

Quantitative Genetics Of Salmon Lice Resistance In Atlantic Salmon At Different Life Stages

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Introduction

During the last years it has been documented significant additive genetic variation in the resistance to the salmon lice (*Lepeophtheirus salmonis*) in Atlantic salmon (Glover, Aasmundstad, Nilsen et al., 2005; Kolstad, Heuch, Gjerde et al., 2005; Gjerde and Ødegård, 2010). Heritability estimates for lice resistance from a controlled infestation tests (Kolstad, Heuch, Gjerde et al., 2005; Gjerde and Ødegård, 2010) are found to be higher than those obtained from field tests (Kolstad, Heuch, Gjerde et al., 2005), most likely due to the higher infestation levels and a more stable environment. The genetic correlation between resistance in a controlled test with post-smolts and in natural infection with older and larger fish is seemingly high (0.88 ± 0.26 ; Kolstad, Heuch, Gjerde et al., 2005) and indicate that a controlled test can be used to accurately rank families as well as individuals with respect to lice resistance in a selective breeding program. However, this correlation was obtained from field data with low infestation levels (on average 1.2 lice per fish) and a relatively low number of families (50) in the infestation test. The main objective of this study was to obtain more reliable estimates of the genetic correlations between lice resistance measured at different life stages of the same individuals and to obtain the first estimate of the genetic correlation between the resistance to sessile and adult lice in Atlantic salmon.

Material and methods

Fish. Records were obtained from the 1764 offspring of 69 sires and 152 dams families) of the 2008 year-class from the breeding nucleus of SalmoBreed AS. The full-sib families were produced at AS Bolaks and were transported as eyed eggs to Nofima Marin, Sunndalsøra where they were kept in separate hatching trays and were startfed on February 18 (19 families) and 26 (134 families) and reared in separate tanks until a body size (mean = 28.6 g; SD = 8.3 g) for individually tagging with PIT-tags in August 2009. The tagged fish, 15 from each family, were kept in a common tank until April 24, 2009 when they were transported to Nofima Marin, Averøy where the fish were randomly divided on two separate 3 m diameter tanks with 1.8 m dept of seawater (Table 1).

First lice infestation. Egg sacs from mature salmon lice (*Lepeophtheirus salmonis*) were harvested from Atlantic salmon at Nofima Marin, Averøy. The eggs were incubated in small containers (1 L) and hatched from April 9 to April 14, 2009. On April 27 a total of 25400

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copepodids (newly hatched lice larvae) were added to tank 1 (on average 37.2 copepodids per fish including 130 rainbow trout) and 28300 copepodids were added to tank 2 (on average 26.2 copepodids per fish including 149 rainbow trout). Just prior to the lice infestation the water level in each tank was reduced to $\frac{3}{4}$ of normal level and the water flow stopped. After two hours the water level was increased to normal level, and thereafter stopped for another two hours. Sufficient oxygen was added through air stones during the four hours infestation period.

First lice count. On May 18 (tank 1) and 19 (tank 2) the number of sessile lice (chalmus II-III stage) per fish was counted on anaesthetised fish and their individual body weights recorded. On May 23 all fish in tank 2 died due to an algae bloom that blocked the water inlet. The fish in tank 1 were deloused and moved to a 125 m³ floating net cage.

Second lice infestation. Egg sacs from mature salmon lice were incubated and hatched from October 29 to November 6, 2009. On November 11 a total of 104,100 copepodids were added to the cage (on average 134 copepodids per fish including 130 rainbow trout). Just prior to the lice infestation the cage volume was reduced to about 50 m³ after which a tarpaulin was placed around the cage and oxygen was added to the seawater through air stones. After four hours the tarpaulin was removed.

Second lice count. On November 30 the number of sessile lice (chalmus II-III stage) per fish was counted on anaesthetised fish and the individual body weight of the fish recorded.

Third lice count. On January 18-21 the number of adult lice per fish was counted on individually anaesthetised fish in a white tub, All lice were removed from the fish, the fish weighted and removed from the tub and the number of pre-adult and adult lice in the tub was counted as well as the number of sessile lice (new settlements). The weight of each the fish was recorded. Thereafter the fish were deloused.

For each of the three recordings the lice density per fish was calculated as $LD = LC/Bw^{2/3}$ where LC is the total number of sessile (May 2009 and Nov 2009) or pre-adult plus adult (Jan 2010) lice per fish and Bw is the body weight of the fish. As most fish have similar proportions, $Bw^{2/3}$ is expected to be proportional to body surface.

Statistical model. Estimates of heritabilities and genetic correlations between the body weight and lice density traits were obtained using multivariate linear animal models:

$$y_i = X_i\beta_i + Z_i a_i + e_i$$

where y_i is the vector of the observations for trait i , β_i is a vector of the tank effect for body weight and lice count in May and the overall cage mean for the other traits; a_i is the additive genetic effect of each individual for trait i ; e_i is a vector of random residuals and X_i and Z_i are appropriate incidence matrices. Initially, an additional random effect common to fullsibs was also included but was found to be not significant for all traits and was therefore omitted from the final model.

