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Bateman's principle and immunity

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The immunocompetence handicap hypothesis (ICHH) of Folstad and Karter has inspired a large number of studies that have tried to understand the causal basis of parasite-mediated sexual selection. Even though this hypothesis is based on the double function of testosterone, a hormone restricted to vertebrates, studies of invertebrates have tended to provide central support for specific predictions of the ICHH. I propose an alternative hypothesis that explains many of the findings without relying on testosterone or other biochemical feedback loops. This alternative is based on Bateman's principle, that males gain fitness by increasing their mating success whilst females increase fitness through longevity because their reproductive effort is much higher. Consequently, I predict that females should invest more in immunity than males. The extent of this dimorphism is determined by the mating system and the genetic correlation between males and females in immune traits. In support of my arguments, I mainly use studies on insects that share innate immunity with vertebrates and have the advantage that they are easier to study.

Keywords: immunocompetence; fitness; sexual dimorphism; parasite-mediated sexual selection; life-history trajectories

1. INTRODUCTION

This paper proposes an alternative perspective of the immunological phenomena associated with honest sexual signalling and parasite-mediated sexual selection. As a result of this perspective I aim to provide a new general framework for understanding patterns of investment in immunity based on the fact that males and females of many species have sexually dimorphic life-history strategies (Rice 1984; Slatkin 1984; Crowley 2000 and references therein). Females gain fitness through increased longevity, whilst males gain fitness by increasing mating rates (Bateman 1948; Trivers 1972; Clutton-Brock 1988), known as Bateman's principle.

In order to provide the background upon which my hypothesis is based, I will start by briefly reviewing the central ideas of the immunocompetence handicap hypothesis (ICHH) and related studies. Moreover, I will summarize the literature on sexual dimorphism in immunity, life history and parasite resistance, focusing on insects. Insects and other invertebrates provide highly suitable models with which to study innate immune function, thereby providing insights into this part of immunity shared by vertebrates and invertebrates (Hultmark 1993; Roitt *et al.* 1993; Vilmos & Kurucz 1998). Innate immunity is 'a set of disease-resistance mechanisms that are not specific to a particular pathogen' (e.g. anatomical, physiological, phagocytotic and inflammatory) (Goldsby *et al.* 2000). My aim is to provide an evolutionary framework within which all animals with innate immunity can be examined. Furthermore, many aspects discussed below using insect examples have been reviewed in vertebrates (e.g. Grossmann 1989; Poulin 1996; Zuk & McKean 1996; Hillgarth & Wingfield 1997; Lochmiller & Deerenberg 2000; Norris & Evans 2000).

The hypothesis of parasite-mediated sexual selection (Hamilton & Zuk 1982) has generated a large and influential literature (see references in Hamilton & Poulin 1997; Hillgarth & Wingfield 1997). This hypothesis predicts that within a species, females ought to prefer to mate with 'bright' males in order to gain indirect genetic benefits: the assumption being that 'brightness' is correlated with parasite resistance. This prediction should also hold among species, such that species with higher average parasite burdens should have more elaborate ornamentation in males (Hamilton & Zuk 1982). However, this mechanism only works if 'brightness' honestly reflects immunocompetence. Folstad & Karter (1992) provided a mechanistic explanation for parasite-mediated sexual selection, the immunocompetence handicap hypothesis (ICHH). In vertebrates, the hormone testosterone is involved in the expression of secondary sex traits (e.g. bill colour). The ICHH proposes that high levels of testosterone lead to increased expression of these traits whilst simultaneously assuming that testosterone also has an immunosuppressive effect. This direct link between trait expression and immunity is suggested as the pivotal mechanism that guarantees that secondary sexual traits honestly reflect immunocompetence, and hence facilitates female choice based on parasite resistance. Several correlational and experimental studies have found empirical support for the ICHH (Skarstein & Folstad 1996; Kurtz & Sauer 1999; Verhulst *et al.* 1999; Ryder & Siva-Jothy 2000; Skarstein *et al.* 2001). Even though the original hypothesis is explicitly related to the function of testosterone, a hormone that is restricted to vertebrates, specific predictions derived from the ICHH have been investigated using invertebrate model systems. Most invertebrates, and all insects, lack male-specific hormones such as testosterone (Nijhout 1994). Kurtz *et al.* (2000) and Kurtz & Sauer (2001) detected higher immunocompetence in female, relative to male, scorpion flies when they examined four immune effector systems (haemocyte numbers, phagocytosis, phenoloxidase expression, lysozyme-like activity). Ryder & Siva-Jothy (2000) found a positive correlation between haemocyte load and courtship song quality in crickets.

