



CHICAGO JOURNALS



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Source: *The American Naturalist*, Vol. 131, No. 4 (Apr., 1988), pp. 544-555

Published by: [The University of Chicago Press](#) for [The American Society of Naturalists](#)

Stable URL: <http://www.jstor.org/stable/2461743>

Accessed: 29/08/2011 15:55

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PARASITE-MEDIATED COMPETITION:  
SOME PREDICTIONS AND TESTS

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*Submitted January 6, 1986; Revised April 20, 1987; Accepted July 27, 1987*

Suppose that two species are in contact, along with a parasite that is capable of using both species as a host. Such a situation could come about if geographic or local ranges change such that the two species come into contact, and the parasite is present in one of them. Alternatively, a new parasite or a new strain could evolve in one of two species that have been in contact for some time. In general, one host will be more tolerant of the parasite, immunologically and/or genetically, than the other host because of longer exposure to the disease.

Because of the presence of the other host, either of the two host species could experience an infestation that was more severe or was more virulent than it would have been in the absence of the other host. The two species therefore have a potentially negative effect on each other via the parasite. They are in indirect, parasite-mediated competition. As Haldane said, "A non-specific parasite to which partial immunity has been acquired, is a powerful competitive weapon" (1949, p. 70). Here we make predictions about outcomes of parasite-mediated competition, using generalizations about parasites and their effects, plus assumptions to the effect that other things are equal. Further, we investigate evidence available for testing these predictions. The evidence is sketchy, but what there is supports the predictions.

Terms used in describing this sort of interaction have included "germ warfare" (Barbehenn 1969), "apparent competition" (Holt 1977), "biological warfare" (Price 1980), "weapons of competition" (Holmes 1982), "agents of interference competition" (Rice and Westoby 1982), and "parasite mediation of interactions" (Price et al. 1986). The concept is thus not a new one; neither do we wish to reiterate the examples that have been described in the above papers. Rather, our aim here is to contribute to the theory about the outcome of such interactions. Until now, no general patterns or predictions have been observed or made about parasite-mediated interactions, except that they are very common in nature (Price et al. 1986). Indeed, Park's (1948) pioneering work on the sporozoan parasite *Adelina tribolii* mediating competition between *Tribolium* beetle species in the

laboratory indicated that clear-cut winners or losers in competition may not occur, and patterns in nature would be difficult to detect as a result.

### THREE GENERALIZATIONS ABOUT PARASITE-HOST RELATIONSHIPS

1. *Relationships between parasitic species and the host area.*—The number of parasitic species per host increases as the area occupied by the host population increases (Janzen 1968, 1973; Dritschilo et al. 1975; Price 1977, 1980; Freeland 1979; Lawton and Price 1979; Price and Clancy 1983). Using a broadly conceived view of a parasite (as in Price 1980), several researchers have found this pattern within one host species (Strong 1974; Tepedino and Stanton 1976; Strong et al. 1977; Freeland 1979) and between different host species (Opler 1974; Strong and Levin 1975; Lawton and Schröder 1977; Cornell and Washburn 1979; Price and Clancy 1983).

From these relationships, we deduce that increases in host range (1) increase the probability that one or more parasitic species can become advantageous to the carrier in parasite-mediated competition; (2) reduce the probability of exposure to novel parasitic species; and (3) result in host populations more genetically and immunologically resistant to the existing parasitic diseases. In addition, the wider the host range, the more parasitic strains are likely to exist, increasing the possibility that our deductions are correct.

We assume that these patterns translate to similar effects at the local level of host-species interaction. (See below for evidence from Europeans landing in the New World.)

2. *Host geographic range, population density, and body size.*—The geographic range of a species correlates positively with local abundance (Hanski 1982; Bock and Ricklefs 1983; Brown 1984). Also, species with higher numbers per unit of area in general have smaller body size (Mohr 1940, 1947; Ghilarov 1967; Clutton-Brock and Harvey 1977; Damuth 1981; Peters 1983; Robinson and Redford 1986). Body mass of species correlates negatively with population growth rate (Farlow 1976; Banse and Mosher 1980; Peters 1983).

