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The context of host competence: a role for plasticity in host–parasite dynamics

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Even apparently similar hosts can respond differently to the same parasites. Some individuals or specific groups of individuals disproportionately affect disease dynamics. Understanding the sources of among-host heterogeneity in the ability to transmit parasites would improve disease management. A major source of host variation might be phenotypic plasticity – the tendency for phenotypes to change across different environments. Plasticity might be as important as, or even more important than, genetic change, especially in light of human modifications of the environment, because it can occur on a more rapid timescale than evolution. We argue that variation in phenotypic plasticity among and within species strongly contributes to epidemiological dynamics when parasites are shared among multiple hosts, which is often the case.

Heterogeneity in host competence

Predicting disease risk to humans, wildlife, and domestic animals has become ever more crucial as the frequency and magnitude of emergence events continue to increase [1,2]. Although host abundance and density strongly influence whether or not a parasite will establish and persist in a population [3,4], a growing body of evidence also suggests that host competence, defined as the proficiency with which a host transmits a parasite to another susceptible host or vector (Box 1), is a key determinant of epidemiological dynamics for parasites with more than one host [5–13]. Thus, a productive step toward improving predictions of such dynamics would include a framework for understanding how heterogeneity in host competence arises and persists.

Competence represents the functional role of an individual or group of individuals (e.g., of the same sex, age, size, life-history stage, population, or species) for infection dynamics in an ecological community [8–10,14,15] (Box 1). In other words, competence is often a relative term because different hosts and combinations of hosts can act to facilitate epidemics or maintain the presence of a parasite in the environment. Although mediated by individual behavioral, physiological, and immunological factors, host competence links what happens inside a host to what happens among hosts comprising communities [16–19]. Crucially, however, competence is governed not only by genetic variation of hosts, parasites, or vectors (Box 1) but also by the environments in which such genetic variants occur [15,20,21]. Consequently, how competence drives variation in community infection dynamics will be mediated by feedbacks among higher and lower levels of biological organization, from individuals to ecosystems [17,18]. We focus here on phenotypic plasticity as a mediator of variation in host competence and environmentally dependent disease risk.

Phenotypic plasticity: a context for host competence?

The environment works on organisms in two ways: it sorts genetic variation via natural selection and it exposes genetic variation via plasticity [22]. Phenotypic plasticity describes the ability of one genotype to express different phenotypes across environmental contexts [22]. Depending on spatial or temporal heterogeneity in the environment, organisms adjust their morphology, physiology, and behavior, often in an adaptive manner [22]. The evolution of phenotypically plastic traits depends in large part on the type and magnitude of spatial and temporal heterogeneity in the environment [22]. When reliable cues accurately signal impending changes in the environment, species may evolve a highly plastic repertoire of traits, including those tied directly and indirectly to their interactions with parasites and vectors [22]. Thus, our insight into disease dynamics might be improved if we consider how different environments affect hosts plastically. For example, resource-rich environments fuel rapid replication of a fungal parasite in its water flea host, *Daphnia dentifera*. This plastic shift in host competence can drive large epidemics in *Daphnia* populations that cause larger host die-offs and more intense parasite-mediated selection for costly resistance to the parasite [15,23–25]. Environmentally mediated epidemics in this system may produce cascading effects on the rest of the ecosystem because the abundance of *Daphnia* also affects the abundance and composition of algal and fish communities [26]. Simply put, plasticity could often underlie unique host (individual, population, and species) contributions to parasite dynamics across contexts but, in general, the role of plasticity has been minimally considered, especially in terms of parasites with more than one host (including zoonoses).

A plasticity-mediated framework for disease dynamics

Figure 1 illustrates how variation in parasite transmission potential or risk of infection (depicted as the transmission coefficient, β) might be driven by plasticity in host competence among individuals, nested within species, nested

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Box 1. Heterogeneity in host competence

Host competence is the ability of a host to transmit infection to another susceptible host or vector effectively. Behaviors of hosts, parasites, and vectors affect the frequency and magnitude of host–parasite encounters [step **(1)** in Figure I], whereas genetic, molecular, and cellular processes generally mediate within-host post-exposure host responses, including susceptibility to infection (e.g., whether hosts become infected upon exposure or not) **(2)**, as well as duration and magnitude of infectiousness and probability and rate of recovery after infection **(3)**. Variation in the way that individual hosts cycle through a progression of exposure, infection, disease, and recovery has obvious individual-level consequences (e.g., life or death), but these individual-level responses are also directly linked to transmission to other susceptible hosts, to vectors, or into the physical environment **(4)**. Host competence at an individual level mediates intraspecific population level (**5**) and interspecific community level (**6**) parasite prevalence and spread. The proficiency with which a parasite is transmitted among susceptible hosts is crucial because it ultimately influences community-level dynamics, including whether a parasite invades, spreads, and persists in a multihost environment.

