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LETTER

Juvenile compensatory growth has negative consequences for reproduction in Trinidadian guppies (*Poecilia reticulata*)

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Abstract

Compensatory or 'catch-up' growth may be an adaptive mechanism that buffers the growth trajectory of young organisms from deviations caused by reduced food availability. Theory generally assumes that rapid juvenile compensatory growth impacts reproduction only through its positive effects on age and size at maturation, but potential reproductive costs to juvenile compensatory growth remain virtually unexplored. We used a food manipulation experiment to examine the reproductive consequences of compensatory growth in Trinidadian guppies (*Poecilia reticulata*). Compensatory growth did not affect adult growth rates, litter production rates or investment in offspring size. However, compensatory growth had negative effects on litter size, independent of the effects of female body length, resulting in a 20% decline in offspring production. We discuss potential mechanisms behind this observed cost to reproduction.

Keywords

Allocation, compensatory growth, fitness, life history, litter size, phenotypic plasticity, *Poecilia reticulata,* reproduction, resource availability, trade-off.

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INTRODUCTION

Young organisms often face considerable challenges in acquiring resources because of the heightened mortality risks, increased competitive pressures and reduced physical capabilities associated with their small body size (Conover & Schultz 1997). These tough circumstances can be exacerbated if young are born during a time of low resources, if they are relatively small because of reduced parental provisioning, or if they are born late in the season (Lindstrom 1999; Metcalfe & Monaghan 2001). Reduced food intake during early growth and development can then have negative consequences for fitness. Low resources early in life can lead to slower rates of growth and development and thereby to maturity at an older age and smaller size. Delayed maturation decreases fitness because it increases generation time and can decrease the reproductive lifespan (Roff 1992). Smaller body size at maturation can also lead to reduced fitness because of the general positive effect that body size has on survival and reproductive success (Roff 1992). Given these potential fitness costs, selection might be expected to favour mechanisms that mitigate the negative effects of early setbacks in size at age, provided that the costs of such responses are not too high (Metcalfe & Monaghan 2001).

Compensatory or 'catch-up' growth is a common form of growth plasticity in which growth is accelerated to a rate above that of normal routine growth in response to an increase in resources following a period of growth restriction (Metcalfe & Monaghan 2001). Compensation can occur through recoupment of energy reserves and through increased investment in structural growth and thereby lead to partial, full or even overcompensation in energy reserves and body size (Ali *et al.* 2003; Metcalfe & Monaghan 2003). Compensatory growth is commonly financed through a hyperphagic response to an increase in resource levels (Ali *et al.* 2003), but it can also be achieved through changes in allocation rules that direct energy towards growth at the expense of other functions and activities (Sogard & Olla 2002).

Compensatory growth may be an adaptive mechanism that buffers the growth trajectory of young organisms from deviations caused by reduced resources early in life (Metcalfe & Monaghan 2001). However, compensatory growth and rapid growth in general are known to have negative consequences for survival. When increased growth

rates are financed by increased food intake, higher foraging rates can lead to increased risk of predation in the short-term (Lankford *et al.* 2001). Compensatory growth can also increase mortality risk and reduce lifespan through its long-term detrimental effects on disease resistance, starvation resistance, oxidative stress and over-winter mortality (Forsen *et al.* 2004; Dmitriew & Rowe 2005; Johnsson & Bohlin 2006; De Block & Stoks 2008) These survival costs are, in turn, thought to shape the evolution of the compensatory growth response (Yearsley *et al.* 2004; Mangel & Munch 2005).

Although great emphasis has been placed on understanding survival costs to compensatory growth, its impact on reproduction, an equally important component of fitness, remains virtually unexplored (but see Morgan & Metcalfe 2001; Dmitriew & Rowe 2007). Current theory generally assumes that compensatory growth impacts reproduction only through its positive effects on age and size at maturation (Abrams *et al.* 1996; Ali *et al.* 2003; Metcalfe & Monaghan 2003; Yearsley *et al.* 2004; but see Lindstrom *et al.* 2005; Mangel & Munch 2005 for exceptions). However, many of the developmental and physiological mechanisms thought to underlie survival costs of compensatory growth may also have negative impacts on reproduction. For example, the increases in metabolic demands (Stoks *et al.* 2006) and reductions in cognitive function (Fisher *et al.* 2006), energy reserves (Stoks *et al.* 2006), and physical performance (Royle *et al.* 2006) associated with compensatory growth could negatively impact the reproductive success of the organism as well as its survival. Alternatively, reproductive costs to juvenile compensatory growth could arise as an indirect result of a trade-off between compensatory growth and some other trait closely tied to reproduction (Reznick 1985). For example, if juvenile compensatory growth reduces adult survival, then adults may respond by investing more energy in survival at the expense of current reproduction. If compensatory growth has negative consequences for later reproduction, then these costs have the potential to influence how compensatory and other plastic growth strategies evolve.

We examined the reproductive consequences of juvenile compensatory growth in Trinidadian guppies (*Poecilia reticulata*) by comparing different components of reproduction between guppies that were experimentally manipulated to undergo either routine or compensatory growth as juveniles. Specifically, we examined the effects of juvenile growth history on adult rates of litter production and investment in offspring size and offspring number. Because allocation of energy to adult growth comes at the expense of investment in reproduction, we also examined treatment differences in adult growth rates. In addition, we looked at the effects of compensatory growth and subsequent reproduction on female energy reserves. We predicted that if there is a reproductive cost to compensatory growth, then guppies that underwent compensatory growth as juveniles would exhibit slower rates of litter production, reduced adult growth rates, fewer offspring per litter and/or smaller, lower quality offspring relative to individuals that exhibited normal routine growth rates during the juvenile stage of life.

METHODS

Guppies are small poeciliid fishes that have internal fertilization and bear live young. Young develop rapidly and can give birth to their first litter as early as 60–70 days of age (Reznick & Bryga 1996). They initiate a new litter of young during or shortly after the birth of the prior litter and can give birth at regular intervals of *c.* 25 days. Energy used to fuel each litter is taken in part from the female's somatic reserves as well as from energy accrued during the time of yolking (Reznick & Yang 1993). Females used in the assay were offspring of laboratory born descendents of fish collected in 2003 from the Turure River in Trinidad, West Indies.

