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REVIEW

Host-parasite interactions and life-history evolution*

Heinz Richner

Zoology Department, University of Bern, CH-3032 Hinterkappelen-Bern, Switzerland
Phone: +41-31 631 91 26; Fax: +41-31 631 91 41; e-mail: richner@esh.unibe.ch

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Summary

Many hosts and parasites are caught in an arms race. Reciprocal selection pressure can lead to sophisticated responses, and an important category of responses involves changes in the host's and parasite's life histories. Life-history traits, however, are often negatively intercorrelated and a change in one trait can only occur at the expense of another trait. Thus, parasites can act on the trade-off between life-history traits, and parasite-induced selection can therefore shift the trade-off from the optimum without parasites to a new optimum under parasitism. Trade-offs can occur at behavioural, morphological, and physiological levels.

Here I review our experimental work on a bird-ectoparasite system, the great tit, *Parus major*, and the hen flea, *Ceratophyllus gallinae*. I first evaluate the potential for coevolution based on the parasite's host specificity and virulence, and then show several host responses including parasite avoidance, changes in reproductive effort, and parasite-induced maternal effects. Finally, it is shown that the change in reproductive effort involves a trade-off with resistance to an important blood parasite, *Plasmodium* spp., causing bird malaria. It also provides a mechanism for the general trade-off between current and future reproduction in iteroparous organisms. A consideration of the consequences of host-parasite interactions for life-history evolution is important for our understanding of population processes, behavioural evolution, and for the design of efficient conservation strategies.

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Introduction

In the coevolutionary process parasites exert selection pressures on their hosts, and hosts on their parasites, leading to an arms race where both win and loose in turn. Winning is commonly not just a matter of gearing up ever more sophisticated arms, but also of adaptive changes in the hosts' and parasites' life-histories. Life-history changes as a coevolutionary defense strategy often imply the alteration of genetically based trade-offs in the allocation of time or resources. Thus, the coevolutionary process will lead to changes in both host and parasite genotype frequencies.

Since each host response selects in the parasite for a specific counter-strategy, and vice versa, it is often assumed that host-parasite coevolution with each host is less intense in a generalist parasite than for a parasite with a narrow host range. Selection pressure on the parasite, however, is rather determined by host specificity, that is the relative importance of each host, than simply by the range of hosts. Thus, high host specificity is a first, important prerequisite for rapid coevolution of hosts and parasites within a given host-parasite association.

A second prerequisite for parasite-induced selection leading to adaptive changes is that a parasite has to significantly affect the host's lifetime reproductive success. The degree of reduction in host fitness is often taken as a measure of the parasite's virulence (Read 1994). Survival to first reproduction, current reproductive success, and survival to

the next reproductive event are three important fitness components of iteroparous organisms. Since the three components are not independent of each other, a parasite-induced change in one component can affect other components both within individual hosts and among host generations. As an example, a parasite-induced change of the age at maturity may affect subsequent reproductive success of an individual host, or a parental compensation for the effects of a parasite on its offspring may affect the parents subsequent survival.

Here I review our experimental work on a bird-ectoparasite system, the great tit *Parus major*, and its most common ectoparasite, the hen flea *Ceratophyllus gallinae*. I will first assess the experimental and comparative evidence concerning the hen flea's host specificity, and the fitness consequences of hen flea parasitism for the host, and then analyse some of the parasite-induced host responses and their life-history consequences for the host.

Prerequisites for host-parasite coevolution

Host specificity: coevolution with a 'generalist' ectoparasite

The common hen flea *Ceratophyllus gallinae* (Schrank) has been found on the bodies or in the nest of over sixty host species (Rothschild 1952, Smit 1957). If *C. gallinae* is a true generalist, rapid coevolution with any particular host species is unlikely, and diffuse coevolution with its several hosts (Futuyma and Slatkin 1983) could be expected instead. The coevolutionary potential of *C. gallinae* with a given host depends on parasite specificity (Holmes *et al* 1977, Rohde 1980), and requires an assessment of the relative prevalence and intensity of infestation for each host species, and the relative abundance of each host species.

C. gallinae breeds during the host's nesting period where it uses the adults and young for regular blood meals. The larvae develop in the nest material and feed on detritus and undigested blood excreted by the parents (Marshall 1981, Lehane 1991). When the nestlings fledge, most flea larvae spin cocoons, pupate, and the imagoes remain quiescent in the cocoons until the next spring (Humphries 1967, Tripet and Richner in press a). Thus the majoritiy of the new flea generation overwinters in cocoons in the abandoned nests (Heeb *et al* 1996, Tripet and Richner in press b). The highest prevalence p (= percentage of infested nests) and intensity i (= mean number of fleas overwintering in infested nests) of flea infestation occurs in hole-nesting bird species (black circles in Fig. 1), i.e. in forest communities. Among the studies surveyed in

