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Life history determines genetic structure and evolutionary potential of host–parasite interactions

Luke G. Barrett[1,2,*], **Peter H. Thrall**[1], **Jeremy J. Burdon**[1], and **Celeste C. Linde**[2]

[1] CSIRO Plant Industry, GPO Box 1600, Canberra, ACT 2601, Australia

[2] School of Botany and Zoology, Australian National University, Canberra, ACT 0200, Australia

Abstract

Measures of population genetic structure and diversity of disease-causing organisms are commonly used to draw inferences regarding their evolutionary history and potential to generate new variation in traits that determine interactions with their hosts. Parasite species exhibit a range of population structures and life-history strategies, including different transmission modes, life-cycle complexity, off-host survival mechanisms and dispersal ability. These are important determinants of the frequency and predictability of interactions with host species. Yet the complex causal relationships between spatial structure, life history and the evolutionary dynamics of parasite populations are not well understood. We demonstrate that a clear picture of the evolutionary potential of parasitic organisms and their demographic and evolutionary histories can only come from understanding the role of life history and spatial structure in influencing population dynamics and epidemiological patterns.

Introduction

Population genetic studies can provide insight into parasite evolutionary histories [1–3] as well as identify causal factors contributing to disease dynamics and distribution [4,5]. Hence, accurately interpreting measures of genetic variation and its distribution within host–parasite systems is central to many applied and basic issues relating to human, plant and animal populations. These include the emergence and spread of new diseases [1,3,6,7], effects of infection on host mortality and reproduction [8], assessing risks posed by invasive parasites [9,10] and predicting the evolutionary response of parasite populations to new host resistance genes or vaccines [11,12].

Parasites are a heterogeneous group of organisms that show a remarkable diversity of transmission modes, life-history strategies and spatial structures. Across the spectrum of plant and animal parasites, examples include sexually transmitted species where infection causes host sterility (e.g. anther smuts, gonorrhoea), parasites with complex life cycles requiring multiple hosts (e.g. rust fungi, digenean trematodes), soil parasites that quickly kill their hosts (damping-off diseases, anthrax) and aerially dispersed species that individually have only limited effects on their hosts (e.g. foliar plant pathogens, common cold). Many species infect hosts opportunistically or can attack multiple hosts, whereas others are more specialised, relying on living tissue for survival. Host species are similarly heterogeneous and differ in key traits such as spatial structure (population size and distribution), longevity and resistance diversity. However, despite the importance of such traits for determining disease incidence, prevalence and severity [13–16], the causal relationships between spatial

Corresponding author: Barrett, L.G. (lukeb@uchicago.edu).
*Current address: Department of Ecology and Evolution, The University of Chicago, 1101 E 57th Street, Chicago, IL 60637, USA.

structure, life history and the evolutionary dynamics of parasite populations have received little attention.

Here we highlight the idea that variability in key host and parasite life-history traits distinctively influences disease epidemiology, genetic variation and underlying evolutionary dynamics within populations of parasitic organisms (Box 1). Importantly, many of the underlying assumptions of classical population genetics theory (e.g. infinite population size, marker neutrality, random mixing and free genetic recombination) on which analyses and interpretations of genetic variation in parasite populations are based are frequently violated as a result of strong demographic fluctuations and variable selection patterns in both hosts and parasites [17,18]. Drawing on these considerations, we derive a series of inferences regarding the likely impact of interacting host–parasite life-history traits on parasite population dynamics and genetics.