Wedekind & Jakobsen (1998) found higher parasite susceptibility in male, relative to female, copepods and stressed that this supported the general findings on gender differences in immunocompetence even in the absence of sex-specific hormones. All three studies recommend examination of the consequences of the predictions made by the ICHH. Since Folstad & Karter (1992) based their arguments on the dual function of testosterone, a hormone restricted to vertebrates, there is a need for a broader hypothesis that explains the similar patterns observed in vertebrates and invertebrates. A much simpler version of the ICHH omits hormones: it states that finite resources can be allocated in different ways, as such investment in ornamentation leads to trade-offs in immunological investment (Sheldon & Verhulst 1996). Only individuals in good condition can invest simultaneously in immunity and ornamentation. However, there is little evidence for a direct physiological link between immunocompetence and ornamentation (for an exception showing a physiological link between a secondary sex trait and an immune effector system see Siva-Jothy (2000)).

Furthermore, testosterone is the key to honest signalling in the ICHH, whereas the simplified approach is strongly condition-dependent and therefore considerably more vulnerable to cheats.

More recently, the immunosuppressive role of testosterone has been challenged by Braude *et al.* (1999), who proposed the concept of immunoredistribution: testosterone mediates the migration of immune cells like leucocytes. Peters (2000) reported an immunosuppressive effect of testosterone in laboratory experiments but observed conflicting patterns in the field. Furthermore, experimental investigation by Bilbo & Nelson (2001) demonstrated an immunity-enhancing rather than suppressing role of testosterone in hamsters. Inherent to the ICHH and the mechanism proposed by Sheldon & Verhulst (1996) is the assumption that males have suppressed or downregulated their immune function due to reproductive behaviour, but that males and females have the same basal levels of immunocompetence. Sexual dimorphism in morphology, behaviour, physiology and ecology, however, is a widespread phenomenon (Rice 1984; Slatkin 1984; Crowley 2000 and references therein) and is likely to occur in immunological traits (e.g. Grossman 1989; Kurtz *et al.* 2000). Furthermore, it has been shown that genes promoting fitness in females can decrease male fitness and vice versa (Chippindale *et al.* 2001).

2. SEXUAL DIMORPHISM AND THE PROPOSED HYPOTHESIS

(a) *Sexual dimorphism in life history*

A striking sexual difference in many species is the way fitness is maximized (Bateman 1948; Trivers 1972). Whereas longevity is a major determinant of female fitness, mating success or even daily mating rate is of much greater importance to males. Female reproduction depends directly on the resources (quality and quantity) gained whereas males should invest in obtaining mates (Trivers 1972). As Williams (1966) stated: 'It is commonly observed that males show a greater readiness for reproduction than females. This is understandable as a consequence of greater physiological sacrifice made by females for the production of each surviving offspring'. Even within species displaying sex-role reversal, such as the mormon cricket, where males invest in a spermatophore, female investment in egg production is still higher (Gwynne 1984).

The classic example for this dichotomy was provided by Bateman's (1948) study on reproduction in *Drosophila melanogaster*. He showed that variance in male mating success was much higher than variance in female mating success. Studies of many insect and vertebrate taxa support the view that mating rate is a major determinant for fitness in males, whereas longevity is of more importance in females (e.g. Fincke *et al.* 1997; Clutton-Brock 1988). If, as seems likely (see above), 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. As a consequence, females should show immune expression that is consistent with increased resistance to infection (see above).

(b) *Sexual differences in parasite burdens*

If testosterone were generally immunosuppressive, we would expect higher parasite burdens in male vertebrates, a phenomenon reported in a recent meta-analysis (Poulin 1996). However, Poulin (1996) also discusses the possibility that gender differences in behaviour might account for the observed pattern by influencing exposure to parasites.

Sheridan *et al.* (2000) did not reveal such a pattern in a meta-analysis of parasitic infection in invertebrates. However, many of the studies that Sheridan *et al.* (2000) cite show a bias of infection towards one sex or the other. It is important to note that patterns of parasitic infection are not a direct indicator of immunocompetence since they might also reflect habitat quality, food preference, behavioural differences etc.

(c) *Sexual differences in parasite mediated fitness costs*

In addition to differences in parasite burdens it is also important to look at variation in the fitness consequences of such burdens to males and females. Even though the literature on this subject is scant, some recent studies have reported differences between males and females in levels of virulence for a certain parasite (e.g. Agnew *et al.* 1999; Braune & Rolff 2001). Abbot & Dill (2001) found decreased longevity in female milkweed beetles infested with sexually transmitted mites but not in males. Female locusts died earlier than males if infected with an entomophagic nematode (van Sambeek and Wiesner, 1999). In contrast, male house crickets showed a higher mortality compared with females when experimentally infested with the bacterium *Serratia liquefaciens* (Gray 1998).

