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New Idea

A hypothesis to explain host species differences in resistance to multi-host parasites

Mark R. Forbes and Julia J. Mlynarek

Mark R. Forbes (mark_forbes@carleton.ca), Department of Biology, Nesbitt Biology Building, Carleton University, 1125 Colonel By Drive, Ottawa, Ontario, Canada K1S 5B6

Julia J. Mlynarek (julia_mlynarek@carleton.ca), Department of Biology, Nesbitt Biology Building, Carleton University, 1125 Colonel By Drive, Ottawa, Ontario, Canada K1S 5B6

Abstract

Here, we offer a novel hypothesis to explain why some host species evolve resistance, whereas other related species remain susceptible to a shared parasite species. We first describe instances of single water mite species that are ectoparasitic on different species of host dragonflies, where the mites are killed by resistance mechanisms and have little to no fitness on some host species. This begs the question of why some host species are susceptible, whereas other host species are (nearly) completely resistant. Earlier logic based on parasites exploiting abundant host species at the cost of exploiting rare host species does not explain such instances well. Rather, a hypothesis based on closed populations of some host species being able to evolve parasite recognition is invoked. Parasite recognition is not expected to evolve in host species from more open populations with considerable gene flow across sites, only some sites of which have the parasite species present. The logic of this hypothesis can be explored with simulation models, whereas empirical tests could involve combined approaches using molecular genetics, population genetics, experimental infections and transplantation experiments.

Keywords: parasite-host co-evolution, host resistance, multi-host parasite, parasite host range.

Many species of parasites exploit several to many host species for a given stage of their life cycle (Lajeunesse and Forbes 2002, Poulin 2007). These broad host species ranges are still the case even after cryptic species are uncovered by genetic barcoding, i.e., some of the cryptic species are generalists (Hebert et. al 2004, Smith et al. 2007). In some parasite-host associations, host phylogeny is a main predictor of host species exploitation by parasite races or species, whereas in other associations, host ecology—particularly temporal and spatial overlap between host species—appears to be an important determinant of host species use (*cf.* McCoy et al. 2001, Perlman and Jaenike 2003, Krasnov et al. 2004, Dare et al. 2008, Mlynarek et al. 2012). Furthermore, host specificity need not be strong if the parasite derives resources from a tissue that has a blood-immunity barrier (e.g., eye flukes of some teleost fishes, Locke et al. 2010). To summarize, host species' phylogeny, ecology, and physiology might all be determinants of specificity (or generality) of host species use by parasite species, but the strengths of these determinants differ across different parasite-host associations. The same is likely true of other host-symbiont associations (Knee et al. 2012).

Amidst all of this potential variation in host species use, there appears a common recurrence, i.e. single species of parasites often exploit 'related' species of hosts. Many of those related species share ecological and life history attributes and, in some cases, show strong temporal and spatial overlap. Notwithstanding, those same host species often show differences in the degree of attempted or successful parasitism, presumably relating to subtle to more obvious interspecific variation in exposure to infective stages of parasites (Lajeunesse et al. 2004), or to interspecific variation in either resistance (Mlynarek et al. 2014) or tolerance

(reviewed by Schmid-Hempel 2011). This short paper is concerned with interspecific variation in resistance to single parasites species.

Researchers ought to study instances of strong differences in resistance between host species to single species of parasites. Such research will help predict factors important to changes in host species range of parasite species over ecological and evolutionary time scales. Broadening of host species range is a relevant research question given it was assumed that specialists were derived lineages subject to extinction risk; however, there are many examples where generalist species are derived from specialist lineages (Stireman 2005). Such research is particularly important during this era of emerging infectious diseases.

Here, we describe three similar patterns each involving a species of multi-host ectoparasitic mite subjected to differential resistance from species of dragonflies and damselflies (hereafter dragonflies). One possible determinant of such obvious differences in resistance to parasites is relative host species abundance. In each comparison, one host species is relatively abundant and the other species is relatively rare. In two of three cases, sibling host species are compared (thereby controlling for phylogeny). In those cases, the less common host species shows absolute or near absolute resistance to the mite species being investigated; whereas, the common species is very susceptible. One suggestion for this recurring pattern is that the common species is a larger target of selection for the parasite than is the less common species (*cf.* Forbes and Robb 2008). Such explanations do not capture other potential determinants of differential host species resistance to single parasite species. Insight is provided by exploring the recurring patterns in greater detail.

