The evolutionary ecology of faking sick

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Abstract

Natural selection often produces traits that enable organisms to detect and avoid infected conspecific or environments deemed to be of high risk for parasite acquisition. We propose that such traits could foster the evolution of dishonest signals of infection. We describe herein instances where dishonest signals of infection could be favored by natural selection and the various costs and benefits likely to be associated with them. We further review the available evidence suggesting that such traits could evolve and the ecological contexts which might foster or impede their evolution. Finally, we provide a model verifying that a stable frequency of dishonest signalers of infection can be maintained in populations, at least in principle, and that the stable frequency of dishonest signalers increases with the prevalence of the infection. We conclude that dishonest signals of infection could evolve and be maintained in a variety of systems and warrant further scrutiny.

INTRODUCTION

Parasites are organisms that have adapted to live on or in a host organism in order to exploit the host's resources (Combes 2001). Parasites (both macro- and micro-) can have considerable effects on host fitness and so act as a strong selective force for many organisms. This has favored traits allowing hosts to identify and avoid risks of parasitism in their environment. Parasite-avoidance mechanisms include identifying and avoiding vectors of parasites, infected conspecifics, contaminated areas, or aggregations of parasites (Curtis 2014). As such, parasite avoidance is anticipated to change how animals behave and utilize their environment. These avoidance behaviors are thought to impact various population, community, and ecosystem outcomes, recently referred to as the "landscape of disgust" hypothesis (Weinstein *et al.* 2018).

Here we examine the possibility that the avoidance of parasites by hosts could be influential in another manner: in fostering the evolution of dishonest signals and otherwise biasing signal evolution. Specifically, we outline situations where dishonest signals of infection could benefit infection-mimics, but also induce costs. We first make the case for the plausibility of the evolution of dishonest signals of infection. We then detail various situations where these signals could impact the fitness of their bearers and discuss systems that meet the preconditions necessary for them to evolve. We support aspects of this argument with a straightforward mathematical model that identifies the criteria under which "faking sick" can persist and which illustrates the dependence of this strategy on the prevalence of true parasites. By outlining scenarios in which the evolution of dishonest signals of infection are theoretically plausible, we hope to encourage consideration of less intuitive ways in which parasitism could impact animal communication systems, competition, social behavior, and sexual selection. For simplicity, we limit our discussion to intraspecific infection-mimicry as a male tactic, but our reasoning could apply to females in some cases.

A Case for the Role of Infection Cues in Signal Evolution

Animal signals often evolve to exploit pre-existing sensory biases in receivers (reviewed in Ryan 1990; Endler & Basolo 1998). For instance, novel signals may resemble stimuli to which a receiver is already attracted, such as food-mimicking orange spots on male guppies and appendages on male tetras (Rodd *et al.* 2002; Arnqvist & Rowe 2005). Sensory bias can also select for signals that exploit receivers' aversion to certain stimuli, such as harmless Batesian mimics resembling a dangerous model (Bates 1862). Sickness behaviours (Hart 1988) and physical symptoms of infection are often used to identify infected conspecifics (Zylberberg *et al.* 2013). Symptoms and sickness behaviours have also recently been framed in terms of signal evolution, with the possibility of dishonesty in these signals being conjectured (Shakhar & Shakhar 2015; Steinkopf 2015; Tiokhin 2016). However, to our knowledge, risk-factors for parasitic infection as a template for sensory exploitation in the context of competition and sexual selection have not been demonstrated in any species. We propose that signs of infection could be promising and flexible sources of dishonest signal evolution.

First, infection-mimicking signals have the benefit of their being weighed and interpreted differently by different receivers. For instance, often some members of a population are more susceptible to infection than others or may incur greater costs upon infection (Zuk 2009; Hawley *et al.* 2011). We would predict these highly susceptible hosts would avoid infected conspecifics more so than less susceptible individuals. This allows dishonest signals of infection to potentially target their effects towards specific subsets of a population, or at least differ in the strength of their effects on different receivers. Facultatively expressed dishonest signals of infection are more powerful still because, even in the absence of differential susceptibilities of receivers, they could be expressed or even directed towards (e.g., sneezes) certain individuals whilst being concealed from others. Additionally, in extreme cases, infection mimicry has the potential to attract some receivers but repel others. These possibilities are discussed in detail later.

Second, dishonest signals of infection have the benefit that they are unlikely to interfere with species recognition systems (e.g., identification by potential mates). Contrast this to a hypothetical scenario where individuals evolve to resemble their predators. Such predator-mimics run the risk of being misidentified as heterospecifics, which could hamper their ability to engage in beneficial social interactions, e.g. mating.

In the following sections, we lay out more specific scenarios in which dishonest signals of infection might evolve and be maintained, or in which aversion to infection cues could be an initially exploited sensory bias resulting in subsequent signal elaboration.

