Parasitism, predation and the evolution of animal personalities

Abstract
Trade-offs between behavioural traits promoting high life-history productivity and mortality may fuel the evolution of animal personalities. We propose that parasites, including pathogens, impose fitness costs comparable to those from predators, and influence the adaptiveness of personality traits associated with productivity (PAPs). Whether personality traits are adaptive or not may also depend on individual immunological capacity. We illustrate this using a conceptual example in which the optimal level of PAPs depends on predation, parasitism and host compensation (resistance and tolerance) of parasitism's negative effects. We assert that inherent differences in host immune function can produce positive feedback loops between resource intake and compensation of parasitism's costs, thereby providing variation underlying the evolution of stable personalities. Our approach acknowledges the condition dependence of immune function and co-evolutionary dynamics between hosts and parasites.

Keywords
Behavioural syndrome, feedback loop, growth-mortality trade-off, parasitism, pathogen, personality.

INTRODUCTION
Observations on consistent, individually characteristic behaviours across a wide variety of taxa have motivated enormous interest in animal personalities and their evolutionary importance (reviewed by Dall et al. 2004; Sih et al. 2004; Dingemanse & Réale 2005; Bell 2007; Réale et al. 2007; Biro & Stamps 2008; Stamps and Groothuis 2010). Even though animal personalities can sometimes appear maladaptive in contrast to fully flexible context-dependent behaviour, their possible adaptive significance is becoming evident (Sih & Bell 2008; Dingemanse et al. 2010; Luttbeg & Sih 2010). In this article, we define (animal) ‘personality’ (also known as ‘temperament’ or ‘coping style’) as behaviour that varies among individuals, but is consistent across time and/or contexts within individuals (Stamps and Groothuis 2010). For example, animals with a ‘shy’ personality are consistently cautious and avoid risk taking, whereas those with a ‘bold’ personality are consistently incautious and prone to take risks (Sih et al. 2004).

Predation is generally considered the major cause of mortality for ‘bold’, risk-taking individuals (e.g. Smith & Blumstein 2008; Biro & Booth 2009), and thus has been identified as an important factor in the evolution of personality traits, such as activity, exploration and boldness (e.g. Dingemanse et al. 2007). Predation incurs not only direct mortality costs, but also the loss of resources via intimidation effects (Preisser et al. 2005; Stamps 2007). Consequently, the adaptive significance of risk taking depends on the relative costs and benefits of being bold at different times and in different microenvironments: bold individuals obtain a fitness advantage (higher resource-intake rates), if predation risk is low, whereas shy individuals can have higher fitness, if predation risk is high (Sih et al. 2004). As the fitness costs of risk taking may increase in parallel with the fitness benefits accompanying increased resource-intake rates, individuals with different levels of boldness may, on average, achieve equal lifetime fitness and a continuum of individuals from shy to bold will remain in a population (Stamps 2007; Biro & Stamps 2008).

Although predation is widely recognized as an important agent in the evolution of personality, the potential effect of parasitism on the evolution of animal personalities has been largely ignored. Nonetheless, parasitism imposes fitness costs comparable to those of predation (Raffel et al. 2008; Rohr et al. 2009; Schmid-Hempel 2009), as parasites...
(including microparasites and pathogens such as viruses and bacteria) are significant sources of mortality and sublethal fitness costs. Very recently, Barber and Dingemanse (2010) outlined the potential importance of parasites in the evolution of animal personality. Here, in accordance with Barber and Dingemanse (2010), we argue that because parasites have major impacts on fitness (including behavioural components of fitness), and because certain personality traits can affect the probability of acquiring and resisting parasites (Lozano 1991; Wilson et al. 1993; Hart 1997), parasites should influence the evolution of personality. We also discuss the implications of this for recent models for the evolution of personality, and explain why parasitism should help generate and maintain consistent interindividual variation in behaviour.

