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PARASITES, BRIGHT MALES, AND THE IMMUNOCOMPETENCE HANDICAP

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Abstract.—It has been argued that females should be able to choose parasite-resistant mates on the basis of the quality of male secondary sexual characters and that such signals must be costly handicaps in order to evolve. To a large extent, handicap hypotheses have relied on energetic explanations for these costs. Here, we have presented a phenomenological model, operating on an intraspecific level, which views the cost of secondary sexual development from an endocrinological perspective. The primary androgenic hormone, testosterone, has a dualistic effect; it stimulates development of characteristics used in sexual selection while reducing immunocompetence. This "double-edged sword" creates a physiological trade-off that influences and is influenced by parasite burden. We propose a negative-feedback loop between signal intensity and parasite burden by suggesting that testosterone-dependent signal intensity is a plastic response. This response is modified in accordance with the competing demands of the potential costs of parasite infection versus that of increased reproductive success afforded by exaggerated signals. We clarify how this trade-off is intimately involved in the evolution of secondary sexual characteristics and how it may explain some of the equivocal empirical results that have surfaced in attempts to quantify parasite's effect on sexual selection.

Darwin (1871) introduced the idea that female mate preference may be a selective force resulting in extravagant male ornamentation but was unable to explain the driving force behind the evolution of female choice. Recently, Hamilton and Zuk (1982) presented a controversial theory that implicates the role of parasitism in this process. They proposed that male secondary sexual characters have a utilitarian function in that they facilitate female appraisal of a potential mate's ability to resist the detrimental effect of parasitism. A basic assumption in the Hamilton-Zuk hypothesis is that the full expression of viability-indicating traits (i.e., secondary sexual characters) depends on the health and vigor of the individual possessing them. Females would select for parasite resistance in their future offspring and enhance their own reproductive success if their mate-selection criteria, based on male secondary sexual characters, were indicative of a male's ability to resist parasite infection and if this ability were heritable.

Hamilton and Zuk (1982) and later Maynard Smith (1985) recognized this parasite–sexual selection hypothesis as a modification of the handicap principle

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first presented by Zahavi (1975, 1977). The development and maintenance of secondary sexual characters may be a considerable handicap (Zahavi 1975, 1977) reducing a male's chances of survival, while also acting as a signal of quality by advertising genetic resistance under sexual selection (Maynard Smith 1985). According to extant theory, honest indicators of genetic quality must be costly to produce or maintain (Zahavi 1975, 1987; Kodric-Brown and Brown 1984). Although the additional cost of producing and maintaining a secondary sexual character under concurrent parasite challenge is poorly understood, recent evidence indicates that parasites could act as a constraint on character exaggeration (Zuk et al. 1990*a*). Here we postulate an explicit mechanistic model that presents a new constraint for the development of vertebrate secondary sexual characters, an endocrinological determinant emphasizing parasitism's role in sexual selection. This hypothesis may help to explain several empirical discrepancies in intraspecific tests of the parasite–sexual selection hypothesis.

Disease (defined to include the deleterious effects of viral, bacterial, protozoan, and metazoan parasites) and nutrition are considered the major external biotic factors influencing vertebrate growth (Vander et al. 1986). As a specialized form of somatic growth, production and maintenance of secondary sex traits are also influenced by these two environmental determinants. The regulation of male secondary sex traits in response to nutritional constraints is mediated by the endocrine system, which "adjusts and correlates the activities of the various body systems making them appropriate to the changing demands of the external and internal environment" (Ganong 1987, p. 229). The titer of the principal androgenic hormone, testosterone, required for the development of many secondary sex traits in males, has been shown to fall sharply during food shortages (Wilson et al. 1979; Wingfield 1980), and thus nutrition can be considered a limiting factor for the expression of any such trait linked to this hormone (cf. Ligon et al. 1990).

However, regulation of testosterone profiles, and consequently the development of secondary sexual characteristics, cannot be explained by energetic considerations alone. Parasitic disease also plays an important role in this process. Although a high testosterone titer confers the benefits of exaggerated secondary sexual development and potentially increased mating success, such a condition simultaneously impairs the functioning of the immune system. This double-edged sword creates a real and potential physiological dilemma for males. Thus, established or incident parasite infections expose a male to substantial costs during the developmental and maintenance period of testosterone-dependent secondary sexual characters. When this detrimental immunological effect is taken into account, the viability-indicating signal broadcasted through secondary sexual characteristics is indeed costly and should thus fit the criteria for honest indicators detailed by the existing handicap principles (see, e.g., Zahavi 1975; Grafen 1990*a*, 1990*b*).

