Ecology Letters, (2007) **10**: 418–434 doi: 10.1111/j.1461-0248.2007.01028.x

REVIEW AND SYNTHESIS

A synthesis of experimental work on parasite local adaptation

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Abstract

The study of parasite local adaptation, whereby parasites perform better on sympatric hosts than on allopatric hosts and/or better on their own host population than do other parasites, is of great importance to both basic and applied biology. Theoretical examination of host–parasite coevolution predicts that parasite migration rate, generation time and virulence all contribute to the pattern of parasite local adaptation, such that parasites with greater dispersal ability, more frequent reproduction and/or high virulence ought to exhibit increased infectivity on local hosts. Here, we present a meta-analysis of experimental work from 57 host–parasite systems across 54 local adaptation studies to directly test theoretical predictions concerning the effect of each attribute on parasite adaptation. As expected, we find that studies of parasites with higher migration rates than their hosts report local adaptation, as measured by infection success, significantly more often than studies of parasites with relatively low migration rates. Furthermore, this synthesis serves to identify biases in the current body of work and highlight areas with the greatest need for further study. We emphasize the importance of unifying the field with regard to experimental methods, local adaptation definitions and reported statistics for cross-infection studies.

Keywords

Coevolution, generation time, meta-analysis, migration, parasites, virulence.

Ecology Letters (2007) 10: 418–434

INTRODUCTION

The spatial and temporal variation typical of antagonistic interactions between hosts and their parasites offers a unique opportunity to examine coevolutionary dynamics. Specifically, the continuous selection imposed by parasites on their hosts and vice versa is predicted to lead to parasite local adaptation, whereby sympatric host–parasite combinations result in higher infection success than do allopatric combinations. The dynamic nature of local adaptation forms the basis for many current theories in evolutionary biology, including predictions concerning the maintenance of sexual reproduction (Jaenike 1978; Hamilton *et al.* 1990; Busch *et al.* 2004) and genetic diversity within and among populations (Haldane 1949; Hutson & Law 1981; Hamilton 1993).

The process of coevolution requires reciprocal, but not necessarily symmetrical, selection pressure such that parasites may gain more in terms of fitness through successful infection than hosts lose by sustaining an infection. Furthermore, the ability of hosts and parasites to coevolve depends both on the strength of selection and the adaptive genetic variation available within each of the populations (Gandon & Michalakis 2002). As parasites are under strong selection and have an adaptive advantage due to their typically shorter generation times, larger population sizes and higher rates of migration, they are predicted to be adapted to their local host populations (Price 1980; Ebert 1994; Gandon & Michalakis 2002).

The numerous host–parasite systems utilized for cross-infection studies differ with regard to impact of infection on host fitness, parasite life cycle and dispersal abilities of each species. This large body of experimental work is therefore ideal for comparing the effects of various host and parasite characteristics on the ability of a population to adapt to its local environment. Examining the importance of factors like migration rate has proven problematic, as it has rarely been possible to do so within a single host–parasite system (but see Forde *et al.* 2004; Morgan *et al.* 2005), and the results of such experiments may not be relevant to other systems. Although formal meta-analyses have proven useful at quantifying trends in local adaptation for a small subset of

organisms, e.g. insect herbivores (Van Zandt & Mopper 1998) and a snail-trematode system (Lively *et al.* 2004), they have not been applied to the body of work as a whole. Thus far, the only comprehensive reviews of local adaptation have focused either on qualitative discussion of experimental studies (Kaltz & Shykoff 1998) or quantitative synthesis for a single parasite trait: host specificity (Lajeunesse & Forbes 2002).

In the present meta-analysis, we build upon past work by utilizing the current body of local adaptation studies to statistically examine the effects of relative migration rates, relative generation times and virulence on parasite local adaptation. An examination of the data reported across local adaptation studies revealed that, to use *P*-values or other reported statistics in calculating the effect size for each study, i.e. the magnitude of the observed local adaptation result (as described by Rosenthal 1994), a prohibitive number of studies would need to be excluded from analysis. Therefore we chose to group studies into two-by-two contingency tables, based on biologically meaningful categories, and use Fisher's exact tests to quantify trends across the current body of empirical work.

Defining parasite local adaptation

A recent review highlighted the widespread use of two different ways of examining parasite local adaptation: (1) comparing parasite performance on 'at home' vs. 'away' hosts; or (2) comparing 'local' parasite performance with 'foreign' parasite performance on a given host population (Kawecki & Ebert 2004). When host populations differ in their degree of resistance to infection or when parasite populations differ in their ability to infect, the two definitions may give conflicting results (Thrall *et al.* 2002; Kawecki & Ebert 2004). Although a substantial number of authors address both definitions in their analyses, many report statistical results for only one definition.

The issue is further complicated by certain experimental designs that require definition-specific statistical comparisons and are therefore not equipped to detect host or parasite main effects. This is problematic as main effects indicate potentially important differences among host and parasite populations in overall susceptibility and infectivity, respectively. Where such differences exist, the conclusions reached concerning local adaptation may depend on the definition used for the statistical analysis, making comparison across studies troublesome. Furthermore, it has been argued that three or more populations are necessary to separate local adaptation from host or parasite main effects (Kawecki & Ebert 2004), and yet many studies use only one or two sympatric host–parasite combinations. To address these potential problems, we tested whether the number of populations examined and the presence or absence of main effects changed the pattern of local adaptation detected under each definition.

Predictions for parasite local adaptation

Given their assumed adaptive advantage, conventional wisdom holds that parasites ought to be, on average, locally adapted to their hosts. It remains unclear, however, if and how this pattern is influenced by significant levels of migration among populations. Classic models suggest that high levels of host or parasite gene flow will homogenize populations over time, thereby reducing the level of local adaptation (Holt & Gomulkiewicz 1997). There is empirical evidence for such a constraint from a sunfish-salamander system, in which populations linked by high levels of gene flow were maladapted with regard to their ability to escape from predation (Storfer 1999). More recent theoretical examination, however, suggests that when parasite gene flow is greater than that of the host, parasite local adaptation is expected to be more pronounced due to the inherent increase in genetic variation of parasite populations, and thus the increased efficacy of selection (Gandon *et al.* 1996; Lively 1999; Gandon 2002). Given these predictions, we expected that studies of parasites with relatively high migration rates would report parasite local adaptation more often than studies of parasites with migration rates equal to or less than those of their hosts.

It has also been suggested that as parasites typically have shorter generation times than their hosts they are able to adapt more readily to changes in host populations (Price 1980), thereby increasing the degree of local adaptation. In contrast to this common assumption, theoretical work suggests that parasite generation time, relative to that of the host, does not inherently alter the pattern of local adaptation (Lively 1999; Gandon & Michalakis 2002). This latter prediction is based on the idea that, while parasites with shorter generation times may have a faster absolute rate of adaptation, the overall coevolutionary pattern is similar to that of parasites with longer generation times and thus local adaptation remains prevalent (Lively 1999). Other simulations indicate that migration and mutation rates, as the primary factors determining the degree of parasite local adaptation, would mask any smaller effects of parasite generation time (Gandon & Michalakis 2002). We tested the prediction that relative generation time has little to no effect on local adaptation by comparing parasites with shorter generation times than their hosts to those with equal to or longer generation times than their hosts.

