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INTERACTIONS OF INTRASPECIFIC, INTERSPECIFIC, AND APPARENT COMPETITION WITH HOST–PATHOGEN POPULATION DYNAMICS[1]

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Abstract. I investigated the interactions of intra- and interspecific competition with parasitism for populations of two terrestrial isopods, *Porcellio scaber* and *Porcellio laevis*, infected with a lethal virus. In field experiments, I varied densities of one or both isopod species in enclosures in which food and virus presence were also manipulated. I estimated the effects of intra- and interspecific competition, additional food, and virus infection on both the survival of *P. scaber* and the prevalence of the virus. In uninfected populations, intraspecific and interspecific competition negatively affected isopod survival, although food resources had no effect. In infected treatments, interspecific competition produced higher levels of virus prevalence than did intraspecific competition. Virus infection and reduced food interacted to produce higher overall mortality in interspecific competition treatments than in intraspecific competition treatments. The lack of significant effect of food on competition suggests that interference interactions rather than resource competition predominated. This mechanism is supported by a laboratory study in which total food availability was controlled. These results indicate that competition and parasitism interacted to produce the observed mortality patterns, and that ignoring mortality due to the virus would produce apparent interspecific competition. The data suggest that the impact of *P. laevis* on *P. scaber* involves both direct and indirect effects.

Key words: apparent competition; host–parasite; host–pathogen; interference competition; interspecific competition; intraguild predation; intraspecific competition; parasitism; population dynamics; Porcellio *spp.; terrestrial isopods; viruses.*

INTRODUCTION

The influence of competition on population dynamics and community structure has been a major focus in ecology (reviewed in Schoener 1983, Connell 1983). However, indirect effects greatly complicate our ability to detect competition in nature. Apparent competition has emerged as a potentially important indirect effect of predation (Williamson 1957, Holt 1977, 1984, Jeffries and Lawton 1984, Schmitt 1987). Differential predation may create apparent competition by reducing the abundance of the more preferred prey species, a result that may be mistaken for a negative competitive effect of the less preferred prey species on the more preferred species. The possibility that apparent competition may be common necessitates considering other forces that may produce similar effects.

That apparent competition may occur in host–parasite systems has been demonstrated theoretically (Holt and Pickering 1985), but not empirically. Although interest in the effects of parasites and pathogens on the dynamics of host populations in nature is rapidly growing (Fenner and Ratcliffe 1965, Holmes and Bethel 1972, Price 1980, Moore 1983, Price et al. 1986, van Riper et al. 1986, Burdon 1989, Dwyer 1991, E. D. Grosholz, *unpublished manuscript*), few attempts have been made to examine the interaction of parasitism with other biotic forces such as competition. Of these, most have been laboratory studies concerned with the effects of a parasite on the outcome of competition between host species (Park 1948, Steinwascher 1979), and none have explicitly addressed the issue of apparent competition. Studies of competition and parasitism, with plants and plant pathogens (Burdon 1989, Burdon et al. 1989) as well as insect parasitoids and their hosts (Settle and Wilson 1990), have recently attracted interest. But to date, no studies have simultaneously manipulated sympatric host species and a common pathogen in nature.

Using field and laboratory experiments, I examined the effects of a lethal, directly transmitted virus on the outcome of competition between two terrestrial isopods, *Porcellio scaber* and *Porcellio laevis*. I also investigated the influence of isopod competition on the probability of being parasitized. In the field experiment, I experimentally investigated the possibility of apparent competition due to parasitism by examining competitive interactions both with and without the pathogen, while partitioning the influence of interspecific and intraspecific competition on the observed patterns. In the laboratory experiment, I experimentally determined the interactions of parasitism and com-

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petition under conditions of controlled food availability.

