Characterization of hub genes involved in sweet cherry fruit cracking

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Sweet cherry fruit cracking is a complex physiological disorder that causes significant economic losses. Despite many years of research there is a lack of understanding of the mechanisms involved in sweet cherry skin cracking. Here, skin and flesh tissue from the cracking susceptible cultivar 'Early Bigi' and the cracking tolerant cultivar 'Regina' were sampled prior and just after water dipping treatment to identify water-affected metabolic networks that are putatively involved in fruit cracking as illustrated in **Figure 1.**

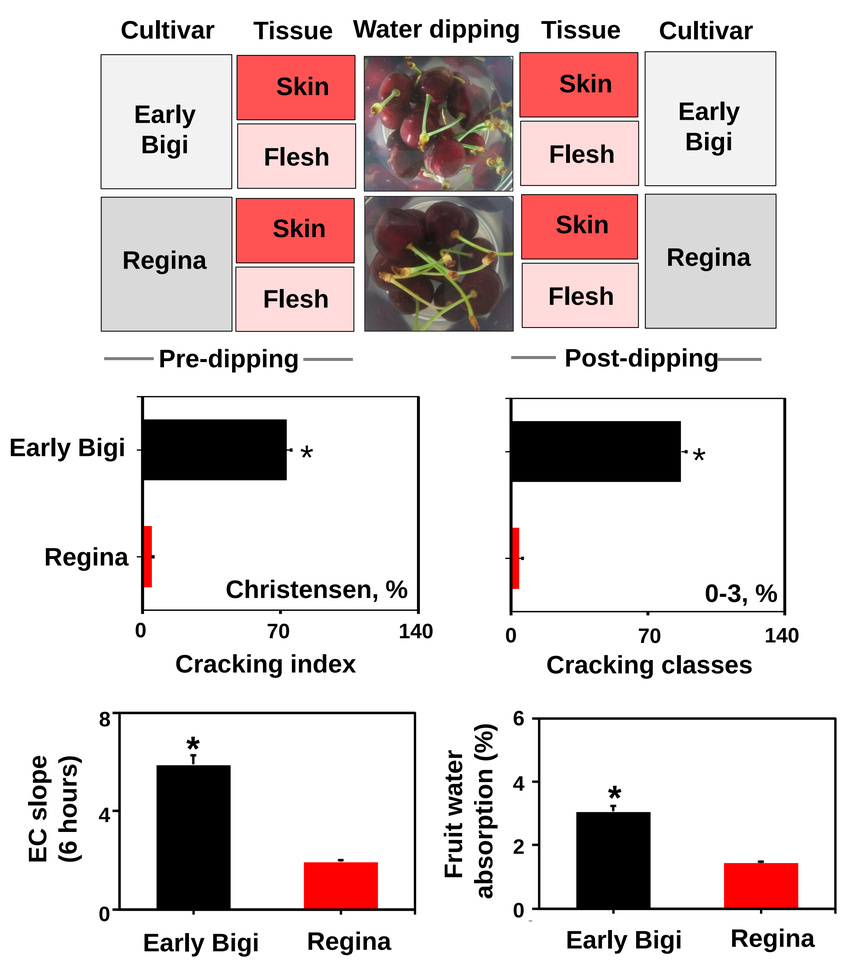


Figure 1. A schematic presentation of the sampling process. Cracking parameters such as cracking index, and cracking classes, electrical conductivity and fruit water absorption.

