When sexual selection in hosts benefits parasites

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Abstract

In host-parasite coevolution, the parasite is selected to increase its infectivity while host is selected to resist the parasite infection. It is widely held that parasite-mediated sexual selection can further amplify the selective pressure on the host to overcome parasite infection. In this paper we focus on certain types of parasites, those that can impair the activity of the host immune function to prevent signs of sickness. We show that the effect of sexual selection can actually reduce the selective pressure on the host immune response to adapt to the parasite infection. We design a simple mathematical model for a population of sexually reproducing organism in which individuals are choosy, preferring traits that are correlated negatively with immune system activity. We introduce to this population a parasite that can suppress activation of the host’s immune response. Our results show that even though the host immune system is likely to ultimately evolve and adapt to the parasite infection, when sexual selection is part of this process, it can slow down this evolution on the host and give the parasite more time to get established.

1. Introduction

In host-parasite coevolution, a parasite is selected to increase its infectivity while a host is selected to resist the parasite infection (Woolhouse et al., 2002). Among the various ways that hosts resist the parasite infection, the immune response is the most sophisticated (Schmid Hempel, 2011). Therefore, there is a constant and strong selective pressure on the host immune response against parasite infection (Zuk and Stoehr, 2002). In this paper, we use the term parasite to refer any organism that has an obligate parasitic life cycle, including pathogens such as viruses and bacteria.

Parasite-mediated sexual selection can further amplify the selective pressure on hosts to overcome parasite infection. For example, under the good genes hypothesis proposed by Hamilton and Zuk (Hamilton and Zuk, 1982), females choose mates based on their secondary sexual traits; full expression of which can be reduced by a parasite infection. In such models, by choosing mates with exaggerated secondary sexual traits, a female can increase the chance that her offspring have the ability to resist infection (Westneat and Birkhead, 1998). Alternatively, under the parasite avoidance hypothesis, females choose males with the most extravagant ornaments to reduce the risk of acquiring contagious parasitic infections (Borgia, 1986). In parasite-mediated sexual selection models, females are generally assumed to be more choosy than males because they invest more energy in the process of reproduction (Lehtonen et al., 2016). However, if parasites are sexually transmitted—or if
transmission is just more likely with close contact—both sexes should be comparably selected to avoid mates showing signs of infection. It is possible for some males to actually benefit from transmitting a pathogen to a mate by reducing her potential reproduction with future partners (Johns et al., 2019). In this case, females should be even more strongly selected to prefer mates who exhibit no evidence of infection.

In the models discussed above, the effect of the sexual selection should generally increase the selection for resistance to parasites and further reduce the fitness of infected males. We will show, however, that this is not always the case. In fact, for certain types of parasites, sexual selection can further facilitate the transmission of the parasites and reduce the selective pressure on the host immune response to resist the parasite infection.

When a host becomes infected by a parasite, its immune defense will be activated to increase its chance of recovery (Adamo, 2014). Since the immune defense is expensive for the host, other energetically expensive behaviors, such as sexual behaviors or foraging for food, should be reduced, shifting resources towards defense against the parasite (Zuk and Stoehr, 2002). This adaptive reduction in normal behaviors that are energetically expensive that occurs as a consequence of activation of the immune response is called sickness behavior (Adamo et al., 2014). In this situation, besides a significant decline in the ability of a host to express sexual behavior, the attractiveness of its secondary sexual traits will also decline (Adamo, 2014). Thus, activation of the host immune response should have a negative effect on the exaggeration of its secondary sexual traits. Moller et al. studied this hypothesis, using meta-analysis of previous studies and concluded that there is a negative correlation between an individual’s secondary sexual ornaments and its immune activation (Moller et al., 1999).

Recent studies show, however, that some parasites can reduce the activity of the host’s immune function, either by limiting the sickness behaviors, or by directly impairing the initial immune activation (Adamo, 2013; Adamo et al., 2014). For example, Adamo et al. identified the pathogen iridovirus IIV-6/CrIV, that transmits through sexual contact between its hosts, the cricket Gyllus texensis, during matings (Adamo et al., 2014). The pathogen suppresses the sickness behavior of these crickets, and the infected males actually show amplified sexual behaviors during matings. Interestingly, males infected by IIV-6/CrIV produce courtship songs with shorter latency compared to those that are uninfected or infected by other pathogens such as Serratia marcescens (Adamo et al., 2014). Note that the latency in courtship song is the period of time between when a male first notices a female and when it start producing the first courtship song. Therefore, shorter latency in producing the courtship song corresponds to showing more sexual behavior.

