Progression of damping-off epidemics in *Glycine* populations of even-age and mixed-age structure

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Development of damping-off epidemics, caused by a random distribution of *Pythium aphanidermatum* in soybean populations, was quantified in even-aged and mixed-aged populations of *Glycine max* and *Glycine soja* (progenitor of cultivated species). Age structures were created to mimic naturally occurring even- and mixed-aged structures of *G. max* and *G. soja*, respectively. Damping-off epidemics developed more rapidly in populations of *G. max* than *G. soja*, in mixed-aged than even-aged populations of *G. max*, and even-aged than mixed-aged populations of *G. soja*. Diseased soybeans were distributed less uniformly, both temporally and spatially, and epidemics were longer in mixed-aged than even-aged structures and in populations of *G. soja* than *G. max*. Incidence of preemergence disease was greater near the inoculum source. Seedlings that damped-off after emergence and (or) those that remained symptomless were peripheral to the inoculum source. Epidemic development was dependent upon plant species and the temporal and spatial distribution of susceptible plants and virulent pathogen.

Key words: *Glycine max*, *Glycine soja*, *Pythium aphanidermatum*, soybean, survivorship analysis.

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Les auteurs ont quantifié le développement d'épidémies de fonte des semis, causées par une distribution au hazard du *Pythium aphanidermatum*, dans des populations de fève soja d'age uniforme et d'age aléatoire, appartenant aux espèces *Glycine max* et *Glycine soja* (progéniteurs des espèces cultivées). Les structures d'age ont été établies afin d'imiter des structures d'age mixtes et uniformes tels qu'on les trouve naturellement, pour *G. max* et *G. soja*. Les épidémies de fonte se développent plus rapidement dans les populations de *G. max* que dans celles de *G. soja*, dans celles dont la structure d'age est mixte plutôt qu'uniforme chez le *G. max*, alors que chez le *G. soja* elles sont plus rapides avec les structures d'age uniformes, plutôt que mixtes. Les plants de soja malades se distribuent moins uniformément dans le temps aussi bien que dans l'espace, et les épidémies sont de plus longue durée dans les populations dont la structure d'age est mixte plutôt qu'uniforme; ceci s'applique également aux populations du *G. soja* comparativement à celles du *G. max*. La maladie survenant au stade de préémergence est plus fréquente au voisinage de la source d'inoculum. Les plantules qui succombent après l'émergence et (ou) celles qui ne montrent aucun symptôme sont périphériques par rapport à la source d'inoculum. Le développement d'une épidémie dépend de l'espèce de plante ainsi que de la distribution spatio-temporelle des plantes susceptibles et du pathogène virulent.

Mots clés : *Glycine max*, *Glycine soja*, *Pythium aphanidermatum*, fève soja, analyse de survie.

[Traduit par la rédaction]

Introduction

The development of an epidemic of Pythium blight in a population of soybeans (*Glycine* Willd.) is comprised of a complex of plant and pathogen processes that interact in both time and space. Disease incidence and rate of epidemic development are determined by the nature of this interaction. For example, the fungus *Pythium aphanidermatum* (Edson) Fitzp. is a pathogen of *Glycine max* (L.) Merr. (11) and *Glycine soja* Sieb. & Zucc. (13), but when incubated under similar conditions, the incidence of disease caused by *P. aphanidermatum* is greater in populations of *G. max* than in populations of *G. soja*. Disease symptoms may develop on these plants within 4 and 9 days after planting, respectively (13).

In soil, a three-dimensional and structured medium, pathogenesis is affected by time and space because inoculation, infection, colonization, and reproduction occur over time and in space. Plant growth, maturation, and resistance to infection also are processes requiring time and are species dependent. For a plant and a pathogen, space may be defined simply as the distance between inoculum and infection site. This space must be reduced to zero before infection can occur. The process for reducing this space can be termed inoculation and the duration of inoculation depends on the rate of movement by the pathogen, the plant, or both.