In eastern Ontario, Canada, two sibling species of dragonflies appear equally parasitized by a single species of mite, *Arrenurus planus* (Forbes et al. 1999). However, whereas one uncommon host species (*Sympetrum internum*) is almost totally resistant to this ectoparasite, the other much more common species (*S. obtrusum*) is almost totally susceptible. An unusual feature of mite-dragonfly associations is that resisted (dead) mites remain on the host individual and thus can be enumerated. Thus, instances of failed parasitism can be documented. For the second comparison, two dragonfly species in the genus *Nehalennia* have statistically indistinguishable levels of parasitism by the same *Arrenurus* sp. mite (Mlynarek et al. 2014). None of the mites on *Nehalennia gracilis* engorged successfully: they were all resisted. In comparison, all mites engorged successfully on the often more abundant and more regionally widespread *Nehalennia irene* (Mlynarek et al. 2014).

A similar finding was found for two distantly related species of dragonflies (*Leucorrhinia frigida* and *Nannothemis bella*) in a sphagnum bog. At that site, the ectoparasitic mite (*Limnochares americana*) timed its egg laying to coincide with the emergence of the more common *L. frigida*: a species very susceptible to this mite (Lajeunesse et al. 2004). That species had higher mite abundance and proportionately more live mites than the less common species. Individuals of the later emerging and less common *N. bella* might have resisted more mites because they were exposed to mites that had already searched for hosts and had exhausted resources needed to successfully parasitize. However, experiments showed that resistance was more often expressed by the less common species, even after controlling for variation in larval mite "vigor" (Lajeunesse et al. 2004).

This collective work suggests that differential *successful* parasitism is perhaps relatively common (see also Christe et al. 2003 for an example with sibling species of bats and ectoparasitic mites). But why does host species discrimination by parasites not evolve in such instances? Host species discrimination not evolveing could relate to costs of discrimination (*cf.* Robb and Forbes 2005) or limited need to discriminate when most hosts encountered are actually individuals of one common species. Intriguingly, other larval *Arrenurus* mites appear able to discriminate host species of mosquitoes at the pupal stage (Smith and McIver 1984). Similarities in measures of parasitism between dragonfly species, however, strongly suggest that host discrimination by these mites, if present, is extremely weak. Suffice it to say that differential host species resistance against parasites would not even exist were it not for non-discrimination of host species by parasites *in the first place*.

As mentioned, one explanation consistent with observations of rare and common species differing in their expression of resistance to single parasite species is that the more abundant and more regionally widespread host species is a stronger target of selection for the parasite than is the less abundant/less widespread host species. As such, mites should track abundant hosts and might evolve to evade their immune systems. One could thus reason that host species that are abundant and widespread are likely to be susceptible to parasites. The "target" hypothesis makes sense as parasites could adapt to common host species by evading immune system recognition, but this would likely trade off with being recognized by the less common species. Such a mechanism could work for the *Sympetrum*- and *Nehalennia-Arrenurus* associations (i.e., where the abundant host species is susceptible, and the rare species is resistant). Intriguingly, in the case of *Nehalennia*, the more resistant species *N. gracilis* actually shows less innate immunity (J. Mlynarek, unpublished data). This means that the differences in resistance are probably aligned with differences in recognition and not host immune ability *per se*.

The target hypothesis might make more sense were it not for the fact that in the case of *A. planus*, several species of *Lestes* dragonflies are also parasitized in addition to *Sympetrum* species. Some of those species show complete susceptibility and some show partial resistance (Yourth et al. 2001, Nagel et al. 2010), but none of the *Lestes* sp. show complete resistance. Here, host abundance at the study site seems unimportant in determining resistance level. Perhaps the other important factor is whether the host species being considered have populations from many sites with considerable gene flow, and at many of those sites, the specific parasites being considered are lacking. Here, having adaptive genetic variation for evading the host's immune system makes little sense (at least it is not parsimonious) as the parasite would have to track several regionally widespread species simultaneously (one evasion mechanism would not work that well).

