Physiological Ecology of Mediterranean Blue Tits (*Parus caeruleus* L.): Effects of Ectoparasites (*Protocalliphora* spp.) and Food Abundance on Metabolic Capacity of Nestlings

Aurélie Simon[1,2,*]
Don Thomas[1,†]
Jacques Blondel[2,‡]
Philippe Perret[2,§]
Marcel M. Lambrechts[2,‖]
[1]Groupe de Recherche en Écologie, Nutrition, et Énergétique, Centre de Recherche en Biologie Forestière, Université de Sherbrooke, Sherbrooke, Quebec J1K 2R1, Canada; [2]Centre d'Écologie Fonctionnelle et Évolutive, Centre National de la Recherche Scientifique, 1919 route de Mende, 34293 Montpellier Cedex 5, France

Accepted 10/21/03

ABSTRACT

The consequences of nest ectoparasites, such as *Protocalliphora* larvae, on nestling birds have been the subject of numerous studies. Despite observed reductions in mass and hematocrit of chicks from parasitized nests, no studies have found any effect of *Protocalliphora* on nestling survival, suggesting that fitness consequences of *Protocalliphora* are either weak or occur after fledging. From experiments on the metabolic performance of chicks, we found that parasitized chicks suffer from reduced thermogenic and metabolic capacities as a result of decreased mass and hematocrit. Hence, *Protocalliphora* may potentially affect nestling survival after fledging, when energetically costly activities such as flight and moult are undertaken. Previous studies have demonstrated an increase in parental feeding rate to compensate for the detrimental consequences of parasite infestation. We tested whether parasite effects on nestling aerobic capacity were dependent on food availability during the feeding period. Measures of caterpillar densities and experimental manipulations of parasite loads allowed us to investigate relationships among host, parasite, and environment. A positive relationship between chick aerobic and thermogenic performances and caterpillar density suggests that negative effects of parasitism may be offset by increased food availability. This study provides the first measurement of the effects of an ectoparasite on metabolic competence in wild birds and documentation of the effect of food availability on ectoparasite virulence using a quantitative measure of food abundance.

* Corresponding author; e-mail: aurelie@hermes.usherb.ca.
† E-mail: donald.thomas@usherbrooke.ca.
‡ E-mail: blondel@cefe.cnrs-mop.fr.
§ E-mail: perret@cefe.cnrs-mop.fr.
‖ E-mail: lambrechts@cefe.cnrs-mop.fr.

Physiological and Biochemical Zoology 77(3):492–501. 2004. 1522-2152/2004/7703-2173$15.00

Introduction

Although the ecological and evolutionary aspects of host-ectoparasite interactions have received much attention in the past decade (Lehmann 1993; Brown et al. 1995; Clayton and Moore 1997; Møller 1997; Heeb et al. 1998, 1999; Richner 1998) and parasites should always have detrimental effects on their hosts (Price 1975; Anderson and May 1978, 1979; Clayton 1990; Loye and Zuk 1991; Hochberg et al. 1992; Clayton and Moore 1997), it remains ambiguous whether host-ectoparasite interactions evolve in a tightly coupled arms race. In the case of virulent pathogens, the evolution of both resistance in the host and virulence in the parasite does not raise any serious conceptual problems. The survival or reproductive consequences of infection select for genotypes and offer enhanced resistance in the host, increase fitness, and allow these genotypes to propagate. Host resistance and death both select for genetic lines in the pathogen that offer the optimal level of virulence and allow these pathogen lines to propagate without decimating the host populations. This relationship is typically referred to as a coevolutionary arms race. The principal conceptual problem posed by ectoparasites is that their effects on host fitness are subtle and may differ depending on whether young or adults are considered. Most bird ectoparasites have relatively clear effects on chick development (e.g., body mass and hematocrit; Table 1). However, these effects are difficult to link to fitness costs: only eight of 20 experimental and correlative studies have shown any effect on survival to fledging (Table 1). This weak effect of ectoparasites on nestling survival and hence on the fitness of the host is particularly impressive in the case of blood-sucking larvae of blowflies, *Protocalliphora* spp. These nest ectoparasites develop impressive biomasses occasionally amounting to 10% of chicks' biomass, yet they do not have any clear effect on nestling survival. However, the nest environment of-

Table 1: Reported effects of ectoparasites on development and survival of nestling birds

Parasite	Host	Mass	Hematocrit	Survival	Study Design	Source
Androlaelaps casalis	*Troglodytes aedon*	NA	NA	0	Experimental	Pacejka et al. 1998
Argas cooleyi	*Hirundo pyrrhonota*	−	−	−	Experimental	Chapman and George 1991
Ceratophyllus gallinae	*Parus major*	−	−	−	Experimental	Richner et al. 1993
Crataerina pallida	*Apus apus*	NA	NA	0	Experimental	Tompkins and Begon 1996
Dermanyssus rognephillus	*Progne subis*	NA	NA	−	Experimental	Moss and Camin 1970
Dermanyssus hirundinis	*A. apus*	NA	NA	0	Experimental	Tompkins and Begon 1996
D. hirundinis	*T. aedon*	NA	NA	0	Experimental	Pacejka et al. 1998
Dermanyssus hirundo	*T. aedon*	NA	0	0	Experimental	Johnson and Albrecht 1993
Ixodes lividus	*Riparia riparia*	NA	NA	−	Experimental	Szep and Møller 2000
Oeciacus hirundinis	*Delichon urbica*	NA	NA	−	Experimental	de Lope et al. 1998
Oeciacus vicarius	*H. pyrrhonota*	−	NA	NA	Experimental	Brown and Brown 1986
Ornithonyssus sylvarium	*Sturnus vulgaris*	NA	NA	−	Experimental	Fauth et al. 1991
Ornithonyssus bursa	*Hirundo rustica*	−	NA	−	Experimental	Møller 1990
Protocalliphora. azurea	*Parus caeruleus*	−	−	0	Experimental	Hurtrez-Boussès et al. 1997
Protocalliphora parorum	*T. aedon*	NA	0	0	Experimental	Johnson and Albrecht 1993
Protocalliphora silia	*Tachycineta bicolor*	0	NA	0	Experimental	Rogers et al. 1991
Protocalliphora spp.	*Parus gambeli*	NA	NA	0	Correlative	Gold and Dahlsten 1983
Protocalliphora spp.	*Parus rufescens*	NA	NA	0	Correlative	Gold and Dahlsten 1983
Protocalliphora asiovora	*R. riparia*	NA	−	0	Correlative	Whitworth and Bennet 1992
Protocalliphora chrysorrhoea	*Pica pica*	NA	−	0	Correlative	Whitworth and Bennet 1992
Protocalliphora hirundo	*R. riparia*	NA	NA	−	Correlative	Shields and Crook 1987