In contrast to the small effect of generation time, the degree to which a parasite reduces host fitness is predicted to have a large impact on coevolutionary dynamics. In this synthesis, we use the term virulence to denote a decrease in host fitness due to infection; this usage is in contrast to plant

pathology literature, in which virulence is indicative of infection success (Thrall *et al.* 2002). In particular, parasite local adaptation is expected more often for highly virulent parasites, i.e. those that significantly increase host mortality or induce sterility, because they exert a stronger selective force on their hosts (Lively 1999; Gandon 2002). This strong selection drives divergence between host populations and subsequently leads to large differences in parasite performance on sympatric vs. allopatric hosts. We therefore expected that studies involving parasites that kill or sterilize their hosts would report local adaptation more often than studies considering less virulent parasites. Similarly, as obligate parasites must live in or on susceptible hosts to complete their life cycle, they should be under greater selection pressure than non-obligate parasites to successfully infect local hosts. As a result, we expected studies of obligate parasites to report local adaptation more often. Lastly, we predicted that vertically transmitted parasites, due to their intimate association with a subset of host genotypes, would be locally adapted more often than horizontally transmitted parasites.

To address the predictions outlined above, we performed a thorough search of the literature for studies examining parasite local adaptation. For each paper considered, we recorded the pattern of local adaptation and collected information on the host–parasite system, treating each experiment as an independent data point. We found that studies involving parasites with high migration rates relative to their hosts were significantly more likely to report local adaptation, while the other factors examined had little to no effect on local adaptation.

METHODS

Literature searches and inclusion criteria

We began the exhaustive literature search by scouring cross-citations from reviews and local adaptation studies. More recent papers (2004–present) were gathered from searches using the ISI Web of Knowledge (2006) and Google™ Scholar (search terms included local adaptation and parasite, parasite specialization, host resistance and local, cross-infection, cross-inoculation, host specificity and population). A few additional studies were gathered from colleagues. We used a broad definition of parasitism that includes mutualist species and insect herbivores, but excludes predator–prey interactions. Studies were incorporated only if they gave a statistical analysis of parasite local adaptation among different populations of a host species (as opposed to *inter*specific local adaptation studies).

We discarded studies that either experimentally manipulated migration rate, did not include an experimental cross-inoculation of parasites, or provided insufficient statistical analyses to make a determination of local adaptation. This last group included two studies that only reported, means of parasite performance (He *et al.* 1991; Carlsson-Granér 1997) and one study that reported only means and standard deviations (Vera *et al.* 1990). Although there is a convention for judging two means to be significantly different based on standard deviation intervals (Browne 1979), there is no corresponding convention for judging the difference to be non-significant. In other words, although we could determine which populations were locally adapted or maladapted, we were unable to evaluate, with confidence, which populations were *neither* locally adapted nor maladapted, and thus we omitted this study.

To characterize the pattern of local adaptation, we focused on the two most common measures of parasite performance: infectivity (ability to infect) and infection intensity (severity of infection). Studies were excluded from the meta-analysis if they did not give data for either measure of parasite performance. Infectivity data were collected primarily in the form of frequency successfully infected hosts or, for invertebrate social parasites, either percentage of host brood raided (Fischer & Foitzik 2004) or per cent survival of parasites (Schönrogge *et al.* 2006). Data on infection intensity were most often estimates of parasite load (Ebert 1994; Ebert *et al.* 1998), or reproductive success during infection, e.g. the number of flowers produced by a parasitic plant (Mutikainen *et al.* 2000). For studies that reported data across multiple time points without statistically combining the results (Karban 1989; McCoy *et al.* 2002), we arbitrarily chose to take information from only the final time point, so as not to give more weight to these studies.

If there were two or more studies on the same host–parasite system, only one study was retained to avoid pseudoreplication (Hurlbert 1984). In all cases, the retained study offered a more thorough analysis or a more rigorous experimental design. In addition, although two studies focused on the same parasite species, *Matsucoccus acalyptus*, each tested a different host species (Unruh & Luck 1987; Cobb & Whitham 1993). Similarly, two others examined parasite performance on multiple host species with a separate analysis for local adaptation within each host species (Leuchtmann & Clay 1989; Sicard *et al.* 2007). These four studies were included in the meta-analysis, with each host–parasite combination treated as an independent data point.

Data collection

For each study included in the analysis, we recorded the pattern of parasite adaptation (whether the parasite populations showed an overall pattern of local adaptation, local

maladaptation or no significant pattern, herein called 'no local adaptation'). As previously mentioned, some studies used a 'home vs. away' definition of local adaptation, comparing parasite performance of a given parasite population among sympatric (home) and allopatric (away) host populations, while others used a 'local vs. foreign' definition, comparing the performance of local (sympatric) vs. foreign (allopatric) parasite populations on a given host population (definitions labelled in Kawecki & Ebert 2004). Whereas the relevant comparison for the home vs. away definition is performance across host populations, the relevant comparison for the local vs. foreign definition is performance across parasite populations.

The most common statistic used to interpret results of cross-inoculation experiments was an analysis of variance (ANOVA), which under some circumstances can address both definitions. For example, if there are no host or parasite main effects, but there is a significant interaction term due to differences between sympatric and allopatric combinations, the result is local adaptation or maladaptation (depending on the direction of the difference) under both definitions. If an ANOVA gives a non-significant interaction term, i.e. there is no interaction between parasite origin and host origin, then the result is no local adaptation under both definitions. If the author(s) did not report an interaction term, we used information on differences in mean performance among individual parasite populations. Specifically, in the cases where authors reported statistics comparing parasite population means, these were used to determine the overall pattern of local adaptation. If, on the other hand, authors did not report such information, means were considered significantly different if they were separated by more than two standard errors. Differences were considered non-significant if standard error intervals overlapped (Browne 1979). In the rare cases where standard error intervals were neither overlapping nor separated by more than two standard errors, the individual population was omitted from the overall local adaptation result for the study.

When means were used, the parasite was determined to be locally adapted when more than half of the populations were significantly locally adapted and was determined to be maladapted if more than half of the populations were significantly maladapted. Two studies were excluded from the local vs. foreign analysis of infectivity because exactly half of the populations were considered locally adapted (Zangerl & Berenbaum 1990; Fischer & Foitzik 2004). When author(s) chose not to compare performance among parasite populations directly, either due to potential differences in parasite dose (Lively 1989) or to differences in the timing of measurements across different parasite populations (Ericson *et al.* 2002), the studies were omitted from the local vs. foreign analysis. There were no analogous cases for comparisons across host populations, and thus no studies were removed from the home vs. away analysis for these reasons.

Studies were categorized based on parasite migration rate and generation time relative to those of the host, virulence, and whether or not the parasite was obligate. Parasites were also classified with regard to their life cycle; a species was considered to have a complex life cycle if it must infect two (or more) different host species for successful reproduction. We recorded the number of host and parasite populations utilized in each experiment, and the number of sympatric combinations (denoted sympatric units after Kaltz & Shykoff 1998).

