Mitochondrial small heat shock protein and chilling tolerance in tomato fruit

Mariela R. Escobar, Martín D. Ré, María L. Sossi, Silvana B. Boggio, Cornelia Herrfurth, Ivo Feussner and Estela M. Valle

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Abstract

Our previous report indicated that tomato (*Solanum lycopersicum*) fruit of two contrasting varieties in the chilling tolerance showed the opposite expression pattern of a mitochondrial small heat shock protein (*M-sHSP23.8*) gene after chilling stress. Thus, the fruit of the relatively tolerant variety Micro-Tom strongly accumulated *M-sHSP23.8* transcripts while the susceptible var. Minitomato fruit did not. To test whether *M-sHSP23.8* is involved in tomato fruit protection mechanisms against chilling stress, Minitomato fruit overexpressing *M-sHSP23.8* (*OE23.8*) and knockdown Micro-Tom fruit with reduced levels of *M-sHSP23.8* (*amiR23.8*) were developed. After chilling treatment, most of the *amiR23.8* fruit failed to ripen normally, showed wilting and skin wrinkles, partial discoloration, and did not reach full red color. On the contrary, these chilling injury symptoms were significantly diminished in *OE23.8* fruit, showing less visible deterioration after chilling. Fruit of *OE23.8* and *amiR23.8* showed opposite patterns of water loss, electrolyte leakage, and expression of the tomato *catalase 1* gene compared to control fruit. Membrane lipidome profile evidenced that *amiR23.8* fruit showed differential adjustment of extraplastidic and plastidic lipids and variations in the lipid remodeling compared to control fruit, suggesting alterations in the membrane integrity. The high sensitivity of Micro-Tom *amiR23.8* fruit and the better performance of Minitomato *OE23.8* fruit to chilling treatment indicate that sHSP23.8 may be crucial in the chilling stress tolerance in tomato fruit.