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Deferred costs of compensatory growth after autumnal food shortage in juvenile salmon

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Growing animals are often able to offset the effects of periods of reduced food availability by subsequently undergoing a phase of elevated compensatory or 'catch-up' growth. This indicates that growth rates are not normally maximized even when food is not limiting, suggesting that fast growth may be costly. Here, we show experimental evidence of a long-term deferred cost of compensatory growth after a period of food shortage. Juvenile salmon subjected to a short-lived low-food regime in autumn subsequently entered a hyperphagic phase, leading to complete restoration of lipid reserves and partial recovery of lost skeletal growth relative to controls. However, several months later they entered a prolonged phase of poorer performance (despite food now being freely available), so that by the following spring they were substantially smaller than controls and had lower lipid reserves for their body size. The incidence of sexual maturation in males the following breeding season was also reduced. Salmon thus appear to trade off the benefits of short-term restoration of fat stores prior to winter against long-term performance.

Keywords: *Salmo salar*; life-history strategy; fat; body size; compensatory growth; fish

1. INTRODUCTION

Many animals experience unpredictable and sometimes prolonged periods of food shortage, which can influence skeletal body size if the shortage occurs during a period when the animal would normally be growing. A commonly observed response to this is a subsequent period of compensatory or 'catch-up' growth, where the animal undergoes a period of accelerated weight gain, usually lasting a matter of weeks, such that it partially or fully recovers from the period of poor growth (Wilson & Osbourn 1960; Ashworth 1986; Boersma & Wit 1997). Compensatory growth is usually achieved through hyperphagia, sometimes coupled with increases in metabolic and/or digestive efficiency (Wieser *et al.* 1992; Ji & Friedman 1999).

The existence of a compensatory response indicates that growth is normally maintained at sub-maximal levels, presumably because elevated rates of growth are costly (Calow 1982; Arendt 1997). Potential short-term costs of rapid growth include developmental deformities (Arendt 1997), reduced investment in tissue maintenance (Morgan *et al.* 2000) and an elevated exposure to predators caused by an increased food demand (Werner & Anholt 1993; Johnsson *et al.* 1999). More intriguingly, there is also some evidence of long-term costs (see Birkhead *et al.* 1999). In this study we show that a period of food shortage and subsequent compensatory growth can have unexpected and dramatic longer-term costs in salmon.

Juvenile Atlantic salmon, *Salmo salar*, have previously been shown to be capable of compensatory growth, sustaining elevated skeletal growth rates for months after a late-summer period of food deprivation (Nicieza & Metcalfe 1997). However, in autumn they must also deposit fat, since fishes reduce the risk of predation in winter by decreasing their foraging effort and using stored lipid as an energy source (Bull *et al.* 1996). Food shortage in autumn therefore prevents the fish from building up this lipid reserve, as well as causing a loss of skeletal growth, which has negative implications for future life-history decisions (Metcalfe 1998); there is thus a potential trade-off in any compensatory response between immediate (lipid) and long-term (skeletal growth) requirements. In examining how juvenile salmon responded to, and compensated for, these contrasting effects of food shortage, we revealed unexpected and previously undocumented costs in terms of growth performance, lipid levels and maturation rates that only became apparent months after the period of compensation had ended.

2. METHODS

The fertilized eggs of a single pair of sea-run Atlantic salmon from the River Shin, Sutherland, UK, were reared under ambient light and temperature conditions at the University Field Station, Rowardennan, UK. In order to standardize the states of the fish, only fish that would delay seaward migration until the age of two years were used. In September 1998, 240 0+ juveniles of less than 75 mm fork length were selected as delayed migrants (body size at this age being a good predictor of migration strategy (Thorpe 1977)) and moved to aquaria in Glasgow University. The fish were measured for wet weight (to the nearest 0.01 g) and fork length (to the nearest 0.1 mm) and given combinations of alcian-blue-dye marks on the ventral body surface to enable individual identification. They were divided equally into a treatment group and a control group, each with two replicates of 60 fish. The two groups, over a period of six weeks (7 September 1998 to 22 October 1998), were treated as follows.

