Genetic parameters for Piscirickettsia salmonis resistance, sea lice (Caligus rogercresseyi) susceptibility and harvest weight in rainbow trout (Oncorhynchus mykiss)

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Piscirickettsiosis caused by the intracellular bacterium Piscirickettsia salmonis and caligidosis produced by the ectoparasite Caligus rogercresseyi, are important diseases which generate great economic losses in salmonid farming in Chile. Selective breeding for pathogen resistance has been proposed as an alternative tool for the control of diseases. The objective of the present study is to determine the levels of genetic variation for resistance to P. salmonis and susceptibility to C. rogercresseyi, in addition to investigating the levels of genetic co-variation between these traits and harvest weight in rainbow trout. Resistance to P. salmonis was defined as individual day of death (SRS_DD) and as binary survival (SRS_BS) after an experimental challenge carried out by intraperitoneal injection of an inoculum based on LF89 strain. Susceptibility to C. rogercressevi (CAL) was measured as the sum of the parasitic load on the pectoral and caudal fins per fish after an experimental challenge. Harvest weight (HW) was recorded in individuals genetically related to challenged fish and analyzed to determine the genetic correlations between this trait and the previous ones. A linear multi-trait animal model was used to estimate (co)variance components for SRS_DD, CAL and HW. A single-trait probit threshold animal model was used to estimate variance components for SRS_BS on the underlying scale. Bivariate linear animal models were used to estimate genetic correlations between SRS_BS on the observed scale and all other traits. The heritabilities for SRS DD, CAL and HW were 0.45 ± 0.06 , 0.08 ± 0.02 and 0.35 ± 0.06 , respectively. The heritabilities for SRS_BS were 0.28 ± 0.03 and 0.38 ± 0.05 on the underlying and observed scale, respectively. The genetic correlation between SRS_DD and CAL and between SRS_BS and CAL were 0.39 ± 0.14 and $?0.34 \pm 0.15$, respectively. All other genetic correlations assessed were not significant. We concluded that there is significant additive genetic variation for P. salmonis resistance and C. rogercresseyi susceptibility, which indicates that it is possible to genetically improve these traits in rainbow trout. In addition, there is an unfavorable genetic correlation between P. salmonis resistance and C. rogercresseyi susceptibility and a null genetic correlation between growth and these traits. These results suggest that resistance to P. salmonis or C. rogercresseyi can be simultaneously improved with harvest weight. However, simultaneous selection for P. salmonis and C. rogercresseyi resistance must account for the unfavorable genetic relationship between both traits in this rainbow trout breeding population.