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Resistance against infectious pancreatic necrosis exhibits significant genetic variation and is not genetically correlated with harvest weight in rainbow trout (*Oncorhynchus mykiss*)

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ABSTRACT

Infectious pancreatic necrosis (IPN) is one of the most prevalent and economically important diseases in rainbow trout aquaculture (*Oncorhynchus mykiss*). Vaccines as a conventional control measure have shown variable results under production conditions. Genetic improvement for resistance to IPN represents an alternative for the prevention of disease outbreaks. The objective of the present work was to estimate the heritability and genetic correlation for IPN resistance and harvest weight (HW) in rainbow trout. To determine the genetic resistance to the IPN virus, a total of 2278 fingerlings from 58 full-sib families were used, which were challenged with IPN virus to induce the disease. Resistance refers to survival to the disease and was defined as the day of death of each fish. HW was also recorded in 13,241 genetically related individuals from the same population. For the genetic analysis we fitted a bivariate mixed linear model including HW and resistance to IPN as dependent variables; tank:year:sex as a fixed contemporary group and age at harvest as a covariate for HW; and final weight as a covariate for resistance to IPN. The animal effect was included as a random effect for both traits. A random effect associated with common environment was also included for HW. The estimated heritability for IPN resistance was 0.39 ± 0.08 and 0.35 ± 0.06 for HW. Genetic correlation between IPN and HW resistance was not significant (0.05 ± 0.25). The results indicate that the heritability for both traits is moderately high in this population, and that there is no significant genetic correlation between them. The presence of significant genetic variation for both IPN and HW resistance and the absence of genetic correlation between both traits indicate the feasibility of improving them simultaneously by means of artificial selection.

1. Introduction

Rainbow trout is one of the most important species among cultured salmonids, with an estimated world production of 813 thousand tons in 2014, and an estimated value of US$ 3933 million (FAO, 2016). Chile makes an important contribution to the worldwide production of rainbow trout, reaching 152 thousand tons in 2015 (SERNAPESCA, 2016). One of the key factors affecting the profitability and sustainability of this industry is health status, which depends mainly on the control of infectious diseases affecting fish under culture (Yáñez and Martínez, 2010).

Infectious pancreatic necrosis (IPN) virus (*Aquabirnavirus* genus; *Birnaviridae* family) causes an acute viral disease, which generates high mortality in first-feeding juveniles (Roberts and Pearson, 2005) and smolts in the first six months after transfer to seawater (Wolf et al., 1960). Fish surviving the infection can become healthy carriers, which can infect susceptible animals (Wolf et al., 1960; Wolf, 1988), either by vertical and/or horizontal transmission. Vaccination may provide some protection against disease in juvenile fish (Ramstad and Midtlyng, 2008), but control in freshwater environments generally depends on the biosecurity measures and innate resistance of juvenile fish.

From a sustainability and animal welfare perspective and to reduce the negative effects of this disease, the aim should be to increase resistance to IPN through genetic improvement. This strategy is a viable

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alternative for the control of infectious and parasitic diseases in domestic animals (Stear et al., 2001; Yáñez and Martínez, 2010; Yáñez et al., 2014a). Genetic improvement increases the intrinsic potential of animals to resist infection and is a complementary tool in the control of infectious and parasitic diseases. However, in order to succeed in the genetic improvement for resistance to IPN, there must be additive genetic variation of this trait in rainbow trout.

Several studies indicate the presence of significant genetic variation for resistance to pathogens within rainbow trout populations (Rye et al., 1990; Dorson et al., 1995; Slierendrecht et al., 2001; Perry et al., 2004; Henryon et al., 2002, 2005; Silverstein et al., 2009). These results are encouraging, since they allow genetic improvement for resistance to infectious diseases to be feasible. It has also been observed that there is a genetic component associated with IPN resistance in Atlantic salmon with heritabilities ranging from 0.31 ± 0.06 to 0.45 ± 0.07 (Guy et al., 2006, 2009; Wetten et al., 2007; Storset et al., 2007; Kjøglum et al., 2008; Gheyas et al., 2010a, 2010b). For example, a heritability between 0.38 and 0.43 in the marine phase has been reported for IPN resistance (Houston et al., 2008), while in the freshwater phase the value is 0.31 (Houston et al., 2010). In addition, it has been shown that genetic variation for IPN in Atlantic salmon is controlled by a locus of major effect (Houston et al., 2008, 2010; Moen et al., 2009). However, to date there is scarce evidence of genetic variation for resistance to IPN in rainbow trout. Okamoto et al. (1993) demonstrated that the progeny of a rainbow trout strain showed genetic resistance to IPN after a spontaneous outbreak. In addition, the use of low-resolution molecular markers has shown the presence of genomic regions associated with resistance and susceptibility to IPN in rainbow trout (Ozaki et al., 2001). There are no available reports of heritability for IPN resistance in rainbow trout in the scientific literature. The possibilities to improve resistance to IPN depend on the genetic correlations between resistance and other traits of economic interest, such as growth rate. The more favorable the genetic correlations, the easier it is to improve traits simultaneously.