Growth-mortality trade-offs, and feedback loops between food intake and fitness: mechanisms for the evolution of animal personalities

Stamps (2007) proposed that animal personalities are relatively consistent over time since risk taking and other behaviours promoting high food intake rates are driven by individually consistent intrinsic growth rates. An animal’s behavioural foraging potential, composed of traits, such as activity, exploration, boldness and aggressiveness, is associated with growth rate, given that growth is regulated by the intake of resources (e.g. Petherick et al. 2002; Ward et al. 2004). As natural selection acts on intrinsic growth rates (Arendt 1997), adaptive individual differences in behaviours (e.g. activity, boldness and aggressiveness) might be explained by interindividual variation in intrinsic growth capacity and trade-offs between growth and mortality that differ among individuals, but allow them to achieve equal fitness (Stamps 2007; Biro & Stamps 2008; see Adriannsens & Johnsson 2008). However, this mechanism for the evolution of personality relies upon pre-existing genetic differences among individuals in intrinsic growth rates (or underlying traits) to drive the evolution of different behavioural types (personalities).

Alternative mechanisms that emphasize links between behaviour and relatively stable state variables have been proposed for the evolution of animal personalities. These mechanisms include asset protection, i.e. trade-offs between current vs. future reproduction and avoidance of starvation (Wolf et al. 2007; but see Massol & Crochet 2008), and positive feedback loops in state-dependent behaviour (McElreath et al. 2007; Luttbeg & Sih 2010). Recently, Luttbeg and Sih (2010) showed that negative feedback mechanisms, such as asset protection and starvation avoidance eventually lead to convergence of behavioural types over timescales comparable to an individual’s lifetime, and therefore cannot account for the long-term stability of personalities. However, long-term stability was achieved when these negative feedback mechanisms were combined with a positive feedback mechanism, state-dependent safety (Luttbeg & Sih 2010). Thus, state-dependent safety, for example, condition-dependent avoidance of predation – or as we argue here, parasitism – could favour the development of stable personalities by maintaining and reinforcing individual differences that arise from small disparities in original assets (Luttbeg & Sih 2010). Importantly, models by Stamps (2007; also see Biro & Stamps 2008) and Luttbeg and Sih (2010) are not mutually exclusive and yield the same prediction: high life-history productivity and bold behaviours will be coupled. Here, we discuss how parasitism can change the predictions of productivity/mortality models (Stamps 2007; Biro & Stamps 2008) and generate positive feedback loops between host immune function and behaviour, analogous to those generated by condition-dependent avoidance of predation (Luttbeg & Sih 2010). We first separate the effects of parasite exposure on personality variation from parasite-induced selection on personality traits, and then discuss how state-dependent safety, generated by the host’s immune function, affects the costs of acquiring parasites through certain behavioural styles.

Parasites and pathogens as selective agents

In general, parasites and pathogens are just as important as predators in determining individuals’ fitness (e.g. Raffel et al. 2008; Schmid-Hempel 2009). Even though many parasites do not directly kill their hosts, they often weaken or manipulate their hosts, so that the hosts become more susceptible to predation (e.g. Milinski 1990; Seppälä & Jokela 2008). Moreover, parasites can cause low fecundity, sterility or low reproductive rates due to poor success in intraspecific or interspecific competition (e.g. Kavaliers et al. 2000; Barber et al. 2004; Newey & Thirgood 2004). They may also decrease the host’s mating success due to the opposite sex’s avoidance of parasitized mates (Hamilton & Zuk 1982).