Box 1. Genetic expectations for host–pathogen interactions

Conclusions regarding parasite evolutionary history and potential that are based on univariate approaches (e.g. characterisation of patterns at neutral marker loci) are likely to be incomplete. Empirical examples presented in this essay support the argument that host and pathogen life-history features partly determine key variables such as connectivity and stochasticity (particularly as it relates to the predictability of encounter between individual host and parasite genotypes, and the overall degree to which local dynamics are endemic versus epidemic) that affect disease patterns, genetic variation and genetic structure (Figure I; Table 1 For example, demographic stability is predicted to decline with reductions in host and pathogen population sizes, decreasing host range, shorter host lifespans, reduced ability of pathogens to survive in the absence of living hosts, and smaller spatial scales of dispersal. Among-population connectivity is a function of both host and pathogen dispersal scale. Based on the assumption that neutral loci are generally more sensitive to changes in either stochasticity or connectivity than loci under selection, it is possible to make general predictions about how changes in connectivity and stochasticity might influence genetic patterns for neutral versus selected loci (Figure I). As implied by Figure I, absolute levels of variation, as well as relative variability in neutral and selected loci, are subject to the influence of suites of host and parasite traits which also determine patterns of disease persistence and among-population movement. An important point is that low genetic diversity *per se* does not necessarily imply that a parasite is of recent origin or newly introduced.

These predictions assume that genes under selection for variation (e.g. avirulence loci) will be buffered to some extent, particularly in natural coevolutionary interactions where pathogens are responding to spatial heterogeneity in host resistance structure. Thus, levels of diversity in host resistance structure will partly determine the degree to which genes under selection respond to changes in either stochasticity or spatial structure. Clearly, the precise shapes of the relationships shown in Figure I could take many forms, particularly given that stochasticity and connectivity are not completely independent of each other. Moreover, one could also imagine situations where these relationships would not hold. Thus, if local selection were particularly strong, genetic structure at selected loci could potentially be stronger than at neutral loci. Characterising host resistance structure is therefore crucial to interpreting patterns of variation in parasite populations. Despite these caveats, this analysis provides a starting point for exploring interrelationships between life history, spatial structure and the epidemiological and genetic dynamics of host–parasite systems. Furthermore, it strongly highlights the value of comparative studies across multiple populations or species as well as modelling studies that integrate demographic and genetic dynamics, particularly where key life-

history features can be varied. Host–parasite systems where genes relevant to the interaction can be characterised in addition to neutral molecular markers are likely to be especially informative.

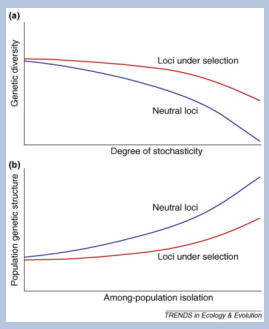

Figure I.
General genetic expectations for neutral versus selected loci in pathogen populations. **(a)** Overall genetic diversity in relation to host and pathogen population stability. **(b)** Population genetic structure (e.g. as measured by standard statistics such as F_{ST}) in relation to among-population connectivity. Expectations for changes in neutral genetic variation are based on metapopulation models exploring the impact of variation in rates of population turnover [62].

Impacts of parasite life history on population demography and genetic structure

Parasite species exhibit a range of life-history strategies that affect disease dynamics and epidemiology and, through this, the genetic composition and spatial structure of their populations. The examples given below, and summarised in Table 1, illustrate the potential for particular traits to strongly influence the genetic structure and evolutionary trajectory of disease-causing organisms.

Host specificity

Parasites vary in the extent of host specialisation as well as overall dependence on a parasitic mode of life [19]. One expectation is that the demographic and evolutionary dynamics of parasites with broad host ranges will generally differ from those that can only infect single host species. For example, parasites with narrow host ranges are more likely to be locally adapted to their hosts than generalist species [20]. In addition, obligate parasites specialised to single host species are more likely to experience frequent local extinction and recolonisation events than generalists, particularly in small and fragmented wild host populations [21,22]. Such among-population processes might promote loss of genetic diversity within parasite populations and generate among-population genetic differences (e.g. through genetic drift), as has been observed for the biotrophic rust pathogen *Melampsora lini* infecting wild Australian flax *Linum marginale*, which is typified by ‘boom-and-bust’ epidemic dynamics [23,24]. By contrast, pathogens able to infect multiple hosts or survive in the absence of a host are unlikely to regularly experience such extreme levels of population stochasticity, and thus should maintain comparatively higher levels of within-population genetic variation. For example, the aerially dispersed wheat pathogen *Mycosphaerella graminicola* (wheat leaf blotch) survives saprophytically between epidemics on plant debris. Populations are genetically stable over time [25], and worldwide studies indicate remarkably low genetic differentiation among populations, with more than 90% of variation distributed within populations [26].

