

A Roadmap for Breeding Sweet Tomatoes

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Received: 22 September 2025 | **Revised:** 14 April 2026 | **Accepted:** 16 April 2026

Keywords: genetic engineering | sugar metabolism | tomato sweetness

ABSTRACT

Sweetness is the most important determinant of the overall desirability of tomato (*Solanum lycopersicum*) fruits. In cultivated tomatoes, sweetness is mainly determined by the contents of fructose and glucose, which are the major carbohydrates in the fruits. In recent years, many key regulators of sugar accumulation in tomato fruit, including sugar metabolism enzymes, sugar transporters, transcription factors, and protein kinases, have been identified through map-based cloning, genome-wide association studies, and reverse genetics. A growing number of studies have also indicated that aromatic volatiles, such as apocarotenoid- and phenylalanine-derived volatiles (PHEVs), can improve tomato sweetness independently of sugar content. In this perspective, we summarise recent advances in our understanding of sugar metabolism in tomatoes and discuss progress in identifying aromatic substances that can affect tomato sweetness. We also discuss challenges in increasing tomato sweetness and propose new breeding strategies to further improve sugar content and sweetness in tomatoes.

1 | Introduction

Humans are naturally fond of sweet foods, and sugar is the most important source of sweetness in foods [1–3]. This fondness of sweet foods has been traced back to our early primate relatives and arises from natural selection [4]. In resource-limited environments, individuals capable of rapidly accessing and utilising sugary foods such as ripe fruit and honey exhibited enhanced survival and reproductive success. In addition, sugar is related to neurochemical changes in the brain, including changes in the levels of dopamine and acetylcholine opioids, which make us feel happy and satisfied, thus reinforcing the preference for sugar [5]. Historically, sugar was a costly commodity symbolising power and status. The Industrial Revolution enabled mass production, transforming it into a daily staple for the general population [6]. Despite its commonality today, the pursuit of sugar has never stopped.

Tomato (*Solanum lycopersicum*) is currently the most valuable vegetable crop worldwide and prized by consumers for its distinct flavour profile, with sweet varieties being particularly preferred. To date, a total of 31 key flavour-associated compounds have been characterised in tomato fruits. Of these, soluble sugars and organic acids act as primary taste-active components by stimulating gustatory receptors, whereas 27 volatile metabolites (e.g., 2-phenylethanol and 6-methyl-5-hepten-2-one) modulate olfactory perception [7–9]. However, the flavour quality of modern tomato cultivars has declined significantly, mainly because flavour-related traits have never been a priority in breeding. Breeders have primarily focused on meeting the needs of growers by prioritising high yield and resistance as the primary selection criteria. During domestication and improvement, the fruit weight of modern tomatoes (111.33 ± 68.19 g) has increased nearly 60 times larger than its wild ancestors (2.04 ± 0.85 g) [10]. Nevertheless, the soluble

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Key Points

- This perspective systematically reviews key genes, loci and molecular mechanisms underlying sugar metabolism and sweetness-enhancing volatile biosynthesis in tomato.
- It proposes an integrated breeding roadmap to synergistically improve tomato sweetness and agronomic traits via multi-omics, gene editing and AI-aided strategies.

solids content (SSC) of modern cultivars has decreased by approximately one-third, and the concentrations of 20 out of the 27 flavour-determining volatile metabolites have been reduced by approximately 20%–70% [7]. Thus, the synergistic enhancement of both fruit flavour and yield has emerged as a critical and urgent breakthrough target for tomato breeding in the future.

Sugar accumulation is inherently tied to fruit ripening, a process divided into four distinct stages: the immature green stage (IMG), green ripening stage (MG), breaker stage (BR), and red ripening stage (RR). Fruit size gradually expands during the IMG stage and is generally fixed during the MG stage, with the substantial accumulation of glucose and fructose occurring after the BR stage [11, 12]. This development time makes it possible

to balance yield and quality during the breeding process. Here, we summarise recent advances in our understanding of sugar metabolism in tomato fruits. We also discuss future challenges in increasing tomato sweetness and propose new breeding strategies to further improve the sugar content and sweetness of tomatoes.