In the absence of sexual selection, when a parasite strategy is to impair or circumvent the host’s immune response, it should amplify the selection pressure on the immune response of the host to resist the parasite infection (Frank and Schmid-Hempel, 2008). But when a host is under sexual selection, mating success also has a key role in its fitness. In this situation, those parasites that are capable of suppressing their host’s immune activation can indeed help their infected hosts to increase their mating successes, because prospective mates cannot detect signs of the diseases. This matters because evolutionary fitness is the product of survival, mating success, and fertility. Though such parasites decrease survival, they increase another component of fitness, mating success (Fig. (1)).

Because this parasite strategy both weakens the host’s immune response and potentially increase contact with other hosts (via mating), it could lead to increased spread of other parasites as well. This in turn would feed back positively on the
immunomannipulative parasite, by increasing the proportion of hosts infected by other parasites; making those that do not express sickness behavior all the more attractive to mates.

In the next section, we use a simple mathematical model to describe how an infection by parasites that suppress the immune response of their hosts can influence overall host fitness. The key component of our model is a trade-off between survival and mating success. Our analysis shows that with sexual selection, the host’s immune response does not always evolve to maximally impede parasites.

Because we are concerned with the initial evolutionary response to a pathogen, we will hold the pattern of sexual selection (i.e., what traits in males are preferred by females) constant in our model. However, we will note in the discussion that the process that we identify will, in the long term, lead to the evolution of sexual preference itself.

2. Model and results

We consider a population of sexually reproducing organism in which individuals are choosy about mates, preferring traits that are generally correlated with health, and therefore are negatively correlated with immune system activity. We assume that in this population several different parasites are circulating and they are capable of infecting individuals. But infection by those parasites activates the host’s immune response which decreases the quality of host’s sexual traits and therefore mating success. We introduce to this population a focal parasite that can manipulate its host’s behavior by suppressing the activation of host’s immune response. Therefore, despite being infected by a parasite, the quality of the host’s sexual traits will remain high.

In sexual selection, individuals choose between different prospective mates. The probability that a particular competitor is chosen as a function of what other prospective mates are available. This makes it an example of frequency dependent selection, meaning that the fitness of a strategy is function of the frequencies of it and other strategy in the population. As a result, we cannot assume that the average fitness in the population will increase (Rice, 2004). Instead, we use a principle from adaptive dynamics (Metz et al., 1995) that says that the mean value of a trait in a population will increase if the following condition holds:

For any one individual, increasing that individual’s expression of the trait adaptive dynamics (Metz et al., 1995) that says that the mean value of a trait in a population will increase if the following

$$\frac{dh}{dt} + dw = 0$$

(1)

Table 1
Symbols and notation.

