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Effects of early resource limitation and compensatory growth on lifetime fitness in the ladybird beetle (*Harmonia axyridis*)

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fitness;
life history;
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mating success;
resource limitation;
trade-off.

Abstract

Acceleration of growth following a period of diet restriction may result in either complete or partial catch-up in size. The existence of such compensatory growth indicates that organisms commonly grow at rates below their physiological maxima and this implies a cost for accelerated growth. We examined patterns of accelerated growth in response to temporary resource limitation, and assayed both short and long-term costs of this growth in the ladybird beetle *Harmonia axyridis*. Subsequent to the period of food restriction, accelerated growth resulted in complete compensation for body sizes, although we observed greater larval mortality during the period of compensation. There were no effects on female fecundity or survivorship within 3 months of maturation. Females did not discriminate against males that had undergone compensatory growth, nor did we observe effects on male mating behaviour. However, individuals that underwent compensatory growth died significantly sooner when deprived of food late in adult life, suggesting that longer-term costs of compensatory growth may be quite mild and detectable only under stressful conditions.

Introduction

Growth conditions experienced in early development may have important fitness costs over the long term (e.g. Metcalfe & Monaghan, 2001; Brakefield *et al.*, 2005; Taborsky, 2006). Most commonly, costs of depressed growth are manifested in reduced size at maturity or prolonged development time, although the direction and magnitude of these effects depend on the duration, severity and timing of the poor growth conditions (e.g. Day & Rowe, 2002, Davidowitz *et al.*, 2003). Reduced size at maturity has been associated with decreased survivorship, fecundity and mating success in a variety of taxa (Roff, 1992; Honek, 1993; Sokolovska *et al.*, 2000), whereas delayed maturation elevates prereproductive mortality and in some circumstances, reduces fitness because of an extended generation time (Roff, 1992; Stearns, 1992). In light of these costs to periods of reduced growth, one might expect selection for accelerated growth should conditions subsequently improve. In fact, there is growing evidence of compensatory growth, defined as 'a phase of accelerated growth when favourable conditions are restored after a period of growth depression' (Ali *et al.*, 2003), particularly in vertebrate taxa (Metcalfe & Monaghan, 2001; Ali *et al.*, 2003; Ozanne & Hales, 2004). Accelerated growth may result in full or partial compensation for body size or stored resources, thereby offsetting the costs arising from the initial period of growth restriction, although both empirical studies and theory indicate that the magnitude of the response will depend on the timing and severity of the period of growth restriction (Roseboom *et al.*, 2000; Yearsley *et al.*, 2004; Mangel & Munch, 2005).

In addition to compensatory growth, researchers have found that early growth restriction has a variety of effects on behavioural, physiological, morphological and life history traits over the short- and long-term, and it is hypothesized that these are adaptive responses that minimize the costs arising from periods of poor growth (Gotthard *et al.*, 1994; Metcalfe & Monaghan, 2001; Ozanne & Hales, 2004; Dmitriew & Rowe, 2005; Stoks *et al.*, 2006). Accelerated growth is often achieved by increasing foraging effort, which may come at an

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immediate cost to survival if foraging behaviour is conspicuous to predators (Abrams *et al.*, 1996; Anholt & Werner, 1998; Gotthard, 2000). Animals that can increase growth rates via pathways other than foraging effort (e.g. Boujard *et al.*, 2000; Dmitriew & Rowe, 2005) may therefore have an advantage. However, in spite of compensatory growth, even brief periods of resource restriction early in life may have negative effects on fitness later in life, either because the strength or duration of the compensatory response is insufficient, or because resources invested in accelerating growth are diverted from other traits and functions (Royle *et al.*, 2005; Johnsson & Bohlin, 2006; Stoks *et al.*, 2006).

A particular emphasis has been placed on the effect of poor conditions on traits (e.g. age and size) measured at eclosion or maturity because they are thought to be highly correlated with fitness and therefore serve as convenient proxies for lifetime fitness, obviating the need to monitor subjects through their entire life. However, conditions during development are known to impact fitness independently of size and age at maturity (Gotthard *et al.*, 1994, Roseboom *et al.*, 2000; De Block & Stoks, 2005). For example, immunocompetence and longevity were reduced despite compensation for size in zebra finches and humans exposed to resource limitation during early development (Birkhead *et al.*, 1999; Roseboom *et al.*, 2000). Furthermore, it is known that food restriction during development can have complex effects on body allometry (e.g. Stevens *et al.*, 1999; Boggs & Freeman, 2005; Dmitriew & Rowe, 2005; Frankino *et al.*, 2005), suggesting that simply measuring body mass or a single size metric may well miss potentially important costs that are manifested in other metrics of size. Finally, compensation for size is often incomplete, and in these cases, it is difficult to determine whether downstream fitness costs arise solely from changes in body size or whether there are additional, hidden costs. Although some studies have attempted to correct for the effects of variation in body size at maturation using statistical adjustments (e.g. De Block & Stoks, 2005), an alternative approach is to study animals in which complete compensation for body size has occurred. Only a few studies of early resource restriction have demonstrated downstream effects on adult traits independent of body size (e.g. Birkhead *et al.*, 1999; Ali *et al.*, 2003; Johnsson & Bohlin, 2006). Yet the connection between observed changes in these traits and fitness is not always clear. As patterns of resource allocation vary in response to resource availability, some traits may be positively affected and others negatively (Ali *et al.*, 2003). It is, therefore, perhaps not surprising that few general patterns regarding the costs of compensatory growth emerge from the literature.

These issues may be circumvented by directly assaying those composite traits that are the components of fitness: fecundity, mating success and longevity. The long-term effects of maternal malnutrition has been linked to reduced survivorship in humans and mammalian model systems via elevated levels of cardiovascular disease and metabolic disorders arising after conditions improve (reviewed in Barker *et al.*, 2002; Ozanne & Hales, 2004). However, to our knowledge, no other studies have looked at the effects of early resource limitation and compensatory growth on the suite of fitness components: fecundity, mating success and longevity.

To address these questions, we conducted an experiment designed to determine the lifetime consequences of a brief but significant period of diet restriction early in the development of the multicoloured Asian ladybird beetle, *Harmonia axyridis*. Preliminary experiments allowed us to determine a feeding regime that would result in delayed growth followed by complete compensation for body size at eclosion. This, in turn, allowed us to assay any costs that were truly independent of size. Lifetime fitness costs were then assessed using direct measures of the components of fitness: longevity and fecundity in females and mating success in males. In addition to these potential long-term costs, we also evaluated potential short-term costs, including mortality and activity rate during the periods of diet restriction and compensatory growth.

