

Fat or lean? The quantitative genetic basis for selection strategies of muscle and body composition traits in breeding schemes of rainbow trout (*Oncorhynchus mykiss*)

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Abstract

Quantitative genetic analyses of fish composition have been strongly biased towards lipid deposition, rather than protein deposition. This is partly at odds with desires of the modern aquaculture industry, to improve the efficiency of lean growth. Using a quantitative genetic approach, we examined the selection potential in both protein and lipid components of wet weight growth in rainbow trout over a two-year growth period. Two diet treatments were applied to test the hypothesis that an experimental, high protein, low lipid diet (HP) would enhance selection potential compared to the current modern, normal protein, high lipid diet (NP). We found that lipid traits (lipid body weight, percent muscle and body lipid; $h^2=0.40$) were more heritable than corresponding protein traits (protein body weight, percent muscle and body protein; $h^2=0.18$), indicating a higher selection potential for lipid traits. The results revealed further that breeding for improved lipid composition over the whole growth period is easier than for protein composition. This was shown by the high favourable genetic correlations between differently aged fish for lipid traits. In contrast, the respective correlations for protein traits were low or even negative. Similarly, the genetic correlations between muscle and body composition were higher for lipid than for protein, enhancing selection efforts to change lipid traits. Heritabilities increased with age, implying that selection practiced on old (>800 g) rather than young (<60 g) fish should be more effective in achieving a compositional response. Although the diet had a significant effect on the composition traits, there was no general trend for diet differences in heritabilities of either protein or lipid traits. Thus, the hypothesis of increased selection potential on HP diet was not supported. In conclusion, lipid traits are both more variable and exhibit more favourable genetic architecture for selection compared to protein traits. © 2006 Elsevier B.V. All rights reserved.

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1. Introduction

Breeding programmes for farmed fish species have been successful in improving wet weight growth (Bolivar and Newkirk, 2002; Kause et al., 2005). There are several reasons why breeders should further control the degree to which lipid and protein components of wet weight growth change in fish. First, the composition of fish has an effect on both sensory and consumer-perceived quality, and the potential to change composition enables the aquaculture industry to adapt to increasing consumer demands on product quality. Second, body composition is a matter of production efficiency; fish farmers aim at converting feed into muscle growth rather than excess visceral lipid and waste. Historically, quantitative genetic studies of muscle and body composition in salmonids have focused more on lipid rather than protein traits (Gjerde and Gjedrem, 1984; Gjerde and Schaeffer, 1989; Elvingson and Johansson, 1993; Gjedrem, 1997; Kause et al., 2002; Neira et al., 2004; Quillet et al., 2005). This is partly at odds with desires of the modern aquaculture industry, to improve the efficiency of lean growth. For instance, in pigs, lean growth is extensively studied and meat percent is included in the selection indices of breeding programmes to improve the protein component of growth (reviewed by Clutter and Brascamp, 1998).

Fish growth is a dynamic process and the compositional components of growth vary throughout life. Most genetic analyses have focused on muscle and fillet composition at a single time point, typically in large size fish (reviewed by Gjedrem, 1997; Kause et al., 2002; Neira et al., 2004; Quinton et al., 2005). This approach is well justified because characteristics of large fish have been of greatest economic interest in these studies. However, globally, rainbow trout are harvested across a large range of body weights and age classes, from portion-sized 300 g fish to large 3 kg trout. Therefore, it is appropriate to assess the extent of the potential correlated genetic responses at various times throughout life when selection is only practiced at one discrete time point. Moreover, it is of interest to assess whether composition traits recorded from small fingerlings could be used as reliable predictors of larger market-size fish.

Here we assessed the potential to breed for both protein and lipid content over the whole growth phase of rainbow trout, *Oncorhynchus mykiss* Walbaum. Previous studies have typically shown moderate heritabilities for lipid but low heritabilities for protein content in salmonids (Gjerde and Schaeffer, 1989; Gjedrem, 1997; Kause et al., 2002; Quillet et al., 2005; Neira et al.,

2004). Consequently, our first hypothesis was that protein content of muscle and whole body display lower heritabilities than the respective lipid traits. Second, we hypothesised that correlations of body and muscle protein between fish of various age classes are likely to be higher and more stable compared to correlations between the lipid content traits. We based this hypothesis on the evidence that the protein component of growth in salmonids is less variable and under greater endogenous control and thus is less affected by exogenous factors than lipid content (Shearer, 1994; Jobling et al., 1998). Lipid deposition, in turn, is more readily influenced by factors such as age, growth rate, diet, feed intake, size and maturation (Grayton and Beamish, 1977; Jobling et al., 1998), potentially reducing correlations between ages.

We further assessed whether testing the same set of families under two nutritional environments, consisting of a modern normal protein, high lipid diet (NP) and an alternative high protein, low lipid diet (HP), would increase our understanding of the mechanisms of protein and lipid deposition. It was originally hypothesised that phenotypic and genetic variation in wet body weight should be higher on HP diet (Kause et al., 2006). The hypothesis is based on the idea that the low protein content of the NP diet restricts the protein component of growth (Kim and Kaushik, 1992), but that the excess lipid of the NP diet facilitates the lipid growth component in all fish. Due to the high lipid content, even inefficient fish (in terms of protein utilisation) are able to obtain considerable body weights, leading to low variation in growth on NP diet. On HP diet, in turn, variance in body weight is elevated because this feed does not allow inefficient fish to grow well. Due to the high protein content, the capacity of inefficient fish to digest and utilise protein is exceeded while the reduced lipid content does not support the lipid component of their wet-weight growth either, thus, both factors lead to their poor growth performance, and hence, to high variation on HP diet. Here this presumption is extended to include the protein and lipid components of growth [the effects of both diets on digestive ability in rainbow trout are reported by Rungruangsak–Torrissen et al. (unpublished)]. Hence, our third test was to assess whether or not protein and lipid traits would show diet-specific differences in heritabilities and phenotypic variation. These three hypotheses were tested by recording muscle and whole body composition as well as protein and lipid body weight in rainbow trout at three stages of growth, and by estimating diet-specific genetic parameters in a split-family design.