Note. Minus sign = negative effect; 0 = no effect; NA = no available data.

fers a high buffering capacity and may not challenge chicks sufficiently to expose severe fitness costs before fledging. One way to demonstrate fitness costs of parasitism that has not been used so far should be to investigate the metabolic performance of nestlings in relation to parasite infestation. Metabolic performance can be measured as the ability to mobilize and invest energy in costly activities such as flight or as the thermogenic capacity and the ability to thermoregulate independently at fledging (Swanson et al. 1996). If this capacity is reduced under the effect of ectoparasite loads, then any costs would be expressed only at or after the fledging period, at a time when fledglings face a considerable energetic and thermoregulatory challenge (and after most studies are terminated). Because hematocrit level indicates the amount of red blood cells, hemoglobin levels, and hence oxygen transport capacity, a decrease in hematocrit should result in a reduced aerobic capacity in nestlings and should impair flight ability and endurance. Because body mass is closely correlated with developmental state and shivering thermogenic capacity in nestling birds (Ricklefs and Starck 1996), a reduction in body mass in parasitized nestlings may indicate decreased thermogenic capacity. Thus, the apparently slight direct phenotypic consequences of parasites on nestlings may potentially have great importance to fledglings and may ultimately affect their survival and future recruitment.

It has also been argued that the apparent variance of the effects of ectoparasites on growth or fitness of young birds and mammals may be due to the capacity of hosts to compensate for the presence of ectoparasites (Munger and Karasov 1989). Ectoparasites compete directly with their host for energy, nutrients, and possibly water, so compensation necessarily means that hosts are able to increase the flow of energy and nutrient into the system. For the host, the cost and consequences of compensation will depend on the quality of the environment. When food is abundant, parents may increase provisioning rates and allow their young to increase food intake sufficiently to offset the consequences of nutrients and energy drained by

ectoparasites (de Lope et al. 1993; Merino and Potti 1994; Dufva and Allander 1995). If this is true, then the effects of ectoparasites on host growth and fitness may be expressed only in years of poor food supply, with productive years hiding detrimental effects of parasites and resulting in a high between-year variance in parasite effects.

In this study, we address these questions in a population of blue tits (*Parus caeruleus*) living in a very constraining habitat because of consistently low food supply and exceptionally high parasite rates. More than 90% of nests are infested with up to approximately 100 larvae per nest; this represents the highest prevalence and infestation rates so far reported in Europe (Hurtrez-Boussès et al. 1997). The birds are parasitized by two species of blowflies (*Protocalliphora azurea* and *Protocalliphora falcozi*). Our aim was to quantify the effects of parasites and the potential interaction between parasite load and food abundance on growth and metabolic capacity of nestlings just before fledging. We tested the following two principal hypotheses: (1) *Protocalliphora* ectoparasites retard not only growth but also the development of metabolic competence of nestling blue tits just before fledging and (2) interannual variation in prey abundance can offset the detrimental effects of *Protocalliphora* infestation. From these hypotheses, we predict that (1) heavily parasitized chicks will have a lower maximal aerobic capacity ($\dot{V}o_{2\ max}$) than unparasitized chicks and (2) the differences between the two categories will disappear in years of abundant food supply.

Material and Methods

Larval blowflies of the genus *Protocalliphora* are nest parasites found in a wide range of bird species across North America and Europe (Bennett and Whitworth 1991). Adult flies lay eggs in the nest material around the time of chick hatching. *Protocalliphora* larvae hatch and develop for approximately 15 d in the nest material and move up to periodically draw blood meals from the nestlings. Larvae attain a mass of approximately 200 mg before pupation. What makes this study model interesting is that parasites attack only the chicks in the nest, which means that fledglings and adults are completely free of them. This implies that the condition of chicks cannot be biased as a result of infestation of their parents. Our study population is located in a Holm oak (*Quercus ilex*) forest in the Fango Valley, Corsica, where a long-term program on population biology of tits has been in progress for 25 yr (see Blondel 1985; Blondel et al. 1987 for a site description). In Corsica, both *Protocalliphora azurea* and *Protocalliphora falcozi* are present, but because the larvae are indistinguishable in the field, we combined the two species and simply referred to them as *Protocalliphora.*

In this population, nestlings attain an asymptotic mass of 9.5–11.0 g by the age of 13 d. The variation in the asymptote of body size is explained by interannual variation in caterpillar abundance (Dias and Blondel 1996) but also by the presence and number of *Protocalliphora* larvae in the nest (Hurtrez-Boussès et al. 1997). In our population, infestation by *Protocalliphora* reduces nestling mass, tarsus length, and hematocrit level but has no significant effect on the survival of nestlings to fledging, which occurs at 17–21 d of age (Hurtrez-Boussès et al. 1997).