Inoculation, the temporal distance between inoculum and infection site (i.e., the root), can be considered as the length of time required for fungus and plant movement to reduce the distance separating them to zero. This unit of time alone does not represent the rate at which the epidemic will progress (R). The rate of epidemic development integrates the rates of inoculation (r_i) and pathogenesis (r_p); these concepts are similar to VanderPlank's (17) description of infection and spore production rates, respectively. Pathogenesis is the process beginning with infection and ending with production of inocu-

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lum; the rate of this process equals r_p and the duration is called the latent period. If $r_p \gg r_i$, then R will approach the value r_p and pathogenesis will be the rate-limiting process. If $r_i \gg r_p$, then R will approach the value r_i and inoculation will be rate-limiting. When $r_p = r_i$, R is complicated by the fact that the time to complete inoculation (r_i) also can affect r_p, the time to complete pathogenesis. The time of inoculation may occur at maturation stage of plant tissue that slows or expediates r_p.

The objective of this study was to conceptualize components of an epidemic that influence r_i and r_p by examining the temporal and spatial components of damping-off epidemics caused by *P. aphanidermatum* in soybean populations of contrasting age structure. Populations were structured as mixed-aged and even-aged for each of two *Glycine* species in controlled environmental conditions favorable to the development of damping-off epidemics. Controlling the effective density of plants and inoculum among them creates conditions for different r_i values directly by influencing the density of susceptible plants and indirectly by influencing the life stage of individual plants when an infection occurs. Although not measured directly, r_p is inferred because it represents an integration of genetic, morphological, and physiological differences between plant species, their differences in population age structure, and period of susceptibility.

Materials and methods

Plant species

Glycine max cv. Williams and *G. soja* (progenitor of cultivated species) were used. Populations of *G. max* and *G. soja* naturally have even-aged and mixed-aged structures, respectively, due to differences in their temporal pattern of germination. Seed of *G. soja* (PI 135.624) was collected originally in the Heilongjiang Province near Harbin, China, and seed quantity was increased in Champaign, Illinois by R. Bernard. Seed of *G. max* was obtained from Illinois Foundation Seeds, Inc. Neither *G. max* or *G. soja* have specific resistance to *Pythium* spp.

Inoculum preparation

Pythium aphanidermatum was isolated from bentgrass (*Agrostis palustris* Huds.); it is a common soil inhabitant and is not host specific (11, 12). Agar cultures were stored in sterile distilled water at 21°C. Oat grains (*Avena sativa* L.) colonized by *P. aphanidermatum* were used to standardize the age, density, and position of the inoculum within a plant population. Oat seeds (mixed 1:1 v/v with water) were autoclaved (6.8 kg/645 mm^2) for 20 min on each of 3 successive days, while the pathogen was subcultured on potato dextrose agar at 21°C for 2 days. Then autoclaved oat grains were aseptically placed on *Pythium* cultures and incubated for 3 days (21−23°C).

Determination of susceptibility period

The period of host susceptibility to *P. aphanidermatum* was determined using methods described by Neher et al. (13). Prior to emergence, seeds and seedlings were considered susceptible to preemergence damping-off. Seedlings of *G. max* and *G. soja* emerged ≤3 and 4.3 days after planting, respectively (13). All postemergence damping-off occurred in ≤1 and 5 days after emergence for *G. max* and *G. soja* seedlings, respectively. Therefore, we concluded that the total period of susceptibility of *G. max* and *G. soja* seedlings to preemergence and postemergence damping-off combined were a maximum of 4 and 9 days after planting, respectively (13).

Epidemic development

For the population studies, plastic trays (45 × 36 × 12 cm) were divided into four equal sections with a Plexiglas partition. Trays were autoclaved and the partitions sterilized with 0.525% sodium hypochlorite. Each tray section, representing one population replicate, was filled with 3 L of sterile vermiculite and saturated with 1.8 L of sterile distilled water. The vermiculite was misted with sterile distilled water every 48 h.

