

SalmoBreed



SF ICELAND

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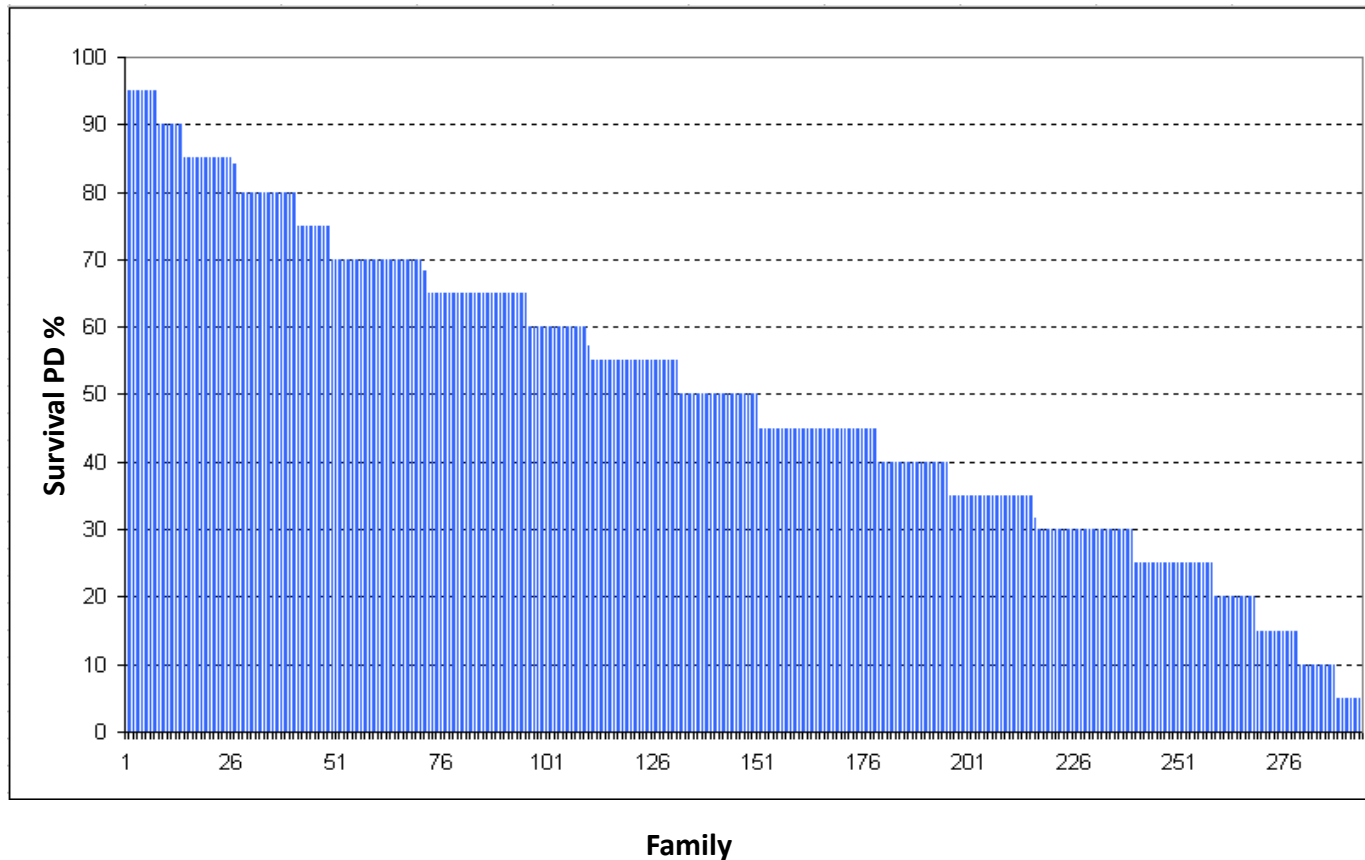
PD TRI NATION Dublin 2015

Genetics and Genomics Manager
Borghild Hillestad
Ph.D.