We conducted this study during the breeding seasons of 1999, 2000, and 2001 in this habitat where about 70 breeding pairs of blue tits occupy artificial nest boxes (Blondel 1985; Blondel et al. 1987). Each year, we determined the dates of nest and clutch initiation and hatching by daily routine inspection of nest boxes. Although hatching can be slightly asynchronous, we assigned all chicks in a brood to the same age on the basis of the first hatched chick.

When nestlings were 3 d old, nests were randomly assigned to two groups. Parasitized nests were left to be infested by natural levels of *Protocalliphora* larvae. To obtain unparasitized nests, we installed an elastic nylon fabric in the nest cup. Previous tests showed that this barrier blocked access by *Protocalliphora* to the nestlings and eliminated parasites without the use of insecticides. Parasite load represents the total of second and third stages of larval development (Gold and Dahlsten 1983), when chicks are 15 d old and stop growing. Hurtrez-Boussès et al. (1997) found that parasite effects on chicks are maximal at this age. We measured caterpillar abundance by collecting caterpillar frass dropping from the canopy of oaks in four 0.25-m^2 collectors at 3-d intervals at five stations scattered throughout the study site (see Zandt et al. 1990 for methods). These data were extrapolated to construct the curve of caterpillar abundance through the breeding season. This curve provided a measure of caterpillar abundance for each nest when chicks were 15 d old.

We ringed all the chicks on day 5, and at 15 d of age, we measured body mass (± 0.1 g) and hematocrit level by collecting a 20-μL blood sample from the brachial vein, centrifuged within 30 min (3 min at 13,000 rpm). For analyses of metabolic competence, we created a relative condition index, which we defined as the product of chick's mass and its hematocrit level.

We experimentally measured the thermogenic response of the nestlings to a cold challenge by using the sliding method of cold exposure (Swanson et al. 1996). One to two hours after the last feeding bout on day 14 (at about 2130 hours), two chicks were randomly selected from a nest and brought to a nearby laboratory. They were weighed and placed in a dark-walled 0.5-L metabolic chamber, which was then placed in a controlled-temperature refrigerator. Dry CO_2-free air (ascarite and drierite) was pumped through each chamber at a rate of 250 mL/min, and a 100 mL/min subsample was directed first through an ascarite/drierite scrubber and then through an oxygen analyzer (Sable Systems FC-1, Henderson, Nev.) for each chamber. A computerized data acquisition system (Sable Systems Datacan V) controlled valves to first calibrate the oxygen

analyzers at the start and end of each trial with fresh scrubbed air (20.95% O_2) before reading and storing O_2 concentration in the chamber outflow at 5-s intervals. The computer also regulated the refrigerator temperature according to a programmed pattern and held it at 35°C for 30 min to allow chicks to acclimate before allowing the refrigerator to cool down to approximately 5°C over the following 60 min. During the cooling period, we monitored O_2 consumption and body temperature (T_b) using temperature-sensitive radio transmitters, and we stopped trials before T_b fell below 30°C, which approximates the lower bound of T_b found in nests. After the experiment, chicks were warmed if necessary and put back into their nest.

The resulting O_2 consumption curves were transformed to $\dot{V}O_2$ (mL/g/h) using Equation (4a) from Withers (1977). Chicks were scored as metabolically competent or incompetent depending on their response to this cold challenge. Metabolically competent chicks continued to increase $\dot{V}O_2$ until the trial was completed, and they maintained body temperature at ≥35°C. Metabolically incompetent chicks showed a clear peak in $\dot{V}O_2$ followed by a decline (Fig. 1). Chick aerobic capacity, which we term "metabolic competence," was thus scored as a binary variable because competent chicks did not show their true maximum aerobic capacity under the limited cold challenge that we used.

Statistical analyses were performed with S-Plus 2000 (MathSoft). Data are presented as mean ± SD unless otherwise indicated. Sample sizes in tests of metabolic performance were limited to the number of chicks for which we had body mass, hematocrit levels, measures of metabolic performance, and parasite load. Normality of data and residuals have been tested using the Kolmogorov-Smirnov test. Using each chick as an independent value could be considered to represent pseudoreplication because each nest generally contributed two chicks to the sample size. However, because we were interested in individual variation and because metabolic competence was scored as a binary variable, we could not use the mean values for nests. To circumvent this problem, we used mixed models and included nest of rearing as a random factor, thus separating nest and individual effects. To account for noncontrolled year effects, we added year as a factor in all statistical tests. Because the results never identify year as a significant variable, we removed it and thus restricted our analyses to the continuous variables, prey abundance, and parasite load. This allowed us to pool data for a more robust analysis.

Results

Over the 3-yr study, mean ectoparasite loads varied from 12.7 ± 3.9 larvae/chick in 1999 to 18.3 ± 7.3 larvae/chick in 2000, with 2001 being intermediate in parasite load (13.6 ± 6.2 larvae/chick). Over the same period, caterpillar abundance also varied from 53.5 ± 22.0 mg frass/m^2 in 1999 to 200.6 ± 64.7 mg frass/m^2 in 2000, again with 2001 being intermediate

