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Infectious diseases, reproductive effort and the cost of reproduction in birds

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SUMMARY

Reproductive effort can have profound effects on subsequent performance. Field experiments on the collared flycatcher (*Ficedula albicollis*) have demonstrated a number of trade-offs between life-history traits at different ages. The mechanism by which reproductive effort is mediated into future reproductive performance remains obscure. Anti-parasite adaptations such as cell-mediated immunity may probably also be costly. Hence the possibility exists of a trade-off between reproductive effort and the ability to resist parasitic infection. Serological tests on unmanipulated collared flycatchers show that pre-breeding nutritional status correlates positively with reproductive success and negatively with susceptibility to parasitism (viruses, bacteria and protozoan parasites). Both immune response and several indicators of infectious disease correlate negatively with reproductive success. Similar relations are found between secondary sexual characters and infection parameters. For brood-size-manipulated birds there was a significant interaction between experimentally increased reproductive effort and parasitic infection rate with regard to both current and future fecundity. It seems possible that the interaction between parasitic infection, nutrition and reproductive effort can be an important mechanism in the ultimate shaping of life-history variation in avian populations.

1. INTRODUCTION

That reproduction is costly constitutes a core assumption in life history theory (Williams 1966*a*,*b*). The idea that reproduction competes with other functions within the individual is as old as biology itself and can be illustrated by the idea of a Darwinian demon. A Darwinian demon can be characterized as an organism that produces an infinite number of offspring, lives for ever and as a consequence does not recognize any cost of reproduction or of living (we are among many that would envy such an organism).

A Darwinian demon simultaneously maximizes life-history traits such as longevity, breeding frequency, size at birth, rate of growth, and number and size of offspring. However, life-history theory predicts that organisms should face a trade-off between investment in current and future reproduction (Williams 1966*a*,*b*). This would be manifested via several intermediate trade-offs, such as current reproduction and survival, current and future reproduction, and number and quality of offspring.

Trade-offs can be either physiological or evolutionary. By physiological trade-offs we mean the allocation between two processes competing for the same limited resources within a single individual. Evolutionary trade-offs are genetically based and can be demonstrated by selection experiments on phenotypes and the correlated response in subsequent generations. Physiological trade-offs as well as ecologically mediated trade-offs (see, for example, Partridge 1987) are the background of all evolutionary trade-offs; this paper mainly considers physiological trade-offs.

Orton (1929) suggested that the physiological costs of reproduction might explain the occurrence of natural death and senescence. When reproductive investment and the associated physiological costs become too high, the organism dies as a result of the deterioration of the body. Jönsson & Tuomi (1994) state that the potential for the physiological costs of reproduction having an evolutionary significance was first indicated by Fisher (1930), who pointed out two evolutionarily interesting aspects of trade-offs. Fisher was interested in the physiological mechanisms that allocate nutrition to either reproduction or maintenance and also under what circumstances one might find specific life histories.

The occurrence of trade-offs is not mysterious, but actually rather trivial. In life-history theory, the important issue is not whether any relations exist, as some are inevitable, but rather, which of the possible combinations occur and are strongest. Stearns (1992) discusses 45 possible trade-offs based on ten

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life-history traits. Precisely which combinations occur will depend on the interaction of selection pressures over evolutionary time and on the precise way in which physiological processes operate in a specific environment. The problem organisms face is to optimize different combinations. There is not one single solution to the problem; it will differ between populations and environments.

In most cases trade-offs can only be demonstrated convincingly by manipulating one of the involved traits. The reason for this is that individuals differ in their resources and in how these resources are allocated. This effect has been referred to as the silver spoon effect or the big house and big car syndrome (Van Noordwijk & de Jong 1986): to those that already have, more shall be given. Therefore, we need to break up the positive correlations between the life-history traits with phenotypic experiments (Gustafsson & Sutherland 1988). A number of studies have confirmed, for example, that experimentally increasing current reproductive effort results in decreased survival, future reproduction or offspring fitness (see reviews in Lessells 1991, Stearns 1992).