Juvenile growth

We initially reared offspring from a given litter together at densities of 8–10 individuals in 8 liter aquaria. Offspring were from 22 litters, each litter from a different female. Fish were fed a diet of liver paste in the morning and *Artemia* brine shrimp nauplii in the evening. Juveniles in group tanks were fed 2 μL per individual in the morning and 2 μL per individual in the evening, amounting to roughly 24 J per individual per day. At age 14 days, we randomly chose two sibling females of similar body mass and length from each of the 22 litters. Females were sexed based on the triangular pattern of melanophore development in the abdominal region that is otherwise absent in males (Reznick 1990). Females were assigned to their own 8 liter aquaria and to one of two food treatments. One sibling per litter was fed an *ad libitum* ration throughout the experiment, hereafter control fish (n = 22). The food ration of the other sibling was reduced by half for the next 2 weeks, hereafter referred to as the 'growth restriction period', from age 14 to 28 days and then fed *ad libitum* throughout the remainder of the experiment, hereafter experimental fish (n = 22). The food level used for experimental fish during the growth restriction period (1 μL in the morning and 1 μL in the evening) was pre-determined to allow some growth and maintain fat reserves comparable with those observed in the wild (Reznick *et al.* 2001). Food levels were otherwise adjusted each week to compensate for changes in female size and maintain rations pre-determined to be at an *ad libitum* level (Reznick 1980). *Ad libitum* food rations were always only partially consumed and uneaten food was removed daily. We

used a randomized block design to control for variation among individuals in their pedigree and microenvironment experienced in the laboratory: sister pairs were placed next to one another in a randomly assigned position in the laboratory.

We measured standard length and mass each week during the juvenile stage. Individuals were anaesthetized in a solution of neutrally buffered MS-222. Each fish was measured under a dissecting scope using digital calipers and then weighed using an Ohaus electronic balance (Pine Brook, NJ, USA). Aquaria were cleaned every 2 weeks with a partial water change to remove build up of nitrogenous waste.

Female reproduction

Females were mated overnight each week beginning at age 35 days, approximately a week before the earliest estimated age at maturation in *ad libitum* fed guppies (Reznick 1980). Females were mated overnight once a week with males randomly selected from the same stock of stud males. Aquaria were checked twice daily for newborn offspring. Offspring were collected when born, killed with a fatal overdose of MS-222 and preserved in 5% formalin. Female body length and mass were measured on the day each litter was born. Females were euthanized and preserved in the same manner after giving birth to their fourth litter.

We calculated litter size (number of offspring per litter), the mean offspring lean dry mass and the mean extractable fat content per offspring for the first four litters of each female as well as the length of the three interlitter intervals between the four successive litters. Interlitter interval was calculated as the number of days between two consecutive litters. To determine the mean offspring lean dry mass, we first dried each litter overnight in an oven at 60 °C. We then weighed the dry mass of each litter and submerged it in anhydrous ether to remove triglycerides until it reached a constant lean dry weight (Reznick & Endler 1982). The mean lean dry mass per offspring was then calculated as the total lean dry mass divided by the number of young in the litter. The difference between the dry weight and lean weight of a litter represents the fat content of that litter, which when divided by the number of offspring in a given litter, gives the mean extractable fat content (in mg) per offspring. Offspring size as well as body condition (fat reserves) are indices of the quality of the offspring since they are both known to influence offspring growth and survival (Berkeley *et al.* 2004; Bashey 2008).

Juvenile and adult body condition

We examined treatment differences in fat reserves in juveniles at the end of the growth restriction period and in adults at the birth of their fourth litter. To assess juvenile fat content, we reared 14 additional pairs of female siblings, each pair from a different mother, under the same conditions as the treatments described above. One sibling of each pair was assigned to the control and the other to the experimental treatment. These siblings were raised until the end of the 2-week growth restriction period (28 days of age), killed, preserved and analysed for fat content, as described above. Fat content of females preserved at the birth of their fourth litter was also analysed using these same procedures.

Statistical analysis

We used a linear mixed model approach to examine the effects of treatment on juvenile growth and adult reproductive traits. For analyses where the dependent variable was measured on only one occasion for each individual, we included block as a random effect to account for variation in pedigree and microenvironment in the laboratory. For analyses of longitudinal data, we accounted for the random effects of female identity – her intercept and slope – and thereby controlled for the non-independence of repeated measures on the same female (Singer & Willett 2003; Bolker *et al.* 2009). We used a model selection approach whereby we tested hypotheses about the contribution of fixed effects in nested models. We started with a full model for each trait and used backward model selection, sequentially eliminating terms with the lowest F-values until all terms in the model were significant. In general, a fixed factor or its interaction with another predictor was dropped from the model when its effect was not significant. However, because we were testing hypotheses about the effect of treatment on the different dependent variables, the main effect of treatment was always retained in the final model. We compared alternative models produced by maximum likelihood estimation, but report parameter estimates for the final model produced by restricted maximum likelihood estimation. If one of the random effects was not estimable due to model overparameterization, we excluded that effect.

Juvenile and adult growth

We first tested for differences in body size of experimental and control fish at the beginning and at the end of the 2-week growth restriction period (age 14 and 28 days). We also examined treatment differences in growth during the growth restriction period. We then tested for treatment differences in juvenile growth in body mass and length once experimental fish returned to *ad libitum* food conditions. Because experimental and control fish were different sizes at the end of the growth restriction period and because growth is often size-dependent, we compared growth rates among treatment groups using a 'size/time' approach whereby growth trajectories of experimental fish during the compensation phase were 'slid back in time' to a common

starting size with control fish (Mangel & Munch 2005; Nicieza & Alvarez 2009). Because experimental fish grew during the growth restriction period, we could not simply slide them back in time to the beginning of the growth restriction period when the control fish were 14 days old. So, we compared body sizes (mass and length) of the control fish at ages 15, 16 and up to 20 days with those of the experimental fish at age 28 days to find the age at which the control fish were the same size as the experimental fish. The mean daily growth rates in mass and length of the control fish calculated for the week spanning ages 14–21 days were used to estimate their mass and length, respectively, on each of these days. When the control fish were 17 days old, they did not differ in body mass ($F_{1,41} = 1.33$, $P = 0.26$) or length ($F_{1,41} = 3.49$, $P = 0.07$) from experimental fish at age 28 days. Thus, experimental fish were slid back 11 days to a common starting size with the control fish at age 17 days. We analysed growth across four ages (controls: 17, 21, 28 and 35 days of age; experimental fish: 28, 35, 42 and 49 days of age). Growth was analysed until experimental fish were 49 days old, the age when our back-calculations indicated most of these individuals were mature. Finally, we examined whether experimental fish caught up in body size to control fish by the time they were 49 days old.