THE IMMUNOCOMPETENCE-HANDICAP HYPOTHESIS

Empirical evidence strongly implicates an endocrinological component that has been ignored in the extant parasite–sexual selection hypotheses. We suggest that

testosterone profiles and/or responsiveness, and consequently both immunosuppression and the expression of male secondary sexual characters, are self-regulated in response to parasite burden. However, this parasite-dependent regulation is not independent of the constraint imposed by genetic resistance (i.e., whether or not the individual possesses "good genes"). Males that possess such a parasite-dependent feedback system would have a clear advantage relative to those that overinvested in viability indicators and thus subsequently suffered because of the concomitant immunosuppression. Although an exaggerated investment in secondary sexual characters may increase a male's immediate reproductive success, future success would be reduced because of the morbidity and possible mortality following immunosuppression.

This endocrinological process would have implications for females as well. Assuming an obligatory immunosuppression associated with the development and maintenance of morphological and behavioral secondary sexual characters, viability-indicating traits would be strictly "honest" handicaps, enabling females to assess the status of a potential partner's parasite burden and resistance.

This feedback system, which we refer to as the "immunocompetence-handicap hypothesis," may explain how parasites influence expression of secondary sexual traits, why such traits are a handicap, and thus how they could evolve as viability indicators. Our hypothesis stresses parasitism and the hidden costs of viability indicators (i.e., immunosuppression). We present our thesis as it applies to testosterone; however, the model would accommodate any biochemical substance that is self-regulated and exerts the two-pronged effect of compromising the immune system and stimulating trait expression. It is a testable hypothesis that further sheds light on how parasitism influences viability-indicating traits used in sexual selection within vertebrates.

EMPIRICAL EVIDENCE

We develop our case by presenting empirical evidence for the interaction between each model component separately (fig. 1). Although we hypothesize causality in the model system as a whole, the interrelationships between components are based on observational correlations and need not have a causal basis. The functional relationships among parasites, plasma testosterone, immunocompetence, secondary sexual characters, and reproductive behavior are complex and by no means completely understood. Not one of the reviewed empirical studies includes all of these model response variables, and a study that simultaneously investigated all components and their interactions in a particular species would facilitate testing of the immunocompetence-handicap hypothesis. Until now, component interactions have been most extensively documented in independent studies of laboratory mice (*Mus musculus*). These studies include parasite's effect on the immune system and vice versa (reviewed in Grossman 1984), parasite's effect on the endocrine system and vice versa (e.g., Huber et al. 1982; Spindler 1988), parasite's effect on dominance (e.g., Rau 1984), the endocrine system's effect on immunity (e.g., Cohn 1979*b*), and, however controversial, the endocrine system's effect on dominance (Adkins-Regan 1981; Wee et al. 1988).

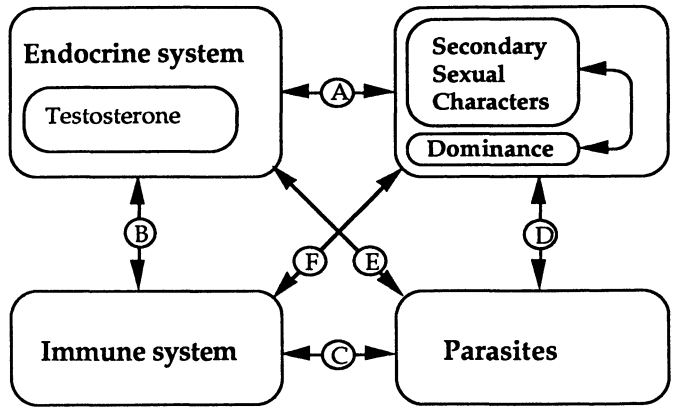

FIG. 1.—Figure detailing model interactions included in the immunocompetence-handicap hypothesis. Profiles of testosterone, the primary androgenic hormone in vertebrates, have a positive effect on the development of secondary sexual characters and dominance, while hampering the immune response (pathways *A* and *B*, respectively). Parasites interact with the immune system (pathway *C*), have a negative effect on secondary sexual development and dominance (pathway *D*), and cause reductions in testosterone profiles (pathway *E*). The development of testosterone-dependent secondary sexual characters also co-occurs with a reduction in immunocompetence (pathway *F*). We hypothesize a feedback system, operating through the direct and indirect relationships connecting model components, a feedback that links secondary sexual development to an individual's genetic resistance to parasites.

