

# Mapping and validation of a major QTL affecting resistance to Pancreas disease in Atlantic salmon

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- Atlantic salmon
  - 2 million tonnes (\$10 billion) production in 2012
- Pancreas disease (alphavirus)
  - Natural outbreaks reported at post-smolt stage
  - High levels of mortality and morbidity
  - Management practices in place

- Selective breeding for improved disease resistance
  - Use full-sibs of challenge survivors
  - Limitations:
    - Requires yearly challenge tests
    - Does not capture within-family variation
- Individual rather than family-based selection
- Genetic markers

- Genetic markers and selection
  - Characterise genetic architecture for resistance
    - Identify resistance QTL
    - Marker assisted selection (MAS)
  - Advantages:
    - Reduce need for sib-challenge tests
    - Exploits within- and between-family variation
- Genetic markers and selection in aquaculture
  - E.g. MAS for IPN resistance
  - Not yet widely applied for pancreas disease resistance

- Quantify and characterise the underlying genetic architecture for resistance to pancreas disease
  - Identify QTL and associated markers for use in selective breeding programs



# Materials and methods



## Population

### POP 1

### POP 2

## Life stage

Fry  
(51 days post-hatch)

Post-smolt  
(333 days post-hatch )

## Origins

Marine Harvest  
2010 year class

SalmoBreed AS  
2009 year class

## Viral strain

SAV3

SAV3

## Challenge protocol

Bath challenge

Intraperitoneal injection

## Number of challenged individuals

5,558

4,946

## Number of full- (half-) sibling families

218 (83)

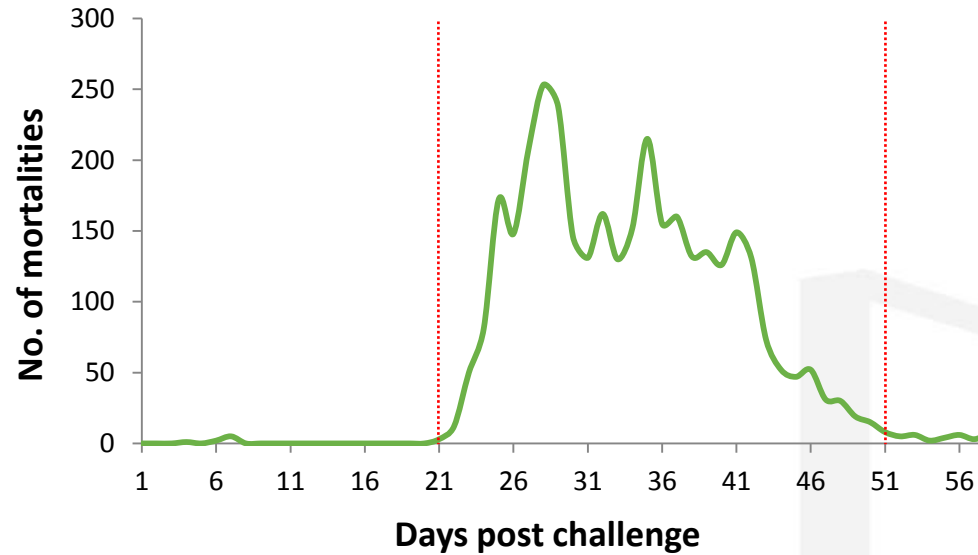
284 (120)

## Average number of offspring per family

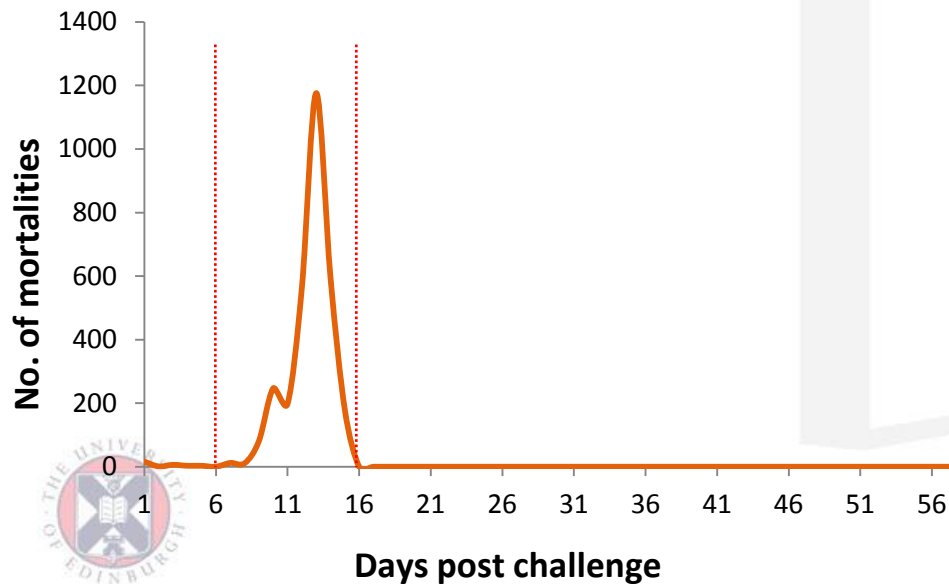
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# Challenge mortality profiles