Inference—The degree to which pathogen species maintain stable population sizes via free-living stages, or the ability to infect multiple host species, strongly influences the effective size, variability and genetic structure of their populations.

Mode of reproduction

Parasite reproductive mode is a complex trait that reflects the interplay between environmental, genetic and demographic factors [27]. Although sexual reproduction is common, many parasite species are either clonal or primarily inbreeding; others exhibit both clonal and sexual reproductive modes that vary in time and space. In some bacteria and fungi, horizontal exchange of genetic information (both within and among species) also occurs [6]. Thus, patterns of reproduction in many species greatly depart from expectations under classical population genetic models [28]. Of perhaps most obvious effect, variation in levels of sexual recombination should strongly influence patterns of genetic variation, with populations that undergo sexual recombination generally exhibiting higher levels of genotypic diversity than populations that are exclusively asexual [18,29,30]. Studies investigating the genetic structure of sexual and asexual populations of the wheat stem rust *Puccinia graminis* f.sp. *tritici* illustrate some of these points. In areas where the alternate host common barberry *Berberis vulgaris* is present, sexual reproduction is common and pathogen populations harbour comparatively high levels of genotypic and phenotypic diversity for infectivity. By contrast, where barberry is absent, the rust reproduces only asexually, and populations contain comparatively low pathogenic diversity [31].

Patterns of disease incidence and prevalence can also strongly influence mating patterns and thus the genetic structure of pathogen populations. For example, the malarial parasite *Plasmodium falciparum* has an obligate sexual phase that occurs in the mosquito vector before transmission. In regions where disease incidence is high, populations maintain high levels of genotypic diversity and low genetic differentiation among locations. In these areas, high disease incidence means the vector is frequently infected by multiple *Plasmodium* genotypes, which then mate and recombine before transmission to a new host. By contrast, parasites in regions with comparatively low disease incidence have a lower probability of encountering novel genotypes in the vectors, leading to frequent self-fertilisation, low genetic diversity and high variation between geographic locations [32].

Inference—Parasite reproductive mode is a variable and complex trait that strongly influences the genetic structure and evolutionary potential of populations. Clonal reproduction and inbreeding are generally expected to decrease genotypic variation within populations.

Transmission and dispersal

Understanding how parasites move within and between host populations is important for correctly interpreting patterns of genetic diversity (Box 2). Disease dispersal gradients vary with mode of dispersal, environmental conditions and vector behaviour. For example, soil-borne and splash-dispersed species generally show steep declines in dispersal with distance, whereas wind-borne diseases show much flatter curves [33]. Host-dispersed and vector-transmitted species might be expected to lie somewhere between these extremes, although in these cases, host and vector behaviour (e.g. in relation to resource distribution) will largely determine the precise shape of parasite dispersal gradients.

The spatial scale of parasite dispersal is an important determinant of extinction–recolonisation dynamics, particularly in wild host metapopulations, and thus influences regional patterns of disease occurrence and persistence. In turn, spatial variation in disease incidence and severity affects the intensity of selection on parasite infectivity and

aggressiveness, as well as host resistance [4,34]. Furthermore, parasites that experience high levels of dispersal (and thus high gene flow) should have higher within-population genetic diversity than those with more limited dispersal. In addition, high gene flow tends to counteract the effects of genetic drift and homogenise adjacent populations, thereby increasing the spatial area encompassed by a deme. However, the outcome of host–parasite coevolution means that frequent dispersal among populations might not always result in a homogeneous population structure. Theoretical studies indicate that parasites are more likely to be locally adapted to their hosts when their among-population migration rates exceed those of their hosts. The reverse is expected when parasites migrate less than their hosts [35].

