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Induced responses of nestling great tits reduce hen flea reproduction

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The dynamics of host–parasite interactions depend to a large extent on the effect of host responses on parasite fitness. Exposure to parasites may induce behavioural or physiological responses in hosts that may reduce the subsequent survival or reproductive output of the parasite. Neonate hosts may further directly obtain immunologically active substances from their mother, for instance via milk in mammals or egg yolk in birds. However, the relative importance of maternally-derived and self-generated responses in inducing parasite resistance is poorly understood, especially in free-living vertebrates. Here we investigate the complementary effect of experimentally induced maternal and neonate responses in great tit (*Parus major*) hosts on the reproductive success of their common ectoparasite, the hen flea (*Ceratophyllus gallinae*). In the laboratory we measured the number of eggs and larvae produced by individual flea females collected from host nests. In addition, the total number of larvae produced by an experimentally set number of flea females in the host's nestbox was assessed under field conditions. There was no indication of maternally-transferred parasite resistance, since exposing the mother to fleas during the laying period did not affect the reproductive rate of fleas exploiting her offspring early or late in the nestling cycle. Independent of the maternal treatment, exposure of neonates to fleas early in the nestling period reduced the reproductive output of fleas late in the nestling cycle. The effect of the induced nestling response was seasonal, reducing flea reproduction in nests of early-breeding hosts but not in nests of late-breeding ones. Larvae production in the nestbox and in the laboratory was positively correlated, but under natural conditions the neonate response did not affect the size of the flea larvae population. Our results indicate induced responses as a means by which neonate avian hosts resist ectoparasites. Other factors, such as the environmental temperature and density-dependent larval competition, may be more important in determining the size of the future parasite populations.

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There is considerable evidence that ectoparasites have detrimental effects on bird reproduction and survival (reviewed by Loye and Zuk 1991, Clayton and Moore 1997). Previous studies have shown that a broad spectrum of life history traits such as laying date, hatching success (Christe et al. 1994, Oppliger et al. 1994), nestling growth and survival (Møller 1990, Møller et al. 1990, Richner et al. 1993) may be negatively affected by parasitism. Ectoparasites thereby exert selection pressure on hosts, and natural selection is presumed to favour hosts that evolved efficient defence strategies against parasites. Parasite-induced host responses are thus predicted to reduce the reproductive success of parasites.

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Host responses against ectoparasites may involve behavioural and physiological responses. Behavioural responses include parasite avoidance, activities that reduce the impact of the parasite, or the active removal of the parasite (Hart 1997). The most important defence mechanisms following parasite infection are based on immune responses (Roitt et al. 1996, Wakelin and Apanius 1997). Ectoparasites transmit saliva during blood sucking (Allen 1994) and the transferred proteins elicit a cascade of reactions including the production of immunoglobulins (Wikel et al. 1996, Wikel and Alarcon-Chaidez 2001). In domesticated vertebrates it was shown that these immune responses affect the morphology, physiology, reproduction or survival of ectoparasites (Brossard et al. 1991, Allen 1994, Randolph 1994, Wikel 1996, Wikel et al. 1996), and thus provide some protection from parasites to the host. It has not been investigated whether this type of host resistance occurs in free-living vertebrates, and how it affects the population growth of the ectoparasite. Under natural conditions resources are usually limited and this may restrict the expression of an efficient host response.

Our model system deals with the interaction between great tit (*Parus major*) hosts and their most common ectoparasite, the hen flea (*Ceratophyllus gallinae*). The case of nest-based ectoparasites is particularly interesting since both the adults and the nestlings may produce a response to the parasites, and the two responses may work separately or in concert (Apanius 1998). Previous studies in this system have shown that parasite-induced responses are beneficial to the host (Heeb et al. 1998): birds with nests that were experimentally infested during egg laying and post-hatching had fewer breeding failures, faster nestling growth, higher fledging success, and a higher offspring recruitment than birds that were exposed to parasites post-hatching only. One suggested explanation for the enhanced resistance of nestlings to fleas is the transfer of maternal immunity to offspring as an induced response to the local parasites. In support, Buechler et al. (2002) found higher immunoglobulin-concentration in the yolk of eggs produced by females that were exposed to hen fleas during egg laying as compared to the eggs of unexposed females. This result is in line with studies on domesticated animals, which demonstrated parasite-induced maternal effects in eggs (Rose et al. 1974, Yamamoto et al. 1975, Gottstein and Hemmeler 1985, Kowalczyk et al. 1985, Smith et al. 1994, Carlier and Truyens 1995). Gasparini et al. (2001) showed higher prevalence of antibodies against *Borrelia burgdorferi* sensu lato, a spirochaete transmitted by ticks, in eggs of kittiwakes (*Rissa tridactyla*) that breed in areas with higher prevalence and abundance of ticks. Further, in barn swallows (*Hirundo rustica*) the allocation of maternal immune factors to the eggs depends on laying order and parental phenotypic quality (Saino et al. 2002). The induced response of nestlings and its effects on parasites is less well documented. Some studies in domestic and captive birds suggest that the immune system of neonates is poorly developed and that small chicks therefore have little competence to build an effective immunological response (reviewed by Apanius 1998, Klasing and Leshchinsky 1998). It has further been suggested that the maternal-transfer of antibodies might prevent the neonatal immune response by binding and blocking antigenic targets (Apanius 1998, Klasing and Leshchinsky 1998).

