Trade-off between growth rate and aggression in juvenile coho salmon, *Oncorhynchus kisutch*

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In juvenile salmon and trout, there seems to be a positive phenotypic correlation between individual aggression level and growth rate. Aggressive fish are dominant, and they obtain and defend territories, giving them access to good feeding sites. Being aggressive may increase predation risk, and may also carry costs such as increased metabolic demand, with effects on growth. To test the hypothesis that there is a trade-off between individual growth rate and aggression, we mated 12 female coho salmon with two unique males each, creating 24 full-sibling families. Growth of individually marked fish from each family was estimated in a situation where food could not be monopolized. Thereafter, individual fish were tested for mirror-elicited agonistic behaviour. We found significant variation between families in early growth rate, with a high heritability (1.04). There was also significant between-family variation in agonistic behaviour, but activity was generally low and heritability was low (0.25) and not significant. Growth rate and agonistic behaviour were negatively correlated. These results imply that aggressive behaviour has an energetic cost.

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subordinate brown trout, but nonaggressive individuals grew as fast as dominants (Højesjø et al. 2002).

Both growth rate and aggression level have a significant additive genetic component in salmonid fish (Bailey & Loudenslager 1986; Gjerde 1986; Rosenau & McPhail 1987; Taylor 1990; Riddell & Swain 1991; Vølestad & Lillehammer 2000). There are suggestions that more aggressive fish generally grow more rapidly, and Lahti et al. (2001) found evidence of positive genetic correlation between growth and aggression. This means that selection for one trait will lead to a correlated positive response in the other (through either pleiotropy or epistasis). However, the evidence for this correlation is indirect (Rosenau & McPhail 1987; Swain & Riddell 1990; Berejikian et al. 1996; Lahti et al. 2001). The hypothesis of a positive genetic correlation between aggression and growth rate has not been directly tested, and there are reasons to expect that the genetic correlation might be negative (Holm & Fernø 1986; Huntingford & Garcia de Leaniz 1997; Jonsson 1997). Aggressive behaviour has costs such as increased metabolic rate, more time is used on increased activity, and predation risk is increased. More aggressive individuals may also have higher standard metabolic rates than less aggressive ones (Metcalfe et al. 1995; Cutts et al. 1998; Lahti et al. 2002) and thus reduced metabolic scope for activity (Cutts et al. 2002). Our objective was to determine whether there is a genetic correlation between growth rate and aggression in juvenile coho salmon. We did this using a half-sibling mating design. In our experiment, all fish had equal access to food in excess; thus we limited the ability of fish to monopolize food (i.e. the benefits of being dominant). Our hypothesis was that the more aggressive fish would experience the inherent costs of being more active, and therefore have reduced growth rates.

**METHODS**

**Experimental Animals**

We used coho salmon from the University of Washington (UW) hatchery population. Mature adults migrate from the ocean into a holding pond where they are kept until ready to spawn. The fully mature fish are then spawned artificially, and the eggs and juveniles incubated and reared under controlled conditions. Standard salmon food is distributed in excess at relatively short intervals to maximize growth and minimize aggression.

**Experimental Set-up**

We experimentally mated each of 12 female coho salmon with two unique males, producing 24 full-sibling families. All females were the same age, having spent two summers at sea (range 920–3830 g). Most males were also of this age, but two had spent only one summer at sea (known locally as jacks). These jacks represent an alternative life history tactic, attempting sneak mating rather than to gain access to females by fighting (Gross 1985; Fleming & Gross 1994). The males ranged in size from 250 to 1750 g. Egg size, estimated by weighing a subsample of about 50 eggs from each female, was 0.126–0.242 g.

From 289 to 747 (mean 466) eggs from each female were dry-fertilized, and incubated in standard Heath stack incubators, at constant temperature (10 ± 1°C). Each full-sibling family was kept separate. During incubation, we monitored development and mortality every day. When the eggs reached the eyed stage, they were given a standard physical shock to identify dead or unfertilized eggs, which were removed. Thereafter, dead eggs were removed daily.

