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Perspective

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Parasitism as a driver of host diversification

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Abstract

Parasites are ubiquitous and frequently impose strong deleterious fitness effects on host individuals. These effects often manifest at the microevolutionary scale through host assortative mating and local adaptation. At the macroevolutionary scale, parasite-mediated effects can not only cause population divergence leading to speciation but also cause extirpation of host populations, leading to extinction. The balance between parasite-mediated effects on both speciation and extinction determines species diversification patterns. However, empirical tests of the hypothesis that parasitism contributes to macroevolutionary dynamics of host speciation and extinction are lacking. In this Perspective, we discuss how parasites can affect host macroevolution and outline an approach to determine whether parasitism and diversification are linked. We predict that parasitism, similar to other species interactions, shapes the process of diversification specifically in host species. Testing this hypothesis will require further empirical research to investigate the role of parasitism driving host diversification across the tree of life.

Sections

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Introduction

Antagonistic interspecific interactions frequently contribute to species diversification[1–3]. As traits evolve to mediate such interactions, populations can undergo divergent selection, experiencing reproductive isolation as a by-product and driving ecological speciation[1,4]. Even within a single species, different populations often face various natural antagonists and thereby experience different selective pressures exerted by local interactions[5]. Theoretical studies also predict that species interactions can cause extinction[6], with one species affecting survival, fecundity and hence population density of another species. Ultimately, the balance between speciation and extinction determines macroevolutionary patterns of lineage diversification.

The role for antagonistic interactions, such as predation and resource competition, as drivers of diversification has been extensively studied[1,4], but a role for parasitism is largely unexplored[7]. However, parasites are ubiquitous across the tree of life and have evolved numerous adaptive strategies to successfully exploit their hosts (Fig. 1). Hosts infected with parasites often suffer considerable fitness losses[8] and are selected to evolve adaptations to overcome these fitness losses. The limited exploration of how parasitism shapes species diversification represents a knowledge gap in understanding the evolutionary processes that have shaped the accumulation and loss of biodiversity through time.

A drive to understand speciation and adaptive radiations has inspired pioneering work attempting to link the fitness costs of parasites to reproductive isolation and ecological speciation of their hosts via three main criteria[7]: parasites are expected to contribute to divergent selection if host populations vary in their infection profile; if these differences in infection profile remain consistent through time; and if parasitic infection imposes fitness costs on the hosts that are strong enough to overrule conflicting fitness costs from other sources[7].

Cichlids[9–11] and sticklebacks[12] have been used as model species for empirical tests of these criteria, although the results are system-specific and inconsistent. Parasite communities of cichlids in Lake Tanganyika[11] and sticklebacks in the Outer Hebrides of Scotland[12] differ substantially in their infection profiles among replicate populations, and these differences remain consistent over 2 (cichlids) and 6 (sticklebacks) years of sampling. Furthermore, stickleback hosts are more resistant to their local (rather than non-local) parasites, highlighting selection against immigrants in driving post-mating isolation[12]. However, cichlids in Lake Victoria do not exhibit signs of parasite-mediated speciation, despite host species consistently varying in their infection profile over time[9,10]. This suggests that although parasites might have contributed to host differentiation after speciation, they probably did not drive it.

Although contributions of parasites to the ecological speciation of cichlids and sticklebacks are inconclusive, understanding the macroevolutionary effects of host immune defences against parasitism can offer some insight. The evolution of the immune system influenced speciation in teleost fish, with lineages that possess a high copy number of the major histocompatibility complex (MHC) I gene having elevated rates of speciation[13]. Although the understanding of parasitism that contributes to speciation dynamics is growing, the contribution of parasitism to extinction remains largely unexplored, especially from an empirical basis.

In this Perspective, we present a conceptual framework highlighting the mechanisms by which parasitism can shape diversification patterns and describe an empirical approach for determining whether parasitism is linked to diversification patterns. This framework will support and guide future investigation of how parasitism can shape species diversification. This will be achieved by building on previous research on parasitism and host evolution by considering the interplay between speciation and extinction to empirically link variation in parasite pressure to macroevolutionary patterns of host lineage diversification.

Linking diversification and parasitism

Parasitism is widespread — up to half of all known organisms may be parasites[14,15] and virtually every organism is parasitized[16], including invertebrates (Fig. 2a,b), plants (Fig. 2c), vertebrates (Fig. 2d,e) and fungal species (Fig. 2f). These parasites can only be parasites during their larval stage (Fig. 2a), force their hosts to defend them (Fig. 2b), become part of their host's anatomy (Fig. 2d) and/or feed off of the host directly (Fig. 2c,f). However, variation among populations in the diversity and intensity of parasitism is common[17–19] and such variation is probably a key facet underlying the diversification process. In this section, we explore the effects and mechanisms of parasite–host interactions in evolutionary diversification.

