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Citric Acid Reduces the Respiration of Fresh-cut Carrots

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Citric acid inhibits the activity of phosphofructokinase (PFK) purified from higher plants in vitro (Ashihara and Stupavska, 1984; Dennis and Coultate, 1966). PFK catalyzes the phosphorylation of fructose 6-phosphate to fructose 1,6-bisphosphate in the glycolic pathway of respiratory metabolism, and it has been suggested that PFK plays an important role in the control of glycolysis (Kennedy et al., 1992; Turner and Turner, 1980). Since citric acid is used commercially as an antibrowning agent on fresh-cut fruits and vegetables (Santerre et al., 1988), it was of interest to determine the effect of citric acid on respiration of these products. We report here the effect of various concentrations of citric acid on CO_2 production of carrot (*Daucus carota* L.) shreds.

Carrot roots, purchased from a local wholesaler, were washed, peeled, trimmed of their root tips and stem plates, and shredded (≈50 mm long × 5 mm wide × 4 mm thick). About 600 g of shreds were dipped in a 2-L solution of 0, 1, 3, 10, 100, or 300 mM citric acid (monohydrate; Sigma Chemical Co., St. Louis) for 2 min at room temperature. The dipped shreds were centrifuged for 15 s at 100 g_n, using the spin cycle of a washing machine to remove the surface moisture. After centrifugation, the shreds were placed in plastic trays and stored in 3.8-L glass jars (100 g per jar) at 15 °C. Distilled water (100 mL) was placed at the bottom of the jar to maintain a high relative humidity, and the tray was elevated above the water as described by Kato-Noguchi and Watada (1996). A stream of air was metered through the jar at 15 $mL \cdot min^{-1}$, which was sufficient to keep CO_2 accumulation below 0.3%. Carbon dioxide production of the shreds was measured with a computer-controlled CO_2 analyzer (CD-3A; Ametec, Pittsburgh). Three entirely separate experiments were conducted, and measurement was repeated four times per day with four replications in each experiment. Data were recorded on a fresh mass basis.

The rate of CO_2 production of control carrot tissue (0 mM citrate) was stable at ≈85 $mL \cdot kg^{-1} \cdot h^{-1}$ for 7 days (Fig. 1), which was similar to that reported by Leshuk and Saltveit (1991). Citric acid at 1 mM and above decreased CO_2 production, and the decrease was greater the higher the citric acid concentration. The decreasing rate leveled off after day 2. The steady-state rates of CO_2 production were 70, 51, 44, 30, 26 $mL \cdot kg^{-1} \cdot h^{-1}$ for 1, 3, 10, 30, and 100 mM citric acid at day 3, respectively. These values represent 82%, 60%, 51%, 35%, and 31% the levels of the respective control shreds. No injury was noted on citric acid-treated carrot shreds following storage. These results indicate that citric acid inhibits respiration of fresh-cut carrot shreds at concentrations of 1 mM or higher. Exogenously applied citric acid probably did not serve as a substrate for respiration because the permeability of mitochondria to citric acid is very low (Wiskich and Dry, 1985).

Respiration is generally stimulated and remains elevated when fresh fruits and vegetables are processed for fresh-cut products (Watada et al., 1996). The elevated rates result in a more rapid depletion of reserve substrates for respiration and consequently reduce shelf life (Kader, 1986). The respiration of fresh-cut carrots can be reduced by citric acid treatment, which may subsequently extend shelf life.

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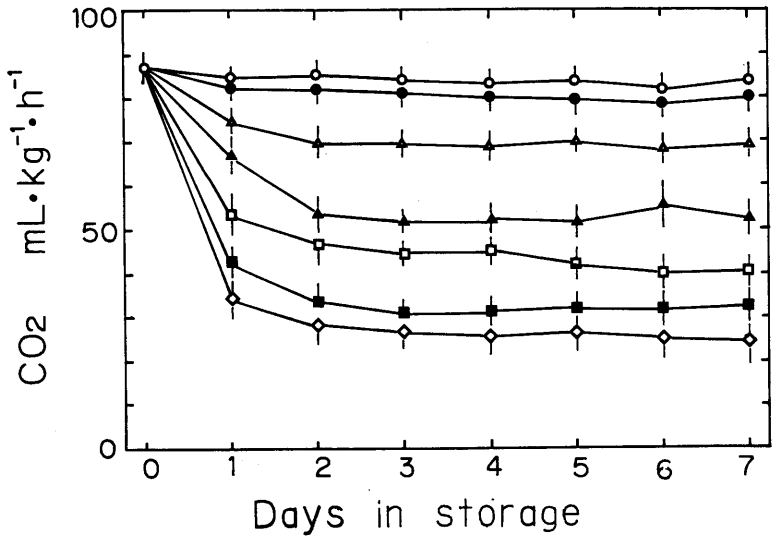

Fig. 1. Effect of citric acid on CO_2 production of carrot shreds at 15 °C. Shreds were dipped in a solution of 0 (○), 0.3 (●), 1 (△), 3 (▲), 10 (□), 30 (■), or 100 mM (◊) citric acid for 2 min before storage (see text). Means ±SE are from three experiments with four replications and four measurements per day for each determination.

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