Sex-specific predation risk and the evolution of sexual dimorphism in immunocompetence: a theoretical analysis

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Sexual dimorphism in immunocompetence, with males having lower immune function, is a prevalent pattern in nature. The main evolutionary explanation for this pattern is that males preferentially allocate resources away from immune function and towards reproductive effort to increase their competitiveness for limited females. However, the role of differential predation risk between the sexes has not been considered, despite predation risk being a major driver of life history strategies and male sexual traits often having associated predation costs. It is unclear whether increased predation risk should increase or decrease investment in immune function, as males have been shown to utilize both behavioural (e.g. decrease foraging activity) and/or life-history (e.g. decrease investment in sexual trait) defense strategies to manage predation risk. Here, we modelled optimal resource acquisition and allocation towards immune function under differential predation risk with multiple defense strategies. If males have limited defense strategies, increasing predation risk caused males to trade-off immune function for reproductive effort, leading to reduced immunocompetence. In contrast, if males can only decrease predation risk through reduction of reproductive effort (e.g. decrease colouration or calling rates), then increasing predation risk causes immune function to increase. If males can utilize multiple defense strategies and sexual selection is low, then males maintain a constant immune function as predation risk increases. Sexual selection robustly resulted in decreased immunocompetence. Overall, our results suggest that predation plays an important role in the evolution of sexual dimorphism in immunocompetence, but predicting its effect requires understanding the integrated defense strategies available.

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Abstract

Sexual dimorphism in immunocompetence, with males having lower immune function, is a prevalent pattern in nature. The main evolutionary explanation for this pattern is that males preferentially allocate resources away from immune function and towards reproductive effort to increase their competitiveness for limited females. However, the role of differential predation risk between the sexes has not been considered, despite predation risk being a major driver of life history strategies and male sexual traits often having associated predation costs. It is unclear whether increased predation risk should increase or decrease investment in immune function, as males have been shown to utilize both behavioural (e.g. decrease foraging activity) and/or life-history (e.g. decrease investment in sexual trait) defense strategies to manage predation risk. Here, we modelled optimal resource acquisition and allocation towards immune function under differential predation risk with multiple defense strategies. If males have limited defense strategies, increasing predation risk caused males to trade-off immune function for reproductive effort, leading to reduced immunocompetence. In contrast, if males can only decrease predation risk through reduction of reproductive effort (e.g. decrease colouration or calling rates), then increasing predation risk causes immune function to increase. If males can utilize multiple defense strategies and sexual selection is low, then males maintain a constant immune function as predation risk increases. Sexual selection robustly resulted in decreased immunocompetence. Overall, our results suggest that predation plays an important role in the evolution of sexual dimorphism in immunocompetence, but predicting its effect requires understanding the integrated defense strategies available.

Introduction

Sexual dimorphism in behaviour, physiology and morphology is common in nature. These differences evolve due to different selective pressures between the sexes, resulting from the interaction between ecology and sexual selection (Fairbairn et al. 2007; Hedrick & Temeles 1989; Shine 1989). In a variety of animal taxa (e.g. birds, mammals, insects), males have been found to have inferior immune function compared to their female counterparts (Nunn et al. 2009; Rolff 2002; Stoehr & Kokko 2006; Zuk & Stoehr 2010). The consequences of this lower immune function are that males often have higher disease prevalence rates (Markle & Fish 2014; Moore & Wilson 2002; Poulin 1996; Zuk & McKean 1996), and suffer more severe symptoms (Guerra-Silveira & Abad-Franch 2013). There is a growing body of research into the physiological mechanisms underlying these differences in mammals, partly driven by humans being sexual dimorphic in immunocompetence (reviewed in Klein & Roberts (2010) and Markle & Fish (2014)). However, the evolutionary mechanisms are poorly understood (Zuk 2009).

Because immune function is costly and strongly affects evolutionary fitness (Lochmiller & Deerenberg 2000; Norris & Evans 2000; Sandland & Minchella 2003), life history theory has been used to explain sexual dimorphism in immunocompetence. It has been proposed that the sexes allocate limiting resources differently between immune function and reproduction (Bacelar et al. 2011; Restif & Amos 2010; Rolff 2002; Stoehr & Kokko 2006). Why the sexes should allocate resources differently has been subject of some debate. Several mechanisms have been proposed: sexual selection (Stoehr & Kokko 2006), disease risk (Restif & Amos 2010), competition for resources (Bacelar et al. 2011) and mortality rates (Bacelar et al. 2011).