Direct effects of parasitism on host behaviour are diverse and relatively well known. Exploitation of hosts by parasites can affect the hosts’ feeding rates, sociability, migration and success in sexual selection (Lozano 1991; Hart 1997; Jog & Warve 2005; Fleurance et al. 2007). In addition, parasites manipulate hosts to increase their own transmission rates, often by increasing the hosts’ risk of predation (Seppälä & Jokela 2008) or the hosts’ aggressiveness (as in rabies; Niegzoda et al. 2002). Therefore, parasites may, by directly altering host behaviour, create apparent personalities or behavioural syndromes during the host’s lifetime (e.g. Coats et al. 2010; see review in Barber and Dingemanse 2010). Our focus here, however, is on parasite-generated selection on genotypes. For example, parasite avoidance can be
considered as a behavioural form of parasite resistance that evolves in response to parasitism. Parasite avoidance is comprised of behaviours that reduce exposure to parasites, for example, moving away from particular areas, such as beddings or nests (Moore 2002; Ezenwa 2004), or removing macroparasites by grooming or scratching. Population-level studies have demonstrated that populations under selection by certain parasites show increased parasite avoidance behaviour (e.g. Cruz et al. 2008). Notably, parasites (especially micro-parasites) are more difficult to avoid through immediate behavioural responses than predators, because the infectious stages of many parasites cannot be observed.

When parasite exposure is associated with personality traits, direct and indirect parasite-induced selection for personality traits may occur. Potential examples of this phenomenon occur in juvenile pumpkinseed sunfish, in which parasite fauna differ among individuals along a shy–bold continuum (Wilson et al. 1993); Siberian chipmunks, in which non-random distributions of parasites among hosts result from personality-related differences in space use (Boyer et al. 2010); and in feral cats, in which higher levels of lethal feline immunodeficiency virus occur in males with more aggressive personalities (Natoli et al. 2005). Likewise, in domestic cats, feline immunodeficiency virus is associated with aggressiveness, large body size and potentially, earlier age at first reproduction (Pontier et al. 1998).

Role of host parasite avoidance and resistance

Links between behavioural traits and the cost of parasitism are necessary for parasitism to affect the evolution of animal personality. To evaluate the effects of past and current behaviour on the cost of parasitism, it is important to consider individual differences in both exposure to parasites and immune function. These two factors have very different natures. While exposure to parasites may be a function of certain behaviours, the cost of that exposure likely varies between individuals in accordance with their capacity to resist parasites. In vertebrates, the effectiveness of the immune system depends on the innate availability of particular major histocompatibility (MHC) alleles and previous exposure to parasites (Woelfing et al. 2009). In contrast, invertebrate immunity relies more on intrinsic, non-specific defences, such as encapsulation and melanization of intruders by the prophenoloxidase-cascade (e.g. Cerneius & Söderhäll 2004; Lee 2006). In addition, parasite resistance and tolerance are energetically costly, and involve trade-offs with other life-history traits (e.g. Zuk & Stoehr 2002; Rantala et al. 2003; Tschirren & Richner 2006). In turn, the higher is the resource availability, the more organisms are expected to invest in immune function (Houston et al. 2007). Therefore, the cost of parasitism will depend on the intrinsic and condition-dependent qualities of an individual and might also reflect an individual’s investment in immune defence in earlier phases of life.

Various models for the evolution of personality (e.g. Luttbeg & Sih 2010) rely upon initial differences in assets among individuals, even if these are very small. However, hypotheses relying on stochastic initial differences in assets or state of individuals do not ultimately explain the adaptive origin of variation in animal personalities. Here, we provide an ultimate explanation for adaptive variation in animal personality by arguing that inherently co-evolutionary and frequency-dependent host parasite interactions maintain interindividual variation in immune function and therefore set different adaptive values for different behavioural styles under the risk of parasitism. We argue that innate differences may be reinforced through positive feedback loops in which high resource-intake rates lead to efficient immune function. Therefore, the immune system is potentially a generator of variation in initial assets, necessary for the development of different personalities (‘behavioural types’, Luttbeg & Sih 2010), and of positive feedback loops that promote and maintain this divergence. Moreover, as individuals with efficient immune function pay a smaller cost for parasite exposure than individuals that accumulate high parasite loads due to inefficient immune function, we predict, under the positive feedback framework, that immunologically competent individuals should generally be more bold and active than less immunocompetent individuals (also see Barber and Dingemanse 2010). We argue that although individuals with inefficient immune function could try to compensate for their innately poor immunocompetence by increasing boldness, the compensation is unlikely to be successful due to frequency-dependent factors or stochastic events involved in feeding behaviour (see also discussion in Dall et al. 2004) and the high potential for extra costs of acquiring more parasites through increased boldness.

