A Meta-Analysis of Factors Affecting Local Adaptation between Interacting Species

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ABSTRACT: Adaptive divergence among populations can result in local adaptation, whereby genotypes in native environments exhibit greater fitness than genotypes in novel environments. A body of theory has developed that predicts how different species traits, such as rates of gene flow and generation times, influence local adaptation in coevolutionary species interactions. We used a meta-analysis of local-adaptation studies across a broad range of host-parasite interactions to evaluate predictions about the effect of species traits on local adaptation. We also evaluated how experimental design influences the outcome of local adaptation experiments. In reciprocally designed experiments, the relative gene flow rate of hosts versus parasites was the strongest predictor of local adaptation, with significant parasite local adaptation only in the studies in which parasites had greater gene flow rates than their hosts. When nonreciprocal studies were included in analyses, species traits did not explain significant variation in local adaptation, although the overall level of local adaptation observed was lower in the nonreciprocal than in the reciprocal studies. This formal meta-analysis across a diversity of host-parasite systems lends insight into the role of both biology (species traits) and biologists (experimental design) in detecting local adaptation in coevolving species interactions.

Keywords: coevolution, host-parasite, local adaptation, mutualism, meta-analysis.

Evolutionary ecologists have long been interested in the question of what causes differences in phenotypic traits among populations (e.g., Levins 1968; Mayr 1970). Recently, much research into this question has focused on how adaptive divergence among populations results in local adaptation, that is, genotypes in their local environments exhibiting greater fitness than genotypes in nonlocal environments. Studies of local adaptation have become a paradigm for testing evolutionary hypotheses about traits that are favored in different environments (Kawecki and Ebert 2004). A subset of this work has focused on local adaptation between pairs of interacting and potentially coevolving species or guilds of species. Unlike adaptation to the physical environment, the adaptive peaks for coevolving species may continually shift in response to changes in the other coevolving species, which can make ongoing evolutionary potential more important than when adaptive peaks are relatively static (Peters and Lively 1999; Thompson 2005; Garant et al. 2007). Local adaptation between species is the starting point for the coevolutionary process, and patterns of local adaptation between species can reveal how species interactions drive adaptive differentiation among populations (Thompson 2005).

The outcomes of experiments testing for local adaptation between coevolving species have varied widely, from strong local adaptation in some species interactions (e.g., Jackson and Tinsley 2005) to strong maladaptation in other species interactions (Kaltz et al. 1999). The reasons for this variation across different systems are still poorly understood because most studies of the causes of local adaptation either are theoretical (e.g., Gandon et al. 1996) or focus on empirical work in particular study systems. A general understanding of the factors affecting local adaptation in coevolutionary species interactions requires a comparative approach across a diversity of natural systems. Here, we present a formal meta-analysis testing hypotheses for variation in local adaptation across a broad cross section of parasitic and mutualistic interactions.

The degree and direction of local adaptation between interacting species is usually predicted to be driven by differences in characteristics between those species, and much of this theory has been developed in the context of host-parasite interactions. First, parasites have been pre-

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dicted to be locally adapted to their hosts because they have shorter generation times, and thus potentially greater evolutionary rates, than their hosts, which may lead to an adaptive advantage (Dawkins and Krebs 1979; Price 1980; Hafner et al. 1994; Ebert and Hamilton 1996; Kaltz and Shykoff 1998). Indeed, a prominent theory for the evolution of sex, referred to as the Red Queen hypothesis, makes this assumption and predicts that reciprocal coevolutionary selection between hosts and parasites should result in parasites being locally adapted to their hosts on average (Van Valen 1973; Ebert and Hamilton 1996; Lively 1999). Some recent models of coevolution in both host-parasite interactions and mutualistic interactions, however, suggest that faster generation time in the symbiotic species does not always lead to greater local adaptation (Lively 1999; Gandon and Michalakis 2002) and sometimes has the opposite effect (Doebeli and Knowlton 1998; Gandon and Michalakis 2002; Bergstrom and Lachmann 2003), especially if genetic variability is limiting (Gandon and Michalakis 2002). One goal of the analysis presented here was to test for a relationship between parasite local adaptation and relative differences in generation time between hosts and parasites across a broad range of host-parasite interactions.

The issue of genetic variability highlights a second set of host and parasite traits commonly used to predict local adaptation: the gene flow rates of the species. Many models of host-parasite coevolution predict that parasite local adaptation will be facilitated by parasites having higher migration rates (and thus gene flow rates) among populations than hosts, and vice versa (Gandon et al. 1996; Gandon 2002), because gene flow introduces new alleles on which local selection can act. These potential benefits of gene flow to adaptation might be greatest in strongly coevolutionary interactions, that is, when the target of adaptation is capable of evolving quickly (Garant et al. 2007). Not all models predict this effect of greater gene flow rates, however (e.g., Lively 1999); for example, high gene flow rates can potentially swamp local selection (Slatkin 1985), preventing local adaptation or causing maladaptation in some populations (Dias 1996; Lenormand 2002; Thompson et al. 2002). Thus, a second goal of our analysis was to test for a relationship between parasite local adaptation and the relative gene flow rates of hosts and parasites.

Third, differences between host and parasite species in their organismal complexity may influence levels of local adaptation. According to the cost-of-complexity hypothesis, greater organismal complexity (e.g., a greater number of cell types or independent body parts in the body of an organism) may be associated with a cost in terms of rate of adaptation, whereby simpler species can adapt faster than more complex species (Orr 2000; Welch and Waxman 2003). We know of no instances where this hypothesis has been applied to coevolution; however, if it holds for coevolving host-parasite interactions, then we predict that parasite local adaptation should be strongest when parasites are relatively less complex than their hosts, and vice versa. A third goal of our meta-analysis was to test for a relationship between parasite local adaptation and relative organismal complexity of host and parasite species.

Fourth, phylogenetic distance between the host and parasite may influence coevolutionary dynamics and local adaptation between species. To our knowledge, no formal theory has been developed to address this relationship directly, but one idea in the literature suggests that it may be worth testing. Emery's rule (Emery 1909; Wilson 1971) is the observation that species often effectively parasitize other closely related species, especially among social insects, although the rule has also been suggested to extend to diverse groups of organisms such as fungi, red algae, and mistletoe (e.g., Goff et al. 1997). One suggested mechanism for Emery's rule is that parasites can pass more easily through the defenses of a sibling species than through those of less closely related species (Lowe et al. 2002). If a close phylogenetic relationship between host and parasite tends to give parasites an evolutionary advantage, then parasites may be more locally adapted to more closely related hosts; however, it is not clear at what phylogenetic scale such a phenomenon should manifest itself. Thus, we explored the potential relationship between phylogenetic distance and local adaptation by using taxonomic similarity as a proxy for phylogenetic distance and testing its relationship to host-parasite local adaptation.

Theory suggests that coevolutionary dynamics of host-parasite interactions should differ, depending on the genetic system governing the interaction (e.g., Agrawal and Lively 2002). For example, interactions involving plant hosts have been thought to often be governed by gene-for-gene genetic systems, whereas interactions involving animal hosts are often thought to be governed by matching-allele genetic systems (Parker 1994). The matching-allele genetic system should be more likely to generate the type of coevolutionary dynamics described by the Red Queen hypothesis than the gene-for-gene system (Parker 1994; Agrawal and Lively 2002), leading to the possibility that local adaptation of parasites to animal hosts will be stronger or more common than local adaptation of parasites to plant hosts. One goal of our analysis was to test whether there are differences in local adaptation of parasites to plant versus animal hosts.