2. Materials and methods

2.1. Experimental animals and set up

All fish used in this study ($n=2931$ fish) originated from the Finnish national breeding programme and were housed at the Tervo Fisheries Research and Aquaculture Station, Finland, for the duration of the experiment. The pedigree for all fish was known through four generations back to 1989. The experimental fish were from 210 families, produced from 89 sires and 109 dams. Each sire was mated to an average of 2.3 dams (range: 1–5) and each dam to 1.9 sires (range: 1–3). Matings were completed over three days in April 2001. The broodstock fish have been selected for high body weight, late maturity age, good skin colour and body shape. The rate of inbreeding has remained low, the values being between 0%–0.7% per generation. The management and characteristics of the broodstock are detailed by [Kause et al. \(2005\)](#).

Full-sib batches of eye-staged eggs were transferred into indoor 150 L family tanks in June 2001. In February 2002, fish were individually tagged with PIT-tags (Trovan Ltd, Germany). For 165 out of the 210 families, an average of 7.0 fish per family were randomly tagged. For the remaining 45 families, an average of 39.6 randomly chosen fish were tagged. Large initial family size was needed for these 45 families because they were destructively sampled for muscle and body composition several times during growth. Prior to initiation of the experimental diets, all fish were fed with commercial rainbow trout dry food (Royal Response, Rehuraisio Inc., Finland).

During tagging, each family was randomly split into two groups to be reared on different experimental diets. In May 2002 (week 20) two dietary treatments were initiated ([Table 1](#)). The diets were a standard modern low protein (39.5–44.9%), high lipid (30.3–33.4%) diet (NP diet), and a novel experimental high protein (49.4–56.4%), low lipid (20.6–23.8%) diet (HP diet), which exceeds the

protein requirements of fish ([Cho, 1992; Hardy, 1996](#)). The weight at tagging of fish allotted to the two dietary groups was very similar (mean \pm SD; NP=62.4 \pm 19.9 g, $n=1355$ and HP=62.3 \pm 19.4 g, $n=1335$). Fish fed each diet were distributed to four replicate 20 m³ indoor test tanks (fish density <20 kg/m³) after an initial 8-month period in 150 L family tanks. Families were equally distributed among the test tanks. Feeding was automated using computer-controlled pneumatic feeders (Arvo-Tec Inc., Finland) and fish were fed to satiation 4 h a day. Water temperature during the experiment was natural and exposed to seasonal fluctuations.

2.2. Traits recorded

A total of 28 traits were recorded across three time points; in February 2002 before the initiation of the diet treatments (time 1, at weight ~60 g), and after the diet treatments were started in November 2002 (time 2, at ~800 g) and November 2003 (time 3, at weight ~2200 g). The recording time is indicated by a subscript in a trait name. At times 1–2 only the 45 families were sampled for the composition analyses, while at time 3 all remaining fish from all 210 families were sampled ([Table 2](#)).

Percent muscle protein and lipid (percentage of fresh matter) were recorded at each of the three sample times (traits: muscleprot% and musclelipid%). Percent body protein and lipid (percentage of fresh matter) (bodyprot% and bodylipid%) were recorded twice at times 2 and 3. To account for lipid deposited on the viscera, percent visceral weight (excluding gonads; visce%=100 \times weight of viscera/wet body weight) was recorded once, at time 3. To calculate protein and lipid body weight, individual wet weights recorded at times 2 and 3 were multiplied by the respective protein and lipid body percentages. Sample sizes for the traits are given in [Tables 3–5](#).

To record muscle composition at times 1, 2 and 3, approximately 10 g of epaxial white muscle was dissected just above the lateral line. For body composition at time 2, the fish were first weighed ungutted. The carcass was then

Table 1

Proximate composition of the normal protein (NP) and high protein diets (HP) over the course of the experiment. 3, 6 and 7 mm pellets were fed from May 2002, August 2002 and July 2003, respectively

Feed, pellet size	Moisture (%)	Ash (%)	CL (%)	CF (%)	CP (%)	NFE (%)	P (g/kg)	Energy (kJ/g)
NP, 3 mm	2.10	7.40	30.50	0.73	44.90	15.10	12.10	23.60
NP, 6 mm	1.50	7.70	30.30	0.72	44.60	15.90	12.70	25.95
NP, 7 mm	4.00	6.70	33.40	1.00	39.50	15.40	9.60	25.88
HP, 3 mm	2.30	9.60	20.70	0.44	56.40	11.10	15.50	25.63
HP, 6 mm	1.70	9.50	20.60	0.48	56.30	11.90	15.40	24.03
HP, 7 mm	7.20	8.30	23.80	1.30	49.40	10.00	11.80	23.26

CL=crude lipid; CF=crude fibre; CP=crude protein; NFE=nitrogen-free extracts; P=phosphorus.

Table 2
Population structure and average family size of a total of 210 full and half-sib families sampled for composition analysis

	165 families	45 families
Sires, dams	81, 99	34, 40
Family size, time 1	–	4.8
Family size, time 2	–	10.1
Family size, time 3	3.8	8.6

Larger family sizes for 45 families were planned to accommodate terminal composition analysis.