We also argue that direct parasite-induced selection on certain personality traits is very likely to occur (also see Barber and Dingemanse 2010). For example, active, social or group-living individuals encounter parasitized conspecifics more often than do sedentary or solitary individuals, which can considerably accelerate the transmission of parasites, particularly horizontally (directly) transmitting parasites and pathogens (Côté & Poulin 1995; Hart 1997; Altizer et al. 2003). Increasing the frequency of social contacts significantly increases parasite spread among mammals (Altizer et al. 2003; Vicente et al. 2007). This increased transmission of parasites via social contact is especially important for infectious diseases, such as influenza or sexually transmitted diseases (e.g. Smith & Dobson 1992; Mosure et al. 1996; Altizer et al. 2003). Indeed, Schaller & Murray (2008) recently proposed that infectious diseases can impose strong selection on human personality.
traits, such as sociosexuality, extraversion and openness to experience.

Another key factor determining the risk of parasitism (reviewed in Altizer et al. 2003) is dominance status, which is usually associated with aggressiveness as well as activity and boldness (e.g. Kortet & Hedrick 2007). Dominance status affects both parasite encounter rates and immune function. Dominant individuals typically have better access to food resources than subordinate individuals (e.g. Stahl et al. 2001). Although this may increase the risk of acquiring indirectly transmitted parasites along with food, enhanced resource intake often improves condition-dependent immune function. Thus, improved immune function may compensate all or some of the negative effects of food-borne parasitism (Moller 1997). Dominant individuals often have stronger immune defence, for example, in field crickets (Rantala & Kortet 2004) and drumming wolf spiders (Ahtiainen et al. 2006). In social species, high dominance status can increase grooming behaviour received from other individuals, which may decrease the cost of ectoparasitism (Foster et al. 2010). Dominance status can also affect endocrine–immunity interactions by affecting the levels of immunosuppressive androgens and stress hormones (Hillgarth & Wingfield 1997). For example, subordination stress in low-ranking fish can increase the risk of parasitism via stress-induced immunosuppression favouring dominant individuals (Conte 2004). Similarly, in some mammals, subordination stress in low-ranking individuals causes immunosuppression (Sapolsky 2005). In contrast, sex hormones that promote aggression but induce immunosuppression can indirectly increase the cost of parasitism for dominant individuals in a number of species (Hillgarth & Wingfield 1997; Easterbrook et al. 2007). Moreover, in some animals including cats and wild rats, dominant individuals have a higher, behaviourally mediated risk of parasitism through injuries from aggressive encounters (Glass et al. 1988; Pontier et al. 1998; Natoli et al. 2005; Easterbrook et al. 2007).

In some cases, parasitism strengthens the behaviourally mediated (via bold, active, aggressive behaviours) trade-off between productivity and mortality compared to the trade-off induced by predation alone. For example, aggressive, dominant rats with a higher risk of parasitism from injury during fights (Glass et al. 1988) might also incur higher risks of predation than shy, subordinate rats, because of their higher activity levels. Here, selection against aggressiveness/boldness/activity would be stronger than that predicted by a predation-only model.

The examples above illustrate the fact that associations between parasite exposure and particular behaviours are probably multifaceted and should often be considered species- and context-specific. Ultimate mechanisms that may influence the impact of parasites on the evolution of personality include the host–parasite populations’ environment and their preceding co-evolutionary history. For example, host social organization and mating system should influence not only parasite diversity and prevalence, but also fitness advantages to parasites of different transmission strategies (Altizer et al. 2003).

