Effects of food ration on SMR: influence of food consumption on individual variation in metabolic rate in juvenile coho salmon (*Onchorhynchus kisutch*)

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Summary

1. Consistency of differences in standard metabolic rate (SMR) between individual juvenile salmonids and the apparently limited ability of individuals to regulate their SMR has led many researchers to conclude that differences in individual SMR are fixed (i.e. genetic).
2. To test for the effects of food ration on individual performance and metabolism, SMR was estimated by measuring oxygen consumption using flow-through respirometry on individually separated young of the year coho salmon (*Onchorhynchus kisutch*) placed on varying food rations over a period of 44 days.
3. Results demonstrate that the quantity of food consumed directly affects SMR of juvenile coho salmon, independent of specific dynamic action (SDA, an elevation in metabolic rate from the increased energy demands associated with digestion immediately following a meal) and indicates that higher food consumption is a cause of elevated SMR rather than a consequence of it. Juvenile coho salmon therefore demonstrated an ability to regulate their SMR according to food availability and ultimately food consumption.
4. This study indicates that food consumption may play a pivotal role in understanding individual variation in SMR independent of inherent genetic differences. We suggest that studies involving SMR need to be cautious about the effects of intra-individual differences in food consumption in communal tanks or in different microhabitats in the wild as disproportionate food consumption may contribute more to variation in SMR than intrinsic (genetic) factors.
5. In general, our results suggest that evolutionary changes in SMR are likely a response to selection on food consumption and growth, rather than SMR itself.

Key-words: coho salmon, consumption, dominance, energy balance, growth depression, individual variation in standard metabolic rate, oxygen uptake, prey-mediated, standard metabolic rate, territoriality

Introduction

Standard metabolic rate (SMR) is the minimal maintenance metabolic rate of ectotherms in a post-absorptive state (Priede 1985; Beck & Gropp 1995). SMR (usually measured in terms of oxygen consumption) is an integrated measure of the physiological energy expenditures involved in tissue maintenance and organism homeostasis and is analogous to basal metabolic rate (BMR) in endotherms. Standard metabolic rate and BMR have been shown to vary up to half an order of magnitude between individuals of species ranging from fish (Cutts, Metcalfe & Taylor 2002; Alvarez, Cano & Nicieza 2006; Finstad *et al.* 2007) to mammals (Boily 2002; Speakman, Krol & Johnson 2004) to birds (Williams & Vezina 2001) with differences that are repeatable over time (Bech, Langseth & Gabrielsen 1999; McCarthy 2000; Seppänen, Piironen & Huuskonen 2010). The consistency of differences in metabolic rate between individuals and the apparent inability of individuals to regulate their metabolism (Priede 1985) has led many researchers to conclude that differences in individual BMR and SMR are fixed (i.e. genetic) (Roonying, Moe & Bech 2005; Pakkasmaa, Pentinnen & Piironen 2006), suggesting that this level of individual variation in baseline metabolism is adaptive.

Differences in SMR within salmon and trout populations have been linked to variation in individual growth, behaviour and life-history strategies, e.g. timing of smolt migration.
(Metcalfe, Taylor & Thorpe 1995; Cutts et al. 1999; Forseth et al. 1999; McCarthy 2001; Finstad et al. 2007). Metcalfe, Taylor & Thorpe (1995) and Cutts, Metcalfe & Taylor (2002) have shown that Atlantic salmon with a higher SMR were more likely to be dominant and that SMR was correlated with aggression, presumably because of the higher energetic demands associated with a higher maintenance metabolism. While it is clear that differences in SMR are associated with differences in life history and performance (e.g. growth), it remains unclear whether SMR is the cause of these differences (i.e. higher SMR is genetically fixed), or a consequence of them.

