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## THINNING REDUCES THE EFFECT OF RUST INFECTION ON JEWELWEED (*IMPATIENS CAPENSIS*)<sup>1</sup>

CURTIS M. LIVELY, STEVEN G. JOHNSON,<sup>2</sup> LYNDA F. DELPH, AND KEITH CLAY  
*Department of Biology, Indiana University, Bloomington, Indiana 47405 USA*

**Abstract.** We examined the relationship between plant density and the probability of infection by a rust (*Puccinia recondita*) in a natural population of jewelweed (*Impatiens capensis*). We also reduced plant density in field manipulations to determine whether there is any synergism between intraspecific competition and infection by the rust. The results showed no relationship between plant density and the probability of infection. However, we found that the effect of infection depends on plant density. Uninfected plants grew significantly faster than infected plants under natural conditions of high density, but infection had no significant effect on growth where density was experimentally reduced. Hence, the effect of disease on jewelweed depends on the plant's competitive environment. Such interactions between competition and disease are likely to be important to the population biology and breeding-system evolution of plants.

**Key words:** conditional outcome; disease; intraspecific competition; *Impatiens capensis*; *Puccinia recondita*.

### INTRODUCTION

Depending upon environmental conditions, the effect of infection by parasites can range from detrimental to beneficial (Thompson 1988, Cushman and Whitham 1989, Michalakis et al. 1992, Thompson and Pellmyr 1992, Clay et al. 1993, Bronstein 1994). This variation in outcome can in turn affect host population dynamics (e.g., Hudson and Dobson 1991), the spatial and temporal distribution of host resistance genes (Burdon 1987), and selection on parasite virulence (Anderson and May 1982, Ewald 1983, May and Anderson 1983). Host density may be a particularly important part of the host environment. A recent model by Hamilton et al. (1990) showed that an outcrossing mating system was favored in competitive environments where the most infected individuals failed to reproduce due to an inability to compete for resources, i.e., where host fitness depended on an interplay between competition and parasitism. In the present study of a natural population of jewelweed (*Impatiens capensis*), we examined (1) the relationship between plant density and the probability of infection, and (2) plant density and the effect of infection on plant growth. We found that the probability of infection is density independent, but the effect of rust infection on plant growth depends heavily on host density.

#### *Natural history of the plant-rust interaction*

Jewelweed is a common annual plant of wetlands and forests throughout eastern North America. Plants

with late germination or slow growth are often suppressed by faster growing plants, which have longer life spans, higher fecundities, and produce proportionally more chasmogamous seeds (Waller 1980, Schmitt et al. 1987a, b). Hence, growth rate is an important component of reproductive success. *Puccinia recondita* is a heteroecious (two hosts) rust that infects jewelweed and other species in the families Balsaminaceae, Boraginaceae, Hydrophyllaceae, and Ranunculaceae (Farr et al. 1989). The rust infects jewelweed in the early spring and summer, when the teliospores produced on primary hosts (various cool-season grasses) come in contact with young jewelweed plants. The resulting lesions, which range from 3 to 20 mm in diameter, can number from one to hundreds per plant. Aeciospores produced on jewelweed then re-infect the grasses that serve as the primary host to the rust. Although the life cycle of *P. recondita* has not been studied in detail, we assume by analogy with similar rusts that there is no direct transfer of infection between jewelweed plants (see Cummins 1971).

### MATERIALS AND METHODS

#### *Plant density and infection prevalence*

To examine the relationship between plant density and the prevalence of rust infection, samples of jewelweed were taken (on 5 May 1993) from 20 different quadrats of 0.1 m<sup>2</sup> at the Lake Griffey Nature Preserve 1 km north of Bloomington, Indiana. The quadrats were distributed to insure a range of plant densities, but were positioned without regard to the presence of infected plants. All plants within the quadrats were clipped at the base, and returned to Indiana University where they were counted and examined for lesions. Prevalence of infection was calculated as the percentage of infected plants per quadrat, and intensity of infection was cal-

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<sup>2</sup> Present address: Department of Biological Sciences, University of New Orleans, New Orleans, Louisiana 70148 USA.

