Title Inhibition of PAL, CHS, and ERS1 in 'Red d'Anjou' Pear (*Pyrus communis* L.) by 1-MCP

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Abstract

The ethylene antagonist 1-MCP was investigated for its potential impact on the transcription of two key flavonoid biosynthetic (phenylalanine ammonia-lyase, PAL, E.C. 4.3.1.5; and chalcone synthase, CHS, E.C. 2.3.1.74), flavonoid transport (glutathione *S*-transferase, GST, E.C. 2.5.1.18) and ethylene perception (ethylene response sensor 1, ERS1) transcripts during the postharvest storage and ripening of pear (*Pyrus communis* L.). 'Red d'Anjou' pear fruit were harvested from Wenatchee, WA, USA, transported to Guelph, Ont., Canada, then treated with 1 μL L^{-1} 1-MCP, and subsequently placed in cold storage (0–1 °C, 90–95% RH) for up to 126 days. After removal, fruit were warmed to room temperature (1 or 7 days) then tissue samples were collected for Northern blot analysis and determination of flavonoid and chlorogenic acid concentration by HPLC. In general, PAL content decreased during storage, with content increasing during the post-storage ripening period in parallel with the increase in respiration rate and ethylene content. In contrast, CHS content decreased dramatically during the 1-week ripening period, while ERS1 remained constant. The expression of PAL, CHS and ERS1 transcripts were all inhibited by 1-MCP. GST transcript abundance decreased during storage, and was largely unaffected by the 1-MCP treatment. The flavonoid concentration remained constant throughout storage and subsequent ripening. However, after the first removal and warming to room temperature, chlorogenic acid concentration increased in the untreated, but not in the 1-MCP-treated fruit. These results suggest that 1-MCP significantly inhibits the transcription of key flavonoid biosynthetic enzymes and ethylene perception proteins, but not the flavonoid transport enzyme. The increase in PAL with the concomitant post-storage decrease of CHS and postharvest decrease of GST suggests a diversion of carbon from flavonoid compounds into chlorogenic acid.