After the embryos had completely absorbed their yolk sacs (late January 2002), the fry from each full-sibling family were transferred to one of a set of randomly assigned, identical circular 4.5-litre flow-through tanks for feeding. The water temperature was kept at 10.6 ± 0.7°C. Fry were fed a standard commercial salmon feed in excess one to three times a day. The food was distributed evenly on the surface of the tank to minimize interactions during feeding and give all individuals equal access (Ryer & Olla 1996). On 25–27 February 2002, we reduced the density in each tank to 11 individuals to minimize density-dependent effects on growth.

Each individual was anaesthetized with MS222 and individually marked with unique combinations of a small dot of alcian blue dye applied with a panjet and a fin clip. Mark type did not significantly influence either survival (nominal logistic regression: Wald \( \chi^2_{10} = 13.21, P = 0.212 \)) or growth rate (ANOVA: \( F_{10,226} = 0.893, P = 0.541 \)). We also individually weighed (to the nearest 0.001 g) and measured (to the nearest 0.1 mm) the fry. They were then fed ad libitum for 30 days before being weighed and measured again to estimate individual growth rates. During this period, 22 fish died. Ten fish were lost during an accident with the water supply; the rest either jumped out of the tanks during the night or died for unknown reasons. To keep density constant within each tank, we replaced these fish with new individually marked fish. These extra fish were not used for estimating growth, nor were they used in the behavioural experiment.

We treated each fish as a replicate of a certain sire–dam combination, to estimate heritability. However, this may lead to the dam effect being confounded by tank effects. We randomly distributed the families between the tanks, thus reducing the importance of a nonrandom tank effect. For this reason, we feel confident that the sire effect is not confounded by tank effects. Juvenile salmonid fish are more aggressive towards nonkin than towards kin (Brown & Brown 1993, 1996), and this effect is most evident when odour concentration is heightened (Griffiths & Armstrong 2000). The juvenile coho in this experiment were exposed only to full siblings, and therefore should not have experienced elevated levels of aggression or variation in odour that might bias the results.

**Mirror Image Stimulation**

After the growth experiment was finished, we assessed individual aggression level by mirror image stimulation.
stimulation behaviour as our measure of aggressiveness when comparing individuals and families within the population. We estimated growth rate both as the specific growth rate ($G$, %/day), and by using an allometric growth model accounting for possible effects of fish size on growth rate ($\Omega$, %/g per day). $G$ was estimated as:

$$G = \frac{\ln {M_2} - \ln {M_1}}{\text{days}} \times 100$$

where $M_1$ and $M_2$ are mass at the start and end of the growth period, respectively. The standardized mass-specific growth rate ($\Omega$; Ostrovsky 1995) was calculated as:

$$\Omega = \frac{M_2^b - M_1^b}{b \times \text{days}} \times 100$$

where $b$ is the allometric mass exponent for the relation between growth rate and body mass. This exponent has not been estimated for coho salmon. However, estimates obtained for brown trout (0.308) and Atlantic salmon (0.310) are very close (Elliott et al. 1995; Elliott & Hurley 1997), suggesting that this exponent may be similar among salmonids. In our analysis we set $b=0.31$.

We used nested mixed-model ANOVA in the JMP statistical package to analyse the variation in early growth rate and behaviour (independent variables; $Y$) using the following model:

$$Y = \mu + \text{dam} + \text{sire (dam)} + \varepsilon$$

where $\mu$ is the mean response and $\varepsilon$ is the residual variance. The dam and sire (nested within dam) effects were set as random, and variance components were estimated by the restricted maximum likelihood method (REML). This method is less sensitive to imbalance in the data than the conventional ANOVA (Roff 2002). We used the sire variance component to estimate heritability ($h^2$) of each trait:

$$h^2 = \frac{4\sigma^2_{\text{sire}}}{\sigma^2_{\text{ser}} + \sigma^2_{\text{dam}} + \sigma^2_{\text{error}}}$$

Standard errors for the estimated $h^2$ were estimated following Roff (1997).

