Behavioral Ecology Advance Access published June 1, 2006

Behavioral Ecology
doi:10.1093/beheco/ark018

Sexual dimorphism in immunocompetence: what does life-history theory predict?

Andrew M. Stoehr[a] and Hanna Kokko[b]
[a]Department of Biology, University of California, Riverside, CA 92521, USA, and [b]Laboratory of Ecological and Evolutionary Dynamics, Department of Biological and Environmental Science, University of Helsinki, Finland

Sexual dimorphism in immunocompetence, usually in the direction of inferior male immunocompetence, has historically been explained as the result of proximate physiological mechanisms such as the immunosuppressive effects of the male hormone testosterone. More recently, it has been argued that this pattern is best understood as a result of resource-based trade-offs between male mating effort and immune defense, a trade-off that females do not make. The central prediction of this hypothesis is that as the strength of sexual selection on males increases, the magnitude of the sex differences in immunocompetence will increase. Two implicit assumptions of this argument are that 1) longevity is of more importance for female than for male fitness and 2) that the primary benefit of immunocompetence is increased longevity. However, both of these assumptions may not be as broadly applicable as has been argued. We have modeled the optimal allocation to immunocompetence for males and females without making these assumptions. We find that the optimal allocation to immune defense for males decreases as the strength of sexual selection increases, as predicted. However, males may still invest more, relative to females, into immunocompetence if the impact of parasites on condition differs for the sexes and/or if the relationship between condition and reproduction differs for the sexes. We argue that these previously overlooked assumptions may be critical for predicting sex-specific patterns of immunocompetence. *Key words:* ecological immunology, immune defense, life-history theory, sexual selection. *[Behav Ecol]*

Immune defense is an important component of fitness. The perspective of "ecological immunity" states that the evolution of immune defenses can only be understood in the proper ecological and behavioral contexts of the organisms under study (Sheldon and Verhulst 1996; Zuk and Stoehr 2002; Schmid-Hempel 2003; Rolff and Siva-Jothy 2004). One currently active subdiscipline of ecological immunology is interest in sex differences in immunocompetence (Zuk 1990; Zuk and McKean 1996; Rolff 2002; Zuk and Stoehr 2002). Historically, explanations of this dimorphism have relied on proximate mechanisms, particularly the immunosuppressive effects of testosterone (Zuk and McKean 1996). More recent attempts have taken a life-history approach, usually invoking trade-offs between costly immune defenses and other aspects of host reproduction (Zuk 1990; Rolff 2002; Zuk and Stoehr 2002). These hypotheses argue that whatever the proximate reasons (e.g., hormones) for inferior male immunocompetence, ultimately it is the result of the different ways the sexes maximize fitness.

The basic hypothesis, which we refer to as the "susceptible male hypothesis," is best summarized by Rolff (2002): "If, as seems likely . . ., immune function is costly and females invest more in longevity than males, then females should invest relatively more in immune function in order to increase their survival probability." In other words, males stand to gain more from sacrificing immune defense than do females, if allocation to mating effort brings higher fitness gains than longevity. Females are not expected to make such a sacrifice because, it is argued, survival (i.e., longevity) is of more importance to female fitness than to male fitness. The central prediction of the susceptible male hypothesis is that as the strength of sexual selection increases, the magnitude of sexual dimorphism in immunocompetence, in the direction of inferior male immunocompetence (except when sexual selection on females is greater), should increase (Zuk 1990; Rolff 2002; Zuk and Stoehr 2002).

Here, we address 2 assumptions implicit in the susceptible male hypothesis. The first assumption is that longevity is of more importance for female than for male fitness. The second, and perhaps more important, assumption is that the primary benefit of immune defense (or, conversely, the greatest cost of parasites) is survival. With these assumptions in place, the hypothesis can be represented graphically (Figure 1a). In this form, the hypothesis clearly predicts that the sex that invests the most in survival—typically females—will be the sex with superior immune defenses.