The objective of the present study is to estimate the level of genetic variation for resistance against IPN virus in rainbow trout from survival data obtained from experimental challenges. In addition, we also estimated the genetic correlation between resistance to IPN and body weight at harvest. This information will be useful for planning of disease control strategies through genetic improvement for IPN resistance in rainbow trout.

2. Material and methods

2.1. Experimental population

The fish belong to the genetic nucleus of Aguas Claras SA, and were challenged at the Aquainnovo Center for Research and Aquaculture Transfer (Puerto Montt, Chile). IPN resistance data were obtained from a sib-testing scheme, in which 58 families of full siblings of rainbow trout, with a representation of 17 to 50 fish per family from the 2014 year-class were experimentally challenged against IPN virus. Growth was measured on relatives from year-classes 2008 and 2011, tied through pedigree connections to the challenged fish. There were 2278 and 13,241 fish with records for resistance to IPN and body weight at harvest, respectively. A 3-generation pedigree was used (2008, 2011 and 2014) with a total of 20,529 recorded fish (Table 1). Fertilization of families took between one to five weeks depending on the spawning year. Fertilized ova from each family were incubated separately until hatching. The fish were then marked at about 2 to 7 g with PIT-tags (Passive Integrated Transponders), which were inserted in the abdominal cavity to preserve genealogical information during the challenge test and the grow-out period, for the fish measured for IPN resistance and body weight at harvest, respectively. Fish recorded for growth were then transferred to fresh water sites until smoltification. An average number of 20 to 83 individuals from each family were randomly divided into two fresh water tanks for year-classes 2008 and 2011. After smoltification, fish were transported to sea cages, maintaining the tank distribution established during fresh water rearing. Fish were then reared until market size at approximately 3 kg (on average 25 months post-spawning), in which harvest weight (HW) was recorded for all fish. The variables recorded for the IPN resistance trait were day of death after inoculation and weight at end of challenge. The variables recorded for the growth trait were body weight at harvest, age at harvest, sea cage, year and sex (determined by ultrasound imaging).

Table 1
Summary information of the pedigree from the rainbow trout (*Oncorhynchus mykiss*) breeding population used in the present study by year.

			Number of offspring	
Year	Number of sires	Number of dams	Total number	Mean per full-sib family
2008	18	48	4946	103
2011	48	106	13,305	126
2014	30	58	2278	39
Total	96	212	20,529	97

2.2. Experimental challenge test

Prior to challenge, the sanitary condition of 30 fries from the full-sib families to be experimentally challenged was evaluated randomly. Quantitative real-time PCR was used to detect *Flavobacterium psychrophilum* following the highly sensitive protocol described by Strepparava et al. (2014), which has shown a detection limit of 20 gene copies. Quantitative real-time reverse transcriptase PCR was used to determine the presence of IPN virus following the highly sensitive protocol described by Bowers et al. (2008), which has shown a detection limit of 10 RNA copies. Negative results were obtained for both pathogens. In addition, the latter method was used to confirm the cause of death generated post challenge. All these diagnostic analyses were carried out in the ALAB SA. (Puerto Montt, Chile). At the time of inoculation with IPN, the fish had an average weight of 2.24 (0.71) gr and 154 (15) days of age. The challenge was performed with an IPN virus isolate (virulent Sp serotype) in a 0.25 m^3 tank with fresh water in a recirculation system at an average temperature of 11 °C, oxygen saturation of 95.74% and salinity of 3.46 ppt. The IPN virus isolate (CD-AQ03) was purchased from Centrovet Ltda. (Puerto Montt, Chile). The virus was isolated from affected Atlantic salmon kidney from a Chilean farm (X^{th} Region) in November 2014 using CHSE-214 cell line and then cryopreserved until the preparation of inoculum in RTG-2 cell line.