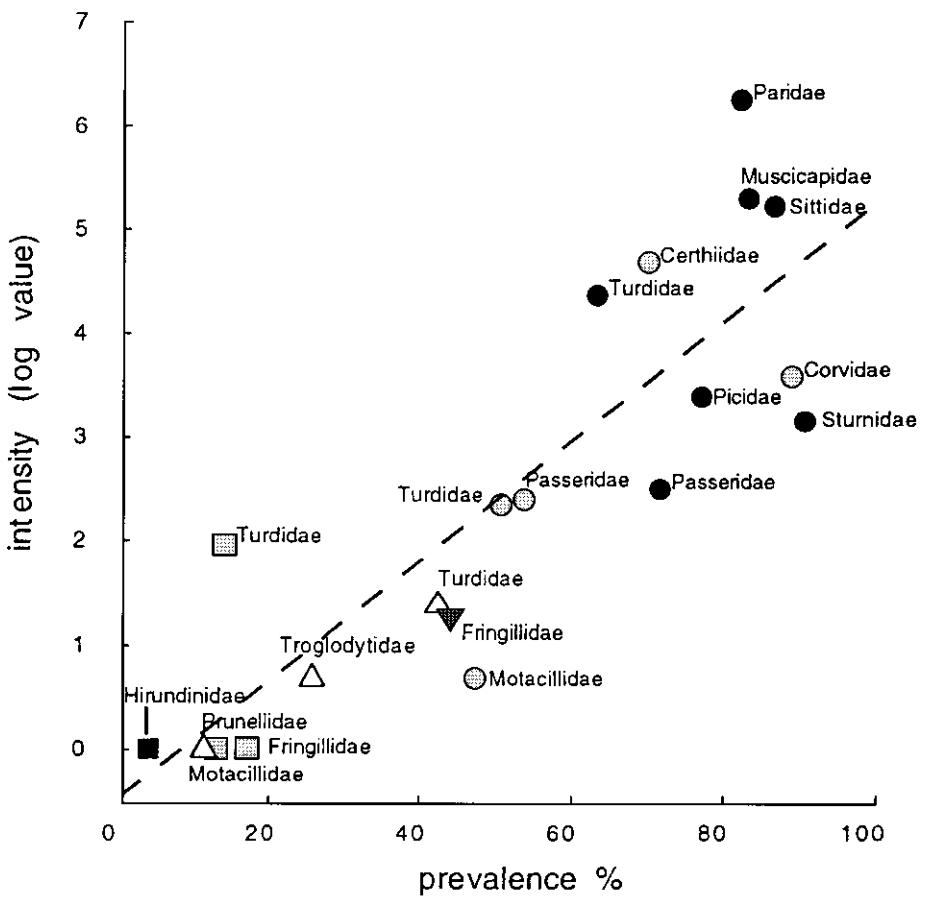

Fig. 1. Relationship between mean intensity *(i)* and prevalence *(p)* of infestation. In order to minimise phylogenetic effects, the values within nest types were calculated from family means. Within nest types, p and i's are family means calculated from the species data. Excluded from this graph are families for which p and $i = 0$ (Ralidae, Podicepididae, Anatidae, Sternidae, Laniidae and Columbidae). Symbols are ● nest-holes, ◍ crevices, ▼ tree crowns, ▩ bushes, △ ground and ■ mud nests (from Tripet and Richner 1997).

Tripet and Richner (1997) *C. gallinae* has been detected in 93% of all examined nests (2469 infested out of 2668). It was found in 61% of the species (60 out of 99) for which *p* or *i* was recorded. There is as positively exponential correlation between the prevalence and intensities (log value) of infestations ($r = 0.857$; $n = 20$; $p < 0.001$) (Fig. 1) among bird families. Avian families using tree hollows and those using various crevices have the highest *p* and *i*'s. At a comparable prevalence of infestation, the Paridae have the highest intensity of flea infestation among the hole nesting families.

For a given bird species *(s)* in a given wood habitat the number of flea produced in one breeding season and surviving to the next spring can be calculated as

$$N(s) = p(s) \cdot i(s) \cdot d(s)$$

where *d* is the density of host's nests for that habitat. Calculating *N(s)* per km^2 for the most common bird species of a oak dominated lowland forest in central Western Europe, with bird densities taken from Glutz and Bauer (1980), shows that 93% of the parasite population (140552 out of 150743 individuals) is produced by the hole-nesting Paridae (Tripet and Richner 1997). Altogether, 99.8% of the hen flea population overwinters in nest holes, and blue and great tits harbour most of *C. gallinae* individuals (Fig. 2). These results underline the hen fleas' potential for coevolution with tits despite their extensive host range (see also Tripet and Richner, in press b).

Fitness costs of hen fleas for great tit hosts

Host-parasite coevolution requires that parasites impose significant fitness costs onto their hosts. In great tits, nestling growth and body mass before fledging closely correlates with post-fledging survival and recruitment (e.g. Perrins 1965, Smith *et al* 1989, Tinbergen and Boerlijst 1990, for a review see Gebhardt and Richner 1998). Fitness costs were investigated by heat-treatment of nests in order to kill all parasites, and subsequent random infestation with 60 adult fleas (for details see Richner *et al* 1993). This number is below naturally occurring numbers in nests which have not been cleaned by humans after the previous breeding