Surprisingly, the role of predation on the evolution of sexual dimorphism in immunocompetence has been relatively ignored, despite predation playing a major role in shaping life history traits (Cressler et al. 2010; Reznick & Endler 1982; Roff 1992), and in sexual selection theory (Andersson 1994; Breden & Stoner 1987). Predation costs are often assumed to counteract strong female preference and stabilize sexual trait expression in males (Breden & Stoner 1987). Males often have more exaggerated sexual traits that hinder locomotion (e.g. long tails, male weaponry; (Basolo & Alcaraz 2003; Emlen 2001; Møller 1989)) and increases conspicuousness

to predators (e.g. calling, courtship displays; (Magnhagen 1991; Stuart-Fox et al. 2003; Tuttle et al. 1982; Zuk & Kolluru 1998)). Furthermore, predation risk has been shown to affect both immune function (Joop & Rolff 2004; Rigby & Jokela 2000; Stoks et al. 2006; Zhang et al. 2003) and sexual trait expression (Fowler-Finn & Hebets 2011; Kotiaho et al. 1998; Magnhagen 1991; Ruell et al. 2013; Taylor et al. 2005). Consequently, differential predation risk between the sexes likely has evolutionary consequences on how sexes trade-off immune function and reproductive effort.

However, how predation risk may affect this trade-off is not readily obvious as the nature of the predation likely depends on the ecological characteristics of the sexual trait. In particular, the temporal phenotypic plasticity of the sexual trait will likely affect the defense strategies used to manage predation risk. For instance, morphological traits (e.g. long tails, bright colouration) are often temporally static and may not only increase predation risk during mating activities, but also during non-mating activities, like foraging. In this case, in order to manage predation risk males must decrease both trait expression and activity. For example, guppies have reduced colouration and activity levels when cohabiting with fish predators (Licht 1989; Millar et al. 2006; Ruell et al. 2013). In contrast, behavioural sexual traits (e.g. vocalizations, courtship displays, mate searching) are only expressed during mating activities and hence are temporally plastic (similar distinction in sexual traits is used in Jennions et al. (2001)). In this case, reducing trait expression is the main defense strategy for mitigating predation risk. For example, Tungara frogs reduce calling when bats are present (Tuttle et al. 1982). Reduction of trait expression may free up additional resources for immune function. Therefore, depending on the sexual trait expressed, males may be able to decrease predation risk by allocating less to the sexual trait and/or decreasing foraging activity. Consequently, the defense strategies used may affect whether immune function should increase or decrease.

Here, we investigate this predation hypothesis using a life history model to identity what conditions may favour the evolution of decreased immunocompetence in males. To this end, we explored the effect of predation risk when males can mitigate predation risk using behavioural (e.g. reduced foraging effort) and/or life history strategies (e.g. decreased trait expression). Furthermore, we modelled these situations under varying levels of sexual selection, the main mechanism assumed to underlie sexual dimorphism in immunocompetence (Stoehr & Kokko 2006).

Model Description

Model overview

We developed a life history model to explore the effect of predation risk on optimal immune function (Fig. 1). The model explores these strategies over the lifetime of an iteroparous animal. During each time period, an animal decides how much to forage (u ; $0 \leq u \leq 1$) and the proportion of reserves to allocate to immune function (a; $0 \leq a \leq 1$) and to reproduction (1-a). Mortality is caused by either disease or predation. Disease mortality is managed by investment in immune function. The model's environment is characterised by resource richness (e_{max}).

Immune function

After acquiring resources, an animal decides how much of those resources to allocate, a, to immune function. Disease susceptibility is managed solely through immune defense. Allocation of resources toward immune function, $z(a, u) = a\ u\ e_{max}$, lowers disease risks as described by:

$$D(z) = d_0\ e^{-Lz} \quad \text{(modified from Houston et al. (2007))} \qquad (1)$$

We assume a decelerating relationship between amount of resources invested and the probability of infection (Fig. S1). The shape of the relationship is controlled by parameter L with higher levels of L resulting in more efficient immune function.

Reproduction

The remaining resources, $r = (1\text{-}a)\ u\ e_{max}$, are all used for reproduction. We broke the mating process into two steps: the probability of attracting the mate and the payoff of a successful mating. We assumed that the payoff was constant (i.e. no sperm competition). However, the mating probability depended on the level of sexual selection, b. With no sexual selection ($b = 1$), probability of successful mating increases linearly with resource investment. When $b > 1$, each additional resource investment into attracting a mate results is associated with an accelerating probability of attracting a mate (Fig. S2):

$$O(r) = \Pr(\text{mating}) \text{ x Payoff} = \left(\frac{r^b}{e_{max}^b}\right) e_{max} \qquad (2)$$

Predation risk

Predation risk stemmed from two sources. The first source is background predation risk (p_b), which is the overall riskiness of the environment. This could be interpreted as predator density. Second, there was additional predation risk associated with trait expression (p_r). This type of predation can be viewed as the added predation risk male incur, such as having long tails that may hinder escape or loud calling rates that attract predators (Magnhagen 1991). Using this framework, we modelled five scenarios (see Table 1 for summary).