A. Endocrine–Secondary Sexual Characters Interplay

The endocrine basis of development of secondary sexual characters is not fully understood, but the conventional wisdom is that sex hormones, such as testosterone, are of fundamental importance. A spectrum of secondary sexual characters, including aggressive and display behavior, vocalizations, pheromones, and visible characters, exhibit dose dependency to androgenic hormone profiles in species of mammals, birds, lizards, and fish (see, e.g., Dorfman 1948; Zeller 1971; Lofts and Murton 1973; Edwards and Rowe 1975; Fernald 1976; Rohwer and Rohwer 1978; Silverin 1980; Adkins-Regan 1981; Wittenberger 1981; Fox 1983; Inga 1984; Wingfield 1984; Hadley 1988; Leader-Williams 1988). In general, male vertebrates from seasonally mating species exhibit a sharp rise in testosterone profile before the initiation of sexual activity. The levels remain elevated during the mating season and then dramatically decrease at the termination of mating activity (Leader-Williams 1979; Silverin 1980; Inga 1984; Stokkan et al. 1988). The exact profile patterns vary among species (Wingfield 1984; Dufty and Wingfield 1986; Wingfield et al. 1990).

Experimental evidence and field studies illustrate the importance of sex hormones to various behavioral and morphological components of male reproductive effort. Testosterone-induced behavioral changes such as alteration of the duration and intensity of territorial behavior (Silverin 1980; Watson and Parr 1981; Miller et al. 1987), vocalizations (Watson and Parr 1981), and aggressiveness (Leader-

Williams 1979; Mossing and Damber 1981) have been documented (cf. Konishi et al. 1989). Experimental studies indicate that social status is testosterone-dependent (Moss et al. 1979; Stokkan et al. 1980), and there is also evidence for a positive association between testosterone ranking and overall fertility (capable females impregnated), libido (estrual females mounted), and fertilizing ability (mounted females impregnated) (Post et al. 1987). Experimental castration has been shown to reduce aggressiveness and induce loss of social status (Moss et al. 1979; Wittenberger 1981), an effect usually reversed by subsequent testosterone injections (Watson and Moss 1971; Lofts and Murton 1973; Moss et al. 1979; Watson and Parr 1981; Wittenberger 1981). Age-dependent social dominance has been explained by the gradual rise in testosterone secretion naturally occurring with male maturation (Leader-Williams 1979). Additionally, testosterone-induced polygamy in normally monogamous birds has been shown experimentally (Wingfield 1984).

Examples of testosterone-dependent morphological developments are numerous. Response to testosterone treatment of birds has been documented to affect the brightness of male breeding plumage (Stokkan 1979) and comb growth in domestic roosters (*Gallus domesticus;* Dorfman 1948; Zeller 1971), willow ptarmigan (*Lagopus lagopus lagopus;* Stokkan 1979), and red grouse (*Lagopus lagopus scoticus;* Moss et al. 1979). Castrated males sometimes show attenuation of sexual ornaments and are known not to take part in sexual competition (Meschaks and Nordkvist 1962; Lincoln et al. 1972; Lofts and Murton 1973). Furthermore, female nuptial plumage shows testosterone dependency in two polyandrous species, northern and Wilson phalaropes, *Lobipes lobatus* and *Steganopus tricolor,* respectively (Johns 1964).

B. Endocrine–Immune System Interplay

The interaction between the endocrine and immune systems is well documented. Current evidence supports the hormonal mediation of the thymus (Grossman 1985; Grossman and Roselle 1986), thymic hormones affecting the maturation of T-lymphocytes, and an immunosuppressive effect of glucocorticoids (Hadley 1988). Hormones have an effect on the immune system, at the level of both individual cells involved in the humoral- and cellular-mediated immunity (i.e., maturation of T-lymphocytes) and the glands or tissues important to the immune system (i.e., thymus gland). It is currently accepted that male-female differences in the immune system are modulated largely by sexual dimorphism in sex hormones (Dörner et al. 1980; Weinstein et al. 1984; Ahmed et al. 1985; Grossman 1985, 1988). Males typically show greater susceptibility to parasites and weaker immune response to a variety of antigens than do females (Cohn 1979*a;* Grossman 1985; Alexander and Stimson 1988).

Testosterone is often implicated in changes in immunocompetence. There is observational evidence from free-ranging marsupials (Bradley 1987) and salmonids (Pickering 1987) of immunosuppression associated with greatly elevated testosterone (and corticosteroid) profiles during male reproductive effort. Castration of mature male rodents results in decreased testosterone production and increased immunoglobulin levels, thymic weight, and humoral and cell-mediated

