

# Genetics of Social Interactions in Atlantic cod (*Gadus morhua*)

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## Introduction

Selection for high production efficiency in terrestrial animals is known to cause undesirable effects in traits like health and reproduction (Rauw, Kanis, Noordhuizen-Stassen *et al.* (1998)), and may affect adaptability, stress and animal welfare (Bakken, Vangen and Rauw (1998); Sandøe, Nielsen, Christensen, *et al.* (1999)) as well as the behaviour of farmed animals (Grommers, Rutgers and Wijsmuller (1995)). Intensifying the domestication process by more efficient selection against cannibalism and aggressive behaviour is particularly important when starting farming wild fish of a new species. In order to consider effects of current breeding practises on fish welfare and possibilities for taking fish welfare more directly into account in breeding programmes, we need estimates of heritability of relevant welfare traits and their genetic correlation with traits selected for today, e.g. growth. Mixed model methodology for studying competitive or aggressive behaviour affecting growth in fish exists (Muir and Shinkel (2002); Rutten, Bovenhuis, Komen *et al.* (2006)), and may be particularly important when improving domestication of new cannibalistic species such as cod. These models may be applicable for analyzing genetic variation of aggression and other welfare traits such as fin injuries and survival. A main advantage of this method is that there is no need for monitoring and recording of fish behaviour. Recent results in other species using such methods show that social interactions may be responsible for the majority of heritable variance for traits such as growth and survival, which implies that they deserve a key role in genetic improvement programs (Bijma, Muir, Ellen *et al.* (2007b); Bergsma, Kanis, Knol *et al.* (2008); Ellen, Visscher, van Arendonk *et al.* (2008)). For a breeding program aiming at improving fish welfare, it may therefore be necessary to consider both the direct genetic effect of an individual fish on its own performance (e.g. growth or survival) and the social effect of the individual fish on the performance of the other fish in the group. In chicken the heritable variation of survival when including social interactions is substantially larger than estimates obtained using classical methods (Bijma, Muir, Ellen *et al.* (2007b); Ellen, Visscher, van Arendonk *et al.* (2008)). This indicates that the classical selection methods, which ignore social interactions, may be less efficient than when accounting for social interactions. In some cases, they may result in undesirable selection response and possibly reduced fish welfare. Mixed models including both direct- and social additive genetic effects (e.g. aggression and cannibalism) on growth (Rutten, Bovenhuis, Komen *et al.* (2006); Bijma, Muir, and van Arendonk (2007); Bijma, Muir, Ellen, *et al.* (2007)) are promising for taking behavior (as social effects) into account without any direct recording of individual behavior.

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The objective of this study is to apply this methodology for modeling and analyzing genetic social interactions on growth and a more direct welfare trait, fin injuries to reveal possible genetic social interactions of Atlantic cod juveniles. Preliminary results of this study are presented in this paper.

## Material and methods

**Experimental Design.** Design of the experiment with number of fish and families per tank was determined after initial simulations and power calculations. As a result, a total of 2100 individually tagged fish from 100 full sib families originating from the Norwegian National Cod Breeding program were used. Each full-sib group was split into three sub-groups (7 individuals in each sub-group, 300 sub-groups in total). Each sub-group was administered into one of 100 available tanks. Three sub-groups was administered semi randomly in each of the 100 tanks, i.e., each family were stocked in three separate tanks. Recordings were conducted three times during the experiment; first recording at stocking (2<sup>nd</sup> and 3<sup>rd</sup> November 2009, weight<sub>1</sub> and length<sub>1</sub>), second recording two weeks after stocking (16<sup>th</sup> and 17<sup>th</sup> November 2009), and third recording four weeks later at the end of the experiment (14-16 December, weight<sub>3</sub> and length<sub>3</sub>). Traits recorded at each sampling appear in Table 1.

Body weight was measured to the nearest 0.1 g, body length to the nearest 0.1 cm. Fin erosion and deformities were scored subjectively on a scale from 0 % fin erosion to 100 % fin erosion in 5 % intervals on first dorsal fin (Erosion1) and the caudal fin (Erosion2). Due to low incidence of deformities (5%), deformities were classified as deformed or not deformed.

**Table 1: Mean body weight and length at the three recordings. Erosion1 (erosion of the first dorsal fin) and Erosion2 (erosion of the caudal fin) were only recorded at recording 3.**

	Body weight (grams)	Body length (cm)	Erosion1 (% erosion)	Erosion2 (%erosion)
Recording 1.	34.6 g	15.4 cm	-	-
Recording 2.	42.2 g	16.2 cm	-	-
Recording 3	63.5 g	18.3 cm	23,1 %	13,3%

The traits condition factor (1-3), changes in condition factor ( $\Delta CF$ ) and growth rate (GR) (from first until third recording) was derived according to the following formulas:

$$GR = \frac{weight_3 - weight_1}{weight_3}, CF_i = \frac{weight_i}{(length_i)^3}, \Delta CF = CF_3 - CF_1$$

The ASReml output gave the variance components for direct genetic effect ( $\sigma_{AD}^2$ ), social genetic effect ( $\sigma_{AS}^2$ ) and the covariance between the two ( $\sigma_{ADS}$ ). Variance of the total genetic ( $\sigma_{TBV}^2$ ) was calculated using the formula  $\sigma_{TBV}^2 = \sigma_{AD}^2 + 2(n-1)\sigma_{ADS} + (n-1)^2\sigma_{AS}^2$ , where n = 21 (number of fish per tank). Total heritable variation,  $T^2$ , was calculated as  $T^2 = \sigma_{TBV}^2 / \sigma_P^2$ .