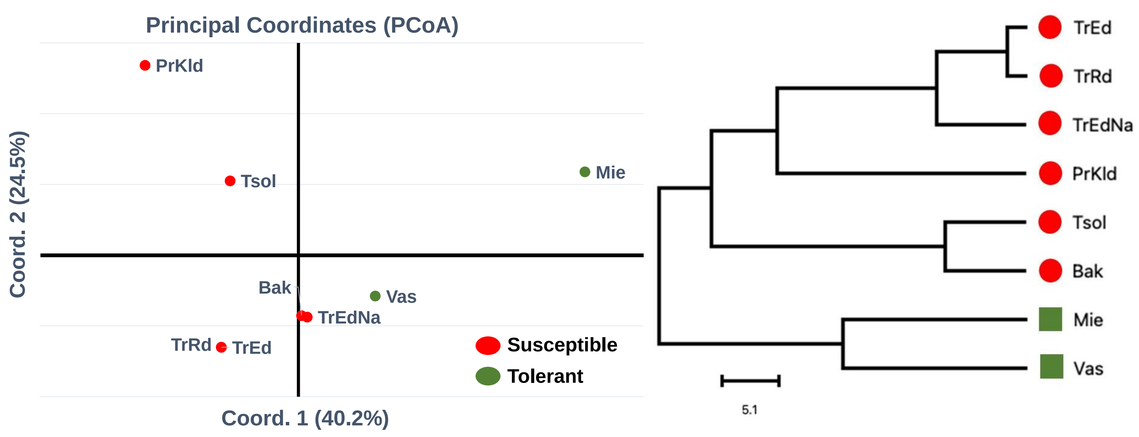
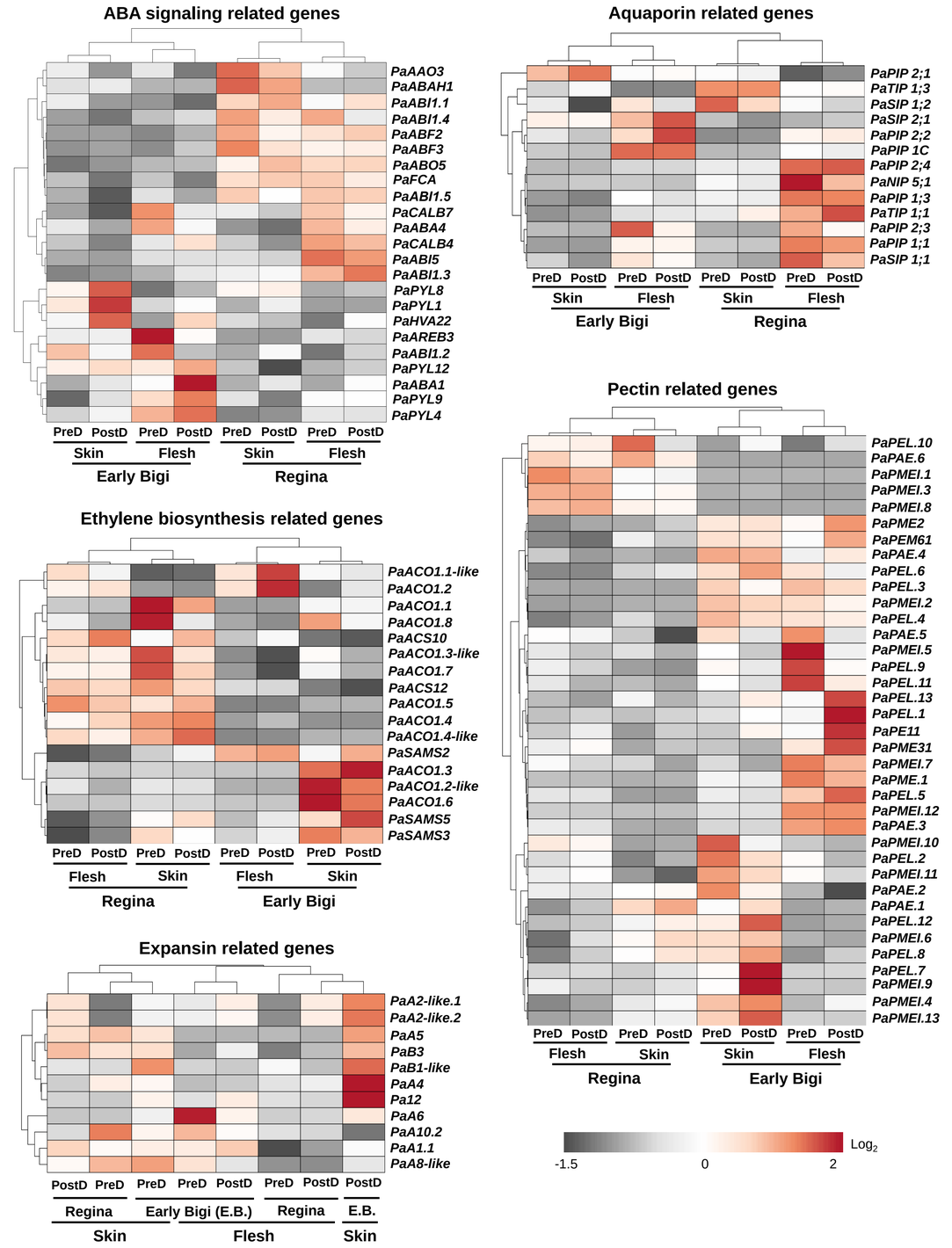


Figure 3. Identification and analysis of Single Nucleotide Variants (SNVs) located in the genomic sequence of pectin related hub-genes of eight sweet cherry cultivars.

