

# Pathogen mortality of tropical tree seedlings: experimental studies of the effects of dispersal distance, seedling density, and light conditions

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**Summary.** We present results of two experiments designed to identify the relative importance of dispersal distance, seedling density, and light conditions on pathogen-caused mortality of tropical tree seedlings. The field experiment on Barro Colorado Island, Panama, demonstrated that both an increase in dispersal distance and a decrease in seedling density reduce levels of damping-off disease among seedlings of *Platypodium elegans*, and that there is an interaction between the two factors. The results indicated significant variation among sites in pathogen activity and suggested that seedlings are more vulnerable to disease when establishing around their parent tree than around other conspecific trees.

The second experiment in a screened enclosure used potted seedlings of 18 wind-dispersed tree species exposed to two levels of sunlight and seedling density. The results indicated that environmental conditions similar to those in light-gaps significantly reduce pathogen activity. They also confirmed that high seedling density increases disease levels, especially under shaded conditions.

Seedlings of 16 of the 18 species experienced pathogen-caused mortality, but in widely varying amounts. Seed weight was not a good predictor of a species' vulnerability to pathogens. Adult wood density, an indicator of growth rate and successional status, was inversely correlated with a species' vulnerability to pathogens. Fast-growing, colonizing species, whose seedlings require light-gaps, lacked strong resistance to seedling pathogens, relative to slow-growing species able to tolerate shade and escape seedling pathogens. We discuss these results in the context of seed dispersal as a means of escaping from seedling pathogens.

## Introduction

Earlier studies revealed the important role of pathogens as causes of mortality during the exponential decline in seedling numbers of tropical trees (Augspurger 1983a, b). Seed dispersal increased the probability that some of a parent's offspring escaped from pathogen death, thus influencing the location of offspring recruitment relative to the parent tree (Augspurger 1983b, c). These studies illustrated that the probability that disease kills a seedling declines with: 1) increasing dispersal distance, 2) decreasing seedling density, and/or 3) increasing light and associated microclimatic factors in light-gaps.

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The three variables, distance, density, and light-gaps, are related. Distance and seed/seedling density are inversely related. Greater dispersal distance increases the area over which seeds are distributed and enhances the probability that some seeds land in a light-gap. Here we report the results of two experimental studies conducted on Barro Colorado Island, Panama, which were designed to separate the relative importance of light, distance, and density on the disease mortality of tropical tree seedlings.

The field experiment in the shaded understory varied distance and density of seedlings. The study species was *Platypodium elegans*, a wind-dispersed tree species known to incur high levels of seedling mortality by fungal pathogens (damping-off) (Augspurger 1983a).

A second experiment in a screened enclosure varied density and light conditions. All 18 species used in this experiment were trees with wind-dispersed seeds. Nine of the species had been observed previously to suffer varying intensities of disease under field conditions (Augspurger 1983b).

A final objective of the multi-species experiment was to test two hypotheses:

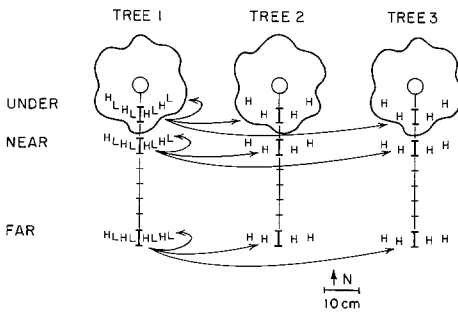
- 1) Among species, seedling vulnerability to disease declines with an increase in dry weight of seeds;
- 2) Among species, seedling vulnerability to disease declines with an increase in successional status, i.e. rapid-growing, colonizing species are more vulnerable than are shade-tolerant, persistent species.

