The first clues on epigenetic modulation on grapevine - *P. viticola* interaction

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State of art

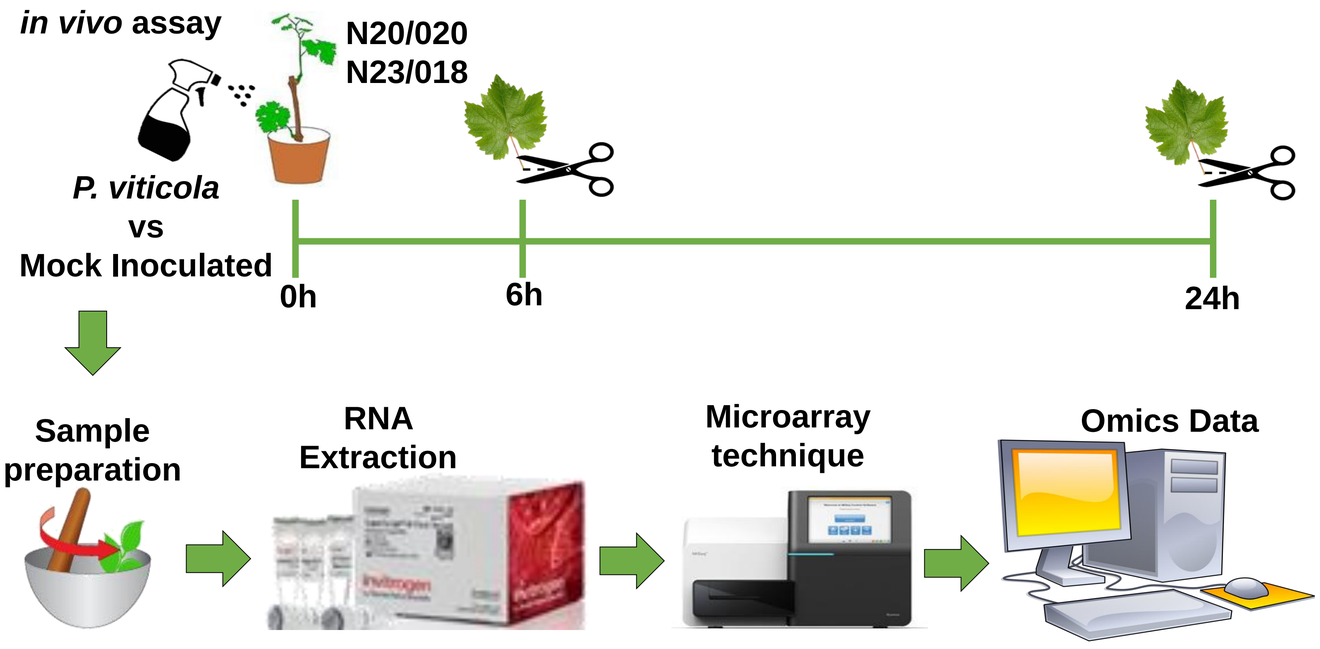
Plants plasticity, adaptation and survival against biotic stresses rely on the plant defense response[1,2]. Epigenetic machinery impacts the gene expression through the influence of DNA, histones, chromatin and RNA modifications[1,2]. Currently, little is known on the epigenetic-plant defense systems relationship[1] and the effect on grapevine-*Plasmopara viticola* interaction.

P. viticola is the causal agent of downy mildew, one of the most important diseases worldwide. Presently, viticulture is concerned with the increase pathogen re-occurrences per year as well as the unhealthy chemical application on crops to avoid harvest loses[3]. Thus, new disease control approaches have been perused and are crucial to deepen the knowledge on this pathosystem evolution.

Aim

Identification of the main epigenetic associated genes that are differentially expressed after *P. viticola* inoculation

Methods



Results

Differentially Expressed Genes (DEGs) → **Defense Related Genes Differentially Expressed (DR-DEGs)** → **Epigenetic Related Genes Differentially Expressed (ER-DEGs)**

