K. Mathias Wegner, Ana Lokmer, Umberto Rosani, Uwe John, Stefan Neuhaus, Eike Petersen
Deborah Chesslett, Stein Mortensen, Paola Venier, Benjamin Morga, JB Lamy,
Marianne Alunno-Bruscia, Bruno Petton

ALFRED-WEGENER-INSTITUT
HELMHOLTZ-ZENTRUM FÜR POLAR-
UND MEERESFORSCHUNG

Genomic signatures of selection across mass mortality events in European populations of Pacific oysters

VIVALDI

Mass mortalities in Europe associated to OsHV1

Mass mortalities in Europe associated to OsHV1

High Mortalities → Strong selection for resistance

A role for genetics?

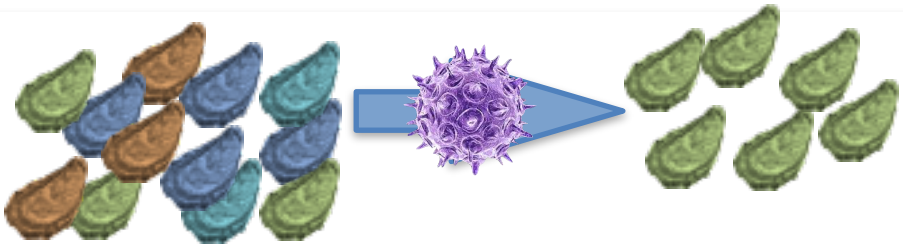
High Mortalities → Strong selection

High heritability
(>0.5, Degremont et al. 2015) → Efficient response to selection

Is selection the same everywhere?

- Does infection select for the same or different genetic variants in different locations?
- Does genetic variation in responsive loci vary between sites?

Which loci respond to selection?



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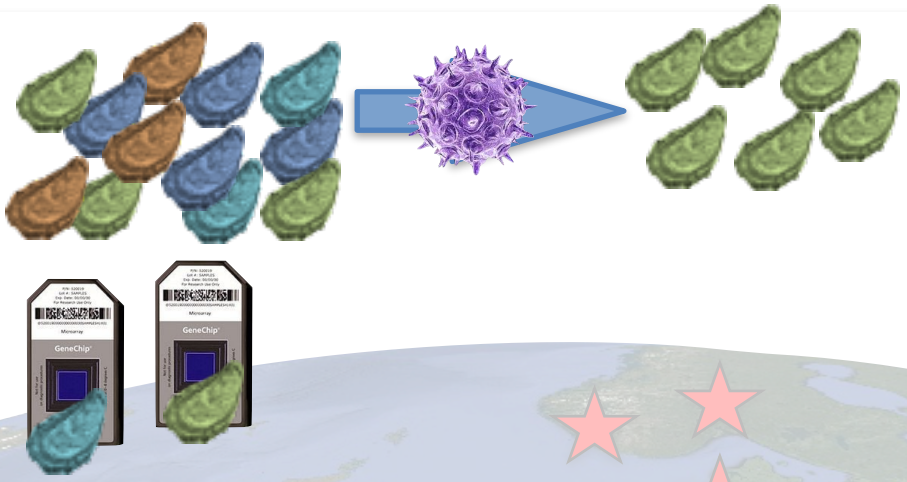

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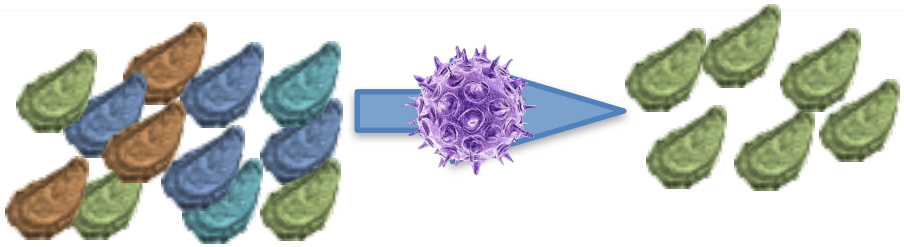

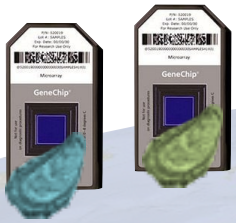

G3 Genes | Genomes | Genetics

INVESTIGATION

A Genome-Wide Association Study for Host Resistance to Ostreid Herpesvirus in Pacific Oysters (*Crassostrea gigas*)

Alejandro P. Gutierrez,* Tim P. Bean,† Chantelle Hooper,† Craig A. Stenton,† Matthew B. Sanders,† Richard K. Paley,† Pasi Rastas,‡ Michaela Bryrom,§ Oswald Matika,* and Ross D. Houston*

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ORCID ID: 0000-0003-1805-0762 (R.D.H.)

ABSTRACT Ostreid herpesvirus (OsHV) can cause mass mortality events in Pacific oyster aquaculture. While various factors impact on the severity of outbreaks, it is clear that genetic resistance of the host is an important determinant of mortality levels. This raises the possibility of selective breeding strategies to improve the genetic resistance of farmed oyster stocks, thereby contributing to disease control. Traditional selective breeding can be augmented by use of genetic markers, either via marker-assisted or genomic selection. The aim of the current study was to investigate the genetic architecture of resistance to OsHV in Pacific oyster, to identify genomic regions containing putative resistance genes, and to inform the use of genomics to enhance efforts to breed for resistance. To achieve this, a population of ~1,000 juvenile oysters were experimentally challenged with a virulent form of OsHV, with samples taken from mortalities and survivors for genotyping and qPCR measurement of viral load. The samples were genotyped using a recently-developed SNP array, and the genotype data were used to reconstruct the pedigree. Using these pedigree and genotype data, the first high density linkage map was constructed for Pacific oyster, containing 20,353 SNPs mapped to the ten pairs of chromosomes. Genetic parameters for resistance to OsHV were estimated, indicating a significant but low heritability for the binary trait of survival and also for viral load measures (h2 0.12 – 0.25). A genome-wide association study highlighted a region of linkage group 6 containing a significant QTL affecting host resistance. These results are an important step toward identification of genes underlying resistance to OsHV in oyster, and a step toward applying genomic data to enhance selective breeding for disease resistance in oyster aquaculture.

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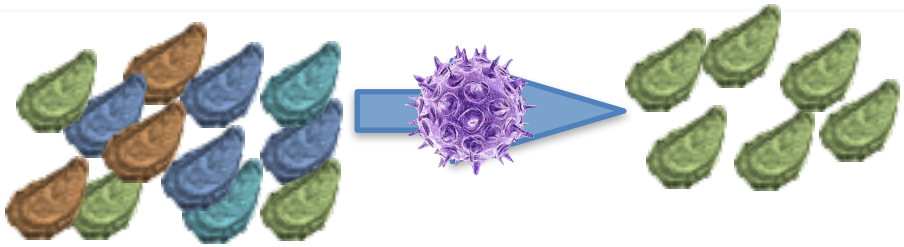

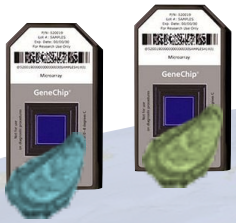

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APPLICATIONS OF NEXT-GENERATION SEQUENCING

Sequencing pools of individuals — mining genome-wide polymorphism data without big funding

Christian Schlötterer[1], Raymond Tobler[1,2], Robert Kofler[1] and Viola Nolte[1]

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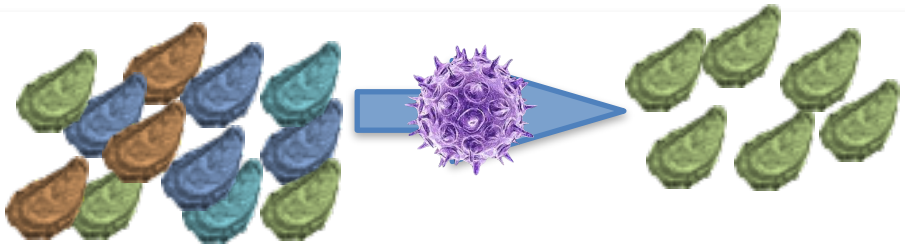

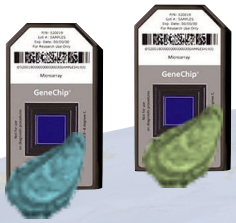

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Sequencing pools of individuals — mining genome-wide polymorphism data without big funding

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- 'Dirty' data estimating population allele frees

- Whole genome coverage (Millions of SNPs)
- Fairly robust estimates
- Can be applied to wild/field populations

Two data sets

1. before vs. after mortality samples from field collected animals
2. before vs. after mortality samples from lab bred animals reciprocally transferred between sites
3. (6 Italian populations with varying histories of disease)

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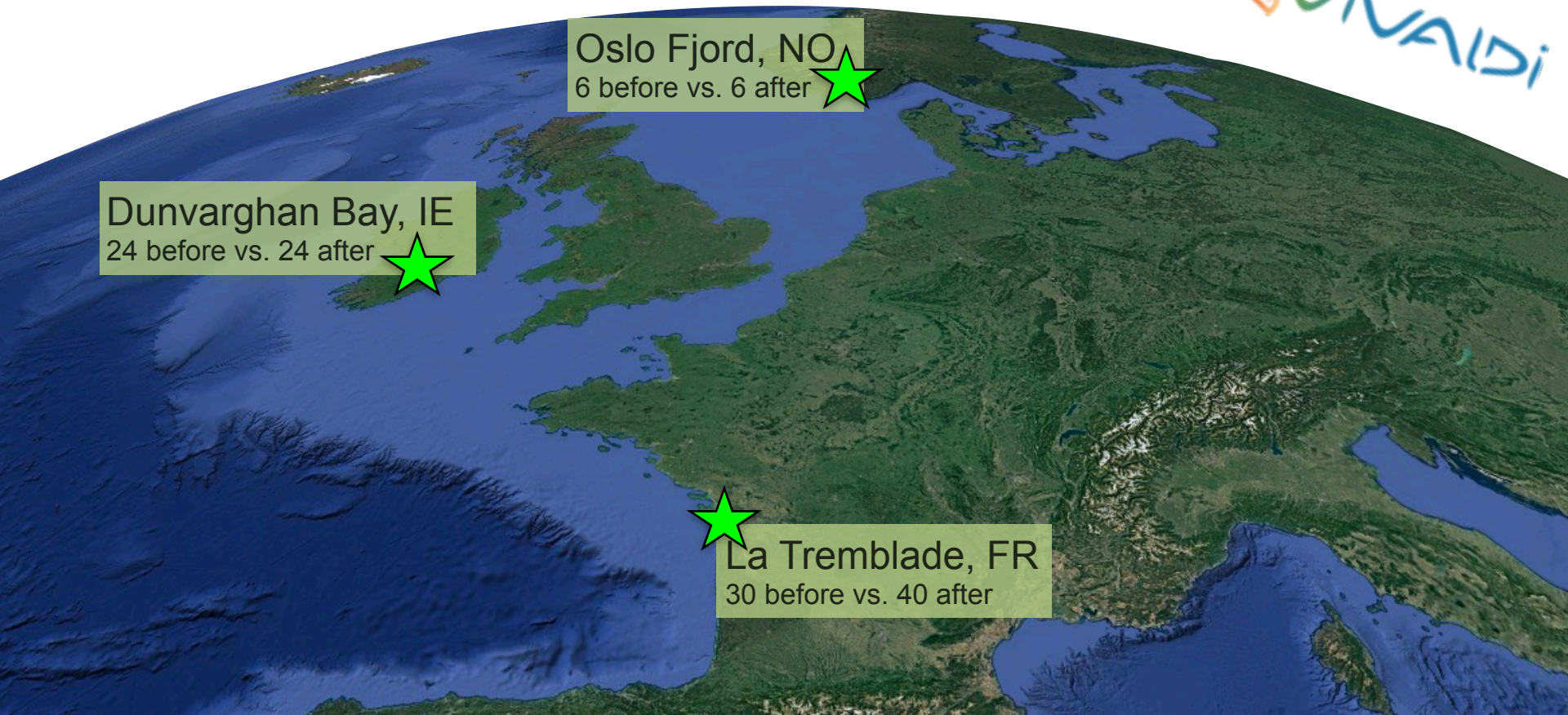

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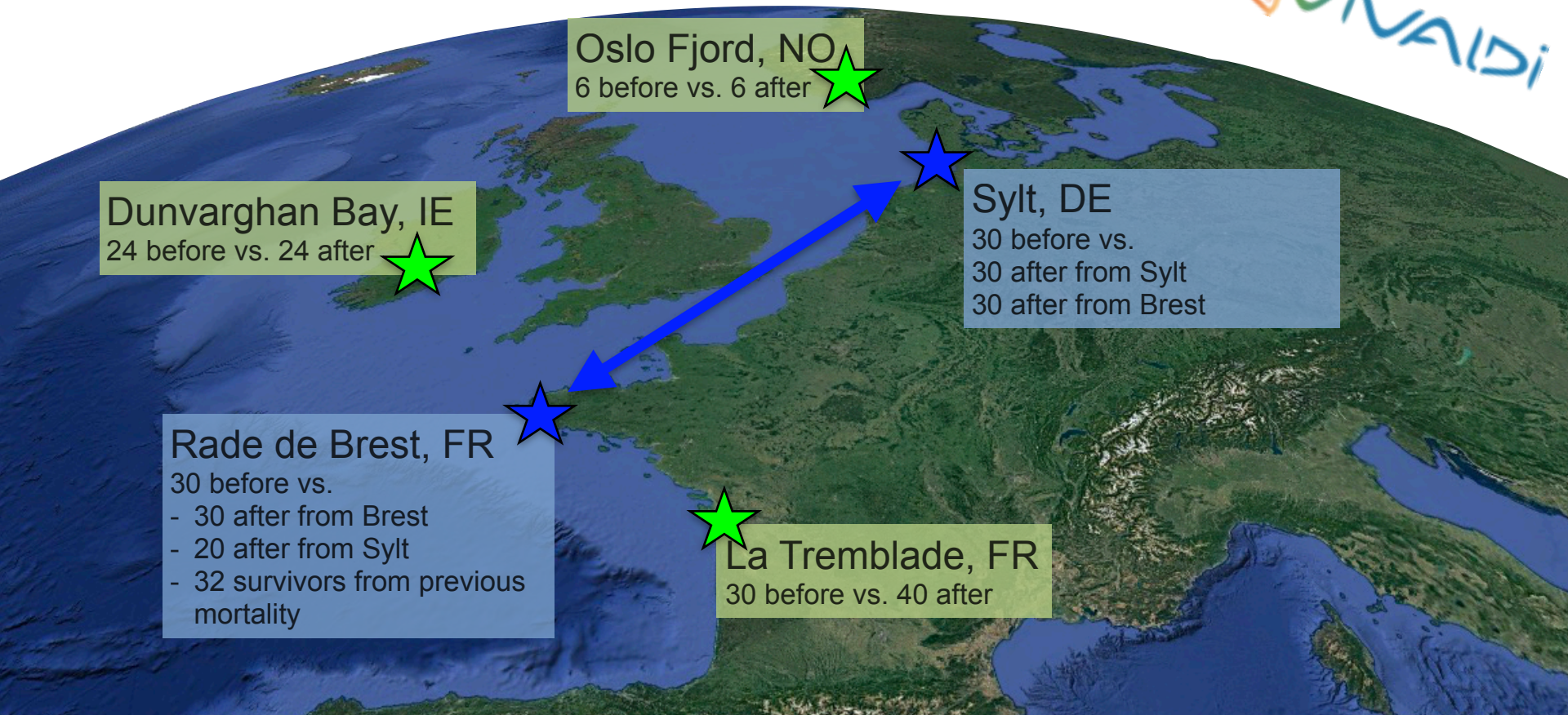