| Symbol | Meaning |
|--------|---------|
| $h^*$  | The ability of an individual host to prevent parasites from interfering with its immune response and sickness behavior. $h = 0$ corresponds to a case in which the host has no defense against the manipulative parasite. |
| $h$    | The mean blocking ability in the host population |
| $t$    | Time |
| $w$    | Individual fitness that measures the expected number of offspring produced by an individual. |
| $s$    | Survival of an infected individual which is a random variable taking values 0 or 1 depending on whether an individual does not ($s = 0$), or does ($s = 1$) survive to sexual maturity. The expected value, $E(s)$, is the probability that an individual survives to sexual maturity. |
| $\tau$ | Mean population survival in the absence of disease |
| $m$    | Mating success of an infected individual which measures the number of times an individual mates. It is a random variable taking possible values $(0, 1, 2, \ldots)$. The expected value, $E(m)$, is the expected number of matings for an individual |
| $\bar{m}$ | Average population mating success |
| $F$    | Individual fecundity of an infected individual. Measures the number of offspring produced from a single mating. It is a random variable taking possible values $(0, 1, 2, \ldots)$. |
| $F^*$  | Maximum fecundity of an individual |
| $r_f$  | Expected virulence of the focal parasite. It measures the proportional reduction in the probability of survival due to a pathogen. $r \geq 0$, with $r = 0$ meaning that the pathogen has no effect on host survivorship. Because it measures a proportional change, it is dimensionless. |
| $I_f$  | Relative frequency in the population of individuals infected by the focal parasite ($= \text{the proportion of individuals so infected}$). $0 \leq I_f \leq 1$. |
| $r_o$  | Expected virulence of other parasites in the environment |
| $b$    | Relative frequency of individuals that are infected by other parasites |
| $r_{s0}$ | Expected virulence of the coinfection |
| $r_{s1}$ | Sickness behavior of an infected individual. Sickness behavior includes reduced effort devoted to courtship and foraging, as well as other behaviors that conserve energy. $r \geq 0$. |
| $r_b$  | Average sickness behavior of the population |
| $\rho^*$ | Baseline sickness behavior of an individual |
| $b$    | Measure the proportional (therefore dimensionless) degree to which the manipulative parasite suppresses sickness behavior in the host. $b = 0$ if the pathogen does not interfere with the expression of sickness behavior. |
| $k$    | Measures the proportional rate at which expected mating success declines as sickness behavior increases. |
| $\alpha$ | Measures the degree to which virulence of a parasite also reduces fecundity (in addition to survivorship). $\alpha \geq 0$, if $\alpha = 0$, then the parasite does not influence fecundity (conditional on survival) even if it reduces survivorship ($r > 0$). |
that are coinfected by both pathogens (\(P(\text{coinfection})\)). In such coinfection cases we assume that the expected virulence of other parasites, and the expected virulence of the coinfection. Note that depending on the interaction between the pathogens, \(P(\text{coinfection})\) can have different forms. For simplicity, in all simulation we assumed \(P(\text{coinfection}) = P_0 + P_1 \cdot P_2\). \(P_0\) and \(P_1\) are the relative (i.e. the proportion of the population) frequencies of individuals that are infected by focal and other parasites, respectively. Simply, the numerator in the exponential term of Equation (3) captures average virulence experienced by hosts. In Equation (3), \(r_{h^*}\) measures expression of the sickness behavior which decreases the effect of virulence and increases the chance of survival. It is expressed as follows:

\[
r_{h^*} = I_f (1 - I_0) r^* \exp \left( - \frac{b}{1 + h^*} \right) + I_0 (1 - I_f) r^* + I_0 I_f r^* \exp \left( - \frac{b}{1 + h^*} \right)
\]

In the above equation \(r^*\) is the baseline sickness behavior of an individual in response to an infection, \(b\) measures the ability of the focal parasite to suppress sickness behavior, and \(h^*\) measures the individual’s ability to block immune suppression by the parasite. Note that we categorize infected individuals (I) into three groups, namely, the ones that are only infected by the focal pathogen \(I_f (1 - I_0)\), the ones that are only infected by other pathogens \(I_0 (1 - I_f)\), and finally the ones that are coinfected by both pathogens \(I_0 I_f\). Also, we assume that the events of being infected by focal and other parasites to be independent, i.e., \(P(\text{coinfection}) = I_f I_0\). Therefore we have:

\[
I_f (1 - I_0) + I_0 (1 - I_f) + I_f I_0 = I
\]

The first term of Equation (4) captures the case in which a host is only infected by the focal parasite and the sickness behavior is reduced by a decreasing exponential term, \(\exp \left( - \frac{b}{1 + h^*} \right)\). The second term of Equation (4) captures the cases in which a host is only infected by other parasites that cannot suppress the host’s sickness behavior. Finally, the third term of Equation (4) represents the effect of possible coinfection by focal and other parasites. In such coinfection cases we assume that the host’s immune response will be also suppressed because of effect of the focal parasite.

The expected mating success of an individual is defined as:

\[
E(m|s) = \bar{m} \exp (-k (r_{h^*} - r_h))
\]

where \(\bar{m}\) is average population mating success. \(k\) measures the proportional rate at which expected mating success declines as sickness behavior increases. If \(k = 0\), then the expression sickness behavior has no effect on that individual’s expected mating success. A large value of \(k\) means that expressing even a small amount more sickness behavior than average greatly reduces expected mating success, and a small amount less than average greatly increases expected mating success.

At the level of individual interactions: If we imagine an individual female choosing between two prospective males that express different degrees of sickness behavior, \(k\) does not measure the female’s preference, but rather the sensitivity of that preference to the difference in sickness behavior between the two males. A large value of \(k\) corresponds to a case in which a small increase in sickness behavior in a male leads to a large drop in the probability that a female will prefer that male.