It remains largely unclear whether induced responses are of functional significance with respect to resistance to parasites under natural conditions. One mechanism is that the host response leads to the control of the parasite population size or population growth, one suggested mechanism that it affects parasite reproduction. In the present study we investigate the effect of both the maternal and the neonates' host response on fertility and reproductive success of the parasite. Maternal effects were induced by exposing females to hen fleas shortly prior to egg laying (Heeb et al. 1998, Buechler et al. 2002), while nestling responses were induced through temporary flea infestation in the first days post-hatching. The two treatments were arranged in a two-way factorial design, which allowed us to investigate the effect of the maternal-induced response, the neonate induced response, as well as their combined effect on hen flea reproduction. If induced responses convey resistance, we expect to find a reduction in the reproductive performance of the parasite.

Materials and methods

The study was conducted in 1998 in a population of great tits in the Bremgartenwald, a forest near Bern, Switzerland (46°57′N, 7°28′E). Prior to the breeding season, i.e. in early March, all nestboxes were cleaned and nests containing fleas from the previous year were kept in the refrigerator at 4°C for later use. During the breeding season nestboxes were visited regularly from mid-April onwards to record the laying date of the first egg, clutch size, and the start of incubation. Between eleven days after the start of incubation and hatching, nests were inspected daily to determine the exact hatching date. Hatching date (here defined as day 0) was defined as the date on which at least half of the eggs had hatched.

The hen flea *Ceratophyllus gallinae* (Siphonaptera: Ceratophyllidae), a common, blood-sucking ectoparasite in great tit nests, was used for the controlled infestations. It occurs in at least 75 bird species (Smit 1957) and is particularly abundant in tits (Harper et al. 1992, Tripet and Richner 1997). The hen flea reproduces in the host's nest. Adult fleas feed on blood from adult and nestling birds, while larvae feed on detritus

and undigested blood excreted by the adult fleas (Marshall 1981, Lehane 1991). A few adult fleas leave with the fledglings and normally hundreds remain inside the nest in cocoons until the next breeding season (Rothschild and Clay 1952, Tripet and Richner 1999b).

We aimed to induce maternal responses by infesting nests shortly prior and during egg laying with 40 fleas, and neonate responses by infesting nests two days post-hatching with 40 fleas. With respect to the pre-laying treatment we will further refer to maternal-control and maternal-infested nests respectively. Similarly, for the post-hatching treatment we will use the terms neonate-control and neonate-infested. This 2×2-design with sets of four nests allowed us to assess the combined effects of the maternal-treatment and the neonate-treatment on parasite reproduction. The grouping criterion for the four nests in a set was based on the date when the newly built nests reached a height of 10 cm. All nests were heat-treated for two minutes in a microwave oven to eliminate nest-based ectoparasites. We then assigned nests randomly to one of the four treatments. On the day the female laid the seventh egg, a second heat treatment was applied. Nests of the control group were treated the same way. There was no effect of the pre-laying infestation on the probability of clutch initiation by female great tits ($n = 128$ nests, $\chi^2 = 0.85$, $df = 1$, $P = 0.36$). Among great tit females that initiated a first clutch, the mean time span between the dates of pre-laying treatment and first egg laying was 9.65 ($SE = 1.01$) and 10.39 ($SE = 0.94$) days for controls and infested nests, respectively (Mann Whitney U, $Z = -0.78$, $P = 0.43$). Similarly, laying date (mean $\pm$ SE: uninfested nests 115.6 ± 1.1, infested nests 113.9 ± 0.7, Mann Whitney U, $Z = -1.03$, $df = 1$, $P = 0.30$) and clutch size (uninfested nests 8.37 ± 0.2, infested nests 8.17 ± 0.15, Mann Whitney U, $Z = -0.93$, $df = 1$, $P = 0.35$) were not significantly related to pre-laying treatment.