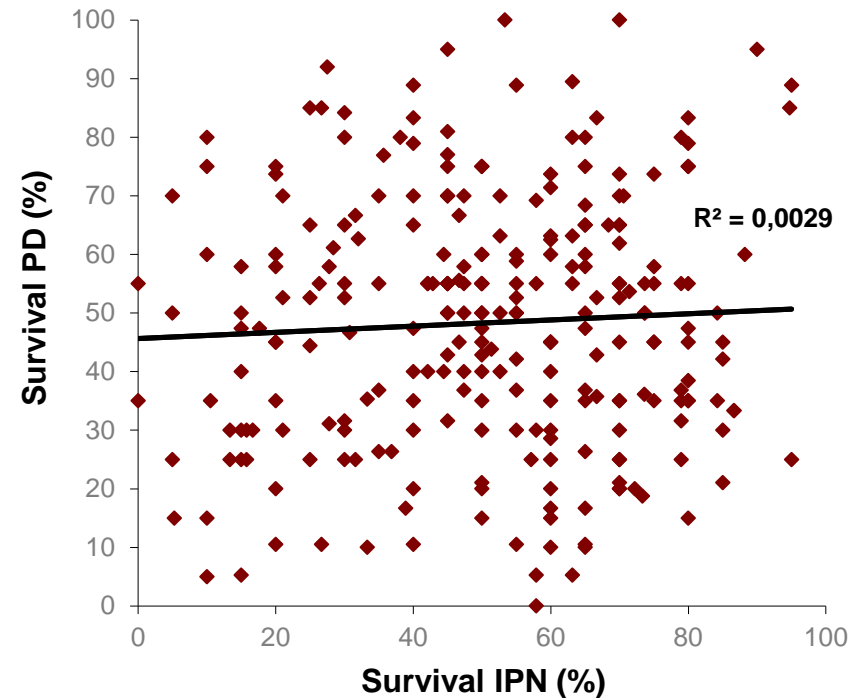
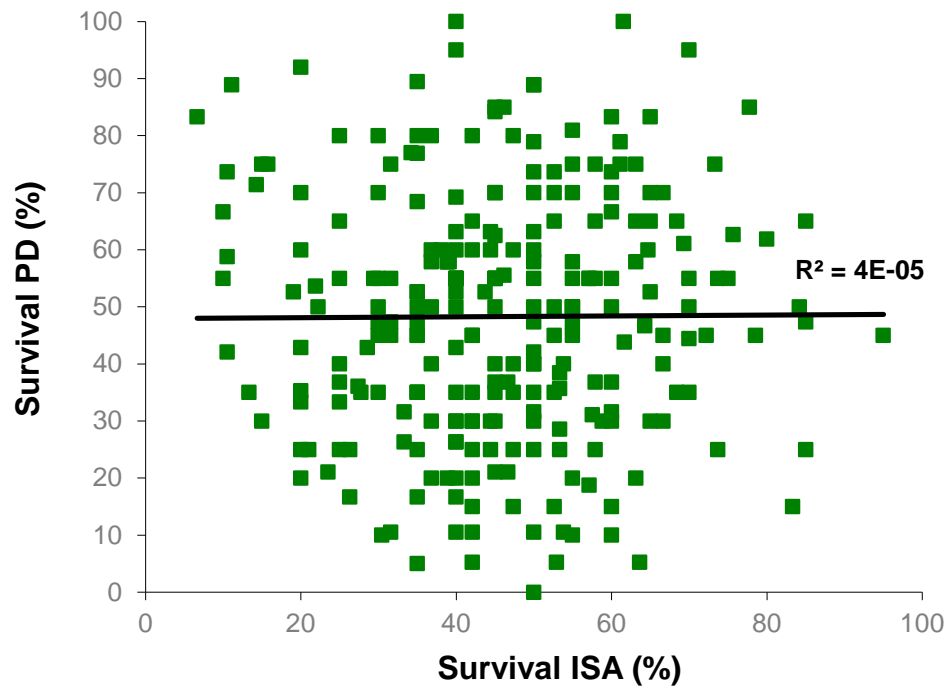
PD breeding in SalmoBreed

- PD is one of many challenges SalmoBreed is facing with selected breeding and use of genetic markers
- Generation of 2008
 - ✓ First year class tested
- First Genetic Marker
 - ✓ Identified AND implemented 2009
- Used different challenge models
 - ✓ VESO fry test and injection test of Pharmaq
 - ✓ Latest years ip challenge