It is well known that nutritional status affects fecundity (Fisher 1930; Drent & Daan 1980) and that nutritional status is also important for defence against infection (Baron 1988). As early as 1871, Darwin proposed that the health and vigour of females determined both the date at which they breed and their fecundity. It is therefore important to measure both condition and disease in life-history studies. Possibly, individuals that spend more energy on current reproduction have relatively fewer resources to devote to immune defence against parasites; or individuals in lower condition that attempt to spend the same amount of resources on reproduction as those in higher condition are constrained to devote fewer resources to immune defence. These conjectures suggest a trade-off between reproductive effort and the ability to resist parasitic infections.

This paper reviews earlier and recent findings on reproductive effort, cost of reproduction and its link to infectious diseases from our long-term study of the collared flycatcher. We also give examples of, and point out the similarities between, effects of diseases and effort on both life-history characters and secondary sexual characters.

2. A CASE STUDY: LIFE HISTORY TRADE-OFFS IN THE COLLARED FLYCATCHER

(*a*) *The species*

The collared flycatcher has been studied since 1980 on the Baltic island of Gotland, where it forms an isolated population with a remarkably high site fidelity of both adults and young (Gustafsson 1985; Pärt & Gustafsson 1989) such that the lifetime reproductive success can be assessed with an accuracy rarely possible under natural conditions (Gustafsson 1989). Adult birds are caught with traps in the nest boxes, while feeding their young, and marked with unique numbered rings. Laying date, clutch size, hatching date, the number of hatched young and number of fledglings in each brood are recorded. Thirteen days after hatching, each young bird is uniquely ringed and its mass and tarsus length are measured. Adult survival and the number of offspring recruited into the breeding population are determined by catching all breeding pairs within the study area in subsequent years. Within the study area we have more than 900 nest boxes in nine woodlands which attract annually about 300–400 pairs of collared flycatchers. Eight deciduous woods are dominated by oak (*Quercus robur*) and ash (*Fraxinus excelsior*), with a dense understory of hazel (*Corylus avellana*) and hawthorn (*Crategus spp.*). The one area of coniferous forest is dominated by pine (*Pinus sylvestris*), with some birch (*Betula pubescens*).

The first male flycatchers arrive on Gotland in the first week of May and immediately take up territories. The latest arrive probably during the first week of June. The first females arrive, on average, one week after the earliest males. Nest building starts in early to mid-May and the first eggs are laid around 10–20 May so that most clutches hatch during the first half of June. Broods contain up to 8 young, which normally fledge on the fourteenth or fifteenth day after hatching. Most have left the nest by 5 July and the latest around 15 July. Only one clutch is laid, except for a few replacement clutches.

(*b*) *Brood size experiments*

In several different years (1983–5, 1988 and 1992–4) the broods of more than 1000 collared flycatchers were manipulated. Broods with the same hatching date were treated in pairs and either one or two young

Table 1. *The effect of experimentally increasing brood size on some life-history parameters for adult male and female flycatchers and offspring*

(Relationships are indicated as significantly positive (+), significantly negative (−) or non-significant (0); n.a., not applicable. Data are from Gustafsson & Sutherland (1988), Gustafsson & Pärt (1990), Schluter & Gustafsson (1993), L. Gustafsson & W. J. Sutherland, unpublished.)

	survival	fecundity	time of breeding	growth	age of first breeding	LRS
male	0	0	0	n.a.	n.a.	0
female	0	−	0	n.a.	n.a.	−
offspring	−	−	+	−	+	−

were, at two days of age, moved from one nest to the other, or for the control group two young were swapped between nests. The number of young added or removed was independent of the original number laid. Small-scale manipulations like these have the disadvantage that large sample sizes are needed to detect any effects, but the advantage that the achieved brood-size variation is similar to the natural heritable variation, which is crucial for a study to elucidate evolution.

The effects of manipulating brood size on life-history parameters are shown in table 1. The survival of both males and females was unaffected by the manipulation, but the subsequent fecundity of females was decreased by the manipulation. Furthermore, the brood size manipulation increased the number of fledged young. However, the growth of the nestlings was affected, such that larger broods resulted in lighter and smaller fledglings that were less likely to survive (Lindén *et al.* 1992). The combination of these trade-offs meant that females laying natural clutch sizes had the highest lifetime reproductive success (L. Gustafsson & W. J. Sutherland, unpublished). Consequently individuals seem, on average, to adjust their clutch to the optimal size.