Methods

Harmonia axyridis, although native to Asia, has become established as an invasive to North America over the last 20 years (Koch, 2003). Since then, it has spread rapidly, becoming the dominant coccinellid species in Southern Ontario, feeding primarily on aphids (Yasuda & Ohnuma, 1999). Larval cannibalism is common, particularly when aphid populations are limiting (Pervez *et al.*, 2006). Females are highly fecund, producing up to several hundred eggs during its lifetime in the lab; both fecundity and lifespan are highly variable and depend on diet (e.g. Lanzoni *et al.*, 2004; Evans & Gunther, 2005).

Experiment 1: Effects of early growth conditions on the components of female fitness

Adult ladybird beetles were collected on 5 November 2005 from Oakville, Ontario and after 3 days in the lab were stored for several weeks at 4 °C. On 14 January beetles were transferred to 8.5 cm diameter Petri dishes at room temperature and fed fruit, sugar water and aphids. On 3 February 24 females were paired with males, allowed to mate and oviposit. To prevent future cannibalism of larvae, adults were removed from the Petri dishes after 24 h. Clutches produced during this period that contained more than 10 eggs ($n = 11$) were collected for use in the experiment. Following hatching on 7 February larvae were haphazardly assigned to one of two food treatments, with families evenly distributed across the treatments (10–30 eggs per family).

Larvae were then housed at room temperature (20–22 °C) individually in 5.5 cm diameter Petri dishes

containing a piece of cotton moistened with sucrose solution (approximately 15 g per 100 mL of water) until emergence. New sucrose solution was added daily, ensuring a constant supply of sugar. Food provided for both larvae and adults were live large Pea aphids (*Acyrthosiphon pisum*), which were reared on broadbean plants in the lab. We chose a schedule of feeding that appeared to be slightly under *ad libitum* in that all food was eaten except on very rare occasions, when one aphid would remain. Any higher food level risked more uneaten food items during normal growth, which compensating larvae could consume to increase growth rate. The schedule of larval feeding was as follows: larvae were fed two small aphids daily until day 2, 4 adult aphids daily until day 6, and 10 aphids daily until emergence. Larvae in the high food treatment ($n = 100$) were fed *daily* according to the above schedule, whereas those in the low food treatment ($n = 100$) were fed according to this schedule, but only *every second day* for the first 8 days after hatching and *daily* thereafter. Thus, larvae in the low food treatment received half the ration of the high food larvae for the first 8 days, but equivalent ration over the remainder of the larval period. Smaller aphids were used for first instar larvae to ensure that ingestion was not limited by prey size. Hatchlings were weighed and head and pronotum width measured within 24 h. These measurements were repeated at subsequent moults and again within a day of eclosion.

Following eclosion, animals were sexed and females were placed in 8.5 cm diameter Petri dishes containing sugar water. For the first 3 days individuals were fed three aphids daily, and the ration was increased to eight aphids per day thereafter. We chose this ration to reflect an intermediate feeding level. It was sufficient for sustained egg production in females but below that required for maximize reproductive output (Lanzoni *et al.*, 2004).

One week after eclosion (allowing for a teneral period between emergence and sexual maturation), males from the lab population that emerged within a week of the focal females were placed individually in each Petri dish (housing a female) and allowed to mate. Petri dishes were checked periodically to ensure that mating occurred, at least once. Males were removed after egg laying commenced (about 9 days). Individuals that were not observed to have produced eggs within 2 weeks were paired with a novel male but if mating still did not occur these individuals were discarded from the analysis of egg production ($n = 14$).

In order to assess the effects of early food deprivation and subsequent compensatory growth on female fitness, mortality, daily egg production and egg hatching rate were recorded for 100 days after the first adults had emerged. At 100 days we applied a severe stress (starvation) and monitored subsequent fecundity and longevity. During this period, the sugar water and aphids were removed and larvae were provided with water-soaked cotton wool as a source of moisture. In our analyses, we tested for the effect of food treatment on the components of lifetime fitness, as well as considering early life fecundity separately because it may have a disproportionately high impact on lifetimes fitness given that mortality rates in the wild can be very high (e.g. Thorne *et al.*, 2006). A period of 25 days following emergence was selected as this was the mean lifespan of *H. axyridis* observed by Lanzoni *et al.* (2004) under abundant food conditions and maximum reproductive rate.

Experiment 2: Effect of early growth conditions on male mating success

In experiment 1, adult fitness was assayed in females only. Therefore, we repeated the larval food manipulation and assayed components of adult male fitness. Adult ladybird beetles were collected from Toronto, ON in June, 2006. Twenty male–female pairs were formed in 4′ Petri dishes containing sugar water and fed aphids daily. Adults were removed shortly before hatching. Larvae hatched on June 30 were distributed between two treatments, low and high food (120 larvae per treatment), and reared individually in Petri dishes. The food rations of the low and high food treatments mimicked those in experiment 1, except that the period of reduced ration for the low food treatment last 6, rather than 8 days. Head width, pronotum width and mass of larvae were measured on day 6 to ensure that the low food treatment resulted in delayed growth. These measurements were repeated within a day post-eclosion to adult.

In order to determine whether larvae increased foraging effort in an attempt to accelerate growth rates, larval activity levels were estimated on two occasions: during the period of diet restriction and later during compensatory growth. Movements of a subset of 6-day-old larvae ($n = 110$) were tracked in the absence of food using a digital camera and the software program ETHOVISION™. During the period of observation, which lasted for 15 min, larvae were held individually in clean 5.5 cm diameter Petri dishes containing moist cotton. Distance travelled and mean velocity during movement were recorded and compared between treatments. This was repeated on day 15 for the same subset of individuals during the period of compensatory growth. One individual from the control treatment had begun pupating by this point and was excluded from the analysis.

In order to determine whether there were treatment effects on adult male fitness, we assayed a variety of mating behaviours and female preference. Mating behaviour in *H. axyridis* is fairly stereotypical. Males mount a female and commence copulation immediately unless females struggle, which occurred only rarely during this experiment. After approximately 1 h, males begin to shake intermittently while still in copula, behaviour that generally lasts for 1–2 h, after which males dismount without guarding (C. Dmitriew, personal observation).

Copulation duration is often correlated with male mating success. The quantity of sperm transferred may increase with copulation duration, delay female remating, or accelerate female oviposition, thereby increasing fertilization success in polyandrous species (Simmons, 2001). In coccinellids specifically, time to oviposition decreases with mating duration and is positively correlated with paternity success and fertility (De Jong *et al.*, 1998; Omkar *et al.*, 2006); we therefore measured copulation duration (and shaking duration alone) as an indicator of male fitness.