Box 2. Pathogen dispersal patterns

Between-host transmission is primarily horizontal for the majority of parasites, with spread occurring between related and unrelated host individuals via a variety of mechanisms (e.g. direct contact, vector transmission or aerial dispersal) (Figure I). In some instances, transmission can be vertical, with the pathogen passing directly from parent to offspring without an intervening free dispersal stage. Differences in transmission can significantly affect the persistence of disease in populations as well as parasite genetic diversity and population structure. Thus, vertical transmission of grass endophytes typically results in populations that maintain high and consistent levels of infection through time [63]. Stable population sizes associated with vertically transmitted species can therefore be expected to promote the retention of variation within populations.

For vector-dispersed species, the extent of gene flow is primarily determined by vector behaviour. Thus, strong local spatial genetic structure in the anther smut *Microbotryum violaceum* is associated with locally restricted insect dispersal [64]. Host preferences shown by vectors might also play a role in sympatric pathogen race formation by promoting reproductive isolation among host-specific strains [65]. Similarly, for parasites of animal species, patterns of host dispersal have the potential to strongly influence parasite gene flow [66]. For example, the dispersal ability of intermediate hosts has been shown to have a strong effect on the genetic structure of different species of trematode parasites of salmonid fishes [67].

By contrast, patterns of dispersal in wind-dispersed species are likely to be highly stochastic and dependent on prevailing environmental conditions. Long-distance aerial dispersal has been shown to result in significant gene flow among populations at both continental and global scales [68]. However, for specialist pathogens with a high degree of host specificity, effective dispersal at any scale can only occur if there is a susceptible host in the new location. For that reason, long-distance dispersal events are likely to be more evident for pathogens that infect widely dispersed species (e.g. agricultural crops, livestock). Occasionally, such events can be detected or reliably inferred. For example, in 1969, two previously undetected genotypes of wheat stem rust (*Puccinia graminis* f.sp. *tritici*) dispersed to Australia from southern Africa in the jet steam [69]. Although such events might be relatively rare, the result will be founder populations that have a lower genetic diversity than the source population because of genetic drift.

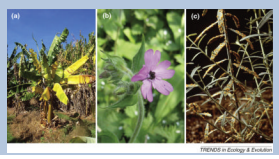

Figure I.
Plant hosts infected by pathogens with variable modes of transmission. **(a)** Panama disease of plantain caused by a soil-borne pathogen, *Fusarium oxysporum cubense* (photo credit: G. Wallduck, Department of Primary Industries, Northern Territory Government); **(b)** vector-transmitted anther smut in flowers of red campion *Silene dioica*, caused by *Microbotryum violaceum* (photo credit: CSIRO Plant Industry); and **(c)** an aerially dispersed rust infection of wild flax *Linum marginale*, caused by *Melampsora lini* (photo credit: CSIRO Plant Industry).

Inference—Pathogen dispersal is a critical factor determining disease dynamics and persistence. Pathogens infecting sessile hosts, or lacking specialisations promoting long-distance dispersal, should show stronger patterns of spatial genetic structure and isolation by distance.

Life-cycle complexity and epidemiology

Parasitic organisms display an array of life histories and infection strategies (Box 3), ranging from species that complete their life cycle on a single host, through species with alternate parasitic and free-living phases, to species that have multiple stages, each on a different host species. For parasites with obligate multi-host life cycles, the selective forces experienced by different genotypes are likely to vary for different stages, given the requirement for establishing on potentially unrelated host species. Such tradeoffs between fitness components might be expected to promote the maintenance of genetic polymorphisms owing to disruptive or fluctuating selection favouring different alleles in different obligatory hosts [36]. The availability of alternate hosts has further been proposed to promote out-crossing and transmission [37] and might also increase population stability, thus reducing the severity of bottlenecks and decreasing genetic drift.