Time 1 was at February 2002 (at weight ~ 60 g), time 2 was at November 2002 (~ 800 g), and time 3 was at November 2003 (~ 2200 g).

minced and homogenised. Each muscle and body sample was stored at $-30\text{ }^{\circ}\text{C}$ until analysis. After sample homogenisation in standard solvent (Mirasolve, Miris AB, Sweden) with a Losmixer (Miris AB, Sweden), lipid and protein in white muscle and whole body were determined using mid-infrared transmission spectroscopy, as described by Elvingsson and Sjauna (1992), using a FMA2001 Milk Analyser (Miris AB, Sweden). Standard analytical methods for lipid (Folch et al., 1957) and nitrogen (Kjeldahl, 1883) were used for constructing calibration curves between single wavelength absorption of infrared light and concentration of the target substance. Due to technical problems, analyses of lipid and protein of approximately 200 and 800 samples at time 2 (whole body) and 3 (white muscle) respectively, had to be conducted using an INFRATEC Food and Feed Analyser (Tecator, Sweden), which is also based on infra red technology. Special care was taken to match the results using the two different analysers by running a number of replicate controls.

Body composition of all fish at time 3 was predicted by multiple regression models. First, a subsample of 200 fish randomly chosen among all the fish remaining at time 3 were determined for their true body composition.

Second, a set of predictive traits was recorded from all fish remaining at time 3. The predictive traits were protein, lipid and water percent from body chop (3-cm thick cutlets were cut directly from behind the dorsal fin from each fish), as well as body weight, head weight, visceral weight and gonad weight ($n=1015$ fish). Third, using the data for 200 fish, true body composition (body protein% and lipid%) was regressed against the predictive traits, to obtain predictive regression models listed in the Appendix. Finally, using these regressions, the predicted protein and lipid body percentage was calculated for all fish and used in further analyses.

2.3. Statistical analysis of diet means

To examine differences between the diets in the trait means, parametric analyses of variance were performed (procedure PROC MIXED, SAS Institute). The fixed effects included in the model were diet, sex/maturity class (males mature at 2, 3, and later years, females mature at 3 and later years, fish with unknown sex and maturity age), and interaction of diet with sex/maturity. The random factors included were test tank nested within diet, family (this consists of both common environment effect and genetic effect of full-sib families), interaction of test tank with sex/maturity, interaction of family with diet, and interaction of family with sex/maturity class. To take into account the data structure, the method of Kenward and Roger (1997) was used to calculate correct standard errors of diet means, F -tests and their degrees of freedom for the fixed effects. Using test tank as a random factor ensures that the number of test tanks primarily determines the denominator degrees of freedom for the F -tests. Protein weight₂, lipid weight₂, and musclelip%₃ were log-transformed to obtain normally distributed residuals.

Because body weight is known to correlate with composition traits, differences between diets in muscle

Table 3
Sample sizes (n), means and their standard errors (se) for protein traits on the two diets

Trait	Unit	Normal protein			High protein			df_1	df_2	F	P -value
		n	Mean	se ^a	n	Mean	se ^a				
ProtWeight ₂	g	227	76.1	-3.05 +3.18	225	73.7	-2.85 +2.96	1	10.3	0.35	0.57
Bodyprot% ₂	%	228	9.81	±0.32	226	10.1	±0.30	1	10.9	0.48	0.50
Muscleprot% ₂	%	197	20.6	±0.12	202	20.5	±0.11	1	8.30	1.09	0.30
ProtWeight ₃	g	416	414	±13.6	482	403	±13.5	1	6.34	0.34	0.58
Bodyprot% ₃	%	416	15.5	±0.05	482	16.1	±0.04	1	7.90	112	<0.01
Muscleprot% ₃	%	467	23.3	±0.42	532	25.2	±0.41	1	6.22	10.8	0.02

Degrees of freedom for nominator (df_1) and denominator (df_2) and P values of F -test for the differences in diet means are given.

^a Means and SEs for ProtWeight₂ were back-transformed to original scale after the analysis of log transformed values, giving different SEs for lower (-) and upper (+) tails of the distribution.

Table 4
Sample sizes (*n*), means and their standard errors (se) for lipid traits on the two diets

Trait	Unit	Normal protein			High protein			<i>df</i> ₁	<i>df</i> ₂	<i>F</i>	<i>P</i> -value
		<i>n</i>	Mean	se ^a	<i>n</i>	Mean	se ^a				
LipidWeight ₂	g	227	154	-4.40 +4.53	225	130	-3.63 +3.73	1	9.28	21.1	<0.01
Bodylipid% ₂	%	228	19.0	±0.22	226	17.0	±0.22	1	7.24	50.2	<0.01
Musclelipid% ₂	%	199	6.78	±0.31	202	6.48	±0.30	1	12.9	0.51	0.49
LipidWeight ₃	g	416	600	±23.1	482	516	±22.9	1	6.20	6.81	0.04
Bodylipid% ₃	%	416	22.3	±0.18	482	20.6	±0.18	1	6.53	44.9	<0.01
Musclelipid% ₃	%	467	8.78	-0.25 +0.26	532	6.79	-0.19 +0.20	1	6.35	44.2	<0.01
Visce% ₃	%	463	10.1	±0.12	536	8.20	±0.12	1	7.07	119	<0.01

Degrees of freedom for nominator (*df*₁) and denominator (*df*₂) and *P* value of *F*-test for the differences in diet means are given.

^a Means and SEs for LipidWeight₂ and Musclelipid%₃ were back-transformed to original scale after the analysis of log transformed values, giving different SEs for lower (-) and upper (+) tails of the distribution.

and body composition traits were further corrected for differences in body weight. This was performed by running additional models in which wet body weight at the recording time was included as a covariate in the model. For musclelipid%, bodylipid% and visce% at times 2 and 3, the interaction of body weight with diet was significant, and it was also included in the models.