On 11 August virgin males were marked using non-toxic paint and placed individually in fresh Petri dishes containing a piece of cotton soaked with sugar water. One previously mated female from lab stock was randomly assigned to each dish, and time to first copulation, start of shaking behaviour, and end of copulation were recorded. All stock females were observed to have mated once (between 9 and 11 August) before being used in the experiment because females that have not mated may be less likely to discriminate between males, and female behaviours may influence copulation duration. If copulation had not begun within 4 h of the start of the period of observation, that individual was recorded as unmated.

To determine whether females discriminated against males that had undergone compensatory growth we used a choice design. A male from each treatment was placed in a Petri dish with a randomly selected stock female. An effort was made to eliminate size differences in paired males, and subsequent analysis determined that there was no significant difference in size of paired males. Female preference was evaluated by measuring the time to first copulation and probability of mating. Although we did not observe competitive interactions between males, they may well have occurred. Thus, any mating bias observed in this experiment could be attributed to either treatment effects on male competitive ability or female preference.

Results

Experiment 1

Patterns of growth and mortality, during and following resource limitation

We first analysed the entire data set on growth with repeated measures ANOVA with sex and food treatment as independent variables (Fig. 1, Table 1). This analysis demonstrated that there was a strong and significant ($P < 0.001$) treatment effects on growth in pronotom width, head width and mass. These treatment effects resulted from depressed growth early in development, followed by accelerated growth later in the treatment in the low food treatment (Fig. 1). There were also significant effects of sex on growth, as well as interactions between sex and the other factors in the model (Table 1). We had initially included family as a random factor in the RM-ANOVA, but found no significant interaction between family and food treatment, therefore, family was not included in further analyses. In using this repeated measures approach, we used moult number, rather than date, as the measure of time for two reasons. First, measures were made immediately following moult and secondly, as moult day varied within treatments, it was unsuitable for RM_ANOVA, which requires a variable that lacks variance. However, size at each subsequent moult cannot be used to assess growth rate (change in size over time) as the duration of each moult can and did vary between treatments. For a more detailed analysis of

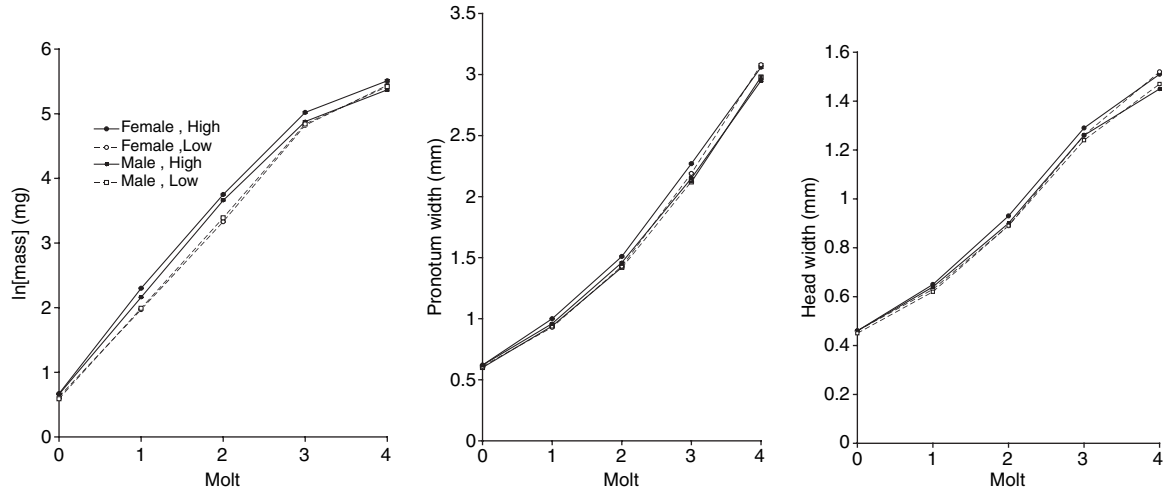

Fig. 1 Larval growth trajectories for male and female ladybird beetles reared at high or low food level in experiment 1. Three measures of body size (mass, pronotum width and head width) were taken at each moult including newly eclosed adults. Error bars indicate ±SE.

Table 1 Results of RM-ANOVA testing for effects of food level, sex and time (repeated measures at hatching and the four subsequent moults) on three measures of body size in *Harmonia axyridis* throughout development.

Variable	Source	d.f.	*F*	*P*
Between subjects				
ln[mass]	Food	1	44.7	< 0.0001
	Sex	1	3.92	0.05
	Food × Sex	1	6.10	0.0015
	Error	139		
Pronotum width	Food	1	13.5	0.0003
	Sex	1	33.5	< 0.0001
	Food × Sex	1	3.57	0.061
	Error	139		
Head width	Food	1	7.9	0.0057
	Sex	1	33.1	< 0.0001
	Food × Sex	1	2.6	0.11
	Error	139		
Within subjects				
ln[mass]	Moult	4,136	8353.0	< 0.0001
	Moult × Food	4,136	11.5	< 0.0001
	Moult × Sex	4,136	0.58	0.68
	Moult × Food × Sex	4,136	0.59	0.67
Pronotum width	Moult	4,136	13341	< 0.0001
	Moult × Food	4,136	9.26	< 0.0001
	Moult × Sex	4,136	7.97	< 0.0001
	Moult × Food × Sex	4,136	3.1	0.018
Head width	Moult	4,136	15606.3	< 0.0001
	Moult × Food	4,136	6.9	< 0.0001
	Moult × Sex	4,136	6.6	< 0.0001
	Moult × Food × Sex	4,136	1.1	0.37

Note that d.f. is reduced because some mass data were missing from three individuals due to moulting on the day of weighing.

growth patterns over the course of the experiment we analysed growth rate in each instar separately with ANOVA (Table 2).

We first contrasted mean size at hatching in the two treatment groups to determine if pretreatment sizes differed. Mass and head width at hatching did not differ between treatment groups (ANOVA: $F_{1,144} = 1.42$, $P = 0.16$ and $F_{1,144} = 1.46$, $P = 0.15$, respectively), although pronotum width was slightly smaller (2.3%) in the low food group ($F_{1.144} = 2.25$, $P = 0.03$). Therefore, we included initial pronotum width as a covariate in all subsequent analyses of pronotum growth rate and size. These analyses suggest that any subsequent differences in growth among treatment groups can be attributed to the treatment itself (see below).