It is relevant that *N. gracilis* is less regionally widespread compared with *N. irene*, but can also be less common at the site with the parasite (Van Gossum et al. 2007). In fact, recent work has shown that *N. gracilis* exists in highly genetically structured populations (Iserbyt 2012), suggesting low gene flow among populations. It would be interesting to compare the genetic structuring of *S. obtrusum* versus *S. internum* populations (and even *L. frigida* versus *N. bella*). One would expect that the latter species in each comparison exist in highly fragmented populations with limited gene flow. Gene flow is expected to have implications for which species might show resistance. We now know that *N. irene* is a widespread species that is not parasitized by the mite at many of its sites, simply because this parasite is absent from those sites. The same might be true of *S. obtrusum* and *A. planus*.

Assuming a genetic basis for the parasite recognition mechanisms (reviewed for insects by Wilson and Cotter 2013), one can ask under what conditions parasite recognition alleles would be favored versus not. To address this question for a closed population, one would have to be concerned with the fitness of four phenotypes of hosts. They are the following: those hosts with mites, and having parasite recognition alleles versus not, and, those hosts without mites and having recognition alleles versus not. From the relative fitness of these four phenotypes and assumptions about whether the recognition allele is dominant or recessive, one could calculate the average fitness of recognition alleles and determine under what conditions of parasite prevalence, recognition is partially or wholly favored. In such closed populations where mites are also present, all host individuals of that species have the possibility of encountering mites. Recognition alleles might be selected for, in such instances, because the costs of parasitism are expected to be more than the costs of recognition followed by resistance.

Contrast this with the other host species like *N. irene*, which are part of more open intraspecific populations. Most of the individuals in these populations do not even encounter mites of particular species. It is safe to assume that the relative fitness of the recognition allele might not be influenced much by the benefits such recognition alleles confer in the proportionately few individuals parasitized by mites. The focus on evolution of parasite recognition by hosts, rather than parasites evading immune systems of common or widespread hosts, seems more productive in explaining nature. Those host species in open populations have not evolved parasite recognition because most do not encounter strong selection from the particular parasites under study. Yet, they seem (somewhat paradoxically) to be the host species that are susceptible to the parasite in zones of overlap (i.e. subsets of their population are under selection).

Our contention is that advances in understanding factors influencing host species use by parasites can be aided by studying variation across related host species in expression of resistance to multi-host parasites. We note that some closed populations of host species can seem removed from selection imposed by parasites because they have evolved fixed resistance to the parasites. This might at first seem paradoxical given the expected asymmetry in selection on parasites and their hosts. This asymmetry in strength of selection is expected because many more parasite offspring are produced relative to host offspring; because all obligate parasites need a host to complete their life cycle, but not all hosts have to resist obligate parasites (in fact, many host individuals remain uninfected), and, because parasites typically have shorter generation times than their hosts. Although mites herein described have generation times *on par* with their dragonfly hosts, the two other conditions for asymmetry in selection are met.

In Figure 1a, we highlight the situation that is being described using two host species in two imaginary populations. One host species, *h1* (filled squares), exists at a single locality in a closed small population (small circle). *h1* is less numerous than host species *h2* (open squares) at that site. *h2* is not only more common at the site harbouring *h1* but also is very common at a nearby locality (larger circle) with considerable bidirectional gene flow (represented by the double-headed arrow). We now consider the parasite in Figure 1b. Here, a single parasite species, *p1* (filled dots), is exploiting two host species (*h1* or *h2* again represented by filled and open squares). All three species are syntopic (which is why the parasite is added to the locality represented by the small circle in Figure 1a). The species represented by the open squares is also present at many other sites (one such population being represented by the larger circle in Figure 1a) and not just the few sites where it co-occurs with its congener and the shared parasite