Control group: an excess daily ration was provided by automatic feeding at 20 min intervals between 10.00 and 16.00 with a commercial diet (BOCM Pauls Keystart 1200 (Renfrew, UK); protein 55%, lipid 16%) that had been ground and re-pelleted to allow incorporation of the label for measurement of food consumption (see below) into a sub-sample of the pellets.

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Low-food (treatment) group: the fish were fed to excess as above, but on only one day per week. This feeding schedule was chosen over reduced daily feeding in order to minimize the establishment of feeding hierarchies and so allow all fish to feed freely on days when food was provided.

All fish were held under simulated ambient (for Rowardennan) temperature and photoperiod in 100 l tanks. The tanks were fitted with a platform raised 5 cm above the tank floor with a continuous gap of 2.5 cm between its edge and the tank wall. The fish were easily able to seek refuge below this platform but had to emerge above it in order to feed; this simulated the microhabitat choice faced by fish in natural streams. At the end of the treatment period all the fish were placed under control conditions until the end of the experiment (26 April 1999). Weight and length were measured at the beginning (8 September 1998) and end (20 October 1998) of the treatment period and then at increasing intervals (9 November 1998, 30 November 1998, 21 December 1998, 25 January 1999, 12 March 1999 and 16 April 1999). Specific growth rates (% day^{-1}) of weight (SGR_W) and length (SGR_L) between each pair of measurement dates were calculated as $\text{SGR} = 100 \times (\ln S_2 - \ln S_1)/(t_2 - t_1)$, where S_1 and S_2 are the initial and final sizes at times t_1 and t_2 (days), respectively. Food consumption (milligrams of food per gram of body weight per day) was measured on each sampling occasion after the end of the experimental treatment using an X-ray technique as described in Morgan *et al.* (2000). X-ray opaque Ballotini beads were observed in the hindgut of less than 1% of fish, indicating that the 6 h feeding period did not exceed gut evacuation time. The whole-body lipid reserves (g) of individual fish were estimated repeatedly from wet weight (W) and fork length (L),

$$\text{lipid} = 0.180W - 0.015L + 0.641 \quad (F_{2,89} = 652.2,\ r^2 = 0.935,\ p < 0.001), \qquad (1)$$

and then expressed as a percentage of wet body weight. Equation (1) was derived from a separate sacrificed sample of salmon from the same population as, and of similar size and condition to, the experimental fish, which were subjected to Soxhlet solvent extraction (Simpson *et al.* 1992).

On each occasion that the above parameters were measured, the fish were replaced in a different tank in a pseudo-random fashion (no group of fish occupied the same tank in consecutive sampling intervals) in order to minimize any tank effects on foraging and growth patterns. On 24 October 1998, 18 fish died in one of the control tanks when a standpipe was replaced incorrectly following routine cleaning. In order to maintain similar numbers of fish in all tanks, individuals were transferred between the duplicate control tanks and the number of replicate tanks for the low-food treatment was increased from two to three. After the end of the experiment in April 1999, the remaining fish were pooled in a single tank per treatment group and held for a further four months so that the sex and maturity status of the fish could be determined by internal examination.

Feeding and growth were analysed using repeated-measures analysis of variance, with time as the within-subjects factor and treatment (low food, control), sex category (maturing male, immature male, female—all of which were immature) and replicate tank as between-subjects factors, tank being nested within treatment. Percentage data were converted to proportions and subjected to arcsine transformation prior to statistical analysis. The location of any difference between sex categories was identified using a post-hoc Tukey honestly significant difference (HSD) test ($p < 0.05/3$). Where significant overall effects of treatment and interactions between time and treatment were found, data at each time were analysed separately using three-way (nested) analysis of variance at an acceptance level of $p < 0.05/k$, where k was the number of sampling times. Although replicate tank was controlled for in all statistical analyses, this factor has minimal biological significance and therefore is not considered in the results and their discussion; values for individual tanks have been pooled for display purposes in tables and figures.

3. RESULTS

Out of the fish alive at the time of determining sex and maturity status, 16 control and 18 low-food fish had undergone smoltification (i.e. were not delayed migrants), while ten controls and eight low-food fish had lost their identification marks. This left a total of 53 control and 55 low-food delayed-migrant fish for use in subsequent analyses. While the two treatments had a similar sex ratio, sexual maturation among males was reduced from 74% in the control fish to 48% in the low-food group (table 1, $\chi^2 = 4.91$, d.f. = 1, $p < 0.05$).

