Predation cost of rapid growth: behavioural coupling and physiological decoupling

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Summary

1. Despite its prominent role in life-history theory, there is no direct empirical evidence for a behaviourally mediated predation cost of rapid growth. Moreover, we know little about how digestive physiology may also influence the shape of the growth/predation risk trade-off function.

2. We determined the role of behaviour and digestive physiology in experiments in which damselfly larvae were induced to grow slowly or rapidly by manipulating photoperiod (time stress), and exposure to a fish predator.

3. We showed that larvae under time stress grew more rapidly. Rapid-growing larvae had a higher foraging activity and a higher growth efficiency.

4. Under predation risk, larvae not only had a lower foraging activity but also a lower growth efficiency.

5. Rapid-growing larvae (i.e. those under time stress) balanced the growth/predation risk trade-off differently and took more risk in the presence of a predator, which resulted in a behaviourally mediated higher predation cost compared to slow-growing larvae. Their higher growth efficiency, however, made this cost smaller compared to a completely behaviourally mediated rapid-growth strategy.

6. Our results provide the first explicit experimental proof of a behaviourally mediated predation cost of rapid growth. Besides a behavioural coupling of growth and predation risk, resulting in the well-known trade-off, we also found a partial decoupling of these two processes by digestive physiology.

Key-words: antipredator behaviour, growth/predation risk trade-off, life-history plasticity, physiological stress, time stress.

Introduction

Understanding variation in growth rates has become a central focus in life-history theory because it determines how animals balance the advantage of short development times against the disadvantage of small sizes at maturity (Abrams *et al*. 1996; Nylin & Gotthard 1998). There is increasing evidence for adaptive variation in growth rates and for the presence of growth rates lower than the maximum possible in a given environment,

which suggest strongly that high growth rates are costly (Arendt 1997; Metcalfe & Monaghan 2001). In particular, predation costs of rapid growth have been invoked in life-history models. These models often assume explicitly that this cost is mediated through increased foraging behaviour; the behaviourally mediated growth/predation risk trade-off (e.g. Werner & Anholt 1993; Crowley 2000). However, empirical studies that tested explicitly predation costs of rapid growth are rare and did not measure foraging activity. Gotthard (2000) showed that faster-growing caterpillars of the butterfly *Parage aegeria* suffered from higher predation and assumed this to be behaviourally mediated. Similarly, Lankford, Billerbeck & Conover (2001) and Munch & Conover (2003) assumed that the higher predation cost of rapid growth in the fish *Menidia menidia*

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was an effect of reduced swimming performance. As a result, explicit proof of a behaviourally mediated predation cost of rapid growth is lacking.

One complicating factor that may affect the behavioural mediation of predation costs of rapid growth is digestive physiology. Rapid growth can be achieved not only through a higher foraging activity but also through a higher growth efficiency, the latter component being largely neglected in life-history studies (but see, for example, Present & Conover 1992). Theoretically, animals may be capable of increasing growth rate without increasing foraging activity and as a result decouple growth rate and mortality by predation. Several recent studies suggest a decoupling of activity, growth and development (e.g. Anholt, Werner & Skelly 2000; Johansson *et al*. 2001; Altwegg 2002), and three studies so far have explicitly identified the underlying role of growth efficiency in causing this (McPeek, Grace & Richardson 2001; Stoks & McPeek 2003; McPeek 2004). No studies so far, however, have considered how this decoupling might affect predation costs of rapid growth.

We present the results of two experiments designed to unravel the mechanistic link between growth rate and predation risk addressing the complicating factor mentioned above. Both experiments used larvae of the damselfly *Lestes sponsa* Hansemann and their predator, perch *Perca fluviatilis* Linnaeus. We manipulated growth rate by imposing time stress on the larvae as in Gotthard (2000). Under time stress, increased growth rates and foraging activities are predicted (Rowe & Ludwig 1991; Werner & Anholt 1993; Abrams *et al*. 1996), and demonstrated empirically in *Lestes* (De Block & Stoks 2005). This is logical, as under time stress animals will speed up development and will try to compensate for the resulting shorter period available for growth by speeding up growth rate (Abrams *et al*. 1996). The strength of this approach is that we manipulate randomly selected larvae to increase both their growth rate and foraging activity (in contrast to when, for example, manipulating food level; Anholt & Werner 1995). In a first experiment, we evaluated the role of foraging activity and digestive physiology in shaping growth rate, and whether their contribution changes with growth strategy and predation risk. In a second experiment, we explicitly test the behavioural mediation of the predation cost of rapid growth.