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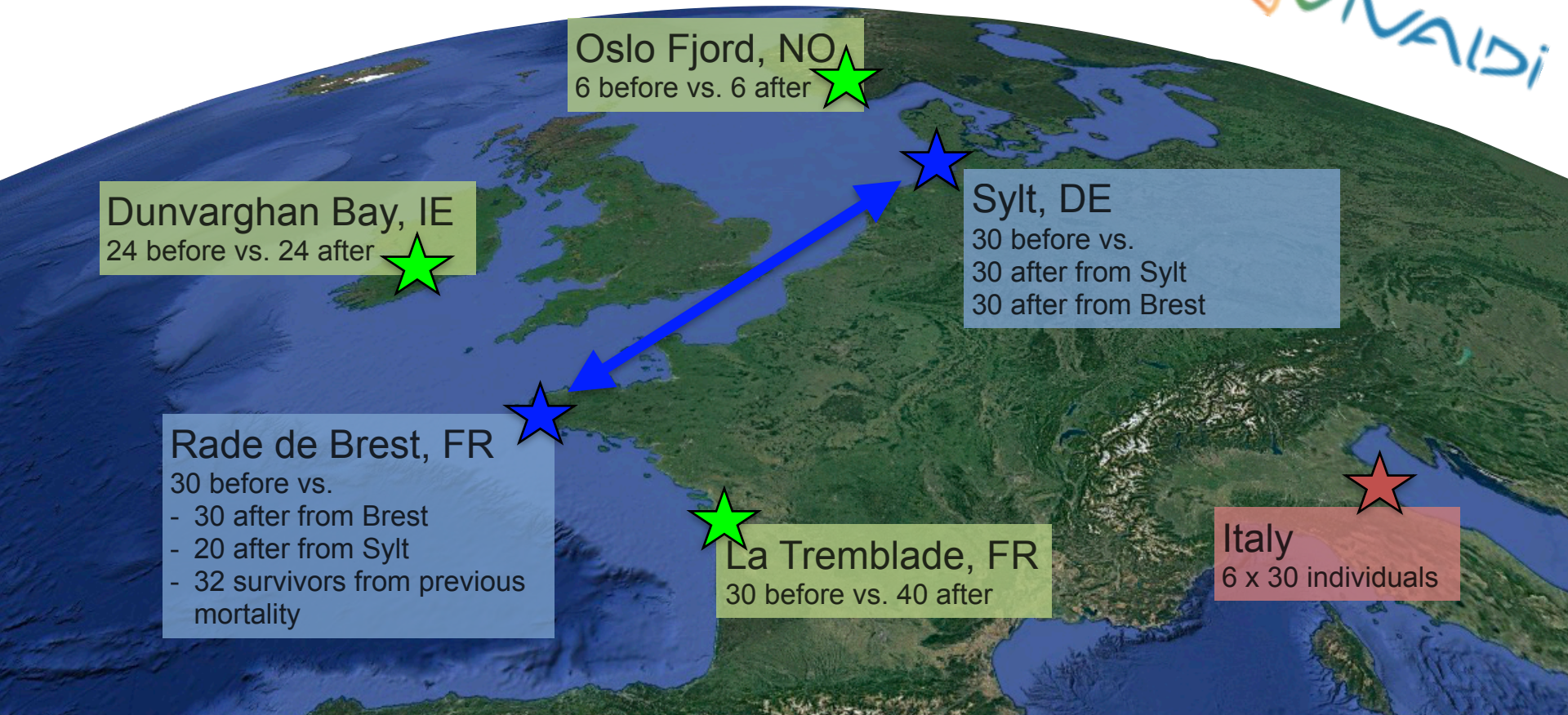

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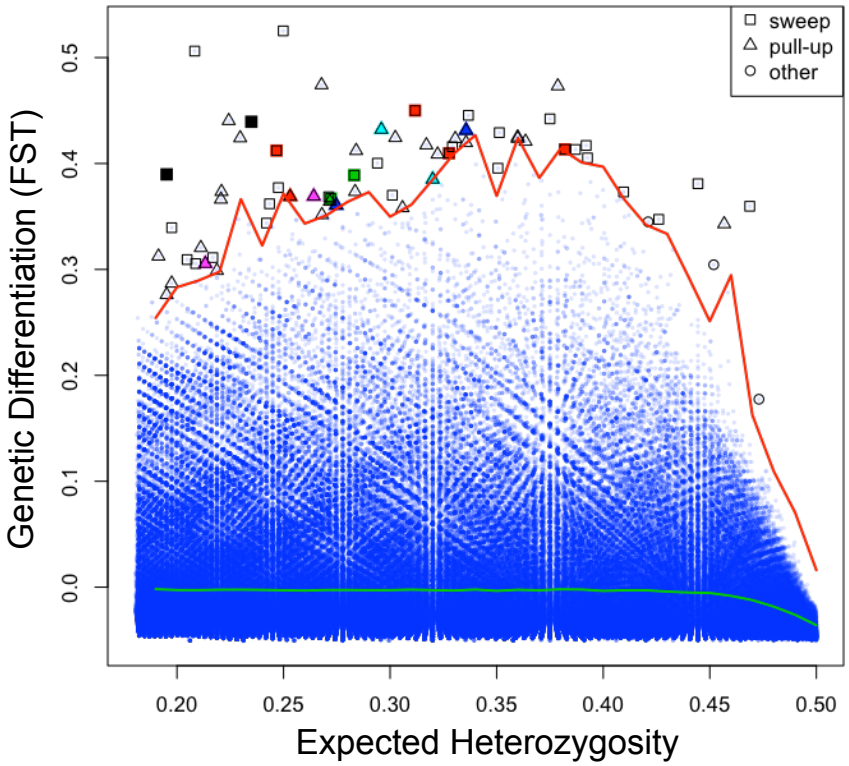

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- 592382 SNPs (minimum coverage: 30, min. minor allele frequency mia = 0.1)
- 68 outliers

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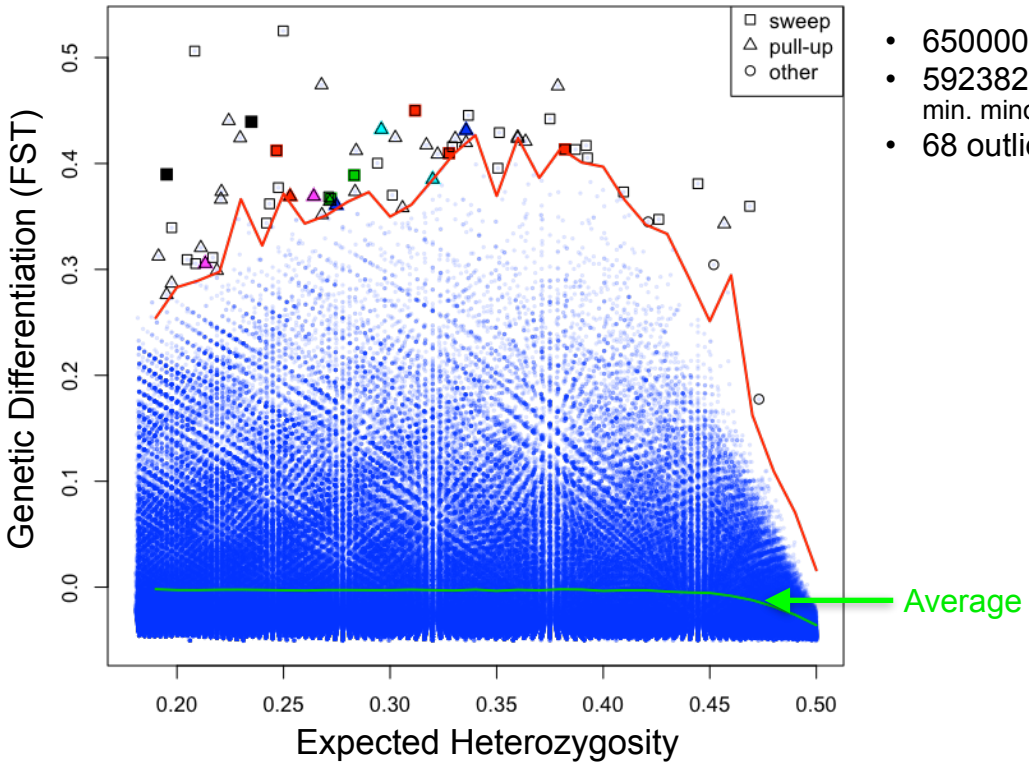

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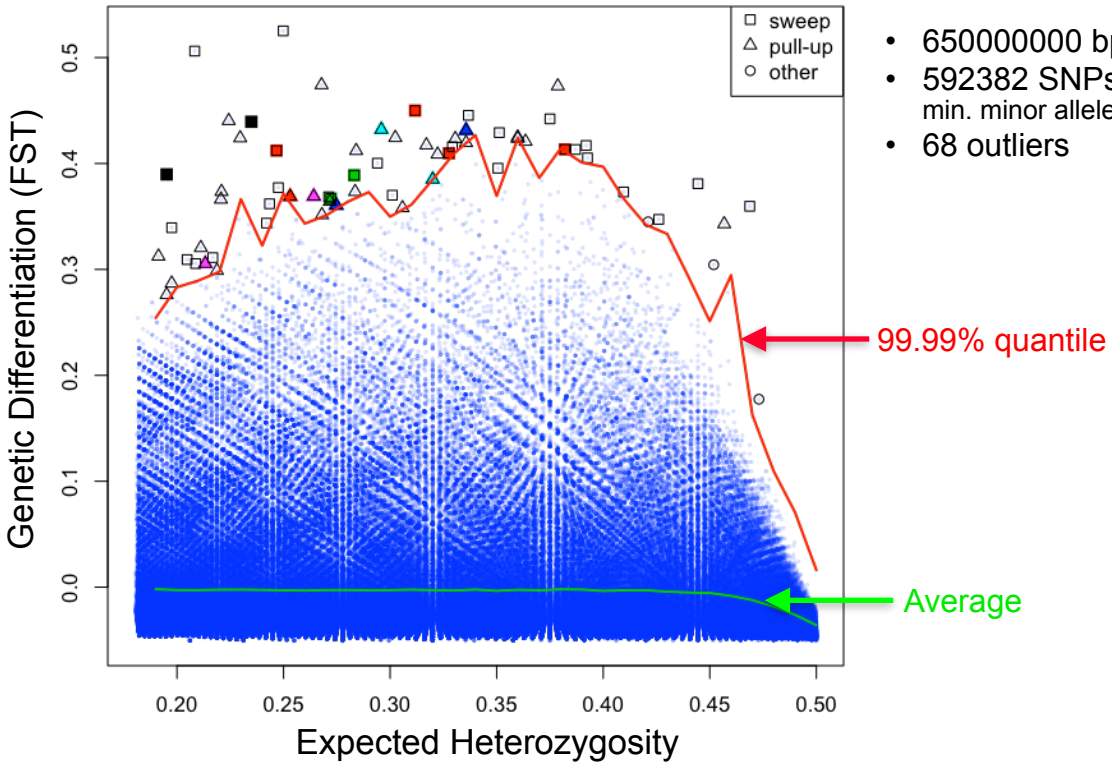

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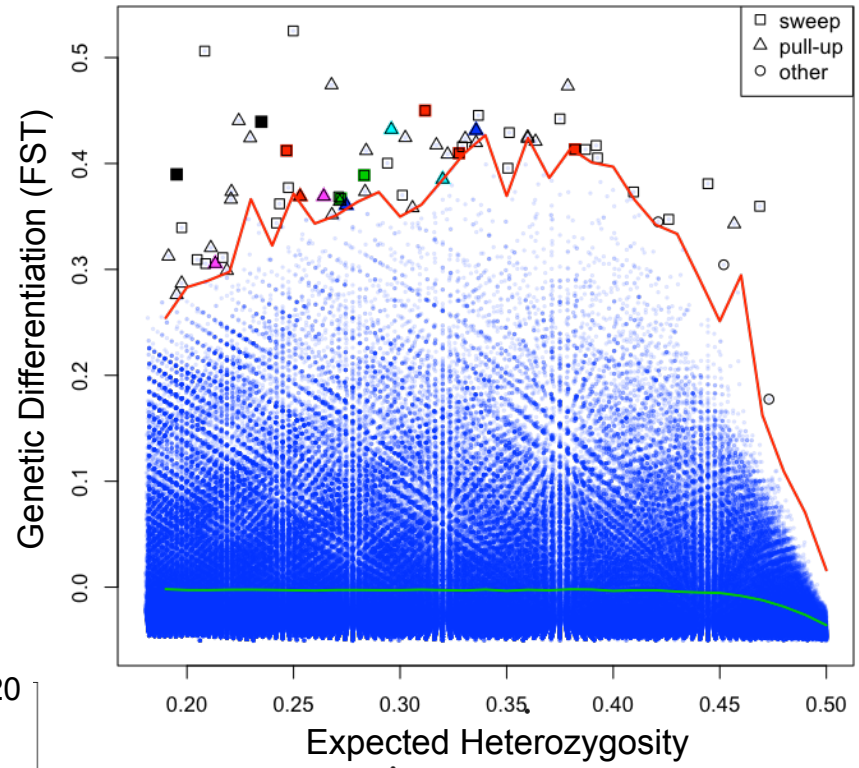

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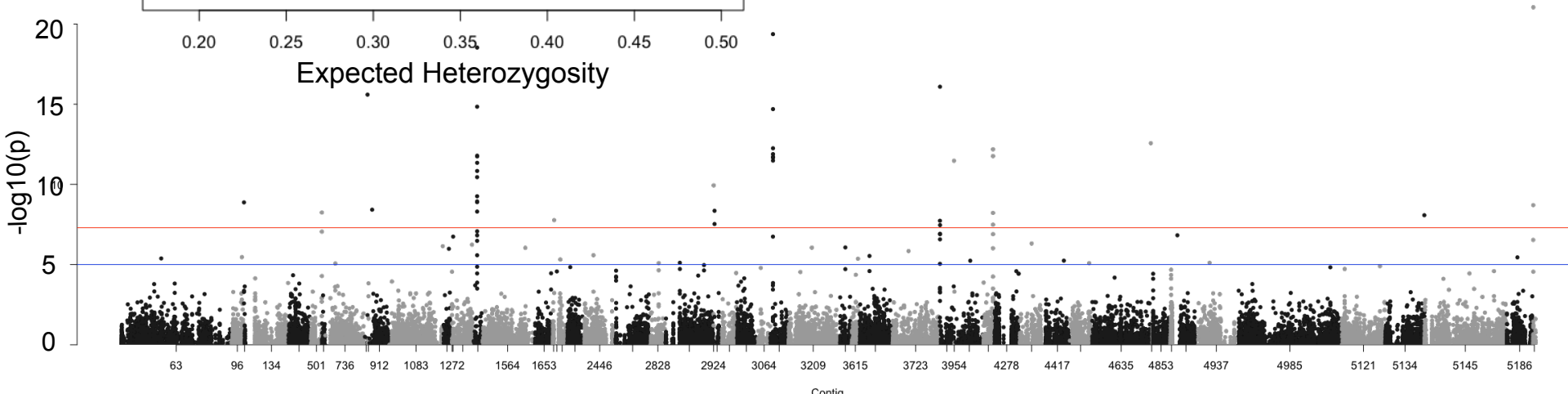

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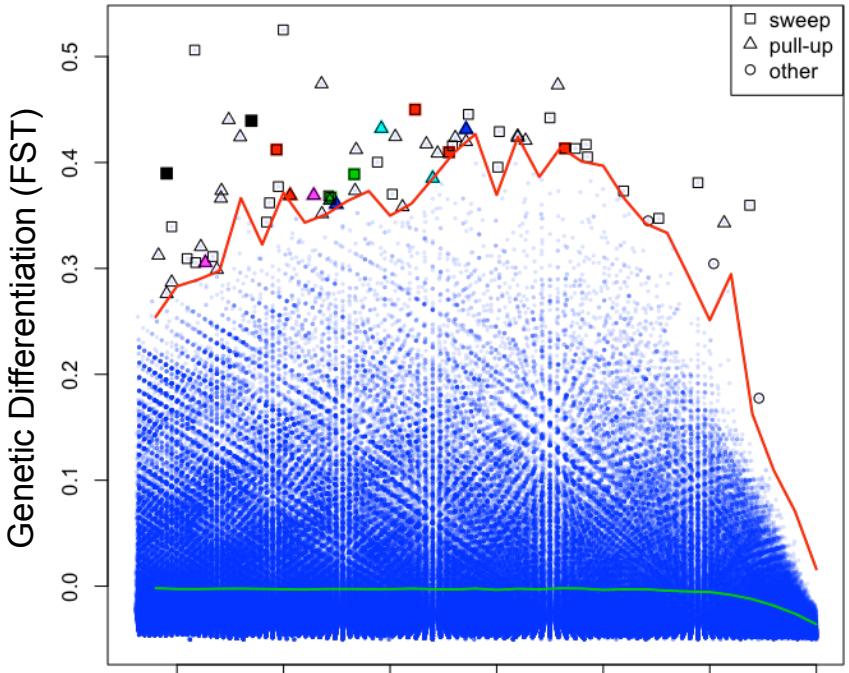