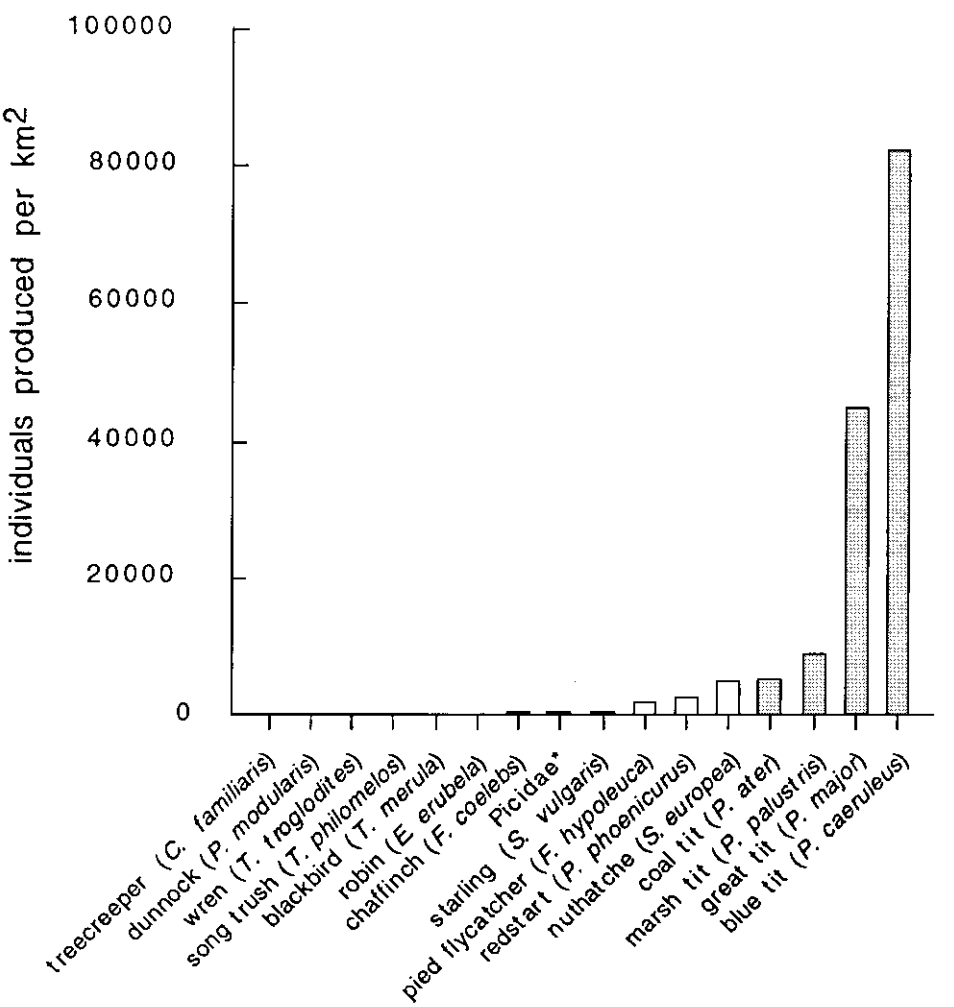

Fig. 2. Number of fleas produced in the nest of different species *(N(s))* in a hypothetical lowland forest community. Shaded bars are hole-nesting Paridae. *Picidae family means were used because no data is available for the great spotted woodpecker *(D. major)* (from Tripet and Richner 1997).

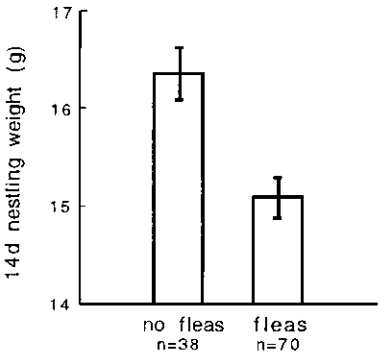

Fig. 3. Mean body mass of nestlings 14 days after hatching in parasite-free and flea-infested nests of great tits. Data from an experimental study in a population near Lausanne, Switzerland, carried out in 1991 and 1992.

period. Fleas reduce 14d nestling weight (Fig. 3; $T = 3.69$, $DF = 106$, $P < 0.001$) but also structural body size and wing length (Richner *et al* 1993).

At the physiological level, the fleas significantly affect haematocrit levels ($P < 0.001$; Richner *et al* 1993). Nestling mortality (number of chicks dead per day) is 0.22 in infested nests, but 0.07 only in parasite-free nests ($P = 0.006$; Richner *et al* 1993). Thus, hen fleas significantly reduce reproductive success, i.e. Darwinian fitness, of great tit hosts, and coevolved host responses can be expected.

Host responses

There are many ways for a host to respond to parasites and, as a general rule, one can expect that natural selection should favour the set of responses with the lowest cost/benefit ratio. The responses can occur at physiological, morphological and behavioural levels. They can be genetically fixed and then be inducible, or invariably present whether parasitism is present or absent, and/or be purely inducible and have no genetic basis. It can be expected that the response itself carries costs which will be measurable as a shift in the allocation of time and/or energy of an individual. Thus, host responses will affect important life-history trade-offs (Richner and Heeb 1995, Richner and Tripet in press).

Parasite avoidance

A simple and often efficient host response is the avoidance of a parasite. In an experiment we investigated the effects of the hen flea on timing of reproduction, nest site choice, and the probability of nest desertion in the great tit (Oppliger *et al* 1994). When great tits were offered a choice, on their territory, between an infested and a parasite-free nest box, they chose the one without parasites (Fig. 4). When there was no choice, the great tits in a territory containing an infested nest box delayed laying of the clutch by 11 days as compared to the birds that were offered a parasite-free nesting opportunity (Fig. 5). Nest desertion between laying and shortly after hatching was significantly higher in infested nests (Fisher Exact Test, $p<0.001$; Table 1).

For great tits it has been shown that survival of juveniles is negatively correlated with hatching date (Dhondt and Olaerts 1981, Perrins 1965, Kluyver *et al* 1977). In a data set covering twenty-five years, Perrins and McCleery (1989) have shown for great tits in Whytham Wood (Oxford, England) that the number of recruits per brood decreases on average by 3.7% for each day's delay in starting to lay. A difference of 11 days would then cause a reduction in reproductive success of 40%. Why then should a bird at a parasitized nesting site delay breeding?

Table 1. Desertion of clutches or broods between laying the first egg and the 4th day after hatching in infested and parasite-free nest boxes (from Oppliger *et al* 1994).

		stayed	deserted
Fleas	absent	**21**	**1**
	present	**8**	**12**

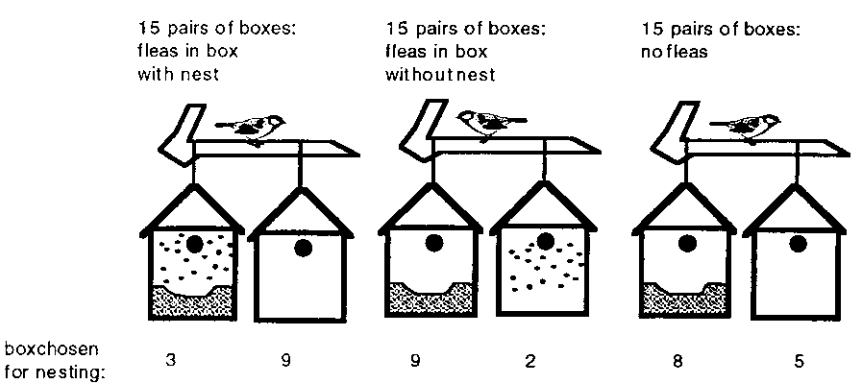

Fig. 4. Design and results of the choice-experiment. A pair of nest boxes was provided in each of 45 territories. One box of each pair contained an old nest, and in thirty of these pairs the haematophagous ectoparasites were injected in either the box with or without the nest. Of the 23 pairs of great tits that started breeding in the 30 experimental pairs of boxes, 78% of bird chose the parasite-free box (Binomial test, $p = 0.01$) (from Oppliger *et al* 1994).

