

**Title** Wound-induced H<sub>2</sub>O<sub>2</sub> and resistance to *Botrytis cinerea* decline with the ripening of apple fruit

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**Citation** Postharvest Biology and Technology, Volume 62, Issue 1, October 2011, Pages 64-70

**Keywords** Apple; Disease resistance; Fruit ripening; Hydrogen peroxide; Wound healing

### **Abstract**

Fruit ripening is a developmental process and is associated with increased susceptibility to mechanical injury, which favours *Botrytis cinerea* infection. Using 'Gala' apples harvested at different stages of ripening, we demonstrated that wounding can activate initial H<sub>2</sub>O<sub>2</sub> accumulation and wound healing ability to defend against *B. cinerea* penetration. Delaying the harvest date attenuated those responses. Superoxide dismutase, peroxidase and catalase, which are all involved in H<sub>2</sub>O<sub>2</sub> metabolism, were differentially activated by wound stress depending on the stage of fruit maturity. Mature fruit were less able to respond to wounding by increasing phenylalanine ammonia lyase and peroxidase activity, which are associated with reduced phenolics and lignin content in local wound sites. The reduced response in late-harvested fruit contributes to the fruit ripening-induced loss of wound healing ability and increases susceptibility to *B. cinerea*. In addition, the rapid increase of H<sub>2</sub>O<sub>2</sub> content immediately after wounding in early-harvested fruit was followed by increased phenylalanine ammonia lyase and peroxidase activity. In late-harvested fruit, the reduced ability to increase phenylalanine ammonia lyase and peroxidase activity in response to wounding was consistent with ripening-reduced generation of H<sub>2</sub>O<sub>2</sub> early after wounding, leading to reduced resistance to *B. cinerea*. Thus, H<sub>2</sub>O<sub>2</sub> accumulation in response to wounding is modulated by fruit maturity and is required for efficient wound healing and resistance to *B. cinerea*.