Methods

For both experiments we reared larvae of *L. sponsa* under manipulated time stress. This species diapauses during winter in the egg stage, has a brief larval period in spring, then emerges and reproduces in summer (Jödicke 1997). During winter, sedge stems containing eggs were kept outside. After winter, these stems were transferred to the laboratory (21 °C), immersed in water and assigned randomly to light regimens mimicking the start [15 March, light : dark (L : D) 12 : 12; early photoperiod] or the end (15 May, L : D 15·5 : 8·5; late photoperiod) of the egg hatching season in Belgium, respectively. Light regimens were adjusted weekly according to the natural progress of the light cycle. Note that by so doing, day lengths were initially longer at the late photoperiod, but then became longer at the early photoperiod. We rotated all larvae and their associated light regimen between two climate rooms every 2 weeks to minimize the possibility of confounding climate room with photoperiod (see Johansson & Rowe 1999). All larvae used in both experiments hatched on 7 March. Newborn larvae were placed individually into small white plastic cups (100 mL), given a toothpick as substrate and fed a ration of, on average, 230 *Artemia nauplii* (SE: 17, $n = 6$) five times a week. This corresponds to high food levels (Johansson *et al*. 2001).

GROWTH EXPERIMENT

We determined the contribution of behaviour and digestive physiology in shaping rapid growth under two levels of predation risk. Initially, 40 larvae were reared in each of the two photoregimens in the absence of a predator. These larvae, 16 full sibs per family, were obtained from five mating pairs the previous summer. On day 70, their wet mass was measured to the nearest 0·01 mg after gently blotting them dry on absorbent paper and their instar was determined. This yielded a measure of growth and development rate up to day 70. Starting on this day, larvae were given a daily ration of 30 *Daphnia pulex* De Geer of standardized size.

The 4-day growth experiment started 24 h after a larva moulted into the penultimate or final instar. Some larvae were used several days after a moult, but this did not affect the results (R. Stoks, unpublished data). We randomly implemented the predation-risk treatment, creating a full factorial design with four replicates of each combination of family, photoregimen (early vs. late) and predation risk (predator vs. no predator). By randomly using larvae from both instars we were successful in avoiding differences in initial larval mass between the four combinations of photoregimen and predation risk (ANOVA, all $P > 0·15$). The methodology closely followed that of Stoks & McPeek (2003). Larvae were kept in individual glass vials (20 mL) floating in 20 10-L aquaria (five per combination of photoregimen and predation risk). Four vials floated in each aquarium. Each larva was randomly reassigned daily to one of the five aquaria with its treatment combination. Predation risk was manipulated by adding one perch (standard length 6 cm) to half of the aquaria. Perch were kept on a diet of mealworm and *L. sponsa* larvae. Damselfly larvae could see the perch through the glass vials and could smell them via water exchange through a submersed cylinder made of Nitex screen attached to the top of each vial. Aquaria were filled with aged tap water that was replaced every 4 days and permanently aerated.

At the start of the 4-day period, wet mass of each larva was measured. At the end, each larva was dried

Table 1. Principal components analysis for the original ln transformed behavioural variables of the predation experiment. Factor loadings > 0·5 are given in bold type