The general validity of these assumptions is unclear. Studies across several taxa suggest that although longevity may not be particularly important to male fitness in some polygynous male mammals, it is "a major cause of variation" in the fitness of both male and female insects and many birds (Clutton-Brock 1988). In a survey of several studies of birds, longevity accounted for 29–86% of the variation in lifetime reproductive success of males and for 29–81% for females, being on average 59% in both sexes (Newton 1989). In one formulation of the hypothesis (Rolff 2002), this sex-dependent importance of longevity is attributed to the classic work of Bateman (1948) on *Drosophila*. However, Bateman (1948) did not show that longevity was more important for female than for male fitness and in fact took measures to insure that longevity was not a factor: "bottles in which any parent had died during the laying period were discarded" (Bateman 1948, p. 360).

Furthermore, parasites can have many and various nonlethal effects on host fitness (Loye and Zuk 1991; Grenfell and Dobson 1995; Clayton and Moore 1997). Parasites reduce the overall health, or "condition," of their hosts, and many traits, including but not limited to survival, are condition dependent. These effects of parasites may influence different

Address correspondence to A.M. Stoehr. E-mail: andrew.stoehr@email.ucr.edu.

Received 18 October 2005; revised 25 April 2006; accepted 3 May 2006.

© The Author 2006. Published by Oxford University Press on behalf of the International Society for Behavioral Ecology. All rights reserved.
For permissions, please e-mail: journals.permissions@oxfordjournals.org

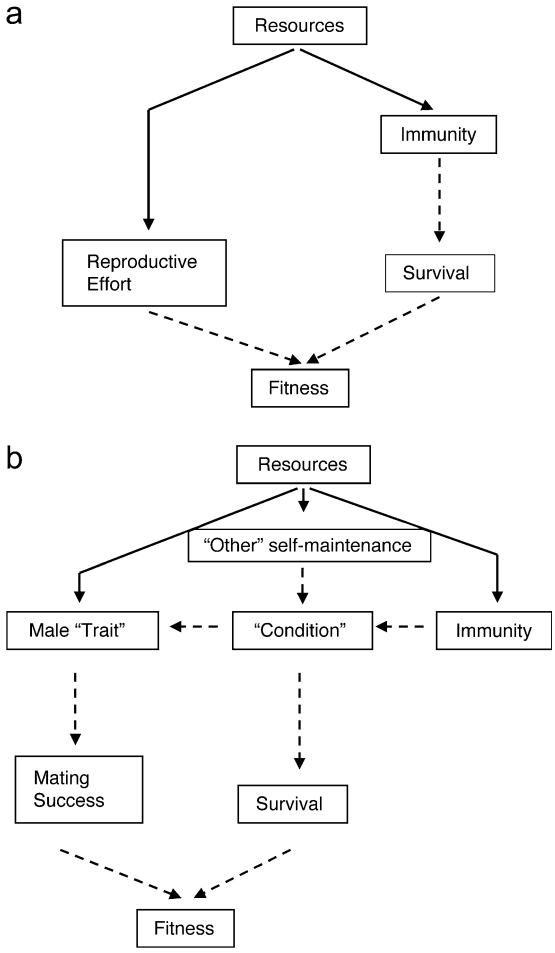

Figure 1
(a) Resource allocation to immune defense and reproductive effort, assuming the benefits of immune defense only affect survival. In this model, the sex that invests the most in survival must necessarily invest more in immune defense. (b) Resource allocation when immune defense benefits both survival and mating success due to the beneficial effect of immunity (one form of self-maintenance) on condition, which in turn increases both survival and a male's sexually selected "trait." Condition is also a function of "other," that is, nonimmunity-based, self-maintenance. Shown is the male case; in the female case, reproductive effort is fecundity. Solid arrows represent resource allocations, and dashed arrows are causal relationships. See text for details.

components of fitness, such as male mating success and female fecundity. The second assumption therefore implicitly asserts that the nonlethal effects of parasites are the same for each sex, for the same investment in immune defense. Given that immune defense may be costly in terms of mating success and fecundity but that parasites may also negatively affect male mating success or female fecundity, immunity is a double-edged sword for both sexes, not just males. It is also worth noting that the general notion that longevity selects for increased immunocompetence has recently been challenged on theoretical grounds (van Boven and Weissing 2004).

