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Parasitism and developmental plasticity in Alpine swift nestlings

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Summary

1. Development plasticity is a common evolutionary and phenotypic response to poor growth condition, in particular in organisms with determinate growth such as most birds and mammals. Because various body structures can contribute differently to overall fitness, natural selection will adjust the degree of plasticity of each structure to its proportionate contribution to fitness at a given life stage.

2. Two non-mutually exclusive mechanisms can account for plasticity in the growth of offspring to compensate for the effect of parasites. First, if parasite infestation levels fluctuate over the nestling period, parasitized young may show reduced growth until peak parasite infestation, and accelerated growth once the conditions improve (the accelerated growth hypothesis). Secondly, if the period of tissue maturation is not fixed in time, hosts may grow slower than parasite-free hosts but for a longer period of time (the delayed maturation hypothesis).

3. To test whether hosts compensate for the effects of parasites on their development, the load of the blood-sucking louse-fly *Crataerina melbae* Rondani in the nests of Alpine swifts, *Apus melba* Linnaeus, was increased or decreased experimentally. Parasite prevalence was 100% in both treatments, but intensity (no. of parasites per nestling) was significantly lower for deparasitized nestlings. In both treatments, parasite intensity increased up to halfway through the rearing period (i.e. 30 days of age) and decreased afterwards.

4. In line with the accelerated growth hypothesis, wings of parasitized nestlings grew at a lower rate than those of deparasitized ones before the peak of parasite infestation, but at a greater rate after the peak. Louse-flies had no significant effect on the growth of body mass. In agreement with the delayed-maturation hypothesis, wings of parasitized nestlings grew for 3 additional days and were of similar size at fledging as in deparasitized birds.

5. In summary, the present study shows in a wild bird population that nestling hosts can compensate for the effect of parasitism on their phenotype. It emphasizes the need to take the dynamics of parasite populations into account in studies of host–parasite relationships, and to investigate the effect of parasites on host development over the entire growing period rather than only at fledging, as employed traditionally.

Key-words: *Apus melba*, compensatory growth, *Crataerina melbae*, delayed maturation, host–parasite interaction.

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Introduction

Parasites extract body resources of other species, and are therefore expected to affect the development of their hosts (Price 1980). Evidence of a cost of parasitism

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is usually based on experiments where parasite load is manipulated early during host development and the effect measured close to the end (e.g. Møller 1990; Richner, Oppliger & Christe 1993; Clayton & Tompkins 1995). In species where the effect of parasites on early growth can be compensated later, the effects of parasites may therefore not be detected. Given the potentially long-term fitness consequences of poor early growth caused by parasites (Gebhardt-Henrich & Richner 1998), selection will favour such compensatory strategies, provided that compensation is not, in itself, too costly (Metcalfe & Monaghan 2001). Plasticity in the development may lead to partial or full compensation, in particular for those body structures where the long-term costs of suboptimal development are high. Developmental plasticity as a response to food shortage or low temperatures is well documented (reviewed in Schew & Ricklefs 1998; Metcalfe & Monaghan 2001) but poorly known as a response to parasites.

Two compensatory strategies can be used to face poor growing conditions. First, after a period with harsh conditions and poor growth, individuals may show accelerated growth if condition improves (Fig. 1, curve II). With parasites, this may occur if populations fluctuate or if the initial host response is very costly. Although the population dynamics of ectoparasites has not attracted much attention, in some species an increase in population size over the first half of the host's rearing period is followed by a decrease (e.g. Summers 1975; Roulin 1998). Secondly, if the length of the maturation is not fixed, parasitized individuals can grow for a longer period of time to compensate for the parasite-induced reduction in growth rate (Fig. 1, curve III). The accelerated growth and delayed maturation hypotheses can work in concert, as parasitized nestlings may simultaneously alter their growth rate in relation to parasitism and extend the growth period. Developmental plasticity may therefore help parasitized individuals to compensate for the effects of parasites on their development and, as a consequence, to catch up in size to reach the level of parasite-free individuals (Fig. 1, curve I). In contrast, if organisms are unable to perform developmental plasticity or show incomplete compensatory growth, they will become permanently stunted at a smaller final size (Fig. 1, curve IV; Richner 1989; Potti & Merino 1996).