Investigations of local adaptation between parasite and host species usually utilize some form of a cross-infection experiment, in which the fitness of the host and/or the parasite is compared between sympatric (hosts and parasites from the same population) and allopatric (host and parasites from different populations) host-parasite pairings. The details of the design and analysis of these cross-

infection experiments can have a significant influence on the inferences that can be made from them regarding local adaptation. One important experimental design consideration is reciprocality, that is, whether or not all parasite and host populations or demes that are included in sympatric pairings are also included in allopatric pairings, and vice versa. Thrall et al. (2002) demonstrated particularly well that when an experimental design is not reciprocal, overall differences in parasite virulence or host resistance among populations can lead to results that superficially appear to indicate local adaptation, because overall main effects of the host and/or parasite populations cannot be separated from host-by-parasite population interactions in the analysis. For example, consider an experiment that tests only the fitness of a focal parasite population with its sympatric host population and an allopatric host population but does not also test the fitness of the allopatric (i.e., nonfocal) parasites on both host populations. In this case, greater performance of the focal parasite on the sympatric host might be interpreted as local adaptation, but it could instead be the result of the sympatric host population having lower overall levels of resistance to all parasite populations. This ambiguity arises because a population (in this case, the allopatric host population) that is included in an allopatric pairing is not included in a corresponding pairing with its sympatric parasite population.

Additional aspects of experimental design may also affect the outcome of experiments testing for local adaptation between hosts and parasites. For example, local adaptation can vary across multiple spatial scales, such that the spatial scale at which an experiment is conducted can greatly affect the outcome. Furthermore, the number of different host and parasite populations that are tested in both sympatric and allopatric combinations may significantly influence the power of an experiment to detect local adaptation. As clearly explained by Kawecki and Ebert (2004), at least two host and parasite populations (or demes) should be included in a cross-infection experiment, and inclusion of additional replicate populations allows more precise estimation of the overall level of adaptive differentiation among populations, above and beyond other processes that may contribute to differentiation. Studies that include only one host or parasite population illustrate the relationship between reciprocality and replication at the population level; such experiments lack sufficient replication at the population level for comparing sympatric and allopatric performance and as a result are also lacking in reciprocality. Studies having at least the minimum number of populations, that is, experiments with multiple sympatric and allopatric pairings, can still be nonreciprocal to a degree if some populations are tested only in either sympatric or allopatric pairings. It may be, however, that the problem of nonreciprocality is gradually reduced as the total number of sympatric and allopatric pairings is increased. A final goal of our analysis was to assess the effect of experimental design features such as reciprocality, degree of replication, and spatial scale on the detection of local adaptation.

Local adaptation in host-parasite interactions has been reviewed from a variety of perspectives, but no formal, weighted meta-analysis has been used to test predictions about relationships between the degree of parasite local adaptation and aspects of experimental design or traits of host and parasite species. The importance of weighted meta-analysis is that it provides a quantitative method for integrating results across studies to assess general predictions, taking into account variation among studies in their precision and providing quantitative estimates for experimental effects and relationships among variables (Hedges and Olkin 1985; Gurevitch and Hedges 1999). Kaltz and Shykoff (1998) reviewed both theoretical and empirical work to assess the commonness of local adaptation in host-parasite systems and to synthesize proposed mechanisms for parasite local adaptation. Kawecki and Ebert (2004) also reviewed both theoretical and empirical studies, focusing especially on developing a conceptual framework and making specific recommendations for investigations of local adaptation. Van Zandt and Mopper (1998), Lively et al. (2004), and Lajeunesse and Forbes (2002) all applied formal meta-analysis to studies of local adaptation, focusing on either specific cross sections of the literature (Van Zandt and Mopper focused on 17 studies testing for adaptive deme formation in phytophagous insects, and Lively et al. analyzed 12 of their own studies on a particular snail-trematode interaction) or very specific questions (Lajeunesse and Forbes used 32 studies to test the effect of parasite host range on local adaptation). Greischar and Koskella (2007) reviewed empirical studies of local adaptation between hosts and parasites and used a vote-counting analysis to explore associations between parasite local adaptation and characteristics of host and parasite species, such as migration rates and generation times, as well as aspects of experimental design. Their review suggested an association between parasite local adaptation and relative host and parasite migration rates but no relationship between local adaptation and relative host and parasite generation times. Their observations suggest the need for a formal weighted meta-analysis on such questions, since vote-counting approaches do not take into account variation in precision and statistical power among studies and thus can be biased toward finding negative results, a problem that is exacerbated when an increasing number of studies are included (Hedges and Olkin 1980, 1985; Gurevitch and Hedges 1999).

We conducted a meta-analysis of studies across a wide variety of host-parasite interactions in order to rigorously

test multiple hypotheses for variation in parasite local adaptation and to quantitatively assess how experimental factors affect the detection of local adaptation. The overall aim of the meta-analysis was to evaluate the effect of both the biology of the organisms (species traits, such as migration rates, generation times, and complexity) and the role of the biologists (experimental design) on observed levels of local adaptation between hosts and parasites.

Methods

Literature Search

We compiled studies for the meta-analysis by searching the literature using the keywords "local adaptation," "maladaptation," "host-parasite," and "mutualism" in the ISI Web of Knowledge Web of Science database. From the hundreds of publications associated with these keywords, we selected only those that (1) report data from cross-infection experiments, including measures of performance of one or both species in an interaction, in at least one sympatric (host and parasite from the same population) and one allopatric (host and parasite from different populations) experimental combination and (2) provide sufficiently detailed information regarding the experimental design that was used. Measures of host and parasite performance were broadly classified as either infection/infectivity (e.g., symbiont abundance, parasite load, percent parasitized, and infection rate; hereafter referred to broadly as "infectivity") or virulence (e.g., host mortality, survival, biomass, and growth). We included the few studies on putatively mutualistic interactions that we found because most putative mutualisms are not thought to be fundamentally different from parasitisms (Ewald 1987; Johnson et al. 1997; Herre et al. 1999; Hochberg et al. 2000; Neuhauser and Fargione 2004), involving species that mutually exploit each other and interactions that often shift on a continuum between mutualism and parasitism. Data from one recent study (Springer 2007) were obtained directly from the author.

Data Extraction

To prevent our conclusions from being overly influenced by the results from a few studies, in cases where papers reported results from more than one independent experiment on the same pair of interacting species, we used the mean value across those experiments to generate a single case in our database, which we call a "study." When independent experiments on multiple different species interactions were reported in a single published paper, each was treated as a separate study. From each study, we collected data on mean infectivity and/or virulence in sympatric and allopatric experimental pairings. When data were presented in graphical form only, data values were estimated using the digitizing software program Engauge Digitizer (http://digitizer.sourceforge.net/). When performance was reported from more than one sympatric or allopatric pairing in a given study, we recorded the number of sympatric and allopatric pairings and then calculated the arithmetic means of the pairings within each of those two groups (sympatric and allopatric) to create overall sympatric and allopatric means for a given study. Altogether, we collected data from 27 papers containing 29 studies on 27 different species interactions, including 24 host-parasite interactions and three putative mutualisms (table 1).