We then tested for treatment differences in adult growth in body mass and length. The yolking of eggs occurs at defined intervals in guppies and requires energy otherwise used for growth (Reznick & Yang 1993), so weekly changes in adult body size can depend on stage of pregnancy (S. K. Auer, unpublished data). We therefore controlled for stage of pregnancy by analyzing growth rates between litters, i.e. between the age at which a female yolked the eggs for the first litter (i.e. at sexual maturity) and each of the ages at which she yolked a subsequent litter. Because guppies are livebearers, sexual maturity in females is only visibly apparent at the birth of the first litter. However, age at maturation can be approximated by subtracting the length of one gestation period from the age at first parturition (Reznick 1982). To estimate age at maturation, we first calculated the interval between the first and second litter for each individual, and then subtracted that interlitter interval (the gestation period) from the age that individual first gave birth. We then used our weekly measurements of body size up until first parturition to estimate body size at the estimated age at maturation for each individual. For subsequent litters, the age and size at yolking corresponded to measurements taken at the birth of the previous litter. Adult growth was calculated as (body $\text{size}_{\text{litter } i+1}$ − body $\text{size}_{\text{litter } i}$)/interlitter interval.

Female reproduction

We first tested individually for differences in age and size at sexual maturation between treatment groups. We then tested for treatment differences in four reproductive traits (interlitter interval, mean offspring lean dry mass, mean extractable fat per offspring and litter size) and how those traits changed with litter number and female body length. Because the effect of treatment on litter size changed with adult body length, we re-centered the intercept in the final model on litter number and the grand mean female length for each litter and tested for treatment differences in litter size at each of the four litters (*sensu* Singer & Willett 2003; West *et al.* 2006).

Juvenile and adult body condition

We examined potential differences in fat reserves between treatments in the subset of female fish preserved at age 28 days and also in females preserved after the birth of their fourth litter. Comparisons of fat content controlled for effects of lean dry body mass (Christians 1999; Packard & Boardman 1999). Residual body mass (after controlling for body length) was a good predictor of the fat content measured in females preserved after the birth of their fourth litter ($F_{1,42} = 6.09$, $P = 0.02$), so we also tested for treatment differences in body condition at maturation.

In all analyses, diagnostics were run to ensure that the functional form of the model was linear or quadratic and to inspect the distribution of the residuals. With the exception of litter size, all traits were normally distributed. Litter sizes are count data and their variance was greater than the mean, so models testing effects of treatment on litter size assumed a quasi-Poisson distribution. Main effects and interactions were regarded as significant when $P < 0.05$. All models were run using SAS version 9.2 (SAS Institute, Cary, NC, USA). Because we were interested in the fixed effects, we present results of parameters estimates and significance values for both fixed and random effects in each final model, but only discuss results for fixed effects.

RESULTS

Juvenile and adult growth

At age 14 days, there was no significant difference between experimental and control fish in either body mass (mean ± 1SE: 17.3 ± 0.6 and 16.4 ± 0.6 mg, respectively; treatment: $F_{1,21} = 2.28$, $P = 0.15$; block: Wald $Z = 2.22$, $P = 0.01$ on ln-transformed data) or standard length (9.5 ± 0.9 and 9.4 ± 0.1 mm, respectively; treatment: $F_{1,21} = 4.51$, $P = 0.05$; block: Wald $Z = 2.54$, $P = 0.01$ on ln-transformed data). During the growth restriction period, experimental fish grew at a significantly slower rate than control fish in both mass (0.5 ± 0.1 vs. 3.8 ± 0.2 mg day^{-1}, respectively; treatment: $F_{1,19} = 98.8$, $P < 0.001$; initial mass: $F_{1,19} = 85.8$, $P < 0.001$; block: Wald $Z = 0.86$, $P = 0.19$) and length (0.1 ± 0.01 vs. 0.4 ± 0.01 mm day^{-1}, respectively;

treatment: $F_{1,19} = 123.8$, $P < 0.001$; initial length: $F_{1,19} = 36.4$, $P < 0.001$; block: Wald $Z = 0.30$, $P = 0.38$) and as a result were significantly smaller than controls at 28 days of age in both mass (24.0 ± 1.2 vs. 70.2 ± 2.5 mg, respectively; treatment: $F_{1,21} = 534.8$, $P < 0.001$; block: Wald $Z = 1.52$, $P = 0.06$ on ln-transformed data) and length (10.9 ± 0.2 vs. 14.8 ± 0.2 mm, respectively; treatment: $F_{1,21} = 415.5$, $P < 0.001$; block: Wald $Z = 1.37$, $P = 0.09$ on ln-transformed data).

Once returned to *ad libitum* rations, experimental fish exhibited a compensatory growth response in both body mass and length, growing at a significantly faster rate than control fish (Table 1, Fig. 1). Compensatory growth in body mass in experimental fish, when controlling for effects of mass at the beginning of each growth period, began immediately after the growth restriction period ended and continued until they were 49 days old; growth of experimental fish during this time was roughly 30% faster than control fish (Fig. 1a,c). Compensation in body length in the experimental fish, on the other hand, did not commence until the second week after the growth restriction period ended. Daily growth in length in experimental fish was initially roughly 90% of that exhibited by control fish, but then accelerated to a rate *c.* 30% higher than the controls (Fig. 1b,d). By 49 days of age, when most experimental fish had matured, experimental fish were still significantly smaller than control fish in both mass (161.3 ± 6.1 vs. 242.5 ± 6.6 mg, respectively; treatment: $F_{1,21} = 153.4$, $P < 0.001$; block: Wald $Z = 1.80$, $P = 0.04$ on ln-transformed data) and length (19.1 ± 0.2 vs. 22.0 ± 0.2 mm, respectively; treatment: $F_{1,21} = 338.1$, $P < 0.001$; block: Wald $Z = 1.57$, $P = 0.06$ on ln-transformed data) and therefore had only partially compensated in body size.

