

BACKGROUND

- Powdery mildew (*Blumeria graminis* f.sp. *Tritici*) is a significant reducer of wheat grain yields in Southeastern U.S. growing conditions.
- Resistant germplasm is the most economical method of fungal control.
- Wheat resistance is driven by a combination of quantitative and qualitative resistance genes.

POPULATION

- Created 15 biparental RIL populations.
- These were divided into 3 nested association map (NAM) groups.
- Planted ~132 individuals from each biparental in three environments.

PHENOTYPE

- Assessed powdery mildew resistance on a 0 (no infection) to 4 (flagleaf infested) scale.
- Calculated BLUEs using a discrete ordered categorized response variable through a cumulative logit link function:

$$y = Xb_{Genotype} + Zu_{Location:Year:Block} + \varepsilon$$

GENOTYPING

- Sequenced all lines with GBS.
 - Filtered raw data and created biparental linkage maps and a whole-population imputed dataset.
- Ran KASP assays for major known powdery mildew genes on parents.

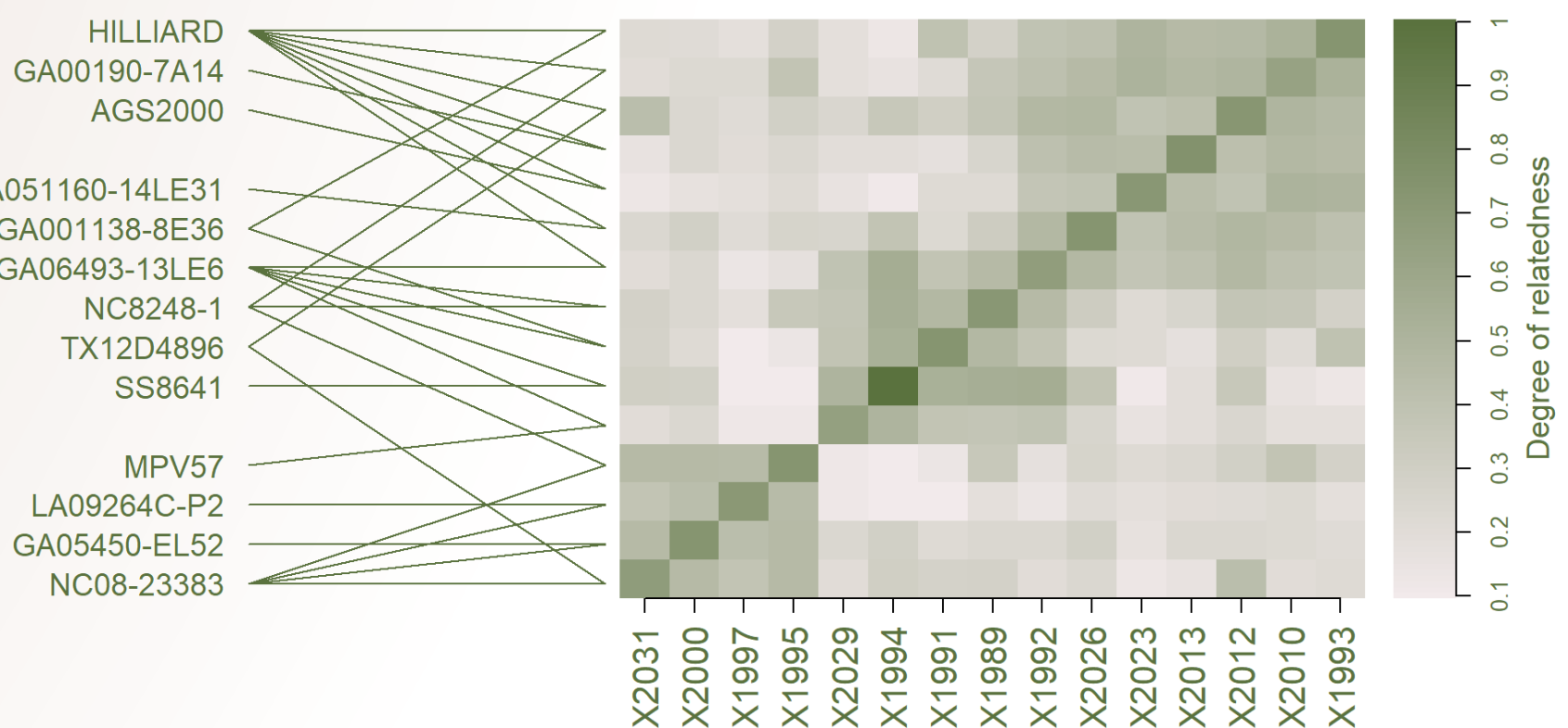


Figure 1: Relatedness of each biparental; color represents whole-population averages. Right column and lines shows parentage of biparental.