For each study, we also compiled five characteristics of the species involved in each species interaction to be used as independent predictor variables in our meta-analysis: (1) the relative gene flow rates of the two species, (2) the relative generation times of the two species, (3) the taxonomic similarity of the two species, (4) the relative organismal complexity of the two species, and (5) whether the host species was a plant or an animal (table 1). Because quantitative estimates of gene flow rates, generation times, and phylogenetic distance are not readily available for most host-parasite species pairs, we created qualitative categorical variables. While these qualitative categories are somewhat arbitrary, we believe that they represent a useful starting point for such an analysis in the absence of quantitative data. In each case, our goal was to create the largest possible number of unambiguous categories. For relative gene flow rates, we created three categories: parasite gene flow rate is higher than that of host, parasite gene flow rate is lower than that of host, or parasite and host have approximately equal rates of gene flow. For relative generation times, we were able to create five categories: parasite's much faster than host's (e.g., bacterium parasite and plant host), parasite's moderately faster than host's, parasite's and host's approximately equal, host's moderately faster than parasite's, and host's much faster than parasite's. All of these qualitative determinations were made using a variety of sources of information: natural-history information and data given by the authors in the local adaptation studies themselves, other published papers or books on the same species, and/or published resources on the internet. As a proxy for phylogenetic distance between host and parasite, we created seven categories for taxonomic similarity, using commonly accepted recent taxonomic classifications from general sources (e.g., Campbell and Reece 2004).

Because models of coevolution between hosts and parasites make predictions about local adaptation based not only on relative gene flow rates and generation times of hosts versus parasites but also on the absolute values of

these traits for host and parasite species (e.g., Gandon et al. 1996), it would be ideal to include at least qualitative estimates for the latter as predictor variables in a meta-analysis such as ours. Unfortunately, quantitative estimates of absolute rates of gene flow for most organisms in our study do not exist, and absolute rates of gene flow for individual species are much more difficult to estimate on a qualitative scale based on natural-history information than are relative rates of host and parasite species. For example, in the host-parasite system studied by Curt Lively and colleagues (e.g., Lively 1989), it is relatively straightforward, using natural-history information provided by those authors, to infer that the trematode parasite *Microphallus* likely has a greater gene flow rate among populations than does its snail host, because of dispersal of parasites (and not snails) among lakes by their alternative bird hosts (as discussed by Gandon et al. [1996]). On the other hand, barring actual quantitative gene flow estimates, it would be entirely more difficult to make a qualitative categorization for absolute gene flow levels in *Microphallus* that could be meaningfully compared to those of very different parasites in other systems. Consequently, it is our opinion that until quantitative estimates of absolute gene flow rates are available for more host and parasite species, meta-analysis of how absolute rates of gene flow affect local adaptation in coevolving species interactions will not be possible. Similar arguments apply for relative versus absolute generation times of host and parasite species, although it would likely be easier (compared to gene flow) to develop meaningful estimates of absolute generation times for diverse host and parasite species.

To compute a quantitative proxy for relative host and parasite organismal complexity, we first created estimates of the number of different cell types in each host and parasite species, using the data compiled by Bell and Mooers (1997). Although Bell and Mooers do not provide estimated numbers of cell types for the specific species in our meta-analysis, they do provide a taxonomically wide-ranging set of estimates that can be used as approximations for most species. Since their initial estimates were only approximate and vary widely among taxa (see Bell and Mooers 1997 for a detailed explanation of how their estimates were obtained), information should not be lost by extrapolating to related species not included in their study. For each species in our database, we determined the most closely related species or group of species included in Bell and Mooers' study and then computed the average number of cell types for those species. We then used these estimates of number of cell types for each host and parasite species in our database to compute the log ratio of host to parasite number of cell types. This ratio is positive when hosts are more complex than their parasites and negative when the reverse is true.

We also evaluated aspects of the experimental design of each study. First, we noted whether studies were fully reciprocal or not. To be classified as fully reciprocal, a study did not need to include all possible combinations of allopatric and sympatric pairings. Rather, studies were classified as nonreciprocal if the experimental design included allopatric pairings in which either population was not also included in at least one sympatric pairing (or sympatric pairings in which either population was not also included in an allopatric pairing), since these types of pairings produce the type of nonreciprocality that can potentially lead to errors in interpreting the outcomes of experimental tests of local adaptation. When studies were nonreciprocal but included a subset of experimental treatments that were reciprocal, we used only the latter subset of data and classified those studies as reciprocal. In addition, we recorded the maximum distance between populations in allopatric pairings in each study, as a proxy for the geographical scale of the study. As mentioned above, the number of sympatric and allopatric pairings included in each study was also recorded. We used the sum of these two numbers as a proxy for the precision of the overall estimate of local adaptation in each study and thus as the weighting variable in our meta-analysis, as described below.

Meta-Analysis

Analyses were performed using a combination of Metawin, version 2.0 (Rosenberg et al. 2000), and SAS 9.1 (SAS Institute, Cary, NC). For each study, we calculated an effect size of local adaptation by parasites or mutualists to their hosts based on mean values of infectivity and/or virulence in sympatric and allopatric experimental pairings. For simplicity, and because our data set contains only a small number of species interactions classified as putative mutualisms, hereafter we refer to both the parasite and mutualist species as the "parasite" and to the other species in the interaction as the "host." Effect size of local adaptation (E) was calculated as the log response ratio of sympatric to allopatric performance: $E = \ln(X_s/X_a)$, where X_a is mean performance of the parasite in allopatric pairings and X_s is mean performance of the parasite in sympatric pairings (Rosenberg et al. 2000). This metric is positive when parasite local adaptation to hosts is observed and negative when parasite local maladaptation to hosts (i.e., local adaptation of hosts to parasites) is observed. We chose to use the log response ratio (rather than other commonly used metrics for effect size, such as Hedges' d) because it provides a standardized measure of overall performance on allopatric hosts relative to sympatric hosts, giving positive values for parasite local adaptation and

Table 1: Summary information for the studies used in the meta-analysis of local adaptation

Reference	Type[a]	Host	Parasite/mutualist	Local adaptation[b]		Rel. gen. time[c]	Rel. gene flow rate[d]	Tax. sim.[e]	Relative complexity[f]	Fully reciprocal?	Max. allopatric distance (km)	Sympatric, allopatric pairings
				Infectivity	Virulence							
Altizer 2001	P	Monarch butterfly (*Danaus plexippus*)	Protozoan (*Ophryocystis elektroscirrha*)	−.0818	.0937	2	2	2	2.53	Yes	2,271	3, 4
Capelle and Neema 2005	P	Plant (*Phaseolus vulgaris*)	Fungus (*Colletotrichum lindemuthianum*)	.00389		2	2	2	1.69	Yes	.5	2, 2
Dufva 1996	P	Great tit (*Parus major*)	Hen flea (*Ceratphyllus gallinae*)	−.182	.305	4	3	4	.79	Yes	300	2, 2
Fischer and Foitzik 2004	P	Ant (*Leptothorax acervorum*)	Ant (*Harpagoxenus sublaevis*)		.164	4	2	7	.00	Yes	250	2, 2
Ganz and Washburn 2006	P	Mosquito (*Ochlerotatus sierrensis*)	Protozoan (*Lambornella clarki*)	.710		2	3	2	2.53	Yes	61	2, 2
Gasnier et al. 2000	P	Snail (*Lymnaea truncatula*)	Trematode (*Fasciola hepatica*)	−.875	.154	2	1	5	−.31	No	1,300	2, 1
Goss and Bergelson 2006	P	Plant (*Arabidopsis thaliana*)	Bacterium (*Pseudomonas viridiflava*)	.0895		1	1	1	3.63	Yes	94.3	5, 25
Hanks and Denno 1994	P	Plant (*Morus alba*)	Scale insect (*Pseudaulacaspis pentagona*)	.190		1	1	2	−.28	Yes	.3	4, 4
Hatcher et al. 2005	P	Amphipod (*Gammarus duebeni*)	Microsporidian (*Nosema granulosis*)	.587	−.375	3	2	2	2.85	No	588	1, 3
Hoeksema and Thompson 2007	M	Plant (*Pinus contorta* var. *contorta*)	Fungus (*Rhizopogon occidentalis*	−.161	−.783	2	3	2	1.20	No	1,573	1, 2
Hoeksema and Thompson 2007	M	Plant (*Pinus radiata*)	Fungus (*Rhizopogon occidentalis*)	.584	−.0416	2	3	2	1.20	No	1,191	1, 3
Imhoof and Schmid-Hempel 1998	P	Bumblebee (*Bombus terrestris*)	Trypanosome (*Cirithidia bombi*)	.130	−.220	2	3	2	2.53	Yes	128	6, 12
Jackson and Tinsley 2005	P	Frog (*Xenopus laevis*)	Flatworm (*Protopolystoma* spp.)	.982		2	2	4	2.19	Yes	4,032	3, 6
Kaltz et al. 1999	P	Plant (*Silene latifolia*)	Fungus (*Microbotryum violaceum*)	−.418		1	3	2	1.44	Yes	166	14, 42