Parasite-mediated effects on diversification

Parasites can be a source of divergent selection among host populations[7], capable of influencing mate choice[20,21] and drastically reducing host fitness[8]. In addition, theoretical models predict that parasites can drive host populations extinct[22,23]. The evolutionary maintenance of sexual reproduction among eukaryotes is thought to

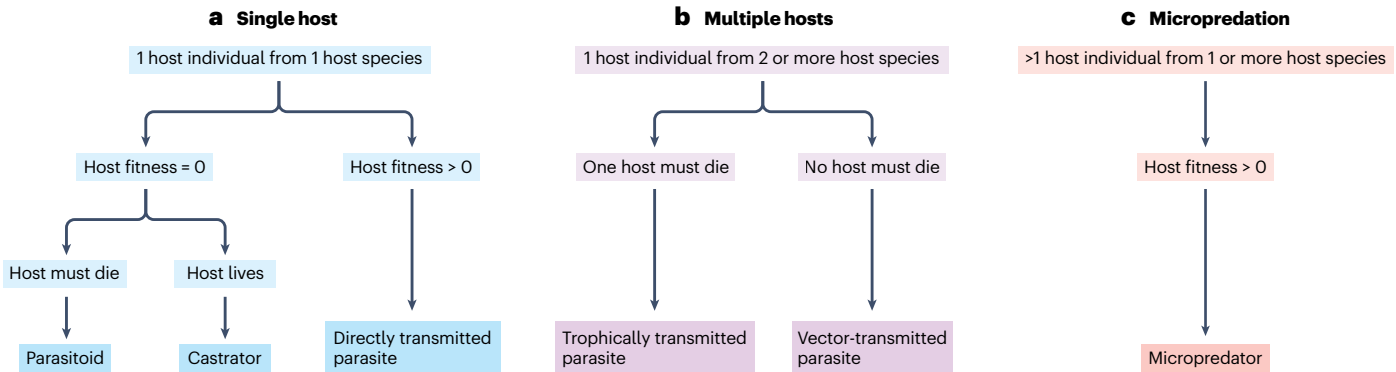

Fig. 1 | Parasite evolutionary strategies. a, Parasite strategies for a single generation using a single host of a single species. **b**, Parasite strategies for a single generation using single host individuals across two or more species. **c**, Parasite strategy for a single generation using multiple host individuals across multiple host species. In total, there are six evolutionary strategies used by the vast majority of parasites[105].