Interindividual and intraindividual variation in behaviour

For personalities to be detected in a population, both low intraindividual and relatively high interindividual variation in behaviour must be present (Bell 2007; Stamps and Groothuis 2010). Inherently frequency-dependent co-evolutionary dynamics between hosts and parasites generate and maintain genetic variation among hosts (Anderson & May 1982; Salathé et al. 2007; Woelfing et al. 2009), as parasites, due to their shorter generation times, usually evolve faster than their hosts, and impose continuously changing selection pressures on their hosts. This is probably not the case in predator–prey co-evolution, as predators usually have longer or similar generation times compared to prey. Co-evolutionary parasite–host dynamics mean that parasites can contribute to the maintenance of genetic variation both in initial assets that favour personality differences, and in direct parasite-induced selection on personality traits. Thus, parasites impose strong selection on host immune function and favour maintenance of variation in several facets of immune defence, including MHC genes (Anderson & May 1982; Woelfing et al. 2009). We therefore argue that host parasite co-evolution, which is ubiquitous in nature, is responsible for intrinsic differences in host immune function and that these induce initial differences in individual state (see Luttbeg & Sih 2010). However, we acknowledge that interindividual variation in initial assets may also be generated by other mechanisms including, for example, differences in metabolic rate (Careau et al. 2009), differences in anti-predator ability, susceptibility to stress or intrinsic growth rate (Stamps 2007), differences in parental investment or luck early in life (Luttbeg & Sih 2010).

We argue here that intrinsic differences in host immune function affect the individual-level cost of parasite exposure. We argue further that due to the lower cost of parasite exposure for individuals with a high intrinsic capacity to resist parasites, condition-dependent improvement of immune defence is unlikely to override but rather reinforces initial state differences. Individuals may decrease the cost
of parasite exposure by investing more resources in parasite resistance, but only in relation to their intrinsic resistance capacity. Therefore, we postulate that the stronger is the selection imposed by parasites, the more likely is low intraindividual variation in behavioural strategies, linked to the cost of parasite exposure. We also emphasize that interindividual variation in behaviours is not random, but adaptive, particularly with respect to individual constraints set by the physiological capacity to resist parasites.

A conceptual example adding parasitism to productivity/mortality models

We argue that the adaptiveness of personality traits associated with productivity (hereafter PAPs, i.e. personality traits that promote fast growth/high fecundity), which in some species contribute to active, bold and aggressive behaviours (Stamps 2007; Biro & Stamps 2008), depend not only on predation risk, but also on how they affect an individual’s parasite exposure (Fig. 1a) and resistance (Fig. 1b). In our conceptual example (Fig. 1), we assume two possibilities with varying magnitudes regarding parasitism risk. In Fig. 1a, increasing PAPs per se increase the risk of parasitism whereas hosts’ parasite resistance is not affected. In Fig. 1b, PAPs are independent of the risk of parasitism but increasing PAPs improve parasite resistance so that the cost of parasitism decreases with increasing level of PAPs. We aim to explain: (1) the consistency of individual behaviour, (2) average level of PAPs and (3) population-level variation in PAPs as a function of the abovementioned factors. We assume that interindividual differences are generated and maintained by the mechanisms we have discussed above.

In our example, the risks and costs of predation and parasitism are additive and independent of host body size. By this definition, cost of one type imposes a moderate trade-off between productivity and mortality, and the maximum cost imposes a strong trade-off. We also assume that individuals do not use special allocation strategies between anti-predator and anti-parasite strategies vs. growth and reproduction, but that once increasing resource-intake rates, they increase investment in both life-history productivity and safety provided by enhanced immune function (Houston et al. 2007). We also assume that food intake rates are independent of population density.