Karban 1989	P	Plant (*Erigeron glaucus*)	Thrips insect (*Apterotrips secticornis*)	.873		1	1	2	−.28	Yes	.5	3, 6
Koskela et al. 2000	P	Plant (*Urtica dioica*)	Plant (*Cuscuta europaea*)	.0643	.0195	2	3	6	.00	Yes	166	5, 20
Krist et al. 2000	P	Snail (*Potamopurgus antipodarum*)	Trematode (*Microphallus* spp.)	.00353		2	2	5	−.31	Yes	.01	2, 2
Laine 2005	P	Plant (*Plantago lanceolata*)	Fungus (*Podosphaera plantaginis*)	.0105		2	2	2	1.69	Yes	37	4, 12
Lively 1989	P	Snail (*Potamopurgus antipodarum*)	Trematode (*Microphallus* spp.)	.691		2	1	5	−.31	Yes	88	4, 8
McCoy et al. 2002	P	Bird (*Rissa tridactyla*)	Tick (*Ixodes uriae*)	.0680	.121	4	3	4	.61	Yes	.8	7, 7
Mutikainen et al. 2000	P	Plant (*Agrostis capillaris*)	Plant (*Rhinanthus serotinus*)	.0965	.105	4	3	6	.00	Yes	60	4, 12
Niemi et al. 2006	P	Plant (*Salix triandra*)	Fungus (*Melampsora amygdalinae*)	.543		2	2	2	1.44	Yes	722	3, 6
Oppliger et al. 1999	P	Lizard (*Gallotia galloti*)	Protozoan (*Haemogregarine* sp.)	−2.505		1	3	2	3.31	No	30	1, 4
Parker 1995	M	Plant (*Amphicarpaea bracteata*)	Bacterium (*Bradyrhizobium* sp.)	.323	−.216	1	3	1	3.63	Yes	860	8, 8
Ruhnke et al. 2006	P	Plant (*Fraxinus excelsior*)	Sawfly (*Macrophya punctumalbum*)	.00553		2	2	2	−.28	Yes	.13	4, 4
Ruhnke et al. 2006	P	Plant (*Fraxinus excelsior*)	Sawfly (*Tomostethus nigritus*)	−.00304		2	2	2	−.28	Yes	.09	8, 8
Springer 2007	P	Plant (*Hesperolinon californicum*)	Fungus (*Melampsora lini*)	.121		2	1	2	1.44	Yes	78.3	10, 90
Thrall et al. 2002	P	Plant (*Linum marginale*)	Fungus (*Melampsora lini*)	.516		1	1	2	1.44	Yes	10	6, 30
Wilkinson et al. 1996	M	Plant (*Amphicarpaea bracteata*)	Bacterium (*Bradyrhizobium* sp.)		−.194	1	3	1	3.63	Yes	1,000	21, 139

[a] Type of interaction: P = parasitism; M = mutualism.

[b] Local adaptation was calculated as the log response ratio of sympatric to allopatric performance: $E = \ln(X_s/X_a)$, where X_a is mean performance of the parasite in allopatric pairings and X_s is mean performance of the parasite in sympatric pairings. Positive values of the log response ratio indicate local parasite adaptation, and negative values indicate local parasite maladaptation.

[c] Relative generation time: 1 = much faster in parasite/mutualist (P/M) than in host; 2 = faster in P/M than in host; 3 = approximately equal in P/M and host; 4 = faster in host than in P/M; 5 = much faster in host than in P/M.

[d] Relative gene flow rate: 1 = greater in P/M than in host; 2 = approximately equal in P/M and host; 3 = greater in host than in P/M.

[e] Taxonomic similarity: 1 = host and P/M extremely divergent (e.g., prokaryote vs. eukaryote); 2 = animal/fungus vs. plant/alga/protist; 3 = fungus vs. animal or protist vs. plant; 4 = different subkingdoms within a kingdom (e.g., vertebrate vs. invertebrate animal); 5 = different phyla within a subkingdom; 6 = different classes/orders/families within a subkingdom; 7 = different genera within a family or subfamily.

[f] Relative complexity was calculated as $\ln(CH/CP)$, where CH and CP are the estimated numbers of cell types in the host and parasite bodies, respectively. Numbers of cell types were estimated for each species using the means for closest relatives given by Bell and Mooers (1997).

negative values for parasite maladaptation, and because log response ratios have been determined to have particularly favorable statistical properties for meta-analysis (see Hedges et al. 1999 for a detailed analysis of the merits of log response ratios for meta-analysis). The log response ratio of sympatric to allopatric fitness does not have units and therefore is a relative measure that can be compared among studies.

We calculated the overall level of local adaptation (weighted cumulative effect size E, that is, the log response ratio of local adaptation), with each study's effect size estimate weighted by the number of sympatric and allopatric pairings included in that study, as well as a corresponding 95% confidence interval (van Houwelingen et al. 2002). We explored the effect of species traits and experimental design on effect size of local adaptation, using multifactor statistical models. In analyses of species interaction traits, the multifactor model included only the main effects of the five factors (relative generation times, relative gene flow rates, taxonomic similarity, relative complexity, and plant vs. animal host) because the degrees of freedom in the data set did not support a more complex model that included all of the two-way interactions. In analyses of experimental design factors, the model contained only the main effects of experimental reciprocality and maximum allopatric distance (log transformed) because there were too few studies in the nonreciprocal group to meaningfully test the interaction effect, that is, the difference in slopes of the maximum allopatric distance effect between reciprocal and nonreciprocal studies.

These multifactor models were fitted to the data using weighted maximum likelihood estimation of parameters in a mixed-model framework, using SAS procedure MIXED, in which the species traits and experimental design factors were considered fixed effects, and a random between-studies variance component was estimated. Mixed-effect models have the advantage of not assuming that all studies being compared share a common true effect size, and thus the results can be generalized beyond the studies included in the analysis (Sokal and Rohlf 1995; Underwood 1997; Rosenberg et al. 2000). Maximum likelihood methods allow simultaneous estimation of the between-studies variance component and the coefficients associated with each independent variable. Significance (at $\alpha = 0.05$) of individual factors in these models was determined using randomization procedures with 10,000 iterations (performed with a combination of macros in SAS), in which effect sizes and their weights were randomly permuted among levels of the independent variables (as discussed by Adams et al. [1997]). Randomization tests, when combined with a mixed-model approach to analysis, provide appropriate controls on Type I error rates in multifactor meta-analysis (Higgins and Thompson 2004). Our SAS code, which is available from the authors on request, was modified from code suggested by van Houwelingen et al. (2002). The effects of each individual independent variable were further characterized using separate one-way mixed-model analyses in Metawin 2.0. These one-way analyses utilized least squares estimation of parameters, and the model sum of squares was obtained for each factor to quantify the heterogeneity in effect size explained by that factor (Q_M). To assess the proportion of total heterogeneity in effect size explained by each factor, we divided Q_M by Q_T, the total heterogeneity in effect size. This ratio (Q_M/Q_T) is analogous to the coefficient of determination (R^2) from ANOVA. For categorical variables, we calculated effect sizes and bootstrapped, bias-corrected 95% confidence intervals for each category, and for continuous variables, we calculated bias-corrected standard errors around regression slope and intercept estimates (Rosenberg et al. 2000).