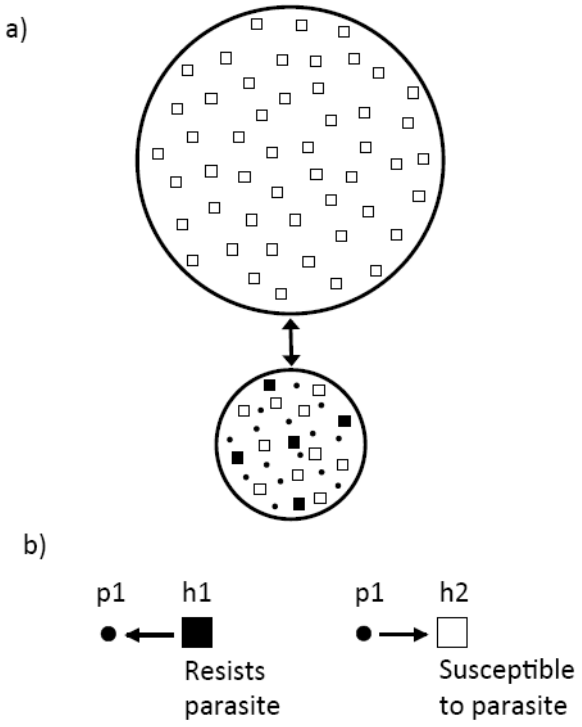

Figure 1. a) A representation of relative abundance and regional representation of two host species, at two localities represented by small and large circles. Double headed arrow connecting localities depicts bidirectional gene flow for the more abundant and widely distributed species (open squares). **b)** *Left panel:* the magnitude and direction of selection, represented by backward-facing arrow, on the parasite (filled dot) by the host (filled square). *Right panel:* the magnitude and direction of selection (forward-facing arrow) on the host (open square) by the parasite. The two panels represent species interactions at the locality where all three species co-exist (within the small circle in Figure 1a). Species represented by filled symbols exist in largely closed populations, whereas *h2*, represented by an open square, is in a more open population. The expression of resistance predicted for each host species is indicated. See text for further details.

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species. The arrows in Figure 1b represent which species has the advantage in the interaction (forward pointing, parasite has advantage; backward pointing, host has advantage despite expected asymmetry in selection). Species represented by filled symbols exist in largely closed populations (*p1* and *h1*); *h2* exists in a largely open population. In this particular series, the host is either a habitat specialist in a closed system and thus rare (*h1*), or is a widespread host that is locally abundant and in an open system (*h2*). The text refers to the prediction about the evolution of host resistance based on the direction and magnitude of the interaction (from complete resistance to complete susceptibility). This situation is what has been observed in nature for at least two study systems already described with sibling host species, and might well be representative of many parasite- multi-host associations.

We know in the case of *Nehalennia*, *h1* (*N. gracilis*) is a habitat specialist, existing in a closed system with the parasite; *h2* (*N. irene*), by comparison, is a widespread host species and is locally abundant at many sites, including those where *N. gracilis* is also present. Importantly, *N. gracilis* shows considerable population genetic structure between sites, as compared with *N. irene*, suggesting little gene flow in the former species (Iserbyt 2012). *h2* is expected to be a target of selection by the parasite because *h2* is common, and because *h2* individuals that resist and pay a cost of recognition or resistance are swamped by gene flow from *h2* individuals with higher fitness from sites where the parasite is absent. *h2* should thus be susceptible, both if found alone or found with *h1* in specialized habitats.

Does consideration of relative host species abundance and population structure (open versus closed populations for both parasites and hosts) actually help explain the evolution of differential resistance? And can this be seen as a special case of the geographical mosaic of coevolution (e.g., Thomspon 2005)? We propose various studies to vet and test predictions and assumptions of this verbal model. First, it might be useful to elucidate more of what is known. For starters, *A. planus* is an ephemeral pond specialist which has many implications for its life history (Nagel et al. 2010), including the possibility of its being in fragmented populations. As mentioned, *S. internum* might also be a habitat specialist. The genetic sub-structuring of *Sympetrum* spp. would be useful to explore. In a similar vein, it is known that *N. bella* is a bog specialist and might well show little gene flow, similar to *N. gracilis*. In comparison, the *L. americana* mite is expected to be in an open population. This scenario might result in the mite being "a moving target" for recognition in a system in which resistance is otherwise selected (the outcome being partial resistance).

