Nitric oxide is involved in melatonin-induced cold tolerance in postharvest litchi fruit

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Abstract

Previous studies indicate that melatonin, as an important bioactive molecule, appreciably improves cold tolerance in postharvest litchi fruit. However, whether other signaling molecules are involved in melatonin-induced cold tolerance in litchi fruit has not been clarified. In this study, the effect of the interplay between melatonin and nitric oxide (NO) on chilling stress tolerance in relation to redox homeostasis in litchi fruit was investigated. In the first experiment, exogenous applications of melatonin and sodium nitroprusside (SNP) showed similar efficacy in decelerating the development of chilling injury (CI), as indicated by lower CI index and higher retention of pericarp redness in litchi fruit during refrigeration, whereas the opposite effect was found with NO scavenger 2-(4-carboxyphenyl)-4,4,5,5-tetramethylimidazoline-1-oxyl-3-oxide (cPTIO). The ameliorating effect of melatonin on CI was evidently impaired under cotreatment with melatonin and cPTIO (melatonin + cPTIO). In the second experiment, the biochemical response of litchi fruit to melatonin and melatonin + cPTIO during cold storage was analyzed. The results exhibited that melatonin triggered NO accumulation by activating nitrate reductase (NR) and nitric oxide synthase (NOS) in refrigerated litchi fruit. Melatonin-induced NO accumulation was reduced under treatment with melatonin + cPTIO, but this combined treatment did not affect the NR and NOS activities when compared to melatonin alone. Reactive oxygen species (ROS) in melatonin-treated fruit were markedly quenched by activating antioxidant enzymes and increasing antioxidant metabolite contents, thus alleviating membrane lipid peroxidation and maintaining membrane integrity. Melatonin also led to increased expression of oxidized protein repair-related genes, including *LcMsrA1*, *LcMsrA2*, *LcMsrB1*, *LcMsB2*, *LcTrx2* and *LcNTR1*. Moreover, an enhanced level of protein *S*-nitrosylation was observed in fruit upon melatonin treatment. However, the above physiological effect associated with improved fruit cold tolerance afforded by melatonin was partially counteracted by cPTIO. The results suggest that endogenous NO could mediate melatonin-induced cold tolerance in litchi fruit via regulation of

redox status.

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