Methods

Natural history

In central California, *Porcellio scaber* and *Porcellio laevis* are among the most common of the 25 species of terrestrial isopods (Miller 1938, Smith and Carlton 1980). The two species overlap widely in their distribution, and are commonly found in dense aggregations in excess of 2000 isopods/m^2 (E. D. Grosholz, *unpublished manuscript*). They are generalist scavengers on rotting plant, animal, and fungal material (Neuhauser and Hartenstein 1978), although they also consume living vegetation. Like many crustaceans, they often cannibalize their own offspring and injured or molting conspecifics. Both species share similar life histories and can live for at least 4 yr. They generally mature in 1 yr and commonly produce up to three directly developing broods each year during the spring and summer (Sutton et al. 1984). Both species have pan-global distributions heavily influenced by human activities.

Both *P. scaber* and *P. laevis* are infected by the same strain of the isopod iridescent virus (IIV) throughout the United States and Europe (Cole and Morris 1980, Federici 1980, Poinar et al. 1985). Previous work has established many details of the serology, pathology, and transmission of this large (130 nm), double-stranded DNA virus (Cole and Morris 1980, Federici 1980, Hess and Poinar 1985). The primary route of transmission occurs when healthy isopods consume infected isopod cadavers (Cole and Morris 1980, Federici 1980, E. D. Grosholz, *unpublished data*); there are no resistant or free-living stages (sensu Anderson and May 1981). Approximately 2 wk after infection, the isopods become bright purple-blue, due to large numbers of virions, which form light-refracting, paracrystalline arrays through most tissues (Cole and Morris 1980, Federici 1980). Death ensues in $\approx$3 wk once this blue stage is reached.

As determined by indirect enzyme-linked immunosorbent assay (ELISA), the blue color is a quantitative indicator of virus infection (Cole and Morris 1980, E. D. Grosholz *unpublished data*). Unapparent infections are rare ($\approx$1%, E. D. Grosholz, *unpublished data*). Since isopods may live for 3 wk with large amounts of virus (up to 1% of total body mass, Cole and Morris 1980), it is likely that the fitness costs of undetectable quantities of virus are small, although reduced reproduction may be possible.

There appears to be negligible vertical transmission via transovarial infection since visibly infected females kept in the laboratory abort their offspring (E. D. Grosholz, *unpublished data*). Transmission via parasitic nematodes has also been suggested (Hess and Poinar 1985), since in some habitats, infected isopods may often harbor endoparasitic nematodes infected with the virus; however, cause and effect remain unclear. At the present study site and other seasonally dry habitats in coastal California, parasitic nematodes are absent or extremely rare (E. D. Grosholz, *unpublished data*).

Field site

I conducted all field experiments at the University of California's Bodega Marine Laboratory, Sonoma County, California, from August through December 1989. The coastal prairie consists of a mixture of introduced and native grasses and forbs described in detail by Barbour et al. (1973). The vegetative structure is fairly simple, with the tallest plants being yellow lupine *Lupinus arboreus* and gold yarrow *Eriophyllum staechadifolium*. The site is on a bluff within 20 m of shore; soils are sandy and rather saline (Barbour et al. 1973).

At the Bodega reserve, *P. scaber* can be found at low to intermediate densities rarely exceeding 200–300 isopods/m^2 throughout the grasslands. However, *P. laevis* is not found on the Bodega reserve, although it can be found within 2 km of the study site. The absence of *P. laevis* from the Bodega reserve is not likely due to lack of introduction since it is widely distributed and readily dispersed by human activities.

Within the reserve, over a 3-yr period I have never observed the virus in isopod populations (E. D. Grosholz, *unpublished manuscript*), although infected isopods can be found within 2 km of the study site. The use of an uninfected field site allowed me to control the presence of the virus in the experimental design. Since the virus is as widely distributed as the isopods, it is unlikely that dispersal has limited establishment of the virus on the reserve. Isopods on the Bodega reserve are susceptible to the virus and it is possible that isopod densities are insufficient to maintain the virus endemically. In habitats where the virus is endemic, isopod densities generally exceed 2000 individuals/m^2, whereas at the Bodega reserve, they rarely exceed 200–300 individuals/m^2 and are often one-tenth of this (E. D. Grosholz, *unpublished manuscript*).