2 | Sugar Metabolism in Tomato Fruits

The sugar content in tomato fruit is a complex quantitative trait controlled by multiple genes [13, 14]. Wild tomato species are widely distributed geographically and exhibit extensive genetic variation, providing valuable genetic resources for improving tomato fruit quality [15]. To date, four quantitative trait loci (QTLs) that regulate sugar accumulation in tomato fruits have been identified based on recombinant inbred lines (RILs) or near-isogenic lines (NILs) derived from wild germplasm (Table 1). Three of these QTLs encode enzymes related to carbohydrate metabolism. Among these, *Sucr* (Suc accumulation), identified in *Solanum chmielewskii*, encodes an acid invertase (TIV1) [16, 17]. Another QTL encodes AGPL1, the large subunit of ADP-glucose pyrophosphorylase [18, 19] (Figure 1). Increasing the activities of these sugar metabolism enzymes can increase the accumulation of sugar in fruits. *Lin5* encodes a flower- and fruit-specific invertase that promotes the cleavage of sucrose in the apoplast, thereby affecting the sugar supply from

TABLE 1 | Functionally characterised sugar metabolism genes in tomato fruit.

Gene name	Gene locus	Biological functions	References
<i>SlTIV1/VI</i>	Solyc03g083910	Acid invertase	[16, 17]
<i>SLAGPL1</i>	Solyc01g109790	ADPglucose pyrophosphorylase	[18]
<i>SILin5</i>	Solyc09g010080	Cell-wall invertase	[20]
<i>SlINVINH1</i>	Solyc12g099200	Inhibitor of CWINV	[90]
<i>SIVPE5</i>	Solyc12g095910	Vacuolar processing enzyme	[91]
<i>SlHT1/STP1</i>	Solyc02g079220	Hexose transporter	[26, 36]
<i>SlHT2</i>	Solyc09g075820	Hexose transporter	[36]
<i>SlHT3</i>	solyc07g006970	Hexose transporter	[36]
<i>SlFgr</i>	Solyc04g064610	Sugar transporter	[21]
<i>SlSWEET7a</i>	Solyc08g082770	Sugar transporter	[37]
<i>SlSWEET14</i>	Solyc03g097560	Sugar transporter	[37]
<i>SlSWEET15</i>	Solyc09g074530	Sugar transporter	[67]
<i>SlZIP11</i>	Solyc08g066500	Zn transporter	[92]
<i>SlGLK2</i>	Solyc10g008160	Transcription factor	[27, 28]
<i>SlGLK1</i>	Solyc07g053630	Transcription factor	
<i>SLARF4</i>	Solyc11g069190	Auxin response factor	[31]
<i>SLARF10</i>	Solyc11g069500	Auxin response factor	[29]
<i>SLARF6A</i>	Solyc00g196060	Auxin response factor	[30]
<i>SlbZIP1</i>	Solyc01g079480	Transcription factor	[32]
<i>SlHY5</i>	Solyc08g061130	Transcription factor	[33]
<i>SlCDPK26</i>	Solyc01g008440	Protein kinase	[48]
<i>SlCDPK27</i>	Solyc11g065660	Protein kinase	[48]