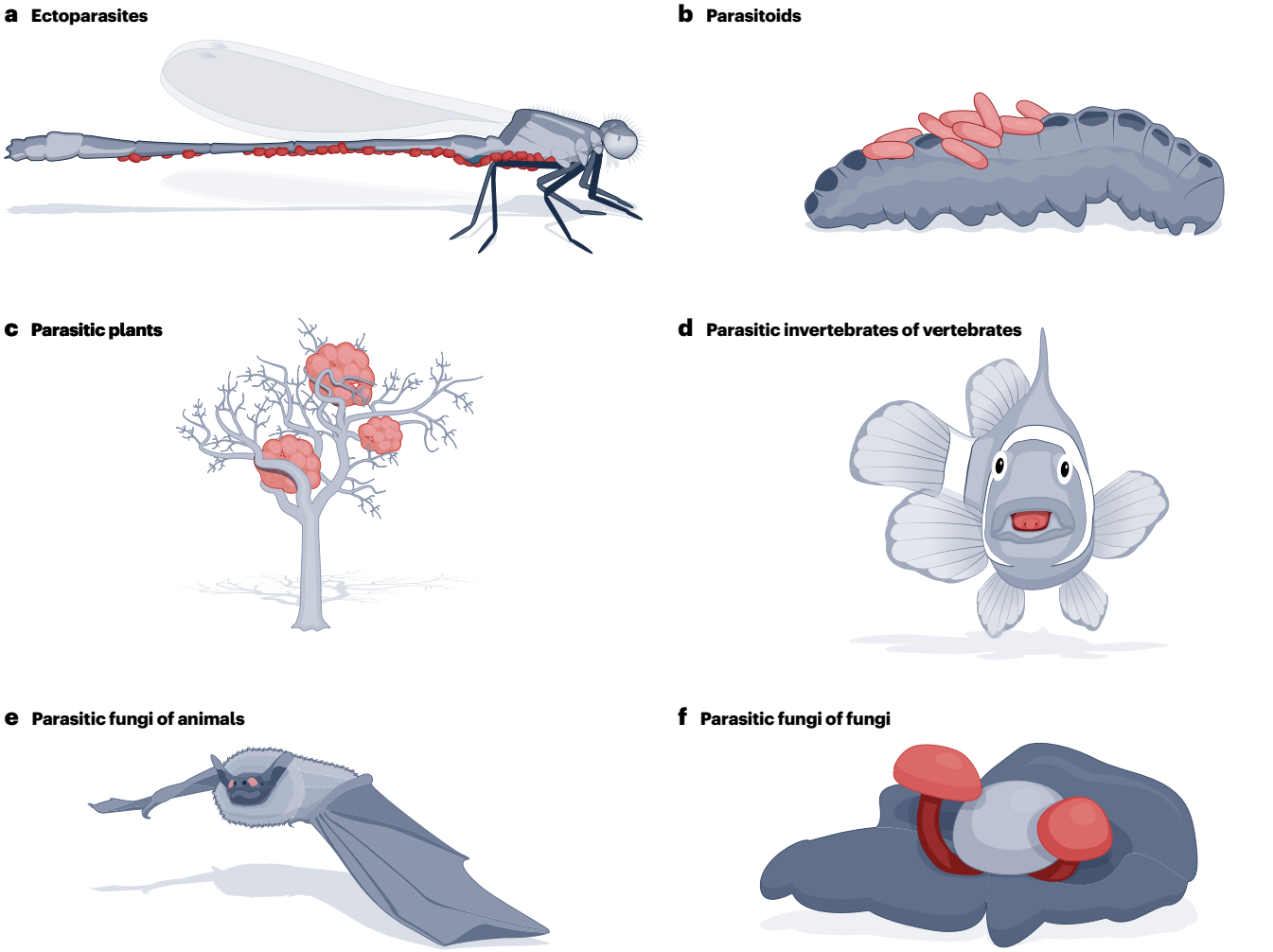

Fig. 2 | Parasite infection strategies. a, Damselfly infected by ectoparasitic mite larvae that reduce flying ability and mating success. **b**, Caterpillar infected by parasitoid wasp larvae that consume the host from the inside and force the host to protect them. **c**, Lime tree infected with mistletoe that reduces growth and survival. **d**, Clownfish infected with a tongue-eating isopod that steals resources, blood and tissue from its host. **e**, Bat infected with a fungal parasite that induces white-nose syndrome and has decimated bat populations. **f**, Mushroom infected with another, parasitic mushroom that affects host growth and reproduction. Host species are coloured grey, and parasites are coloured red.

result from parasite-mediated selection[24,25], although predators can also select for sexual reproduction in their prey[26]. The resulting genetic variation that sexual reproduction produces provides the raw material for selection to act upon, subsequently driving host diversification[7].

The hypothesis that parasites can cause host speciation is not new[27,28]. Parasites can increase opportunities for host selection at the microevolutionary scale among diverse taxa[8], and parasites are also responsible for the incredible diversity found in some regions of the host genome, especially those involved in disease resistance (such as R genes in plants[29,30] and the MHC in vertebrates[31,32]). Plant systems particularly support the role of parasites in driving and/or contributing to speciation. Specifically, hybrid necrosis results in either differentiating populations (in the case of active speciation occurring) or co-occurring species (in the case of already-speciated taxa) producing offspring in the F_1 generation that suffer from deleterious phenotypes (such as greater susceptibility to viral infections[33–35] or lethal necrosis[36,37]). Owing to their deleterious phenotypes, these offspring rarely reach reproductive maturity[38].

Theoretical models of host switching support the idea of parasites increasing host diversity at the macroevolutionary scale, with lineages harbouring greater numbers of parasites experiencing greater turnover[39]. However, insights from empirical analyses conflict with the theory-driven expectation. Host populations of the bacterium *Pseudomonas aeruginosa* display more rapid adaptive evolution and diversification when parasitized by more diverse assemblages of viral parasites under experimental conditions[40], supporting the model of parasite-driven diversification. By contrast, parasites strengthen differences among host species in African cichlids[10], but do not drive speciation in a radiating clade[9]. Therefore, it is necessary to discriminate between parasites being the main drivers initiating host diversification and parasites contributing alongside other ecological factors to host diversification. Additionally, although both parasite-mediated

extirpation of hosts[41] and host population declines[42] are not uncommon, definitive empirical evidence for parasite-driven extinction is lacking[43].

In natural populations, the direction and magnitude of the effect of variation in parasitism on host diversification remain unclear. Given that parasitism occurs in a broad ecological context, it is often difficult to disentangle the role of parasitism in host diversification from that of other ecological factors. For example, trait evolution to escape predators or avoid competitors could also result in a shift to habitats with different parasites, which could in turn affect mate choice, contributing to the evolution of reproductive isolation[44]. Such complexities probably account, in part, for the lack of studies linking parasitism to diversification. Three key microevolutionary mechanisms could underlie the effect of parasites (alone or in tandem with other species interactions) on host macroevolutionary dynamics: assortative mating, local adaptation and extirpation (Fig. 3). Although we focus on these three mechanisms in this section, we encourage the development of understanding other ways in which parasitism may drive host diversification.

Assortative mating

Assortative mating can be positive (matings between phenotypically similar individuals) or negative (matings between phenotypically dissimilar individuals)[45]. Phenotype, in this context, can be broad — reflecting not only morphological or behavioural traits but also features such as habitat choice. Assortative mating in animals tends to be positive, although weak, according to a meta-analysis of 269 studies[46]. Assortative mating could lead to speciation through parasitism if quantitative or qualitative differences in infection lead to reduced gene flow between hosts differing in their infection profile (Fig. 3a), with infection profile being the phenotype that affects mating. For instance, infection with acanthocephalan parasites can alter the microhabitat choice of their isopod hosts[47,48]. As a result, non-infected hosts are more likely to encounter and mate with one another than with infected hosts, whereas infected hosts are more likely to mate with other infected hosts. Thus, assortative mating arises through parasite-induced habitat choice.