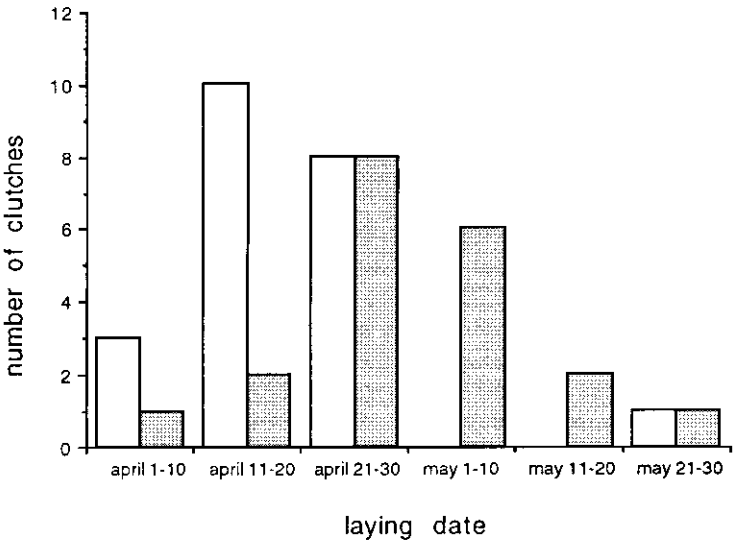

Fig. 5. Frequency distribution of the number of clutches initiated between April 1 and May 30. Light bars show the number of clutches laid in parasite-free nest boxes, dark bars the number of clutches in nest boxes that were infested with a haematophagous ectoparasite, the hen flea. The birds in the infested nest boxes started their clutch on average 11 days later than the ones in the parasite-free boxes. Hatching of the chicks in the infested boxes was delayed by 10 days and the difference between infested and parasite-free broods for both laying date and hatching date is higly significant (laying date: $t_{20,22} = 3.2$, $p = 0.003$; hatching date: $t_{13,21} = 2.7$, $p = 0.011$) (from Oppliger *et al* 1994).

The fleas in a nest box survive in large numbers from one breeding season to the next one. Hundred to thousands of flea larvae are present when the fledglings leave the nest (Heeb *et al* 1996, Tripet and Richner, in press b). Only few adult fleas leave the nest with the fledglings (Humphries 1968). The flea larvae in the nest develop into adults after fledging of the chicks, but then stay in a dormant stage inside cocoons, mostly attached to the nesting material. As described by Humphries (1968), mechanical stimulation of the old nest, as naturally provided by a nesting bird, causes the fleas to emerge from the cocoons. Similarly, a change in temperature causes emergence. In spring, the emerged fleas wait around the entrance hole for visiting birds, often forming a black ring, but also emigrate by crawling out of the nest box and up the tree trunk (Humphries 1968). If the nest box is not occupied, fewer and fewer fleas will therefore be left in time in the box and finally the flea load will be so small that the site becomes acceptable for a bird intending to breed. Nevertheless, later breeding has costs (e.g. Perrins and McCleery 1989), and a bird will therefore have to trade off the reduction in reproductive success caused by the delay in breeding against the loss it would incur if breeding earlier with a higher load of ectoparasites.

Clutch size

Clutch size varies widely both among and within species and much of this variation has been attributed proximately to variance in phenotypic quality of the parents, variance in food abundance, nest predation, nestling competition, nest parasitism by other birds, phylogenetic inertia, physiological constraints, and ectoparasites (for review see e.g. Murphy and Haukioja 1986, Godfray *et al* 1991, Poiani 1993a, b). Ultimately much of this variation can be understood in terms of variation in reproductive trade-offs, such as clutch size with offspring or adult survival and fecundity (for reviews see Linden and Møller 1989, Dijkstra *et al* 1990, Stearns 1992). Ectoparasites can strongly reduce reproductive success (Moss and Camin 1970, Møller *et al* 1990, Møller 1993, Richner *et al* 1993, Clayton and Tompkins 1994) of their hosts, and are therefore most likely to affect reproductive trade-offs. This life-history point of view predicts that hosts may reduce the impact of parasites by altering their own reproductive effort (Forbes 1993, Poulin 1994).

In hen fleas the length of a generation cycle is dependent on environmental factors, e.g. temperature, and varies from 17 to 40 days. Nestling great tits develop within approximately 20 days from hatching to fledging. Thus the parasite population grows, at best, very slowly over the time where nestlings are available as a resource. Indeed, we found no correlation between parasite load per nest and brood size ($r = -0.14$, $n = 28$, $p = 0.48$). As a consequence, it can be predicted that parasite load per nestling decreases with brood size. In other words, parasites get more diluted in a larger brood than in a smaller one. As predicted, observational and experimental data show a strongly negative correlation (observational data: $r = -0.42$, $p<0.001$, $n = 74$ broods; experimental data with controlled infestations: $r = -0.75$, $n = 35$, $p < 0.0001$; Fig. 6).

With long-cycled ectoparasites the parasite cost per chick decreases with increasing brood size due to the dilution of the parasites, and this by itself would favour larger broods with ectoparasites (pbs*) than without ectoparasites (bs*) (Fig. 7). To which extent larger broods will be favoured, will depend on the importance of the positive dilution effect relative to the negative effect of food competition in a larger brood.