Food treatment affected larval growth rate for each of the three size metrics in each of the four instars (Fig. 2, Table 2). For some instars and size metrics there were sex by treatment interactions. The general pattern was one where growth rates were lower in the low food treatment during larval instars one to three, and higher growth rates in the low food treatment during the final instar (Fig. 2, Table 2). The period of restricted diet lasted 8 days, which was equivalent to the mean egg to second instar duration in the low food treatment. This accounts for the reduced growth rate of low food larval in instars one and two. As expected, a comparison of the three size metrics at completion of the moult to third instar reveals that this food treatment was sufficient to cause a significant divergence in body size between the two treatments (ANOVA, $F_{1,144} = 16.0$, $P < 0.001$; $F_{1,144} = 38.0$, $P < 0.001$; $F_{1,144} = 18.0$, $P < 0.001$ for mass, pronotum and head width respectively; Fig. 1).

During the third instar, food ration was equivalent for both treatments, yet growth rates were still reduced in the low food treatment (Fig. 2, Table 2). Presumably this is a carryover effect of the lower food ration for these larvae in the preceding instar. The fact that growth rates were higher during the forth instar in the low food

Table 2. Effect of sex and food treatment on growth rates(change in size/duration of instar) during each instar and adult size for three measures of size(mass, pronotum width and head width).

			Growth rate											
			1 st instar		2nd instar		3rd instar		4th instar		Final size		Development time	
			F	*P*	*F*	*P*	*F*	*P*	*F*	*P*	*F*	*P*	*F*	*P*
ln[mass]	Food	1	66.3	< 0.0001	60.3	< 0.0001	0.25	0.62	7.19	0.0082	0.0003	0.99	161.5	< 0.0001
	Sex	1	1.48	0.23	1.48	0.23	0.0889	0.77	0.13	0.72	12.4	0.0006	3.12	0.08
	Food*Sex	1	1.32	0.25	0.11	0.74	0.13	0.72	0.23	0.63	4.09	0.045	0.69	0.41
	Error	142												
Pronotum width	Food	1	90.7	< 0.0001	88.7	< 0.0001	27.8	< 0.0001	8.03	0.005	1.25	0.27		
	Sex	1	2.41	0.12	2.81	0.096	0.088	0.77	0.085	0.77	18.2	< 0.001		
	Food*Sex	1	4.64	0.033	4.97	0.027	0.83	0.036	0.14	0.71	0.022	0.88		
	Error	142												
Head width	Food	1	77.5	< 0.0001	66.5	< 0.0001	35	< 0.0001	13.41	< 0.0004	2.59	0.11		
	Sex	1	4.98	0.027	1.63	0.2	1.14	0.29	1.86	0.17	22.45	< 0.0001		
	Food*Sex	1	1.38	0.24	4.05	0.46	1.05	0.31	0.17	0.68	0.66	0.42		
	Error	142												

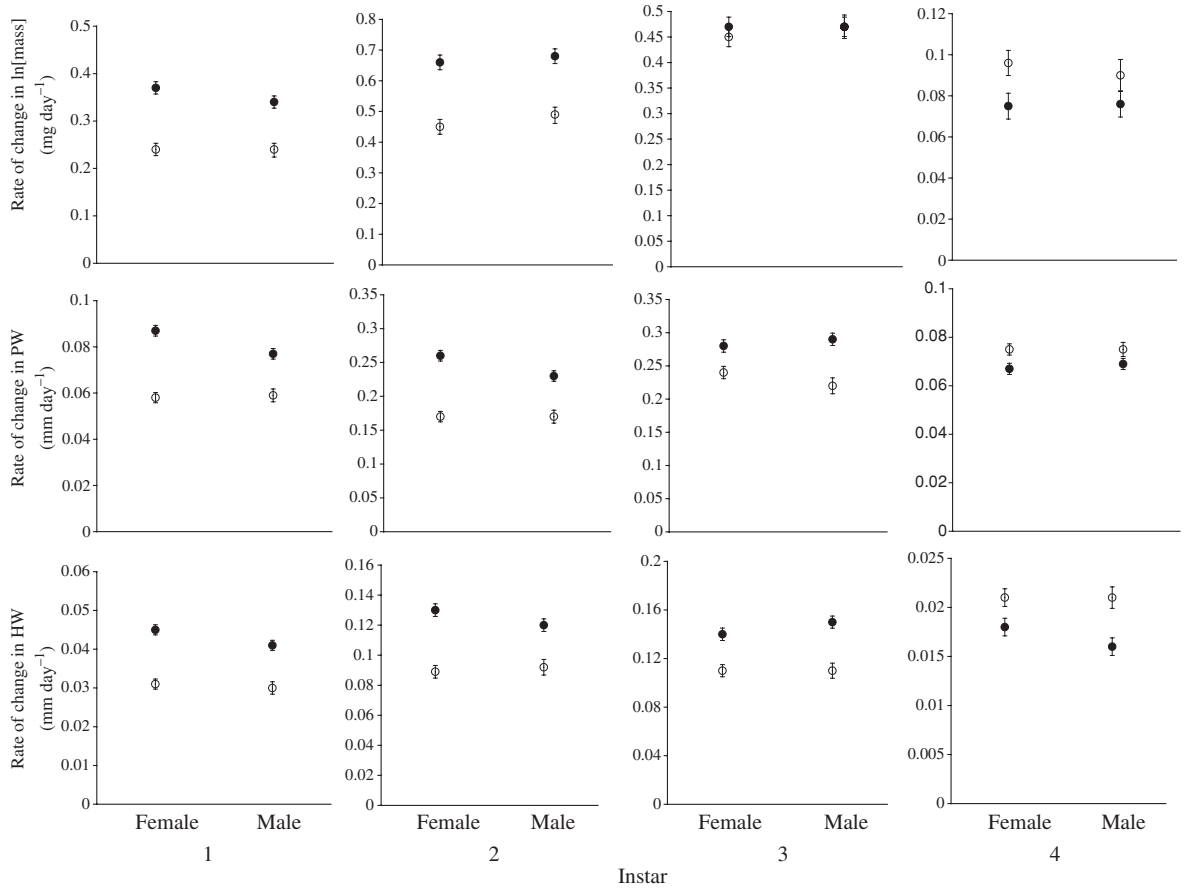

Fig. 2 Mean growth rates (change in size/duration of instar) for each instar of larvae reared at high (filled circles) or low (open circles) food in experiment 1. Size was measured as mass (top row), pronotum width (middle) and head width (bottom). Error bars indicate ±SE.

treatment is a clear demonstration of compensatory growth (Fig. 2, Table 2). It may well be that compensatory growth commenced during the third instar, but this effect was masked by the carryover effect of differences in food ration in the preceding instar.

Age and size at maturation

By the time of maturation to adult, the accelerated growth of the low food larvae observed in the forth instar was sufficient to completely compensate for the reduction in structural size observed in these larvae immediately following diet restriction. At maturation, both head width and pronotum size did not differ between treatments (Fig. 1, Table 2). Likewise, there was no treatment effect on mass, however there was an interaction between sex and food treatment on adult mass, with low food males having a greater mass and females a lower adult mass relative to high food beetles. Recall that there was a small difference between treatments in pronotum size at hatch; using hatchling pronotum width as a covariate (to account for the initial difference between treatments in this metric) in an ANCOVA did not affect the outcomes for either adult size or growth rate during the final instar and was thus not included in the analysis presented here ($P > 0.5$).

