sampling) is based on considering the information-processing mechanisms involved in choice. The subjects remember the properties of each food supply with a degree of uncertainty and then make choices between samples taken from their memory for each source of reward, choosing always the sample that appears to lead to a better reward. The way uncertainty is included derives from what is known of perception of food amounts and time intervals. This model (Gibbon et al., 1988; Reboreda and Kacelnik, 1991) is described in full as applied to the present experiment and extended to more general cases by Kacelnik and Brito e Abreu (1998). It predicts risk aversion for desirable outcomes such as food amounts and risk proneness for aversive outcomes such as food delays. It also predicts partial preferences because in different trials sampling from memory yields different values for the two food supplies. We found a remarkable fit between the predictions of this model and the average results of our group of animals, but the fit is weaker when applied to each individual subject.

In conclusion, we found that starlings are persistently risk averse when amount variability is involved and that this is unaltered by energy-budget manipulations. These results, together with those of other studies showing that risk attitude for delays has the opposite sign and is equally resistant to budget manipulations, are consistent with the hypothesis that in natural environments maximization of mean rate of gain may be paramount, but that the mechanisms by which mean rates are computed may lead to paradoxical (not rate-maximizing) choices under experimental circumstances.

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REFERENCES

Bateson M, Kacelnik A, 1995a. Accuracy of memory for amount in the foraging starling (*Sturnus vulgaris*). Anim Behav 50:431–443.

Bateson M, Kacelnik A, 1995b. Preferences for fixed and variable food sources: variability in amount and delay. J Exp Anal Behav 63:313–329.

Bateson M, Kacelnik A, 1996. Rate currencies and the foraging starling: the fallacy of the averages revisited. Behav Ecol 7:341–352.

Bateson M, Kacelnik A, 1997. Starlings preferences for predictable and unpredictable delays to food. Anim Behav 53:1129–1142.

Bautista L, Tinbergen J, Wiersma P, Kacelnik A, 1998. Beyond optimal foraging: how starlings cope with changes in food availability. Am Nat 152:543–561.

Box GEP, Cox DR, 1964. The analysis of transformations (with discussion). J R Stat Soc B 26:211–252.

Caraco T, 1981. Energy budgets, risk and foraging preferences in dark-eyed juncos (*Junco hyemalis*). Behav Ecol Sociobiol 8:213–217.

Caraco T, Blanckenhorn WU, Gregory GM, Newman JA, Recer GM, Zwicker SM, 1990. Risk-sensitivity: ambient temperature affects foraging choice. Anim Behav 39:338–345.

Caraco T, Chasin M, 1984. Foraging preferences: response to reward skew. Anim Behav 32:76–85.

Caraco T, Kacelnik A, Mesnik N, Smulewitz M, 1992. Short-term rate maximization when rewards and delays covary. Anim Behav 44:441–447.

Caraco T, Martindale S, Whittam TS, 1980. An empirical demonstration of risk sensitive foraging preferences. Anim Behav 28:820–830.

Case DA, Nichols P, Fantino E, 1995. Pigeon's preference for variable-interval water reinforcement under widely varied water budgets. J Exp Anal Behav 64: 299–311.

Davison M, McCarthy D, 1988. The Matching Law: a research review. Hillsdale, New Jersey: Erlbaum Associates.

Gibbon J, Church RM, Fairhurst S, Kacelnik A, 1988. Scalar expectancy theory and choice between delayed rewards. Psychol Rev 95:102–114.

Gilliam JF, Green RF, Pearson NE, 1982. The fallacy of the traffic policeman: a response to Templeton and Lawlor. Am Nat 119:875–878.

Ha JC, Lehner PN, Farley SD, 1990. Risk-prone foraging behaviour in captive grey jays *Perisoreus canadensis*. Anim Behav 39:91–96.

Hamm SL, Shettleworth SJ, 1987. Risk aversion in pigeons. J Exp Psychol Anim B 13:376–383.

Herrnstein RJ, 1970. On the law of effect. J Exp Anal Behav 13:243–266.

Kacelnik A, Bateson M, 1996. Risky theories—the effects of variance on foraging decisions. Am Zool 36:402–434.

Kacelnik A, Bateson M, 1997. Risk-sensitivity: cross-roads for theories of decision-making. Trends Cog Sci 1:304–309.

Kacelnik A, Brito e Abreu F, 1998. Risky choice and Weber's Law. J Theor Biol 194:289–298.