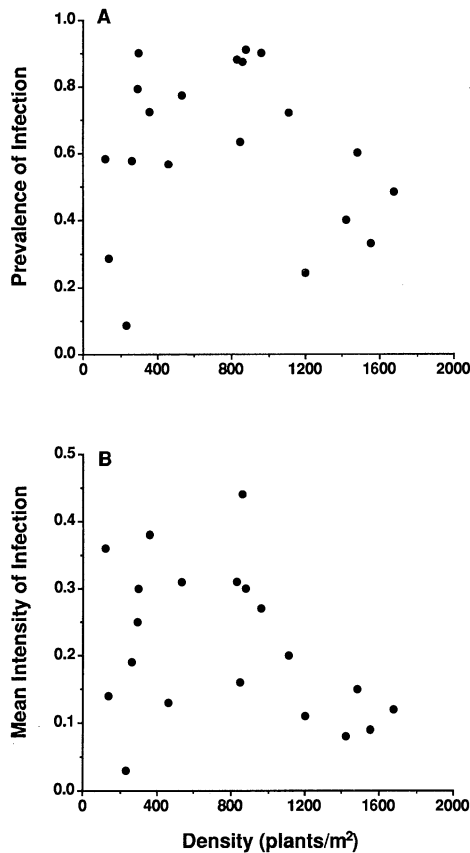


FIG. 1. The relationship between density (plants/m<sup>2</sup>) of *Impatiens capensis* and (A) prevalence of rust infection (frequency of infected individuals per plot), and (B) mean intensity of rust infection (average frequency of infected leaves per plant per plot). Neither of the relationships is statistically significant.

culated as the percentage of leaves on a plant with lesions, averaged over all plants in a quadrat.

#### Plant density and effect of infection

To determine the effect of plant density on the effect of infection, 40 plots were randomly chosen (also on 5 May) throughout the understory of silver maple (*Acer saccharinum*), birch (*Betula nigra*), and sycamore (*Platanus occidentalis*). In each plot, two plants of similar height were selected, one infected with rust and one uninfected; the plants were usually within 20 cm of each other. In half (20) of the plots, we clipped at the base and removed all conspecific individuals within a 15 cm radius of the focal plants (thinned treatment). Plant density was not altered in the other 20 plots (control treatment). The height and number of leaves were recorded for all plants, and the number of leaves bearing lesions was recorded for infected members of the pair. All plants were then individually marked for later identification. On 10 June (21 d after the start of the experiment), the heights of the plants were again mea-

sured. The study site was flooded on 14 June, and all experimental plants were lost.

A second experiment of the same design was initiated on 26 May. Thirty thinned and 30 control plots were established as above, and the heights of all plants were measured 15 d later on 10 June, 4 d before they were lost in the same flood. Finally, we established a third experiment in a site that was not flooded. On 26 June, 20 thinned and 20 control plots were established, and the height of experimental plants was recorded 24 d later on 20 July.

The results of the plant-density manipulations were analyzed by a two-way ANOVA. The dependent variable was calculated as the difference in growth rates (in millimetres per day) between paired uninfected and infected plants in the same plot. Analysis of covariance was used to determine if the growth rate of infected plants varied as a function of the intensity of infection (measured by the percentage of infected leaves) and treatment.

## RESULTS

### Plant density and infection prevalence

Plant density ranged from 132 to 1848 plants/m<sup>2</sup> with a mean of  $853 \pm 125$  plants/m<sup>2</sup> (all means are given  $\pm 1$  SE). A total of  $61 \pm 5\%$  of the plants was infected, and the mean intensity was  $21.6 \pm 3\%$ . Plant density was not significantly correlated with the prevalence of infection ( $r = -0.11$ ;  $n = 20$ ;  $P > 0.5$ ; Fig. 1A), or with the intensity of infection ( $r = -0.35$ ;  $n = 20$ ;  $P = 0.13$ ; Fig. 1B), in contrast with other published studies (Burdon and Chilvers 1982, Alexander 1984, Burdon 1987). However, a lack of correlation between density and disease is perhaps not surprising here, given

TABLE 1. ANOVA summary table for the effects of plant density (thinned vs. controls) and time of experiment (Experiment 1: 5 May–10 June; Experiment 2: 26 May–10 June; Experiment 3: 26 June–20 July) on growth/day differences (in mm) between uninfected and infected plants (*Impatiens capensis*).\*