The treatment period had the desired effect of reducing the growth and condition of the low-food group. While the mean weights and lengths of the two groups of juvenile salmon were similar prior to the treatment (weight: $F_{1,92} = 1.87$, $p = 0.175$; length: $F_{1,89} = 2.66$, $p = 0.107$) (table 1), the low-food group were significantly lighter ($F_{1,92} = 28.53$, $p < 0.001$) and shorter ($F_{1,89} = 15.75$, $p < 0.001$) than the controls by October. As a result, weight and length were dependent upon treatment and showed a significant interaction with time (table 2).

The food consumption rate of the juvenile salmon after treatment was independent of sex category but was strongly dependent on treatment group (table 2). After the end of the treatment period, intake rates of fish previously given restricted food were several times greater than those of the controls ($F_{1,84} = 71.26$, $p < 0.001$) (figure 1). This compensatory hyperphagia was lost within five weeks of the end of the experimental treatment and both treatments subsequently showed a gradual decline in feeding rates until January. However, while control fish increased their intake in the spring, the fish previously given the low-food treatment did not, leading to a significant difference in intake by March ($F_{1,84} = 71.23$, $p = 0.004$).

As a consequence, specific growth rates in both weight (SGR_W) and length (SGR_L) showed a highly significant interaction between treatment group and time (table 2): the low-food fish tended to have higher growth rates than controls immediately following the experimental treatment period in autumn but lower growth rates than controls by the following spring (figure 2). As a result, the size discrepancy between the treatment groups initially decreased due to catch-up growth but then became ever more extreme (table 1; differences in April in weight: $F_{1,92} = 33.92$, $p < 0.001$; and length: $F_{1,89} = 27.32$, $p < 0.001$). These differences in growth trajectory were not simply due to a continuation of the differences induced by the experimental treatment: control fish of a given weight at the end of the treatment period had grown significantly more by April than had low-food fish

Table 1. *Size of salmon in relation to autumnal food availability*

(Letters (a,b), where different, indicate a significant difference between sex categories (maturing male, immature male, female) within a treatment (Tukey HSD, $p < 0.05$).)

	weight (g)				length (mm)			
treatment (*n*)	initial (September)	after treatment (October)	final (April)		initial (September)	after treatment (October)	final (April)	
control								
maturing males (17)	3.22 ± 0.15	3.81 ± 0.17	7.41 ± 0.48	a	67.6 ± 0.8	71.8 ± 1.0	87.3 ± 1.7	a
immature males (6)	2.73 ± 0.32	3.19 ± 0.18	5.30 ± 0.45	b	64.7 ± 0.8	68.8 ± 1.0	79.3 ± 1.8	b
females (26)	3.29 ± 0.17	3.88 ± 0.19	7.22 ± 0.40	a	68.0 ± 1.0	71.9 ± 1.1	86.8 ± 1.4	a
low food								
maturing males (10)	3.16 ± 0.21	3.10 ± 0.22	5.40 ± 0.58	a	66.9 ± 1.3	68.7 ± 1.5	80.8 ± 2.2	a
immature males (11)	2.76 ± 0.19	2.61 ± 0.20	4.20 ± 0.46	b	63.6 ± 1.5	64.8 ± 1.5	74.5 ± 2.4	b
females (29)	2.93 ± 0.11	2.92 ± 0.12	4.80 ± 0.29	a	65.7 ± 0.7	67.4 ± 0.8	78.0 ± 1.4	a

Table 2. *Repeated-measures analyses of variance of the post-treatment food-consumption rate and growth of salmon in relation to food availability in autumn*

(Treatment: low food or control (excess) in autumn; sex: maturing male, immature male or female; SGR: specific growth rate in weight (SGR_W) or length (SGR_L); occasional missing values result in differing sample sizes for the different analyses.)

