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Host condition and host immunity affect parasite fitness in a bird–ectoparasite system

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Summary

1. Parasites might preferentially feed on hosts in good nutritional condition as such hosts provide better resources for the parasites' own growth, survival and reproduction. However, hosts in prime condition are also better able to develop costly immunological or physiological defence mechanisms, which in turn reduce the parasites' reproductive success. The interplay between host condition, host defence and parasite fitness will thus play an important part in the dynamics of host–parasite systems.

2. In a 2×2 design, we manipulated both the access to food in great tit *Parus major* broods and the exposure of the nestlings to hen fleas *Ceratophyllus gallinae*, a common ectoparasite of hole-breeding birds. We subsequently investigated the role of manipulated host condition, host immunocompetence, and experimentally induced host defence in nestlings on the reproductive success of individual hen flea females.

3. The food supplementation of the nestlings significantly influenced the parasites' reproductive success. Female fleas laid significantly more eggs when feeding on food-supplemented hosts.

4. Previous parasite exposure of the birds affected the reproductive success of fleas. However, the impact of this induced host response on flea reproduction depended on the birds' natural level of immunocompetence, assessed by the phytohaemagglutinin (PHA) skin test. Flea fecundity significantly decreased with increasing PHA response of the nestlings in previously parasite-exposed broods. No relationship between flea fitness and host immunocompetence was, however, found in previously unexposed broods. The PHA response thus correlates with the nestlings' ability to mount immunological or physiological defence mechanisms against hen fleas. No significant interaction effect between early flea exposure and food supplementation on the parasites' reproductive success was found.

5. Our study shows that the reproductive success of hen fleas is linked to the hosts' food supply early in life and their ability to mount induced immunological or physiological defence mechanisms. These interactions between host quality and parasite fitness are likely to influence host preference, host choice and parasite virulence and thus the evolutionary dynamics in host–parasite systems.

Key-words: early developmental conditions, host–parasite interactions, immunocompetence, *Parus major*, phytohaemagglutinin.

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Introduction

Parasites often show a clumped distribution within a host population; while some hosts experience very large parasite infestation levels, others have only few or even no parasites (Anderson & May 1978; Anderson & Gordon 1982). In some host–parasite systems this distribution pattern might result from differences in host behaviour, and thus from variation in the chance of encountering a certain type of parasite (Reimchen 2001). However, variation in parasite abundance might

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also reflect variation in the attractiveness of hosts to their parasites (Zuk & McKean 1996; Poulin 1998; Krasnov, Khokhlova & Shenbrot 2003).

Parasites live in or on hosts from which they derive resources for their own growth, survival and reproduction (Price 1980; Clayton & Moore 1997). Generally, the quality of the resources an individual host offers for a parasite varies within a host population, and might depend on the host's nutritional status, i.e. its access to high-quality food. Parasites might thus have a preference for hosts in good condition (e.g. Keymer, Crompton & Walters 1983; Blanco, Tella & Potti 1997; Dawson & Bortolotti 1997). However, hosts in good condition are also better able to develop physiological or immunological defence mechanisms (Gershwin, Beach & Hurley 1985; Cook 1991; Lochmiller, Vestey & Boren 1993; Saino, Calza & Møller 1997; Ing *et al*. 2000), which in turn can reduce the reproductive success of the parasite (Wakelin & Apanius 1997). Such defence mechanisms might include a combination of immunological (e.g. the production of specific immunoglobulins or local hypersensitivity reactions) or biochemical and mechanical mechanisms (e.g. the modification of the skin thickness or blood viscosity), which reduce the feeding efficiency of (ecto-) parasites (see reviews in Marshall 1981; Allen & Nelson 1982; Nelson 1984; Lehane 1991; Allen 1994; Wakelin 1996; Wikel, Bergmann & Ramachandra 1996; Wikel & Bergman 1997; Apanius 1998) or increase the costs associated with the digestion of the host's blood (Sarfati *et al*. 2005).

Both building up an efficient immune system and its use are considered to be costly in terms of energy and metabolites (reviewed in Sheldon & Verhulst 1996; Norris & Evans 2000; but see Klasing 1998). Consequently, only individuals in prime condition can afford to invest their limited resources in immune defence rather than in other life-history traits such as growth or reproduction (e.g. de Lope, Møller & de la Cruz 1998; Siva-Jothy, Tsubaki & Hooper 1998; Veiga *et al*. 1998; Tschirren, Fitze & Richner 2003; Tschirren & Richner 2006). Parasites might thus benefit from infesting hosts of lower quality, which are easier to exploit due to their weaker immune system (Coop & Holmes 1996; Christe, Møller & de Lope 1998; Roulin *et al*. 2003; Simon *et al*. 2003).