(d) *Sexual differences in immunity*

If parasitism affects each host sex in a different way, there are three possible explanations: (i) differences in condition and/or life-history investment (Braune & Rolff 2001), (ii) differences in the expression of immune effector systems (Hazarika & Gupta 1989; Radhika *et al.* 1998; Kurtz *et al.* 2000), or (iii) a combination of both. Clearly, measuring immune function by assaying single effector

system parameters without showing how this expression is related to a successful immune response is of restricted use (Siva-Jothy 1995; Norris & Evans 2000). The insect literature cited is either based on studies showing *in vitro* rates of phagocytosis of haemocytes (Kurtz *et al.* 2000), or the activity of phenoloxidase, which is known to confer higher resistance when expression is higher (Nigam *et al.* 1997). The best evidence that high haemocyte loads are related to successful encapsulation is provided by an artificial selection study in *D. melanogaster* (Kraaijeveld *et al.* 2001). This finding is further supported by comparative studies (Eslin & Prévost 1998; Fellowes & Godfray 2000) showing that among *Drosophila* species those with higher encapsulation rates have higher haemocyte loads. The few studies carried out on gender differences in immunity in invertebrates show a higher female immunocompetence, often already present in late larval instars (Hazarika & Gupta 1989; Nigam *et al.* 1997; Radhika *et al.* 1998; Kurtz *et al.* 2000; Kurtz & Sauer 2001; Siva-Jothy *et al.* 2001). In vertebrates many measures of immune effector systems can be confounded by interactions with sex steroid hormones, however the overall picture seems to be that females are more immunocompetent than males (Grossman 1989). Furthermore, in a study on Siberian hamsters carried out by Bilbo & Nelson (2001) where the effects of sex steroid hormones were experimentally removed, females showed a highly elevated antibody production against lipopolysaccharide (LPS) relative to males. Therefore, in this species at least, immunity differs between the sexes without sex-specific hormonal immunoregulation.

In order to understand the evolution of immunity it is necessary to show that immune traits are heritable. There is now good evidence that the expression of immune function is heritable in insects (Fellowes *et al.* 1998; Kurtz & Sauer 1999; Kraaijeveld *et al.* 2001; Ryder & Siva-Jothy 2001) and vertebrates (Goater & Holmes 1997 and references therein).

(e) *Costs of immunity*

Most concepts in ecological immunology (Sheldon & Verhulst 1996) revolve around the costs of immunity. Two major potential costs exist, those of evolving effective immunity, and those of maintaining and utilizing an immune effector system. The costs of evolving immunity have been demonstrated by Kraaijeveld & Godfray (1997) and Fellowes *et al.* (1998) who showed that *D. melanogaster* selected for resistance against parasitoids had lower larval competitive ability. In addition, Webster & Woolhouse (1999) demonstrated reduced fecundity in snails selected for resistance against larval trematodes. The costs of using an immune system have also been experimentally demonstrated in terms of reduced fecundity (Fellowes *et al.* 1999) and survival (Moret & Schmid-Hempel 2000). *Drosophila melanogaster* that mounted an immune response were less fecund and bumble bee workers with challenged immune sytems showed lower survivorship under starvation. Under conditions of food shortage immunity is also downregulated (Feder *et al.* 1997; M. T. Siva-Jothy and J. J. W. Thompson, personal communicaton). Thus, it can be assumed that maintaining immunity is costly for invertebrates (for vertebrates see Lochmiller & Deerenberg (2000)).

(f) *Sexual conflict*

As Rice (1984) pointed out, once the genders evolved and shared genes, expression of certain genes might be beneficial for one, but not necessarily for the other gender. Shared genes might even be deleterious for one gender. Therefore, even without a direct conflict of interest between the sexes, sexually antagonistic genes may exist (Lessells 1999). Chippindale *et al.* (2001) clearly demonstrated such a conflict between the sexes in the development of *D. melanogaster*. Whereas juvenile fitness was strongly positively correlated between the sexes, adult fitness was negatively correlated. Genomes that increased male fertilization success reduced egg production and vice versa. Another example is the study by Rice (1998), which showed that male fitness increased when females were experimentally removed from the gene pool. Males with experimentally created Y-chromosomes performed better in three out of four fitness correlates measured. However, survivorship and fecundity of females mated to such males with synthetic Y-chromosomes decreased.

Sexual conflict is likely to be a powerful mechanism for the evolution of sexual dimorphism (Rice & Chippindale 2001). Here, due to the costliness of immunity, a sexual conflict over immunity is envisaged, as males and females are assumed to have different adaptive peaks in immunity.

