Abstract

With field-grown 'Eureka' cucumber (*Cucumis sativus* L.) fruit, I explored effects of storage programs on ascorbate-glutathione cycle responses during chilling injury development. After harvest, fruit that underwent no preharvest chilling (<12°C) (warm-field fruit) were continuously chilled (5°C), continuously tempered (13°C), preconditioned (4 or 8 days at 13°C before chilling), or intermittently warmed (one to four one-day cycles at 13°C after 3 days at 5°C) without light; fruit that underwent mild chilling one to two nights preharvest were also chilled (5°C) without light postharvest. Surface tissue was assayed for ascorbate, glutathione, ascorbate free radical reductase (EC 1.6.5.4) activity, and glutathione reductase (EC 1.8.1.7) activity during storage. I measured visible onset of chilling injury by assessing phloem exudate declines and translucency increases upon sampling; this method estimated injury onset as effectively as electrolyte leakage in chilled samples. 2,3,5-Triphenyltetrazolium chloride reduction failed to measure injury onset.

In warm-field fruit, injury was observed after 8.7 days chilling; preconditioning (4 and 8 days) delayed observed injury 7.5 and 12.2 days respectively; intermittent warming delayed observed injury 5.3, 0.4, -0.7, and -0.7 days from the preceding cycle; field-chilling delayed observed injury 4.3 days. Senescence (yellowing) limited tempered storage after 4 weeks.

Warm-field fruit harvested later season encountered lower growth temperatures, developed higher initial ascorbate and glutathione, and lasted longer in chilled storage. When intermittently warmed, fruit encountering lower growth temperatures lasted longer. When preconditioned 4 days, fruit encountering lower temperature minima just before harvest lasted longer.

With all programs, ascorbate ranged between 450 and 900 nmol/gfw until shortly before observed injury, then declined; ascorbate did not decline in tempered storage. Delays in ascorbate decline accompanied delays in observed injury. Glutathione ranged between 200 and 600 nmol/gfw through observed injury then declined afterward. Treatments that elevated low ascorbate or glutathione levels within these ranges did not elevate high levels further. All programs sustained or enhanced ascorbate free radical reductase and glutathione reductase activities past observed injury. These patterns conform with a hypothesis that a functional ascorbate-glutathione cycle responds to chilling stress until membranes are compromised by lipid peroxidation.