immunity (Eidinger and Garrett 1972; Grossman 1984; Alexander and Stimson 1988). Involution of the thymus and shrinkage of other lymphoid tissue in mammals (Dörner et al. 1980; Weinstein et al. 1984) and bursa reduction in birds (Norton and Wira 1977) are examples of testosterone's direct effect on the immune system. Short-term, intensive dosing of testosterone is documented to reduce both humoral and cell-mediated immunity (Inman 1978; Grossman 1984; Ahmed et al. 1985), whereas sustained administration of lower dosages reduces activity of natural killer cells and antibody-dependent, cell-mediated cytotoxicity (Hou and Zheng 1988). The production of immunoreactant factors and the presence of lymphocyte membrane antigens have been demonstrated to be modulated by "physiological levels" of testosterone (Stimson and Crilly 1981). Additionally, studies have shown that testosterone inhibits lymphocyte transformation in a dose-dependent manner (Wyle and Kent 1977). Testosterone may also have an indirect effect on immunocompetence by reducing the levels of corticosteroid-binding globulins, which, in turn, can increase levels of free corticosteroids (Gala and Westphal 1965; Kley et al. 1973; Bradley et al. 1980; McDonald et al. 1981; Bradley 1987). Low immune performance in male mice has also been shown to correlate with high target-organ responsiveness to testosterone (Cohn and Hamilton 1976; Cohn 1979*a*, 1979*b*).

Circulating levels of sex hormones, such as testosterone, are in turn affected by the functioning of the immune system, an interaction appearing to be mediated by the hypothalamic-pituitary-gonadal-thymic axis (Grossman 1984, 1985). This negative-feedback loop between testosterone production and immune-system function enables individuals to regulate testosterone profiles to changing internal demands.

C. Parasite–Immune System Interplay

Parasites activate and are influenced by the immune system (Cohen and Sadun 1976; Cox 1982). Even ectoparasites can elicit a strong host immune response. Domestic white leghorns, which are frequently infected by the hematophagus northern fowl mite (*Ornithonyssus sylviarum*), show increased antibody production followed by a reduction in mite populations (DeVaney and Augustine 1988). Infected male reindeer (*Rangifer tarandus tarandus*) have decreased concentrations of circulating antibodies to the nematode *Elaphostrongylus rangiferi* during the breeding season, which coincides with an increase in first stage *E. rangiferi* larval density in the feces (cf. Gaudernack et al. 1984; Halvorsen et al. 1985). It was suggested that larval production is normally suppressed by the male immune responses during the nonreproductive period. Fecal samples taken from females at the same time period showed no increase in larval density (Halvorsen et al. 1985), a relationship also documented for *E. cervi* in red deer (Ottestad 1983). Thus, the dynamic interplay between parasite fecundity and host immune system exhibits a seasonality that closely tracks or is tracked by changes in male sex-hormone profiles.

D. Parasite–Secondary Sexual Characteristics Interplay

Several recent studies have reported an inverse relationship between male mating success and degree of parasite infestation (Kennedy et al. 1987; Schall and

Dearing 1987; Borgia and Collis 1989; Zuk et al. 1990*a*). This may occur because parasitism influences the development of secondary sexual characteristics. Experimental parasitic infections reduced the expression of ornamental traits (combs and hackle feathers) in red jungle fowl (*Gallus gallus*), and females chose controls as mates more frequently than infected males (Zuk et al. 1990*a*). Conversely, traits not associated endocrinologically with reproduction (tarsus length, bill size, and saddle feathers) were not influenced by nematode intensity nor used as criteria for female choice. Loss of behavioral dominance in response to parasitic infections was documented in male mice experimentally infected with *Trichinella spiralis* (Rau 1983, 1984), and the nematode *Heligmosomoides polygyrus* prevented males from achieving behavioral dominance (Freeland 1981). Additionally, male guppies (*Poecilia reticulata*) experimentally infected with *Gyrodactylus* spp. showed a reduction in rate of mating-display behavior (Kennedy et al. 1987).

On the other hand, social status and testosterone-dependent secondary sexual coloration (Kimball and Erpino 1971) were positively associated with parasite prevalence in male lizards (*Sceloporus occidentalis*) infected with *Plasmodium* spp. (Ressel and Schall 1989). Additionally, Hausfater and Watson (1976) observed a positive correlation between dominance in male baboons (*Papio cynocephalus*) and fecal output of helminth eggs and protozoan cysts. In another study, Hausfater et al. (1990) found no correlation between the burden of a wide range of parasites and call duration of male tree frogs (*Hyla versicolor*), a trait considered important in female sexual selection. As illustrated by the studies above, the correlation between the expression of secondary sexual characteristics and parasite burden has been shown to be positive in some cases and absent or negative in others.

E. Endocrine-Parasite Interplay

The development, behavior, or reproduction of parasites is often correlated with its host's reproductive and metamorphic events (Lawrence 1991). Furthermore, the reproductive hormones regulating these events have been shown to influence parasite life-history behavior in vitro, as well as in vivo (Lawrence 1991). Parasites may have been selected to use changing hormone levels as a cue for impending changes in host environmental conditions (e.g., testosterone-induced immunosuppression). Experimental treatment with high doses of testosterone increases intensity of parasitic infection, leading to decreased host survival (Solomon 1969; Huber et al. 1982; Arcay 1985; Nakanishi et al. 1989). Testosterone implants in hosts can result in increased egg production, increased differentiation of proglottids (cestodes), and increased longevity of parasites (Solomon 1969; Chomicz 1984; Alexander and Stimson 1988). Castration of hosts, on the other hand, reduces host susceptibility and decreases somatic growth and reproductive output of parasites (Beck 1952; Hosier and Durning 1975; Noble and Noble 1976), whereas administration of testosterone reverses these effects (Lincicome and Emejuaiwe 1963; Solomon 1966, 1969).