Here, we present a model of the susceptible male hypothesis, which has up to now only been articulated as a verbal model (Zuk 1990; Rolff 2002; Zuk and Stoehr 2002). More specifically, we asked whether the main prediction of the hypothesis—inferior male, relative to female, immunocompetence when sexual selection is strong—is likely to apply when we allow that 1) survival is important to the fitness of both sexes (not just females) and that 2) immunocompetence will have benefits beyond increased survival due to effects on condition. Our model of optimal immunocompetence includes the essence of the argument: female fitness is a product of fecundity and longevity, whereas male fitness is a product of mating success and longevity. We have adopted an optimality approach to the problem because the susceptible male hypothesis, as it is usually articulated, is based on the sex-specific costs and benefits of immune defense. An important limitation of such an approach is that it ignores genetic correlations between the sexes; we will deal with the likely consequences of this assumption in the Discussion.

THE MODEL

Deriving a relationship between immunocompetence and fitness can be fairly uncomplicated if immunocompetence influences survival only (Figure 1a). However, it becomes necessarily complicated if individual condition is determined by several allocation decisions and if condition has consequences for both survival and reproduction. This is particularly true for males with condition-dependent sexual displays. In this case, immunocompetence is clearly one aspect that determines an individual's condition. However, increasing investment in immunocompetence can diminish other forms of self-maintenance, and the net effect on condition depends on the parasites that are actually encountered. Moreover, males' direct investment in sexual traits can trade-off with his investment in condition (Figure 1b).

Our goal is not to explore every possible functional relationship of such trade-offs. Instead, we ask if there are cases in which simple verbal relationships no longer apply. We begin by supposing that an individual has a limited pool of resources to be allocated to reproductive effort, immunocompetence (one form of self-maintenance), and "other" forms of self-maintenance. The latter 2 allocations together determine an individual's condition. Condition, in turn, has positive effects on both reproductive effort and survival. Fitness is a function of both reproductive effort and survival, and so allocation to immunocompetence manifests its positive effects on fitness through its effect on condition (Figure 1b).

Resource allocation is modeled as the sum of the relative proportions allocated to reproductive effort (p_r), immune defense (p_i), and other necessary forms of self-maintenance (p_s): $p_r + p_i + p_s = 1$. Condition, C, is a function of the direct allocation of resources to self-maintenance as well as of allocation to immune defense:

$$C = p_s^{\gamma} p_i^{\beta}. \quad (1)$$

Exact shapes of trade-offs are hardly ever known in nature. We have chosen a functional form that captures some biologically essential features as well as allowing for nonlinear effects. β and γ describe the nonlinearity of the relationship between allocation and condition: condition cannot improve without any boundaries even if one component of condition receives ever-increasing allocation. The multiplicative form of Equation 1 ensures that C lies between 0 and 1 and that $C = 0$ if an individual neglects one aspect of condition completely. The parameter β describes the sensitivity of condition to the allocation of resources to immunocompetence and we call it "parasitic impact." Parasitic impact can include effects of virulence, but we use our term in a much broader sense: it includes any factor that influences the relationship between immunocompetence and condition. For example, an individual's behavior can be sex specific and hence contribute to its exposure to parasites. Thus, β is one of the parameters relevant to the assumption that the (often) nonlethal effects of parasites on fitness are the same for the sexes.

We measure longevity of both males and females as annual survival S and assume it is a linear function of condition, $S = C$ for both sexes. Our model formulation ensures that both condition and survival always fall between 0 and 1. The linear relationship can be justified because condition is a nonlinear function of the resource allocation decisions and can therefore take many shapes; we are hence essentially scaling condition in such a way that it is measured in terms of its effects on survival. However, to ensure that our results are robust when relaxing this assumption, we also considered a curvilinear scaling, $S = C^{0.5}$, and find little qualitative difference in the outcome (see Results).

