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Influence of age structure of plant populations on damping-off epidemics

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Summary. Germination synchrony may facilitate damping-off epidemics by creating a high density of uniformly susceptible individuals. We tested the hypothesis that synchronous germination causes increased seed and seedling mortality from damping-off in two legume species attacked by the fungal pathogen, *Pythium aphanidermatum*. *Glycine max* exhibited rapid, synchronous germination compared to its progenitor, *G. soja*, and suffered greater mortality from both pre- and postemergent damping-off in controlled environment experiments. However, when mixed-aged populations of *G. max* were created experimentally by staggering planting times, a significant increase in damping-off mortality occurred. In *G. soja*, which typically has mixed-aged populations due to asynchronous germination, experimental populations with an even-aged distribution also suffered increased damping-off mortality. Hence, the relationship between population age structure and damping-off mortality was species-specific. We propose that species differences in the duration of individual seedling susceptibility to disease interact with population age structure to control the outcome of damping-off epidemics.

Key words: Population age structure – Damping-off epidemics – Seed-seedling pathogens – *Glycine* – *Pythium*

Damping-off disease by fungal pathogens (e.g. *Pythium* spp.) causes seed and seedling mortality in a multitude of both agronomic crops and indigenous plant populations worldwide (Middleton 1943; Barton 1958; Hendrix and Campbell 1973). It influences the demography (Augspurger 1984), spatial distribution (Wilde and White 1939; Augspurger 1983), and genotype diversity (Hendrix and Campbell 1973) of plant populations. Epidemic development of damping-off requires the spatial and temporal overlap between susceptible host plant and virulent pathogen populations (Leach 1947; Garrett 1970; Wallace 1978), and a conducive environment. Earlier studies of damping-off have emphasized the spatial component of disease epidemics. Disease severity is greater with increased plant density (Burdon and Chilvers 1975a, b; Augspurger and Kelly 1984). It can be reduced in mixed-species stands where the rate of pathogen movement is decreased by the reduced density of susceptible individuals within the stand (Burdon and Chilvers 1976).

Less emphasis has been placed on temporal aspects of damping-off. The number of susceptible individuals in a population changes through time and depends on two factors: 1) the relative period of susceptibility of individual seeds/seedlings and 2) the degree of synchrony of germination and subsequent development among individuals in the population. Thus, to understand differences in epidemic development between populations or species, it is necessary to quantify these temporal factors, and to analyze interactions between them.

Individual seeds become susceptible to damping-off as they become non-dormant and begin to germinate. Resistance of individual seedlings to damping-off increases rapidly with maturity due to secondary wall thickening and lignin formation (Populer 1978; Ride 1983). Additionally, species may differ in their inherent susceptibility of individuals to damping-off, independent of temporal factors (McCarter and Littrell 1970).

In populations the changing density of susceptible seeds/seedlings through time depends on the temporal pattern of germination. Synchronous germination leads to an even-aged structure and hence relatively uniform susceptible populations. Asynchronous germination increases the range of ages, and creates populations of nonuniformly susceptible individuals. It has been proposed that a plant population with a mixed-aged structure would reduce the rate of pathogen reproduction and dispersal, thereby decreasing epidemic development (Burdon and Chilvers 1976; Cowling 1978; Schmidt 1978).

The objective of this study was to test the hypothesis that the population's level of damping-off disease is influenced by the density of susceptible individuals, as determined by germination synchrony. Using *Pythium aphanidermatum* (Edson) Fitz, a fungal pathogen causing damping-off, the hypothesis was tested experimentally by both interspecific and intraspecific comparisons under controlled environmental conditions. First, we compared two congeneric plant species with sharp contrasts in temporal germination pattern. *Glycine max* (L.) Merr. cv. Williams has been bred to germinate rapidly and synchronously, while *Glycine soja* Sieb. & Zucc., its progenitor, germinates more slowly and asynchronously (R.L. Bernard, unpublished work). Second, for intraspecific comparisons, germination synchrony was manipulated by either timed asynchronous

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plantings (*G. max*) or seed scarification to synchronize germination (*G. soja*) and achieve the desired contrasting age structures. The basic experiment was designed to quantify seed (preemergent) and seedling (postmergent) damping-off in populations with contrasting age structures exposed to randomly located inoculum. Two subsequent experiments examined the influence of delayed inoculation time and frontal disease invasion (border distribution of inoculum) on the development of damping-off epidemics. In a separate paper, we analyzed in detail the temporal and spatial dynamics of the damping-off epidemics in the first experiment (D.A. Neher, unpublished work).

