Aquaculture 294 (2009) 172–179

Contents lists available at ScienceDirect

Aquaculture

journal homepage: www.elsevier.com/locate/aqua-online

Genetic variation of gross gill pathology and survival of Atlantic salmon (*Salmo salar* L.) during natural amoebic gill disease challenge

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ARTICLE INFO

Article history:
Received 12 March 2009
Received in revised form 1 June 2009
Accepted 2 June 2009

Keywords:
Atlantic salmon
Salmo salar
Heritability
Quantitative genetics
Aquaculture
Salmonid
AGD
Disease

ABSTRACT

Survival in an experimental disease challenge test or to natural disease challenge is utilised by aquaculture breeding programs as the selection trait for disease resistance. However, these trials are expensive and do not offer the ability to retest animals. The aim of this study was therefore to estimate genetic parameters for resistance to amoebic gill disease (AGD) measured by a categorical scale of gross gill signs ("gill score") and survival in a field challenge in order to establish whether gill score provides adequate measurement of genetic variation for AGD resistance compared to an AGD challenge survival. A total of 1504 Atlantic salmon smolt, representing 140 full-sib families, was transferred to a marine site in SE Tasmania. The gills were assessed by gill score prior to freshwater bathing on the first two rounds of infection, and then the disease was allowed to develop until mortalities began. Gill score was reassessed after 50 days and mortality was allowed to continue until it had reached a plateau at 100 days. The overall survival rate was 32.3% but varied from 0% to 69% between families. Estimated narrow sense heritability for AGD resistance assessed by gill score varied between 0.23 and 0.48 over the three rounds of infection. Heritability of AGD survival challenge was 0.40 to 0.49 on the observed scale using binary and longitudinal measures. Gill score and survival showed a weak (−0.19) to strong (−0.96) negative genetic correlation which improved when assessed closer to the survival challenge. Estimated genetic gains by selection of the top one hundred estimated breeding values for gill score indicated that up to 82% of the expected gain in survival can be achieved when compared to estimated gain by selection upon survival (days to death), thus minimising selection costs and improving fish welfare whilst allowing repeat measures to be made. The results show that genetic variation of gill score at the early onset of losses closely compares with survival results if the disease is allowed to progress without subsequent freshwater bathing. Gill score may therefore be utilised as a nondestructive and repeatable selection trait for breeding Atlantic salmon with greater resistance to AGD.
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1. Introduction

Amoebic gill disease (AGD) is the main disease affecting marine Atlantic salmon aquaculture in Tasmania, Australia. The aetiological agent is the protozoan ectoparasite *Neoparamoeba perurans* (Young et al., 2007, 2008), which causes multifocal alterations in gill morphology, including severe epithelial hyperplasia, hypertrophy, lamellar fusion and interlamellar vesicle formation (Adams and Nowak, 2001, 2003, 2004a,b; Adams et al., 2004). Untreated, the disease causes inappetance, lethargy, respiratory distress, hypertension, cardiovascular compromise and eventual death (Munday et al., 1990; Powell et al., 2008). AGD is estimated to add up to 20% to the cost of production (Munday et al., 2001) due to growth loss, direct mortalities and the high infrastructure, labour and operating expenses of freshwater bathing to control the disease. The reiterative process of freshwater bathing each pen of fish eight to 12 times uses approximately 500 l freshwater per fish over the 15 to 18 month production cycle (Taylor et al., 2009).

A recent approach to minimising the impact of AGD has been breeding for disease resistance. A prerequisite for a successful commercial selective breeding program is to establish that genetic variation of economically important traits exists. There is ample evidence that a significant genetic basis exists to resistance of many important viral, bacterial and parasitic Atlantic salmon diseases (Chevassus and Dorson, 1990; Gjedrem et al., 1991; Gjedrem and Gjøen, 1995; Mustafa and MacKinnon, 1999; Kolstad et al., 2005; Guy et al., 2006; Ødegard et al., 2007b; Wetten et al., 2007; Kjøglum et al., 2008; Norris et al., 2008).

The characteristics assessed as a selection trait must adequately predict the objective trait and be cost effective to measure. In many aquaculture breeding programs the selection trait for disease resistance is measured as survival to a controlled challenge or natural field

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doi:10.1016/j.aquaculture.2009.06.007

infection. Lethal testing does not offer the ability to retest the same individuals. Considerable additive genetic variation in resistance to infectious finfish diseases has previously been measured through survival challenge tests in controlled tank experiments (Gjedrem et al., 1991; Gjedrem and Gjøen, 1995; Gjøen et al., 1997; Henryon et al., 2002; 2005; Kettunen et al., 2007; Ødegard et al., 2007a; Wetten et al., 2007; Silverstein et al., 2009). The advantage of these challenges is that the test environment is controlled and the host can be exposed to known quantities of a single pathogen, mortalities are easy to collect and the cause of death can be readily defined. However, due to space limitations, researchers are often curtailed in the number of animals they can trial, thus limiting the number and size of families that can be assessed. In addition, the facilities required for challenge assessment are expensive to establish and operate.

In breeding programs aimed at improving disease resistance in farmed fish, individuals and families should ideally be selected based on disease resistance in commercial production environments (Gjøen et al., 1997; Ødegard et al., 2006). Since the marine environment is an open system, field trials of aquatic animal diseases in aquaculture pens may be affected by environmental effects and non-target diseases, but are reflective of commercial infection conditions. The outcomes of disease outbreaks in the field have been shown to be highly correlated with those of tank challenge tests (Gjøen et al., 1997; Ødegard et al., 2006; Storset et al., 2007) though field genetic measures tend to be slightly lower due to higher error variance (Wetten et al., 2007). Using natural infections as a selection criterion is problematic due to a number of factors, including unpredictable timing and magnitude of infection (Kolstad et al., 2005); conversely, the biotic and abiotic stressors in the natural environment may be an essential factor in inducing typical field pathology that cannot be recreated in a tank challenge (Norris et al., 2008).