Original variable	PC1	PC2	PC3
Number of walks	−0·23	**−0·95**	−0·06
Total distance of walks	−0·26	**−0·94**	−0·06
Number of swims	0·16	0·16	**0·87**
Total distance of swims	0·08	−0·10	**0·70**
Number of push-ups	0·25	0·18	**0·79**
Number of advances	**0·94**	0·13	0·12
Total distance of advances	**0·93**	0·08	0·05
Number of orientations	**0·72**	0·41	0·39
Number of unsuccessful strikes	**0·81**	0·37	0·31
Number of captures	**0·76**	0·43	0·36
Explained variance (%)	37·24	23·73	22·69

for > 24 h at 65 °C to determine final dry mass. Initial wet mass was converted into dry mass using the formula: dry mass = 0·14 × wet mass ($R^2 = 0·99$, $n = 80$). Growth rate was calculated as [ln(final dry mass) − ln(initial dry mass)]/4 days. This measure of growth proved independent of initial larval wet mass ($r = −0·11$, $P = 0·32$, $n = 80$) and therefore also of instar. To quantify digestive variables, we determined dry masses of given food rations, uneaten food and faecal pellets to the nearest 0·1 µg using a electrobalance (Cahn C-35, Massachusetts, USA). The amount of ingested food was estimated as (total dry mass supplied food) − (total dry mass uneaten food). The amount of assimilated food was calculated as (total amount ingested food) − (total dry mass of faecal pellets). Assimilation efficiency was calculated as (amount of assimilated food)/(amount of ingested food), conversion efficiency was calculated as (gain in dry mass by the larva)/(amount of assimilated food) and growth efficiency was calculated as their combined effect (gain in dry mass by the larva)/(amount of ingested food) (Slansky & Rodriguez 1987). Growth rate and the digestive parameters were analysed with three-way mixed model ANOVAs. Photoregimen and predation risk were considered as fixed, family as a random effect. Additionally, we performed a general linear model (GLM) testing the contribution of the behavioural component (food ingested) and the physiological component (growth efficiency) in shaping growth rate. Food ingested should be a good surrogate for direct estimates of behaviour because it is impossible to ingest food without performing some of the behaviours estimated below in the predation experiment (food ingested and foraging activity load on the same principal component axis, see Table 1). To evaluate potential treatment-dependent contributions of both growth components, we added their two-way interactions with photoregimen and predation risk. We did not consider family as a focal variable and will not report its effects. Means are given ± 1 SE.

PREDATION EXPERIMENT

In the predation experiment, we determined directly the contribution of behaviour in shaping predation costs of rapid growth. We reared 60 larvae, using the same set-up as in the growth experiment, at each of the two photoregimens in the absence of a predator. A subset of 30 larvae of each photoregimen were used for the predation trials 2–4 days after their moult in the final instar. The other larvae were used to score growth rate, age and mass at emergence.

We scored the behaviour of each larva during two successive 15-min periods, in the absence and presence of one perch, respectively. We followed closely the methodology of Stoks & Johansson (2000). After addition of 200 *Daphnia*, one larva was placed at one side of a 10-L aquarium that was divided into two equal parts by a screen. After 30 min we scored its behaviour continuously for 15 min. We then introduced one perch (standard size 6·5 cm) into the other compartment and scored the behaviour of the larva for another 15 min. The larva could see and smell the perch through holes in the transparent screen. Larvae do not show saturation during the second observation period (Stoks & Johansson 2000). After this second period we lifted the screen. The time between the moments the fish started foraging on *Daphnia* and the capture of the larva was defined as the survival time of the larva. Mortality rate was calculated as 1/survival time. Artificial vegetation was provided by vertical plastic rope strands spaced 2 cm from each other. Strands were fixed to a PVC plate, which was covered with a 3-cm layer of fine grind.

We recorded 10 behavioural components (Table 1). For swimming, walking and advancing we calculated frequency and distance travelled, summed across a 15-min observation period. Walking is a random change in position, while advancing toward prey is a walk to chase a *Daphnia*. Push-ups are up- and downwards movements of the body by bending the legs. Orientating towards prey was defined as a larva turning its head or body towards a *Daphnia* without changing its position. Finally, we scored separately unsuccessful strikes at the *Daphnia* prey and captures.