To predict evolution, we make the following simplifying assumptions about how trade-off strength relates to the

![Figure 1 Predicted level of personality traits associated with productivity (PAPs) as: (a) a function of the cost of predation and cost of parasitism, and (b) as a function of the cost of predation and resource-intake-dependent immunity (compensation). The predictions apply both in evolutionary time at a population level and in ecological time at an individual level given that individuals vary in their initial state with respect to both parasite resistance and anti-predator ability. (a) We assume that the risks of predation and parasitism are additive and no compensation of either type of cost occurs. (b) We assume that PAPs mediate compensation of the cost of parasitism. The cost of parasitism is assumed to be high at the x-axis (when no compensation occurs). At an individual level, the degree of compensation also applies to differences in the efficiency of immune function. Phenotypically plastic compensation of the cost of predation does not occur in either (a) or (b). The darker the circle, the more active/bolder an individual/individuals should be, and the lighter the circle the less active an individual/individuals should be. The greater the diameter of the circle, the stronger is the expected consistency in personality traits, i.e. the more likely it is that animal personalities may develop/evolve given that interindividual variation occurs. In contrast to the left lower corner in (a), in the upper left corner of (b) a high level of interindividual variation in current parasite resistance (from reinforcement of initial differences through state-dependent positive feedback, see the main text) induces a high level of intraindividual behavioural consistency. Note that the amount of interindividual variation in PAPs is not demonstrated in the figure, but likely depends on the strength of selection and variation in individual assets (see the main text).](image-url)
optimal level of PAPs and the population-level variation in PAPs. If the fitness costs of increasing PAPs increase more rapidly than the benefits, i.e. the costs are accelerating, for example because intake-dependent compensation is inefficient against a high risk of parasites, selection is likely leading to a single evolutionary endpoint (c.f. Hoyle et al. 2008). In contrast, when PAPs increase survival, for example, by decreasing the cost of parasitism, and the costs are strongly decreasing, evolution may proceed towards maximal average levels of activity/boldness (c.f. Hoyle et al. 2008). However, with more weakly accelerating, linear or decelerating costs, the evolutionary outcomes are more difficult to predict but may, at least in non-sexual models, include a stable polymorphism of different behavioural types (de Mazancourt & Dieckmann 2004; Hoyle et al. 2008). In this respect, note that Fig. 1 should be interpreted only as an explanatory example and not as a quantitative model. Our assumed scenarios differ in that Fig. 1a assumes no feedback – host resistance varies, but not in response to PAPs, whereas Fig. 1b assumes a positive feedback, where PAPs bring in the energy that drives an increase in host resistance. Host parasite interactions are inherently frequency-dependent (Anderson & May 1982), and negative frequency-dependent selection pressure arising from the host population itself or from parasites could complicate our simple predictions.

**Personality and parasite exposure**

According to our conceptual example, the trade-off between PAPs and mortality in the productivity-mortality model will be strengthened when parasites are acquired through PAPs and there is no resource-dependent improvement of parasite resistance (Fig. 1a, along y-axis). The stronger the trade-off is, the more consistently an individual should follow the behavioural trajectory set by its initial and current assets and the more likely it is that animal personalities will develop (Fig. 1a). This is explained by the increasing cost of deviating from an optimal behavioural trajectory that is set by initial and current assets. For example, an individual with inefficient immune function will do poorly by behaving in a way that increases parasite exposure, if a behavioural way to avoid parasites exists, and similarly, an individual with efficient immune function loses potential benefits by unnecessarily avoiding parasites. The same is true with predators if individuals differ in qualities that affect their likelihood of being killed by a predator. When the risks of predation and parasitism are close to zero, we would expect to see a maximum level of PAPs, but intraskill variation in PAPs may exceed that of interindividual variation, and personalities may not develop (Fig. 1a, lower left corner). The situation is even more pronounced if parasites are not acquired through PAPs, but through behaviours that are coupled with low productivity rates (e.g. shy and sedentary behaviour). In such cases, parasites will decrease the cost of boldness/high activity and weaken the trade-off further (not shown in the figure).

Thus, when we abandon the potential for condition-dependent variation in parasite resistance, and the costs of parasitism and predation are additive, animal personalities most likely develop in environments with high risks of both parasites and predators (Fig. 1a, upper right corner), as both predators and parasites can affect the adaptiveness of behaviours in relation to intrinsic variation in individual state. This occurs because we assume that interindividual variation in initial assets is constant along the lower left to upper right diagonal (Fig. 1a), and consistency in individual behaviour increases as the cost increases of deviating from the optimal trajectory set by initial assets. However, in nature the likelihood of detecting personalities in a population depends also on the amount of interindividual variation in personality traits. This is set by variation in individual assets determining how tightly individuals follow the same behavioural trajectory (see also Bell 2007).