Results and discussion

Descriptive statistics are given in Table 1. The variation in the lice count (LC) and lice density (LD) per fish was in particular large for sessile lice; with a coefficient of variation (CV) for LD of 78% and 68% for the two tanks in May 2009 and 80% in Nov 2009, while it was lower but also substantial for adult lice (CV=36%). The mean LC and LD values increased from Nov2009 (sessile lice) to Jan2010 (adult lice); most likely due to the very high average lice count for rainbow trout in November 2009 (99 and 0.53 for LC and LD, respectively) than in January 2010 (50 and 0.25 for LC and LD, respectively).

Tabel 1: Means and standard deviations (SD) for body weight (Bw), lice count (LC) and lice density ($LD = LC/Bw^{2/3}$) per fish at each recording.

Date	Tank /Cage	N	Bw, g		LC		LD	
			Mean	SD	Mean	SD	Mean	SD
May09								
Sessile lice	T1	683	201	54	14.2	12.9	0.40	0.31
	T2	1081	197	53	11.9	9.2	0.34	0.23
Nov09								
Sessile lice	C1	649	1411	354	12.4	10.1	0.10	0.08
Jan10								
Adult lice	C1	638	1615	419	19.8	7.4	0.14	0.05

Estimates of heritabilities and genetic and residual correlations are given in Table 2. The magnitude of the heritabilities for both sessile and adult LD coupled with the large phenotypic variation for LD (Table 1) show that there are substantial additive genetic variation in lice resistance in Atlantic salmon which is in accordance with earlier reports (e.g. Kolstad, Heuch, Gjerde et al., 2005; Gjerde and Ødegård, 2010).

Table 2: Estimates of heritabilities (on diagonal) and genetic correlations (above) and residual correlations (below) among the lice density ($LD = LC/Bw^{2/3}$) and body weight (Bw) traits at different life-stages of the fish and the lice¹.

	Lice density (LD)			Body weight (Bw)		
	May09	Nov09	Jan10	May09	Nov09	Jan10
LD						
May09	0.22±0.04	0.80±0.16	0.51±0.15	0.47±0.11	0.33±0.14	0.37±0.14
Nov09	0.02	0.19±0.07	0.87±0.12	0.33±0.17	0.18±0.20	0.23±0.20
Jan10	0.00	0.37	0.31±0.08	0.00±0.13	-0.12±0.17	-0.11±0.17
Bw						
May09	0.09	0.04	0.07	0.58±0.06	0.72±0.07	0.70±0.07
Nov09	0.01	-0.05	-0.13	0.55	0.46±0.09	0.98±0.01
Jan10	0.05	-0.02	-0.11	0.51	0.96	0.53±0.09

¹ Sessile lice in May09 and Nov09 and adult lice in Jan10.

The genetic correlation between sessile LD in May 2009 and November 2009 was high (0.80 ± 0.16) and is thus in agreement with the high genetic correlation between LC in a controlled infestation test and in natural infection (Kolsted, Heuch, Gjerde et al. (2005). Furthermore, the genetic correlation between sessile LD in November 2009 and adult LD in January 2010 was also high (0.87 ± 0.12).

These results strongly indicate that resistance to the salmon lice in Atlantic salmon measured at different life stages of both the fish and the salmon lice may be regarded as the same genetic trait. The genetic correlation between LD and body weight in May 2009 was significantly positive and medium in magnitude, and thus unfavourable. However, the genetic correlations between LD in May 2009 with body weights in November 2009 and January 2010 were lower (although not significantly lower), while correlations between LD in November 2009 and January 2010 and the corresponding body weights were not significantly different from zero. The latter is in agreement with Gjerde and Ødegård (2010) who found that the genetic correlation between sessile LD of post-smolt (mean body weight 260 g) and the harvest body weights of their sibs reared at two farms (mean body weight 4.6 and 5.0 kg) were not significantly different from zero.

Conclusions

These results show that there is substantial additive genetic variation in the resistance to the salmon lice in Atlantic salmon and that the resistance measured at different life stages of the fish and the lice may be regarded as the same genetic trait. This knowledge may be utilized in selective breeding programs to obtain increased resistance to the salmon lice in Atlantic salmon and thus provide significant economic gains in at least three ways: (a) The farmed salmon will require fewer treatments against salmon lice; (b) reduced risk of developing lice resistant to the medicaments presently used and to prolong the life time of these medicaments; and (c) reduced infestation pressure on wild salmonids. The first two is of value of the salmon farming industry while the latter is of value to the society and the environment.

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