Methods

Plant populations

G. max cv. Williams seed was obtained from Illinois Foundation Seeds, Inc. *G. soja* seed (PI 135.624) was collected originally in the Heilongjiang Province near Harbin, China and seed quantity was increased in Champaign, IL. The natural synchronous germination of *G. max* leads to an even-aged structure. To experimentally create mixed-aged populations of *G. max*, seeds were planted at staggered intervals. In *G. soja*, asynchronous germination normally leads to a mixed-aged structure. To experimentally create even-aged populations of *G. soja*, seeds were scarified with 95% sulfuric acid for $2^1/_2$ min and washed in sterile deionized water. The altered age structures of *G. soja* and *G. max* were constructed to simulate the natural germination pattern of the other species (Fig. 1).

Populations with even-aged and mixed-aged structures were planted over 1 d and 6 d, respectively. Although this planting schedule was identical for both species, seedling emergence was slower and more asynchronous for *G. soja* (for even-aged and mixed-aged populations, range = 10 d and 16 d, respectively) than *G. max* (range = 4 d and 10 d, respectively) (Fig. 1). All plant populations were uniform in density (2500 seeds $\cdot m^{-2}$) and sample size (n = 80 seeds arranged on a 16 cm × 20 cm grid). Prior to planting, seeds were surface sterilized in 0.525% sodium hypochlorite and 95% ethanol (1:1 ratio) and rinsed in deionized water.

Pathogen populations

Pythium aphanidermatum (Edson) Fitz was used as the fungal pathogen because it is virulent on both *G. max* (Morgan and Hartwig 1964; McCarter and Littrell 1970; Sinclair 1982) and *G. soja* (D.A. Neher, unpublished work). Neither species has specific resistance to *Pythium* spp. (R.L. Bernard, unpublished work). The pathogen was isolated originally from bentgrass (*Agrostis palustris* Huds.); it is a common soil inhabitant and is not host-specific (Middleton 1943; McCarter and Littrell 1970).

Colonized oat grains (*Avena sativa* L.) were used as the source of inoculum to allow standardization of the age and quantity of the pathogen population. To prepare the inoculum, first *Pythium* cultures were grown on potato dextrose agar at 21 C for 2 d. Then sterile oat grains were placed on the cultures and incubated for 3 d at 21–23 C. Ten oat grains served as individual sources of inoculum and were inserted 1 cm deep into the vermiculite and adjacent to different seeds. Summed over all treatment populations, the mean and maximum distance of a given seed/seedling from an initial inoculum source was 2.8 cm and 8.0 cm, respectively. The control populations received ten autoclaved colonized oat grains, lacking viable *Pythium*.

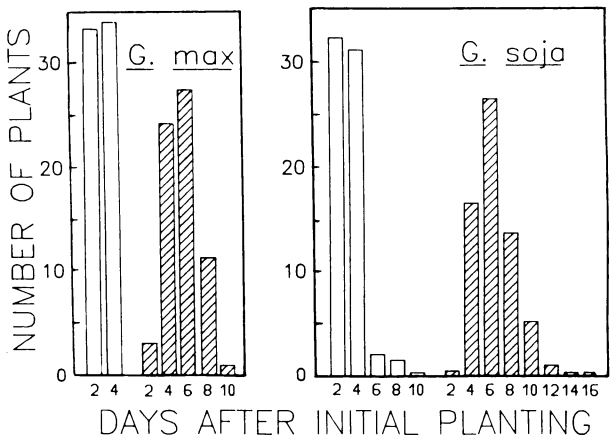

Fig. 1. Contrasting populations with even-aged (blank) and mixed-aged (hatched) structures of *G. max* and *G. soja*. Although data collection for seedling emergence was restricted to 48 h intervals, observations of populations with even-aged structure indicated that almost all *G. max* seedlings emerged by day 3, whereas most of *G. soja* seedlings emerged by day 4, although some delayed emergence until day 6–10. The data represent the mean emergence of three replicate poulations ($n = 80$ seeds). In the poulations, 80–84% of the seeds germinated for both species