Source of variation	df	ss	F	P
Density	1	83.21	15.26	<0.001
Experiment	2	16.89	1.93	0.152
Interaction	2	1.67	0.15	0.858
Error	83	452.53		
Linear contrasts				
Density in Experiment 1	1	31.2	5.68	0.019
Density in Experiment 2	1	27.9	5.09	0.027
Density in Experiment 3	1	27.0	4.93	0.029

\* Density is a fixed factor, and Experiment is a random factor in this mixed-model two-way analysis (see Sokal and Rohlf 1981). The Density effect in the table is tested over the MS error term, because the Interaction is nonsignificant. (The Density effect tested over the Interaction MS is nonetheless significant [ $F_{1,2} = 99.74$ ;  $P = 0.01$ ].) The linear contrasts give the effect of Density within each Experiment, and are less sensitive in general to the effects of variance due to interactions (see McKone and Lively 1993).

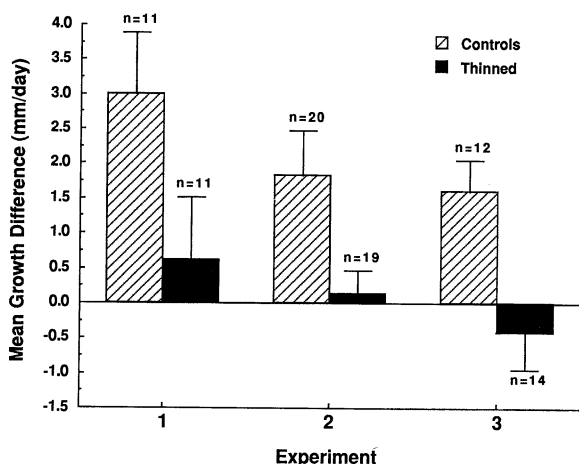


FIG. 2. Mean ( $\pm 1$  SE) difference in growth rate (mm/day) between paired uninfected and infected individuals of *Impatiens capensis* growing under different densities, for the three experiments (Experiment 1: 6 May–10 June; Experiment 2: 26 May–10 June; Experiment 3: 26 June–20 July). Numbers refer to sample size for each group. Standard error bars of the thinned plots overlap zero.

that the rust is heteroecious with no direct transmission among plants.

#### Plant density and effect of infection

Plant density had a significant effect on the growth difference between infected and uninfected plants in the field study (Table 1). In all three experiments, infection had a detrimental effect on plant growth only in the high-density control plots. For example, the average difference in growth between infected and uninfected plants was nearly 5 times greater in control plots than thinned plots in the first experiment (Fig. 2). Growth rate differential was not significantly different from zero in thinned plots (Fig. 2). The differences among experiments were not statistically significant (Table 1).

In the first experiment, we observed a significant positive relationship in the control plots between growth differential and the percentage of infected leaves [ $\beta$  (slope) =  $0.18 \pm 0.08$ ,  $P = 0.049$ ]. The same relationship, however, was not significant in the thinned (experimental) plots ( $\beta = -0.12 \pm 0.11$ ,  $P = 0.32$ ). The intensity of infection was correlated with the difference in growth between infected and uninfected plants only under high density (Fig. 3). However, in the second and third experiments, which began later when plants were larger, there was no significant relationship between growth differential and intensity of infection in either control or thinned treatments (Experiment 2, controls:  $P = 0.86$ , thinned:  $P = 0.13$ ; Experiment 3, controls:  $P = 0.80$ , thinned:  $P = 0.68$ ). Mitchell-Olds (1987) found that 56% of the variation in final plant size of *I. capensis* resulted from early size differences. Similarly, small differences in growth

caused by rust infection early in life appear to be magnified over the growing season.

#### DISCUSSION

Infection by rust (*Puccinia recondita*) had a deleterious effect on growth in *Impatiens capensis* under the high-density conditions of our unmanipulated control plots. No such effect was observed in thinned plots in which plant density was experimentally reduced. These results indicate that the effect of infection is conditional on the host's competitive environment. Thinned plots may have had higher light, lower humidity, greater air circulation, and/or greater moisture. Decomposition of the roots from plants clipped in the process of experimental thinning may also have altered nutrient levels in the soil. Regardless of the mechanism, infected and uninfected plants responded differently to experimental thinning. As jewelweed typically occurs in high-density populations (Schmitt et al. 1987a, Waller 1980), rust infection will be deleterious in most circumstances, and it would appear that the effect of infection is determined in part by the local competitive environment.