Table 1 also gives examples of intergenerational trade-offs, that is the effect increased parental effort may have on the next generation. Both male and female offspring bred later in their life when they came from a brood that had been increased in size. Female offspring from such broods were less fecund which resulted in a reduced lifetime reproductive success for female offspring from larger broods (L. Gustafsson & W. J. Sutherland, unpublished).

Clutch size, as is the case with other life-history traits, is typically age-related (Gustafsson & Pärt 1990). It has been suggested that selection for high early fecundity is related to reduced later fecundity (Medawar 1952; Williams 1957, 1966*b*). This is supported by the observation that females that do not breed in their first year of life, and thereby have a lower early effort, have larger clutches later in life than those that bred in their first year (Gustafsson & Pärt 1990). This is confirmed by the effect of brood-size experiments on birds one year old. Those that had an increased brood had smaller clutches later in life than did the control birds. Consequently there is a trade-off between early and late reproduction (Gustafsson & Pärt 1990).

When the expectation of successful future reproduction at older ages is reduced by senescence, as it is in the collared flycatcher, life-history theory predicts that reproductive effort should increase with age. This is because, above certain ages, the trade-off between current and future reproduction should tip towards current and individuals are expected to make a terminal investment in reproduction (Clutton-Brock 1984). We (Pärt *et al.* 1992) investigated whether collared flycatchers tend to make a terminal investment in reproduction by comparing nestling feeding rates, energy expenditure (measured with the double-labelled water technique) and mass loss in females 5 years old compared with middle-aged females (e.g. 2–4 years old), which are at their peak performance in life (Gustafsson & Pärt 1990). Old females fed their young more frequently, consumed more energy and lost more mass. This increased investment resulted in higher mortality for old females with many young. It clearly demonstrates the effect of the allocation of resources between reproduction and survival, and indicates that there is a tendency to terminal investment.

3. MECHANISMS OF LIFE-HISTORY TRADE-OFFS

It is relatively easy to imagine how reproductive effort early in a season might affect effort later in the season (see, for example, Møller 1993, 1994), but how is an effort in one year mediated into a reproductive cost the following year? Figure 1 shows the annual cycle of the collared flycatcher and the experimental and sampling periods. It also illustrates that an effort during egg-laying or when feeding the young must have an impact on the bird during moulting, during the migration to Africa in winter, and during the spring return migration. Finally (if the bird has survived) this impact is expressed during the following breeding season. To explore possible mechanisms, we make experiments during the laying period and the nestling period. We take blood samples from the birds when they arrive and later during the breeding period. Serological tests give us measures of the birds' condition and health before breeding, during breeding and after our experiments during the feeding period.

(*a*) *Breeding time, phenotypic quality and reproductive success*

What determines individual variation in breeding time? Darwin (1871) proposed that the health and vigour of females determined both the date at which they breed and their fecundity. Fecundity and reproductive success decline with season in the collared flycatcher. The earliest birds seem to match their laying date to anticipate the peak period of food abundance. Later and less fecund birds have fledged

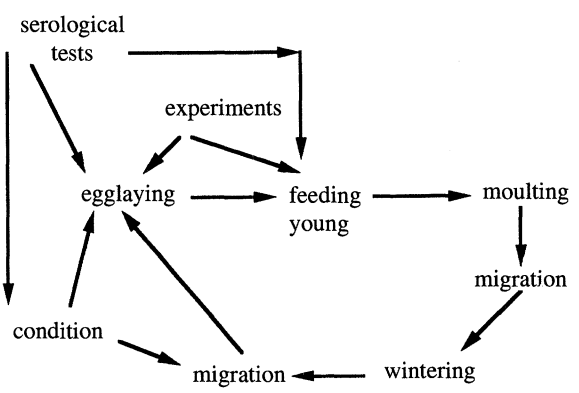

Figure 1. The annual cycle of the collared flycatcher and the experimental and sampling periods.

