The Effect of Calcium Uptake, Translocation, and Distribution on Blossom-end Rot in Tomato Fruit

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Abstract

Blossom-end rot in tomato is recognized as a physiological disorder symptom caused by apoplastic Ca^{2+} deficiency in part of fruit tissue and usually happens during the rapid growth stage of fruit. Calcium imported into fruit is determined by the combination of the root absorption from soil solution, the partitioning to fruit, the translocation and allocation within fruit, and the distribution at cell level. The amount of calcium uptake increased as root temperature rose to 26℃, but decreased as the nutrient solution salinity increased from 2 $dS \cdot m^{-1}$ to 17 $dS \cdot m^{-1}$. Tomato plants grown in high salinity nutrient solution had lower xylem vessel density in stalk, pericarp and placenta, and higher blossom-end rot incidence than those in low salinity. High relative humidity and ABA spray resulted in reduced leaf transpiration rate, and increased calcium accumulation in fruit. However, GA spray upregulated CAX (vascular Ca^{2+}/H^{+} exchangers) and Ca-ATPase expression in tomato fruit, and enhance calcium transport to storage organelles, reduced apoplastic calcium concentration, and increased ion leakage and blossom-end rot incidence.

Key words: abscisic acid; gibberellin; leaf transpiration; xylem vessel

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