The challenge was carried out in two stages; i) intraperitoneal inoculation at a rate of 0.05 mL/inoculum fish, at a concentration of $10^{7.82}$ TCID50/mL, determined by the Kärber-Spearman method (Hamilton et al., 1977); and ii) the immersion was then carried out, with 1.1 L of the inoculum diluted in 5 L of water and then poured into the tank which contained 130 L of water and kept at retained flow for 4 h and 17 °C. At the end of the immersion, fresh water was incorporated at 10 °C, causing a thermal shock. Mortality was withdrawn from the tank on a daily basis and each time it occurred, in order to record the day of death for each fish. At the end of the evaluation period (day 63), all surviving fish were euthanized. All the experimental challenge procedures were approved by The Comité Institucional de Cuidado y Uso de Animales (CICUA) from the University of Chile (Certificate N° 17,019–VET-UCH).

2.3. Estimation of co-variance components

To estimate the components of variance and covariance for HW (y_1) and resistance to IPN (y_2), the following bivariate mixed linear model was used:

$$\begin{bmatrix} y_1 \\ y_2 \end{bmatrix} = \begin{bmatrix} x_1 & 0 \\ 0 & x_2 \end{bmatrix} \begin{bmatrix} b_1 \\ b_2 \end{bmatrix} + \begin{bmatrix} z_1 & 0 \\ 0 & z_2 \end{bmatrix} \begin{bmatrix} u_1 \\ u_2 \end{bmatrix} + \begin{bmatrix} w_1 & 0 \\ 0 & 0 \end{bmatrix} \begin{bmatrix} c_1 \\ 0 \end{bmatrix} + \begin{bmatrix} e_1 \\ e_2 \end{bmatrix}$$

where, y_1 and y_2 are vectors of phenotypic records measured in animals for HW and resistance to IPN, respectively; b_1 is the vector of fixed effects for HW, including contemporary group as factor (tank:year:sex) and age at harvest as covariate; b_2 is a fixed effects vector for IPN resistance, including body weight at the end of the experiment as a covariate; u_i and e_i are vectors of random animal genetic effect and residual effects for HW and resistance to IPN, respectively;$_{c1}$ is the random effect vector of common environment associated with families of full siblings for HW; X_i and Z_i are the design matrices for HW and resistance to IPN; and $w_1$is the design matrix for HW.

For both traits the random effects associated to animal, common and residual environment effects, were assumed:

$$u = [u_1' u_2']' \sim N(0, G_0 \otimes A)$$

$$c = [c_1']' \sim N(0, C_0 \otimes I_C)$$

$$e = [e_1' e_2']' \sim N(0, R_0 \otimes I_N)$$

Where, A is the matrix of additive genetic kinship among all the fish included in the pedigree; I_c and I_N are identity matrices of dimension C and N, respectively; and ⊗ indicates the direct operator of the products; G_0 and R_0 denote the matrices of variances and covariances of 2 × 2 of animal and residual additive genetic effect, respectively. A random effect of common environment associated with families of full siblings was evaluated in a preliminary analyses using a single-trait likelihood ratio test (Lynch and Walsh, 1998). This effect was only significant for HW (p-value < 0.05). Therefore, only the final bivariate model included the common environment effect for HW. C_0 represents a 1 × 1 scalar matrix of common environment effects for HW. The parameters of the bivariate mixed linear model were estimated using the restricted maximum likelihood method (REML) implemented in ASREML version 3.0 (Gilmour et al., 2009).

2.4. Heritability and genetic correlation

The following formula was used to calculate h^2 for the traits of body weight to harvest (HW) and resistance to IPN:

$$h_i^2 = \frac{\sigma_{Gi}^2}{\sigma_{Gi}^2 + \sigma_{Ci}^2 + \sigma_{Ei}^2}$$

Where, i represents HW or resistance to IPN, σ_{Gi}^2 is the additive genetic variance of the matrix G_0, σ_{Ci}^2 is the variance explained by the common environment effect associated with the families of full siblings of the matrix C_0 and $\sigma_{Ei}^2$is the residual variance of the matrix R_0. It must be noted that for the case of IPN resistance, the common environment effect was not taken into account. The genetic correlation (r_{xy}) between the two traits, HW and IPN resistance was calculated according to Falconer and Mackay (1996). Because the traits were recorded on different animals, the residual covariance was set to zero.