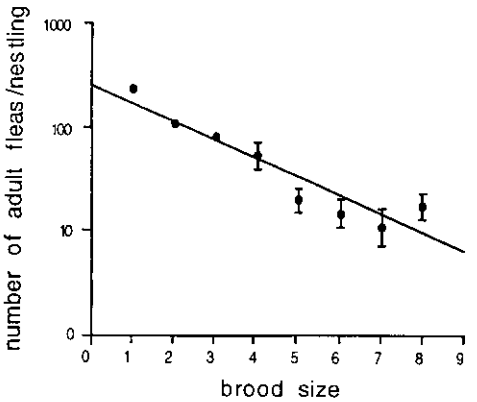

Fig. 6. Flea load of great tits in relation to brood size. Mean number (±1 s.e.) of adult hen fleas (on a $\log_{10}$-scale) per nestling (n = 35 nests) at fledging. Nests had been inocculated with 40 adult fleas at the beginning of egg laying by great tits.

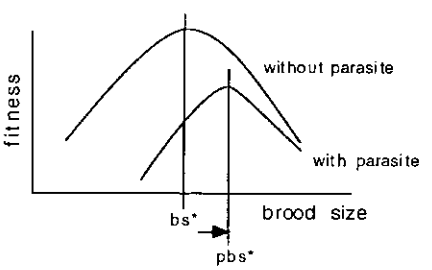

Fig. 7. Fitness with and without ectoparasites when parasites have relatively stronger effects in small broods. Optimal brood size with parasites (pbs*) ist larger than optimal brood size without parasites (from Richner and Heeb 1995).

Empirical support comes from both intra- and interspecific studies. In our studies, female great tits which were experimentally infested after laying the second egg layed, in some years but not in others, a larger clutch than unparasitized females. We do not yet fully understand the causes for the variance in the females' response. Further support is provided by a recent comparative study (Poiani 1993b) showing that clutch size of some North American and Australian passerine families increased with an increasing importance of ectoparasitism for each species which is commonly infested by dipteran parasites (i.e. long-cycled ectoparasites). As one interesting example, it may be noted that the blue tit, which is according to our observations and to the findings by Harper *et al* (1992) the most heavily infested host of hen fleas, lays the largest clutch for a songbird of its size.

Maternal effects

Evidence for epigenetic processes in host-parasite interactions in natural populations is scarce. Facultative parasite-induced responses, however, should be favoured by natural selection when the risk of infection is unpredictable and host responses costly. In vertebrates, induced responses are generally viewed as being adaptive although evidence for fitness benefits arising from these responses in natural host populations is lacking. In domesticated or captive vertebrates, parasite-induced host responses have been shown to affect food intake, reproductive rate and survival of ectoparasites (Devaney and Augustine 1988, Allen 1994, Randolph 1994, Wikel 1996). Examples of inducible responses are the acquired immunity of vertebrates, which generally refers to the antibody-mediated responses to the invasion of foreign antigens (Mitchison 1990, Roitt *et al* 1996, Wakelin 1996) but can also include behavioural changes triggered by parasites (Hart 1990, Milinski 1990, Clayton 1991, Christe *et al* 1994, 1996a, b, Keymer and Read 1991). There is also growing evidence that female birds, when exposed to parasites, can transfer antibodies through the eggs to the newly hatched chicks (Graczyk *et al* 1994, Smith *et al* 1994). We therefore tested for the presence of maternal effects in our host-parasite system. If such adaptive responses occur, we predict that great tits exposed to fleas during egg-laying raise young in better conditions and achieve higher current reproductive success than birds unexposed to fleas during egg-laying.

In the experiment (Heeb *et al* 1998) we exposed some great tits to fleas during egg laying (exposed group) and thereby allowed for induced responses and, as a control, kept others free of parasites (unexposed group) over the same time period. At the start of incubation, we killed the parasites in both groups and reinfested all nests with fleas. Nestlings from birds exposed to fleas during egg laying (solid line) grew faster than nestlings from birds unexposed to fleas during egg laying (dashed line), and sixteen days after hatching, nestlings of the exposed group were significantly heavier than nestlings of the unexposed group (means ± s.e.; t-test: $t_{37} = 3.42$, $p_{dir} = 0.0012$) (Fig. 8).

In 1995, a total of 94 young fledged from nests in the unexposed control group and 13 (13.8%) were recaptured as recruits in 1996. From nests in the exposed group a total of 121 young fledged of which 24 (19.8%) were recaptured in 1996. There was a significant effect of treatment on the number of young recruited (Poisson regression model: change in deviance = 3.39, df = 1, $p_{dir} = 0.041$). Among birds which recruited young, pairs from the exposed group recruited a median of 2 young (range: 1–4, n = 12) whilst pairs in the unexposed

group recruited a median of 1 young (range: 1–3, n = 9). The number of grandchildren raised in 1996 by the recruits was higher for mothers in the exposed group in 1995 than for mothers of the unexposed group (Mann-Whitney U-test, $p_{dir} = 0.05$) (Fig. 9). In summary, we showed (Heeb *et al* 1998) that birds with nests infested during egg laying (1) had fewer breeding failures and raised a higher proportion of hatchlings to fledging age, (2) have offspring that reach greater body mass, grow longer feathers, and fledge earlier, and (3) have a higher number of recruits and first-year-grandchildren, than unexposed birds. Our results provide the first experimental evidence for the occurrence and the adaptiveness of induced responses against a common ectoparasite in a wild population of vertebrates.