2.4. Genetic analysis

(Co)variance components were estimated using the DMUAI software. The software analyses multivariate mixed models using the restricted maximum likelihood method, and accounts for all relationships between all animals in the pedigree using a relationship matrix (Jensen et al., 1996). The model for muscle composition at time 1 before the initiation of the diet treatments was:

$$y_{ijk} = \text{anim}_i + \text{famtank}_j + \text{SEXMAT}_k + e_{ijk}, \quad (1)$$

where anim_i is a random genetic effect of an animal ($i=1..$ number of observations), famtank_j is a random family tank effect ($j=1-210$ tanks), SEXMAT_k is a fixed sex and maturity effect ($k=1-6$), e_{ijk} is the residual, and y_{ijk} is an observation of an individual.

A trait measured on the two diets was regarded as two different traits. The model for the diet-specific traits recorded after the initiation of the diet treatments was:

$$y_{ijkl} = \text{anim}_i + \text{famtank}_j + \text{SEXMAT}_k + \text{TESTTANK}_l + e_{ijkl}, \quad (2)$$

where TESTTANK_l is a fixed test tank effect ($l=1-4$ tanks per diet). When two traits were not measured from the same individuals, residual covariance was set to zero.

Using these models, genetic (V_A), common environment (V_{famtank}), residual (V_R) and phenotypic variances ($V_P = V_A + V_R + V_{\text{famtank}}$), as well as phenotypic (r_P) and genetic correlations between traits (r_A) were obtained. Heritability was calculated as $h^2 = V_A/V_P$ and common environment ratio as $c^2 = V_{\text{famtank}}/V_P$. If the common environment ratio was lower than 0.01, the family tank effect was removed from the model of that trait. To compare phenotypic variation of traits with different means (x), coefficient of phenotypic variation was calculated as $(CV_P = \sqrt{V_P}/x)$. Conditional genetic variation (V_A^*) was estimated to determine the genetic variance in the composition traits that was independent of the genetic variation in body weight (Kause et al., 2002). This was calculated as follows, $V_A^* = V_{A1} - \text{COV}_{12}^2/V_{A2}$, where V_{A1} and V_{A2} are the initial genetic variances for a body composition trait and body weight respectively, and COV_{12} is the initial genetic covariance between the two traits. Then, conditional heritability (h^{2*}) was calculated as $h^{2*} = V_A^*/V_P$.

3. Results

3.1. Differences in trait means

The lipid traits were strongly affected by the diet treatments. Protein traits were also affected but to a lesser extent (Tables 3 and 4). None of the protein traits

Table 5
Phenotypic variances (V_P), heritabilities (h^2), common environment ratios (c^2) and their standard errors for muscle composition measured at sampling 1, before diets started

Trait	<i>n</i>	Mean	V_P	h^2	se	c^2	se
Muscleprot% ₁	217	19.20	1.01	0.00	0.21	0.15	0.13
Musclelipid% ₁	216	3.50	1.96	0.14	0.28	0.07	0.14

measured at time 2 differed significantly between the diets (Table 3). Diet effects on protein traits appeared only at measurement time 3. High protein feed (HP) resulted in fish with higher muscle and body protein percent at time 3. There was no significant difference in protein weights between diets at any time point (Table 3), showing that an increase in dietary protein did not result in an increase in protein retention.

In contrast to protein traits, lipid traits were strongly affected by the diets (Table 4). Fish fed with low protein and high lipid NP diet deposited significantly more lipid. This was shown by the significant diet effects for all lipid traits, except muscle lipid%₂ (Table 4).

NP fish were heavier compared to HP fish (Kause et al., 2006). When wet body weight was included as a covariate in the statistical models, the results for diet differences in protein traits remained the same. This shows that for protein traits, the difference in body weights between diets did not influence protein traits. For lipid traits, the case was more complex. Visual inspection of the significant body weight-by-diet interactions showed that at low body weights, lipid traits were of similar magnitude on both diets. In contrast, with increasing body weight, fish on NP diet displayed higher lipid trait values than fish on HP diet (results not shown). Thus, lipid traits standardised to common body weight were higher for NP fish only for heavy, and not light, fish.

In general, these results confirm that our diet treatments functioned as expected, creating a firm experimental set-up for the subsequent genetic analysis.

3.2. Genetic variation

As hypothesised, lipid traits displayed higher heritabilities (average h^2 across all lipid traits equals 0.40) compared to those of corresponding protein traits (average h^2 across all protein traits equals 0.18), irrespective

of diet (Tables 5–7). For instance, the average heritabilities for percent muscle and percent body protein were 0.06 and 0.25, and for percent muscle and body lipid 0.22 and 0.48, respectively. This indicates a higher selection potential for lipid traits.

In general, there was a trend to increasing heritabilities with age. Muscle composition was the only trait that was recorded during three time points, and the average heritabilities across diets for percent muscle protein at times 1, 2 and 3 were 0.00, 0.03, 0.12 and for percent muscle lipid 0.14, 0.08 and 0.41, showing that the highest heritabilities were observed at time 3 (Tables 5–7). The increase observed for percent body protein and protein weight over time was greater than that for percent muscle protein. Average heritabilities across diets for lipid traits also increased in time, although, unlike protein traits, the increase was more dramatic between times 1 and 2 (0.14 to 0.41) with subsequent stabilisation between times 2 and 3 (0.41 to 0.43). This was partly due to heritabilities for percent body lipid and lipid weight traits being already high at time 2 ($h^2 \geq 0.54$) (Table 7).