When chicks were two days old we first heat-treated all nests, and then infested half of the previously infested nests and half of the previous control nests with 40 adult fleas, thus completing the 2×2-design. Fleas were then left in the nests for three days. Five days post-hatching we collected ten flea females from the infested nests to investigate their reproductive performance in the laboratory and then heat-treated all nests. To collect individual female fleas, nests were put in a plastic-tray (50×40 cm) and by shaking we induced the fleas to leave the nest material. Control nests received a similar treatment as experimental nests. The collected fleas were put individually in polypropylene tubes (length: 100 mm, width: 12 mm) for egg laying. In the field, the tubes were temporarily stored in a cooling box at 4°C. In the laboratory the tubes were placed in an incubator at 20°C and 75% humidity. After two days we examined the number of eggs laid in the tubes using a pair of binoculars. Five days later we counted the larvae in order to assess hatching success of flea eggs.

To assess the effect of the maternal response, the neonate response, and their combination on flea reproduction simultaneously, we infested all four groups 10 days post-hatching with 40 female and 20 male fleas. Six days later, i.e. 16 days post-hatching, 10 female fleas were collected from each nest and kept in the laboratory to assess reproductive rate. Further, all nests were collected and put individually into round containers (height: 20 cm, width: 10 cm, with a net lid). To minimise cannibalism among flea larvae in the collected nests we added 30 grams of food supplement, consisting of 20:3:2 dry pulverised dog food, dried pig blood, and yeast (Silvermann et al. 1981) into the nest material. The nests were then placed in an incubator at 20°C and 75% of humidity.

Hen fleas stop egg laying after one day without a blood meal (M.W., pers. obs.) and the eggs develop into larvae in 5 to 7 days under the given conditions (M.W., pers. obs.). On day 8 we removed the containers from the incubator and emptied them over a net into a funnel. These funnels were placed in a Berlese apparatus. The combination of a temperature, humidity and light gradient caused larvae to move downwards within the nest. At the lower edge of the nest the flea larvae then dropped into a collecting glass that was filled with ethanol. The nests were left in the funnels for 120 h. No further larvae were collected beyond this time period. The larvae were counted under 6-time magnification. To estimate the repeatability of the counting, 8 samples were counted twice and blindly. The repeatability (Lessels and Boag 1987) was high ($r = 0.99$, $F_{7,8} = 274.79$, $P < 0.001$).

Statistical analyses were performed using the JMPIn statistical package (Sall and Lehmann 1996) and GLMStat (Beath 2002). We fitted generalised linear models using normal or binomial error. Following a stepwise backward procedure we reduced the maximal model to a minimal adequate model by removing non-significant terms in a hierarchical fashion. Significance levels in all statistical tests are two-tailed. Maximal models included the following predictor variables: pre-laying treatment (factor), post-hatching treatment (factor), date (covariate), and their mutual interactions. To control for potential variation in the extent of maternal effects between nests due to differences in the duration of pre-laying flea exposure, we added the time span between the dates of pre-laying infestation and the laying of the first egg as a covariate (duration of pre-laying flea exposure). In addition, final tests included a covariate indicating the expected percentage of eggs within the clutch with elevated immunoglobulin (IgG) levels, calculated using the minimal time lag of seven days for an induced antibody response indicated by Buechler et al. (2002). To assess

in which stage of reproduction our experiment affected flea reproductive output we analysed, first, the mean probability of egg laying, second, flea fecundity given egg-laying, and third, the hatching success of eggs produced. Finally we investigated the total mean production of larvae per female and nestbox in relation to treatment.

Binominal error (or logistic regression) was used to analyse the probability of egg laying by female fleas, where the number of eggs was taken as response variable, and the number of females as the binominal denominator. We conservatively used F-tests instead of χ^2-tests to evaluate the significance predictor variables, because of overdispersion in the data (i.e. scale parameter > 1). Normal error (or analysis of covariance) was used to analyse the mean egg and the mean larvae production of flea females, as well as the mean hatching success of flea eggs. Prior to the analysis the data on mean hatching success, calculated per female and then per nest, were arcsin-transformed. All data sets passed tests (i.e. $\alpha > 0.05$) for normality (Shapiro–Wilk W-test) and heterogeneity of variance (O'Brien T-test). Differences in sample sizes between analyses are due to the exclusion of nests, in which all chicks died. Only nests that fledged chicks were taken into account for the analyses of flea reproduction on day 16. Sample size differences between the analyses of egg number and hatching success on day 16 are due to the loss of five samples prior to hatching. Mean values are presented with SE.