Survival in challenge test for PD (50%) L10



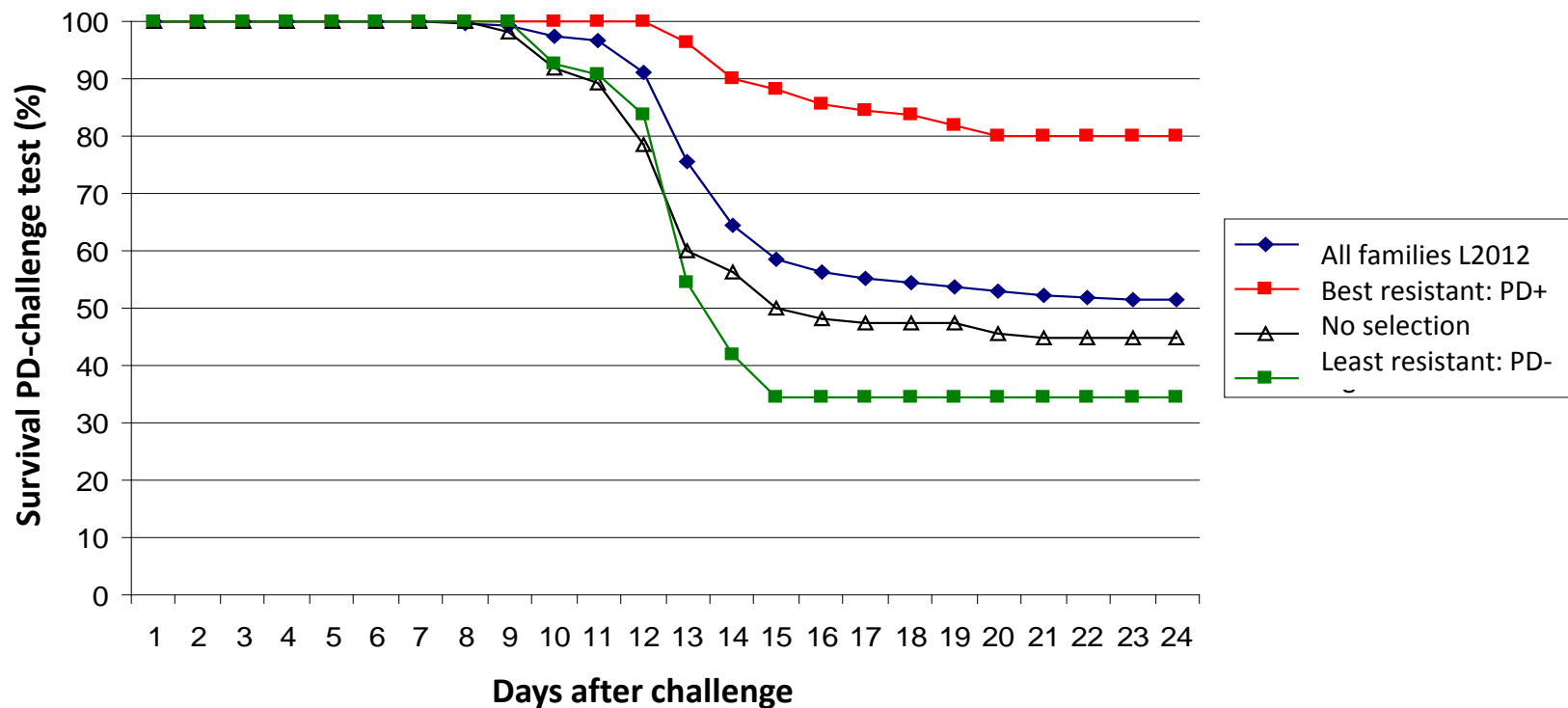
PD's correlation to ISA and IPN

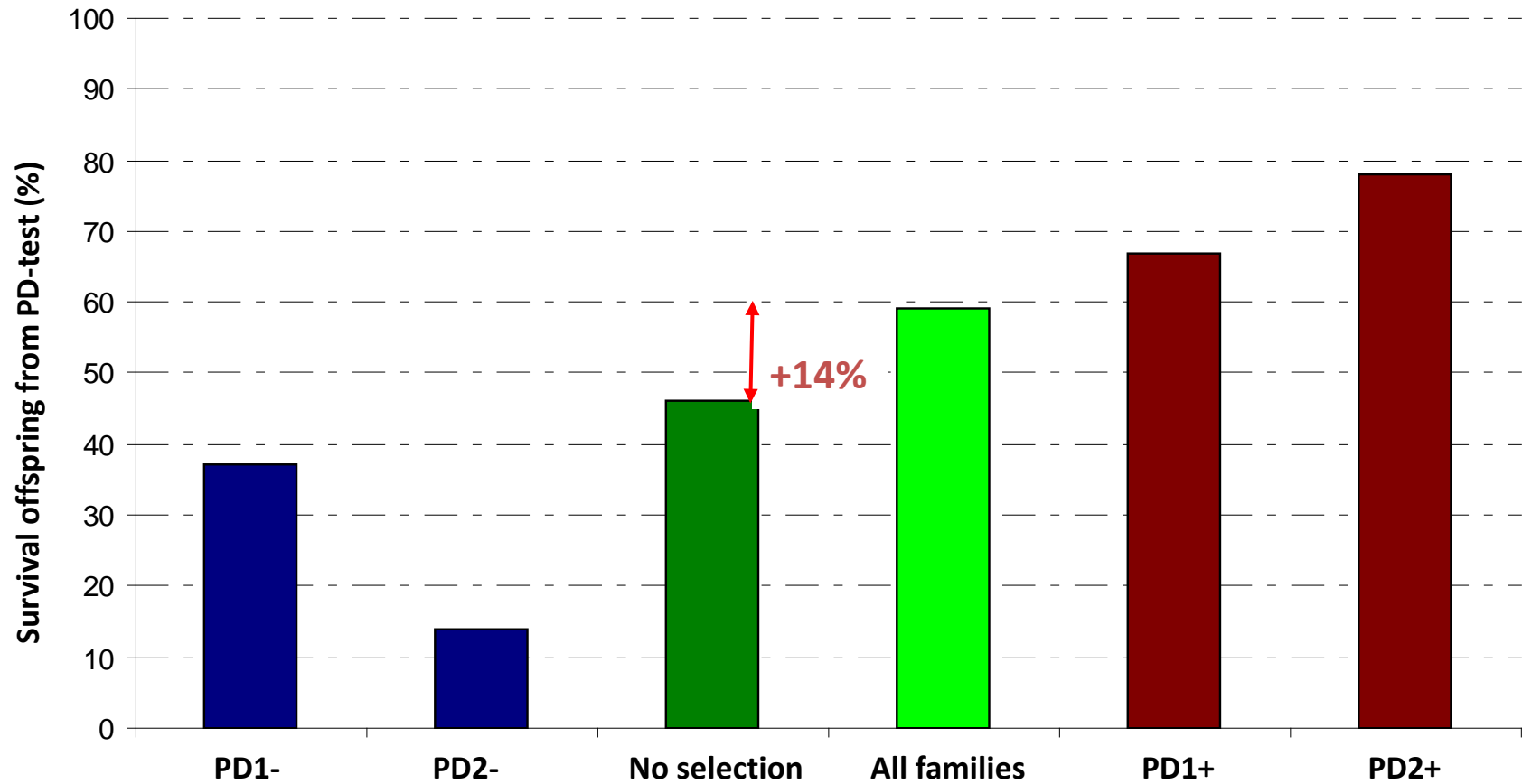


No correlation between PD and ISA
Very small correlation IPN and PD

L2012:

Survival in challenge test for PD+ and PD- families





ORIGINAL ARTICLE

Mapping and validation of a major QTL affecting resistance to pancreas disease (salmonid alphavirus) in Atlantic salmon (*Salmo salar*)

S Gonen^{1,7}, M Baranski^{2,7}, I Thorland³, A Norris⁴, H Grove⁵, P Arnesen⁴, H Bakke⁶, S Lien⁵, SC Bishop¹ and RD Houston¹

Pancreas disease (PD), caused by a salmonid alphavirus (SAV), has a large negative economic and animal welfare impact on Atlantic salmon aquaculture. Evidence for genetic variation in host resistance to this disease has been reported, suggesting that selective breeding may potentially form an important component of disease control. The aim of this study was to explore the genetic architecture of resistance to PD, using survival data collected from two unrelated populations of Atlantic salmon; one challenged with SAV as fry in freshwater (POP 1) and one challenged with SAV as post-smolts in sea water (POP 2). Analyses of the binary survival data revealed a moderate-to-high heritability for host resistance to PD in both populations (fry POP 1 $h^2 \sim 0.5$; post-smolt POP 2 $h^2 \sim 0.4$). Subsets of both populations were genotyped for single nucleotide polymorphism markers, and six putative resistance quantitative trait loci (QTL) were identified. One of these QTL was mapped to the same location on chromosome 3 in both populations, reaching