To determine relative migration rates of hosts and parasites, we utilized any information regarding population structure based on genetic markers, first from the study itself, or else in related work. When this information was not available, we used anecdotal discussion of relative dispersal ability or, in one case, an analysis of relative re-colonization rates (Ganz & Washburn 2006). Determinations were also made based on information gathered for dispersal mechanisms or life cycles of hosts and parasites. Parasites were additionally classified as vertically transmitted, horizontally transmitted, or as having a mixture of transmission modes. Virulence was determined such that, if parasites significantly increased mortality and/or sterility in host populations, they were recorded as highly virulent. We also recorded whether the parasite was obligate, meaning it must live on or in host tissue for a portion of its life cycle, or non-obligate (adapted from Roberts & Janovy 2000).

As there is no standardized way of reporting information on migration rate, generation time or virulence, and as estimates were not always provided in the study itself, we often utilized further resources and personal communications with authors. In the case that reported information was too scarce to make a determination, the study was omitted from the comparisons involving the trait in question. In addition, we chose not to test for patterns between local adaptation and transmission mode, due to the paucity of studies examining parasites that are strictly vertically transmitted and the difficulty of reliably classifying parasites with mixed transmission modes (Lipsitch *et al.* 1995; Kover *et al.* 1997). To minimize potential errors due to cross-system comparison and grey areas within the parasite literature, all categories were kept deliberately broad, e.g. highly virulent parasites vs. all other parasites (see Table 1).

Statistical analysis

Using the data supplied within each paper, we focused on four comparisons with respect to parasite local adaptation: (1) infectivity on sympatric vs. allopatric hosts (home vs. away definition of local adaptation); (2) infectivity of

Table 1 (a) Summary of system characteristics and local adaptation results for plant hosts. Microparasites include viruses, bacteria and protozoa, while macroparasites include parasitic worms and arthropods (Anderson & May 1979). Virulence (harm to the host) is listed as beneficial (B), increased mortality (**M**) or sterility (**S**), little or no effect (N), reduced fecundity (F), and reduced fitness (W). Parasites causing significantly increased mortality (**M**) or sterility (**S**) were considered highly virulent. 'N/A' indicates that the category was not applicable to the organism studied, that researches could not distinguish between the effects of two or more parasites species, or that the experimental design precluded the relevant statistical analysis. (b) Summary of system characteristics and local adaptation results for bacteria and animal hosts

Host–parasite system	Reference	Host	Parasite	Complex life cycle?	Migration rates*	Generation times*	Virulence	Obligate?	Transmission†	Infectivity‡			Infection intensity‡			Sympatric units§	Parasite populations	Host populations	Main effects¶	
										HvA	LvF	Overall	HvF	LvF	Overall				Parasite	Host
(a)																				
Plant–fungus	1	*Triticum aestivum*	*Septoria tritici*	N	**p**>h	p<**h**	W	Y	H				**LA**	**LA**	LA	2	2	2		
	2	*Spartina pectinata*	*Puccinia* sp.	Y	**p**>h	p<**h**	N	Y	H				NLA	NLA	NLA	3	3	5		
	3	*Pinus sylvestris*	*Crumenulopsis sororia*	N	**p**>h	p<**h**	W	Y	H	NLA	NLA	NLA				3	3	3	Yes	No
	4	*Filipendula ulmaria*	*Triphragmium ulmariae*	N	**p**>h	p<**h**	W	Y	H	**NLA**		NLA	**NLA**		NLA	4	4	6		
	5	*Silene latifolia*	*Microbotryum violaceum*	N	p<**h**	p<**h**	**S**	Y	H	MA	MA	MA				14	14	14	Yes	Yes
	6	*Plantago lanceolata*	*Podosphaera plantaginis*	N	**p**>h	p<**h**	F	Y	H	**LA**		LA	**NLA**		NLA	4	4	20	No	No
	7	*Danthonia compressa*	*Atkinsonella hypoxylon*	N	p<**h**	p<**h**	B	Y	H/V	NLA	NLA	NLA				2	2	2		
	7	*Danthonia spicita*	*Atkinsonella hypoxylon*	N	p<**h**	p<**h**	B	Y	H/V	NLA	NLA	NLA				3	3	3		
	7	*Stipa leucotricha*	*Atkinsonella hypoxylon*	N	p<**h**	p<**h**	W	Y	H/V	NLA	NLA	NLA				3	3	3		
	8	*Salix triandra*	*Melampsora amygdalinae*	N	**p**>h	p<**h**	W	Y	H	**LA**	NLA	LA				3	3	4		
	9	*Amphicarpaea bracteata*	*Synchytrium decipiens*	N	p<**h**	p<**h**	**M**	Y	H	**LA**		LA	**LA**		LA	1	1	3	N/A	N/A
	10	*Podophyllum peltatum*	*Puccinia* sp.	N	p<**h**	p<**h**	**M**	Y	H/V	NLA	NLA	NLA	NLA	NLA	NLA	6	6	6		
	11	*Arabis holboellii*	*Puccinia monoica*	N/A	**p**>h	p<**h**	**M**	Y	H	**MA**		MA				3	3	3	Yes	Yes
	12	*Phaseolus coccineus*	*Colletotrichum lindemuthianum*	N	p<**h**	p<**h**	F	Y	H/V	NLA	NLA	NLA	NLA	NLA	NLA	3	3	3	No	No
	12	*Phaseolus vulgaris*	*Colletotrichum lindemuthianum*	N	**p**>h	p<**h**	F	Y	H/V	LA	LA	LA	LA	LA	LA	3	3	3	No	Yes
	13	*Linum marginale*	*Melampsora lini*	N	**p**>h	p<**h**	**M**	Y	H	LA	LA	LA				6	6	6	Yes	Yes
	14	*Lactuca sibirica*	*Puccinia minussensis*	N	**p**>h	p<**h**	W	Y	V	**LA**		LA				1	1	5	N/A	N/A

Table 1 (*Continued*)