Field results also give support to the testosterone-parasite interplay. It was demonstrated that females and castrated male reindeer had a significantly lower intensity of third-instar warble fly larvae (*Hypoderma tarandi*) than intact males

(Folstad et al. 1989). Moreover, *H. tarandi* intensity was positively correlated to body weight of intact males and this same correlation of intensity with body weight was negative for females and castrated males. The relatively lower testosterone profiles among females and castrates and the positive association between profiles and intact-male body weight (Inga 1984) may provide an explanation for this parasite-intensity pattern. In male rainbow (*Salmo gairdneri*) and brown trout (*S. trutta*), an increase in prevalence and intensity of parasitic and fungal infection has been shown to coincide with sexual maturation (Richards and Pickering 1978; Robertson 1979; Pickering and Christie 1980).

The endocrine-parasite interplay may be responsible not only for a portion of the intra- and intersexual variability of parasite intensity and prevalence but also for regulation and sensitivity to testosterone itself. Decreased testosterone titers following parasitic infections are also documented in several species of mammals (Spindler 1988; Hublart et al. 1990; Villette et al. 1990). Consistent with the previously mentioned sex-specific pattern, white leghorn roosters show higher prevalence and intensity of the northern fowl mite (*Ornithonyssus sylviarum*) relative to females (Maw et al. 1935; Kirkwood 1968). Within the male population, hosts appear to exhibit plasticity in terms of testosterone profiles in response to the levels of parasitic infection. DeVaney et al. (1977) showed that 22-wk-old white leghorn roosters experimentally infected with northern fowl mites significantly decreased their plasma testosterone levels in response to moderate infections. The drop in testosterone profiles started 2 wk after infection and stabilized at a difference of about 6% after 10 wk. Both infected and uninfected birds were fed ad lib. and gained weight during the 20-wk-long study.

F. Immune System–Secondary Sexual Characteristic Interplay

Reduced immune performance has been documented to co-occur with the mating season in vertebrates (Gaudernack et al. 1984; Pickering 1986; Bradley 1987). To our knowledge, however, there are no good studies that directly measure the correlations between the variation in immune response and the development or quality of secondary sexual characteristics. There is quite likely a series of confounding variables and intermediate steps in the biochemical pathway between these two model components.

DISCUSSION

There exist several problems with the interspecific component of the Hamilton and Zuk (1982) hypothesis (see Read 1990). Additionally, empirical evidence (Hausfater and Watson 1976; Ressel and Schall 1989; Hausfater et al. 1990) challenges the model's intraspecific predictions of a negative correlation between secondary sexual development and parasite burden. Predictions based on our intraspecific model differ in some cases with those of Hamilton and Zuk because of our addition of an essential layer of detail, a negative-feedback connection between signal intensity and parasite burden and its interaction with several mediating variables. We suggest that androgen-dependent signal intensity is a plastic response, adjusted according to the potential costs of parasite pathogenicity ver-

sus the benefits of increased reproductive success afforded by signal exaggeration. When the signal is "on," there is a marked reduction in immunocompetence that increases the present and future costs incurred from parasite-induced pathogenicity. Paradoxically, this seemingly maladaptive trait sends an honest signal of an individual's ability to withstand parasitic infection, a signal of "good genes" for resistance. This embodies the tenet of handicap principles in general and provides a biological explanation for the costs that ensure honesty and evolutionary stability in a signaling system. This detailing of costs was lacking in the original Hamilton and Zuk hypothesis.

The empirical evidence from several vertebrate taxa clearly indicates that testosterone increases susceptibility and pathogenicity of parasitic infections and suppresses the immune system and, at the same time, enhances the expression of secondary sexual characters. Hormonal self-regulation in response to parasitic infection, and subsequent modification of the development of secondary sexual characters, is a pivotal element in our proposed hypothesis and is also corroborated by empirical evidence (e.g., DeVaney et al. 1977; Zuk et al. 1990*a*, 1990*b*). However the relationship between parasitic burden and signal intensity, the two variables measured in most tests of the Hamilton and Zuk predictions, is clouded by the interaction and confounding effects of three main factors: genetic resistance, timing of infection, and nutritional status.

