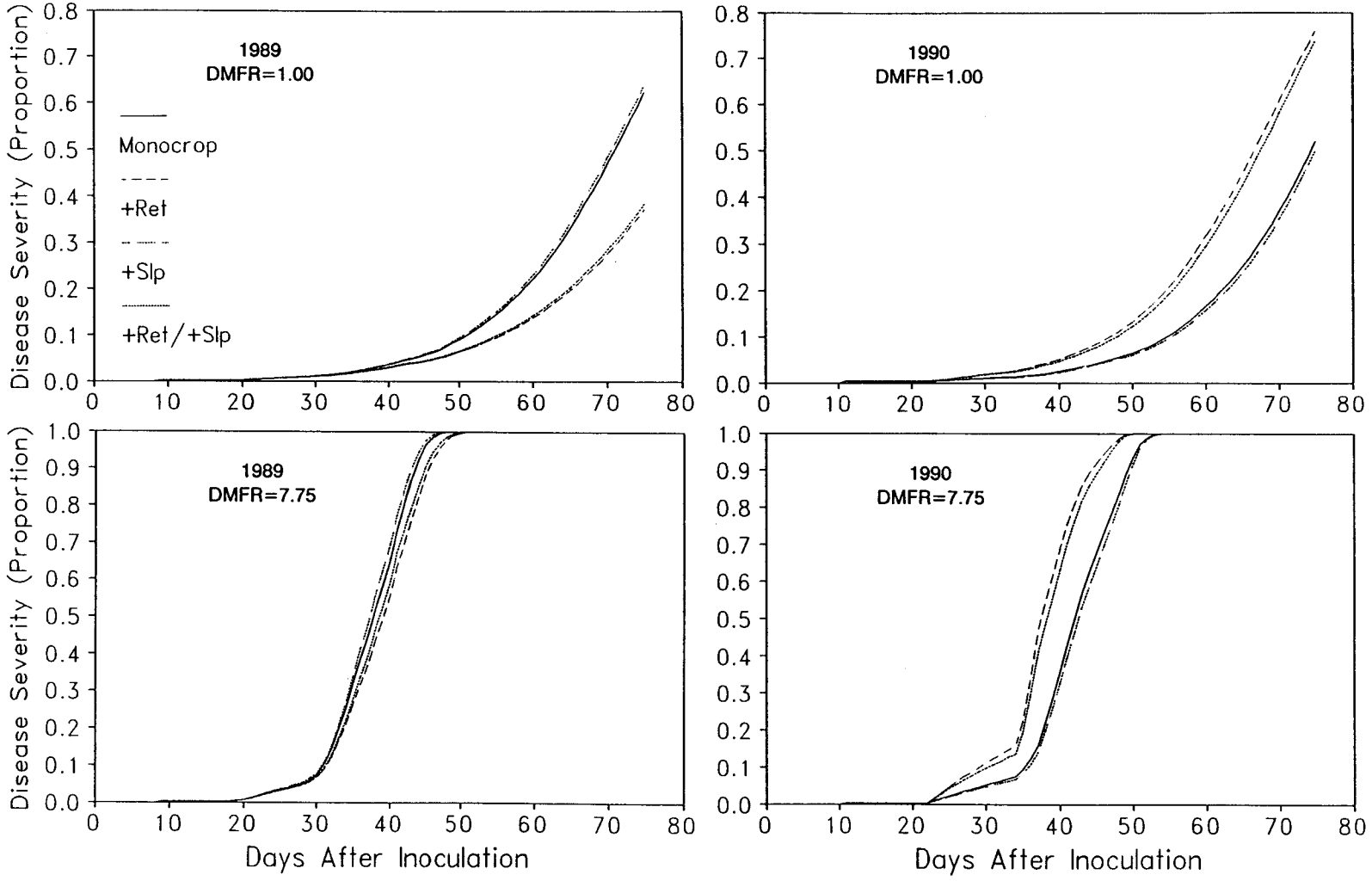

1989
DMFR=1.00
Monocrop
+Ret
+Slp
+Ret/+Slp
1990
DMFR=1.00
1989
DMFR=7.75
1990
DMFR=7.75
Disease Severity (Proportion)
Days After Inoculation

FIGURE V.5. Disease progress curves for computer-simulated bean rust epidemics in bean monocrops, and as affected by components of maize competition with beans acting on spore dispersal, based on field data from 1989 and 1990. +Ret = maize competition effect on spore retention in bean plot included; +Slp = maize competition effect on gradient slope steepness included. Note that Monocrop and +Slp lines are indistinguishable in 1989. Combined effects (+Ret/+Slp) are equivalent to total dispersal effects of maize due to competition (+Cmp in Figs. V.2 and V.3). DMFR = base daily multiplication factor (progeny lesions produced/infectious lesion/day in monocrop treatment), a determinant of the rate of disease progress. See text for explanation of input variable determination.

