WHEAT YELLOW RUST, BEHIND THE UNDERSTANDING OF THE GENETIC ARCHITECTURE AND IN SEARCH OF RESISTANCE SOURCES IN GENETIC RESOURCES OF URUGUAY

VENANCIO RIELLA[1,2], BETTINA LADO[2], PAULA SILVA[1], LUCÍA GUTIÉRREZ[3], SILVIA GERMÁN[1]

[1] INSTITUTO NACIONAL DE INVESTIGACIÓN AGROPECUARIA (INIA), ESTACIÓN EXPERIMENTAL LA ESTANZUELA, COLONIA, URUGUAY
[2] DEPARTAMENTO DE BIOMETRÍA, ESTADÍSTICA Y COMPUTACIÓN, FACULTAD DE AGRONOMÍA, UNIVERSIDAD DE LA REPÚBLICA
[3] DEPARTMENT OF PLANT & AGROECOSYSTEMS SCIENCES, UNIVERSITY OF WISCONSIN - MADISON

INTRODUCTION. Yellow rust (YR) in Uruguay

- Caused by *Puccinia striiformis* f. sp. *tritici* (Pst)
- Pst population composed of races virulent over many local cultivars
- Breeding for cultivars resistant to *Pst* is the most cost-effective and environmentally friendly way to control YR

1929-1930 → **2017** →

Widespread, severe epidemics. Significant grain yield losses
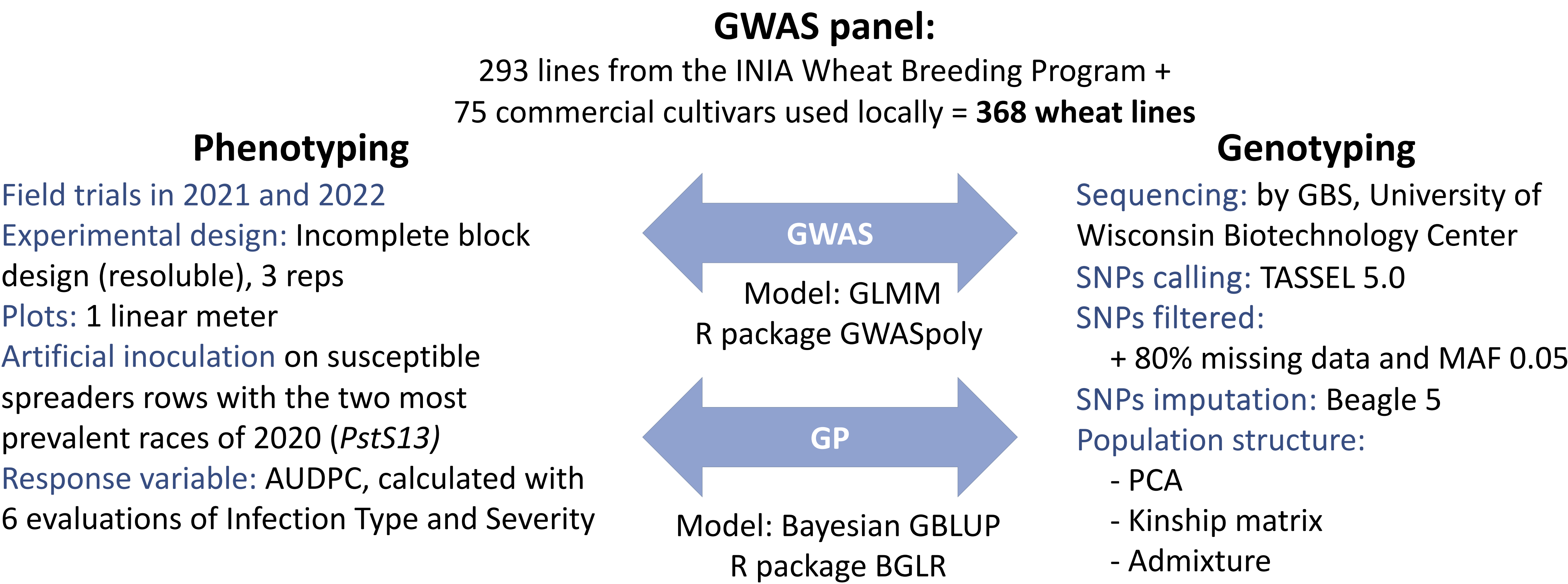
Sporadic occurrence, rarely epidemic