Population	POP 1	POP 2
Challenge duration	58 days	16 days
Number of mortalities	3,415 (61 %)	3,029 (62 %)
Number of survivors	2,328	1,888



- Within each population:
  1. Estimate heritability for resistance
  2. QTL mapping
  3. Association analysis





# Heritability

Population	POP 1	POP 2
Number of individuals	3,949	4,946
Number of full- (half-) sibling families	150 (72)	284 (120)
Average full-sibling family mortality	61 %	62 %
Heritability (observed binary)	0.34 ( $\pm 0.05$ )	0.23 ( $\pm 0.05$ )
Heritability (underlying liability)	0.5	0.4



- POP 1 (fry)
  - Two-step approach
    - Step 1: Detect QTL
      - Sparse marker panel, sire to offspring
    - Step 2: Confirm and position QTL
      - Dense marker panel, dam to offspring
  - 20 paternal half-sib (55 full-sib) families
    - Intermediate levels of mortality
- POP 2 (post-smolt)
  - Single step combined approach
  - 120 paternal half-sib (284 full-sib) families

- GridQTL
  - Half-sib regression-based interval mapping
  - Sib-pair IBD-based interval mapping
  - QTL significance using F-ratio thresholds
    - Chromosome-wide
      - 10,000 permutations
    - Genome-wide
      - 10,000 permutations
      - Bonferroni corrected P-value at the 5 % significance level

# QTL mapping

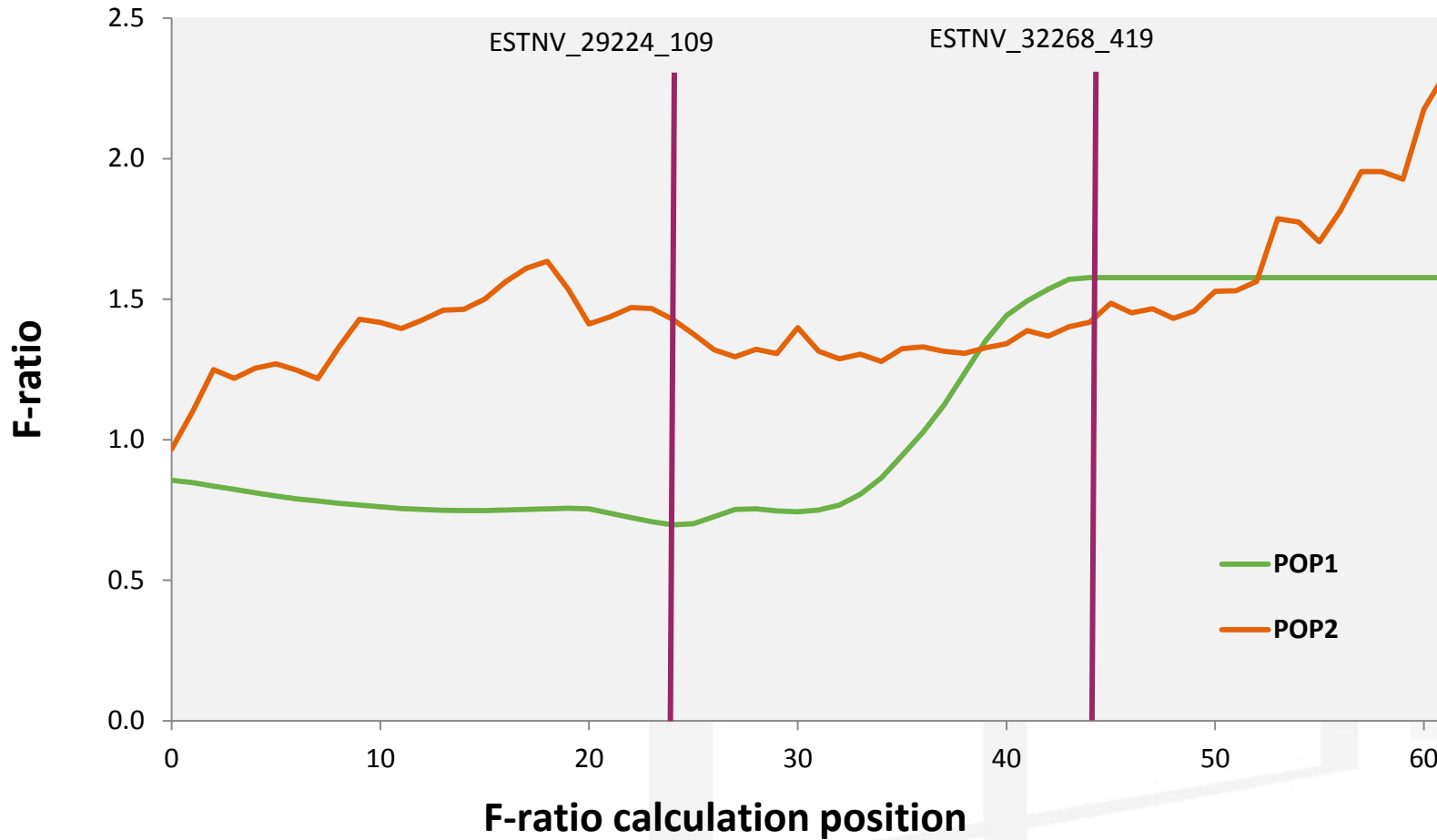
Population	Chromosome	PVE
POP 1	3 <sup>\$</sup>	10
	4	6
	7	5
	23*	6
POP 2	2	10
	3*	23
	14	NA