Host–parasite system	Reference	Host	Parasite	Complex life cycle?	Migration rates*	Generation times*	Virulence	Obligate?	Transmission†	Infectivity‡			Infection intensity‡			Sympatric units§	Parasite populations	Host populations	Main effects¶	
										HvA	LvF	Overall	HvF	LvF	Overall				Parasite	Host
Plant–herbivore	15	*Pinus ponderosa*	*Nuculaspis californica*	N	p<**h**	p<**h**	W	Y	H	NLA	NLA	NLA				5	5	5	No	Yes
	16	*Pinus edulis*	*Matsucoccus acalyptus*	N	p<**h**	p<**h**	W	Y	H	NLA	NLA	NLA				20	23	20	No	Yes
	17	*Morus alba*	*Pseudaulacaspis pentagona*	N	p<**h**	p<**h**	W	Y	H	**LA**		LA				10	10	10		Yes
	18	*Erigeron glaucus*	*Apterothrips secticornis*	N	p<**h**	p<**h**	W	N	H				**LA**		LA	3	3	3		
	19	*Cupressus lusitanica*	*Cinara cupressi*	N	**p**>h	p<**h**	W	N	H	NLA	NLA	NLA				8	8	8		
	20	*Salix triandra*	*Gonioctena linaeana*	N	**p**>h	p<**h**	W	N	H	LA	LA	LA				2	2	3	Yes	Yes
	21	*Quercus rubra*	Various herbivores	N	p<**h**	p<**h**	W	N	H	MA	MA	MA				3	3	3	No	No
	22	*Rhus glabra*	*Blepharida rhois*	N	**p**>h	p<**h**	W	N	H	NLA	NLA	NLA				8	8	8	Yes	Yes
	23	*Pinus monophylla*	*Matsucoccus acalyptus*	N	p<**h**	p<**h**	W	Y	H		**NLA**	NLA				15	17	15	No	No
	24	*Fagus sylvatica*	*Cryptococcus fagisuga*	N	**p**>h	p<**h**	**M**	Y	H		**LA**	LA				5	5	5	Yes	Yes
	25	*Pastinaca sativa*	Various herbivores	N	N/A	N/A	N/A	N	H	**NLA**		NLA				2	2	2	Yes	No
Plant–macroparasite	26	*Roridula gorgonias*	*Pameridea marlothi*	N	**p**>h	p<**h**	B	N	H	**NLA**	NLA	NLA				2	2	2		
	27	*Vitis arizonica*	*Daktulosphaira vitifoliae*	N	p<**h**	p<**h**	W	Y	H	**NLA**		LA	**NLA**		NLA	3	3	6	No	
	28	*Quercus geminata*	*Stilbosis quadricustatella*	N	**p**>h	p<**h**	W	Y	H	**LA**		LA				1	1	2	N/A	N/A
	29	*Borrichia frutescens*	*Asphondylia borrichiae*	N	**p**>h	p<**h**	W	Y	H	LA	LA	LA	LA	LA	LA	4	4	4	Yes	
Plant–microparasite	30	*Arabidopsis thaliana*	*Pseudomonas viridiflava*	N	p<**h**	p<**h**	W	N	H				NLA	NLA	NLA	6	6	6		
Plant–parasitic plant	31	*Urtica dioica*	*Cuscuta europaea*	N	**p**>h	p<**h**	F	Y	H	**LA**		LA				5	5	5	No	
	32	*Agrostis capillaris*	*Rhinanthus serotinus*	N	p<**h**	p=h	W	N	H	**NLA**		NLA	**NLA**		NLA	4	4	4	No	
(b)																				
Bacteria–microparasite	33	*Pseudomonas fluorescens SBW25*	*SBW25ϕ2*	N	p=h	p<**h**	**M**	Y	H	**MA**	MA	MA				12	12	12		

Table 1 (*Continued*)

Host–parasite system	Reference	Host	Parasite	Complex life cycle?	Migration rates*	Generation times*	Virulence	Obligate?	Transmission†	Infectivity‡			Infection intensity‡			Sympatric units§	Parasite populations	Host populations	Main effects¶	
										HvA	LvF	Overall	HvF	LvF	Overall				Parasite	Host
Invertebrate–macroparasite	34	*Leptothorax acervorum*	*Harpagoxenus sublaevis*	N	p<**h**	p=h	**M**	N	H	NLA		NLA				2	2	3	No	
	35	*Lymnaea truncatula*	*Fasciola hepatica*	Y	**p**>h	p<**h**	**M**	Y	H				MA	MA	MA	2	2	2	N/A	N/A
	36	*Acyrthosiphon pisum*	*Aphidius ervi*	N	p<**h**	p=h	**M**	Y	H	NLA	**NLA**	NLA				3	3	4	No	Yes
	37	*Potamopyrgus antipodarum*	*Microphallus* sp.	Y	**p**>h	p=h	**S**	Y	H	**LA**		LA				4	4	4	Yes	Yes
	38	*Bulinus globosus*	*Schistosoma mattheei*	Y	**p**>h	p<**h**	**M**	Y	H	LA	LA	LA	NLA	NLA	NLA	2	2	2	No	Yes
	39	*Biomphalaria glabrata*	*Schistosoma mansoni*	Y	p=h	p<**h**	**S**	Y	H	**NLA**		NLA				4	4	5	Yes	Yes
	40	*Anodonta piscinalis*	*Paraergasilus rylovi*	N	p<**h**	p<**h**	W	N	H	**LA**		LA	**LA**		LA	2	2	2		
	41	*Formica lemani*	*Microdon mutabilis*	N	**p**>h	p<**h**	**M**	N	N/A	**LA**		LA				15	33	28		
Invertebrate–microparasite	42	*Danaus plexippus*	*Ophryocystis elektroscirrha*	N	p<**h**	p=h	W	Y	V				NLA	NLA	NLA	3	3	3	Yes	No
	43	*Daphnia magna*	*Glugoides intestinalis*	N		p<**h**	F	Y	H				LA	LA	LA	3	3	9		
	44	*Daphnia magna*	*Pasteuria ramosa*	N	**p**>h	**p**>h	**S**	Y	H	NLA	**NLA**	NLA	NLA	**NLA**	NLA	3	3	4	Yes	No
	45	*Ochlerotatus sierrensis*	*Lambornella clarki*	N	p<**h**	p<**h**	**M**	N	H	NLA	**LA**	LA	NLA	**NLA**	NLA	2	2	4	Yes	Yes
	46	*Gammarus duebeni*	*Nosema granulosis*	N	p<**h**	p=h	F	Y	V	**NLA**		NLA	**LA**		LA	1	1	4	N/A	N/A
	47	*Bombus terrestris*	*Crithidia bombi*	N	p<**h**	p<**h**	W	Y	H/V		**NLA**	NLA		**NLA**	NLA	3	3	3		No
Vertebrate–brood parasite	48	*Turdus migratorius*	*Molothrus ater*	N	p=h	p=h	F	N	N/A	**MA**		MA				1	1	2	N/A	N/A
Vertebrate–macroparasite	49	*Phoxinus phoxinus*	*Diplostomum phoxini*	Y	**p**>h	p<**h**	W	Y	H				**LA**		LA	2	2	2		
	50	*Parus major*	*Ceratophyllus gallinae*	N	p=h	p<**h**	N	N	V					**NLA**	NLA	1	2	1	N/A	N/A
	51	*Xenopus laevis*	*Protopolystoma* spp.	N	p<**h**	p<**h**	W	Y	H				**LA**		LA	3	3	4		
	52	*Gasterosteus aculeatus*	*Diplostomum pseudospathaceum*	Y	**p**>h	p<**h**	**M**	Y	H				**MA**		MA	1	1	2	N/A	N/A
	53	*Rissa tridactyla*	*Ixodes uriae*	N	**p**>h	p<**h**	W	N	H	**LA**		LA	**NLA**		NLA	32	32	32		
Vertebrate–microparasite	54	*Gallotia gallotia*	Haemogregarine	Y	p<**h**	p<**h**	W	Y	H	MA	MA	MA				1	1	3	N/A	N/A

*Relative host and parasite values are represented by 'h' and 'p', respectively.

†Transmission mode is classified as 'H' for horizontal, 'V' for vertical and 'H/V' for any combination of horizontal and vertical transmission.

‡Bold entries indicate that authors reported results by that definition (HvA refers to the home vs. away definition, while LvF refers to the local vs. foreign definition). See Introduction for discussion of the two definitions. Blank cells indicate that the information could not be obtained from the study. 'LA' indicates parasites local adaptation; 'MA' indicates parasite local maladaptation; 'NLA' indicates no local adaptation (no significant pattern found).