Parasite-mediated assortative mating could also result in situations in which alleles associated with parasite resistance or avoidance tend to remain with less-infected hosts, whereas alleles associated with susceptibility to parasites tend to remain with more-infected hosts. Therefore, mate choice could be under parasite-mediated selection, as parasites can affect general host condition and the display of secondary sexual traits[20]. For example, the male nuptial coloration of cichlid and of stickleback fish covaries with their infection status[49,50] and it affects female mate choice[51,52]. As a result, less-infected males that are more colourful mate more often and their offspring are more resistant to parental parasites[53]. Assuming minimal gene flow between the two host types (less infected versus more heavily infected), this could eventually lead to speciation as hosts differing in infection profiles become genetically distinct, leading to the evolution of reproductive isolation. Importantly, for such parasite-mediated selection to drive assortative mating, infection prevalence would have to be high in the host population, infection would have to be concurrent with and/or affect host reproduction and infection would have to impose strong fitness costs. That said, if the fitness costs of infection are too severe, parasite-mediated assortative mating might not evolve fast enough, leaving ample opportunity for the heavily infected and less-infected hosts to mate and reduce reproductive isolation.

An intriguing counterexample is that of the MHC in vertebrates, which is involved in both adaptive immunity against parasites and mate choice. Whereas the majority of assortative mating in animals is positive[46], assortative mating tends to be negative with respect to the MHC[54,55]. The MHC hypothesis suggests that vertebrates seek to maximize offspring fitness by mating with individuals that possess complementary (but not identical) MHC genes, thus mating with dissimilar individuals[56]. Heterozygosity at MHC loci enables an immune response against a broad array of parasites, favouring gene flow between hosts differing in infection profiles and hence hampering host speciation. Despite the prevailing idea of negative assortative mating being associated with MHC, positive assortative mating for MHC is possible[57–59] through homogenizing selection if some alleles are more effective against a specific parasite[60]. If some MHC alleles are more efficient against a specific parasite, they will be selected in environments in

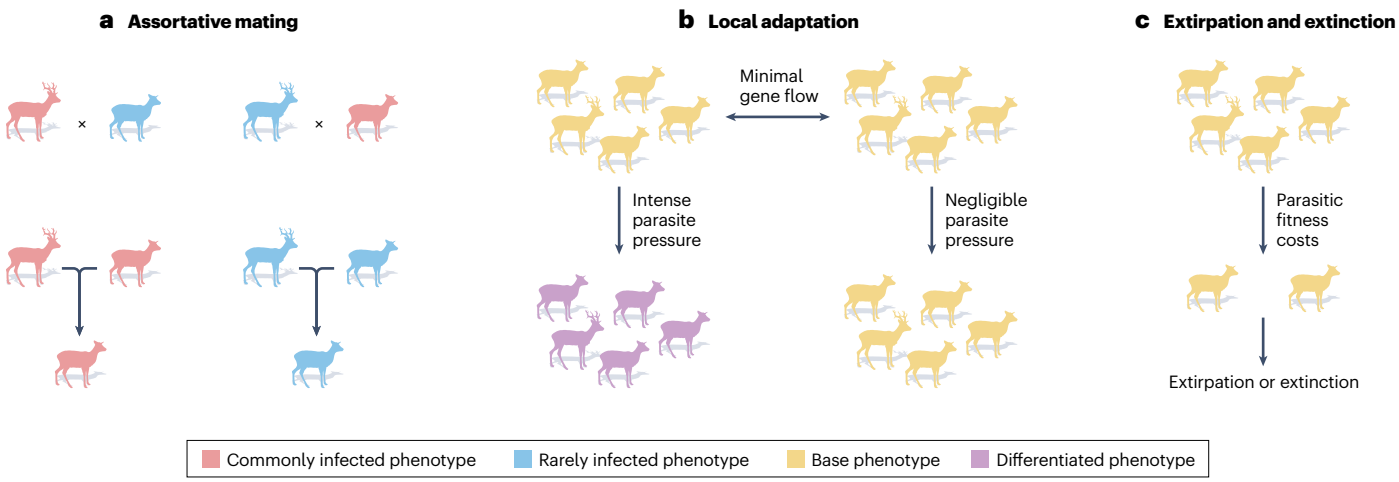

Fig. 3 | Parasite-induced evolutionary effects. a, Parasite-driven assortative mating, in which individuals mate with phenotypically similar individuals (red and blue denote two different phenotypes), which differ in their ability to defend against parasites. **b**, Parasite-mediated local adaptation can drive phenotypic divergence between two similar, non-interacting populations (yellow) as selection for parasite resistance occurs in one population (purple) but not another. **c**, Parasite-induced extinction via extirpation can occur as rising parasite pressures apply intense fitness costs inducing population decline.

which such parasites exert strong selection[61], potentially leading to positive assortative mating and providing offspring with higher resistance as a by-product and favouring host diversification[60].

