CaM enhances chilling tolerance of peach fruit by regulating energy and GABA metabolism

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Abstract

The effects of calcium chloride ($CaCl_2$), calmodulin antagonist trifluoperazine (TFP) and different storage temperatures on chilling injury (CI) of peach fruit during postharvest storage were studied. The results showed that 0 °C storage and $CaCl_2$ treatment reduced the CI index, reduced the ion leakage and maintained the integrity of cell membrane. Storage at 0 °C and $CaCl_2$ treatment increased calmodulin (CaM) expression and protein content, energy charge (EC), adenosine triphosphate (ATP) content and adenosine diphosphate (ADP) content. At the same time, it also promoted the expression and activity of Ca^{2+}-ATPase and other enzymes related to energy metabolism. In addition, storage at 0 °C and $CaCl_2$ treatment promoted the expression and activity of glutamic acid decarboxylase (GAD), and increased the content of γ-aminobutyric acid (GABA) and the expression of GABA transaminase (GABA-T). On the contrary, TFP treatment significantly increased CI index and ion leakage, significantly inhibited *PpCaM* expression and protein content, and decreased EC and GABA content. Moreover, the molecular properties of PpCaM were studied. The results showed that PpCaM was a highly conserved hydrophilic protein, located on the nucleus and plasma membrane. PpCaM could induce the expression of *PpCa^{2+}-ATPase*, increase the activity of Ca^{2+}-ATPase, and participate in the regulation of energy metabolism. In addition, PpCaM could activate the activity of PpGAD, promote the synthesis of GABA, and participate in the regulation of GABA metabolism. These results suggested that 0 °C storage and $CaCl_2$ treatment promoted the expression of *PpCaM* and increased the protein content of PpCaM, suggesting PpCaM enhanced the cold resistance and reduced the occurrence of CI by participating in the regulation of energy metabolism and GABA metabolism.