Results

Flea reproduction on day 5: laboratory data

The effect of the experimentally induced maternal response on flea reproduction during the early nestling stage was investigated by exposing flea females to nestlings from day 2 until day 5. The mean probability of egg laying on day 5 was 0.93 ± 0.036 ($n = 22$) for maternal-control nests and 0.94 ± 0.022 ($n = 21$) for maternal-infested nests. This difference was not significant (logistic regression, $F_{1,41} = 0.11$, $P = 0.75$). In maternal-control nests, laying fleas produced on average 6.71 ± 0.30 eggs, hatching success was $79 \pm 3\%$ and the overall mean larvae production was 5.21 ± 0.33. In maternal-infested nests the mean egg production was 6.24 ± 0.38, hatching success was $83 \pm 2\%$ and the overall mean larvae production was 5.07 ± 0.28. Pre-laying infestation, date and their interaction did not explain a significant part of the variation in flea fecundity, hatching success and number of larvae (all P-values > 0.13). In addition, measures of flea reproduction were not associated to duration of pre-laying flea exposure (all P-values > 0.63) or the expected percentage of eggs with elevated IgG-levels (all P-values > 0.21).

Flea reproduction on day 16: laboratory data

The effect of the maternal response, the neonate response and their combination on flea reproduction was investigated after allowing flea females to feed on nestlings from day 10 to 16. The mean probability of egg laying among treatment groups varied between 0.89 and 0.94, and was independent of the pre-laying treatment, the post-hatching treatment, and their mutual interaction (logistic regression, all P-values > 0.14). Further, the duration of pre-laying laying flea exposure ($F_{1,71} = 1.32$, $P = 0.25$) and the expected percentage of eggs with elevated IgG-levels ($F_{1,71} = 1.35$, $P = 0.25$) were not significant. The mean number of eggs, the mean hatching success and the overall mean larvae production were significantly related to post-hatching treatment, collection date and the interaction between post-hatching treatment and collection date (Table 1). In addition, the pre-laying treatment and the interaction between the pre-laying treatment and the post-hatching treatment were not significant in either analysis (Table 1). There was also no indication for an effect of the duration of pre-laying laying flea exposure (all P-values > 0.70) and the expected percentage of eggs with elevated IgG-levels (all P-values > 0.45) on any measure of flea reproduction. The significance of the interaction between post-hatching treatment and collection date indicates that the effect of treatment was dependent on the season. Flea reproduction increased with season in the neonate-infested nests, while the reproduction was independent of date for neonate-control nests (Fig. 1).

Flea reproduction under natural condition

The effect of the maternal response, the neonate response and their combination on flea reproduction was further assessed using the total number of larvae produced by female fleas that were allowed to reproduce in the nestbox from day 10 to 16. The overall mean number of progeny per nestbox was 2750 larvae, ranging between 178 and 4757 individuals. The mean larvae production was significantly related to date (ANCOVA, $F_{1,71} = 5.49$, $P = 0.02$). In addition, the pre-laying treatment, post-hatching treatment, their mutual interaction and their second order interactions with date were not statistically significant (all P-values > 0.06). Maternal transfer of resistance was also not indicated by the duration of pre-laying laying flea exposure ($F_{1,70} = 0.0009$, $P = 0.97$) or the expected percentage of eggs with elevated IgG-levels ($F_{1,70} = 0.12$, $P = 0.73$). The overall larvae production increased progressively with season (Fig. 2a).

We further investigated whether the effect of season on larvae production could be explained by the increase in the mean environmental temperature with season.

Table 1. Analyses of the mean number of eggs, hatching success (arcsin-transformed) and the mean number of larvae per female per nest on day 16. The final model contains significant terms only. Rejected terms of major interest are also given. The total variance is given by the null model, which contains the constant only. The full model gives the residual variance. The difference between the two variances represents the variance explained by the model.

