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Genetic architecture of robustness in *Capsicum annuum* for resistance to *Phytophthora capsici* and Potato virus Y under a temperature stress

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BACKGROUND Current climate has an impact on the expression of plant diseases and therefore their management [1]. The plant-pathogen interaction resulting from an infection can be difficult to predict when one or several parameters of the environment are changed. For example, increased mean temperature was reported to inhibit the expression of resistance to pathogens. Usually, the higher the temperature, the higher the virus multiplication. However, the opposite can also be observed: Tobacco rattle virus multiplies faster at 18-22°C than at 26°C [1]. The pathogen multiplication rate is also linked to the host behaviour, which depends on its genetics. This leads to the definition of robustness, sometimes called canalization: the absence or low variation of a phenotypic trait faced to a specific environmental change [2]. Our goal is to identify Quantitative Trait Loci (QTL) and genes involved in the robustness of pepper resistance to *Phytophthora capsici* and Potato virus Y (PVY).

MATERIALS & METHODS A collection of 176 pepper (*Capsicum annuum*) accessions was phenotyped for resistance to *P. capsici* under two temperatures, 22/24°C or 28/30°C (the lowest being the night temperature) [3]. The same accessions were inoculated with PVY in two conditions (20°C or 28°C) and relative concentration of virus in apical leaves was measured with Enzyme Linked ImmunoSorbent Assay (ELISA). The collection was genotyped for 10,308 Single Nucleotide Polymorphisms (SNPs). Robustness was calculated as the inverse of the difference of resistance mean between both environmental conditions. Best Linear Unbiased Predictor (BLUP) values of robustness were used for Genome Wide Association Studies (GWAS).

RESULTS The temperature regime and the accession have an effect on the mean resistance level (Fig. 1). For both *P. capsici* and PVY, different responses were identified between the two conditions. Mortality at 30 dpi was more frequent at 20°C than at 28°C for PVY. Further analyses will underline the genetic part involved in robustness. QTL determining resistance for each pathogen at each temperature regime and robustness of resistance to these pathogens under thermic variation will be looked for with a GWAS approach. Then, a search into *C. annuum* reference genomes will enable us to identify robustness genes.

DISCUSSION & CONCLUSION Thanks to this project, we expect to identify different QTL of robustness for each pathogen. A larger temperature spectrum will allow the determination of reaction norms for each accession across multiple environments. A precise characterization of the response could help the breeding for changing environments. Robustness for different stress (flooding, drought, and other pathogens) and combination of stresses will also be assessed.

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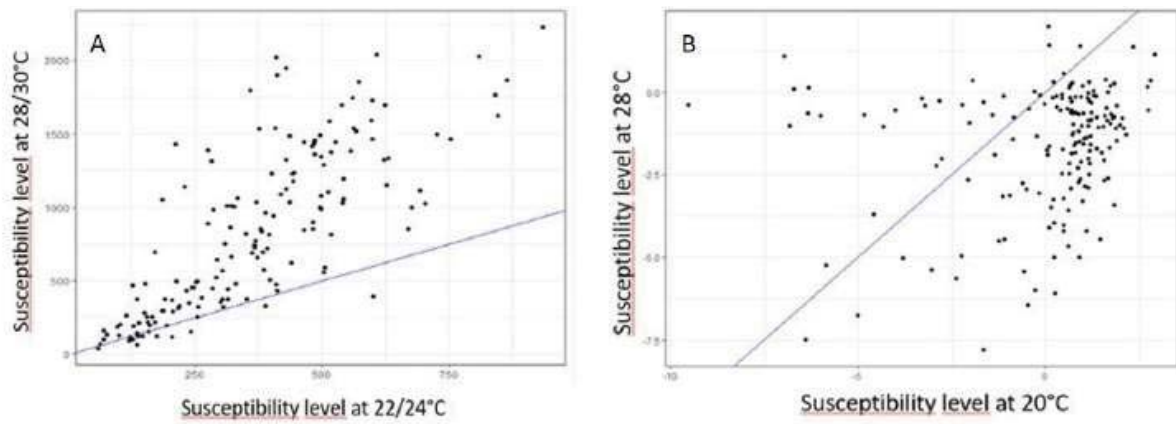


Figure 1: Plot of the susceptibility levels of the 176 accessions in both environments. The blue line corresponds to the first bisector *i.e.* the response is similar in both environments. A) For *P. capsici*, susceptibility level corresponds to the AUDPC index. B) For PVY, susceptibility level corresponds to the log transformation of virus concentration.



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