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Genetic architecture of rainbow trout survival from egg to adult

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Summary

Survival from birth to a reproductive adult is a challenge that only robust individuals resistant to a variety of mortality factors will overcome. To assess whether survival traits share genetic architecture throughout the life cycle, we estimated genetic correlations for survival within fingerling stage, and across egg, fingerling and grow-out stages in farmed rainbow trout. Genetic parameters of survival at three life cycle stages were estimated for 249 166 individuals originating from ten year classes of a pedigreed population. Despite being an important fitness component, survival traits harboured significant but modest amount of genetic variation ($h^2 = 0{\cdot}07$–$0{\cdot}27$). Weak associations between survival during egg-fry and fingerling periods, between early and late fingerling periods ($r_G = 0{\cdot}30$) and generally low genetic correlations between fingerling and grow-out survival (mean $r_G = 0{\cdot}06$) suggested that life-stage specific survival traits are best regarded as separate traits. However, in the sub-set of data with detailed time of death records, positive genetic correlations between early and late fingerling survival ($r_G = 0{\cdot}89$) showed that during certain years the best genotypes in the early period were also among the best in the late period. That survival across fingerling period can be genetically the same, trait was indicated also by only slightly higher heritability ($h^2 = 0{\cdot}15$) estimated with the survival analysis of time to death during fingerling period compared to the analysis treating fingerling survival as a binary character ($h^2 = 0{\cdot}11$). The results imply that (1) inherited resistance against unknown mortality factors exists, but (2) ranking of genotypes changes across life stages.

1. Introduction

Survival from birth to reproductive adult is a series of challenges created by a multitude of mortality factors whose incidence varies in time and space. Thus only individuals robust enough to overcome these challenges will become parents of the next generation. Survival is an ultimate robustness trait because it is a measure of an individual's resistance against multiple mortality factors occurring in an environment (Vehviläinen *et al*., 2008).

In animal breeding, a common goal is that animals would be robust against multiple environmental disturbances, stressors and mortality factors throughout all life stages (Mulder & Bijma, 2005; Pertoldi *et al*., 2007; Vehviläinen *et al*., 2008). Genetic analyses of survival, and its underlying component traits, across environments and during different growth or life stages will increase our ability to utilize survival as a selected trait in breeding programmes. Likewise, in the wild, natural selection favours genotypes capable of producing optimal phenotypes at different life stages and across multiple environments (Pigliucci & Schlichting, 1995; Miller & Vincent, 2008), making understanding of survival genetics and its developmental mechanisms of importance for evolutionary biology (Félix & Wagner, 2008).

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Table 1. *Population structure, mean survival, sample sizes and mating designs in each year class*

Population/ Generation	Fertilization year	Mean survival (%)			Number of fish			No. of sires	No. of dams	Mean (range) dams per sire	Mean (range) sires per dam	No. of full-sib families	No. of family tanks	No. of sea test stations
		Fingerling	Freshwater grow-out	Sea station grow-out	Fingerling	Freshwater grow-out	Sea station grow-out							
Population I														
1	1995	–	80	–	–	13 643	–	92	272	3·0 (1–5)	1·0 (1–1)	272	370	–
2	1998	97	74	–	16 169	6096	–	71	128	1·8 (1–4)	1·0 (1–1)	128	132	–
3	2001	92	50	56	50 962	8399	7877	121	154	2·5 (1–6)	2·0 (1–3)	303	303	2
4	2004	98	71	84	37 265	7501	7493	130	93	1·9 (1–5)	2·7 (1–4)	250	250	2
Population IIa														
1	1996	97	78	–	21 324	7480	–	75	150	2·0 (1–4)	1·0 (1–1)	150	150	–
2	1999	89	74	70	16 362	4459	3633	48	109	2·3 (1–4)	1·0 (1–1)	109	150	2
3	2002	88	65	60	40 157	5503	1456	113	139	2·5 (1–6)	2·1 (1–3)	287	287	1
Population IIb														
1	1997	96	73	–	37 712	10 262	–	65	79	2·9 (1–5)	2·4 (1–3)	191	228	–
2	2000	97	77	78	29 215	7916	9730	98	122	2·0 (1–5)	1·6 (1–3)	200	200	2
3	2003	–	71	70	–	10 240	10 216	168	155	2·0 (1–5)	2·2 (1–3)	341	341	2

Previous studies on survival at different periods have revealed that genetic variation can vary depending on the age or life stage of an organism. For instance, reported heritabilities for lamb survival show large variation during the first year of life (h^2 range: 0·01–0·33; Southey *et al*., 2001, 2003; Sawalha *et al*., 2007; Riggio *et al*., 2008). Similarly, resistance to multifactorial mastitis disease in cattle is not the same trait between different lactations, and the proper selection method is thus based on a multitrait index rather than defining mastitis as one trait over all lactations (Negussie *et al*., 2007, 2008). Different variants of candidate genes have age-specific influence on human survival (Passarino *et al*., 2006).