Although we collected data on local adaptation of parasite virulence, we did not analyze the effect of species traits or experimental design factors on local adaptation of parasite virulence, for two reasons. First, many fewer studies reported virulence data, compared to infectivity data, and thus replication for most of the desired analyses was insufficient. Second, infectivity is actually the parasite performance measure that is predicted by theory to be the target of selection for local adaptation (Dybdahl and Storfer 2003), whereas virulence is predicted to vary among systems and to influence the degree of local adaptation in infectivity (Lively 1999; Gandon 2002). Thus, we report data on local adaptation in virulence for the studies that provided virulence data (table 1), but we focus our meta-analysis of the effects of species traits and experimental design factors on local adaptation of parasite infectivity. For analysis of the effect of species interaction traits on local adaptation of infectivity, all analyses were performed on two different subsets of the database: the full set of studies and the subset of studies having reciprocal experimental designs. We consider the latter subset to be the more conservative and informative analysis, given the potential for misinterpretation of data from nonreciprocal cross-infection studies, as explained above. We also tested whether our results differed when we excluded the studies on putative mutualisms. We tested for publication bias in our database by examining funnel plots of effect size versus variance and number of replicates and by calculating correlations between effect size and variance.

Results

Of the 29 studies that fit the criteria for inclusion in our analysis, 27 reported response variables that we classified as infectivity, while only 13 reported response variables

that we classified as virulence. Five studies were classified as nonreciprocal (table 1). We included only four studies on three different species interactions that are considered putative mutualisms. We performed all analyses both with and without these studies on mutualisms and found that the results did not differ qualitatively, perhaps because of the small number of mutualisms in the data set, and that the estimates of local adaptation in these interactions did not represent outliers in any analysis. Thus, we present results from analysis of the full data set (both host-parasite and mutualistic interactions). Examination of funnel plots and the results of correlation tests between effect size and variance did not indicate significant publication bias in our data set.

Across the 27 studies reporting infectivity data, the overall weighted effect size of local adaptation of parasites to their hosts was positive but not significantly different from 0 ($E = 0.117$, 95% confidence interval [CI] $= -0.093$ to 0.328). The maximum likelihood estimate of the between-studies variance component was 0.1736 (95% CI = 0.068–0.696). When nonreciprocal studies were excluded from the analyses, the overall weighted effect size of local adaptation by parasites was positive and significantly greater than 0 ($E = 0.184$, 95% CI = 0.0264–0.342). The maximum likelihood estimate of the between-studies variance component was 0.0516 (95% CI = 0.0205–0.288).

Effects of species traits. In the full data set (i.e., including nonreciprocal studies), none of the independent variables on species traits explained significant variation in local adaptation of parasite infectivity. For relative gene flow rate, $Q_M = 3.53$, df = 2, $P = .226$; effect size for parasites > hosts = 0.301, 95% CI = 0.0103–0.5667, $n = 7$; effect size for parasites = hosts = 0.224, 95% CI = 0.0240–0.5037, $n = 9$; and effect size for parasites < hosts $= -0.0916$, 95% CI $= -0.5834$ to 0.1973, $n = 11$. For relative generation time, $Q_M = 0.152$, df = 1, $P = .404$, slope $= 0.0432 \pm 0.112$ (SE), and intercept $= 0.0348 \pm 0.236$ (SE). For taxonomic similarity, $Q_M = 0.029$, df = 1, $P = .843$, slope $= 0.0111 \pm 0.0653$ (SE), and intercept $= 0.0882 \pm 0.204$ (SE). For relative complexity, $Q_M = 0.599$, df = 1, $P = .869$, slope $= -0.0581 \pm 0.0751$ (SE), and intercept $= 0.1936 \pm 0.137$ (SE). For plant versus animal host, $Q_M = 0.392$, df = 1, $P = .544$; animal effect size = 0.0338, 95% CI $= -0.5892$ to 0.4353, $n = 11$; and plant effect size = 0.1625, 95% CI = 0.0113–0.3179, $n = 16$.

When nonreciprocal studies were excluded from the analyses, the relative gene flow rate of the host and parasite had the most substantial effect on parasite local adaptation ($Q_M = 3.553$, df = 2, $P = .0295$; fig. 1), accounting for a significant proportion of the observed heterogeneity in local adaptation among studies ($Q_M/Q_T = 0.172$). Specifically, parasites exhibited significant local adaptation to hosts only in studies in which parasites had greater rates of gene flow than their hosts; local adaptation was positive but not significantly different from 0 when parasites and their hosts had approximately equal rates of gene flow, and local adaptation was close to 0, with a CI ranging widely into maladaptation, when hosts had greater rates of gene flow than parasites (fig. 1). None of the other independent variables explained significant variation in effect size (fig. 2). For relative generation time, $Q_M = 0.262$, df = 1, $P = .469$, slope $= -0.0458 \pm 0.0895$ (SE), and intercept $= 0.276 \pm 0.181$ (SE). For taxonomic similarity, $Q_M = 0.0887$, df = 1, $P = .166$, slope $= 0.0154 \pm 0.0517$ (SE), and intercept $= 0.146 \pm 0.162$ (SE). For relative complexity, $Q_M = 0.0003$, df = 1, $P = .562$, slope $= -0.0011 \pm 0.0630$ (SE), and intercept $= 0.190 \pm 0.113$ (SE). For plant versus animal host, $Q_M = 0.939$, df = 1, $P = .167$; animal effect size = 0.309, 95% CI = 0.0570–0.6150, $n = 8$; plant effect size = 0.140, 95% CI $= -0.0361$ to 0.3020, $n = 14$.

Effects of experimental design. As described above, we found different effects of species traits on local adaptation

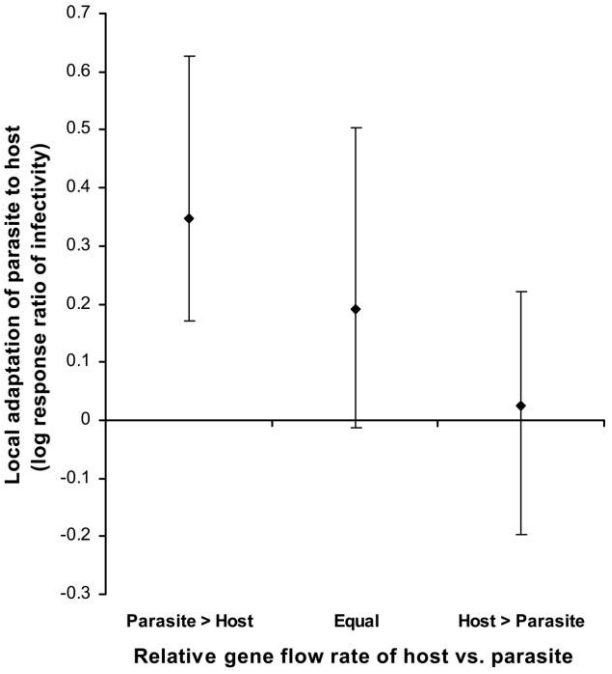

Figure 1: Local adaptation of parasites to their hosts varies as a function of the estimated relative gene flow rates of host versus parasite species ($Q_M = 3.553$, df = 2, $P = .0295$, $Q_M/Q_T = 0.172$, where Q_M is heterogeneity in effect size explained by a given factor and Q_T is total heterogeneity in effect size). Data shown are weighted mean effect sizes from reciprocal experiments only (parasite > host: $n = 6$; equal: $n = 8$; host > parasite: $n = 8$). Error bars represent a bootstrapped, bias-corrected 95% confidence interval.