**Analyses.** Genetic analyses were conducted using ASReml (Gilmour, Gogel, Cullis *et al.* (2006)) using linear animal models including a social genetic effect as proposed by Bijma, Muir, and van Arendonk (2007); Bijma, Muir, Ellen, *et al.* (2007). The model used was:

$$\mathbf{y} = \mathbf{X}\mathbf{b} + \mathbf{Z}_D\mathbf{a}_D + \mathbf{Z}_S\mathbf{a}_S + \mathbf{Z}_m\mathbf{f} + \mathbf{Z}_e\mathbf{t} + \mathbf{e},$$

where  $\mathbf{b}$  includes the fixed effects of population mean and deformities,  $\mathbf{a}_D$  is a vector of random direct additive genetic effects,  $\mathbf{a}_S$  is a vector of random social additive genetic effects,  $\mathbf{f}$  is a vector of random common full sib environmental effects,  $\mathbf{t}$  is a vector of random effects of experimental tank environments, and the  $\mathbf{X}$  and  $\mathbf{Z}$  matrices are the appropriate incidence matrices.

## Results and discussion

Estimates of genetic parameters for the traits  $\Delta$ CF, Erosion1, Erosion2, GR and Weight3 are shown in Table 2. The traditional heritability estimates of the traits  $\Delta$ CF, Erosion2 and GR were low. Weight3 (weight at the end of the experiment) showed a moderate heritability, while Erosion1 (erosion of the first dorsal fin) was very high. When including social effects the proportion of heritable variation of the phenotypic variation ( $T^2$ ) increased for all traits, although the estimated  $T^2$  was significant ( $P < 0.05$ ) for the two traits erosion1 and weight3, only. GR was the trait that was least affected by social components.

**Table .2: Genetic parameters of growth and fin erosion traits, including phenotypic variance ( $\sigma_P^2$ ), variance of the total breeding value ( $\sigma_{TBV}^2$ ), variance of the direct additive genetic effect ( $\sigma_{AD}^2$ ), heritability ( $(\sigma_{AD}^2)/(\sigma_P^2)$ ),  $T^2$  - proportion heritable variance of total variance ( $\sigma_{TBV}^2/\sigma_P^2 \pm$  s.e).**

	$\Delta$ CF	Errosion1	Erosion2	GR	Weight3
$\sigma_P^2$	0.17	213.4	63.04	0.132	416
$\sigma_{TBV}^2$	0.03	283.5	29.76	0.016	239
$\sigma_{AD}^2$	0.01	177.18	0.058	0.015	154.6
$h^2$	0.05 ( $\pm 0.04$ )	0.83 ( $\pm 0.13$ )	0.08 ( $\pm 0.07$ )	0.11 ( $\pm 0.10$ )	0.37 ( $\pm 0.14$ )
$T^2$ (s.e)	0.21 ( $\pm 0.13$ )	1.32 ( $\pm 0.32$ )	0.47 ( $\pm 0.36$ )	0.12 ( $\pm 0.14$ )	0.57 ( $\pm 0.19$ )

Heritable social effects seem to have an effect on fin damages and growth traits in cod. Erosion of the fins was not expected to show high direct genetic variation, as damages are more likely to be inflicted by other fish. Erosion of the caudal fins (erosion2) was in accordance with this, while erosion of the first dorsal fin showed a remarkable high heritability. More knowledge and understanding of the underlying biological processes is needed before deciding how to possibly account for aggression using e.g. fin erosion in a cod breeding program. In these analyses, we did not consider status of fin erosion prior to the trial. The results may therefore be biased by initial variation in fin erosions rather than social interactions during the experiment. In a future study, images captured of all individual fish at all recordings will be analysed, change in fin erosion during the experiment will be calculated and genetic parameters for this change will be estimated.

For the trait  $\Delta CF$ ,  $\sigma_{TBV}^2$  was ~3 times the magnitude of  $\sigma_{AD}^2$ , while GR was not significantly affected by social interactions. Adipose tissue has much higher energy content per gram compared with muscle tissue. Hence, condition factor may be more affected than growth by insufficient access to feed, making  $\Delta CF$  more suitable for estimating social interactions than GR.

Weight at the end of the experiment (weight3), seems to be strongly affected by heritable social interactions although growth was seemingly hardly affected. However, weight3 was also much determined by the initial weights. Further analyses will be conducted before drawing final conclusions on the social effects on growth and fin erosion of Atlantic cod. Furthermore, genetic correlations between the traits should be estimated to predict correlated responses in social interactions.

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