chromosome-wide significance in both the sire- and dam-based analyses in POP 1, and genome-wide significance in a combined analysis in POP 2. This independently verified QTL explains a significant proportion of host genetic variation in resistance to PD in both populations, suggesting a common underlying mechanism for genetic resistance across lifecycle stages. Markers associated with this QTL are being incorporated into selective breeding programs to improve PD resistance.

Heredity advance online publication, 20 May 2015; doi:10.1038/hdy.2015.37

INTRODUCTION

Infectious diseases present a significant threat to the sustainability of

influenced by many factors, such as feeding rate, season, temperature, stocking density, co-infection with other pathogens and host genetics

Explore genetic architecture of resistance to PD

POP1

- Marine Harvest
- Challenged with SAV3 as fry (VESO Vikan)
- Sire- and dam-based analysis
- $h^2 \sim 0.5$

POP2

- SalmoBreed
- Challenged with SAV3 as post-smolt (PHARMAQ AS)
- Genome-wide analysis
- $h^2 \sim 0.4$

QTL findings

- 6 QTLs were found!
- 1 QTL mapped on the same location at CHR3 in both populations
- Suggesting a common underlying mechanism for genetic resistance across lifecycle stages
- Markers associated with this QTL are being incorporated into SalmoBreed's breeding program to improve PD resistance

SUMMARY

- SalmoBreed was early with both PD challenge test (year class 2008) and identification of QTL PD (in market 2009)
- Our work, together with partners, has recently been acknowledged with a publication in Nature, and is the basis of our breeding strategy against PD