We asked whether the structural size–mass functions were affected by the treatment (ANCOVA on both pronotum width and head width, with ln[mass] as the covariate, and the factors sex and treatment, and their interactions). In addition to mass, sex affected structural size, but there was no significant effect of treatment or any interactions.

Recovery of mass and body size was achieved through a combination of the acceleration of growth rate (described above) and a prolonged development time. Low food individuals emerged 2.8 days later on average than those reared at high food, with no effect of sex or interaction between the two variables (Table 2). The observed delay was the result of prolonged development in the first three instars when growth was depressed, with no significant difference in duration of the final instar (ANOVA, $F_{1,145} = 0.31$, $P = 0.58$). Similarly, there

were no effects of food or sex on pupation duration (ANOVA, $F_{1,142} = 0.78$, $P = 0.44$ and $F_{1,142} = 0.41$, $P = 0.68$ respectively, and no interaction effect).

Lifetime fitness

There was some indication of a survival cost of the low food treatment (33% mortality in low food and 21% in high food; $\chi^2_1 = 3.65$, 198, $0.1 < P > 0.05$). Mortality rates were higher though not significantly so in both period of diet restriction (instars 1 + 2; 11% in low food, 9% in high food) and the period of compensatory growth (instars 3 + 4; 20% in low food, 9% in high food).

Seventy-eight females (37 high and 41 low) survived to emergence, with 68 of these females (33 high and 35 low) eventually producing eggs. Four individuals escaped during the course of the experiments and were therefore not included in the analysis. There was no treatment effect on probability of producing eggs ($\chi^2_1 = 1.58$, 77, $P > 0.05$), or on the teneral period (the number of days between emergence and the production of the first clutch; ANOVA: $F_{1,63} = 0.03$, $P = 0.87$). There was no relationship between maternal body mass at emergence and total egg ($R^2 = 0.002$, $t_{60} = 032$, $P = 0.75$) or larval production ($R^2 = 0.013$, $t_{60} = 0.87$, $P = 0.39$) and therefore we dropped body mass for the remaining analyses.

Total egg production and larval production did not differ between treatments (ANOVA: $F_{1,63} = 0.33$, $P = 0.57$ and $F_{1,63} = 0.16$, $P = 0.69$); Fig. 3). Ladybird beetles reared at high food laid an average of 554 ± 49 eggs whereas the compensating treatment produced 515 ± 47. Mean hatching success was 28 ± 4.5% in the high food treatment and 32 ± 4.4% in the low food ($F_{1,63} = 0.41$, $P = 0.52$). Early life fecundity (first 25 days of eclosion) was also unaffected by treatment (*t*-test: $F_{1,63} = 0.83$, $P = 0.41$ and $F_{1,63} = 0.05$, $P = 0.83$ respectively).

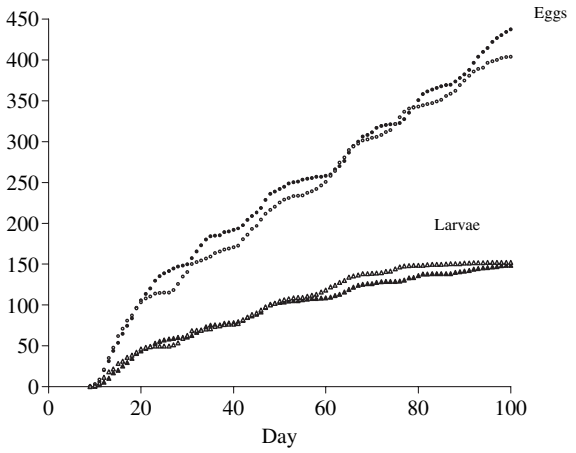

Fig. 3 Cumulative production of eggs (circles) and larvae (triangles) over time. Each point represents the mean daily production by treatment for all fertile individuals. Solid markers represent the high food treatment and open markers the low food treatment.

Although there was no difference in the probability of surviving to 100 days past emergence ($\chi^2_1 = 0.02$, $P > 0.05$), we found that adults from the low food treatment were less resistant to a period of starvation imposed at this time. At the time of the starvation treatment, there were 26 and 27 adults alive in the low and high food treatments. Of these remaining beetles, the mean time to death was 9.4 ± 0.63 days for high food beetles and 7.6 ± 0.60 days for low food beetles, and this difference was significant (ANOVA $F_{1,54} = 4.03$, $P < 0.05$). There was no difference in the number of eggs produced after feeding ended (high food: 2.4 ± 0.8, low food: 1.9 ± 0.8, $F_{1,52} = 0.21$, $P = 0.65$) and no eggs hatched in either treatment.

Experiment 2

Larval growth, mortality, and age and size at maturity

We again analysed the larval growth trajectories (between the end of the treatment and emergence) with repeated measures ANOVA (Table 3). This analysis demonstrated that there was again a strong and significant ($P < 0.001$) treatment effect on growth in mass and pronotum and head width during the post-treatment period (Fig. 4). We analysed body size in greater detail using ANOVA, and found that all three measures of size were again reduced by the food restriction treatment (Table 4). As in experiment 1, low food larvae had a longer development time (19 days vs. 18.1 days; $F_{1,194} = 75.7$, $P < 0.0001$), but in this case individuals in the low food treatment actually emerged at larger body size than those reared at high food (Table 4, Fig. 5), indicating that overcompensation for poor resource availability occurred, although the effect on mass was not significant. An ANCOVA on both pronotum width and head width, with ln[mass] as the covariate, and sex and treatment as factors, indicated that in addition to mass, both sex and treatment affected structural size, and there were some interactions with sex. For our purposes the important element is a consistent effect of treatment. Treatment effects of both head width ($F_{1,186} = 38.9$, $P < 0.0001$) and pronotom width ($F_{1,186} = 13.0$, $P < 0.0001$) were significant and this resulted from a larger structural size for any given mass in the low food that than the high.

A total of 100 males survived to emergence (58 and 42 for control and low food treatments respectively), with a greater proportion of larvae reared at high food surviving to emergence than those reared at low food (respectively; $\chi^2_1 = 5.66$, $P = 0.017$). Mortality rates of low food larvae were significantly greater than high food larvae during both the period of diet restriction (instars 1 + 2; 7% in low food, 3% in high food) and the subsequent period when compensatory growth occurred (instars 3 + 4; 11% in low food, 5% in high food), (instars 1 + 2, $\chi^2_1 = 5.85$, $P < 0.05$; instars 3 + 4, $\chi^2_1 = 3.85$, $P < 0.05$).