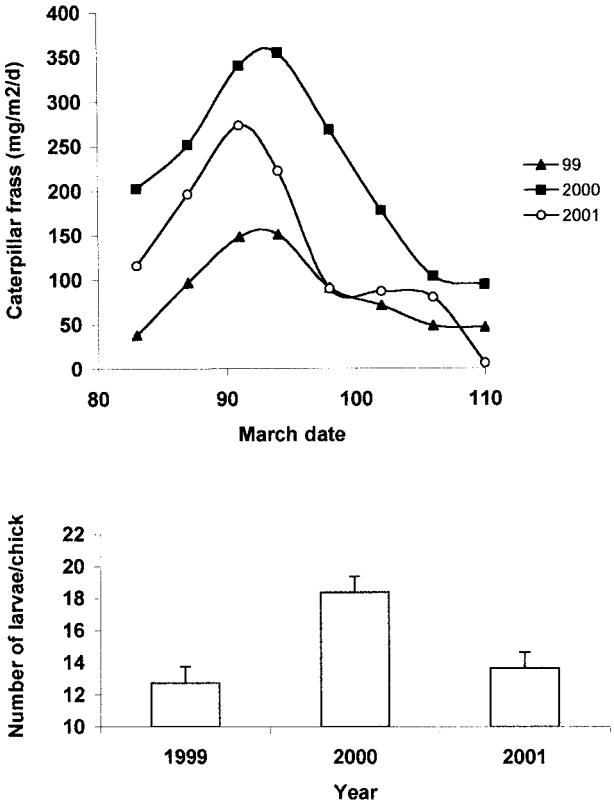

Figure 1. Seasonal variation in caterpillar abundance measured by frass fall in 1999, 2000, and 2001 (*top*). Dates are "March date," that is, 32 = April 1. Between-year variation in parasite abundance in untreated blue tits nests (*bottom*).

(69.8 ± 31.6 mg frass/m^2). We thus refer to 1999 as a poor year and 2000 as a rich year in terms of food abundance. The lowest caterpillar abundance for any study nest in 2000 (103 mg frass/d/m^2, estimated from the frass fall value when nestlings of the nest were 15 d old) was greater than the highest abundance for any nest in 1999 (91 mg frass/d/m^2), and in 2000, nests had approximately 1.5 times higher parasite loads than in 1999. Because 1999 and 2000 represent the two extremes in food abundance and parasite loads, we first contrast the results obtained in those years and thereafter pool the 3 yr of data for a more robust analysis (Fig. 1).

Interannual Variation of Parasite Load and Caterpillar Abundance

Because they were randomly selected among active nests each year, parasitized and unparasitized nests had similar caterpillar abundance in 1999 (parasitized, 55.3 ± 24.8 mg frass/d/m^2; unparasitized, 51.4 ± 19.1 mg frass/d/m^2; $F_{1,35} = 0.28$, $P = 0.60$) and in 2000 (parasitized, 211.5 ± 75 mg frass/d/m^2; unparasitized, 176.2 ± 24.5 mg frass/d/m^2; $F_{1,11} = 0.28$, $P = 0.40$).

Body Mass and Hematocrit Levels in Poor and Rich Years

We checked for between-year variability in body mass and hematocrit levels for each treatment group. Despite contrasted environmental conditions, we found that body mass did not differ between years. Only hematocrit from unparasitized nests differed significantly between 1999 and 2000 (Table 2).

The four times higher caterpillar abundance in 2000 compared with 1999 masked any effect of food supply on growth patterns of chicks in 2000. Chick mass was significantly affected by caterpillar abundance in 1999 (positive effect, $P < 0.05$), but this effect disappears ($P > 0.50$) in 2000 (Table 3). Ectoparasite load also affected chick mass differently depending on year. In the poor year (1999), chick mass was significantly depressed by ectoparasite load ($P < 0.05$), but in the rich year of 2000, this effect disappeared ($P > 0.10$; Table 3) despite the fact that ectoparasite load was nearly twice as high. In both 1999 and 2000, increasing ectoparasite load decreased hematocrit level (Table 3). Although high caterpillar abundance counteracts the negative effect of ectoparasites on chick mass, this was not the case for hematocrit; caterpillar abundance had no effect on hematocrit level in either year ($P > 0.05$ in both years).

In a multiple regression analysis for which data for 1999, 2000, and 2001 were pooled to increase the range of ectoparasite loads and caterpillar abundance, chick mass was negatively affected by ectoparasite load ($P < 0.01$) but positively by caterpillar abundance ($P < 0.01$; Table 3). Hematocrit level was negatively affected only by parasite load ($P < 0.01$).

Metabolic Performance

Resting metabolic rate (RMR) for chicks, estimated by $\dot{V}O_2$ at 35°C (thermoneutral zone), did not significantly differ between parasitized and unparasitized nestlings at 14 d (parasitized, 4.08 ± 0.25 mL O_2/g/h; unparasitized, 4.12 ± 0.17 mL O_2/g/h; ANOVA, $F_{1,54} = 0.18$, $P > 0.05$). Our procedures did not allow us to determine the true peak or summit metabolism of metabolically competent chicks. However, our measurements showed that the chicks we classed as metabolically incompetent were able to boost $\dot{V}O_2$ to only approximately two times resting levels. It is important to remember that we evaluated metabolic competence by the presence or absence of a peak ($\dot{V}O_{2\,max}$) in the metabolic rate for chicks facing a cold challenge (Fig. 2). Metabolic competence is thus a binary variable. We therefore used a logistic regression to evaluate metabolic competence as a function of ecological (parasite load, caterpillar abundance) and physiological variables (body mass, hematocrit level, and condition index).

The interannual variation of ectoparasite loads and caterpillar abundance had a complex interaction that affected physiological parameters such as hematocrit level and body mass and determined the expression of metabolic competence. In the poor 1999 year, only 12% of parasitized individuals tested were classed as metabolically competent. Parasite load had a strong negative effect ($P < 0.01$) and hematocrit a strong positive effect ($P = 0.01$) on metabolic competence, but caterpillar abundance and chick mass do not significantly affect competence ($P > 0.10$; Table 4). In contrast, despite the fact that neither hematocrit level nor body mass was significantly higher in 2000 compared with 1999, parasitized as well as unparasitized chicks were all metabolically competent in the rich year of 2000. This means that in 2000, metabolic competence was unaffected by either ecological factors (parasite load and caterpillar abundance) or physiological parameters (body mass or hematocrit level).