The first hypothesis has been proposed in the literature (Grime and Jeffrey 1965, Ng 1978), but lacks an empirical, quantitative test. The second hypothesis originated from studies suggesting that early successional species have low defense against generalized herbivores (Cates and Oriens 1975, Feeny 1976, Rhoades and Cates 1976). Coley (1980, 1982) reported that leaves of fast-growing, colonizing tropical species are palatable throughout their life to insects, while leaves of shade-tolerant tropical species are scarcely eaten after the initial leaf flush. If pathogens function as a specialized form of herbivore, then the second hypothesis follows.

## Methods

### *Dispersal distance and density experiment: Platypodium elegans*

This experiment was carried out on Barro Colorado Island, Panama, in the wet season of 1982. The ecology of this



**Fig. 1.** Details of the experimental design for the distance-density experiment using seedlings of *Platypodium elegans*. *H* High Density Treatment (100 seeds per 0.25 m<sup>2</sup>); *L* Low Density Treatment (100 seeds per 2 m<sup>2</sup>; Tree 1 only). Replicates were separated by a minimum of 2.5 m. Arrows indicate that seeds of Tree 1 from a given distance interval were replaced at the same distance at Trees 1, 2, and 3. Canopy radius averaged about 8–9 m

semi-deciduous lowland tropical forest has been described in Leigh et al. (1982). Three reproductive canopy trees of *Platypodium elegans* were selected for study. The trees were separated from one another by more than 1 km. Seedlings of Trees 2 and 3, observed in 1980, had more fungal disease (damping-off) near than far from the trees; the disease was extreme near Tree 2 and moderate near Tree 3 (Augspurger 1983a). No seedlings appeared in 1981 at these two trees. The history of damping-off was unavailable for Tree 1, as its seedlings were first observed in 1982.

The experimental year was one of exceptionally low fruiting activity in *Platypodium*. Only Tree 1 had many fruits in 1982; it served as the seed source for the experimental design at all three trees. This design allowed for a test of the effect of site-to-site variation in disease mortality of one parent's seedlings.

Details of the experimental design are outlined in Fig. 1. Three distance intervals were selected south of each tree: under, representing non-dispersed fruits, near, and far. The majority of the 1-seeded fruits were dispersed in this downwind direction; also observations of damping-off in 1980 were made in this direction. Two density treatments were employed. The high and low densities approximated the natural density at germination at 5–15 m and 50–100 m, respectively, from the parent. In previous studies, roughly one-third of the seeds germinated.

Prior to the experiment, all naturally dispersed fruits of Tree 1 were removed in the late dry season, mid-April, within each of the three distance intervals. The fruits from different distances were kept separate and were stored in paper bags in a screened room for one month. This storage period reduced seed predation by ground mammals, although it was still high at Tree 3.

The fruits were replaced in the experimental quadrats when the rainy season and germination began. Consistent rains began by late May in 1982. Germination was synchronous, primarily in the first 2 weeks of June. Weekly observations of the number of seedlings dying from damping-off began 1 June and continued for 8 weeks, the time interval known from previous studies of *Platypodium* to encompass the major activity of damping-off fungi. Ascribing death to damping-off disease posed no difficulties; *Platypodium* showed the following classic symptoms: initial necrosis near soil level (the major criterion), wilting of leaves, and finally lodging from the weakened stem.

**Table 1.** Details of experimental design for density-light experiment in screened enclosure

	Low density 18 species	High density 13 species
Sun <sup>a</sup>	20 pots per species <sup>c</sup> 1 seedling per pot pot = 15 cm diam; 20 cm deep species randomly located	containers = 15 × 20 cm; 12 cm deep 8 species: 4 replicates 6 species: 50 seedlings per container 2 species: 30–40 seedlings per container 5 species: no replicates density range (5–43) species randomly located
Shade <sup>b</sup>	Identical to above	Identical to above
17.5 microeinsteins per m <sup>2</sup> per sec watered monthly		

<sup>a</sup> Sun treatment established on top tier of bench. Bench lined with plastic to prevent water runoff to underlying plants in shade. A layer of clear acetate, 1 m above the upper (sun) tier, diverted rainfall from all plants in sun and shade