For example, there is evidence that SMR increases with food ration (O’Connor, Taylor & Metcalfe 2000; Van Leeuwen, Rosenfeld & Richards 2011); this makes physiological sense, as SMR is an integrated measure of anabolic and catabolic activity, and an increase in food consumption and growth should conceivably elevate SMR (West, Brown & Enquist 2001). Here we provide evidence that variation in SMR is influenced by differences in food consumption, which commonly varies in nature between habitats (e.g. pools vs. riffles; Rosenfeld & Boss 2001; Rosenfeld & Taylor 2009) and with rank in dominance hierarchies, where the dominant fish commonly receives more food (Sloman & Armstrong 2002). We examined the relative importance of food consumption and individual differences in metabolism to observed variation in SMR and tested the causation between SMR and food consumption by measuring oxygen consumption of separately reared young of the year (YOY) coho salmon (Onchorhynchus kisutch) placed on varying food rations over a period of 44 days. Objectives of the analysis were to determine (i) whether SMR is elevated under high-food ration; (ii) the size of a food consumption effect on SMR relative to intrinsic differences in SMR between individuals; and (iii) whether juvenile coho salmon are able to downregulate their SMR at very low food levels.

Materials and methods

FISH COLLECTION AND REARING

Young of the year coho salmon were obtained by seining and minnow trapping in Chapman Creek near Sechelt, British Columbia, Canada [Universal Transverse Mercator (UTM) 448100E 5478100N]. Fish were transported back to the University of British Columbia and held indoor under quarantine in a temperature-controlled environment chamber at 14 °C. Fish were housed in a 350-L flow-through tank supplied with dechlorinated tap water and acclimated together for approximately 3 months prior to experimentation. During the acclimation period, fish were fed ad libitum a combination of Aqua Pride Trout 48 : 16 pellets (Unified, Okotoks, Alberta; composition: crude protein 48%0, crude fat 16%, crude fibre 5%, sodium 0-4%, calcium 2-5%, phosphorus 1-5%) supplemented with a natural diet of earth worms.

EXPERIMENTAL PROTOCOLS

Forty fish were initially weighed and measured to the nearest 0.01 g and 0.1 cm, respectively (26 ± 0.5 g SD average weight), and stocked individually in one of twenty 114-L tanks that were divided transversely in half to optimize available space. Tanks were divided using a 1-mm-mesh partition, which allowed fish to see each other and permitted circulation of water in the tank. Food, however, was too large to pass through the screen divider ensuring each fish received the quantity of food appropriate for their treatment. Fish were housed separately to prevent the development of dominance hierarchies that are inevitable among fish held communally and can lead to large differences in food consumption even at a fixed food ration, as well as associated stress and agonistic behaviours that have all been shown to elevate SMR (Metcalfe, Taylor & Thorpe 1995; Cutts, Metcalfe & Taylor (1998)). Housing fish individually allowed us to control for these factors and isolate the effect of food consumption on SMR. Although Millidine, Armstrong & Metcalfe (2009) have shown that the visual presence of a dominant (i.e. when seen through a transparent barrier) is sufficient to alter SMR of subdominants, we assume that such an effect is likely small compared with the effects of changes in food ration associated with direct competition in a dominance hierarchy. An Aquaclear 200 filter was placed in the middle of each tank divider, and aquarium was vacuum siphoned weekly followed by a 50% water change to maintain water quality. A large rock with green flag tape attached (artificial plant) was added for enrichment. Fish were acclimated to a 12L : 12D cycle and fed ad libitum for 2 weeks after stocking and observed closely to ensure that fish were feeding and behaving normally with no visible signs of stress associated with being housed individually (e.g. not feeding, sluggish and lethargic).

FEEDING TREATMENTS

After the 2-week acclimation period, fish were again measured to the nearest 0.01 g and 0.1 cm and placed on an intermediate-food ration. The experiment was designed as a simple crossover trial (Jones & Kenward 2003) with two groups of fish that received different sequential food treatments over time (Fig. 1). The intermediate-food ration consisted of 40% of satiation fed once daily based on the fishes body weight using the satiation equation from (Brett 1971) for juvenile sockeye salmon [Consumption (% body dry weight) = 14.5 – (5.15 *Log10 (Wet weight of fish (g)))]. This was done to ensure that all fish received the same level of food based on the allometry of food consumption (smaller fish consume less food than larger fish to achieve an equal degree of fullness, but satiate at a much higher per cent of body weight). Food treatments consisted of 75% natural diet (chopped up earth worm) and 25% pellets (see above for composition) and were staggered to ensure that all fish were on each treatment for the same length of time prior to oxygen consumption measurements. Standard metabolic rate was measured after fish had been on the fourteen day intermediate-food treatment. Half of the remaining fish were then haphazardly placed on either a low-food diet that

