IP3 mediates NO-enhanced chilling tolerance in postharvest kiwifruit

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Abstract

The function of inositol 1,4,5-trisphosphate (IP3) in nitric oxide (NO)-induced chilling tolerance in postharvest kiwifruit was revealed. The fruit were treated using sodium nitroprusside (SNP; exogenous NO donor) and neomycin (IP3 inhibitor). Data demonstrated that compared with the control, chilling injury (CI) index and firmness decreased and increased upon SNP treatment in kiwifruit. SNP treatment enhanced phosphoinositide-specific phospholipase C (PI-PLC) activity, and consequently induced IP3 production. Moreover, SNP treatment down regulated malondialdehyde (MDA) content and electrolyte leakage as well as the activity and gene expression of lipoxygenase (LOX) in kiwifruit. In addition, the gene expression of transcription factors, including C-repeat binding factor1 (CBF1), WRKY1 and NAC5 was induced by SNP treatment. The above effects induced upon SNP treatment were inhibited by neomycin treatment. Neomycin treatment alone also led to the increase in CI index, MDA content and electrolyte leakage as well as the activity and gene expression of LOX, and the decrease in firmness, PI-PLC activity and IP3 production and gene expression of *CBF1*, *WRKY1* and *NAC5*. Thus, IP3 mediated the alleviation of membrane damage, and the induction of CBF1, WRKY1 and NAC5 by SNP, thereby delaying CI in kiwifruit.