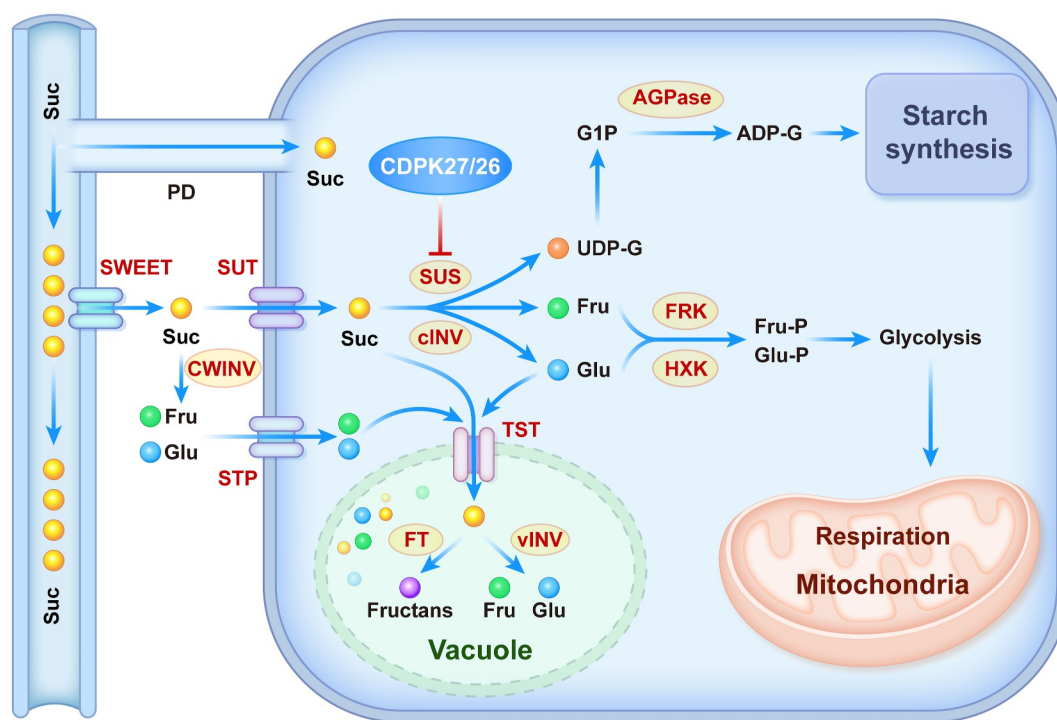


FIGURE 1 | Simplified schematic presentation of sugar metabolism in tomato fruit cells. Following long-distance phloem transport, sucrose (Suc) is unloaded into the apoplast via SWEET transporters. In the apoplast, Suc is hydrolysed by cell wall invertase (CWINV) into glucose (Glu) and fructose (Fru), which are then imported into fruit cells through Sugar Transport Proteins (STP). Alternatively, Suc enters cells directly via sucrose transporters (SUT) or plasmodesmata (PD). Once inside the cell, Suc, Glu, and Fru are transported into the vacuole for storage by tonoplast-localised Tonoplast Sugar Transporters (TST). Within the vacuole, Suc is either hydrolysed into Glu and Fru by vacuolar invertase (vINV) or converted into fructans by fructosyltransferase (FT). In the cytoplasm, Suc is hydrolysed into Glu and Fru by cytoplasmic invertase (cINV) or cleaved into Fru and UDP-glucose (UDP-G) by cytoplasmic sucrose synthase (SUS); notably, CDPK27/26 regulates SUS3 protein stability via phosphorylation. Glu and Fru are phosphorylated into glucose-phosphate (Glu-P) and fructose-phosphate (Fru-P) by hexokinase (HXK) and fructokinase (FRK), respectively, which then undergo glycolysis and subsequent mitochondrial respiration. UDP-G is converted into glucose-1-phosphate (G1P) under the action of enzyme UDP-Glc pyrophosphorylase (UGPase), and then G1P is converted into ADP-glucose (ADP-G) by ADP-glucose pyrophosphorylase (AGPase), which is subsequently channelled into starch biosynthesis. Colour spheres coding: yellow, Suc; blue, Glu; green, Fru; orange, UDP-G; purple, fructans.

tomato source organs to fruits (Figure 1). The conversion of glutamic acid at position 348 in *Lin5* to aspartic acid increased its enzyme activity and fruit sink strength, thus increasing sugar yield in tomato [20]. *Fgr* (Fructose to glucose ratio), identified from *Solanum habrochaites* (LA1777), encodes a glucose efflux transporter of the SWEET (Sugars Will Eventually be Exported Transporter) family that regulates the ratio of fructose to glucose in tomato fruits (Figure 1). Overexpressing *Fgr* increased the fructose-to-glucose ratio but decreased the total sugar content in tomato fruits [21].

Multi-omics analysis has provided considerable insight into the extent of metabolic and genetic variation in tomato and shed light on the degree to which human domestication and genetic improvement have influenced fruit metabolite content [15]. In terms of genome-wide association studies (GWAS) on fruit metabolic traits, Sauvage et al. conducted GWAS using 163 tomato accessions, identifying 9, 3, and 2 loci associated with SSC, sucrose, and fructose contents, respectively [22]. Subsequently, Zhao et al. performed GWAS on 174 tomato varieties, leading to the identification of 10, 4, and 6 quantitative trait loci (QTLs) involved in regulating sucrose, glucose, and fructose contents, respectively [23]. Furthermore, through meta-analysis