During the adult stage, daily growth rates in both mass and length declined with litter number in both treatments, but experimental fish grew at the same rate as control fish (Table 2, Fig. 1e,f). Thus, differences in size at age between experimental and control fish at 49 days of age persisted through adulthood.

Female reproduction

Experimental fish matured at a significantly later age than control fish (49.4 ± 1.3 vs. 43.3 ± 1.5 days of age, respectively; treatment: $F_{1,21} = 30.9$, $P < 0.001$; block: Wald $Z = 1.02$, $P = 0.15$). However, there was no significant difference in body size between experimental and control fish in either mass (167.4 ± 12.4 vs. 187.4 ± 16.8 mg, respectively; treatment: $F_{1,21} = 1.20$, $P = 0.29$; block: Wald $Z = 0.58$, $P = 0.28$ on ln-transformed data) or length (19.6 ± 0.4 vs. 20.1 ± 0.5 mm, respectively; treatment: $F_{1,21} = 0.91$, $P = 0.35$; block: Wald $Z = 1.37$, $P = 0.09$ on ln-transformed data).

Interlitter interval increased with litter number, whereas mean offspring lean dry mass and mean offspring extractable fat increased with female body length. However, none

Table 1 Results of final linear mixed model testing for effects of juvenile food level regime (treatment) on juvenile growth in body mass (mg day^{-1}) and body length (mm day^{-1}). Results are given for control (=0) and experimental (=1) fish. Size and growth trajectories of experimental fish starting at the end of the 2-week growth restriction period (28 days of age) were slid back in time back to a common starting size with the control fish (17 days of age). Parameter estimates for each model are centered on age 28 days for experimental fish and age 17 days for control fish

	Juvenile growth in mass (mg day^{-1})			Juvenile growth in length (mm day^{-1})		
Fixed effects	Estimate	SE	*t*	Estimate	SE	*t*
Intercept	1.75	0.33	5.35***	0.38	0.01	32.3***
Age†	0.31	0.07	4.37***	0.01	< 0.01	3.19**
Age^2	−0.02	0.01	−4.14***	< −0.01	< 0.01	−5.14***
Initial size‡	0.05	0.01	4.89***	n/s		
Treatment	1.29	0.23	5.70***	−0.03	0.02	−1.84
Age × Treatment	n/s			0.01	< 0.01	4.96***
Age^2 × Treatment	n/s			n/s		
Random effects	Estimate	SE	Wald *Z*	Estimate	SE	Wald *Z*
Variance in female slope	0.01	< 0.01	2.09*	< 0.01	< 0.01	1.37
Residual variance	1.14	0.18	6.49***	< 0.01	< 0.01	6.59***

***$P < 0.001$; **$P < 0.01$; *$P < 0.05$; $^{n/s}P > 0.05$ and term dropped from model.

†Age is measured in days.

‡Initial size at the beginning of each growth period; initial mass (in mg) was used for the juvenile growth in mass model and initial length (in mm) was used in the juvenile growth in length model.