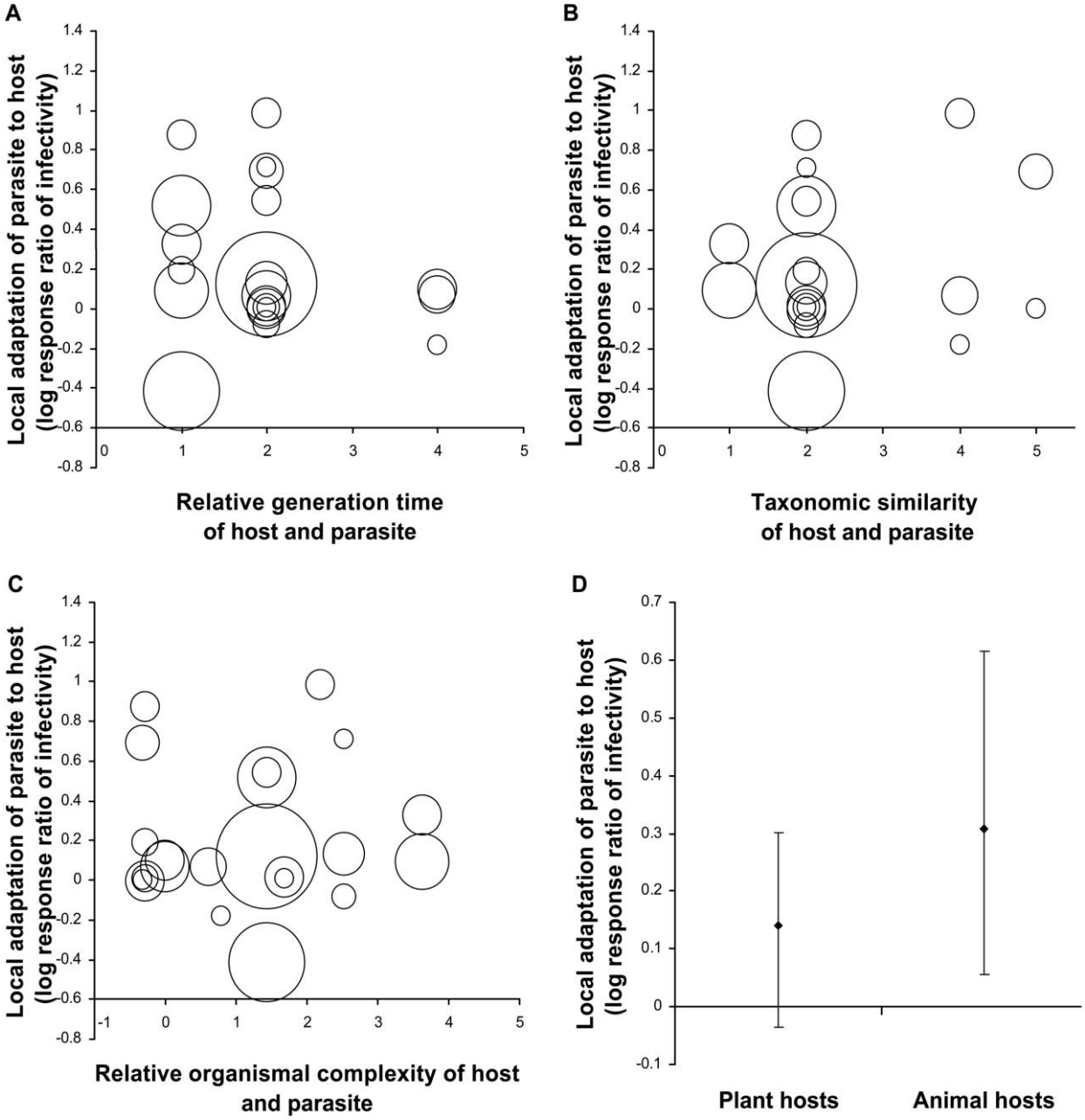

Figure 2: Local adaptation of parasites to their hosts as a function of (*A*) relative generation time of the parasite and host, (*B*) taxonomic similarity of the parasite and host, (*C*) relative organismal complexity of the parasite and host, and (*D*) plant versus animal hosts (plant hosts: $n = 14$; animal hosts: $n = 8$; error bars represent a bootstrapped, bias-corrected 95% confidence interval around the cumulative weighted mean effect size). Data shown are from reciprocal experiments only. In *A*–*C*, circle size is proportional to weight in meta-analysis, that is, the number of sympatric plus allopatric pairings, and larger values on the *X*-axis indicate that the host has a faster generation time than the parasite, is more taxonomically similar to the parasite, or is more complex than the parasite, respectively.

in reciprocal versus nonreciprocal experiments. In addition, reciprocality as an independent variable explained significant variation in the effect size of local adaptation in the full data set ($Q_M = 6.23$, df $= 1$, $P = .0216$, $Q_M/Q_T = 0.197$), with local adaptation being significantly lower (and having a much wider 95% CI) in the nonreciprocal studies than in the reciprocal studies (fig. 3). The natural log of maximum allopatric distance did not explain

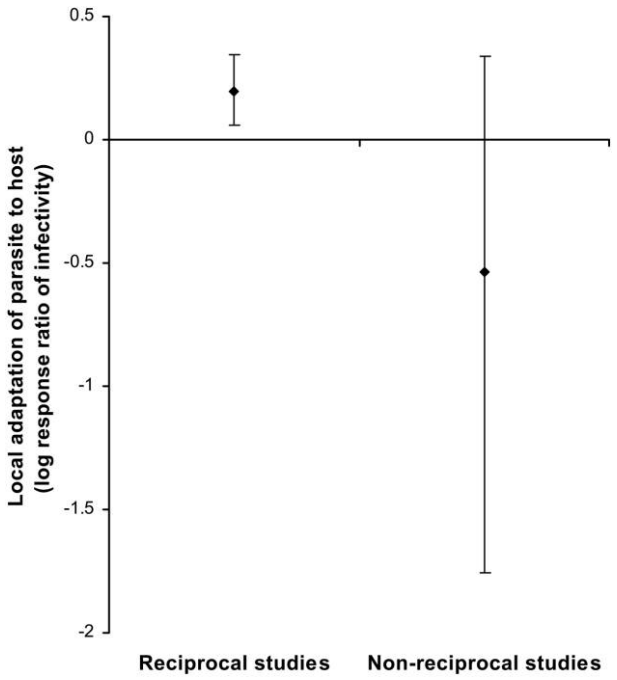

Figure 3: Local adaptation of parasites to their hosts differs between reciprocal and nonreciprocal studies ($Q_M = 6.23$, df = 1, $P = .0216$, $Q_M/Q_T = 0.197$, where Q_M is heterogeneity in effect size explained by a given factor and Q_T is total heterogeneity in effect size). Data shown are weighted mean effect sizes from the full data set (reciprocal studies: $n = 22$; nonreciprocal studies: $n = 5$). Error bars represent a bootstrapped, bias-corrected 95% confidence interval.

significant variation in the effect size of local adaptation ($Q_M = 0.0813$, df = 1, $P = .352$, slope = 0.0085 ± 0.0298 [SE], intercept = 0.0885 ± 0.144 [SE]).