The way condition and reproductive effort influence reproductive output differs between the sexes. For females, reproduction equals fecundity, F, which is a function of direct allocation to reproductive effort and of condition. For similar reasons as above, fecundity obeys a multiplicative form:

$$F = p_r^{\alpha} C^{\phi}, \tag{2}$$

where the parameters α and ϕ describe nonlinearities as above. We refer to the parameter ϕ as the "condition dependence of reproduction." For males, reproductive effort manifests as a male trait, T, which in turn determines his mating success, M. This trait refers to secondary sexual traits such as bright plumage or elaborate song but also to other forms of mating effort, such as mate searching. The male trait, T, is a function of direct allocation and condition:

$$T = p_r^{\alpha} C^{\phi}. \tag{3}$$

Unlike the female case, male reproductive success depends on a focal male's trait T_m relative to the value of the trait for the rest of the population, T_{pop}:

$$M = \left(\frac{T_m}{T_{\text{pop}}}\right)^{\delta}. \tag{4}$$

The parameter δ determines how male mating success increases with relative trait value; when $\delta > 1$, marginal gains from increases in relative trait value are increasing, whereas $\delta = 0$ describes complete absence of sexual selection. Thus, δ is a measure of the strength of sexual selection on T. The structure of our model, particularly Equation 3, allows that survival and reproduction may be inversely related despite positive effects of condition on reproduction.

Male fitness equals his lifetime reproductive success, $\text{LRS}_{\text{M}} = M/(1 - S)$, and likewise for females, $\text{LRS}_{\text{F}} = F/(1 - S)$. Although the proper choice of a fitness measure depends on the type of density dependence that operates in a population (Mylius and Diekmann 1995; Brommer et al. 2002), a different fitness measure is unlikely to change our conclusions (see Discussion), nor is the fact that male and female fitness must be equal on average (Kokko and Jennions 2003; Arnqvist 2004). We can ignore this latter fact: we do not assume that males can influence female allocations, hence multiplying male fitness to equal average female fitness would treat every male equally, only changing the scale of male fitness measurements without changing the optimal allocation.

Our goal is to determine the allocation of resources to immune defense that maximizes lifetime reproductive success. Solutions were found numerically, which was straightforward for females. Because male mating success is frequency dependent, finding the pattern of allocation for males requires an evolutionarily stable strategy (ESS) approach. To find the ESS, we start with an arbitrary allocation strategy for the population and determine the best response (i.e., allocation strategy) to that population strategy. If the response strategy is different, it becomes the new population strategy, and we repeat the process until the best response to the population is the population strategy itself.

RESULTS

The interactions between sexual selection, the condition dependence of reproduction, and parasitic impact determine both the magnitude and direction of sex differences in allocation to immune defense. When we assume that both parasitic impact and reproduction condition dependence are the same for the sexes (i.e., $\beta_{\text{males}} = \beta_{\text{females}}$, $\phi_{\text{males}} = \phi_{\text{females}}$), then in general males invest more in immunocompetence than do females when sexual selection is weak but less than females when sexual selection is strong (Figure 2a,b heavy line). However, if the impact of parasites and/or the condition dependence of reproduction are the same for the sexes and of sufficient magnitude, sex differences in immune defenses are eliminated and insensitive to increases in the strength of sexual selection (not shown). When parasites are particularly bad for male fitness, they do not sacrifice immune defense even in the face of stronger sexual selection on the "trait."

If parasitic impact and condition dependence of reproduction differ between the sexes, conditions exist under which male allocation to immunocompetence may be higher or lower than female allocation over the entire range of sexual selection considered. For example, assuming equal and linear condition dependence of reproduction (e.g., $\phi_{\text{male}} = 1$, $\phi_{\text{female}} = 1$) for both sexes, a relatively small difference in the impact of parasites on condition (e.g., $\beta_{\text{females}} = 1.0$, $\beta_{\text{males}} = 1.2$) is sufficient to change the direction of sex differences in immunocompetence from female superior to male superior even when sexual selection is strong (Figure 2a). Greater sex difference in parasitic impact (e.g., $\beta_{\text{females}} = 1.0$, $\beta_{\text{males}} = 2.0$) substantially magnifies the sex difference in immune defense (Figure 2a). Likewise, similar changes in parasitic impact for females will have a corresponding but opposing effect on the direction of sex differences in immune defense.