integrating data from multiple GWAS populations, Zhao et al. successfully screened four candidate genes that modulate glucose and fructose contents in tomato fruits [24]. Additionally, Tieman et al. identified two major QTLs, namely *Lin5* and *SSC11.1*, by utilising 398 accessions encompassing modern cultivars, heirlooms, and wild relatives [7]. More recently, a graph pangenome of tomato constructed using an even larger population was used for GWAS and showed great power to capture missing heritability, leading to the identification of two new genes that might contribute to sugar accumulation in tomato fruit [25]. Notably, although multi-omics analyses have uncovered numerous QTLs regulating tomato fruit sugar content, *SIHT1/STP1* (*Solanum lycopersicum* Sugar Transport Protein 1) stands out as one of the few loci that have been recently demonstrated to directly regulate fruit SSC (Table 1). A 21-bp insertion in the promoter region of *SIHT1/STP1* is correlated with high SSC in tomato fruits; this insertion facilitates the binding of ZAT10-LIKE to the *SIHT1/STP1* promoter, thereby enhancing *SIHT1/STP1* expression and promoting sugar accumulation in fruits [26] (Figure 1).

In parallel with QTL analysis and GWAS, reverse genetics approaches have also been extensively employed to dissect the key

regulatory factors governing sugar metabolism in tomato (Table 1). For instance, the MYB transcription factor *SlGLK2* (*Solanum lycopersicum* Golden 2-like 2) enhances chloroplast abundance and chlorophyll content in immature green fruits, thereby boosting photosynthetic capacity and ultimately elevating sugar accumulation in mature fruits [27, 28]. Consistently, *SlGLK2* expression is positively modulated by *SlARF10* (*Solanum lycopersicum* Auxin Response Factor 10) and *SlARF6A* [29, 30], while the expression of *SlGLK1* is negatively regulated by *SlARF4* in tomato fruits [31], thus implicating these ARF family members participating in the regulation of tomato fruit sugar levels. Moreover, fruit-specific overexpression of the bZIP (Basic Region Leucine Zipper) transcription factor gene *SlbZIP1* resulted in the production of tomato fruits with enhanced sweetness [32]. Most recently, *SlHY5* (*Solanum lycopersicum* Elongated Hypocotyl 5), a core regulator of light-induced signal transduction networks, has been demonstrated to modulate tomato fruit sugar content by directly binding to the promoter of *SlSWEET12c* [33]. Additionally, functional inactivation of *SlINVINH1* (*Solanum lycopersicum* Cell Wall Invertase Inhibitor 1) has been shown to increase sugar content in tomato fruits [34].

Notably, sugar transporters also contribute substantially to sugar accumulation in tomato fruits [35] (Table 1). For instance, RNAi-mediated knockdown of the STP (Sugar Transport Proteins) subfamily monosaccharide transporter genes *SlHT1/STP1*, *SlHT2*, and *SlHT3* (formerly *LeHT1*, *LeHT2*, and *LeHT3*, respectively) caused a 55% decrease in hexose accumulation in tomato fruits [36] (Figure 1). Additionally, *SlSWEET7a* and *SlSWEET14* localise to the plasma membrane and mediate the transport of fructose, glucose, and sucrose; silencing of *SlSWEET7a* or *SlSWEET14* markedly increased sugar accumulation in tomato fruits [37].

More recently, tonoplast sugar transporters (TSTs) have been shown to regulate the accumulation and distribution of sugars in the fruits of various vegetable and fruit crops, thereby influencing their flavour quality [35]. *CITST2* is the causal gene of the major-effect QTL *QBRX2-1*, which regulates sugar content in watermelon (*Citrullus lanatus*) fruit [38]. Similarly, three *CmTSTs* were identified in melon (*Cucumis melo*), among which *CmTST2* is highly expressed during fruit development; heterologous overexpression of *CmTST2* significantly increased sucrose, glucose, and fructose levels in strawberry (*Fragaria × ananassa*) and cucumber (*Cucumis sativus*) fruits [39]. Six *MdTSTs* have been identified in apple (*Malus domestica*), with *MdTST1* and *MdTST2* expression showing a strong positive correlation with sugar accumulation during fruit development. Overexpressing *MdTST1* or *MdTST2* increased sucrose, glucose, and fructose contents in apple callus tissues [40, 41]. The orthologs *SlTST1* and *SlTST2* exhibit similar functions in tomato fruit, as the simultaneous knockout of *SlTST1* and *SlTST2* significantly reduced the sugar content in tomato fruits [40] (Figure 1). In addition, orthologous *TSTs* in other vegetable and fruit crops, such as sweet orange (*Citrus sinensis*), strawberry (*Fragaria vesca*), and pear (*Pyrus* L.), also regulate sugar content in fruits [42–44]. These findings underscore the crucial roles of TST proteins in regulating sugar accumulation in fruits, suggesting that manipulating *TST* gene expression or transport activity could modulate sugar content in tomato fruits.