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their young when food is less abundant (L. Gustafsson, T. Pärt & A. Lundquist, unpublished). To separate the proximate effect of time *per se* from the phenotypic quality of the parents we (L. Gustafsson *et al.*, unpublished; Wiggins *et al.* 1994) experimentally made birds hatch one week earlier or later by swapping clutches that would have hatched at different dates. If parental quality were the main factor explaining reproductive success we predicted that we should not observe a change in success compared with control birds. If the main effect were due to time *per se*, reproductive success in the experimental group would decline in parallel with control birds.

The results were intermediate. For delayed birds the reproductive success was above that of the control birds (indicating higher parental quality) but below the expected value from their breeding time (indicating a presumable environmental effect of time *per se*). The same results were obtained for the advanced group: later birds were not as successful as early birds, but their earlier hatching increased their success compared with the late controls. This demonstrates that there is both an environmental (time *per se*) and parental quality effect on the seasonal decline in reproductive success in the collared flycatcher (L. Gustafsson, T. Pärt & A. Lundquist, unpublished).

(*b*) *Condition and reproductive success*

What is this quality effect? To be able to estimate condition in the period before breeding, we measured the amount of glycosylated haemoglobin when the birds arrived at the breeding ground (Andersson 1993; M. S. Andersson & L. Gustafsson, unpublished). Hazelwood (1972) and Bairlein (1983) studied the seasonal fluctuation of blood glucose levels in migratory birds. On the basis of their results we expect birds in bad condition to stop more frequently and for longer feeding periods during migration, and to have lower average blood glucose levels. Glycosylated haemoglobin reflects the average blood glucose level during a period of two to three weeks before the blood was sampled (Andersson 1993; M. S. Andersson & L. Gustafsson, unpublished).

The proportion of glycosylated haemoglobin decreased rapidly with arrival time, indicating that late birds had been in poor condition during migration. That was further supported by a positive relation between glycosylated haemoglobin and plasma protein, which is also an indication of nutritional status (Andersson 1993; M. S. Andersson & L. Gustafsson, unpublished). Furthermore, there was a positive relation between glycosylated haemoglobin and reproductive success when breeding time was controlled for (Andersson 1993; M. S. Andersson & L. Gustafsson, unpublished). This result shows that birds in good condition not only arrived early, but also produced more offspring than expected from their breeding date. This relationship could perhaps partly explain the difference in parental quality between early and late birds.

(*c*) *Infectious diseases and reproductive success*

What causes these differences in condition? Infectious diseases have been a neglected field in evolutionary ecology (see, for example, Sheldon 1993). Much recent work on host–parasite interactions has focused on macroparasites such as haematozoa and ectoparasites, which are relatively easily quantified (Loye & Zuk 1991). The impact that parasites have on their hosts is usually expressed in terms of pathogenicity or virulence of a specific type of parasite. However, the impact of a particular parasite on its host can never be generalized, because it depends to a large extent on the quality of the host's immune system, which is highly variable. We have used an alternative way of studying parasite–host interaction simply by measuring the activity of the birds' immune systems and using this as a guide to whether any parasite is currently causing disease in each individual bird. We performed several serological tests on blood samples collected from the birds. In concert these tests function as a screening procedure that detect disease processes rather than as specific diagnostic tests (Hunter 1989; Nordling 1993).

(i) *Serological tests*

The total white blood cell (WBC) count is the most widely used and perhaps the single most important serological test. It offers general information about infection status and can thus function as a screening test. Many different avian species respond to a wide range of pathogenic agents with increasing WBC counts (Dein 1986). In birds, this increase has been associated with infection by macroparasites and bacteria, including tuberculosis, as well as different viruses, such as herpes. However, the total WBC count is the most difficult parameter to measure in avian blood because of the interference of lysed red blood cells (RBC) and thrombocytes. To overcome this problem, we chose an alternative way of measuring WBC in this study. The amount of WBC was estimated from the height of the buffy coat layer in a hematocrit capillary tube (Wardlaw & Levine 1983) by using digital callipers after centrifugation. A portable microscope with a halogen light source was constructed for this purpose. For some samples the proportion of different WBC types was determined from counts of blood smears. A selective increase in different types of white blood cell indicates what type of parasite is likely to be the cause of infection.