Determination of susceptibility period

The period of an individual's susceptibility to the pathogen was determined as follows. First, a seed was considered susceptible to preemergent damping-off from planting until seedling emergence. This period was 3 d and 4.3 d, on average, for *G. max* and *G. soja*, respectively (Fig. 1). Second, in a separate experiment, seedlings were grown individually in plastic tubes filled with vermiculite. Six to seven seedlings in each of four age categories (0–1, 3–5, 7–8, and 10 d postemergence) each were inoculated with one oat grain colonized by the pathogen, and grown in environmental conditions described below. All postemergent damping-off occurred in seedlings $\leqq 1$ d and $\leqq 5$ d of age for *G. max* and *G. soja*, respectively. These observations on individual seedlings were verified by repeated observations of seedlings in populations. Therefore, we concluded that individuals were susceptible to pre- and postemergent damping-off combined for a maximum of 4 d (*G. max*) and 9 d (*G. soja*).

Environmental conditions

For the population studies, plastic tubs (45 × 36 × 12 cm) were divided into four equal sections with a plexiglass partition. First, tubs were autoclaved for 20 min and the partitions sterilized with 0.525% sodium hypochlorite. Then each section, representing one replicate population, was filled with 3 l of sterile vermiculite saturated with 1.8 l of deionized water. Vermiculite was chosen as the medium because of its practicality and strong retention of soil moisture, which is conducive to damping-off development. Following inoculation, the sides of the tubs were surrounded by wet paper towels and enclosed in a plastic bag to maintain high humidity. The vermiculite was misted with sterile deionized water every 24 h before inoculation and every

48 h following inoculation. The tubs were incubated in an environmental chamber at 31 C with a 14:10 h light regime and a light intensity of 120 μmol m^{-2} s^{-1}.

Experimental design

Each tub contained one plant species. The four sections in each tub were assigned randomly to the two age structures, with and without viable *Pythium*.

Experiment 1. Preemergent inoculation, random distribution of inoculum. On the initial day of planting seeds, the oat grains were placed in a spatially random pattern within each plant population to represent a residual pathogen population. The locations of oat grains in Experiments 1 and 2 were uniquely random for each population.

Experiment 2. Postemergent inoculation, random distribution of inoculum. The time of inoculation was adjusted to each species' relative period of individual susceptibility (see previous section) so that within each age structure treatment, the two species had approximately equivalent proportions of susceptible to resistant individuals. Inoculation occurred after seedling emergence when 100% and 74% of the individuals in populations with even-aged and mixed-aged structures, respectively, were susceptible to postemergent damping-off. For populations with even-aged structures, *G. max* and *G. soja* were inoculated 2 d and 4 d, respectively, after the day of seed planting. For populations with mixed-aged structures, *G. max* was inoculated 2 d after the last seed was planted (when seeds had been in soil for 2–7 d) and *G. soja* was inoculated 4 d after the last seed was planted (when seeds had been in soil for 4–10 d).

Experiment 3. Postemergent inoculation, border distribution of inoculum. Time of inoculation was the same as in Experiment 2. The oat grains were adjacent to ten seedlings along one border of the population. This distribution was designed to simulate the initial invasion of a pathogen into the population.

Each 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. An equal number of control and treatment populations occurred in each plot.