In the first scenario (*No defense*), predation risk cannot be modified. This scenario would be if males may have higher predation risk unrelated to a sexual trait. For simplicity, we assumed that there is no p_r and total predation is just the background predation risk:

$$P_{total} = p_b \quad (3)$$

Next, we modelled a scenario (*Acquisition*) in which an animal can manage predation risk by decreasing foraging activity, routinely used defense strategy (Lima & Dill 1990). Therefore, total predation is as follows (Fig. S3):

$$P_{total}(u) = p_b\, u^2 \quad (4)$$

Next, we modelled a scenario (*Allocation*) in which an animal can manage predation risk by decreasing the allocation of resources towards that sexual trait. This scenario reflects the case in which the sexual trait is temporally static. Therefore, total predation is as follows:

$$P_{total}(a) = p_r(1-a)^2 \quad (5)$$

The final scenarios assume that males can manage predation risk by decreasing foraging intensity and/or changing allocation to the sexual trait. The fourth scenario (*Behavioural*) assumes that the sexual trait is a behavioural trait that is only expressed during a proportion of the time (w_r).

$$P_{total}(u,a) = (1-w_r)p_b u^2 + w_r p_r[(1-a)u]^2 \quad (6)$$

The fifth scenario (*Morphological*) assumes the sexual trait is a morphological trait and expressed consistently.

$$P_{total}(u,a) = (1 - w_r)(p_b + p_r[(1-a)u]^2)u^2 + w_r p_r[(1-a)u]^2 \quad (7)$$

Fitness function

We found the optimal acquisition (u^*) and allocation (a^*) strategies under the varying scenarios using dynamic programming that maximizes lifetime reproductive success. V(t) is the reproductive value for an animal that follows the optimal time dependent strategy from *t* onwards:

$$V(t+1) = \underbrace{max}_{u,a}\ H(u,a;t) \quad (8)$$

where $H(u,a;t)$ is the reproductive value of the animal foraging at intensity *u* and allocates *a* resources for immune function.

At each time point, an animal survives until the next day with probability

$$S(u,a) = \big(1 - P_{total}(u,a)\big)\big(1 - D(z(u,a))\big) \quad (9)$$

Therefore, $H(u,a;t)$ is the combination of future reproductive success (aka, residual reproductive effort) plus current reproductive effort (Houston & McNamara 1999):

$$H(u,a;t) = S(u,a)[V(t+1) + O(r(u,a))] \quad (10)$$

The upper limit on lifespan, T, was chosen such that patterns on day 1 remain stable with additional increases in T. The reproductive value of an animal at time T is zero $[V(T) = 0]$. We numerically solved the dynamic equations 8 and 10 by finding the optimal values at T-1,T-2,…,1. We assume that population size and environment are constant between generations (Houston & McNamara 1999).

Results

Scenario 1: *No defense*

Increasing p_b causes optimal allocation to immune function (a*) to decrease, resulting in decreased immune function (Fig. 2). Since animals cannot mitigate this predation risk, background predation discounts future reproductive and causes animals to invest more in current reproduction 'live hard, die young'. Increasing sexual selection had a straightforward effect, decreasing allocation towards immune function independent of predation risk. As a consequence, disease mortality rates increases with predation risk (Fig. 3).

Scenario 2: *Acquisition*

When an animal can adjust foraging activity to manage predation risk, increasing p_b results in immune function consistently decreasing, but optimal reproductive effort (r*) may either increase or decrease (Fig. 2). Under low predation risk, the optimal strategy is to trade-off immune function for extra reproductive effort, but once predation risk increases too much, the optimal strategy is to manage predation risk by decreasing foraging activity. Again, mortality from disease increases with predation risk (Fig. 3). Increasing sexual selection delays the onset of reduced foraging activity.

Scenario 3: *Allocation*

If predation risk is associated with reproductive effort, then an animal can adjust reproductive effort to manage predation risk. Under no sexual selection, increasing p_r causes decrease allocation to reproductive effort and the extra resources can then be used for immune function (Fig. 2). Not surprisingly, mortality from disease decreases with increased predation risk (Fig. 3). Once sexual selection increases, the relationship between p_r and allocation to immune function changes from positive to negative, resulting in an optimal strategy similar to *No defense* scenario.

Scenario 4: *Behavioural*

Males often have multiple defense mechanisms available. The previous scenarios only included up to one defense mechanism. For this scenario, we assumed reproductive effort only increases predation risk during a short proportion of the time (w_r = 0.1; Table S1). As predation risk is constant during foraging (i.e unaffected by sexual trait), the optimal strategy is to maintain a fairly constant level of reproduction and immune function (Fig. 2). Sexual selection only had a small effect on the

relationship between p_r and immune function. Under no sexual selection, immune function increases with predation risk p_r, as reproductive effort slightly decreases and the extra resources are used for immune function. As sexual selection increased, the relationship changed to negative. As in the other scenarios, sexual selection overall strongly decreased investment in immune function (Fig. 2). As mortality from predation risk is only a small proportion of the risk, predation risk only slightly increased with p_r (Fig. 3).