Local adaptation

Within species, host populations often vary in the infection profile they experience[62–66]. Although these differences in parasitism can often be attributed to the local environment hosts are exposed to, local adaptation provides another explanation[67]. Populations of hosts and parasites tend to vary in their ability to defend themselves from parasitic attack[68–70] and successfully infect hosts[64,71], respectively. Parasites can impose strong selective pressure on their hosts[8], and when combined with varying diversity and/or levels of parasitism among host populations, differential adaptive evolution may occur. Specifically, host populations experiencing greater parasite-mediated selection might adapt by developing defences, such as heightened immunocompetence or specialized behaviours. Such adaptations would not be expected to emerge within populations experiencing weaker parasite-mediated selection as defences are energetically costly, or if they do, such differentiation could be trivial (Fig. 3b). Subsequently, non-local hosts (immigrants or hybrids between non-local and local individuals) should have reduced fitness compared with local hosts owing to parasite-mediated selection[12,28], reducing gene flow between local and non-local hosts. Heavily infected hosts might also be unable to disperse as far as lightly infected hosts[72], offering another avenue by which gene flow between heavily infected and lightly infected populations can be reduced. Without extensive gene flow between populations experiencing varying parasite profiles, such divergent parasite-mediated local adaptation could lead to reproductive isolation and eventually ecological speciation[1,4].

However, it should be noted that in some cases, local adaptation of parasites could act against host diversification. Namely, because parasites are often more adapted to infect their local hosts over non-local hosts[67], migrant hosts can actually gain a fitness advantage. Damselflies[64], guppies[73] and sticklebacks[74] have all demonstrated that non-local hosts experience reduced parasitism relative to the local hosts in experimental settings. This creates a situation in which parasitism reduces the evolution of reproductive isolation between populations by enhancing gene flow.

Extirpation, extinction and population declines

Little is known about parasites driving or mediating host population extirpation and host species extinction in natural systems. Mathematical theory suggests that host populations will be extirpated if the individuals comprising them are unable to overcome fitness costs associated with parasitism[22,23]. Such extirpation is expected to be less likely for specialist parasites, as smaller or threatened host populations have fewer specialist parasites[75]. Therefore, assuming density-dependent transmission, specialist parasites will be extirpated once host population density drops to a certain point (see ref. 76 for an example of parasites persisting in low-density populations). Direct empirical evidence of extirpation in wild systems is lacking[43], although studies of invasive species offer insight into how parasite-induced extirpation could lead to host extinction. Specifically, co-introduced parasites have been implicated in the replacement of native red squirrels (*Sciurus vulgaris*) by invasive grey squirrels (*Sciurus carolinensis*)[77]. Squirrel parapoxvirus causes deleterious disease in red squirrels but not in grey squirrels, which act as a reservoir. Similar cases of parasite amplification in native hosts by invasive hosts have also been seen in amphibians[78]. Other examples of parasite-mediated extirpations such as trypanosomes in Christmas Island rats[79], chytrid fungus in Panamanian frogs[80] and white-nose syndrome in bats[81] can all be linked to human activities already impacting these host species. Quantifying parasite-induced extinction needs to be addressed with further study, and the documented parasite-mediated extirpations and population declines from myriad host species represent another pathway by which parasites can affect hosts on the macroevolutionary scale (Fig. 3c). It remains an important goal to understand whether parasites can drive host populations extinct in natural systems absent human influence.

Quantifying parasite-mediated effects

Testing the hypothesis that parasitism contributes to host diversification should use phylogenetic models to investigate how traits associated with parasitism, and/or measures of the extent of parasitism, are correlated with the accumulation and loss of species among host clades over their evolutionary history (Fig. 4). In this section, we outline the broad steps necessary for empirically linking parasitism to lineage diversification.

Field data

Broadly sampling host lineages across the host tree (Fig. 4a) requires collecting field data on parasitism or traits mediating parasitism (such as MHC alleles) from multiple host species across multiple populations across the range of each host species sampled, with each species mean weighted by its sampling error. Failure to sample broadly among multiple host lineages will limit any kind of inference, as results could be biased depending on taxa included in the analysis. Broad sampling also ensures that traits of interest are representative of the whole clade. That is, if all samples (host lineages) come from one taxonomic group that is rarely parasitized, it will be difficult to link any macroevolutionary trends when all host taxa included rarely encounter parasites. Similarly, if all populations and lineages are heavily parasitized, there would be no phylogenetic variance, making comparative study infeasible. Thus, it is critical to sample host species that vary in the extent of parasitism or traits mediating parasitism.

An additional and important consideration relates to measuring parasite-associated traits of the hosts. A trait-based approach could be illuminating and offer insight into mechanisms by which parasites affect patterns of host lineage diversification, yet it might not always be possible to identify and/or quantify such important traits in the wild. Therefore, an approach linking variation in parasitism to diversification is ideal for such situations in which these key traits are unknown. This task is not easy, especially given spatial and temporal variation in host parasitism.