Similarly, for host–pathogen interactions typified by epidemic dynamics, pathogen traits under selection will vary at different stages in the epidemiological cycle. During the transmission and establishment phase, selection for the ability to encounter and infect a broad range of hosts will be high; later, as population growth enters an exponential phase, fecundity can become increasingly important; while later still, as populations collapse, there is likely to be a strong shift toward traits favouring off-season survival [16]. For example, end-of-season population reductions can be massive for specialist pathogens of plants that have no reliable resting spore stages or alternate hosts (e.g. some biotrophic rusts [38]). By contrast, survival might be less problematic for generalist species, although evolutionary constraints imposed by differential patterns of selection between parasitic and free-living phases of the disease cycle might act to reduce diversity [39]. Particularly for systemic parasites, tradeoffs can occur such that greater dispersal ability might allow genotypes to persist even when they are inferior within-host competitors [40]. A key point is that inferences regarding pathogen evolutionary history must account for within-and among-population variation in within-season disease dynamics, severity and incidence.

Box 3. Parasite infection strategies

Parasite species vary in infection strategies that, in combination with different suites of host life histories, broadly determine opportunities for disease invasion, spread and persistence, and thus the rate and direction of coevolution. The ways in which parasites impact host fitness can be broadly classified into those that cause rapid host death (‘killers’), those that directly attack host reproductive organs (‘castrators’) and those that reduce overall host fitness (‘debilitators’) [70]. Results from a recent metapopulation model exploring the dynamics of pathogens that decrease host fertility versus ones that

increase mortality support the contention that these groups might differ broadly in persistence, the degree to which they reduce host population size, and the strength of selection on host resistance [15]. In particular, the evolution of resistance was more rapid for pathogens affecting host mortality than those reducing fecundity. Interestingly, whereas increasing pathogen effects on fecundity resulted in a monotonic decrease in the number of patches occupied by the host, the impact of increasing mortality was nonlinear, with the greatest reductions in host population size at intermediate virulence levels; when virulence was high, the parasite temporarily reduced host occupancy of patches to the point that it caused its own extinction. The magnitude of these effects is at least partly determined by host life history (e.g. longevity) [71]. Parasites also differ with regard to which parts of their hosts they colonise. Resultant variability in the durability of infection is likely to strongly influence the genetic diversity and demographic dynamics of parasite populations. For example, foliar plant pathogens frequently undergo annual epidemic cycles with population sizes fluctuating greatly, whereas systemic pathogens generate long-term infections of perennial hosts and maintain relatively stable population sizes [16].

Inference—Increasing life-cycle complexity is likely to generate higher levels of genetic variation in response to increased demographic stability and the potential disruptive selection pressures being experienced by different infective stages.

Inference—Variation in selection intensity during the epidemiological cycle provides opportunities for diversifying selection, competition and genetic drift to influence the diversity and structure of pathogen populations.

Impact of host life history and spatial structure on parasite dynamics and evolution

The obligate dependence of many parasitic organisms on their hosts for long-term survival makes the size, structure and distribution of host populations an important determinant of the genetic structure of parasite populations. Hosts represent an inherently patchy and dynamic resource that varies spatially and temporally in the time available for infection, levels and types of resistance (e.g. quantitative versus genes of major effect) and population size and persistence. Furthermore, host species differ in life-history traits such as longevity, mating system and phenology, which also influence parasite population dynamics. Despite this, there has been little consideration of how such heterogeneities affect the genetic structure and evolutionary trajectories of parasite populations.

Spatial structure

The extent to which the spatial structure of parasite populations mirrors patterns seen in host populations depends on the degree to which pathogens are obligately dependent on that host. For animal parasites, estimates of spatial structure must also take into account host dispersal [41] (Box 2) as well as heterogeneities in host social behaviour and group structures. Generally, increasing among-population isolation should reduce genetic variability within parasite populations and promote higher levels of genetic differentiation among demes [42]. Population subdivision also increases parasite population vulnerability to environmental stochasticity and the possibility of local extinction of particular genetic variants, or even whole demes.