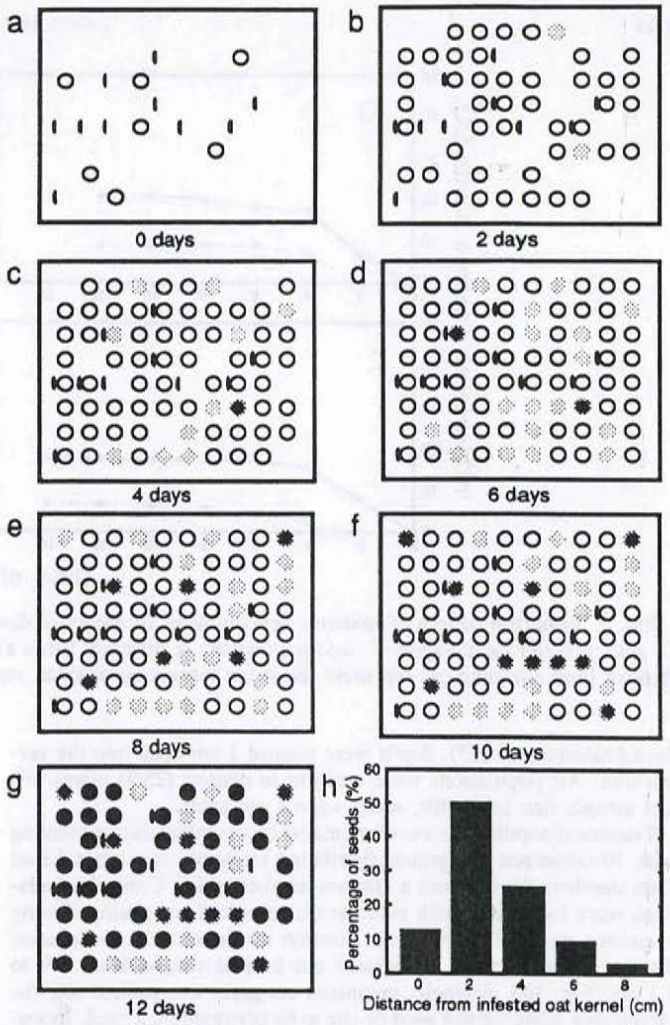

FIG. 1. Cumulative changes in the spatial aspects of a damping-off epidemic in one representative mixed-aged population of *G. max* at (*a*) 0 days, (*b*) 2 days, (*c*) 4 days, (*d*) 6 days, (*e*) 8 days, (*f*) 10 days, and (*g*) 12 days after inoculation. Illustrated are positions of infested oat kernels (solid line), seeds at time of planting (open circles), symptomless seedlings (stippled circles), postemergence damping-off (black stippled circles), and preemergence damping-off (solid circles). All seeds were planted 2 cm apart between 0−6 days after inoculation; however, the seeds are shown for 0-10 days because the cause of nonemergence could not be determined until after epidemic development ceased. Preemergence damping-off is illustrated only at day 12 because the time occurrence was unknown. Seeds considered to be nonviable owing to causes other than preemergence damping-off (probability estimated from control populations and position assigned randomly in inoculated populations) are eliminated on day 12. The lower right graph (*h*) illustrates the spatial pattern of seeds from the nearest infested oat kernel. These values represent the mean distribution across populations of both species and age structures.

Age structures were created to mimic naturally occurring even- and mixed-age structures of *G. max* and *G. soja* (13). Mixed-age structures for both plant species were created by staggered plantings; even-aged structures of *G. soja* were created by scarifying seeds with 95% sulfuric acid for $2\frac{1}{2}$ min and rinsing in sterile distilled water. No scarification was necessary for *G. max* because it naturally germinates synchronously. Contrasting age structures were developed by planting seeds on a single day (even-aged) and over 6 successive