Preemergent damping-off, quantified only in Experiment 1, was estimated by subtracting the seedling emergence frequency of each treatment population (with *Pythium*) from the mean emergence of the respective controls (without *Pythium*) for each plant species and age structure. Postemergent damping-off was directly quantified for each experiment. Seedlings dying from postemergent damping-off toppled over due to necrosis of their stem base. The day of emergence and postemergent damping-off for each seedling was recorded. Observations were recorded every 48 h until the incidence of diseased seedlings no longer increased.

Statistical analysis

The data were analyzed as a four-dimensional cross-categorical analysis (Fienberg 1978). A maximum-likelihood ratio was calculated because of small sample size. In the analysis, plant species, age structure, and replicates were treated as factor variables; total seedling mortality, preemergent or postemergent damping-off mortality were treated as dependent response variables. A saturated model was used to test the significance of all main effects and interactions in all experiments.

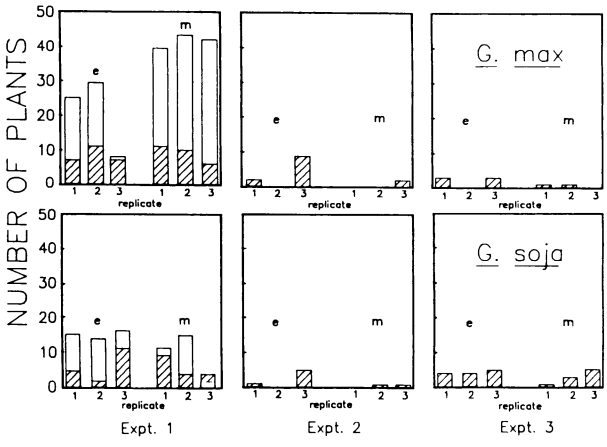

Fig. 2. Number of seeds and seedlings dying from preemergent (blank) and postemergent (hatched) damping-off in populations with even-aged (e) and mixed-aged (m) structures of *G. max* (top row) and *G. soja* (bottom row). The bars for pre- and postemergent damping-off are overlapping, not stacked. Values represent totals for individual replicates ($n = 80$ seeds) for each age structure and plant species. Results are shown for experiment 1 (preemergent random inoculum), experiment 2 (postemergent random inoculum), and experiment 3 (postemergent border inoculum)

Results

No damping-off occurred in any control population. Therefore control populations in Experiment 1 were used to estimate preemergent damping-off in treatment populations (see methods).

Experiment 1. Random distribution of preemergent inoculum. Generally, mortality was greater from preemergent than postemergent damping-off (Fig. 2). For pre- and postemergent damping-off combined, there were significant effects due to plant species ($X^2 = 70.91$, d.f. = 1, $P < 0.001$), the interaction of age structure with plant species ($X^2 = 29.57$, d.f. = 1, $P < 0.001$), and the three-way interaction among plant species, age structure and replicates ($X^2 = 20.48$, d.f. = 2, $P < 0.001$). Overall, total mortality was greater for *G. max* than *G. soja*. Within *G. max*, total mortality was greater in populations with mixed-aged than even-aged structures. The opposite relationship held for *G. soja*. For preemergent damping-off, the effects of plant species ($X^2 = 48.02$, d.f. = 1, $P < 0.001$), the interaction of plant species with age structure ($X^2 = 29.32$, d.f. = 1, $P < 0.001$), and the three-way interaction ($X^2 = 16.58$, d.f. = 2, $P < 0.001$) were significant. For postemergent damping-off, only plant species effects were significant ($X^2 = 19.17$, d.f. = 1, $P < 0.001$). Therefore, preemergent damping-off accounted for the interaction effects and the differences between plant species were observed for both preemergent and postemergent damping-off.

Experiment 2. Random distribution of postemergent inoculum. Seedling mortality caused by postemergent damping-

off was not significantly affected by age structure, plant species, or replicates (Fig. 2). Total mortality was significantly lower in Experiment 2 (postemergent inoculum) than Experiment 1 (preemergent inoculum) ($X^2 = 243.36$, d.f. = 1, $P < 0.001$). The reduction in mortality in Experiment 2 can be attributed to the absence of preemergent damping-off because there was no significant difference in mortality by postemergent damping-off between experiments.