(g) *Theory of sexual dimorphism*

Based on the preceding brief review I will examine immune function in terms of sexual dimorphism caused by ecological factors. Investigating the ecological causes of sexual dimorphism, Slatkin (1984) distinguished three different cases: (i) competition between the sexes for limited resources, (ii) at least two optima for both sexes, and (iii) different selection exerted on females and males. The last two causes have greater relevance to the case discussed here. Because of different life-history trajectories in males and females, investment in immunity should differ. Lande (1980) provides a model for the evolution of quantitative traits in males and females on the basis of genetic correlations between the sexes. This model suggests that natural selection alone can act sufficiently upon males and females so that they achieve different adaptive peaks. Because of the genetic correlation for fitness traits, each sex is dragged away from its adaptive peak. Under both natural and sexual selection, males might be further away from their adaptive peak than females (Lande 1980).

3. DISTINGUISHING BATEMAN'S PRINCIPLE IN IMMUNITY FROM OTHER HYPOTHESES

Looking at immunity from a natural rather than a sexual selection perspective could provide an alternative explanation for several findings reported in the context of the ICHH. As the predictions of the idea proposed here are similar to those of Folstad & Karter (1992) and Sheldon & Verhulst (1996) I shall explore criteria to distinguish between these three frameworks. First, Folstad and Karter's model was originally proposed for species with testosterone (e.g. vertebrates) as this is the key for honest signalling in their framework. Clearly, therefore, the ICHH is not applicable to insects as male-specific hormones with functions analogous to testosterone are not described (Nijhout 1994). The approach of Sheldon &

Verhulst (1996) is mainly based on resource trade-offs: 'possession or maintenance of the ornament is considered to be costly in terms of some limiting resource (energy?) with the result that less of that resource can be devoted to other important functions (e.g. immune functions)' (p. 319). Again it is mainly concerned with the downregulation of immunity in adult males.

By contrast, the approach suggested here envisages life-history trade-offs due to different life-history trajectories in males and females. This predicts considerable differences in immunity between males and females before the onset of reproduction and would work without costly secondary sex traits in males.

Bateman's principle in immunity would be supported by comparative studies of immunity in insects looking for positive correlations between an index of male/female reproductive success and the difference in immunity between the sexes. Furthermore, classic quantitative genetic approaches could be used to look for negative covariance between male and female fitness in immune traits (as suggested in a different context by Rice & Chippindale (2001)).

A recent study by Svensson *et al.* (2001) demonstrates a genetic correlation between an immune trait and a signal trait due to linkage disequilibrium. This supports the view presented here that linkage disequilibrium rather than pleiotropic effects should underlie a correlation between signal traits and immunity. Good quality (immunocompetent) females mate with high quality (attractive) males (e.g. Poulin 1994) and the offspring inherits attractiveness and immunocompetence.

4. CONCLUSIONS

The idea proposed is essentially based on a bottom-up approach: because of genetic and life-history differences, males and females differ in their investment in immunity. In contrast, the ICHH and related hypotheses adopt a top-down approach: endocrine regulation in adults differs between the sexes and therefore immunity is driven in different directions. This 'top-down' approach poses the question: why are different endocrinological mechanisms altering the immune system in different directions? Is it because the evolution of compensatory mechanisms is constrained? It is important to note that hormone-mediated changes in immunity, either by immunosuppression or by immunoredistribution, can act in addition to the mechanism proposed here. Grossman (1989) has already suggested a link between female immunocompetence and longevity but devoted his paper to the role of hormones from a functional perspective. Whatever the proximate mechanism for sexual dimorphism in immunity, the Batemanian approach discussed could be the ultimate reason. The idea of sexual dimorphism in immunity is also much more parsimonious. Whether it is the causal basis for the cited studies remains to be investigated. To date, few studies have provided separate data on immune function in the sexes whilst controlling for behavioural and ecological differences. This is particularly true in insects and it is therefore too early to gain a broader picture from a meta-analytical approach. It is interesting to note that testosterone might indirectly influence immunity by altering male behaviour and hence resource acquisition (e.g. Buchanan *et al.* 2001). This would not conflict with the idea of sexually dimorphic immunity but would question the mechanism that guarantees honest signalling as proposed by Folstad & Karter (1992).

Mike Siva-Jothy followed all stages of these ideas with encouragement and coined the title. Andy Barnes, David Richardson, Mark Fellowes, Sophie Armitage and especially Helen Crudgington (beware of social insects) commented on several drafts and helped to keep my train of thought on track and to clarify the presentation. An anonymous reviewer provided further clarifying criticism, and the DFG provided financial support. To all I am grateful.

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