In the pooled data set, including 1999, 2000, and 2001, parasite load had a negative effect and caterpillar abundance a positive effect on metabolic competence (Table 4). However, in this combined sample, hematocrit had a slightly weaker effect on metabolic competence than in 1999 (Table 4). To test for an interaction between mass and hematocrit, we combined them by creating a relative condition index (see "Material and Methods" section). In 1999, condition index had a weaker effect on metabolic competence than hematocrit (Table 4). In the pooled sample, the condition index appeared to be a stronger determinant of metabolic competence than hematocrit level (Table 4; *P* value and likelihood ratio statistic).

Discussion

This study provides a first measurement of the effects of an ectoparasite on metabolic competence in wild birds and doc-

Table 2: Mass and hematocrit level of nestling blue tits as a function of parasite load and year

	1999	2000	U^a	*P*
Mass (g):				
Parasitized	9.5 ± 1.0 ($N = 18$)	10.3 ± .3 ($N = 9$)	46	.07
Unparasitized	10.0 ± .6 ($N = 16$)	10.4 ± .5 ($N = 4$)	22.0	.34
Hematocrit (%):				
Parasitized	34.3 ± 6.5 ($N = 15$)	32.8 ± 5.6 ($N = 9$)	79.5	.47
Unparasitized	46.3 ± 2.8 ($N = 15$)	53.5 ± 5.5 ($N = 4$)	8.0	.03

[a] Mann-Whitney statistic for across-year contrasts.

Table 3: Effects of parasite load and caterpillar availability on mass and hematocrit level, using a linear mixed model

Year and Dependent and Independent Variables	df	Slope	t	F	P
1999:					
Mass ($N = 34$):					
Parasite load	15	−.06	−2.55	6.09	.03
Caterpillar abundance	15	.25	2.37	5.56	.03
Hematocrit ($N = 30$):					
Parasite load	11	−.81	−5.49	30.20	<.01
Caterpillar abundance	11	−.57	−.90	.82	.39
2000:					
Mass ($N = 13$):					
Parasite load	7	−.01	−.84	.64	.44
Caterpillar abundance	7	.02	.33	.11	.75
Hematocrit ($N = 13$):					
Parasite load	7	−1.18	−6.79	43.53	<.01
Caterpillar abundance	7	1.98	2.57	6.61	.07
Pooled data (1999–2001):					
Mass ($N = 62$):					
Parasite load:	31	−.05	−4.46	10.31	<.01
Caterpillar abundance	31	.13	4.50	20.28	<.01
Hematocrit ($N = 59$):					
Parasite load	28	−.80	−9.22	82.75	<.01
Caterpillar abundance	28	.34	1.57	2.48	.13

Note. See "Material and Methods" section. Nonsignificant interactions were removed from the model.

umentation of the apparent effect of food availability on ectoparasite virulence by using a quantitative measure of food abundance. Although it could be argued that our results are correlational and that other unmeasured year effects caused changes in metabolic capacity, we underline that year was not significant in our global regression analyses.

The story that emerges from our data is the following. Ectoparasite load depresses both circulating red blood cells (hematocrit) and asymptotic body mass of chicks, with this effect being expressed only during the nestling stage before fledging. High food abundance can completely mask the negative impact of ectoparasites on growth and asymptotic body mass and can explain the between-year variation in ectoparasite effect on mass. Low hematocrit levels result in a diminished ability to mobilize energy for thermoregulation. This effect can be masked in years of high food availability through a relatively subtle effect of food abundance on asymptotic body mass. Relatively minor (and statistically undetectable) interannual differences in body mass do not directly affect metabolic capacity but rather interact with hematocrit (condition index) to determine thermogenic capacity (Table 4). Although we cannot definitively establish a causal relationship between these variables, it is likely that ecological factors (ectoparasite load and food availability) directly affect physiological variables (mass and hematocrit), which in turn determine metabolic capacity.

The pathway by which ectoparasite *Protocalliphora* larvae affect metabolic capacity appears to include both hematocrit and, to a lesser degree, body mass. Hematocrit level provides a quantitative measure of the volume of circulating red blood cells and represents both the oxygen-carrying capacity of the blood and its ability to deliver oxygen to the tissues and cells for fuel oxidative metabolism at the mitochondrial level. Hematocrit level fell from 46.3% in unparasitized nestlings to 34.3% in parasitized nestlings in 1999 and from 53.5% to 32.8% for unparasitized and parasitized nestlings in 2000 (Table 2). This represents a loss of 29% (1999) and 39% (2000) in oxygen-carrying capacity due to the effects of ectoparasites. One would expect the major reduction in thermogenic capacity found in

