Effects of the blood parasite *Leucocytozoon simondi* on growth rates of anatid ducklings

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Abstract: The blood parasite *Leucocytozoon simondi* is often associated with heavy mortality of ducks and geese, especially domestic ones, in North America. In contrast, in a previous study we found no mortality from *L. simondi* in our wild stock of mallard (*Anas platyrhynchos*) and American black duck (*Anas rubripes*) ducklings. However, because parasites can slow growth, which could extend the interval during which ducklings are susceptible to predators, we tested for parasite effects on growth rates. We analysed growth rates over the first 20 days of life, based on tarsus length, culmen, bill width, body mass, and a principal component of structural size. Growth rates of infected ducklings were not lower than those of uninfected ducklings. Similarly, more intense infections did not have a greater effect on growth rates. Hence, growth rates were not negatively affected by *L. simondi*, which suggests that effects of this parasite on wild duck populations have been overestimated.

Résumé : L'hématozoaire *Leucocytozoon simondi* est souvent associé à un taux de mortalité élevé des canards et des oies en Amérique du Nord, surtout chez les variétés domestiques. En revanche, au cours d'une étude antérieure, nous n'avons pas observé de mortalité causée par *L. simondi* chez nos stocks sauvages de canetons du Canard colvert (*Anas platyrhynchos*) et du Canard noir (*Anas rubripes*). Cependant, étant donné que les parasites peuvent ralentir la croissance, ce qui a pour effet d'étirer la période au cours de laquelle les canetons sont sujets à la prédation, nous avons examiné les effets des parasites sur les taux de croissance. Nous avons analysé les taux de croissance au cours des 20 premiers jours de la vie par mesure de la longueur du tarse, du culmen, de la largeur du bec, de la masse totale et d'une composante principale de la taille structurale. Les taux de croissance n'étaient pas plus faibles chez les canetons parasités que chez les canetons sains. De même, les infections graves n'avaient pas plus d'effets sur les taux de croissance que les infections bénignes. Donc, les taux de croissance ne sont pas affectés négativement par la présence de *L. simondi*, ce qui laisse à penser que les effets de ce parasite sur les populations de canards sauvages ont été surestimés dans le passé.

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Introduction

Leucocytozoon simondi is a blood parasite transmitted by black flies (Simuliidae) that can cause heavy mortality in domestic ducks and geese in eastern North America (Bennett et al. 1974; Atkinson and van Riper 1991; Desser and Bennett 1993). However, Shutler et al. (1996) found no mortality or signs of disease from *L. simondi* in a captive population of wild-strain mallard (*Anas platyrhynchos*) and American black duck (*Anas rubripes*; hereafter black duck) ducklings. Shutler et al. (1996) argued that previously reported high rates of mortality from *L. simondi* could be ascribed largely to low resistance in domestic versus wild genetic stocks (also see Bennett et al. 1993). However, parasites can have significant negative effects on host development or growth that result from increased allocation to immune function (such as fever), repair of tissues (such as blood) that have been damaged by parasites, or reduction in appetite (Seebeck et al. 1971; Batt 1980; Crompton 1991; Goater et al. 1993; Richner et al. 1993; Goater 1994; Spalding et al. 1994). Moreover, predators can preferentially take more heavily parasitised prey (e.g., Temple 1987; Hudson et al. 1992). Because ducklings are most vulnerable to predators when they are small, slow growth rates could indirectly translate into higher mortality rates. Because *L. simondi* destroys red blood cells directly and also causes healthy cells to rupture (Maley and Desser 1977), this could result in energy being diverted from growth to tissue repair and related metabolic processes. Hence, we tested whether *L. simondi* affected duckling growth.

Leucocytozoon simondi is specific to waterfowl and is endemic in much of North America (Bennett et al. 1974; Atkinson and van Riper 1991; Desser and Bennett 1993). *Leucocytozoon simondi* infections arise when infected black flies (Simuliidae) take blood meals, at which point asexual stages of the parasite can enter the bloods stream of duck hosts. Once inside the host, the parasites undergo a few asexual stages, each lasting a few days, in most organs, especially the liver. After a total of about 10 days, the parasites return to the blood, invading erythrocytes as gametocytes. Mature gametocytes are first present in the blood about 10 days post infection, and it is this time and the fol-

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Table 1. Numbers of ducklings, from a total of 115, allocated to treatments according to type.