The effects of changes in the condition dependence of reproduction (ϕ) are qualitatively the same as for the impact of parasites but quantitatively of lower magnitude (Figure 2b). Thus, when considered in tandem, ϕ and β may work to increase or decrease sexual dimorphism in immunocompetence depending on the relative values of each for the different sexes. However, because sex differences in parasitic impact have a greater effect on immunocompetence than sex differences in condition dependence of reproduction (because the impact of parasites is "upstream" of the relationship between condition and reproduction, Figure 1), relatively small differences in the former can offset larger differences in the latter. The results are qualitatively the same when we assume that survival is a diminishing function of condition (i.e., $S = C^{0.5}$), but the effect is to shift all curves upward, further into the area of greater male immunocompetence.

DISCUSSION

Some parameter combinations confirmed the verbal ideas of the susceptible male hypothesis. We predict that strong sexual selection decreases optimal allocation to male immunocompetence. Because sexual selection only influences optimal immunocompetence in males, the net result is that sexually selected males compromise their immunocompetence relative to females. An exception occurs when parasites very strongly impact condition: allocation to immune defense is then insensitive to sexual selection. Our results also suggest that

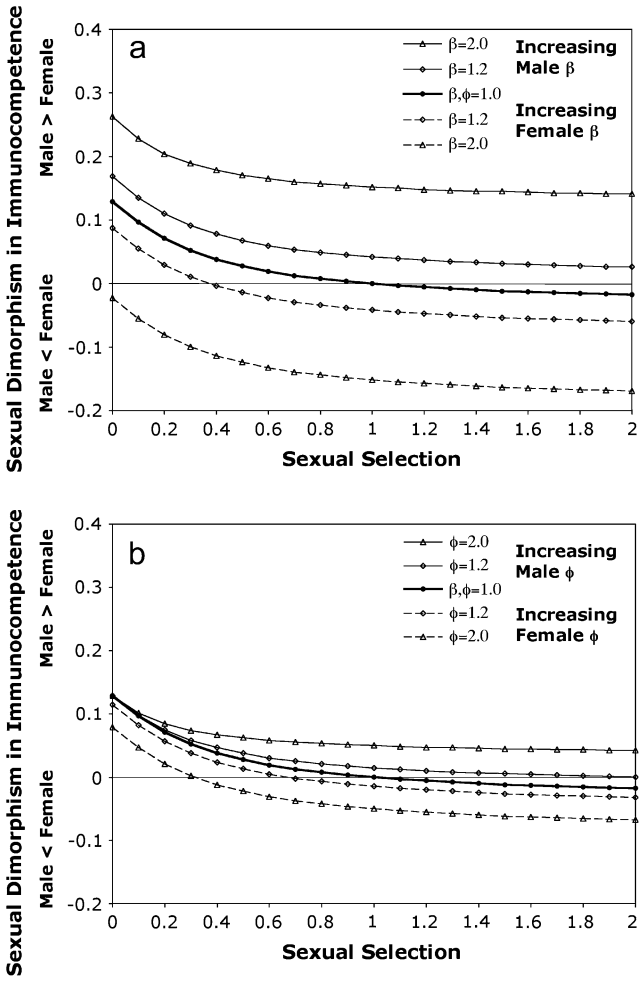

Figure 2
Sexual dimorphism in immunocompetence as a function of the strength of sexual selection, the impact of parasites on condition (β), and the condition dependence of reproduction (ϕ). Sexual dimorphism in immunocompetence is the difference (male – female) in optimal allocation to immunocompetence: positive values indicate greater male immunocompetence and negative values greater female immunocompetence. For both sexes, $\alpha = 0.5$ and $\gamma = 0.5$. (a) Sexual dimorphism in immunocompetence when $\phi_{males} = \phi_{females} = 1.0$ and $\beta_{males} \neq \beta_{females}$. Heavy line shows the case where $\phi_{males} = \phi_{females} = \beta_{males} = \beta_{females} = 1.0$ for reference. Solid lines show effects of increasing β_{males} (with $\beta_{females} = 1.0$) and dashed lines increasing $\beta_{females}$ (with $\beta_{males} = 1.0$). (b) SDI when $\phi_{males} \neq \phi_{females}$; $\beta_{males} = \beta_{females} = 1.0$; lines as in (a).

weak or absent sexual selection may make males invest substantially more in immune defense than females do. Complete or near complete absence of sexual selection on males is probably unlikely in most systems, however.