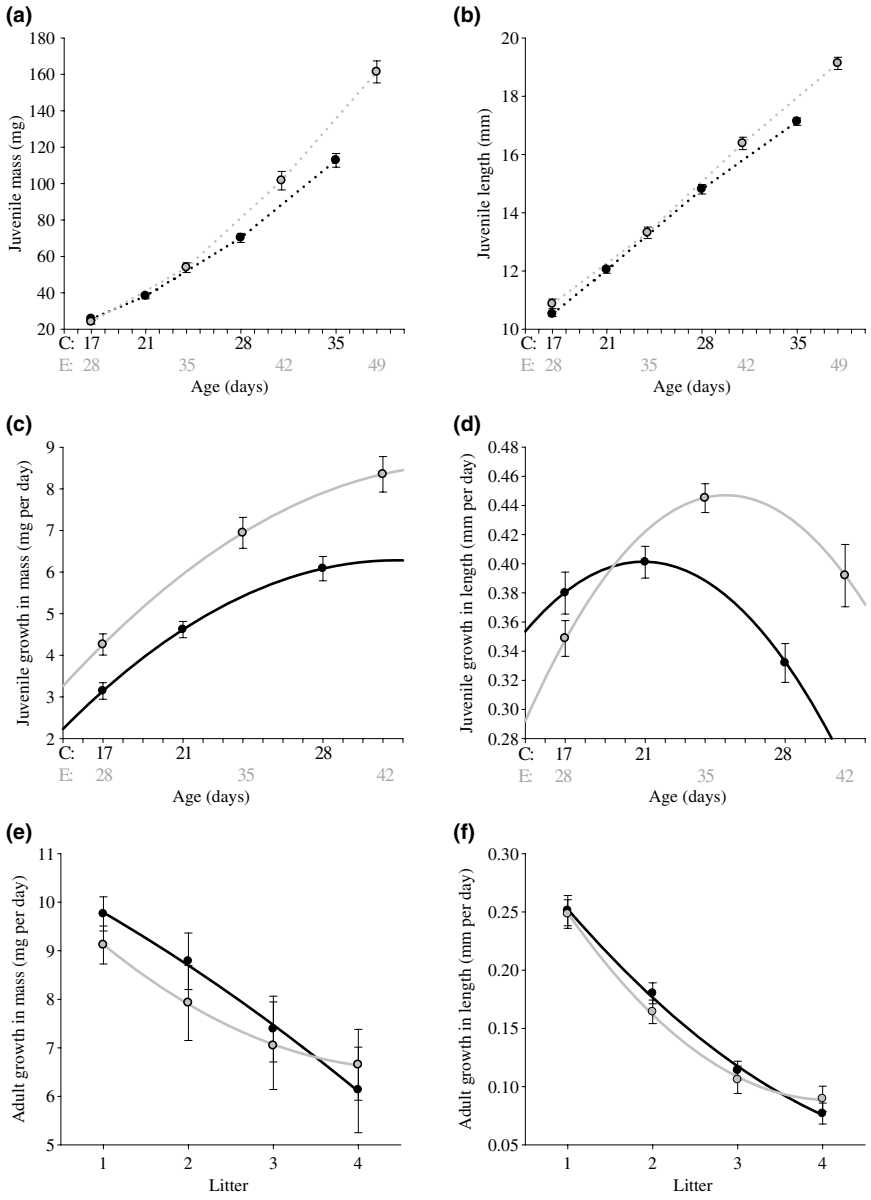

Figure 1 Trajectories of (a) juvenile body mass, (b) juvenile body length, (c) juvenile daily growth in mass (d) juvenile daily growth in length, (e) adult daily growth in mass and (f) adult daily growth in length in experimental (grey) and control (black) fish. Trajectories of juvenile body size and juvenile growth are during the post-growth restriction period. For juvenile growth analyses, size and growth trajectories (for both mass and length) of experimental fish at 28 days of age (the end of the growth restriction period) were slid back in time to a common starting size with the control fish at 17 days of age. Estimates are presented as mean ± 1SE; solid lines are predicted growth trajectories for each treatment.

were affected by treatment (Table 3, Fig. 2a–c). In contrast, treatment had a significant effect on litter size and how it changed with female body length (Table 3, Fig. 2d). When accounting for effects of litter number, litter size increased at a slower rate with female length in experimental fish relative to controls (Table 3, Fig. 2d). Because of this significant treatment by female length interaction, litter size was similar during the first litter (Table 3), but increasingly lower in the second (intercept: $t = 24.0$, $P < 0.001$, estimate ± 1SE: 28.1 ± 1.2; treatment: $t = -3.4$, $P < 0.001$, estimate: −5.47 ± 1.6), third (intercept: $t = 31.9$, $P < 0.001$, estimate: 42.0 ± 1.3; treatment: $t = -4.19$, $P < 0.001$, estimate: −7.6 ± 1.8), and fourth (intercept: $t = 32.8$, $P < 0.001$, estimate: 54.1 ± 1.7; treatment: $t = -4.42$, $P < 0.001$; estimate: −9.2 ± 2.1) litters when estimated at the grand mean length at the yolking of each of the litters. Predicted differences in litter size therefore amounted roughly to 5, 8 and 9 offspring in the second through fourth litters, respectively. This difference reflected a *c.* 20% decline in litter size in experimental relative to control fish.

Juvenile and adult body condition

In the subset of individuals preserved at age 28 days, experimental fish had a significantly lower amount of fat reserves than control fish when controlling for lean dry body mass (Fig. 3; treatment: $F_{1,12} = 9.70$, $P = 0.01$; lean

Table 2 Results of final linear mixed model testing for effects of juvenile food level regime (treatment) on adult daily growth in body mass (mg per day) and body length (mm per day) between litters. Results are from control (=0) and experimental (=1) treatment fish. Parameter estimates are centered on litter 1

	Adult growth in mass (mg day^{-1})			Adult growth in length (mm day^{-1})		
Fixed effects	Estimate	SE	t	Estimate	SE	t
Intercept	9.34	0.52	17.9***	0.25	0.01	27.1***
Litter	n/a			−0.12	0.01	−7.95***
Litter^2	−0.20	0.04	−5.75***	−0.01	< 0.01	4.11***
Treatment	−0.31	0.66	−0.47	< −0.01	0.01	−0.30
Litter × Treatment	n/s			n/s		
Litter^2 × Treatment	n/a			n/s		
Random effects	Estimate	SE	Wald Z	Estimate	SE	Wald Z
Variance in female intercept	3.07	1.09	2.80**	< 0.01	< 0.01	3.35***
Residual variance	7.87	0.86	7.87***	< 0.01	< 0.01	7.89***

***$P < 0.001$; **$P < 0.01$; $^{n/s}P > 0.05$ and term dropped from model; $^{n/a}$term not included in model.

Table 3 Results of final linear mixed model testing for effects of juvenile food level regime (treatment) on adult changes in interlitter interval (days), mean offspring lean dry mass (mg), mean offspring extractable fat (mg), and litter size. Results are given for control (=0) and experimental (=1) fish. Parameter estimates for each model are centered on litter 1 and the grand mean body length (19.8 mm) at the yolking of the first litter

	Interlitter interval (days)		Mean offspring lean dry mass (mg)		Mean offspring extractable fat (mg)		Litter size	
Fixed effects	Estimate (SE)	t	Estimate (SE)	t	Estimate (SE)	t	Estimate (SE)	t
Intercept	23.22 (0.30)	77.0***	0.56 (0.02)	32.8***	0.10 (0.01)	16.8***	10.67 (1.21)	8.82***
Litter	0.53 (0.11)	4.97***	n/s		n/s		7.56 (1.29)	5.88***
Body length†	n/a		0.01 (< 0.01)	6.91***	0.01 (< 0.01)	11.0***	1.67 (0.28)	5.96***
Treatment	−0.05 (0.42)	−0.12	0.03 (0.02)	1.61	0.01 (0.01)	1.92	−2.15 (1.63)	−1.31
Litter × Treatment	n/s		n/s		n/s		n/s	
Body length × Treatment	n/a		n/s		n/s		−0.56 (0.15)	−3.66***
Random effects	Estimate (SE)	Wald Z	Estimate (SE)	Wald Z	Estimate (SE)	Wald Z	Estimate (SE)	Wald Z
Variance in female intercept	1.33 (0.43)	3.08**	0.01 (< 0.01)	3.31***	< 0.01 (< 0.01)	2.59**	18.93 (5.83)	3.24***
Residual variance	2.44 (0.31)	7.96***	0.01 (< 0.01)	7.64***	< 0.01 (< 0.01)	7.83***	1.14 (0.15)	7.69***