However, there is a lack of comprehensive coverage of the genetic architecture of age-specific survival traits across life stages (Wilson *et al*., 2008). The main mortality factors (diseases, parasites, predators and abiotic conditions) change during the life cycle of most organisms, and susceptibility to different factors, e.g. bacterial versus viral diseases, can be weakly or even negatively genetically correlated (e.g. Gjøen *et al*., 1997; Cotter *et al*., 2004; Bubliy & Loeschcke, 2005; Ødegård *et al*., 2007; Kjøglum *et al*., 2008). Most of the studies mentioned above concentrate on a relatively short period of time, a specific disease, or are conducted within one life stage of an animal. Therefore it is not evident whether the same genotypes have superior survival across all life cycle stages. Moreover, genotypes can re-rank even within a life stage if the stage is long with respect to variation in the presence of mortality factors. It is also possible that there are trade-offs between resistance mechanisms at different ages, thus creating re-ranking even when mortality factors remain constant. These questions can be approached by defining survival in physiologically different life stages as separate traits and then calculating the genetic correlations between the traits. Finding positive genetic correlations would then mean a set of genotypes exist that on average survive best in all life stages. Negative correlations, on the other hand, would mean that there is a degree of reversed genotype ranking and that survival in different life stages is best regarded as separate traits.

Salmonids are well suited for studying the genetic architecture of traits across life stages. These fish lack an internal embryonic stage and have several clearly defined life stages (egg, yolk-sac fry, fingerling and post-smoltification grow-out). Thus, survival of individuals can be tracked from embryo to adulthood. In this paper, we investigate genetic architecture of survival traits within and across life stages (egg-fry, fingerling and grow-out) in farmed rainbow trout (*Oncorhynchus mykiss* Walbaum) using data from ten year classes of a pedigreed population. These results provide evidence of the extent to which (1) survival is

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heritable and (2) survival traits within and across life cycle stages share genetic architecture.

2. Materials and methods

Survival records were obtained from the Finnish national rainbow trout breeding programme maintained by the Finnish Game and Fisheries Research Institute (FGFRI) and MTT Agrifood Research Finland. The freshwater breeding nucleus is held at the FGFRI Tervo Fisheries Research and Aquaculture station in Central Finland. The breeding population was established in 1992 and the pedigree is known back to a common base population in 1989 (Kause *et al*., 2005).

(i) *Population structure*

The data consisted of 814 full-sib family-level survival observations from the egg-fry period (fertilized egg to first feeding fry), 249 166 individual survival observations from the juvenile fingerling period (the first months of feeding), and 121 905 individual survival records from the grow-out period in freshwater or seawater (growth from 50 to 1000 g, after which the first individuals reach maturity).

The fish originated from three subpopulations sharing a common base population and from ten year classes belonging to four generations (Table 1). Each year class consisted of 109–341 full-sib families generated from 48 to 168 sires and 79 to 272 dams, mated using either nested paternal or partial factorial designs. Pedigrees of all individuals were known at all life stages because families with known parents were held separately before individual tagging. During the fingerling period, the total number of fish within each year class was 16 169–50 962. During grow-out in each year class, fish were kept either in the freshwater nucleus station (range: 4459–13 643 fish/year class), or sent to one or two sea test stations (range: 1456–5165 fish/year class, Table 1).

(ii) *Rearing conditions*

The parents for each generation were selected based on their estimated breeding values for growth (since 1992), maturity age (since 2001), external appearance (since 2001), skeletal deformations (since 2002), fillet colour (since 2003) and cataracts due to *Diplostomum* parasite (since 2003) (Kause *et al*., 2005). Parental fish were mated at the Tervo freshwater nucleus station during April–June.

(a) *Egg-fry period*

The egg-fry period lasted from egg fertilization to first feeding. Full-sib egg batches of equal volume (0·5 dl) were incubated separately in subdivided trays of vertical incubators, and at the eyed-egg stage, each full-sib family was transferred to one or two indoor 150 litres family-tanks (Table 1). Eggs hatched in July. To estimate egg-fry period survival, the number of fish alive at the end of the egg-fry period in August was counted. This resulted in one observation per full-sib family. At the same time, full-sib families were graded to similar family size (mean = 150 individuals, range = 17–170) and the average individual weight of fish was determined (mean = 3·37 g, range = 0·56–17·3 g).

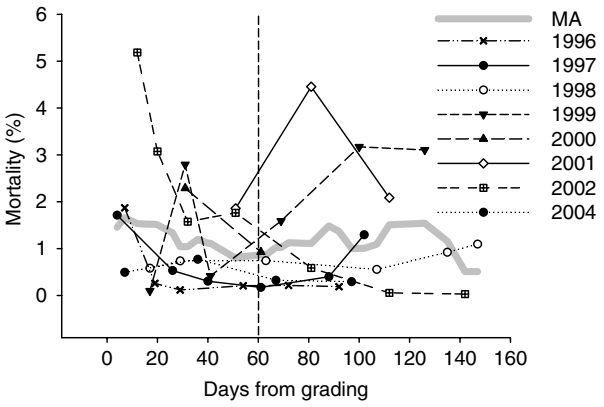

Fig. 1. Timing of mortality (% of fish died in each observation period from total number of fish in family tanks after grading) during juvenile fingerling period in different year classes. Monthly moving average (MA) calculated as mean of ±15 days around each observation point over all year classes in grey bold. Dashed vertical line marks the division of fingerling period to early and late fingerling survival traits.