Koops MA, Giraldeau LA, 1996. Producer-scrounger foraging games in starlings—a test of rate- maximizing and risk-sensitive models. Anim Behav 51:773–783.

Mazur JE, 1984. Tests of an equivalence rule for fixed and variable reinforcer delays. J Exp Psychol Anim Behav Proc 10:426–436.

McNamara JM, 1996. Risk-prone behaviour under rules which have evolved in a changing environment. Am Zool 36:484–495.

McNamara JM, Houston AI, 1992. Risk-sensitive foraging: a review of the theory. Bull Math Biol 54:355–378.

Reboreda JC, Kacelnik A, 1991. Risk sensitivity in starlings: variability in food amount and food delay. Behav Ecol 2:301–308.

Stephens DW, 1981. The logic of risk-sensitive foraging preferences. Anim Behav 29:628–629.

Tarpy R, 1997. Contemporary learning theory and research. New York: McGraw-Hill.

Templeton AR, Lawlor LR 1981. The fallacy of the averages in ecological optimization theory. Am Nat 117:390–393.

Vasquéz RA, 1995. Decision making in variable environments: individuals, groups and populations (D.Phil. thesis). Oxford: University of Oxford.

Stress, testosterone, and the immunoredistribution hypothesis

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Recent interest in parasites and sexual selection has focused attention on the paradox that the sexual displays which indicate parasite resistance in male vertebrates are triggered by testosterone, an apparently immunosuppressive hormone. We question the underlying assumption that testosterone is immunosuppressive and offer here the alternative of immunoredistribution to explain the changes in circulating leukocytes associated with male displays and elevated testosterone. First, we briefly examine three hypotheses that have attempted to resolve the testosterone immunosuppression paradox (Folstad and Karter, 1992; Hillgarth et al., 1997; Wedekind and Folstad, 1994). Although the immunoredistribution hypothesis under-

mines the premise of these hypotheses, there are other problems intrinsic to each one.

The immunocompetence handicap hypothesis

Folstad and Karter (1992) proposed the immunocompetence handicap hypothesis as an extension of Zahavi's (1975) handicap hypothesis for the evolution of secondary sexual characteristics. While Folstad and Karter's hypothesis offered an explanation for higher parasite loads in males than in females, it has been used to explain both correlation and lack of correlation between testosterone and reduction in indices of immunity. Specifically, if males with high levels of testosterone have higher indices of immunity or lower parasite loads, the interpretation would be that those males have such high-quality immune systems that they can overcome the immunosuppression of testosterone (Zuk, 1996). On the other hand, if high-testosterone males have higher parasite loads, the interpretation would be that those males are of such high quality overall that they can display and attract females despite higher infection due to immunosuppression (Salvador et al., 1996; Weatherhead et al., 1993). And if no relationship is found between testosterone and parasite loads, the argument is that the high-quality males ''are reliably signaling their resistance to parasites since they are still able to fend off parasites in the presence of high circulating levels of androgens'' (Saino and Moller, 1994:1331). The invocation of the immunocompetence handicap to support such contradictory trends undermines the utility of the hypothesis.

The resource allocation hypothesis

Wedekind and Folstad (1994) suggested that testosterone suppresses the immune system so that essential resources can be allocated instead to produce secondary sexual characteristics such as horns, songs, or stamina in repeatedly performing a display (Enstrom et al., 1997; Hunt et al., 1997). Although this would explain the adaptive trade-off underlying the immunocompetence handicap, it could also stand alone as an explanation for testosterone immunosuppression. However, Hillgarth and Wingfield (1997) pointed out that this explanation is unlikely to account for immunosuppression because the metabolic resources saved by suppressing immunity would be trivial compared to the associated risk of infection.

The sperm protection hypothesis

Hillgarth et al. (1997) offered the alternative hypothesis that the immunosuppressive effect of testosterone protects haploid spermatozoa, which are antigenic because they are formed long after the development of the immune system. Despite the partial protection of spermatocytes by Sertoli cells and the blood–testis barrier, some lymphocytes can pass into the seminiferous tubule (Turek and Lipshultz, 1994). Hillgarth et al. (1997) suggest that testosterone suppresses antibody production in order to protect antigenic sperm. Thus, testosterone could pleiotropically reduce antibody production in general when it is in the systemic circulation to regulate other secondary sexual characteristics. Penn and Potts (1998) question the likelihood of this hypothesis because there would be strong selection against sensitivity of immune cells to testosterone outside the testes. Although the sperm protection hypothesis might account for some reduced antibody-mediated immunity in high-testosterone males (Folstad and Skarstein, 1997), it still does not explain the change in numbers of circulating leukocytes associated with elevated testosterone.