Additionally, initial sampling efforts should focus on only one higher parasite taxon, chosen either for their known fitness impacts or because they are the most common parasites within the chosen host group. Mixing multiple parasite types (for example, gastrointestinal helminths and ectoparasitic mites) will make interpretation of the results difficult. On a microevolutionary scale, parasite-mediated effects on host species interactions have no overall effect on host survival and fecundity, according to a meta-analysis, yet the individual effect sizes (mostly among species) varied wildly in direction[82]. It is therefore possible that a macroevolutionary analysis including multiple parasite groups would erroneously reveal the lack of a relationship with host diversification, despite such relationships existing for individual parasite taxa. This scenario could occur if, for example, one parasite was associated with increased diversification, whereas another

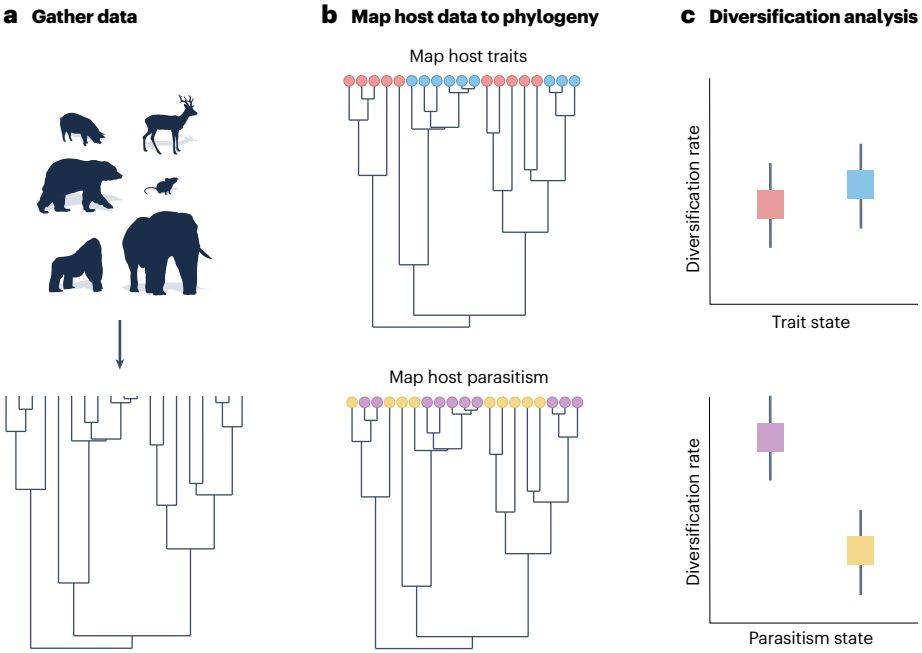

Fig. 4 | Framework to explore the link between parasitism and host diversification. a, Data collection from multiple host–parasite pairs and the construction of a phylogenetic tree. **b**, Mapping host parasitism-mediation traits (red denotes no mediation trait, blue denotes presence of a mediation trait or host parasitism levels, purple denotes high parasite infection and yellow denotes low parasite infection) onto the phylogenetic tree. **c**, Perform phylogenetic analyses to determine a link between host traits or parasite data and macroevolutionary diversification dynamics. In this hypothetical example, the purple parasitism state is associated with increased diversification rates.

was associated with reduced diversification, resulting in a null overall relationship and thus further obscuring interpretation. Given that most hosts are infected with multiple parasites (which could interact with one another), understanding how multiple parasites might affect diversification is an important future direction. It could well be that interactions among multiple parasites (or their combined, synergistic effects) are a key driver of host diversification. The lack of a detectable effect at the macroevolutionary level, despite seeing such an effect at the microevolutionary scale, is biologically meaningful and provides insight into the mechanisms by which evolutionary processes do or do not occur. The accumulation of many microevolutionary processes might not simply scale up to a detectable macroevolutionary pattern. Thus, this possible scaling effect emphasizes the need for macroevolutionary investigations. Furthermore, given the need to analyse multiple host types together, it could be just as informative and important to include multiple parasite types.

After collecting trait data on host hosts and parasitism, the next step is building a host phylogenetic tree to conduct macroevolutionary analyses (Fig. 4a). Parasitism can refer to various individual-level or population-level metrics[83]; however, macroevolutionary analyses are concerned with trends among lineages within clades. Selection is imposed on individual organisms, yet it is the population that responds to selection, with such responses of multiple populations manifesting at the scale of the species. It is, therefore, essential to relate either host traits associated with parasitism (such as immune defences or preening to remove ectoparasites) or infection data to parasitism of host lineages. For the latter, prevalence will serve as a useful measure of parasitism for macroevolutionary models. Populations evolve in response to selection, and prevalence provides a measure of how frequent parasite pressure is within that population. Therefore, using mean prevalence across all populations sampled provides an estimate of the strength of parasite-mediated selection imposed on a given host species, in which mean prevalence values are weighted by sampling error. Although prevalence measures from field studies are often the best estimates available for understanding parasite pressure, early epidemiological theory suggests that it can be a misleading measure of the effect of parasites on their hosts[84]. Alternatively, in systems in which parasitism is highly prevalent across most populations of most host species, abundance can be used. One difficulty with using such measures of parasitism is that, much like the ghost of competition past concept[85], contemporary measurements of parasitism abundance might not reflect past evolutionary history in response to selection imposed by a parasite. This ghost of parasitism past[86] can make detecting a phylogenetic signal of parasitism from such data challenging. The measure of parasitism or the trait chosen to represent parasitism must then be mapped onto the host phylogeny (Fig. 4b).