To test ideas outlined here, more research is needed on the factors accounting for differential resistance to parasites among related host species (or differential parasite-induced mortality, e.g. Thomas et al. 1995). Such occurrences are probably common in nature, but can be 'invisible'. One problem is that the immune systems of the resistant species might remove all signs of past infections. Such studies should be supplemented with genetic barcoding on live and dead parasites to ascertain whether they are the same species. One can also use experimental infections to address whether differential resistance is real. Following such investigations, researchers could examine the population

genetics of related host species to determine if the partially or wholly resistant species exists in more genetically substructured populations than the susceptible species. Such surveys would be improved by monitoring the presence or absence of the parasite at sites where samples for population genetics are taken. Finally, studies could be supplemented with molecular genetics work to determine if adaptive genetic variation for parasite recognition resides in either or both of the host species. Many of these studies could be conducted concurrently rather than in a linear fashion: identifying associations showing differential resistance → genetic barcoding of parasites and experimental infection→ population genetics while recording presence and absence of all species at sites → molecular genetics of recognition mechanisms.

Transplantation experiments of hosts from the sites where differences in representation of host and parasite species exist also can be insightful. We might otherwise simply be ignoring a lot of how nature packages itself. For example, at sites lacking *h2*, it is quite possible the parasite has gone extinct because of *h1's* resistance but subsequent selection has been relaxed in terms of recognition followed by resistance. Parasitizing *h1* from such sites could elucidate whether adaptive genetic variation in recognition has been lost from the population as expected. If it is just that parasites have evolved repeatedly to evade immunity of common host species, then complete resistance will still be expected. The prediction is not risky in that there simply might not have been enough time elapsed since the parasite has gone locally extinct to select against recognition alleles. Predictions involving the relative fitness of different parasite lineages can be complex (see Vesakoski and Jormalainen 2013 for treatment of this problem in the study of local adaptation largely in herbivorous insects).

Finally, this problem could be tackled with mathematical (simulation) models, which are particularly useful when investigating scenarios where the behaviour of a system involves multiple contributing factors. Such models could simulate the dynamics of open and largely closed host populations to determine how host species interact with parasites both within and across populations. Models could also elucidate what other conditions in fragmented populations—including probability of becoming parasitized, costs of parasitism, cost of genetic basis for recognition, and cost of resistance—favour recognition becoming fixed. Such models are currently being developed. Once complete, the predictions of these models can then be tested quantitatively with detailed field studies, which include population size estimates and prevalence of infections.

In summary, evolving parasite recognition might be more important than evolving host immunity evasion for explaining instances of differential resistance to multi-host parasites. Such recognition might not be selected against in closed populations of hosts. Counter-selection by the parasite might not be expected providing the host species, which shows resistance, is actually a small target of selection. In a similar vein, host nondiscrimination by parasites might not be selected against and might be one reason why such spectacular examples of failed parasitism due to host resistance are visible in nature.

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Referees

Alex Cordoba-Aguilar -- acordoba@ecologia.unam.mx
Universidad Nacional Autónoma de México

George Lozano – dr.george.lozano@gmail.com
Estonian Centre of Evolutionary Ecology

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Response to referee

In *The Extended Phenotype*, Dawkins (1982) cajoles us about advocating a point of view: "As is the case with advocates, I shall make the strongest case I can, and this means the case for wilder hopes rather than the more cautious minimum expectations." Lozano (2014) has aptly expanded our hypothesis on resistance evolution in relation to multi-host parasites to other questions including the evolution and efficacy of innate anti-parasite defenses, the evolution of sexual ornaments as putative signals of parasite resistance, and the

importance of different host species as reservoirs of emerging infectious diseases. Lozano has jarred us from our narrow focus on the evolution of dragonfly resistance to their ectoparasitic mites. True, our "coevolutionary release hypothesis" might have merit wherever there is a parasite recognition element involved in the parasite-host phenomenon under study. Simply stated, some host species are expected to be good hosts, providing those host species are abundant and/or regionally widespread, and exist in open interconnected populations. Closed or fragmented populations of different host species (or other evolutionarily significant units) might evolve recognition of multi-host parasites and this could lead to effective behavioral or immunological resistance to those parasites.