Genetic Resistance

Our hypothesis is based on the same assumption as Hamilton and Zuk's (1982); secondary sexual characteristics signal "good genes" for parasitic resistance. However, signaling of genetic quality, while beneficial in terms of increased mating success, is constrained by the cost of immunosuppression (i.e., parasite pathogenicity and increased susceptibility). We suggest a plasticity in signaling amplitude that optimizes the trade-off between costs and benefits. This trade-off, between parasite-induced pathogenicity (cost) and increased mating success resulting from trait development (benefits), depends on genetic quality. An individual with "good genes" for resistance would pay less in the form of parasite-induced pathogenicity relative to an individual with low genetic resistance for a given signal quality. Thus, genetic quality in terms of parasite resistance modifies expression of secondary sexual characteristics.

Nutritional Status

The importance of nutritional status for the functioning of the immune system is well established (Scrimshaw et al. 1968). We suggest that parasites have an endocrinologically based, inhibitory effect on the exaggeration of secondary sexual characteristics, whereas nutritional plane has a positive effect on the same response variable. At the onset of reproductive activity, males exhibit variability in both the level of parasitic infection and their nutritional plane. Individual variability in nutritional status is determined to a large extent by variation in food availability and metabolic rate, factors that are controlled for or randomized in experimental, ad lib. feeding–regime studies. Differential development of ornamental versus nonornamental traits cannot be attributed to nutritional constraints

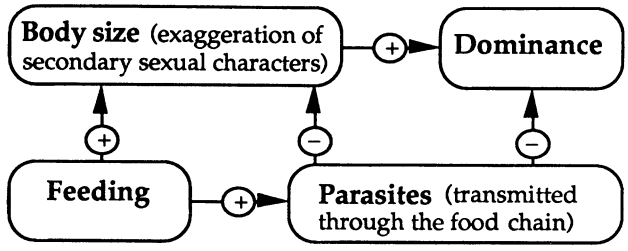

FIG. 2.—Increased food consumption positively influences an individual's somatic growth and exposure to parasites through the food chain. In turn, parasitic infection has a negative effect, whereas body size (secondary sexual characters) has a positive effect on an individual's social status.

under such experimental design. These studies corroborate the concept of parasite infection-dependent testosterone profiles (DeVaney et al. 1977), as well as a parasite's stronger negative effect on the testosterone-dependent, ornamental, secondary sexual traits (e.g., combs) relative to nonornamental traits (e.g., bill length; Zuk et al. 1990*a*, 1990*b*). It is conceivable that given low parasitic intensities, nutritional plane may have an overriding importance as a determinant of reproductive effort, which may explain some of the contradictory results found in observational studies (see Hausfater et al. 1990; Read 1990).

The importance of nutrition relative to parasitism as predictors of trait exaggeration is complicated by the fact that food consumption is positively associated with exposure to food-chain transmitted parasites. Male dominance is often related to body weight (Krebs and Davis 1983) and, thus, indirectly is a function of food consumption. Inductively, consumption-dependent, somatic growth increases an individual's probability of attaining higher status but simultaneously increases the potential for exposure to the many food-chain transmitted parasites (see fig. 2). The trade-off between growth and parasite transmission strengthens the reliability (i.e., costs) of dominance and exaggerated secondary sex traits as indicators of a male's ability either to evade behaviorally or to resist immunologically food-chain transmitted parasites. This is especially so if we consider the relationship between development of secondary sexual characteristics and immunosuppression. It is interesting to note that in many species, males refrain from eating during the mating season, reducing the transmission probability of food-chain transmitted parasites. This may be a behavioral adaptation to minimize the risks of infection during the immunosuppression associated with the reproductive activity.

Timing of Infection

A few of the field measurements of the proposed predictions of the Hamilton and Zuk hypothesis have been equivocal (Hausfater et al. 1990; Read 1990), and it has been suggested that a positive correlation in this relationship would refute their model (Price et al. 1987). However, the puzzling inconsistency may be explained by the immunosuppressive effect of testosterone and the timing of

incident cases relative to reproductive activity. This will have an impact on (1) optimal host reaction in terms of testosterone regulation and (2) the severity of parasite virulence in terms of pathology, within-host longevity, and proliferation. Parasite intensity normally fluctuates, and hosts that harmonize immunocompetence in relation to present parasite intensity immediately before or during the mating season reduce the potentially serious consequences of parasitism. Such regulation could conceivably influence behavioral dominance, and thus mating success may be sensitive to the timing and severity of parasitic infections. Subsequently, endocrine regulation that harmonizes ornament expression in relation to phenotypic condition should be favored by selection (Andersson 1982, 1986).