(b) *Juvenile fingerling period*

The fingerling period lasted from full-sib family grading to individual tagging. After grading of the families to a similar family size, full-sib families were kept separately indoors in 150 litres family tanks until the start of individual tagging at November. Dead individuals were collected from the tanks during routine maintenance, and the individuals alive at the end of the period were counted, providing individual-level data on survival. Individual mortality during the fingerling period (length range in different year classes: 92–147 days, Fig. 1) was recorded periodically (mean interval of recording = 22 days, range: 4–51).

(c) *Grow-out period*

The grow-out period lasted from individual tagging at the end of first growing season to measurements at the end of the second growing season. At the size of 50–100 g, fish were individually tagged with Passive Integrated Transponders (Trovan Ltd, Germany). After tagging, the fish were transferred either to an

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outdoor raceway at the freshwater station or sent to one or two Baltic Sea test stations during April in a split-family design (Table 1). In commercial sea farming, it is a standard protocol to vaccinate fish before transportation from freshwater to sea grow-out. Thus, all fish sent to the sea test stations were vaccinated one month before transportation with intraperitoneal injection (1995–1997: 0·1 ml of Lipogen Duo, Aquahealth Ltd, Canada; 1998–2004: 0·2 ml of Apoject 1800, Pharmac, Norway) against bacterial diseases caused by *Aeromonas salmonica* ssp. *salmonica* and *Listonella* (*Vibrio*) *anguillarum*. These diseases do not occur in significant incidence in freshwater grow-out, and thus the fish remaining in freshwater are not vaccinated.

At the freshwater station, the fish were held in a flow-through earth-bottomed raceway. All sea stations were located in South-West Finland within a maximum distance of 163 km from each other, but they were not the same ones from generation to generation. At each sea station, the fish were reared under commercial farming conditions in a single net-pen. All fish were fed commercial fish feed pellets throughout the rearing cycle. The rearing procedure is detailed by Kause *et al*. (2005). The individual grow-out survival of fish from tagging to the end of the second growing season (fingerling period + grow-out season) was determined in May at freshwater (mean weight of fish = 964 g) and in late summer–autumn (July–December) at the sea stations (mean weight of fish = 1095 g). In each year class and environment, grow-out survival recording lasted 2–4 weeks.

(iii) *Trait definitions for linear model analysis*

(a) *Egg-fry survival*

Full-sib family size before grading was used as an estimate of family-level survival from egg fertilization to first feeding fry. For each family, an equal volume of fertilized eggs (0·5 dl) was initially placed in incubators and all eyed eggs were transferred into family tanks. If all eggs were the same size, the number of fish at grading would precisely describe family-level survival. However, egg size likely differed among families, and therefore 'egg-fry survival' is only an approximation. Egg-fry survival was not recorded for year class 2002 and could not be obtained in a few families throughout the study, resulting in 814 family-level records.

(b) *Fingerling survival*

A trait 'juvenile fingerling survival' (JuvTotal) was defined as survival from grading to the starting date of individual tagging of the first tagged family within each year class. Individual fish that survived this period were scored as survived (= 1), while the individual fish that died were coded as dead (= 0). The length of period from grading to the start of tagging varied between year classes (year class period length range: 61–147 days, Fig. 1). This was due to both variability of fish growth in different years (fish need to reach a certain size before individual tagging is feasible) and practical logistic challenges. To standardize the trait definition across families, the end of the fingerling period was defined as the date when the first family was tagged. This is because tagging all families takes months. Out of all eight year classes, fingerling survival records for three families were deleted because they experienced non-natural mortality due to management accidents (e.g. failure in water flow system).

(c) *Early and late fingerling survival*

To assess whether mortality during early and late fingerling periods are the same trait, two additional fingerling survival traits were defined by dividing JuvTotal into two periods. 'Early juvenile fingerling survival' (EarlyJuv) was defined as survival until 60 days after grading (year class range: 51–69 days; individual fish surviving until the end of the period = 1, individual fish died during the period = 0). 'Late juvenile fingerling survival' (LateJuv) was defined as survival from 60 post-grading until the start of individual tagging (range: 30–91 days; individual fish that survived = 1, individual fish that died = 0). The 60-day threshold was based both on the experience of practitioners and on the preliminary inspection of the data, which suggested that mortality is not stable through the fingerling period (Fig. 1).

The fingerling survival records for year class 2000 extended only to 61 days. Therefore LateJuv and JuvTotal were not defined for this year class, resulting in 219 951 observations for these traits.

