

Genetic contributions to tolerance for downy mildew pathogen *Peronospora variabilis* in a South American panel of quinoa

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Introduction

- ▶ *Chenopodium quinoa* is an emerging South American crop with high nutritional quality and tolerance to salinity and drought¹.
- ▶ Downy mildew (*Peronospora variabilis*) is the most common disease of the crop². Resistance to the pathogen has been suggested as polygenic^{3,4}.
- ▶ Identifying markers and QTLs associated with tolerance to mildew via Genome Wide Association Studies (GWAS) is expected to become an important objective for current and future quinoa breeding programs⁵.

Objectives

- ▶ Characterize the phenotypal response to downy mildew from an association panel of quinoa.
- ▶ To unravel the genetic architecture behind resistant and susceptible quinoa genotypes towards downy mildew disease.

I. Phenotypic response to downy mildew

Responses to downy mildew disease were phenotyped from an association panel of 132 quinoa genotypes. *P. variabilis* was isolated from wild species of *Chenopodium album* in Tåstrup, Denmark and inoculated under humid greenhouse conditions. Three experiments, each with four blocks with replicated controls and unreplicated genotypes, were set up. To analyze the data we use the model under the formula: $y_{ijk} = \mu + \rho_i + \beta_{ij} + \alpha_k + (\rho\alpha)_{ik} + e_{ijk}$

Where (ρ_i) represents the effects for the experiments, (β_{ij}) the nested blocks, (α_k) the genotypes and ($\rho\alpha_{ik}$) the Experiment-by-Genotype interaction. All design effects were fitted as random to allow the estimation of variance components and account for the structure of the experimental design (Fig 1a). Additionally, a highly-significant effect of the genotype on the response to *P. variabilis* was found (p-value = 1.18×10^{-18}).