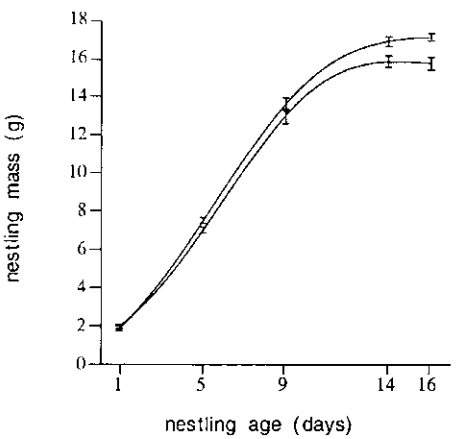

Fig. 8. Growth of great tit nestlings in flea infested nests following exposure or non-exposure of parents to fleas during egg laying (from Heeb *et al* 1998).

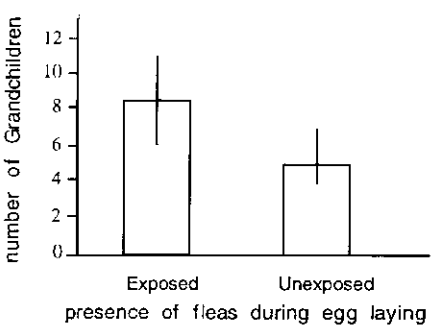

Fig. 9. Number of first-year-grandchildren in relation to exposure of parent great tits to fleas during egg laying (median ± interquartile ranges). Birds of the group exposed in 1995 had significantly more first-year-grandchildren in 1996 than birds of the unexposed group (from Heeb *et al* 1998).

Parasite compensation

As shown above, hen fleas reduce both body mass and number (Richner *et al* 1993) of great tit offspring. Since it is well documented that fledging body mass correlates with the probability of survival of the offspring (Perrins 1965, Smith *et al* 1989, Tinbergen and Boerlijst 1990), it is reasonable to conclude that ectoparasites therefore lower the value of the current brood. A reduction in offspring quality and number could be the direct physiological consequence of the blood-sucking ectoparasites or, alternatively, the consequence of a lowered begging and parental food provisioning response due to parasitism. The optimal response of the parents to parasitism is shaped by life-history trade-offs, and theoretical models predict that parental effort should increase in our host-ectoparasite system (Perrin *et al* 1996, Christe *et al* 1996, Tripet and Richner 1997).

In the model (Perrin *et al* 1996) current reproductive success ($p_o b_t$) and future reproductive success ($p_a V_{t+1}$) are functions of reproductive effort for both unparasitized and parasitized hosts. Offspring survival (p_o) and offspring number (b_t) both increase with parental effort, while adult survival (p_a) and adult residual reproductive value (V_{t+1}) decrease with parental effort. The optimal reproductive effort lies where the slopes of the tangents to the curves are equal with opposite signs. Fleas decrease offspring condition, and thereby survival in such a way (fat line in Fig. 10) that an additional parental investment Δ_{RE} would be necessary to compensate for the fleas' effect (i.e. achieve the same survival as non-parasitized). The optimal reproductive effort RE (P^*) is higher than that of non-parasitized, but not as much as Δ_{RE}, so both current and residual reproductive success remain below the non-parasitized level.

To test whether parents partly compensate for the effects of parasites, as predicted by the model,

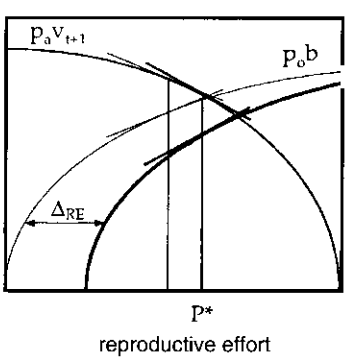

Fig. 10. Current reproductive success ($p_o b_t$) and future reproductive success ($p_a V_{t+1}$) as functions of reproductive effort (RE) for both unparasitized and parasitized hosts (from Perrin *et al* 1996).

we manipulated flea load of nests during the laying period by infesting half of the nest boxes three times with 20 fleas at each infestation. The other nests were heat-treated with a microwave appliance at intervals from 4–8 days (for details see Richner *et al* 1993). Thirteen days after hatching we monitored the rate of begging of nestlings, and the rate of food provisioning of the male and the female parent by use of a video camera equipped with an infra-red light source. For individual recognition, the nestlings were marked with minute spots of paint on their heads. Fourteen days after hatching we measured nestling body mass and size, and caught the parents by use of a trap-door at the nest (for further details see Christe *et al* 1996).

Mean body mass of nestlings, 14 days after hatching, was significantly lower in infested nests than mass of nestlings in parasite-free nests (14.5 ± 0.32 s.e. versus 15.9 g ± 0.20 s.e.; *t*-test, $t = 2.59$, $P = 0.007$). Body mass of parents 14 days after hatching of their offspring was not significantly affected by the flea treatment (parasite-free vs infested broods: females 16.9 ± 0.24 vs. 16.7 ± 0.26, $t = 0.66$, $P = 0.52$; males 17.9 ± 0.21 vs. 17.7 ± 0.28, $t = 0{,}71$, $P = 0.49$).

Begging rate, expressed as the sum of all nestlings' begging time per hour, increased significantly (*t*-test: $t = 2.45$, $p = 0.021$) if broods were infested with hen fleas. Nestlings of infested broods begged on average 140% more than nestlings of parasite-free broods (Christe *et al* 1996). Hen fleas increase competition among nestlings (Christe *et al* 1996), and may also affect food distribution among young (Kölliker *et al* 1998).

Male parents of infested broods increased the rate of food provisioning to the nest significantly (*t*-test: $t = 2.59$, $p = 0.015$). Males of infested broods return 24.4 times per hour with food, and the ones in parasite-free broods 15.5 times, an increase of 57% (Fig. 11). Ectoparasites do not significantly (*t*-Test: $t = 0.47$, $p = 0.64$) affect the females' rate of food provisioning to the nest. The power of the latter test (Cohen 1988) is below 10% however, and the null hypothesis may therefore not be safely accepted (Christe *et al* 1996).