(d) *Grow-out survival*

For grow-out survival (from tagging to the end of the second growing season), two environment-specific traits were defined: (1) 'Grow-out survival in freshwater' (FreshwG) and (2) 'Grow-out survival in sea water' (SeaG). Individual fish that survived from tagging to the end of the second growing season were scored as survived (= 1), while fish not present were coded as missing (= 0).

(iv) *Trait definitions for survival analysis*

For the detailed survival analysis of the fingerling period, the survival trait analysed was the number of days from grading until death. Individuals still alive at the start of individual tagging for each year class were treated as censored records. Three year classes (1997, 1998 and 1999) consisted of enough detailed data that

allowed a genetic survival analysis. In these year classes, time at death was recorded both more frequently (6 times) than in other year classes (mean = 4·4 times), and the recording period was longer (102–126 days) than in the others (61–142 days).

(v) *Accounting for selection bias*

In artificially selected populations, traits are recorded from a non-random sample of individuals. The impact of such selection bias on the genetic parameter estimates can be accounted for with a multitrait analysis that includes the selected trait(s) (Henderson, 1984). In our data, body weight was the main selected trait encompassing approximately 60% of the selection index weights. Thus, genetic parameters for survival traits were estimated by including three body weight traits into all linear model multitrait analyses. The body weight traits included were: (1) 'JuvBW' – individual body weight of fingerlings recorded at tagging ($n = 189\,299$ fish); (2) 'FreshwBW' – individual body weight recorded at the end of the grow-out period in freshwater in April–June ($n = 58\,724$) and (3) 'SeaBW' – individual body weight recorded at the end of grow-out period in sea in October–April ($n = 41\,678$). Genetic analysis of body weights has been reported previously (Kause *et al*., 2003, 2005).

(vi) *Linear model genetic analyses*

Linear model heritabilities and genetic correlations were estimated using restricted maximum likelihood and multitrait animal models (DMU-AI software; Madsen & Jensen, 2008).

Juvenile fingerling survival traits (JuvTotal, EarlyJuv and LateJuv) and grow-out survival in freshwater (FreshwG) were modelled as

$$y_{\mathrm{ijk}} = \mu + \mathrm{year}_i + \mathrm{year}_i * c_j + \mathrm{anim}_k + \varepsilon_{ijk}, \quad (1)$$

grow-out survival at sea (SeaG) as

$$y_{ijkl} = \mu + \mathrm{year}_i * \mathrm{site}_l + \mathrm{year}_i * c_j + \mathrm{anim}_k + \varepsilon_{ijkl}, \quad (2)$$

body weight at tagging (JuvBW) as

$$y_{ijk} = \mu + \mathrm{year}_i + \mathrm{year}_i * c_j + \mathrm{anim}_k + \varepsilon_{ijk} \times \mathrm{Tsum(year)}, \quad (3)$$

body weight after freshwater grow-out (FreshwBW) as

$$y_{ijkmn} = \mu + \mathrm{year}_i * \mathrm{sex}_m * \mathrm{mat}_n + \mathrm{year}_i * c_j + \mathrm{anim}_k + \varepsilon_{ijkl} \quad (4)$$

and body weight after sea grow-out (SeaBW) as

$$y_{ijklmn} = \mu + \mathrm{year}_i * \mathrm{site}_l * \mathrm{sex}_m * \mathrm{mat}_n + \mathrm{year}_i * c_j + \mathrm{anim}_k + \varepsilon_{ijkl}, \quad (5)$$

where y_k is the survival or body weight observation for an individual (k = number of animals), μ is a mean of a given trait, year_i is the fixed effect of fertilization year ($i = 1, \ldots, 8$ years for fingerling period and 1, …, 10 years for grow-out period), $\mathrm{year}_i * \mathrm{site}_l$ is the fixed interaction effect of birth year and sea test station ($l = 1, 2$; site A and B), $\mathrm{year}_i * \mathrm{sex}_m * \mathrm{mat}_n$ is the fixed interaction effect of birth year, sex ($m = 1, 2, 9$; male, female and unknown) and maturation ($n = 0, 1$; mature or immature), $\mathrm{year}_i * c_j$ is the random interaction effect of birth year with common environment effect shared by full sibs before tagging (j = number of family tanks), anim_k is the random genetic animal effect (k = number of animals) taking into account full pedigree information and ε is a random error term. Tsum(year) is a covariate of the cumulative temperature at date of recording, nested within a birth year. The common environment effect was modelled separately for each birth year because tanks did not have a consistent effect every year.

Heritabilities and genetic correlations were derived from seven-trait analyses. Estimation of parameters with all fingerling survival traits in the same analysis was not feasible because of the close to unity correlation structure between EarlyJuv versus JuvTot and LateJuv versus JuvTot, combined with near zero correlation between EarlyJuv and LateJuv. Thus, to obtain all correlations three separate trait combinations were run. In each run, different two-trait combinations of fingerling survival traits were analysed with both of the grow-out survival traits and the three body weight traits. The correlation matrices were bent to be positive definite using the method of Hayes & Hill (1981). Bending changed the genetic correlations by an average of 0·005, the maximum change being 0·022, and the common environment correlations by an average of 0·017, the maximum change being 0·093. The standard errors reported for the correlations are means from the separate multitrait runs.