We hypothesize that the dynamics of host–parasite interactions including host preference and the optimal level of host exploitation by the parasite will thus depend on the relative importance of the quality of the resources a host offers, and the negative effect of the host's immunological or physiological defence on parasite fitness (see also Lee & Clayton 1995). However, while the impact of parasite infestation on host fitness has been demonstrated in numerous studies (reviewed in Lehmann 1993; Møller 1997), the role of host condition and host immunity on parasite fitness has so far received very little attention (but see, e.g. Krasnov *et al*. 2005).

In an experimental field study on great tits *Parus major* and their most common ectoparasite, the hen flea *Ceratophyllus gallinae*, we thus investigated the role of early nutritional condition, induced host response and their interaction with the hosts' natural level of immunocompetence on the parasites' reproductive success. The relationship between host characteristics and parasite fitness can elucidate how host-induced selective pressures can shape the parasite's life history and host preference, and thus how hosts can shape the co-evolutionary dynamics in host–parasite systems.

Materials and methods

STUDY SITE AND SPECIES

The experiment was performed during the breeding season of 2005 in a great tit *Parus major* population breeding in nest boxes in the 'Forst', a forest composed of a mixture of deciduous and pine trees, near Bern, Switzerland (46°54′N 7°17′E/46°57′N 7°21′E).

The great tit is one of the main hosts of the ectoparasitic hen flea *Ceratophyllus gallinae* (Tripet & Richner 1997a). Hen fleas live in the nest material of hole-nesting birds and suck blood from nestlings, but also from adults that visit the nest, e.g. during nest building, roosting, incubation or feeding of the young (see Tripet & Richner 1997a, 1999 for further information on the parasite's life cycle). Most great tit nests are naturally infested with hen fleas, however, there is large variation in the infestation intensity among nests (Heeb *et al*. 1996; Fitze, Clobert & Richner 2004).

MANIPULATION OF THE NESTLINGS' FOOD SUPPLY

We regularly visited the nest boxes from the beginning of the breeding season (1 April) onwards to determine the start of egg laying, incubation and hatching. After hatching, we manipulated the food supply of the nestlings by supplementing half of the broods with 30 g of maggots (*Sarcophaga* sp.) provided in a cup within the nest box on days 3, 5, 7, 9 and 11 after hatching. All maggots were eaten within 2 days. The other half of the broods did not receive extra food, but they had an empty cup in their nest box and were visited and handled similarly on all 5 days.

MANIPULATION OF THE NESTLINGS' EXPOSURE TO PARASITES

We heat-treated the nesting material of all nests 3 days after hatching to kill all nest-based parasites that were naturally present in the nests (Richner, Oppliger & Christe 1993; Tripet & Richner 1997b). Simultaneously, the nest height was standardized to 7 cm to avoid density-dependent effects on the flea population (Tripet & Richner 1999). Thereafter, half of the nests were experimentally infested with 40 female and 20 male hen fleas originating from old nests collected within the same forest at the beginning of the breeding season, allowing the nestlings of this treatment group to

mount immunological or physiological responses against hen fleas or flea-transmitted pathogens during the feeding treatment. The other nests were kept free of parasites (no induced host response). Nests were randomly assigned to one of the four treatment groups of this 2×2 design including the manipulation of early parasite exposure and food abundance. A total of 43 nests were included in the experiment.

REPRODUCTIVE SUCCESS OF HEN FLEAS

On day 12 post-hatching (i.e. 1 day after the last food supplementation), all nests were heat-treated and thereafter infested with 40 female and 20 male hen fleas from the stock population. The fleas were allowed to suck blood on the nestlings for 2 days, then 10 random female hen fleas were collected from each nest (as described in Walker *et al*. 2003). The females were placed into small tubes, transferred to the lab and placed in an incubator at 20 °C and 75% humidity. Two days later we counted the number of eggs laid by each female flea using a binocular microscope. Walker *et al*. (2003) have shown that the mean egg production of 10 individual females strongly correlates with the total reproduction of fleas in a nest. The mean egg production of the 10 female fleas collected within a nest was used in the statistical analyses.