The genetic correlation ($r_g$) was estimated using the Pearson product–moment correlation between family means (Roff 2002). Standard errors were estimated using the Z transformation of the correlation coefficient ($r$):

$$Z = \frac{1}{2} \ln \left[ \frac{1 + r}{1 - r} \right]$$

The standard error is approximated by

$$\sqrt{\frac{1}{N - 3}}$$

where $N$ is the number of families. Standard error and 95% confidence limits on the $Z$ scale can then...
be estimated and back transformed to the \( r \) scale (Zar 1996):

\[
r = \frac{e^{2r} - 1} {e^{2r} + 1}
\]

Mean observed activity level (\( s \)) was ln transformed to normalize variance.

To test for a negative correlation between aggression level and growth rate we used logistic regression, with aggression and nonaggression coded as 1 and 0, respectively, as the response variable, full-sibling family as the main effect and growth rate as the covariate.

**Ethical Note**

The experiment was approved by the University of Washington Institutional Animal Care and Use Committee. After the fish had been used in the growth experiment, and observed in the swim against mirror experiment, they were released into holding tanks at the University of Washington hatchery. All surplus fish not used in the experiment were also released in the same way. The fish were then allowed to migrate to sea as part of the ordinary hatchery programme.

**RESULTS**

Mean size of the juveniles at the start of the growth experiment ± SD was 0.505 ± 0.175 g, with significant differences in size between families (one-way ANOVA: \( F_{23,236} = 14.00, P < 0.001 \)). There were also significant differences in the weight–length relation between families (ANCOVA on ln-transformed data: \( F_{23,211} = 3.32, P < 0.001 \) for the length x family interaction). There was, however, no significant linear relation between mean family egg size and mean juvenile size at the start of the growth experiment (length: \( r^2 = 0.089, N = 24, P = 0.156 \); mass: \( r^2 = 0.067, N = 24, P = 0.221 \)).

Estimated mean individual specific growth rate ± SD was 3.78 ± 0.77%/day (range 1.79–6.12%/day). The specific growth rate was negatively correlated with mass at the start of the growth experiment (\( r_{2.255} = -0.498, P < 0.001 \)). Mean mass-specific growth rate (\( \Omega \)) was estimated as 3.73 ± 0.77%/g per day (range 1.75–6.23%/g per day), and was not correlated with mass at the start of the experiment (\( r_{2.255} = -0.073, P = 0.261 \)). We therefore used this estimate of growth in the subsequent analyses.

There was a highly significant sire effect on mass-specific growth rate (Table 1), indicating significant additive genetic variance for this trait. The dam effect was not significant. The estimated heritability (\( h^2 ± SE \)) was 1.04 ± 0.34. Mass-specific growth rate was not correlated with egg size (regression on family means: \( r^2 = 0.036, N = 24, P = 0.451 \)), male size (\( r^2 = 0.005, N = 24, P = 0.741 \)) or male age (ANOVA: \( F_{1,22} = 1.829, P = 0.189 \)).

The majority (65.9%) of the individuals tested for aggression (\( N = 220 \) since some fish died during the growth experiment) did not respond to the mirror challenge, leading to a highly skewed distribution of total duration of LD and SAM. Mean duration of agonistic activity (LD+SAM) for all fish ± SD was 24.0 ± 58.3 s for 360 s of total observation. There were also highly significant differences between families (Kruskal–Wallis test: \( \chi^2_{23} = 55.7, P < 0.001 \)). Mean duration of agonistic activity for those 75 fish that responded to the challenge ± SD was 70.3 ± 82.2 s for 360 s of total observation. There was a weak sire effect on mean duration of agonistic activity (Table 1), indicating a weak but nonsignificant additive genetic variance for this trait. The dam effect was not significant. Heritability was estimated as 0.250 ± 0.138.