Scenario 5: *Morphological*

Many sexual traits are morphological and can increase predation risk during both mating and foraging. In this scenario, as p_r increases, the optimal strategy is to maintain a constant level of immune function, but decrease reproductive effort. This is achieved by increasing allocation to immune function and decreasing acquisition levels (Fig. 2). Interestingly, mortality rates from disease were similar to *Behavioural* scenario (Fig. 3). Mortality from predation is much higher and follow similar mortality patterns as the *Acquisition* scenario (Fig. 3). Sexual selection had little effect on p_r pattern with immune function, but sexual selection did affect pattern between p_r and reproductive effort. This was due to sexual selection inhibiting when it is optimal to begin to trade-off foraging effort for decreased predation risk.

Effects of disease rate and immune function efficiency

Overall, background disease rate had little effect on overall patterns between predation risk and immune function. Higher disease environments and/or decreasing immune function efficiency increased the optimal immune function, but did not interact strongly with predation risk (see Fig. S4-S6).

Discussion

Suppressed immunocompetence in males is a prevalent pattern in nature (Nunn et al. 2009; Rolff 2002; Zuk 2009), suggesting that a common evolutionary mechanism(s) may be responsible. Previous research has shown that increased predation risk can reduce immune function (Rigby & Jokela 2000; Stoks et al. 2006; Zhang et al. 2003) and that predation risk can be a significant cost of sexual traits in males (Magnhagen 1991; Stuart-Fox et al. 2003; Taylor et al. 2005; Woods et al. 2007). Therefore, we proposed a novel hypothesis that higher predation rates on males may lead to the evolution of suppressed immunocompetence. Our results suggest that the evolution of suppressed immunocompetence depends on the nature of the predation risk. If additional predation risk is associated with being male and hence cannot be mitigated using life history trade-offs (*No defense* and *Acquisition* scenarios), then males should evolve suppressed immune function. However, if predation risk is associated with a sexual trait in which expression is affected by resource allocation (*Allocation*, *Behavioural* and *Morphological* scenarios), then our model predicts that higher predation risk will either increase or have no effect on immune function levels.

Differential predation independent of sexual traits

Our simplest model (*No defense*) assumed that predation risk was independent of sexual trait expression. With increasing predation risk, the model predicted that sexual traits should increase and immune function decrease. The results from the *No defense* scenario can be viewed as an example of 'live fast, die young' strategy in life history (Promislow & Harvey 1990). For instance, higher mortality risk is associated with larger litter size in female mammals and increasing reproductive investment to younger ages (Promislow & Harvey 1990). Similarly, female aphids increase fecundity when exposed to increased mortality risk (Barribeau et al. 2010). Not much else is known about how males alter reproductive investment with increased predation risk that is not associated with a sexual trait. A recent study with amphipods provides support that increased background predation risk may increase sexual trait expression. Exposing amphipods to predation cues increased expression of a morphological sexual trait (gnathopods: enlarged claw to grab females during copulation) in males, but no change in female gnathopod expression (Cothran et al. 2012). Furthermore, these high-predation males had enhanced sexual competitiveness compared to low-predation males.

Whether males and females differ systematically in background predation risk is, however, unclear. Sex differences in background predation rate could emerge from differences in the ecology of males and females. For instance, in several mammals, females utilize grouping behaviour, which is assumed to decrease predation risk (e.g. elephants (Dublin 1983), elk (Childress & Lung 2003), gazelles (Fitzgibbon 1990)). It is thought that for this reason cheetahs preferentially chase male gazelles, since males are typical on the periphery of social groups (Fitzgibbon 1990)). In a similar vein, males are often the dispersing sex, reducing their genetic relatedness in groups and hence the potential for altruistic defense behaviours that could reduce predation risk. These sex differences could lead to predators having different sex-specific predation rates.

We next expanded the *No defense* scenario to include behavioural defenses. Animals often utilize behavioural defenses to manage predation risk, such as adjusting foraging behaviour (*Acquisition* scenario). There are numerous examples of animal decreasing foraging activity in response to increased predator risk (reviewed in Lima & Dill (1990) and Lima (1998)). The *Acquisition* scenario models this case and found a decrease in immune function across all conditions. The *Acquisition* scenario differed from the *No defense* that eventually reproductive investment should decrease with increasing predation risk, depending on the strength of sexual selection.

The literature mostly supports the above model results that increased background predation risk should decrease immune function. The reduction in immune function with increasing predation risk has been shown in several species, though studies are mainly limited to phenotypic plasticity (e.g. (Mikolajewski et al. 2008; Navarro et al. 2004; Seiter 2011; Stoks et al. 2006; Zhang et al. 2003)). For instance, exposure to fish predator cues reduced phenoloxidase in larval damselflies (Stoks et al. 2006). It should be note, though less common, increased predation risk has been also shown to have no effect (Slos et al. 2009) or increase immune function (Joop & Rolff 2004). Little research has been done at the evolutionary scale (e.g. natural selection under different predation regimes).