<sup>b</sup> Shade treatment established on bottom tier of bench. Shade created by covering 1 m height with green shade cloth made of fine plastic mesh

<sup>c</sup> Sample size of *Platypodium elegans* was 228 seedlings in both sun and shade

Differences in the absolute number of seedlings arose within and among trees due to low germination levels under all three trees, and predation of seeds by mammals, particularly at near and far distances from Tree 3. To eliminate these differences in absolute numbers, the proportion of seedlings dying from damping-off disease was first calculated for each replicate, prior to performing an analysis of variance (fixed effect model). Separate analyses using the transformation arcs in  $\sqrt{p}$  gave the same results.

#### Density and light experiment: multi-species comparisons

This experiment was carried out on benches in a screened enclosure in the laboratory clearing on Barro Colorado Island, Panama, in the wet seasons of 1980–1982. The experimental design, outlined in detail in Table 1, included four treatments: sun-high density, sun-low density, shade-high density, and shade-low density. Germination rates and therefore seedling density varied among species and among replicates for a given species. Insufficient seed source prevented replicates from being established for some species.

Light conditions (photon flux density in the photosynthetically active range) in the enclosure and adjacent forest were measured at 2-h intervals on 4 cloudless days in July 1982. The mean daily maximum photon flux density for plants in the shade (17.5 microeinsteins per m<sup>2</sup> per sec) approximated those in the nearby forest understory (4.8, 9.3, 18.0, 25.0 at four forest sites). The light conditions for plants in the sun (330) were similar to those in a small light-gap or the edges of a large light-gap. Buildings, forest trees close to the small enclosure, and the screening of the

enclosure restricted its light level well below that found in the center of a nearby large light-gap (1640).

This experiment used 18 species of wind-dispersed canopy trees (see Croat 1978 for descriptions of the trees). Data were collected in 1980 for *Platypodium elegans*, in 1981 for 15 other species (see Table 4 for their names), and in 1982 for *Bombacopsis sessilis* and *Terminalia amazonica*. Differences between years in environmental conditions were minimized by initiating the experiment at the same time, late May, when the wet season with its rather uniform conditions was well underway, and by artificially delivering the same amount of water to the plants in all years. The soil in all years came from the same homogeneous mixture from a common soil pit established under one adult tree of *Dalbergia retusa*, one of the species used in the experiment. The soil had a sandy texture and drained well.

The seeds of most species were gathered in the late dry season, March to May, when wind dispersal activity was at its peak. The seeds were stored in paper bags until late May, when seeds with obvious insect or fungal damage were eliminated. Apparently healthy seeds were evenly distributed on the top of the soil, one per pot or 50 per container, in late May when the watering regime was initiated. No litter was added over the seeds. Seeds of *Ochroma pyramidale* were scarified to ensure their rapid germination. The above schedule was delayed by 3 wk for *Aspidosperma cruenata* and *Lonchocarpus pentaphyllus* because of their later time of seed dispersal.

Weekly observations of seedling mortality were made for the first 9 weeks when the great majority of seedling mortality occurs. Disease was assigned as the cause of death only if 1) the dead seedling was healthy the previous week, i.e. death was rapid, and 2) the dead seedling showed overt symptoms of disease. Dead seedlings were not removed, but were identified by driving a slender plastic marker into the soil.

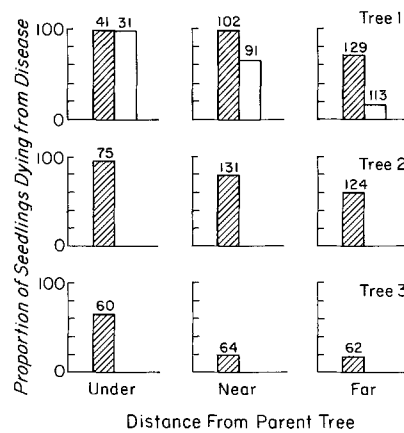
Mean dry weight of seed for each species was based on a sample of 15 seeds with all dispersal appendages removed. Such seeds were oven dried at 40° C to constant weight and reweighed. Values for basic wood density (green volume, oven dry weight) were obtained largely from Chudnoff (1979).