Simple predictions from Fig. 1a are that: (1) when the cost of parasitism and risk of predation increase, the expected average level of PAPs (e.g. activity/boldness) decreases; and with lower costs of parasites and lower risk of predation, the expected level of PAPs increases. We also predict that (2) when the trade-off between PAPs and fitness costs is strong, it is more likely that individual behaviour will match the assets generated by immune function (or other physiological factors), and consequently, it is more likely that animal personalities will develop or evolve within a population. In this scenario, personality variation is based wholly on initial rank order state differences, which may remain even when individual state feeds back positively to individual state (see below).

**Personality and the compensation of parasitism**

In Fig. 1b, we assume that high levels of PAPs increase the efficiency of immune function through enhanced food intake rates and thus generate safety and decrease the strength of the trade-off between PAPs and fitness costs. We argue that the stronger is the selection imposed by parasites, the more likely it is that low intraskill and high interindividual variation in behavioural strategies, linked to the cost of parasite exposure, will be maintained (Fig. 1b). Consequently, animal personalities will most likely develop in a situation where immune defence is efficient at decreasing the cost of parasite exposure and when predation risk adds to the cost of deviating from the individually optimal behavioural trajectory set by initial and current assets (Fig. 1b, upper right corner).

The safety generated by high levels of PAPs decreases the fitness cost of PAPs and therefore favours higher levels of PAPs than would be expected by the non-compensatory
predation cost model only. This leads to the prediction that individuals with efficient immune function should show higher levels of PAPs than individuals with less efficient immune function (Fig. 1b). Similarly, if individuals can decrease the cost of predation, for example, by growing quickly to a large (safe) size, individuals are expected to show higher levels of PAPs than in situations where PAPs only increase the cost of predation (not shown in the figures). If there is full compensation of increased parasite exposure by condition-dependent improvement of immune defence (Fig. 1b, upper line), the productivity-mortality trade-off in predation-only models (Stamps 2007; Biro & Stamps 2008) will not change except for the fact that asset differences generated by immune function may still contribute to individual consistency of behaviour.

Predictions based on Fig. 1b are that: (1) when increasing levels of PAPs promote compensation of the cost of parasites more than they increase the cost of predation, the expected level of PAPs should increase (in individuals or over evolutionary time) and (2) when increasing levels of PAPs increase the cost of predation more than they provide compensation for parasites, the expected level of PAPs should decrease (in individuals or over evolutionary time). Therefore, the maximum level of PAPs (e.g. maximally active and bold behaviour) is expected when PAPs reduce the cost of parasitism and there are no predators (as in Fig. 1b, or when anti-predator strategies are positively dependent on PAPs).

PREDICTIONS AND CONCLUSION

Our analysis yields predictions that are readily testable. First, an empiricist could test whether individuals with efficient immune function are bolder, more active or explore more than individuals with less efficient immune function and whether this relationship is genetically determined. This prediction arises from the claim that intrinsically immune-competent individuals suffer a small cost of acquiring parasites along with food, whereas all individuals can improve their immune function and parasite tolerance by acquiring more resources (e.g. Houston et al. 2007). The prediction should hold for a wide continuum of taxa and it does not separate intrinsic immunity from condition-dependent immunity, as according to Luttbeg and Sih’s (2010) positive-feedback model with state-dependent safety, the positive correlation between boldness and immune efficiency is reinforced over time. If resource-intake-dependent improvement of immune function fully compensates for increased parasite exposure, bolder individuals should have lower parasite loads. If the compensation is not efficient enough, they should have higher parasite loads. Note that if the cost of higher tolerance of parasites requires higher activity, positive coupling between boldness and resource acquisition is still likely to be generated (Andrew Sih, personal communication). Second, comparative studies could reveal whether species or populations living in environments with high predation and parasitism risk more often show personalities than species or populations living in low predation and parasitism areas (c.f. Dingemanse et al. 2007). We predict that high risks of predation or parasitism favour high levels of boldness, when compensation of predation and parasitism induced fitness costs is efficient, but low levels of boldness, when the animals have little or no means of compensating for the high costs of predation and parasitism. Third, comparative studies or studies using selected lines (e.g. selected for increased and decreased immune function) could reveal whether abundant but non-virulent parasites, against which the host displays condition-dependent parasite resistance, select for higher boldness or stronger behavioural consistency under constant risk of predation than virulent parasites that cannot be compensated for by foraging more.