Population size and the degree of population subdivision are both likely to differ significantly between parasites infecting human or domesticated species, and those found on

wild hosts. For example, agricultural crops are typically planted in large genetically uniform stands, which massively amplify available host resources for suitable pathogen races, thereby increasing effective pathogen population size. By contrast, wild plant populations are often demographically small and at least partially isolated, thus decreasing the chance of migration between pathogen populations, and increasing the propensity for drift, divergent selection and genetic differentiation among populations. The possibility of near, or total, extinction of pathogen populations also depends on host numbers. For example, an 11 year survey of >130 populations of the rust pathogen *Triphragmium ulmariae* infecting its wild host meadowsweet *Filipendula ulmaria* found that extinction and recolonisation events were common, and that disease incidence was positively correlated with host population size [43].

Ecological differences between the plant host species red campion *Silene dioica* and white campion *S. latifolia* provide an interesting comparison of the effects of host life history on populations of the anther smut *Microbotryum violaceum* in Europe [44]. *S. latifolia* is found in ruderal habitats associated with frequent population extinction and recolonisation. By contrast, *S. dioica* is found in more undisturbed natural habitats, and has more stable population dynamics. As might be predicted based on these differences, *M. violaceum* collected from *S. latifolia* showed less microsatellite variation and higher differentiation among populations than that from *S. dioica*. Another key factor is host population connectivity; more continuous host populations favour the evolution of resistance as disease spreads globally [45], which could in turn influence the evolution of pathogen virulence.

Inference—Host population distribution affects parasite population dynamics and among-population movement. The metapopulation structure typical of many wild hosts results in complex heterogeneous mosaics of local populations, dictating that pathogen population sizes are limited, near-extinction events are common and drift and gene flow are influential.

Host longevity and phenology

Theoretical models indicate that long-term persistence of horizontally transmitted parasites exploiting single host species within populations increases with lower intrinsic host mortality [46]. Thus, if hosts are short lived, pathogen populations are more likely to experience regular extinction and colonisation dynamics. By contrast, if hosts are long lived and provide a perennial resource, then pathogen populations are likely to be more stable. Empirical support for this prediction is found in the interaction between *M. violaceum* and plant host species in the Caryophyllaceae, which vary in longevity, mating system and vector identity [47]. Variation in the timing of different host life-history stages (or ephemerality of the tissues being attacked) can also alter host availability and hence disease epidemiology within populations. For example, because of seasonal differences in leaf availability, foliar pathogens of perennial deciduous trees experience demographic dynamics more akin to pathogens of short-lived host species than systemic pathogens of long-lived species [48].

The timing of host availability is also a strong selective force at multiple levels of spatial organisation. Thus, temporal variation in the timing of leaf production and abscission among individual trees promotes reproductive isolation and adaptation of specialist insect herbivores [49]. Experimental studies of entomopathogenic nematodes indicate that variation in host availability and transmission opportunities among host populations might play a role in the evolution of infection strategies, as well as maintaining overall diversity [50], whereas differences in flowering phenology of the congeneric hosts *S. latifolia* and *S. dioica* promote strong host-related genetic differentiation in associated populations of the anther smut *M. violaceum* [51].

Inference—Host longevity and availability affect the predictability and timing of substrate availability for pathogens and, through this, the size of populations, adaptation to local conditions and the potential for gene flow among populations.