Heritability for linear animal models was quantified as $h^2 = V_G(V_G + V_C + V_R)^{-1}$, where V_G is genetic, V_C common environment and V_R residual variation. Although genetic variance is assumed to be mainly due to additive genetic effects, the potential effects of dominance and epistasis cannot, however, be excluded. The common environment effect was quantified as $c^2 = V_C(V_C + V_G + V_R)^{-1}$. In addition to the common environment effects of full-sibs, V_C may potentially include parts of dominance variance. Asymptotic standard errors for the genetic parameters were computed based on a Taylor series approximation (Madsen & Jensen, 2008).

Heritabilities and their standard errors estimated by the linear model were transformed to the underlying liability scale using the formula of Dempster & Lerner (1950). Genetic correlations of binary traits estimated using linear models are unbiased

Table 2. *Sample sizes, mean survival, heritabilities* (h^2), *common environment effects* (c^2) *and their standard errors* (*SE*), *genetic* (V_G) *and phenotypic* (V_P) *variances*

Trait	Sample size	Survival (%)	$h^2 \pm$SE	$c^2 \pm$SE	V_G	V_P
EarlyJuv	249 166	96·1	0·20 ± 0·04	0·09 ± 0·00	0·0014	0·0368
LateJuv	219 951	97·4	0·27 ± 0·07	0·12 ± 0·01	0·0009	0·0240
JuvTotal	219 951	93·4	0·19 ± 0·04	0·10 ± 0·00	0·0031	0·0594
FreshwG	81 499	72·3	0·16 ± 0·02	0·05 ± 0·00	0·0178	0·1955
SeaG	40 405	71·3	0·07 ± 0·02	0·04 ± 0·00	0·0071	0·1914

(Mäntysaari *et al*., 1991). Because fish in the sea were not recorded for traits in fresh water, and vice versa, residual covariances between the sea and freshwater traits were set to zero in the analysis. Because individuals were not yet individually tagged during the fingerling survival data collection, a fingerling survival record of an individual could not be attached to the individual's grow-out observation, and thus the residual covariances between the fingerling and grow-out survival traits were also set to zero.

Because egg-fry survival was recorded at a family level, genetic correlations with the other survival traits could not be estimated using the animal model. Instead, we explored the associations between egg-fry and fingerling survival traits by calculating sire-family mean Spearman correlations between egg-fry and fingerling survival traits within each year class (Proc CORR in SAS v.9.1.3; SAS, 2005). A sire-mean correlation is an approximation of the true genetic correlation, and more conservative (i.e. produces a weaker genetic correlation) than Restricted Maximum Likelihood (REML) (Roff, 1997; Astles *et al*., 2006).

(vii) *Survival analysis*

Survival analysis for the timing of death during the juvenile fingerling period was run for the sub-set of data (year classes 1997, 1998 and 1999) using SurvivalKit software (Ducrocq & Sölkner, 1998). Rate of mortality over time was modelled as a continuous time with frailty Cox's proportional hazard model.

In the frailty model, survival was modelled as

$$h_{ijk}(t) = h_0(t) e^{\text{year}_i + \text{year}_i * c_j + \text{anim}_k}, \quad (6)$$

where $h_{ijk}(t)$ is the hazard function for the kth individual from the jth family tank in the ith year and $h_0(t)$ is a baseline hazard function. The covariance structure of the random animal effect was modelled as a multivariate normal, and that of the common environment as a log-gamma distribution.

Heritability of frailty model was quantified as $h^2 = V_G(V_G + V_C + \pi(6)^{-1})^{-1}$, where V_G is the mode of genetic and V_C the mode of common environment variance estimated from a Laplacian approximation of the corresponding marginal posterior distribution (Ducrocq & Casella, 1996). Due to failure in iterating Gauss–Hermite quadrature of the approximate marginal posterior densities, the standard errors for variances could not be conclusively obtained and therefore are not reported.

The sub-set data was also analysed using linear models in which survival was defined as binary traits (EarlyJuv, LateJuv and JuvTotal). This was done to compare results from the linear and survival models.

3. RESULTS

(i) *Heritability of survival traits*

All three juvenile fingerling survival traits displayed significant amounts of genetic variation (h^2: JuvTotal = 0·19, EarlyJuv = 0·20 and LateJuv = 0·27; Table 2). Heritabilities for the grow-out survival traits were significant, but slightly lower than those for the fingerling period (h^2: FreshwG = 0·16, SeaG = 0·06; Table 2). The common environment effect was significant, but fairly low, for all traits (c^2 range: 0·04–0·12; Table 2).

