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Perspective

Calcium Chloride Treatment Decreases Postharvest Blueberry Softening *Via* Abscisic Acid Pathway Modulation

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DESCRIPTION

Blueberries (*Vaccinium* spp.) have emerged as a globally popular berry crop, appreciated for their unique balance of sweetness and tangy acidity, coupled with their rich nutritional profile. These berries are particularly celebrated for their high anthocyanin content and other bioactive compounds that confer health benefits. While blueberries are primarily marketed in their fresh form, they are subject to significant postharvest challenges, largely stemming from their inherent botanical characteristics. One major issue is the rapid softening of the fruit during storage due to the concentrated harvest period, which exacerbates this problem. Consequently, addressing the issue of postharvest softening has become a critical focus for the industry, as it directly affects both economic viability and consumer satisfaction.

In recent years, advancements in preservation techniques have been developed to mitigate postharvest softening, with calcium chloride ($CaCl_2$) treatment emerging as a potential approach. This method is recognized for being environmentally friendly, cost-effective and sustainable. Calcium treatments have been extensively studied and applied to a variety of fruits and vegetables, including apricots and papayas, yielding significant preservation benefits. Calcium ions (Ca^{2+}) play a vital role in maintaining structural integrity in plant cell walls, ensuring proper membrane functionality and participating in cellular signal transduction. These properties make calcium a critical macronutrient for preserving freshness, quality and extending the shelf life of fruit and vegetable products.

Research has established that Ca^2 serves as a central hub within the difficult signaling networks of plants, integrating inputs from developmental signals and environmental signals. However, decoding and transducing these signals rely on specialized calcium sensors, such as Calmodulin (CaM), Calmodulin-Like Proteins (CMLs), Calcineurin B-Like Proteins (CBLs) and their associated kinases, such as Calcium-Dependent Protein Kinases (CDPKs). Calcium's influence on fruit ripening is well-documented, with notable effects on cell wall modifications and the senescence process. As soluble calcium levels in fruit decrease, the structural integrity of cell walls is compromised, triggering physiological and metabolic disorders that culminate in fruit softening and deterioration.

In addition to calcium, the plant hormone Abscisic Acid (ABA) plays an important role in fruit development and ripening. ABA is involved in regulating a wide range of plant growth and developmental processes, including interactions with ethylene during fruit maturation. For non-climacteric fruits such as citrus and cherries, ABA influences sugar accumulation and starch hydrolysis, which are key processes in fruit ripening. The ABA signal transduction pathway is regulated by key components, including ABA receptors Pyrabactin Resistance/Pyrabactin Resistance-Like (PYR/PYL), Type 2c Protein Phosphatases (PP2Cs) and Sucrose Non-Fermenting 1-Related Protein Kinases 2 (SnRK2). These components mediate ABA's effects on fruit physiology, often in concert with calcium signaling.

Research suggests that calcium and ABA signaling pathways may have a synergistic relationship during fruit maturation and ripening. For instance, ABA-responsive elements have been shown to significantly influence the expression of Ca^{2+}-related genes, indicating a regulatory interplay between the two signaling systems. Additionally, calcium and ABA signaling share downstream target proteins, modulating their activity through protein phosphorylation. These shared targets underscore the interconnected roles of calcium and ABA in maintaining fruit quality, structural integrity and proper physiological function during the ripening process.

Despite the promising results of $CaCl_2$ treatments on postharvest quality in various fruits, the specific mechanisms underlying calcium and ABA signaling in the softening process of blueberries remain incompletely understood. Preliminary studies suggest that calcium treatments can mitigate softening in blueberries, but the precise molecular pathways and interactions involved warrant further investigation.

CONCLUSION

This study aims to eliminate the network regulatory mechanisms of $CaCl_2$ treatment in mitigating blueberry fruit softening, using an integrated approach combining physiological and molecular biology techniques. By elucidating the interaction between

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calcium and ABA signaling, this research seeks to provide a deeper understanding of the processes driving fruit softening and to identify effective methods for postharvest storage. The findings are expected to contribute valuable theoretical insights into blueberry storage, helping to enhance the economic sustainability and quality of this widely consumed fruit. Transcriptomic and proteomic analyses have begun to shed light on these pathways, revealing how calcium and ABA signals regulate gene expression and protein activity during fruit softening.