New insights into the effects of ethylene on ABA catabolism, sweetening and dormancy in stored potato tubers

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Abstract

Continuous ethylene supplementation suppresses postharvest sprouting, but it can increase reducing sugars, limiting its use as an alternative to chlorpropham for processing potatoes. To elucidate the mechanisms involved, tubers were treated after curing with or without the ethylene binding inhibitor 1-methylcyclopropene (1-MCP at 1 μL L^{-1} for 24 h), and then stored in air or air supplemented with continuous ethylene (10 μL L^{-1}). Across three consecutive seasons, changes in tuber physiology were assessed alongside transcriptomic and metabolomic analysis.

Exogenous ethylene alone consistently induced a respiratory rise and the accumulation of undesirable reducing sugars. The transient respiratory peak was preceded by the strong upregulation of two genes encoding 1-aminocyclopropane-1-carboxylate oxidase (ACO), typical of wound and stress induced ethylene production. Profiles of parenchymatic tissue highlighted that ethylene triggered abscisic acid (ABA) catabolism, evidenced by a steep fall in ABA levels and a transient rise in the catabolite phaseic acid, accompanied by upregulation of transcripts encoding an ABA 8′-hydroxylase. Moreover, analysis of non-structural carbohydrate-related genes revealed that ethylene strongly downregulated the expression of the *Kunitz-type invertase inhibitor*, already known to be involved in cold-induced sweetening. All these ethylene-induced effects were negated by 1-MCP with one notable exception: 1-MCP enhanced the sprout suppressing effect of ethylene whilst preventing ethylene-induced sweetening.

This study supports the conclusions that: i) tubers adapt to ethylene by regulating conserved pathways (*e.g.* ABA catabolism); ii) ethylene-induced sweetening acts independently from sprout suppression, and is similar to cold-induced sugar accumulation.