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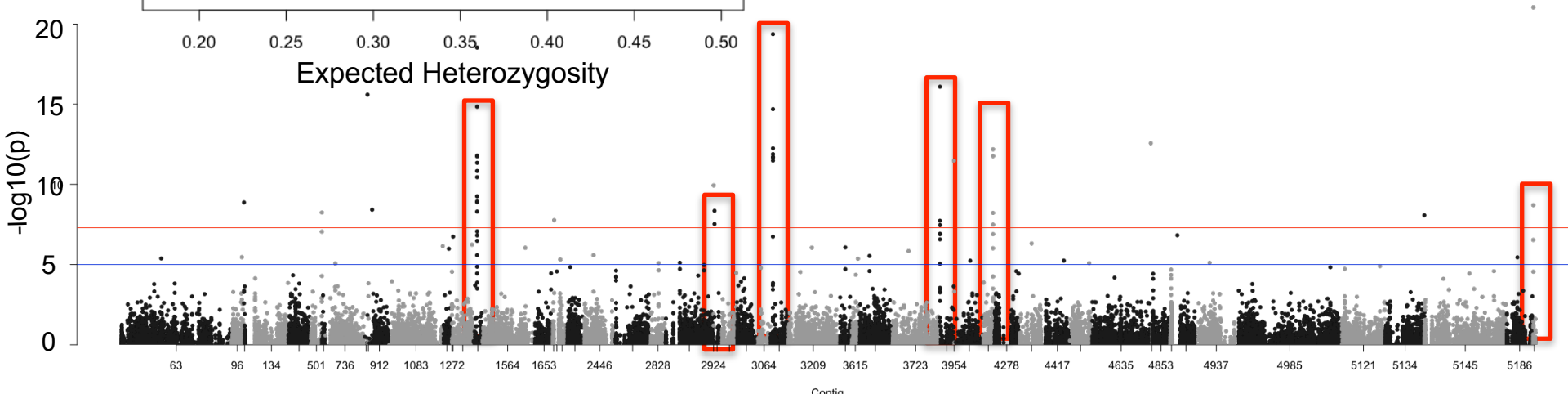

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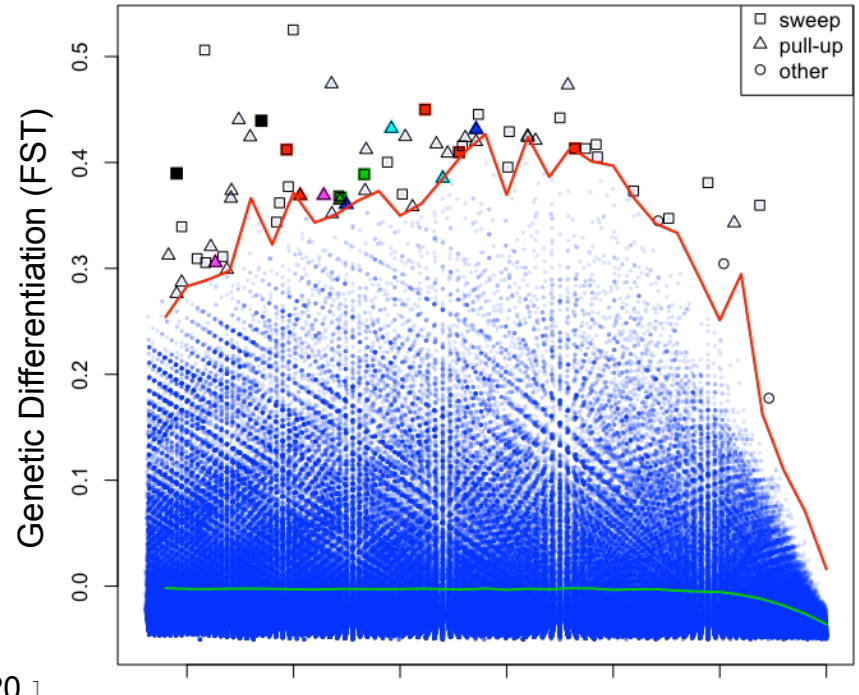

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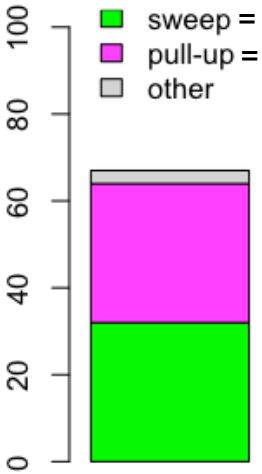
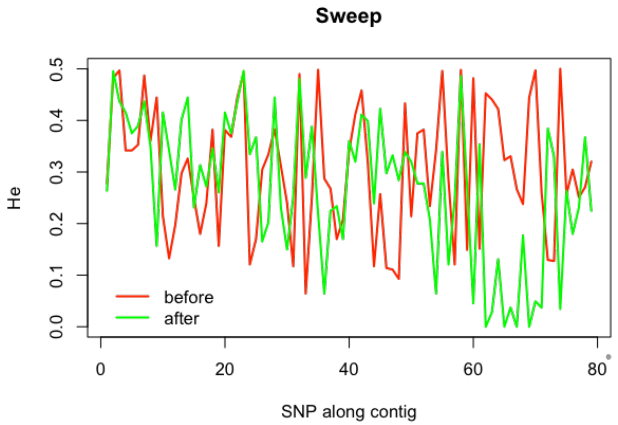
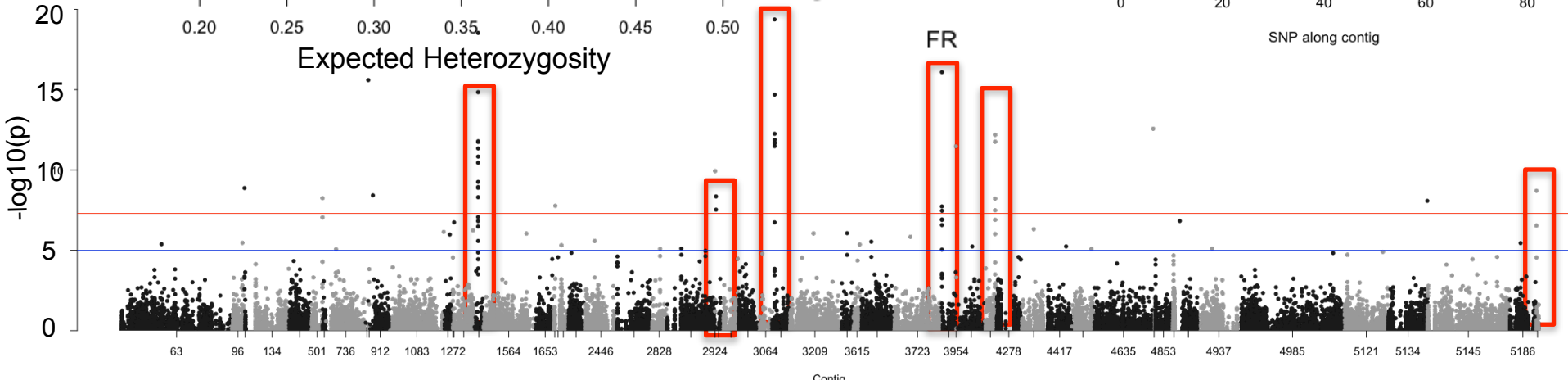
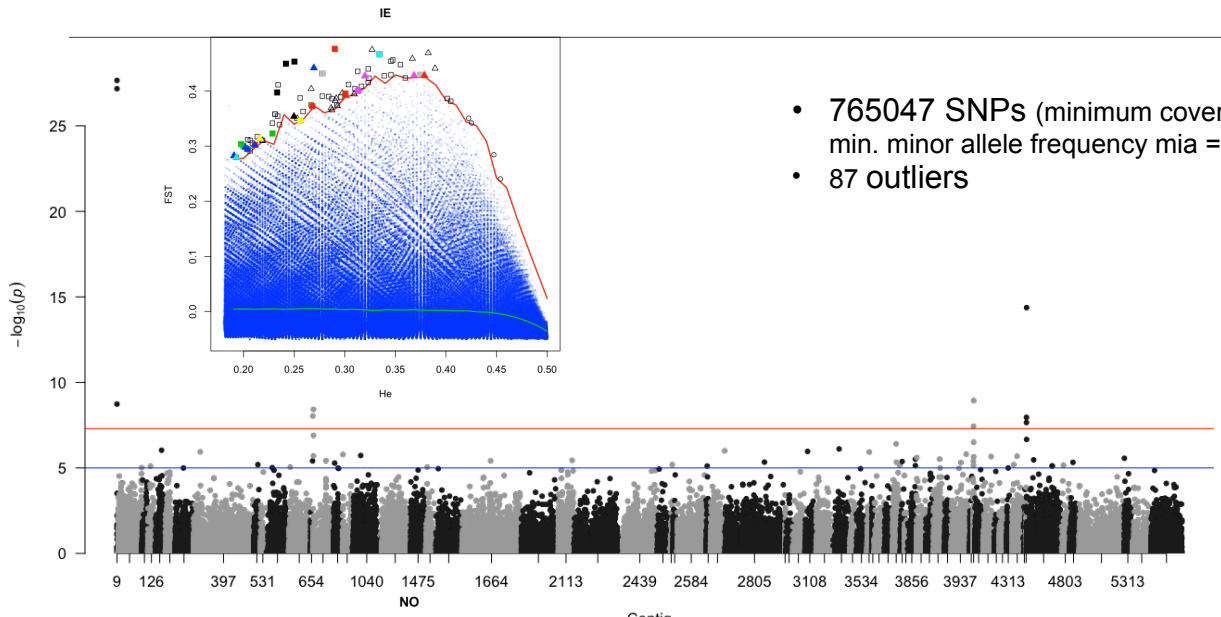






IRELAND & NORWAY



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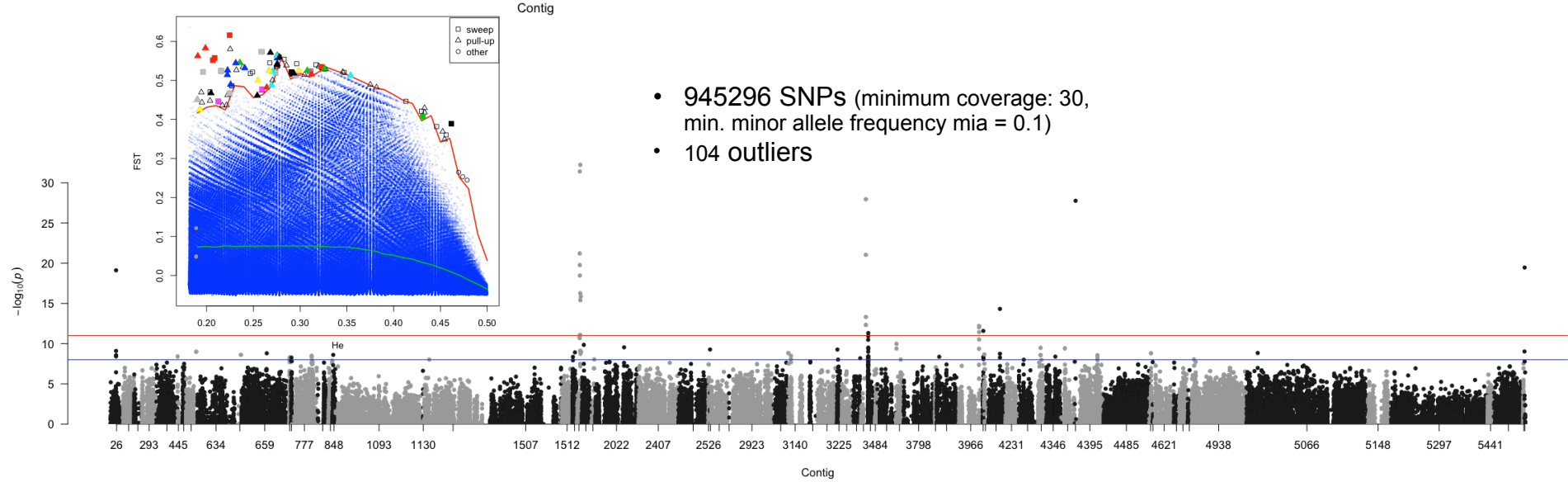

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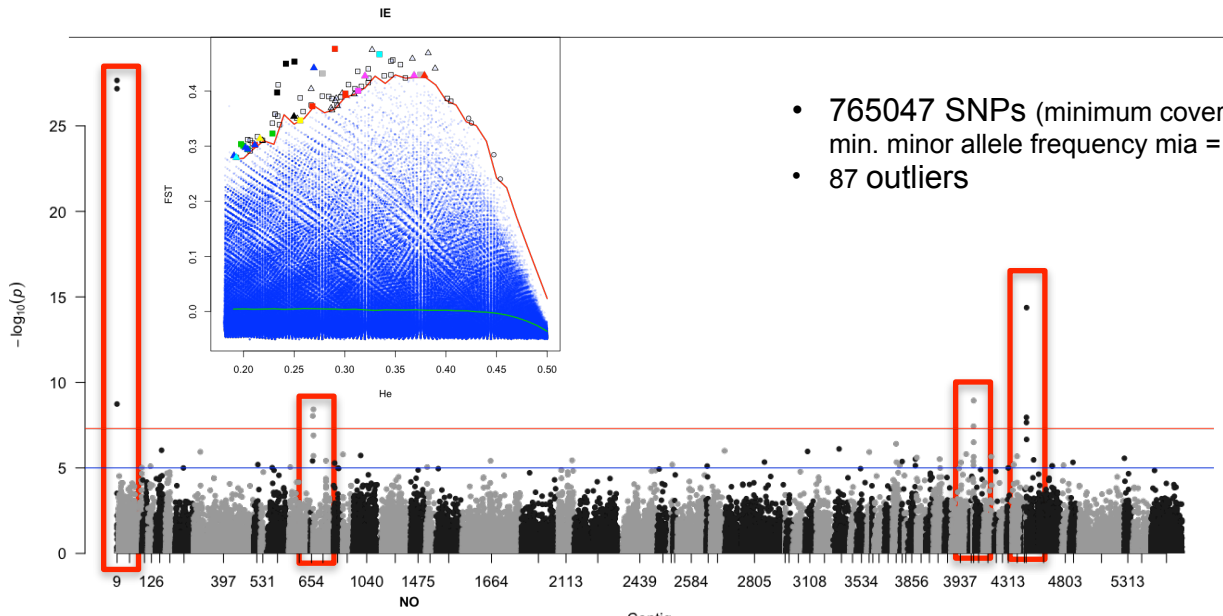