3 | Releasing Sugar Brakes Generates Sweeter Tomatoes

Increasing both SSC and fruit weight is a major goal of modern tomato breeding, but this has largely proven elusive [7]. Recent studies have suggested that it should be possible to break the yield-sugar trade-off in tomato, as revealed using an integrative model by coupling a biophysical model of fleshy fruit growth processes [45], with simulations for recurrent genomic selection [46], as well as using a population large enough to utilise a phenotypic trait-driven selection approach [47].

We recently discovered two ‘sugar brake’ genes, *Calcium-Dependent Protein Kinase 27* (*SlCDPK27*; also known as *SlCPK27*) and its paralog *SlCDPK26*, which regulate the total amount of sugar in fruit by phosphorylating the sucrose synthase *SlSUS3*, thereby promoting its degradation (Figure 1). Genome editing of *SlCDPK27* and *SlCDPK26* increased glucose and fructose contents in tomato fruits by up to 30%, and all mutants exhibited comparable fruit size and total yield to the wild type. In addition, a 12-bp insertion within the promoter region of *SlCDPK27* was significantly associated with high SSC content, but the low-sugar allele without the 12-bp insertion was predominantly present in the modern, large-fruited (BIG) population (97%) owing to co-evolution of *SSC11.1* and the three fruit weight (*fw*) loci [48]. Thus, the selection of large-fruited varieties has led to a dramatic decline in the frequency of the high-sugar allele, resulting in reduced flavour in modern commercial tomato varieties.

We created two null mutants for *SlCDPK27* named MM-CDPK27-CR1 and MM-CDPK27-CR2. Interestingly, MM-CDPK27-CR1, which harbours a 6-bp deletion in the sequence encoding the ATP-binding domain, accumulated more sugar than MM-CDPK27-CR2, a typical mutant with 5-bp deletions, resulting in the premature termination of protein translation [48]. Because the deletion of two key amino acids is required for ATP binding, the recombinant *SlCDPK27*-CR1 protein nearly lost its kinase activity, but its protein-binding ability remained unaffected. In vitro phosphorylation assays demonstrated that *SlCDPK27*-CR1 competes with *SlCDPK26* for interaction with *SlSUS3*, which in turn prevents the phosphorylation of *SlSUS3* by *SlCDPK26*. *SlCDPK27* and *SlCDPK26* can be viewed as functionally redundant, endowing plants with better environmental adaptability and a wider range of uses, but at the same time, this situation increases the complexity and uncertainty of genetic breeding. Nonetheless, we created a 6-bp deletion in the sequence encoding the ATP-binding domain of *SlCDPK27*, representing a novel allele created through gene editing and providing a beneficial allele for tomato improvement that encodes a dominant-negative interactor.

4 | Sweetness-Enhancing Volatiles

Human perception of sweetness involves the integration of taste and olfactory sensations [7]. Taste in tomato mainly involves sensing sugar and substances that are inherently sweet, and some aromatic volatiles can contribute to perceived sweetness independent of sugar concentration through olfactory pathways.

These volatiles are mainly derived from phenylalanine, branched-chain amino acids (BCAAs), fatty acids, or carotenoids [8, 49, 50]. In strawberry, more than 20 volatile compounds were shown to significantly enhance the perceived intensity of sweetness independent of glucose, fructose, and sucrose [51, 52]. Thirteen volatile compounds are positively correlated with sweetness in apples, seven of which are esters [53]. In tomatoes, sweetness was associated with 12 compounds; geranial, 2-methylbutanal, and 3-methyl-1-butanol were independent predictors of sweetness after accounting for fructose [54]. Recently, using machine learning models, 2-phenylethanol, 1-penten-3-one, *E*-2-pentenal, and 4-carene were also estimated to be important for perceived sweetness in tomatoes [55].

