GENETIC BASIS OF HOST RESISTANCE AND TOLERANCE TO SCUTICULOCILIATOSIS IN TURBOT

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BACKGROUND

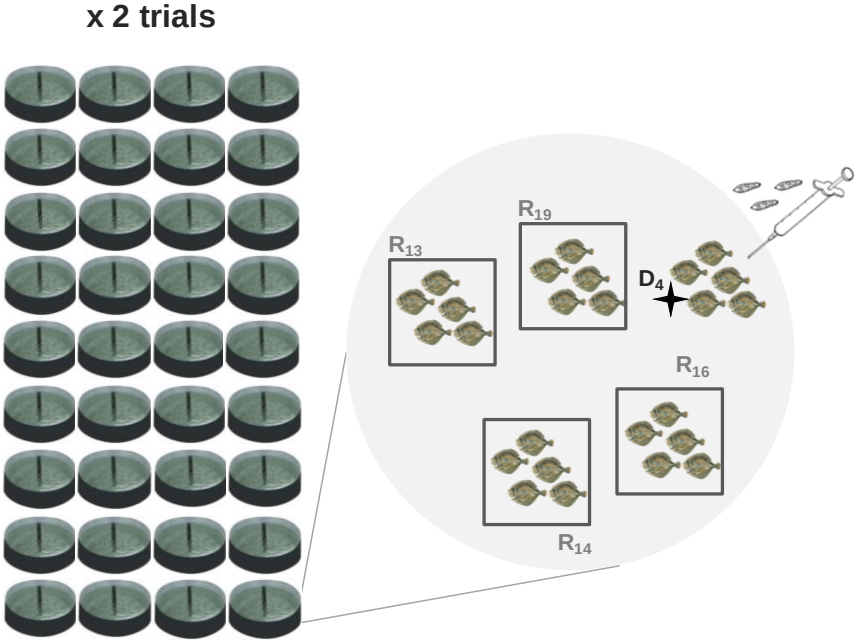
Genetic analyses of infectious disease data usually focus on (overall) disease resistance, but recent developments point towards additional host traits that may influence the risk and severity of infectious disease outbreaks, namely (i) resistance; i.e. the ability of host to reduce pathogen replication; (ii) tolerance; i.e. the ability of an infected individual to survive the infection; and (iii) infectivity; i.e. the ability of the host to transmit the infection (Lipschutz-Powell et al. 2014).

Here, framed within the EU project FISHBOOST (http://www.fishboost.eu/), for the first time an experiment designed for disentangling these three traits was developed. Empirical data from turbot (*Scophthalmus maximus*) challenged with the parasite *Philasterides dicentrarchi* have been analysed with the aim of estimating the heritability for resistance and tolerance. *Philasterides* is a parasite (protozoan ciliate) that causes scuticulociliatosis, a disease that produces high economic loss to the sector, for which Spain is the first producer in Europe and third worldwide. The experiment provided the rare opportunity to obtain data for both time of onset of infection as well as time of death, leading to a unique data set that is appropriate for our purpose.

METHODS

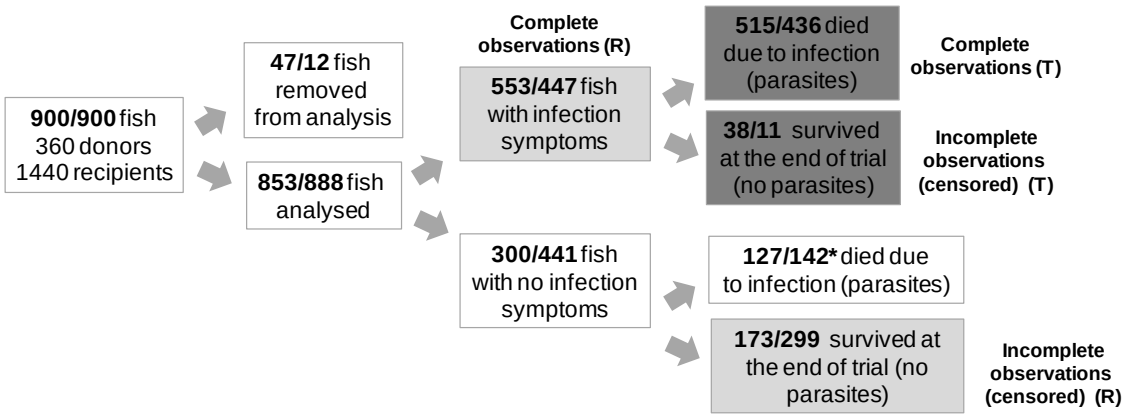
The experiment comprised eight turbot 'donor' families that were injected with the parasite, and these were used to infect 36 'recipient' families by cohabitation. Recipient families were distributed into 72 tanks, with one donor family per tank. Due to logistic matters, the experiment was carried out in two phases (trials) (Figure 1).