Experiment 3. Border distribution of postemergent inoculum. Mortality was low and not significantly affected by age structure, plant species, or replicates (Fig. 2). The contrasting spatial distribution of inoculum between Experiments 2 and 3 did not result in significantly different levels of postemergent damping-off.

Discussion

Overall, synchronously germinating populations with even-aged structure did not experience consistently greater seed and seedling mortality compared to asynchronously germinating populations with mixed-aged structure. We reject the hypothesis that the density of susceptible individuals, as determined by germination synchrony, alone influences development of damping-off epidemics in populations. The data suggest instead that the influence of plant population age structure on damping-off epidemics depends on a combination of the time of pathogen introduction, the period of individual susceptibility, and plant species.

Time of pathogen introduction

The initiation of damping-off epidemics requires the temporal coincidence between susceptible plant tissue and a virulent pathogen. The period of susceptibility differs for seeds and seedlings. Germinating seeds prior to seedling emergence above the soil are continually susceptible to preemergent damping-off. Within 1 day (*G. max*) and 5 days (*G. soja*) after emergence, seedlings become resistant to postemergent damping-off. When the pathogen was introduced on the day of seed planting, preemergent mortality was greater than postemergent mortality. Also, the rate of epidemic development was influenced largely by the magnitude of preemergent mortality (D.A. Neher, unpublished work). These results may be due to differences in the degree and/or period of susceptibility between seeds and seedlings.

The incidence of preemergent damping-off greatly increases at temperatures that favor the growth rate of the pathogen relative to the plant (Leach 1947). The experimental temperature (31 C) was within the range most favorable to *P. aphanidermatum* (Adams 1971; Thompson et al. 1971). This high temperature accompanied by high soil moisture and high humidity provided conditions favorable to *P. aphanidermatum* growth and reproduction. High soil moisture and low oxygen levels can reduce germination and seedling emergence (Woodstock and Grabe 1967; Carver and Matthews 1975) and increase the sugar and amino acid exudates of seed compared to normal soil atmospheres (Kuan and Erwin 1980), thereby enhancing the growth and chemical attraction of the pathogen to the plant.

A disease gradient developed around the inoculum foci. Preemergent mortality was greatest near the source of inoculum; postemergent mortality and seedlings that "escaped" disease development were progressively further from the inoculum source (D.A. Neher, unpublished work). Extensive preemergent mortality increased the diameter of the inoculum source through time and distributed the pathogen more evenly through the plant population.

The time of disease development may be delayed until after seedling emergence, either by delayed artificial introduction as in this study, or a delay due to environmental conditions in nature, e.g. temperatures that favor the growth rate of the plant over the pathogen (Leach 1947; Garrett 1970). A delay may shorten the period of coincidence between susceptible tissue and active pathogen. Predictably, mortality was less in this study when the pathogen was introduced after seedling establishment than before seed planting. Earlier studies that focus on postemergent damping-off neglect the potentially greater impact of preemergent damping-off on the demography and selection of indigenous plant populations (Burdon and Chilvers 1976; Augspurger and Kelly 1984).

Spatial pattern of inoculation

The low disease incidence in the two experiments with postemergent inoculation made it impossible to distinguish between epidemic development from inoculum randomly spaced throughout the population and a line of inoculum at one border of the population. The growing inoculum foci increased less than 0.5 cm per day (D.A. Neher, unpublished work) and adjacent seedlings were 2 cm apart. Therefore, in either experiment, it is likely that the pathogen did not move rapidly enough to intercept additional susceptible seedlings before natural maturation allowed the seedlings to "escape" infection by developing resistance. Chi and Hanson (1962) found seedling age at the time of inoculation to be more influential on susceptibility of two legume species to *Pythium debaryanum* than soil type, moisture, or temperature.