Fig. 11. Mean number of feeding visits per hour (±1 SE) made by males and females of parasite-free and infested broods of great tits (from Christe *et al* 1996).

Life-history trade-offs with host responses

If follows inherently from evolutionary theory that host responses must carry costs to the responding individual, and they are particularly intriguing when the best response leads to a deviation from an individual's optimal life-history. The deviation implies a shift in one or several life-history trade-offs, which can arise at morphological, physiological, or behavioural levels. These shifts are at the heart of the co-evolutionary process in host-parasite interactions. I will illustrate the life-history consequences of two host responses exposed above, parasite compensation and a shift in clutch size.

The trade-off with parasite compensation

From the model shown in Fig. 10, a consequence of an increase in parental effort as a response to parasitism is a decrease in the parents' future reproductive success. This decrease is not a consequence of fleas directly exploiting parents as a food source, but is caused by the higher parental effort. How is this decrease mediated? A basic trade-off exists between investment in current versus future reproduction, and one way to mediate an increase in parental effort for current reproduction into future reproduction, is to decrease investment in immune function, which is supposedly costly. Thus the predictions are (1) that parents with an ectoparasite-induced increase in the effort for current reproduction will show weakened immune responses, and (2) will therefore be more likely to contract infections by other common pathogens.

The first prediction has been confirmed by an experiment with zebra finches (Deerenberg *et al* 1997), where parents were assigned randomly to care for 2, 4 or 6 nestlings. During the period of parental care, the parents received an injection with a novel antigen (sheep red blood cells), and then the immune response was measured. Parents which had to care for more young were significantly less likely to respond.

The second prediction was tested (Richner *et al* 1995) in one of our great tit populations where around 20% of females and 35% of males are commonly infected with *Plasmodium*, a pathogen causing bird malaria. In order to exclude the possibility of a direct *Plasmodium* transmission by fleas, we eliminated all fleas by a heat-treatment and then manipulated parental effort by manipulation of brood size

after hatching. We measured feeding effort of both males and females, and assessed the prevalence of the hemoparasite from blood smears. Males of enlarged broods fed their chicks with a rate nearly 50% higher than males of reduced (Fisher's Least-Significant-Difference Test, P = 0.008) and unmanipulated broods (P = 0.03) (Fig. 12). In contrast, the females' rate of feeding visits to nest boxes was not significantly higher in enlarged than in reduced or unmanipulated brood (all *P*-values > 0.2).

Given that only males adjusted the feeding rate to the increased demand, we predicted a higher prevalence of hematozoa in males of enlarged broods than in males of reduced and unmanipulated broods, but no effect on females. The experimental manipulation of brood size was significantly associated with the occurrence of *Plasmodium* spp. in male parents (Fig. 12) but not in females. In reduced and unmanipulated broods male prevalence was 38% and 32% respectively, whereas in enlarged broods prevalence of *Plasmodium* in males was 76% (G = 9.9, df = 0.009, n = 61) (Richner *et al* 1995). None of the females in the reduced group, and two females each in the unmanipulated and the enlarged group showed an infection with *Plasmodium*. The findings show that there is a trade-off between reproductive effort and parasite susceptibility, and also suggest a mechanism for the well documented trade-off between current reproductive effort and parental survival (see Richner *et al* 1995, Richner and Tripet in press).

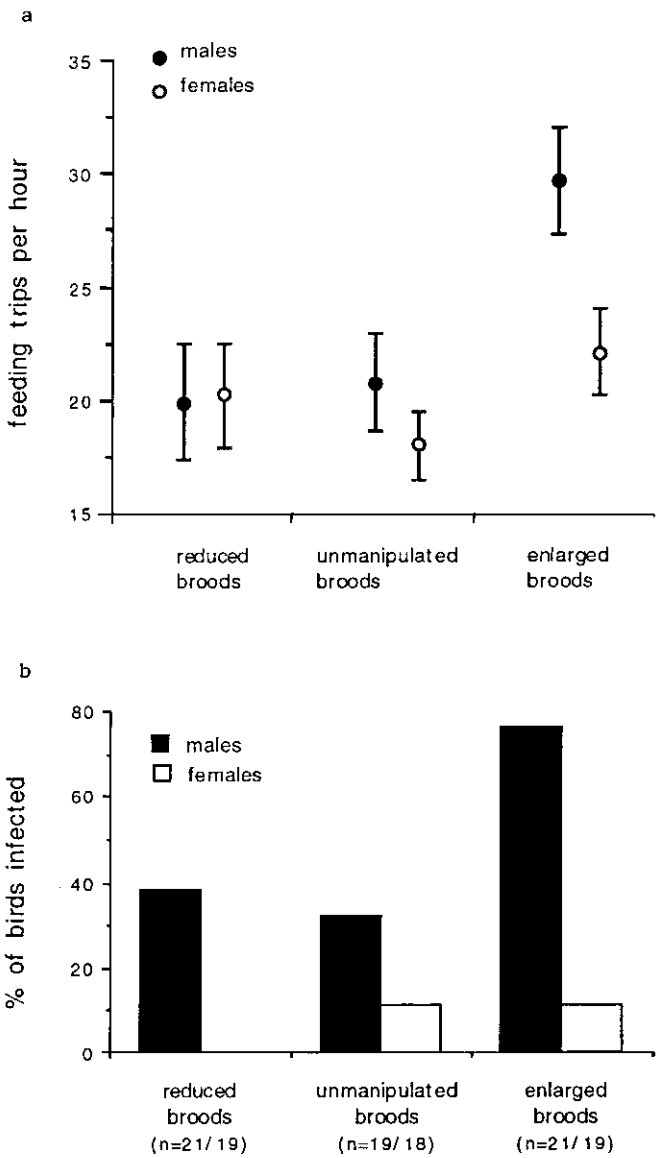

Fig. 12. a) Number of feeding trips to the nest box per hour (± s.e.) by male and female parent of reduced (n = 13), unmanipulated (n = 8), and enlarged (n = 13) broods. **b)** Prevalence of *Plasmodium* spp. in blood smears of parents rearing reduced (21 males/20 females), unmanipulated (19 males/18 females), and enlarged (21 males/19 females) broods.

