5 Molecular Regulation of Sugar–Acid Balance in Kiwifruit

5.1 Key structural genes involved in sugar and acid metabolism

Genes controlling starch degradation and sucrose metabolism are major determinants of sugar accumulation during ripening. Multi-omics analyses identify *AMY*, *BAM*, *SUS*, *SPS*, *INV*, *HK*, and *FK* as core structural genes associated with soluble sugar metabolism in kiwifruit (Wang et al., 2021). Among them, *BAM* genes are repeatedly highlighted as the main drivers of starch-to-sugar conversion, and AcBAM3.5 shows especially strong ripening-associated expression in 'Hongyang' (Gong et al., 2025). Zhang et al. (2018) further demonstrated in an ethylene-response study of 'Hayward' kiwifruit that *AdBAM3L* is a key gene involved in starch degradation, and its overexpression reduces starch content, supporting a direct structural role for *AdBAM3L* in soluble sugar formation. Recent family-wide analysis also expanded the BAM repertoire to 17 *AcBAM* genes, with *AcBAM5* and *AcBAM13* most highly expressed during postharvest storage and strongly induced by ABA; silencing either gene increases starch content (Gong et al., 2025). In yellow-fleshed kiwifruit, Lin et al. (2024) pointed out six *AcBAM* genes are significantly upregulated during ripening, especially *AcBAM3.1* and *AcBAM3.2*, and their expression parallels rising β-amylase activity and sugar accumulation. Starch synthesis capacity also contributes to final sweetness, because AGPS1.1, AGPS1.2, and WAXY1 are upregulated during development in sweeter *A. eriantha* fruit, while BAM3.2, BAM3.3, PHO1, PHO2.2, SUS2.1, and SUS2.2 support subsequent remobilization (Li et al., 2024).

Genes associated with citrate and malate metabolism regulate the acid side of the balance through both metabolic flux and transport. For citrate, accumulation depends on strong synthesis and weak degradation, with citrate synthase, mitochondrial aconitase, and NAD-dependent isocitrate dehydrogenase identified as key enzymatic control points (Wang et al., 2021). A major kiwifruit-specific advance is the identification of AcALMT1 as a citrate transporter rather than a malate transporter in this context; its expression correlates with citrate levels, and transient overexpression increases citrate accumulation. Malate regulation appears partly distinct: AcPNAD-MDH1 is negatively correlated with malate content during 'Hongyang' development, and overexpression lowers both malate and citrate in leaves (Qi et al., 2025). In *A. eriantha*, malate accumulation is linked mainly to NAD-MDH and PEPC activity, with AeMDH2 implicated as an important regulator, while quinate metabolism has been associated with DAHPS, DHQS, and QDH expression.

Sugar and organic acid metabolism are interconnected through carbon allocation, respiratory metabolism, and intracellular compartmentation. The major structural genes and metabolic pathways involved in starch–sugar interconversion and citrate–malate metabolism collectively constitute the sugar–acid metabolic network in kiwifruit and contribute to the establishment of sugar–acid balance through coordinated carbon flux (Figure 2).

5.2 Transcriptional regulation of sugar–acid metabolism

MYB, bZIP, NAC, ERF, bHLH, C2H2, Dof, and related transcription factor families all contribute to sugar–acid regulation, but they do so through different targets and developmental signals. Network analysis places NAC, C2H2, AP2/ERF, MYB, and bHLH among the dominant candidate families associated with soluble sugar accumulation in kiwifruit (Song et al., 2025). Within sugar metabolism, AcERF182 directly emerges as a central positive regulator because it is tightly correlated with AcBAM3.5 and embedded in the soluble sugar regulatory network (Wang et al., 2021). Additional ripening regulators act upstream of BAM genes through different mechanisms: AdDof3 activates the AdBAM3L promoter in an ethylene-responsive pathway, while under cool temperature AcCTS1 directly activates AcBAM3.3 and AcBAM3.5 and is negatively controlled by the E3 ligase AcPUB11 (Zhang et al., 2018). Co-regulation can also be negative: spatiotemporal multiomics identified an A. chinensis ERF1 that negatively controls sugar accumulation and ethylene production, indicating that not all ERFs promote ripening sugar gain (Zeng et al., 2025).

On the acid side, NAC regulation is the clearest experimentally validated mechanism. AcNAC1 directly binds the AcALMT1 promoter and activates its expression, and CRISPR mutagenesis of AcNAC1 causes a dramatic citrate decline without major effects on malate or quinate (Fu et al., 2023). Malate control involves a different transcriptional module, because AcSQBP9 represses AcPNAD-MDH1 and its mutagenesis decreases both malate