We first extracted principal component scores from the correlation matrix of the original $\ln(x + 1)$ transformed behavioural variables. The resulting axes were rotated using the normalized varimax method to aid
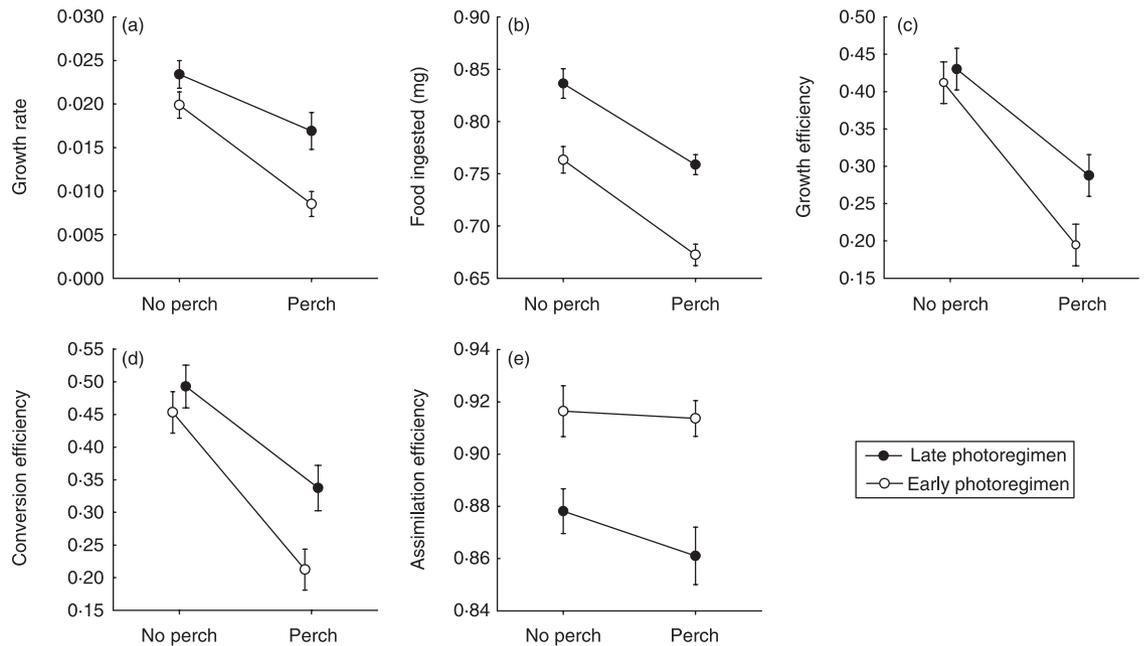
Fig. 1. (a) Growth rates and digestive variables, (b) amount of food ingested, (c) growth efficiency, (d) assimilation efficiency and (e) conversion efficiency of *Lestes sponsa* larvae studied in the growth experiment. Means are given ± 1 SE.

their interpretation (Sokal & Rohlf 1995). We tested the effect of photoregimen and presence of perch on the behavioural principal components with repeated measures ANOVAS (RM-ANOVAS). The successive behavioural principal component scores of each larva during the first and second observation period, hence in the absence and presence of perch, respectively, were included as repeats. To test the effect of the behavioural variables in the presence of fish in shaping mortality rate we used a general linear model with photoregimen as the categorical predictor variable and the behavioural principal components as continuous predictor variables. Interactions between photoregimen and the behavioural principal components on mortality rate were not significant (all $P > 0.10$), and dropped from the final model.

Results

GROWTH EXPERIMENT

Already during the first 70 days it became apparent that, consistent with the imposed time stress, larvae at the late photoregimen had higher development and growth rates than larvae at the early photoregimen. At day 70, larval instar varied from F-3 to F-0 (F-0: final instar, F-1: penultimate instar, …). Larvae reared at the late photoregimen (hereafter called late larvae) were in a more advanced instar than larvae reared at the early photoregimen (early larvae) (instar distribution F-3 : F-2 : F-1 : F-0, late: 1 : 7 : 20 : 12 vs. early: 10 : 26 : 4 : 0) (log-linear analysis, photoregimen × instar: $x_1^2 = 38.68$, $P < 0.0001$). Late larvae had a higher mass at day 70 than early larvae (27·32 ± 0·74 mg vs. 18·59 ± 0·61 mg) (ANOVA: photoregimen: $F_{1,70} = 89.22$, $P <$

0·001). At emergence early larvae would probably have caught up in size with late ones as they have a longer development; see Predation experiment.