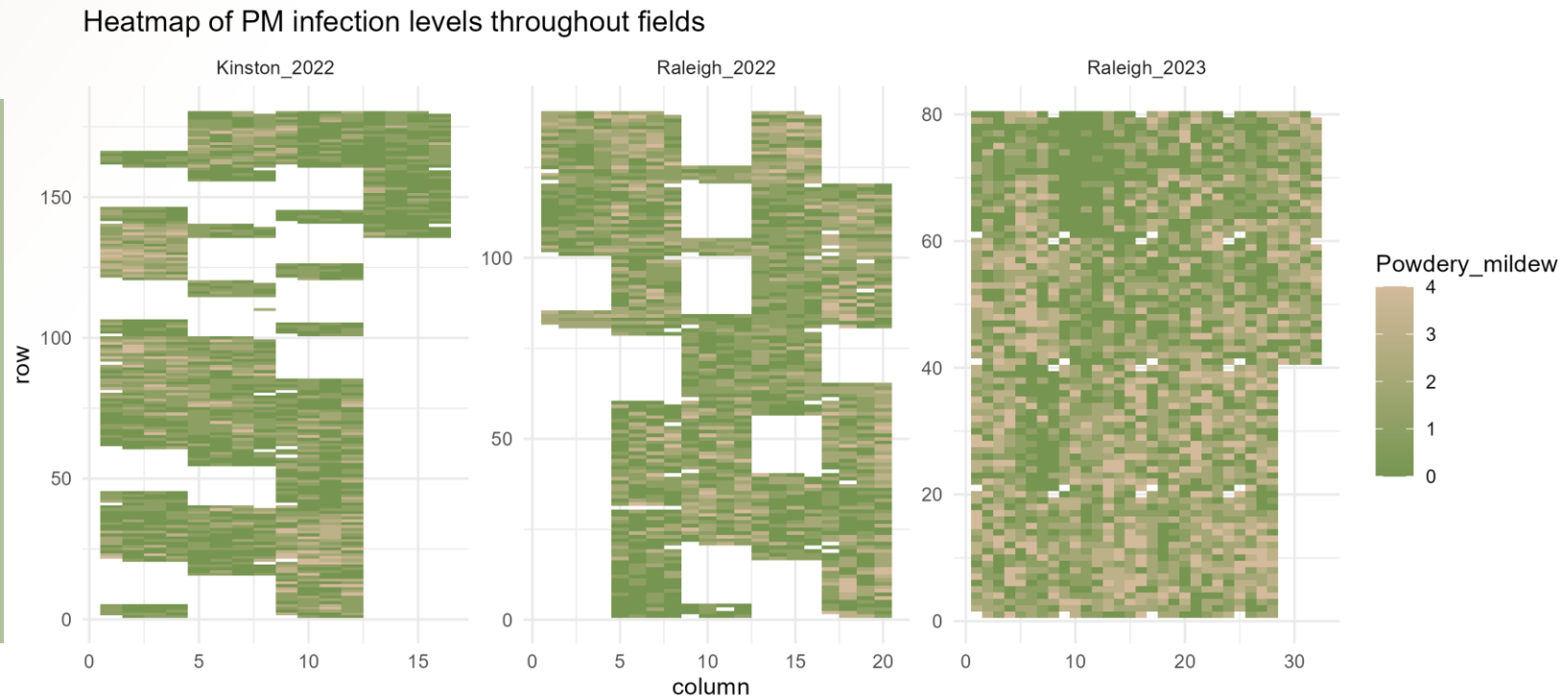


Figure 2: Field ratings for all lines, separated by environment. Rated on 0 (no disease) to 4 (flagleaf infested) scale