The sedimentation rate (SR) of red blood cells is not a standard measure in avian diagnostics. However, it has been experimentally verified as a useful measure of infection intensity (Sharma *et al.* 1984). SR is highly species-specific and can only be informative if red blood cells sediment through plasma at an intermediate rate. This turns out to be the case in the collared flycatcher. The speed is very similar to the human SR. The SR depends on plasma factors as well as blood-cell factors. It is enhanced in a wide range of infectious and inflammatory disease owing to increasing concentrations of one of the major acute-phase proteins (fibrinogen) and immunoglobulins.

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The buffy layer (Wintrobe 1993) and the layer under it are useful areas for detection of blood parasites (G. F. Bennett, personal communication). Just above the buffy layer is the site where trypanosomes and microfilaria are encountered. In the buffy layer *Leucocytozoon* spp. and *Hepatozoon* may form a thin darker layer. The thin layer under the buffy coat may contain *Haemoproteus* and *Plasmodium* spp. (G. F. Bennett, personal communication). These parasite indicators were found to exist in the collared flycatchers in our study; blood smears from the buffy layer suggest a parasite prevalence of 60% (authors' unpublished results). The usual measurement of haematozoan parasite prevalence is made by counting parasites in ordinary blood smears. This suggests a parasite prevalence of about 20% in this population. Hence measurements based on ordinary blood smears may underestimate the prevalence of Haematozoa.

Proteins in general are not viewed as an energy store and are used only after both glycogen and lipid reserves are nearly or completely exhausted (Blem 1990). During times of heavy energy expenditure, such as feeding nestlings, the plasma protein concentration is likely to be very informative as a nutritional status parameter. Infectious and inflammatory diseases are another major cause of plasma protein degradation. Low concentrations signal disease. Standard spectrophotometric measurements were therefore made of plasma protein concentrations.

Different serum proteins involved in defence against infection, such as acute-phase proteins and immunoglobulins, were quantified by standard agarose gel electrophoresis. Typical patterns of infection determined from the plasma electrophoresis correlated strongly with our serological tests done in the field (figure 2 and authors' unpublished results).

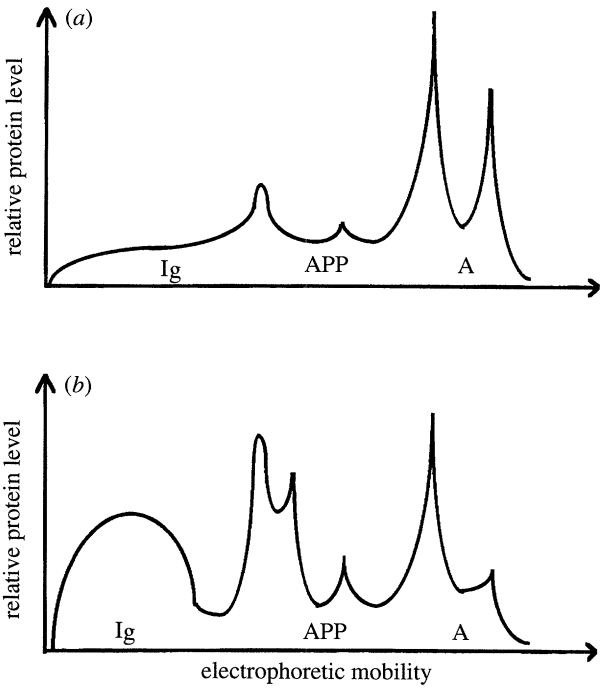

Figure 2. Schematic graph of results from standard agarose gel electrophoresis of plasma proteins performed to quantify different serum proteins (albumin A) acute-phase proteins (APP) and immunoglobulins (Ig) involved in infection defence. (*a*) A typical 'healthy collared flycatcher' (high levels of albumin, low levels of acute phase proteins and immunoglobulins): (*b*) a bird with strong signs of infectious disease (lower levels of albumin, higher levels of acute phase proteins and immunoglobulins).

(*d*) *Infectious diseases and reproductive performance*

That disease might negatively affect reproductive success seems obvious. More pertinent is the extent to which this mechanism is operating in natural populations. In the collared flycatcher we found that a number of measures of reproductive performance correlates significantly with all three infection parameters as well as with plasma protein concentrations in females (table 2). WBC counts, as expected, were related to parasite intensity. There was also a strong negative association between reproductive success and the immune response, including WBC counts, indicating overall that birds with low success had more infections and parasites. There was a particularly strong association for heterophil counts which indicate that bacteria may be important in this population, since bacterial infection results in increased levels of heterophils (Dein 1986).

