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Niche construction: evolutionary implications for parasites and hosts

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Organisms can determine the environment they experience through the process of niche construction. This may have important evolutionary consequences by exposing them to new selection pressures, producing a faster response to selection, and building suites of co-adapted traits. Traits of the parasite which influence the likelihood of encountering different host environments, or which change the host environment, can be regarded as niche construction traits, as can traits of the host which influence the likelihood of the host being infected by parasites. A niche construction perspective may provide new insights into the evolution of host/parasite interactions; this is illustrated with several examples from the viewpoint of both parasite and host traits.

Parasites, hosts, and niche construction

Niche construction is the process by which the traits of an organism determine the biotic and abiotic environment in which it lives (see Glossary and Box 1). By actively or passively choosing where they live, or by modifying their surroundings, organisms alter their environment. If this altered environment persists to influence the phenotypic traits of subsequent generations, the evolutionary trajectory of a species might be substantially changed. In host/parasite systems, niche construction can be viewed from the perspective of either the parasite (considering the host as part of the environment) or the host (considering the parasite as part of the environment). Microparasites have much shorter generation times than their hosts, and there has been some investigation of the potential role of niche construction in the evolutionary dynamics of bacterial and viral populations in a static host environment. There has been little consideration, however, of the possible evolutionary implications of niche construction in macroparasites, which may live for an appreciable fraction of their host’s lifespan and which must usually leave the host to complete their life cycle. Nor has there been much consideration of how the evolutionary dynamics of hosts may be affected by the way in which their physiological and behavioural activities shape their parasite community.

This Opinion article considers how a niche construction perspective might provide new insights into genetic change by parasites in response to selection pressures exerted by hosts, and genetic change by hosts in response to selection pressures exerted by parasites. These considerations are speculative and are intended to suggest examples of host/parasite interactions where a niche construction view may be productive. I take a quantitative genetic approach to evolutionary dynamics, in part because I believe that most traits which influence the interaction of hosts and parasites are polygenic in nature (Box 2), but also because the evolutionary implications of niche construction are most far-reaching for polygenic traits.

Niche construction may alter selection pressures

The most obvious way in which niche construction can influence evolutionary dynamics is by altering exposure to existing selection pressures or by exposing organisms to new selection pressures. This change in selection pressures may affect the evolution of any trait for which genetic variation exists in the population and does not require genetic variation in the niche construction trait itself.

Glossary

Additive genetic variance: the extent to which phenotypic differences among individuals are due to differences in allelic composition. Correlational selection: selection which acts upon the covariance between two traits, in other words, favours certain combinations of trait values. Ecosystem engineering: modifications of the abiotic environment by an organism that create habitat for organisms of different species. Extended phenotype: an adaptation of an organism which manifests outside of the body of the individual to affect the abiotic environment, conspecifics or other species. Genetic covariance (correlation): the proportion of variance shared by two phenotypic traits due to genetic causes. Genotype-environment correlation (ρGE): occurs when particular genotypes are more likely to experience particular environmental conditions than expected by chance. Genotype-environment covariance (CovGE): non-random association between genetic and environmental effects on the phenotype. Linkage disequilibrium: non-random association between alleles at different gene loci. Niche construction: the process whereby organisms, through their choice of habitat, behaviour, or metabolic activities, influence the environment they experience. Personality: behavioural differences among individuals in a population which are consistent over time and in different contexts. The term ‘behavioural syndrome’ refers, strictly speaking, to correlations among personality traits measured at the population level, but is often used as a synonym for personality, as are the terms ‘temperament’ and ‘coping style’. Phenotypic plasticity: the capacity of a single genotype to exhibit a range of phenotypes in response to environmental variation. Positive assortative mating: a pattern of sexual reproduction in which individuals with similar phenotypes (and/or genotypes) mate more often than would be expected by chance. Reaction norm: the pattern of phenotypes expressed by a single genotype over a range of environmental values.
Box 1. Niche construction