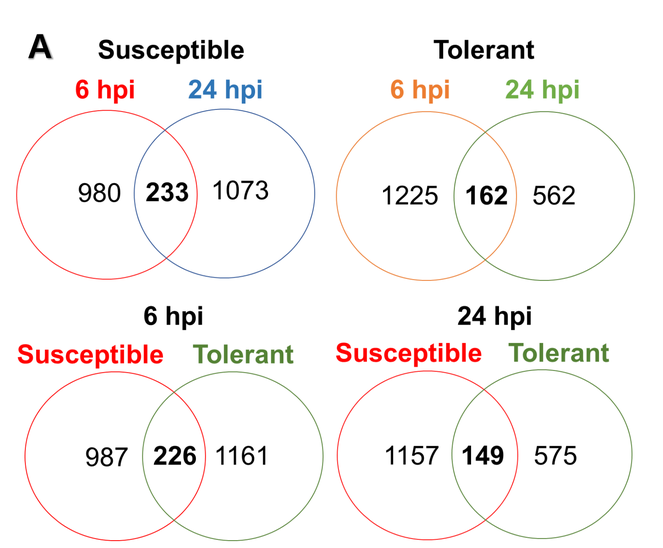


Figure 1. Identification of DEGs after *P. viticola* inoculation. Venn diagrams (A), and Principal component analysis (B) of differentially expressed genes obtained from the comparison between inoculated with control (mock) samples after the FDR cut-off<0,20 at different hours post inoculation (hpi) in N20/020 (S - susceptible) and N23/018 (T – tolerant) genotypes.

- **N20/020** (S) showed a more **delayed response** to *P. viticola* while **N23/018** (T) a **quicker response**;
- The Two genotypes are clustering by the period of infection;

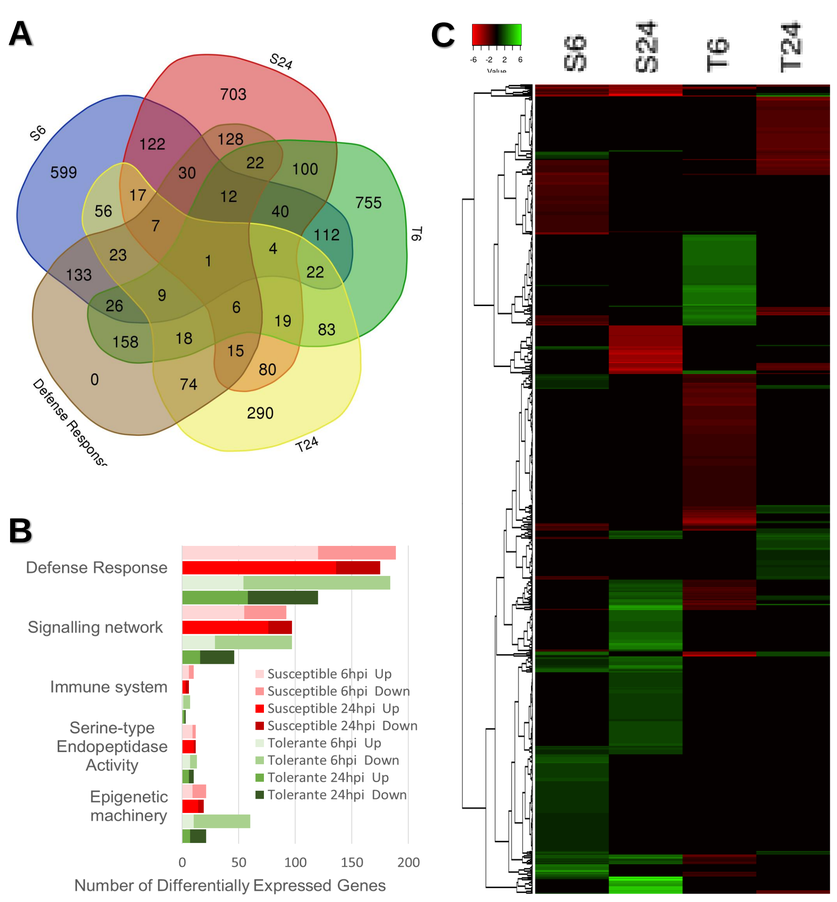


Figure 2. Defense response gene modulation after *P. viticola* inoculation. A Venn diagram shows DR-DEGs distribution at different hours post inoculation (hpi) in susceptible (S) and tolerant (T) genotypes. A histogram of the more representative defense pathways was obtained to show the different gene modulation at the several timepoints after pathogen colonization (B). An analysis of the microarray expression profile (log2FC) at 6 and 24hpi of an both genotypes, was observe through a heatmap (C).