Since 2017, associated with the presence of more aggressive *Pst* races migrated from other continents, **yellow rust** has become the **most important wheat foliar disease**
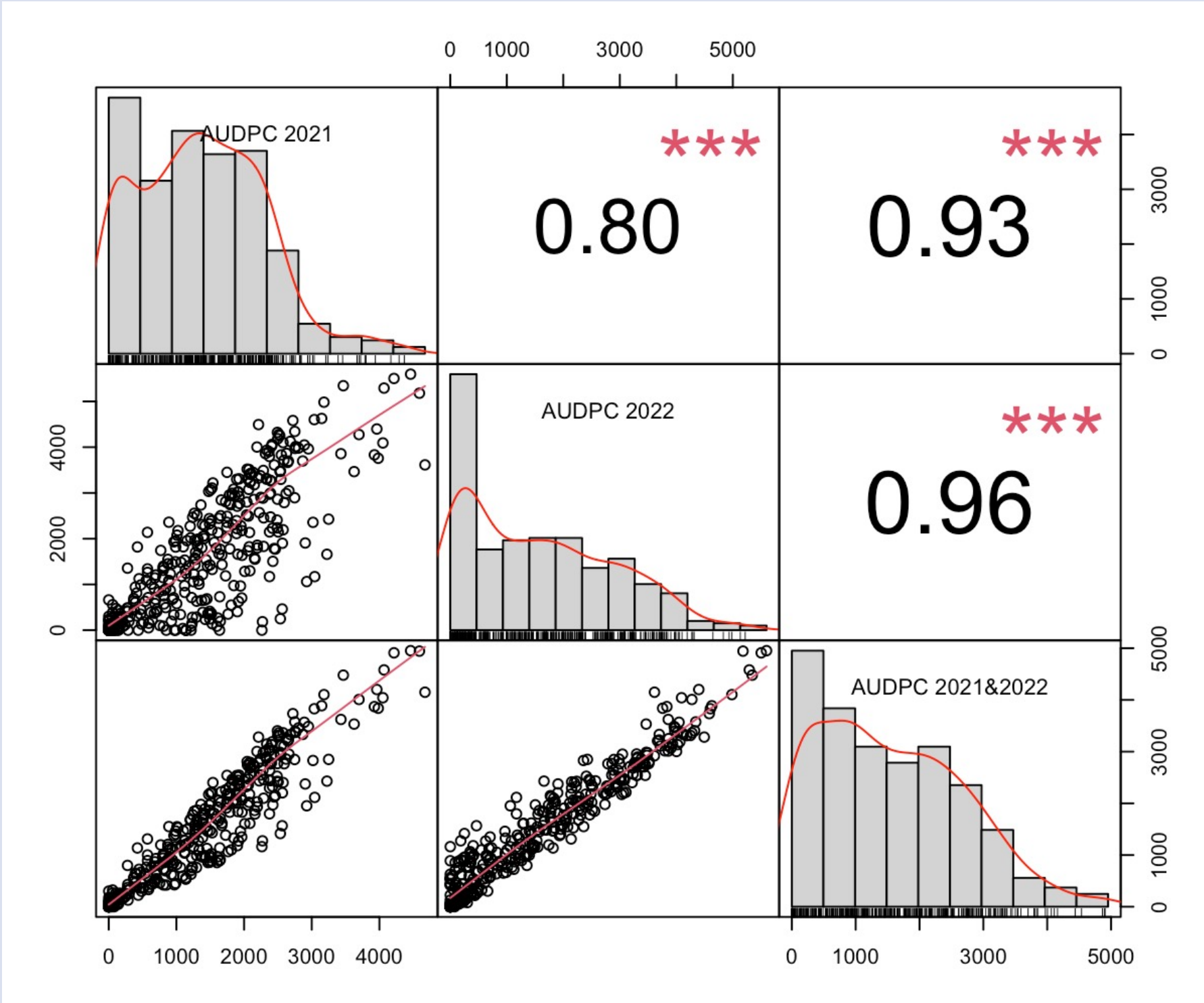
Generalized epidemics in Uruguay and Argentina. Yield losses of up to 80%

OBJECTIVES

- **Characterize** the wheat **resistance architecture** using genome-wide association studies (**GWAS**), and
- **predict wheat lines' behavior** against yellow rust employing genomic prediction (**GP**) models,
as prerequisites for the breeding of wheat cultivars with resistance to yellow rust.