Niche construction is "the process whereby organisms, through their metabolism, their activities and their choices, modify their own and/or each other’s niches” [55]. According to this broad definition, niche construction is any changes that organisms have on their environment; it is not limited only to those changes that are adaptive or that impact upon the niche constructing organism. Niche construction includes obvious examples, such as the construction of dams by beavers and webs by spiders, but also more subtle processes such as the choice of a particular habitat or alteration of the environment through metabolic activities. For example, the feeding activities of earthworms alter soil chemistry, structure, aeration and drainage. This benefits plants, leading to increased plant growth, which in turn provides increased plant litter supply for earthworms. Both earthworms and plants now experience a different environment than did their ancestors, and this has shaped their evolutionary responses [56].

While this is a fairly straightforward concept, there has in recent years been a surge of interest in the ecological and evolutionary importance of niche construction (reviewed in [3,56]). There has also been much controversy over whether niche construction has been adequately accounted for in standard evolutionary theory [57]. Proponents of the niche construction perspective argue that the environment, because it is in effect manipulated by organisms, plays a much more active role in both micro- and macroevolution than is commonly believed. Critics argue that niche construction is often defined so broadly that it can be applied to almost any trait of an organism, and that concepts such as the extended phenotype and ecosystem engineering adequately account for any creative role that the environment plays during evolution.

A distinction should be made between the process of niche construction, of which there are a great number of interesting examples, and niche construction theory, which proposes niche construction as a fundamental cause of evolutionary change, equal in importance to natural selection [57]. A pragmatic approach is to consider whether viewing a trait from the perspective of niche construction provides novel insights into evolutionary and ecological processes, in other words whether a niche construction approach leads to lines of enquiry which would not otherwise have been followed.

There are, however, many examples of genetic variation in niche construction traits, in which case different genotypes may consistently experience different environments, a phenomenon known as gene–environment correlation (rGE); this can bias selection pressures, leading to different evolutionary trajectories for niche construction genotypes [3].

Niche construction by the parasite

Hosts are essential for completion of the life cycle of a parasite and are therefore a predominant feature of the selective environment. From the viewpoint of the parasite, different hosts available to the same life cycle stage represent alternative environments. Any trait which influences the likelihood of encountering different host genotypes or species can be regarded as a niche construction trait and may alter selection pressures on parasite traits which interact with the host environment. Genetic variation in niche construction traits may lead to rGE, favouring the evolution of host-adapted races. The formation of host races has been studied most closely in phytophagous insects, starting with Walsh [4], who reported the shift of the apple maggot fly Rhagoletis pomonella from native hawthorn (Crataegus spp.) to introduced apple (Malus pumila) trees. There are now well-characterised host races in several species of phytophagous insects, many of which are consistent with a sympatric mode of divergence [5].

Phytophagous insects are able to actively choose their host. This may also be the case for parasites of animals, particularly ectoparasites and those with free-living stages. For example, infective stages of the parasitic copepods Lepeophtheirus thompsoni and L. europaensis preferentially colonise turbot (Psetta maxima) and brill (Scophthalmus rhombus), respectively, and these sympatric fishes are the hosts on which each species of copepod is found in the Western Mediterranean [6]. For many parasites of animals, however (and certainly many endoparasites), transmission is passive. Nevertheless, there are parasite traits which will influence host occurrence. Heritable differences have been found in cercarial emergence time between populations of Schistosoma mansoni infecting either humans or black rats (Rattus rattus) in Guadeloupe [7]. Genotypes with an early pattern of shedding are found more commonly in humans, whose activity is principally diurnal, while genotypes with a late pattern of shedding are found more commonly in rats, which are nocturnal. Genotypes which differ in cercarial emergence time will therefore be associated with different host environments and different selection pressures.

