

# Behavioral competence: how host behaviors can interact to influence parasite transmission risk

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Hosts vary in their ability to transmit new parasite infections (i.e. competence). Although behavior is suggested as a source of individual-level variation, the contribution of host behavior to host–parasite dynamics at the population-level remains largely enigmatic. Here we advocate that *behavioral competence* be characterized as a syndrome of behaviors that interact to directly or indirectly influence transmission potential. These behaviors can be plastic in response to environmental conditions and/or infection state, and appear linked to immunological traits through shared physiological regulation. By integrating behavioral variation and covariation into a whole-organism view of host competence, disease ecologists might more realistically characterize an individual's role in host–parasite systems.

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## Introduction

The typical epidemiological approach for predicting parasite outbreaks and dynamics employs theoretical models of host transition through stages of infection (e.g. susceptible, exposed, infected, and recovered) [1]. While such models traditionally assumed minimal variation in host characteristics, researchers have now established that hosts vary widely in their probability of contacting and spreading parasites (i.e. competence; **Box 1**) even within a single species [2,3], and that such heterogeneity can strongly influence dynamics of the host population [1,4–6]. Given the extensive variation in host traits among and even within individuals, our ability to predict the nature and severity of parasite outbreaks will be improved when the consequences of this variation are better understood. Here we

highlight the importance of host behavior in shaping individual competence, and illustrate how behavioral variation and covariation could influence broader dynamics of host–parasite systems. Specifically, we discuss the (A) importance of behavior in determining an individual's propensity to contact and spread parasites, (B) necessity of simultaneously considering multiple dimensions of host behavior, (C) potential for plasticity to shift behavioral competence, and (D) interactions between behavioral and immunological elements of host competence.

## Host behaviors mediate variation in competence

Host competence to transmit parasites is not solely the product of the immune system, but is also simultaneously determined through behaviors that limit contact with parasites, vectors, or other hosts [7,8]. This notion has met support across numerous studies documenting behavioral variation across individuals, populations, or species that differ in parasite burden [9]. However, the predominance of correlational studies makes it difficult to determine whether infection differences are driven by behavioral defenses. More direct evidence comes from three recent studies that experimentally restricted relevant host behaviors and documented a corresponding increase in parasite success [10,11\*,12\*]. The one study pairing behavioral and immunological manipulations found their effects to be additive, and ultimately suggested that behavioral defenses may be more effective than immune defenses at reducing parasite burden [11\*]. These findings strengthen the argument that behaviors play an important role in mediating individual parasite risk (**Figure 1a**). Moreover, these behaviors likely impact population-wide host–parasite dynamics. For example, the behavioral restrictions in one of the above studies altered spatial aggregation of the parasites and increased host heterogeneity in parasite burden [11\*]. Similarly, a recent simulation revealed that avoidance of infected conspecifics decreased the intensity and duration of outbreaks, sped extirpation, and reduced viral prevalence [13\*].