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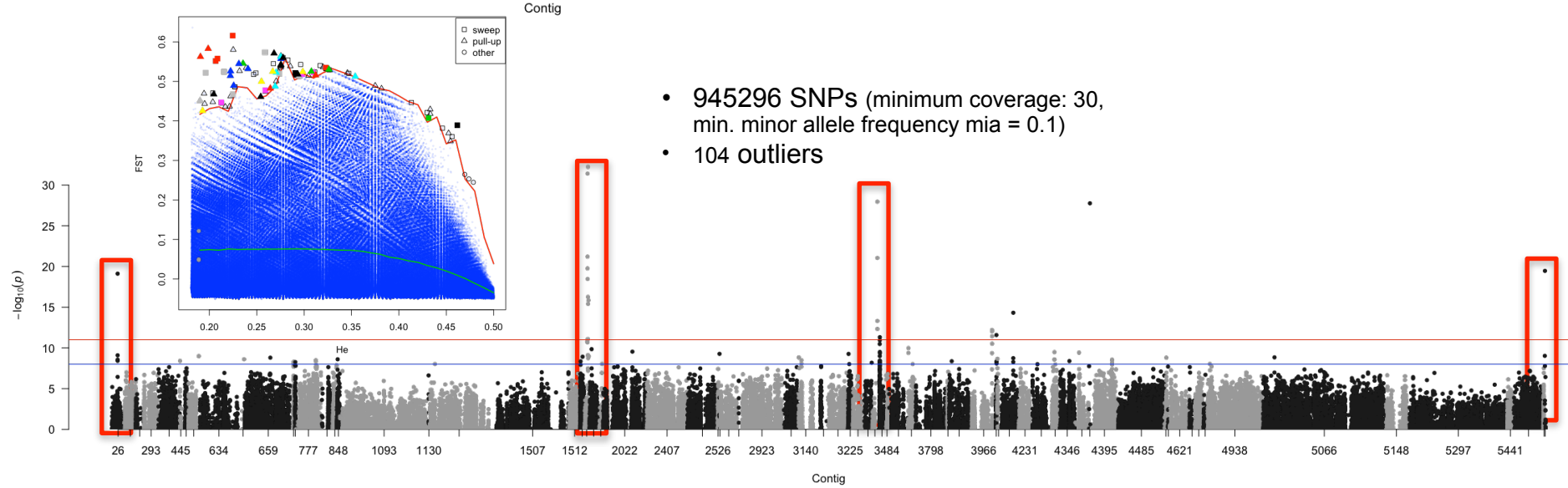

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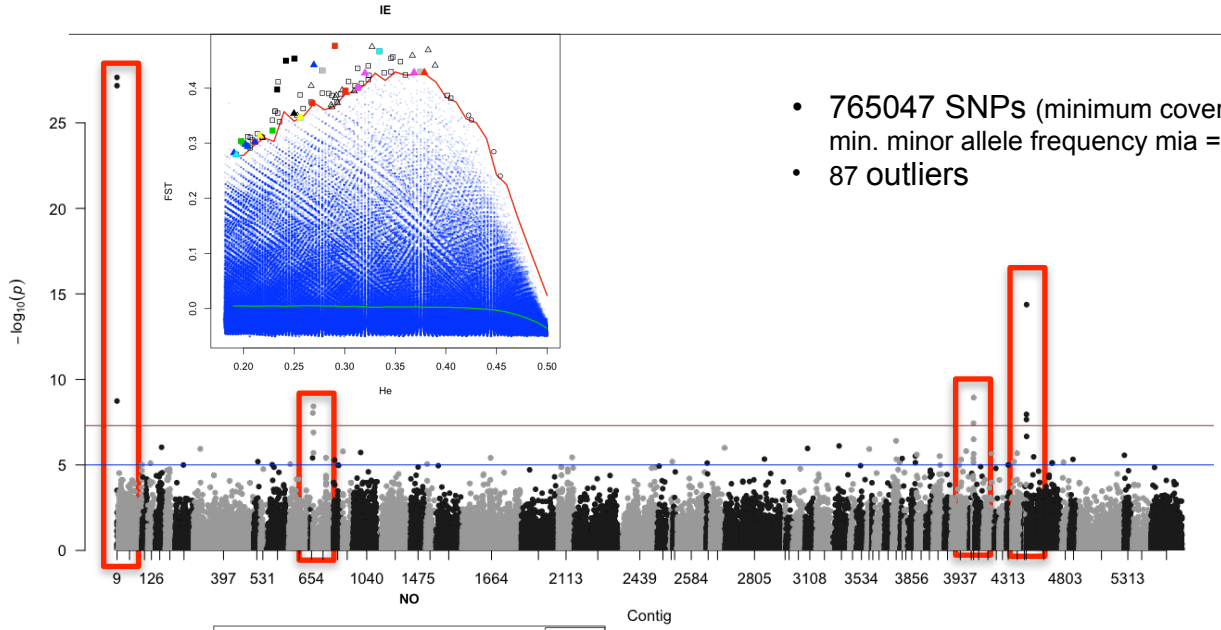

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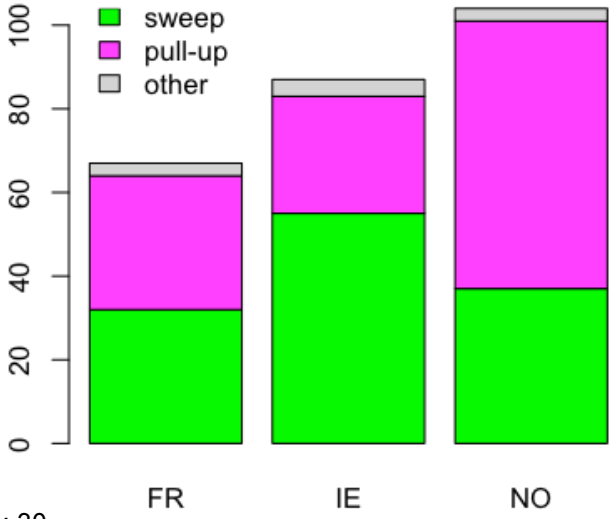

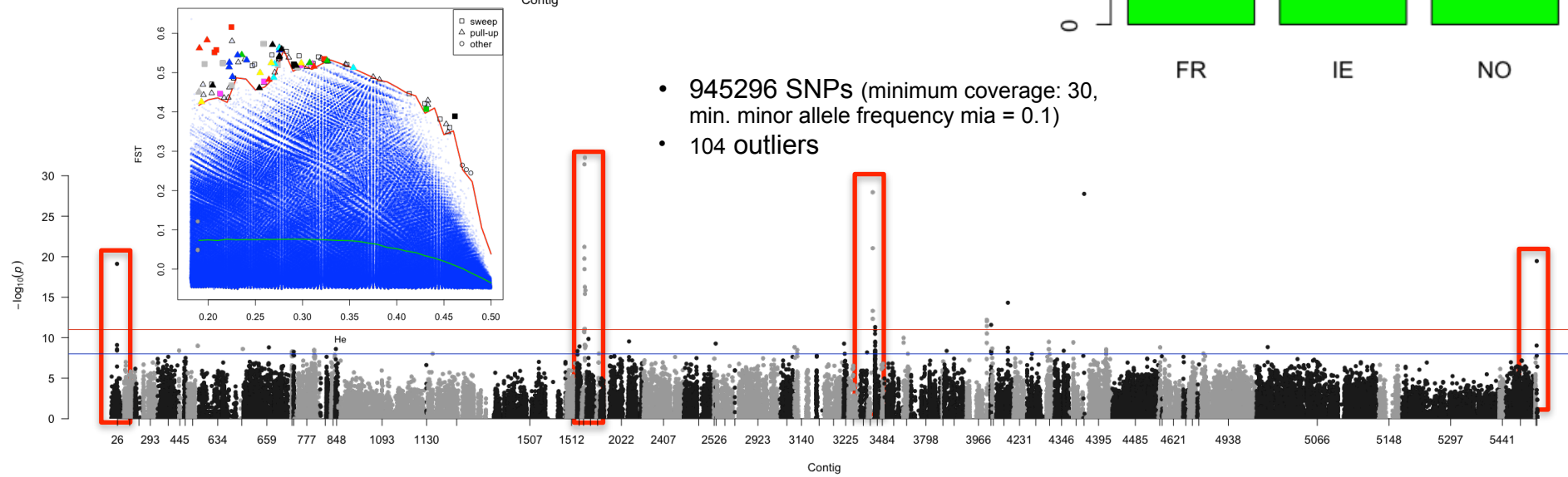

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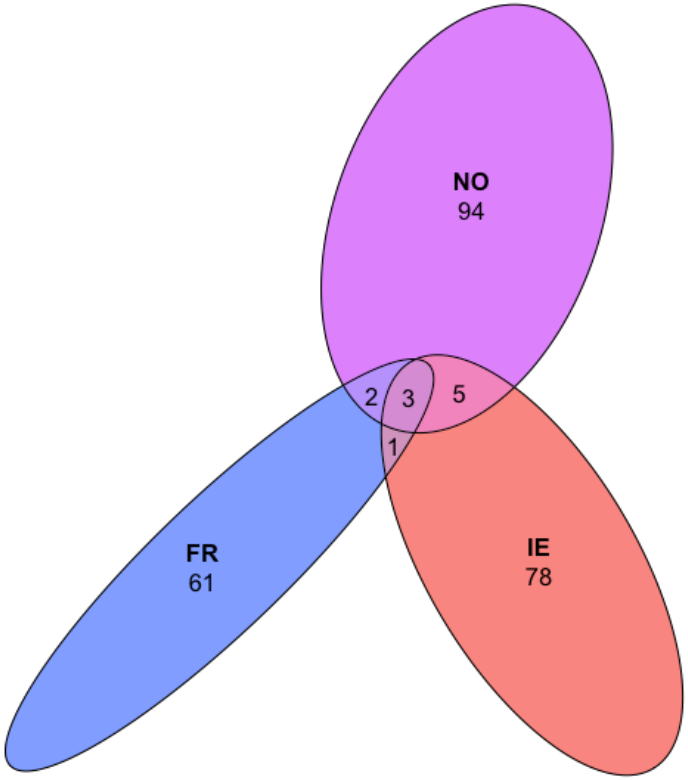

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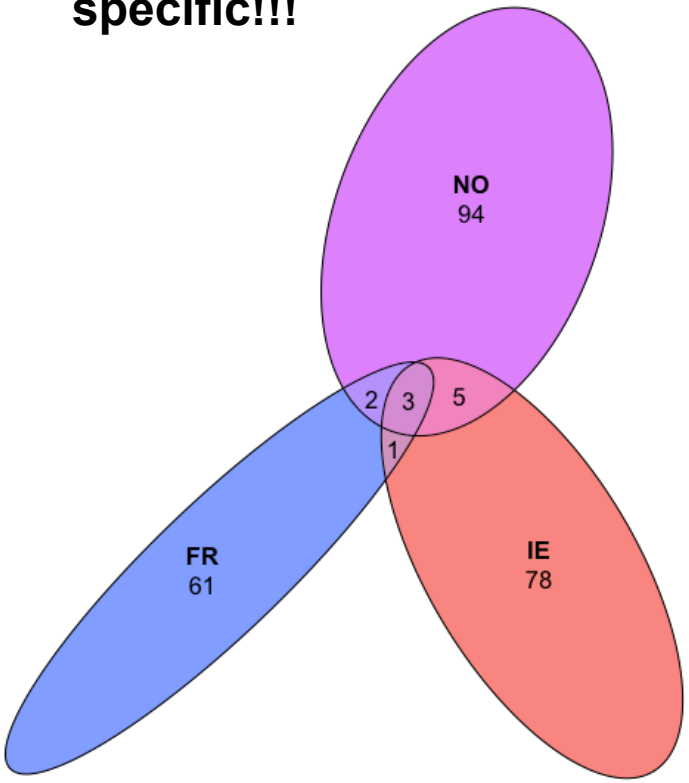

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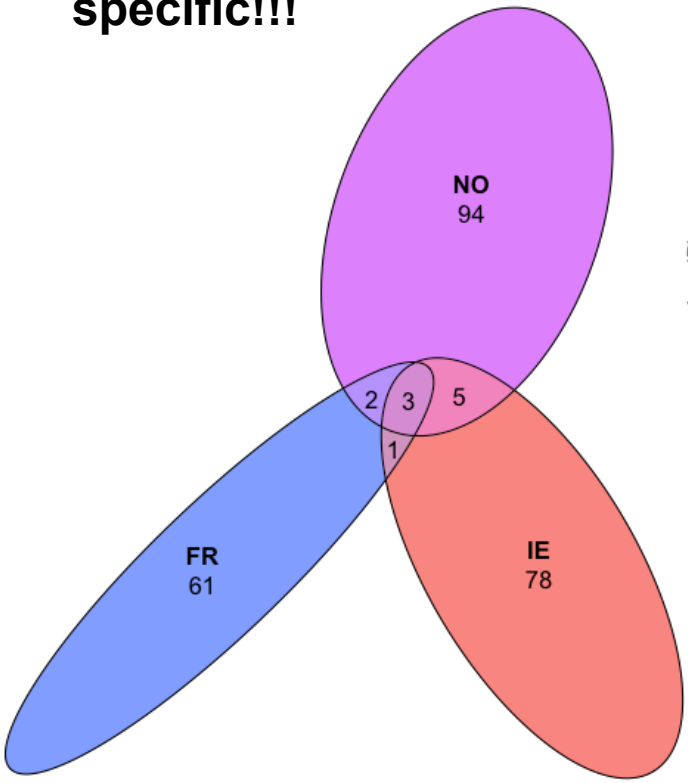
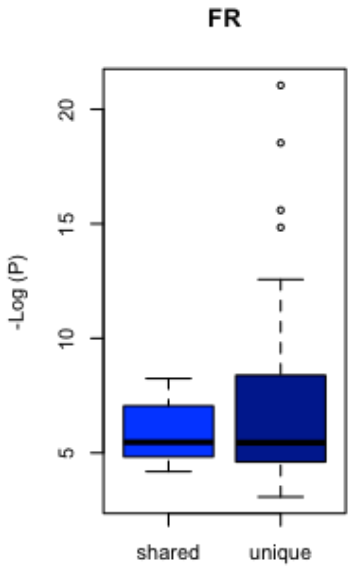
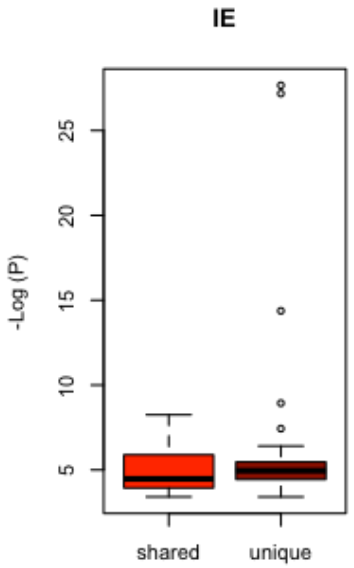
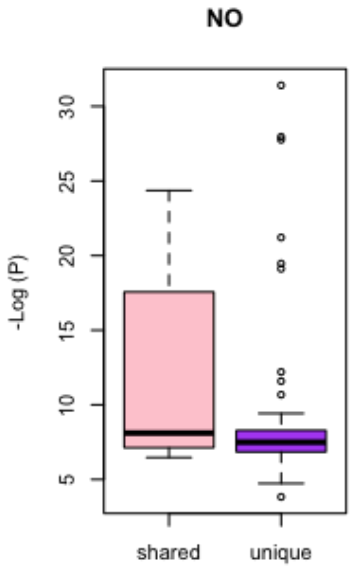
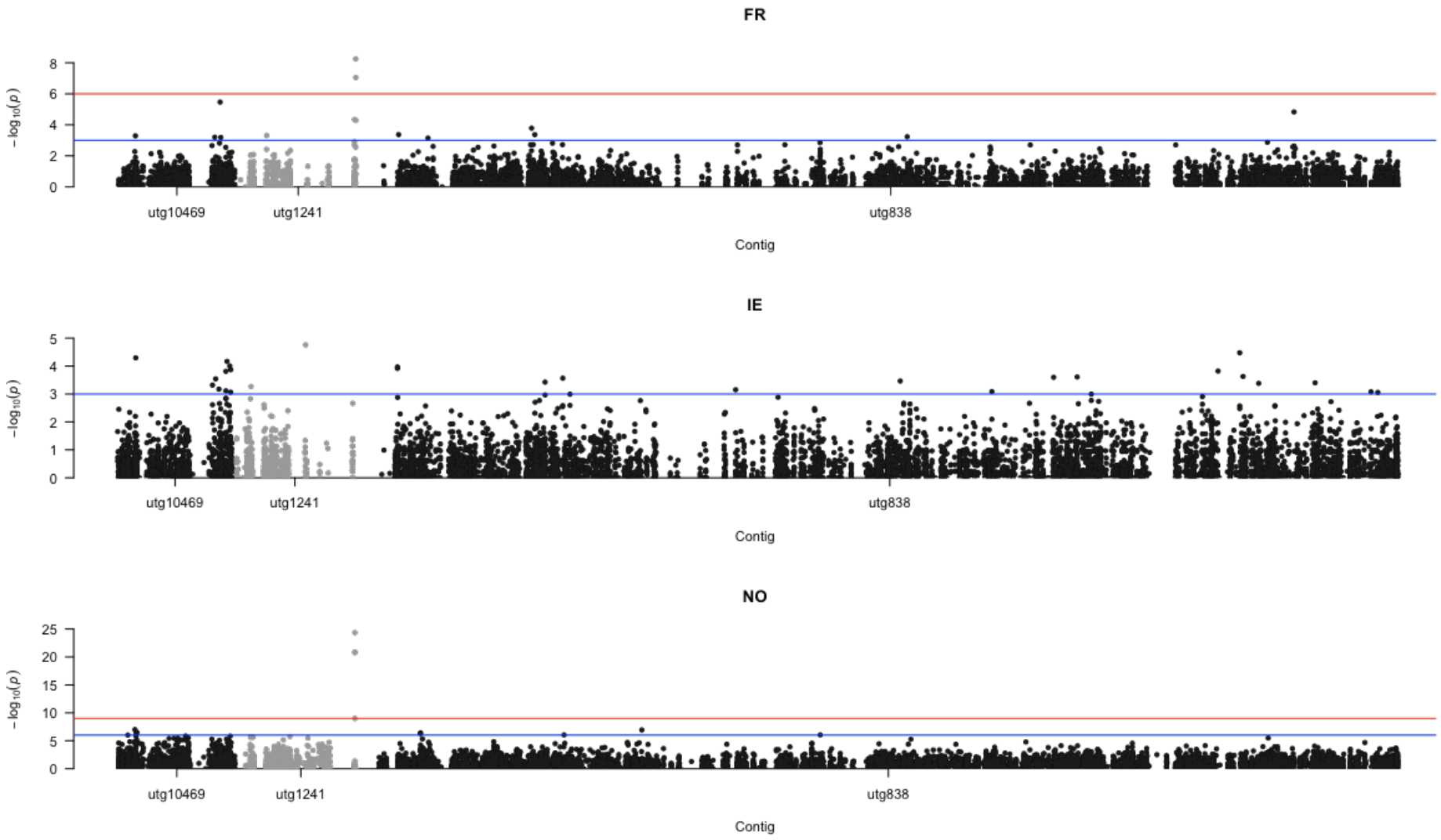