Box 2. Polygenic traits and quantitative genetic analysis

The evolutionary significance of niche construction does not depend on any particular genetic model. Laland et al. [58,59] used a Mendelian, two-locus population genetic model to analyse the evolution of a niche construction trait and an additional recipient trait whose selection depended on the environmental change caused by niche construction. This led to unexpected evolutionary dynamics, including momentum effects (continuing evolutionary change when selection has stopped), inertia (delayed response to selection), the fixation of otherwise deleterious alleles, and the maintenance of stable polymorphisms. More recent studies have considered the effect of niche construction on complex, polygenic traits, and suggested novel ways by which niche construction can influence evolution by affecting the genetic variance of these traits [3].

What is the appropriate approach for considering the evolutionary implications of niche construction for interactions between parasites and hosts? Initial models of parasite/host coevolution were largely based on single locus, two allele systems for the genetic determination of parasite infectivity and host resistance [60]. The empirical basis for these models came from interaction loci identified in plant/pathogen (mostly fungi) associations [61]. More recent theoretical and empirical studies, however, suggest that coevolutionary interactions between parasites and both animal and plant hosts are more often mediated, at least in part, by complex, polygenic traits (e.g., [62–70]).

The inheritance of polygenic traits has traditionally been analysed by the statistical techniques of quantitative genetics, which enable the observed phenotypic variance of the trait (Vp) to be partitioned into components due to genetic (Vg) and environmental (Ve) effects: $V_p = V_g + V_e$. Vg is the phenotypic variance due to differences in environments among individuals and Vg is the phenotypic variance due to differences in allelic composition among individuals (for simplicity ignored non-heritable components of genetic variance due to dominance and epistatic interactions). A quantitative genetic approach to predicting the evolution of polygenic traits assumes that these traits are determined by a very large number of genes, each contributing infinitesimally small additive effects. In recent years genomic mapping studies have generally found that the infinitesimal model provides a good approximation to the inheritance of polygenic life-history traits [71,72].
The selection (either active or passive) of different host genotypes or species is not the only way in which parasites may construct their niche. Parasites may also affect many aspects of the physiology, behaviour, or immune response of their host, potentially leading to a change in selective environment for themselves and their descendants. The rabies virus (Lyssavirus), for example, causes physiological and behavioural changes in its host, manifesting as increased aggression [8]. Because viruses have multiple generations within an individual host, the viral population which initiates an infection produces modified selection pressures for its descendants, and this may have important implications for the evolution of parasite traits affecting virulence and transmission [2]. A theoretical model of the evolutionary dynamics of antibiotic-resistant and antibiotic-sensitive bacterial strains suggests that if pathogens can change host behaviour (e.g., if an increase in frequency of resistant bacteria discourages antibiotic use), then strain polymorphism can be maintained even when it would not be expected [1].

Changes in host phenotype induced by parasites may affect selection pressures on infecting species, as well as the niche constructing species itself. This may be a byproduct of niche construction or an adaptation by the niche constructor to provide a competitive advantage. For example, parasites may be able to provoke a non-specific immune response to eliminate competitors. A model of such 'proactive invasion' shows that virulent, immune-provoking invaders can competitively exclude commensal residents at both within-host and among-host levels [9].

Niche construction by the host
Parasites may play a key role in the selective environment of their hosts - they are numerous, in many cases they may significantly reduce survival and reproductive success of individual hosts, they have been shown to be important regulators of host population dynamics and, in addition, they may mediate the outcome of other interspecific interactions within ecosystems [10,11]. As is the case for parasites, any trait which influences the likelihood of the host being infected by parasites can be regarded as a niche construction trait, and genetic variation in such traits may lead to $r_{GE}$, changing the evolutionary trajectory of host populations.