Discussion

Host-Parasite Trait Differences and Local Adaptation

Our results indicate that the relative gene flow rate of host and parasite species is the strongest predictor, among the variables we tested, of local adaptation in parasite infectivity. In some ways, this result is consistent with recent theory on the effect of gene flow on local adaptation in coevolving-species interactions. Some amount of gene flow among populations may enhance local adaptation by providing genetic variation on which local selection can act, whereas greater levels of gene flow may inhibit local adaptation by swamping local selection (Slatkin 1985; Gandon et al. 1996; Gandon 2002; Gandon and Michalakis 2002; Garant et al. 2007). In coevolutionary interactions specifically, when two species are both under selection to adapt to the other species, the relative gene flow rates of the two species may be more relevant than the absolute rate of either species (Gandon et al. 1996; Gandon and Michalakis 2002), although the absolute rates are also important. Specifically, parasite local adaptation to hosts is predicted to be strongest when parasite gene flow rates are higher than host gene flow rates and absolute levels of gene flow in the host are low overall (Gandon et al. 1996). Our results are consistent with this prediction, in that we observed significant parasite local adaptation in systems in which parasites were estimated to have higher gene flow rates than their hosts.

Some models also predict that host local adaptation to parasites should be strongest (i.e., parasites should be maladapted to their hosts) when hosts have greater gene flow rates than parasites, especially when absolute parasite gene flow rates are low overall (Gandon et al. 1996). In our analysis, however, in studies in which hosts had greater gene flow rates than parasites, parasites exhibited wide variation in local adaptation, ranging from parasite local adaptation to strong maladaptation (fig. 1). There are at least three potential explanations for the high variability in local adaptation among systems in this subgroup of studies. One possibility is that in many systems in which hosts have greater gene flow rates than their parasites, absolute rates of parasite gene flow are also quite high, preventing host local adaptation (Gandon et al. 1996). Unfortunately, quantitative estimates of absolute rates of gene flow in host and parasite species are not available for most systems and are not as easily estimated in a qualitative fashion as relative rates of host versus parasite gene flow. Thus, we could include only the latter in our analysis. As more data become available on absolute gene flow rates in host and parasite species, it will be ideal to test simultaneously the effects of both absolute and relative host and parasite gene flow rates on local adaptation. A second potential explanation is that factors other than gene flow, such as the relative generation times of hosts and parasites or spatial variation in the degree of coevolutionary selection (i.e., a selection mosaic sensu Thompson 1994, 2005) may influence local adaptation, adding to the variation in effect size observed within categories of relative host versus parasite gene flow. For example, recent theoretical studies have shown that relative gene flow rates of hosts and parasites may in some circumstances be much less important for local adaptation than selection mosaics (Nuismer 2006) and that parasite maladaptation should be relatively uncommon when host-parasite interactions are governed by polygenic traits (Ridenhour and Nuismer 2007). Third, although the confidence interval we calculated for local adaptation in studies where hosts have greater gene flow rates than parasites overlaps 0, it does include a significant region of parasite maladaptation (fig. 1); it may be that variability in local adaptation among systems in this category is greater than that in the other categories but that

parasites in this category are more often maladapted to their hosts than those in other categories of host-parasite interactions. In this case, a larger sample of studies will be required to more accurately estimate local adaptation in this subset of systems.

If parasite local adaptation is most often observed to be strongest when gene flow rates of parasites are greater than those of hosts, as suggested by our results, then it may be that local adaptation of parasites to their host species is often limited by genetic variation. Thus, it may be that the net effect of parasite gene flow is often to provide genetic variation on which local selection can act, rather than to swamp selection for locally adapted parasite genotypes. It is also possible, however, that we did not see evidence of gene flow swamping adaptation because of publication bias. It is likely that some results indicating no evidence of adaptation or of maladaptation (e.g., no difference in infectivity of a parasite between sympatric vs. allopatric hosts) go unpublished, and these results could be due to high levels of gene flow. On the other hand, the studies included in our analysis showed a wide range of results with respect to adaptation, including negative results and maladaptation, and graphical and statistical tests showed no evidence of publication bias. Furthermore, our results are consistent with the conclusions of the recent review of local adaptation studies by Greischar and Koskella (2007). They found that studies of systems in which parasites were estimated to have greater gene flow rates than their hosts more often report significant parasite local adaptation, while the reverse prediction—a greater likelihood of studies reporting significant parasite maladaptation when hosts have greater gene flow rates than parasites—did not hold.

Parasites have often been predicted to have an evolutionary advantage over their hosts because of faster generation times and thus faster evolutionary rates (Price 1980; Ebert and Hamilton 1996; Kaltz and Shykoff 1998). Our data, however, suggest that other factors may be more important in predicting current variation in local adaptation among systems or that relative generation times may have variable effects on local adaptation. Indeed, recent theory has shown ways in which faster generation times can result in an evolutionary disadvantage in some circumstances. For example, Gandon and Michalakis (2002) showed how, when mutation and migration limit genetic variability in parasite populations, faster parasite generation times can serve to deplete genetic variation, preventing further adaptation to a changing environment. In addition, it is important to note that we found relatively few studies in which hosts had greater generation times than their parasites (table 1). There may be little variation among systems in the relative generation times of host and parasites, and thus surveys across existing species interactions may lend little insight into the evolutionary importance of relative host and parasite generation times. Greischar and Koskella (2007) also found no evidence for an association between parasite local adaptation and relative host and parasite generation times.

Additional insight into the importance of relative host and parasite generation times and gene flow rates for coevolutionary dynamics must come from experimental systems in which these variables can be experimentally manipulated. In one such recent experiment, Morgan and Buckling (2006) indirectly manipulated generation times in a microbial host-parasite system and found that the relative number of generations of each species did not influence parasite local adaptation. Forde et al. (2004) and Morgan et al. (2007) used phage-bacterium interactions to show that changes in simultaneous migration rates of the host and the parasite can have a significant influence on parasite local adaptation. Independent manipulations of host and parasite migration demonstrated that parasite migration results in parasite local adaptation, but host migration alone had no effect (Morgan et al. 2005).

We found no evidence that parasite local adaptation is significantly influenced by the taxonomic similarity of the host and parasite or by the relative complexity of the host and parasite. Although the latter idea is grounded in a plausible theoretical argument—that less complex species should be able to adapt faster than more complex species (Orr 2000; Welch and Waxman 2003)—the trend we observed is in the opposite direction (fig. 2*C*). Thus, if there is a relationship between local adaptation and relative host and parasite complexity, additional explanations should be sought beyond the cost-of-complexity hypothesis. For example, it may be that the cost-of-complexity argument does not apply to adaptation in the context of many coevolutionary species interactions. If the genetic basis of a species interaction is very simple and involves similar numbers of loci and alleles for the host and parasite species, then the overall complexity of either species may be irrelevant to the expected ability to adapt to the other species. We also found little evidence to support the prediction that local adaptation of parasites to animal hosts should be greater than local adaptation to plant hosts because of expected differences in the genetic control of these two types of interactions (Parker 1994; Agrawal and Lively 2002; but see Frank 1993 regarding the difficulty of empirically distinguishing between the two models of genetic control). In the full data set, the trend we observed was in the opposite direction, with a tendency toward greater local adaptation of parasites to plant hosts than to animal hosts. In the reciprocal subset of experiments, the trend was in the predicted direction, but with the two groups exhibiting widely overlapping confidence intervals (fig. 2*D*).