Paul and Ayres (1986) found, in contrast, that the growth of infected groundsel (*Senecio vulgaris*) in the greenhouse was inhibited over a wide range of plant densities, and that there was no interaction between host density and infection. Hence the results of our field experiments may not be generally applicable. Nonetheless, Burdon et al. (1984) showed (in a greenhouse study) that infected *Chondrilla juncea* had slower growth than uninfected resistant genotypes, which resulted in plant-size hierarchies. It is these size hierarchies that would be expected to be important in high-density field situations, especially if fast-growing uninfected plants can further suppress the slow-growing infected individuals. This kind of synergism be-

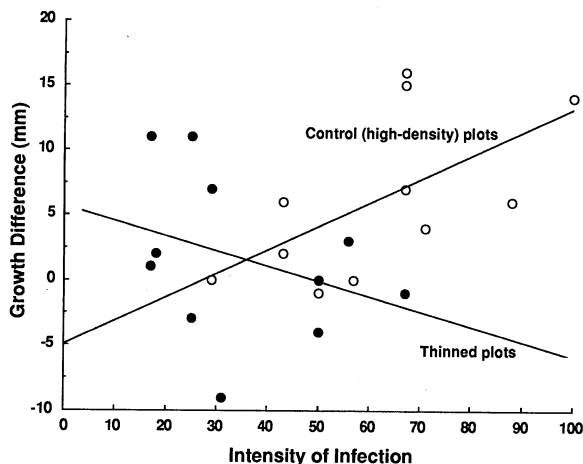


FIG. 3. Relationship between intensity of infection and differences in growth between paired uninfected and infected *Impatiens capensis* growing in thinned plots (●) and in high-density control plots (○) in Experiment 1.

tween infection and competition appears to be the simplest explanation for our results on jewelweed. Infected plants seem less able to compete for resources in dense stands, and become suppressed by faster growing, uninfected plants. Suppression of slow-growing and late-germinating plants is well known from the field studies of jewelweed by Schmitt et al. (1987a, b).

Interactions between competition and infection, if general, also bear directly on the current controversy regarding the evolutionary stability of cross fertilization. It is well known that diseases could pose a potent selective force against asexual reproduction, as they would tend to select against common host genotypes (Jaenike 1978, Hamilton 1980, Price and Waser 1982, Hamilton et al. 1990, Howard and Lively 1994). Nonetheless, a caveat by May and Anderson (1983) has been overlooked; they show that diseases must have very severe effects on host fitness in order for outcrossed sexual individuals to have an advantage that would overcome the twofold reproductive advantage of asexual individuals. Other than castrating species of trematodes and fungi, it would seem that such severe effects of parasites per se are not to be expected. However, if the severity of disease in field conditions is greatly increased by competition, then the parasite hypothesis for sex becomes more tenable. Hamilton et al. (1990) showed in computer simulations that sex is evolutionarily stable when the most infected individuals fail to contribute to the next generation due to an inability to compete for resources. The effects of infection may otherwise be minor. It is not known for any species whether such a scenario is reasonable in nature, but we have shown that disease and competition do interact in natural populations of jewelweed. Additional field studies of the interacting effects of disease and competition seem warranted.