None of the current explanations for immunosuppression by testosterone is particularly satisfying because of the huge selective cost of any significant impairment of immunity to pathogens. If immunity to parasites and disease is as important as Hamilton and Zuk's (1982) model suggests, suppression of immunity should be a rare and limited phenomenon. This has led us to question the underlying assumption that testosterone actually causes immunosuppression.

The immunoredistribution alternative

New insights into the immune response to stress offer an alternative explanation for the correlation between high levels of testosterone and changes in the immune system. We propose that leukocytes are temporarily shunted to different compartments of the immune system in response to testosterone, as they are in response to other steroids. This process, called immunoredistribution, would be easy to confuse with immunosuppression if immunity is assessed by leukocyte counts or if the measure of immunity is sampled at only one time or in only one tissue.

Unlike immunosuppression, redistribution is a temporary shifting of immune cells to compartments where they are likely to be more useful. This is far more than a semantic distinction. Immunosuppression implies that there is a single immune system which is inhibited from acting throughout the body and that lower cell counts result from elimination or reduced production of immune cells. Immunoredistribution is a quickly reversible relocation of immune cells to sites where they are most likely to be useful but perhaps less likely to be detected by researchers. Immunoredistribution is a well-documented response to stress and the associated elevation in circulating corticosteroids.

Stress and immunosuppression

Although stress-induced immunosuppression is assumed to be a common phenomenon, there are a number of systematic problems with the evidence and interpretation of it. First, Keller et al. (1992) point out that few of the studies that describe inhibition of immune cell function controlled for changes in the proportions of different types of immune cells, and thus may not have been measuring inhibition at all. Equally important is the empirical evidence that the endocrine and immune responses to stress are complex and depend on a number of factors, including the sex ratio and social composition of the group under study (Sapolsky, 1986; Taylor et al., 1987). These factors are typically not controlled or taken into account. When they have been controlled, no suppression of immune cell activity was found (Klein et al., 1992). In addition, acute and chronic triggers are often lumped together. Despite these problems and the fact that responses may differ in different species, we do not question the evidence of suppression of immune cell activity under pharmacological doses of steroids. However, these examples only demonstrate that the activity of immune cells can be artificially suppressed, not that suppression is a normal physiological response to corticosteroids or testosterone.

Stress and immunoredistribution

Immunoredistribution is an alternative explanation for the apparent immunosuppressive effect of stress. Environmental stress and social stress are both known to increase the level of circulating corticosteroids which in turn affect the immune system (Harbuz and Lightman, 1992; Morrow-Tesch et al., 1994; Taylor et al., 1987). This common mechanism is due to activation of the hypothalamus-hypophysis-adrenal axis. In response to the elevated corticosteroid levels, leukocytes exit peripheral blood circulation and enter lymph nodes, skin, and