3. Results

3.1. Experimental challenge test

During the 63 days of challenge, the tank reached an average number of dead fish per day of 6 (SD = 4), with a minimum of 0 and a maximum of 23 dead fish per day. The percentage of total accumulated mortality was 13.77%. The Kaplan-Meier survival curve (Kaplan and Meier, 1958) was plotted for the best and worst full-sib family, according to the cumulative percentage of mortality, as well as for the mean of all families (Fig. 1). These results confirm the presence of significant variation (p-value < 0.05) for resistance to IPN between families, based on the Kaplan-Meier (Log-rank) survival analysis. The percentage of accumulated mortality per family ranged between 0% and 47.62%.

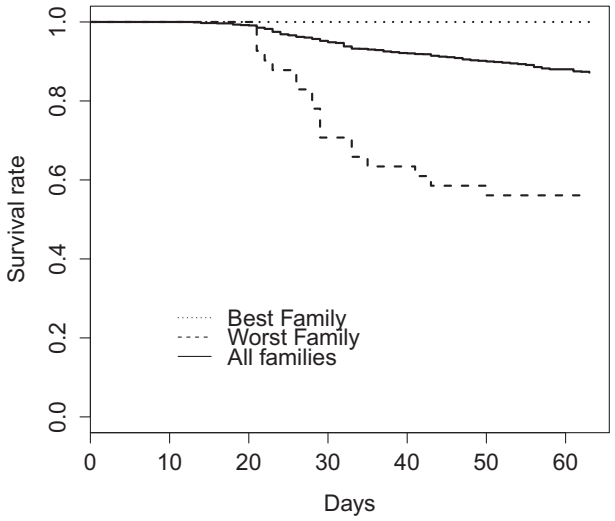

Fig. 1. Kaplan–Meier survival curve of an average of the 58 full-sib families, the worst and the best family after an experimental challenge with infectious pancreatic necrosis virus in rainbow trout (*Oncorhynchus mykiss*).

3.2. Phenotypic variation for HW and IPN

The general mean for HW was 2.76 kg (SD = 0.89) and the coefficient of variation was 32.18%. The minimum and maximum values for HW were 0.20 kg and 6.09 kg, respectively. In these fish, body weight was recorded approximately 25 months after spawning. On the other hand, resistance to IPN measured on days of survival to the challenge test, presented an average of 57 days (SD = 9 days), a coefficient of variation of 16.52%, a minimum value of 13 days and a maximum of 63 days (Table 2).

3.3. Heritability and genetic correlation

Significant additive genetic variation was identified for both traits (Table 3). The estimated heritability values were moderate for resistance to IPN (0.39 ± 0.08) and HW (0.35 ± 0.06), respectively. The effect of common environment associated with families of full siblings represented 7.9% of the phenotypic variance of body weight at harvest. The genetic correlation between resistance to IPN and HW is not significantly different from zero. The phenotypic correlation could not be estimated because both traits were measured in different individuals.

4. Discussion

Previous studies on rainbow trout have reported significant genetic variation for body weight. For example, low to moderate heritability values for body weight have been estimated, ranging from h^2 = 0.16 ± 0.02 to 0.37 ± 0.04 (Aulstad et al., 1972; Gunnes and Gjedrem, 1981; Gjerde and Gjedrem, 1984; Gjerde and Schaeffer, 1989; Su et al., 1996; Henryon et al., 2002; Pante et al., 2002; Haffray et al., 2012; Janhunen et al., 2012; Sae-Lim et al., 2015). In the present study the heritability estimate for body weight at harvest was moderate (0.35 ± 0.06) and is within the upper range of previous reports. In addition, Pante et al. (2002) demonstrated the significance of common environmental effect on body weight in three rainbow trout populations, where they estimated that heritabilities decreased from 0.37 to 0.16; 0.36 to 0.10 and 0.46 to 0.17 in the different populations, when the common environment effect was included in the model. On the other hand, Janhunen et al. (2012) reported a heritability of 0.35 ± 0.05 and Sae-Lim et al. (2015) reported heritabilities of 0.26 ± 0.03 and 0.22 ± 0.03 for body weight, including the common

Table 2
Summary statistics for resistance against infectious pancreatic necrosis (IPN) and harvest weight, by each replicated tank in the rainbow trout (*Oncorhynchus mykiss*) breeding population used in the present study (standard deviation in parenthesis).