FRIENO shared contigs



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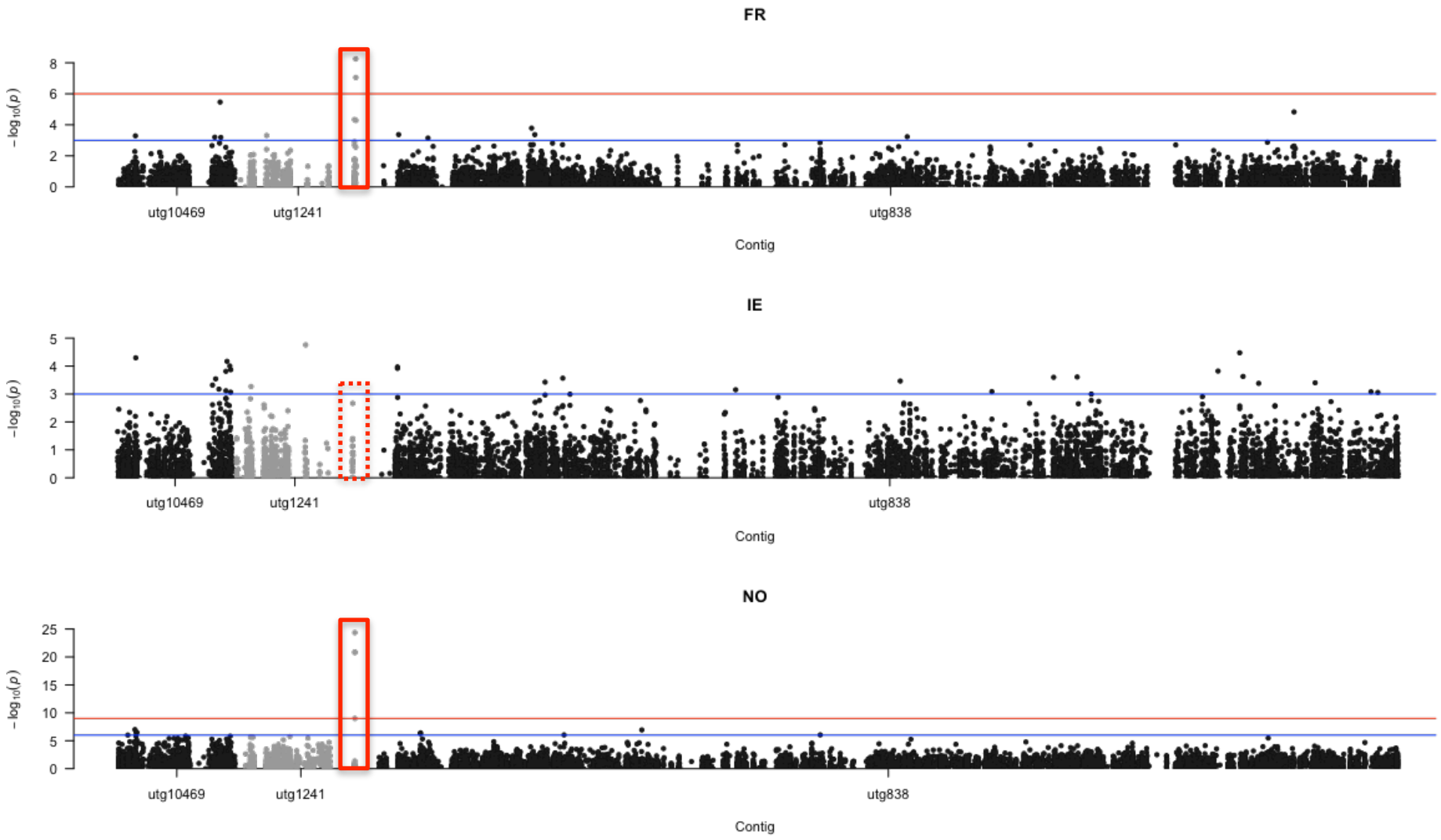

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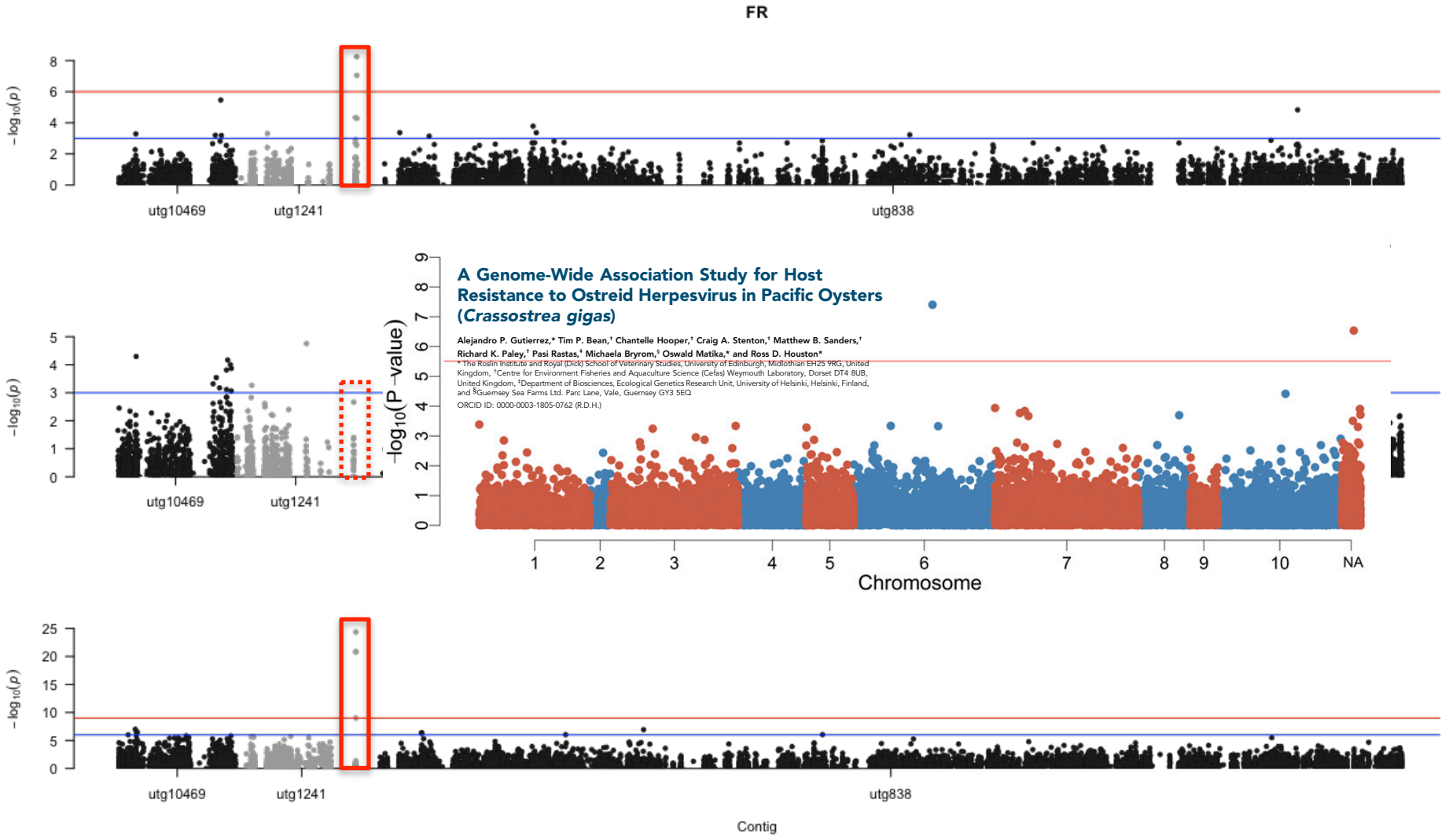
FRIENO shared contigs



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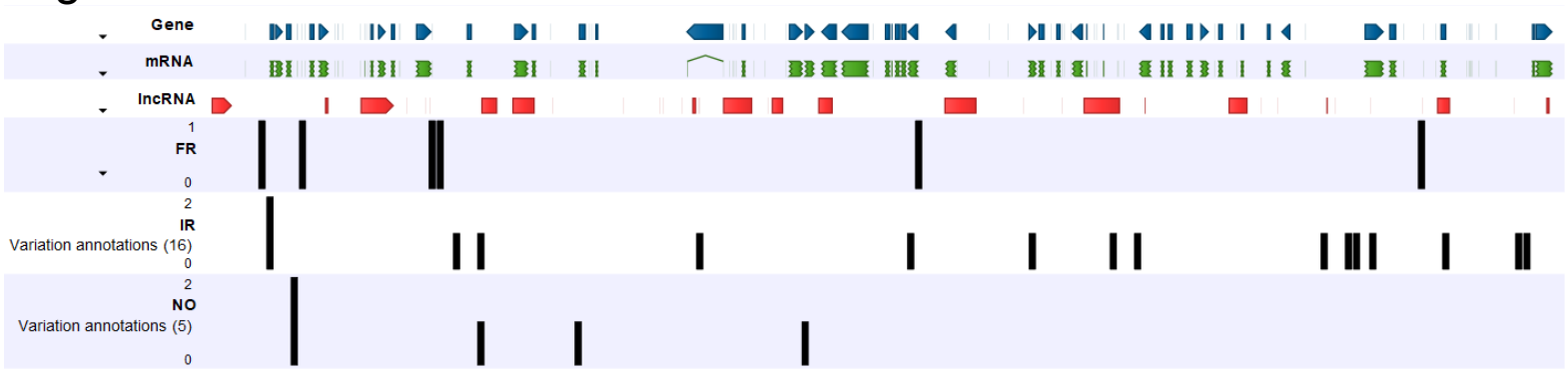

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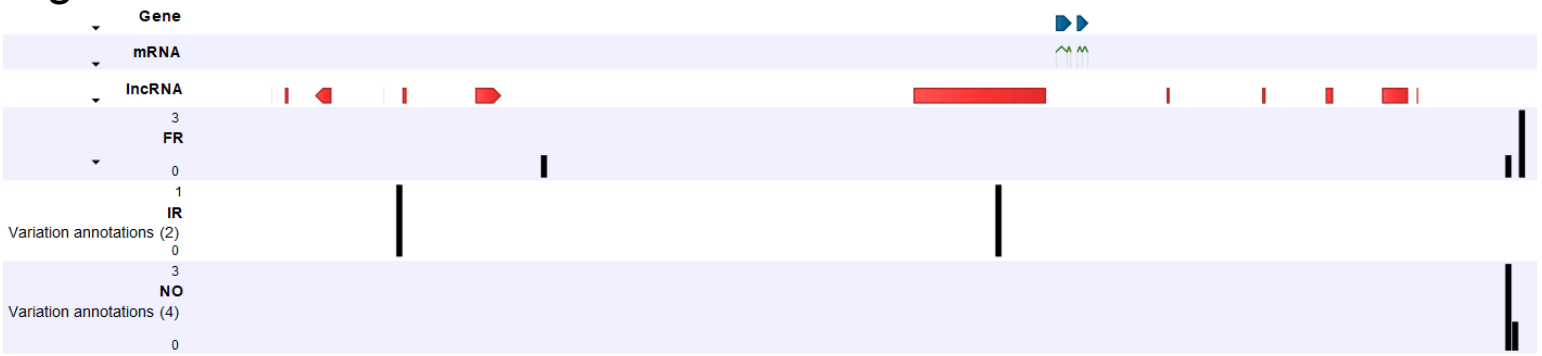

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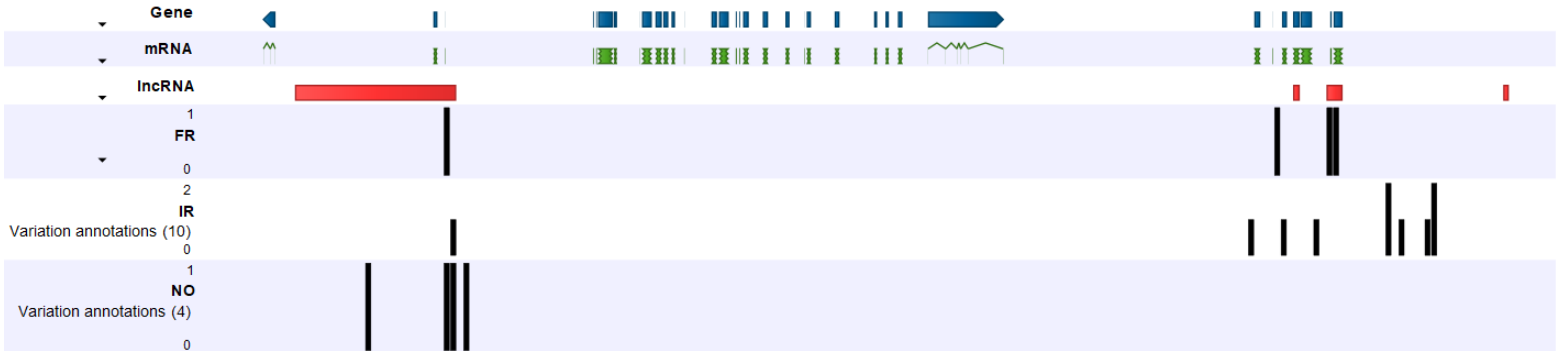