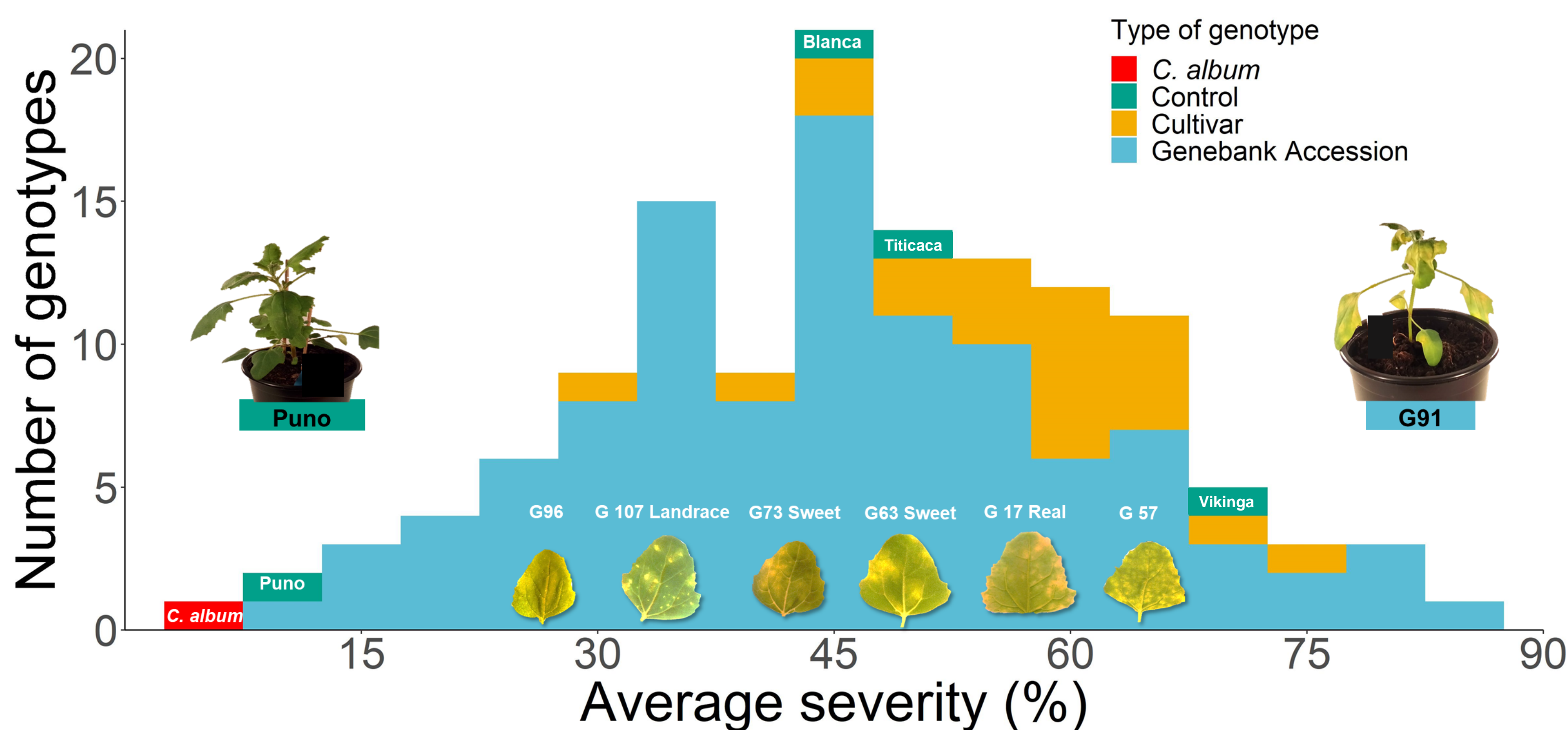


Fig 1a: Distribution of the average severity (%) on leaves of 133 genotypes

- ▶ Large variation in the response to *P. variabilis* was found (Fig 1a)
- ▶ The heritability of the average disease severity was estimated to be ≈ 0.72 indicating that is feasible to select for the trait.

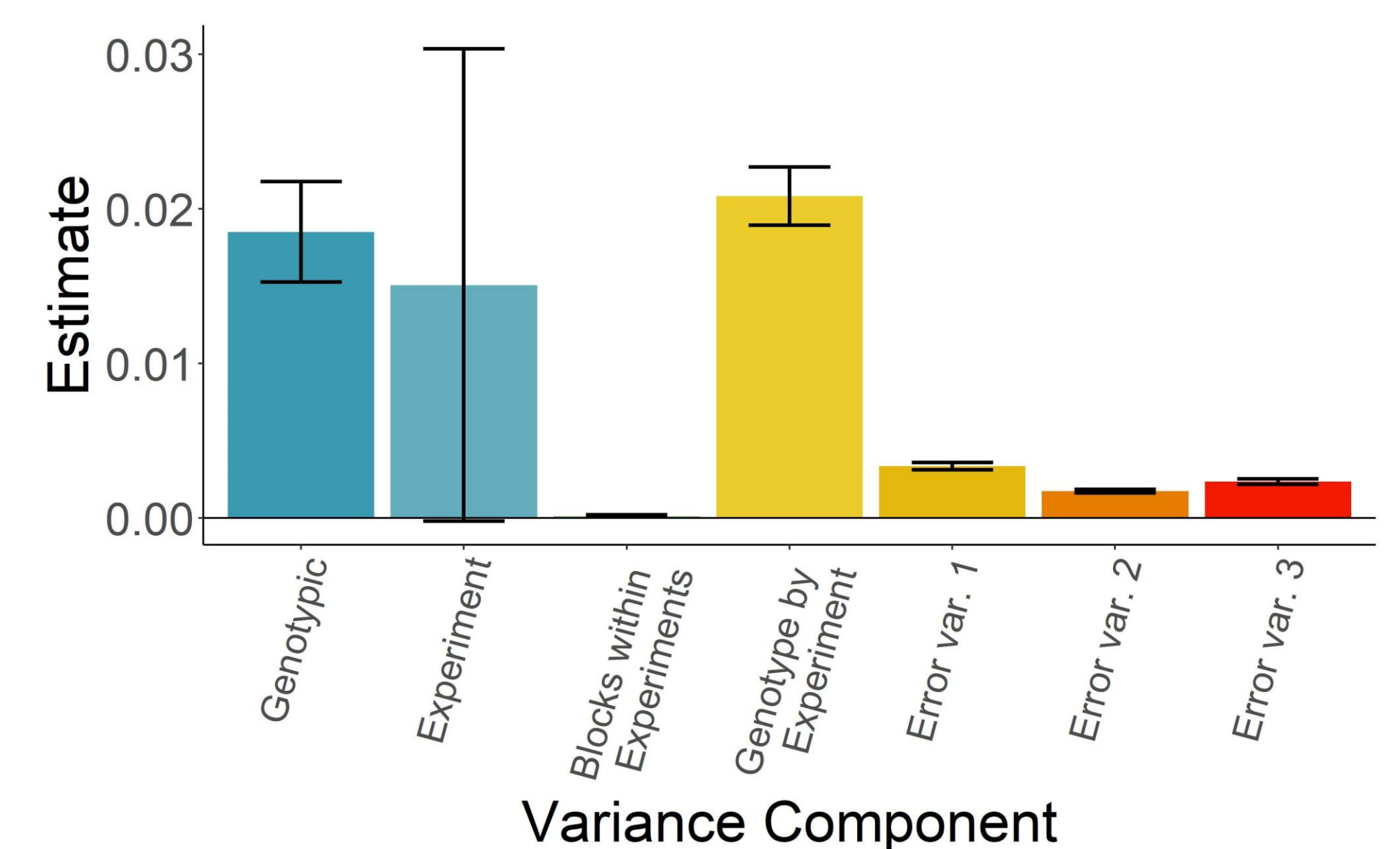


Fig 1b: Variance components and their standard error for the model with all effects fitted as random