	Duckling type[a]						
Treatment	SM × SM	SM × OM	SM × OB	OM × OM	OM × OB	OB × OB	Total
Control	2	5	2	14	2	5	30
Experimental	3	18	4	38	8	18	85

[a]SM, Saskatchewan mallard; OM, Ontario mallard; OB, Ontario black duck.

lowing few days that are associated with the most pathology, particularly in ducklings. Although numerous pathological signs of *L. simondi* have been described, such as anemia and splenomegaly (Bennett et al. 1974; Atkinson and van Riper 1991; Desser and Bennett 1993), the parasite's effects on growth rates have not been assessed. We predicted that metabolic costs of dealing with infection *by L. simondi* would result in slower growth of infected ducklings.

Methods

A stock of adult ducks was obtained by raising ducklings that were hatched from 9 different clutches in Saskatchewan, Canada, and from 16 different clutches collected in and around Algonquin Provincial Park, Ontario, Canada (ducks were designated as American black ducks from Ontario (OB), mallards from Ontario (OM), and mallards from Saskatchewan (SM)). Adults were maintained at Lake St. Clair, Ontario, an area that is probably free of vectors because suitable habitat is lacking (Herman 1978). Moreover, no *L. simondi* was detected in blood smears from individuals that were kept there (Shutler et al. 1996). Adults were bred (avoiding sib–sib pairings) in spring 1992 and 1993 to produce 6 egg types (Table 1) for use in experiments designed to compare the susceptibility of different genetic stocks of ducklings to *L. simondi* (for additional details see Shutler et al. 1996). Eggs were hatched in incubators so that ducklings could not acquire parasites from parents. *Leucocytozoon simondi* caused no mortality, and infection intensities did not differ significantly among types (Shutler et al. 1996). Although a comparison of genetic stocks was not a focus of this study, we controlled for duckling type in analyses (see below).

Ducklings were randomly assigned to the control or experimental category (for further details on the procedures and study sites described below, see Shutler et al. 1996; Table 1). There was no practical way to prevent vectors in Algonquin Park from getting inside cages while we measured or fed ducklings. Consequently, controls were retained at Lake St. Clair, where they remained free from *L. simondi* infection. A maximum of 2 control ducklings was randomly chosen from a brood; the remainder were experimental ducklings. For 5 successive weeks, all experimental ducklings that had hatched during the preceding week were transported together to Algonquin Park (all ducklings were first exposed when they were between 2 and 9 days old). Experimental ducklings were randomly assigned to one of three locales (Lake Sasajewan, Swan Lake, or Kennisis Lake) in and around Algonquin Park. Experimental ducklings were housed until the end of July in wire cages situated on lake shores, where black fly vectors are most abundant (Bennett 1960). Controls were retained at Lake St. Clair in identical cages situated on shorelines and treated similarly to experimental ducklings. For both control and experimental ducklings, exposure to the elements (and for experimental ducklings, to vectors) was similar to what would occur in the wild.

Tarsus length (the tarsus bone in Dzubin and Cooch 1992), culmen (culmen 1 in Dzubin and Cooch 1992), bill width (preceding all to the nearest 0.1 mm), and body mass (to the nearest 0.1 g up to 50 g, then to the nearest 1 g up to 100 g, and to the nearest 5 g thereafter) of ducklings were measured every 2 days from date of hatch (or 1 day of age) up to 14 or 15 days of age, and at least every 4 days thereafter. Because the most severe parasite effects occur 7–10 days post infection (Bennett et al. 1974; Atkinson and van Riper 1991; Desser and Bennett 1993), we focused on the first 20 days of duckling growth, as blood smears (see below) indicated that more than 85% of ducklings were infected for at least the last 10 days of this interval (Shutler et al. 1996). For each duckling, we calculated growth rates of each morphological measure; these growth rates were our response variables. (We also analysed Richards' growth curves (Richards 1959; White and Brisbin 1980; Brisbin et al. 1986; McCallum and Dixon 1990) for the first 80 days of life. Results of analyses comparing growth rate, time required to reach final size, and asymptotic size were qualitatively similar to those reported here.)

We collapsed structural measures (i.e., excluding body mass) using principal components (PC) analysis based on the correlation matrix. The first PC (from 3125 measurements) explained 97% of the variation in these structural measures (eigenvalue 3.88); duckling scores for this PC are referred to as SIZE. The remaining PCs explained less variation than expected by chance (according to the broken-stick criterion; Frontier 1976; Jackson 1993) and were not retained for analysis. Morphological characters grow partially independently of each other (Ricklefs 1973), so that, for example, fast growth in tarsus length could mask slow growth in bill width within a PC analysis. Hence, we also analysed growth rates of each morphological character independently.