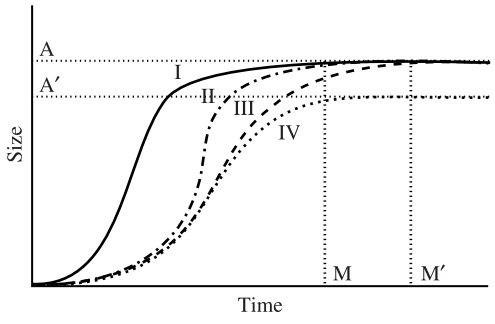

Fig. 1. Growth curves of parasitized (II–IV) and parasite-free (I) individuals. The growth curve I represents the optimal development of an individual facing no parasites, where A and M are the optimum asymptotic size and age at maturity. Although parasites can reduce the growth of their hosts, parasitized individuals may compensate for their slower growth by accelerating their development once the population of parasites decreases (II) or by taking longer (M′) to reach final size (III). If the development of organisms is not plastic, parasitized individuals become permanently stunted at a smaller final size (A′; IV).

The goal of the present study is to examine the effects of the blood-sucking louse-flies *Crataerina melbae* on nestling development in the Alpine swift *Apus melba*. This non-passerine bird is suited to examine whether nestlings alter their growth pattern adaptively in response to varying levels of parasite infestation because they reduce growth rate and delay fledging in prolonged periods of food deprivation, indicating that development is plastic (Koskimies 1950). When populations of parasites fluctuate we predict, under the accelerated growth hypothesis, that experimentally parasitized nestlings grow less rapidly at the peak of parasite infestation but more rapidly once this peak has passed, compared to deparasitized ones. Under the delayed maturation hypothesis, we predict that experimentally parasitized nestlings grow for a longer time period. If these two mechanisms allow nestlings to reduce the cost of parasitism, parasitized nestlings should fledge with a similar size to deparasitized ones.

Materials and methods

MODEL ORGANISMS

The Alpine swift is a 90-g migrant insectivorous apodiform bird that spends most of its life on the wing, landing only for breeding purposes. It is socially monogamous and reproduces in colonies of a few to several hundred pairs located in holes of cliffs or tall buildings. One clutch of one to four eggs (mean 2·6) is produced per year, and both parents incubate for approximately 20 days (Arn 1960). Offspring fledge at an age of 50–70 days (Arn 1960). Before fledging, nestlings lose about 4·5% of their body mass (Arn 1960). At fledging, parental care ceases and young do not return to the colony until the following year (P.B. personal observation; see also Tarburton & Kaiser 2001 for the common swift *Apus apus* L.). Body mass and wing/tail shapes need, therefore, to be particularly fine-tuned in swifts (Martins 1997). Alpine swifts are sexually mature in the second or third year, and the oldest recorded individual was 26 years old (Arn 1960).

Alpine swifts are parasitized by a 7-mm-long blood-sucking louse-fly *C. melbae* (Diptera, Hippoboscidae) that feeds exclusively on this species (Tella *et al*. 1998). Larvae develop in the maternal abdomen (viviparity) until the prepupae stage, when they are released into the surroundings of the nests and pupate immediately (Bequaert 1953). Adult parasites emerge slightly before their nestling hosts hatch. *C. melbae* is flightless and can switch hosts rapidly by walking (P.B. personal

observation). Several Hippoboscidae have been shown to transmit blood parasites to their host (Baker 1967), but to date this hypothesis has not yet received support for *C. melbae* (Tella *et al.* 1995; P.B. personal observation).

MANIPULATION OF ECTOPARASITE LOAD

Fieldwork was carried out between May and August in 1999, 2000 and 2001 in a Swiss Alpine swift colony located under the roof of an old building in Solothurn (47°12′ N, 7°32′ E) holding about 50 breeding pairs. At the beginning of each breeding season nests were visited every other day to determine the date of clutch completion, and daily around hatching to determine the hatching date of the first egg (day 0).