Design of field experiment

In a 6 × 8 grid I established 48 square plots each measuring 0.36 m^2, with each plot separated by at least 1 m from others in its row or column. Along the perimeter of each plot I dug a trench and placed a strip of sheet aluminum flashing 0.25 m wide, halfway into the soil, to surround each area. Preliminary experiments demonstrated that this was an effective barrier to isopod migration. In the middle of each area I placed a fiber mat 0.16 m^2, which I periodically dampened with a garden hose to provide a moist microhabitat.

Into each area I introduced combinations of *P. scaber* and *P. laevis* in one of three treatments (referred to as "species treatments"): (1) 50 *P. scaber*, (2) 100 *P. scaber*, or (3) 50 *P. scaber* and 50 *P. laevis*. These densities

were within the natural range at this site, based on counts from 10 additional fiber mats adjacent to the experimental area but with no aluminum flashing (mean = 348.8 isopods/m^2, range 178–518 isopods/m^2). This design allowed me to examine intraspecific competitive effects on *P. scaber* by comparing treatments (1) and (2), and interspecific effects on *P. scaber* by comparing treatments (2) and (3) (Underwood 1986).

I manipulated the presence or absence of the virus ("virus treatments') by introducing either infected conspecific cadavers or uninfected control cadavers. The virus controls were very effective; no infected isopods were observed in uninfected treatments. Every 2–3 wk I added infected or uninfected cadavers equal to 15–20% of the experimental populations. This infective dose is within the range experienced by isopods in naturally infected populations with virus prevalence of 15–20% (E. D. Grosholz, *unpublished manuscript*).

Finally, sliced carrots and potatoes, added or not, were a third factor ("food treatments"). Since food availability in nature is difficult to measure for generalist scavengers like isopods, food was added ad libitum at each census.

The overall design included 12 treatment combinations with each replicated four times in a balanced design. I divided the grid of 48 enclosures into four blocks of 12 enclosures and assigned one replicate of each treatment to each block. Within each block I randomized treatments, but did not subsequently examine block effects. Once a week I counted adult isopods and scored them as either healthy or infected. Since the experiment was conducted late in the reproductive season, I did not include the few recruits in the census.

Analysis of field experiment

I analyzed the effects of: (1) the density and species of isopods, (2) the presence or absence of the virus, and (3) the presence or absence of additional food on isopod survival and virus prevalence using ANOVA (SAS Institute 1988) with all main factors fixed. To determine whether competition was occurring in the absence of the virus, I analyzed survival in virus control treatments (uninfected cadavers added) using a two-way ANOVA with species and food as factors. This permitted determination of both intraspecific and interspecific competitive effects in the absence of the pathogen. To determine the effects of competition on virus prevalence, I analyzed virus prevalence in infected populations only (infected cadavers added) using a two-way ANOVA with species and food as factors. Control populations yield no information on virus prevalence. To analyze the effects of the viral pathogen on the outcome of competition, I analyzed survival in all treatment using a three-way analysis of variance with species, virus, and food as factors.

I performed univariate analysis of variance on isopod survival and virus prevalence with data from the final census. For each replicate, I calculated survival as the percentage of the starting population present on the final census date, and virus prevalence as the cumulative percentage of the starting population scored as infected throughout the experiment. All percentage data were arcsine transformed and met the requirements of homoscedasticity for ANOVA.

Since the univariate analysis used only data from the final sampling date, repeated-measures ANOVA was performed to ensure that the observed patterns were consistent over time. This analysis used all census dates and the same dependent variables as the univariate analysis, with tests designed to detect interactions of the main factors with time.

Laboratory environment and experimental design

I conducted a preliminary laboratory experiment from July to October 1988 at the University of California, Berkeley. Isopods were obtained locally (Tilden Regional Park, Berkeley, California; University of California Richmond Field Station, Richmond, California) from populations known to be uninfected for at least 3 yr (E. D. Grosholz, *unpublished data*). They were kept at room temperature in plastic boxes (13 × 18 × 7 cm) painted black to reduce light. The bottom of each box had a moistened layer of plaster of Paris and a damp, cellulose sponge to maintain high humidity, plus chalk to supplement calcium.