Many traits of the host, including sex, age, behaviour, diet, and immunity, can affect exposure and susceptibility to parasitic infection. While the relative importance of these factors is poorly understood for most host/parasite systems, recent studies have emphasised the role of host behaviour in influencing the likelihood of infection [12,13]. In particular, studies on several taxonomic groups have found consistent, correlated differences among individuals in their exploratory activity, boldness (reaction to risky situations), and aggressiveness to conspecifics, such that they can be aligned on an axis from proactive/bold/aggressive to reactive/shy/non-aggressive [14]. Such personality differences can lead to differences in parasitc infection among host individuals by affecting the level of exposure to infectious stages. We might expect individuals that are more proactive to be more likely to encounter new environmental sources of infection [15]. Similarly, animals with different personality types may differ in their frequency and type of social interactions, which has been linked to level of parasite exposure [16,17]. There is some empirical support for these expectations, with differences in trematode communities between bold and shy pumpkinseed sunfish (Lepomis gibbosus) [18], a greater prevalence of feline immunodeficiency virus in feral cats (Felis catus) [19], and a positive relationship between boldness and tick (Ixodes spp.) load in chipmunks (Tomias sibiria) [12].

Host personality may therefore be viewed as a niche construction trait, with more proactive individuals being subject to greater selection pressures for physiological, immunological, or behavioural traits that influence the reproductive success of parasitised hosts. It is important to note, however, that associations between personality types and parasite exposure may be affected by many factors and are likely to be species- and context-specific [20]. For example, an experimental study which exposed wood frog (Lithobates sylvaticus) tadpoles to parasitic trematodes (Echinopharyum spp.) found that more active and exploratory individuals had a lower, rather than a higher, parasite load [21].

Niche construction may increase or decrease genetic variance
As well as altering the selection pressures experienced by organisms, niche construction may also change the expression of genetic variance in polygenic traits. This arises because polygenic traits, as well as being determined by many genes, each of relatively small effect, are also subject to environmental influences (phenotypic plasticity). Niche construction may affect the expression of genetic variance in phenotypically-plastic traits in one of two ways (Box 3). First, for any trait in which environmental effects differ among genotypes, niche construction, by changing the environment, can either increase or decrease the magnitude of additive genetic variance, which in turn may increase or decrease the rate of response to selection. More importantly, if there is genetic variation in the niche construction trait leading to $r_{GE}$, then niche construction may create a covariance between genetic and environmental effects on the phenotype (genotype–environment covariance or $C_{GE}$), such that effects of allelic composition on trait differences among individuals will not be independent of the effects of the environment they experience. This may increase or decrease the expression of additive genetic variance in the niche construction trait itself, again increasing or decreasing the rate of response to selection.

Niche construction by the parasite
Phenotypic plasticity in parasite life-history traits is not well understood, but is likely to be common [22,23]. Unfortunately, we know very little about reaction norms for phenotypically-plastic parasite traits, and therefore the extent to which niche construction may alter the expression of genetic variation can only be speculative. One promising line of enquiry may be gametocyte sex ratio in Plasmodium spp. In P. chabaudi, for example, gametocyte sex ratio not only differs among parasite genotypes, but can also be facultatively adjusted in response to host anaemia.
Opinion

Box 3. Niche construction and genetic variance

For a quantitative trait (z), the response to selection or change in mean trait value between generations (Δz) is given by Δz = Gb, where G is the additive genetic variance of the trait and b is the selection gradient (the covariance of trait value and fitness). Niche construction, as well as changing selection pressures and therefore altering the covariance between trait values and fitness (see text), can also affect the expression of additive genetic variance in a trait. In other words, the extent to which genetic differences among individuals in a population are reflected in phenotypic differences.

To see why niche construction can affect the expression of additive genetic variance we need to consider the variance partitioning equation of quantitative genetics. The standard form of this equation (\( V_p = V_G + V_E \)) assumes that genotypes do not differ in their response to environmental variation. If they do, then a genotype \( \times \) environment interaction term (\( V_{G\times E} \)) is required: \( V_p = V_G + V_E + V_{G\times E} \). The significance of this term is that differences between genotypes are environment-dependent, such that additive genetic variance and therefore the response to selection will be greater in some environments than others [73]. If niche construction changes the environment for a population of genotypes, and if G×E exists, then the expression of additive genetic variance may be increased or decreased (Figure IA).