- ▶ The largest variance component, was found in the genotype by experiment (GxE) interaction. This could be due to the combination of environmental conditions and the interactions between genotype and pathogen (Fig 1b).

II. Association Mapping

- ▶ From the association panel we have currently conducted GWAS for 61 quinoa accessions using a published reference genome for mapping⁶. Approximately, 600.000 Single nucleotide polymorphisms (SNPs) markers have been identified and used for FarmCPU. Interestingly, one suggestive marker was found on chromosome 4 (Fig 2a).
- ▶ These results show strong effect of minor genes on the interaction quinoa-*P. variabilis* and lack of power due to a small sample size (Figs 2a-b).
- ▶ The p-values from the GWAS show a normal trend with no indication of inflated values (Fig 2b).

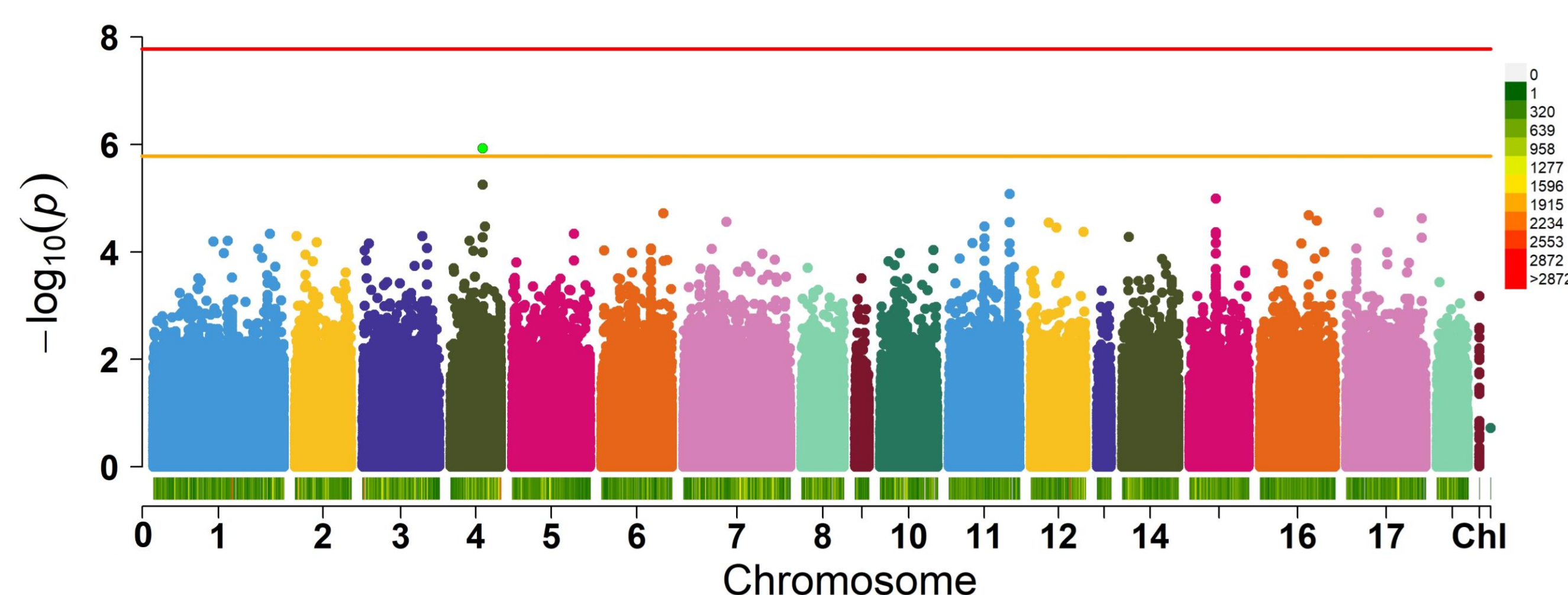


Fig. 2a: GWAS Mapping for downy mildew severity using FarmCPU. Red line denotes the Bonferroni corrected threshold for $p=0.01$; orange line is the suggestive threshold ($1/\text{number of markers}$). Bars at the bottom indicate marker density