factor		weight	length	lipid content	SGR_W	SGR_L	feeding rate
time	*F*	53.88	106.49	71.05	150.56	13.68	31.60
	d.f.	7,86	7,83	7,87	5,83	5,82	4,81
	p	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
treatment	*F*	17.27	13.43	17.48	0.24	0.37	11.07
	d.f.	1,92	1,89	1,93	1,87	1,86	1,84
	p	<0.001	<0.001	<0.001	0.626	0.547	0.001
treatment × time	*F*	29.28	14.68	26.61	46.59	11.35	18.65
	d.f.	7,86	7,83	7,87	5,83	5,82	4,81
	p	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
sex	*F*	4.96	5.21	3.64	2.50	4.21	0.18
	d.f.	2,92	2,89	2,93	2,87	2,86	2,84
	p	0.009	0.008	0.030	0.088	0.018	0.837
sex × time	*F*	1.26	1.10	0.91	0.49	0.70	0.38
	d.f.	14,172	14,166	14,174	10,166	10,164	8,162
	p	0.236	0.359	0.550	0.898	0.723	0.930

of the same post-treatment size (analysis of covariance, $F_{1,92} = 15.61$, $p < 0.001$). Throughout the experiment there were significant differences between the sex categories in both weight and length (table 2): females and maturing males were both heavier (Tukey HSD, $p = 0.002$ and $p = 0.017$, respectively) and longer (Tukey HSD, $p = 0.002$ and $p = 0.012$, respectively) than immature males. However, both SGR_W and SGR_L were independent of sex category (table 1).

The lipid reserves of the juvenile salmon were significantly affected by experimental treatment and showed a significant treatment × time interaction (table 2). The two treatments initially had similar lipid reserves of approximately 7% of body weight (figure 3) but, as expected, the fish given the low-food regime had significantly lower lipid reserves by late October than the controls ($F_{1,93} = 36.54$, $p < 0.001$). The low-food fish then showed a strong compensatory response, rapidly increasing their lipid reserves to control levels within two weeks of the end of the experimental treatment, and the two groups then maintained similar lipid reserves until December. However, between December and March the lipid reserves of the low-food fish decreased whilst those of the control fish increased slightly, such that there was a significant difference between the two treatments in January ($F_{1,93} = 10.87$, $p < 0.001$), which became greater by March ($F_{1,93} = 38.81$, $p < 0.001$) and was maintained through to April ($F_{1,93} = 41.84$, $p < 0.001$) despite both groups showing an increase in lipid levels. The difference in lipid reserves between the two groups at the end of the experiment was not simply due to the difference in average size, as fish in the previously low-food group had a lower percentage lipid than control fish of the same body length (analysis of covariance based on arcsine-transformed data, $F_{1,90} = 11.85$, $p = 0.001$). The lipid reserves of immature males were significantly lower than those of either