Hence, our second set of results is more important: when we relaxed the assumption that the impact of parasites on condition and the condition dependence of reproduction are the same for the sexes, our ability to predict both the direction and magnitude of sex differences in immunocompetence was substantially compromised. If condition has little effect on male mating success (very low ϕ) and sexual selection is strong, we might expect males to sacrifice immune defense (and thus survival) to a great degree, leading to a greater female bias in the direction of sexual dimorphism in immunocompetence if condition is still important for female fecundity. This raises some interesting questions, such as whether sexual selection can become so strong that it can threaten the persistence of populations (Kokko and Brooks 2003; Le Galliard et al. 2005). However, most male ornaments are condition dependent (Johnstone 1995), and our model suggests that when parasites have a relatively greater impact on male, rather than female, condition and/or a male's sexually selected trait is more condition dependent than is female fecundity, male investment in immunocompetence exceeds that of females, even in the face of the trade-off imposed by sexual selection. Thus, our simple model of the susceptible male hypothesis shows that the assumptions implicit in the verbal model are very important for its main prediction. Relatively inferior male immunocompetence is not an inevitable consequence of strong sexual selection on males.

Whether our conclusion is widely applicable depends on how often and to what degree the impacts of parasites on the fitness of the sexes differ, due to sex-specific effects on condition (β), sex-specific effects of condition on reproductive effort (ϕ), or both. The inherent differences between males and females, particularly in the different forms that reproductive effort may take, may make it difficult to measure these parameters accurately. It is precisely these differences, however, that suggest that the assumption of largely similar effects of parasites on males and females is probably rarely, if ever, valid. For example, differences in behavior may affect exposure to parasites (e.g., Tinsley 1989; Zuk and Kolluru 1998; Reimchen and Nosil 2001), and similar infections may have sex-specific effects on condition (e.g., Blanco et al. 2001; Tseng 2003).

Sexual differences in the components of the pathways leading to immunocompetence can affect sexual dimorphism in immunocompetence in ways that may not be obvious from a more superficial examination of the system, for example, only considering the strength of sexual selection. This could be viewed as somewhat discouraging from the perspective of predicting sex differences in immune defense. However, the primary value of this result is that it should encourage a proper investigation of the causes behind patterns of immunocompetence, particularly when they do not conform to the expectations of the susceptible male hypothesis. Indeed, in both invertebrates and vertebrates, where multiple measures of immunocompetence were used and/or immunocompetence was assessed under varying conditions (e.g., diet quality, reproductive history), the direction and/or presence of sex differences in immunocompetence depended on these different factors (Klein 2000; Adamo et al. 2001; Hosken 2001; Fedorka et al. 2004; Zuk et al. 2004; McGraw and Ardia 2005; McKean and Nunney 2005; Rolff et al. 2005). Our results suggest that differences in the strength of sexual selection are not solely responsible for these varying patterns: sex differences in the relationships between immune defense and condition or condition and reproduction can play a large role.

Some limitations of the model

We have made some simplifying assumptions in the construction of the model. First, we have modeled immunocompetence simply as something that has a positive effect on condition. Thus, we do not consider host–parasite coevolution, which may affect the evolution of host resistance (Gandon et al. 2002; Day and Burns 2003). We also ignore that immune defense responses are complex and comprised numerous components, for example, both humoral and cell-mediated components, innate and acquired resistance, and constitutive and inducible defenses (Gupta 1991; Roitt 1998). If the per-unit resource effect of allocation (i.e., costs and/or benefits) is different for different components of defense, the optimal allocation for each arm of the immune system may differ

(e.g., Boots and Bowers 2004). The direction and/or magnitude of sex differences in immunocompetence could, therefore, be different for each component of defense (e.g., Fedorka et al. 2004). Our simplified approach also emphasizes the maintenance costs of immunocompetence, not the deployment costs (i.e., the costs associated with mounting a defense). Sex-specific deployment costs might be expected to result in plasticity in the direction of sex differences in immune defense, depending on factors such as the diet quality or current sex ratio, which indeed has been found (McKean and Nunney 2005).