This is not to suggest that all parasites which affect current reproduction should elicit a compensatory response. There is good empirical evidence that ectoparasites may also cause a decrease in reproductive effort. Ticks, for instance, can provoke complete nest desertion (the ultimate reduction in reproductive effort) in some seabirds such as brown pelicans (e.g. King *et al* 1977), sooty terns (Feare 1976) and cormorants (Duffy 1983). House martins have been shown to reduce investment in response to hemopterans (Møller *et al* 1994), which may also induce nest desertion in cliff swallows (Emlen 1986, Loye and Carroll 1991). Depending on how parasites affect the shape of the relationship between reproductive effort and current reproductive success (or between maintenance effort and residual reproductive success), the optimal response may be either an increase, or a decrease in current reproductive effort.

Interestingly, the males, but not the females, of infested nests increased the rate of food provisioning. This finding agrees with the finding above where only males but not females compensated for the effect of ectoparasites. Why should only the males respond? One possibility is that for females the trade-off between investment in the current versus future broods is in favour of future broods, whereas for the male the trade-off is in favour of investing in the current brood. In great tits and blue tits the females readily divorce males after low breeding success or breeding failure (Lindén 1991, Dhondt and Adriansen 1994) and this may indicate that a female puts a relatively high premium on future broods. Males could then reduce the probability of a divorce by a heavier investment in the current brood. Another possibility is that the females' most critical trade-off during reproduction arises at the stage of egg production. This is illustrated below.

The trade-off with a parasite-induced shift of optimal clutch size

Evolutionary theory predicts that, for iteroparous organisms, the optimal clutch size is the result of a trade-off between current and future reproduction (Williams 1966, Charnov and Krebs 1974, Stearns 1992), i.e. natural selection maximises lifetime reproductive success. At the physiological level, this trade-off predicts that parents which invest heavily in their current offspring will have fewer resources to allocate to parasite defence, thereby impairing their future reproduction. Thus, an ectoparasite-induced increase in clutch size should render the female parent more susceptible to other pathogens common in their environment, in our population to *Plasmodium*. We found a strong correlation between clutch size and malaria prevalence (Oppliger *et al* 1997). Since this correlation can be caused by many other factors, we performed an experiment to investigate the causal pathway between clutch size and malaria prevalence. Again, to exclude the more simple possibility of a direct *Plasmodium* transmission by fleas, we eliminated all fleas by a heat-treatment and then manipulated female effort by forcing females to lay a larger clutch (Oppliger *et al* 1996). In half of the nests of our breeding population we removed the first two eggs laid. As a consequence, females in this experimental group compensated by laying, on average, one more egg (t = 1.99; d.f. = 74; P = 0.05) than females in a control group (Fig. 13), but raised, on average, one offspring less. Fourteen days after hatching of the chicks, we assessed the prevalence of *Plasmodium* in the peripheral bloodstream of females. If there is a trade-off between egg production and parasite defence, one would predict a higher parasite prevalence in the females of the experimental group since they laid a larger clutch. Prevalence in males of the experimental group should not increase because the number of offspring to be raised is no greater than in the control group. Our study showed that an increase of the clutch size by one egg leads to an increase in the prevalence of *Plasmodium* from 20% in the control females to 50% in the experimental females (χ^2 = 6.49, d.f. = 1; P = 0.011) (Fig. 13), thus supporting the hypothesis of a trade-off between egg production and parasite defence. As expected, there was no significant difference in prevalende of *Plasmodium* in males (χ^2 = 0.30, d.f. = 1, P = 0.59) of the two groups (Fig. 13).

Thus, if ectoparasitic hen fleas give rise to a larger optimal clutch size, the female parent which adjusts clutch size to the new optimum can only do this at the expense of a lowered resistance against other pathogens. Hen fleas, therefore, will influence the host's trade-off between current and future reproduction.

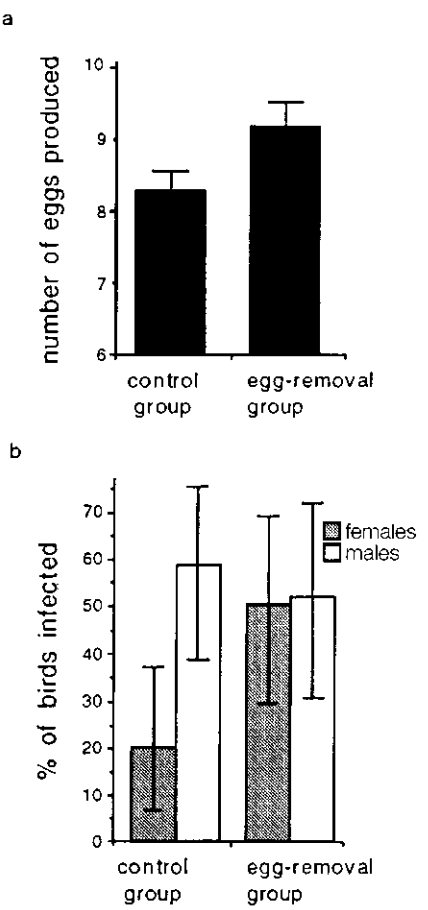

Fig. 13. a) Mean numer of eggs (± s.e.) laid by females of a control group (n = 41 broods), and of an experimental group in which the first two eggs were removed the day of laying (n = 35 broods). **b)** Prevalence of *Plasmodium* spp. (±95% confidence limits of binomial distribution) in male and female great tits in the egg-removal group (n = 30) and in the control group (n = 35).

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