(ii) *Genetic correlations within fingerling period*

The positive genetic correlation between early and late fingerling survival traits was moderate, but non-significant ($r_G = 0{\cdot}30$; Table 3), suggesting that re-ranking occurs even within a life stage. In line with this observation, mortality during the fingerling period tended to occur during two separate peak times, at the beginning and end of the period (Fig. 1). Fingerling period mortality was constant only in two year classes (1998 and 2000). Three year classes (1997, 1999 and 2000) had higher mortality both at the beginning and end with a plateau in the middle, and the remaining three year classes had higher mortality either in the beginning or end of the period (Fig. 1). In addition, the common environment correlation between EarlyJuv and LateJuv did not differ from zero ($r_C = 0{\cdot}04$; Table 3).

Genetic correlations of both EarlyJuv and LateJuv with JuvTotal were highly positive (r_G: 0·86 and 0·76, respectively; Table 3), as can be expected for traits that are components of a whole. Similarly, the common environment correlations between the

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Table 3. *Genetic associations of different survival traits during ontogeny estimated from the whole data: below diagonal = genetic correlations (SE), above diagonal = common environment correlations (SE). Significant (zero not within estimate ± 1·96 SE) correlations in bold. All correlations significantly different from unity*

	EarlyJuv	LateJuv	JuvTotal	FreshwG	SeaG
EarlyJuv		0·04 (0·03)	**0·74** (0·02)	0·06 (0·04)	0·09 (0·06)
LateJuv	0·30 (0·16)		**0·69** (0·02)	−0·02 (0·05)	0·03 (0·05)
JuvTotal	**0·85** (0·05)	**0·76** (0·08)		0·03 (0·04)	0·09 (0·06)
FreshwG	−0·08 (0·12)	0·27 (0·14)	0·09 (0·12)		**0·59** (0·05)
SeaG	−0·22 (0·17)	0·33 (0·18)	0·03 (0·16)	**0·62** (0·09)	

component and total fingerling survival traits were highly positive ($r_C = 0{\cdot}71$–$0{\cdot}73$; Table 3).

(iii) *Detailed survival analysis of time at death within fingerling period*

For the year classes 1997, 1998 and 1999, the heritability estimate from the frailty model for time at death during the fingerling period was moderate ($h^2 = 0{\cdot}15$) and only slightly higher than the heritability from the linear model [h^2 (SE): JuvTotal 0·11 (0·04)]. Thus, modelling survival as a continuous time with the frailty model added some but minor additional information.

Surprisingly, genetic correlations between the fingerling survival traits in the sub-set data were much higher [r_G (SE): EarlyJuv versus LateJuv = 0·89 (0·38), EarlyJuv versus JuvTotal = 0·95 (0·16) and LateJuv versus JuvTotal = 0·99 (0·05)] than in the analysis of the whole dataset (r_G: EarlyJuv versus LateJuv = 0·30, EarlyJuv versus JuvTotal = 0·86, and LateJuv versus JuvTotal 0·76; Table 3). This means that in the sub-set data, survival was almost a single trait across the fingerling period, a conclusion contrary to the results of the whole data.

Although EarlyJuv and LateJuv were highly correlated in the sub-set of data, the estimated survival curve for these year classes substantiated the finding that the survival probability drops at the beginning and end of the fingerling period separated by a mid-period plateau (Fig. 2).

(iv) *Sire-family correlations across egg-fry and fingerling stages*

The sire-family mean correlations of egg-fry survival with fingerling survival traits were in most cases close to zero [mean r_S across year classes (range): JuvTotal = −0·03 (−0·18–0·23), EarlyJuv = 0·01 (−0·15–0·29) and LateJuv = −0·09 (−0·19–0·00)]. Only three correlations out of 19 differed significantly from zero [year class 1996: EarlyJuv $r_S = 0{\cdot}29$, $P = 0{\cdot}01$; year class 2004: JuvTotal $r_S = -0{\cdot}18$, $P = 0{\cdot}04$, LateJuv $r_S = -0{\cdot}19$, $P = 0{\cdot}03$). The low sire-family mean correlations indicate that survival during egg-fry and fingerling periods are genetically separate traits.

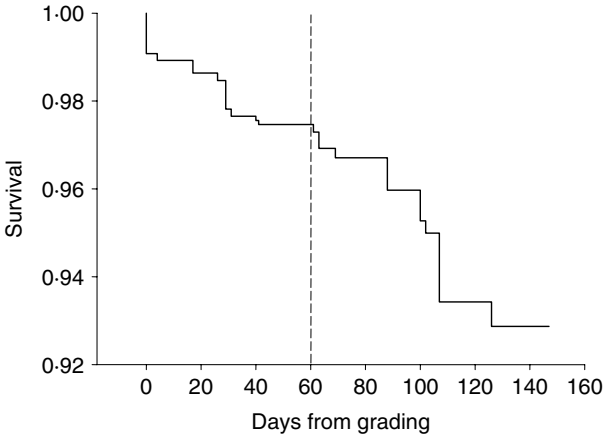

Fig. 2. Kaplan–Meier estimated survival curve for year classes 1997, 1998 and 1999. Dashed vertical line marks the division of fingerling period to early and late fingerling survival traits.