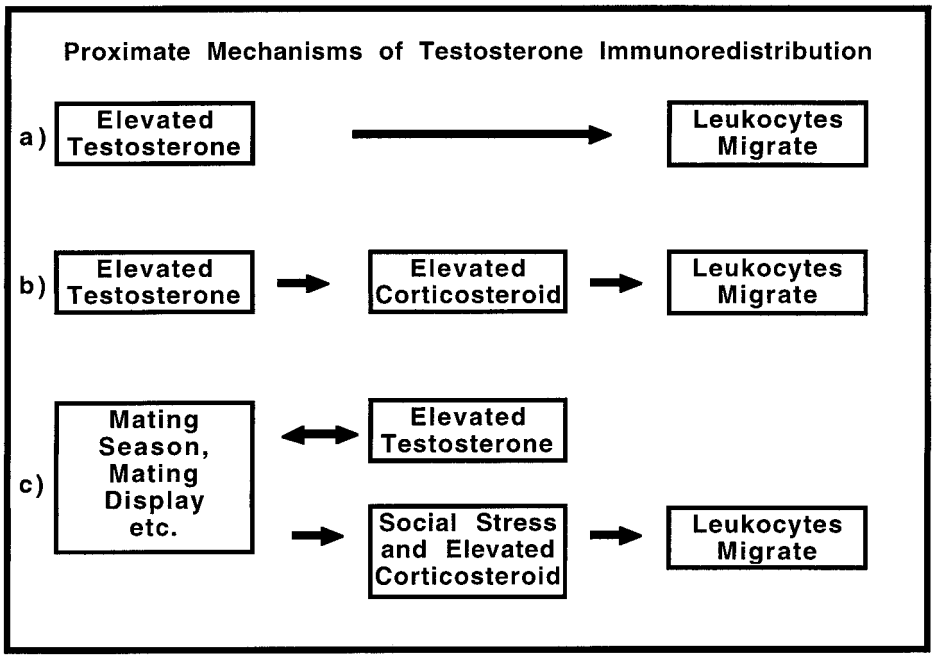

Figure 1
In addition to the two mechanisms by which testosterone is likely to trigger immunoredistribution (a and b), it is also possible that increases in testosterone correlate with redistribution but are not a direct cause of the phenomenon (c).

other tissues where they are well positioned to combat challenges from new trauma (Dhabhar, 1998; Dhabhar and McEwen, 1996). Circulating leukocytes then return to normal levels within a few hours after the stress ceases (Dhabhar et al., 1995).

Dhabhar has provided the clearest demonstration that temporary immunoredistribution associated with stress is triggered by corticosteroids (Dhabhar, 1998; Dhabhar and McEwen, 1996, 1997; Dhabhar et al., 1994, 1995, 1996), but the phenomenon of immunoredistribution has been recognized as an important trade-off response for at least the past 25 years. Fauci (1975) had already shown that corticosteroid treatment in guinea pigs leads to a temporary lowering of lymphocytes in peripheral circulation as they migrate to bone marrow. Chung et al. (1986) further showed that glucocorticosteroids in mice cause lymphocytes to migrate to peripheral lymph nodes and bone marrow. In fact, the temporary redistribution of various leukocytes in response to hormone stimulation is well documented (Claman, 1972; Cohen, 1972; Dhabhar, 1998; Dhabhar and McEwen, 1996, 1997; Dhabhar et al., 1994, 1995, 1996; Fauci and Dale, 1974; Gross, 1990; Landmann et al., 1984; Lundin and Hedman, 1978; Moorhead and Claman, 1972; Schedlowski et al., 1993 ; Spry, 1972) and can even be traced back to Dougherty and White (1944).

The testosterone immunoredistribution hypothesis

We propose, testosterone has a similar effect on the immune system to corticosteroids (i.e., immunoredistribution rather than suppression). Not only would suppression of the whole immune system be wasteful and maladaptive, but redistribution of immune cells would be an extremely valuable adaptive trade-off (Dhabhar, 1998). Just as emotional stress is often a good indicator of imminent trauma and immunochallenge, the testosterone surge in a displaying and competing male is also a good predictor of potential injury and immunochallenge. Male–male competitions are often not orderly, ritualized battles that avoid actual fighting. Rather, males in a wide range of species are often seriously injured, even killed, in competition over mates (Clutton-Brock, 1982; Cox, 1981; Geist, 1966, 1974; Silverman and Dunbar, 1980; Wilkinson and Shank, 1977). Moreover, courtship interactions are always likely to involve some danger and stress because of the unpredictability of a potential mate's response (Hinde 1953,1954). Therefore, we should expect immune resources to be temporarily redeployed to sites of potential injury.

We envision three possible mediating mechanisms for testosterone-mediated immunoredistribution. First, testosterone may directly activate immunoredistribution by binding to receptors in leukocytes or endothelium, thereby triggering migration to specific tissues (Figure 1a). Such receptors for steroid hormones have already been identified (Cupps and Fauci, 1982; Fox, 1995; Dhabhar, 1998). Second, testosterone may enhance corticosteroid levels which, in turn, trigger immunoredistribution (Figure 1b). Ketterson et al. (1991) and Ketterson and Nolan (1992) have demonstrated that experimentally elevated testosterone in dark-eyed juncos causes elevated corticosterone levels. Johnsen (1998) also found that seasonal elevations of corticosteroids and testosterone were significantly correlated. Finally, high testosterone levels may merely correlate with high corticosteroid levels due to the stress associated with male–male competition or courtship (Figure 1c). For example, the changes in circulating leukocyte frequencies in high-testosterone red jungle fowl males reported by Zuk et al. (1995) are well-known responses to stress and corticosteroids in domestic chickens (Gross and Siegel, 1983, 1985).