Leucocytozoon simondi infections in ducklings were assessed from blood smears (Bennett 1970; for details see Shutler et al. 1996) 10 days after initial exposure to vectors, and again 7 days later. Infection intensity was measured as the number of parasites per 25 microscope fields examined at 400× magnification (approximately 18 000 blood cells). We predicted that experimental ducklings would have lower growth rates than (uninfected) controls. We also predicted that among infected ducklings, more intense infections would cause greater reductions in growth rates.

Statistical analyses were performed in SAS (SAS Institute Inc. 1990). Morphological measures were normally distributed (assessed with Shapiro–Wilk's tests), and were thus suitable for parametric analyses; it was necessary to (log + 1)-transform infection intensity data to achieve a normal distribution.

We used analysis of covariance to compare growth of control and experimental ducklings. Because of limited sample sizes, we were restricted in the number of covariates and interaction terms we could include in models. We chose those we judged would have the most influence on growth rates. Full models had growth rates of a morphological character as response variable, and year, duckling sex, duckling type, treatment, and the interaction between type and treatment as explanatory variables. If the interaction was not significant, it was dropped from the full model (Alisauskas and Ankney 1994; Sorci et al. 1996). Thereafter, nonsignificant variables, other than treatment, were iteratively removed to produce final models.

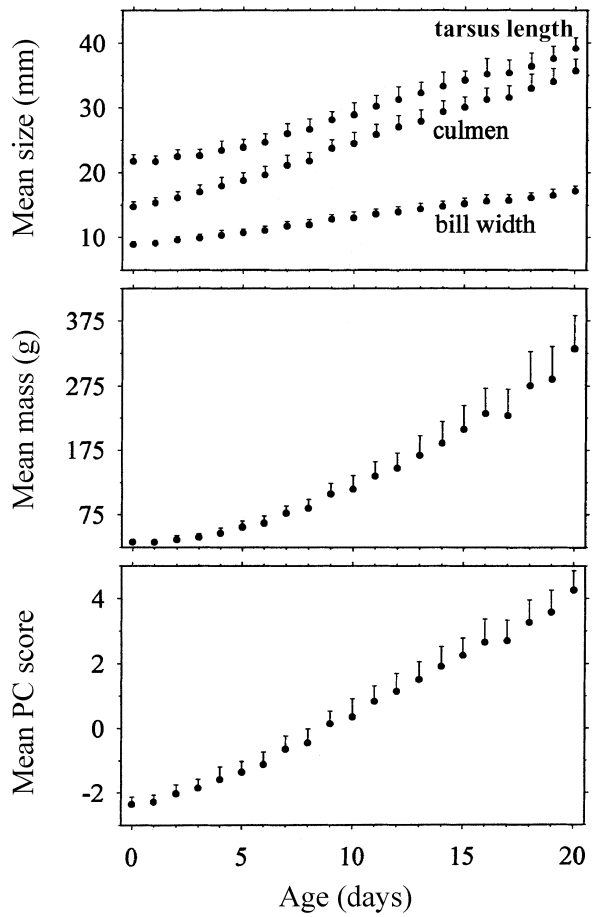
The next analysis used only experimental ducklings and focused on whether infection intensity (the maximum obtained from two smears for an individual) affected the growth rate. As was the case for the preceding models, growth rate of a morphological character was the response variable, and year, type, duckling sex, location of exposure, infection intensity, and an infection intensity by type interaction term were explanatory variables. We followed the same protocol as above, always retaining infection intensity in final models.

Table 2. Factors associated with variation in growth rates of control versus experimental ducklings.

	Morphological measure				
Variable in model	Tarsus length	Culmen	Bill width	Body mass	SIZE
Year	1.0		9.2**		
Sex	5.3*				
Type	3.1*				
Treatment	0.3	0.2	2.3	12.5***	1.6
Type × treatment	2.8*				
R^2	0.24**	0.00	0.09**	0.09***	0.01

Note: The values shown are F values for final models (following iterative removal of nonsignificant variables). There are 5 degrees of freedom associated with the interaction and type terms; all other variables had a single degree of freedom. Because of the significant interaction for tarsus length, each type was analysed separately (Table 3); *, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$.

Fig. 1. Mean size of each morphological character for all ducklings by age. All ducklings were measured on the day of hatch. Thereafter, they were measured only on predetermined days, which may have fallen 1 or 2 days after hatch. After a minimum age of 12 days, measurements were made only every 4 days. This caused variation in the number of ducklings measured at each age (sample sizes by age, beginning at 0 days, are 125, 48, 71, 50, 68, 57, 68, 59, 64, 66, 51, 67, 53, 67, 49, 58, 32, 29, 30, 29, and 34). To reduce clutter, only positive standard deviations are shown.