Equation (6) assumes that \(k\) is a constant, so mating success drops off exponentially with increasing sickness behavior. This corresponds to a case in which even a small amount of sickness behavior is quickly noticed by potential mates and influences their preference. In the Appendix, we consider the case of a inverse logistic function for mating success (as well as for survivorship). In that case, individuals largely ignore small expressions of sickness behavior in prospective mates, only allowing it to significantly influence their preference when it becomes more pronounced. The Appendix shows that our principal results follow from both forms of the \(E(s)\) function and the \(E(m|s)\) function. This suggests that our results are not just artifacts of particular functions chosen.
rh measures the average sickness behavior of the population and has the following equation:

$$rh = If(1 - l0)r^* \exp\left(-\frac{b}{1 + h}\right) + l0(1 - lf)r^* + l0lf r^* \exp\left(-\frac{b}{1 + h}\right)$$

Equation (7) has the same form as Equation (4), but with h substituted for h*. We are thus assuming that different hosts differ only in their immune response to focal parasites.

As we mentioned in the introduction, expressing sickness is negatively correlated with mating success. An individual gains mating success by expressing less sickness behavior than in the population, if rh - rh* < 0. Note that in Equation (7), a term h represents average ability of hosts to block suppression of their immune response by the focal parasite.

Finally, we model a expected fecundity as follows:

$$E(f|m, s) = \frac{F^*}{1 + \alpha(\eta f I f(1 - l0) + \eta d l0(1 - If) + \eta d f l0)}$$

In the above equation, F* denotes the maximum fecundity and α denotes the degree to which the fecundity is reduced by the expected virulence. For simplicity, we are assuming that expected fecundity (as distinct from survival and mating success) is not directly influenced by the ability to express sickness behavior. This may well not be the case. Sickness behavior might reduce fertility while it is being expressed, but increase future fertility after the pathogen has been defeated.

Now, by substituting Equations (3), (6) and (8) into Equation (2) we obtain the following equation for the fitness of the infected individual:

$$w = \frac{3MF^* \exp\left(-\frac{\eta f I f(1 - l0) + \eta d l0(1 - If) + \eta d f l0}{1 + \alpha(\eta f I f(1 - l0) + \eta d l0(1 - If) + \eta d f l0)} - k(rh - rh*)\right)}{1 + \alpha(\eta f I f(1 - l0) + \eta d l0(1 - If) + \eta d f l0)}$$

To find the strength of selection at the beginning of infection, we calculate the fitness gradient by taking a partial derivative of fitness with respect to host’s ability to block the parasite, rh*, and set that and average population blocking ability, h, both equal to zero. Fig. (2) shows a plot of selection gradient versus sexual selection intensity. We see that in the absence of sexual selection, the selection gradient is positive and therefore selection favors increasing the host’s ability to block the manipulative parasite. But when a host is under sexual selection, the fitness gradient becomes negative and selection favors decreasing ability to resist the parasite.

Selection on the host’s blocking ability is also influenced by the relative frequency of the focal parasite (i.e. its proportion among all parasites). Fig. (3) shows the relation between selection gradient and sexual selection intensity at the three levels of the focal parasite’s frequencies. When the relative frequency of the focal parasite increases, sexual selection makes it increasingly adaptive to not resist the parasite blocking the immune response. This is because at the low relative frequency of focal parasite, most of the infections in the population involve other parasites that impose sickness behavior, which is selected against by sexual selection. As the relative frequency of the focal parasite increases, hosts that do not block it actually gain a fitness advantage through sexual section.

If the virulence of the focal parasite is too high such that the degree in which it decreases the host’s survival is much higher than the benefit it gives to the mating success, then the influence of sexual selection on reducing the strength of selection on the immune system will decrease. Fig. (4) depicts selection on the host’s blocking ability for different levels of focal parasite’s

![Figure 2](image-url)  
**Fig. 2.** Strength of selection at the beginning of the infection. The figure shows the plot of the fitness gradient versus sexual selection intensity. Note that fitness gradient calculated by taking the partial derivative of fitness with respect to host’s blocking ability (h*), then setting both host and average population blocking ability to zero, i.e. h = h* = 0. As the intensity of sexual selection increases, the fitness gradient becomes negative which means that selection favors decreasing the host’s ability to prevent parasite from manipulating its (host’s) immune response.
virulence. When virulence of the parasite is high then increasing sexual selection is less effective for selection on host’s ability to block manipulation by the parasite.