In the 4-day growth experiment, late larvae had a higher growth rate than early larvae (ANOVA, photoregimen: $F_{1,60} = 27.72$, $P < 0.001$; Fig. 1a). This was associated with a higher food ingestion ($F_{1,60} = 189.52$, $P < 0.0001$; Fig. 1b) and a higher growth efficiency ($F_{1,60} = 9.49$, $P < 0.05$; Fig. 1c). The higher growth efficiency in late larvae was due to a higher conversion efficiency ($F_{1,60} = 18.57$, $P < 0.05$; Fig. 1d), as late larvae showed a lower assimilation efficiency ($F_{1,60} = 49.17$, $P < 0.01$; Fig. 1e). All larvae reduced their growth rate in the presence of the predator (predator: $F_{1,60} = 76.35$, $P < 0.0001$), but the early larvae did so more (photoregimen × predator: $F_{1,60} = 11.08$, $P < 0.05$; Fig. 1a). The growth reduction under predation risk was associated with a lower food ingestion ($F_{1,60} = 44.02$, $P < 0.01$; Fig. 1b) and a lower growth efficiency ($F_{1,60} = 116.56$, $P < 0.001$; Fig. 1c). The lower growth efficiency was due to a lower conversion efficiency ($F_{1,60} = 70.77$, $P < 0.001$; Fig. 1d), while assimilation efficiency was not affected ($F_{1,60} = 1.17$, $P = 0.34$; Fig. 1e).

Both food ingested and growth efficiency contributed significantly to growth rate (GLM, food ingested: $F_{1,70} = 4.98$, $P < 0.03$; growth efficiency: $F_{1,70} = 28.94$, $P < 0.0001$). However, their contribution to growth rate depended on predation risk (predator × food ingested: $F_{1,70} = 3.46$, $P = 0.067$, predator × growth efficiency: $F_{1,70} = 6.98$, $P = 0.010$). In the absence of a predator, both food ingested (partial correlation: 0·55, $t_{37} = 4.04$, $P < 0.0003$) and growth efficiency (partial correlation: 0·38, $t_{37} = 2.53$, $P = 0.016$) had significant positive contributions to growth rate. In the presence of a predator,
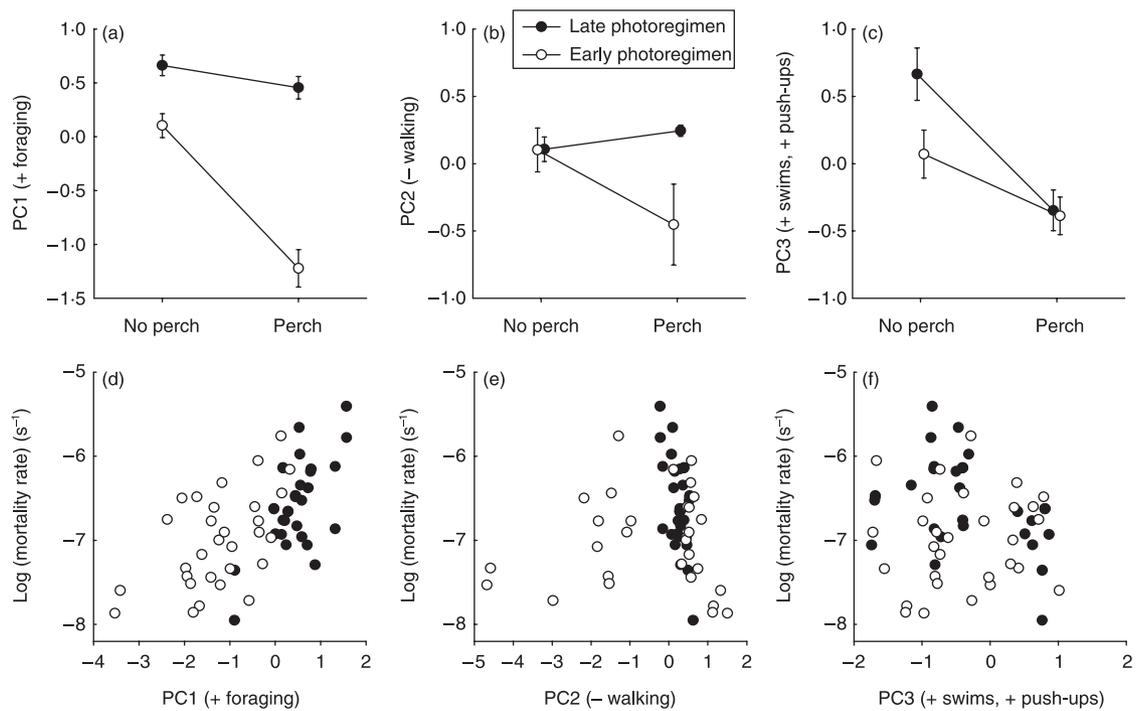
Fig. 2. (a–c) Behavioural responses of *Lestes sponsa* larvae in function of photoregimen and predation risk in the predation experiment. (d–f) Relationships between mortality rate (ln transformed) and scores of the behavioural principal components in the presence of perch. Means in the upper panels are given ± 1 SE.