In the data analysis, individual pots in the low density treatment could not be considered replicates; the experimental unit consisted of 20 individual pots. Therefore, high density containers were also not analyzed as replicate samples; instead, the counts of diseased seedlings from all containers were combined. The data were analyzed as a three-dimensional contingency table (Fienberg 1978). In the analysis, light (sun or shade) and density (high or low) were set experimentally as independent factors, each, potentially, influencing disease (present or absent) as the dependent variable. The analysis tested for interactions among these factors. The interpretation of, for example, a significant disease-light interaction is that light influences disease.

## Results

### Density-distance experiment

For each tree, the proportion of seedlings dying from disease was lower at the far distance than under the tree; for Trees 2 and 3, it was also lower at the near distance



**Fig. 2.** The proportion of seedlings of *Platypodium elegans* dying from damping-off disease in the distance-density experiment. Values represent totals summed for 4 replicates of a given treatment. The total number of seeds germinating is given above each bar. Shaded bars represent high density quadrats; unshaded bars represent low density quadrats

than under the tree (Fig. 2). Distance was a significant factor in explaining levels of disease at Tree 1 ( $F_{2,18} = 37.4$ ,  $P < 0.001$ ) and Tree 2 ( $F_{2,9} = 6.8$ ,  $P < 0.025$ ); it was not significant at Tree 3 ( $F_{2,9} = 1.0$ , NS), the tree with the lowest proportion of seedlings dying from disease at all distances (Fig. 2).

Density was a second significant factor in explaining the level of disease at Tree 1 ( $F_{1,18} = 26.0$ ,  $P < 0.001$ ). Although no differences in disease level existed between high and low density treatments under the tree, the effect of density became more pronounced at greater distances (Fig. 2). There was a significant interaction between the distance and density variables ( $F_{2,18} = 6.8$ ,  $P < 0.01$ ).

Whether offspring of Tree 1 were dispersed around their parent or around another conspecific tree further influenced the proportion of seedlings dying from disease (Fig. 2). In comparing the high density treatment at each of three distances, there were significant differences among trees in the level of disease ( $F_{2,27} = 46.9$ ,  $P < 0.001$ ). Distance remained a significant factor in this analysis ( $F_{2,27} = 8.1$ ,  $P < 0.005$ ), but there was no significant interaction between the tree and distance variables ( $F_{4,27} = 0.7$ , NS). Offspring of Tree 1 experienced more disease at all distances around their own parent (Tree 1) than around either Tree 2 or Tree 3 (Fig. 2). Seedlings around Tree 3 had less disease than those around Tree 2 (Fig. 2), a pattern consistent with disease levels observed in 1980.

### Enclosure experiment: disease symptoms

Seedlings of 16 of the 18 species died from disease, but in widely varying amounts (Table 2). Classic symptoms of damping-off due to fungal pathogens were apparent in 13 of the 16 species showing disease symptoms. These included stem necrosis at or near the soil line with advancement upward. In some species necrosis also arose at the petiole base of the cotyledon or on the stem at the cotyledon node; such necrosis led to the dropping of cotyledons. Many other species showed wilting leaves and lodging as a result of weakened stems. Two of the three species lacking stem necrosis at the soil line had necrotic patches on their cotyledons; this necrosis of *Cavanillesia platanifolia* and *Triplaris*

**Table 2.** The proportion (p) of seedlings of 18 tree species dying from disease under different conditions of light and density in a screened enclosure on Barro Colorado Island, Panama. Also shown are the number (N) of initial seedlings in each treatment and the mean dry weight of seed and basic wood density for each tree species