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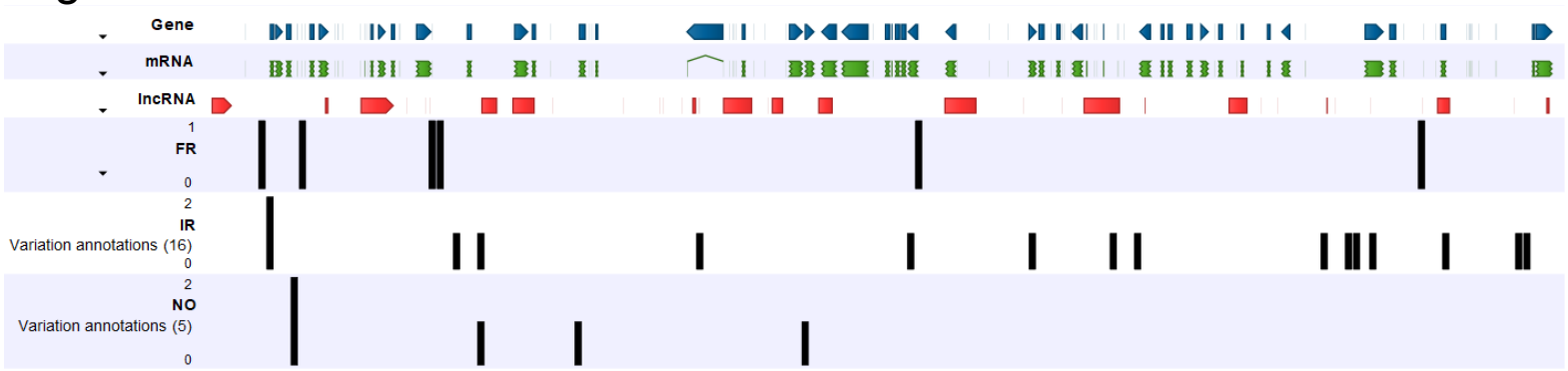

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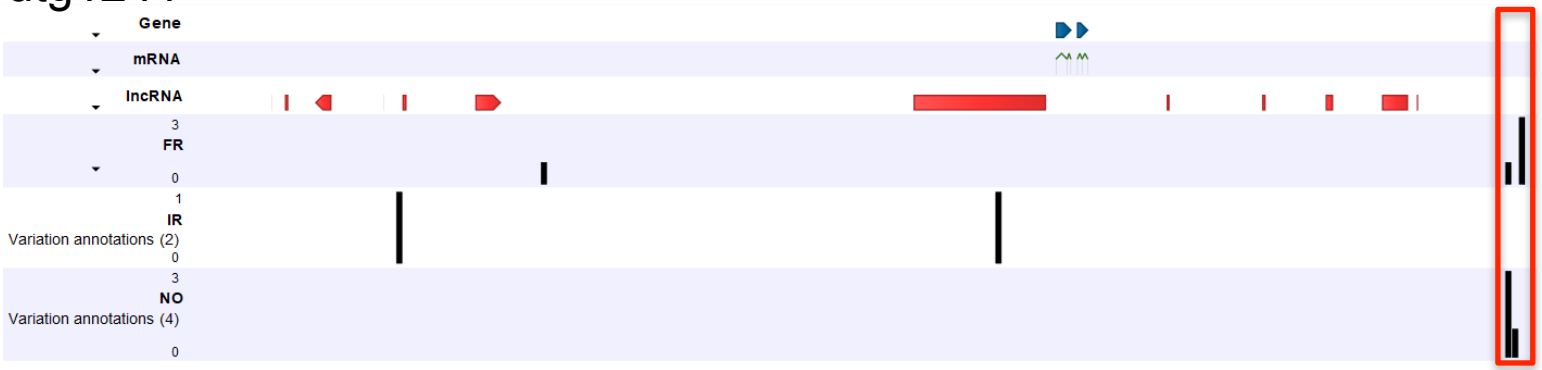

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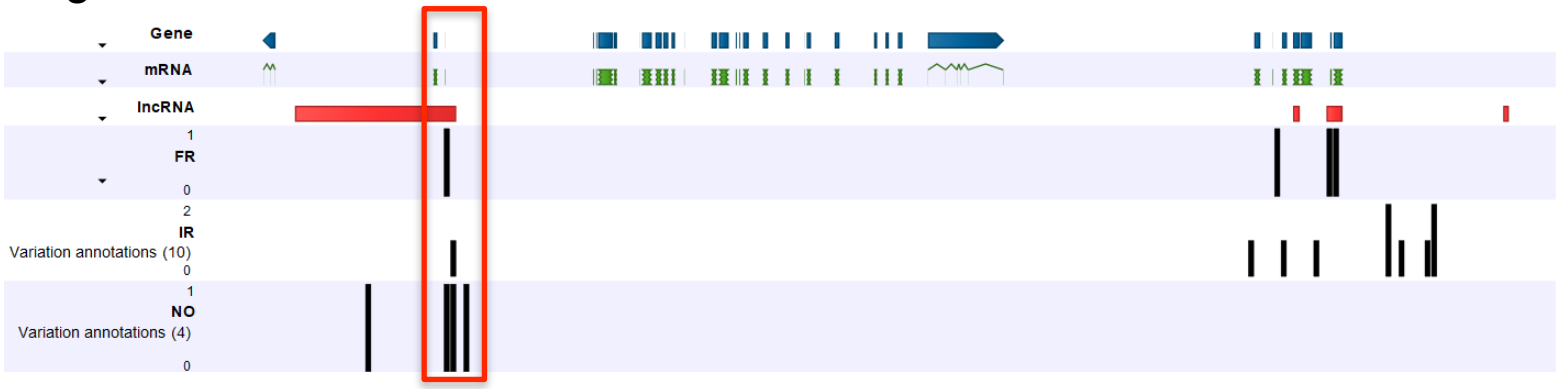
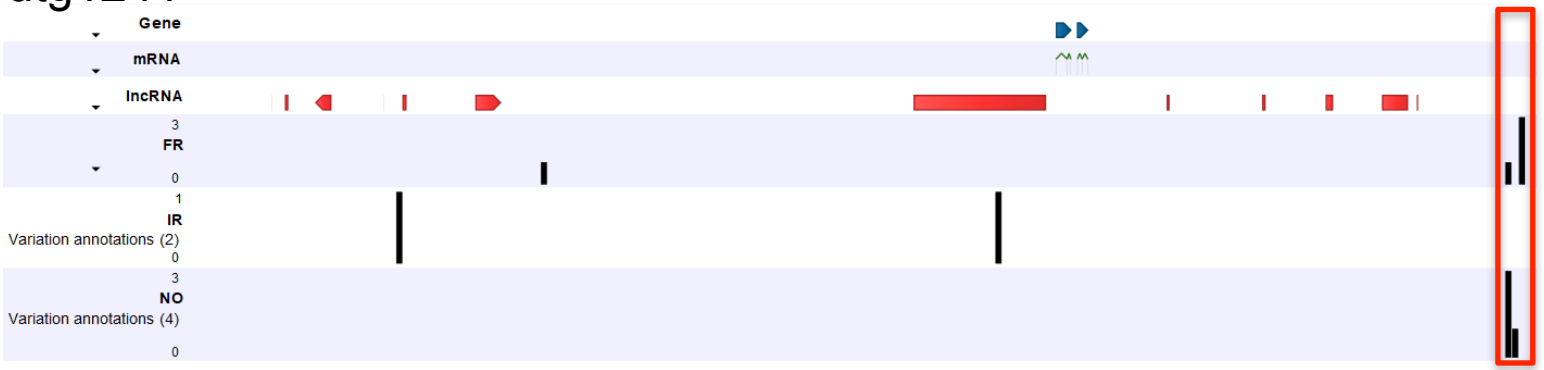


utg878

All over the place…

utg1241



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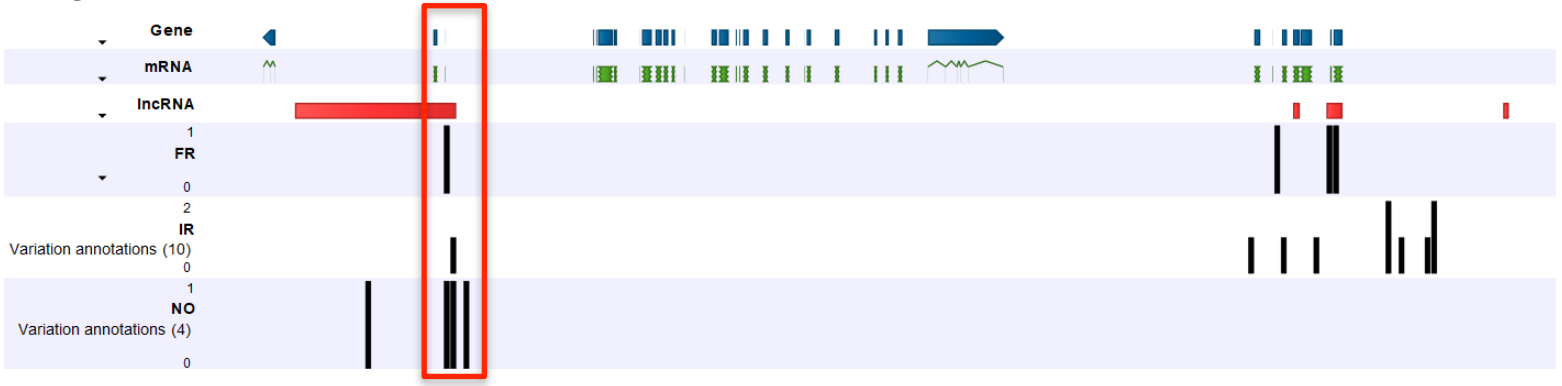
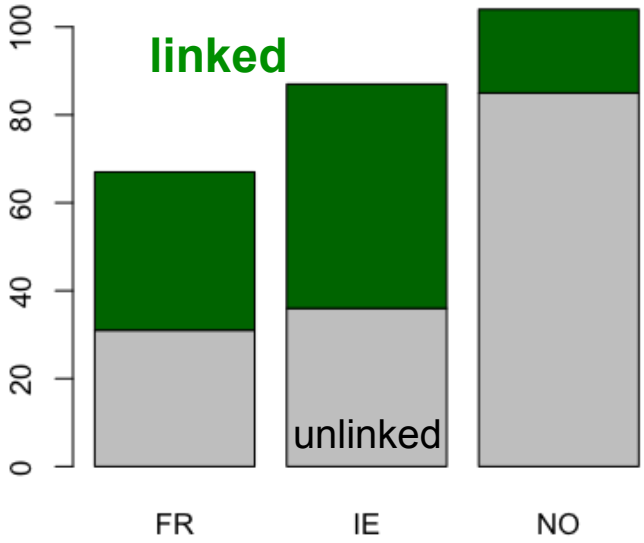


FR-IE-NO Functions



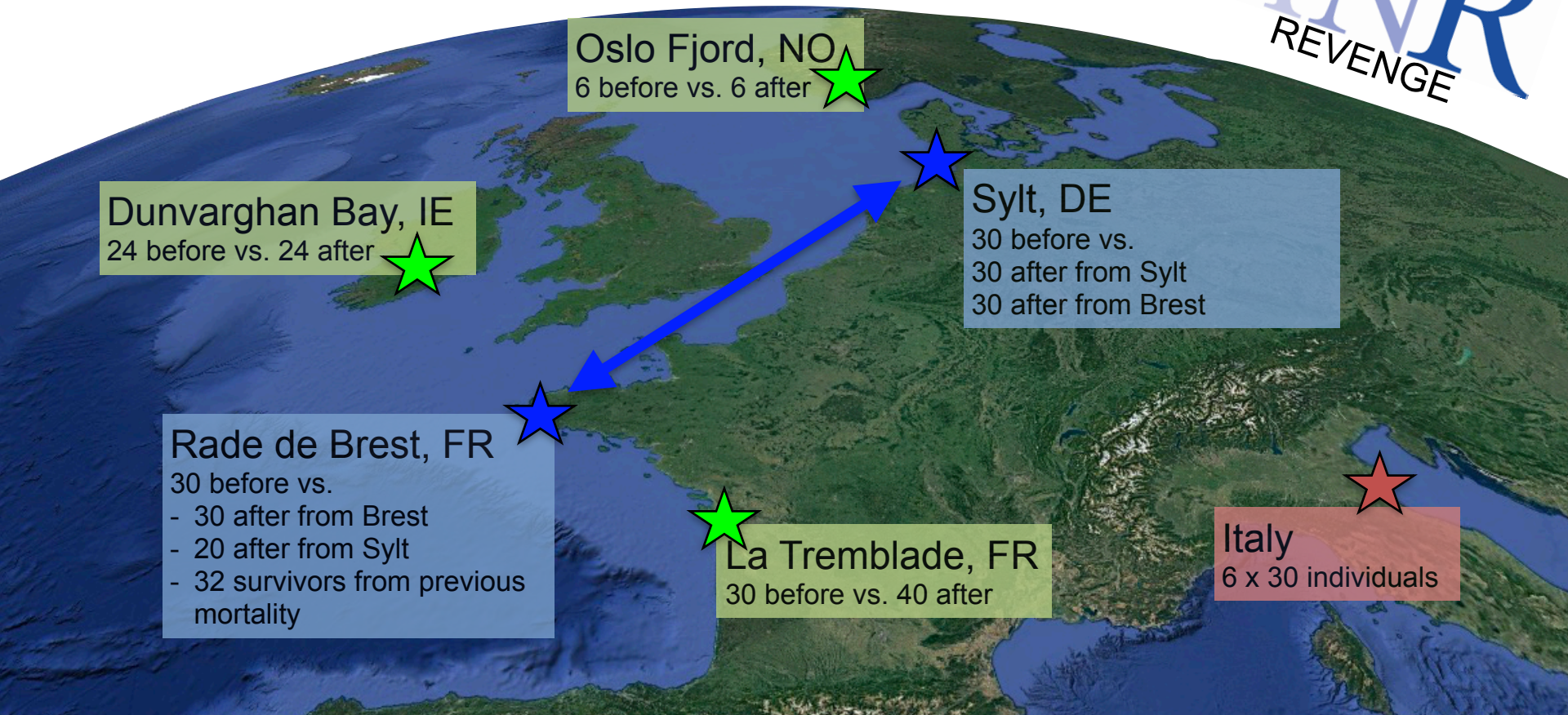
- only few outlier SNPs in coding regions or closely linked to predicted genes
- many predictions without annotation
- Ntoables:
  - lncRNA - regulatory function?
  - Cathepsin Z - phagocytosis?
  - Scavenger receptors

- **caveat**: just old genome annotation squeezed on new assembly (~18000 genes)
- new annotation is on the way

Transplant (assaying the same gene pool in different mortality events)

1. before vs. after mortality samples from field collected animals
2. before vs. after mortality samples from lab bred animals reciprocally transferred between sites
3. (6 Italian populations with varying histories of disease)