§The number of sympatric host–parasite combinations examined in the study.

¶Evidence of significant differences among populations in terms of infectivity for parasites, or susceptibility for hosts. Blank cells indicate that such information was not reported, or that no statistical significance was reported. 'N/A' indicates that the experimental design precluded the detection of main effects.

sympatric vs. allopatric parasites (local vs. foreign definition); (3) infectivity of sympatric vs. allopatric host–parasite combinations (across definitions); (4) infection intensity of sympatric vs. allopatric host–parasite combinations (across definitions). Overall local adaptation results were pooled for comparisons (3) and (4) to maximize statistical power by including as many studies as possible. The cross-definition results were obtained using the pattern reported by the author(s) when only one definition was used or when both definitions yielded the same overall result. When the pattern of local adaptation changed depending on the definition employed, the pattern discussed by the author(s) was recorded as the cross-definition result since reported statistics tended to be definition-specific.

Fisher's exact tests were performed for all comparisons of parasite local adaptation results across system characteristics (as described above), number of sympatric units in a study, or host/parasite main effects. Specifically, studies finding local adaptation were compared to those finding no local adaptation across two categories, e.g. obligate vs. non-obligate parasites, in a two-by-two contingency table. We also compared the number of studies finding local maladaptation to the number finding no local adaptation to identify trends in the opposite direction. In both cases, local adaptation results were tested separately from maladaptation results to ensure that, when data were condensed into these two-by-two tables, statistical results were not misleading due to grouping of non-equivalent patterns, such as maladaptation and no local adaptation. Given that there could be differences among types of host–parasite systems with respect to the traits listed above, we also performed Mantel–Haenszel tests of conditional independence to determine if the same correlations with local adaptation could be seen across different host types (plant vs. invertebrate, as these were the most common host types in the data set). Tests for pairwise interactions between factors (complexity of life cycle, relative migration rate and generation time, virulence, and whether the parasite was obligate or non-obligate) were also run using Fisher's exact tests.

The number of sympatric units tested (the number of sympatric host–parasite combinations, after Kaltz & Shykoff 1998) was compared across studies to determine if testing fewer than three sympatric units would increase the likelihood of finding an overall significant pattern, i.e. local adaptation or maladaptation. We also tested for a correlation between likelihood of finding local adaptation and the presence of host or parasite main effects. All analyses were run using SPSS software (2005) and all reported *P*-values are two-sided.

Power analysis

To analyse the power of our Fisher's exact tests, we used estimates of small, medium and large effect sizes for χ^2 tests ($w = 0.1$, 0.3 and 0.5, respectively) in a two-by-two contingency table ($\alpha = 0.05$) (Table 7.3.15 in Cohen 1988). For a contingency table, each effect size specifies a deviation from the result predicted by the null hypothesis, with greater effect sizes resulting from larger deviations from the null hypothesis. In each case, the null hypothesis is that the probability of getting an entry in any one cell is given by the product of the relevant marginal probabilities. For example, the probability (under the null hypothesis) of finding a study with an obligate, locally adapted parasite would be equal to the probability of finding an obligate parasite times the probability of finding a locally adapted parasite. Thus, effect size (w) can be defined as a function of the difference between the probability predicted by the null hypothesis (P_{0i}) and the probability given by the alternate hypothesis (P_{1i}) for each of the m cells in a contingency table (equation 7.2.1 of Cohen 1988):

$$w = \sqrt{\sum_{i=1}^{m} \frac{(P_{1i} - P_{0i})^2}{P_{0i}}}$$

The effect size can be equivalently defined as a function of the difference between the number of studies in a certain cell expected by the alternate hypothesis (k) and the number expected by chance alone, such that:

$$w = \sqrt{\frac{(kn - r_1 c_1)^2}{r_1 r_2 c_1 c_2}} \quad (1)$$

where r and c refer to row and column totals, respectively, and n refers to the total number of studies in the two-by-two contingency table. Thus, for each table in our analysis, we were able to estimate the size of the deviation (from the null hypothesis) required to obtain a given effect size and power (the probability of finding a significant result for a predicted effect size). For all but one comparison, the alternate hypothesis was taken to be that given by theory. For generation time, however, the theoretical prediction is roughly equivalent to the null hypothesis, and thus we utilized the conventional logic that shorter parasite generation times would increase local adaptation. The k values for the relevant cells are presented in Table 2, along with the k values predicted under the null hypothesis.

RESULTS

We analysed experiments from 57 different host–parasite systems across a total of 54 studies (data summarized in Table 1). When the two host categories sufficiently large for comparison, plant and invertebrate, were examined, there was no difference in the number of studies reporting local adaptation vs. those finding no local adaptation by infectivity across definitions (Fisher's exact test, $P =$

Table 2 Summary of results for comparisons of local adaptation studies. Maladaptation results are excluded from comparisons in this table, and all *P* values are two-tailed. For infectivity across both definitions, the results of the power analysis are shown. The expected count for the bolded entry is given under the null hypothesis, as well as the expected counts assuming small, medium and large effect sizes ($w = 0.1$, 0.3 and 0.5, respectively), with the corresponding percentage likelihood of finding a significant result for the given effect size and sample size of 40 (power). These expected counts are intended to give the reader an idea of the size of the deviation required to obtain a certain effect size and power.

		Relative migration rates		Relative generation times		High virulent?		Obligate?	
Definition	Comparison	p≤**h**	**p**>h	p<**h**	**p**≥h	No	Yes	No	Yes
Home vs. away (infectivity)	Number of studies finding local adaptation (LA)	3	13	15	1	11	5	4	12
	Number of studies finding no local adaptation (NLA)	14	6	15	5	14	6	7	14
	Fisher's exact test	*P=0.003*		*P=0.196*		*P=1.000*		*P=0.723*	
Local vs. foreign (infectivity)	Number of studies finding local adaptation (LA)	1	6	7	0	4	3	2	5
	Number of studies finding no local adaptation (NLA)	10	6	14	2	13	3	3	13
	Fisher's exact test	*P=0.069*		*P=1.000*		*P=0.318*		*P=0.621*	
Across both definitions (infectivity)	Number of studies finding local adaptation (LA)	4	**14**	**17**	1	11	**7**	5	**13**
	Number of studies finding no local adaptation (NLA)	15	6	16	5	16	5	6	16
	Fisher's exact test	*P=0.004*		*P=0.190*		*P=0.488*		*P=1.000*	
Expected counts	**Under null hypothesis**	**9.2**		**15.2**		**5.5**		**13.1**	
	Power 10% ($w = 0.1$)	**10.2**		**15.9**		**6.4**		**13.9**	
	Power 47% ($w = 0.3$)	**12.1**		**17.3**		**8.2**		**15.7**	
	Power 89% ($w = 0.5$)	**14.1**		**N/A***		**10.0**		**17.5**	
Across both definitions (infection intensity)	Number of studies finding local adaptation (LA)	5	4	9	1	9	1	2	8
	Number of studies finding no local adaptation (NLA)	9	6	12	3	11	4	5	10
	Fisher's exact test	*P=1.000*		*P=0.626*		*P=0.615*		*P=0.659*	

*For the relative generation time comparison, the expected count assuming a large effect was not possible given the total number of local adaptation results in the comparison.