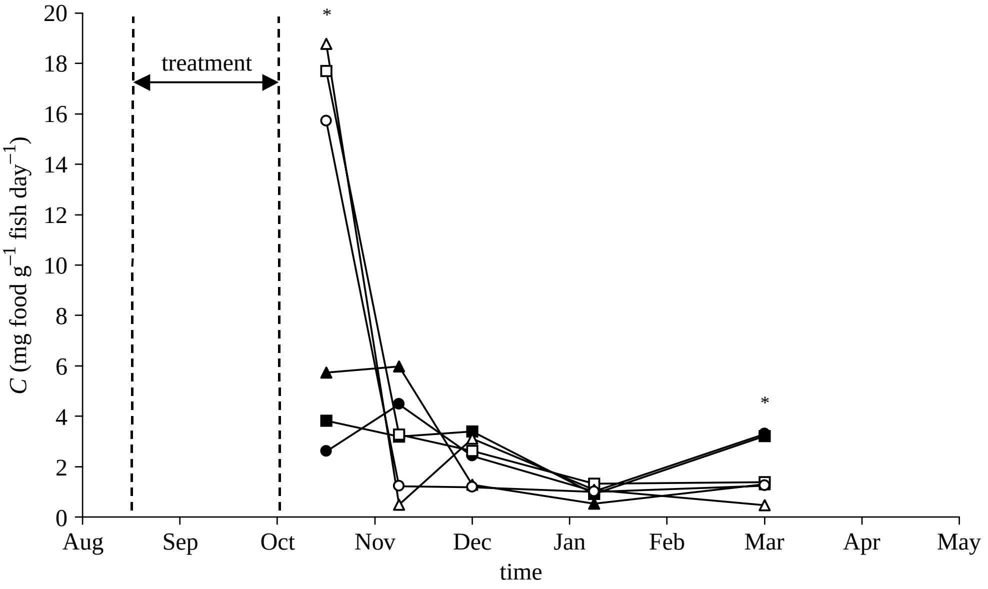

Figure 1. Mean food-consumption rate (C, milligrams of food per gram of body weight per day) of juvenile salmon following temporary manipulation of food availability (during the treatment period). Control (filled symbols) and low-food (open symbols) fish are each divided into males that will become sexually mature in the following autumn (squares), immature males (triangles) and females (circles). Error bars have been omitted for clarity; asterisks indicate a significant difference between control and low-food fish (three-way ANOVA, $p < 0.05$). See table 2 for other statistical analyses.

mature males (Tukey HSD, $p = 0.007$) or females (Tukey HSD, $p = 0.020$) throughout the experiment (table 2 and figure 3).

4. DISCUSSION

The low-food fish responded to increased food availability with a dramatic but short-lived increase in food consumption. This hyperphagic response resulted in the restoration of lipid reserves to control levels within two weeks, but only partial compensation for lost body growth. This may be because lipid levels are more important than body size *per se* at this time of year. Although a number of studies have reported that the probability of overwinter survival in juvenile salmonids is size dependent, they have attributed this to the greater energy reserves of larger fish (Pickering & Pottinger 1988; Meyer & Griffith 1997). During the compensatory period the fish thus appeared to preferentially allocate resources to fat replenishment rather than skeletal growth, presumably because the fitness costs of low lipid reserves in early winter outweigh those of lost skeletal growth, which can be made up over a longer time-period (Bull *et al.* 1996).

Once any compensatory phase had passed, the two treatments would be expected to show similar patterns of food consumption and growth. However, from December onwards (two months after the end of the treatment period and six weeks after the end of compensatory hyperphagia) the performances of the two treatment groups diverged. The growth rates of the previously low-food fish fell consistently below those of the controls, resulting in significant differences in weight and length, which were greater than those observed at the end of the experimental treatment. However, the most striking difference between the treatment groups in the latter part of the experiment was in their lipid reserves. Although the fish had similar reserves in early November, control fish showed a continual slight increase in body lipids from December to March whereas those previously deprived of food showed a continual decrease, so that a widening difference in lipid reserves was sustained through to the end of the experiment in April. This treatment difference was significantly greater than would be expected from the differences in body size.

The loss of lipid in the low-food fish had major life-history implications as it was linked to a significantly lower incidence of sexual maturation in males. The probability of a male maturing in its second autumn is positively correlated with its size and lipid reserves at the onset of the first winter (Bohlin *et al.* 1990; Simpson 1993) but maturation appears to be switched off in males that fail to build up reserves in the spring (Rowe *et al.* 1991; Berglund 1995). Maturation may begin as early as November (Simpson 1993), but by that time in the present experiment the lipid levels of the low-food fish had recovered from the experimental treatment. It is therefore probable that the unexpected lipid loss observed from December to March in the low-food fish was a direct cause of the reduced incidence of male maturation.

These unexpected results demonstrate that, while the juvenile salmon may have recovered in the short term from a temporary reduction in food availability, they subsequently paid costs in terms of impaired future growth, depleted energetic reserves and deferred sexual maturation. Negative effects on adult performance of poor nutrition at critical stages of foetal development have been documented in mammals (for a review, see Desai & Hales 1997). In rats, these effects have been attributed to the 'thrifty phenotype' hypothesis (Hales & Barker 1992), which proposes that permanent metabolic changes that occur during malnutrition are adaptive for subsequent survival under such conditions but become maladaptive if nutrition is improved. However, these effects appear to be restricted to nutritional perturbations