As the relative frequency of focal parasite increases, the incidence of coinfection for other parasites also increases. Indeed, it is more advantageous for other parasites to coinfect with the focal parasites since they can benefit from the strategy of the focal parasite in suppressing the host’s sickness behavior. Fig. (5) explains such cases by showing the relationship between the change in fitness with respect to the relative frequency of other parasites ($w_{Io}$) and intensity of sexual selection ($k$) at three levels of focal parasite’s frequencies ($I_f$). Interestingly, we see that when the relative frequency of the focal parasite increases the fitness cost to other parasites is reduced ($\Delta w_{Io}$). This means that as focal parasite becomes more common in the population, more cases of coinfection occur which further helps the other parasites. Note that as other parasites increase in relative frequency, more and more hosts will become infected. Those not also infected by the focal parasite will exhibit increased sickness behavior further increasing the intensity of sexual selection favoring those infected by the focal parasite.

3. Discussion

When a parasite infects a host, the host’s immune response becomes activated to increase its chance of survival against the parasite infection. Therefore, in the coevolutionary battle between host and parasite, there is an strong selective pressure on
the parasite to circumvent the host immune response, and an strong selective pressure on the host to resist the parasite infection. There are some known mechanisms by which infection could increase male reproductive output. In some cases, a male might benefit from passing a pathogen to a female if this reduces her future reproductive potential with other males (Johns et al., 2019). Also, an individual (male or female) may increase its short-term reproductive output if a pathogen threatens its long-term survival (Caswell, 2000). In both of those cases, sexual selection by potential mates should work against the pathogen.

Previous models of parasite-mediated sexual selection have assumed that sexual selection should amplify the selective pressure on the host’s immune response to resist infection (Hamilton and Zuk, 1982; Møller, 1996; Westneat and Birkhead, 1998). But this assumption cannot be generalized for all types of parasites. Some parasites can impair their host’s immune activation, such that the host shows reduced sickness behavior and normal sexual behaviors. In such cases, the host’s mating success need not be reduced, and may in fact increase if many potential mates are expressing high immune system activity due to other parasites. System like this are known to exist. For example, an experimental study on the cricket Gyllus texensis and its obligate parasites, iridovirus IIV-6/CrIV, showed that sexual behaviors in infected male crickets surprisingly increase and there was less latency in their courtship song, comparing to both uninfected males or those infected with other parasites (Adamo et al., 2014). Although the mating success of male crickets infected by IIV-6/CrIV was the same as uninfected male crickets, their life span and fecundity were reduced relative to uninfected crickets. There is thus still selection to block the parasite, but it is less intense that it would be in the absence of sexual selection.

We showed that when a host is under parasite-mediated sexual selection and the parasite is able to suppress the host’s immune activation, then sexual selection reduces the strength of selection to resist the parasite infection. Therefore, parasites have more time to get established in population. Any novel immune response against the parasite infection will result the

**Fig. 5.** Plot of the change in fitness with respect to the relative frequency of other parasites \( (\frac{v}{v_0}) \) versus sexual selection intensity \( (k) \). As the focal parasite becomes common in the population, there is a less degree on reduction level of \( (\frac{v}{v_0}) \). Therefore, more cases of coinfection occur, which help the other parasites by suppressing the host’s sickness behavior.

**Fig. 6.** Strength of selection at the beginning of the infection. The figure shows the plot of the fitness gradient versus sexual selection intensity. Note that fitness gradient calculated by taking the partial derivative of fitness with respect to host’s resistance substituting both host and average population resistance to zero, i.e \( h = h^* = 0 \). As the intensity of sexual selection increases, the fitness gradient becomes negative which means that selection favors decreasing the host’s resistance.
sickness behavior which in turn reduces mating success. In this situation, infected hosts with impaired immune response can gain mating success, while sacrificing survival. Therefore, we predict that selection favors the novel immune response that balances this trade-off between survival and mating success.

We also showed that since such parasites are able to suppress the host’s immune activation, as they become more common in the population, there is a higher chance for the incidence of coinfection. This is because by coinfecting a host, other parasites can benefit from the suppression of the host’s immune activity and increase their chance of transmission.

Parasites that can impair and evade their host’s immune system are well documented in the literature (Schmid-Hempel, 2008), and a few are known that can actively enhance the host’s sexual behavior (Heil, 2016). For example, infection by the protozoan parasite Toxoplasma gondii manipulates sexual behavior and enhances attractiveness in male brown rats (Dass et al., 2011). A study by Lim et al. further showed that infection by Toxoplasma gondii enhances testosterone production in male rats (Lim et al., 2013).

