Nitric oxide alleviates mitochondrial oxidative damage and maintains mitochondrial functions in peach fruit during cold storage

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Abstract

Mitochondrial oxidative damage induces aging and apoptosis of organisms, so improving the antioxidant capacity is vital to maintain mitochondrial functions. Peaches were treated with water (control), 15 μmol L^{-1} nitric oxide (NO), and 5 μmol L^{-1} c-PTIO (NO scavenger), and stored at 0 °C. Mitochondrial reactive oxygen species (ROS) content, mitochondrial swelling, mitochondrial membrane potential, mitochondrial membrane fluidity, and the activities of two mitochondrial respiratory electron transport pathways were measured. Results indicated that NO could extenuate mitochondrial swelling, maintain mitochondrial membrane potential and membrane fluidity, and delay the decrease in activities of the cytochrome pathway and cyanide-insensitive pathway of the mitochondrial respiratory chain. Simultaneously, NO maintained the lower mitochondrial oxygen consumption and cytochrome c content. In summary, NO could efficiently sustain the mitochondrial structure and functions by alleviating the mitochondrial oxidative damage of peaches during cold storage.