Fig. 1. Schematic showing the sequence of food treatments throughout the experiment. Intermediate-food ration was 40% of satiation, whereas low food and high food were 1% body wt day⁻¹ and satiation fed once daily respectively, based on a consumption equation from Brett (1971).
consisted of 1% of body wt day\(^{-1}\) or a high-food diet (satiation) fed once daily based on the satiation equation described previously (Brett 1971). After an additional 10 days on each food ration, SMR, weights and lengths were measured immediately following respirometry measures. Food treatments were then switched, with the high-food group placed on the low-food ration and the low-food group placed on the high-food ration for a further 10 days (Fig. 1). Standard metabolic rate, weights and lengths were again measured at the end of the treatment. To assess the ability of fish to downregulate metabolism in the absence of food, fish were then placed on a starvation treatment for 10 days, at the end of which SMR, weights and lengths were again measured (Fig. 1).

Of the forty fish at the start of the experiment, twenty-two were available for final analysis because a number of fish escaped during transfer to metabolic chambers, failed to grow, escaped through the partition in their tank and were no longer individually identifiable or showed obvious signs of poor health (discolouration, not feeding, etc.) and were excluded from the analysis.

**MEASURING STANDARD METABOLIC RATE**

Aquaria holding fish were vacuum siphoned to remove any food and debris the evening before fish were to be placed in respirometry chambers. This was to ensure that fish had sufficient time to evacuate their guts and were unfed for twenty hours prior to oxygen consumption measurements; twenty hours post-feeding has been shown to be adequate for the SDA response to subside (McCarthy 2000; Cutts, Metcalfe & Taylor 2002). Specific dynamic action is an elevation in metabolic rate from the increased energy demands associated with digestion, immediately following a meal (Kleiber 1961; Jobling 1981; Alsop & Wood 1997) and is generally not considered part of SMR.

The morning following aquaria cleaning, fish were introduced into a flow-through respirometer set-up in the same environmental chamber at 14 °C in preparation for respiration measurements to be carried out the following night. Oxygen consumption was measured using flow-through respirometry by placing individual fish into one of three individual glass respirometry chambers, arranged in parallel, ten hours prior to oxygen consumption measurements. This allowed for simultaneous measurements on three fish to be carried out nightly. Respirometers were constructed of 130 cm lengths of 28-cm-diameter glass tubing, covered in black plastic to minimize fish activity during measurements (Cutts, Metcalfe & Taylor 2002). Measurements of background oxygen consumption for individual respirometry measurements were found to be negligible. Oxygen consumption was measured continuously overnight in complete darkness using a four-channel fibre optic oxygen meter (Foxy-Or125 microvalves to ensure that there was at least a 10% drop in oxygen tension between the inlet and outlet of each respirometer. Oxygen sensors; Ocean Optics, Dunedin, FL, USA) calibrated daily using nitrogen and aerated water at 14 °C. To minimize disturbance to fish when measuring oxygen consumption, three oxygen probes were placed individually into each of three low-volume (approximately 5 mL) in-line glass chambers receiving outlet water from each respirometer containing a fish. The fourth oxygen probe was placed into a larger sealed container, supplied with water from the header tank. This was done to ensure inflow water to the respirometers was indeed completely air saturated. An airstone in the header tank of the respirometer apparatus was used to keep inflow water fully saturated with air. Flow to each respirometer was adjusted using microvalves to ensure that there was at least a 10% drop in oxygen tension between the inlet and outlet of each respirometer. Flow was measured by collecting the outlet water for 60 s and weighing to the nearest 0.01 g periodically throughout the experiment. The rate of oxygen consumption was determined using the following equation (Ege & Krough 1914):

\[
MO_2(\text{whole}) = V_w \Delta C_{wO_2} \cdot bw^{-1}
\]

where bw is the mass of the fish, \(V_w\) is the flow rate of water through the respirometer and \(\Delta C_{wO_2}\) is the difference between the oxygen tension of inflow water into the respirometer and the oxygen tension of the outflow water. Concentration of oxygen was calculated by taking \(P_{O_2}\) (partial pressure of oxygen) corrected for barometric pressure and multiplying by \(\alpha_{O_2}\) (umol L\(^{-1}\) torr\(^{-1}\)), the solubility coefficient at the observed temperature.