No great differences between diets were observed for the amount of variation. In contrast to the hypothesis, heritabilities for protein traits were marginally higher on NP diet (average $h^2=0.20$) than on HP diet (average $h^2=0.18$), although, with overlapping confidence limits (Table 6). For lipid traits (average $h^2=0.43$ and 0.42 on NP and HP), the difference in heritabilities between diets was similar to that between protein traits. Coefficients of phenotypic variation (CV_P) for protein and lipid traits showed weak or non-existent differences between diets. The largest differences in CV_P were observed for protein and lipid body weights, the values being consistently higher on NP diet (Tables 6 and 7).

Conditional heritability values demonstrated that genetic variation for composition traits was equal or lower when the genetic variation of body weight was taken into account (Tables 6 and 7). However, the general trends

Table 6

Phenotypic variances (V_P), coefficients of phenotypic variation (CV_P), heritabilities (h^2), their standard errors (se) and conditional heritabilities (h^{2*}) for protein traits on both diets

Trait	Normal protein					High protein				
	V_P	CV_P	h^2	se	h^{2*}	V_P	CV_P	h^2	se	h^{2*}
ProtWeight ₂	737	33.4	0.13	0.11	–	600	32.4	0.06	0.11	–
Bodyprot% ₂	8.57	29.4	0.07	0.10	0.00	8.92	30.1	0.15	0.12	0.07
Muscleprot% ₂	1.28	5.5	0.06	0.11	0.06	1.75	6.5	0.00	0.11	ne
ProtWeight ₃	7325	20.4	0.34	0.11	–	5417	18.1	0.43	0.11	–
Bodyprot% ₃	0.23	3.1	0.39	0.12	0.36	0.28	3.3	0.39	0.10	0.39
Muscleprot% ₃	13.30	15.7	0.19	0.10	0.15	11.10	13.2	0.06	0.07	ne

ne=non-estimable. Low heritabilities for protein trait heritabilities do not enable the estimation of the genetic correlation between composition and body weight and thus the conditional heritability.

Table 7

Phenotypic variances (V_p), coefficients of phenotypic variation (CV_p), heritabilities (h^2), their standard errors (se) and conditional heritabilities (h^{2*}) for lipid traits on both diets

Trait	Normal protein					High protein				
	V_p	CV_p	h^2	se	h^{2*}	V_p	CV_p	h^2	se	h^{2*}
LipidWeight ₂	1106	21.5	0.62	0.17	–	553	18.1	0.54	0.17	–
Bodylipid% ₂	2.42	8.2	0.59	0.15	0.56	2.07	8.5	0.54	0.16	0.14
Musclelipid% ₂	9.14	44.4	0.13	0.14	0.13	9.39	45.7	0.03	0.11	ne
LipidWeight ₃	19302	23.0	0.32	0.11	–	9839	19.4	0.40	0.11	–
Bodylipid% ₃	1.61	5.7	0.32	0.11	0.32	1.60	6.2	0.46	0.10	0.39
Musclelipid% ₃	5.79	27.0	0.40	0.11	0.39	3.53	28.4	0.41	0.10	0.31
Visce% ₃	2.61	15.8	0.62	0.11	–	1.27	14.0	0.54	0.10	–

ne=non-estimable. Low heritability for musclelipid%₂ does not enable the estimation of the genetic correlation between this trait and body weight and thus the conditional heritability.

observed for uncorrected heritabilities in the differences between protein and lipid traits, between differently aged fish and between diets remained unaltered.

3.3. Re-ranking of families across diets

Where estimable, the genetic correlations between diets were all positive and high, with estimates higher than 0.85 recorded in all cases (detailed values not shown). The strongest genetic relationships ($r_A > 0.9$) were observed for percent muscle and body protein, protein weight at time 2, and for percent muscle and body lipid. For all genetic correlations, the standard errors of the correlations included unity. Hence, the re-ranking of families was very weak across the diets.

3.4. Correlations over time

In contrast to our hypothesis, correlations of composition traits recorded at different ages revealed stronger positive correlations for lipid traits compared to protein traits. Genetic correlations between protein weights at

times 2 and 3 were weakly negative or moderately positive but the standard errors were high (NP: -0.13 ± 0.58 ; HP: 0.44 ± 0.59). The only estimable genetic correlation between percent muscle protein was between times 2 and 3 on NP diet, and the estimate was close to zero (Table 8). The genetic correlation between percent body protein recorded at time 2 and 3 was either zero (NP diet) or strongly negative (HP diet) (Table 8). Our inability to estimate the other muscle protein correlations was due to the close-to-zero heritabilities of these traits.

In contrast to protein weight, the correlations between lipid weight at times 2 and 3 were positive and close to unity (NP: 0.81 ± 0.23 ; HP: 0.97 ± 0.11). For percent muscle and body lipid, the genetic correlations between ages were positive and moderate-to-high on both diets (Table 9). For percent muscle lipid, the genetic correlations ranged between 0.19 to 0.48, but none of these estimates reached significance. For percent body lipid the genetic correlations between time 2 and 3 were higher than 0.75 and with low standard errors (Table 9) indicating weak re-ranking of families across both time points.

Table 8

Correlations between protein muscle and body composition traits

	Muscleprot% ₁	Muscleprot% ₂	Muscleprot% ₃	Bodyprot% ₂	Bodyprot% ₃
<i>Normal protein</i>					
Muscleprot% ₂	ne	•	.	-0.01	.
Muscleprot% ₃	ne	-0.03 ± 0.50	•	.	0.11
Bodyprot% ₂	ne	0.02 ± 0.80	0.19 ± 0.63	•	.
Bodyprot% ₃	ne	-0.97 ± 0.46	0.20 ± 0.30	0.00 ± 0.63	•
<i>High protein</i>					
Muscleprot% ₂	ne	•	.	-0.09	.
Muscleprot% ₃	ne	ne	•	.	0.00
Bodyprot% ₂	ne	ne	-0.27 ± 0.81	•	.
Bodyprot% ₃	ne	ne	-0.02 ± 0.50	-0.67 ± 0.30	•

Phenotypic correlations above and genetic correlations (\pm standard error) below the diagonal.

ne=non-estimable due to low heritability.