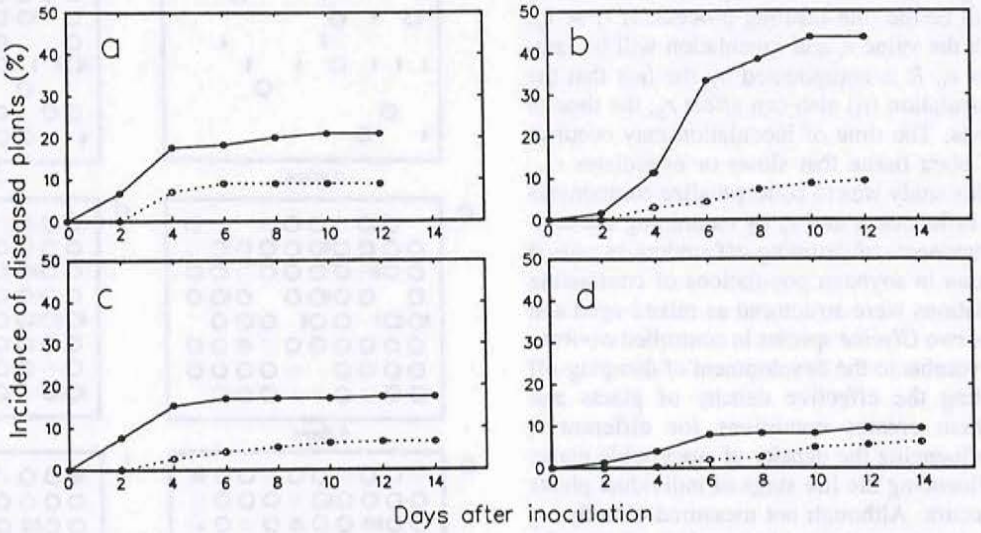

FIG. 2. Temporal pattern of epidemic development. Incidence of disease in (*a*) even-aged *Glycine max*, (*b*) mixed-aged *G. max*, (*c*) even-aged *G. soja*, and (*d*) mixed-aged *G. soja* populations at different times after inoculation. Values of preemergence (solid line) and postemergence (broken line) damping-off represent the mean frequency of three replicate populations.

days (mixed-aged) (13). Seeds were planted 1 cm deep into the vermiculite. All populations were uniform in density (2500 plants/m^2) and sample size (n = 80); seeds were 2 cm apart.

Treatment populations were inoculated on the initial day of planting with 10 colonized oat grains distributed randomly (positions based upon numbers drawn from a random number table). Control populations were inoculated with autoclaved, colonized oat grains. Owing to unique randomization of the position of oat kernels, the nearest distance between pairs of infested oat kernels ranged from 3.0 to 4.1 cm (Fig. 1*a*). A single, colonized oat grain was pushed into the vermiculite adjacent to a seed or site to be occupied by a seed. Inoculation occurred on day 1 of planting. Control populations allowed (*i*) estimation of preemergence damping-off in treatment populations (see below) by comparison and (*ii*) a check for mortality caused by factors other than *P. aphanidermatum*. Across all populations, 12.5, 50.2, 25.2, 9.3, and 2.8% of the seeds or seedlings were distributed approximately 0, 2, 4, 6, and 8 cm, respectively, from an infested oat kernel (Fig. 1*a*, 1*h*).

Summed over all treatment populations, the mean and maximum distance of a given seed or seedling from an infested oat kernel were 2.8 and 8.0 cm, respectively. Conservatively, 8 cm was chosen to be the maximum radius of disease development from an infested oat kernel. Although the pathogen may have moved >8 cm, the nearest infested oat kernel was assigned as the inoculum focus by necessity to calculate the maximum radius.

Following placement of inoculum, the trays were surrounded by wet paper towels enclosed in a plastic bag to maintain a saturated environment and incubated in an environmental chamber at a constant temperature of 31°C with a 14 h light : 10 h dark (120 μmol m^{-2} s^{-1}) cycle. These conditions favor pathogenesis (1,2,16).

The experiment was replicated three times and included control populations (without viable *Pythium*) replicated in the same design as treatment populations. A split-plot design was employed. Main plots were plant species and subplots were age structures. Each plot contained an equal number of treatment and control populations.

Data collection and statistical analyses

Day of seedling emergence and the occurrence of postemergence mortality were recorded for each seedling every 48 h until disease increase ceased, i.e., 12 days for *G. max* and 14 days for *G. soja*. Each 48-h time period for each seedling was considered a separate observation in the data set, to allow for temporal analyses of epidemic development. Preemergence damping-off was quantified indirectly by subtracting the seedling emergence frequency in a given treatment population from the mean emergence of respective controls for each plant species and age structure treatment on each observation day. In control populations, an average of 18% seeds did not germinate and (or) emerge for reasons other than the presence of the introduced pathogen, *P. aphanidermatum*. In treatment populations, we assumed an identical percentage of seeds failed to germinate and (or) emerge for reasons other than preemergence damping-off. Therefore, in spatial analyses, seedlings that did not emerge because of causes other than damping-off were randomly assigned a position. Postemergence damping-off was quantified directly. Postemergence damping-off was assigned if a seedling toppled because its stem base was weakened by necrosis.