An important determinant of reproductive success is time of breeding: first, food supply, especially caterpillar availability, declines quickly with season; second, good territories are occupied early in the season (L. Gustafsson, T. Pärt & A. Lundquist, unpublished). However, the causal basis for delayed breeding is poorly understood. The results in this study, with late birds showing high levels of all three infection parameters, suggest that disease might cause later breeding and also smaller clutch sizes. One can rule out the alternative suggestion that it is solely late breeding that causes disease (birds on poor territories may be exposed to more vectors, or have to look harder for food, thereby increasing susceptibility to parasites) because disease was also measured with the same result close to the arrival date, before these effects could have occurred. Because disease influences time of arrival and the start of breeding, there may also be additional physiological mecha-

Table 2. *Relations between condition and infection parameters and reproductive performance*

(Abbreviations: % HBG, proportion glycosylated haemoglobin; protein, plasma proteins; WBC, white blood cell count; SR, sedimentation rate; Ig, immunoglobulins. Data from Andersson (1993); Nordling (1993); L. Gustafsson, unpublished.)

	% HBG	protein	WBC	SR	Ig	heterophils
laying date	−	−	+	+	+	+
clutch size	+	+	−	0	−	−
number of fledged young	+	+	−	−	−	−

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nisms, caused by disease, that directly influence clutch size. If this is the case, the nutritional constraints on clutch size and reproductive success shown to correlate with a late start to breeding (L. Gustafsson, T. Pärt & A. Lundquist, unpublished) may be caused by direct influences from disease.

(*e*) *Diseases, reproductive effort and future reproduction*

We have seen earlier that present reproductive effort can have profound effects on subsequent reproductive performance. Such reproductive trade-offs clearly must be mediated physiologically (see, for example, Calow 1979; Stearns 1992) rather than circuitously through 'ecological mechanisms'; (see, for example, Partridge 1989). The exact nature of the mechanism, however, still remains obscure (see Gustafsson 1990; Lessells 1991). Anti-parasite adaptations based on, for example, cell-mediated immunity can certainly also be costly. As a consequence, a trade-off between reproductive effort and the ability to resist parasitic infection can be expected. Some experimental evidence indicates that birds that are artificially given larger clutches are more likely to become infected (Norris *et al.* 1994). Festa-Bianchet (1989) showed that female bighorn sheep (*Ovis canadensis*) that raised male offspring or that were lactating had higher faecal counts of lungworm larvae than females that raised female offspring or that were not lactating.

Serological tests on collared flycatchers whose brood sizes were manipulated (L. Gustafsson & D. Nordling, unpublished) showed that parasitic infection rate increased with brood size. Unsurprisingly, immune response also increased, perhaps directly owing to increasing infection rate by haematozoan parasites (see, for example, Norris *et al.* 1994) but perhaps owing to other infectious diseases.

These findings suggest that the results from previous brood-size experiments in the collared flycatcher, which demonstrated a link between high reproductive effort early in life and a reduced performance later, could be explained by a link between increased effort, increased infectious disease and reduced subsequent reproduction.

A recent analysis of the effect of experimentally increased effort in one year on fecundity in the following year shows that female collared flycatchers that are parasitized by Haematozoa lay relatively smaller clutches in the year following the experiment than females that are unparasitized in the year of the experiment, where both groups receive an increase in brood size (L. Gustafsson, B. C. Sheldon, D. Nordling, F. Widemo, T. Pärt & G. F. Bennett, unpublished). Within the subset of birds that were scored as not being infected by haematozoan parasites there was no apparent effect of brood-size manipulation on future fecundity, indicating that a decline in future fecundity due to experimental manipulation arose mainly through its effect on parasitized birds. We suggest that the interaction between the immune response to parasitism, nutritional status and the reproductive effort of birds subjected to brood-size manipulation may therefore be the mechanism through which the current versus future reproduction trade-off is mediated between years (figure 3*a*). This result also demonstrates that there are physiological mechanisms that can linger on throughout the annual cycle of the collared flycatcher (figure 1) and be manifested in the subsequent breeding.