Differential predation associated with sexual traits

In contrast to sex difference in background predation rates, much evidence supports differential predation risk associated with male sexual traits (Andersson 1994; Magnhagen 1991), as well as males modifying trait expression in response to predation rates (Lima 2009; Taylor et al. 2005; Tuttle et al. 1982). The *Allocation* scenario shows that, unlike acquisition, increased predation risk on a sexual trait could lead to the reverse pattern in which males have higher immune function, due to more resources allocated to immune function. However, the more realistic scenario includes both behavioural and morphological defenses (*Behavioural* and *Morphological*).

The *Behavioural* scenario assumes that additional predation risk is only associated during a proportion of the time. We modelled this scenario in which only 10% of the time an animal is engaging in expressing the sexual trait. Because of the fairly short duration, our model predicts additional predation risk should have the optimal investment in immune function and sex trait expression. Interesting, if the trait is morphological and expressed during the entire breeding period, additional predation risk associated with the trait decreased trait expression, but did not affect immune function. This mainly occurred through a combination of changes in acquisition and allocation strategies.

The evolution of guppy colouration patterns under predation risk is a well-studied example and an instructive comparison to our *Morphological* model results. When predatory fish are absent, male guppies evolve more elaborate colouration relative to males inhabiting streams with predatory fish (Endler 1980; Millar et al. 2006). This is likely due to fish predators preferentially attacking more colourful males (Godin & McDonough 2003). Furthermore, fish predators alter foraging activity in guppies and hence likely reduced overall resource intake (Fraser et al. 2004). Thus, male guppies utilize both life history and behavioural defense strategies for this morphological trait. Physiological measures of immune function have not been measured, though levels of parasitism do not differ consistently across predation environments (Gotanda et al. 2013; Perez-Jvostov et al. 2012), matching our model predictions.

Comparison with other theoretical models

Several other theoretical models have explored the evolution of sex differences in immune systems, with each exploring a different potential mechanism. The first

model was based on Bateman's principle: that is, reproductive success is often more variable for males than females. This is because male reproductive success is limited by female availability, while females are limited by physiological constraints (Rolff 2002; Stoehr & Kokko 2006). As a result, sacrificing immune function for additional investment in reproduction (e.g. higher sperm amounts, faster call rate) may allow males to outcompete other males and obtain a disproportionally higher amount of matings. Using a game theory approach, Stoehr & Kokko (2006) found that males should allocate less to immune function. Instead of using the game theory approach, we modelled sexual selection by assuming that the relationship between reproductive success and reproductive investment for males is an accelerating curve. We obtained similar results as Stoehr & Kokko (2006), with stronger sexual selection (higher *b*) causing lower immune function. We also found a predation-by-sexual selection interaction, in which the strength of sexual selection affects whether reproduction-related predation risk has a positive, neutral, or negative effect on immune function.

Sex differences in disease and mortality risks have also been modelled. Using a host-pathogen population dynamics approach, Restif & Amos (2010) found that increasing disease risk in males can lead to increased or decreased recovery rates from infections. Our model only found that increasing background disease prevalence increased disease risk. This difference likely stems from our model ignoring ecological feedbacks and linking the trade-off between immune and reproductive investment to death rates, whereas, Restif & Amos (2010) linked trade-off between recovery rate and reproductive investment to reductions in reproductive potential, not death.

In a third model, Bacelar et al. (2011) showed that increasing mortality risk in males consistently led to decreased immune function, using a similar modelling approach as Restif & Amos (2010). In this model, mortality is modelled as generic background mortality and hence cannot be mitigated by adjusting acquisition or allocation mechanisms (Bacelar et al. 2011). Therefore, it is not surprisingly that their model found that increasing mortality rates in just males resulted in the evolution of decreased immune function (similar to our background predation scenario). However, we showed that understanding when and if mortality risk can be mitigated by acquisition and/or allocation patterns can greatly change predictions.

Conclusion

437

438 In this study, we proposed the novel hypothesis that predation risk plays a role in the
439 evolution of sexual dimorphic immunocompetence. Empirical data supports that
440 predation risk can affect immune function and male sexual traits are often increase
441 predation risk. The key insight from our model is that suppressed immunocompetence
442 in males should only evolve when predation risk can not be managed through life
443 history strategies (i.e. allocation mechanisms). If males can re-allocate resources
444 away from sexual trait expression to immune function, males should either maintain
445 or increase immune function with increasing predation risk. Our models highlighted
446 the importance of classifying the type of sexual trait (behavioural vs. morphological)
447 in understanding the role of predation risk. Encouragingly, where our model overlaps
448 with other models on the evolution of sexual dimorphism, we find similar
449 conclusions, suggesting a general robustness of the conclusions drawn (Bacelar et al.
450 2011; Houston et al. 2007; Stoehr & Kokko 2006). Overall, predation risk may play a
451 complex and intriguing role in the evolution sexual dimorphism in
452 immunocompetence.