Figure 1. *Experimental design. Each trial comprised 36 tanks with 25 individuals each from five different families that included one donor (D) and four recipient (R) families.*



Phenotypes were defined as: (i) time (days) from the start of the experiment to the onset of symptoms (resistance), and (ii) time (days) from the onset of symptoms to death (tolerance). Figure 2 summarizes the number and type of data used for the analysis of resistance and tolerance. Phenotypic and genetic analyses were performed using survival techniques.

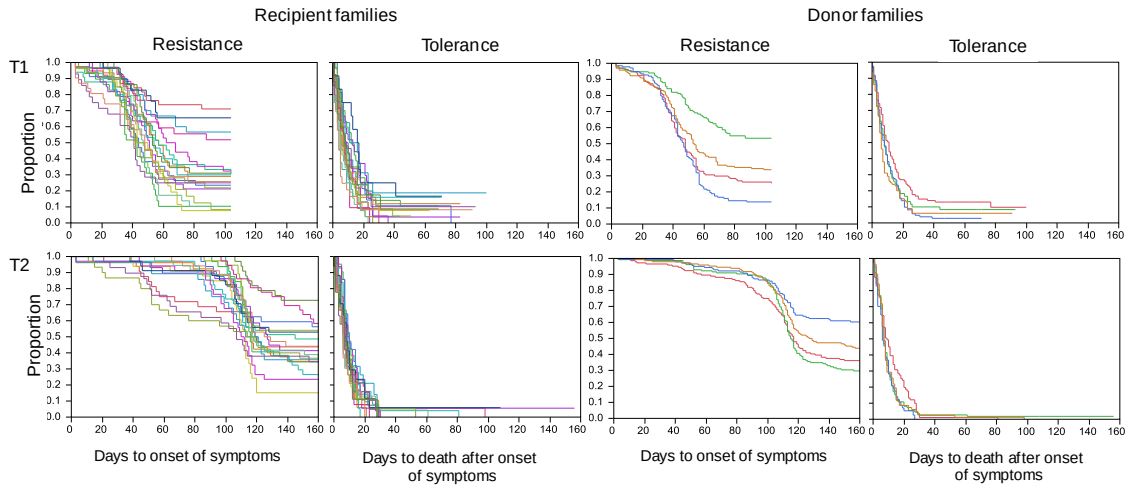
Figure 2. *Number and type of data used for the analysis of resistance (R, light grey) and tolerance (T, dark grey) for trials 1 and 2, respectively (separated by a slash). Records were considered incomplete (censored) when individuals did not show symptoms (resistance) or death (tolerance) at the end of the trial but could have shown it if the experiment continued.*



PHENOTYPIC ANALYSIS

Phenotypic variation was visualized with survival curves obtained with the non-parametric Kaplan-Meier estimator of the survivor function using JMP (SAS Institute Inc. 2010). High variation was observed in both recipient and donor families and this variation was higher for resistance than for tolerance (Figure 3). Also, the pattern observed for two trials was different, probably due to uncontrollable differences in the parasite strains between the trials.

Figure 3. *Kaplan-Meier survival curves representing the probability of surviving (from the start of the experiment to the onset of symptoms -resistance- or form symptoms to death -tolerance) across time (days) for the two trials (T1 and T2). Individuals are grouped by recipient or donor families, respectively.*



GENETIC ANALYSIS

Genetic variation was investigated using a proportional Hazards model with the software Survival Kit (Mészáros et al. 2013). For both resistance and tolerance, a Cox model including tank (fixed effect) and the additive genetic effect of the animal (random effect) was fitted. For resistance, donor family and initial weight were additionally included as fixed effects.

All fixed effects showed significant contribution to both the risk of onset of symptoms (resistance) and to the risk of death (tolerance), according to the likelihood ratio test. For resistance, the factor showing the largest contribution to the likelihood was the donor family (suggesting that variation in infectivity also exists), followed by the tank. Recipients from trial 2 had lower risk of symptoms than from trial 1, which is in agreement with the patterns observed in survival curves. Initial weight showed a lower contribution to the risk of onset of first symptoms, although a trend was observed towards a higher risk when individuals had lower weight.

Estimates of heritability were 0.20 for resistance and 0.05 for tolerance. These estimates are in agreement with the results obtained from phenotypic analyses. Thus, our results suggest the existence of genetic variation for both traits and that this variation is greater for resistance. Although preliminary, our results are promising for the application of genetic selection of host resistance traits in the turbot breeding industry.

ACKNOWLEDGEMENTS

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