HOST IMMUNOCOMPETENCE

The nestlings' natural level of immunocompetence was assessed by the phytohaemagglutinin (PHA) skin test (Goto *et al*. 1978; McCorkle, Olah & Glick 1980; Cheng & Lamont 1988). The PHA response is related to the immune cell activity (including lymphocytes, basophils, eosinophils, heterophils, macrophages and thrombocytes) at the injection site, and involves both innate and adaptive components of the immune system (Martin *et al*. 2006). The PHA skin test is widely used as a general measure of immunocompetence in avian ecological research (see, e.g. Smits, Bortolotti & Tella 1999; Tella, Scheuerlein & Ricklefs 2002; Martin *et al*. 2006 for reviews), but it can obviously only assess a limited aspect of the very complex vertebrate immune system (Klein 1993; Martin *et al*. 2006). Previous studies have shown that the PHA-induced swelling is correlated with nestling survival (Christe *et al*. 1998; Hõrak *et al*. 1999) and the probability of recruiting locally (Moreno *et al*. 2005).

Nestlings were injected subcutaneously with 0·1 mg of PHA-P (SIGMA Chemicals, Deisenhofen, Germany) dissolved in 0·02 mL of sterile phosphate-buffered saline in the centre of the left wing-web (patagium) 12 days post-hatching (Smits *et al*. 1999; Tschirren & Richner 2006). The thickness of the patagium at the injection site was measured with a micrometer (Mitotuyo, Type 2046FB-60) to the nearest 0·01 mm before and 24 h (± 1 h) after injection. The micrometer applies a constant pressure on the wing web and the measure stabilizes after a short time. The thickness of the wing web 5 s after applying the micrometer was used as a standardized measurement. The difference between the wing-web thickness before and 24 h after PHA injection was calculated for each nestling (Smits *et al*. 1999), and the mean change of the wing-web thickness per nest was used in the statistical analyses. As the birds' investment in immune defence was not directly manipulated in this study, the PHA response is an assay of their natural level of immunocompetence.

MORPHOMETRIC MEASURES

On days 3 and 15 post-hatching, we measured the body mass of the nestlings using an electronic Sartorius® balance with a precision of 0·01 g. On day 15 we also measured the length of the metatarsus to the nearest 0·1 mm using a calliper (Svensson 1992). Means per nest were calculated and used in the statistical analyses. Mean body mass was not significantly different between treatment groups at the start of the experiment (flea treatment: $F_{1,40} = 0{\cdot}30$, $P = 0{\cdot}59$; food supplementation: $F_{1,40} < 0{\cdot}01$, $P = 0{\cdot}98$; fleas × food: $F_{1,39} = 1{\cdot}11$, $P = 0{\cdot}30$). Similarly, brood size at the start of the experiment did not significantly differ between treatment groups (flea treatment: $F_{1,40} = 1{\cdot}43$, $P = 0{\cdot}24$; food supplementation: $F_{1,40} = 0{\cdot}51$, $P = 0{\cdot}48$; fleas × food: $F_{1,39} = 0{\cdot}95$, $P = 0{\cdot}34$), indicating that there was no experimental bias due to initial body mass or brood size differences between treatment groups. All measurements were collected blindly with respect to the treatment of the birds.

STATISTICAL ANALYSES

We used two-way ANOVAs to analyse the effects of flea exposure (i.e. the induced host response) and the food supplementation on body mass, body size and PHA response of the nestlings. Parasite exposure and food supplementation and their interaction were included as fixed factors into the model. The reproductive success of female fleas was analysed with the same model as described above. Additionally, we included mean PHA response per nest (and its two- and three-way interactions with the parasite exposure and food supplementation) as a covariate into the model (ANCOVA). Interactions were backward eliminated if they were nonsignificant. All tests were two-tailed with a significance level of 0·05. Residuals of the models were normally distributed and homoscedastic. Mean ± 1 SE are presented in Results and figures. Statistical analyses were performed using JMP IN 4·0 (Sall & Lehmann 1996).

Results

REPRODUCTIVE SUCCESS OF FLEAS

The egg production of female fleas was influenced by the food treatment of their hosts ($F_{1,38} = 6{\cdot}25$, $P = 0{\cdot}02$).