- The **defense system** of grapevines against *P. viticola*, was more expressed on **N23/018** (T) at an **early stage** of infection, **contrarily to N20/020** (S);
- The **susceptible** (S) genotype presents a modulated **signalling and defense related pathways**;
- Interestingly, **somatic recombination mechanism** is affected on the **N23/018** (T) cultivar at an **early stage of infection**;

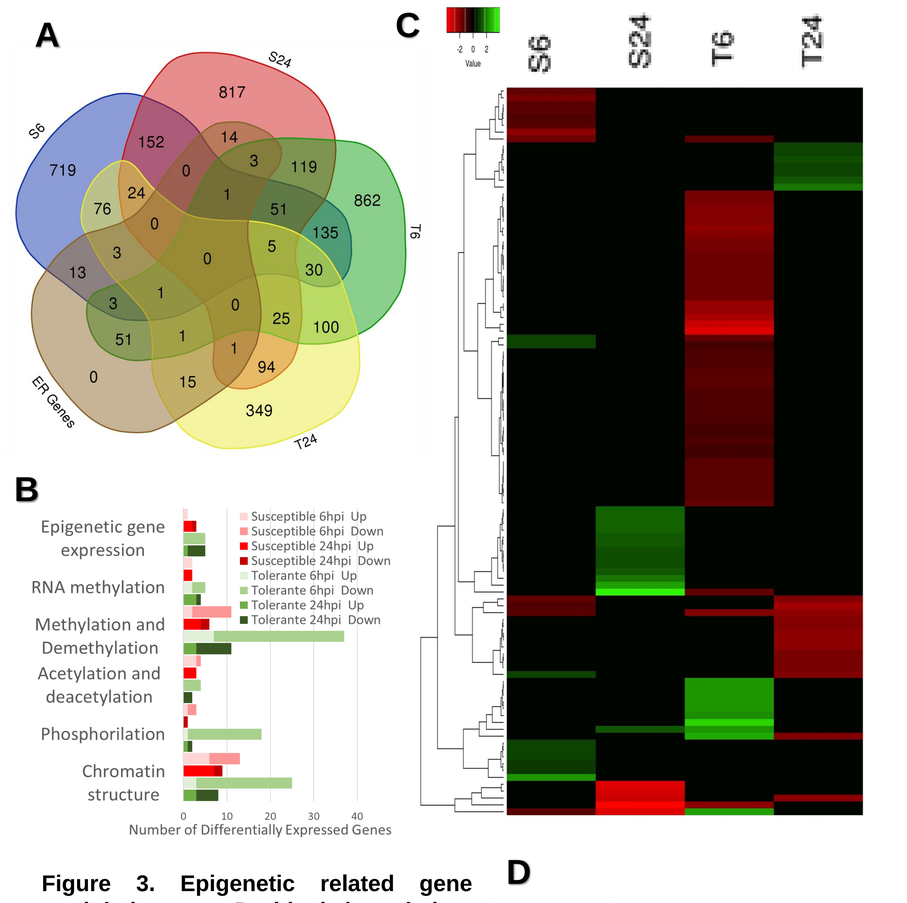


Figure 3. Epigenetic related gene modulation once *P. viticola* inoculation. The total number of GO epigenetic related (ER-) DEGs were cross-compared at different hours post inoculation (hpi) in both genotypes (A). The ER-DEG's were grouped by the epigenetic representative terms to observe the gene modulation on both varieties and timepoints (B). An analysis of the microarray expression profile (log2FC) at both timepoints and genotypes was observed by a heatmap (C). Also, the ER- and DR-DEGS were compared to observe the gene similarity (D).

- Genes encoding **Histone, DNA and Chromatin modification** are affected by *P. viticola* infection specially at the tolerant variety;
- **Chromatin remodelers** are modulated on the **ER-DEGs dataset** as well as the **DR-DEGs dataset**;
- **~25% of the ER-DEGs** also have **defense related functions**;

Take home message

Our results suggest that the epigenetic machinery might be part of tthe grapevine-*P. viticola* interaction through chromatin remodelers possible role on plant defense.

References: [1]Alonso, C. *et al.* The role of plant epigenetics in biotic interactions (2019) 221: 731–737; [2]Espinas, N.A., Saze, H. and Saijo, Y. Epigenetic Control of Defense Signaling and Priming in Plants (2016) 7:1201; [3]Brilli M et al. A multiomics study of the grapevine-downy mildew (*Plasmopara viticola*) pathosystem unveils a complex protein coding and noncoding-based arms race during infection. (2018) 8:757

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