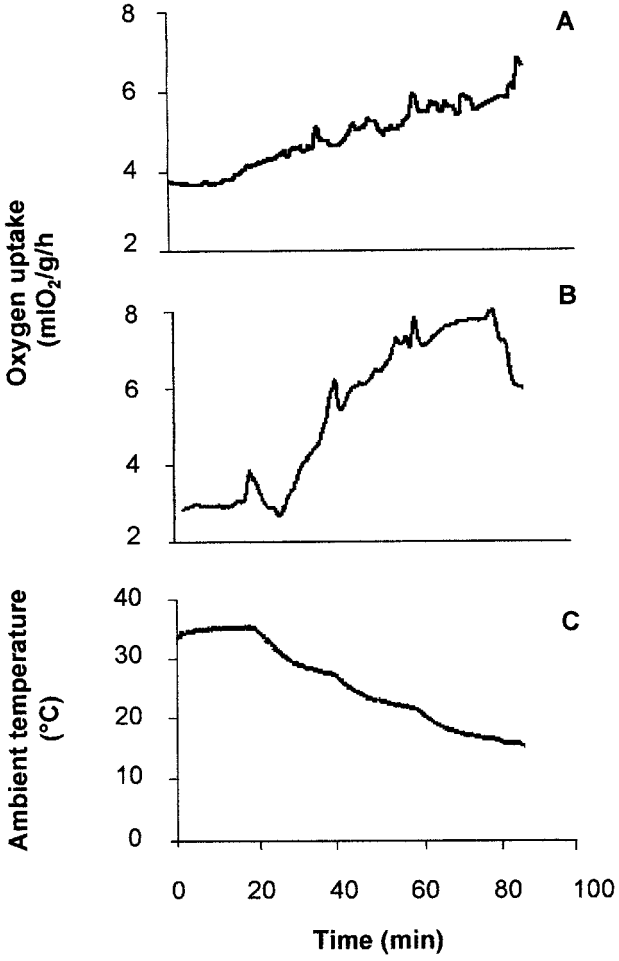

Figure 2. Changes in oxygen uptake (*A*, *B*) and ambient temperature (*C*) over time for unparasitized (*A*) and parasitized (*B*) nestling blue tits exposed to a cold challenge. A continuous increase in oxygen uptake characterizes a metabolically competent chick (*A*), whereas a peak in oxygen consumption characterizes a metabolically incompetent chick (*B*).

Table 4: Effects of environmental variables (parasite load and caterpillar abundance) and physiological variables (body mass, hematocrit, and condition index) on metabolic competence

Year and Parameter	N	b	P Value for Parameter	Likehood Ratio Statistic	P Value for Model (χ^2)
1999:					
Caterpillar abundance	30	−.27	.38	14.79	<.01
Parasite load	30	−.40	<.01	14.79	<.01
Hematocrit level	26	.21	.01	10.0	<.01
Mass	26	.39	.46	.58	.44
Condition index	26	.02	.02	9.13	<.01
Pooled data:					
Caterpillar abundance	56	.53	<.01	21.22	<.01
Parasite load	56	−.15	<.01	21.22	<.01
Hematocrit level	55	.07	.04	4.59	.03
Mass	55	.89	.08	6.30	.02
Condition index	55	.01	.01	7.04	<.001

Note. Data in 2000 are not presented because no chicks were metabolically incompetent.

metabolically incompetent chicks to be also associated with a proportional reduction in body mass. Indeed, in birds, heat production depends on oxidative shivering thermogenesis, which is primarily based in the pectoralis (flight) muscle because of its size. In altricial birds, the acquisition of a strong thermogenic capacity and thermoregulatory competence (or independence) is tightly correlated with both the size of the pectoralis muscle and its developmental state, which appears to implicate mitochondrial and cellular maturation (Ricklefs and Starck 1996). The fact that we did not statistically detect any direct effect of mass on metabolic capacity suggests that any size-related effects should be slight. However, because we found metabolic performance to be affected by food availability (Table 4) and because caterpillar abundance only offsets parasite effect on mass but not on hematocrit (Table 3), we suspected that mass and hematocrit interacted to determine metabolic capacity, with slight but statistically insignificant changes in mass having an important effect when combined with hematocrit. Our results show that the metabolic capacity of chicks does not result from either body mass or hematocrit separately but from their combination, as suggested by the condition index. Chicks that had low hematocrit combined with high mass were more likely to be metabolically competent than chicks having the same hematocrit level but low mass. We suppose that the pathway by which hematocrit and chick mass combine to set a chick's metabolic capacity probably involves a bottleneck on the transport of oxygen to the muscle tissues and mitochondria themselves.

By compensating for the effects of parasite load on body mass, high caterpillar abundance increased the condition index and enhanced metabolic competence. The compensatory effect of caterpillar abundance likely reflects changes in parental provisioning rather than adjustments by the chicks themselves, as indicated by the much higher feeding rates in parasitized nests (Richner et al. 1993; Hurtrez-Boussès et al. 1998; Bouslama et al. 2002). It is interesting that in rich years, increasing feeding rate appears to totally offset the effect of ectoparasites on mass but not on hematocrit values. It indicates that even under good food conditions, the direct costs of parasites are shared between the nestlings that support the hematocrit decrease and the parents that compensate for chick loss in mass by increasing their provisioning rates. The increase in parental provisioning in infested nests (see Hurtrez-Boussès et al. 1998) reveals the possibility for the parents to work much more than they would if their broods were not parasitized, an excess capacity almost certainly enhanced in years of high food abundance. From a mean abundance of 56.7 ± 53.0 mg frass/d/m^2 for the years 1987–1999, caterpillar abundance rose to the high value of 212.1 mg/d/m^2 in 2000. We argue that in 2000, the ease with which parents could provision nestlings alleviated the detrimental effects of blowflies and explained why chicks could cope with ectoparasites (when caterpillar abundance increased fourfold) without any decrease in asymptotic mass or metabolic competence.

The capacity of food abundance to offset the detrimental impact of ectoparasite virulence likely explains the high variation in virulence and fitness costs reported for ectoparasites (Whitworth and Bennett 1992; Johnson and Albrecht 1993) and underlined in Table 1. Dufva and Allander (1995) had reported that parasite virulence was higher in years when weather conditions were bad in early spring, and de Lope et al. (1993) had found that parasites had a greater impact on

chick quality and survival in second rather than first broods. Both these effects can be explained by the decrease in prey abundance (or access to prey) under bad weather or as the season progresses. Although this pattern has been speculated and these mechanisms need to be confirmed, we believe that our study is one of the first to establish a link between ectoparasite virulence and a quantitative measure of prey abundance.