Continuous oxygen consumption measurements were averaged over half-hour periods and plotted graphically (Fig. 2) to discriminate periods of complete rest (SMR) from spontaneous activity, which appeared as distinct spikes in the oxygen consumption trace. Activity level of fish, observed as distinct spikes in oxygen consumption during measurement, did not differ between food treatments, making differences in activity level an unlikely explanation for differences in SMR (Fig. 2). All fish regardless of treatment achieved periods of complete rest in excess of one hour at some point during the night. Standard metabolic rate was estimated using the lowest one-hour duration oxygen consumption values observed during each respirometry trial.

**DATA ANALYSIS**

Fish mass and length were measured at the end of each food treatment, immediately following respirometry measurements. Instantaneous growth rates of fish (per cent per day) over each of the four treatment intervals were calculated as \(\frac{\log_{10}(\text{final mass}) - \log_{10}(\text{initial mass})}{\text{days}}\).

![Fig. 2. The relationship between oxygen consumption rate and time for a single coho salmon on low (a) and high food (b), measured over an 18-hour period using flow-through respirometry. Note: activity level is similar between treatments with standard metabolic rate being achieved for a period between approximately 8–10 hours in between two bouts of spontaneous activity that correspond to the lights in the chamber being turned off and on.](image-url)
mass)/(duration) \times 100 (Ricker 1975). Mass and SMR data were log10 transformed to linearize data and meet assumptions of normality and homogeneity of variance.

We tested for the effects of food (ration; four levels – high, intermediate, low and starvation), fish mass, time (four intervals) and fish group on growth (% body weight per day) and SMR (\mu\text{mol g}^{-1} \text{ h}^{-1}) separately as a mixed model analysis of variance (ANOVA) using the MIXED procedure in SAS (Littell et al. 2006), including interaction terms between mass and food ration, and time and food ration. To account for autocorrelation of repeated measures on the same fish over sequential time periods, individual fish identity was treated as a random variable and covariance structure was modelled as either simple (independent observations with homogenous variance, the default in PROC MIXED), compound symmetric (observations on individuals covary) or autoregressive order 1 (covariance of observations is greatest in adjacent time intervals; Littell, Prendergast & Natarajan 2000). The covariance structure with the lowest AIC value was used in the final analysis (simple for SMR, autoregressive for growth). Mass was included as a covariate to control for any potential allometric effects of mass on SMR. Interaction terms and independent variables that were not significant at $P < 0.05$ were removed from the model.

In addition to the mixed model ANOVA described previously, we evaluated whether there was a difference in SMR between fish on the first high- and low-food treatment using a paired $t$-test. A $t$-test was appropriate because all fish were on the same food ration prior to half being placed on high- and low-food rations. We also tested for an overall positive relationship between average SMR in all treatment groups ($n = 8$) and food ration by regressing average SMR (log10 transformed) against estimated food ration as a per cent of body weight (also log10 transformed, calculated using equations from Sullivan et al. 2001). Lastly, we tested for a positive correlation between the average change in SMR between time intervals (i.e. increase or decrease) and the change in food ration between intervals for group treatment means ($n = 6$) using a one-tailed Wilcoxon rank sum test; the change in SMR for a given time interval was calculated by subtracting the mean SMR value in the previous interval, as with a paired $t$-test.

To assess whether individual fish had an SMR that was consistently higher or lower than the average in their treatment groups, we tested whether residuals (observed SMR subtract modelled SMR) from the mixed model ANOVA were significantly different from zero over the four time intervals for each fish ($t$-test, $n = 4$ observations per fish for 22 fish). As a more formal test, we also repeated the mixed model ANOVA described previously as a random coefficients model with empirical best linear unbiased predictors (Littell et al. 2006) to test for significant deviation in average SMR of individual fish from treatment means.