Species	High density				Low density				Light	Density	Seed weight (mg)	Wood density
	Sun		Shade		Sun		Shade					
	N	p	N	p	N	p	N	p				
<i>Platypodium elegans</i>	196	0.01	208	0.89	228	0.004	228	0.08	*	**	444	0.75
<i>Lafoensia punicifolia</i>	198	0	200	0.61	20	0	20	0.05	*	**	18.8	0.72
<i>Pseudobombax septenatum</i>	194	0.02	197	0.83	20	0	20	0.20	*	**	57.0	0.14
<i>Triplaris cumingiana</i>	200	0.01	199	0.20	20	0	20	0	*	**	44.5 <sup>a</sup>	0.56
<i>Luehea seemanii</i>	113	0.04	95	0.64	20	0	20	0.25	*	**	1.9	0.50
<i>Tabebuia rosea</i>	205	0.05	200	0.36	20	0.40	20	0.70	*	**	31.6	0.52
<i>Cordia alliodora</i>	157	0.01	157	0.09	20	0	20	0.05	*		2.9 <sup>a</sup>	0.44
<i>Aspidosperma cruenata</i>	200	0	198	0.01	20	0	20	0			686	0.71
<i>Terminalia oblonga</i>	43	0	44	0.11	20	0	20	0.10	*		41.3 <sup>a</sup>	0.70
<i>Terminalia amazonica</i>	33	0.09	30	0.67	20	0	20	0.50	*		2.4 <sup>a</sup>	0.68
<i>Ochroma pyramidale</i>	13	0	12	1.00	20	0	20	0.95	*		5.6	0.13
<i>Cochlospermum vitifolium</i>	5	0.20	5	1.00	20	0.20	20	0.95	*		20.2	0.30
<i>Ceiba pentandra</i>	27	0.04	21	0.29	20	0	20	0.25	*		45.0	0.24
<i>Cavanillesia platanifolia</i>					20	0.20	20	0.95	***		412	0.12
<i>Dalbergia retusa</i>					20	0	20	0.40	***		121	0.95
<i>Lonchocarpus pentaphyllus</i>					20	0	20	0.20	***		51	0.75
<i>Bombacopsis sessilis</i>					20	0	20	0			221	0.42
<i>Myroxylon balsamum</i>					20	0	20	0			218	0.78

\* Light-disease interaction significant ( $P < 0.05$ ) from three-dimensional contingency table analysis

\*\* Density-disease interaction significant ( $P < 0.05$ ) from three-dimensional contingency table analysis (Fienberg 1978)

\*\*\* Light-disease interaction significant ( $P < 0.05$ ) from two-dimensional contingency table analysis; density-disease interaction was not tested for in these species due to insufficient seed source

<sup>a</sup> Weight includes fused ovary wall

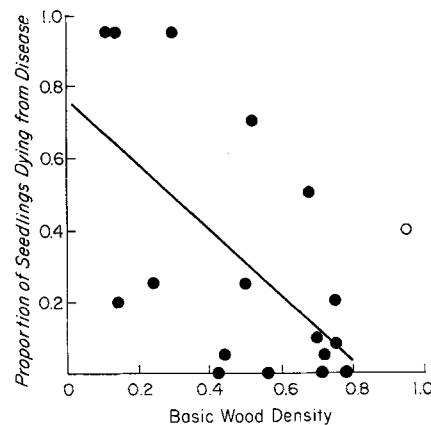
*cumingiana* advanced to the petiole and terminal meristem; their cotyledons fell off and their meristems were destroyed. *Tabebuia rosea* first displayed necrosis near the terminal meristem and its leaves blackened. Only *Aspidosperma cruenata* showed any resprouting from previously diseased stems. No isolations of pure cultures of the disease-causing organisms were made.

#### Density-light experiment

Light conditions and/or the associated environmental factors greatly affected disease activity. For each species with diseased seedlings, the proportion of seedlings dying from disease was greater in shade than in the sun (Table 2). Fifteen of the 16 species with disease-caused mortality showed a significant effect of light on disease (Table 2).