We have also modeled condition in a form that ignores the possibility that individuals may differ in their initial pools of resources (another definition of condition—see Hunt et al. 2004) and that condition itself may have feedback effects on immunity. A state-dependent modeling approach would be appropriate in this case. However, addressing any of the model limitations is likely to strengthen rather than change our general conclusion: due to the complex nature of the relationship between immunocompetence and fitness, attaining sex-specific predictions of immunocompetence is harder than simple verbal models predict.

In addition, we have also ignored the underlying genetic architecture of immunity. It has been convincingly shown that there may be genetic correlations between the sexes for immunocompetence (Rolff et al. 2005) or between different components of defense (Cotter et al. 2004; Lambrechts et al. 2004; Rolff et al. 2005). This will prevent the sexes from reaching their optima, possibly for long stretches of evolutionary time (Rhen 2000). However, for any correlations less than 1.0, at equilibrium we expect sexual dimorphism (Lande 1980; Zeng 1988), and genetic correlations are not expected to change the direction of this dimorphism. Thus, adding this feature would only change the magnitude of dimorphism, not our main message, which is to highlight the possibility that males can be sometimes selected to invest more in immunocompetence than females, despite the effects of sexual selection. The same basic conclusion is also likely to be unchanged had we chosen a different measure of fitness. That is, considering additional measures of fitness would increase, not decrease, the diversity of possible patterns emerging from the model.

We thank Daphne Fairbairn, David Reznick, Marlene Zuk, and 3 anonymous reviewers for comments that improved the quality of the manuscript. Support was provided by the University of California, Riverside and a Department of Education Graduate Assistance in Areas of National Need Fellowship (to A.M.S.) and by the Academy of Finland (to H.K.).

REFERENCES

Adamo SA, Jensen M, Younger M. 2001. Changes in lifetime immunocompetence in male and female *Gryllus texensis* (formerly *G. integer*): trade-offs between immunity and reproduction. Anim Behav 62:417–25.

Arnqvist G. 2004. Sexual conflict and sexual selection: lost in the chase. Evolution 58:1383–8.

Bateman AJ. 1948. Intra-sexual selection in *Drosophila*. Heredity 2: 349–68.

Blanco G, De La Puente J, Corroto M, Baz A, Colas J. 2001. Condition-dependent immune defence in the Magpie: how important is ectoparasitism? Biol J Linn Soc 72:279–86.

Boots M, Bowers RG. 2004. The evolution of resistance through costly acquired immunity. Proc R Soc Lond B Biol Sci 271:715–23.

Brommer JE, Merilä J, Kokko H. 2002. Reproductive timing and individual fitness. Ecol Lett 5:802–10.

Clayton DH, Moore J, editors. 1997. Host-parasite evolution: general principles and avian models. New York: Oxford University Press.

Clutton-Brock TH. 1988. Reproductive success. In: Clutton-Brock TH, editor. Reproductive success: studies of individual variation in contrasting breeding systems. Chicago, IL: University of Chicago Press. p 472–85.

Cotter SC, Kruuk LEB, Wilson K. 2004. Costs of resistance: genetic correlations and potential trade-offs in an insect immune system. J Evol Biol 17:421–9.

Day T, Burns JG. 2003. A consideration of patterns of virulence arising from host-parasite coevolution. Evolution 57:671–6.

Fedorka KM, Zuk M, Mousseau TA. 2004. Immune suppression and the cost of reproduction in the ground cricket, *Allonemobius socius*. Evolution 58:2478–85.

Gandon S, van Baalen M, Jansen VAA. 2002. The evolution of parasite virulence, superinfection, and host resistance. Am Nat 159:658–69.

Grenfell BT, Dobson AP, editors. 1995. Ecology of infectious diseases in natural populations. Cambridge, UK: Cambridge University Press.

Gupta AP, editor. 1991. Immunology of insects and other arthropods. Boca Raton, FL: CRC Press.

Hosken DJ. 2001. Sex and death: microevolutionary trade-offs between reproductive and immune investment in dung flies. Curr Biol 11:R379–80.