FIGURE V.5

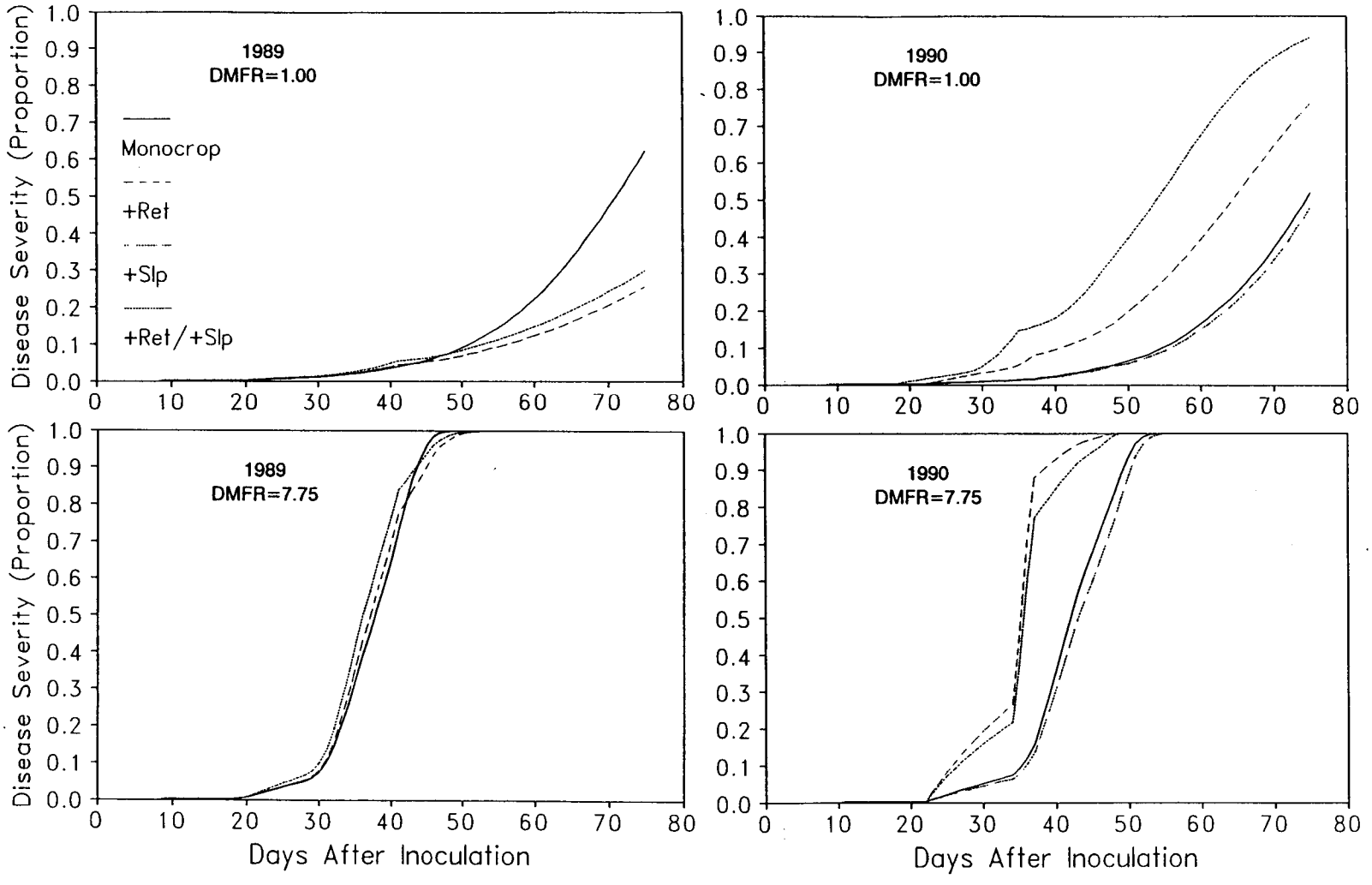
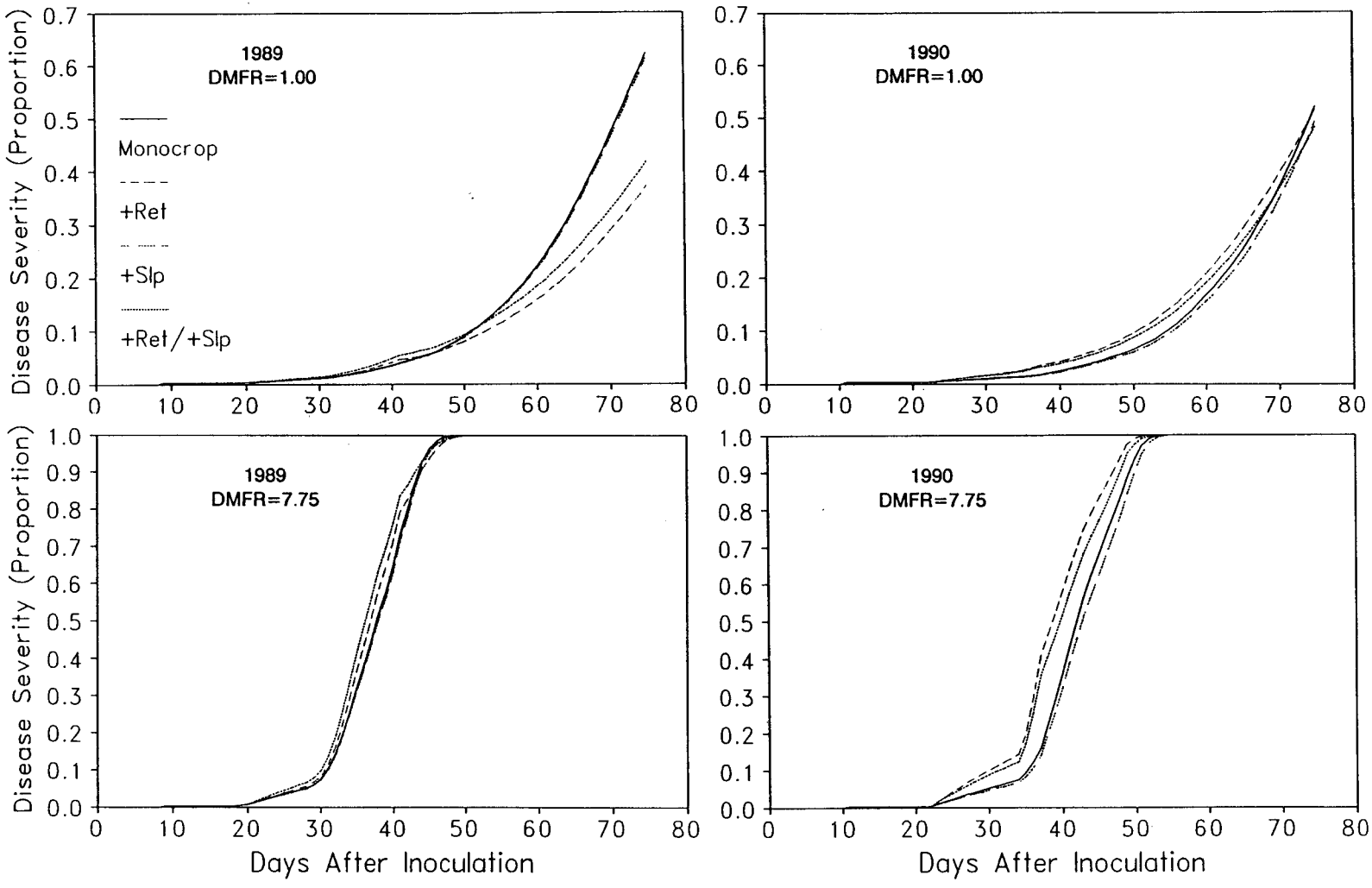

1989
DMFR=1.00
Monocrop
+Ret
+Slp
+Ret/+Slp
1990
DMFR=1.00
1989
DMFR=7.75
1990
DMFR=7.75
Disease Severity (Proportion)
Days After Inoculation

FIGURE V.6. Disease progress curves for computer-simulated bean rust epidemics in bean monocrops, and as affected by components of overall maize influence on spore dispersal (i.e., competition + interference effects), based on field data from 1989 and 1990. +Ret = maize influence on spore retention in bean plot included; +Slp = maize influence on gradient slope steepness included. Combined effects (+Ret/+Slp) equivalent to dispersal effects of maize due to both interference and competition (+Int/+Cmp in Figs. V.2 and V.3). DMFR = base daily multiplication factor (progeny lesions produced/infectious lesion/day in monocrop treatment), a determinant of the rate of disease progress. See text for explanation of input variable determination.

FIGURE V.6.


1989
DMFR=1.00
Monocrop
+Ret
+Slp
+Ret/+Slp
1990
DMFR=1.00
1989
DMFR=7.75
1990
DMFR=7.75
Disease Severity (Proportion)
Days After Inoculation

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