***$P < 0.001$; **$P < 0.01$; $^{n/s}P > 0.05$ and term dropped from model; $^{n/a}$term not included in model.
†Body length is measured in millimeters.

dry body mass: $F_{1,12} = 8.26$, $P = 0.01$; block: Wald $Z = 1.43$, $P = 0.08$ on ln-transformed data). Differences in body condition between treatment groups, estimated as residual ln-transformed body mass after removing effects of ln-transformed body length, were no longer evident at the estimated age of maturation (treatment: $F_{1,20} = 0.64$, $P = 0.43$; length: $F_{1,20} = 259.8$, $P < 0.001$; block: Wald $Z = 0.97$, $P = 0.17$). By the end of the experiment when females gave birth to their fourth litter, there was no significant difference in fat reserves between treatments (Fig. 3; treatment: $F_{1,20} = 2.71$, $P = 0.12$; lean dry body mass: $F_{1,20} = 66.3$, $P < 0.001$; block: Wald $Z = 0.17$, $P = 0.43$ on ln-transformed data).

DISCUSSION

Our food manipulation was successful in inducing compensatory growth in both mass and length. Experimental fish accelerated their growth rates after food returned to *ad libitum* levels, increasing growth in mass immediately but delaying the increase in growth in length until 1 week after the growth restriction period ended. They then maintained the same

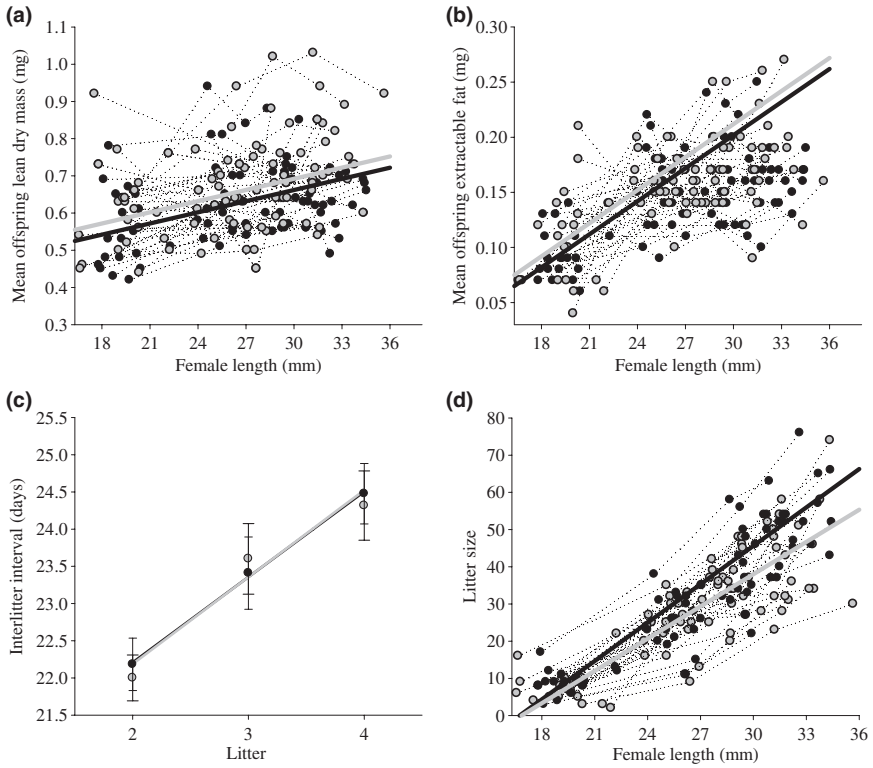

Figure 2 Trajectories of (a) mean offspring lean dry mass, (b) mean offspring extractable fat content, (c) interlitter interval and (d) litter size for experimental (grey) and control (black) fish. For mean offspring lean dry mass, mean offspring extractable fat and litter size, dotted lines represent observed individual trajectories; solid lines represent predicted trajectories for each treatment. For interlitter interval, estimates are presented as mean ± 1SE; solid lines are predicted trajectories for each treatment. Note that because estimated values for interlitter interval for experimental and control fish are nearly identical, only one trajectory is visible.

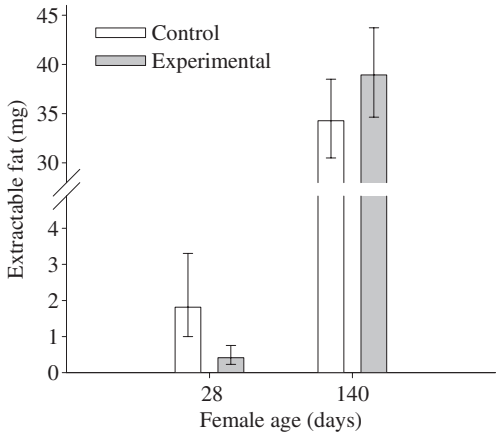

Figure 3 Extractable fat (mean ± 95% CI) for a subset of experimental and control females preserved at the end of the growth restriction period (age 28 days) and for experimental and control females preserved at the birth of their fourth litter (mean age = 140 days). Estimates of fat content are estimated marginal mean values after controlling for variation in lean dry body mass.

growth rates as controls during the adult stage after only partially compensating in mass and length. Experimental fish matured *c.* 6 days later but at the same mass and length as controls. Interlitter interval, mean offspring lean dry mass and mean offspring extractable fat were not affected by juvenile growth history. In contrast, juvenile compensatory growth led to reduced fecundity. After controlling for the effects of litter number and female length, litter size was similar in the first litter but was increasingly lower in the second, third and fourth litters in experimental relative to control fish. Although compensatory growth has been shown to reduce the occurrence of precocious sexual maturation in males (Morgan & Metcalfe 2001), this is the first study, to our knowledge, to demonstrate that it can have negative effects on female reproduction and that the magnitude of these effects increases throughout adulthood.