TABLE 1. Temporal progression of epidemics

Time of damping-off	Wilcoxon statistic	df	P
Plant species			
PRE	18.62	1	0.0001
POST	6.78	1	0.0092
Age structure			
PRE	0.330	1	0.5656
POST	0.002	1	0.9644
Plant species × age structure			
PRE	30.19	3	0.0001
POST	9.06	3	0.0285

NOTE: Separate survivorship analyses (BMDP1L) were used to calculate Wilcoxon statistics for preemergence (PRE) and postemergence (POST) damping-off. The data used in the analyses for preemergence and postemergence damping-off represent frequencies of nonemergent seeds and diseased seedlings for each observation period, respectively (see Fig. 1).

The temporal pattern of preemergence and postemergence epidemic development was analyzed separately using BMDP survivorship analysis (6); a Wilcoxon (Breslow) statistic was computed. The cumulative spatial pattern of damping-off throughout the epidemic was analyzed with least squares means analyses to compare the mean distance between the nearest infested oat kernel and plants with (*i*) no disease symptoms, (*ii*) symptoms of preemergence damping-off, and (*iii*) symptoms of postemergence damping-off for each plant species and age structure treatment. Only inoculated populations were analyzed because no damping-off was observed in control populations. Rates of epidemic development (cm/day) were calculated by subtracting the

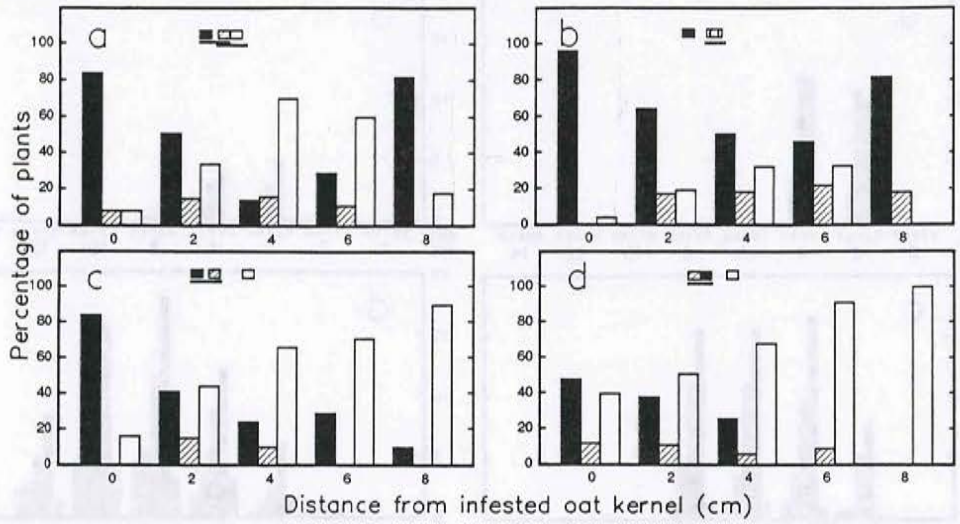

FIG. 3. Percentage of seed and seedling mortality at various distances (cm) from infested oat kernels on the final observation day (day 12 and 14 for *G. max* and *G. soja*, respectively). Values of preemergence damping-off (■), postemergence damping-off (▨), and symptomless seedlings (□) represent means of three replicate populations for (*a*) even-aged *Glycine max*, (*b*) mixed-aged *G. max*, (*c*) even-aged *G. soja*, and (*d*) mixed-aged *G. soja* populations. The position of the boxes above the bars (in reference to the *x*-axis scale) represent the mean distance of seeds or seedlings expressing pre- (■) or post-emergence damping-off (▨) or no symptoms (□) from infested oat kernels. Boxes not connected by horizontal lines indicate significant ($P < 0.05$) differences.