The Sickly Defender Hypothesis

The ability to detect and avoid socially transmitted infections has been selected for in many species. Many studies demonstrate that individuals can identify and avoid infected conspecifics based on visual cues, chemical cues, or sickness behaviours (Kiesecker *et al.* 1999; Behringer *et al.* 2006; Tobler & Schlupp 2008; Zylberberg*et al.* 2013; Poirotte *et al.* 2017; Stephenson *et al.* 2018). Were an individual to mimic being infected, it could potentially deter others from approaching or from attempting to utilize a shared environment. This could, for instance, result in a rival foregoing aggression in favor of avoidance (Figure 1). The mechanism by which this might occur would differ depending on how parasites are transmitted and the sensory modalities by which infection is detected and mimicked.

If infections are transmitted by direct contact with infected individuals, or via transfer of blood, it might be beneficial for a healthy individual to avoid combat or social interactions with an apparently infected conspecific. This, in turn, could benefit an infection-mimic by diminishing the frequency and intensity of aggressive bouts. For males with low resource-holding potential (RHP: Parker 1974), false infection might then allow access to more resources than would be possible otherwise, at a reduced cost. Faking sick is likely to be a particularly potent strategy for low-quality males to deter high-quality rivals, as more attractive and higher-quality males are often more risk averse (Hedrick 2000; Fowler-Finn & Hebets 2011; Ory *et al.*2015; Rypstra *et al.* 2015), likely because they stand more to lose if their residual reproductive value is reduced due to infection or predation (Stoehr & Kokko 2006; Engqvist *et al.* 2015).

Dishonest signals of infection may also decrease the odds that rivals will invade an infection-mimic's territory or attempt to consume his resources. Many parasites adopt a sit-and-wait strategy, and aggregations can

form due to limited dispersal of certain life stages (McCoy *et al.* 2003). This means that an infected resident could indicate local infection risk to any would-be intruder, even if the resident is ultimately displaced. In contests, the value of the contested resource is an important determinant in an animal's decision to risk escalated combat for that resource (Parker 1974). If a territory appears to carry with it a higher risk of infection than other territories, its value should be discounted.

Maintenance of Mimicry in the Sickly Defender Hypothesis

The plausible maintenance of dishonest signals of infection are contingent on several factors. The transmissibility of parasites and their associated fitness costs to its host must be weighed against the benefits gained by displacing or attacking infection-mimicking opponents. In cases where an opponent's genuine infection status is ambiguous, evolution is anticipated to select against making the costlier error (Wiley 1994). Thus, if the costs of acquiring a certain infection are higher than the costs of losing a subset of winnable contests, then animals should err on the side of "believing" any infection cues they see.

The frequency of mimics relative to truly infected individuals will alter the relative costs to receivers of ignoring or believing mimics, meaning that the success of mimicry is likely contingent on mimics remaining beneath a certain frequency threshold (negative frequency-dependent selection). When mimics become too common, this will select for receivers to ignore mimics. As mimics decrease in frequency, however, the benefits of ignoring mimics is predicted to be outweighed by the costs of mistakenly confronting genuinely infected opponents. The precise stable frequency of mimics will depend on the fitness impacts of the parasite, its transmissibility, and the relative benefits of acquiring territories or other resources (see Box 1A).

The frequency of mimics might also be shaped by the costs of the strategy (see Box 1A). For instance, there may be costs associated with increased conspicuousness to predators or, very likely, decreased attractiveness to mates. These costs may differ depending on male condition. Seeing as low-RHP males in species with large differences in RHP are perhaps the most likely mimics (Mokkonen & Lindstedt 2016), it may be that their attractiveness to females is already sufficiently low that the costs of exhibiting false infection might be comparatively modest. Conversely, the larger reproductive potential of high-quality males will cause any decrease in their attractiveness to have a greater absolute cost to reproductive success (Engqvist *et al.* 2015), likely causing infection-mimicry to be a suboptimal strategy. In terms of benefits, high-quality males will presumably be capable of competing for mates and resources by conventional means, reducing any benefits of mimicry. Additionally, due to greater risk aversion in high quality males (Engqvist *et al.* 2015), the efficacy of mimicry for deterring high-quality opponents is predicted to be greater than for low-quality opponents (Figure 1). In aggregate, infection mimicry is anticipated to impose steep costs to high-quality males with low returns, whereas low-quality males are anticipated to experience lower costs and greater returns. These differential costs and benefits of mimicry may be sufficient to ensure that the mimic strategy is not optimal for all males, thus preventing the frequency of mimics from crossing the threshold at which receivers begin to ignore apparent infection cues. The attractiveness costs of mimicry, and ways in which they may be altered or mitigated, are discussed in more detail later.

If infection mimicry is most beneficial and least costly to low-quality males, then it is most likely to evolve as a condition-dependent strategy. Condition-dependent models of alternative reproductive tactics posit that an individual's condition will affect an animal's developmental decision to go down alternative strategic routes (e.g., if big, be a fighter, if small, a sneaker) (Repka & Gross 1995; Gross & Repka 1998). This allows for the maintenance of different tactics with different fitness outcomes. If costs to low-quality males of conspecific aggression exceed the benefits of maximizing their attractiveness, then infection-mimicry could be a fitness optimizing strategy, even if their overall fitness is lower than high quality males. Mimicry, then, becomes a classic "making the best of a bad situation" strategy.