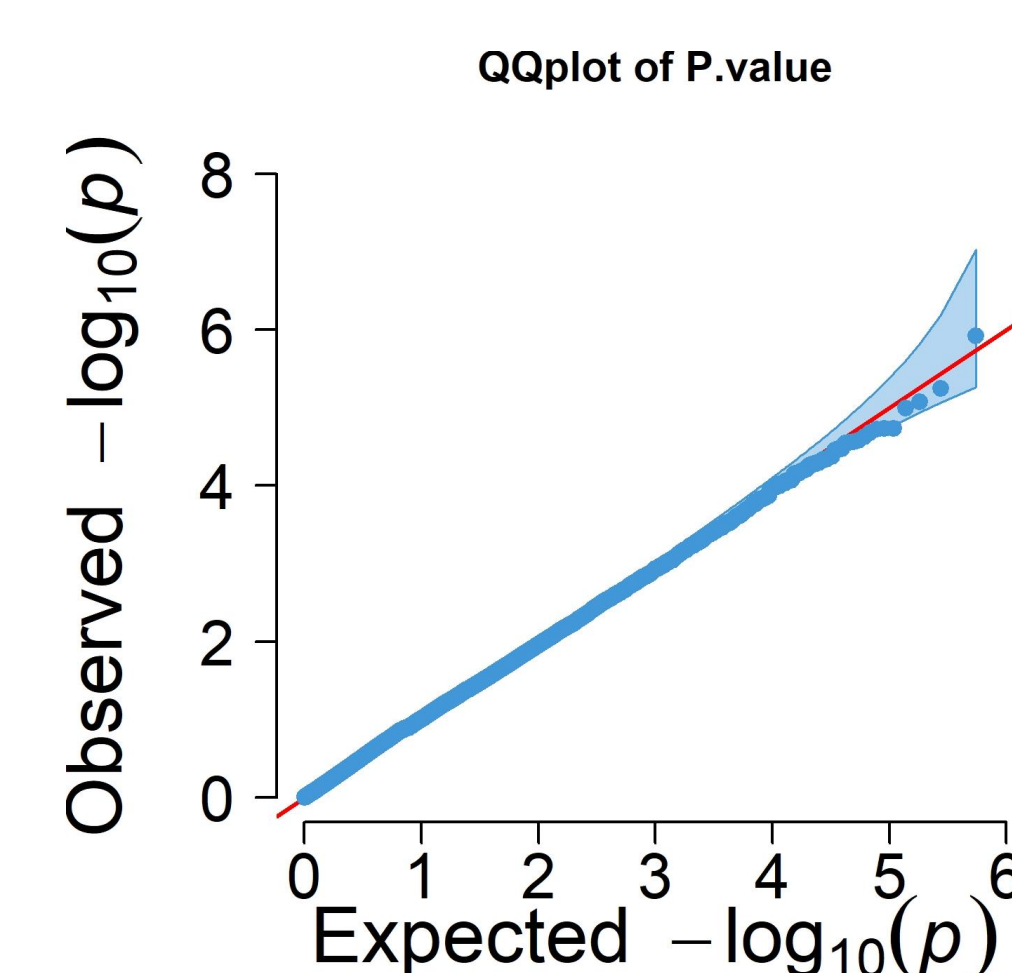


Fig. 2b : QQ-plot for the Farm CPU model. Red line draws expected distribution for p-values

Conclusions and Perspective

- ▶ The data strongly indicates that the resistance to downy mildew has a quantitative nature. It does not, however, rule out the presence of qualitative resistance.
- ▶ Future assessments of resistance to *P. variabilis* should expect large GxE interactions. Therefore, selection of genotypes, experimental designs and breeding strategies should account for it.
- ▶ Minor mildew symptoms were observed in few plants from the *C. album* complex used in this study. Thus, this weed could potentially become an alternative and overwintering host for downy mildew, affecting resistance breeding efforts in quinoa.

References

¹Zurita-Silva et al. 2014. Mol Breeding 34(1):13–30. ²Danielsen and Munk. 2004. Crop Protection 23(3):219–228. ³Kitz et al. 2009. Phytopathology 99:S184. ⁴Kumar et al. 2006. Crop Prot 25:879–889. ⁵Maughan et al. 2004. Theor Appl Genet 109:1188–1195. ⁶Jarvis et al. 2017. Nature 542:307–312.