PVE – Proportion of within-family variation explained by QTL

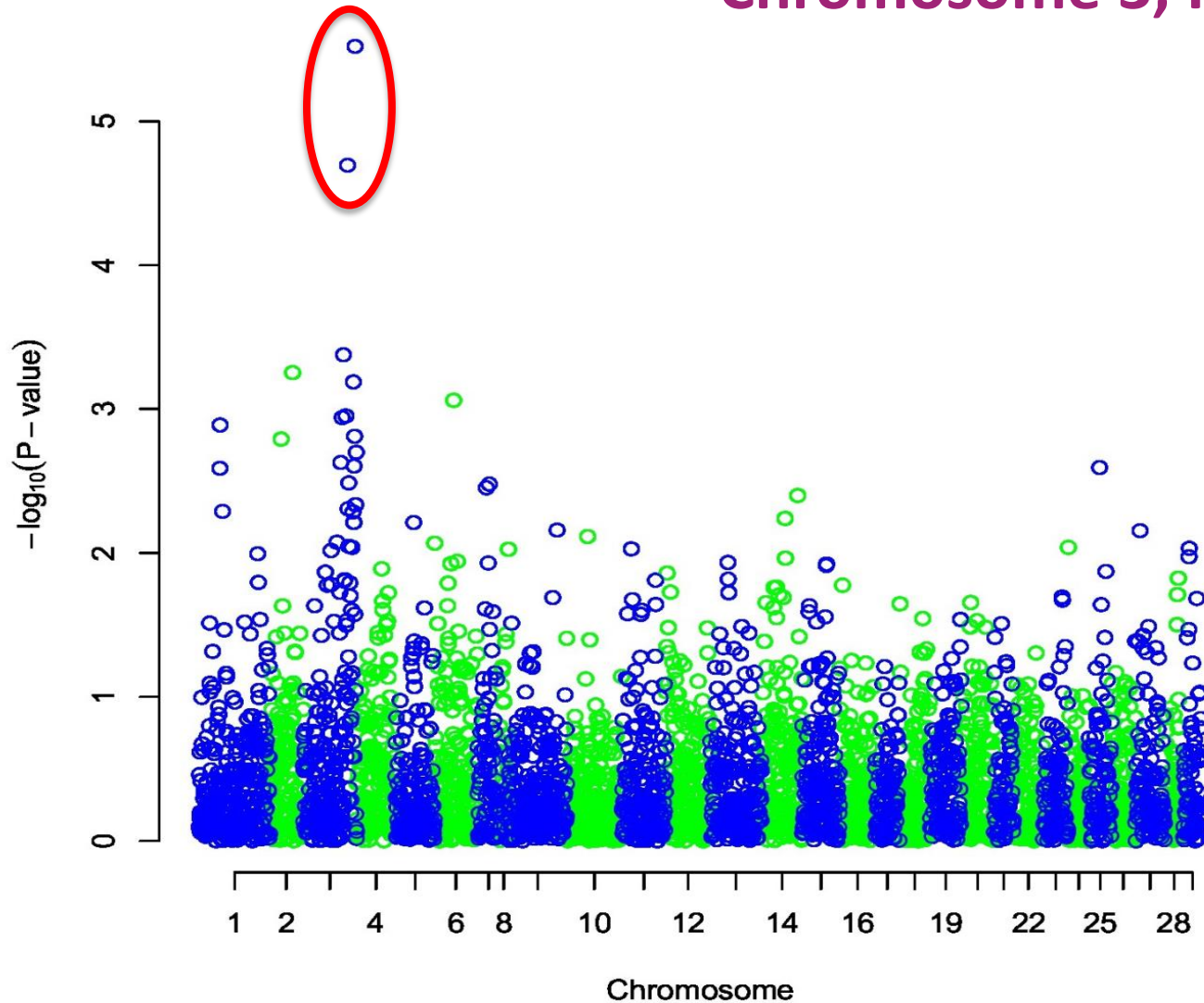
<sup>\$</sup> Significant across sire and dam analyses

\* Genome-wide significant

# Is chromosome 3 the same QTL?



## Chromosome 3, POP 2



- High heritability for resistance to pancreas disease
  - Selection for resistance is possible
- Common QTL on chromosome 3
  - Replicated in two independent populations
  - Similar mechanisms underlying resistance
    - Unrelated to barrier function
- Additional independent QTL
  - Life stage specific QTL

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**Thank you for your attention!**



Read more: Gonen et al. 2015, Heredity doi: [10.1038/hdy.2015.37](https://doi.org/10.1038/hdy.2015.37)