1.000). In addition, there were no differences between host types with regard to migration rate ($P = 0.517$) or the frequency of obligate vs. non-obligate parasites ($P = 1.000$). Therefore, host groups were lumped together for these analyses, except where otherwise noted. The host types were not, however, equivalent in terms of relative generation time, with almost all plant hosts having generation times longer than their parasites ($P = 0.001$), or virulence, with the majority of plant studies involve less virulent parasites ($P = 0.004$). Lastly, plant hosts had significantly fewer parasites with complex life cycles than did invertebrate hosts ($P = 0.021$). Thus Mantel–Haenszel tests were used to account for the confounding effects of host type in addition to Fisher's exact tests for these categories.

We found that studies of parasites with higher migration rates than their hosts found local adaptation by infectivity, across definitions, more often than did studies in which parasite migration rate was less than or equal to that of the host ($P = 0.004$, Fisher's exact test, Table 2). The same pattern was found using both the home vs. away and local vs. foreign definitions of local adaptation ($P = 0.003$ and $P = 0.069$, respectively, Table 2). These patterns were also detected when type of host (plant or invertebrate) was treated as a confounding variable (Mantel–Haenszel test across definitions for infectivity, $P = 0.010$).

Parasites exhibiting a migration rate less than or equal to that of their hosts were not, however, more likely to be locally maladapted in terms of infectivity (across definitions, $P = 1.000$). In addition, there was no significant pattern found for local adaptation by infection intensity with regard to relative parasite migration rate ($P = 1.000$, Table 2). Studies of parasites with complex life cycles were not more likely to report local adaptation in any of the comparisons ($P \geq 0.304$) and were not associated with higher relative migration rates ($P = 0.125$). Furthermore, when these studies were removed, the effects of migration rate on local adaptation by infectivity were still significant (with the exception of the local vs. foreign definition, $P = 0.149$).

Parasites with shorter generation times than their hosts did not show local adaptation more often than other parasites (Table 2). Moreover, parasites with generation times equal to or longer than their hosts were not more likely to show maladaptation (across definitions, for infectivity and infection intensity, $P = 1.000$). When host

Table 3 Effect of number of sympatric units tested on significance of results. A unit denotes a single combination of sympatric host and parasite populations. Note that all *P*-values are two-tailed.

		Number of sympatric units tested	
Definition	Comparison	1–2	3+
Home vs. away (infectivity)	Number of studies finding significant results*	8	14
	Number of studies not finding significant results	6	15
	Fisher's exact test	*P=0.747*	
Local vs. foreign (infectivity)	Number of studies finding significant results*	4	7
	Number of studies not finding significant results	2	14
	Fisher's exact test	*P=0.187*	
Across both definitions (infectivity)	Number of studies finding significant results*	9	15
	Number of studies not finding significant results	5	17
	Fisher's exact test	*P=0.346*	
Across both definitions (infection intensity)	Number of studies finding significant results*	7	5
	Number of studies not finding significant results	3	12
	Fisher's exact test	*P=0.057*	

*Includes local adaptation and maladaptation.

type was separated as a confounding variable, there was still no correlation between relative generation time and the pattern of local adaptation (infectivity across definitions, Mantel–Haenszel test, $P = 0.221$). However, the statistical power of these comparisons was low due primarily to the unequal class sizes within the table, as only eight of the systems included parasites with equal or longer generation times than their hosts (Table 2).

Studies of highly virulent parasites did not find local adaptation more often than studies of less virulent parasites (Table 2). This result was robust when host type (plant or invertebrate) was separated as a confounding variable (Mantel–Haenszel test across definitions for infectivity, $P = 0.370$). Highly virulent parasites were also not more likely to show maladaptation with regard to infectivity than less virulent parasites (across definitions, $P = 0.319$). While there was no significant correlation between virulence and generation time ($P = 0.228$), there was an interaction between virulence and type of life cycle, with highly virulent parasites being significantly more likely to have a complex life cycle than less virulent parasites (Fisher's exact test, $P = 0.038$). When parasites with complex life cycles were excluded from analysis, there was still no interaction between virulence and local adaptation (across definitions, infectivity, $P = 0.470$; infection intensity, $P = 0.616$). In addition, when host type was examined as a confounding variable, there was no correlation between complex life cycle and pattern of local adaptation (infectivity across definitions, Mantel–Haenszel test, $P = 0.860$).

No significant differences were found between obligate and non-obligate parasites with regard to local adaptation (Table 2). When comparing the contingency tables separately for plant and invertebrate hosts, there remained no correlation between obligate parasites and local adaptation by infectivity (Mantel–Haenszel test across definitions, $P = 0.936$). Not surprisingly, there was a marginally significant interaction, such that all parasites with complex life cycles were also obligate (Fisher's exact test, $P = 0.089$). There were no other significant pairwise interactions among the factors of complex life cycle, relative migration rate, relative generation time, virulence or obligate/non-obligate.

Parasite main effects were not correlated with an increased likelihood of finding local adaptation (as opposed to no local adaptation) by infectivity under either the home vs. away or local vs. foreign definition ($P = 1.000$, $P = 0.315$, respectively; across definitions, $P = 0.414$). There was, however, a trend towards finding parasite local adaptation by infectivity more often when host main effects were present under the local vs. foreign definition ($P = 0.044$) and a marginally significant trend across definitions ($P = 0.070$). There were no significant interactions between infection intensity and main effects (parasite, $P = 1.000$; host, $P = 0.375$).

The number of sympatric units, i.e. the number of parasite populations tested on their sympatric hosts, within a study had no effect on whether or not a significant pattern of local adaptation or maladaptation emerged with respect to infectivity (Table 3). There was a marginally significant pattern for infection intensity, such that studies comparing one or two host–parasite combinations were more likely to find a significant pattern (local adaptation or maladaptation) than studies testing three or more host–parasite combinations ($P = 0.057$, Fisher's exact test, Table 3).

DISCUSSION

Parasite local adaptation is commonly predicted due to the strong selective pressure exerted by parasites on their

host populations. Assuming some degree of genetic specificity for infection, parasites should be under selection to infect common host genotypes resulting in increased fitness of rare host genotypes (Jaenike 1978; Hamilton 1980; Hutson & Law 1981; Bell 1982). Specifically, when parasites drive changes in host allele frequencies within geographically distinct populations, it is predicted that parasites will be better at infecting sympatric than allopatric hosts, which are most likely in a different phase of the coevolutionary cycle due to both selection and drift. Although frequency-dependent dynamics do not necessarily lead to local adaptation of parasites, evidence of local adaptation across parasite populations is considered suggestive of parasite-mediated, negative frequency-dependent selection (Parker 1985, 1989; Roy 1998). Our meta-analysis utilized results from 54 studies across a diverse set of organisms, to test theoretical predictions concerning the effects of relative migration rate, generation time and virulence on parasite local adaptation. In line with the conventional wisdom that parasites are ahead in the coevolutionary cycle, 18 studies found evidence of parasite local adaptation across definitions for infectivity, compared with only six showing maladaptation.