To create two groups of nests differing in ectoparasite load, we transferred ectoparasites from a donor brood (referred to as 'deparasitized brood') to a receiver brood ('parasitized brood'). For this purpose, nests with a similar clutch size (Pearson's correlation, $r = 0{\cdot}39$, $N = 50$, $P = 0{\cdot}005$), hatching date ($r = 0{\cdot}95$, $N = 50$, $P < 0{\cdot}001$), brood size at 10 days after hatching ($r = 0{\cdot}57$, $N = 50$, $P < 0{\cdot}001$) and ectoparasite load at 10 days after hatching ($r = 0{\cdot}45$, $N = 50$, $P = 0{\cdot}001$) were matched in 11, 20 and 19 pairs of nests in 1999, 2000 and 2001, respectively. Manipulations of louse-fly load started 10 days after hatching because ectoparasites are rarely found on offspring before this age (Roulin *et al.* 2003). As *C. melbae* is highly mobile and deparasitized nests were re-infested frequently by ectoparasites, we transferred ectoparasites every 5 days until nestlings were 50 days old. For each pair of nests we extracted and counted ectoparasites on all nestlings, and then put all ectoparasites into the parasitized nest of its pair. At the start of the experiment there were no differences between treatments in clutch size, hatching date or brood size and ectoparasite load 10 days after hatching (all comparisons $P > 0{\cdot}43$). Although we added parasites to nests already infested naturally, the number of louse-flies per parasitized nestling remained within the natural range of infestation, as observed in other Swiss colonies (Roulin *et al.* 1998; P.B. personal observation). At each manipulation, nestling wing length and body mass were measured to the nearest millimetre and 0·1 g, respectively. After 20 days of age, Alpine swift nestlings start to invade neighbouring families and are frequently adopted by foster families (Arn 1960; Bize, Roulin & Richner 2003). To prevent adoption, we checked all nests daily between 20 and 50 days after hatching, and returned each nest-switcher immediately to its natal nest (Bize *et al.* 2003). Shortly before fledging, nests were visited daily and nestlings measured as described above. We did not assess the growth of tarsus in relation to the manipulation of ectoparasite load because, in this species with very short tarsi (mean 17·6 mm), the tarsus has already reached its final length at the time when we started to manipulate ectoparasite load (P.B. personal observation).

STATISTICS

To avoid pseudo-replication the breeding pair was used as the unit of analysis, and hence if a pair was recorded in more than one year we included only its first breeding attempt. This restriction reduced our sample to 78 experimental broods (38 of 50 parasitized and 40 of 50 deparasitized broods). For the same reason, statistical tests were carried out on mean sibling values rather than on individual nestlings. Only nestlings that survived up to fledging were included in the analyses. The number (x) of louse-flies found on nestlings was $\log_{10}(x + 1)$ transformed before analyses to fit a normal distribution. Growth rates (R) of wing length (W) and body mass (M) between interval t and $t + i$ were calculated with the formulae: $R_W = (W_{t+i} - W_t)/i$, and $R_M = (M_{t+i} - M_t)/i$. Growth rates of wing and body mass were calculated for intervals between days 10 and 20, 20 and 30, 30 and 40, 40 and 50, and 50 and fledging. Body condition at fledging was calculated as the residuals from a linear regression of body mass on wing length. The effect of experimental manipulation on parasites load was analysed using repeated-measures ANOVA with mean number of parasites per nestling and per nest at 10, 20, 30, 40 and 50 days after hatching as repeats. Effect of parasite manipulation on growth rate was analysed using repeated-measures ANOVA with wing growth rate on day intervals 10–20, 20–30, 30–40, 40–50 and 50–fledging as repeats. The same procedure was applied to growth rate of body mass. Mean values are given ±1 SE, statistical tests are two-tailed and P-values smaller than 0·05 are considered significant.

Results

EFFECT OF EXPERIMENTAL MANIPULATION ON ECTOPARASITE LOAD

Although parasite prevalence was 100% in both treatments, the experimental manipulation of parasite load significantly affected mean parasite intensity over the period of 10–50 days after hatching with 7 ± 1 louse-flies per nestling in deparasitized nests vs. 17 ± 1 per nestling in parasitized ones (repeated-measures ANOVA with log-transformed louse-fly load on days 10, 20, 30, 40 and 50 as repeats; treatment as factor: $F_{1,64} = 44{\cdot}35$, $P < 0{\cdot}0001$; Fig. 2). The number of louse-flies per nestling varied significantly over the rearing period with a peak of infestation at day 30 (age as factor: $F_{4,61} = 74{\cdot}80$, $P < 0{\cdot}0001$; Fig. 2). The treatment by age interaction was also significant ($F_{4,61} = 12{\cdot}59$, $P < 0{\cdot}0001$), indicating a treatment effect on age-related dynamics of parasite infestation (Fig. 2).

