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Genetic parameters for resistance to Infectious Pancreatic Necrosis in pedigreed Atlantic salmon (*Salmo salar*) post-smolts using a Reduced Animal Model

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ABSTRACT

Infectious Pancreatic Necrosis Virus (IPNV) is an important cause of mortality and economic loss across all species of commercially farmed salmonids, and genetic variation in survival to IPN challenge has been previously demonstrated. In order to exploit this variation in the development of resistant strains, robust procedures are required to quantify the extent of genetic variation and to provide estimated breeding values used to select candidates for breeding. This paper applies a recently developed implementation of the Reduced-Animal Mixed-model procedure (RAM) to field data describing percent mortality following IPN epidemics in Scottish farmed Atlantic salmon, covering 1369 full-sib family groups distributed over four years and a total of seven sites. Pedigrees were established through a combination of electronic (PIT) tagging and parentage assignment using microsatellite DNA analysis. Heritabilities between 0.07 and 0.56 (s.e.<0.04) were obtained, genetic correlations between sites sharing the same families were uniformly high, 0.70 to 0.85, (s.e.<0.06) and low levels of fullsib family effect due to common environment (proportion of phenotypic variance 0.04, s.e. 0.002) were observed. These results confirmed that exploitable genetic variation exists for mortality to IPNV over a range of epidemiological conditions inherent in field data, which can be used to select strains of salmon with increased resistance to IPNV.

1. Introduction

Mortality from Infectious Pancreatic Necrosis (IPN) is an important cause of economic loss across all species of commercially farmed salmonids. In anadromous species mortalities can occur at both the juvenile post-hatch freshwater stage (typically 30–80%) and the early post-smolt seawater stage (5–30%), both stages being when the fish are particularly vulnerable, both immunologically and physiologically (Roberts and Pearson, 2005). A significant genetic component to mortality from the IPN virus (IPNV) has been clearly demonstrated from experimental and field challenges of pedigreed Atlantic salmon *Salmo salar* L. populations, in both freshwater stage fry (Wetten et al., 2007; Kjoglum et al., 2008) and in seawater post-smolts (Guy et al., 2006). Heritabilities of 0.11 to 0.24 for seawater mortality to IPN on the observed binary scale, and 0.31 to 0.69 on the underlying liability scale, were demonstrated by Guy et al. (2006) using simple expressions derived from the variance and covariance of family mean prevalence.

Given a significant heritability, and a population measured on one site, a ranking of family means is sufficient to apply a selection differential and to generate genetic gain for decreased mortality from disease. However, breeding from survivors is undesirable where biosecurity may be compromised through a risk of viral transmission, as is the case with IPN. In commercial breeding programs, therefore, the information on disease is more likely to come from one or more test sites, possibly with differing levels of infection and testing sibs of candidates with only a subset of the families in common. These data then require to be linked back to the breeding population, which are maintained separately with the intention of remaining unexposed. Additionally, information on disease requires to be properly weighted in combination with other traits. This process is usually addressed through application of multi-trait mixed models, in particular parameterised using the Individual Animal Model, IAM; (see Henderson, 1986; Mrode, 2005) to estimate variance components and predict breeding values (EBVs). The IAM provides genetic evaluations for any animal that is linked via the pedigree to any other animal having a data record. This is especially appropriate when breeding for disease resistance where the animals providing data from disease challenge (in aquaculture, these are usually mortalities) are different to candidates for breeding.

Mixed model methodology allows for complex genetic and environmental modelling, for example: estimates of direct additive genetic effects, dam maternal genetic, dam environmental effects, and effects due to common environment where family members are

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reared together. While maternal effects may seem less relevant to species which do not gestate or rear their young and which invest little compared to mammals in parenting their offspring, the female fish parent does invest resources in the egg that may affect survival and initial growth, which may then persist to affect performance measurements taken later in life. Moreover, broodstock families are often reared in separate 'family' tanks until the fry are large enough to have electronic tags inserted at around 20 g weight and up to six months of age. Rearing families separately may induce a common environment effect on growth records (e.g. Winkelman and Peterson, 1994) which can inflate estimates of genetic, or maternal effects if the data does not enable them to be independently estimated.

With binary mortality data, recorded on the scale of observation as zero (alive) and one (dead), family information based on data from individual animals can be summarised without loss of information by the full-sib sample mean prevalence p within year and location. Summarised family information includes the sample variance, equal to $p(1\text{-}p)$ for binary traits, allowing suitable variance partitioning, the basis of mixed models. An implementation of mixed models and the IAM in particular, based on family mean prevalence may therefore be more computationally efficient in some cases and has attracted some attention in the past (Simianer and Gjerde, 1991). One such implementation has recently been described by White et al. (2006) based on a revival of the Reduced Animal Model (RAM) of Quaas and Pollak (1980). RAM separates the data into two layers, parents and non-parents. Only the parental layer is represented in the pedigree relationship matrix used for analysis and data on non-parents is accumulated into the parental layer, from which the variance components and associated estimated breeding values are derived, without directly processing the non-parents. If required, estimates for non-parents can be obtained indirectly using simple linear functions (backsolving).