These patterns suggest that, in general, species with larger geographic ranges, which, according to generalization 1 above, carry more parasitic species, also have higher population densities, smaller body sizes, and higher population growth rates. These kinds of species have a higher rate of production of young individuals, naive and susceptible to disease, resulting in a higher effective population size for maintaining parasitic populations that induce rapid and persistent immune responses.

3. *Threshold theorem in epidemiology.*—There exists a threshold host-population density, below which a disease cannot establish and persist (Kermack and McKendrick 1927; R. M. Anderson 1981, 1982*a,b*). The more virulent the disease, the greater the host population needed to sustain it. Equations for threshold densities differ depending on mode of transmission between hosts (R. M. Anderson and May 1979; May and Anderson 1979), but threshold densities are important in the majority of diseases and are frequently high. For example, maintenance of measles in human populations on islands occurs only at a population size of

550,000 (Hawaii), and not at 346,000 (Fiji) (Black 1966). A highly significant correlation exists between population size ( $N$ ) and the percentage of months in which cases were reported ( $P_m$ ) ( $P_m = 16.07 + 0.00016N$ ,  $n = 19$ ,  $r^2 = 0.59$ ,  $p < 0.01$ ; our analysis of Black's data).

These patterns indicate that the larger the population is, the higher the probability is of sustaining any disease, the more likely it is that more-virulent forms of a disease will be maintained, and the higher the probability is that individuals capable of transmitting the disease are present in the population at any given time.

#### PREDICTIONS FROM GENERALIZATIONS

Which host species will be favored in parasite-mediated competition and displace the other host species in terms of relative abundance or spatial distribution? As a general tendency, we expect that hosts with many parasitic species, some of which are novel or more virulent to the other competitor, tend to displace hosts with fewer parasitic species. Therefore, we suggest the following two hypotheses.

1. *The geographic-range hypothesis.*—Species with a wide geographic range tend to displace species with a narrower range when a parasite common to both has differential impact. Wide-ranging species have a greater probability of acquiring new parasitic species from the various parts of their ranges, which become incorporated into the general parasitic species pool for that host.

The geographic-range hypothesis may be effective independent of body size, which is invoked in the next hypothesis. Populations of the same species (i.e., with the same body size) coming together for the first time may interact in the predicted way. We discuss this case for human populations below (under "Tests of hypotheses").

2. *The body-size hypothesis.*—Species with smaller body mass tend to displace species with larger body mass when a parasite common to both has differential impact. This hypothesis is not independent of the geographic-range hypothesis, but it is another testable aspect of the same mechanistic relationships.

In some taxa, positive correlations exist between body size and number of parasitic species (e.g., Lawton and Schröder 1977; Price 1977, 1980; Strong and Levin 1979). If these are sufficiently strong or general to compensate for the effects of geographic range and population density, our hypothesis would fail.

The net effect of the geographic-range, population-density, and body-size correlations is increased parasite numbers as range and density increase. This is because the relationship of the host area to the parasitic species number is quite general, although not universal (e.g., exceptions in Dunn 1966; Kennedy 1978; Betterton 1979), and the relationship of host body size to the number of parasitic species has important exceptions (e.g., Lawton and Price 1979; Price and Clancy 1983). In addition, the effects of local density, so well established in the epidemiological literature, have never been tested simultaneously for importance relative to geographic range and body size, so far as we know. We assume, then, that the more-established patterns of geographic range and threshold density have a stronger effect on parasitic species number and virulence than the less-understood direct effect of body size.

The body-size hypothesis differs distinctly from predictions often made about direct competition for resources; that is, species of larger body size win, either because they exploit a broader range of resources (Wilson 1975; Hutchinson 1978) or because of their advantage in behavioral interactions or in plants' competition for nutrients and light (Clements et al. 1929; Klopfer 1962; Harper 1977).

#### TESTS OF HYPOTHESES

To test these predictions, we need cases in which one species is disadvantaged in response to a parasite carried by another species, and the reverse effect is smaller or absent; for brevity, we call the displacing species the "winner" and the other the "loser." Examples have previously been collected (Price 1980; Rice and Westoby 1982); these examples and the sources cited should be consulted for full details. Tests of hypothesis 1 also require us to know which of the species had the wider geographic range, and tests of hypothesis 2 require us to know which species has larger body size. The quality of the data supporting the interactions and the resulting winners and losers varies enormously (see below). However, the cases were selected objectively because Price (1980) presented no expectation of patterns and Rice and Westoby (1982) drew on all the available literature on the subject. Since these publications, we have learned of no new cases.