Box 1A: The Sickly Defender Hypothesis: Proof of Concept Model

To demonstrate the plausibility of constitutive dishonest signals of infection, we use a simple model of their evolution. Our model is based on the *Sickly Defender Hypothesis* , in which a mimicking male accrues fitness benefits through conflict-avoidance but incurs costs due to foregone mating opportunities. We represent

male fitness as the difference between fitness gains (benefits) from reproduction and fitness losses (costs) from competition with other males. The general expression of this, for a male of any type i , is:

$W_i = R_i - C_i$.

We consider three male phenotypes: Males may be sick (infected, I), or healthy. Healthy males may either mimic (M) a diseased state, or not (N). We assume that the choice of mimicry is made either at birth or early on in development, and is consistent throughout an animal's lifetime (e.g., a healthy individual either mimics or does not, but does not switch between these strategies).

Both reproductive benefits and competition costs are affected by a male's phenotype. For a healthy male, reproductive benefits occur at a rate r , whereas sick or mimicking males have a reduced benefit *r(1-)* due to female avoidance of obviously unhealthy individuals. Here, δ is the proportion of reproductive fitness lost due to illness. Male-male conflict also depends on phenotype. Conflicts are the most intense (and most costly) between two healthy males, which incur costs at a rate c per encounter. Conflicts between sick (or mimicking) and healthy males incur reduced costs for the sick (or mimicking) individuals because healthy males choose to avoid contact that may transmit infection. Because mimics are healthy and wish to avoid infection, they also reduce contact (and conflict) with sick or mimicking individuals. Thus, the cost to a healthy male of conflict with a sick or mimicking male is c(1-α), and the cost to a mimicking male of conflict with a non-mimicking healthy male is c(1-β), where α and β are the proportionate reductions in conflict costs for avoidance of sick individuals, and avoidance by healthy individuals, respectively. Infected males experience the same landscape of intraspecific interactions as mimicking males, except that they incur an additional fitness cost s from being sick.

The fitness functions for each type of male are therefore:

$W_N = r - c\left[N + (1-\alpha)(M+I)\right]$

$W_m = r - c\left[N + (1-\alpha)(M+I)\right]$

$W_I = r - c\left[N + (1-\alpha)(M+I)\right] - s$

We can express these fitness functions in terms of the frequency of each phenotype in the environment (such that $N + M + I = 1$), where $I = p$, the proportion of individuals who carry the disease,$N = x(1-p)$, the proportion of healthy individuals who fake sickness, and $M = (1-x)(1-p)$, the proportion of healthy non-mimics:

$$W_N = r - c\left[1 - \alpha\left(x - xp + p\right)\right]$$

$$W_M = r\left(1-\delta\right) - c\left[1 - \beta\left(1-x\right)\left(1-p\right) - \alpha\left(x - xp + p\right)\right]$$

$$W_I = r\left(1-\delta\right) - c\left[1 - \beta\left(1-x\right)\left(1-p\right) - \alpha\left(x - xp + p\right)\right] - s$$

We focus on the evolution of x , the frequency of mimicry among healthy individuals. To determine the equilibrium frequency of mimicry, we must study the dynamics of the differential equation

$$\dot{x} = x\left(1-x\right)\left[W_M\left(x\right) - W_N\left(x\right)\right]$$

which describes how the frequency of mimicry changes over time (Nowak 2006). This equation has three possible equilibria: $x^* = 0$ (no mimicry), $x^* = 1$ (all healthy individuals mimic), and $x^* = \hat{x}$ where$\hat{x}$ satisfies $W_M\left(\hat{x}\right) = W_N\left(\hat{x}\right)$(and mimicry persists in the system). If all healthy males mimic ($x = 1$), the value of the mimicked signal should quickly erode; therefore we are most interested in the latter equilibrium, which implies that mimicry can evolve, and that its evolved frequency is $0 > \hat{x} > 1$.

For mimicry and non-mimicry to co-occur, two conditions must be met:

1. The mimicry-free ($x^* = 0$) equilibrium must be unstable, which implies that a mutant, mimicking male in a population of non-mimics must have a higher fitness than the non-mimics, or, mathematically, that $W_M(0) > W_N(0)$ so that$c\beta(1-p) > r\delta$. Biologically, this means that if the benefits from conflict avoidance exceed the costs of lost mating opportunities when mimicry is rare, mimicry should invade.
2. The mimicry-exclusive ($x^* = 1$) equilibria must be unstable, meaning that a mutant non-mimicking male in a population of mimics should have a higher fitness. Mathematically:$W_M(1) < W_N(1)$ so that $r\delta > 0$. Biologically, this means that, so long as there is a fitness cost to mimicry (in this case due to reduced reproductive opportunity), mimicry will never be the sole evolutionarily stable strategy and will coexist with non-mimicry.

If these conditions are met, the evolved frequency of mimicry (which satisfies $W_M(x) = W_N(x)$) is:

$$\hat{x} = 1 - r\delta c\beta(1-p).$$

The larger the costs of mimicry (due to reductions in mating opportunities, $\rho\delta$), the lower the frequency of mimicry. The greater the benefits of mimicry (due to reductions in competition, $\varsigma\beta$), the greater the frequency of mimicry.

