Molecular and biochemical differences underlying the efficacy of lovastatin in preventing the onset of superficial scald in a susceptible and resistant *Pyrus communis* L. cultivar

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Abstract

The molecular and biochemical events underlying the onset of superficial scald in two pear cultivars with different susceptibility ('Blanquilla' and 'Conference') was investigated in fruit untreated and treated with lovastatin, 1-MCP or ethylene. 'Conference' pears were characterized by higher content of flavonols and linolenic acid (18:3), two metabolites related to chilling injury resistance. In this cultivar, the expression level of three genes belonging to the ascorbate glutathione pathway (*APX*, *DHAR* and *MDHAR*) were constitutively over-expressed, highlighting the role that endogenous antioxidant potential played in scald control. In the scald-susceptible cultivar ('Blanquilla') the lovastatin treatment, in contrast to 1-MCP, effectively prevented superficial scald development and α-farnesene production without affecting fruit ripening. Moreover, lovastatin stimulated an increased production of ethanol and oleic + cis vaccenic acid (18:1), both compounds being also involved in cold stress tolerance. In both cultivars, and in contrast to 1-MCP, lovastatin did not impair the expression level of the genes devoted to ethylene production (*ACO*, *ACS*) and perception (*ERS1*, *ERS2*). As a consequence, the expression levels of the genes involved in texture modifications (*PG1*) and volatile emission (*LOX*, *HPL*, *ADH* and *AAT*) were maintained in lovastatin-treated samples allowing the fruit to reach an adequate final quality.

The results from this study are discussed to highlight the complex regulatory network underlying superficial scald development in different pear cultivars.