the contribution of food ingested was much lower (partial correlation: 0·27, $t_{37} = 1·70$, $P = 0·098$), and it was growth efficiency that mainly shaped variation in growth rate (partial correlation: 0·70, $t_{37} = 6·04$, $P < 0·0001$).

In summary, the growth experiment shows that late larvae increased development and growth rate and suggests this increase is due to a combination of a higher amount of food ingested and a higher conversion efficiency. Perceived predation risk reduced growth rate, owing to a combination of reduced food intake and reduced conversion efficiency. Interestingly, early larvae reduced their growth rate more under predation risk than late larvae.

PREDATION EXPERIMENT

Late larvae increased growth rate by about 11% (late vs. early photoregimen: 0·0613 ± 0·0005 vs. 0·0550 ± 0·0005, ANOVA $F_{1,58} = 78·65$, $P < 0·0001$), and emerged on average 11 days earlier (day 101 ± 1 vs. day 112 ± 1 day, with day 0 = day of hatching; $F_{1,58} = 57·15$, $P < 0·0001$). Both life-history effects balanced, as mass at emergence was not affected by time stress (late vs. early photoregimen: 50 ± 18 mg vs. 48 ± 18 mg; $F_{1,58} = 0·35$, $P > 0·55$).

Three principal components, each having an eigenvalue > 1, summarized 84% of the variation in the original behavioural data set (Table 1). PC1 was highly positively correlated with variables describing foraging activity, such as the number of advances, distance travelled during advances, the number of orientations, strikes and captures. PC2 was strongly negatively associated

with walking. Finally, PC3 receives high loadings from swimming and push-up frequencies.

Late larvae had a higher foraging activity (PC1) than early larvae (RM-ANOVA, photoregimen: $F_{1,58} = 63·25$, $P < 0·0001$; Fig. 2a). All larvae reduced their foraging effort in the presence of the predator (predator: $F_{1,58} = 51·48$, $P < 0·0001$), but early larvae reacted much more (photoregimen × predator: $F_{1,58} = 27·45$, $P < 0·0001$). Larvae from both photoregimens did not differ in walking (PC2) in the absence of the predator. However, early larvae increased walking after introduction of the perch while late larvae did not (photoregimen × predator: $F_{1,58} = 6·66$, $P = 0·012$; Fig. 2b). Late larvae made more swims and push-ups (PC3) than early larvae in the absence of perch, but they also decreased these behaviours more, resulting in similar intensities in the presence of perch (photoregimen × predator: $F_{1,58} = 8·49$, $P < 0·005$; Fig. 2c).