These requirements leave a limited but still suggestive body of evidence. Of cases that test hypothesis 1 (table 1), the winner is the species with the wider geographic range in 9 of 11 cases ( $p < 0.025$ , one-tailed Wilcoxon matched-pairs signed-rank test,  $n = 11$ , critical value of  $T = 11$ ; Siegel 1956). Of cases that test hypothesis 2 (table 2), the winner is the species with the smaller body size in 12 of 15 cases ( $p < 0.018$ , one-tailed sign test,  $n = 15$ ; Siegel 1956). (The 3 cases of roughly equal-sized hosts do not enter the analysis. The test enables use of qualitative data such as large versus small.) Note that tables 1 and 2 include some cases in which one competitor has a wider geographic area but the other has a smaller body size. Such cases necessarily confirm one of the hypotheses but are counter to the other, depending on which effect has greater weight. In fact, every apparent counter case in either table 1 or 2 was an example of this kind. Therefore, the evidence in tables 1 and 2 is even more consistent with the two hypotheses than appears at first glance.

The meningeal worm (*Parelaphostrongylus tenuis*; examples 1–5, tables 1, 2) is one of the best-studied wildlife pathogens. Pronghorn antelope (*Antilocapra americana*) and cervids other than whitetail deer (*Odocoileus virginianus*) suffer severe neurological disease with only small infections of the brain. Repeated efforts to introduce cervids where meningeal worm was present have been unsuccessful (R. C. Anderson 1976; R. C. Anderson and Prestwood 1981). The extensive field data and introduction "experiments" constitute strong evidence of parasitic mediation in our estimation. If each introduction and every case of displacement recorded were entered into an analysis of meningeal worm–whitetail deer impact, the pattern would be convincing.

The interaction between deer mice (*Peromyscus maniculatus*) and woodrats (*Neotoma cinerea*) in lava caves (example 6) is not as well documented as that of

TABLE 1  
TEST OF THE GEOGRAPHIC-RANGE HYPOTHESIS

Parasite	Winner	Range* (million ha)	Loser	Range* (million ha)
1. Meningeal worm, <i>Parelaphostrongylus tenuis</i>	whitetail deer ( <i>Odocoileus virginianus</i> )	1088	moose ( <i>Alces alces</i> )	843
2. <i>P. tenuis</i>	whitetail deer	1088	mule deer ( <i>Odocoileus hemionus</i> )	748
3. <i>P. tenuis</i>	whitetail deer	1088	woodland caribou ( <i>Rangifer caribou</i> )	535
4. <i>P. tenuis</i>	whitetail deer	1088	elk ( <i>Cervus canadensis</i> )	169
5. <i>P. tenuis</i>	whitetail deer	1088	pronghorn antelope ( <i>Antilocapra americana</i> )	313
6. Sylvatic plague, <i>Yersinia pestis</i>	deer mouse ( <i>Peromyscus maniculatus</i> )	1518	bushytail woodrat ( <i>Neotoma cinerea</i> )	319
7. Stomach worm, <i>Graphidium strigosum</i>	European rabbit ( <i>Oryctolagus cuniculus</i> )	254	European hare ( <i>Lepus europaeus</i> )	4727
8. White pine blister rust, <i>Cronartium ribicola</i>	red currant ( <i>Ribes triste</i> )	1227	white pine ( <i>Pinus strobus</i> )	529
9. Parasites	Norway rat ( <i>Rattus norvegicus</i> )	large (New World)	black rat ( <i>Rattus rattus</i> )	small (New World)
10. Parasites	Norway rat	1718 (Old World)	black rat	787 (Old World)
11. <i>Strongyloides robustus</i>	southern flying squirrel ( <i>Glaucomys volans</i> )	359	northern flying squirrel ( <i>Glaucomys sabrinus</i> )	887

NOTE.—Of the 11 cases, two (7 and 11) do not fit the hypothesis.