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461 ## Author Contributions

462 BGF developed model, analysed model results, and wrote-up manuscript
463 ES developed model, analysed model results, and revised drafts of manuscript

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Tables

Table 1. Summary of modelling scenarios.

For each model scenario, a short description, the predation equation used, and the effect of predation risk on immune function. '+'/'-' indicates that immune function increase/decreases with increasing predation risk.

b=1.5 is low sexual selection scenario. *b*=5 is high sexual selection scenario.

Model Id	Description	Predation Equation	Model Results	
			b =1.5	*b* = 5
1) No defense	Males cannot mitigate background predation	$P_{total} = p_b$	-	-
2) Acquisition	Males can mitigate predation risk by reducing foraging activity (no sexual trait predation risk)	$P_{total}(u) = p_b * u^2$	-	-
3) Allocation	Males can mitigate predation risk only by reducing sexual trait expression	$P_{total}(a) = p_b * (1-a)^2$	+	-
4) Behavioural	Males can use both defense strategies for a behavioural sexual trait	$P_{total}(u,a) = (1-w_r)p_b u^2 + w_r p_r * [(1-a) * u]^2$	+	-

5) Morphological	Males can use both defense strategies for a morphological sexual trait	$P_{total}(u,a) = (1-w_r)(p_b + p_r * [(1-a) * u]^2 * u^2 + w_r p_r * [(1-a) * u]^2$	+	-

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Figure Legends

Figure 1. Diagram of resource acquisition and allocation model.
Resource movement is indicated by black arrows. Dotted grey arrows indicate ecological parameters that can affect acquisition and allocation decisions. Solid grey arrows indicate variables that directly affect either survival or reproductive output. Letters (e.g. a, u, d_0) indicate variable name in the model equations.

Figure 2. Optimal responses as background predation risk increases for all scenarios.
Top row shows optimal reproductive effort, followed by immune, allocation and acquisition. Different columns are each scenarios (Table 1). Line types reflect different levels of sexual selection (b).

Figure 3. Mortality patterns for all five scenarios.
Top and bottom rows show mortality rates for predation and disease, respectively. Different columns are each scenarios (Table 1). Line types reflect different levels of sexual selection (b).

Figure 1(on next page)

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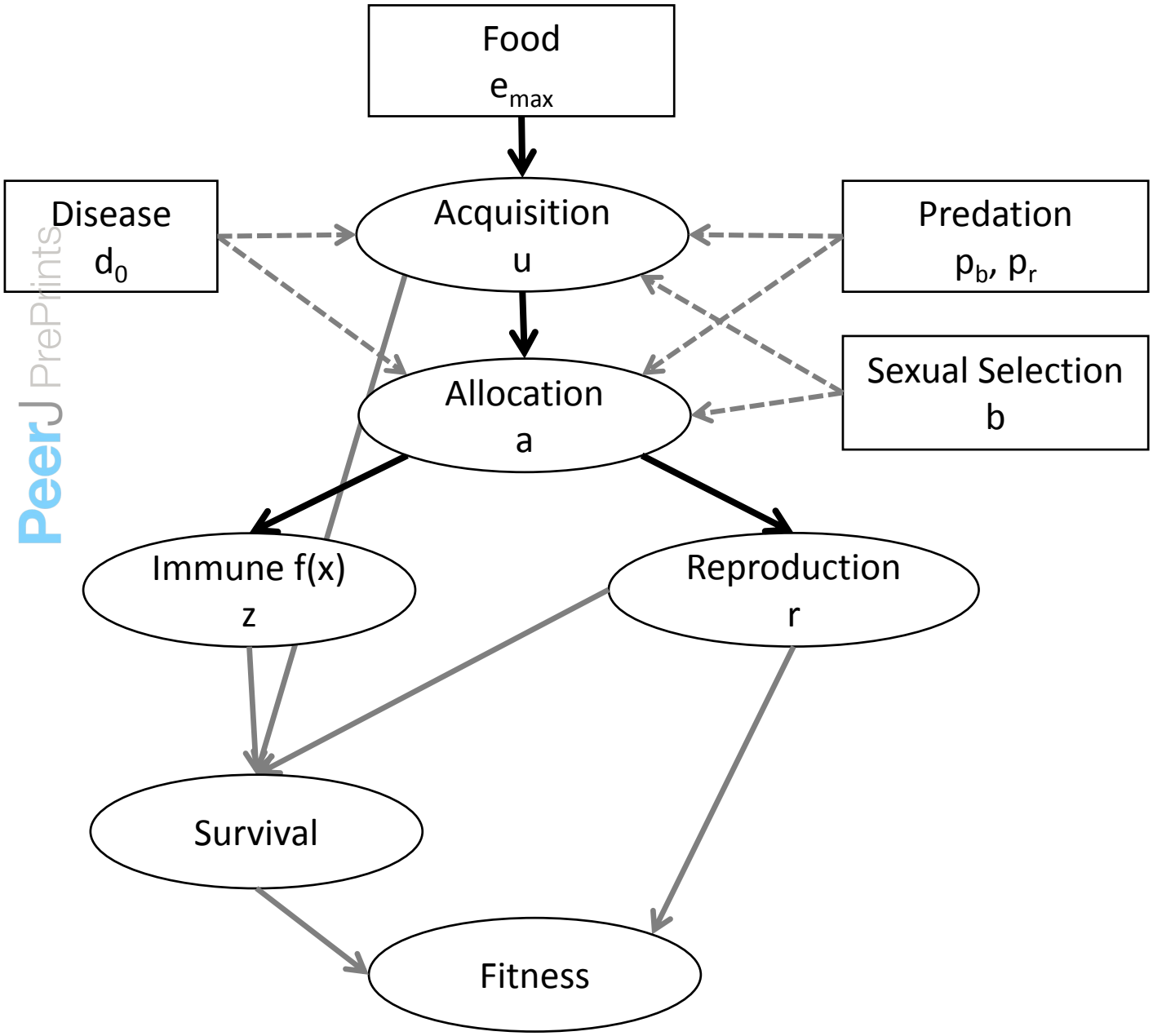