Phylogenetic methods

Macroevolutionary analyses using either parametric or non-parametric approaches provide a means for estimating diversification for the character mapped onto the phylogeny (Fig. 4c). Popular parametric approaches, such as state-dependent speciation and extinction (SSE) models, estimate evolutionary rates with respect to the presence or absence of a given trait (Box 1). An important caveat to interpretation of SSE models is that a positive correlation between a trait and diversification can be found when there are few instances of said trait[87]. Likewise, although a given trait could emerge multiple times across a tree, it could be that only a single host clade has increased rates of diversification[88]. However, these caveats relate to the broader issue of whether the focal traits are the actual drivers of diversification. Hidden state speciation and extinction (HiSSE) models provide a more refined understanding of whether and how a trait of interest relates to diversification rates[89] and thus should be preferred over SSE models.

In contrast to parametric approaches, non-parametric approaches do not use an explicit model for character evolution and diversification. Instead, they compare observed data (such mean evolutionary

rates for each parasitism state or trait) with a distribution of simulated data, with evidence for an effect of a trait being based off of rejection of the null hypothesis of no difference between the states[90]. Non-parametric approaches testing for differences in evolutionary rates are, therefore, robust to model misspecifications and inadequacies that often come with parametric approaches that only focus on a focal character of interest[91]. However, HiSSE models are an innovative parametric framework that allows for inferences of diversification shifts in response to a focal agent (for example, parasitism state or trait) amidst a background where many other factors are unknown, unmeasured or simply covary[89,92]. Thus, despite parasitism being embedded in a complex ecological framing, the HiSSE approach can not only test for a signature of parasitism contributing to diversification patterns among host lineages but also quantify the strength of that signature.

Results need to be carefully considered to interpret what the patterns might mean. For example, although the expectation outlined earlier is that parasitism would be associated with increased rates of macroevolutionary parameters, it could very well be that studies would find no evidence of such a relationship. Such negative results could be due to the noisy nature of ecological interactions. That is, despite the abilities of HiSSE models to tease apart the influence of a focal trait of interest against a background where many other traits vary, the complex ecological networks that hosts are embedded in could be too intricate to disentangle, even if a relationship between parasitism and host diversification exists. Another important consideration is the size of the tree being analysed. Trees used to conduct similar analyses of diversification parameters require hundreds of tips[89,92,93], and estimating diversification parameters on smaller trees with low levels of representation runs the risk of ascertainment bias if the trees used are not representative of the overall clade[92]. As in any observational study, these models provide estimates for correlations between the focal trait and macroevolutionary parameters, meaning that causality cannot be inferred. Finally, another important caveat of our proposed framework is extinct lineages. Although the fossil record is not always available for inclusion in phylogenetic analyses, including extinct lineages could help to limit the ambiguities and issues associated with basing phylogenetic analyses on extant lineages alone[94–96]. Parasite data do exist in the fossil record[97], thus we recommend including extinct lineages if and when possible (but see ref. 98).

Outlook

We see our framework as a call to explore the possible link between parasitism and host diversification and suggest three important steps be taken. First, synthesize parasite prevalence (and/or other measures of parasite selective pressure) data across the host tree of life, assimilating data collected over decades as interest in disease ecology has rapidly grown. Second, given the ubiquity of parasitism, we suggest applying this framework to multiple clades to provide a more robust test of the link between parasitism and diversification. Good candidates are clades including both host groups that experienced bursts of diversification and those that did not diversify (such as cichlid fish). Third, to move beyond a phenomenological understanding, we suggest investigating mechanisms underlying these patterns, exploring how and why differential extents or profiles of parasitism contribute to patterns of species diversification. Population-level studies might be insightful[40]; however, establishing links between population-level microevolutionary processes and macroevolutionary patterns is difficult[99,100]. The question of whether microevolutionary patterns mimic and/or generate macroevolutionary patterns is longstanding, but an analysis of the paradox of predictability provides promise for linking the two timescales[101].

Differences among host–parasite systems could affect relationships between host diversification and parasitism. For example, some

Box 1 | Phylogenetic metrics and their interpretations

Phylogenetic analyses are useful for understanding how and why some clades are incredibly diverse compared with those that have only a few species (see refs. 121,122 for more detailed information on such analyses). For example, the Placozoa phylum contains only a few species[123], whereas the Arthropoda phylum contains well over one million species[124]. To understand these differences in clade diversity and richness, it is useful to compare traits of interest (in this case, parasitism) with key macroevolutionary parameters underlying diversification patterns: speciation, extinction, net diversification and net turnover. Speciation is the evolution of reproductive isolation yielding a new species, whereas extinction is the loss of a species. Net diversification is the speciation rate minus the extinction rate, giving an estimate for the net gain (or loss, in cases when the extinction rate is greater than the speciation rate) of lineages within a clade over time. By contrast, net turnover is the sum of the speciation rate and the extinction rate, providing information on how much a given clade has changed as lineages appear and disappear over evolutionary time.

Differences in one or more macroevolutionary parameter(s) among lineages with differing traits can be informative of how and why some clades are more diverse than others. For example, plant lineages that have developed buds on their underground structures (geophytes) have a net diversification rate ~30% greater than non-geophytes[125], implying that geophytism provides a selective advantage at the macroevolutionary level. Specifically, geophytes have a belowground, resource-rich structure that non-geophytes do not have, which allows them to escape aboveground disturbances such as fires[126]. This escape from harsh, aboveground conditions might make geophytes more competitive and thus result in greater diversification rates[125].