Table 9
Correlations between lipid muscle, body and visceral composition traits

	Musclelipid% ₁	Musclelipid% ₂	Musclelipid% ₃	Bodylipid% ₂	Bodylipid% ₃	Visce% ₃
<i>Normal protein</i>						
Musclelipid% ₂	0.33±0.48	●	.	0.08	.	.
Musclelipid% ₃	0.45±0.29	0.33±0.36	●	.	0.58	0.08
Bodylipid% ₂	0.14±0.30	0.21±0.36	0.82±0.17	●	.	.
Bodylipid% ₃	0.28±0.36	0.66±0.41	0.67±0.14	0.77±0.19	●	0.24
Visce% ₃	-0.07±0.32	0.50±0.40	0.07±0.20	0.18±0.24	0.20±0.20	●
<i>High protein</i>						
Musclelipid% ₂	0.19±0.65	●	.	0.14	.	.
Musclelipid% ₃	0.29±0.28	0.48±0.60	●	.	0.45	0.04
Bodylipid% ₂	0.11±0.29	0.20±0.59	0.76±0.17	●	.	.
Bodylipid% ₃	0.24±0.28	0.24±0.58	0.78±0.14	0.99±0.11	●	0.23
Visce% ₃	0.49±0.32	0.73±0.76	0.37±0.21	0.33±0.25	0.25±0.19	●

Phenotypic correlations above and genetic correlations (±standard error) below the diagonal.

3.5. Correlations between composition traits

The correlations between muscle composition and body composition were more favourable for lipid traits than for protein traits. Correlations between percent muscle and body protein were low or even negative (Table 8). Phenotypic correlations between percent muscle and body protein were low (from -0.09 to 0.11) and the majority of the genetic correlations between percent muscle and body protein were between -0.27 and 0.20 with large standard errors. The only exception was the negative genetic correlation between percent muscle protein at time 2 and percent body protein at time 3 on NP diet (-0.97±0.46) (Table 8).

In contrast to protein traits, most of the phenotypic correlations between percent muscle lipid and percent body lipid were positive and moderate, ranging from 0.08 to 0.58 (Table 9). On both diets both phenotypic and genetic correlations between percent muscle and percent body lipid increased over time (time 2: $r_P=0.08$ to 0.14, $r_A=0.20$ to 0.21; time 3: $r_P=0.45$ to 0.58, $r_A=0.67$ to 0.78). Moreover, on both diets there were strongly positive genetic correlations of percent muscle lipid at time 3 with both percent body lipid at times 2 and 3 ($r_A \geq 0.67$). Apart from strong positive albeit insignificant genetic correlations between percent muscle lipid at time 2 and visceral percent at time 3 on both diets ($r_A \geq 0.50$), there was no apparent genetic relationship between visceral percent and the remaining lipid traits.

3.6. Relationship between protein and lipid

The relationships between protein and lipid traits were dynamic, changing with time. For muscle and body composition on both diets, phenotypic and genetic correlations between protein and lipid percent tended to

be positive in younger smaller fish but strongly negative in older larger fish (Table 10). For muscle composition, the phenotypic correlation between protein and lipid percent was positive at time 1 ($r_P=0.20$), but switched to negative or close-to-zero at time 2 (from -0.26 to -0.01) and to moderately negative at time 3 (from -0.40 to -0.23). The same trend was observed for the phenotypic correlations between protein and lipid percent in the whole body. At time 2, genetic correlations between protein and lipid for muscle or body traits varied from positive to negative, albeit with large standard errors. By time 3, genetic correlations between percent protein and lipid in both muscle and body were all strongly negative and mostly significant, ranging from -0.77 to -1.0 (Table 10).

Table 10
Genetic correlations (r_A), their standard errors (se) and phenotypic correlations (r_P) between protein and lipid traits for both diets

Protein trait	Lipid trait	r_A	se	r_P
Muscleprot% ₁	Musclelipid% ₁	ne	ne	0.20
<i>Normal protein</i>				
Protweight ₂	Lipidweight ₂	0.85	0.27	0.43
Bodyprot% ₂	Bodylipid% ₂	0.54	0.72	0.04
Muscleprot% ₂	Musclelipid% ₂	-0.50	1.46	-0.26
Protweight ₃	Lipidweight ₃	0.89	0.05	0.94
Bodyprot% ₃	Bodylipid% ₃	-1.00	0.06	-0.37
Muscleprot% ₃	Musclelipid% ₃	-0.89	0.15	-0.40
<i>High protein</i>				
Protweight ₂	Lipidweight ₂	0.99	0.75	0.34
Bodyprot% ₂	Bodylipid% ₂	0.92	0.39	0.09
Muscleprot% ₂	Musclelipid% ₂	ne	ne	-0.01
Protweight ₃	Lipidweight ₃	0.87	0.05	0.90
Bodyprot% ₃	Bodylipid% ₃	-0.98	0.06	-0.38
Muscleprot% ₃	Musclelipid% ₃	-0.77	0.76	-0.23

ne = non-estimable.

On both diets, protein and lipid weights were strongly and positively correlated at phenotypic and genetic levels during both recording times ($r_p=0.34$ to 0.94 ; $r_A=0.85$ to 0.99) (Table 10). This result indicates that individuals and families with high protein component growth also had high lipid component growth.