We also regressed individual SMR on growth using all data combined (irrespective of food treatment) to test for any apparent relationship between metabolism and growth for the entire data set, to simulate presentation of results from a typical observational study on SMR and growth. All analyses other than the mixed model ANOVA were conducted using R version 2.8.1 statistical software (R Development Core Team, Vienna, Austria).

**Results**

Mean weight of coho salmon ($\pm 95\%$ CI) at the beginning of the experiment was $4.29 \pm 0.81$ and $4.63 \pm 0.52\ \text{g}$ for the two groups and did not differ between groups throughout the experiment ($F_{1,64} = 0.85, P = 0.36$). As expected, there was a significant positive effect of food ration on growth ($F_{3,63} = 14.9, P < 0.001$; Fig. 3).

There was no effect of fish group on SMR ($F_{1,59} = 0.05, P = 0.83$), but there was a significant negative effect of fish mass on SMR ($F_{1,59} = 8.4, P = 0.005$) as expected based on allometry. There was also a significant effect of food ration on SMR ($F_{3,59} = 7.9, P < 0.001$), with fish on high- and low-food rations experiencing significantly elevated SMR relative to starved fish (Tukey-Kramer adjusted difference of least square means, $t_{59} = 3.47, P = 0.001$ for starved vs. high-food ration, $t_{59} = 2.78, P = 0.04$ for starved vs. low-food ration). Between the second and third time intervals, SMR increased more rapidly with elevated food ration than it declined with reduced food ration (Fig. 4), suggesting an asymmetry in the rate of SMR decrease or elevation depending on prior fish condition. Although we could not test this statistically with our design, it suggests a lag in down-regulation of anabolic and catabolic pathways that elevate SMR following a high-food ration.

Growth rate was significantly correlated with SMR for the whole data set ($R^2 = 0.17, P = 0.001, n = 20$), indicating that fish with a higher SMR had a higher growth rate (Fig. 5). The regression of log10 SMR on log10 food ration for treatment means ($n = 8$) was highly significant ($R^2 = 0.83, P = 0.002$) with a positive slope. The direction of change in SMR between time periods (i.e. average increase or decrease in mean SMR by treatment group) was significantly positively related to the direction of change in food ration between time periods (Fig. 4; $S = 11, P = 0.05, n = 6$, for a one-tailed Wilcoxon rank sum test).

Only one of twenty-two fish had SMR residuals from the mixed model ANOVA that were significantly different from zero ($t_{4} = -3.9, P = 0.025$) or intercepts (empirical best linear unbiased predictors) that were significantly different from zero ($t_{38} = -2.6, P = 0.015$); as this is approximately the number that would be expected by chance at $\alpha = 0.05$, this

![Fig. 3. Instantaneous growth rate of juvenile coho salmon during the experiment. Grey and black circles represent different groups of coho salmon placed on contrasting food treatments as illustrated in Fig. 1. Grey circle group went from mid food to low food to high food followed by starvation. Black circle group went from mid food to high food to low food followed by starvation. (error bars represent 95% CI).](image-url)
result provides little support for individual variation of SMR being large (or at least detectable) once the effects of food ration and weight are taken into account.

To determine whether our food ration levels achieved the desired degree of satiation, we back-calculated estimated consumption based on observed growth rate and temperature during the experiment using a bioenergetic model for coho salmon from Sullivan et al. (2001). Coho salmon on intermediate-food ration were estimated to be growing at 51% of satiation, whereas coho at high and low food were estimated to be growing at 69% and 30% of satiation, respectively.