Signal Interpretation

Compared to the relatively stable expression of morphological sex traits, display behavior and aggression are considerably more labile characters and track testosterone profiles more closely than the morphological sex traits (Wingfield et al. 1990). Labile traits may amplify viability differences between signalers and thus have a particular revealing importance (Hasson 1988). This lability increases signal reliability with respect to genetic quality above that afforded by stable morphological traits and facilitates a time-specific evaluation of an individual throughout the breeding season. Our model does not require that the quality of behavioral or ornamental signal directly reveal external signs of parasite presence or intensity (i.e., plumage quality changing as a direct function of feather mite infestation; see Read 1990). Although such short causal pathways certainly occur and may influence female choice, we emphasize rather a more indirect signal interpretation, relating handicap, costs, and trait exaggeration to the heritable parasite resistance of the possessor.

Signal Costs

Parasitic virulence may be intensified in males with compromised immune systems that invest heavily in reproductive effort and thus have high levels of plasma testosterone. Following a reduction in immunocompetence, parasitic site-specificity can be altered, a process that may increase pathogenicity dramatically (Rose 1972). Parasites could have a selective advantage by proliferating within, transmitting to, or undergoing the vulnerable phase of their life history during this immunosuppression associated with mating. Testosterone and secondary sexual characteristics may even be used by vagile parasites and transmission stages as an indirect cue, indicative of a host habitat rendered hospitable by immunosuppression (Dobson et al. 1970; Christensen and Dobson 1979; Post et al. 1987), similar to the way predators use mating vocalizations to locate prey during their mating period (Cade 1979; Ryan et al. 1982). Subsequently, on an evolutionary time scale, it is reasonable to speculate that parasitic life-history strategies adapt to the timing of the male breeding season.

Community-Level Effects

Hosts typically are challenged by a whole community of co-occurring parasite species, each with its own set of traits and habitats. The presence of a one-on-one

association between the variability in quality of the host's sexual trait and intensity of one parasite species has been suggested (Hamilton and Zuk 1982; Price et al. 1987). However, by only evaluating the effects of one parasite species, rather than the more common case of infection by multiple parasite species, it becomes impossible to predict the consequences of the total parasite community on the host species. A ranking of males, in terms of the costs accrued as a result of one particular parasite species, may be quite different than a ranking based on costs accrued as a result of the total parasite community (infracommunity). It would seem reasonable that modification of sexual traits because of a highly virulent parasite would be a more important index for a mate-searching female since resistance to such a parasite would have greater fitness payoffs. It is reasonable that the "immunocompetence handicap" is a mechanism in which viability indicators are responsive to the total infracommunity of parasites, with heightened sensitivity to the most virulent species.

Transmission versus Susceptibility

Parasite burden may be considered a result of two components of host quality, behavior and susceptibility. Host behavior, which influences the contact rate between the parasite and host, and susceptibility (i.e., lack of resistance), which influences how many of the contacted parasites eventually manage to establish in the host, are likely to have a genetic component (Hutt 1958; Madhavi and Anderson 1985; Price 1985; Wakelin 1985*a*, 1985*b*). If a male is not exposed to any parasites before mating because of heritable parasite-avoidance behavior, then he can devote himself fully to the development of secondary sexual characteristics without running the immediate risks involved with immunosuppression, have increased reproductive success, and pass on the behavioral resistance to offspring. However, evading transmission may have a nonheritable behavioral component or be due to chance alone. In this case, a male is still able to develop viability indicators without facing the endocrinological consequences. Such a male's condition is thus a poor indicator of heritable parasite resistance and provides low diagnostic value since there has been no challenge (Read 1990). Additionally, experimental manipulation of parasitic challenge has been shown to result in females' choosing males not exposed to parasites (Zuk et al. 1990*a*). If, however, there are homogeneous transmission levels, subsequent variance in parasite intensity among males indicates variability in male susceptibility, providing a basis for female mating preferences. Accordingly, the differences in the quality of an indicator become more easily distinguished and significantly more reliable with increasing variation in parasitic burden. Consequently, the diagnostic quality of the viability indicators, and thus the evolution of the proposed mechanism, is likely to be functionally related to the parasites' distribution in space and time.

Cheating

Cheating may confer an even more serious attenuation of the signal's informational value to a female searching for a mate. Zahavi (1975) suggested that signals must be costly in order to evolve, and thus there is a limit to which characters

fulfill this status. Grafen (1990*a*) evaluated the effect of cheating on the evolutionary stability of signaling systems based on Zahavi's handicap hypothesis (1975, 1977, 1987). He suggested that a signal's impressiveness is a function of its cost, and the cost should be directly related to what quality is being signaled. Thus, the signal demonstrates excess resources in terms of that quality. Assuming equal quality, a cheater is an individual paying less immediate costs for a given signal relative to an honest signaler. If receivers cannot distinguish between the two morphs, the cheater benefits. It must be noted that cheating has nothing to do with the actual quality of the signaler, because cheaters can be of either high or low quality, but is rather related to the cost of the signal. Although some cheating is expected in evolutionarily stable signaling systems, the maintenance of such stability is conferred by signaling—which, on the average, is honest—and by a reduced fitness in cheaters (see Grafen 1990*a*, 1990*b*).