The frequency of mimicry is also affected by the prevalence of disease in the population (Figure 2): The more frequent the disease, the less effective mimicry becomes until, ultimately, a mimic in a population of non-mimics has a lower fitness than the non-mimics and thus mimicry is lost from the population. This is because, in high disease environments, intense conflicts between healthy males are relatively rare, and individual fitness is primarily driven by differences in attractiveness to females.

Infection mimicry & Could-Be Mates: Costs to Attractiveness

Though deterring same-sex rivals via appearing infected could be beneficial, the difficulty posed by infection mimicry is that the benefits of deterring one class of conspecifics (same-sex rivals) must be weighed against the costs of deterring others, such as potential mates. Indeed, many theories of sexual selection posit that extravagant sexual signals evolved as honest indicators of the signaller's lack of parasites and underlying ability to resist infection (Hamilton & Zuk 1982; Andersson 1994). This suggests that females should select against individuals who appear infected, particularly if parasites can be transmitted directly (Able, 1996). There are, however, factors that may reduce such costs or eliminate them entirely, as discussed below.

There is often sexual dimorphism in susceptibility to, and costs of, infection. For many kinds of parasites, males are at greater risk due to immune suppression via testosterone (Folstad & Karter 1992), higher stress (reviewed in Sapolsky 2005), or energy allocation trade-offs (reviewed in Nunn *et al.*2009). In these cases, males may have a higher susceptibility to becoming infected when they encounter infectious material (Zuk 2009; Hawley *et al.* 2011). These sex-differences could result in male and female receivers being deterred to differing degrees, allowing false infection to be a powerful deterrent to male rivals while only imposing modest costs in terms of attractiveness to females, at least for certain kinds of parasites.

Parasites may also evolve to be more virulent and costly for one sex than other, causing there to be disparate costs to becoming infected. Many infections are differentially spread by males because of their wider ranges, greater contact with conspecifics, and greater immune susceptibility (Hawley *et al.* 2011). Therefore, we may expect parasites to optimize their pathology on male bodies as opposed to females (Duneau & Ebert 2012; Duneau *et al.* 2012), leading to differences in the costs of infection in hosts of different sexes (e.g. Blanco *et al.* 2001; Tseng 2004). Sexual dimorphism in infection outcomes is especially pronounced in polygynous species, in which males must compete intensely for mates, and this is associated with greater parasite-induced male morality (Moore & Wilson, 2002). When fitness costs from infection are sufficiently steep for males relative to females, males should be more averse to risk of infection (Stoehr & Kokko 2006). Thus, in certain scenarios, we should predict the behavioural elements to immunity (Schaller & Park 2011) to be particularly active in males relative to females, and this should be especially true for high-quality

males who have a greater residual reproductive value to protect (Engqvist *et al.* 2015). This could further enable the evolution of dishonest signals of infection with comparatively modest costs to a male mimic's attractiveness relative to their intra-sexual deterrent ability.

An ideal case of asymmetrical risks of infection is when a parasite targets sex-specific tissue. For instance, certain species of myxosporeans, a microscopic parasite, specifically parasitize male gonads in fish and amphibians (reviewed in Sitjà-Bobadilla 2009). When infection is pronounced, this can produce externally visible infection cues (Sitjà-Bobadilla 2009). These parasites can occasionally be transmitted via direct contact, but infection is more commonly acquired via free-floating spores. As such, faking sick could make a territory unappealing to could-be opponents, while the cues could be mostly irrelevant to prospective females, which lack the tissues necessary to harbor the infection.

For the arguments presented above, the infection being mimicked need not be entirely benign to females, as long as the costs of female deterrence are outweighed by the benefits of male deterrence. As was discussed in the section describing the *Sickly Defender Hypothesis* , costs in terms of loss of attractiveness may not be equivalent for males of high and low quality. Thus, even a slight decrease in the immediate attractiveness of high-quality males may be enough to make infection-mimicry suboptimal, while the factors described in this section may allow mimicry to be a more plausible condition-dependent strategy for low-quality males.

The Allure of Infection

While mates typically discriminate against infected partners (Hamilton & Zuk 1982; Borgia 1986; Able 1996), there may be some scenarios in which infection (or infection-mimicry) could enhance an individual's attractiveness. Here we consider three such cases: i) situations in which infections can indicate terminal reproductive investment, ii) instances in which tolerance of parasites is an indicator of potential genetic benefits, and iii) situations in which infection could provide species-identification cues. Mimicry in these scenarios would of course not be maintained by costs to attractiveness as outlined in the*Sickly Defender Hypothesis* , but could be maintained by more conventional mechanisms (Andersson 1994).

Direct Benefits Due to Terminal Investment

Life history theories predict a trade off between investment in growth, reproduction, and survival (Stearns 1992; Roff 1993). As opportunities for future reproduction are diminished in iteroparous animals (e.g., via aging, injury, or disease), an individual's best strategy may be to invest more heavily in their current reproductive event (Williams 1966). This is called terminal investment (Clutton-Brock 1984). Parasites often castrate their host or otherwise decrease their longevity or ability to reproduce in the future, thus reducing the residual reproductive potential of the individual and so promoting increased investment in current reproduction (Agnew *et al.* 2000; Gandon *et al.*2002; Duffield *et al.* 2017). In iteroparous species, selecting terminally-investing males as current reproductive partners could provide direct benefits to females. For instance, infected partners might provide better parental care (Velando *et al.* 2006), increased fertilization success due to increased spermatogenesis and sperm storage (McCurdy *et al.* 2000; Derting & Virk 2005; Brannelly *et al.* 2016), or more nutritious or preferred nuptial gifts (Hurd & Ardin 2003; Duffield *et al.* 2015).