SOURCES.—1–5, R. C. Anderson 1972, 1976; R. C. Anderson and Prestwood 1981; 6, Nelson and Smith 1976, 1980; 7, Broekhuizen and Kemmers 1976; 8, Boyce 1948 (for more biology, see Mielke 1943; Rice and Westoby 1982); 9, 10, Barbehenn 1969; 11, Weigl 1968, 1975.

\* Range estimates: for animals, Burt and Grossenheider 1964, Corbet 1978, van den Brink 1968; for plants, Fernald 1950.

meningeal worm. However, the descriptive details of two plague (*Yersinia pestis*) outbreaks provide strong support for parasitic mediation. During an epidemic, most of the woodrat population of 20 caves became extinct because of plague, whereas little mortality of deer mice occurred in the same caves (Nelson and Smith 1976, 1980). Deer mice carry *Yersinia*, but they are resistant to the pathogen and probably act as the reservoir host for it.

The differential pathogenicity of the stomach worm *Graphidium strigosum* on rabbit and hare (example 7) is well established; rabbits are the reservoir host and must be present for hares to become infected. In rabbits, both the proportion of infected individuals in a population and the number of parasites per individual are higher (Broekhuizen and Kemmers 1976). When rabbit numbers decline, the hare population increases; but the detailed studies needed to distinguish between direct

TABLE 2  
TEST OF THE HOST-BODY-SIZE HYPOTHESIS

Parasite	Winner	Mass* (g)	Loser	Mass* (g)
1. Meningeal worm, <i>Parelaphostrongylus tenuis</i>	whitetail deer	136,000	moose	454,000
2. <i>P. tenuis</i>	whitetail deer	136,000	mule deer	136,000
3. <i>P. tenuis</i>	whitetail deer	136,000	woodland caribou	227,000
4. <i>P. tenuis</i>	whitetail deer	136,000	elk	409,000
5. <i>P. tenuis</i>	whitetail deer	136,000	pronghorn	45,000
6. Sylvatic plague, <i>Yersinia pestis</i>	deer mouse	28	bushytail woodrat	425
7. Stomach worm, <i>Graphidium strigosum</i>	European rabbit	1,500	European hare	4,000
8. White pine blister rust, <i>Cronartium ribicola</i>	red currant	small	white pine	large
9. Parasites	Norway rat	241 (New World)	black rat	213 (New World)
10. Parasites	Norway rat	397 (Old World)	black rat	180 (Old World)
11. <i>Strongyloides robustus</i>	southern flying squirrel	57	northern flying squirrel	142
12. <i>Cronartium comptoniae</i>	myrtle ( <i>Myrica spp.</i> )	small	pine ( <i>Pinus spp.</i> )	large
13. <i>Chrysoomyxa pyrolae</i>	wintergreen ( <i>Pyrola spp.</i> )	small	spruce ( <i>Picea spp.</i> )	large
14. <i>Chrysoomyxa</i> spp.	<i>Rhododendron spp.</i> and <i>Ledum spp.</i>	small	spruce ( <i>Picea spp.</i> )	large
15. <i>Pucciniastrum padi</i>	cherry ( <i>Prunus spp.</i> )	small	spruce ( <i>Picea spp.</i> )	large
16. <i>Cronartium quercum</i>	oak ( <i>Quercus spp.</i> )	large	pine ( <i>Pinus spp.</i> )	large
17. <i>Cronartium fusiforme</i>	oak ( <i>Quercus spp.</i> )	large	pine ( <i>Pinus spp.</i> )	large
18. <i>Tranzschelia pruni-spinosae</i>	<i>Anemone spp.</i>	small	Rosaceae	large

NOTE.—Of six exceptions to the hypothesis (2, 5, 9, 10, 16, 17), three have hosts of roughly equal size (2, 16, 17).

SOURCES.—1–11, see table 1; 12–14, Rice and Westoby 1982; 15, Peace 1962, Rice and Westoby 1982; 16, 17, Hedgecock and Siggers 1949, Rice and Westoby 1982; 18, Arthur 1929, Rice and Westoby 1982.