Assuming the family mean duration of agonistic activity (including all fish) is a good estimator of aggression, we analysed for a genetic correlation (\( r_g \)) between growth (\( \Omega \)) and aggression using product–moment correlation. Mean aggression level showed a nonsignificant correlation with growth rate (\( r_g = -0.360, 95\% \) confidence interval \(-0.681–0.076, P = 0.084 \)). The proportion of aggressive individuals varied significantly between families (logistic regression: \( \chi^2_{23} = 65.67, P < 0.001 \), and was negatively associated with growth rate (\( \chi^2_{23} = 4.51, P = 0.034 \); parameter estimate: \(-0.636 ± 0.306 \)). The genetic correlation was significantly negative (\( r_g = -0.468, 95\% \) confidence interval \(-0.745–0.053, P = 0.021; \text{Fig. 1} \).

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**Table 1. Summary results for nested mixed-model ANOVA**

<table>
<thead>
<tr>
<th>Effect</th>
<th>df</th>
<th>Sum of squares</th>
<th>( F )</th>
<th>Variance component estimate</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Growth rate</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dam</td>
<td>11</td>
<td>3.27</td>
<td>0.88</td>
<td>0.236</td>
<td>0.586</td>
</tr>
<tr>
<td>Sire (dam)</td>
<td>12</td>
<td>20.79</td>
<td>5.11</td>
<td>0.433</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Residuals</td>
<td>213</td>
<td>72.16</td>
<td>0.339</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aggression level</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dam</td>
<td>11</td>
<td>123.16</td>
<td>2.28</td>
<td>0.345</td>
<td>0.086</td>
</tr>
<tr>
<td>Sire (dam)</td>
<td>12</td>
<td>59.00</td>
<td>4.92</td>
<td>0.218</td>
<td>0.074</td>
</tr>
<tr>
<td>Residuals</td>
<td>196</td>
<td>573.67</td>
<td>2.927</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The dependent variables were growth rate (\( \Omega, \%/\text{day per g} \)) and aggression level (time spent in agonistic behaviour+1, ln transformed, \( s \)). The effects dam and sire within dam were given as random.
not mean that there is no additive genetic variance for aggression, merely that we were not able to document it. There were clearly differences in aggression level between families. This suggests the presence of some additive genetic variance for this trait, but that residual variance is high, producing a low estimate of heritability. This is commonly observed in studies of behavioural traits (Stirling et al. 2002). Most studies showing a genetic basis for variation in aggression have used population comparisons (Rosenau & McPhail 1987; Taylor 1990; Dunbrack et al. 1996; Lahti et al. 2001). Furthermore, some studies have documented differences in aggression between juveniles from wild and domestic strains of the same species (reviewed by Einum & Fleming 2001). Documentation of a significant population difference in aggression level does not give any indication about the level of heritability of the trait. The populations may have no remaining additive genetic variance and still be different because of historical differences in selection and drift. Furthermore, some of the observed differences between populations may be between-family differences, as we found. For example, Rosenau & McPhail (1987) documented inherited differences in agonistic behaviour between two populations of coho salmon but all individuals used in parts of the experiment were the progeny of one female and one male from each population. Given the between-family variation we observed, it is not possible to demonstrate between-population differences based on such a design.

Growth rate was negatively correlated with aggression level, showing that the two traits cannot be maximized at the same time under the test environmental conditions. This result was predicted by Martin-Smith & Armstrong (2002), who suggested that in an environment with pulses of high food availability which prevent food from being successfully defended, intake rate is similar among differently ranked fish (fish with different aggression levels). Less aggressive fish will then grow most quickly because of their low energy expenditure. This result is contrary to between-population comparisons showing a positive correlation between level of aggression and growth rate (Lahti et al. 2001). Furthermore, it is often found that aggressive and dominant juvenile salmonids grow better than less dominant individuals (Fausch 1984; Huntingford et al. 1998; Martin-Smith & Armstrong 2002). However, this is only the case when there is spatial variability in food availability and thus profitability. If there is no possibility to compete for and monopolize food, dominant fish will not necessarily grow faster. Furthermore, the subordinate fish (i.e. the less aggressive fish) will not incur the physiological cost (stress) associated with subordination (Abbott & Dill 1989). In our experiment, food was delivered in excess, and with equal access for all individuals. Such food delivery will not facilitate food monopolization. Thus, aggressive individuals will not benefit by being dominant, but will still have to pay the cost. The negative genetic correlation also suggests that, in a hatchery environment where there is selection for rapid growth, less aggressive individuals may be selected. This conclusion has also been reached using game-theoretical analyses (Doyle & Talbot 1986). Indeed, we did not find significant additive genetic variance for aggression level, and \( h^2 \) was low. However, this need