Although numerous volatile compounds have been identified to be associated with sweetness in tomato, only a few key regulators have been characterised to date. A small family of aromatic amino acid decarboxylases, including *SIAADC1A*, *SIAADC1B*, and *SIAADC2*, is involved in the conversion of phenylalanine to phenethylamine. Overexpression of either *SIAADC1A* or *SIAADC2* can increase the accumulation of PHEV, including 2-phenylacetaldehyde, 2-phenylethanol, and 1-nitro-2-phenylethane, in tomato fruits [56]. We recently identified a long non-coding RNA (*lncRNA*), designated *lncRNA-PEL8*, through multi-omics analysis of fruit tissues from 404 tomato accessions. Overexpression of *lncRNA-PEL8* significantly upregulated the expression of *SIAADC1A*, *SIAADC1B*, and *SIAADC2*, thereby promoting the accumulation of 2-phenylethanol [9]. Using a segregating population, *FLORAL4* was identified as the candidate gene underlying the PHEV QTL on chromosome 4. *FLO-RAL4* belongs to the mitochondrial 2-oxoisovalerate dehydrogenase/decarboxylase family, which functions in BCAA catabolism. CRISPR/Cas9-mediated knockout of *FLORAL4* decreased the contents of 2-phenylethanol, phenylacetaldehyde and 1-nitro-2-phenylethane [57]. In addition, *SIPAR1* and *SIPAR2*, belonging to the large and diverse short-chain dehydrogenase/reductase family, can catalyse the conversion of 2-phenylacetaldehyde to 2-phenylethanol. Overexpression of either *SIPAR1* or *SIPAR2* in petunia significantly increased the accumulation of 2-phenylethanol in flowers [58].

Similarly, 1-penten-3-one and *E*-2-pentenal are classified as C5 volatiles derived from fatty acid cleavage. *TomloxC*, a 13-lipoxygenase (LOX), plays a critical role in the biosynthesis of 1-penten-3-one and *E*-2-pentenal, and silencing the expression of *TomloxC* significantly reduces the levels of these two volatiles in tomato fruits and leaves [59]. Furthermore, metabolite-based GWAS combined with genetic mapping and functional analyses have identified *Sl-LIP8*, encoding a novel member of the class III lipase family, which is associated with the accumulation of short-chain fatty acid-derived volatile organic compounds (FA-VOCs) in tomato fruits. Knockout of *Sl-LIP8* significantly reduces the levels of multiple C5 and C6 FA-VOCs in ripe tomato fruits. Further investigations revealed that *Sl-LIP8* participates in the first step of FA-VOC biosynthesis, where it cleaves acylglycerides to release linolenic acid and linoleic acid, the two major precursors of C6 and C5 volatiles in tomato [60].

Compared with phenylalanine-derived and fatty acid-derived volatiles, the regulatory mechanisms underlying several other volatile biosynthesis pathways remain largely unclear. For

example, 2-methylbutanal is derived from isoleucine, while 3-methyl-1-butanol is derived from leucine. The biosynthetic pathways of BCAA-derived volatiles have been well characterised in microorganisms such as yeasts and bacteria. In many fleshy fruits, including tomato, several steps in the biochemical pathway of BCAA-derived volatiles have been shown to be conserved with those in yeast and bacterial pathways [61]. Although multiple enzymes, including branched-chain amino acid transaminases (BCATs), aldehyde dehydrogenases (ADs), and α -keto acid decarboxylases, are known to participate in this pathway, the precise biosynthetic route and regulatory mechanisms of 2-methylbutanal and 3-methyl-1-butanol in tomato fruit remain poorly understood [62].

5 | Conclusions and Perspectives

Although numerous QTLs have been implicated in sugar accumulation in tomato fruit, only a few key regulatory genes have been cloned, and the sugar content in cultivated tomato is still generally low. Furthermore, the molecular regulatory network underlying sugar accumulation in tomato fruits is yet to be fully elucidated. Notably, many of the cloned regulatory genes involved in sugar accumulation exert pleiotropic effects on other agronomically important traits. Thus, enhancing the sweetness of tomato fruits while preserving or improving other desirable agronomic traits remains a major challenge in tomato genetic improvement and molecular breeding.