Costs to reproduction could arise through a direct trade-off between compensatory growth and later reproduction. For example, compensatory growth could have negative impacts on reproductive development and physiology if it decreases the efficiency with which energy is assimilated or if it interferes with the development of reproductive structures. There is some evidence suggesting that rapid juvenile growth can interfere with the development of non-reproductive structures (Ricklefs *et al.* 1994; Arendt *et al.* 2001; Arendt 2003), but its impact on reproductive development and physiology are unknown. Compensatory growth could also negatively affect reproduction if it increases metabolic needs and thereby decreases the amount of energy available for reproduction. However, the effects of compensatory growth on metabolic rates are currently equivocal (Wieser *et al.* 1992; Stoks *et al.* 2006; Criscuolo *et al.* 2008).

Alternatively, the observed trade-off between compensatory growth and later reproduction could arise if juvenile growth impacts another trait closely tied to reproduction and alters how the trade-off between that trait and reproduction is resolved. Investment in reproduction is intimately connected to investment in adult growth and survival. Because energy is a finite resource, adults must 'decide' how to partition energy among the competing demands of these fitness-related traits (Roff 2002). How these trade-offs are resolved often depends on the state of the individual (McNamara & Houston 1996). Thus, if compensatory growth has long-term negative effects on adult growth potential or survival probability, these impacts may change how the trade-off between these traits and reproduction is resolved. For example, the well-known long-term negative effects of rapid juvenile growth on adult survival may favour increased investment in survival at the expense of reproduction and lead to the reproductive costs we observed. Although we did not measure investment in adult survival or changes in growth efficiency, these potential mechanisms certainly remain plausible.

Finally, the observed reduction in litter size could stem from long-term effects of the period of early growth restriction. Low food levels during early growth and development can have long-term negative effects on reproduction (Lindstrom 1999; Lummaa & Clutton-Brock 2002). However, they are also known to have no effect as well as positive effects on different components of reproduction (e.g. Taborsky 2006; Painter *et al.* 2008). In this study, experimental fish were subjected to a food level during the growth restriction period that allowed them to grow approximately 1.3 mm during the 2-week growth restriction period, a rate of growth that falls well within the range of 0.25–2.25 mm growth per 2-week period observed in the wild (Grether *et al.* 2001; Reznick *et al.* 2001). In addition, treatment differences in body condition present at the end of the growth restriction period were no longer evident at maturation. Thus, it seems unlikely that there were any long-term negative effects of early growth restriction. However, we cannot discount the possibility that the long-term reduction in reproductive potential is a direct consequence of the growth restriction period.

Events during the juvenile stage are thought to be tightly coupled with fitness in the adult stage through the size at which maturation occurs. In this study, juvenile growth history had a significant impact on litter size that was independent of the effects of female size. However, it is presently unclear what mechanisms underlie this observed cost to reproduction and why costs were delayed until later litters. Although further studies are needed to fully tease apart the separate effects of the initial growth restriction from the subsequent growth acceleration, our results suggest that reproductive costs to accelerated juvenile growth may play an important role in the evolution of compensatory growth and other rapid juvenile growth responses.

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REFERENCES

Abrams, P.A., Leimar, O., Nylin, S. & Wiklund, C. (1996). The effect of flexible growth rates on optimal sizes and development times in a seasonal environment. *Am. Nat.*, 147, 381–395.

Ali, M., Nicieza, A. & Wootton, R. (2003). Compensatory growth in fishes: a response to growth depression. *Fish Fish*, 4, 147–190.

Arendt, J. (2003). Reduced burst speed is a cost of rapid growth in anuran tadpoles: problems of autocorrelation and inferences about growth rates. *Funct. Ecol.*, 17, 328–334.

Arendt, J., Wilson, D.S. & Stark, E. (2001). Scale strength as a cost of rapid growth in sunfish. *Oikos*, 93, 95–100.

Bashey, F. (2008). Competition as a selective mechanism for larger offspring size in guppies. *Oikos*, 117, 104–113.

Berkeley, S.A., Chapman, C. & Sogard, S.M. (2004). Maternal age as a determinant of larval growth and survival in a marine fish, *Sebastes melanops*. *Ecology*, 85, 1258–1264.

Bolker, B.M., Brooks, M.E., Clark, C.J., Geange, S.W., Poulsen, J.R., Stevens, M.H.H. *et al.* (2009). Generalized linear mixed models: a practical guide for ecology and evolution. *Trends Ecol. Evol.*, 24, 127–135.

Christians, J.K. (1999). Controlling for body mass effects: is part whole correlation important? *Physiol. Biochem. Zool.*, 72, 250–253.

Conover, D.O. & Schultz, E.T. (1997). Natural selection and adaptation of growth rate: what are the tradeoffs? In: *Early Life History and Recruitment in Fish Populations* (eds Chambers, R.C. & Trippel, E.A.). Chapman and Hall, London, pp. 305–332.

Criscuolo, F., Monaghan, P., Nasir, L. & Metcalfe, N.B. (2008). Early nutrition and phenotypic development: 'catch-up' growth leads to elevated metabolic rate in adulthood. *Proc. R. Soc. Lond. B Biol. Sci.*, 275, 1565–1570.

De Block, M. & Stoks, R. (2008). Compensatory growth and oxidative stress in a damselfly. *Proc. R. Soc. Lond. B Biol. Sci.*, 275, 781–785.

Dmitriew, C. & Rowe, L. (2005). Resource limitation, predation risk and compensatory growth in a damselfly. *Oecologia*, 142, 150–154.

Dmitriew, C. & Rowe, L. (2007). Effects of early resource limitation and compensatory growth on lifetime fitness in the ladybird beetle (*Harmonia axyridis*). *J. Evol. Biol.*, 20, 1298–1310.

Fisher, M.O., Nager, R.G. & Monaghan, P. (2006). Compensatory growth impairs adult cognitive performance. *PLoS Biol.*, 4, 1462–1466.

Forsen, T., Osmond, C., Eriksson, J.G. & Barker, D.J.P. (2004). Growth of girls who later develop coronary heart disease. *Heart*, 90, 20–24.