4. Discussion

4.1. Genetic variation

We found that averaged heritabilities and conditional heritabilities for lipid traits were higher than for protein traits, indicating a higher selection potential for the lipid traits. This observation was consistent with our hypothesis based on previous studies. The observed heritabilities for lipid traits were similar in magnitude to previous findings for salmonids, showing moderate to high heritabilities (Elvingson and Johansson, 1993; Rye and Gjerde, 1996; Gjedrem, 1997; Kause et al., 2002; Neira et al., 2004). Moreover, our findings were in line with previous studies on percent muscle protein, where heritabilities have been lower compared to those of lipid percent (Gjerde and Schaeffer, 1989; Kause et al., 2002). However, moderate to high heritabilities for percent body protein and protein weight for large 2.5 kg fish, indicate that considerable genetic variation is available for selective breeding of these specific traits. Although protein percent is known not to vary much through life (Kiessling et al., 1991; Shearer, 1994; Jobling et al., 1998), high heritabilities for percent body protein and protein weight can still be expected. This is because there is genetic variation for body weight, fillet percent and percent dressing loss, all of which contribute directly to variation in percent body protein and protein weight. All in all, the current results together with the fact that rapid growth is related to increased fat deposition (Elvingson and Johansson, 1993; Gjedrem, 1997; Kause et al., 2002; Neira et al., 2004) suggest that much of the difference in growth capacity between families is because of a difference in the capacity for lipid deposition. If selection in genetic improvement programmes is solely based on wet weight gain, it is inevitable that the population will develop in the direction of more fatty fish.

We deduced that selection on older larger fish rather than younger smaller fish should be more effective in eliciting a selection response. Averaged heritabilities of lipid and protein traits were higher for older larger fish (>800 g, time 2 onwards) compared to smaller fish (<100 g, time 1), verifying that selection on large fish should be more effective. It is suggested that the

increased heritabilities for lipid and protein traits were associated with the increase in lipid deposition as the fish grow. The family differences may be expressed more strongly when fish obtain high levels of lipid deposition. This trend has also been observed in several other domesticated animals. Hassen et al. (2003, 2004) found that heritabilities for both fat and lean measures (percent intramuscular fat and longissimus muscle area) in Angus cattle increased as a function of age. In his review of genetic parameters of sheep traits, Fogarty (1995) reported a similar finding across several sheep breeds for fat depth traits, as trait heritability increased from post weaning to hogget. Further to this, Lambe et al. (2004) found that heritabilities for carcass and intramuscular fat traits in sheep varied seasonally, being highest when approaching mating season when ewes had optimal feeding resulting in best body condition and high fat deposits. These findings further highlight the fact that heritabilities for composition traits are not static, but vary with age and body condition.

The expression of genetic variation of a trait may change as a result of exposure to an alternative environment (Falconer, 1952; Charmantier and Garant, 2005). We hypothesised that protein and lipid traits would show diet-specific differences in both heritabilities and phenotypic variation. Counter to expectations, the genetic correlations between diets were strong for all traits ($r_A>0.85$) and there was no general trend for diet-induced differences in heritabilities of either protein or lipid traits. Thus, we found that increasing the protein content and decreasing the lipid content of the diet does not provide any supplementary selection potential for compositional traits.

It should be noted that trait means were strongly influenced by the diets. Moreover, in an accompanying study, we demonstrated re-ranking of families across these same diets for feed intake and, to a lesser extent, for wet body weight (Kause et al., 2006). Hence, the experimental set-up was effective for the study of genotype-by-diet interactions.

4.2. Correlations over time

From previous studies of rainbow trout we know that strong positive genetic correlations exist between differently aged fish for traits such as wet weight and body appearance (Elvingson and Johansson, 1993; Kause et al., 2003, 2004). Similar to these findings the current results revealed that re-ranking of families across ages for lipid content was weak. This was in stark contrast to our findings for protein traits, where significant re-ranking of families was observed. These findings are in

contrast to our initial hypothesis. Unfortunately, to the best of the authors' knowledge, there is a lack of comparative data for other fish species to confirm if re-ranking of protein traits is unique to rainbow trout and further research on other species is required to authenticate this. However, it is clear from our findings that for rainbow trout, changing lipid content over the whole growth through selection would be more successful than changing protein content. When selecting at one time point, favourable correlated genetic changes are thus expected at all other ages for muscle and body lipid percent, although, to a lesser degree for muscle lipid. This finding is consistent with reports from other production animals. For example, in his review of genetic parameters of sheep, Fogarty (1995) reported an average genetic correlation of 0.6 collated from several studies correlating fat depth measurements in sheep at post-weaning, yearling and hogget stages.

Recalling that heritability for percent muscle lipid at time 1 was less than 0.15 but 0.40 to 0.41 at time 3, and that the genetic correlations between percentage muscle lipid at time 1 and all subsequent time points were only moderate (0.19 to 0.45) with large standard errors, it is not advisable to use fingerlings to select for increased muscle content. This further emphasises that selection potential for flesh quality traits would be maximised by focusing on market-sized fish. Unfortunately, the genetic architecture for protein traits was less favourable compared to lipid traits and genetic correlations for protein traits were difficult to estimate reliably between measurement times. Thus, there is no reason to suspect that using young fish to select for muscle and body protein percent would confer a strong breeding advantage.