Density also affected the level of disease activity, but was less important than the light variable. All species with a high density treatment showed some mortality from disease; 14 of 18 species showed disease in the low density treatment (Table 2). For each species with diseased seedlings, the proportion of seedlings dying from disease was greater in the high density treatment. Of the first eight species listed in Table 2, which had high density replicates and thus a large sample size, six showed a significant effect of density on disease. None of the species without high density replicates showed a significant effect of density on disease; these species also tended to have a lower density in their high density treatment than the six species showing a significant effect of density on disease (Table 2).

To summarize the overall relationships including all species, comparisons were made between environmental condi-



**Fig. 3.** Regression of the proportion of seedlings dying from disease on basic wood density ( $r = -0.61$ ,  $n = 17$ ,  $P < 0.01$ ). Data are from the shade-low density treatment of the enclosure experiment. The analysis excluded *Dalbergia retusa* (open circle), because the experimental soil was obtained from beneath it. Including *Dalbergia retusa* weakens the correlation ( $r = -0.52$ ,  $n = 18$ ,  $0.025 < P < 0.05$ ). Separate analyses using the transformation  $\arcsin \sqrt{p}$  gave the same results

tions on the proportion of seedlings dying from disease. Such mortality was greater in shade than in sun under both low ( $n = 18$ ,  $P < 0.001$ ) and high ( $n = 13$ ,  $P < 0.001$ ) densities. In the shade it was significantly greater at high than low densities ( $n = 13$ ,  $P < 0.005$ ). It was also greater at higher densities in the sun, but the effect was not significant at conventional levels ( $n = 13$ ,  $P < 0.07$ ) (all tests used Binomial Exact Test of Paired-Sign Comparisons).

Dry weight of seed and the proportion of seedlings dying from disease (shade-low density treatment) were not significantly correlated among the species ( $r = -0.17$ ,  $n = 17$ , NS). Likewise dry weight of seed was independent of basic wood density ( $r = 0.16$ ,  $n = 17$ , NS). Wood density is one indicator of successional status. Rapid-growing, colonizing species, in general, have low values of wood density relative to shade-tolerant, slow-growing species (Budowski 1965, Whitmore 1975, Williamson 1975). The proportion of seedlings dying from disease showed a significant decline as wood density increased among the species (Fig. 3). Fast-growing, colonizing species were more vulnerable to seedling disease than slow-growing, persistent species.

## Discussion

The field experiment demonstrated that both an increase in dispersal distance and a decrease in seedling density lowered damping-off disease of *Platypodium* seedlings; density increased in importance at greater distances. The study also showed that the particular conspecific tree around which a parent's seeds were dispersed affected disease levels. The multi-species experiment indicated that environmental conditions comparable to light-gaps greatly lowered disease activity. It also confirmed that high seedling density increased disease levels, especially under shaded conditions. Adult wood density of a species, but not seed weight, was negatively correlated with the vulnerability of its seedlings to pathogens.

These results will be discussed in the context of seed dispersal by a parent tree. Dispersal affects three factors important in the escape of a tree's offspring from pathogens. First, it increases the distance between offspring and parent; a related outcome is that some offspring may occur under other conspecific or non-conspecific parents. Second, it lowers seedling density, as the offspring are distributed over a greater area. Third, because of the greater area, it increases the probability that some offspring land in light-gaps.

An increase in dispersal distance lowered, but did not eliminate, disease of *Platypodium* seedlings; pathogens were active even in low density quadrats 50 m from the parent tree. An interpretation of this distance effect requires knowledge of the pattern of inoculum of the pathogens. Generally, dispersal is viewed as a means to remove offspring from a focus of inoculum, e.g., from an infected parent or adjacent saplings, or from the soil where seedlings were infected in previous seasons. In this study, primary infection by the pathogen is likely to have first occurred from soil inoculum because fungal pathogens causing damping-off are known to be soil-borne (Garrett 1970, Weber 1973). Later, secondary infections may arise from previously infected seedlings (Burdon and Chilvers 1975). In general, species of damping-off fungi are low in their host specificity (Walker 1969) and can exist in the soil as saprophytes or as resting spores during low availability of living host tissue (Walker 1969, Garrett 1970).