Table 3 Results of RM-ANOVA testing for effects of food level, sex and time on three measures of size (mass, pronotum width and head width) in experiment 2 following the food manipulation on the second moult and at emergence.

Variable	Source	d.f.	*F*	*P*
Between subjects				
ln[mass]	Food	1	172.9	< 0.0001
	Sex	1	4.95	0.027
	Food × Sex	1	1.85	0.18
	Error	187		
Pronotum width	Food	1	0.01	0.93
	Sex	1	3.6	0.059
	Food × Sex	1	0.09	0.76
	Error	187		
Head width	Food	1	26.6	< 0.0001
	Sex	1	12.0	0.0007
	Food × Sex	1	4.3	0.039
	Error	187		
Within subjects				
ln[mass]	Moult	1, 184	18567.4	< 0.0001
	Moult × Food	1, 184	223.4	< 0.0001
	Moult × Sex	1, 184	5.30	0.022
	Moult × Food × Sex	1, 184	1.32	0.25
Pronotum width	Moult	1, 184	9869.4	< 0.0001
	Moult × Food	1, 184	80.3	< 0.0001
	Moult × Sex	1, 184	0.3	0.57
	Moult × Food × Sex	1, 184	0.09	0.76
Head width	Moult	1, 184	5073.1	< 0.0001
	Moult × Food	1, 184	72.2	< 0.0001
	Moult × Sex	1, 184	24.5	< 0.0001
	Moult × Food × Sex	1, 184	11.0	0.0011

Table 4 ANOVAS on mass, pronotum width and head width of *H. axyridis* post-treatment larvae and newly emerged adults. Analyses test the effect of sex and a food manipulation lasting for the first 6 days of development in experiment 2.

			Size after treatment		Final size	
Size metric	Source	d.f.	*F*	*P*	*F*	*P*
Ln[mass]	Trt	1	233.3	< 0.0001	3.48	0.06
	Sex	1	0.0003	0.99	32.5	< 0.0001
	Trt × Sex	1	1.87	0.17	0.061	0.81
	Error	194				
Pronotum width	Trt	1	13.7	0.0003	24.40	< 0.0001
	Sex	1	2.4	0.12	1.64	0.20
	Trt × Sex	1	2.0	0.16	0.00	0.99
	Error	194				
Head width	Trt	1	70.6	< 0.0001	51.2	< 0.0001
	Sex	1	6.7	0.011	22.3	< 0.0001
	Trt × Sex	1	0.55	0.46	9.15	0.0028
	Error	194				

Larval movement rates

There was a strong effect of food treatment and activity level during the period of resource depression, but not during compensation (Fig. 6). Mean velocity of movement was accelerated in the low food treatment, although total distance moved was not affected. Following restoration of rations, neither distance nor velocity differed between treatments, although both measures of activity declined among all larvae (RM-ANOVA: between-subjects for velocity $F_1, = 7.32$, $P = 0.0079$; within-subjects: Time $F_{1,107} = 28.7$, $P < 0.0001$, Time × Treatment $F_{1,107} = 2.42$, $P = 0.12$. Between factors for distance: $F_{1,107} = 0.74$, $P = 0.39$; within-subjects: Time $F_{1,107} = 14.51$, $P < 0.0001$, Time × Treatment $F_{1,107} = 0.38$, $P = 0.54$).

Male mating behaviour

A total of 54 males from the control food and 38 males from the low food survived for at least one mating trial. Among these males, there was no significant effect of food treatment on probability of mating in these trials (high food: 50/54 mated; low food 33/38 mated, $\chi^2 = 0.84$, $P > 0.9$). Given that adult size differed between treatment groups, size was included as a covariate in the analyses using a principle component axis combining

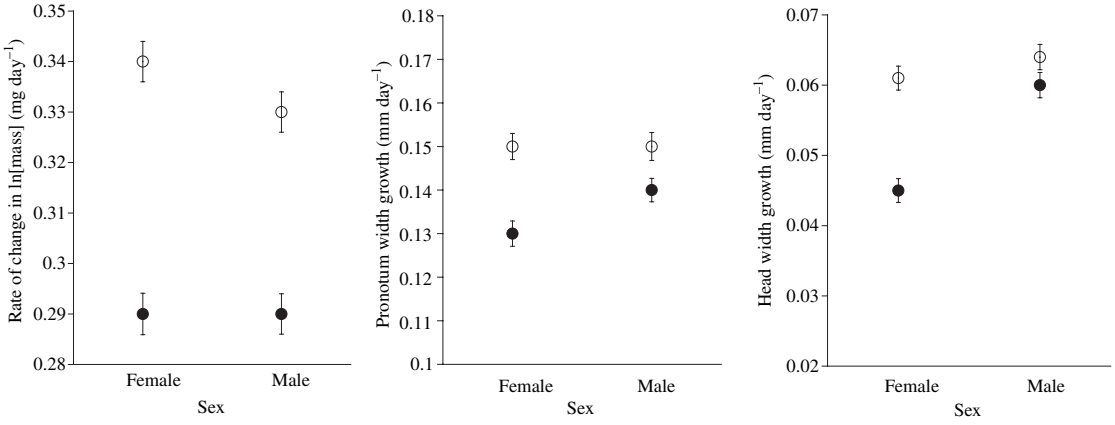

Fig. 4 Mean growth rates of mass, pronotum width, and head width of high (filled circles) and low food (open circles) larvae at the end of the food manipulation (day 6) and at eclosion, during the period when compensatory growth occurred in experiment 2. Error bars indicate ±SE.

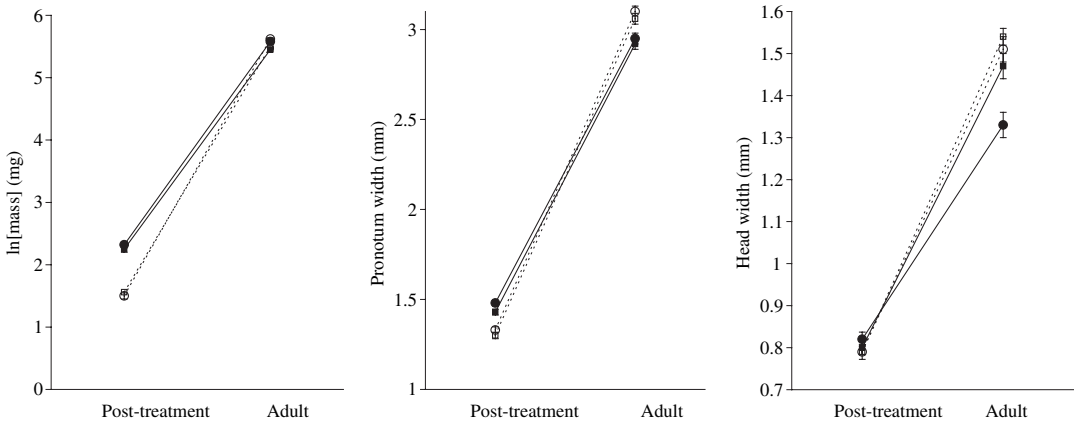

Fig. 5 Effect of sex and food treatment on body size in experiment 2. Measures of mass, pronotum width and head width were taken for larvae after the food treatment and of adults. Females and males are represented by circles and squares, respectively at high (filled symbols) and low food (open symbols). Error bars indicate ±SE.