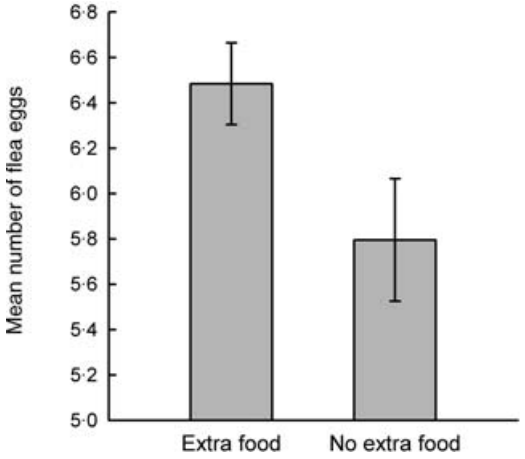

Fig. 1. Egg production of female fleas feeding on food-supplemented and control nestlings. Mean ± 1 SE are shown.

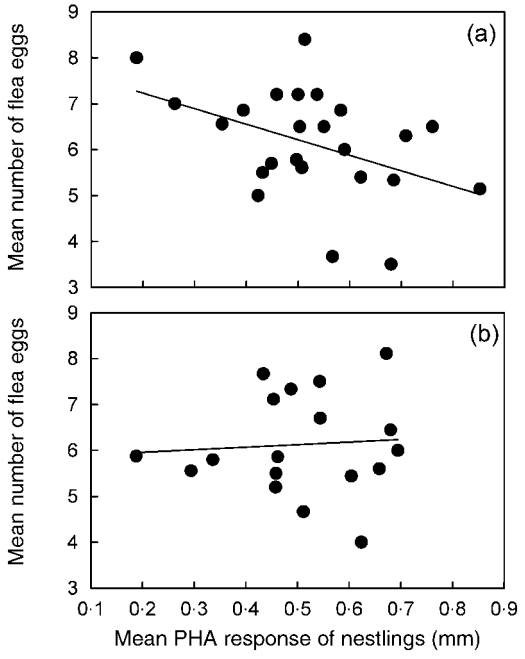

Fig. 2. Relationship between flea fecundity and PHA response of the hosts in (a) previously flea-exposed broods, and (b) broods with no previous flea exposure.

Female fleas laid significantly more eggs when their hosts had received extra food early in life (mean number of eggs in nests with extra food: 6·5 ± 0·2, without extra food: 5·8 ± 0·3; Fig. 1).

Our results further indicate that previous parasite exposure of the host reduced hen flea fecundity. The strength of this induced host response on fleas fitness depended, however, on the hosts' natural level of immunocompetence, as demonstrated by the significant interaction effect between early flea exposure and mean PHA response on flea reproduction ($F_{1,38}$ = 4·07, P = 0·05, Fig. 2). Flea reproduction significantly decreased with increasing PHA response in broods where a host response was experimentally induced, explaining 18·8% of the variation in flea fecundity ($F_{1,23}$ = 5·31, P = 0·03, Fig. 2a). In nests where a host response to fleas was experimentally prevented, however, there was no significant relationship between flea reproduction and PHA response of the host, with mean PHA response explaining only 0·5% of the variation in flea fecundity ($F_{1,16}$ = 0·08, P = 0·77, Fig. 2b). Neither mean host body mass ($F_{1,37}$ = 0·01, P = 0·92), mean host body size ($F_{1,37}$ = 0·06, P = 0·82) nor the hosts' brood size ($F_{1,37}$ = 0·09, P = 0·78) had a significant effect on the reproductive success of fleas. There was no significant interaction effect between early flea exposure and food supplementation on the parasites' reproductive success ($F_{1,37}$ = 0·73, P = 0·40; all other two- or three-way interactions: P > 0·21).

NESTLING BODY SIZE, BODY MASS AND PHA RESPONSE

The food supplementation significantly increased the mean nestling size measured as the mean metatarsus length per nest ($F_{1,40}$ = 7·11, P = 0·01; mean size with extra food: 19·7 mm ± 0·06, without extra food: 19·4 mm ± 0·08) and tended to increase the mean body mass of the nestlings ($F_{1,40}$ = 3·81, P = 0·06; mean body mass with extra food: 16·24 g ± 0·26, without extra food: 15·41 g ± 0·31) shortly before fledging. Neither the flea exposure early during the nestling period nor the interaction between flea exposure and food supplementation significantly influenced mean body mass (flea treatment: $F_{1,40}$ = 0·58, P = 0·45; fleas × food: $F_{1,39}$ = 1·69, P = 0·20) or mean body size (flea treatment: $F_{1,40}$ = 1·00, P = 0·32; fleas × food: $F_{1,39}$ = 3·00, P = 0·09) of the nestlings.