Measurement of genetic variation of disease resistance, as part of a selective breeding strategy, offers substantial economic benefits for industry and potential long-term welfare improvement for farmed fish. Although the number of test animals can be minimised in natural or controlled challenges, there are fish welfare concerns in testing fish to mortality. Researchers are ethically bound to produce as much knowledge as possible from each animal used (Johansen et al., 2006). The ability to test resistance to a field outbreak of disease using a nondestructive assessment method would therefore offer significant cost saving and fish welfare benefits to the selection process as long as the accuracy of selection is maintained.

AGD is fully diagnosed by histopathology to confirm the presence of amoebae, containing a nucleus and symbiont parasome(s), in association with regions of hyperplastic gill (Adams and Nowak, 2001, 2003) that are formed by the host in response to the parasite. This method is destructive so is of limited value for selective breeding. The salmon farming industry utilises a simple presumptive gross "gill score" to schedule freshwater bath treatments. This categorical scale measures the prevalence and intensity of damaged gill which presents grossly as visible white mucoid spots and patches (Clark and Nowak, 1999; Adams and Nowak, 2001). This method is known to have a moderate to good agreement with histopathology in advanced infections (Adams et al., 2004) and a close phenotypic link between gill score and the level of mortalities was confirmed by Taylor et al.(2009). Resistance to AGD, measured by variation in gill score, is presumed to relate to the degree of resistance to *N. perurans* infection, but may also include elements of host tolerance, differential exposure to the parasite or a refractory response to prior infection. Because the gill score method is nondestructive, rapid and utilised by industry to schedule freshwater bathing, it is favoured as a selection trait for the breeding program.

Evidence of varying levels of inherent resistance to AGD was suggested by Bridle et al. (2005) who noted that a subpopulation survived a severe first infection of AGD in a challenge trial and showed relatively minor gill pathology. The first measure of genetic variation for resistance to AGD was provided by Taylor et al. (2007) reporting a broad sense heritability (H^2) of 0.16 ± 0.07, measured in a challenge test at first infection. Resistance of Atlantic salmon to AGD after secondary exposure has previously been reported on the basis of gill pathology (Findlay and Munday, 1998). Vincent et al. (2006) presented evidence of enhanced survival of Atlantic salmon previously exposed to AGD and demonstrated that resistance is associated with systemic anti-*Neoparamoeba* spp. antibody development when compared to naïve control fish. The nature of this acquired response is poorly understood, but future research may support development of a more specific measure of AGD resistance that can be exploited for selective breeding.

In this study, the aim was to establish the accuracy of the "gill score" as a selection trait for AGD resistance compared to survival to the disease in a natural challenge trial. Specifically, the aims were to (i) estimate additive (narrow sense) heritability for resistance to AGD assessed by gill score and survival challenge to a natural summertime AGD infection, (ii) establish whether gill score and AGD survival are under common genetic control, (iii) estimate the relative proportion of genetic gain in AGD survival that could be achieved by using selection strategies based upon different measurements of gill symptoms compared to survival data and (iv) compare relative sampling costs to the breeding program of gill score and survival challenge testing.

2. Materials and methods

2.1. Mating design, freshwater rearing and marine transfer

Broodstock (141 sires and 141 dams) were randomly selected from commercial stock as founder individuals to spawn the first generation offspring (2005 cohort) at the Salmon Enterprises of Tasmania Pty Ltd (SALTAS) Wayatinah hatchery in central Tasmania. Adipose fin samples were taken from all broodstock, stored in 95% ethanol and genotyped by a microsatellite multiplex by Landcatch Natural Selection (Scotland).

The 2005 cohort families were produced in May 2005 using a fractional factorial mating design, where each male was crossed with two females and each female with two males to create 282 full-sib families (i.e. 141 paternal and 141 maternal half-sib families). The performance of the brood fish was unknown so there was no intentional trait selection. The families of fertilised eggs were each allocated to separate egg tray compartments (two compartments per tray) and maintained there until just prior to hatching. Due to variable egg survival during incubation, 109 families were discarded, leaving 173 viable full-sib families (56 paternal and 70 maternal half-sib) from crosses between 115 males and 103 females. Eyed eggs were then transferred from each family to a communal tank to ensure a common environment for swim-up, early feeding and rearing through to pre-smolt stage under natural lighting and ambient water temperatures.

In June 2006, a random sample of pre-smolt (mean = 158 g, SD = 48 g) was anaesthetised, a caudal fin-clip dissected from each individual and a 12 mm × 2 mm passive integrated transponder (PIT, Sokymat, Switzerland) injected into the left flank muscle above the lateral line. Microsatellite genotyping and parentage determination was later performed on the fin-clips to allocate each tagged animal to family. The fish were held in the hatchery for 6 weeks under lights (22L:2D) at ambient temperature. On 17th August 2006, 1504 of these fish (mean = 228 g, SD = 47 g) and one thousand non-genotyped untagged adipose clipped fish (mean = 167 g, SD = 38 g) were transferred to a 10 m × 10 m × 8 m (800 m^3) marine fish cage on a commercial lease operated by Tassal Operations Pty Ltd. (Dover, Tasmania). The untagged fish were included to ensure a reasonable approximation to commercial stocking densities.

2.2. AGD field challenges and subsequent survival trial

The fish were subjected to two rounds of natural AGD and subsequent freshwater bathing, followed by a further natural re-infection through to a 100 day AGD survival trial as described in Taylor et al. (2009). A

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