The standard partitioning of phenotypic variance also assumes that genotypes are distributed randomly over environments. When there is genetic variation in a niche construction trait, then genotypes will differ both in their DNA sequence and in the environments they experience [3]. An extra term (\( Cov_{GE} \)) therefore needs to be incorporated to account for the covariance between the effects of the genotype and the effects of the environment on the phenotype: \( V_p = V_G + V_E + V_{G\times E} + 2Cov_{GE} \). The significance of this term is that it explains the extent to which expression of genetic variance is either reduced or amplified by the non-random assortment of genotypes among environments. If \( Cov_{GE} \) is negative (allelic composition and environment have opposite effects on the expressed phenotype), then evolution will be constrained because phenotypic differences among genotypes will be minimised. However, if allelic composition and the environment have synergistic effects on the expressed phenotype (positive \( Cov_{GE} \)), then phenotypic differences among genotypes will be enhanced and the rate of evolutionary change will be more rapid than expected (Figure IB).

![Figure 1](https://example.com/figure1.png)

**Figure 1.** (A) The effect of genotype \( \times \) environment interaction (G×E) in a trait on the expression of genetic variance. Phenotypic value for a polygenic trait is shown on the y axis for two different genotypes (black and white circles) expressing the trait in each of two environments which are determined by niche construction (x axis). (i) No G×E, phenotypic difference between genotypes is not affected by the environment. (ii) G×E, genetic variance is increased in environment 2. (B) The effect of genotype-environment covariance (\( Cov_{GE} \)) in a niche construction trait on the expression of genetic variance. Phenotypic value for a polygenic niche construction trait is shown on the y axis for two different genotypes (black and white circles) expressing the trait in their preferred environment (x axis). White genotypes are found in environment 1 and black genotypes are found in environment 2, as a result of niche construction. Grey circles show the phenotypic values that the two genotypes would express in their non-preferred environment. (i) Negative \( Cov_{GE} \), phenotypic difference between genotypes is reduced as a result of niche construction. (ii) Positive \( Cov_{GE} \), phenotypic difference between genotypes is enhanced as a result of niche construction.

...and immune status, and resource competition with infecting genotypes [22]. Intrinsic variation among genotypes in sex ratio pattern will therefore be expressed differently in different infection scenarios, and this may influence the response to selection on the trait.

The evolution of host races in parasites may be enhanced by a positive covariance between genetic and environmental effects on host encounters, as a result of niche construction. In phytophagous insects, an important aspect of host race formation is that differences in host occurrence will lead to positive assortative mating if mating is linked to host choice [24,25]. Any genetic differences in host plant preference will then be reinforced by environmental (host) effects, and genetically differentiated host races may evolve into distinct species if selection is strong relative to gene flow [26,27]. For parasites of animals, a positive covariance between genetic and environmental effects on host encounters will occur when parasites are transmitted vertically from parents to offspring, but is also likely to be the case for horizontally transmitted parasites with direct life cycles if there is aggregation of host types in the environment (hosts are philopatric or social) because host encounter rates will be correlated over time, independently of parasite genotype [28]. Even for parasites with
indirect life cycles, environmental heterogeneity may lead to spatial associations of definitive and intermediate hosts [29].

**Niche construction by the host**

There is, as we have seen, some evidence to suggest that hosts with a more proactive personality type are more likely to be infected by parasites. Parasitised hosts, because they experience increased energy demand, may become bolder and more exploratory in an effort to increase food intake, potentially leading to a positive covariance between genetic and environmental effects, thereby increasing the expression of genetic variance and the response to selection in these personality traits [15]. This suggests that parasitism may have played an important and underappreciated role in the evolution of animal personalities [15,20].