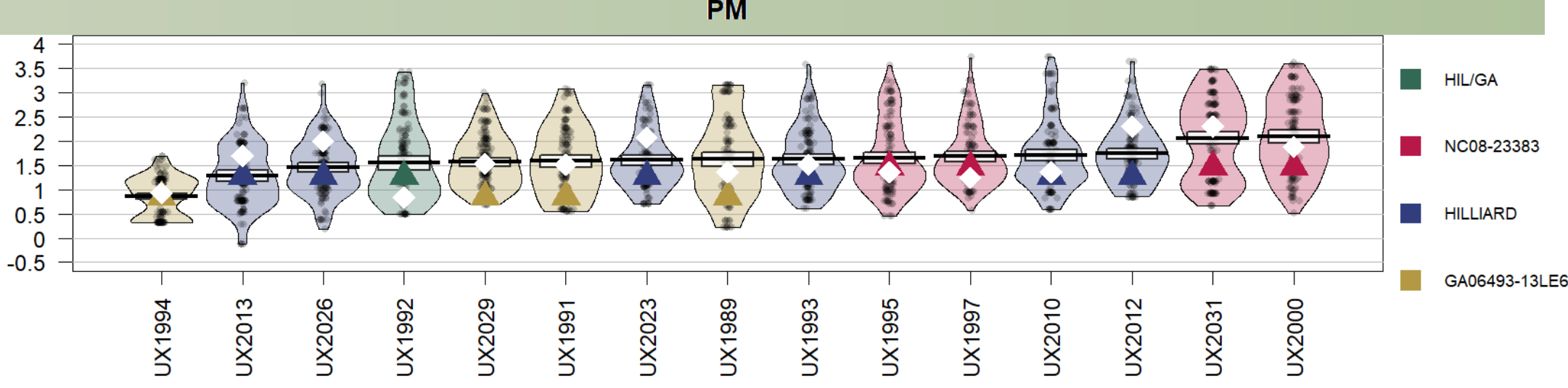
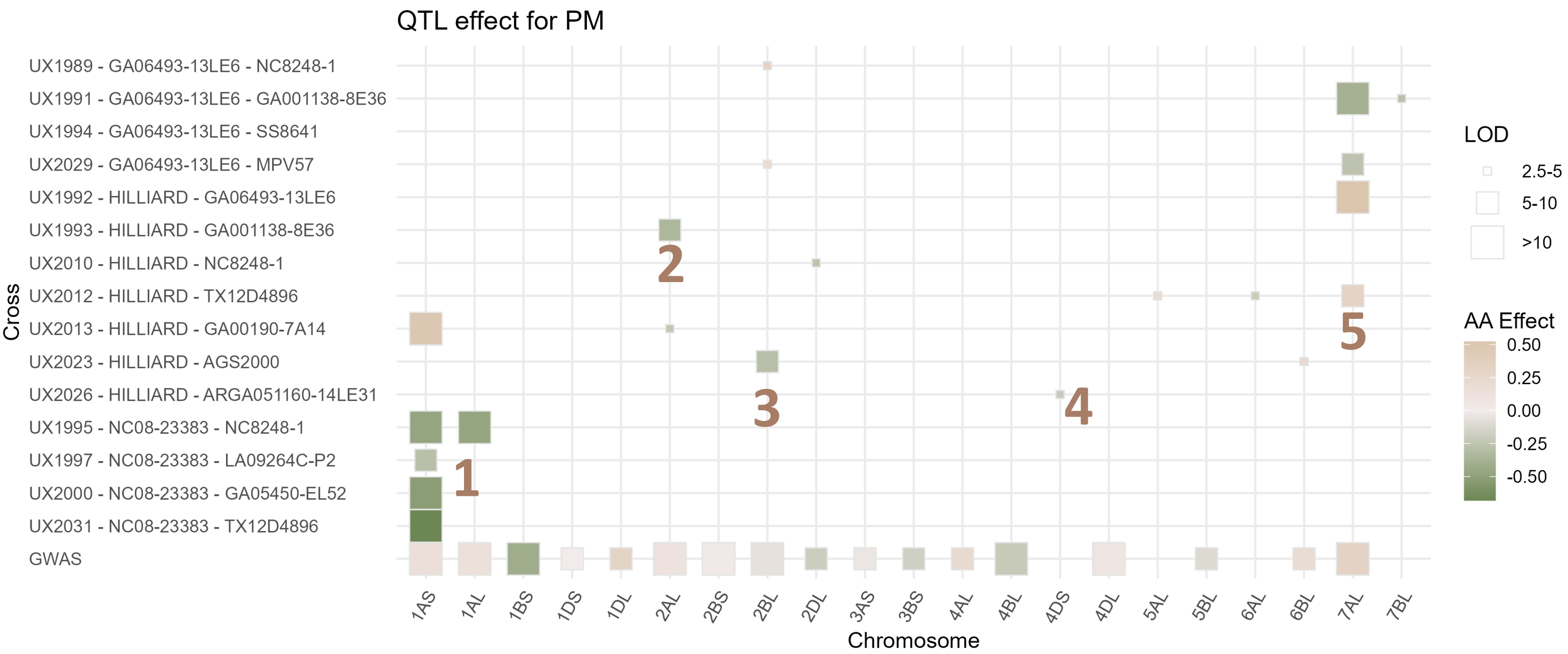


Figure 3: BLUEs for all lines plotted in black dots by biparental. Violin shows distribution of BLUE and color designates NAM founder, with colored triangle designating NAM founder BLUE. White diamond shows diversity parent BLUE, black line shows population mean.