MATERIALS & METHODS

GWAS panel:
293 lines from the INIA Wheat Breeding Program + 75 commercial cultivars used locally = **368 wheat lines**

Phenotyping
Field trials in 2021 and 2022
Experimental design: Incomplete block design (resoluble), 3 reps
Plots: 1 linear meter
Artificial inoculation on susceptible spreaders rows with the two most prevalent races of 2020 (*PstS13*)
Response variable: AUDPC, calculated with 6 evaluations of Infection Type and Severity

GWAS
Model: GLMM
R package GWASpoly

GP
Model: Bayesian GBLUP
R package BGLR

Genotyping
Sequencing: by GBS, University of Wisconsin Biotechnology Center
SNPs calling: TASSEL 5.0
SNPs filtered:
+ 80% missing data and MAF 0.05
SNPs imputation: Beagle 5
Population structure:
- PCA
- Kinship matrix
- Admixture

Correlations between means of AUDPC scores for 2021, 2022 and 2021&2022

RESULTS

### GWAS – AUDPC 2021&2022

Manhattan plot for AUDPC 2021&2022
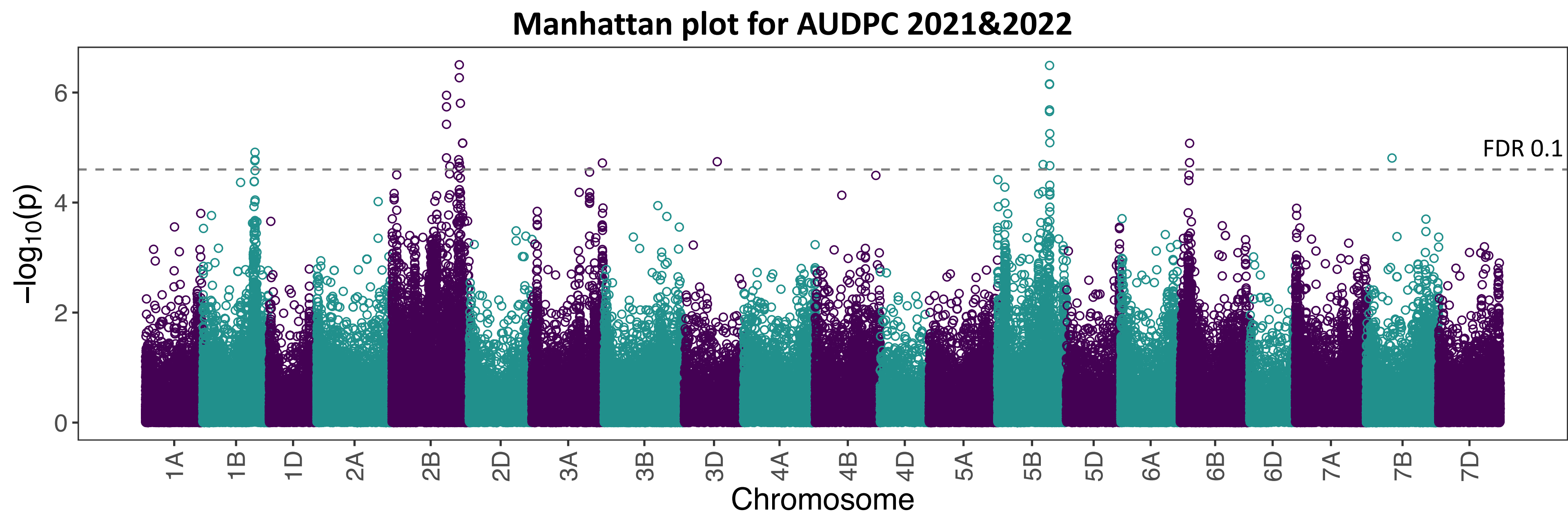
Marker information for SNPs with significant association with AUDPC

Chrom	Position	Marker	pval	Score	Effect	% variance explained
1B	541,787,212	SNP1	0.000897	4.91	355.83	2.95
2B	564,816,352	SNP2	0.000036	5.95	-399.20	4.53
2B	697,387,393	SNP3	0.000007	6.50	-300.57	5.37
3A	547,640,746	SNP4	0.000019	5.01	810.27	4.85
5B	538,482,302	SNP5	0.000002	6.49	430.35	5.91
6B	95,515,580	SNP6	0.000245	5.08	-271.95	3.59
7B	266,136,929	SNP7	0.000222	4.97	-231.44	3.77