Into each container I placed one of three species treatments: (1) 20 *P. scaber,* (2) 20 *P. laevis,* or (3) 10 *P. scaber* and 10 *P. laevis.* For the first week of the experiment I added one of three food treatments to each container: (1) 10 uninfected cadavers, (2) 10 infected cadavers, or (3) 10 infected cadavers plus 0.4 g of decayed willow leaves. Thereafter, all containers received willow leaves ad libitum. Preliminary experiments showed isopods prefer decayed willow leaves over cadavers (E. D. Grosholz, *unpublished data*). I replicated each of the nine treatment combinations three times and randomly assigned each container a position in a 9 × 3 table-top array. For the first 4 wk I censused isopods every 7–10 d, and, thereafter, every 4–5 d. At each census, all adult isopods were scored as either healthy or infected.

Analysis of laboratory experiment

I analyzed the effects of species and food treatments on isopod survival and virus prevalence with ANOVA using SYSTAT (SYSTAT 1989) with main factors fixed. At each census, I counted all isopods and scored them as healthy or infected. In each replicate, I calculated final survival and virus prevalence as described above.

RESULTS

Field experiment

Interspecific and intraspecific competition.—In uninfected treatments, the two-way ANOVA showed a

TABLE 1. Analysis of variance for uninfected populations, examining the effects of species and food treatments on final isopod survival. Data were the arcsine-transformed proportions of isopods surviving since the start of the experiment.

Source of variation	df	SS	MS	F	P
Species	2	0.129	0.064	3.65	<.05
Food 1		0.030	0.030	1.71	NS
Species × Food	2	0.029	0.015	0.85	NS
Error	18	0.317	0.018		

significant effect of species treatment on final survival of *Porcellio scaber* (Table 1). There was no significant effect of food and no significant interaction, so I pooled data across food to show differences between species treatments (Fig. 1). Below (see *Discussion*), I discuss the lack of a significant effect of food in the presence of significant competition. Survival of *P. scaber* was highest in the treatment with only conspecifics at low density, whereas survival was lower in both the high-density treatments. Interestingly, there was no difference between treatments with only conspecifics vs. those with *P. laevis,* which implies that the impact of intraspecific competition on survival was equivalent to that of interspecific competition.

Effects of competition on parasitism.—In infected treatments the two-way ANOVA showed a significant effect of species treatment on total virus prevalence in *P. scaber* (Table 2). Again, there was no significant effect of food and no significant interaction, so I pooled data across food to show differences between species treatments (Fig. 2). Virus prevalence in *P. scaber* was higher in treatments with *P. laevis* than in treatments with only conspecifics at either high or low density. Prevalence was slightly higher in high-density *P. scaber* treatments relative to those at low-density, but this difference is not significant (Table 2).

Effects of parasitism on competition.—In both infected and uninfected treatments, the three-way analysis of variance showed a highly significant main effect of species treatments on overall survival and several significant interactions (Table 3). There was a significant interaction of species with food and virus with food as well as a three-way interaction. In uninfected populations in Fig. 3, additional food had no effect on overall survival. In infected populations, both in treatments with *P. laevis* and those with low-density *P. scaber,* the virus interacted with lack of added food to produce lower overall survival.

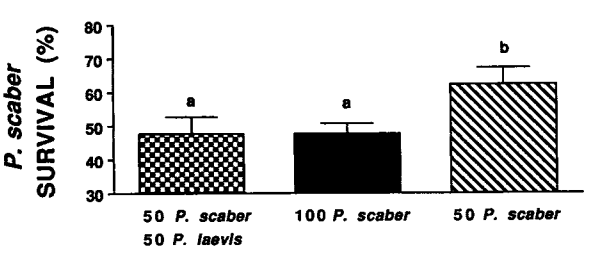

FIG. 1. Survival of *Porcellio scaber* in the field for the three species treatments in uninfected treatments only, pooled across food treatments. Final survival is the percentage of isopods at the start of the experiment that survived to the final date. Error bars represent 1 SE of the mean, and different letters indicate means that are significantly different (Student-Newman-Keuls, $n = 8$).