Although there is evidence that stress and cortisol inhibit testosterone under some situations (Rivier and Vale, 1984), testosterone levels do correlate with corticosteroid levels under more relevant circumstances, such as in the presence of reproductive females (McDonald et al., 1986; Silverin, 1998; Taylor et al., 1987). In addition, Sapolsky (1986) suggests that corticosteroids have different effects on testosterone level depending on a male's status in the group.

Our model contradicts what many believe to be settled science: that testosterone is immunosuppressive. The concept of immunosuppression by testosterone has been broadly accepted in the behavioral ecology literature and can be traced back to a number of reviews by Grossman (1984, 1985) and Alexander and Stimson (1988). These reviews have been repeatedly cited as the authority for the phenomenon of immunosuppression by testosterone (Folstad and Karter, 1992; Gal-

eotti et al., 1997; Hillgarth and Wingfield, 1997; Ros et al., 1997; Saino and Moller, 1994; Saino et al., 1995; Wedekind and Folstad, 1994; Zuk, 1994; Zuk and McKean, 1996). However, Grossman (1984, 1985) and Alexander and Stimson (1988) discuss the varying effects of steroids on different components of the immune system and are careful not to imply that all elements of immunity are stimulated or suppressed. They also review evidence for enhancement of different measures of immunity by both androgens and estrogens. The lack of strong evidence for immunosuppression by testosterone was recently noted by Hilgarth and Wingfield (1997), but they concluded that this points to a need for more research, rather than a need to question whether immunosuppression exists.

The reason for originally suspecting that testosterone is immunosuppressive appears to have come from the epidemiological finding that males suffer higher rates of infection and disease than females. However, Zuk (1990) pointed out that many incidences of disease and mortality in the males of many species are due to indirect effects of testosterone on behavior. For example, males often engage in riskier behaviors and are thereby exposed to different infectious agents than females. Hence, Zuk and McKean (1996) distinguish ecological from physiological causes in the sex differences in infection. In addition, we suggest that the sex differences in the rates of certain diseases may be due to the action of testosterone if redistribution leaves respiratory, digestive, or other systems less protected from infection. For example, Deerenberg et al. (1997) found that there was a lower immune response to an antigenic agent injected into the abdominal cavities of zebra finches during work or brooding stress. Similar reduced protection of the abdominal and thoracic organs due to immunoredistribution is also likely to result from increased testosterone. However, this would be a secondary effect of testosterone and not immunosuppression.

Mounting evidence against testosterone immunosuppression

There is a wide array of published data on enhancement of immunity correlated with testosterone. For example, Dunlap and Schall (1995) found that uninfected male fence lizards had unexpectedly higher levels of testosterone than infected males; Zuk et al. (1995) found that monocytes, heterophils and eosinophils all increased with increasing testosterone in red jungle fowl. Klein and Nelson (1998) found that testosterone correlated with higher specific immune response in voles. Ros et al. (1997) found that testosterone-treated male gulls had increased antibody titers. Although these are confounding results for any immunosuppression model, they are consistent with an immune system that temporarily redistributes its immune resources.

Predictions and tests

The suppression and redistribution models offer different explanations for phenomena such as reduction in circulating leukocytes and a wide range of different predictions follows from each hypothesis (Table 1). On the other hand, we share the prediction of the suppression model in expecting a priori to find similar effects of chronic and acute elevation of testosterone. Our expectation follows from the idea that testosterone would be a reliable predictor of potential trauma, whether acute or chronic. However, the immune response to chronic and acute stress may differ (Dhabhar, 1998; Dhabhar and McEwen, 1997). Therefore, if the effect of testosterone on immunoredistribution is mediated by corticosteroids (Figure 1b) or is correlated with stress (Figure 1c), there may be differences in the response to chronic and acute elevations of testosterone.

Table 1
Predictions of the testosterone immunosuppression and the testosterone immunoredistribution models

Type of response to elevated testosterone	Immunosuppression	Immunoredistribution
Leukocyte counts in blood and tissue	Decrease	No change in total number of leukocytes: quickly reversible decrease in source tissue(s) is accounted for by increase in sink tissue(s)
Response to acute elevation in T (e.g., during territorial display, mating display, or competition	Decrease in number and activity of leukocytes	No change in total number of leukocytes: quickly reversible decrease in source tissue(s) is accounted for by increase in sink tissue(s)
Response to chronic elevated T (e.g., mating seasonal elevation)	Persistent decrease in numbers and activity of leukocytes	Redistribution may revert to baseline after an initial period if the mechanism of immunoredistribution is modulated by corticosteroids
Dermal response	Decrease	Increase because skin is an expected sink for redistribution to help reduce the risk of infection in the event of injury
Rate of respiratory, digestive, and parasitic infection	Increase due to suppression of immunity throughout the body	Increase because redistribution may result in a trade-off of reduced protection of some systems while enhancing protection against infection at wounds
Infection of wounds	Increase	Decrease
Activity of leukocytes	Decrease	Increase