Such pathogens are therefore potentially ubiquitous and seedlings may encounter them at any dispersal distance. Evidence of this ubiquity comes from the enclosure experiment. Seedlings of most species were infected by pathogens, presumably from the common soil used to simulate the soil found at some distance away from the parent.

Despite their widespread nature, these pathogens are not likely to be uniform in their distribution and/or activity. A wide range of disease levels was observed in 1982 among replicates of the same density at a given distance from the same tree and among seedlings at the same density and distance, but at different trees. In 1980 pathogens were much more active under than away from *Platypodium* trees (Augsburger 1983 a).

Variation in disease levels may, in part, be due to density effects of previous years affecting current inoculum levels. This experiment demonstrated that density significantly affected disease levels. A density effect on damping-off disease is frequently observed in nurseries (Gibson 1965, Walker 1969, Cowling 1978). Secondary infection from previously infected seedlings accounts for the spread of the disease in high density, monospecific plantings (Burdon and Chilvers 1975). High seedling density may increase infection levels by increasing humidity (Garrett 1970) or by slowing growth rates and delaying cell-wall thickening (Walker 1969).

At greater distances from the parent, *Platypodium* seedlings encounter fewer conspecific seedlings; they also experience a lower mean seedling density of all species. This lower density of non-conspecifics arises because: 1) species vary in their seedling phenology, 2) adult trees dispersing seeds are not uniformly spaced in the forest, 3) species vary in the number of seeds dispersed and/or seedlings surviving, and in their spatial distribution. In a related field study of nine of the species involved in the enclosure study, seedling densities of conspecifics ranged among the species from 0.2/m<sup>2</sup> to 47/m<sup>2</sup> near the parent and from 0.05/m<sup>2</sup> to 5.6/m<sup>2</sup> at 30–40 m from the parent (Augsburger 1983 b).

Relative to other species, *Platypodium elegans* has large fruit crops, although in alternate years, and high seedling densities under and near the parent. Inoculum levels may be higher under a *Platypodium* tree than under or away from canopies of most non-conspecific adults. If so, historical factors may account for the distance results; past seedling densities may be lower, on average, at most distances away from a *Platypodium* parent. Therefore, past pathogen activity and present inoculum levels are lower away from a *Platypodium* parent and dispersal results in partial escape from pathogens by this species. In a non-experimental study, other tree species, with lower natural seedling densities than *Platypodium*, suffered less density-dependent mortality by pathogens than *Platypodium* seedlings (Augsburger 1983 b). For those species, dispersal to high density areas near and under *Platypodium* trees may be disadvantageous.

Finally, a distance-density interaction was observed in the experiment. The effect of density was especially pronounced at great distances. Density had no effect on pathogen-caused mortality under the tree; only at greater distances did a lower density enhance seedling escape from pathogens. This distance-density interaction may also result from patterns of infection in previous years. High density of seedlings near the parent in past years resulted in high inoculum for subsequent years, thus having a large effect on primary infection, even at low experimental seedling densities under the parent. In contrast, low seedling densities far from the parent in past years led to low inoculum levels and hence low primary infection at the current low density; only when secondary infection from adjacent infected seedlings was possible at high experimental densities did disease levels rise far from the parent. In summary,

density effects of the past set up a gradient of inoculum, high near the parent and low away from the parent. This gradient greatly influenced the distance effect and the distance-density interaction observed in the experiment.