Regarding parasitism, differences in macroevolutionary parameters among lineages experiencing elevated parasite pressure can provide crucial insight into how and why parasitism affects host evolution. For example, greater extinction rates among highly parasitized lineages could point to a pattern whereby the severe fitness costs of parasites at the microevolutionary scale manifest at the macroevolutionary scale, removing those lineages that cannot adapt and overcome these reductions in fitness. Conversely, greater net diversification among highly parasitized lineages would suggest that parasites act as an engine of diversity, as they promote adaptations within their hosts that, over time, serve to differentiate them and increase diversity at a faster rate than those lineages that are less infected.

Glossary

Abundance
The mean number of parasite individuals per host individual, considering both infected and uninfected hosts.

Coevolutionary arms-race
Evolutionary scenario in which a competing set of co-evolving genotypes or phenotypes develop increasingly escalating adaptations and counter-adaptations.

Diversification
The process by which the diversity of a group of organisms increases over time.

Ecological speciation
The process by which new species form owing to the influence of divergent selection experienced in differing ecological environments.

Extinction
The termination of a taxon when the last member dies.

Extirpation
Complete elimination of a species from a specific area.

Gene-for-gene models
Infection model whereby a parasite has a 'universal virulence' allowing it to infect all host genotypes.

Hybrid necrosis
A type of postzygotic incompatibility seen across many species in which hybrid offspring are rarely able to reach their reproductive stage.

Infection profile
Both the number of individual parasites and parasite species infecting a given host.

Intensity
The mean number of parasite individuals per infected host individual.

Local adaptation
Process by which an individual (or group of individuals) performs better in their local habitat than they would in another locality within their species' geographical range.

Matching allele infection models
Infection model whereby a parasite's genotype must exactly match a host's genotype to successfully infect the host.

Parasites
Organisms that have evolved to acquire resources from a host organism at the expense of the host.

Prevalence
The proportion of individuals within a given host population that are infected with a parasite.

Red Queen dynamics
Evolutionary scenario in which species must adapt to and overcome the fitness costs imposed on them by antagonistic species that are also evolving.

Reproductive isolation
Processes preventing members of different populations from successfully mating or producing viable offspring.

Stockholm Paradigm
An evolutionary framework explaining macroevolutionary mechanisms that incorporate evolutionary and ecological processes.

Tolerance
The strategy whereby hosts limit the harm suffered from increasing parasite loads.

Turnover
The process by which a clade changes as lineages appear and disappear over time.

Virulence
The harm to hosts caused by parasite infection.

hosts adopt resistance strategies, whereas others use tolerance. Hosts that tolerate parasites might not be expected to experience increases in diversification, as they are not engaged in the same kind of coevolutionary arms-race or Red Queen dynamics with their parasites as hosts that resist parasites are. However, tolerance and resistance strategies themselves are traits that may be associated with differences in macroevolutionary rates. Beyond resistance and tolerance, previous work suggests that the defensive evolutionary strategies of animal and plant hosts differ, with the former focusing on matching allele infection models and the latter on gene-for-gene models[102]. These evolutionary histories might also affect the speed at which host diversification responds to parasite pressure. Comparisons of host diversification that occurred within different time frames (such as cichlids radiating at varying temporal scales in different lakes) might shed light on this aspect. Furthermore, parasite taxa differ in multiple properties that shape their coevolution with hosts[103–105], such as virulence, host specificity, transmission mode and life cycle complexity. Whether and how these properties determine whether parasites can act as drivers of host diversification remain to be seen.

There remains the possibility that parasites do not drive host diversification. Parasitism is very different as a species interaction compared with other antagonistic interactions such as predation. With parasitism there is a modest average fitness cost experienced by a large proportion of the population, whereas with predation there is a high fitness cost experienced by few members of the population. Furthermore, hosts can experience diffuse selection from a plethora of co-infecting parasite taxa[66,106–109], which could fail to generate consistent selection over macroevolutionary timescales. Finally, the Stockholm Paradigm suggests that ecological disruptions generate novel combinations of species interactions[110], which then drive host–parasite evolution. Conversely, host phylogenetic history appears to be driving patterns of host–parasite associations in mammal hosts and helminth parasites[111].

Parasites are a major selective force for host species[8,40,112], engaging hosts in a constant battle to adapt to and overcome the negative fitness impact of antagonists[40,113,114]. Despite widespread attention to the ecological effects of parasites on hosts, no previous study has examined their effects on host lineage diversification. Associations between host diversity and parasite diversity have been identified, with more diverse host clades harbouring more diverse parasite assemblages[115–117]. Given the many parasites that infect hosts, the cyclical nature of parasite prevalence in long-lived hosts[118,119], the important effects of virulence[103] and behavioural responses to infection[104,120] and the logistical difficulties associated with exhaustively sampling host taxa of interest, it will be challenging to extract a signal of parasite pressure in host lineage diversification. However, the framework developed here offers an approach to empirically link parasitism to host macroevolutionary dynamics. Understanding the potential of parasites to drive host speciation and extinction will deepen understanding of how species interactions drive biodiversity.

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