Traditionally, key genes regulating sugar metabolism have been identified through GWAS analysis, map-based cloning and reverse genetics analysis methodologies. However, most studies on sugar accumulation in tomato fruits have focused on enzymes or transporters involved in fruit sugar metabolic pathways. Notably, the genes encoding these proteins typically exhibit broad tissue expression patterns; thus, genetic manipulation of these sugar metabolism-related genes frequently perturbs normal plant growth and development [13]. In recent years, abundant transcriptomic and metabolomic data of tomato across various developmental stages are available, facilitating the identification of genes with developmental stage-specific expression [15, 63]. Integration of traditional GWAS, genetic linkage analysis and developmental gene expression profiles enable the screening of genes with specific functions in fruit sugar accumulation, thus resolving the trade-off between sugar accumulation and plant growth. Therefore, genetic engineering of key regulatory factors specifically expressed during the sugar accumulation stage in fruits is a promising approach to develop tomato cultivars with improved sugar content while maintaining yield potential.

In addition, tomato fruit ripening is accompanied by changes in the contents of substances related to the specific quality of fruit, which occurs simultaneously with seed maturation [64]. Sucrose is the primary assimilate transported in tomatoes, and the sugars in tomato fruits predominantly come from photosynthetic sucrose synthesised in the leaves [65]. Following its synthesis, sucrose is transported long distances via vascular tissues to fruits, where it undergoes redistribution [66]. A portion of the sucrose is delivered to fruit flesh cells, supporting their

development and expansion, while another portion is allocated to seeds, thus promoting their growth [67]. However, seeds within fleshy-fruited tomato varieties are primed during the later stages of development, and seed dry weight reaches a plateau after the fruit has reached the MG stage [68, 69]. Interestingly, the MM-CDPK27-CR1 mutant exhibited a significant (18%) reduction in seed number per fruit, and the *Slcdpk27 Slcdpk26* double mutant showed a 13% decrease in 1000-seed weight, but with normal germination, indicating that photo-assimilates can be reallocated from seeds into fruits during fruit ripening [48]. Although it remains to be further clarified how SICDPK26/27 regulates sugar distribution between the pericarp and seeds, these findings indicate that more attention should be paid to manipulating energy distribution between seeds and fruits. In recent years, a suite of advanced technical platforms has become available for the identification of tissue- and cell-specifically expressed genes, such as spatiotemporal transcriptomics and integrated metabolomics technologies [70]. These cutting-edge techniques allow precise identification of key genes regulating tissue-specific distribution of fruit sugars. Perhaps tomato varieties could be created with seeds that germinate normally but with more energy stored in fruits, although this may increase seed production costs, and induce the risk of unstable seed germination rates.

Moreover, different types of sugars exhibit distinct effects on sweetness. For instance, fructose possesses superior taste and sweetness compared to sucrose and glucose, with a sweetness intensity 1.5 times that of sucrose and twice that of glucose [71, 72]. The composition of soluble sugars also differs across various fruit species; fruits with a sweeter taste generally tend to accumulate higher levels of fructose and sucrose. In apples and pears, fructose constitutes the highest proportion of soluble sugars, accounting for up to nearly 60% [73]. For the fruits of bananas, mango and pineapple, sucrose serves as the dominant soluble sugar, with the sucrose content accounting for approximately 8% of the fresh weight [74]. Tomato fruits mainly accumulate glucose and fructose, with the ratio of glucose to fructose being close to 1:1, while the ratio could be regulated by manipulating a SWEET protein [21], and a small number of wild tomato fruits primarily accumulate sucrose [16]. Given that fructose and sucrose have higher sweetness intensities than glucose, enhancing the sweetness of cultivated tomato fruits, without altering their total soluble sugar content, could be achieved by either increasing fructose accumulation (instead of glucose accumulation) or promoting substantial sucrose accumulation in the commercial tomato fruits.

More interestingly, a growing number of recent studies have demonstrated that volatile aromatic compounds can modulate human sweetness perception, with numerous volatile compounds significantly correlated with perceived sweetness in tomato fruits [7, 49]. Given that these volatile compounds predominantly accumulate after the BR stage and enhancing their levels does not compromise fruit development or other physiological processes, it is imperative to assess these volatile compounds that enhance sweetness perception and characterise their biosynthetic pathways. However, to our knowledge, the aim of improving the content of these sweetness-enhancing volatiles has not received adequate attention in tomato quality improvement. Moving forward, if the contribution of these

volatile compounds to sweetness perception can be systematically evaluated via sensory assessment assays, and their biosynthetic pathways further deciphered, it will be feasible to generate novel tomato germplasm with unaltered sugar content but significantly enhanced sweetness perception through gene editing and other advanced biotechnologies.