(v) *Genetic correlations across fingerling and grow-out stages*

The differences in genetic architecture between fingerling and grow-out survival traits were similar to those found for traits within the fingerling period. Genetic correlations between fingerling and grow-out survival were generally low (mean $r_G = 0{\cdot}06$; Table 3) suggesting that survival traits during different life stages do not share common genetic architecture.

Furthermore, the grow-out traits also displayed genotype × environment interaction, as revealed by the non-unity genetic correlation between FreshwG and SeaG ($r_G = 0{\cdot}62$; Table 3).

The common environment correlations between fingerling and grow-out survival traits were low (mean $r_C = 0{\cdot}05$; Table 3).

4. DISCUSSION

The genetic analysis of rainbow trout survival across life stages revealed two major patterns. First, strong

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genotype re-ranking across life-stages was evidenced by the weak sire-family correlations between egg and fingerling stages, and near zero genetic correlations within fingerling stage and between fingerling and grow-out stages. Second, the results changed considerably depending on the dataset analysed. These results reveal the transient nature of the genetic architecture of survival, a composite trait recorded without knowing the exact mortality agents.

(i) *Survival within fingerling stage*

Non-significant genetic correlation (0·30) between early and late fingerling survival in the whole data suggested that the genetic architecture of survival can vary even within a life stage. Indeed, fingerling mortality in many year classes displayed seasonal variation: there was a peak in mortality either early (late summer) or late (late autumn–winter) in this period, or both (Fig. 1). Mortality showed constant rate through fingerling period in only two out of eight year classes. The early and late periods typically have different diseases, e.g. *Flavobacterium columnare* in summer and *Flavobacterium psychrophilum* in winter, and also abiotic conditions such as water temperature change during the fingerling period.

To examine whether more exact timing of death during the fingerling period provides additional information on the genetics of survival, the sub-set data (year classes 1997, 1998 and 1999) with more frequent and longer observation period of fingerling survival were analysed with both linear and frailty Cox's proportional hazard model. The frailty model produced only slightly higher heritability compared to that of the linear model [h^2: linear model (JuvTotal) = 0·11, frailty model = 0·15]. The small change in the heritability estimate between the models indicates that in the sub-set data, time at death provides only limited novel information on the genetics of fingerling survival, and that linear model results are robust. In the sub-set data, early and late fingerling survival were also more strongly (0·89) genetically correlated than in the whole data. This sort of difference between datasets is expected if there is spatio-temporal variability in mortality factors causing variation in genetic parameters (Vehviläinen *et al.*, 2008). For instance, in our previous study, we showed that heritability for rainbow trout survival during grow-out stage ranges between 0·04 and 0·71 depending on the year class analysed (Vehviläinen *et al.*, 2008).

Accordingly, this supports the view that during particular years genetic architecture of survival can be rather homogeneous across a fingerling period. Causative mortality factors are unknown in the present study, but the conclusion is that during some years, it is possible to find a set of families that on average survive better than others through the challenges of the fingerling stage.

Previous studies have found favourable genetic associations between resistance to some mortality factors within a life stage. At the same time, however, these studies report that genetic correlations with other mortality factors, even within life stage, can be weak. In Atlantic salmon (*Salmo salar*), resistance against different bacterial diseases is usually favourably genetically correlated, whereas the genetic correlations between bacterial and viral diseases are weak or even negative (Gjøen *et al.*, 1997; Henryon *et al.*, 2005; Ødegård *et al.*, 2007; Kjøglum *et al.*, 2008). Bubliy & Loeschcke (2005) also showed that survival after different stressors in a fruit fly (*Drosophila melanogaster*) exhibit correlated responses to selection, even though results did not support existence of a single resistance mechanism.

(ii) *Survival across life stages*

Weak correlations across life stages suggested that the genotypes surviving best during the fingerling period were not among the best survivors in other life stages. This was evidenced by two results. First, survival from fertilized egg to the first feeding fry was in most year classes not correlated with the subsequent juvenile fingerling survival (mean $r_S = -0{\cdot}03$). Second, genetic correlations between fingerling and grow-out survival were very low (mean $r_G = 0{\cdot}06$).

If any trend for similar ranking of genotypes based on survival is visible across the life stages, it would be between late fingerling survival and grow-out survival traits ($r_G = 0{\cdot}29$–$0{\cdot}32$). This seems logical because during late fingerling period fish are gaining weight reaching body weight of 50–100 g, finishing the smoltification phase and approaching grow-out phase. In our previous study on the grow-out survival of rainbow trout across production environments, we found moderate genotype × environment interaction [mean (range) $r_G = 0{\cdot}70$ (0·17–0·98)] between survival in freshwater and different sea environments. This means that to some extent genotypes rank differently for survival in separate environments even within life stage (Vehviläinen *et al.*, 2008). In the current study, we found a slightly lower between environment genetic correlation (freshwater versus sea grow-out $r_G = 0{\cdot}62$). The present study thus revealed that the genetic correlations of survival across life stages are clearly lower than the genetic correlations of survival across environments.

