

parthenocarpic fruits, and the combination of poor fruit set at 26°C, fewer flowers, and altered fruit growth resulted in low yield. Temperature also modifies the cellular basis of fruit growth: although fruit growth rate was lower at 20/20°C than at warmer regimes, final fruit size was maintained by compensation between cell number and cell size, whereas heavier fruit load reduced fruit size mainly by slowing cell expansion rather than by reducing cell number.

5.3 Effects on fruit quality characteristics

Temperature regulation affects tomato fruit quality as strongly as it affects yield, but the response is trait-specific. Elevated temperature often increases total soluble solids, titratable acidity, and ascorbic acid, while decreasing lycopene, and high-temperature field screening further showed increases in soluble solids, acidity, total phenols, and vitamin C in tolerant genotypes, with concurrent decreases in pH, electrical conductivity, flavonoids, lycopene, and β -carotene (Vijayakumar et al., 2021). These quality shifts indicate that heat does not uniformly degrade composition; instead, it tends to re-balance primary and secondary metabolites, sometimes improving acidity- or vitamin-related traits while reducing carotenoid-based color and nutritional value.

The effect of temperature on fruit quality also depends on developmental stage, humidity, and the specific metabolite considered. Increasing fruit temperature from 21°C-26°C reduced total carotene without affecting lycopene, whereas a further rise from 27°C-32°C reduced ascorbate, lycopene, and precursor contents but increased rutin, caffeic acid derivatives, and glucosides, showing that antioxidant pathways are highly temperature-sensitive. Under combined high temperature and high relative humidity, enzyme activities linked to sucrose breakdown and organic acid metabolism shifted in ways that reduced soluble sugar, vitamin C, total sugar, and the sugar/acid ratio, while increasing titratable acidity; notably, 32°C with 70% relative humidity was identified as the best condition for maintaining fruit quality during the reproductive period under high-temperature stress (Zheng et al., 2022).

6 Physiological and Molecular Mechanisms of Temperature Regulation Effects

6.1 Hormonal regulation under temperature stress

Temperature stress in tomato triggers broad hormonal reprogramming rather than a single-pathway response. Across heat-stress studies, hormone-associated genes are repeatedly among the temperature-responsive transcripts, and genotype-dependent thermotolerance is linked in part to differential regulation of auxin- and ethylene-related genes (Hu et al., 2020). Brassinosteroid signaling appears to be one important branch of this response, because BR treatment in tomato increases RBOH1 expression and apoplastic H₂O₂, while silencing RBOH1 compromises heat tolerance (Li et al., 2021).

Hormonal regulation also integrates developmental temperature responses and cross-stress protection. Day-night temperature difference regulates stem elongation through changes in gibberellin and IAA biosynthesis, with negative DIF suppressing both hormone levels and elongation-related gene expression (Ohtaka et al., 2020). Under extreme temperatures, strigolactones act upstream of ABA-dependent protection, since heat and cold induce strigolactone biosynthesis genes, and ABA deficiency abolishes strigolactone-induced transcription of HSP70, CBF1, and antioxidant-related genes (Chi et al., 2021).

6.2 Heat and cold stress response pathways

The core heat stress response in tomato is organized around HSF-HSP networks that preserve protein homeostasis and cellular survival. Hsfs control transcriptional reprogramming at high temperature and activate canonical HSR genes, especially heat shock proteins, which function as molecular chaperones to prevent protein misfolding and aggregation. Within tomato, HsfA1 has a uniquely central role, because plants with HsfA1 cosuppression become extremely heat-sensitive and fail to induce normal synthesis of chaperones and other Hsfs under elevated temperature.

Cold and heat pathways also intersect with ROS and kinase signaling, but the specific regulators differ by stress type. In heat-stressed tomato, SIMAPK3 acts as a negative regulator of thermotolerance, since knockout mutants show less wilting and membrane damage, lower ROS, and higher antioxidant enzyme activity together with