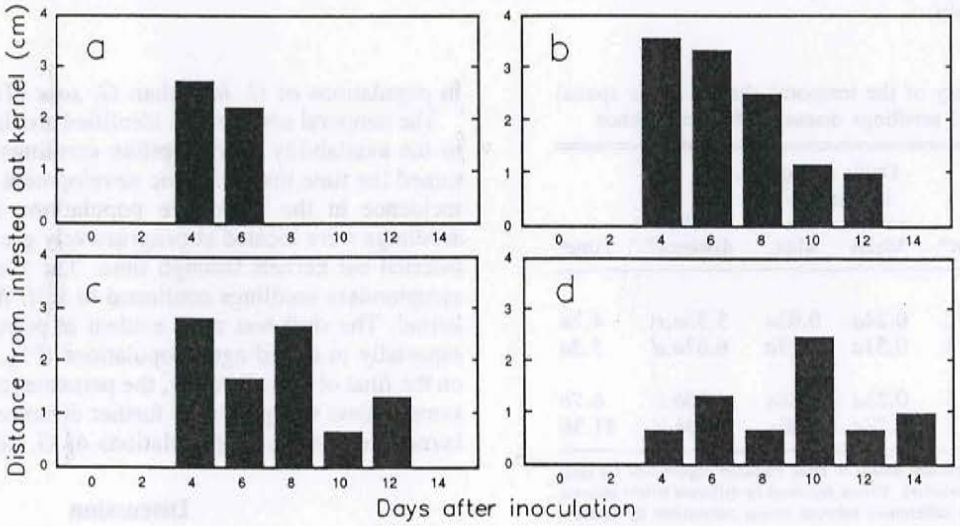

FIG. 4. Actual distance (cm) of seedlings with postemergence damping-off from infested oat kernels. Values represent the mean distance (of three replicate populations) from the kernels to seedlings that became symptomatic since the previous observation day in (*a*) even-aged *Glycine max*, (*b*) mixed-aged *G. max*, (*c*) even-aged *G. soja*, and (*d*) mixed-aged *G. soja* populations.

mean distance of emerged, newly diseased seedlings to the nearest infested oat kernel at time x days from the corresponding mean distance at time $x-2$ days for each treatment. This difference was divided by 2 because observation periods were separated by 2 days.

Results

Total mortality (preemergence and postemergence damping-off) was greater in populations of *G. max* than *G. soja*, in mixed-aged than even-aged populations of *G. max*, and in even-aged than mixed-aged populations of *G. soja* (Table 1; Fig. 2). The greatest total mortality was observed in mixed-aged populations of *G. max* and the least in mixed-aged populations of *G. soja*. More rapid epidemic development (R), indicated by the slope of the curve, was associated with greater total mortality. Maximum mortality, indicating no further epidemic development, was reached earlier in even-aged than mixed-aged populations and in populations of *G. soja* than *G. max*, especially preemergence damping-off. Mortality due to preemergence damping-off was greater than mortality due to postemergence damping-off for all populations (Fig. 2).

A spatial dimension can be added to the above temporal pattern. The epidemic developed in a consistent pattern around infested oat kernels. On the final observation day, the nearest zone was dominated by preemergence damping-off; seedlings that damped-off after emergence and (or) those that remained symptomless were peripheral to infested oat kernels (Figs. 1*g*, 3). This pattern was more distinct for populations of *G. soja* than *G. max* (Fig. 3). Epidemic development appeared more