**Discussion**

Our results demonstrate that the quantity of food consumed directly affects SMR of juvenile coho salmon and indicates that high food consumption is a cause of elevated SMR rather than a consequence of it. Not surprisingly, we also found that higher food ration resulted in higher growth rate, and consequently SMR and growth were positively correlated, with fish on the high-food treatment having the highest SMR and fish on the starvation treatment the lowest, consistent with earlier studies (e.g. O’Connor, Taylor & Metcalfe 2000). As higher food consumption and growth require an upregulation of anabolic pathways even when digestion is not taking place, it is not surprising that increased food consumption and growth elevate SMR independent of the costs of digestion (SDA). This positive relationship between SMR and growth has also been demonstrated in a number of earlier studies under high-food ration (Cutts, Metcalfe & Taylor 1998; Yamamoto, Ueda & Higashi 1998). Our results, however, indicate that the apparent relationship between SMR and growth illustrated in Fig. 5 and in previous studies may be spurious (i.e. does not indicate causation) and suggest that elevated growth and SMR are both a consequence of increased food consumption.

There is a strong theoretical basis for expecting SMR to increase with food consumption and growth (e.g. West, Brown & Enquist 2001). Cellular metabolism that contributes to SMR has two components – SMR associated with cell maintenance, which is assumed to be constant (mass-specific) as an animal ages and metabolism associated with growing new cells. When organisms are small, most of their SMR is dominated by growth rather than maintenance metabolism. As they approach maximum body size, maintenance and reproductive metabolism dominate; the classic allometric decline in mass-specific SMR is associated with a decline in the absolute contribution of metabolism associated with creating new tissue (i.e. growth; West, Brown & Enquist 2001).

If metabolism associated with growth is the main component of SMR for juveniles, then it follows that variation in food ration should be a significant source of variation in juvenile SMR. This is widely recognized among mammalian and avian physiologists, who commonly compare BMR of adult individuals (e.g. Bozinovic et al. 2009). The much larger size disparity among juvenile and adults fishes (relative to birds and mammals) likely amplifies any sensitivity of juvenile SMR to food ration.

Many earlier studies have documented large differences in SMR between individual juvenile salmonids (Cutts, Metcalfe & Taylor 2002; Alvarez, Cano & Nicieza 2006; Finstad et al. 2007), which were shown to be consistent and repeatable over time (McCarthy 2000; Seppanen, Piironen & Huuskonen 2010). In contrast with these studies, we could not detect a significant effect of individual fish identity on SMR when fish were held separately and fed individual food rations in the absence of competition. This may be due in part to low statistical power to detect individual differences in this study. However, our results indicate that any individual (fixed genetic) differences in SMR are relatively small compared with the effect of food ration, which was pronounced.

Previous research has shown that salmonids with a high SMR tend to be more dominant and aggressive relative to...
fish with low SMR (Metcalfe, Taylor & Thorpe 1995; Cutts, Metcalfe & Taylor 2002). Dominant salmonids generally outcompete lower SMR conspecifics for preferential feeding territories and access to food. This correlation between dominance and SMR has also lead researchers to identify high SMR as a cause of dominance (e.g. Alvarez, Cano & Nicieza 2006). Most metabolic studies, however, are carried out in the laboratory with fish housed in communal tanks, often at high densities leading to competition for food, particularly at food rations below satiation. Under these conditions, dominance hierarchies readily form, leading to large variation in food consumption, growth and body size (McCarthy, Carter & Houlihan 1992; Cutts, Metcalfe & Taylor 1998) with disproportionate food consumption and growth by more aggressive dominant fish. When fish were reared individually in this study, preventing the establishment of dominance hierarchies and allowing more precise control of individual food ration, we showed that SMR was elevated or lowered depending on food consumption, and we were not able to detect the effects of individual identity on SMR. While there is a genetic component to individual variation in SMR, this result suggests that increased food consumption in dominance hierarchies is likely a driver of much of the individual variation in SMR observed in earlier studies. As dominance hierarchies are typically size dependent and stable over time, they should result in consistent and repeatable measures of high or low SMR for individual fish. Therefore, we propose that a high SMR for fish reared in communal tanks may simply be a consequence of high food consumption associated with a dominant rank in a competitive hierarchy, rather than high SMR being a cause of dominance.