Tenebrio molitar represents a particularly intriguing example of the potential for terminal investment to influence attraction and reproductive success in the mate of a terminal investor. Cuticular hydrocarbons and volatile glandular pheromones in immune-challenged *T. molitor* males are more attractive to females than are those of healthy males (Nielsen & Holman 2012). In addition, the nutrient content of spermatophores of infected *T. molitor* males is superior to that of healthy males, resulting in a positive relationship between male parasite intensity and female reproductive output (Hurd & Ardin 2003). In cases such as this, it is plausible that females may be selected to prefer terminal investors, and for mimics to evolve to capitalize on the attractiveness of apparent infection.

The virulence of parasites is likely to affect how drastically individuals increase investment during terminal investment, and hence the plausible evolution of female preference, and mimicry. In general, with increased parasite virulence, terminal investment becomes adaptive because future prospects become increasingly bleak

(Gandon *et al.*2002). If females attempt to capitalize on the terminal investment of their mates, virulence and attractiveness may be similarly linked. Additionally, increased virulence should increase the deterrent effect on same-sex conspecific rivals (Stoehr & Kokko 2006) as, even if infection were to produce a temporary spike in attractiveness, becoming legitimately infected reduces fitness overall. Virulence, thus, provides a potential link between these effects of mimicry.

Indirect Benefits Due to Genes for Parasite Tolerance

Females may also choose mates based on their ability to tolerate parasites. Hosts may diminish the negative impacts of parasites through resistance or tolerance: Resistance includes behaviours and physiological responses that allow individuals to avoid becoming infected, while tolerance involves diminishing the negative effects of infection once infected (Råberg *et al.* 2007; Best *et al.*2008; Råberg *et al.* 2009). Theoretical models of sexual selection and female choice often posit that extravagant male traits are an indicator of a male's underlying ability to resist infection, and so females choose flashy males in order to gain indirect benefits of increased resistance in their offspring (Hamilton & Zuk 1982; Andersson 1994). However, becoming infected involves an element of chance, whereas being able to tolerate infection is only possible if that animal truly has a physiology capable of such tolerance. There could, therefore, be a selective advantage for females to attend to a male's quality and vigor despite his infection, which would set the stage for the evolution of mimicry.

There is some circumstantial evidence rendering it plausible that females could prefer infected mates due to their ability to tolerate infection. The ability of individuals to tolerate infection has been shown to vary in wild populations of dace, and this variability seems to be genetically mediated (Blanchet *et al.* 2010), potentially allowing inherited offspring tolerance to be an indirect benefit to females. In these dace, and in mice, parasite resistance and tolerance are negatively correlated, suggesting a trade-off between investment in each mechanism (Råberg *et al.* 2007; Blanchet *et al.* 2010). Female white-footed mice prefer to mate with males who are infested with bot fly larvae, possibly because their ability to continue functioning in the face of infection indicates tolerance to females (Cramer & Cameron 2007). Thus, under certain circumstances, it is possible that females might select males as fathers who will provide indirect genetic benefits in terms of parasite tolerance rather than resistance.

Tolerance is usually measured as the steepness of the slope of a regression of host fitness against infection burden (Simms & Triplett 1994; Koskela *et al.* 2002). As such, females could use the mismatch between a male's level of parasitic infection and his performance of other fitness-enhancing behaviours to evaluate tolerance. For instance, a male who can perform an energetically vigorous display while being heavily parasitized could be selected by females (i.e., a parasite-mediated handicap). By this same logic, a male who artificially augments his apparent parasite burden and so appears to be more heavily infected than he really is, could make any energetic display he does seem more impressive. A parasite that is highly costly to males would allow a mismatch between infection status and vigour to be particularly informative to females, while also effectively deterring rivals.

How prevalent a parasite is in a population is likely to influence the benefits of female choice for tolerance and resistance. In the white-footed mouse example mentioned above, the authors proposed that tolerance may be highly beneficial due to the ubiquitous nature of bot fly infections, which infect 69.8% of males (Cramer & Cameron 2006, 2007). It is likely that, the more ubiquitous a parasite is in a population, the stronger will be the selection pressures on females to ensure their offspring inherit genes promoting tolerance. This is because the chances of offspring experiencing infection are high. In populations in which a certain infection is ubiquitous, it is unlikely that many mimics will avoid becoming truly infected. However, depending on how females evaluate potential mates, if a male were to exaggerate or augment his apparent parasite-load, he could still increase the above-mentioned mismatch between his vigour and apparent parasite burden. Thus, if females evaluate male vigour and apparent parasite load on a continuous scale (see: Kennedy *et al.* 1987; Zuk 1988; Buchholz 1995), rather than classifying males dichotomously as either infected or not, mimic signals which exaggerate infection could still make males appear more parasite-tolerant. As such, parasite prevalence and female evaluation heuristics will shape the likelihood that mimicry will evolve.