In the course of a whole life cycle, both external environment and physiology of an individual fish change, providing potential for the reduced correlations. Our results imply that survival during different life stages are genetically different traits and that there are no superior genotypes that are able to tolerate all

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mortality factors through life stages from fertilized egg to 1–2 kg fish.

Similar to our results, previous studies have suggested that genetic correlations of survival across life stages are weak. Campbell (1997) found low and non-significant genetic correlation between seedling emergence and survival to flowering in a monocarpic herb Scarlet Gilia (*Ipomopsis aggregata*). Studies on piglet mortality from farrowing to weaning around 4 weeks of age have found close to zero or even negative genetic correlations between different periods, thus suggesting that different piglet survival traits do not share a common genetic background and should be treated as separate traits (Su *et al*., 2008, and references therein). Ducrocq *et al*. (2000) found that survival of laying hens during rearing (from birth to housing at 106 days) and productive periods (from housing to 313 days of age) are genetically different traits with different mortality rates and genetic correlation close to zero. In Pacific oyster (*Crassotrea gigas*), survival to 0·5 year is a genetically different trait than survival to 1·5 years (Ernande *et al*., 2003, 2004; Dégremont *et al*., 2007). However, Gjøen *et al*. (1997) studying resistance against a pathogenic bacteria *Aeromonas salmonicida* in Atlantic salmon, found a strong (0·95) genetic correlation between juvenile pre-smolt challenge test and post-smolt grow-out field data.

(iii) *Factors causing weak correlations between life stages*

That survival traits at different stages have partly different genetic architecture can be explained by two likely mechanisms.

Firstly, survival is caused by multiple mortality factors, whose incidence may differ between life stages. Resistances to different mortality factors do not necessarily share a common genetic determination (Gjøen *et al*., 1997; Henryon *et al*., 2005; Bubliy & Loeschcke, 2005; Ødegård *et al*., 2007; Kjøglum *et al*., 2008). This is for example highlighted by the fact that moderate genotype × environment interaction was also found for rainbow trout survival during the grow-out period, as shown by the non-unity genetic correlations between environments (Vehviläinen *et al*., 2008; present study). Similarly, different alleles of a single gene may provide resistance against different diseases or mortality factors (Shook & Johnson, 1999; Grimholt *et al*., 2003). In fact, one pattern behind the weak correlations across life stages was that sea grow-out survival, not freshwater, had the lowest correlations with the fingerling survival traits recorded in freshwater. Although speculative, it is possible that fish in sea grow-out confront different mortality factors compared to fingerlings and grow-out fish in freshwater.

Secondly, it is possible that mortality factors are the same in different life stages, but the ranking of genotypes in resistance against the mortality factor(s) changes during ontogeny. When two traits are selected in the same direction, a non-favourable genetic correlation (genetic trade-off) is assumed to evolve between the two traits (reviewed by Roff, 1996). This applies well to the survival traits in different life stages analysed here. It is clear that egg, yolk-sac, fry, fingerling and grow-out fish have very different resistance mechanisms and that resistance level to a multitude of mortality factors may change during ontogeny. Thus a single family does not need to be superior for all of these mechanisms.

(iv) *Survival heritability and multiple mortality factors*

The low heritability of survival is generally hypothesized to be a result of strong selection on this important fitness component, reducing additive genetic variance for survival (Fisher, 1930; Mousseau & Roff, 1987; Roff & Mousseau, 1987). In this study, we found significant, but fairly low heritabilities (0·06–0·27) for survival traits. Low heritability of survival has been found in diverse taxonomic groups in farmed terrestrial animals (e.g. van Arendonk *et al*., 1996; Ducrocq *et al*., 2000; Knol *et al*., 2002; Goyache *et al*., 2003; Casellas *et al*., 2007; Su *et al*., 2008) and in wild organisms (e.g. Futuyma *et al*., 1995; Campbell, 1997). Moreover, low or even negative genetic correlations between mortality factors across life stages (present study) and across environments (Vehviläinen *et al*., 2008) may easily lead to low heritability for birth-to-adulthood survival. When multiple agents of mortality (e.g. different diseases, predators and physiological effects) do not share common genetic determination, heritability of overall survival may be reduced through decreased genetic variance and/or increased residual variance (Vehviläinen *et al*., 2008). This can happen even when individual component traits of survival display moderate levels of genetic variation (Price & Schluter, 1991; Houle, 1992; Hoffmann & Merilä, 1999; Merilä & Sheldon, 1999; Vehviläinen *et al*., 2008).

(v) *Conclusions*

Taken together, the amount of genetic variance and mostly positive genetic correlations found within the fingerling period support the existence of some genotypes with superior survival within the fingerling stage. However, weak associations between survival during egg-fry and fingerling periods, and generally low genetic correlations between fingerling and grow-out survival suggested that survival across life stages is best regarded as separate traits. The study

demonstrates extensive spatio-temporal variation in the genetic (co)variance structure of survival.

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