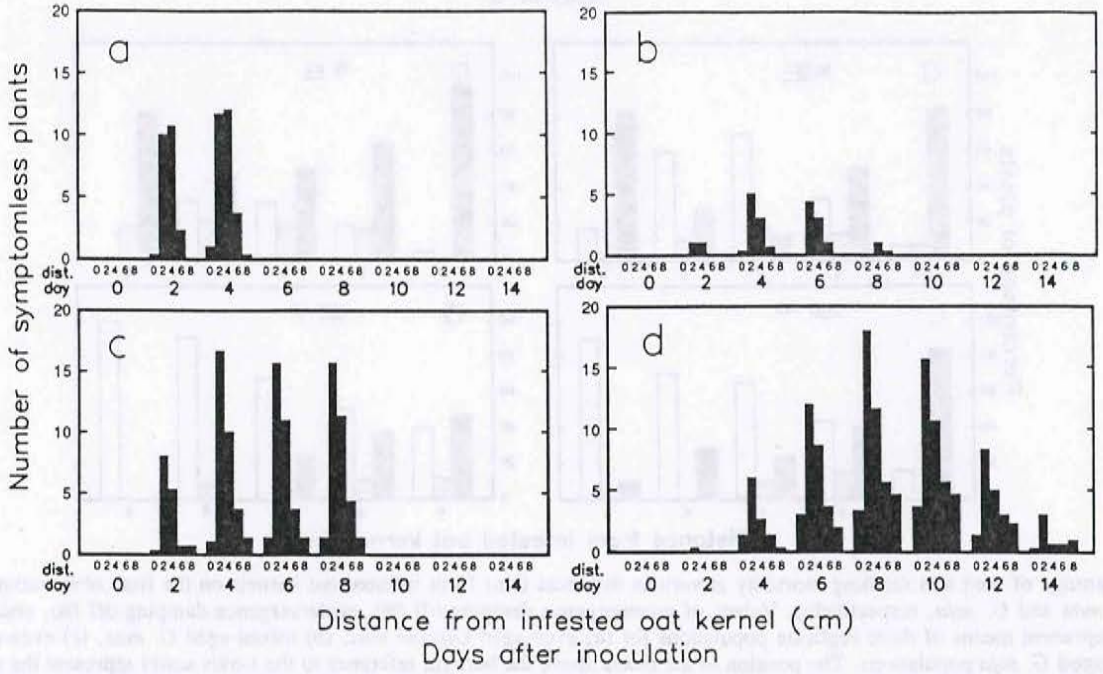

FIG. 5. Estimated temporal changes in the spatial distribution of symptomless seedlings within the age period considered susceptible to damping-off for (*a*) even-aged *Glycine max*, (*b*) mixed aged *G. max*, (*c*) even-aged *G. soja*, and (*d*) mixed-aged *G. soja* populations. These changes are based theoretically on earlier pathogenicity studies that estimated the susceptibility period for seedlings of *G. max* and *G. soja* as 1 and 5 days after emergence, respectively. The labels dist and day on the *x*-axis refer to distance from infested oat kernel and days after inoculation, respectively.

TABLE 2. Summary of the temporal change in the spatial distribution of seedlings diseased after emergence

Species and age structure	*n**	Daily change in distance† Mean	Daily change in distance† Max.	Max. distance‡	Time§
G. max					
Even-aged	2	0.24*a*	0.95*a*	5.33*a*,*c*	4.7*a*
Mixed-aged	4	0.51*a*	1.17*a*	6.67*a*,*d*	5.3*a*
G. soja					
Even-aged	5	0.23*a*	0.88*a*	4.00*b*,*c*	6.7*b*
Mixed-aged	5	0.23*a*	1.00*a*	5.33*b*,*d*	11.3*b*

NOTE: All values represent means of three replicate populations for each plant species and age structure. Values followed by different letters indicate significant ($P < 0.05$) differences between means determined by Tukey's procedure. When two letters appear, the first compared plant species and the second compared age structures.

*Number of bi-daily observation periods with additional seedlings expressing symptoms.

†The daily net change (cm/day) in mean and maximum distance of newly diseased seedlings from previously diseased seedlings.

‡The greatest distance (cm) between a diseased seedling and the nearest infested oat kernel across all observation periods.

§Days after inoculation when symptoms at the maximum distance were observed.

complex when both the temporal and spatial components were considered concurrently. The spatial distribution of seedlings expressing symptoms changed through time (Figs. 1, 4). The change in the mean distance between infested oat kernels and newly diseased seedlings ranged from 0.23 to 0.51 cm/day and was not significantly different among treatments (Table 2). The maximum distance of diseased seedlings from infested oat kernels was greater in mixed-age than even-age structures and in populations of *G. max* than *G. soja* and was reached sooner in populations of *G. max* than *G. soja* (Table 2).

The temporal components identified are those that contributed to the availability of susceptible seedlings and largely determined the time that epidemic development reached maximum incidence in the respective populations (Fig. 2). Diseased seedlings were located at progressively greater distances from infested oat kernels through time. The spatial distribution of symptomless seedlings continued to shift further from the oat kernel. The shift was most evident in populations of *G. soja*, especially in mixed-aged populations (Fig. 5). Consequently, on the final observation day, the percentage of plants that were symptomless was greater at further distances from infested oat kernels, especially in populations of *G. soja* (Fig. 3).

Discussion

The development of damping-off epidemics in *Glycine* spp. depended upon the distribution of susceptible plants through time and in space. This distribution was dictated by a combination of factors including plant species, population age structure, life stage at the time of infection, and period of susceptibility. Each of these components contributed to the net rate of epidemic development (R).