False Infection & Species Recognition

Due to parasite-host specificity, false signals of infection could evolve as means of reinforcing species recognition. Hybrid zones and hybrid reproduction by allopatric species are common phenomena (Harrison 1993). Often hybrids are at a fitness disadvantage compared to non-hybrid individuals of either species (Sage *et al.* 1986; Bleeker & Matthies 2005). This favors reliable mechanisms of species recognition (Andersson 1994). Parasites are often highly species-specific, even when closely related species occur sympatrically (Van As & Basson 1987; Bittencourt & Rocha 2003; Dick 2007). As parasites are obliged to identify their correct hosts, and do so by directly sampling the host's physiology, the presence of visible infection, or symptoms of a host-specific parasitic infection, could be used to increase the reliability of conspecific recognition.

Females tend to be especially attentive to reliable indicators of species identity, because the costs of mistakenly mating with heterospecifics are typically greater for females (Parker, 1979; Parker 1983; Parker & Partridge 1998). Thus, attending to cues of species-specific infection could provide indirect benefits to females via increased offspring vigour, and direct benefits due to reduced mate searching costs. Signals to emphasize or exaggerate, or even "imitate" being infected could then be advantageous for increasing male reproductive success. Over time, a runaway process could cause these signals emphasizing or imitating species-specific cues of parasitism to become species-identifying sexual signals and possibly spread to fixation if costs are low (Fisher 1930). In this hypothetical, infection mimicry is likely not to be dishonest as such, because males displaying false infection would do best to be most attractive to their own species to avoid reduced hybrid fitness in their offspring.

Other Potential Benefits of Intra-Specific Infection Mimicry

Deterring Truly Infected Individuals

Dishonest signals of infection may help to keep legitimately infected conspecifics at bay. Parasites often adaptively disrupt host defence mechanisms against other infections (Wakelin 1984). For example, helminths can suppress the immune response of their hosts in order to improve their own survival (Maizels *et al.* 2004) which can cause increased host susceptibility to other parasites (Helmby *et al.*1998; Su *et al.* 2005; Hartgers & Yazdanbakhsh 2006; Kamal & El Sayed Khalifa 2006) or make the costs of infection more severe (Marshall *et al.* 1999; Graham *et al.* 2005). Thus, genuinely sick individuals are predicted to exhibit heightened levels of disgust to promote avoidance of further infection (Oaten *et al.*2009). By mimicking being parasitized, a healthy individual could, therefore, deter truly infected conspecifics and reduce their chances of becoming legitimately infected (Loehle 1995).

Deterring Unwanted Mates

Dishonest signals of infection could be used by individuals to avoid harassment by the opposite sex when infection cues reduce attractiveness to mates (Thomas *et al.* 1995; Arnqvist & Rowe 2005). This strategy is much more likely to be employed by females rather than males (Arnqvist & Rowe 2005). An analogous case exists in damselflies, in which some females mimic males (Cordero *et al.* 1998). This reduces the level of male harassment but increasing their chances of remaining unmated. Stability of this male-mimicry strategy is posited to be maintained via negative frequency-dependent selection.

Social Exploitation

Symptoms of illness have recently been discussed as being signals, and dishonest signalling has been suggested as a means of social exploitation (Tiokhin 2016). Dishonest symptoms are posited to be a means for individuals to gain social advantage, such as avoiding engaging in costly cooperative behaviours and eliciting social or material support. See Tiokhin (2016) for a thorough discussion of these ideas.

Interspecific Infection Mimicry: False Infection as an Anti-Predator Strategy

For prey, exhibiting dishonest signals of infection could deter predators under a range of circumstances. Dishonest signals to predators are not uncommon in prey: for example, Batesian mimicry is the well-studied phenomenon of non-harmful prey mimicking the honest signals and cues of dangerous species (Bates, 1862).

We propose that cases in which parasites are generalists (i.e., could infect both predator or prey) or merely negatively impact the predator (i.e., make it sick or render the prey distasteful) could foster the evolution of dishonest signals of infection or sickness in prey.

Lafferty (1992) produced a model and classic review paper demonstrating that predators do not tend to be deterred by parasitized prey and may prefer them. He explains this as a result of parasites causing their prey to become easier to catch and handle, often via parasite-induced behavioural changes or increased conspicuousness. While this article is often cited as an argument against predators avoiding parasitized prey, it should be noted that this model specifically considers trophically-transmitted parasites. As such, these parasites have evolved to be consumed by a secondary (predator) host and use behavioural manipulations of their intermediate hosts to complete this lifecycle. By contrast, parasites that are *not* adapted to this multi-host lifecycle may not induce host behaviours that increase ease of capture. In extreme cases, some parasites cause aposematism (Fenton *et al.*2011) or other defensive mechanisms in their hosts (Chailleux *et al.* 2013) to reduce the risk of predation. Thus, we argue that there could be a range of conditions where the evolution of infection-mimicry is favored by causing prey to become less appealing to predators.