AGENCE NATIONALE DE LA RECHERCHE
ANR
REVENGE



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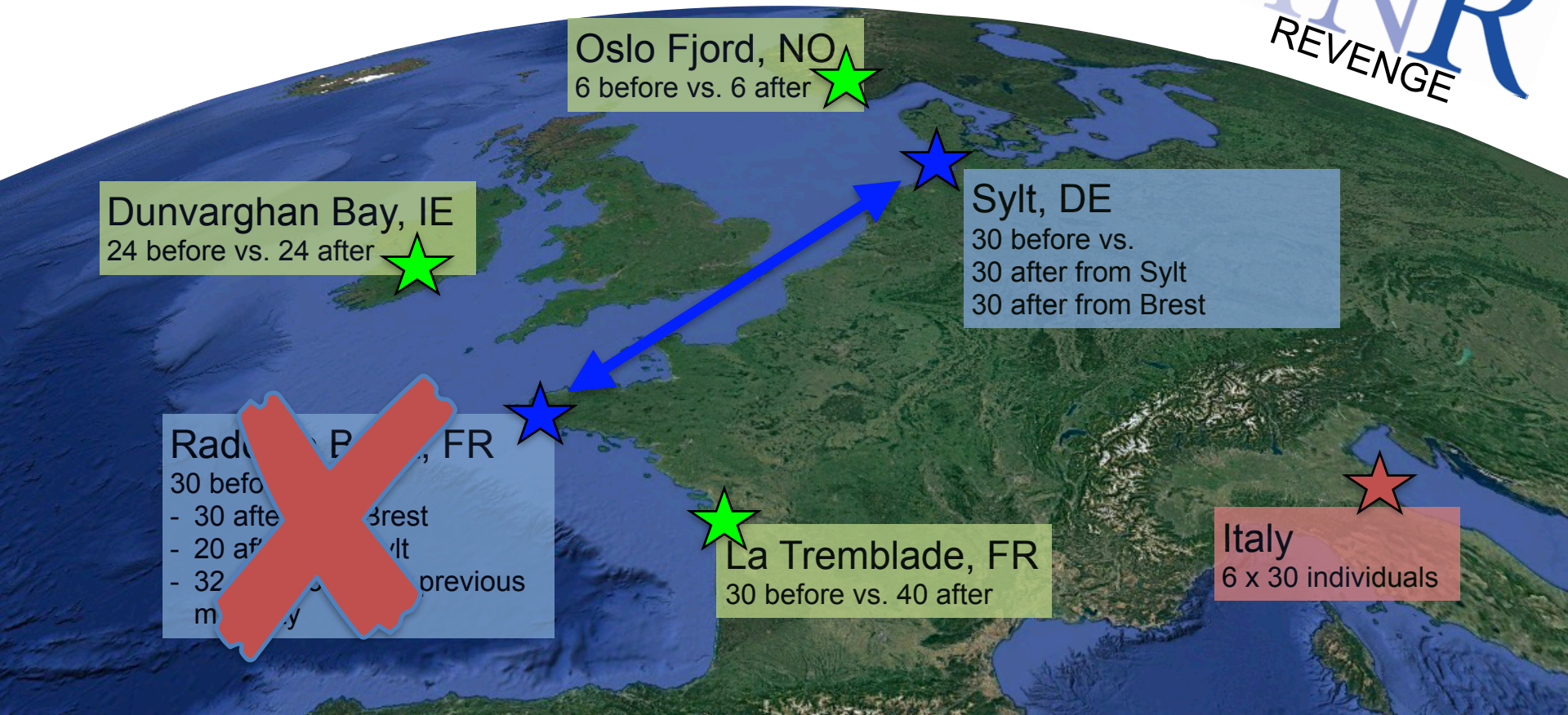
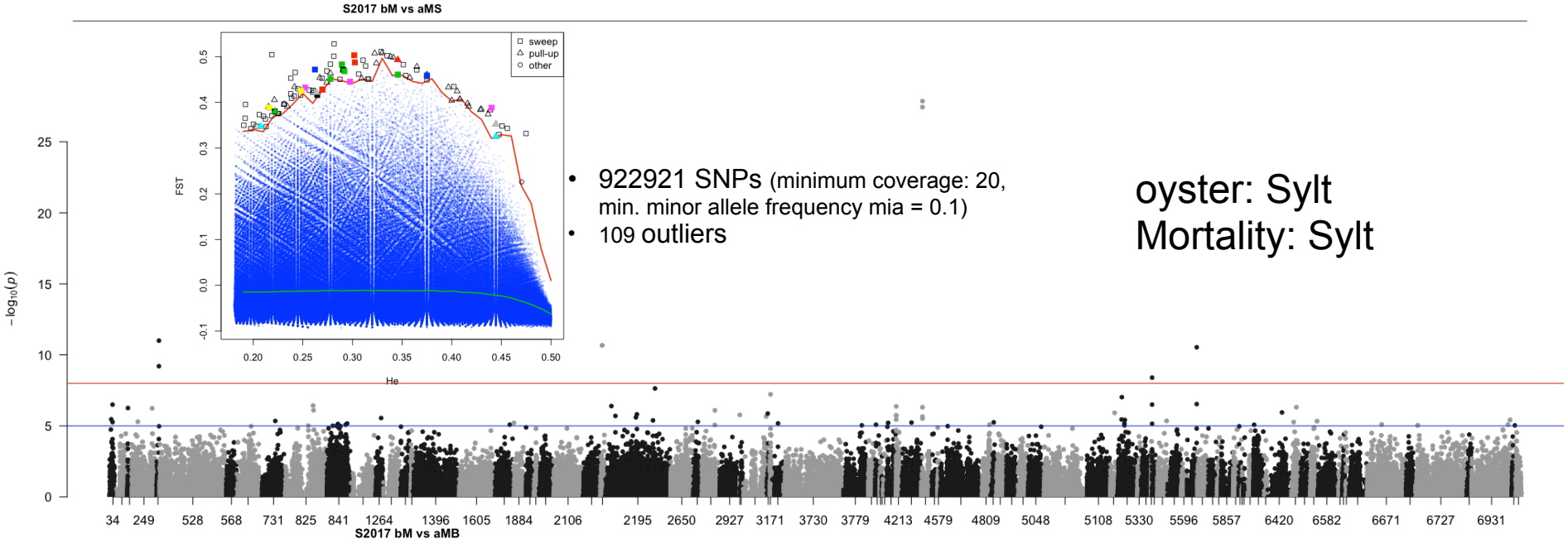
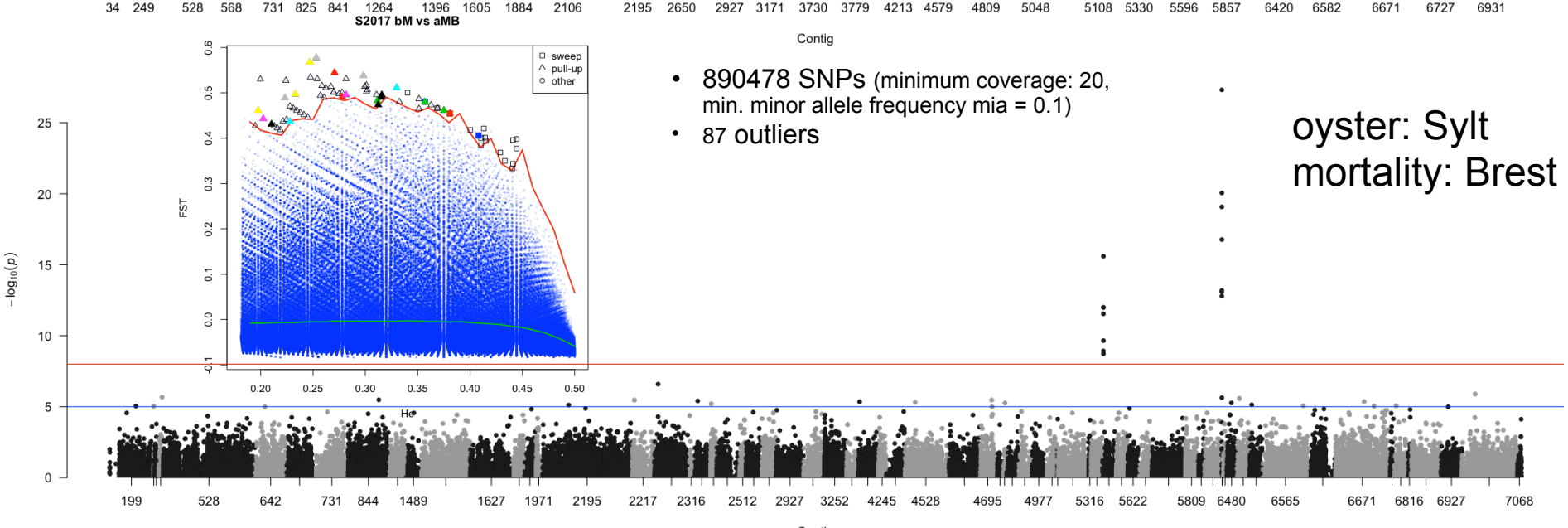


Sylt oysters: mortality Sylt vs Brest



- 922921 SNPs (minimum coverage: 20, min. minor allele frequency mia = 0.1)
- 109 outliers

oyster: Sylt
Mortality: Sylt



- 890478 SNPs (minimum coverage: 20, min. minor allele frequency mia = 0.1)
- 87 outliers

oyster: Sylt
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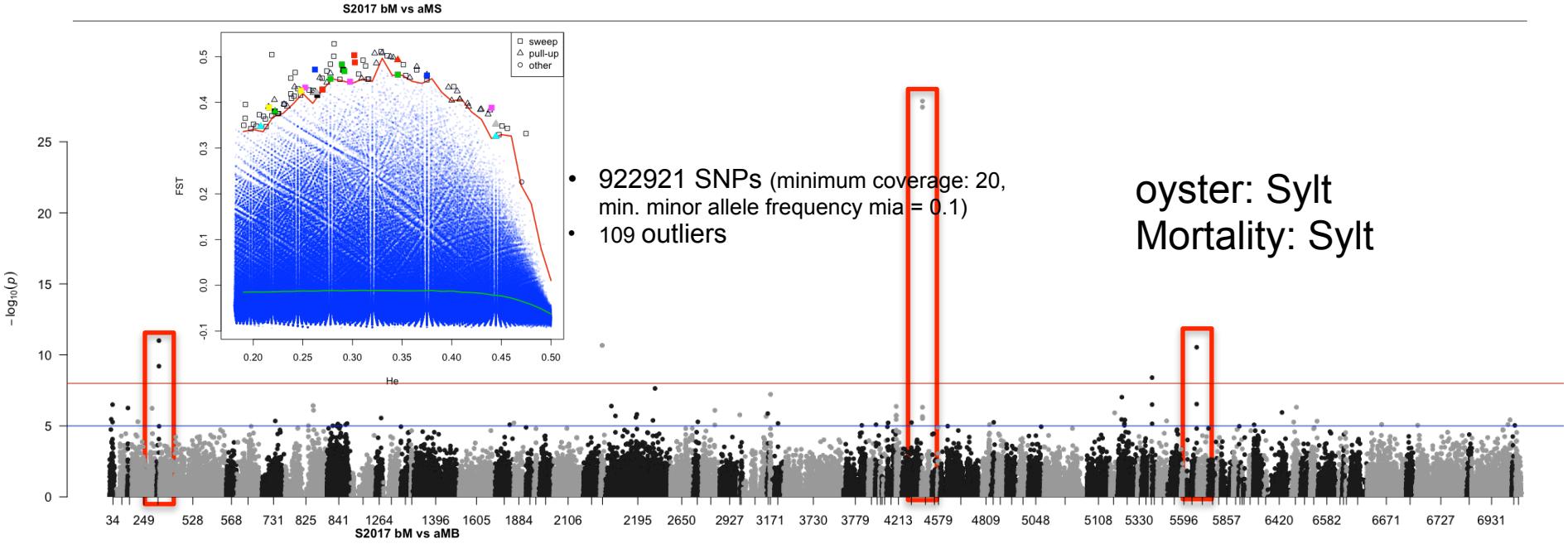
Sylt oysters: mortality Sylt vs Brest



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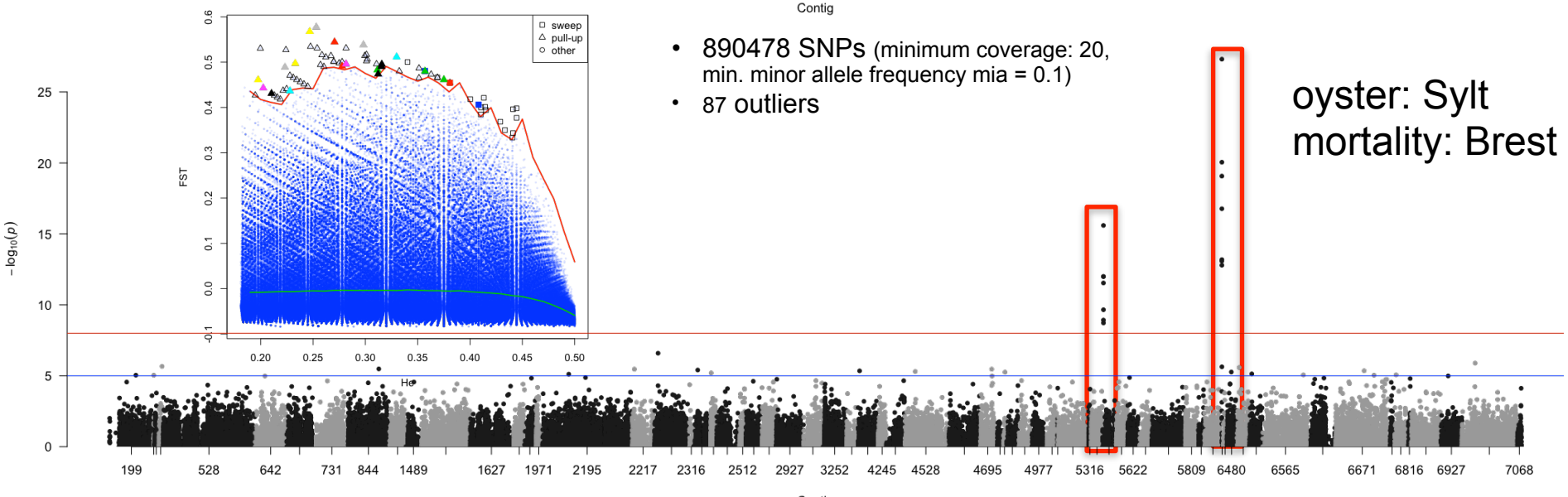

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FR vs IE vs NO

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- 2 shared SNPs

FR vs IE vs NO

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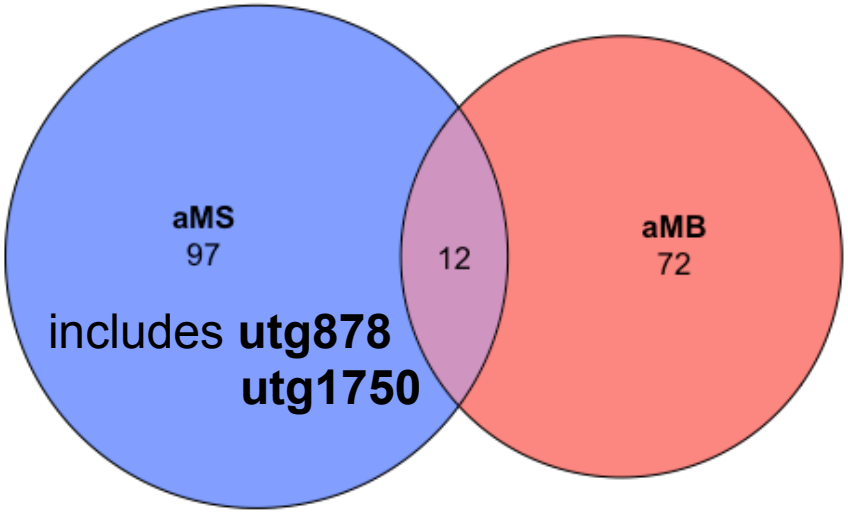
- 12 contigs shared between different sites