Our model focuses on the initial spread of a novel parasite that influences its host’s immune response, and the host’s initial evolutionary response. There are other kinds of feedback, however, that will likely affect the short and long-term dynamics of evolution.

In the short timescale, the spread of a new parasite may reduce host population density, influencing parasite dynamics (Ashby et al., 2019). Population density is not explicitly represented in our model, but it could affect the evolutionary outcome if reduced host population density influences the strength of sexual selection. The intensity of sexual selection has been shown to increase at low population densities in seed bugs (McLain, 1992), dung beetles (McCullough et al., 2018), and soay sheep (Coltman et al., 1999). This may be due to reduced interference competition between males, leading to the increased importance of female choice (McLain, 1992), or an increase in the importance of initial mate choice due to reduced subsequent mating opportunities (McCullough et al., 2018). By increasing the impact of mate choice on fitness, reduced host density will amplify the benefit that a pathogen that reduces sickness behavior will gain from sexual selection in the host population.

In other cases, reducing population density can reduce the strength of sexual selection. For example, in the broadcast spawning sea urchin Strongylocentrotus franciscanus, sexual selection on males declines as density gets low (Levitan, 2004). The effects that we describe here may thus be reduced in organisms with external fertilization if the parasite significantly reduces host population density.

Over a longer timescale, a parasite that reduces host sickness behavior will impose new selection in host mate choice preferences. In nearly all models for parasite-mediated sexual selection, the basic assumption is that the host’s secondary sexual traits are honest traits, meaning that they accurately reflect the host’s physiological state (Ashby and Boots, 2015; Hamilton and Zuk, 1982; Knell, 1999; Poulin and Vickery, 1996). The idea is that maintaining such an expensive trait is possible only in highly fit individuals. If the parasite is able to enhance that secondary sexual trait, however then it is no longer an honest trait. As a result of this parasite strategy, we expect to see antagonistic coevolution between the male’s secondary sexual trait and the female’s preference, similar to chase-away sexual selection (Holland and Rice, 1998).

One possible scenario for the future of such a system is that the prospective mates evolve to prefer a more exaggerated secondary sexual trait, or to focus on a different trait altogether. This will, in turn, change the selective pressure on the parasites.

Even though the host immune system is likely to ultimately evolve and adapt to the parasite infection, our model shows that when sexual selection is part of this process, it can slow down this evolution on the host and gives the parasite more time to get established.

Appendix

Here we show that our results hold even when survivorship and mating success are not negative exponential functions. We can replace Equation (3) with the following sigmoidal equation for survivorship:

$$ E(s) = \frac{\frac{\pi}{2}}{1 + e^{-\frac{n}{2}}} $$  \hspace{1cm} (10)

Also, we use the following sigmoidal equation for mating in place of Equation (6):

$$ E(m|s) = \frac{\frac{\pi}{2}}{1 + e^{\frac{n}{2}}} $$  \hspace{1cm} (11)

Fig. (6) depicts the strength of selection at the beginning of infection compare to Fig. (2). As we see, the overall effect of sexual selection on selection to block the parasite is the same, though the slope of the line lower.

Figs. (7)–(9) correspond to Figs. (3)–(5) in the main text, but using Equations (10) and (11), respectively. In each case, substituting sigmoid functions for negative exponentials reduces the slopes, but does not change the signs of the slopes or the fact that sexual selection can make blocking the parasite maladaptive.
Fig. 7. Strength of selection versus sexual selection intensity at three levels of focal parasite relative frequency. As the relative frequency of the focal parasite increases, more infections occur as a result of such parasite. Therefore, because of the sexual selection, it is increasingly adaptive for an infected individual to not resist the parasite blocking the immune response.

Fig. 8. Strength of selection versus sexual selection intensity for different levels of focal parasite’s virulence. When virulence of focal parasite increases, then there is a decrease in the influence of sexual selection on evolution of the host’s ability to block the parasite.

Fig. 9. Plot of the change in fitness with respect to the relative frequency of other parasites ($\nu wvI_0$) versus sexual selection intensity ($k$). As the focal parasite becomes common in the population, there is a less degree on reduction level of ($\nu wvI_0$). Therefore, more cases of coinfection occur, which help the other parasites by suppressing the host’s sickness behavior.