There is evidence to suggest predators do avoid sick or parasitized prey. For example, *Macrolophus pygmaeus* , an egg predator, shows a preference for *Tuta absoluta* eggs unparasitized by *Trichogamma* parasitoids (Chailleux *et al.* 2013). Late-stage parasitized eggs turn black due to melanisation by the parasitoid larvae, and these black eggs are highly discriminated against. Similarly, rejection of parasitized prey has been noted in a variety of other vertebrate and invertebrate taxa (Holling 1955; MacLellan 1958; Hulscher 1973; Quezada & DeBach 1973; Sloan & Simmons 1973; Cowan 1981; Hoelmer *et al.* 1994; Roger *et al.* 2001; Al-Zyoud & Sengonca 2004). In such cases, it is plausible for mimics to exploit these predator preferences and to be maintained at some frequency.

The same general argument can apply to mimicry as a means of avoiding attack by parasitoids. Multiple parasitism often increases the odds of premature host mortality or can otherwise reduce parasitoid fitness, so parasitoids regularly evolve preferences against pre-parasitized hosts (Salt 1961; Prince 1976; Iwasa *et al.* 1984). In such cases, selection could favor the evolution of mimics that can falsely convey pre-infection.

The maintenance of dishonest signaling in these circumstances depends on the particulars of the system. For example, characteristics of the parasite are important as the parasite must either cause infection in the predator, render it ill, or otherwise decrease the value of the prey relative to alternatives. The plausibility of mimic strategies will also depend on the availability of alternative prey (Kokko *et al.*2003), meaning that the success of mimicry will depend on how specialized the predator is, and the composition of the prey community as a whole. Highly specialized predators will be under strong selection to counter mimicry as a deterrent strategy.

Facultative or Hidden Signals of False Infection

Until now we have treated infection mimicry as a permanent trait. However, the evolution of facultatively expressed dishonest signals of infection could be particularly beneficial. Many sickness behaviours, or behavioural symptoms such as sneezing, could be exhibited flexibly, and so could be shown to rivals, but concealed from potential mates or social partners. This flexibility would mitigate the trade-off between attraction and repulsion of different subsets of conspecifics. Situational expression of sickness behaviours mediated by the social setting has been demonstrated in some case studies (Owen-Ashley & Wingfield 2006; Lopes *et al.* 2012). For instance, zebra finch males who were made sick showed sickness behaviours in isolation, but not in the presence of a female or in a group (Lopes *et al.* 2012; Lopes *et al.* 2013), indicating flexibility in the expression of these behaviours based on audience composition. Sickness behaviours and behavioural symptoms are likely the most conducive to flexibility. Permanent visual signals would likely not be able to be changed in real-time, and longer-lasting signals such as scent marks on a territory (Kavaliers *et al.* 2005) would not be amenable to flexible deployment.

Just as flexibility in signal deployment could reduce the costs to attractiveness of mimicry, so too could taking advantage of polymorphisms in the sensory perception of receivers. Sensory systems often differ markedly

among species, and even within species (reviewed in: Dangles *et al.* 2009). For example, in some New World primate species, males and females differ in their ability to perceive colours (Jacobs 1994). Situations such as this could allow certain signals to be perceived disparately between the sexes. Taking advantage of such differences in sensory capabilities might be especially useful in interspecific infection signalling, such as deterring predators, as mimicry in a signalling channel only available to predators would allow species to be mimics to predators without compromising cues important for intraspecific communication.

Flexible or "hidden" signals in intra-specific mimicry, however, raise some theoretical difficulties: It is difficult to reconcile why the low costs of these signals (i.e., near zero reduction in attractiveness to mates) would not result in these signals spreading to high frequency and, thus, select for receivers to ignore them. Maintenance could perhaps be achieved if there are steep cognitive costs to receiver's ability to detect the trait, or if the signal itself were costly to produce, meaning not all individuals can generate the signal. Alternatively, what began as a signal exploiting a sensory bias against infection cues could be coopted for other purposes, such as becoming a general warning or courtship signal. The selective benefits leading to the origin of a trait (including signals) can differ from the benefits that cause a trait to be maintained or elaborated (Williams 1966; Arnqvist 2006). Thus, even if the costs of mimic signal production are low (Szamado 2003), if it is reliably deployed in specific circumstances such as agonistic interactions, then what began as an infection-mimicking signal, based on the arguments outlined above, could subsequently evolve to be part of a normal species' repertoire of agonistic behaviours.

Conclusions

We have outlined herein a variety of selection pressures and ecological conditions that could drive the evolution of dishonest signals of infection and maintain them. Preconditions necessary to foster the evolution of these traits are met by a variety of animal systems but these hypotheses have never been considered in depth by theoreticians or empiricists. Yet, there are numerous plausible conditions where dishonest signaling of infection could be favored. Where do we go from here? In truth, almost any contribution would be a novel one. Empiricists could experimentally impose the traits of infection in otherwise healthy individuals and compare their performance in staged interactions with legitimately infected and healthy rivals in contrasting ecological conditions (e.g., predators +/-, high/low resources). Likewise, one could evaluate infection-mimicry in terms of the residuals of a regression of symptoms versus parasite load: do some individuals appear particularly healthy despite heavy parasite loads (parasite tolerance) while others seem idiopathically sickly despite low parasite loads (possible mimics)? How do such individuals fare in terms of their overall fitness or performance in contrasting contexts, like acquiring mates, securing fine territories, and so on? Theoreticians could contribute by analyzing models that link more familiar models of dishonest signaling with models of the evolution of virulence, optimal diet theory, animal contests, kin selection, and so on. We freely admit that this is all, for now, conjecture. Yet, we hope that the plausibility of the arguments herein will inspire evolutionary biologists and ecologists alike to consider the possibility of mimicry within their own study systems and to evaluate their plausibility in silico.