Model and predictor variables	df	SS	F	P
Number of eggs:				
Null model	72	93.66		
Minimal adequate model	69	78.46		
Collection date	1	11.47	10.08	0.002
Post-hatching treatment	1	4.98	4.38	0.040
Collection date × post-hatching treatment	1	4.77	4.19	0.044
Rejected terms:				
Pre-laying treatment × post hatching treatment	1	1.71	1.50	0.23
Pre-laying treatment	1	0.21	0.18	0.67
Hatching success:				
Null model	67	1.52		
Minimal adequate model	64	1.17		
Collection date	1	0.12	6.40	0.014
Post-hatching treatment	1	0.077	4.22	0.044
Collection date × post-hatching treatment	1	0.24	13.03	0.0006
Rejected terms:				
Pre-laying treatment × post hatching treatment	1	0.0029	0.16	0.69
Pre-laying treatment	1	0.013	0.69	0.41
Number of larvae:				
Null model	67	147.92		
Minimal adequate model	64	122.90		
Collection date	1	12.82	6.67	0.012
Post-hatching treatment	1	10.37	5.39	0.023
Collection date × post-hatching treatment	1	9.76	5.08	0.027
Rejected terms:				
Pre-laying treatment × post hatching treatment	1	3.08	0.62	0.208
Pre-laying treatment	1	1.62	0.84	0.362

Larvae production was significantly and positively related to the mean environmental temperature ($F_{1,71} = 12.13$, $P < 0.001$, Fig. 2b), and thereby statistically explained the effect of date ($F_{1,70} = 0.0042$, $P = 0.95$). There was an increase of 84 larvae per 1°C raise of the mean temperature.

We finally compared the mean larvae production per nestbox with the laboratory data on the reproductive output of individual females collected on day 16. Flea reproduction measured in the field highly correlated to the estimates obtained in the laboratory from the corresponding host nest ($F_{1,71} = 9.86$, $P = 0.0025$; Fig. 3).

Discussion

In this study we investigated the effect of maternal-induced and neonate-induced responses against hen fleas in great tits. There was no indication of maternal-transferred resistance, since none of the parameters of flea reproduction investigated were affected by the maternal treatment. Controlled flea infestation during the pre-laying period did not influence later flea reproduction neither under laboratory or field conditions. Maternal transfer of resistance could be expected, since Buechler et al. (2002) found significantly elevated IgG-concentrations in the eighth egg of the laying sequence in great tit clutches of mothers that were exposed to hen fleas during the laying period from the first egg onwards. In the present study the duration of pre-laying flea exposure exceeded the minimal time span of seven days needed for antibody transfer suggested by Buechler et al. (2002). Accordingly, 58% of eggs in clutches initiated after a shorter exposure period likely conveyed elevated IgG-values as well. The IgG-increase in eggs might enhance the flea tolerance of newborn nestlings (Buechler et al. 2002), but the present flea reproduction data and field data by Heeb et al. (1998) do not indicate maternal transfer of parasite resistance. It is important to note, however, that both studies used quantitative measures of flea reproduction only, and it cannot be excluded that maternal effects affected other flea life history traits such as larval development, larval survival or adult size. For instance, Casu et al. (1997) showed that induced responses in sheep engender growth inhibition in parasitic fly larvae. Future studies should therefore include quantitative as well as qualitative measurements.

Induced neonate responses affected the later reproductive output of individual flea females, but this effect was dependent on season. Exposure to hen fleas during the initial days post-hatching reduced the hatching success of flea eggs and the number of larvae produced at the end of the nestling cycle in early host broods,

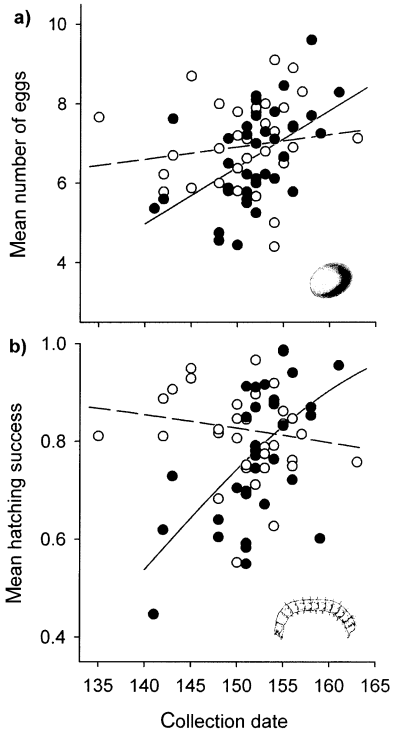

Fig. 1. Seasonal variation in a) the mean egg number and b) the mean hatching success of flea clutches, calculated per female and per nest, with respect to the post-hatching treatment. The open circles and the broken line (a: $y = 2.29 + 0.031 \times x$; b: $y = (\sin(-1.26 - 0.0030 \times x))^2$ indicate the variation among non-infested nests, closed circles and solid line (a: $y = -14.99 + 0.143 \times x$; b: $y = (\sin(-1.88 + 0.017 \times x))^2$ indicate the infested nests. The equations and plots for hatching success reflect back-transformation of arcsin-transformed data used in the analysis.