We have applied the RAM method of White et al. (2006), to mortality data obtained from natural seawater field challenges of Atlantic salmon post-smolt families to IPNV. This paper formalises the earlier results of Guy et al. (2006) that were based on simpler algebraic functions of family means applied to a single year group, and extends them by including three further year groups. Additionally, it explores the importance of common environment effects and estimates genetic correlations between sites.

2. Materials and methods

2.1. Description of test populations

Data consisted of mortality to IPN following seawater transfer, recorded as alive or dead, and covered seven sites, two recorded in year 2000, one in year 2001, three in year 2002 and one in year 2005. Different sites in the same year were replicates of the same families. The populations were coded as *year_site*. For example *2001_3* refers to the population of full-sib families derived from parents stripped (i.e. mated and eggs fertilised) in November 1999 and transferred to seawater at *site 3 in April 2001*. *Populations 2002_4*, *2002_5* and *2002_6* correspond to *sites 1*, *2* and *3* respectively described in Guy et al. (2006), which gives further details of population construction and mortality sampling. For brevity we will refer to a family represented on a particular site at any one time as a 'full-sib group'. Note that populations *2001_3* and *2005_7* were the only populations tested in those years, so in those cases only, families were not replicated across sites.

Table 1 shows the mating scheme giving rise to these families. In total 783 dams and 500 sires were crossed, with each dam being crossed to a single sire, so forming progeny groups of paternal half-sib and full-sib families. With replication of families over sites within year, these formed 1369 full-sib groups. 315 sires were single-pair mated (i.e. one mate only), 126 sires produced two full-sib families each, and 59 sires produced three to seven full-sib families each. The overall level of single pair mating (40% of all dams mated) was a consequence of post-mortem diagnostic testing for the presence of IPNV in asymptomatic parents of eggs destined for export (Roberts and Pearson, 2005) since single pair matings minimised the potential cull rate as a consequence of a male being detected as infected. Post-mortem disease testing also necessitated that males and females contributed offspring to only a single year. Between 0 and 58% of matings each year involved parents from adjacent year-classes (one-sea-winter and three-sea-winter broodstock respectively). However, although identification of individuals through DNA microsatellite analysis commenced with the parents used in the 1996 matings, identification of families common to adjacent year-groups could not start to be determined until four years later, when the offspring of family assigned individuals became parents themselves. Therefore this mortality data was collected when there was only weak genetic linkage between the early year-groups. However, note that parents of the 2005 year group, mated in 2003, were themselves offspring of parents of the 2001 year group, mated in 1999. In total the pedigree included 1980 further ancestors in addition to the 239,535 offspring recorded as being challenged with IPN.

Each female produced a single spawning of 10,000–20,000 eggs, which were split and eventually distributed as batches of fullsib groups of various size to the appropriate site. At smolting in April of each year at 15 months of age (when the fish physiologically adapt to the seawater environment) batches containing equal numbers of each family were transferred as juveniles to the various seawater sites. Adjacent cages used in year 2000 were treated as separate sites, (*2000_1* and *2000_2*). The other sites were single cages in proximity to commercial stocks, apart from *2002_5*, representing a typical IPN event in seawater tanks of mixed broodstock families reared for egg production. The disease challenges occurred June to August, when fish were approximately 18 months of age.

Early mortalities within four weeks of transfer to sea occurred on each site and were assumed to be related to failure in the smolting process. Exposure to low levels of IPN virus in the aquatic environment was assumed to be immediate on transfer, but clinical signs specific to infection by the IPN virus and consequential mortality typically did not present until five to six weeks following transfer. This

Table 1
Numbers of parents and family structure for each *year_site* and the age of the parents at mating.

Sites	Cohort born	Parents born	Age of parents at mating	Number of female parents	Number of male parents	Numbers of males with differing mating ratios			
						1	2	3	4+
2000_1 and 2000_2	1998	1993	5	113	71	45	24	1	1
		1994	4	79	51	30	10	4	7
2001_3	1999	1994	5	4	20	11	4	2	3
		1995	4	197	110	80	23	3	4
		1996	3	0	8	7	1	0	0
2002_4, 2002_5 and 2002_6	2000	1996	4	197	149	104	42	3	0
2005_7	2003	1999	4	186	83	31	21	22	9
		2000	3	7	8	7	1		0
All families				783	500	315	126	35	24