Research suggest that SlIDI1 is involved in tomato carotenoid synthesis in a complex way

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In recent work, researchers from the Beijing Academy of Agriculture and Forestry Sciences and the Chinese Academy of Sciences characterized the molecular mechanism of color formation in an orange-

fruited tomato inbred line, orange fruited tomato3 (oft3). Using high-performance liquid chromatography (HPLC), they found that oft3 fruit had a markedly reduced carotenoid content, as well as a higher β-carotene/lycopene ratio during ripening. Further genetic analysis through crossing experiments suggested that oft3 was controlled by a single recessive gene. Bulk segregant analysis by high-throughput sequencing (BSA-Seq) and fine mapping combined with genome sequence analysis identified SlIDI1, which harbored a 116-bp deletion, as the candidate gene for the oft3 locus. Functional complementation and CRISPR-Cas9 knockout experiments confirmed that SlIDI1 was the causal gene.

Next, the authors confirmed that SlIDI1 produced both long and short transcripts simultaneously by alternative transcription initiation and alternative splicing. Expression of a green fluorescent protein fusion revealed that the long isoform was mainly localized in plastids and that an N-terminal 59-amino acid extension sequence was responsible for its plastid targeting. Short transcripts were identified in leaves and fruit by 5′ RACE and in fruit by 3′ RACE; their corresponding proteins lacked transit peptides and/or putative peroxisome targeting sequences, respectively.

It is widely known that IDI1 functions in the MEP pathway upstream of PSY1, which catalyzes the first committed step of carotenoid biosynthesis. Intriguingly, the authors found that tomato carrying a mutated SlIDI1 gene showed an orange-fruited phenotype and not a yellow color as observed in the r (SlPsy1) mutant. To explain this surprising phenomenon, they measured the expression levels of key carotenoid biosynthesis genes (SlPSY1, SlPDS, SlCRTISO, SlLCY-B1, SlLCY-B2, and SlLCY-E) in two oft3-genotyped BC1F2 individuals. However, no significant changes were observed in any of these genes relative to their expression in wild-type plants. Finally, SlBCH1, which is reported to encode β-carotene hydroxylase, was found to show transcriptional repression in oft3 and CRISPR-Cas9-generated mutants.

Because SLBCH1 catalyzes the transformation of β-carotene into other xanthophylls, the authors speculated that the decrease in SlBCH1 transcripts delayed β-carotene catabolism, minimizing reductions in β-carotene accumulation and contributing to the orange-fruited phenotype of the Slidi1 mutant. These results suggested the existence of a novel feedback loop in carotenoid pathway flux. This study appears in the journal *Horticulture Research*.

The authors said, "SlIDI1 could be targeted to multiple organelles via the expression of different isoforms. However, whether SlIDI1–5′S and SlIDI1–3′S were derived from the same transcript, as well as whether SlIDI1 could be targeted to mitochondria or peroxisomes, were not confirmed yet. Further studies are needed to address this question."

More information: Ming Zhou et al, Alternative transcription and feedback regulation suggest that SlIDI1 is involved in tomato carotenoid synthesis in a complex way., *Horticulture Research* (2022). DOI: 10.1093/hr/uhab045

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