Overall, each of the three outcomes of coevolution (local adaptation, local maladaptation and no local adaptation) was well-represented in the set of studies analysed. It is therefore unlikely that published studies are biased towards significant findings. Indeed, non-significant results (no local adaptation) were reported more often than local adaptation (Table 1). A similar pattern was found in an earlier review of parasite local adaptation, with half of the studies finding either no local adaptation or maladaptation (Kaltz & Shykoff 1998). In fact, the abundance of non-significant findings may, in itself, indicate a bias. For one thing, while type I errors are avoided in these studies, the probability of type II errors are not calculated (Cohen 1965), making it impossible to determine if no local adaptation is indeed a reliable result.

This problem is further complicated by the possibility that, within a given study, either too small or too large a geographical scale has been chosen for detection of local adaptation (Kaltz & Shykoff 1998). For example, some authors find evidence for local adaptation at larger geographic scales and no local adaptation at smaller scales (Hanks & Denno 1994; Imhoof & Schmid-Hempel 1998), while others find evidence for local adaptation at the local scale that cannot be duplicated at larger scales (McCoy *et al.* 2002). Therefore, in addition to power analyses examining the likelihood that local adaptation can be detected statistically, cross-infection experiments should incorporate a discussion as to why a given geographical scale of analysis is appropriate.

Within the data set examined, authors more often utilized measures of infectivity to estimate parasite performance than infection intensity when determining the pattern of local adaptation. The measure of performance used has important implications for interpretation of results. Specifically, infection intensity can give misleading results when comparing across systems or individual populations. Unlike infectivity, infection intensity is not predicted to be under directional selection and, in fact, may be maintained at an intermediate level via balancing selection. In particular, whereas parasites should always be under selection to infect a greater number of hosts, the optimal infection intensity presents a tradeoff in which more severe infections might have a negative effect on the rate of transmission. This tradeoff would result in parasites that are adapted to some optimal value of infection intensity that may be continually changing due to the dynamic nature of the host–parasite relationship (Dybdahl & Storfer 2003). Thus, two different parasite populations are likely to have different optimal infection intensities and, as a result, would not appear to be locally adapted based on mean differences in absolute infection intensity. Although authors often examine infection intensity across multiple populations, these data cannot yield information relevant to local adaptation unless researchers have *a priori* hypotheses regarding optimal infection intensity. Therefore, local adaptation studies should incorporate data on the more relevant and predictable measure of parasite performance, infectivity.

Making generalizations across host–parasite systems is inevitably difficult given the vast differences in basic biology across host–parasite systems. Thus, grouping studies into biologically meaningful categories presents a challenge and forces broad characterization of parasite and host attributes. Under these categories, some host types inevitably contain more variation than others. For example, although there was some variation in relative generation times for non-plant host systems, plant hosts had longer generation times than their parasites in all but one system (Table 1). In addition, some host–parasite systems, particularly plant–fungus interactions, are examined much more frequently than others, such as vertebrate hosts, bacteria–microparasite or plant–parasitic plant systems (Table 1). Lastly, most of the species studied undergo at least some sexual reproduction, and thus more studies on strictly asexual organisms are required before any informative analysis can be performed to examine the importance of reproductive mode to local adaptation.

Relative migration rate

Theoretical work suggests that, although high levels of gene flow tend to homogenize populations, low to intermediate

levels of gene flow facilitate local adaptation by providing genetic variation upon which selection can act (Gandon *et al.* 1996; Lively 1999; Gandon & Michalakis 2002). We tested this prediction by comparing systems in which parasites migrate more than their hosts to systems in which parasites migrate at a rate less than or equal to that of their hosts. The results strongly support the notion that parasite populations with high relative migration rates are more likely to be adapted to infect their local hosts (Table 2). These findings are consistent with experimental and simulation results in which higher rates of phage migration increased phage local adaptation compared with host migration alone or the absence of migration (Morgan *et al.* 2005). Likewise, experiments using another bacteria-phage system show increased phage local adaptation in communities linked by dispersal and increased phage maladaptation in isolated communities (Forde *et al.* 2004).

Conversely, low parasite migration relative to host migration has been suggested to account for finding local maladaptation in a smut fungus-plant system (Kaltz *et al.* 1999). Interestingly, in the present analysis, there was no evidence for parasites that migrate less than their hosts to be maladapted more often. This finding is surprising given past simulation results showing that parasites should be locally maladapted when they migrate less than their hosts, and when parasite migration rates are low in absolute terms (Gandon *et al.* 1996). However, as data were rarely available for absolute migration rates in these systems, this latter theoretical prediction could not be tested directly. Although we found no significant relationship between relative migration rate and infection intensity, this measure of parasite performance is not a reliable indicator of local adaptation, as discussed previously.

Relative generation time

We found no significant pattern with respect to relative generation time and local adaptation. This is congruent with previous simulations showing that, as parasites act as the primary and consistent driving force for host adaptation, relative generation times of hosts and parasites should not impact parasite local adaptation (Lively 1999). In other words, parasites are able to track common hosts regardless of their relative generation times, and the rate at which they drive changes in the host population should not alter the pattern of local adaptation. Furthermore, when relative generation time was experimentally manipulated in a bacteria–bacteriophage system, it was not found to alter parasite local adaptation (Morgan & Buckling 2006).

Theoretical simulations suggest that factors other than generation time, particularly migration and mutation rates, are more likely to limit the evolutionary potential of parasites and therefore act to mask any potential effect of generation time (Gandon & Michalakis 2002). The significant pattern for migration rate in the present analysis, and the lack of a significant pattern for generation time, is consistent with these predictions. Despite the fact that there are a small number of studies for parasites with generation times equal to or longer than their hosts, thus limiting the power of the comparison (Table 2), there was a slight trend in the direction commonly assumed, where parasites with shorter generation times were somewhat more likely to show local adaptation. These patterns are in agreement with the idea that generation time plays a minor role in determining the pattern of coevolution and that other factors, such as migration rate, are likely to be the primary predictors for local adaptation.

Virulence

Local adaptation is predicted to be more pronounced when parasites are harmful to their hosts, due to stronger parasite-mediated selection on host populations yielding greater between-population divergence (Lively 1999; Gandon 2002). Indeed, relatively high virulence may lead to parasite local adaptation even in the face of low relative migration rates (Gandon 2002). The present results, however, did not support this prediction, indicating that highly virulent parasites were not more likely to be locally adapted than their less harmful counterparts. It is likely that virulence must be scaled by the prevalence of parasite infection to make accurate predictions about the magnitude of selection acting on host populations. For example, a highly virulent parasite with low prevalence within a population may constitute a negligible selective force on hosts and may consequently fail to generate a pattern of local adaptation.

While there was no significant correlation between virulence and local maladaptation, parasites with complex life cycles were significantly more likely to be highly virulent, and marginally more likely to be obligate. The interaction between virulence and parasite life cycle is interesting in that it may reflect underlying dynamics in the virulence evolution of parasites with multiple required hosts. In addition, the interaction between obligate parasites and type of life cycle is likely a product of the way we define obligate parasitism; as parasites with complex life cycles require multiple host species to reproduce, it necessarily follows that they are obligate under our definition.