To account for the fact that we measured growth rates of five different morphological characters, we used a sequential Bonferroni correction to judge significance (Holm 1979; Rice 1989). This meant that significance was 0.05/5 = 0.01 for each set of analyses (treatment and intensity). In the text we report where Bonferroni significance was not met for the probabilities indicated in tables.

Results

Infections were detected in 43 of 44 (97%) experimental ducklings in 1992, in all 41 experimental ducklings in 1993, and in 0 of 13 controls (sampled in 1992 only; Shutler et al. 1996). Most ducklings had the highest intensity of blood parasites at 10 days post exposure (Shutler et al. 1996). For all morphological characters but body mass, growth rates were linear over the first 20 days (Fig. 1), and linear models were not improved by including a curvilinear term (R^2 values without squared term all >0.91, R^2 values with both linear and squared terms increased by <0.01, all F values >0.10). For body mass we used body mass squared as our dependent variable (R^2 = 0.86 with linear term, R^2 = 0.91 with squared term alone, R^2 = 0.92 with both terms).

Effect of treatment

Tarsus growth rates for unparasitised control and parasitised experimental ducklings were affected by duckling type (significant interaction in Table 2), so each type was analysed separately. Although treatment did not affect growth rates of five duckling types, counter to prediction, the 9 experimental OB × OM ducklings had faster tarsus length growth rates than their three control counterparts (Table 3; least square means were 0.69 mm per day for controls versus 0.94 mm per day for experimental ducklings). Culmen, bill width, and SIZE were also not affected by the presence of parasites (Table 2). However, again counter to prediction, ducklings gained mass more rapidly when parasitised than when unparasitised (12.0 g per day for controls versus 14.0 g per day for experimental ducklings) (Table 2). Final mass (at 80 days of age) of control ducklings (708 ± 405 g (mean ± SD)) did not differ from that of experimental ducklings (726 ± 407 g; t = –0.22, P = 0.83).

Some covariates were also associated with variation in growth rates. Although sex and type were significantly related to tarsus growth rate in the full model (Table 2), tarsi did not grow differently according to sex within duckling types (Table 3). Type appeared to be a significant covariate solely because of tarsus growth in OB × OM ducklings (Table 3). Year was the only other covariate that was significantly related to growth rate; the tarsus growth rate in OB × OM ducklings was higher in 1992 than in 1993 (but not significantly so after Bonferroni adjustment), whereas other

Table 3. Factors associated with variation in tarsus growth rates of control versus experimental duckling according to duckling type.

	Duckling type[a]					
Variable in model	SM × SM	SM × OM	SM × OB	OM × OM	OM × OB	OB × OB
Year					6.5*	
Sex						
Treatment	1.7	0.6	0.3	0.7	32.2***	0.5
R^2	0.20	0.03	0.06	0.01	0.80***	0.03

Note: The values shown are F values for final models (following iterative removal of nonsignificant variables); *, $P < 0.05$; ***, $P < 0.001$.
[a]OB, Ontario black duck; OM, Ontario mallard; SM, Saskatchewan mallard.

Table 4. Factors associated with variation in growth rates of experimental ducklings.

	Morphological measure				
Variable in model	Tarsus length	Culmen	Bill width	Body mass	SIZE
Year			4.8*		3.9*
Sex	5.3*				
Location	3.6*				4.0*
Type					
Intensity	0.8	0.2	0.4	0.7	0.8
Type × intensity					
R^2	0.15**	0.00	0.08*	0.01	0.13**

Note: The values shown are F values for final models (following iterative removal of nonsignificant variables); *, $P < 0.05$; **, $P < 0.01$.

duckling types grew similarly in both years. In contrast, across duckling types, bill width grew faster in 1993 than in 1992 (Table 2).

Effect of infection intensity

Infection intensity was not associated with variation in growth rates of any of the morphological variables (Table 4), but some covariates were. Year (bill-width growth was 0.41 mm per day for 1992 versus 0.43 mm per day for 1993; corresponding values for SIZE were 0.32 versus 0.34 PC units), sex (the tarsus growth rate for males was 0.91 mm per day versus 0.86 mm per day for females), and location (the tarsus growth rate was 0.87 mm per day for Lake Sasajewan versus 0.93 mm per day for Kennisis Lake versus 0.83 mm per day for Swan Lake; corresponding values for SIZE were 0.32, 0.35, and 0.31 PC units) were associated with variation in growth rates, but none of these associations remained significant after Bonferroni correction.