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#### LITERATURE CITED

- Anderson, R. M., and R. M. May. 1982. Coevolution of hosts and parasites. *Parasitology* **85**:411–426.
- Alexander, H. M. 1984. Spatial patterns of disease induced by *Fusarium moniliforme* var. *subglutinans* in a population of *Plantago lanceolata*. *Oecologia* **62**:141–143.
- Bronstein, J. L. 1994. Conditional outcomes in mutualistic interactions. *Trends in Ecology and Evolution* **9**:214–217.
- Burdon, J. J. 1987. Diseases and plant population biology. Cambridge University Press, Cambridge, England.
- Burdon, J. J., and G. A. Chilvers. 1982. Host density as a factor in plant disease ecology. *Annual Review of Phytopathology* **20**:143–166.
- Burdon, J. J., R. H. Groves, P. E. Kaye, and S. S. Speer. 1984. Competition in mixtures of susceptible and resistant genotypes of *Chondrilla juncea* differentially infected with rust. *Oecologia* **64**:199–203.
- Clay, K., S. Marks, and G. P. Cheplick. 1993. Effects of insect herbivory and fungal endophyte infection on competitive interactions among grasses. *Ecology* **74**:1767–1777.
- Cummins, G. B. 1971. The rust fungi of cereals, grasses and bamboos. Springer-Verlag, New York, New York, USA.
- Cushman, J. H., and T. G. Whitham. 1989. Conditional mutualism in a membracid-ant association: temporal, age-specific, and density-dependent effects. *Ecology* **70**:1040–1047.
- Ewald, P. W. 1983. Host-parasite relations, vectors, and the evolution of disease severity. *Annual Reviews of Ecology and Systematics* **14**:465–485.
- Farr, D. F., G. F. Bills, G. P. Chamuris, and A. Y. Rossman. 1989. Fungi on plants and plant products in the United States. American Phytopathological Society Press, St. Paul, Minnesota, USA.
- Hamilton, W. D. 1980. Sex versus non-sex versus parasite. *Oikos* **35**:282–290.
- Hamilton, W. D., R. Axelrod, and R. Tanese. 1990. Sexual reproduction as an adaptation to resist parasites (a review). *Proceedings of the National Academy of Sciences (USA)* **87**:3566–3573.
- Howard, R. S., and C. M. Lively. 1994. Parasitism, mutation accumulation and the maintenance of sex. *Nature* **367**:554–557.
- Hudson, P. J., and A. P. Dobson. 1991. The direct and indirect effects of the caecal nematode *Trichostrongylus tenuis* on red grouse. Pages 209–253 in J. E. Loye and M. Zuk, editors. Bird-parasite interactions: ecology, evolution, and behaviour. Oxford University Press, Oxford, England.
- Jaenike, J. 1978. An hypothesis to account for the maintenance of sex within populations. *Evolutionary Theory* **3**:191–194.
- May, R. M., and R. M. Anderson. 1983. Epidemiology and genetics in the coevolution of parasites and hosts. *Proceedings of the Royal Society London*, **B 219**:281–313.
- McKone, M. J., and C. M. Lively. 1993. Statistical analysis of experiments conducted at multiple sites. *Oikos* **67**:184–186.
- Michalakis Y., I. Olivieri, F. Renaud, and M. Raymond. 1992. Pleiotropic action of parasites: how to be good for the host. *Trends in Ecology and Evolution* **7**:59–62.
- Mitchell-Olds, T. 1987. Analysis of local variation in plant size. *Ecology* **68**:82–87.
- Paul, N. D., and P. G. Ayres. 1986. Interference between healthy and rusted groundsel (*Senecio vulgaris* L.) within mixed populations of different densities and proportions. *New Phytologist* **104**:257–269.
- Price, M. V., and N. M. Waser. 1982. Population structure, frequency-dependent selection, and the maintenance of sexual reproduction. *Evolution* **36**:35–43.
- Schmitt, J., J. Eccleston, and D. W. Ehrhardt. 1987a. Density-dependent flowering phenology, outcrossing, and reproduction in *Impatiens capensis*. *Oecologia* **72**:341–347.
- Schmitt, J., J. Eccleston, and D. W. Ehrhardt. 1987b. Dominance and suppression, size-dependent growth and self-thinning in a natural *Impatiens capensis* population. *Journal of Ecology* **75**:651–665.
- Sokal, R. R., and F. J. Rohlf. 1981. Biometry. W. H. Freeman, New York, New York, USA.
- Thompson, J. N. 1988. Variation in interspecific interactions. *Annual Review of Ecology and Systematics* **19**:65–87.
- Thompson, J. N., and O. Pellmyr. 1992. Mutualism with pollinating seed parasites amid co-pollinators: constraints on specialization. *Ecology* **73**:1780–1791.
- Waller, D. M. 1980. Environmental determinants of outcrossing in *Impatiens capensis* (Balsaminaceae). *Evolution* **34**:747–761.