Genetic and biochemical bases of superficial scald storage disorder in apple and pear fruits

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Abstract

Superficial scald is a physiological storage disorder affecting apple and pear fruits. The disorder develops during cold storage and intensifies after removal to market temperatures. Scald symptoms result from necrosis of a few hypodermal cell layers and manifest as brown or black patches on the fruit skin. Susceptibility to scald varies greatly by cultivar, and is also influenced by preharvest factors such as growing climate and maturity, and postharvest factors including storage atmosphere, ventilation, and temperature. Despite many years of investigation, the biochemical mechanisms underlying scald are still in question. The disorder is thought to be a type of chilling injury induced by oxidative stress. A long-standing hypothesis holds that oxidation products of the sesquiterpene α-farnesene are directly involved via generation of free radicals. Variation in the antioxidant defense mechanisms required to scavenge radicals and combat oxidative stress is postulated to also play a key role in susceptibility or resistance to scald. A marked rise in α-farnesene synthesis typically occurs shortly after scald-susceptible fruit are placed in storage and oxidation of the α-farnesene to conjugated trienols (CTols) proceeds rapidly after about 6-8 weeks, particularly in air storage. High level accumulation of CTols during storage is usually correlated with the incidence and severity of scald. Further evidence supporting a link between α-farnesene oxidation and induction of scald was the finding that prestorage treatment of apples or pears with 1-methylcyclopropene (1-MCP), a blocker of ethylene action, drastically reduced α-farnesene synthesis and scald development. Silencing genes that control α-farnesene biosynthesis or conversion to CTols should prove or disprove a major role of α-farnesene oxidation in the induction of scald. The primary target for gene knockout is *AFS1* encoding α-farnesene synthase, which catalyzes the final step in α-farnesene biosynthesis. Enzymatic production of CTols is as yet hypothetical, but possibly a glutathione peroxidase or

glutathione S-transferase catalyzes reduction of farnesyl hydroperoxides to the corresponding alcohols. In addition, the precise mechanisms whereby 1-MCP, diphenylamine, UV-visible light irradiation, initial low oxygen stress, and intermittent warming ameliorate or prevent scald are currently being investigated using biochemical, functional genomics, and metabolomics approaches.