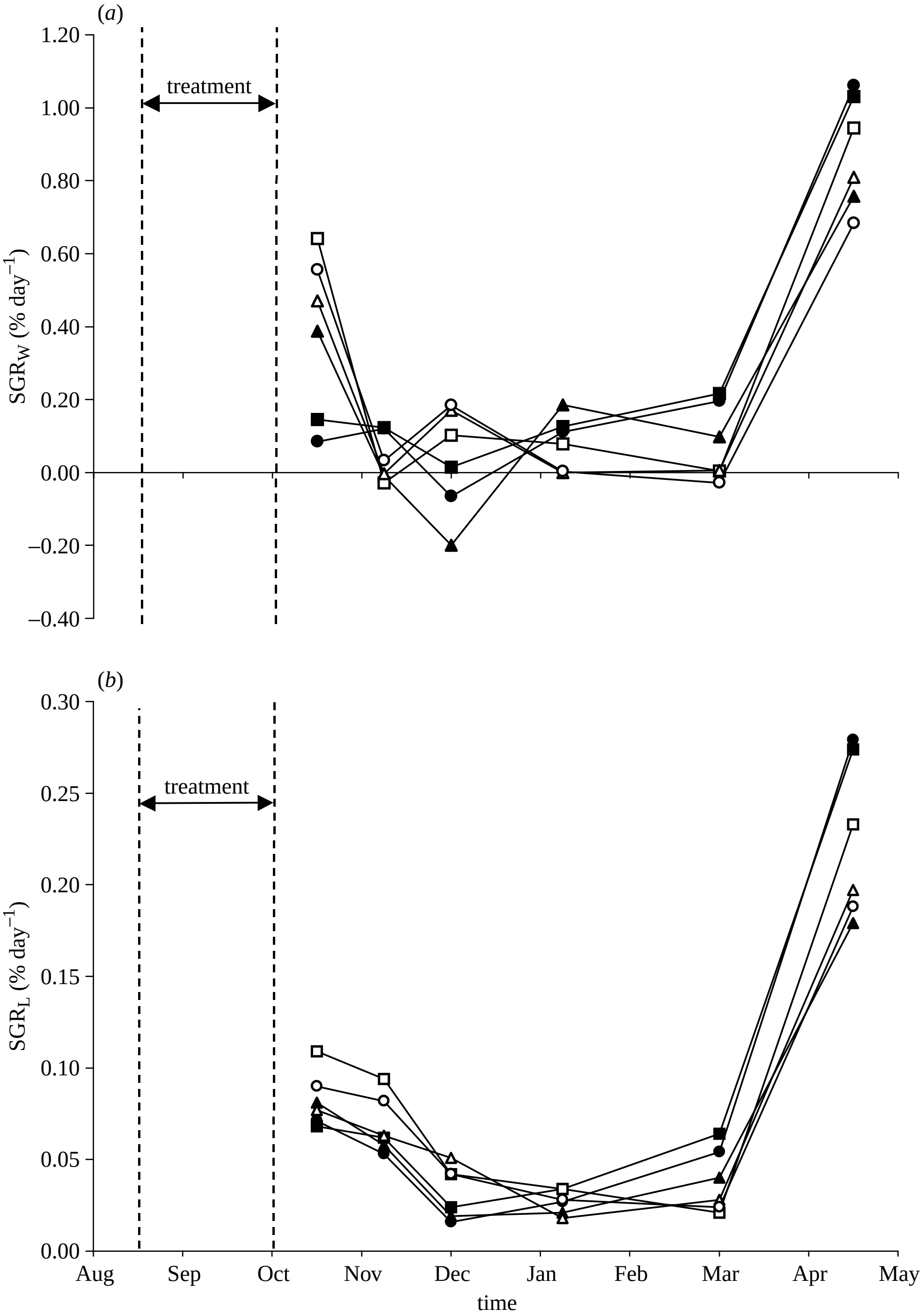

Figure 2. Specific growth rates in terms of (*a*) weight, SGR$_W$, and (*b*) length, SGR$_L$, of juvenile salmon following temporary manipulation of food availability. Data are presented as in figure 1; see table 2 for statistical analyses.

during the phase of metabolic programming at the foetal or neonatal stage and the nature of the compensatory response in mammals is known to vary ontogenetically due to changes in the relative importance of hyperplasia and hypertrophy (Pitts 1986). Our results are more analogous to those of Birkhead *et al.* (1999), who found that while male zebra finches, *Taeniopygia guttata*, could compensate for stunting induced by a poor-quality nestling diet, and so attain normal adult body size and primary and secondary sexual traits, their life span was reduced by approximately 25%. Birkhead *et al.* (1999) suggested that the birds might maintain immune function and secondary sexual characteristics in order to increase short-term reproductive success, at the expense of longevity. In our study, it would appear that the short-term survival benefits to the low-food fish of restoring lipid to normal levels at the start of winter may have been traded off against long-term performance costs.