\[ \text{Figure 1. Family mean aggression level (arcsine-transformed proportion of aggressive individuals) versus family mean mass-specific growth rate (g/g per day) of juvenile coho salmon.} \]
in our experiment we observed a low aggression level, with most individuals showing no aggression in the mirror image test. The UW population of coho salmon has been maintained as a pure hatchery population since about 1967 (ca. 16 generations).

In our experiment, we first estimated individual growth rates and then aggression level. Fish with a high hunger level are often more active and aggressive than less hungry fish (Metcalfe et al. 1995), so the negative genetic correlation between growth rate and aggression could be an artefact of slow-growing fish having a different hunger level to fast-growing fish. However, all fish received food in excess at the same time and rate. Thus, fish with a generally high hunger level would be expected to eat more food and thus grow faster than fish with a lower hunger level. Randomly drawn individuals from all families were tested under identical conditions on the same days. We therefore find it highly unlikely that a systematic difference in hunger level between families would produce the negative genetic correlation reported here.

Another reason for the low aggression level in the mirror image test is the timing of the test. In tests with brown trout fry, 72% of young fry (1 month post-emergence) reacted to the mirror whereas only 10 of 120 older fry (5–6 months postemergence) reacted (Bohlin et al. 2002). This indicates that there is an increase in the ability to distinguish real competitors from ‘fakes’ with age. However, Berejikian et al. (1996) did not find any significant difference in the response of steelhead, *Oncorhynchus mykiss*, fry tested at 30 and 105 days post-emergence. There may be species-specific differences in how different-aged fish react to the mirror image challenge. However, no data are available on this subject. The coho juveniles used in our study were 3–4 months postemergence when tested.

In our experiment, we provided food in only one way. By distributing the food evenly and in excess the food could not be monopolized. Under these experimental conditions, a significant negative genetic correlation between growth rate and aggression level was found. We cannot determine whether the same result would have been found using another experimental design where the benefits of being aggressive (and possibly dominant) could be gained. There are no estimates of genetic correlations available for such an experimental design.

What are the costs of being aggressive? There may be a relation between aggression level or the probability of being dominant, and standard metabolic level (SMR). A number of studies have found that dominant individuals are more likely to have higher SMR than subordinates (Metcalfe et al. 1995; Cutts et al. 1998, 1999; Lahti et al. 2001). In a competitive situation, a high SMR may be beneficial if it allows for higher food intake and faster growth. However, if food intake cannot be increased, a high SMR will lead to reduced growth. Cutts et al. (2002) found that juvenile Atlantic salmon with relatively high SMRs have small metabolic scopes. Furthermore, aggressive individuals seem to have higher levels of growth hormone than less aggressive individuals (Jonsson et al. 1998). Growth hormone treatment also increases the ability to compete for food in pairwise tests, probably by increasing appetite and general feeding activity (Johnsson & Björnsson 1994; Johnsson et al. 1996). Again, this increased level of growth hormone will be beneficial only when it is possible to compete for food; otherwise it will incur costs. In a natural situation, aggressive individuals may incur a number of other costs not observed in the laboratory. Predation risk may be higher because of the higher activity of aggressive fish, and intense defence of a territory may mean less time is available for feeding. Thus it is necessary to study the trade-off between aggression and growth (as a proxy for fitness) in a more natural setting than the laboratory.

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**References**