To determine whether acute changes in testosterone levels result in males suffering from immunosuppression or benefiting from temporary immunoredistribution, one must time sample the indices of immune response. Dhabhar et al. (1995) found that a variety of leukocytes returned to baseline levels 3 h after stress treatments ended. Therefore, it would be crucial to test whether testosterone and circulating leukocytes experience parallel changes over time in displaying and competing males.

More specifically, we expect that some leukocytes are differentially locating to potential sources of injury and that peripheral immunity should increase with testosterone. Resistance to cutaneous infection should increase with testosterone, perhaps by the migration of macrophages and neutrophils which would best enhance defense against bacterial infection of a wound. Thus, we expect an enhanced dermal response in high-testosterone males similar to that found in stressed individuals (Dhabhar and McEwen, 1996).

Although the redistribution hypothesis specifically predicts changes in circulating leukocyte populations, it is founded on a general expectation that suppression is extremely maladaptive and should not be a common phenomenon (outside of normal negative feedback regulation of immunostimulation). Consequently, we would also expect enhancement of antibody-mediated immunity in response to elevated testosterone

as has been found in response to stress (Dhabhar and McEwen, 1996).

We look forward to testing these predictions experimentally in the immediate future. Until then, we believe that this hypothesis will offer a useful framework for interpreting the apparently confounding data collected by others who are currently examining the interactions between testosterone, immunity, and sexual selection.

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REFERENCES

Alexander J, Stimson WH, 1988. Sex hormones and the course of parasitic infection. Parasitol Today 4(7):189–193.

Chung H, Samlowski W, Daynes R, 1986. Modification of the murine immune system by glucocorticosteroids: alterations of the tissue localization properties of circulating lymphocytes. Cell Immunol 101: 571–585.

Claman HN, 1972. Corticosteroids and lymphoid cells. N Engl J Med 287(8):388–397.

Clutton-Brock T, 1982. The red deer of Rhum. Nat Hist 91:42–47.

Cohen J, 1972. Thymus-derived lymphocytes sequestered in the bone marrow of hydrocortisone-treated mice. J Immunol 108:841–844.

Cox C, 1981. Agonistic encounters among male elephant seals: frequency, context, and the role of female preference. Am Zool 21: 197–209.

Cupps T, Fauci A, 1982. Corticosteroid-mediated immunoregulation in man. Immunol Rev 65:133–155.

Deerenberg C, Arpanius V, Daan S, Bos N, 1997. Reproductive effort decreases antibody responsiveness. Proc R Soc Lond B 264:1021–1029.

Dhabhar F, 1998. Stress-induced enhancement of cell mediated immunity. Ann NY Acad Sci 840:359–372.

Dhabhar F, McEwen B, 1996. Stress-induced enhancement of antigen-specific cell-mediated immunity. J Immunol 156:2608–2615.

Dhabhar F, McEwen B, 1997. Acute stress enhances while chronic stress suppresses cell-mediated immunity in vivo: a potential role for leukocyte trafficking. Brain Behav Immunity 11:286–306.

Dhabhar F, Miller A, McEwen B, Spencer R, 1995. Effects of stress on immune cell distribution: dynamics and hormonal mechanisms. J Immunol 154:5511–5527.

Dhabhar F, Miller A, McEwen B, Spencer R, 1996. Stress-induced changes in blood leukocyte distribution. J Immunol 157:1638–1644.

Dhabhar F, Miller A, Stein M, McEwen B, Spencer R, 1994. Diurnal and acute stress-induced changes in distribution of peripheral blood leukocyte subpopulations. Brain Behav Immunity 8:66–79.

Dougherty T, White A, 1944. Influence of hormones on lymphoid tissue structure and function. The role of the pituitary adrenotropic hormone in the regulation of the lymphocytes and other cellular elements of the blood. Endocrinology 35:1–14.

Dunlap KD, Schall JJ, 1995. Hormonal alterations and reproductive inhibition in male fence lizards (*Sceloporus occidentalis*) infected with the malarial parasite *Plasmodium mexicanum*. Physiol Zool 68: 608–621.