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Figure 2(on next page)

Figure 2. Optimal responses as background predation risk increases for all scenarios.

Top row shows optimal reproductive effort, followed by immune, allocation and acquisition. Different columns are each scenarios (Table 1). Line types reflect different levels of sexual selection (*b*).

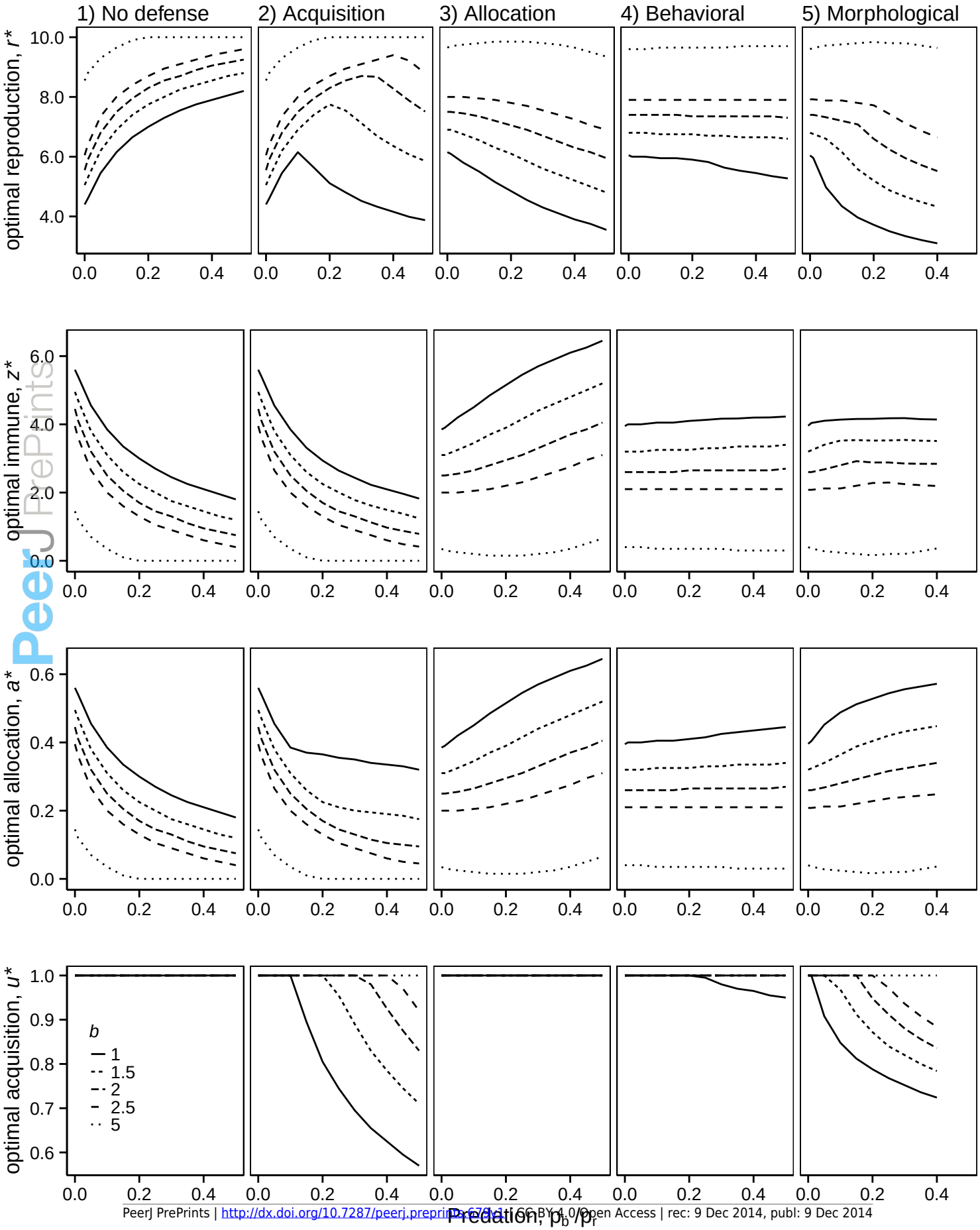