In the context of our hypothesis, the signal is the extent of secondary sexual development, the cost is immunosuppression, and genetic parasite resistance is what is being signaled. Cheaters do not pay the costs of immunosuppression in conjunction with an exaggerated secondary sexual development, regardless of their genetic quality. Thus, a cheater's signal loses all informational value because, no longer constrained by costs, a cheater's secondary sexual development does not honestly relate true genetic quality. Assuming heritability of cheating and discriminating ability, evolutionary stability is maintained because, regardless of the cheater's genetic quality and despite low short-term costs, average inclusive fitness is attenuated in cheaters and the nondiscriminating signal receiver. This occurs because the variation in genetic quality (i.e., parasite resistance) among a cheater's male offspring is not expressed in their signal. Thus, a cheater's male offspring with the most parasite-resistant genotype have no increased probability of being selected in sexual selection. Daughters inheriting the nondistinguishing trait would have a reduced inclusive fitness because they would choose mates with lower average genetic quality. On the average, genetic resistance to parasites, and thus inclusive fitness in the offspring of cheaters and nondiscriminating receivers, would be reduced relative to that of honest signalers and discriminating receivers. This difference between a cheater's inclusive fitness and that of an honest signaler could be expected to increase relative to the intensity of sexual selection and the detrimental effect of parasites. In summation, this honest (i.e., costly) signaling system for genetic parasite resistance is evolutionarily stable because genotypes coding for cheaters or nondiscriminating receivers would have reduced inclusive fitness because of the detrimental effects of parasitism on future generations relative to those that share an honest signaling system.

The proposed immunocompetence-handicap hypothesis is general enough to embrace ecological processes in mating systems with female choice based on outcomes of male-male competition. This generality depends on the obligatory nature of testosterone-dependent immunosuppression because of the issues raised earlier concerning cheating. The case of male-male competition is supported by the hypothesis if an individual assesses a potential opponent by the same viability-indicating criteria used by choosy females, as suggested by Zahavi (1987). Clearly, an individual that has devoted energy to full development of

secondary sexual characteristics before the mating season, displays a high level of testosterone-dependent aggression during the mating period, has risked the dire consequences of immunosuppression under this whole process, and is still vigorous and healthy during competition will be viewed as a formidable opponent. Freeland (1976) even suggested that females may incite male-male competition to reveal diseased males and thus avoid selecting an inferior mate. This would further ensure a high signal cost and thus provide for the maintenance of the signal's reliability.

CONCLUSIONS

The immunocompetence-handicap hypothesis is a proximate model that introduces several important concepts to the discourse on the role of parasitism and genetic resistance in the evolutionary process of sexual selection initiated by Hamilton and Zuk (1982) and on the evolutionary stability of signaling systems initiated by Zahavi (1975). An abundance of empirical evidence from vertebrate taxa suggests that secondary sexual development is sensitive to testosterone, which in turn compromises the immune system. We suggest the presence of a feedback loop between parasite burden and trait expression via this immunosuppression associated with signal development. The addition of this immunological effect provides a new explanation of how these signals impose the cost required for reliability and evolutionary stability in signaling systems (Grafen 1990*a*). Previously, hypotheses relied on energetic explanations of these costs (Halliday 1987). In this view, testosterone-dependent ornaments and display behaviors should be reliable indicators of heritable parasite resistance. However, testosterone's dual role may not be unique and any biochemical substance that exerts such an effect would fit the model assumptions. We further suggest that males regulate testosterone profiles to balance the competing demands of immunomodulation of parasite infections and promotion of the expression of secondary sexual characteristics. Additionally, intervening variables such as genetic quality, timing of infection, and nutritional status may complicate the relationship between parasite burden and signal intensity predicted under the Hamilton and Zuk hypothesis. We make the same general prediction as the original Hamilton and Zuk hypothesis while adding several important stipulations. Assuming genetic resistance, timing of infection, and nutritional status are equal, there should be a negative association between parasite burden and signal intensity on an intraspecific level. Additionally, we do not categorically exclude the role of secondary sex traits in reflecting other components of male quality (e.g., predator evasiveness and food-finding abilities; Andersson 1982, 1986; Partali et al. 1987; Slagsvold and Lifjeld 1988). Selective mechanisms can work in parallel resulting in similar outcomes, and this model describes a particular mechanism without being exclusive of other such mechanisms. Moreover, it has not escaped our attention that the immunocompetence handicap may have a far wider breadth of application. The hypothesis could be extended to embrace any kind of testosterone-related social behavior at one extreme and primary sexual development (e.g., spermatogenesis) at the other.

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