Enstrom D, Ketterson E, Nolan V, 1997. Testosterone and mate choice in the dark-eyed junco. Anim Behav 54:1135–1146.

Fauci AS, 1975. Mechanisms of corticosteroid action on lymphocyte subpopulations. I. Redistribution of circulating T and B lymphocytes to the bone marrow. Immunology 28: 669–680.

Fauci AS, Dale DC, 1974. The effect of in vivo hydrocortisone on subpopulations of human lymphocytes. J Clin Invest 53:240–246.

Folstad I, Karter A, 1992. Parasites, bright males, and the immunocompetence handicap. Am Nat 139:603–622.

Folstad I, Skarstein F, 1997. Is male germ line control creating avenues for female choice? Behav Ecol 8:109–112.

Fox H, 1995. Sex steroids and the immune system. In: Non-reproductive actions of sex steroids (Bock G, Goode J, eds) (Ciba Foundation Symposium 191). Chichester: Wiley. 203–217.

Galeotti P, Saino N, Sacchi R, Moller AP, 1997. Song correlates with social context, testosterone and body condition in male barn swallows. Anim Behav 53:687–700.

Geist V, 1966. The evolution of horn-like organs. Behaviour 27:175–213.

Geist V, 1974. On fighting strategies in animal conflict. Nature 250: 354.

Gross WB, 1990. Effect of exposure to short duration sound on the stress response in chickens. Avian Dis 34:759–761.

Gross WB, Siegel HS, 1983. Evaluation of the heterophil/lymphocyte ratio as a measure of stress in chickens. Avian Dis 27:972–979.

Gross WB, Siegel HS, 1985. Selective breeding of chickens for corticosterone response to social stress. Poultry Sci 64: 2230–2233.

Grossman C, 1984. Regulation of the immune system by sex steroids. Endocr Rev 5(3):435–455.

Grossman C, 1985. Interactions between the gonadal steroids and the immune system. Science 227:257–261.

Hamilton W, Zuk M, 1982. Heritable true fitness and bright birds: a role for parasites? Science 219: 384–387.

Harbuz MS, Lightman SL, 1992. Stress and the hypothalamo-pituitary-adrenal axis: acute, chronic and immunological activation. J Endocrinol 134:327–339.

Hillgarth N, Ramenofsky M, Wingfield J, 1997. Testosterone and sexual selection. Behav Ecol 8:108–109.

Hillgarth N, Wingfield J, 1997. Parasite-mediated sexual selection: endocrine aspects. In: Host-parasite evolution: general principles and avian models (Clayton D, Moore J, eds.) Oxford: Oxford University Press; 78–104.

Hinde RA, 1953. The conflict between drives in the courtship and copulation of the chaffinch. Behaviour 5:1–31.

Hinde RA, 1954. Courtship and copulation of the green finch (*Chloris chloris*). Behaviour 7:207–230.

Hunt K, Hahn T, Wingfield J, 1997. Testosterone implants increase song but not aggression in male Lapland longspurs. Anim Behav 54:1177–1192.

Johnsen T, 1998. Behavioral correlates of testosterone and seasonal changes of steroids in red-winged blackbirds. Anim Behav 55:957–965.

Keller S, Weiss J, Schleifer S, Miller N, Stein M, 1992. Suppression of immunity by stress: effect of a graded series of stressors on lymphocyte stimulation in the rat. Science 213:1397–1400.

Ketterson E, Nolan V, 1992. Hormones and life histories: an integrative approach. Am Nat 140:S33-S62.

Ketterson ED, Nolan V, Wolf L, Ziegenfus C, Dufty AM, Ball GF, Johnsen TS, 1991. Testosterone and avian life histories: the effect of experimentally elevated testosterone on corticosterone and body mass in dark-eyed juncos. Hormones Behav 25:489–503.

Klein F, Lemaire V, Sandi C, Vitiello S, Van der Logt J, Laurent P, Neveu P, Le Moal M, Mormede P, 1992. Prolonged increase of corticosterone secretion by chronic social stress does not necessarily impair immune functions. Life Sci 50:723–731.

Klein S, Nelson R, 1998. Adaptive immune responses are linked to the mating system of arvicoline rodents. Am Nat 151:59–67.

Landmann R, Muller F, Perini C, Wesp M, Erne P, Buhler F, 1984. Changes of immunoregulatory cells induced by psychological and physical stress: relationship to plasma catecholamines. Clin Exp Immunol 58:127–135.