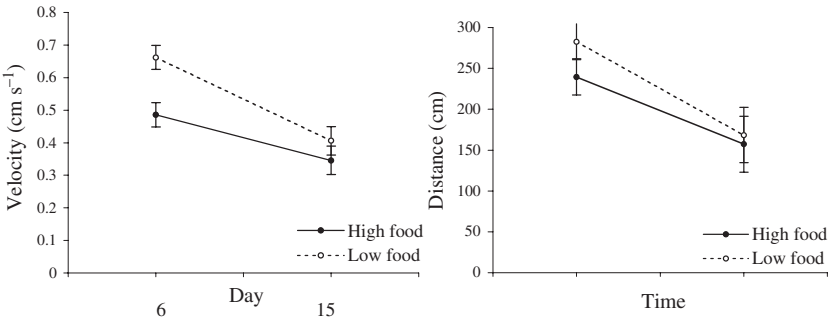

Fig. 6 Effect of food treatment on larval movement, velocity and distance travelled over a 15-min observation period on day 6 (during the treatment period) and on day 15 (during the period of compensatory growth).

mass, head width and pronotum width (PC1). Food treatment and adult size had no effect on time to first copulation (ANOVA, $F_{1,82} = 0.72$, $P = 0.40$ and $F_{1,82} = 2.94$, $P = 0.09$) or time to commencement of male shaking behaviour ($F_{1,82} = 0.82$, $P = 0.37$ and $F_{1,82} = 0.0027$, $P = 0.96$). Male shaking duration was longer in the high food treatment ($F_{1,82} = 6.03$, $P = 0.016$) but this effect was apparently due to an interaction between treatment and PC1, with a few individuals of small body size having a strong effect on the relationship (treatment by size interaction: $F_{1,81} = 4.94$, $P = 0.03$). If these three individuals are removed, there is no longer a significant effect of either treatment or body size on shaking duration ($P > 0.7$).

Mate choice experiment

Mating occurred in 37 of 38 pairs. Age and size had no effect on the probability of mating and was therefore not included in further analyses [size (PC1): $F_{1,70} = 0.0012$, $P = 0.97$; age: $F_{1,70} = 1.73$, $P = 0.19$]. Females demonstrated no preference for males in the high food treatment ($\chi^2_1 = 0.054$, $P = 0.82$), nor were there any treatment effects on time to copulation, preshaking duration or shaking time ($P > 0.05$ for all behaviours).

Discussion

We have found that following a substantial period of food deprivation early in life, *H. axyridis* was able to completely compensate for lost size by the time of maturation. There were short and long-term costs to both growth restriction and acceleration. Emergence was delayed, juvenile mortality was increased and potentially costly increases in activity rate were observed. Complete compensation for body size allowed us to assess, for the first time, long-term costs to fitness without the confounding effects of differences in body size. Our results demonstrate remarkably few fitness consequences to adults of growth restriction and subsequent compensatory growth of juveniles. Lifetime female fecundity, male mating success and longevity were unaffected, however we did find that compensated adults tend to die more quickly when faced with starvation late in life. We discuss these results in light of prior work below.

Compensatory growth

Compensatory growth is well known in vertebrates, although it is almost always associated with elevated foraging effort and hyperphagia (Metcalfe & Monaghan, 2001; Ali *et al.*, 2003). The current study represents one of the few examples of compensatory growth in an invertebrate and one that did not involve hyperphagia (Tammaru *et al.*, 2004; Dmitriew & Rowe, 2005; Stoks *et al.*, 2006). We have found that significantly depressed growth for the first two (of four) instars in this beetle can be completely offset by compensatory growth in the forth instar, leading to mass and structural size at eclosion that was at least that of larvae that had never experienced depressed growth (Fig. 1 and Table 2). Although the switch from low to high rations occurred at the end of the second instar, in experiment 1 growth in structural size continued to be depressed in low food third instars, presumably as a carry-over effect of low food in the second instar (Fig. 2 and Table 2). An earlier study of the damselfly *Ischnura verticalis*, similarly reported complete compensation for size following a substantial period of depressed growth (Dmitriew & Rowe, 2005). Two other studies provide evidence for compensatory growth in invertebrates, but in both cases the period of depressed growth was very brief and was imposed late in juvenile life (the final instar; Tammaru *et al.*, 2004; Stoks *et al.*, 2006),

There were interesting differences between Experiments 1 and 2 in treatment effects on mass and structural size. In both experiments body mass at eclosion in larvae that had undergone compensatory growth was indistinguishable from body mass of those that had not, thus compensation was complete. Likewise, compensation for structural size (head and pronotum width) at eclosion was complete in Experiment 1, but in Experiment 2, structural size was actually greater in compensated larvae. This phenomenon of over compensation has been observed rarely in vertebrates, but is poorly understood (Metcalfe & Monaghan, 2001; Ali *et al.*, 2003; Mangel & Munch, 2005). The only difference in the low food treatment in Experiment 2, relative to Experiment 1, was a slightly reduced food deprivation period (6 vs. 8 days). In at least one theoretical treatment of compensatory growth, overcompensation is predicted to be most prevalent when food deprivation is imposed relatively early in life and for a relatively brief period (Mangel & Munch, 2005).

The different responses of mass and structural size in experiment 2 suggests that allocation to these to size components was effected by the treatment. This was reflected in a treatment effect on mass corrected head and pronotum width. Similar effects on allocation have been reported in a variety of species and components of size. In fish, the tendency is to see compensation for body condition before investment in skeletal growth prior to the onset of winter (e.g. Metcalfe *et al.*, 2002; Johnsson & Bohlin, 2006). Dmitriew & Rowe (2005) found complete compensation in structural size, but incomplete compensation in mass in their study of a damselfly. They interpreted this result as a prioritizing of structural size over mass at eclosion, as there is substantial mass gain in adult damselflies, which may be limited by structural size (Anholt *et al.*, 1991; Richardson & Baker, 1997). This may well be happening in these beetles, yet the fact of overcompensation remains difficult to explain.