Late larvae had a higher mortality rate than early larvae ($F_{1,58} = 9·11$, $P < 0·004$, Fig. 2d–f). When including foraging activity in the model, the effect of photoregimen on mortality was no longer significant ($F_{1,55} = 2·36$, $P = 0·13$), indicating that it worked entirely through foraging activity. More active foraging larvae indeed had a higher mortality rate ($F_{1,55} = 24·68$, $P < 0·0001$; Fig. 2d). The two other behavioural principal components did not affect mortality (both $P > 0·50$).

In summary, late larvae had not only an accelerated life history but had also a higher foraging activity compared to early larvae and reduced this activity less in the presence of the predator. The results suggest

that this higher activity caused the higher mortality by predation.

Discussion

Our results show a mortality cost of rapid growth as induced by time stress, and prove that this cost is mediated by foraging activity. In accordance with optimality models (Rowe & Ludwig 1991; Houston, McNamara & Hutchinson 1993; Werner & Anholt 1993; Abrams *et al*. 1996), time-stressed larvae increased their foraging activity to speed up growth and development in both experiments. Note that these differences between larvae from both photoregimens cannot be attributed simply to different feeding times associated with different day lengths. Indeed, day lengths were adjusted weekly and summed across the entire growth period day lengths are equal between photoperiods. Moreover, damselfly larvae can feed very successfully in the dark (Johansson 1993), and during the 4-day growth and predation experiment day lengths were actually longer at the early photoperiod. In addition, during the predation experiment larvae were exposed to predators only for a short amount of time before they were eaten by the predator. Hence, the time exposed to the predator was very short and affected only by behaviour, not by the 'actual time spent' under light conditions.

The rapid-growth strategy under time stress was beneficial because it allowed late larvae to emerge at the same mass as early larvae despite their shorter development time. Mass at emergence is linked positively to survival in the adult stage (De Block & Stoks 2005). However, this behaviourally mediated growth increase was also costly as it resulted in higher predation rates. This was especially true because late, rapid-growing larvae reduced their foraging activity less in the presence of the perch predator than early, slow-growing larvae. As a consequence, the difference in activity between late and early larvae was larger in the presence of a predator. We note that the smaller reduction in foraging activity under predation risk was significant only in the predation experiment, probably because foraging in the small vials of the growth experiment was less risky. Alternative explanations, besides foraging activity, for shaping the mortality cost of rapid growth are unlikely. First, early and late larvae did not differ in mass at emergence and all larvae were scored in their final instar, making size-biased predation by the fish on the faster growing larvae unlikely. Secondly, although reduced swim burst speeds in larvae with a high growth rate may, in principle, generate a similar pattern (Lankford *et al*. 2001), this is unlikely with the fish predator because damselfly larvae are not able to escape a fish attack by swimming away (Stoks & De Block 2000). Thirdly, changes in other behaviours not linked to foraging activity (PC1) did not explain the pattern in our experiment. In the presence of the restrained fish, walking activity (PC2) was even higher in the early larvae, and no differences in swimming and push-ups (PC3) were

found. Moreover, these two other behavioural principal components were not linked to mortality rate. Finally, the fact that time stress did not affect predation rates when we corrected for foraging activity strongly suggests that we have provided the first empirical proof for a behaviourally mediated predation cost of rapid growth.

Increased behavioural risk-taking under time stress has been predicted by several models (Rowe & Ludwig 1991; Werner & Anholt 1993; Abrams *et al*. 1996), and may be a general pattern causing higher mortality. In a previous study on *L. sponsa* we found similar increased foraging but only a trend for higher risk-taking under time stress (Johansson *et al*. 2001). However, in the latter study we compared foraging activity between larvae that were never and continuously exposed to a perch, respectively. When applying a risk pulse as in the present study, more pronounced antipredator responses are to be expected (Lima & Bednekoff 1999), making it more likely to see differences in antipredator responses among time stress treatments. This may also explain the weak or no support for higher behavioural risk-taking under time stress in other studies (Laurila & Kujasalo 1999; Altwegg 2002; Lane & Mahony 2002). Indirect support for higher risk-taking under time stress comes from other laboratory studies showing that time-stressed animals suffer higher mortality (Johansson & Rowe 1999; Gotthard 2000).