Age structure differences

A population with synchronous germination provides a high density of susceptible individuals at one given time, while a population of equal density, but asynchronous germination provides a lower daily density of susceptible individuals over a longer period of time. Whether the pathogen responds to the greater daily density of susceptible individuals depends on its proximity, abundance, and ability to move rapidly from diseased to nondiseased, susceptible seedlings. For pre-emergent damping-off, there was a significant interaction of age structure with plant species. First, comparing natural germination patterns, the synchronously germinating *G. max* had greater mortality than the asynchronously germinating *G. soja*. This result supports the hypothesis that a greater density of susceptible individuals increases epidemic development. However, the result simply may reflect the fact that *G. max* is overall more susceptible than *G. soja* to this pathogen (see below).

Second, comparing contrasting germination patterns within each species, opposing results arose. In both species, populations forced to germinate in altered germination patterns incurred greater mortality than populations with their natural age structure. It appears that the natural age structure for each species allowed seed germination and seedling maturation to occur without correspondence with high levels of pathogen activity (Leach 1947; Garrett 1970). The

contrasting results between species may be caused by their difference in period of individual susceptibility.

G. max has more rapid germination and a shorter period of susceptibility as a seedling than *G. soja*. In its natural population with even-aged structure, the period of coincidence between plant and pathogen may have been too restricted for severe epidemic develoment. The expansion of inoculum foci was relatively slow (see above) compared to development of resistance. In contrast, in the population of *G. max* with a mixed-aged structure, it appears that inoculum foci continued growth when newly emerging susceptible individuals were continually available, even if they were of lower density and further from the initial inoculum point source.

For *G. soja*, mortality was slightly greater in populations with even-aged than in mixed-aged structures. *G. soja* has slower germination and remains susceptible as a seedling for a longer period of time than *G. max*. Hence, in *G. soja*, epidemic development increased in synchronous populations with a greater density of susceptible individuals, although the difference in mortality between populations was not great. The difference might be exaggerated in indigenous populations of *G. soja* because we arbitrarily eliminated the long tail of the temporal germination pattern occurring in indigenous *G. soja* populations (R.L. Bernard, unpublished work).

Plant species differences

Regardless of age structure, total mortality was greater for *G. max* than *G. soja*, the cultivar's progenitor. The differences may be attributed to: 1) the difference in seed quality between the species and/or 2) the loss of passive or active resistance over many generations of selective breeding. First, seeds of *G. max* are much larger than *G. soja* and may provide a richer nutrient source for pathogens (Harper et al. 1970), thereby increasing the chemotactic attraction of *Pythium* to plant roots (Wallace 1978). Second, the rate of seed decay may be faster in *G. max* than *G. soja*, thereby releasing oospores faster for secondary infection; following successful infection of the plant, oospores can not germinate until released by decay of plant tissue (Stanghellini 1974).

Breeding to produce *G. max* both shortened the individual's period of susceptibility and altered the population's germination to a highly synchronous pattern. Either or both temporal characteristics may have lowered the species' passive resistance to the pathogen, *Pythium aphanidermatum*. The influence of selective breeding on the species' active resistance mechanism(s) is not known. Breeding to produce *G. max* may have led to greater genetic uniformity for susceptibility to *Pythium*. *G. soja* may retain greater genetic diversity and offer more resistance to the pathogen. The net result of the above factors is that *G. max* has become more susceptible than its progenitor to disease epidemics due to this common damping-off pathogen.

Conclusions

The results indicate that several aspects play an important role in the development of damping-off epidemics. First, a delay in pathogen activity lessens the severity of the epidemic by shortening the overlap period between susceptible plants and a virulent pathogen. Second, species differ in their inherent susceptibility to damping-off pathogens. Third, by itself, the age structure of a poulation can not predict the severity of the epidemic; its effect is species-specific. Fourth, within a species, epidemic development depends on the interaction between the individual's period of susceptibility and the population density effect accruing from its age structure. When a high population density of susceptible tissue resulting from even ages among individuals is coupled with a short period of individual susceptibility, as in *G. max*, the population collectively "escapes" infection in time. In contrast, in *G. soja*, an even-aged population coupled with a longer period of individual susceptibility, increases the poulation's vulnerability to epidemic development.

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