The PHA response was not significantly influenced by the food supplementation early during the nestling period ($F_{1,40}$ = 0·06, P = 0·81), the flea exposure ($F_{1,40}$ = 0·16, P = 0·69) or the interaction between food supplementation and flea exposure ($F_{1,39}$ = 0·54, P = 0·47). However, there was significant positive relationship between the mean PHA response and the mean body mass of the nestlings at the end of the nestling period ($F_{1,41}$ = 5·02, P = 0·03; b = 3·42 ± 1·53).

Discussion

Similar to the study of Walker *et al.* (2003), we found a negative effect of an induced host response on parasite fecundity. However, we show that this effect is not unconditional, but it depends on the hosts' natural level of immunocompetence, assessed by the PHA skin test. In broods that were previously exposed to fleas (i.e. broods with an induced host response), the reproductive success of fleas significantly decreased with increasing host PHA response. Interestingly, however, no relationship between PHA response and flea fecundity was found in nests that had no previous contact to fleas (i.e. broods without an induced host response). This second result is in accordance with the findings of two recent studies on Sundevall's jirds *Meriones*

crassus (De Bellocq *et al*. 2006) and greenfinches *Carduelis chloris* (Saks *et al*. 2006), and indicates that the PHA response *per se* is not a good predictor of parasite fecundity. The negative relationship between PHA response and parasite fitness in previously flea-exposed nests, however, shows that the PHA response is correlated to the bird's ability to *mount* an induced physiological or immunological reactions against parasites. It indicates that standard immune challenge protocols, like the PHA skin test, can reveal valuable information about the host's parasite defence capacity.

The investment in parasite defence is most likely associated with costs for the host in terms of energy and metabolites, and thus limited to individuals in good condition (e.g. Sheldon & Verhulst 1996; Norris & Evans 2000). Previous work has demonstrated strong and consistent evidence for condition-dependent effects on parasite defence and the strength of immune reactions in general (including the reaction against a PHA injection; e.g. Gershwin *et al*. 1985; Cook 1991; Lochmiller *et al*. 1993; Saino *et al*. 1997; Ing *et al*. 2000; Alonso-Alvarez & Tella 2001). Given this positive relationship, parasites might preferentially feed on hosts in bad condition (Coop & Holmes 1996; Christe *et al*. 1998; Roulin *et al*. 2003; Simon *et al*. 2003) or benefit from actively reducing their hosts' condition to keep defence mechanisms at low levels, leading to high exploitation rates and parasite virulence. Our results demonstrate, however, that beside the potential beneficial effects of exploiting a host in bad condition, low host condition can also hamper parasite fitness: hen flea females had a significantly higher reproductive success when the host was raised under favourable food conditions early in life. As neither the hosts' body mass, nor body size at the end of the nestling period significantly influenced the reproductive success of fleas (see also Heeb *et al*. 1996), this effect is unlikely due to differences in the nestlings' body surface, as for example found in bee-eaters *Merops apiaster* infested with ectoparasitic flies *Carnus hemapterus* (Valera *et al*. 2004). The food supplementation early in life might have permanently changed the composition of the nestlings' blood (Nijdam *et al*. 2005), its digestibility (Sarfati *et al*. 2005), or accessibility instead, and might thereby have increased the host's value for the parasite. It indicates that early developmental conditions experienced by the host can have long-term consequences for host–parasite interactions.

Interestingly, our results are in contrast to the findings of a recent study on *Xenopsylla ramesis*, a flea species exploiting rodent hosts (Krasnov *et al*. 2005). In this system, fleas laid more eggs when feeding on experimentally underfed hosts, which might be explained by the lower immunocompetence of animals in bad nutritional condition (Nelson 1984). These opposing results could be due to differences in the experimental design of the two studies: Krasnov *et al*. (2005) compared the reproductive success of parasites feeding on food deprived and control animals in the lab, while we used a food supplementation to increase the condition of the hosts in the wild. Alternatively, it might reflect differences in the co-evolutionary history of the two host–parasite systems.

We suggest that host-induced selective pressures can differentially shape parasite life history depending on the direction, strength and relative importance of host condition and host defence on parasite fitness. Assessing both the parasite effect on the host and the impact of the host on the parasite is thus essential for the understanding of the evolutionary dynamics of host–parasite systems.

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