Title When calcium is not the primary cause of calcium deficiency

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Abstract

Calcium deficiency disorders affect quality and yield of many crop species. The first known event leading to calcium deficiency symptoms is plasma membrane breakdown, which results in cell dealth. Calcium deficiency is caused by a low concentration calcium ions; however, calcium deficiency cannot always be explained by total tissue calcium content. Often, fruit showing calcium deficiency symptoms have higher levels of calcium than sound fruit grown under the same conditions. It is relevant to consider that total calcium content in the tissue is important to reduce the risk of calcium deficiency development, but more important is the localization of calcium in the cells. Experiments in our laboratory have shown that tomato plants sprayed weekly with gibberellins (GA4+7) showed a steady increase in calcium deficiency disorder; blossom end rot (HER) developed to 90% by 45 days after pollination. The control plants that were sprayed weekly with water increased in BER incidence up to 30% within 31 days after pollination, but no further BER developed after that time. Fruit from both treatments had the same level total calcium in the fruit, but different degrees of membrane permeability and BER incidence. Gibberellin levels increase tomato fruit during cell division and expansion, decreasing thereafter. This internal increase in gibberellin concentration is consistent with the time of BER development in the control fruit. The presence of gibberellin during cell division and expansion has been proposed as one of the main factors in calcium deficiency development in plants. However, the mechanism involved not well understood. Further analysis of the gibberellin and water treated fruit 31 days after pollination revealed that the expression of three genes thought to encode organellar Ca++/H+ transporters (CAX3, CAX4, and CAX7) had increased significantly with gibberellin treatment. It is possible that high levels of gibberellins increase the expression of Ca++/H+ transporters resulting in more calcium being sequestered inside storage organelles, like the vauole. This can potentially decrease the amount of free calcium present in the apoplastic solution, reducing calcium bound to the plasma membrane and the integrity of membrane structure, eventually leading to cell collapse and death. These results may explain most of the 'contradictory' data in which tissue with higher levels of calcium show higher levels of calcium deficiency.