4. DISEASES, REPRODUCTIVE EFFORT AND SECONDARY SEXUAL TRAITS

Parasitism and secondary sexual traits have recently been of major focus in sexual selection theory (Hamilton & Zuk 1982; Bradbury & Andersson 1987; Loye & Zuk 1991; Møller 1994). In many species, males display gaudy and elaborate ornaments, whereas females are relatively plain. It is common in these sexually dimorphic species that females show reduced expression of the same ornament as males (Darwin 1871). There is now general agreement that these display characters evolved in response to sexual selection (Bradbury & Andersson 1987; but see Alatalo *et al.* 1994). When a secondary sexual trait is costly to its bearer, it is possible that the causal path diagram of the factors affecting the expression of the trait is of a similar structure to that for life-history traits (figure 3*a*). For instance, diseases and condition may affect the size of the trait. The size of the trait affects effort, which affects the prevalence to diseases. This in turn affects the subsequent size of the trait (figure 3*b*).

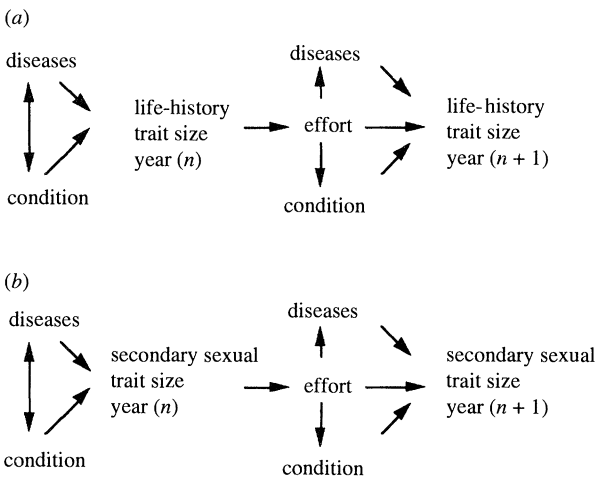

Figure 3. Causal path diagram of the effects on the expression of life-history (*a*) or secondary sexual (*b*) traits. We suggest that the interaction between infectious disease, nutritional status and the reproductive effort of birds subjected to brood-size manipulation may be the mechanism through which the current against future reproduction trade-off is mediated between years. We also suggest that the causal pathway for life-history traits is paralleled in secondary sexual traits where the same mechanisms might occur: diseases and condition affect the size of the trait and the size of the trait affects effort which in turn affects the prevalence to diseases. Finally those affect the subsequent size of the trait.

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Male collared flycatchers apparently use the white areas of their plumage as signals. The white forehead patch is displayed in male–male interactions and the extensive white patches on the wings are displayed when the male tries to attract a female to the nest hole. These white patches (badges) seem to be a reliable signal of male quality: males with large badges survive better and produce more recruited offspring (table 3). Birds in good condition have large white badges; the size of the badges correlates positively with the amount of plasma protein (table 3). Birds that have large white badges also seem to be less affected by parasites; the size of the patches correlates negatively with the WBC count (table 3). In this context a Darwinian demon should be in such good condition, and so healthy, that it would have turned completely white, and be able to displace all other males and to attract all females in the world! This is, of course, not the case. What are the trade-offs that prevent this from happening?

Does a large badge induce more effort? When we enlarged and reduced the badge in a paint experiment (A. Qvarnström, unpublished) there was a significant difference in the sedimentation rate of the red blood cells later in the breeding season. The males with enlarged badges had a higher sedimentation rate, which seems to indicate that they had developed more infections than the males with reduced badges. Interestingly, in unmanipulated males one year old there was a negative relation between the proportion of glycosylated haemoglobin and badge size, indicating that males one year old with large badges are in bad condition. Furthermore, such males also displayed a positive correlation between badge size and amount of WBC; these results also indicate that they had been exposed to more infections. This was not the case for males older than one year (two to four years), for which there was no relation between badge size and percentage glycosylated haemoglobin but a negative relation between badge size and amount of WBC. These results indicate that males one year old with large badges, as well as birds with experimentally increased badges, were having to expend more effort, possibly in male–male interactions.