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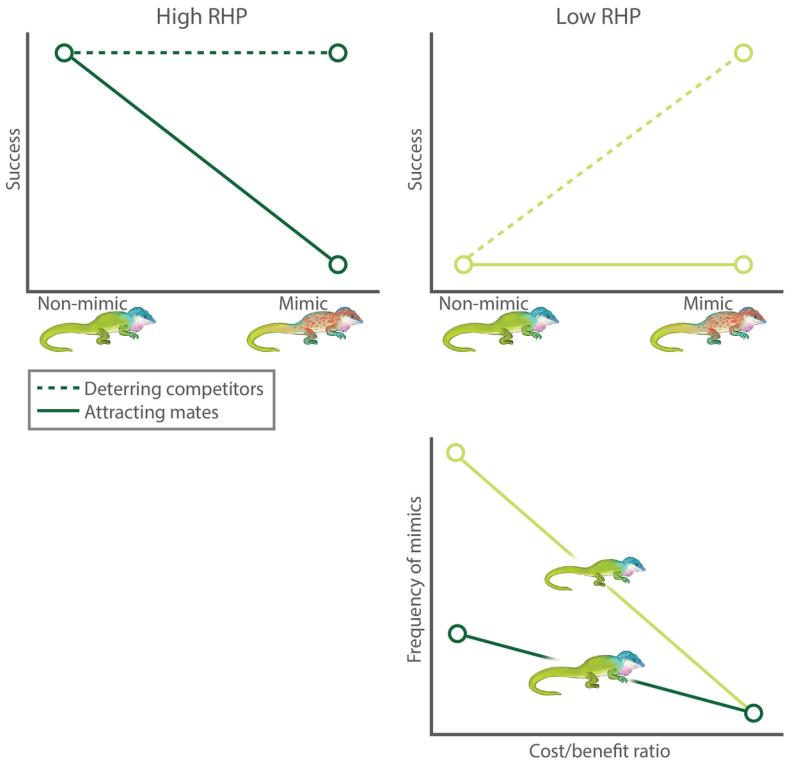

Figure 1 : Anticipated cost and benefits of providing dishonest signals of infection in territorial males of contrasting resource holding potential (RHP). High RHP mimics (top left, dark green) are predicted to experience no benefit to territory defense, because they are already physically capable of defending their territories, but are predicted to experience a drop in their ability to attract females, which devalue infected males. Low RHP mimics (top right, light green) are predicted to experience an increase in territory defense, as rivals may avoid infected conspecifics for fear of transmission, but low RHP males suffer little costs in terms of mate attraction, because they are already physically unappealing. In aggregate, the stable frequency of mimics is predicted to be higher among low RHP males than high RHP males for most sets of ecological conditions.

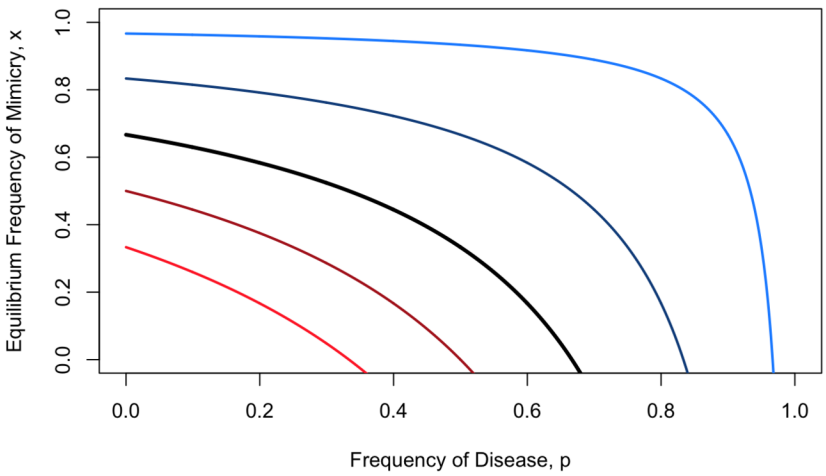

Figure 2: The evolutionarily stable frequency of mimicry is a declining function of disease prevalence. As the frequency of disease p increases, the frequency of mimicry maintained in the population x decreases. Lines show solutions for different values of the parameters δ, the reduction in reproductive fitness due to mimicry, and β, the reduction in competition costs due to mimicry. The base case (δ=0.1, β=0.3) is shown in black; blue lines show the effect of decreasing δ (to 0.05 and 0.01), and red lines show the effect of decreasing β (to 0.2 and 0.15). For all cases, r=c= 1.