Grether, G.F., Millie, D.F., Bryant, M.J., Reznick, D.N. & Mayea, W. (2001). Rain forest canopy cover, resource availability, and life history evolution in guppies. *Ecology*, 82, 1546–1559.

Johnsson, J.I. & Bohlin, T. (2006). The cost of catching up: increased winter mortality following structural growth compensation in the wild. *Proc. R. Soc. Lond. B Biol. Sci.*, 273, 1281–1286.

Lankford, T.E. Jr, Billerbeck, J.M. & Conover, D.O. (2001). Evolution of intrinsic growth and energy acqusition rates, II. Trade-offs with vulnerability to predation in *Menidia menidia*. *Evolution*, 55, 1873–1881.

Lindstrom, J. (1999). Early development and fitness in birds and mammals. *Trends Ecol. Evol.*, 14, 343–348.

Lindstrom, J., Metcalfe, N.B. & Royle, N.J. (2005). How are animals with ornaments predicted to compensate for a bad start in life? A dynamic optimization model approach *Funct. Ecol.*, 19, 421–428.

Lummaa, V. & Clutton-Brock, T. (2002). Early development, survival and reproduction in humans. *Trends Ecol. Evol.*, 17, 141–147.

Mangel, M. & Munch, S.B. (2005). A life-history perspective on short- and long-term consequences of compensatory growth. *Am. Nat.*, 166, E155–E176.

McNamara, J.M. & Houston, A.I. (1996). State-dependent life histories. *Nature*, 380, 215–221.

Metcalfe, N.B. & Monaghan, P. (2001). Compensation for a bad start: grow now, pay later? *Trends Ecol. Evol.*, 16, 254–260.

Metcalfe, N.B. & Monaghan, P. (2003). Growth versus lifespan: perspectives from evolutionary ecology. *Exp. Geront.*, 38, 935–940.

Morgan, I.J. & Metcalfe, N.B. (2001). Deferred costs of compensatory growth after autumnal food shortage in juvenile salmon. *Proc. R. Soc. Lond. B Biol. Sci.*, 268, 295–301.

Nicieza, A. & Alvarez, D. (2009). Statistical analysis of structural compensatory growth: how can we reduce the rate of false detection? *Oecologia*, 159, 27–39.

Packard, G.C. & Boardman, T.J. (1999). The use of percentages and size-specific indices to normalize physiological data for variation in body size: wasted time, wasted effort? *Comp. Biochem. Physiol., A*, 122, 37–44.

Painter, R.C., Westendorp, R.G.J., de Rooij, S.R., Osmond, C., Barker, D.J.P. & Roseboom, T.J. (2008). Increased reproductive success of women after prenatal undernutrition. *Hum. Reprod.*, 23, 2591–2595.

Reznick, D.N. (1980). *Life history evolution in the guppy (Poecilia reticulata)*. PhD Thesis, University of Pennsylvania, Philadelphia.

Reznick, D.N. (1982). The impact of predation on life history evolution in Trinidadian guppies: genetic basis of observed life history patterns. *Evolution*, 36, 1236–1250.

Reznick, D.N. (1985). Costs of reproduction: an evaluation of the empirical evidence. *Oikos*, 44, 257–267.

Reznick, D.N. (1990). Plasticity in age and size at maturity in male guppies (*Poecilia reticulata*): an experimental evaluation of alternative models of development. *J. Evol. Biol.*, 3, 185–203.

Reznick, D.N. & Bryga, H.A. (1996). Life-history evolution in guppies (*Poecilia reticulata*: Poeciliidae). 5 Genetic basis of parallelism in life histories. *Am. Nat.*, 147, 339–359.

Reznick, D.N. & Endler, J.A. (1982). The impact of predation on life history evolution in Trinidadian guppies (*Poecilia reticulata*). *Evolution*, 63, 160–177.

Reznick, D.N. & Yang, A.P. (1993). The influence of fluctuating resources on life-history: patterns of allocation and plasticity in female guppies. *Ecology*, 74, 2011–2019.

Reznick, D.N., Butler, M. IV & Rodd, H. (2001). Life-history evolution in guppies. VII. The comparative ecology of high- and low-predation environments. *Am. Nat.*, 157, 126–140.

Ricklefs, R.E., Shea, R.E. & Choi, I.-H. (1994). Inverse relationship between functional maturity and exponential growth rate of avian skeletal muscle: a constraint on evolutionary response. *Evolution*, 48, 1080–1088.

Roff, D.A. (1992). *The evolution of life histories: theory and analysis*. Chapman and Hall, New York.

Roff, D.A. (2002). *Life history evolution*. Sinauer Associates, Sunderland, MA.

Royle, N.J., Lindstrom, J. & Metcalfe, N.B. (2006). Effect of growth compensation on subsequent physical fitness in green swordtails *Xiphophorus helleri*. *Biol. Lett.*, 2, 39–42.

Singer, J.D. & Willett, J.B. (2003). *Applied longitudinal data analysis: Modeling change and event occurrence*. Oxford University Press, Oxford.

Sogard, S.M. & Olla, B.L. (2002). Contrasts in the capacity and underlying mechanisms for compensatory growth in two pelagic marine fishes. *Mar. Ecol. Prog. Ser.*, 243, 165–177.

Stoks, R., De Block, M. & McPeek, M.A. (2006). Physiological costs of compensatory growth in a damselfly. *Ecology*, 87, 1566–1574.

Taborsky, B. (2006). The influence of juvenile and adult environments on life-history trajectories. *Proc. R. Soc. Lond. B Biol. Sci.*, 273, 741–750.

West, B.T., Welch, K.B. & Galecki, A.T. (2006). *Linear mixed models: a practical guide using statistical software*. Chapman and Hall CRC Press, New York.

Wieser, W., Krumschnabel, G. & Ojwang-Okwor, J.P. (1992). The energetics of starvation and growth after refeeding in juveniles of three cyprinid species. *Env. Biol. Fishes*, 33, 63–71.

Yearsley, J.M., Kyriazakis, I. & Gordon, I.J. (2004). Delayed costs of growth and compensatory growth rates. *Funct. Ecol.*, 18, 563–570.

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