\* Approximate mass values obtained from Burt and Grossenheider 1964 and van den Brink 1968 for animals. Values are not available for the plant examples.

competition and parasite-mediated competition have not been made. Given the differential pathogenicity and high infection rate of rabbits, it is hard to conceive that *Graphidium* would not play an important role.

White pine blister rust (*Cronartium ribicola*; example 8) kills pines (Boyce 1948), but currants (*Ribes triste*) soon recover vigor (Lachmund 1934). Where pines were being killed, the best control has been to remove currants (Boyce 1948; Large 1962), clearly demonstrating the impact of the disease in the presence of

currants. This disease differs from other cases discussed here because both hosts are essential for completion of the full life cycle of the parasite. However, the repeating asexual uredial stage is on the *Ribes* host, meaning that the parasite can persist and increase in the absence of pine (Alexopoulos 1952) and suggesting that *Ribes* should evolve more resistance to the pathogen.

Because the interactions between the Norway rat (*Rattus norvegicus*) and the black rat (*Rattus rattus*) are not well studied, examples 9 and 10 are largely speculations by Barbehenn (1969).

Northern flying squirrels (*Glaucomys sabrinus*) died in field cages close to southern flying squirrels (*G. volans*), which did not die when both species were infected with *Strongyloides robustus*. Laboratory-reared *G. sabrinus* and wild animals without *S. robustus* did not die (example 11; Weigl 1975, pers. comm.). Where the species occur in the same locality in the Appalachians, *G. sabrinus* is largely excluded from habitat occupied by *G. volans*. This kind of evidence is suggestive but not conclusive. Differential susceptibility to an important parasite is clear, but its real role in nature needs more study, as is usually the case (Barbehenn 1969; Weigl 1975).

Examples 8 and 12–18 (table 2) have all been discussed earlier (Rice and Westoby 1982). The evidence in these cases that the rusts damage one host more than the other is derived from the literature cited in tables 1 and 2 and personal communication from experts. As Rice and Westoby acknowledged, no studies have quantified rust effects on individual plant fitness. Such studies will not be undertaken until evolutionary and ecological hypotheses that need to be tested are developed. Nevertheless, the pattern they discovered is remarkably consistent: the plant host that is smaller, earlier in succession, or in the understory suffers less damage than the larger host. Except for the case of white pine blister rust, we do not really know how this differential susceptibility translates into differential demography, but the pattern suggests that demographic effects will become more evident when they are adequately studied.

Another test of the geographic-range hypothesis uses human examples of population interactions. This intraspecific evidence keeps body size constant in order to reveal more purely the effects of population size and density. Crosby noted, "The records of every European people who have had prolonged contact with the native peoples of America are full of references to the devastating impact of Old World diseases" (1972, p. 42). As McNeill (1976, 1980) explained, extensive travel across Europe and Asia brought new diseases into urbanized centers in Europe where "disease-experienced populations" (1980, p. 20) developed and maintained many pathogens in a virulent state. When these diseases were carried to the isolated and smaller populations of the New World, to "disease-inexperienced peoples" (1980, p. 20), the results were catastrophic, and undermined any organized attempts to resist settlement by Europeans. Crosby (1972) outlined many epidemics from 1519 onward, principally in South and Central America. Only cases from Alaska may deviate from this pattern because contact across the Bering land bridge through prehistory may have lessened the impact of European diseases in historical time (Reinhard 1974). The results of contact between Europeans and Amerindians repeatedly support the geographic-range



hypothesis (Cook 1946; Ashburn 1947; Cook and Borah 1960; Dobyns 1963; Borah 1964; Crosby 1972).

#### DISCUSSION

The evidence in tables 1 and 2 and the human example support our two predictions. We would not want to overinterpret this, because the examples we have found are not so broadly representative as we would wish. For example, five of our cases involve whitetail deer and the meningeal worm, although with different competing hosts. Similarly, all the cases with plant hosts involve heteroecious rusts as the parasites. Nevertheless, the number of examples available, and rigorous testing for the existence of parasite-mediated competition, will not improve until the community of evolutionary biologists is aware of the potential of this phenomenon. This is our major justification for this paper, not the quality of the data we have available for testing.