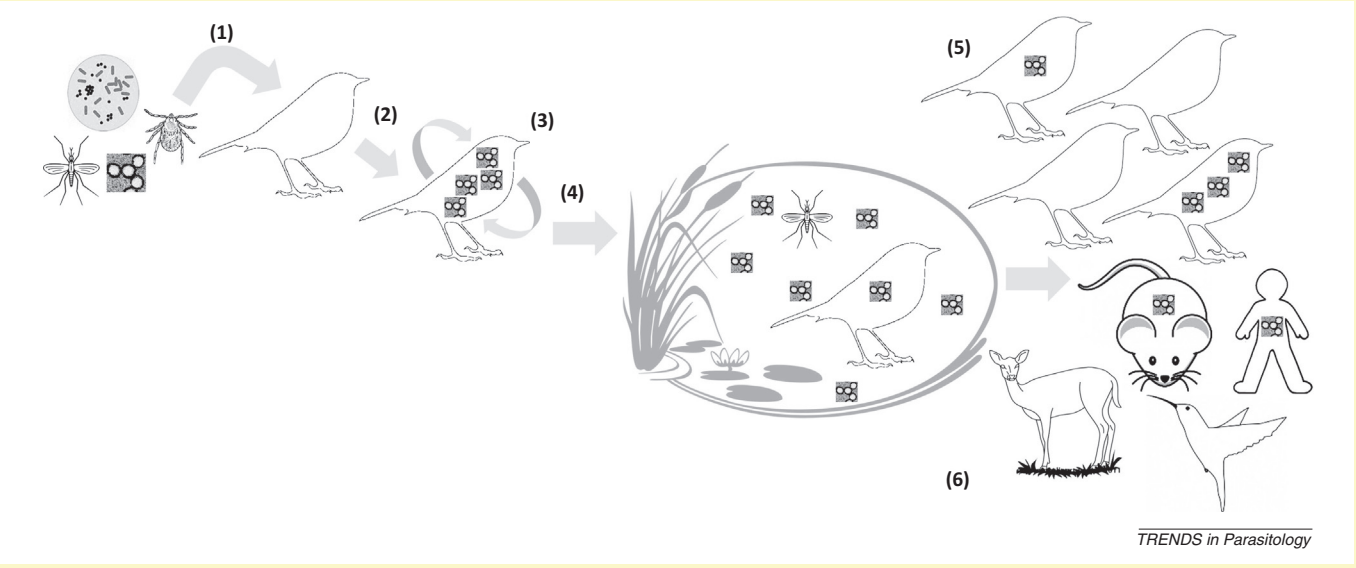

Figure I. Heterogeneity in host competence.

within ecological communities (Figure 1) [27,28]. In this hypothetical example, individuals vary in competence-related traits contributing to transmission depending on their genotype, the environment, and gene–environment interactions (e.g., plasticity including but not limited to epigenetic regulation of gene expression, acquired immunity, and parental effects) (Figure 1A) [27,28]. At the species level (Figure 1B), the range of genotypes present in the population determines the mean and variance of a host species' contribution to parasite transmission potential. In this particular example, despite variation in shape, all reaction norms for all three species have the same average contribution to β across all environments. However, β and variation in β via host competence differs among species, depending on environment. Some species will therefore transmit consistently, whereas others might transmit differently across environments or be extremely variable in particular environments. Host energetics, immunity, behavior, and other traits could mediate differences in these reaction norms [29–31]. At the community level (Figure 1C), species composition and plasticities within and among species differ across environments, thus giving a different community value of β (e.g., the contributions of all species to parasite transmission among a multi-host community) contingent on the site considered. Figure 1C highlights a surprising outcome about β when both community composition and heterogeneity in plasticities are considered: two identical communities can have very different β values contingent on how the species respond to variable environments. For example, in Environment 4, average β is double that of Environment 3 simply as a result of different plasticities among species in different contexts.

To further illustrate the consequences of plasticity in host competence on disease dynamics, we developed a general susceptible-infected (SI) epidemiological compartment model [3]. This classic modeling formalism categorizes hosts by their infection status, and tracks changes in these groups as individuals 'move' through them via births, deaths, and infective contacts, which occur at rates depending on traits and densities [3]. From this model we can calculate the parasite reproductive ratio, R_0, a fundamental index of the potential for parasite spread. Parasites can initiate epidemics when $R_0 > 1$, and larger values of R_0 generally produce larger epidemics with greater effects on host density [3] (Box 2). We show here that a plastic increase in host competence, and thus the contribution to β in one species across an environmental gradient, can facilitate parasite invasion ($R_0 > 1$), increase equilibrial infection prevalence, and increase infected host density. Without this plasticity, the potential for parasite invasion is reduced; with plasticity, the parasite can establish in environments that were previously unsustainable. The qualitative behavior of this model is robust to allowing host species to compete and variation in parasite virulence. This example illustrates only one possibility.