EFFECT OF ECTOPARASITES ON REPRODUCTIVE SUCCESS

Complete brood failure tended to be more frequent in parasitized than deparasitized broods (eight of 38

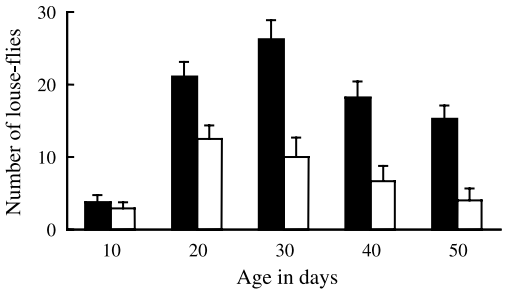

Fig. 2. Mean number of louse-flies per parasitized (solid bars) and deparasitized (open bars) Alpine swift nestlings in relation to age. Bars represent one standard error.

parasitized broods vs. three of 40 deparasitized broods failed, 21·1 vs. 7·5%; Fisher's exact test: d.f. = 1, $P = 0{\cdot}11$). Among successful breeders, brood size at fledging was not significantly different between the two treatments (parasitized broods: $1{\cdot}87 \pm 0{\cdot}12$ nestlings at fledging, $N = 30$; deparasitized broods: $2{\cdot}11 \pm 0{\cdot}11$, $N = 37$; Wilcoxon's two-samples test: $Z = 1{\cdot}40$, $P = 0{\cdot}16$).

EFFECT OF ECTOPARASITES ON NESTLING DEVELOPMENT

As expected from the accelerated growth hypothesis, before the peak of parasite infestation wing growth rate was lower in parasitized nestlings than deparasitized ones, but higher thereafter (repeated-measures ANOVA with wing growth rate on day intervals 10–20, 20–30, 30–40, 40–50 and 50–fledging as repeats; treatment by age interaction: $F_{4,56} = 2{\cdot}88$, $P = 0{\cdot}031$, Figs 3a and 4). Overall, there was no significant difference in wing growth rate between parasitized and deparasitized nestlings (treatment as factor: $F_{1,59} = 1{\cdot}84$, $P = 0{\cdot}18$, Fig. 3a), and in the two treatments wing growth rate slowed down when wings approached final size (age as factor: $F_{4,56} = 175{\cdot}67$, $P < 0{\cdot}0001$, Fig. 3a). Growth rate of body mass did not differ between parasitized and deparasitized nestlings (repeated-measures ANOVA with growth rate of body mass on day intervals 10–20, 20–30, 30–40, 40–50 and 50–fledging as repeats; treatment as factor: $F_{1,59} = 0{\cdot}20$, $P = 0{\cdot}66$; treatment by age interaction: $F_{4,56} = 0{\cdot}81$, $P = 0{\cdot}52$, Fig. 3b). Growth rate slowed down until the age of 50 days, followed by a typical body mass recession (age factor: $F_{4,56} = 161{\cdot}73$, $P < 0{\cdot}0001$, Fig. 3b).

In agreement with the delayed-maturation hypothesis, parasitized nestlings fledged 3 days later than deparasitized ones (Table 1). At the age of 50 days, wings of parasitized nestlings were significantly shorter (parasitized nestlings: $200{\cdot}3 \pm 1{\cdot}9$ mm, $N = 29$ broods; deparasitized nestlings: $207{\cdot}8 \pm 1{\cdot}7$ mm, $N = 37$ broods; Student's t-test: $t = 3{\cdot}00$, $P = 0{\cdot}0038$). Combined with the fact that parasitized nestlings grew their wings at a faster rate at the end of the rearing period, the longer time period spent in the nest allowed parasitized nestlings to fledge with wing lengths similar to deparasitized ones (Table 1). Body mass and body condition at fledging were similar in parasitized and deparasitized young (Table 1).