Even so, our predictions from empirical patterns do converge on recent development of theory by Holt and Pickering (1985). Since species are closely related in the animal examples listed in tables 1 and 2, cross-species infection is likely to occur as frequently as within-species infection. This seems to be the case for *Parelaphostrongylus*, *Yersinia*, *Graphidium*, and *Strongyloides*, at least. Then, host 1 excludes host 2 if  $\hat{I}_2\beta_{22} < \hat{I}_1\beta_{21}$ , where  $\hat{I}_i$  is the density of infected individuals in host  $i$  when it is alone and at equilibrium, and  $\beta_{ij}$  is a measure of the rate at which host  $j$  infects host  $i$ . If transmission occurs at the same rate within and between species, then exclusion occurs when  $\hat{I}_2 < \hat{I}_1$ . Whichever host has the higher density of infected individuals tends to exclude the other host, which is likely to be the smaller species with the higher population density and higher  $r$ . Indeed, this is explicitly incorporated in the Holt and Pickering model; their equation (3) defines  $\hat{I}_1$ , as  $\hat{I}_1 = r_1 d_1 / \beta_{11} (d_1 - e_1)$ , where  $r$  is the intrinsic rate of growth of host 1,  $d_1$  is the rate of decline of the pool of infective individuals through death and recovery, and  $e$  is the rate of entry into the pool of susceptible individuals (individuals that have been born to infected hosts or that have recovered from infection). Therefore, species of smaller mass with higher  $r$  have higher  $\hat{I}_i$  than species of larger mass and tend to exclude the larger species, according to the model. To this extent, the model is consistent with the empirical data presented in table 2.

We clearly need data sets that indicate the number of parasitic species and their density per host, the local abundance of hosts, the geographic ranges of hosts and parasites, and host body sizes and density. With these data we could examine whether the various correlations among these variables were independent and additive or not and then test the extent to which they reflect mechanistic relationships. In addition, more-detailed studies are needed about the local interaction of hosts with and without parasites.

In summary, we believe the available evidence is strong enough to be considered a tentative confirmation of our two predictions. Bakker (1983) suggested that extinctions of South American mammals that came into contact with the North American fauna were more consistent with parasitic action than with predation or

competition for resources. We suggest that parasite-mediated competition may have been one of the important causes of extinctions when previously separate biotas have been mixed over evolutionary time. Further, we suggest that the geographic-range hypothesis and the body-size hypothesis should apply to these extinctions.

Application and extension of these hypotheses are limited by the inadequate record of disease in wild organisms and how it mediates interactions, although such interactions are common in nature (Price et al. 1986). However, the potential importance of such interactions is growing as more plants and animals are moved across geographic barriers, as nature reserves are planned and implemented, as human tribes are forced to coexist, as agricultural crops grow in new associations with wild plants, as genetic engineering radically alters disease epidemiology, and as the paleobiology of extinction is increasingly explored.

#### SUMMARY

Two new hypotheses on parasite-mediated competition are developed from well-defined patterns in nature involving the positive relationship of parasitic species number and host area, the negative relationship of population density and body mass, and the threshold theorem in epidemiology. The geographic-range hypothesis predicts that species with larger geographic ranges, carrying more parasites as "biological warfare" agents, usually displace species with smaller ranges. The body-size hypothesis predicts that smaller species, with higher density and with a higher intrinsic rate of population increase, sustain more parasitic species than do larger species and, counter to much competition theory, exclude larger species more frequently. These hypotheses are tested using all suspected cases of parasite-mediated competition for which the relevant data are available. Nine cases of 11 support the geographic-range hypothesis. For species of different size, 12 cases of 15 support the body-size hypothesis. No examples run counter to both hypotheses. More examples and more-detailed studies of the examples in current use are needed before these patterns can be considered firmly established.

#### ACKNOWLEDGMENTS

We greatly appreciate valuable constructive comments by P. W. Ewald, J. C. Holmes, R. D. Holt, J. K. Moore, J. Pickering, M. L. Rosenzweig, J. J. Schall, P. D. Weigl, and three anonymous reviewers. R. C. Anderson provided valuable information and reference sources on the meningeal worm. This work was supported by grant BSR-8314594 from the National Science Foundation.

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