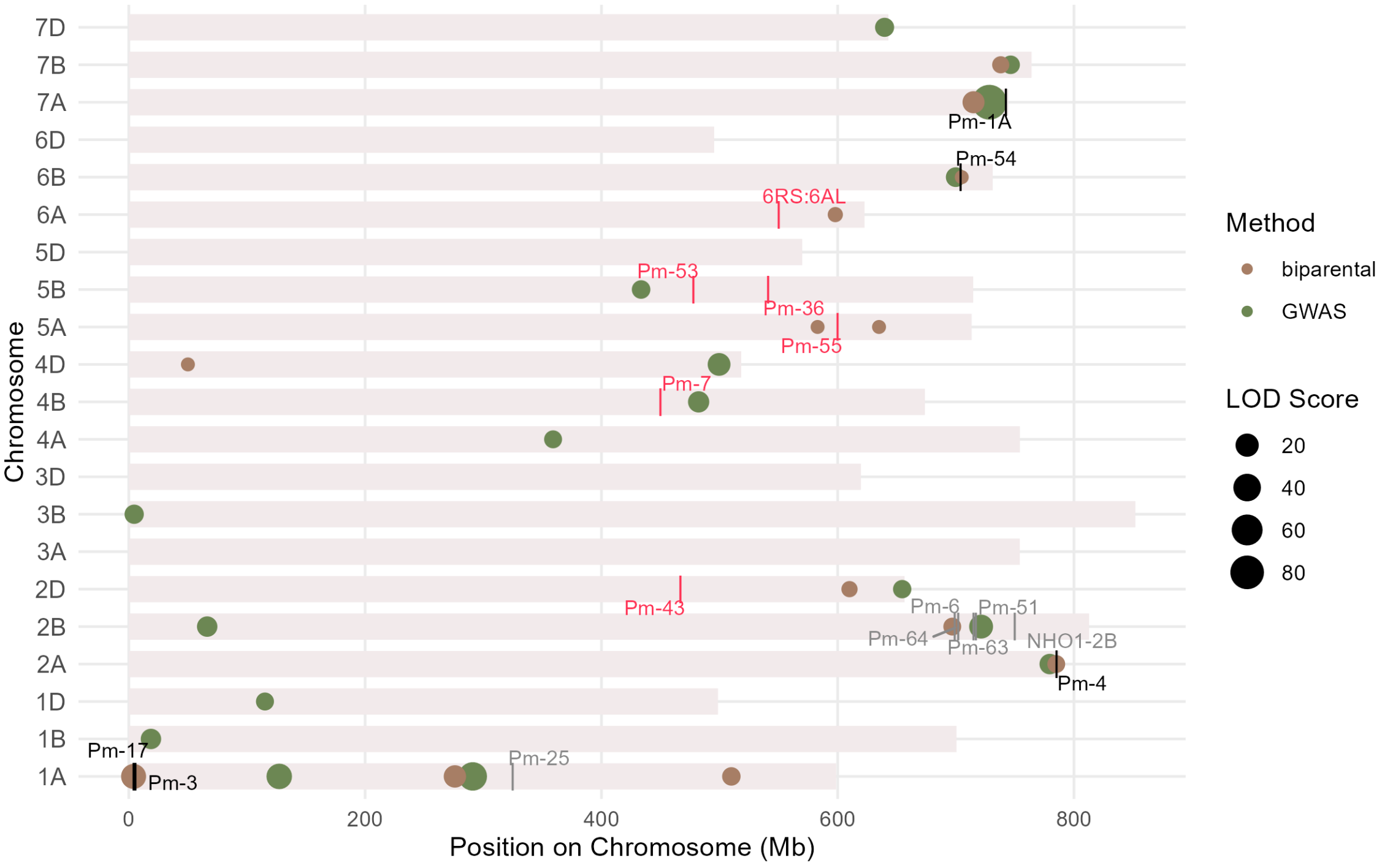
ANALYSIS

- Performed linkage analysis using r/qt12.
- Performed GWAS using r/ASReml, rrBLUP, and GAPIT.



Comparison on linkage analysis from all biparentals and aggregated GWAS results. Chromosomes are split into short (S) or long (L) arms.

1. Both *Pm3* and *Pm17* are in the 0-400 Mb region; due to the 1RS:1AL introgression there is no recombination in this span. Neither are expected to confer field resistance, but there is no resolution for fine-mapping the loci of greatest effect for gene identification.
2. Location of *Pm4*.
3. There are many putative genes for this loci, but none are very likely.
4. There are many small-effect loci identified by only 1-2 tests from 2D-6B. None have known genes associated with them.
5. Major resistance comes from *Pm1a*; UX2012 does not segregate for this gene and thus that peak represents a new gene.



Pictogram showing linkage analysis and GWAS results in chromosomal position, with identified genes, putative genes, and proximal but rejected genes for each QTL peak with relevant literature.