The field results indicated differences among three sites in the amount of disease experienced by one tree's seedlings. Offspring of Tree 1 incurred less disease around other conspecific trees than around their parent. These results have two possible explanations. First, pathogen-host coevolution may be somewhat parent-specific, and hence site-specific. Alternatively, the variation may be due to differences among the three trees in environmental conditions and/or past history of levels of infection by these pathogens. A more complete experimental design having seeds from each tree around both itself and all other trees would help to separate the two explanations. Such a reciprocal design was not possible in 1982 as only Tree 1 bore a sufficiently large fruit crop. The possibility of site-specific coevolution has important implications for a parent tree existing in a clump of conspecific trees. Under these conditions dispersal may not greatly reduce the seedling density a parent's offspring encounters. Nevertheless, dispersal may be advantageous in the escape of a site-specific pathogen.

Dispersal also increases the likelihood that some seedlings encounter seedlings of other species. The effect of mixed-species plantings on seedling disease was not included in this study. Species vary in the vulnerability of their seedlings to a specific pathogen (Burdon and Chilvers 1976). The seedling density of mixed species at distances away from parent trees is seldom equal to the high monospecific density under and near parent trees (personal observation). Also, because of phenological differences among species, seedlings of all species are unlikely to be at the same stage when pathogen invasion is possible; cell wall thickening and lignification within an aging seedling increase its resistance to pathogens (Populer 1969, Walker 1969). Thus the effect of density on disease is likely to be more pronounced in intraspecific than interspecific stands of seedlings.

Seedlings, on average, experience more light away from the parent than under the parent. Although some seedlings away from the parent are in the shade of non-parent canopies, others benefit from slight irregularities in canopy structure increasing the light level. Dispersal also increases the likelihood that some of a parent's seedlings are in light-gaps where disease activity is lower. The enclosure experiment indicated that light is potentially important for almost all of the 18 study species in the escape of their seedlings from pathogens. This advantage of dispersal may be particularly important for those species, e.g., *Ochroma pyramidale*, *Cochlospermum vitifolium*, and *Cavanillesia platanifolia*, showing a high vulnerability to pathogens under shaded conditions. Whether the dependence of these species on large sunny, disturbed areas for establishment (personal observation) results from their vulnerability to pathogens, their photosynthetic requirements *per se*, or both remains to be determined experimentally. Correlations have been shown between falling levels of sugars and increased infectivity in shaded plants (Baker 1946, Horsfall and Dimond 1957 in Grime 1966). It is possible such species could persist in shade, provided they were in a pathogen-free environment (cf. Vaartaja 1962).

Recent field studies verified the importance of light in lowering disease-caused mortality of seedlings (Augsburger

1983a, b). In that study of nine species, all suffered less disease-caused mortality in light-gaps than in the shaded understory. Fungal pathogens of seedlings are known to be favored by low sunlight, high humidity, and low temperatures (Weber 1973, Rotem 1978), conditions of the shaded understory of tropical forests (Bazzaz and Pickett 1980, Garwood 1983). Although light was the designated variable in the enclosure experiment, associated microclimatic factors, such as higher humidity and lower temperatures in the shade treatment, likely contributed to the results.

The multi-species enclosure experiment showed that seedlings of nearly all species were vulnerable to disease, although to varying degrees. The species ranged from fast-growing, colonizing species to slow-growing, shade-tolerant species (Augsburger 1983d). Their seed weights ranged from 1.9 to 686 mg. However, seed weight, indicative of initial energy reserves, was not a good predictor of seedling vulnerability to disease among the 18 species. The same conclusion was reached in the field study of nine of the same species (Augsburger 1983b). Instead, adult wood density, an indicator of past growth rates and successional status, was negatively correlated with vulnerability of seedlings to pathogens. Wood density of adults is not necessarily correlated with wood density of juveniles (B. Williamson, personal communication). Therefore, the above correlation does not indicate that higher wood density itself lowers invasion by pathogens. Rather it appears that fast-growing species, whose seedlings require light-gaps, lack strong resistance to seedling pathogens, relative to species able to tolerate shade as seedlings. Their shade tolerance must include adequate defense against seedling pathogens that are much more active under shaded conditions.

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