Host resistance

Hosts vary widely in resistance mechanisms (Box 4) as well as the diversity of resistance genotypes within and among populations. The potential for variation in host resistance to influence the generation and maintenance of genetic polymorphisms for pathogen virulence is well established both theoretically (e.g. [46,52]) and empirically. For example, high levels of virulence diversity within and between populations [53], local adaptation of pathogen and host populations [54], correlations between levels of host resistance and pathogen virulence in local populations, and tradeoffs between spore production and virulence [17] all implicate selection by *L. marginale* as an important source of variation in populations of *M. lini*. By contrast, genetically homogeneous crops grown over large areas impose strong directional selection on local pathogen populations, resulting in selective sweeps favouring particular genotypes and lower overall virulence diversity in pathogen populations [55]. For pathogens that either infect multiple hosts or are vector transmitted, population structure can reflect selection pressure from different host defences. For example, allele and genotype frequencies in populations of the cattle intracellular parasite *Theileria parva* are markedly changed after passage through both the tick vector *Rhipicephalus appendiculatus* and the bovine host [56]. Similarly, within species, male and female hosts can differ in infection patterns, and thus harbour parasite populations with different levels of genetic diversity [57].

Box 4. The influence of host resistance mechanisms on pathogen population structure

Hosts resist attack by parasites and pathogens via a diverse array of mechanisms that have the potential to influence pathogen population genetic structure. However, broad relationships between host resistance mechanisms and the genetic structure of pathogen populations remain largely unconsidered. For example, in vertebrate hosts, differential exposure to either acquired or innate immune mechanisms should have the potential to strongly influence the genetic structure and evolutionary dynamics of pathogen populations (e.g. [72]). Similarly, pathogen evolutionary dynamics can also be strongly influenced by qualitative versus quantitative genetic control over innate resistance in plant and invertebrate hosts [73]. However, to date, the general influence of such variation on the strength and direction of selection on pathogen genes and the genetic consequences for populations is largely unknown.

Currently, the clearest example of how differences in mechanisms of host resistance can influence the genetic structure of pathogen and parasite systems is in gene-for-gene interactions between plant hosts and their microbial pathogens. Under the current paradigm, there are essentially two models to explain how hosts and pathogens interact (via gene-for-gene dynamics) at the molecular level. First, the products of host resistance genes can interact directly with the pathogen molecules they recognise (known as effector or avirulence proteins) to trigger resistance (e.g. flax and flax rust [73]). Second, hosts can detect certain effector proteins indirectly by responding to changes induced in host target proteins. For example, the *RPS2* and *RPM1* resistance proteins in *Arabidopsis* apparently recognise corresponding *Pseudomonas syringae* effector gene products by detecting changes induced in the intermediary host protein *RIN4* by the pathogen products [74,75]. It has been suggested that these differences are likely to lead to qualitatively different outcomes in terms of the diversity of host resistance and pathogen infectivity [76]. Thus, indirect recognition will lead to simple, binary, balanced polymorphisms for host resistance and pathogen infectivity, such as observed for the

RPM1 and *RPS2* loci in *Arabidopsis* [77,78]. By contrast, direct interactions might favour the classical 'arms race' model of host–pathogen coevolution, whereby continual changes in pathogen *Avr* genes are matched by changes in host *R* genes. This could lead to high levels of phenotypic, allelic and nucleotide diversity, such as are observed at interacting host and pathogen loci in the flax/rust system [79].

Inference—Genetic heterogeneity for host resistance can result in complex patterns of disruptive selection being imposed on pathogen populations and, through this, dramatically influences levels of diversity in host–pathogen systems.

Inferring demographic and evolutionary dynamics

Host–parasite systems represent suites of interacting life histories that collectively determine disease epidemiology, and therefore patterns of genetic variation and evolutionary trajectories of parasite populations (Box 1). In particular, parasite genetic diversity might be affected in an extreme fashion when life-history parameters interact additively (Table 1). For example, high levels of demographic and genetic stochasticity resulting from a combination of traits including host specialisation, metapopulation dynamics (with associated extinction–recolonisation dynamics) and asexual reproduction should significantly increase the fixation rate of neutral and nearly neutral mutations and reduce the effective size of parasite populations [18] relative to more demographically stable situations. Moreover, selective sweeps leading to the fixation of favourable alleles might further lead to reductions in neutral genetic diversity across parasite genomes [55,58].