Lundin P, Hedman L, 1978. Influence of corticosteroids on lymphocyte recirculation. Lymphology 11:216–221.

McDonald I, Lee A, Than K, Martin R, 1986. Failure of glucocorticoid feedback in males of a population of small marsupials (*Antechinus swainsonii*) during the period of mating. J Endocrinol 108:63–68.

Moorhead J, Claman H, 1972. Thymus derived lymphocytes and hydrocortisone: identification of subsets of theta-bearing cells and redistribution to bone marrow. Cell Immunol 5:74–86.

Morrow-Tesch J L, McGlone J, Salak-Johnson J, 1994. Heat and social stress effects on pig immune measures. J Anim Sci 72:2599–2609.
Penn D, Potts W, 1998. Chemical signals and parasite-mediated sexual selection. Trends Ecol Evol 13:391–396.
Rivier C, Vale W, 1984. Influence of corticotropin releasing factor on reproductive functions in the rat. Endocrinology 114:914–921.
Ros AFH, Groothuis TGG, Apanius V, 1997. The relation among gonadal steroids, immunocompetence, body mass, and behavior in young black headed gulls (*Larus ridibundus*). Am Nat 150:201–219.
Saino N, Moller AP, 1994. Secondary sexual characters, parasites and testosterone in the barn swallow, *Hirundo rustica*. Anim Behav 48: 1325–1333.
Saino N, Moller AP, Bolzern AM, 1995. Testosterone effects on the immune system and parasite infestations in the barn swallow (*Hirundo rustica*): an experimental test of the immunocompetence hypothesis. Behav Ecol 6:397–404.
Salvador A, Veiga J, Martin J, Lopez P, Abelenda M, Puerta M, 1996. The cost of producing a sexual signal: testosterone increases the susceptibility of male lizards to ectoparasitic infestation. Behav Ecol 7:145–150.
Sapolsky R, 1986. Stress-induced elevation of testosterone concentrations in high ranking baboons: role of catecholamines. Endocrinology 118:1630–1635.
Schedlowski M, Falk A, Rohne A, Wagner T, Jacobs R, Tewes U, Schmidt R, 1993. Catecholamines induce alterations of distribution and activity of human natural killer (NK) cells. J Clin Immunol 13: 344–351.
Silverin B, 1998. Territorial behavior and hormones of pied flycatchers in optimal and suboptimal habitats. Anim Behav 56:811–818.
Silverman HB, Dunbar MJ, 1980. Aggressive tusk use by the narwhal, *Monodon monoceros*. Nature 284:57–58.
Spry C, 1972. Inhibition of lymphocyte recirculation by stress and corticotropin. Cell Immunol 4:86–92.
Taylor G T, Weiss J, Rupich R, 1987. Male rat behavior, endocrinology and reproductive physiology in a mixed-sex, socially stressful colony. Physiol Behav 39:429–433.
Turek P, Lipshultz L, 1994. Immunologic infertility. Urol Clin North Am 21:447–468.
Weatherhead P, Metz K,. Bennett G, Irwin R, 1993. Parasite faunas, testosterone and secondary sexual traits in male red-winged blackbirds. Behav Ecol Sociobiol 33:13–23.
Wedekind C, Folstad I, 1994. Adaptive and non-adaptive immunosuppression by sex hormones. Am Nat 143: 936–938.
Wilkinson PF, Shank CC, 1977. Rutting-fight mortality among musk oxen on Banks Island, Northwest Territories, Canada. Anim Behav 24:756–758.
Zahavi A, 1975. Mate selection—a selection for a handicap. J Theor Biol 53:205–214.
Zuk M, 1990. Reproductive strategies and disease susceptibility: an evolutionary viewpoint. Parasitol Today 6(7):231–233.
Zuk M, 1994. Immunology and the evolution of behavior. In: Behavioral mechanisms in evolutionary ecology (Real L, ed.). Chicago: University of Chicago Press; 354–368.
Zuk M, 1996. Disease, endocrine-immune interactions, and sexual selection. Ecology 77:1037–1042.
Zuk M, Johnsen T, Maclarty T, 1995. Endocrine-immune interactions, ornaments and mate choice in red jungle fowl. Proc R Soc Lond B 260:205–210.
Zuk M, McKean KA, 1996. Sex differences in parasite infections: patterns and processes. Intl J Parasitol 26:1009–1024.