Prereproductive costs

Our data suggests that both the periods of depressed growth and subsequent compensatory growth entail prereproductive costs. Larvae in the low food treatment were more likely to die prior to maturation and this was associated with both the periods of depressed and compensatory growth itself. It is typical that accelerated growth rates are associated with higher predation rates (e.g. Anholt & Werner, 1998; Gotthard, 2000), but this was not the cause of mortality in our experiments because there were neither predators nor cannibals present. Increased juvenile mortality in the absence of predators has also been shown in faster growing, time-constrained dung flies, as well as within populations of butterflies with higher intrinsic growth rates (Gotthard *et al.*, 1994; Blanckenhorn, 1998). We also assayed activity rate during the juvenile period, because of its potential association with predation mortality in the field. These analyses indicated that one measure of activity (velocity) was elevated during the period of depressed growth, but not during subsequent compensatory growth. It is quite possible that, in the presence of predators, this may elevate mortality.

Compensation for adult body size was accompanied by a prolonged development time in *H. axyridis*. The trade-off between reaching a large adult size and maturing faster has been well studied (Rowe & Ludwig, 1991; Abrams *et al.*, 1996; Nylin & Gotthard, 1998). The outcome of this trade-off seems to depend on the relative importance of maturing rapidly (reduced generation time) or on schedule in seasonal environments vs. the benefits of attaining a large body size (e.g. Rowe & Ludwig, 1991; Johansson & Rowe, 1999, Taborsky *et al.*, 2003). When models include flexible growth rate (and associated costs of rapid growth), the picture becomes more complex, particularly when both short-term and long-term costs of accelerating growth are considered (e.g. Abrams *et al.*, 1996; Yearsley *et al.*, 2004; Mangel & Munch, 2005). The delay in pupation observed in the current study is potentially very costly, and not just in terms of generation time. There is a substantial incidence of cannibalism among larval *H. axyridis*, so fast growth is doubly beneficial; in addition to reducing the probability of being cannibalized by larger individuals, it increases access to an alternative prey (conspecifics) with substantial nutritional gains to be had (Pervez *et al.*, 2006).

Maturation is a refuge from this source of mortality as adults are not at risk of cannibalism.

Compensatory growth and lifetime fitness

We predicted that the effects of poor early conditions would carry over from the larval stage to affect lifetime fitness, with the expectation that compensatory growth merely reduces or masks costs arising from poor conditions early in life. In experiment 1, females reared at low food followed by compensatory growth were less able to resist a period of starvation occurring late in life, suggesting that they had a reduced capacity to survive stress. A similar response was shown in butterflies where individuals from populations with relatively high intrinsic growth rate lost weight faster than those from slow-growing populations during a period of starvation, possibly reflecting reduced investment in energy reserves (Gotthard, 1994). Although we found no effect on body condition (mass/body size) in experiment 1, we are currently performing a more detailed analysis of body composition in ladybird beetles undergoing resource limitation and compensatory growth. Alternatively reduced survivorship could be a consequence of accelerated senescence in low food individuals, the result of a hypothesized trade-off between maintenance functions such as protein turnover and growth (e.g. Samuels & Baracos, 1995), or because low quality nutrition during development reduces antioxidant activity even when conditions later improve (Blount *et al.*, 2003). Both oxidative damage and reduced protein turnover are believed to contribute to cellular senescence and aging (e.g. Bokov *et al.*, 2004). These effects may only be evident under stressful conditions, as evidenced by the fact that mortality was not affected over the first 100 days after emergence, during which larvae received a regular ration of aphids.

We found no effects on female reproductive traits, nor were there effects on male mating behaviours or female preferences. Overall hatching rates were low, but both groups fell within the range previously reported for this species (Bazzocchi *et al.*, 2004). In the current study, the period of resource limitation may have been too brief, or the resource limitation too mild, to cause significant detrimental effects. However, we believe that this is unlikely given that mortality was elevated in the low food treatment, and preliminary experiments demonstrated that increasing the duration of the period of resource limitation by even 2 days resulted in a large and significant reduction in adult size and mass (C. Dmitriew, unpublished data). It has also been suggested that periods of low growth during development may actually cause overall growth efficiency to increase as the maintenance costs for a small body are lower (Skalski *et al.*, 2005); therefore, it may not be the case that resources must necessarily be diverted from other traits or functions to permit growth acceleration later in life. The fact that rearing conditions in this experiment were relatively benign may also have caused costs to be less evident. For example, larvae may be selected to forage at submaximal rates in order to minimize predation risk; costs of a compensatory increase in foraging effort following growth restriction would therefore not be apparent in the lab. This is unlikely for two reasons; first, during the period of compensation we found no differences in activity rates (velocity and distance travelled) and secondly, because there are few major predators of adult *H. axyridis* in North America (Koch, 2003). Previous studies that have found long-term costs of poor conditions during development were conducted in natural or semi-natural conditions (Birkhead *et al.*, 1999; De Block & Stoks, 2005; but see Royle *et al.*, 2005).

Finally, individuals fed near *ad libitum* may be foraging at a rate that is unusually high, and therefore not representative of natural or optimal rates. *Harmonia axyridis* presumably evolved against a backdrop of highly variable resource availability. As such, it may be the case that continuous access to large amounts of food may not, in fact, maximize fitness. If so, then the lack of an observed cost of compensatory growth may reflect the low fitness of 'control' high food adults as much as it does a lack of costs to fitness in low food adults. It is well known from studies of humans and other mammalian model systems that unlimited diet leads to obesity and cardiovascular disease, and has recently been shown to reduce both longevity and fecundity in mice (Johnston *et al.*, 2006). These effects may be particularly strong if the *ad libitum* diet is preceded by resource limitation during development (Roseboom *et al.*, 2000; Ozanne & Hales, 2005). This is important because tests for costs of compensatory growth, this one included, assume that fitness is maximized at growth achieved under continuous abundant food. A profitable approach for future studies would be to explore a range of food levels (including *ad libitum*) to first identify the ration that maximizes fitness, and use this ration as the control.

Summary

In conclusion, we have demonstrated that ladybird beetles exposed to a substantial period of resource restriction early in development fully compensated by increasing growth rate. Growth restriction and subsequent compensation reduced juvenile survival, delayed maturation and reduced starvation resistance late in life. Yet, there were no effects on longevity over the first 100 days, female fecundity or male mating success. To answer our original question: does compensatory growth mask long-term costs of early resource limitation? The answer appears to be largely affirmative. Future experiments should likewise focus on fitness. These experiments would be improvements if they first determine whether 'control' food treatments actually maximize fitness, and fitness should be assayed in more realistic settings.

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