whereas no effect was found in late broods. The seasonal decline in the effect of neonate-induced responses on flea reproduction may have resulted from a decline in host phenotypic or genetic quality with breeding date. Immunocompetence of hosts strongly depends on nutritional condition, as determined by the quality or quantity of food supply (Gershwin et al. 1985, Cook

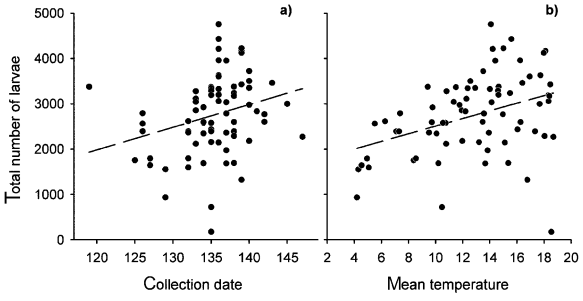

Fig. 2. Total larvae production under natural conditions in relation to a) date ($r^2 = 0.072$; $y = -4832.41 + 50.09 \times x$) and b) mean ambient temperature ($r^2 = 0.14$; $y = 1663.30 + 84.29 \times x$).

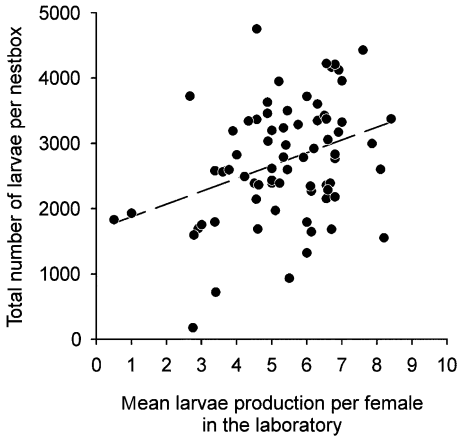

Fig. 3. Within-nest relationship between the total larvae production under natural conditions and the larvae production of individual females in the laboratory ($F_{1,71} = 9.86$, $P = 0.0025$, $r^2 = 0.12$; $y = 1675.71 + 196.35 \times x$).

1991, Lochmiller et al. 1993, Saino et al. 1997). Nestling condition declined with season in the year of the present study (unpubl.), which may have rendered chicks in late-hatched broods less capable of mounting an immune response than early-hatched ones. Such a seasonal decline in immunocompetence was for instance shown in magpies *Pica pica* (Sorci et al. 1997). Further, genetic variation in resistance against hen fleas that is associated to breeding date might be important. Origin-related variation in cell-mediated immunity is documented for the great tit population here studied (Brinkhof et al. 1999), but it is not known whether this variation reflects heritable resistance to hen fleas. Finally, the seasonal variation in traits of the hen fleas, to which the neonates were initially exposed, might have affected the induced response quantitatively or qualitatively. For instance, the antigenic challenge by the parasite might have varied quantitatively or qualitatively with season, due variation in abiotic factors like temperature or humidity, or alternatively, due to genetic variation in the composition of the parasite population. Unfortunately we have no data to substantiate these hypotheses.

The total reproductive output of fleas measured under field condition at the end of the host nestling cycle increased with season, and this effect was largely explained by the increase in environmental temperature. However, there was no additional effect of the induced neonate response on the size of the larvae population at the end of the nesting period. A similar seasonal increase in the reproductive output of ectoparasites was shown in studies on barn owls (*Tyto alba*; Roulin 1999) and barn swallows (Møller 2000), while the strong effect of ambient temperature on the reproduction of ectoparasites is in accordance with a previous study on hen fleas (Tripet and Richner 1999b). Flea reproduction is further density-dependent (Tripet and Richner

1999a), due to food competition among flea larvae (Tripet et al. 2002). Compared to the effect of temperature and density, the neonate-induced resistance apparently plays a minor role in determining the future parasite population under natural conditions.

The novel finding of our study is that a neonate-induced response may generate host resistance against an ectoparasite under natural conditions. The mechanisms behind this effect and the costs and benefits associated to this self-induced responses to offspring fitness are the subject of future study.

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