Although we tested only a response to a cold challenge, maximum thermogenic capacity is strongly correlated with maximum exercise-induced metabolic rate. For this reason, we believe that chicks showing a reduced metabolic capacity in the face of a cold challenge would also have limited aerobic capacity under any other kind of energetic challenge. Normally, the high buffering capacity of the nest environment would not be expected to challenge chicks sufficiently to allow them to express their reduced metabolic capacity. By huddling in a shared nest, brood members benefit from a reduction in the surface area to volume ratio and reduce the individual cost (Hill and Beaver 1982). As long as parents continue to provision them, chicks never face any substantial locomotory costs, although in highly parasitized nests, chicks do move and scratch themselves in an apparent attempt to escape parasites, which could increase metabolic costs (Simon et al. submitted). However, at fledging, which occurs only about 4 d after our measures, chicks must abandon the buffered nest environment and suddenly confront the aerobic challenges of solitary thermoregulation (often >2 × BMR) and flight (>10 × BMR). This is the paradox posed by these nest ectoparasites: nestlings will experience the fitness consequences only after they have escaped ectoparasite attacks. Weak metabolic and aerobic capacity may partly explain why nestlings are vulnerable to predator attacks (especially by jays, *Garrulus glandarius* L., in our study population) and suffer high mortality in the first days after fledging (Naef-Daenzer et al. 2001).

The importance of metabolic capacity on physiological performance and fitness components has only recently received attention; physiological performance is now recognized as a reliable measure of potential fitness (Arnold 1983). In anurans (Goater and Ward 1993) and lizards (Bennet 1980; Schall 1982; Garland et al. 1990), variation in dispersal, social dominance, and predator avoidance has been shown to be directly linked with physiological performance. We argue that physiological performance must also be considered in the context of parasite burdens, food availability, and body condition. The interaction between ectoparasites and environmental quality (e.g., food abundance) complicates the analysis of the fitness (e.g., survival) consequences associated with parasite infestation. Because 2000 was an exceptionally rich year, with prey abundance exceeding any of the previous 14 yr, it allowed us to detect the interaction between food abundance and parasite loads. However, it is likely that normal years of far lower prey abundance will result in the expression of low metabolic capacity among a larger proportion of chicks than was found in this study. It may well be that *Protocalliphora* represents a significant selective pressure on blue tit reproductive and physiological traits.

Acknowledgments

We thank Claude Poulin-Gingras, Daniel Munro for running many of the tedious metabolic trials, and Marie Maistre for caterpillar counts. This study was supported by grants from the Centre National de la Recherche Scientifique (France) to J.B., from Natural Sciences and Engineering Research Council grants to D.T., and grant EKV2-ct-1999-0017 from the European Commission to M.M.L. A.S. was supported by a Canadian government international scholarship and by funding from the Programme de Coopération France-Québec. This is publication 155 of the Groupe de Recherche en Écologie, Nutrition et Énergétique, Département de Biologie, Université de Sherbrooke.

Literature Cited

Anderson R.M. and R.M. May. 1978. Regulation and stability of host-parasite population interactions. I. Regulatory processes. J Anim Ecol 47:219–247.

———. 1979. Population biology of infectious diseases. I. Nature 2800:361–367.

Arnold S.J. 1983. Morphology, performance and fitness. Am J Zool 23:347–361.

Bennet A.F. 1980. The thermal dependence of lizard behaviour. Anim Behav 28:752–762.

Bennett G.F. and T. Whitworth. 1991. Studies on life history of some species of *Protocalliphora*. Can J Zool 69:2048–2058.

Blondel J. 1985. Breeding strategies of the blue tits and coal tit (*Parus*) in mainland and island Mediterranean habitats: a comparison. J Anim Ecol 54:531–556.

Blondel J., A. Clamens, P. Cramm, H. Gaubert, and P. Isenman. 1987. Populations studies of tits in the Mediterranean regions. Ardea 75:21–34.

Bouslama Z., M.M. Lambrechts, N. Ziane, R. Djenidi, and Y. Chabi. 2002. The effects of nest ectoparasites on parental provisioning in a north-African population of the blue tit *Parus caeruleus*. Ibis 144:E73–E78.

Brown C.R. and M.B. Brown. 1986. Ectoparasitism as a cost of coloniality in cliff swallows. Ecology 67:1206–1218.

Brown C.R., M.B. Brown, and B. Rannala. 1995. Ectoparasites reduce long-term survival of their avian host. Proc R Soc Lond B Biol Sci 262:313–319.

Chapman B.R. and J.E. George. 1991. The effects of ectoparasites on cliff swallows growth and survival. Pp. 69–92 in J.E. Loye and M. Zuk, eds. Bird-Parasite Interactions: Ecology, Evolution and Behaviour. Oxford University Press, Oxford.

Clayton D.H. 1990. The influence of parasites on host sexual selection. Parasitol Today 7:329–334.

Clayton D.H. and J. Moore. 1997. Host-Parasite Evolution: General Principles and Avian Models. Oxford University Press, Oxford.

de Lope F., G. Gonzalez, J.J. Perez, and A.P. Møller. 1993. Increased detrimental effects of ectoparasites on their bird host during adverse environmental conditions. Oecologia 95:234–240.

de Lope F., A.P. Møller, and C. Delacruz. 1998. Parasitism, immune response and reproductive success in the house martin *Delichon urbica*. Oecologia 114:188–193.

Dias P. and J. Blondel. 1996. Breeding time, food supply and fitness components of blue tits *Parus caeruleus* in Mediterranean habitats. Ibis 138:644–649.

Dufva R. and K. Allander. 1995. Variable effects of the hen flea *Ceratophyllus gallinae* on the breeding success of the great tit *Parus major* in relation to weather conditions. Ibis 138: 772–777.

Fauth P.T., D.G. Krements, and J.E. Hines. 1991. Ectoparasitism and the role of green nesting material in the European starling. Oecologia 88:22–24.