To date, in-depth characterisation of sweetness-enhancing metabolites and their regulatory networks has yielded abundant valuable breeding targets for high-quality tomato improvement. First, for major identified QTLs regulating sugar accumulation or sweetness perception, molecular markers can be developed to facilitate marker-assisted selection (MAS), thereby accelerating the genetic improvement of high-quality tomatoes [75]. Second, for key regulators with well-characterised functions, such as *SIL1n5*, *SICDPK27* and *SITST2*, direct gene editing technologies, including prime editing (PE) and protein-directed evolution, can be employed to create elite allelic variants not found in nature, thus advancing tomato breeding efforts [48, 76–78]. Furthermore, wild germplasm harbours extensive genetic variation, which has traditionally been exploited in breeding via backcrossing and introgression [20, 75]. However, desirable alleles in wild germplasm are frequently tightly linked to genes controlling undesirable traits, for example, fruit weight, disease resistance, and stress tolerance, making it difficult to simultaneously achieve high quality, high yield, and strong stress resistance in breeding [7, 10, 15]. With the simultaneous advancement of tomato functional genomics, genome editing, and genetic transformation technologies, *de novo* domestication strategies can now be applied to directly modify wild tomato species, accelerating breeding progress and developing elite varieties with balanced multiple traits [79, 80]. Currently, artificial intelligence (AI)-driven precision design breeding has been increasingly applied in crop genetic breeding [70, 81]. With the accumulation of large-scale tomato genomic, transcriptomic, metabolomic, and high-throughput phenomic datasets [7, 15, 25, 82], AI-assisted genomic selection strategies may also be leveraged in the future to enable breeders to rapidly select target materials and develop ideal tomato germplasms integrating multiple desirable traits [70, 83].

Fruits can be categorised into dry fruits and fleshy fruits according to the morphological and structural characteristics of their pericarps. Within the group of fleshy fruits, the tomato has emerged as a well-recognised model system for investigating the mechanisms underlying plant fruit development and ripening [64, 84, 85]. Specifically, the tomato fruit is classified as a berry, characterised by a fleshy and thick pericarp that develops from the ovary [86, 87]. In contrast, other commonly consumed fruits exhibit distinct developmental origins. For instance, strawberry, pineapple, and apple are derived from the receptacle tissue or from expansion of the sepals [88, 89]. Despite these differences in morphology and developmental origin, most fruits share common traits, including the presence of seeds and a synergistic regulatory interaction between seed development and fruit ripening. Notably, the sugar brake genes, *SICDPK27/26*, are conserved across various fruit crops [48]. Additionally, sweetness-enhancing volatile metabolites have been identified in a range of other fruits [51–53]. These findings collectively indicate that the aforementioned strategy for enhancing sweetness in tomatoes is also applicable to the improvement of other

fruit species. Particularly for high-sugar fruits such as lychees and mangoes, if the biosynthetic pathways of sweetness-enhancing volatiles could be precisely modulated, it would enable a reduction in soluble sugar content without impairing the perceived sweetness of these fruits. Such an approach preserves the sensory properties of the fruits while lowering their glycaemic index, thereby providing healthier fruit alternatives for individuals with diabetes or those seeking balanced dietary options.

Collectively, these combined strategies mentioned above hold significant potential for improving the sweetness of tomato and other fruits while maintaining other agronomic performance.

Author Contributions

Jie Chen: writing – original draft, funding acquisition, writing – review and editing, investigation, formal analysis, validation. **Hongjun Lyu:** investigation, funding acquisition, writing – original draft, writing – review and editing, validation, formal analysis. **Jinzhe Zhang:** conceptualization, writing – review and editing, project administration, funding acquisition.

Acknowledgements

This work was funded by the Key Research and Development Program of Guangdong Province (2021B0707010005), National Natural Science Foundation of China (Grant Nos. 32522094 and 32302581), National Key Research and Development Program of China (2021YFF1000103), Key R&D Program of Shandong Province, China (2023LZGCQY004).

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

Data sharing is not applicable to this article, as no new raw data were generated in this study.

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