Genomic Prediction

Boxplots for correlations between observed and predicted AUDPC values using Bayesian GBLUP, for k-fold cross-validation (k=10)

non-fixed effects | SNP3 and SNP5 as fixed effects | SNP1 to SNP7 as fixed effects
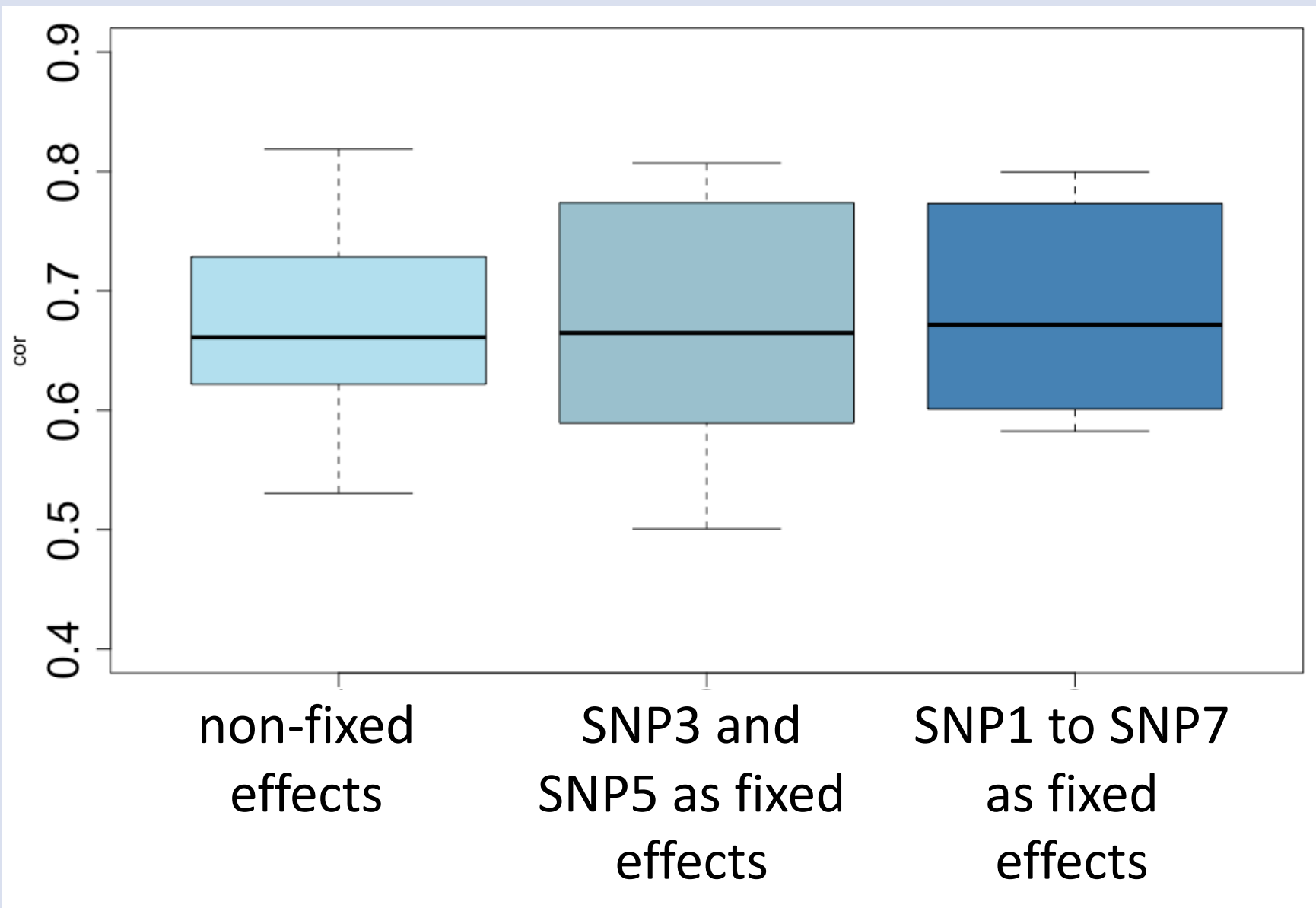
DISCUSSION

- YR phenotyping data showed a strong genetic basis of resistance. A wide variability was observed for AUDPC between genotypes and high correlations between replications (data not shown) and years.

- 7 genomic regions statistically associated with resistance to YR were identified, explaining 32% of the phenotypic variability.

- SNP1 is located close to *Yr29/Lr46* gene. The remaining SNPs could be new QTL.

- We obtained highly accurate predictions for AUDPC (>0.66) generated by Bayesian GBLUP. Adding the SNPs obtained in the GWAS analysis as fixed effects to the GP model (either the 2 QTL with the greatest effect or the total of the 7 QTL) did not lead to an important increase in the predictive ability of the model.

vriella@fagro.edu.uy