As there is currently no standardized way of quantifying virulence, we had to utilize broad categories when classifying the effects of parasites on their hosts. Thus, it was necessary to group what is most likely a continuous trait into two discrete categories. Except in cases where the parasite kills or sterilizes its host, quantifying the virulence of a parasite population is difficult, particularly as environmental factors, especially availability of resources, have been shown to

significantly alter virulence. For example, in a year when fewer resources were available to the host, tick parasites showed a stronger negative effect than when resources were plentiful (McCoy *et al.* 2002). In contrast, there is also evidence that protozoan parasites have higher virulence on *Daphnia galeata* hosts with abundant food compared with hosts given fewer resources (Bittner *et al.* 2002).

Within this analysis, the majority of studies examined parasites that have some intermediate value of virulence, which may have contributed to the discrepancy between theoretical predictions and the results presented here. It is possible that, as previously predicted, virulence does not alter the direction of local adaptation, but only affects the degree to which a parasite is locally adapted (Gandon 2002). Future work would greatly benefit from efforts to quantify virulence, thus allowing for more fine-scale cross-system analysis.

Obligate parasites and transmission mode

We predicted that, due to stronger selection on obligate parasites to adapt to their local hosts compared with non-obligate parasites, local adaptation should be found more often for studies examining obligate parasites, at least in terms of infectivity. We observed no trend with respect to obligate vs. non-obligate parasites, perhaps again due to the use of only two discrete categories. For example, ticks and fleas, which require hosts but do not have to live on or inside their hosts, were grouped with parasites that can exist in free-living form in the complete absence of the host, such as the facultative parasite *Lambornella clarki* (Ganz & Washburn 2006).

We did not test for a correlation between transmission mode and parasite local adaptation because more data are needed to make informative comparisons. In particular, as many parasites are transmitted both vertically and horizontally, it is often difficult to assess the impact of vertical transmission in a given host–parasite system. Experimental work has shown that established genotypes of a fungal pathogen were mostly due to horizontal transmission, despite the fact that the pathogen is usually transmitted vertically, indicating that the usual mode of transmission may not be the primary predictor of parasite fitness (Kover *et al.* 1997). Depending on how widely these results can be generalized, a low frequency of horizontal transmission may be sufficient to classify a parasite as being horizontally transmitted. Theoretical work also suggests that the rate of vertical transmission may not reflect its importance for coevolutionary dynamics (Lipsitch *et al.* 1995). The current analysis further emphasizes the need to quantify how the frequency of vertical transmission contributes to the establishment of parasite genotypes in natural systems.

Biases in experimental design and publication

Given the dynamic nature of host–parasite coevolution, it is possible that the experimental design used to examine local adaptation would, in itself, introduce a bias. As cross-population data are often used to make inferences about the coevolutionary pattern through time (Frank 1991), the outcome of a given experiment may depend on the number of populations sampled. For studies measuring infectivity, there was a slight, although insignificant, trend such that studies sampling one or two sympatric units more often found significant results (local adaptation or maladaptation) than studies sampling three or more units (Table 3). For studies using infection intensity as a measure of parasite performance, this pattern was significant (Table 3). As discussed above, infection intensity is a problematic measure of parasite performance because individual parasite populations may be adapted to different optimal infection intensities. The relationship between the number of sympatric units and the result reflects a significantly greater chance of finding no pattern of local adaptation as more populations are compared. Researchers should therefore be cautious in using this measure of parasite performance because of its sensitivity to the number of populations sampled. Although the corresponding trend for infectivity was not significant, it still raises concerns that researchers might be misled by results if they examine too few sympatric host–parasite combinations.

The number of sympatric units incorporated into an experiment is also related to the ability to detect host and parasite main effects, i.e. innate differences among host populations or among parasite populations (Thrall *et al.* 2002). The pattern of local adaptation cannot be statistically separated from these main effects when only one or two sympatric units are sampled (Kawecki & Ebert 2004). Many studies either did not test for main effects or did not report statistics for these tests, and a few were unable to detect main effects because of the experimental design used (Table 1). Of the studies reporting such information for infectivity, significant host main effects were found in 15 of 24 studies and significant parasite main effects were detected in 14 of 27 studies.

As discussed previously, there were two definitions of local adaptation used to analyse the data from cross-infection studies: (1) home vs. away, where the performance of parasite populations on 'home' (sympatric) hosts is compared to the performance on 'away' (allopatric) hosts; and (2) local vs. foreign, in which the performance of local (sympatric) parasites is compared to that of foreign (allopatric) parasites on a given host population. It has been suggested that host main effects can mask parasite local adaptation when the home vs. away definition is used (Kawecki & Ebert 2004), and

conversely, that parasite main effects can mask local adaptation when the local vs. foreign definition is used (Thrall *et al.* 2002). However, we found no significant differences in the likelihood of finding local adaptation (as opposed to no local adaptation) under the home vs. away definition when host main effects were present vs. when they were absent. Likewise, there were no significant differences in the likelihood of detecting local adaptation (compared with no local adaptation) under the local vs. foreign definition when parasite main effects were present vs. absent. Surprisingly, there was a trend towards finding parasite local adaptation more often when host main effects were present under the local vs. foreign definition. This result emphasizes the need to distinguish between host and parasite main effects and the interaction pattern that is characteristic of local adaptation.

While the majority of local adaptation studies incorporate three or more sympatric combinations into a partially or fully reciprocal design, most do not report analyses of main effects. Unless researchers account for host and parasite main effects (after Thrall *et al.* 2002), they cannot conclusively detect a pattern of local adaptation. Statistical methods must be used to isolate and identify main effects that may otherwise mask the pattern of local adaptation.

CONCLUSIONS

We found that high parasite migration rates relative to those of their hosts were associated with parasite local adaptation by infectivity, regardless of the definition used to analyse results, suggesting that this may be a particularly robust result. In accordance with theoretical expectations, relative generation time did not appear to have a significant effect on parasite local adaptation, but further analysis indicates this comparison had low statistical power. In addition, we did not find the expected patterns of local adaptation with respect to virulence, and there were no apparent differences for obligate vs. non-obligate parasites.

Consistent with prior analyses of cross-infection experiments, we found evidence that local adaptation studies should examine at least three sympatric host–parasite combinations and analyse host and parasite main effects to separate the pattern of local adaptation from population-specific differences in parasite infectivity and/or host susceptibility. If these guidelines are not followed it is more likely that the experimental design itself will influence the reported outcome of the study. Ultimately, more experimental and theoretical work is needed to explore fine-scale patterns of parasite local adaptation, and we hope this synthesis serves to emphasize where future studies could most profitably focus their attention.

ACKNOWLEDGEMENTS

We would like to thank F. Bashey-Visser, S. Hall, K.C. King, C.M. Lively, Y. Michalakis, P. Thrall, J. Wolinska, and two anonymous referees for helpful discussion and/or comments on previous drafts. In addition, we thank Y. Brandvain and E. Housworth for invaluable statistical advice. This work was supported by funding from the Indiana University Science, Technology, and Research Scholars (M.G.) and a NSF pre-doctoral fellowship to B.K.

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Editor, Peter Thrall
Manuscript received 3 November 2006
First decision made 20 December 2006
Manuscript accepted 13 February 2007