In addition to host personality influencing the risk of parasitic infection, parasites may also alter host behaviour. From the perspective of the parasite, this may, as previously discussed, be regarded as a niche construction trait, but from the perspective of the host it is an environmental effect. Key personality traits such as boldness, exploration, activity levels and sociability often appear to be affected by parasites, and may enhance trophic transmission from intermediate to definitive hosts [15,30]. Infection with *Toxoplasma gondii*, for example, has been shown to increase activity level and exploratory behaviour and reduce aversion to predator odour in laboratory rats (*Rattus norvegicus*) [31,32]. Similar changes in behaviour have been reported for other hosts, such as sticklebacks (*Gasterosteus aculeatus*) infected with the tapeworm *Schistococcus solidus* [33] and killifish (*Fundulus parvipinnis*) infected with the trematode *Euhaplorchis californiensis* [34]. While these studies may suggest a positive covariance between genetic (host) and environmental (parasite) effects on host personality traits, similar to that proposed due to the energetic demand that parasites place upon infected hosts, more detailed analyses are required on the effect of parasite infection on a broader range of host traits before we can draw any firm conclusions about how parasite manipulation might feed back on niche construction to affect the evolution of host personality traits. Manipulative parasites may affect multiple characteristics of their host’s phenotype and it has been suggested that the consistency of trait expression or trait correlations, rather than individual traits themselves, may be the real targets of manipulation [35].

**Box 4. Niche construction and genetic covariance**

Natural selection occurs, not at the level of individual traits, but at the level of the whole phenotype. The equation for response to selection in **Box 3** is therefore complicated by the need to consider genetic covariances among traits because selection on a particular trait produces not only a direct response on that trait but also indirect responses on all correlated traits. The multivariate response to selection is therefore given by:

\[ \mathbf{\Delta z} = \mathbf{G} \mathbf{b} \]

where \( \mathbf{\Delta z} \) is a vector of responses to selection for all traits being considered, \( \mathbf{G} \) is the additive genetic variance/covariance matrix (see below), and \( \mathbf{b} \) is a vector of selection gradients, or partial regression coefficients of fitness on each trait [36].

The G matrix summarises additive genetic components of variance and covariance in a group of phenotypic traits. Genetic covariances may arise because of pleiotropy, where the same gene influences more than one polygenic trait, or through linkage disequilibrium, where non-random associations arise among alleles at different gene loci, either because the loci are physically linked on the same chromosome or because particular allelic combinations are favoured by selection. Genetic covariances that are built through correlational selection will decay rapidly if selection is relaxed, but can persist indefinitely if selection is maintained or if there is co-inheritance of allelic combinations, for example because of inbreeding or population genetic structure [74,75]. This provides time for the evolution of recombination suppression or modifier genes, which may maintain optimal trait combinations even when selection is relaxed [37].

Niche construction can generate linkage disequilibrium between loci which influence the niche construction trait and loci which influence other traits, which are acted upon by the new selective environment [89,90]. The strength of linkage disequilibrium will be enhanced by environmental heterogeneity and spatial clustering of similar niche constructing phenotypes [76]. The genetic covariances built by niche construction will enhance fitness, but may also act as a constraint upon the direction of evolutionary change because they channel the response to selection towards trait combinations with the greatest additive genetic (co)variance; evolution will therefore occur along the axis of greatest genetic (co)variation in multivariate phenotypic space [77,78] (Figure 1).

![Figure 1](image_url) The effect of genetic covariance on the direction of adaptive evolutionary change. The black ellipses illustrate the genetic covariance in values for two traits, shown on the x and y axes, for two different populations (or species). The different orientation of the ellipses indicates a positive covariance between the traits in population A and a negative covariance between the traits in population B. The fitness heat-map suggests a peak of global optimum fitness in the top-right corner (high values for both traits), with a local optimum in the top-left corner (low values for trait 1, high values for trait 2). Population A will evolve towards the global optimum (illustrated by the direction of the arrow), whereas population B will evolve towards the local optimum, because that is in the direction of trait covariance.
Niche construction may enhance genetic covariance