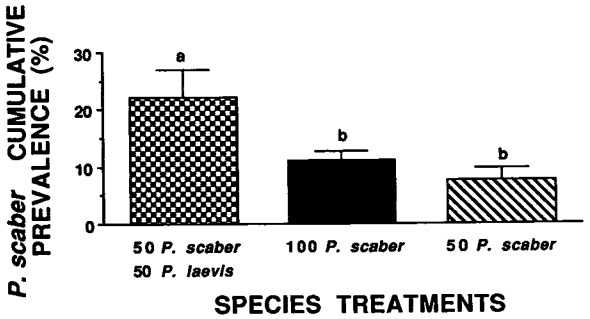

FIG. 2. Virus prevalence in *Porcellio scaber* in the field for the three species treatments in virus-infected treatments only, pooled across food treatments. Cumulative prevalence is the percentage of isopods at the start of the experiment that were scored as blue (i.e., virus infected) during the course of the experiment. Error bars represent 1 SE of the mean, and different letters indicate means that are significantly different (Student-Newman-Keuls, $n = 8$).

Repeated-measures analysis.—None of the factors or combinations of factors within replicates showed any interaction with time, and these results are not presented. The analysis confirmed that patterns analyzed at the end of the experiment were representative of the entire period of the experiment.

Laboratory experiment

In the uninfected-food treatment, survival for *P. scaber* was significantly lower in treatments with *P. laevis* ($t = 4.725, P < .01$). In Fig. 4, survival with conspecifics was twice that with *P. laevis.* Virus prevalence in *P. scaber* was higher in treatments with *P. laevis* (15% in treatments with both leaves and cadavers), although this difference was not significant ($n = 3$) (Fig. 5). Finally, there was no difference in prevalence between *P.*

TABLE 2. Analysis of variance for infected populations, examining the effects of species and food treatments on the total virus prevalence. Data were the arcsine-transformed proportion of isopods that were infected since the start of the experiment.

Source of variation	df	SS	MS	F	P
Species	2	0.221	0.111	4.98	<.05
Food	1	0.000	0.000	0.01	NS
Species × Food	2	0.009	0.004	0.19	NS
Error	18	0.400	0.022		

TABLE 3. Analysis of variance of uninfected and infected populations, examining the effects of species, food, and virus treatments on isopod survival. Data were the arcsine-transformed proportion of isopods surviving since the start of the experiment.

Source of variation	df	SS	MS	F	P
Species	2	0.263	0.131	10.99	<.05
Food	1	0.019	0.019	1.56	NS
Virus	1	0.009	0.009	0.81	NS
Species × Virus	2	0.001	0.000	0.05	NS
Species × Food	2	0.174	0.087	7.24	<.005
Virus × Food	1	0.147	0.147	12.22	<.005
Spec × Virus × Food	2	0.095	0.048	3.97	<.05
Error	36	0.432	0.012		

scaber and *P. laevis* in single-species treatments, suggesting a similar degree of susceptibility for the two species ($t = 0.004$, $P > .90$).

DISCUSSION

The results demonstrate the importance of examining the effects of both intraspecific and interspecific competition on parasitism, as well as the reciprocal effects of parasitism on competition. The importance of considering both types of competition has been underscored numerous times in a variety of systems (Strong et al. 1984, Underwood 1986, Keddy 1989). In the field study, intraspecific competition was equivalent to interspecific competition in determining survival in the absence of the virus. However, in the presence of the virus, interspecific interactions produced higher virus prevalence than intraspecific interactions. Therefore, this study emphasizes the importance of experiments that explicitly partition intraspecific and interspecific competition.