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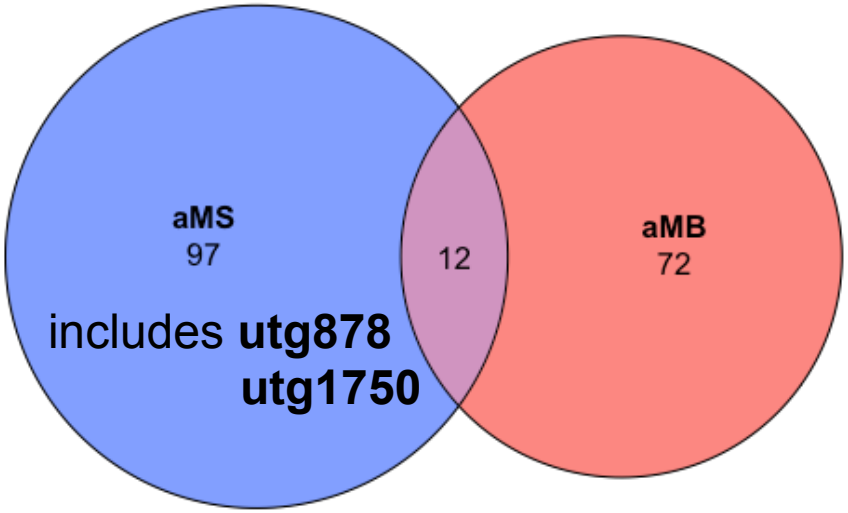
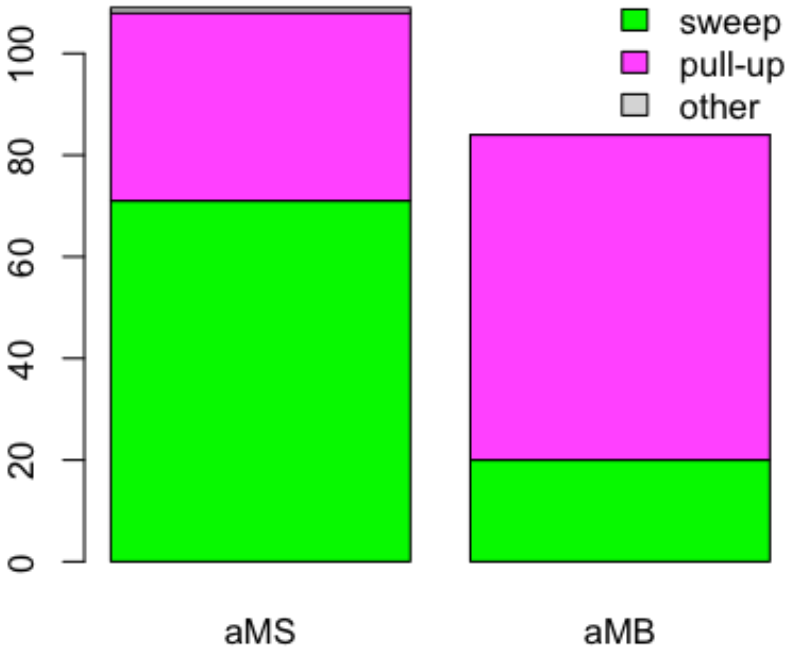
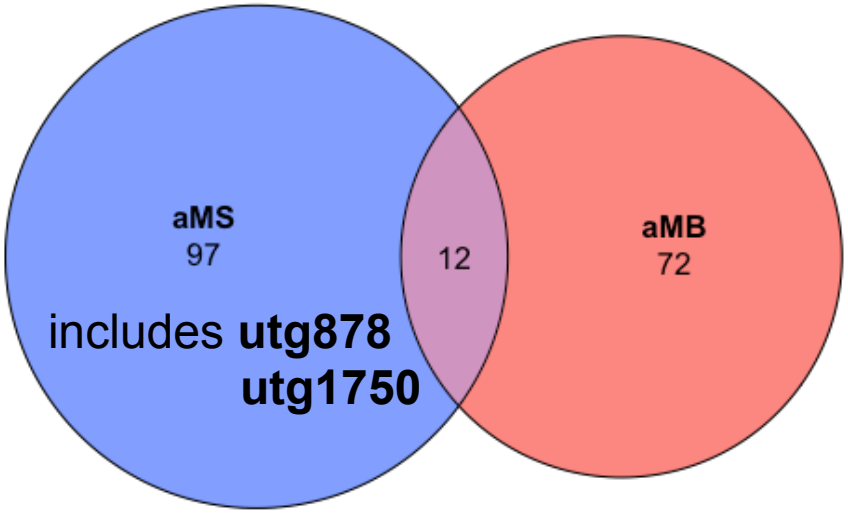
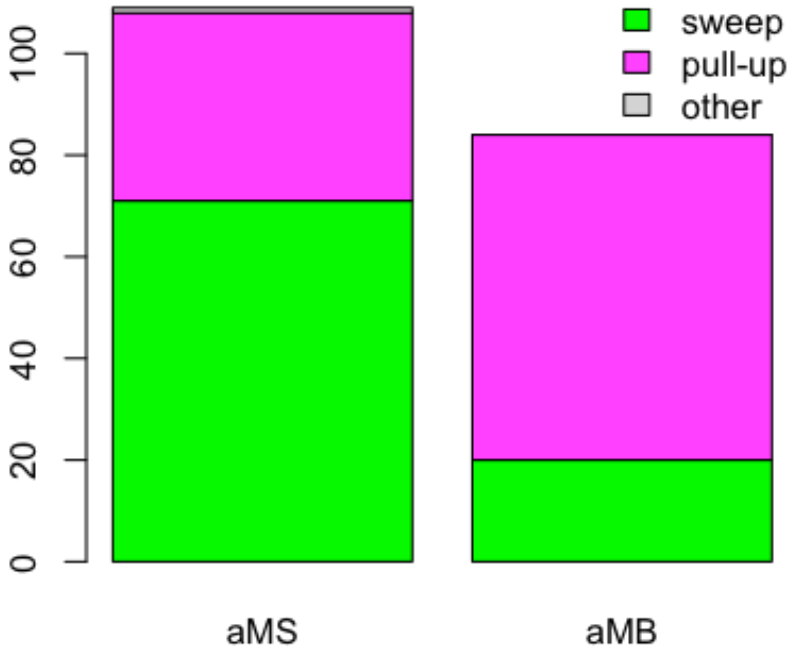




FR vs IE vs NO

- 2 shared SNPs
- 12 contigs shared between different sites
- **selection acts differently on the same gene pool!**





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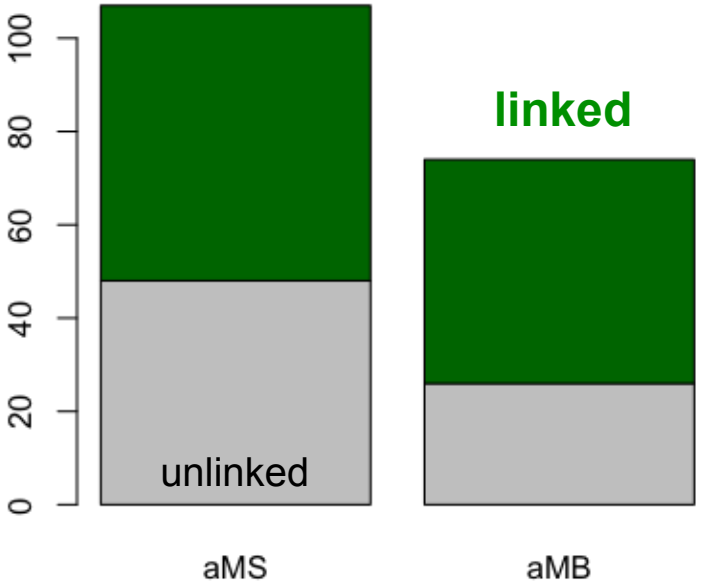
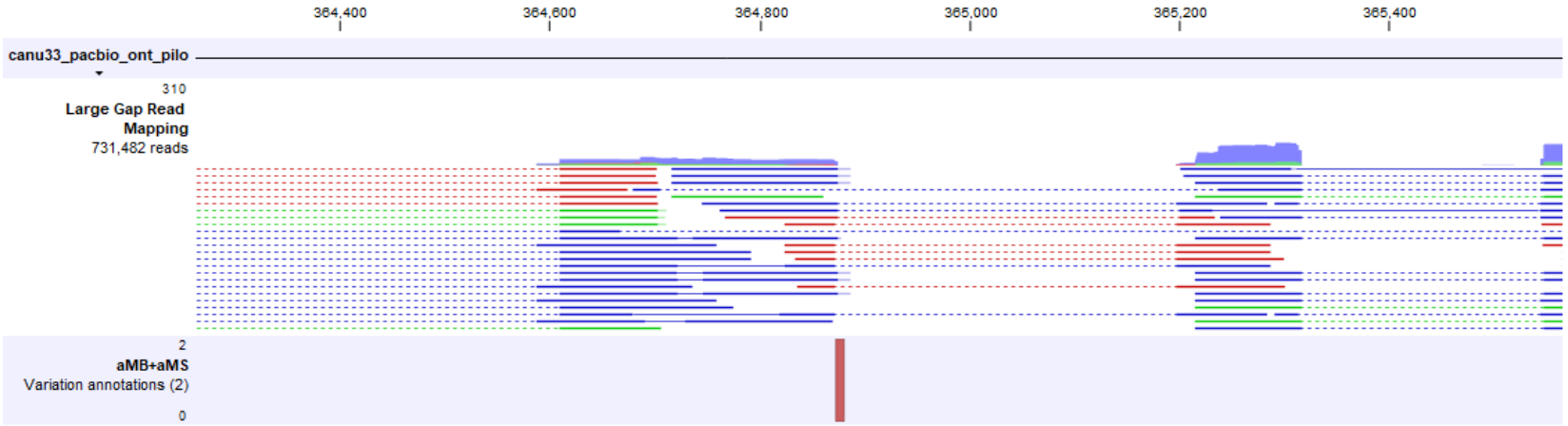
Sylt oysters: Functions



- Notables:
  - Cathepsin Z
  - exonucleases
  - Gramicidin synthetase



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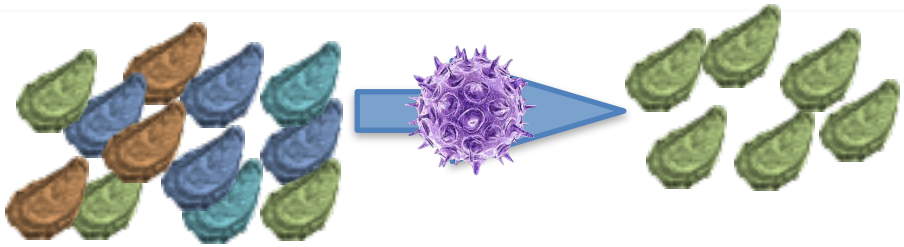
Conclusion



Good news:

- There are common genomic regions shared between distinct mass mortality events - resistant lines might work globally to a given extent

Not so good news:

- Many more genomic regions specific to different populations and/or mortality events
- Specific regions show stronger allele frequency shifts
- real targets of selection (i.e. genes) are still elusive

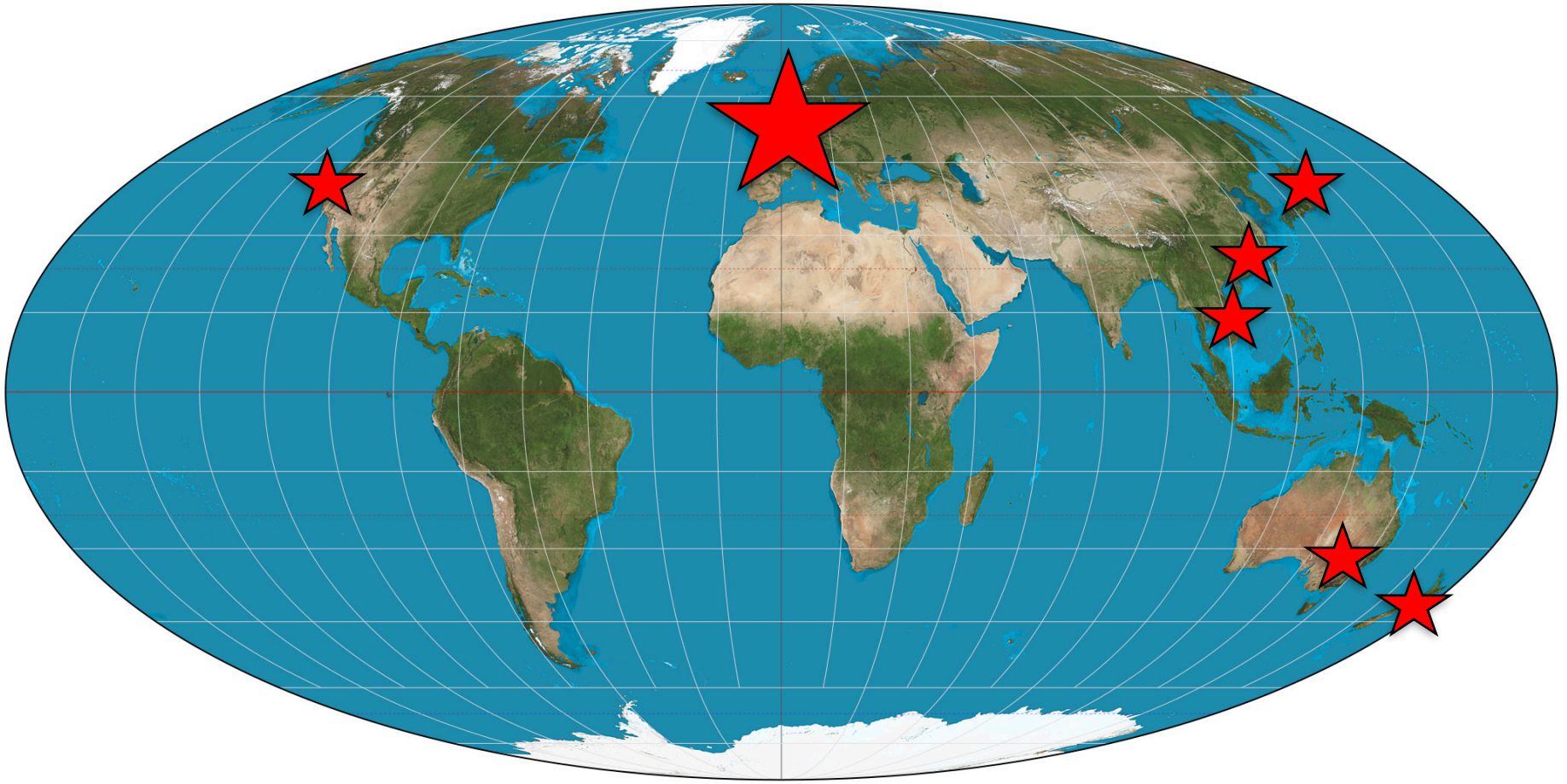
K. Mathias Wegner, Ana Lokmer, Umberto Rosani, Uwe John, Stefan Neuhaus, Eike Petersen
Deborah Chesslett, Stein Mortensen, Paola Venier, Benjamin Morga, JB Lamy,
Marianne Alunno-Bruscia, Bruno Petton

ALFRED-WEGENER-INSTITUT
HELMHOLTZ-ZENTRUM FÜR POLAR-
UND MEERESFORSCHUNG

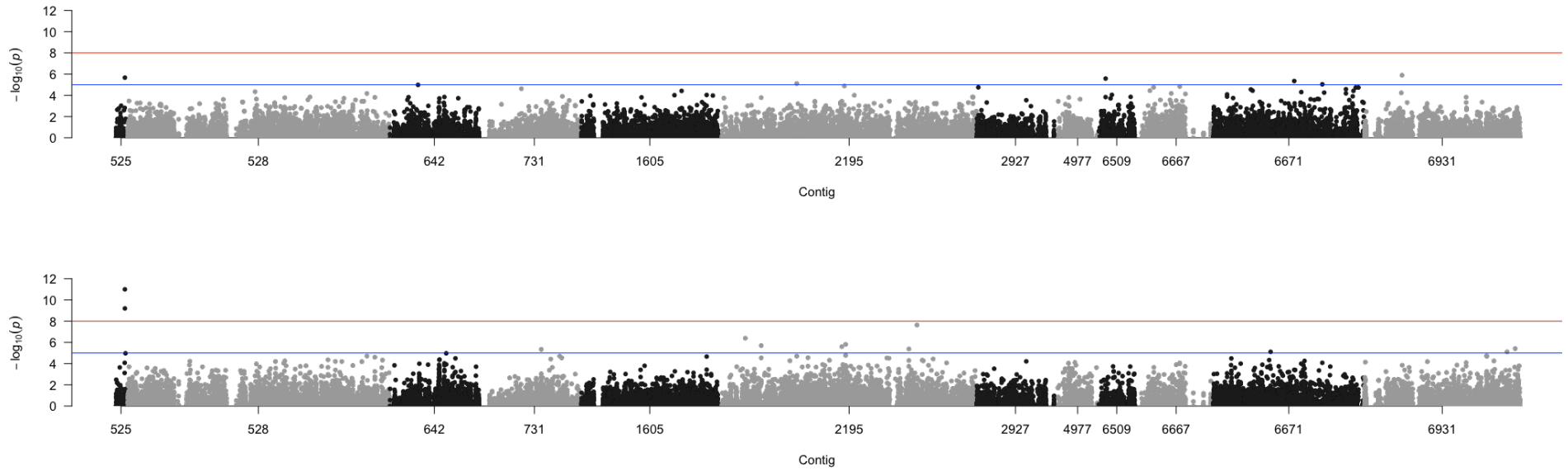
Genomic signatures of selection across mass mortality events in European populations of Pacific oysters

VIVALDI

Os-Hv1 mortalities



Sylt: shared contigs



- **not the strongest allele frequency shifts in the shared contigs**