Discussion

Our results were not consistent with the prediction that *L. simondi* would impair growth of ducklings. In fact, the only two significant results were that infected ducklings grew faster than controls (OB × OM experimental ducklings had faster tarsus growth, and gained mass more rapidly, than controls). We can only speculate as to the cause of these results; possibly they were due to chance allocation of particularly large individuals to the experimental treatments (a sampling problem) or to differences among rearing locations. If faster growth of infected ducklings is a genuine phenomenon, we might expect that more intense infections would be associated with more rapid growth rates, but this was not observed. Finally, we found little evidence that the effects of *L. simondi* on duckling growth rates were related to local versus allopatric adaptations of hosts or parasites (cf. Lively 1989; Ebert 1994). The lack of effect of *L. simondi* on growth rates extends the results of our previous study, wherein we found no mortality from these parasites (Shutler et al. 1996). Together, the results of these studies suggest that the importance of *L. simondi* to wild waterfowl populations has been overestimated (Chernin l952; Herman et al. 1975; Hollmén et al. 1998; also see the introduction in Bennett et al. 1991).

Many other researchers have also concluded that blood parasites have no effects on wild birds (Bennett et al. 1988, 1993; Gibson l990; Weatherhead and Bennett 1991, 1992; Davidar and Morton 1993; Weatherhead et al. 1993). However, the effects of parasites may be subtle and yet have significant, cumulative evolutionary importance (Anderson and May 1979). Some experimental manipulations, such as experimental clutch enlargement, led to a higher prevalence of blood parasites in great tit (*Parus major*) parents (Norris et al. 1994; Richner et al. 1995; Ots and Hõrak 1996). In addition, direct experimental increases in parasite intensities can have substantial fitness effects on hosts (e.g., Clayton l990; Zuk et al. l990). Alternative experimental manipulations, such as keeping individuals free from parasites, as we did, or using drugs to remove parasites (e.g., Hillgarth l990), may be less able to reveal effects because most infected individuals may be able to buffer the effects of natural parasite intensities (e.g., Shutler et al. 1999*a*, l999*b*). On the other hand, at least one review of experimental and observational studies failed to show any evidence that experiments were more likely to detect effects of parasites (Møller 1997).

Parasites such as *L. simondi* that are transmitted between genetically unrelated hosts (i.e., are horizontally transmitted) generally cause greater pathology than parasites that are

only transmitted between parents and offspring (i.e., vertically) (Ewald 1994; Poulin 1998). This is because horizontally transmitted parasites have more opportunities to be transmitted even when they seriously incapacitate their hosts (Bull et al. 1991; Herre 1993; Clayton and Tompkins 1994; Ebert 1994). A second factor that may select for increased virulence is vector transmission relative to direct transmission, because even seriously incapacitated hosts continue to transmit parasites to mobile vectors (Ewald 1983). Despite this selection for virulence, the present and a previous study (Shutler et al. 1996) have failed to provide evidence that *L. simondi* has significant effects on wild ducklings. A third factor that may select for virulence is co-infections, because of resulting competition among parasites for host resources (van Balen and Sabelis 1995). The only other blood parasite detected in ducklings was a species of *Trypanosoma*, but it was seen in only one blood smear, and could usually be detected only if blood was centrifuged (which was done on only 5 of our ducklings; D. Shutler, unpublished data). The lack of hematozoa other than *Trypanosoma* sp. may have limited competition in this host habitat and limited selection for virulence in local strains of *L. simondi*. However, competition among genetic lineages of *L. simondi* could also select for virulence, and there is evidence suggesting that hosts carry multiple genetic lineages of *L. simondi* (Read et al. 1995). If such competition occurred within our ducklings, it also did not appear to be associated with substantial virulence.

There are countervailing selective pressures that could cause *L. simondi* to be benign. Although it is horizontally transmitted, in order to be passed on, it still requires that its host survive. Hence, excessive virulence that caused death directly, or made hosts more susceptible to predators, would not be in this parasite's best interests (Holmes and Zohar 1990; Poulin 1994). Nevertheless, virulence may be favored in areas of high host density, so that virulent strains are transmitted more readily than less virulent strains (Herre 1993; Ewald 1994). However, host density is less than one pair of ducks per lake in the Algonquin area (McNicol et al. 1987), which severely constrains parasite transmission (see also Fialho and Schall 1995; Shutler et al. 1996).

In sum, we found no evidence that *L. simondi* negatively affects growth of wild ducklings, suggesting that most wild mallard and black duck ducklings are equipped to buffer the parasite's effects. Alternatively, the parasite may serve its own best interests by remaining relatively benign, at least in natural, wild situations.

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