This was also the case for males approaching the end of their life (those more than four years old). For these males there was a significant positive relation between badge size and amount of WBC, and a negative relation between badge size and percentage glycosylated haemoglobin, indicating that these males also apparently had too large a badge for their condition. This may be a parallel case to the terminal reproductive effort in relation to brood size discussed earlier (Pärt *et al.* 1992). In all three cases (young males with big patches, experimentally increased patches, ageing males with big patches) these relationships seem to indicate that these birds are displaying too large a badge for their condition, which possibly could result in increased morbidity and subsequent mortality. It is especially interesting that these results might offer a mechanistic explanation for how the honesty of the trait is maintained.

5. CONCLUSION AND PERSPECTIVES

The discussion of the effect of diseases on life-history traits and secondary sexual characters in an evolutionary context requires that there be some heritability of resistance to infections for natural selection to work on. Almost every organism must experience an infectious disease at least some time during its life (Price 1980). Defence against infections, including acquired immunity, can be modulated by environmental factors such as nutrition, stress and age. However, the genetic influence on the infection defence system, such as that resulting from the expression of major histocompatibility (MHC) haplotypes, sets the ultimate limits of reaction patterns in every individual. Accordingly, specific MHC alleles have been shown to be associated with resistance against disease (Van Eden *et al.* 1983; Hedrick 1994). One classic example concerns chickens, in which individuals with one specific haplotype have a much higher resistance to Marek's disease, a type of viral leukaemia (Briles *et al.* 1977). In West African children, malaria has selected for an unusually high incidence of rare MHC genes, which provide protection from severe malaria equal to the protection afforded by the sickle-cell haemoglobin variant (Hill *et al.* 1991). Some MHC B genes produce resistance to the bacterium causing fowl cholera (Lamont *et al.* 1987). The genetic variance in MHC haplotypes could be maintained by the cyclic nature of host–parasite interaction (Hamilton 1982; Anderson & May 1985; Hedrick 1994). The conclusion is that high immunological quality may offer a strong selective advantage with evolutionary consequences. Work in progress on diseases in the collared flycatcher involves the study of how MHC haplotypes interact with disease occurrence and its consequences, i.e. natural selection of genotypic variance in MHC.

As a first step towards demonstrating inheritance of

Table 3. *Associations between white plumage patterns and viability variables in male collared flycatchers*

(Abbreviations as for table 2; *x*, not measured separately. Data from L. Gustafsson, D. Nordling, M. Andersson & A. Qvarnström, unpublished.)

amount of white	survival	no. recruits	protein	% HBG	WBC
all males	+	+	+	0	−
1 year old	*x*	*x*	*x*	−	+
2–4 years old	*x*	*x*	*x*	0	−
5–7 years old	*x*	*x*	*x*	−	+

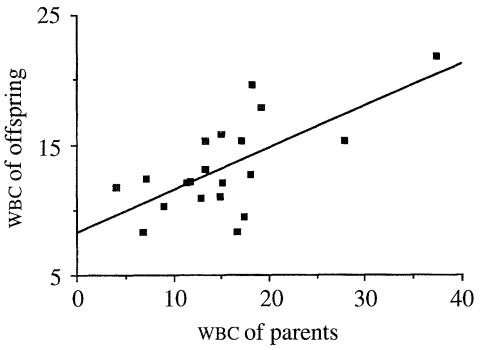

Figure 4. The parent–offspring regression of amount of white blood cells (WBC): $y = 8.30 + 0.33x$, $r^2 = 0.45$, $p < 0.001$. WBC was measured in the birds as adults.

the immune response, figure 4 shows the parent–offspring regression of amount of WBC. The amount of WBC was measured in the offspring during their first year of breeding. There is a surprisingly strong resemblance between offspring and their parents suggesting a simple heritability of *ca.* 0.66. However, beside the genetic contribution to the resemblance it must be borne in mind that there will probably also be maternal effects (Schluter & Gustafsson 1993) and possibly also common environmental effects (Gustafsson & Merilä 1994) that inflate this estimate.

In conclusion, it seems possible that the interaction between infectious diseases, nutrition and reproductive effort can be an important mechanism in the ultimate shaping of both life-history variation and secondary sexual traits in avian populations.

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