We predict that predation-associated costs will dominate the evolution of personalities in systems where the cost of parasitism is not clearly associated with host immune function. Similar patterns will occur when host immune functions are inefficient at reducing the costs of parasitism. However, as some anti-predator strategies, including behavioural traits, are also condition-dependent (e.g. Petterson & Brönmark 1999; Skjaa et al. 2004), we predict that fitness compensation through PAPs (e.g. ‘state-dependent safety’; Luttbeg & Sih 2010) also occurs in predation-driven systems, and suggest that the consequences of this mechanism for the maintenance of variation in animal personalities should be evaluated further using the foundation laid by Luttbeg and Sih (2010). Most importantly, we argue that parasites maintain variation in personality traits by maintaining variation in immune function through host-parasite co-evolution. This process is inherently frequency-dependent and therefore unquestionably generates variation (Anderson & May 1982). How frequency-dependent factors affect interindividually and intraindividually in PAPs, depending on population-level feedback mechanisms such as density-dependent food availability and probability of aggressive encounters, is less clear. Whereas maximum activity and low consistency is the apparent outcome in our simplified scenarios with zero costs, frequency- and density-dependent factors will probably come into play in real situations, and maximal aggressiveness, for example, would be an evolutionarily stable strategy in a predator and parasite-free environment only if an individual was short of food (see e.g. Houston & McNamara 1988). On the other hand, certain frequency-dependent behaviours are capable of maintaining rank order differences in individual state and do not affect life-history trajectories induced by differences in initial state (Dall et al. 2004). In any case, the effect of factors such as
density-dependent availability of food and developmental and physiological constraints in food intake rates and growth rates should be taken into account to fully understand the effects of frequency dependence. Thus, a more detailed formal modelling approach is needed.

To date there are very few empirical tests of the ideas presented here. However, Kortet et al. found a positive correlation in field cricket between boldness and lytic activity, a measure of the crickets’ defence against bacterial parasites (Kortet et al. 2007). Correlations such as this are difficult to interpret without knowing the relationships between behaviour, parasitism and the condition dependence of measured immune function. In this particular example (Kortet et al. 2007), the correlation could suggest that bold behaviour facilitates a high resource-acquisition rate, which facilitates an aspect of strong immune function. Alternatively, innately resistant individuals may be better able to cope with the risk of acquiring parasites with food, and for this reason might behave more actively, aggressively or boldly than their less resistant conspecifics (see also López et al. 2005). Indeed, it may be possible that both of these two causal mechanisms are at work. Tests of immunity–personality interactions are warranted and the results will help to further develop the ideas presented here. For example, positive feedback loops between PAPs and immune functions, particularly during ontogeny, should generate divergent personalities which may appear heritable in quantitative genetic analyses due to the underlying inheritance of immune components.

In conclusion, we assert that parasites and pathogens can strongly affect the costs and benefits of many personality traits. We also propose that initial variation in individual assets can be generated by host parasite co-evolution, and that the consequently arising differences in immunity can induce positive feedback loops between fitness and behaviours promoting high food intake rates, favouring different, individually consistent behaviours (personality traits) over long periods of time. Our work adds to the premises that individually consistent behaviours (personality traits) over ours promoting high food intake rates, favouring different, induce positive feedback loops between fitness and behav-

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