In addition to behaviour, digestive physiology also played a role in shaping growth rate, which may partly decouple the behaviourally mediated predation cost of rapid growth. Time stress affected the digestive physiology of the larvae in opposite ways. While time-stressed larvae had a lower assimilation efficiency, they had a higher conversion efficiency. Shifts in assimilation efficiency may be due to morphological plasticity (e.g. changes in the part of the gut that is responsible for absorption) or to physiological plasticity (e.g. retention time of meals within gut) (e.g. Flanagin, Haase & Juliano 2000). We have no data to test these mechanisms directly. However, the increased feeding rate and increased general activity of *L. sponsa* under time stress (Johansson *et al*. 2001; this study) may be associated with shorter gut-retention times. The higher conversion efficiency indicates that they converted more of the assimilated food into biomass. This may be achieved by allocating a smaller proportion of ingested energy to maintenance and tissue synthesis (Billerbeck, Schultz & Conover 2000). The net effect was an increased growth efficiency under time stress. As a result, to achieve their rapid growth strategy larvae under time stress needed to increase foraging activity less than they would have, had they not increased their growth efficiency. Moreover, the relative contribution of growth efficiency compared to foraging activity in shaping growth rate was especially important under predation risk. Such physiological mediation of growth rate will reduce the predation cost of rapid growth and may therefore partly decouple the growth/predation risk trade-off. We think that this pattern of physiological compensation to increase growth rate may be widespread. Although we are not aware of other

studies showing this at a within-population level, similar increases in growth efficiency underlie increasing growth rates among populations along latitudinal gradients with decreasing time available for growth (Billerbeck *et al.* 2000; Robinson & Partridge 2001).

Apparently, the physiological compensation mediated by increasing growth efficiency was limited and could not completely drive acceleration of growth rate, so that behavioural growth mediation and its associated predation cost were still present. It is likely that increases in growth efficiency are themselves costly and are traded off against other agents of mortality such as winter survival or other fitness components such as fecundity (Arendt 1997; Metcalfe & Monaghan 2001; Munch & Conover 2003). Preliminary results suggest long-term costs on adult fitness of rapid growth in the larval period of the study species (De Block & Stoks, in press). Our results suggest that physiological compensation was more important under predation risk, as expected. A non-adaptive outcome is that larvae not only reduced foraging, a well-known antipredator response (Lima 1998), but also growth efficiency under predation risk. This suggests interference from other limiting factors. There is accumulating evidence for such negative effects of predators on physiology in damselflies (McPeek *et al.* 2001; Stoks & McPeek 2003) and in other taxa [birds: Scheuerlein, Van't Hof & Gwinner (2001); fish: McCormick (1998); mammals: Boonstra *et al.* (1998)].

Predators can affect prey directly through lethal effects or indirectly through sublethal effects on life history (McPeek & Peckarsky 1998; Werner & Peacor 2003), and the relative importance of both mechanisms will depend on how prey balance the growth/predation risk trade-off. The mechanistic base of growth-rate variation may play an important role in this balance. Our study and others indicate that when prey adopt a rapid growth strategy, lethal predator effects may become relatively more important because of higher risk-taking of the prey (see also Altwegg 2002). However, our results suggest that rapid growth is also mediated partly physiologically, especially under predation risk, making rapid growth less costly in terms of predation compared to an entirely behaviourally mediated rapid growth. Increasing physiological mediation of growth will therefore allow the prey to balance the growth/predation risk trade-off more towards rapid growth. The emerging picture from this and other studies is that behaviour and (digestive) physiology may jointly shape the trade off. While behavioural mediation may act to couple both fitness components, our study suggests that physiological mediation may act partly to decouple them. Future efforts must determine whether the role of behavioural mediation of the trade-off, which makes it a key mechanism linking individual behaviour to population-level processes (Werner 1992; Anholt & Werner 1995; Biro, Post & Parkinson 2003a,b), is widespread and to what extent physiological compensation may affect behavioural decisions and balance the growth/predation risk trade-off.

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