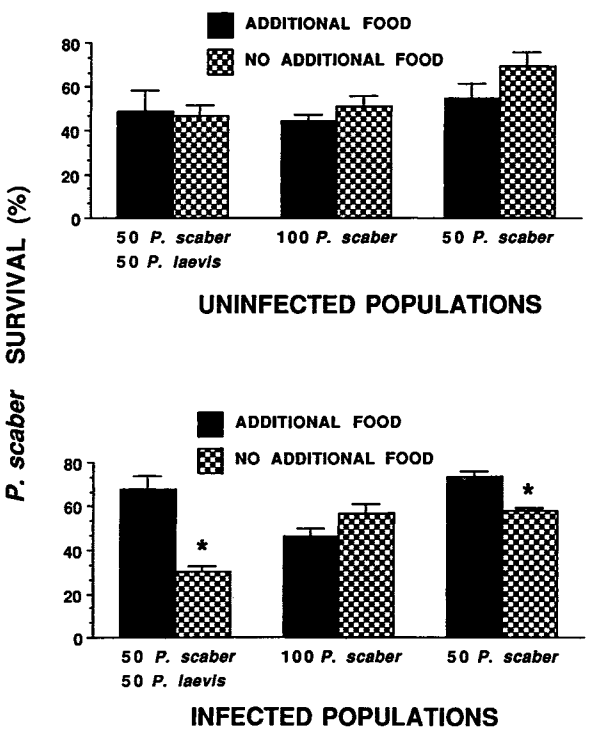

FIG. 3. Survival of *Porcellio scaber* in the field as a function of species and food treatments, for both uninfected and virus-infected populations. Error bars represent 1 SE of the mean, and asterisks indicate pairs of means within a species treatment that are significantly different (Student-Newman-Keuls, $n = 4$).

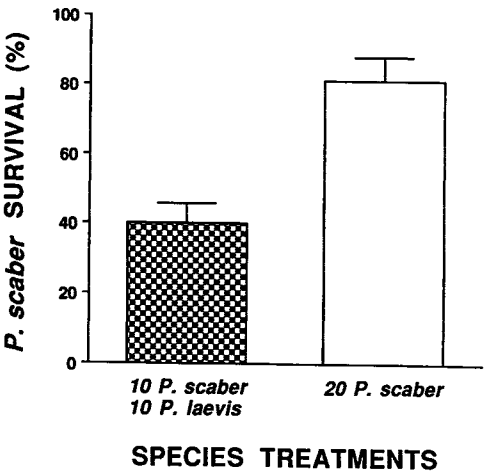

FIG. 4. Survival of *Porcellio scaber* in the laboratory as a function of species treatments for uninfected controls only. Error bars represent 1 SE of the mean, and means are significantly different ($n = 3$).

Competition effects on parasitism

The field experiment showed that interspecific competition with *Porcellio laevis* increased the level of virus prevalence, and thus the impact of the virus on *P. scaber* populations. By contrast, increasing intraspecific competition in *P. scaber* with the same number of conspecifics had no significant effect on the virus prevalence.

Intraguild aggression may be a possible mechanism by which *P. laevis* increases virus prevalence in *P. scaber*. Predation and cannibalism within a guild are common amongst many organisms (Polis et al. 1989), and both species display these behaviors in natural populations (E. D. Grosholz, *personal observation*). These behaviors are especially common when isopods are molting or otherwise weakened. Females will routinely cannibalize their offspring in the lab, and adults have been observed to cannibalize juveniles in the field (E. D. Grosholz, *personal observation*). Isopods are very susceptible to virus infection through the hemocoele (Hess and Poinar 1985, Kelley 1985), so damage to the exoskeleton as the result of interspecific interactions may increase the probability of virus infection.

This mechanism is partially supported by the similar results of the laboratory experiment in which higher virus prevalence in *P. scaber* occurred in treatments with *P. laevis*. Unlike the field experiment, in the laboratory I controlled the availability of all food resources to examine competitive mechanisms. Virus

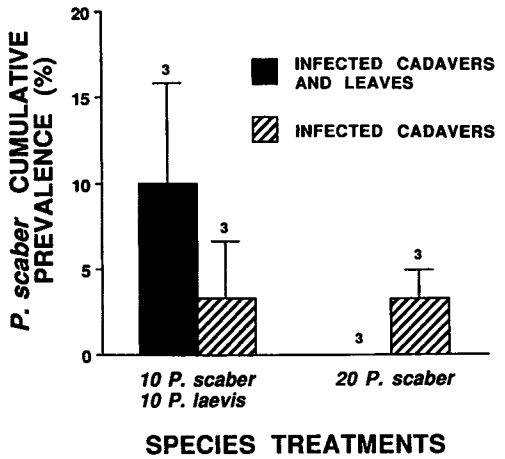

FIG. 5. Virus prevalence in *Porcellio scaber* in the laboratory as a function of species and food treatments. Error bars represent 1 SE of the mean. Means are not significantly different ($n = 3$).

prevalence in *P. scaber* was higher in treatments with *P. laevis* with two food sources rather than one, so simple exploitation seems an unlikely mechanism. It is possible that aggressive interactions over food types may have contributed to the trend of elevated infection rates.