Discussion

Adaptive developmental plasticity is the process by which an organism alters its growth and maturation to counter the impact of detrimental rearing conditions (Schew & Ricklefs 1998). Previous studies found that parasites reduce body size or body condition of their hosts (e.g. Richner *et al.* 1993), and some studies also

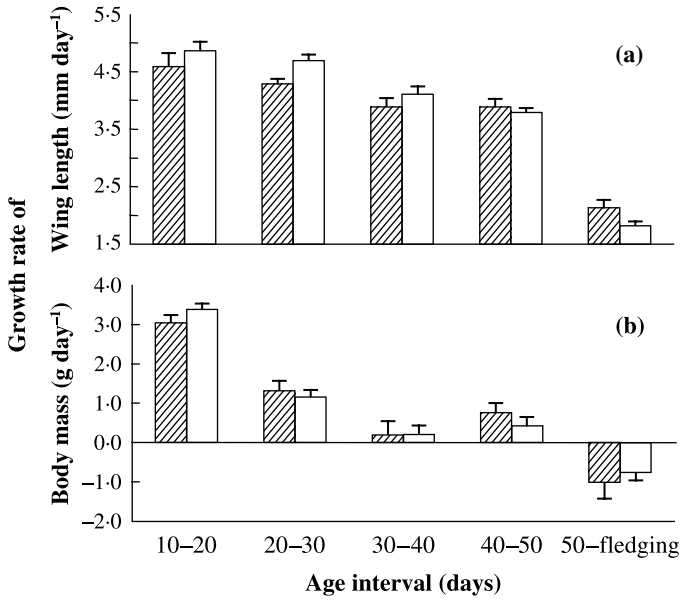

Fig. 3. Mean growth rate of (a) wing length and (b) body mass of parasitized (hatched bars) and deparasitized (open bars) Alpine swift nestlings in relation to age. Fledging took place on average at 61 and 58 days after hatching in parasitized and deparasitized nestlings, respectively. Bars represent one standard error.

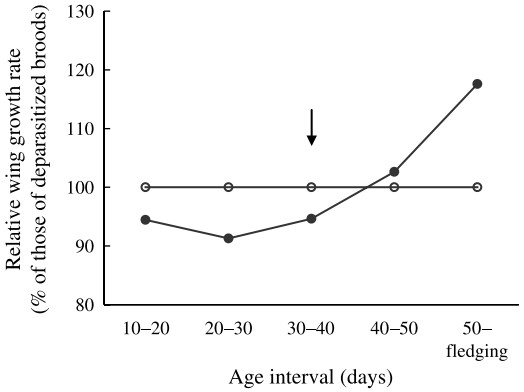

Fig. 4. Age-related wing growth rate of parasitized Alpine swift nestlings (closed symbols) in relation to deparasitized ones (open symbols). The arrow signals the peak of parasite infestation.

reported an increase in the wing growth rate of experimentally parasitized hosts (Saino, Calza & Møller 1998; Szép & Møller 1999). To our knowledge the present experimental study is the first to investigate developmental plasticity in relation to the intranest population dynamics of parasites. We showed that wing growth rate of parasitized hosts decelerated during the peak infestation and accelerated thereafter. Parasitized nestlings were also found to fledge later but at a similar size to deparasitized ones. These findings support the hypothesis that developmental plasticity is not only a strategy to compensate the impact of temporary poor feeding conditions or low ambient temperatures, but also to compensate the effect of parasitism. As developmental plasticity occurs in nestlings of many species, such as aerial insectivorous birds (Emlen *et al*. 1991), seabirds (Boersma 1986), ducks (Street 1978) and ptarmigans (Morse & Vohra 1971; Turner & Lilburn 1992), adaptive parasite-induced developmental plasticity might have been overlooked.

PARASITISM AND ACCELERATED GROWTH

The accelerated growth hypothesis predicts that parasitized nestlings may show reduced growth until the peak parasite infestation, and accelerated growth once the conditions improve. Under this scenario, parasitized hosts adaptively allocate more resources into physiological processes associated with body maintenance and parasite resistance during periods of heavy infestation while favouring physiological processes associated with growth during less stressful periods. In line with the accelerated growth hypothesis, parasitized nestlings grew their wings at slower rates than deparasitized nestlings during the first half of the growing period (i.e. up to peak infestation), and at faster rates thereafter (i.e. once infestation levels decreased). This suggests that, as in the barn swallow *Hirundo rustica* L. (Saino *et al*. 1998) and the sand martin *Riparia riparia* L. (Szép & Møller 1999), where parasitized young grew wings at a faster rate than deparasitized ones, wing growth rate is adjusted adaptively to the level of parasitism in the Alpine swift. With respect to growth rate of body mass, we did not find similar effects of parasitism. This may reflect a hierarchy of tissue preservation, with fat reserves having priority over flight feather growth. In other words, nestlings cannot afford to reduce their mass growth because of the high risk of starvation in case of an upcoming food shortage. Indeed, Alpine swifts prey solely on aerial insects, and this food resource shows large and unpredictable variations over the breeding season. An alternative explanation is that body mass growth reflects mainly parental quality (Bize, Roulin & Richner 2002), thereby masking any weak parasite-induced offspring adjustment of resource allocation between body mass growth and other functions.