Hunt J, Bussiere LF, Jennions MD, Brooks R. 2004. What is genetic quality? Trends Ecol Evol 19:329–33.

Johnstone RA. 1995. Sexual selection, honest advertisement and the handicap principle: reviewing the evidence. Biol Rev 70:1–65.

Klein SL. 2000. Hormones and mating system affect sex and species differences in immune function among vertebrates. Behav Processes 51:149–66.

Kokko H, Brooks R. 2003. Sexy to die for? Sexual selection and the risk of extinction. Ann Zool Fenn 40:207–19.

Kokko H, Jennions MD. 2003. It takes two to tango. Trends Ecol Evol 18:103–4.

Lambrechts L, Vulule JM, Koella JC. 2004. Genetic correlation between melanization and antibacterial immune responses in a natural population of the malaria vector *Anopheles gambiae*. Evolution 58:2377–81.

Lande R. 1980. Sexual dimorphism, sexual selection, and adaptation in polygenic characters. Evolution 34:292–305.

Le Galliard J-F, Fitze PS, Ferrière R, Clobert J. 2005. Sex ratio bias, male aggression, and population collapse in lizards. Proc Natl Acad Sci USA 102:18231–6.

Loye JE, Zuk M, editors. 1991. Bird-parasite interactions: ecology, evolution, and behaviour. New York: Oxford University Press.

McGraw KJ, Ardia DR. 2005. Sex differences in carotenoid status and immune performance in zebra finches. Evol Ecol Res 7:251–62.

McKean KA, Nunney L. 2005. Bateman's principle and immunity: phenotypically plastic reproductive strategies predict changes in immunological sex differences. Evolution 59:1510–7.

Mylius SD, Diekmann O. 1995. On evolutionarily stable life histories, optimization and the need to be specific about density dependence. Oikos 74:218–24.

Newton I. 1989. Synthesis. In: Newton I, editor. Lifetime reproduction in birds. San Diego, CA: Academic Press. p 441–69.

Reimchen TE, Nosil P. 2001. Ecological causes of sex-biased parasitism in threespine stickleback. Biol J Linn Soc 73:51–63.

Rhen, T. 2000. Sex-limited mutations and the evolution of sexual dimorphism. Evolution 54:37–43.

Roitt I, Brostoff J, Male D, editors. 1998. Immunology. London: Mosby.

Rolff J. 2002. Bateman's principle and immunity. Proc R Soc Lond B Biol Sci 269:867–72.

Rolff J, Armitage SAO, Coltman DW. 2005. Genetic constraints and sexual dimorphism in immune defense. Evolution 59:1844–50.

Rolff J, Siva-Jothy MT. 2004. Invertebrate ecological immunology. Science 301:472–5.

Schmid-Hempel P. 2003. Variation in immune defence as a question of evolutionary ecology. Proc R Soc Lond B Biol Sci 270:357–66.

Sheldon BC, Verhulst S. 1996. Ecological immunology: costly parasite defences and trade-offs in evolutionary ecology. Trends Ecol Evol 11:317–21.

Tinsley RC. 1989. The effects of host sex on transmission success. Parasitol Today 5:190–5.

Tseng M. 2003. Sex-specific response of a mosquito to parasites and crowding. Proc R Soc Lond B Biol Sci 271:S186–8.

van Boven M, Weissing FJ. 2004. The evolutionary economics of immunity. Am Nat 163:277–94.

Zeng Z-B. 1988. Long-term correlated response, interpopulation covariation, and interspecific allometry. Evolution 42:363–74.
Zuk M. 1990. Reproductive strategies and disease susceptibility: an evolutionary viewpoint. Parasitol Today 6:231–3.
Zuk M, Kolluru GR. 1998. Exploitation of sexual signals by predators and parasitoids. Q Rev Biol 73:415–38.
Zuk M, McKean KA. 1996. Sex differences in parasite infections: patterns and processes. Int J Parasitol 26:1009–24.
Zuk M, Simmons LW, Rotenberry JT, Stoehr AM. 2004. Sex differences in immunity in two species of field crickets. Can J Zool 82:627–34.
Zuk M, Stoehr AM. 2002. Immune defense and host life history. Am Nat 160:S9–22.