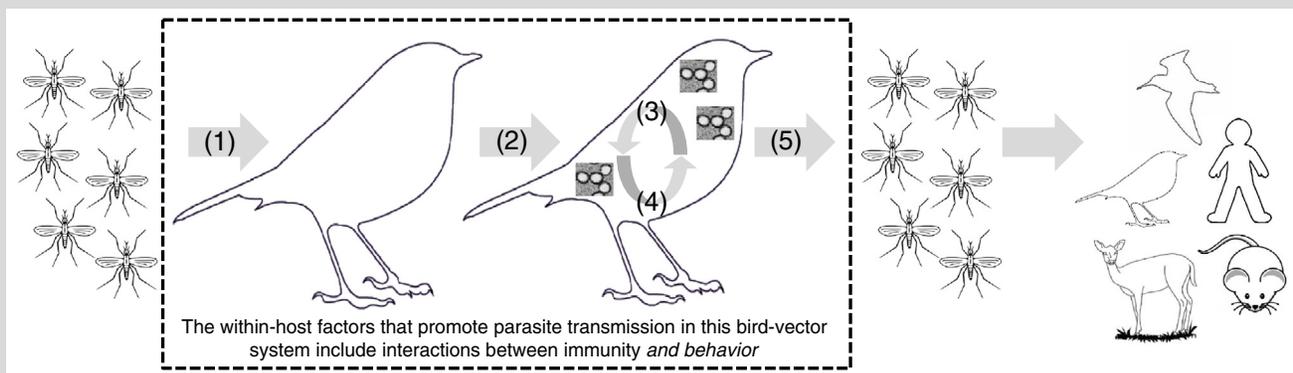
## Behavioral competence as a syndrome

Single host behaviors are unlikely to mediate all variation in competence [14]. Rather, the whole behavioral repertoire of an individual determines its probability of parasite exposure and transmission [15]. Thus, a realistic representation of host competence requires simultaneous consideration of multi-dimensional behavioral networks, or behavioral syndromes (behavioral correlations across contexts; **Box 1**).

**Box 1 Host competence: The ability of a host to transmit parasites effectively to another host or vector.** Here we consider competence as a continuous trait with more competent hosts contributing proportionately more to transmission dynamics. Host competence is a product of an individual's *behavioral competence* (i.e. the extent to which an individual's behaviors directly or indirectly promote parasite exposure and transmission) and *immunological competence* (i.e. the extent to which an individual's immunological response promotes parasite infection and transmission). Throughout the text we refer to behaviors that decrease host competence (and thus limit transmission) as *anti-parasite behaviors* even though such behaviors also function in other contexts (e.g. mating, foraging).

**Behavioral competence:** Individuals and groups of individuals often show distinct *personalities* (i.e. consistent behavioral differences across time and/or contexts) and *behavioral syndromes* (i.e. correlations among behavioral traits). Most disease ecology research has focused on single or few anti-parasite behaviors, but rarely the covariation among them. Likewise, personalities and syndromes (e.g. proactive-reactive, bold-shy) are generally studied in contexts unrelated to infection. However, these and many other correlated behaviors are likely linked with exposure risk and transmission probability.

**High competence** results from the expression and covariation of traits that promote parasite transmission across the following stages of interactions with parasites or vectors: (1) before exposure (2) upon exposure (i.e. susceptibility) (3) during infection (i.e. parasite clearance versus proliferation). Behavior during the infection period is especially important because (4) traits that promote host survival while infected, at least until transmission (e.g. duration of infectiousness) and (5) traits that promote contact between infectious hosts and susceptible hosts or vectors [19] will increase transmission chances.



Element of host competence	Behaviors mediating host competence
(1) Exposure	Activity level, boldness and risk aversion, exploratory behavior, dominance, sociality, learned avoidance, specific anti-vector behaviors (preening and social grooming)
(2) Susceptibility after exposure	Mostly mediated by immunity, but could also relate to tradeoffs with energetically expensive behaviors (e.g. reproduction) across different life stages or times of year.
(3) Supporting parasite maintenance and proliferation	Sickness behaviors such as behavioral fever, lethargy, anorexia, diminished reproduction, and social isolation. Most behaviors arise as energy conservation measures to counter the energetic expense of fighting the parasite immunologically.
(4) Survival (duration of infectiousness)	Behaviors described in (3) that act to conserve energy to counter immunological costs, expanded to include those related to other survival demands (e.g. antipredator defenses).
(5) Transmission	Same as (1) except that post-exposure/infection sickness behaviors can modify contact rates with susceptible hosts or vectors