Experimental Design and Local Adaptation

Our main finding with respect to experimental design was that eliminating nonreciprocal experiments, which constituted approximately one-fifth of the studies, allowed us to detect an influence of relative host and parasite gene flow rates on local adaptation. This result was somewhat surprising because, although the results of nonreciprocal experiments can be difficult to interpret (see Thrall et al. 2002), one might predict that the aggregate results of multiple experiments of this kind should reasonably approximate the results of reciprocal experiments. It may be, however, that the nonreciprocal experiments added disproportionate variation to our meta-analysis because results on local adaptation from such experiments can also be heavily influenced by overall differences (e.g., of average host resistance) among populations, regardless of the true level of local adaptation. Alternatively, other factors that differ between the reciprocal and nonreciprocal subsets of studies could be contributing to the higher variability observed in the nonreciprocal studies. For example, studies with animal hosts, which exhibited higher variability than those with plant hosts, are represented to a greater degree among the nonreciprocal studies.

We observed a wide range of variation in experimental designs with respect to the host-parasite pairings that were utilized from those that were theoretically possible, and we recognize that such choices are sometimes not under the control of the experimenter because of logistical and other constraints. One common constraint is that as additional populations are added to a study, the number of possible allopatric combinations grows at an increasing rate, rapidly making it more difficult to include all possible combinations of host and parasite populations with adequate replication. One efficient solution to this problem was utilized by three different studies in our analyses: Parker (1995), Kaltz et al. (1999), and Ruhnke et al. (2006). All three of these studies maintained a reciprocal design in which all populations were represented in both sympatric and allopatric pairings, but they used an efficient subset of all possible allopatric combinations. This approach allows a larger number of populations to be included without sacrificing the reciprocality that is essential for unambiguous interpretation of results. As larger numbers of sympatric and allopatric pairings are added to a study, it may be that the importance of reciprocality is reduced; it would be useful to explore this possibility using simulated data sets and a variety of experimental designs.

Perhaps surprisingly, we did not find that the spatial scale of the study had any influence on the degree of local adaptation detected. The distance among allopatric populations has been predicted to correlate negatively with adaptation, especially when physical distance corresponds to genetic distance among those populations (Gandon et al. 1996). Specifically, if more distant host populations are more genetically differentiated, parasites are predicted to be more adapted to hosts from nearby populations than to those that are further away. Thus, larger-scale studies should be more likely to detect a signal of local adaptation than smaller-scale studies, all else being equal. On the other hand, there are several reasons why we might not expect to find general relationships between the spatial scale of experiments and local adaptation. First, geographic distance affects gene flow of both the host and the parasite, and as our results suggest, relative gene flow rates of the host and parasite may be more relevant to local adaptation than absolute rates. Second, local adaptation may occur at multiple scales, and researchers may tend to study interactions at the spatial scales where they expect to find local adaptation; thus, a comparative approach across published studies may not be appropriate for testing the relationship between spatial scale and local adaptation. Rather, a comparison of local adaptation across different spatial scales within individual systems would be more informative. Third, the relationship between genetic distance and geographic distance may vary greatly among study systems; for example, to the extent that a host-parasite interaction is governed by a small number of loci and alleles, a long, gradual spatial gradient in host-parasite compatibility may not even be theoretically possible. Finally, in systems where local adaptation does vary with the spatial scale of the study, regression of host and parasite fitness against the distance between populations in experimental pairings, using the slope of this regression as an estimate of the degree of local adaptation, may be more powerful than a pairwise sympatric-allopatric ANOVA approach for detecting local adaptation (e.g., Ebert 1994; Thrall et al. 2002; Hoeksema and Thompson 2007).

Although asymmetries in characteristics between species have long been thought to influence their coevolution, empirically testing these theoretical predictions requires a comparative approach across a diverse collection of natural systems to include the full continuum of traits. Further, experimental design must be seriously considered if we are to accurately evaluate patterns of local adaptation. Additional individual studies of local adaptation are still badly needed, especially on species interactions that are putatively mutualistic or span the mutualism-parasitism continuum, on interactions with relatively rare combinations of host and symbionts traits (such as hosts having faster generation times than their parasites), and in systems in which species traits can be directly manipulated (but see Forde et al. 2004; Morgan et al. 2005, 2007; Morgan and Buckling 2006). Furthermore, we need additional information about the traits of hosts and parasites, such as quantitative estimates of absolute gene flow rates, and the

genetic basis of traits governing the interactions. Our results represent progress in using the comparative approach to understand variability in local adaptation and suggest that host and parasite trait differences do influence the outcome of local adaptation in species interactions.

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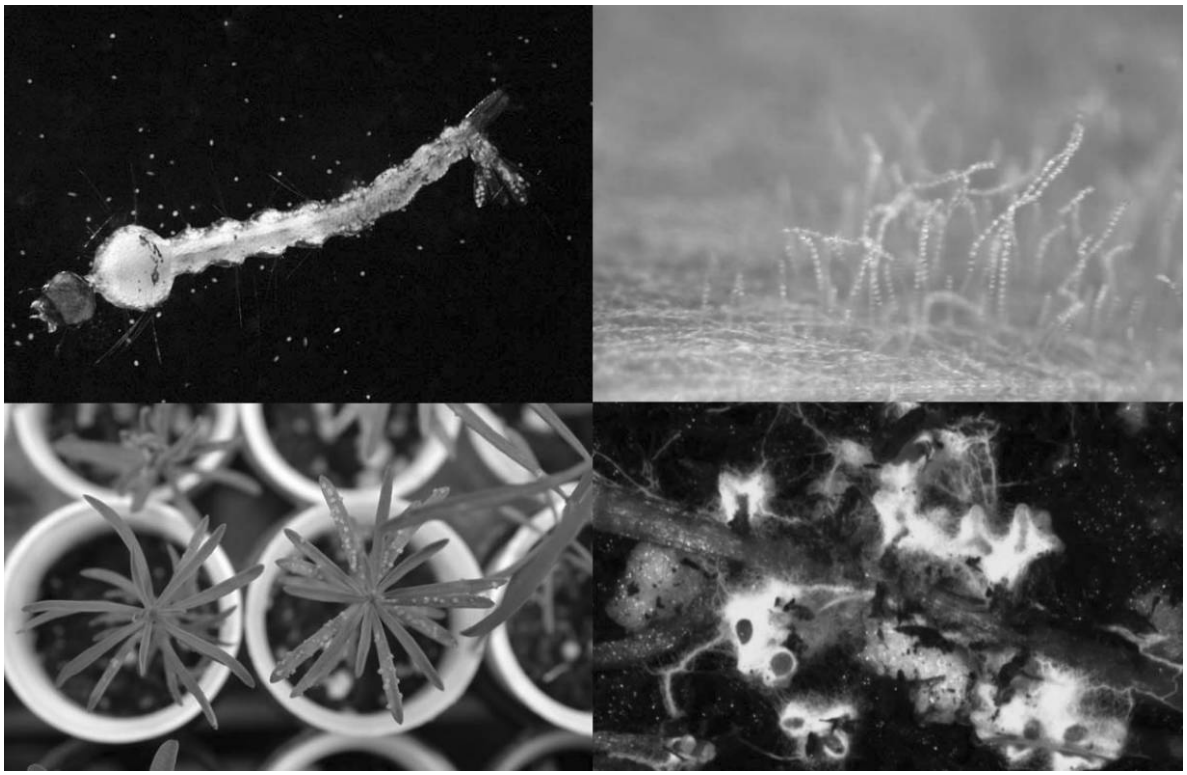

Host-parasite interactions included in the comparative study by Hoeksema and Forde, clockwise from top left: protozoan parasite *Lambornella clarki* and its mosquito host *Ochlerotatus sierrensis* (photograph by Holly Ganz), fungal parasite *Podosphaera plantaginis* and its plant host *Plantago lanceolata* (photograph by Anna-Liisa Laine), fungal parasite *Melampsora lini* and its plant host *Hesperolinon californicum* (photograph by Yuri Springer), fungal mutualist *Rhizopogon occidentalis* and its plant host *Pinus radiata* (photograph by Jason Hoeksema).