Trait	Tank/sea cage	n[a]	n/family	Mean	CV[b]	Min	Max	BW test[c]	Age[d]
IPN resistance (days)[e]	2014A	2278	39.28 (8.85)	57.17 (9.44)	16.52	13	63	0.010 (0.005)	–
Harvest weight (kg)[f]	2008A	940	20.00 (4.37)	1.89 (0.43)	23.01	0.73	3.02	–	743.9 (8.99)
	2008B	4006	83.46 (14.03)	2.65 (1.25)	47.27	0.20	6.09	–	770.3 (32.79)
	2011A	4177	39.26 (4.17)	3.24 (0.94)	29.40	0.30	6.00	–	755.6 (6.21)
	2011B	4118	38.80 (3.83)	3.26 (0.95)	29.27	0.30	5.76	–	755.5 (6.27)

[a] Number of fish included in the analysis after removing outliers by interquartile range rule and discarding missing values.
[b] Coefficient of variation.
[c] Body weight measured in kilograms at the end of the challenge test.
[d] Age at the recording time measured in days post spawning.
[e] IPN resistance measured as the day of death after 63 days of the experimental challenge.
[f] Harvest weight measured in kilograms.

Table 3
Phenotypic, additive, common environment and residual variance components ($\sigma_p^2, \sigma_a^2, \sigma_c^2, \sigma_e^2$ respectively), heritabilities (h^2) and genetic correlation (r_g) for harvest weight (HW) and infectious pancreatic necrosis resistance (IPN) in a rainbow trout breeding population. (± standard error).

		HW	IPN
	σ_p^2	0.63 ± 0.02	83.94 ± 4.32
	σ_a^2	0.22 ± 0.04	32.81 ± 7.80
	σ_c^2	0.05 ± 0.001	–
	σ_e^2	0.36 ± 0.02	51.13 ± 4.47
	h^2	0.35 ± 0.06	0.39 ± 0.08
r_g	HW	–	0.05 ± 0.23

environment effect in the statistical model. In the present study, we also detected a significant common environmental effect for body weight at harvest, which is associated with the common breeding of full sibling groups in family tanks prior to labeling using PIT-tags. The magnitude of common environment effect can be expressed as a proportion of phenotypic variation, which is slightly higher ($c^2 = 0.079$) than the values reported by Pante et al. (2002) ($c^2 = 0.038 - 0.062$), Janhunen et al. (2012) ($c^2 = 0.05 \pm 0.009$) and Sae-Lim et al. (2015) ($c^2 = 0.04 \pm 0.006$). In the present study the relative importance of the estimated components of variance suggests a lower residual variation and greater additive genetic variation for body weight at harvest compared to previous studies. Therefore, the additive genetic variance represents 34.92% and the residual variance represents 57.14% of the total phenotypic variance for body weight to harvest in the population. The magnitude of the additive and residual genetic components explaining the total phenotypic variation of body weight at harvest in a particular population may vary according to environmental conditions, and consequently heritability may change from one population to another (Falconer and Mackay, 1996; Visscher et al., 2008).

We believe the present study constitutes the first report on level of additive genetic variation for IPN resistance in a rainbow trout population, whose heritability estimate was moderate (0.39 ± 0.06). Previous studies have reported variation in resistance to IPN among different cultured varieties of rainbow trout. In this way, Okamoto et al. (1993) showed that the progeny of a rainbow trout population acquired genetic resistance to IPN following a spontaneous outbreak, and the average mortality in the resistant strain (YN-RT201) was 4.3% while it is 96.1% in a highly sensitive strain (YK-RT101). Recent studies have shown significant heritabilities for resistance against IPN in Atlantic salmon (Guy et al., 2006; Storset et al., 2007). Using the same trait definition (day of death) and similar models of genetic analysis, values of heritability of 0.31 and 0.43 in Atlantic salmon have been reported, showing a correspondence with the estimated value in the present work in rainbow trout. In addition, heritability values for IPN resistance between 0.26 and 0.55 have been reported in this same species, estimated from the binary survival trait by threshold models (Wetten et al., 2007; Kjøglum et al., 2008; Guy et al., 2009; Gheyas et al., 2010b; Houston et al., 2010). Similar heritability values have been reported for resistance to other viral diseases in salmonids. For example, ISA resistance in Atlantic salmon has reported heritability values between 0.13 ± 0.03 and 0.40 ± 0.04 (Gjøen et al., 1997; Olesen et al., 2007; Ødegård et al., 2007a, 2007b; Kjøglum et al., 2008; Gjerde et al., 2009). In addition, values between 0.13 and 0.63 ± 0.26 have been reported for VHS resistance in rainbow trout (Dorson et al., 1995; Henryon et al., 2002, 2005). Heritability indicates that selection to improve resistance to IPN in rainbow trout is feasible and will allow the generation of animals intrinsically resistant to the virus.