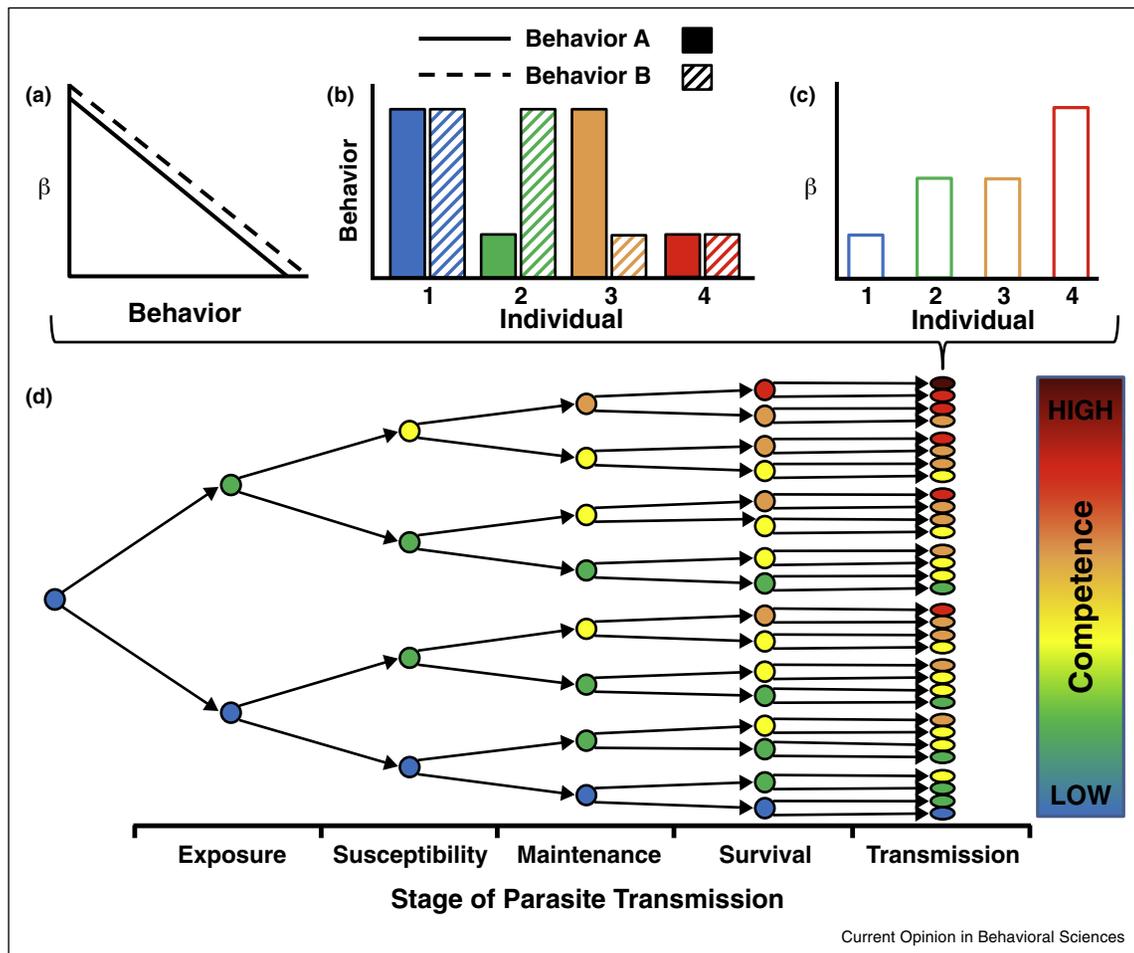
\*Redrawn with permission from [19]

One of the first steps toward unraveling the complexities of host competence is to establish the strength and directionality of relationships among anti-parasite behaviors. Theoretically, these behaviors could covary positively or negatively, each with unique implications for parasite transmission (Table 1). Further, behavioral covariation can occur across two different scales of the transmission cycle (Box 1). First, behaviors could be related *within a single stage of the transmission cycle* (Figure 1a-c). For example, exposure to vector-borne parasites is less likely for individuals that strongly avoid and defend against vectors than those relying solely on defense. Second, behaviors could be separated temporally or *across stages of the transmission cycle* (Figure 1d). This

scenario is exemplified by individuals whose behaviors promoting parasite exposure are positively coupled with those promoting subsequent transmission ('superspreaders' [2]); at another extreme are individuals whose behaviors promoting exposure are inversely related to those promoting transmission ('supersponges'). Importantly, individuals with different behavioral suites can play similar roles in parasite transmission, contingent upon the particular cocktail of behaviors they exhibit (Figure 1c,d).