Another important point by Lozano is that defense can occur at different layers. For example, it would be useful to compare traits like grooming efficacy against, and engorgement rates of, shared ectoparasites on different host species. The host species of interest for comparison would be those that come from both open and closed populations. Our coevolutionary release hypothesis currently does not make predictions about which traits should evolve as defense against parasites, but rather only that resistance should be more likely to evolve when selection for recognition of parasites is not countered by selection on parasites to evade host recognition (i.e., for hosts from closed populations attacked by multi-host parasites). Here, the evolution of a parasite species viz à viz its interactions with one host species constrains its evolutionary trajectories in the context of its interactions with one or more other host species (no surprise there). What is surprising perhaps is the ease of testing which host species should be targets of co-evolutionary covenants and which become more or less removed from selection imposed by the parasite.

Lozano further extends our hypothesis to look at interspecific variation in resistance in his second example on the evolution of sexual ornaments. Some populations within species can become isolated from others, but still have had multi-host parasites to contend with. Here, the relative fitness of four types of phenotypes again becomes important: those unexposed to parasites, but with expression of recognition alleles (1) versus not (2); and, those exposed to parasites but with expression of recognition alleles (3) versus not (4). If a sufficient proportion of hosts are exposed to parasites, then parasite recognition and resistance might evolve. However, if those hosts are swamped by hosts that are unexposed to parasites, but which otherwise pay costs of parasite recognition or resistance expression, then resistance evolution would falter. Such swamping is unlikely to occur in closed populations of host species; so, resistance might become fixed (producing either intra- or interspecific variation in resistance). The logic of such arguments can be tested using simulation models, as alluded to above. But such models would not address intra- or interspecific variation in expression of condition-dependent sexual ornaments. More sophisticated models are needed that would link parasite resistance to condition, and condition to expression of such ornaments and mate choice based on expression of those ornaments (*cf*. Rowe and Houle 1996). However, Lozano's thought experiment does raise the issue of what happens when resistance becomes fixed (is there less variation in certain sexual ornaments than in other host populations/species where parasites are still present and resistance is variably expressed?). Sexual ornaments are complex and thought to reflect general condition dependence so empirical tests would have to consider situations where many parasites species are lost (e.g., birds in isolated populations on islands or breeding in the Arctic). If many parasites are lost then perhaps there is less variation in offspring survival based on parasitism and less need for elaborate sexual ornaments as putative signals.

The final area that Lozano highlights is in the realm of emerging infectious diseases. Here, he argues about the importance of knowing which of a suite of potential host species will act as disease reservoirs and aid in disease transmission. There is, as Lozano notes, considerable research effort being directed at testing suitability of host species to emerging parasites and pathogens. Our hypothesis might aid in predicting which host species are predisposed to evolve resistance to parasite or pathogen attack, thereby thwarting parasite reproduction and transmission, but whether it can be useful in making predictions on ecological time scales remains to be seen. As stated earlier, the hypothesis does not make predictions about which specific resistance traits should evolve in closed populations, nor does the hypothesis make predictions about whether tolerance to parasitism or infections evolves more readily than resistance. Yet hosts that tolerate an infection will be better at transmitting the disease as compared with hosts that resist such an infection (Råberg et al. 2009). We think future studies should ask which populations (closed or open) or species are more likely to evolve tolerance to infection as opposed to resistance to infection. Such studies might inform which species mixes in communities are more suitable for disease control.

We currently do not know the full reach of the hypothesis we have offered. It was intended to provide testable approaches to simply explain interspecific variation in resistance evolution. Our danger is one of inductive reasoning. Our hypothesis might have little reach, but we do not think this is so and are heartened by Lozano's critique and logical extensions.

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