However, the cause of the long-term cost is not clear. Elevated growth rates, such as those observed during the compensatory period, have been shown to be costly (Arendt 1997), and they occurred at a time when the fish may have been metabolically stressed by the autumn–winter transition (Cunjak 1988). The late-winter divergence in lipid reserves between the treatment groups began to develop at a time when there were no differences

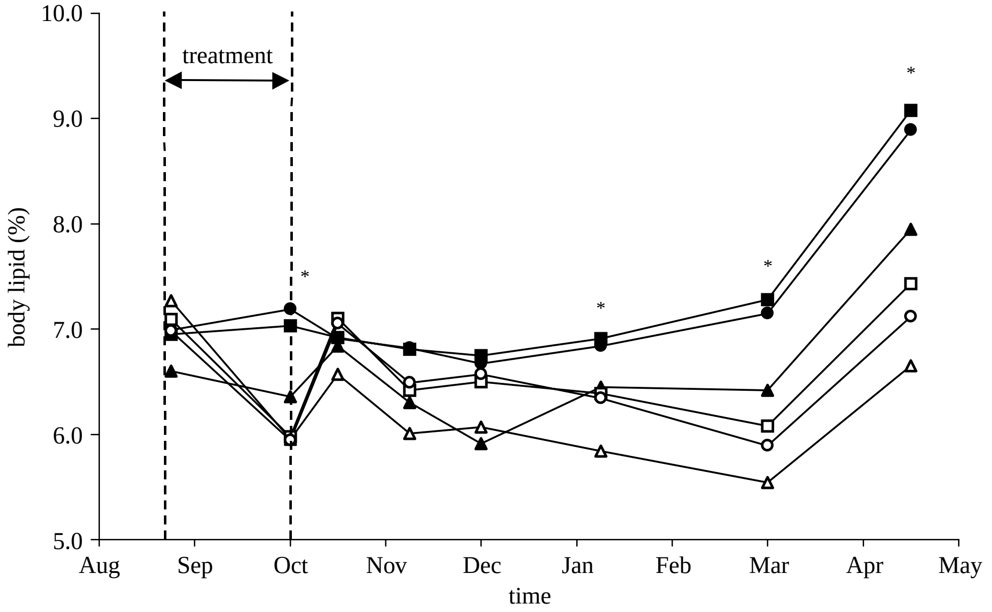

Figure 3. Lipid reserves of juvenile salmon following temporary manipulation of food availability (data are presented as in figure 1). Asterisks indicate a significant difference between control (filled symbols) and low food (open symbols) fish (three-way ANOVA, $p < 0.05$). See table 2 for other statistical analyses.

in food availability, food consumption or growth rates between them. This suggests that the low-food fish were allocating more energy than the controls to some other function(s). Fast-growing fish have low rates of protein turnover (McCarthy *et al.* 1994; Morgan *et al.* 2000) and hence it is possible that an increase in protein turnover following compensatory growth may have an energetic cost. Another possibility is that the basal metabolism or maintenance costs of the low-food fish were greater than those of the controls. However, food deprivation is known to suppress metabolism (O'Connor *et al.* 2000) and, indeed, one explanation for the increased growth rate and efficiency observed when food is again available after a period of shortage is that metabolism is slow to adapt to re-feeding (Jobling 1993). Moreover, it is difficult to envisage how changes in metabolism could explain the fact that the observed costs only begin to be seen months after the end of an apparent post-treatment recovery. Whatever the mechanism, it is apparent that animals may face trade-offs between long- and short-term gains when their typical growth trajectory is disturbed.

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As this paper exceeds the maximum length normally permitted, the authors have agreed to contribute to production costs.