Although it is impractical to investigate all behaviors simultaneously, there is an efficient approach to identifying the key behavioral traits that drive parasite dynamics. First, researchers could assess the relative importance of

Figure 1



*Within-stage behavioral covariation and host competence:* (a) Parasite transmission rate ( $\beta$ ) is negatively correlated with multiple anti-parasite behaviors. (b) Individuals vary in the extent to which they express each behavior. (c) Individuals that express multiple strong anti-parasite behaviors (blue) are less likely to transmit parasites than those with low expression of one (green & orange) or both (red) behaviors. Here we illustrate the implications of covariation within the transmission stage, though concepts are generalizable to other stages of infection. *Among-stage behavioral covariation and host competence:* (d) individuals exhibit variable levels of behavioral competence at each transmission stage, with the color scheme indicating higher (redder) or lower (bluer) competence within a stage. The terms along the x-axis in (d) depict the stages of infection as describe in Box 1. Ultimately the most (reddest; 'superspreaders') and least (bluest; 'supersponges') competent individuals are determined by their behavioral variation and covariation within and among stages of an infection. Here for simplicity each behavior and stage is treated as having an equivalent influence on competence.

the major categories of host behavior (e.g. defense, sociality, activity) for a single host-parasite system, and then evaluate whether those patterns are generalizable to other systems with similar transmission characteristics (e.g. sexually-transmitted, vector-borne). This framework has been successful for elucidating key physiological predictors of host competence [16,17], and may also prove an effective means of evaluating behavioral traits. Second, researchers can focus on the well-established behavioral associations from the animal personality literature, such as the proactive (e.g. bold, aggressive, exploratory) versus reactive (neophobic, submissive) coping styles, as these coping styles are thought to influence parasite susceptibility [18]. When paired with the associated

knowledge of molecular mechanisms underlying syndromes (e.g. hormonal and genetic pleiotropy) one might also begin elucidating the mechanistic basis of behavioral competence.

### The importance of behavioral plasticity

Behavioral responses to parasites can often be engaged flexibly, in which case they can generate context-contingent variation in the nature and magnitude of the anti-parasite response [8]. Whereas we know that such plasticity is often variable within and across populations, we have yet to investigate in much detail how the environment shapes an individual's behavioral propensity for parasite transmission. Various environmental factors could drive plasticity in

Table 1

**Correlations among anti-parasite behaviors can take many forms. These alternatives are not necessarily mutually exclusive.**

Association	Direction	Description
Unrelated	None	Behaviors operate independently. This outcome would be most likely if behaviors are regulated by different physiological systems.
Condition-dependent	Positive	Better resources simultaneously promote expression of multiple anti-parasite behaviors. This outcome could generate a strong continuum of host competence from individuals with consistently effective anti-parasite behaviors (good condition) to those with consistently ineffective behaviors (poor condition).
Permissive	Negative	Strong expression of one behavior allows individuals to express other behaviors that would otherwise increase parasite risk. The relative equivalence of alternative behavioral profiles could dampen variation in competence across the spectrum.
Trade-off	Negative	High expression of one behavior limits the expression of another through resource or time constraints. The relative equivalence of alternative behavioral profiles could dampen variation in host competence across the spectrum.
Pleiotropic	Positive or negative	A gene or physiological factor (e.g. hormone) influences multiple anti-parasite traits, creating either positive or negative covariation. Implications dependent on directionality.

anti-parasite behaviors (e.g. temperature, resource availability and predictability, competition, and predation risk). If only one host manifests plasticity in competence, a parasite can persist in a community in which it would otherwise be extirpated [19].

Given the commonness of parasites (and even co-infection) in most natural systems, particularly salient drivers of plasticity are the parasites themselves. Parasites might mediate behavioral plasticity in two ways. First, environments can vary in risk of parasite exposure, which may induce different coping styles in hosts residing there. This idea has been investigated heavily in regards to predation, where it was demonstrated that predator abundance promoted strong anti-predator behaviors [20] and selected for unique syndrome structures [21]. Although this pattern likely extends to parasites [22], it has yet to be tested.