The effect of plant species on r_p can be measured only indirectly but is apparent by the differences in rate and severity of disease development among species in relationship to total mortality. Rapid pathogenesis (r_p) was assumed to be associated with populations with the greatest total mortality (Fig. 2). The difference in mortality between plant species observed in this and our previous study (13) suggests that r_p is greater in *G. max* than *G. soja*. The differences in susceptibility between plant species may be due to differences in (*i*) genetic variation within species, (*ii*) the interaction of the abiotic environment with the seeds and seedlings, and (or) (*iii*) the nutritional qual-

ity of the seeds. Saturated soils may increase host susceptibility (14,15) by delaying germination (9) and increasing the sugar and amino acid content of nutrient exudations from seeds and seedlings (2, 8). These conditions appear to have been more detrimental to *G. max* than *G. soja* because frequency of emergence was less in *G. max* than *G. soja* in wet soils in the presence of the pathogen (D. A. Neher, unpublished data).

The quantity and quality of plant tissue also influence pathogen growth and reproduction. In field soil, *P. aphanidermatum* can survive as mycelium or oospores. Oospores require more than a week to form in plant tissue. Mycelium will commence growth rapidly (about 0.72 cm/day) in the presence of nutrients and moisture (14), but oospores must be freed from plant debris before they will germinate. Germination of oospores can be direct, forming hyphae, or indirect, forming zoospores, depending on the environmental conditions (7). The r_p values for mycelium would be greater than those for oospores. Based on the rapid rate of epidemic development (Table 2), mycelium was the more likely infection structure in this study.

Within a plant species, age structure will affect r_i and r_p by creating an effective density and composition of plant maturity stages, respectively. Plant populations with high densities of seedlings (3) or numerous inoculum foci (10) have greater r_i and thus incidence of damping-off than populations with lower densities of seedlings or fewer inoculum foci because the temporal period between inoculum and susceptible seedlings is shorter. Heterogeneity in a population of plants decreases the density of susceptible individuals and decreases disease incidence (4,5) and thus r_i. The greater age diversity within mixed-aged structures and populations of *G. soja* may have provided greater structural diversity than populations of *G. max*, especially its even-aged structures. Although mixed-aged populations reduced the density of susceptible seedlings at one time, by increasing the distance between inoculum and infection sites and time between individual infections, they contained susceptible hosts for longer periods of time. The effect of mixed ages on disease should be even more pronounced and beneficial in indigenous populations of *G. soja*. The mixed-aged populations in this study were structured after only a portion (30%) of the natural temporal germination schedule for *G. soja*. Indigenous populations of *G. soja* extend germination over 2 years (R. L. Bernard, unpublished data).

The susceptibility of individual plants within even-aged or mixed-aged populations also changed through time (13); r_p differed between plants at different life stages, thus changing the average r_p of a plant population. Based on relative mortality levels, seeds were more susceptible to preemergence damping-off than seedlings were to postemergence damping-off (Figs. 2, 3; ref. 13). Assuming rates of pathogenesis are proportional to susceptibility of plant tissue, r_p was greater for seeds than seedlings. As a seedling matured, resistance increased and consequently r_p became lower, reducing the probability of completing pathogenesis. Two hypotheses may explain the greater vulnerability of seeds than seedlings to damping-off; (*i*) the period of susceptibility for preemergence damping-off may be longer than for postemergence damping-off or (*ii*) seeds are inherently more susceptible to damping-off than seedlings. The difference in vulnerability of seeds and seedlings to damping-off may partly explain why the incidence of preemergence damping-off was greatest adjacent to infested oat kernels.

Periods of susceptibility to preemergence and postemergence damping-off differed between the plant species evaluated (see above; ref. 13). The longer period of seedling susceptibility for *G. soja* than *G. max* may explain why the spatial distribution of susceptible seedlings shifted over a longer period of time in populations of *G. soja* (especially mixed-aged populations) than populations of *G. max* (Fig. 4).

This study provides an example of a disease system for conceptualizing factors that influence rates of inoculation (r_i) and pathogenesis (r_p), two components of the net rate of epidemic development (R). Plant species influenced r_p by differing in genetic variation, interaction with the abiotic environment, and (or) nutritional quality. Plant–population age structure influenced r_i and r_p by influencing the spatial and temporal distribution of susceptible individuals. Life stage at the time of infection and period of susceptibility influenced r_p by affecting the probability of completing pathogensis.

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