Social interactions in dominance hierarchies may influence SMR through effects on both food ration or induced stress responses. Interference competition by dominants potentially reduces both subdominant food ration and SMR, but the visual presence of a dominant may elevate SMR of subordinates (Millidine, Armstrong & Metcalfe 2009) and may slightly reduce their maximum food ration even in the complete absence of direct interference competition (Abbott & Dill 1989), presumably through an induced stress response (e.g. Gilmour, DiBattista & Thomas 2005; DiBattista et al. 2006). Because we did not directly manipulate dominance in our experiment (other than completely removing direct interference competition), we cannot differentiate the relative effects of interference competition and stress on SMR in dominance hierarchies. However, our results demonstrate that interference competition alone (i.e. reduction in subordinate energy intake by dominant fish) has the potential to cause a significant elevation in dominant SMR and SMR reduction in subordinates, and is consistent with a higher elevation of dominant SMR reported in the literature (e.g. Metcalfe, Taylor & Thorpe 1995; Cutts, Metcalfe & Taylor 2002).

It is likely that the correlation of SMR with food consumption that we observed in juvenile coho salmon also occurs in other salmonid species and potentially BMR of other vertebrate species as well and may be relevant to understanding interactions between growth and metabolism in diverse areas of ecology. For instance, there is some debate concerning the role of metabolism in mediating growth depression under predation risk. A number of studies (e.g. Steiner & Van Buskirk 2009) have demonstrated a depression in metabolic rate in the presence of long-term exposure to predators. This is usually attributed to a selected physiological response for minimizing the costs of anti-predator behaviour, such as increased shelter use and reduced foraging by the prey species. However, if the correlation between SMR and food consumption we observed is widespread, then decreased SMR and growth in the presence of predators may simply be a result of decreased food consumption associated with reduced foraging activity. Similarly, higher observed SMR of northern fish populations (e.g. Atlantic silversides), exhibiting counter gradient variation (Conover & Present 1990), may simply be a consequence of selection for higher food consumption and growth at higher latitudes as opposed to direct selection on SMR.

Our results also suggest that juvenile coho salmon have the ability to downregulate their SMR according to food availability and ultimately food consumption. If SMR were inflexible, salmonids with a high SMR would rapidly deplete their energy stores during prolonged food deprivation, as experienced in freshwater during overwintering in temperate climates (Shuter et al. 1980). Instead salmonids seem to show a capacity to regulate their SMR to maximize growth during periods of high food and minimize weight loss during periods of food deprivation (e.g. O’Connor, Taylor & Metcalfe 2000; Van Leeuwen, Rosenfeld & Richards 2011). However, there is no evidence that the reduction in SMR under food deprivation is a voluntary suppression of metabolism at low food; rather, decreased metabolism is consistent with an involuntary reduction in anabolic and catabolic processes associated with reduced food consumption and growth.

Juvenile coho growth in the high-food treatment was considerably lower than their physiological maximum. Back-calculated consumption based on a bioenergetical model for coho (Sullivan et al. 2001) indicated that fish in the high-food treatment were well below satiation, despite an intention to feed them to excess. Reasons for lower than expected growth and food consumption are unclear as fish did not appear stressed (e.g. not feeding and sluggish), but food consumption most likely would have been higher had fish been fed more than once daily. It would be expected that food consumption associated with reduced foraging activity would be expected growth and food consumption are unclear as fish did not appear stressed (e.g. not feeding and sluggish), but food consumption most likely would have been higher had fish been fed more than once daily. It would be expected that food consumption associated with reduced foraging activity would have been even more pronounced.

This study clearly demonstrates that food consumption affects SMR independent of SDA and that juvenile salmonids regulate their SMR in proportion to consumed food ration. We propose that microhabitat and dominance-mediated effects on food consumption as well as genetic differences play pivotal roles in individual variation in SMR. Therefore, we suggest that studies involving SMR need to be.

cautious about disproportionate food consumption because of formation of dominance hierarchies in communal tanks, as disproportionate food consumption may contribute more to variation in SMR than generally thought and may explain a significant amount of individual variation in SMR that is usually attributed to intrinsic (genetic) factors. While our conclusion that food ration affects SMR is consistent with the results of earlier studies, carefully designed experiments are necessary to better understand the relative contributions of genetic effects and food ration to SMR, which have hitherto been confounded in most studies with juvenile salmonids.

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References


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