Thus, if relevant biological parameters are not considered, expectations for population genetic patterns that are based on particular demographic or evolutionary episodes (e.g. a recent host shift) are likely to be inaccurate, and might lead to a failure to detect the predicted pattern or to false detection of a nonexistent pattern [59]. For example, some wild species, even ones with nearly cosmopolitan distributions, possess little variation at neutral marker loci [60,61]. Thus, low genetic variation *per se* does not necessarily indicate recent bottlenecks caused by invasion or population declines, and instead might reflect various aspects of the underlying biology of the species concerned. Conclusions regarding pathogen evolutionary potential and history must therefore explicitly consider the influence of both host and parasite life history on patterns of genetic variation within contemporary parasite populations.

Conclusions

Understanding the evolutionary drivers of disease outbreak and emergence is of clear importance, especially given the health, economic and ecological costs associated with infectious diseases. Here we demonstrate that variation in key pathogen life-history features drives epidemiological and genetic dynamics within and between pathogen populations. Similarly, we propose that pathogen populations are likely to respond to a range of variable host life-history traits and population structures (Table 1). This argues for better integration of information on host and pathogen life history into theoretical and empirical studies of disease. Of particular value will be focused comparative studies of the genetic structure of pathogen and parasite species that differ in key features of their life-history, life-cycle and associated traits of the host, using similar sampling designs and genetic markers. In addition, we highlight the importance of combining demographic and genetic studies at the population level. Asexamplesfrom both natural and anthropogenically modified systems accumulate and better genomic tools become available, it should be possible to gain a clearer, more mechanistic understanding of the variability and adaptability of pathogen populations in relation to life history and population structure. Quantifying the consequences of these

interactions should lead to predictions that can advance our understanding of host–parasite interactions as well as contribute to the development of a broad conceptual framework for understanding the role of life history in parasite evolution.

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Glossary

Biotroph	an obligate parasite that can only derive nutrition from living host tissues
Deme	an interbreeding population that is isolated from other such populations
Heteroecious	a parasite occupying two or more different hosts at different stages of the life cycle
Metapopulation	a group of partially isolated subpopulations that are connected by dispersal. Extinction–recolonisation dynamics are facilitated by exchange of individuals or propagules between sites
Parasite	any organism that is intimately associated with and metabolically dependent upon a host species for the completion of its life cycle, and which reduces the fitness of the host to some extent
Pathogen	a parasitic microorganism
Phenology	the timing of biological events in relation to the time of year, such as flowering time of host plants
Saprophyte	an organism able to derive nutrition from dead or decaying plant matter
Systemic parasite	a parasite that spreads throughout the internal tissues of the host (e.g. circulatory system and other internal organs in animals, xylem and phloem of plants)
Transmission	the movement of a parasite from an infected source to an uninfected individual or population. Transmission can occur through several different means, although parasites are often specialised for specific modes of transmission
Virulence	the capacity of a pathogen to invade host tissue and reproduce

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Table 1

Some factors associated with host and pathogen life history and population structure that are likely to influence the genetic structure and effective size of populations.

	Factors that generally increase effective population size	**Factors that generally decrease effective population size**
Host exploitation and specificity	Opportunistic and/or generalist species, multiple host species	Specialised parasite, single host species
Mode of pathogen reproduction	Sexual	Clonal or inbreeding
Pathogen dispersal	Long-distance dispersal	Restricted, local dispersal
Environmental stochasticity	Stable environment and host population dynamics	Frequent population extinction and recolonisation, short-lived hosts
Host longevity; ephemerality of tissues attacked	Perennial or long-lived host	Annual or ephemeral hosts
Host population size and structure	Large, interconnected host populations	Small, fragmented host populations
Epidemiological dynamics	Endemic, systemic	Epidemic, boom and bust