4.3. Correlations between different body locations

Similar to the correlations across age groups, the genetic correlations between muscle and body composition were higher for lipid than for protein. Moreover, muscle and body lipid percent were more strongly interrelated in large fish when lipid deposition was high. These correlations are a result of part-whole relationship, and are consistent with the previous observations on the increasing importance of muscle as a lipid storage location as fish grow (Jobling and Johansen, 2003). Several authors have observed positive relationships between body and muscle lipid traits in salmonids (Elvingson and Johansson, 1993; Kause et al., 2002; Neira et al., 2004). However, the genetic relationship between different visceral lipid stores and body or muscle lipid has been weak or even negative (Gjerde

and Schaeffer, 1989; Kause et al., 2002). This is consistent with our finding that for large market sized fish, visceral percent (which consists mostly of lipid reserves) correlated weakly with both percent muscle and percent body lipid. Hence, selective breeding for flesh quality should not be followed by a strong correlated response in visceral lipid storage. Consequently, it is advisable to breed simultaneously for muscle lipid and visceral lipid stores, to efficiently control for lipid deposition. The former is more closely related to product quality itself, whereas the latter to production efficiency because visceral lipid storage is often considered as waste. Similarly, in broilers, Zerehdaran et al. (2004) showed that lipid at different body locations was only weakly correlated, enabling a reduction in abdominal fat without adversely decreasing intra-muscular lipid.

4.4. Relationship between lipid and protein

Phenotypic and genetic correlations between protein and lipid percentage traits recorded from large fish (time 3) suggest that the traits are negatively linked, a condition previously documented in rainbow trout by Gjerde and Schaeffer (1989). Thus, selection for improved protein percent (or decreased lipid) would result in a decrease in lipid percentage (or increase in protein). This is a strong reflection of the fact that both percentage protein and lipid are components of a closed system and a change in one trait will necessitate a corresponding change in the other, and it is a condition that has been reported for other animal species (Eisen, 1989; Fogarty, 1995). It should be noted, however, that the relationship of protein and lipid percentage was age dependent, the correlations being positive in younger fish but strongly negative in older fish. This suggests that within individuals, protein and lipid deposition are not alternative strategies of wet weight growth when fish are small.

Both phenotypic and genetic relationships between protein and lipid weight were consistently strong and positive on both diets. Any attempt to improve protein weight will lead to an increase in lipid weight. These are gross reflections of whole body trends, as large fish tend to be both protein and lipid heavy. In farm animals, it has been observed that selection for lean growth results in a decreased rate of lipid component growth demonstrating that they are alternate growth strategies (Clutter and Brascamp, 1998). It has been further shown in sheep, that to achieve the best improvement in lean growth one must simultaneously include both lean weight and fat weight in the selection strategy rather than adopting an independent strategy of selecting purely for lean weight (Jones et al., 2004).

4.5. Selection strategies to improve components of growth

Considering the pivotal importance of the protein growth component of fish for aquaculture and consumers, it is surprising to note that there has been such a paucity of quantitative genetic data for protein traits in fish. Previous work in fish has instead paid more attention to lipid traits (Gjerde and Schaeffer, 1989; Kause et al., 2002; Neira et al., 2004; Quillet et al., 2005). This study aimed to provide baseline genetic data that could be used to assess the selection potential for a number of protein and lipid traits and the associated responses between composition traits over the whole growing period in farmed fish. Our findings show that, in general, relative to lipid traits, there is limited potential to improve protein content traits through selection in rainbow trout. At least five factors make selection of lipid traits more attractive compared to protein traits.

First, unlike protein, a strong genetic correlation exists between growth and lipid deposition, with sole selection for rapid growth leading to increased lipid deposition, which should be avoided (Elvingson and Johansson, 1993; Gjedrem, 1997; Kause et al., 2002; Neira et al., 2004). Second, intestine lipid and abdominal lipid on the belly flap are removed when dressing and filleting fish, and thus, breeders are under a strong economic pressure to decrease intestine and abdominal lipid. Third, collection of data measuring the protein content of muscle and body is costly and laborious, and more difficult to determine from live fish compared to the lipid content of muscle and body. Fourth, many of the protein traits measured here were only weakly to moderately heritable and subsequent genetic correlations over time were low or non-estimable. Instead, there is sufficient genetic variation in lipid traits which guarantee that they will respond well to selection. Fifth, because in large fish, protein and lipid percent were inversely related, selection for decreased lipid percent should result in an indirect increase in protein percentage.

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Appendix A. Multiple regressions for whole body composition at time 3

Whole body protein and lipid concentrations at time 3 were estimated using multitrait regression models. The potential predictor variables were protein and lipid content of the chop, and weights of body, entrails, gonads and head. The models were fitted as general linear models with the potential predictor variables and the final models were selected on the basis of incremental parameters and the extra sums of squares principle (*F*-test). All models were estimated with the R language (R Development Core Team, 2005) and its Design library (Harrell, 2001).

For whole body protein percentage (Bodyprot%₃) the prediction equation was:

$$\begin{aligned} \text{Bodyprot}\%_3 &= 6.71(1.50) \\ &+ 0.63(0.06)\text{Chopp}\%_3 \\ &- 0.058(0.02)\text{Choplip}\%_3, \end{aligned}$$

where Chopp%₃ is percent chop protein and Choplip%₃ percent chop lipid. Parameter standard errors are in parentheses. All parameters differed significantly from zero ($P < 0.05$). The residual standard error of the model was 0.5049, and adjusted R^2 0.58.

For whole body lipid (Bodylip%₃) the corresponding equation was:

$$\begin{aligned} \text{Bodylip}\%_3 &= 8.39(1.22) \\ &+ 0.58(0.05)\text{Choplip}\%_3 \\ &- 0.023(0.004)\text{Head}_3 \\ &+ 0.005(0.002)\text{Entrails}_3 \\ &+ 0.001(0.0005)\text{Weight}_3, \end{aligned}$$

where Choplip%₃ is percent chop lipid and Head₃, Entrails₃ and Weight₃ are the weights (g) of head, entrails and body, respectively. All parameters differed significantly from zero ($P < 0.05$). The residual standard error of the model was 1.156, and adjusted R^2 0.62.

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