Interference competition has been demonstrated for other terrestrial isopods. Ganter (1984) found that growth rate, survivorship, and reproduction of both *Armadillidium vulgare* and *A. nasatum* decreased with density in the presence of excess food. Increased food actually increased mortality for *A. nasatum*. In contrast to the present study, intraspecific interference was greater than interspecific. Mortality was lower and growth rates higher for *A. nasatum* in treatments with *A. vulgare* relative to treatments with conspecifics only (Ganter 1984).

Although food levels are not comparable, both laboratory and field studies showed little effect of food on interspecific interactions. This result is consistent with interference rather than resource competition. The data further suggest that at this field site food may not be limited. Both biotic (Brereton 1957, Sunderland and Sutton 1980, Rushton and Hassall 1983, Ganter 1984) and abiotic (Paris and Pitelka 1962, Paris 1963, McQueen 1976, Warburg et al. 1984) factors have been suggested as limiting to terrestrial isopod populations. Determining whether the processes operating in this study ultimately limit isopod populations awaits further study.

Parasitism effects on competition

The field data indicated that the presence of the virus created apparent competition. In the absence of the virus, interspecific competition was equivalent to intraspecific competition. In the presence of the virus, if one was unable to discern the mortality due to the virus, as would be the case with most parasites and pathogens, the apparent effect would be that interspecific competition had a greater effect than intraspecific competition. Therefore, the additional mortality due to virus in the interspecific treatments could erroneously be mistaken for interspecific competition, and, hence, apparent competition. With the three-fold higher densities in the laboratory, there was a negative effect of *P. laevis* on *P. scaber*. This suggests that with equivalent densities in the field, interspecific competition might have been greater than intraspecific competition. However, the virus would still compound the negative effects of competition.

Although the idea of apparent competition has been well developed (Holt 1977, Holt 1984, Jeffries and Lawton 1984, Holt and Pickering 1985), experimental demonstrations are rare in natural communities. Schmitt (1987) found that the indirect effects of shared predators (*Octopus bimaculatus, Kelletia kelletii*) resulted in apparent competition between gastropods (*Tegula* spp.) and co-occurring bivalves (*Chama* spp.). Settle and Wilson (1990) experimentally demonstrated that a shared parasitoid, *Anagrus epos,* altered the competitive symmetry between competing leaf hoppers, *Erythroneura variabilis* and *E. elegantula,* resulting in apparent competition.

Examples of apparent competition due to a common parasite or pathogen are also few (reviewed in Price et al. [1988]). White-tailed deer, *Odocoileus virginianus,* are known to carry a meningeal worm, *Parelaphostrongylus tenuis,* that is pathogenic to sympatric cervids such as moose, *Alces americana,* and caribou, *Rangifer tarandus* (Anderson 1976). The deer mouse, *Peromyscus maniculatus,* is less susceptible to sylvatic plague, *Yersinia pestis,* than the woodrat, *Neotoma cinerea,* and after an epidemic, will colonize cave habitats previously occupied by woodrats (Nelson and Smith 1976). It has been suggested that heteroecious rust fungi may dictate the interactions between competing species (Rice and Westoby 1982). However, these studies are generally non-experimental.

This study is perhaps the first experimental demonstration of apparent competition due to a shared pathogen in which both host species and the pathogen are manipulated in the field. The results indicate that the impact of *P. laevis* on *P. scaber* should be considered not simply as an isolated direct or indirect effect, but rather as a compound effect involving a direct effect (interspecific competition) and an indirect effect (apparent competition due to parasitism).

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