PARASITISM AND DELAYED MATURATION

The delayed maturation hypothesis states that parasitized nestlings can compensate for an initial poor growth by taking longer to complete development and maturation. In addition, given that nestling swifts do not receive parental care and do not roost in the colony after their first flight (Tarburton & Kaiser 2001; P.B. personal observation), they should not fledge prematurely and should wait until optimal body mass and shape are reached. Accordingly, our study shows that the nestling period of parasitized young was prolonged by 3 days in comparison to deparasitized ones, which allowed parasitized offspring to fledge with a similar body mass and wing length. Recaptures of eight deparasitized nestlings and four parasitized ones in the subsequent year indicates that their growth was probably completed at fledging, as there was no significant

Table 1. Effects of experimental manipulations of ectoparasite load on fledging age, wing length, body mass and condition

	Parasitized broods			Deparasitized broods			Student's *t*-tests	
Variable	Mean	SE	N	Mean	SE	N	*t*-value	*P*-value
Fledging age (day)	61·3	0·9	27	58·1	0·8	36	2·66	0·009*
Fledging wing length (mm)	220·2	0·8	27	221·5	0·7	36	−1·22	0·23
Fledging body mass (g)	90·2	1·5	27	91·3	1·3	36	−0·54	0·59
Fledging condition (g mm^{-1})	−0·7	1·5	27	0·1	1·3	36	−0·37	0·71

*Also significant after Bonferroni correction for multiple testing.

difference in wing size between fledging and adult stage at return (repeated-measures ANOVA with treatment as factor, and wing length at fledging and adult stage as repeats; treatment as factor: $F_{1,10} = 0{\cdot}27$, $P = 0{\cdot}62$; change in wing size: $F_{1,10} = 2{\cdot}89$, $P = 0{\cdot}12$; interaction: $F_{1,10} = 0{\cdot}74$, $P = 0{\cdot}41$). The fact that, 50 days after hatching, wing length was still shorter in parasitized than deparasitized nestlings is consistent with the idea that parasitized young delayed fledging to grow for 3 supplementary days in order to fledge with a similar final body size to deparasitized ones.

COSTS AND EVOLUTION OF DEVELOPMENTAL PLASTICITY

Adaptive compensatory growth and delayed fledging may carry significant costs for nestlings and their parents, although such costs are as yet poorly documented, and also supposed to occur later in life (Metcalfe & Monaghan 2001). For instance, in parasitized offspring fast growth of flight feathers might reduce the quality of their wings (Dawson *et al*. 2000), and profoundly penalize juveniles on the wing and during long-distant migration. Compensatory growth and delayed maturation in altricial nestlings may also require extra work from their parents, which in turn may impair the parental reproductive success in following years (Richner & Tripet 1999). However, developmental plasticity brings quick benefits by allowing young facing poor growing conditions to recover in size. Hence, developmental plasticity is expected to arise because benefits brought early in life have a stronger impact on the lifetime reproductive fitness of individuals than costs incurred late in life.

PERSPECTIVES

Our study reveals two possible pitfalls associated with studies of host–parasite relationships. First, the lack of effects of parasites on wing length, body mass and condition in Alpine swifts at fledging, despite a significant effect of parasites earlier in their development, points out that the effects of parasites on some host features can be transient. This stresses the importance of investigating the consequences of parasite manipulations from birth to independence. Effects of parasite manipulations on nestlings reported in birds exhibiting post-fledging parental care may therefore overestimate the real impact of parasites on the final size reached by their hosts or on reproductive success. Secondly, because hosts can adjust growth rate to the level and timing of infestation, effects of parasites on host development can vary greatly over the rearing period alongside variation in the level of parasitism. Therefore, future studies on host–parasite relationships should consider parasite fluctuations and assess the effects of parasites at different stages in host development. Finally, the long-term effects of early exposure to parasites on host reproductive success should also be addressed.

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