Title Understanding the mechanisms involved in calcium deficiency disorders in tomato and apple fruit

Author Sergio de Freitas, Elizabeth J. Mitcham, Cai-Zhong Jiang and Eduardo Blumwald

Citation Thesis, Doctor of Philosophy (Plant Biology), University of California. 2010.

Keywords Apoplast; Calcium deficiency disorders; Cell wall; Tomato; Plasma membrane; Vacuole; Apple fruit

Abstract

Calcium (Ca) deficiency disorders in fruit can be triggered by low Ca concentrations, but often fruit showing Ca deficiency symptoms have higher Ca concentrations than sound fruit. Perhaps Ca deficiency disorders are not only caused by low tissue Ca concentration, but also by abnormal regulation of cellular Ca partitioning and distribution. The objectives of this work were to better understand (1) fruit Ca uptake in response to the plant growth regulators abscisic acid and gibberellins, as well as (2) the effect of these growth regulators, Ca binding to the cell wall, and Ca movement into storage organelles on cellular Ca partitioning and distribution and fruit susceptibility to Ca deficiency disorders. In this study, tomato was used as a model system to understand Ca deficiency disorders, and the work was extended to apple fruit. Treating tomato plants with ABA prevented BER development by increasing the number of functional xylem vessels in the fruit and the xylem/phloem ratio of fruit water uptake, which increased fruit Ca uptake. Treating tomato plants with a gibberellin biosynthesis inhibitor, prohexadione-calcium, also increased the number of functional xylem vessels, Ca uptake, and suppressed BER development in the fruit. High expression of *sCAX1* increased total fruit tissue and vacuolar Ca content, but reduced water soluble apoplastic and cytosolic Ca contents, and increased fruit susceptibility to BER. Moreover, treating tomato plants with gibberellins did not change the total fruit tissue Ca concentration, but increased the expression of organellar Ca^{2+}/H^{+} *exchangers* and *Ca-ATPases,* reduced water soluble apoplastic Ca, and increased fruit susceptibility to BER. Silencing pectin methylesterases in tomato did not change total fruit tissue Ca content, but reduced cell wall bound Ca, increased water soluble apoplastic Ca, and reduced fruit susceptibility to BER. The data also show that high Ca accumulation inside storage organelles and Ca binding to the cell wall are also potential mechanisms involved in development of the Ca deficiency disorder, bitter pit, in apple fruit. Our results suggest that fruit susceptibility to Ca deficiency disorders is determined by mechanisms that control fruit Ca uptake, as well as cellular Ca partitioning and distribution.