A second way that parasites can induce behavioral plasticity involves the infection status of individuals, whether that variation be discrete (uninfected vs. infected) or continuous (light to heavy infection). Perhaps surprisingly, it is largely unknown whether an individual's behavior before parasite exposure predicts its behavior following infection [15]; are those most prone to encountering parasites also most likely to transmit them? As individuals of many species can vary strongly in their immunological tolerance of infection [23,24], or the degree to which they maintain fitness at a given level of parasite burden, it follows that some individuals would be more prone to generate new infections than others [12<sup>\*</sup>]. An individual's tolerance should not only positively predict its parasite burden, but could also influence its transmission opportunities through so-called 'sickness behaviors' characterized by decreased activity, sociality, and feeding (Box 1) [25]. Given the commonness of parasites' co-option of host behavior for transmission it is likely that much post-infection behavior in hosts will increase parasite transmission [26<sup>\*\*</sup>,27,28], such as when lethargy and/or social

isolation promote vector feeding or parasitism [29,30<sup>\*</sup>]; however, these same host behaviors would act to decrease transmission in other systems (e.g. socially-transmitted or sexually-transmitted parasites). What has been little considered though is how infection affects behavioral consistency (i.e. personality) and/or covariation (i.e. syndromes) in hosts [26<sup>\*\*</sup>,31,32]. We need to determine to what extent infection strengthens or weakens associations among orthogonal anti-parasite behaviors and hence mediates variation in host competence.

### Integrating behavioral and immunological competence

Host competence results from a combination of behavioral and immunological traits, and given their shared regulatory mechanisms (e.g. nervous and endocrine systems) it seems likely that they covary [15] (Box 1). Unfortunately few studies to date have considered covariation among behavioral and immunological dimensions of competence (Table 1, Figure 1), though the connections between these systems are likely strong. In humans, for example, the mere sight of infection cues (e.g. skin lesions, sneezing) caused amplified production of the proinflammatory cytokine interleukin-6 [33], and recent infections heightened attention to and avoidance of disfigured individuals [34]. While these transient shifts suggest real-time feedback between behavioral and immunological systems, it is also probable that pleiotropic control can facilitate their long-term coordination. For example, researchers found that human cytokine gene variants associated with increased parasite susceptibility were also associated with personality traits expected to reduce infection risk [35,36]. Similarly, transient early-life immune stimulation can permanently organize adult behavioral and immunological traits [37,38], though surprisingly no research has investigated whether exposure to parasites in early-life affects host competence in adulthood. The mechanistic and functional links between behavioral and immunological traits are mostly speculative, yet fruitful, areas for future experimentation.

## Discussion and future directions

This article highlights how individual heterogeneity in competence for parasites might arise from behavioral variation and covariation. Research objectives for the future include identifying syndromes of anti-parasite behaviors, considering behavioral plasticity across contexts, and integrating behavioral and immunological elements of competence. In the end, our ability to elucidate the role of individuals in parasite dynamics will hinge on several related paths of inquiry. First, a reaction norm approach could prove useful in differentiating plasticity versus constraints in host traits [19,39]. Similarly, a comprehensive view of host competence demands knowledge of its physiological underpinnings, and seeing that both the nervous and endocrine systems coordinate cross-talk between behavioral and immunological functions [38], they should be key foci. As empirical insight accumulates, epidemiological models including heterogeneity in host traits should be employed to determine how behavioral regulation of competence (in isolation and in coordination with other traits) might alter expected patterns of parasite transmission and host demography. For instance, much insight could be gained from establishing the stage(s) of infection in which host heterogeneity exerts the greatest influence on population-level transmission patterns, or by determining how covariation in competence across stages influences an individual's cumulative contribution to parasite spread (Box 1, Figure 1d). This pursuit of a whole-organism view of host competence nicely joins basic and applied realms, and is thus poised to enhance our scientific understanding of host–parasite dynamics and their implications for the health of wild or human populations.

## Conflict of interest statement

None declared.

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