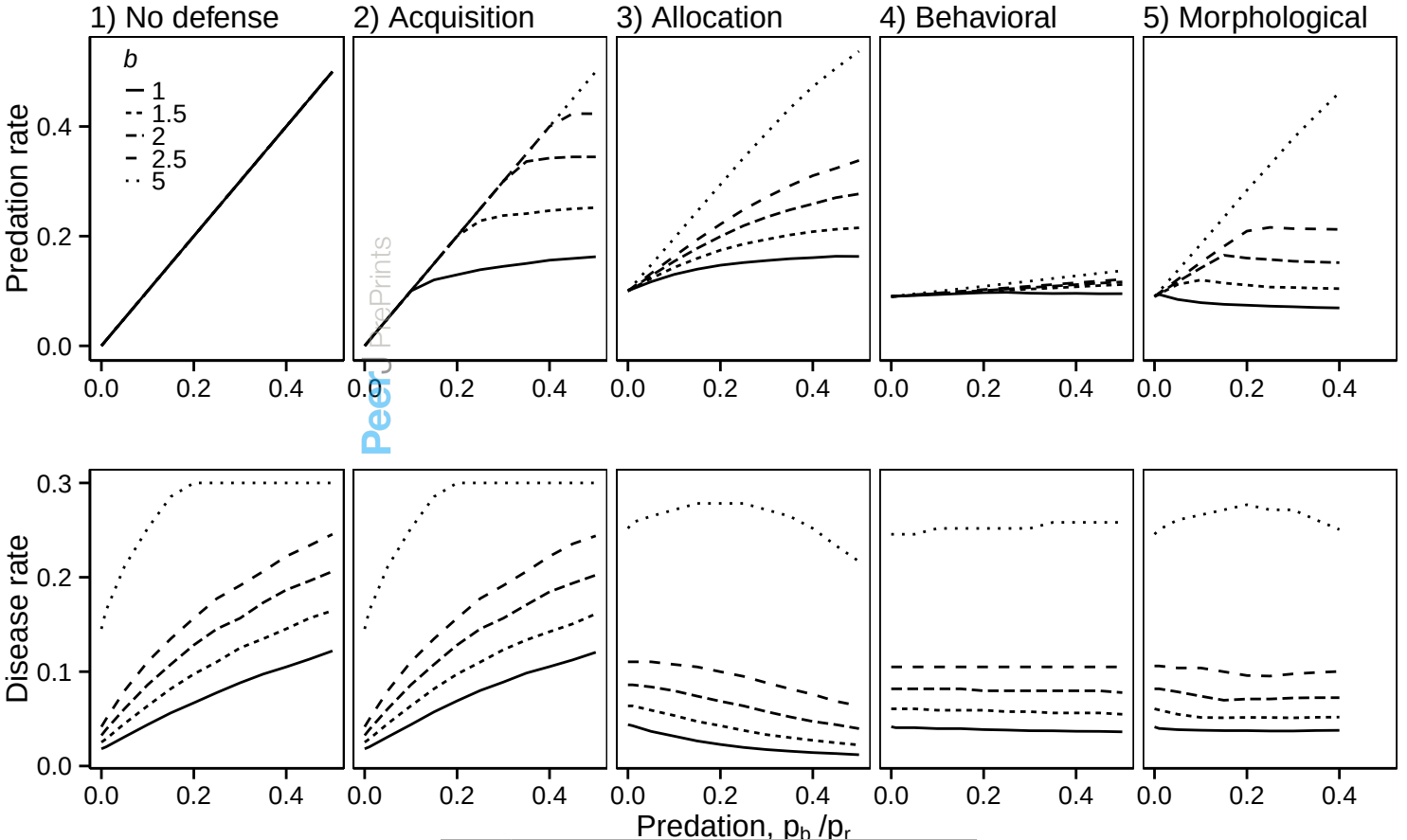


Figure 3(on next page)

Figure 3. Mortality patterns for all five scenarios.

Top and bottom rows show mortality rates for predation and disease, respectively. Different columns are each scenarios (Table 1). Line types reflect different levels of sexual selection (*b*).



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