The foregoing has considered the potential effects of niche construction on the evolution of single traits. In reality, traits do not evolve in isolation, but as part of a complex, co-adapted phenotype. Adaptive evolutionary change in a trait therefore depends not only on the genetic variance of the trait itself, but also on the genetic covariance with other traits. When two or more traits affect fitness in an interactive way (i.e., particular trait combinations have higher fitness than others), correlational selection can build favourable genetic covariances through the formation of linkage disequilibrium at loci influencing the traits [36]. Niche construction, by determining the selective environment in which genotypes are expressed, can provide strong and sustained correlational selection, allowing the construction and maintenance of favourable genetic covariances [37]. This can provide rapid evolution to local fitness optima, but may also constrain the direction of future evolutionary change along lines of least resistance (i.e., along the major axes of genetic covariation) (Box 4).

Niche construction by the parasite

The evolution of host races in parasites will be enhanced by genetic covariance between traits which influence host encounters and those which influence survival and reproduction within the host (i.e., host compatibility). Theoretical studies have shown that this covariance may evolve as a consequence of assortative mating based on host occurrence, which leads to disequilibrium between host choice and host compatibility genes [38,39]. Positive genetic covariances between host choice and host compatibility traits have been found in some phytophagous insect species [40], but there have been no empirical investigations of this question in parasites of animals, despite its recognised importance for host race formation [41,42].

Hamblin et al. [2] speculated that niche construction by the rabies and Seoul (Hantavirus) viruses, which both cause increased aggression in their hosts, may have led to the evolution of virus transmission through saliva. These traits are fixed within the species, and we therefore cannot examine genetic covariances between host manipulation and transmission traits, but a comparative study of induced behavioural changes in hosts and mode of viral transmission across a broader taxonomic range might be used to test the hypothesis.

Niche construction by the host

If more proactive personality types have an increased risk of parasite exposure, and especially if there is a positive covariance between genetic and environmental effects on personality, then we might expect selection to maintain genetic covariances between personality traits and other traits, such as immune function, that mitigate the impact of parasitic infections. The establishment and maintenance of genetic covariances will be enhanced if personality types mate assortatively. There is some evidence for this in humans [43,44] but not, as far as I am aware, in other animals, although wild populations of Trinidadian guppies have been found to assort on personality types in social networks [45].

Studies comparing immune function in animals with different personality types have found mixed results, with more proactive individuals having a stronger immune response in some cases [46,47], but a weaker immune response in other cases [48–50]. This limited evidence does not appear to support a genetic covariance between bold personalities and greater immunocompetence. These studies, however, have examined phenotypic, rather than genetic correlations, and given the complexity of the immune response to parasite infections [51], there is clearly a need for much more detailed analyses before we can make any sensible conclusions about genetic covariance between personality traits and immunocompetence.

Concluding remarks

A niche construction perspective emphasises the active role that organisms play in creating their own environment, leading to a systematic bias in the direction and strength of selection. Can this provide us with any novel insights into the ecological and evolutionary processes of parasites and their hosts? I suggest that it can, although, with the exception of host race formation in phytophagous insects, the arguments in favour of a niche construction approach are speculative and need to be developed by more theoretical and empirical studies.

From a theoretical perspective, there is a need to incorporate niche construction into models of parasite/host coevolution, particularly in models which take a quantitative genetic approach to trait inheritance. From an empirical perspective, we would be in a better position to understand whether niche construction has played a role in the evolution of parasite and host traits if we better understood the genetic and environmental components of (co)variance in these traits. Convincing evidence of an important evolutionary role for niche construction may also come from experimental studies. Niche construction traits have been shown to respond to selection in free-living organisms [52,53]. Although experimental evolutionary studies are more challenging to develop for parasite/host systems, they have been used in other contexts (e.g., [54]) and may provide important insights into the role of niche construction in parasite/host coevolution.

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