Primary metabolites that are mainly involved in sugars and amino acid metabolisms such as glucose and asparagine are shifted in 'Early Bigi' compared with 'Regina' tissues following water exposure. Comparisons between cultivars, tissues and dipping identify significant differentially expressed genes. Particularly, genes related to abscisic acid, ethylene biosynthesis, pectin metabolism, expansins and aquaporins were altered in water-exposed tissues (**Fig. 2**). To further characterize the role of these genes in cracking, their single nucleotide variants of the coding regions was studied in another eight sweet cherry cultivars, which differ in their sensitivity to cracking, revealing a strong link mainly between pectin metabolism-related genes and cracking-phenotypes (**Fig. 3**). Integrated metabolomic and transcriptomic profiling uncovered genotypic- and tissue-specific metabolic pathways, including tricarboxylic acid cycle, cell enlargement, lipid and ethanol biosynthesis, and plant defense that putatively are involved in fruit cracking. Based on these results, a model which describes the skin and flesh metabolic reprogramming during water-induced fruit cracking in the susceptible 'Early Bigi' cultivar is presented. This study can help to explore novel candidate genes and metabolic pathways for cracking tolerance in sweet cherries (**Fig. 4**).

Figure 2. Specific categories of sweet cherry genes that affected by water exposure. Heat diagrams showing the temporal expression pattern in selected genes associated with ABA, ethylene, expansins, pectin and aquaporins signaling/metabolism/function in skin and flesh tissues of 'Regina' and 'Early Bigi' prior and after water dipping.



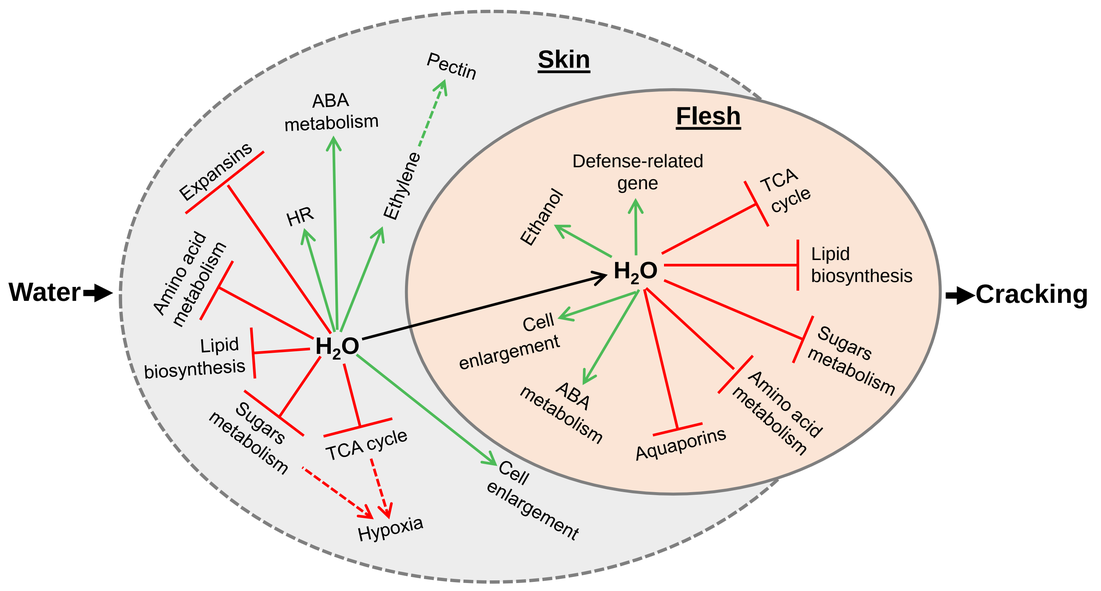


Figure 4. A putative genotype-specific model of water-induced key downstream metabolic events in skin and flesh 'Early Bigi' tissues eventually leading fruit cracking.

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