Garland T., E. Hankins, and R.B. Huey. 1990. Locomotor capacity and social dominance in male lizards. Funct Ecol 4: 243–250.

Goater C.P. and P.J. Ward. 1993. Negative effect of *Rhabdias bufonis* (*Nematoda*) on the growth and survival of toads (*Bufo bufo*). Oecologia 89:161–165.

Gold C. and D.L. Dahlsten. 1983. Effects of parasitic flies (*Protocalliphora* spp.) on nestlings of mountain and chestnut-backed chickadees. Wilson Bull 95:560–572.

Heeb P., I. Werner, M. Kölliker, and H. Richner. 1998. Benefits of induced host responses against an ectoparasite. Proc R Soc Lond B Biol Sci 265:51–56.

Heeb P., I. Werner, A.C. Mateman, M. Kölliker, and W.G. Brinkhof. 1999. Ectoparasite infestation and sex-biased local recruitment of hosts. Nature 400:63–65.

Hill R.W. and D. Beaver. 1982. Inertial thermostability and thermoregulation in broods of redwing blackbirds. Physiol Zool 55:250–266.

Hochberg M., Y. Michalakis, and T. de Meeüs. 1992. Parasitism as a constraint on the rate of life-history evolution. J Evol Biol 5:491–504.

Hurtrez-Boussès S., J. Blondel, P. Perret, J. Fabreguettes, and F. Renaud. 1998. Chick parasitism by blowflies affects feeding rates in a Mediterranean population of blue tits. Ecol Lett 1:17–20.

Hurtrez-Boussès S., J. Blondel, P. Perret, and F. Renaud. 1997. High blowfly parasitic loads affect breeding success in a Mediterranean population of blue tits. Oecologia 112:514–517.

Johnson L.S. and D.J. Albrecht. 1993. Effects of haematophageous ectoparasites on nestling house wrens: who pays the cost of parasitism? Oikos 66:255–262.

Lehmann T. 1993. Ectoparasites: direct impact on host fitness. Parasitol Today 9:8–13.

Loye J.E. and M. Zuk. 1991. Bird-Parasite Interactions: Ecology, Evolution and Behaviour. Oxford University Press, Oxford.

Merino S. and J. Potti. 1994. Mites decrease growth and survival in nestling pied flycatchers. Oikos 73:95–103.

Møller A.P. 1990. Effects of parasitism by a haematophageous mite on reproduction in the barn swalllow. Ecology 71:2345–2357.

———. 1997. Parasitism and the evolution of host life history. Pp. 105–127 in D.H. Clayton and J. Moore, eds. Host-Parasite Evolution: General Principles and Avian Models. Oxford University Press, Oxford.

Moss W.W. and J.H. Camin. 1970. Nest parasitism, productivity and clutch size in purple martins. Science 168:1000–1002.

Munger J.C. and K.W. Karasov. 1989. Sublethal parasites and host energy budgets: tapeworm infection and white-footed mice. Ecology 70:904–921.

Naef-Daenzer B., F. Widmer, and M. Nuber. 2001. Differential post-fledging survival of great and coal tits in relation to their condition and fledging date. J Anim Ecol 70:730–738.

Pacejka A.J., C.M. Gratton, and C.F. Thompson. 1998. Do potentially virulent mites affects house wren (*Troglodytes aedon*) reproductive success? Ecology 79:1797–1806.

Price P.W. 1975. The parasitic way of life and its consequences. Pp. 1–13 in P.W. Price, ed. Evolutionary Strategies of Parasitic Insects and Mites. Plenum, New York.

Richner H. 1998. Host-parasite interactions and life-history evolution. Zoology 101:333–344.

Richner H., A. Oppliger, and P. Christe.1993. Effect of an ectoparasite on reproduction in great tits. J Anim Ecol 62:703–710.

Ricklefs R.E. and J.M. Starck. 1996. Avian Growth and Development: Evolution within the Altricial-Precocial Spectrum. Oxford University Press, Oxford.

Rogers C., R. Robertson, and B. Stutchbury. 1991. Patterns and effects of parasitism by *Protocalliphora sialia* on tree swallow nestlings. Pp. 123–139 in J.E. Loye, M. Zuk, eds. Bird-Parasite Interactions: Ecology, Evolution and Behaviour. Oxford University Press, Oxford.

Schall R.W. 1982. Lizards infected with malaria: physiological and behavioral consequences. Science 217:1057–1059.

Shields W.M. and J.R. Crook. 1987. Barn swallow coloniality: a net cost for group breeding in the Adirondacks? Ecology 68:1373–1386.

Swanson D.L., M.W. Drymalski, and J. Brown. 1996. Sliding versus static cold exposure and the measurement of summit metabolism in birds. J Therm Biol 21:221–226.

Szep T. and A.P. Møller. 2000. Exposure to ectoparasites increases within-brood variability in size and body mass in the sand martin. Oecologia 125:201–207.

Tompkins D.M. and M. Begon. 1996. Parasites can regulate wildlife populations. Parasitol Today 15:311–313.

Whitworth T. and G.F. Bennett. 1992. Pathogenecity of larval *Protocalliphora* parasitizing nestling birds. Can J Zool 70: 2184–2191.

Withers P.C. 1977. Measurements of $\dot{V}O_2$, $\dot{V}CO_2$ and evaporative water loss with a flow-through mask. J Appl Physiol Respir Environ Exercise Physiol 42:120–123.

Zandt H., A. Strijkstra, J. Blondel, and H. Van Balen. 1990. Food in two Mediterranean blue tit population: do differences in caterpillar availability explain differences in timing of the breeding season? Pp. 145–155 in J. Blondel, A. Gosler, J.D. Lebreton, and R. McCleery, eds. Population Biology of Passerine Birds. NATO ASI Series G, Vol. 24. Springer, Berlin.