Previous studies have determined the molecular basis of resistance to IPN in rainbow trout and Atlantic salmon using genomic techniques. For example, QTL (*Quantitative trait loci*) analyses have been carried out for IPN resistance in rainbow trout (Ozaki et al., 2001, 2007) and Atlantic salmon (Houston et al., 2007, 2008, 2009, 2012; Storset et al., 2007; Moen et al., 2009, 2015; Gheyas et al., 2010a, 2010b). These studies have determined genomic regions involved in the resistance to IPN in both species. However, further progress has been made in determining the causal genes of resistance in Atlantic salmon compared to rainbow trout. Resistance to IPN in Atlantic salmon is determined by a locus of major effect located on chromosome 26, which is probably generated by variations in a gene coding for epithelial cadherin (Moen et al., 2015). However, further studies are needed in rainbow trout to perform fine mapping of genomic regions and genes associated with IPN resistance. This information will be useful to better understand the biological mechanisms between host-pathogen associated with resistance and to incorporate these markers within the selection schemes to accelerate the genetic progress for this trait. With the advent of new genomic technologies, such as next-generation sequencing (NGS) and *Single nucleotide polymorphisms* (SNPs) genotyping methods of high performance (Houston et al., 2014; Palti et al., 2015; Yáñez et al., 2014b, 2016b), a better understanding of the genomic basis of disease resistance traits is expected for salmonid species (Yáñez et al., 2015).

Some caveats need to be mentioned due to the technical limitations of the challenge procedure used here. First, only a single tank was used and thus the reproducibility of the heritability estimate is uncertain and probably biased if tank effect represents a significant source of variation for IPN resistance. Second, given the difficulty to reach intermediate mortalities in preliminary assays, fish were challenged simultaneously by both injection and immersion in order to ensure an appropriate cumulative mortality rate. However, we only reached nearly a 14% of total cumulative mortality at the end of the experiment. If resistance against both routes of viral inoculation has a different basis, both traits are confounded in the present study. These two limitations of the experimental challenge lead to some uncertainty regarding the heritability reported here and further studies are needed to corroborate this estimate.

Results from previous studies in rainbow trout have reported that the genetic correlation between growth and resistance to *F. psychrophi-*

lum is not different from zero (Silverstein et al., 2009). Similar results have been found between resistance to two bacterial diseases, columnaris disease (*F. columnare*) and bacterial cold water disease (*F. psychrophilum*) and body weight measured at 9 and 12 months, with genetic correlation ranging from $r_g = -0.15 \pm 0.08$ to -0.19 ± 0.24 (Evenhuis et al., 2015). The evaluation of genetic correlations between resistance to viral haemorrhagic septicemia (HSV) and traits related to growth (body weight, body length or feed conversion efficiency) in rainbow trout, shows values ranging from $r_g = -0.01$ to -0.33, suggesting a slightly negative relationship between growth and resistance to the virus (Henryon et al., 2002). In the same species, a positive phenotypic relationship between body weight and resistance to diseases caused by *F. psychrophilum* and *Yersinia ruckeri* has been reported. However, the phenotypic relationship between HSV and body weight was negative for one population and positive for another (Henryon et al., 2005). Recently, a moderate and negative genetic correlation between resistance against the intracellular bacteria *Piscirickettsia salmonis* and weight at harvest has been reported for coho salmon (*Oncorhynchus kisutch*), a closely related salmonid species (Yáñez et al., 2016a). In general, there is no pattern in the relationship between growth characteristics and resistance to specific pathogens in genetic terms. The present study demonstrates that there is no significant genetic correlation between weight at harvest and resistance to IPN in rainbow trout. These results indicate that genetic selection to increase growth, in terms of body weight at harvest, will not have an effect on genetic resistance to IPN in this species, and vice versa. Further studies are needed to determine whether the mechanisms involved in IPN resistance are genetically correlated with other characters of productive interest in rainbow trout.

From a practical point of view, these results show that it is possible to simultaneously include these two traits in breeding programs in rainbow trout through the use of selection indexes.

5. Conclusion

The levels of genetic variation determined for resistance to IPN and body weight at harvest demonstrate the feasibility of improving these traits through artificial selection in rainbow trout, which is an alternative for the control of the viral disease and the increase in the productive efficiency in the culture systems. The absence of a significant genetic correlation between these two commercially important traits in rainbow trout, suggests that artificial selection for resistance to IPN will not influence body weight at harvest and vice versa.

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