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Regulation of trehalose/sucrose-non-fermenting-related protein kinase1 and its signaling pathways involved in drought stress responses

Abstract

Trehalose/sucrose-non-fermenting-related protein kinase1 (Tre/SnRK1) interaction regulates a myriad of plant responses to drought stress by regulating cell signaling pathways implicated in gene expression, metabolism, protein and membrane stabilization, sink-source balance, hormone homeostasis, carbohydrate allocation and use, and photosynthesis. It has been shown that protein-protein interactions, post-translational modifications, and phytohormones such as abscisic acid (ABA) regulate the activity and signaling pathways of SnRK1. The aim of this review is to incentivize further studies on the elucidation of molecular mechanisms underlying the interactions between Tre/SnRK1 signaling pathways with mitogen-activated protein kinases (MAPKs), domain of unknown function (DUF) 581, and calcium-dependent protein kinase (CDPK) for improved drought tolerance and performance.

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Abbreviations: ABA, abscisic acid; CDPK, calcium-dependent protein kinase; DUF, domain of unknown function; JA, jasmonic acid; MAPK, mitogen-activated protein kinases; NO, nitric oxide; PA, phosphatidic acid; PLDα1, phospholipase Dα1; SnRK1, sucrose-non-fermenting-related protein kinase 1; TOR, target of rapamycin; Tre, trehalose; TPP1, trehalose phosphate phosphatase1; Tre6P, Trehalose-6-phosphate; Tre6PP, trehalose-6-phosphate phosphatase; Tre6PS, trehalose-6-phosphate synthase; UDP, uridine diphosphate

Introduction

Trehalose (Tre; α-D-glucopyranosyl-(1→1)-α-D-glucopyranoside) is a non-reducing disaccharide composed of two molecules of glucose that functions as a compatible solute for the stabilization of biological structures (proteins and membranes) under various abiotic stresses in plants, bacteria, fungi, yeasts, insects, and invertebrates.[1–4] Genetic modification of Tre biosynthesis and regulation of its signaling pathways has the potential to improve plant performance and stress responses to meet the rising world's demand for food, which is in direct correlation with demographic trends.[5] Accordingly, this short review discusses the regulatory pathways of Tre biosynthesis and its interactions with other molecular signals that require further studies for designing drought tolerant plants with improved performance.

Trehalose biosynthesis and its regulation

In plants, Tre is synthesized in a two-step process. Trehalose-6-phosphate synthase (Tre6PS) generates trehalose-6-phosphate (Tre6P) from uridine diphosphate (UDP)-glucose and glucose-6-phosphate followed by dephosphorylation to yield Tre by trehalose-6-phosphate phosphatase (Tre6PP).[2] Tre is not thought to accumulate to detectable levels in most plants, with the exception of the desiccation-tolerant "resurrection plants". Among vascular plants only a few desiccation-tolerant resurrection plants, such as *Selaginella lepidophylla* and *Myrothamnus flebellifolius*, accumulate substantial amounts of Tre.[6] In maize, expression of rice (*Oryza sativa) TREHALOSE PHOSPHATE PHOSPHATASE1* (*TPP1*) from the rice *MADS6* promoter, which was most active in reproductive tissues, altered the expression and activity of the gene for SnRK1. Consequently, Tre6P/SnRK1 acted as the central regulator to promote the primary and secondary metabolism balance, assimilate distribution and use, sink-source balance and thus optimized photosynthetic capacity and yield improvement under water stress condition.[7] Upregulation of Tre6P has been shown to repress the expression of genes encoding SnRK1, the molecular mechanisms of which remains to be investigated. Further studies are yet required to elucidate how Tre6P/SnRK1 signaling pathways regulate gene expression at molecular levels. Interestingly, Griffiths et al.[5] study showed that a chemical intervention strategy based on a 'signaling-precursor' concept for permeability can be employed to directly modulate Tre6P uptake and sunlight-triggered release in plants, leading to markedly higher grain yield, drought tolerance and recovery in wheat. Their results suggested that stimulation of synthetic exogenous small-molecule signal precursors can be used to directly enhance plant performance[5]. The finding that Tre6P activates nitrite reductase suggests a possible role for Tre6P in ABA-mediated regulation of stomata conductance, involving covalent modifications of nitrite reductase and SnRK2.6, with nitric oxide (NO) as an intermediary signal.[6] The findings suggest that the correct interpretation of Tre6P effects require the evaluation of variations in Tre6P:sucrose:starch ratio in different cell, tissue, and organ types during day/night cycles at different growth stages in response to environmental changes.[5,6]

Regulation of Tre/SnRK1 activity and its signaling pathways

As a signaling molecule, Tre functions at least partially as an inhibitor of sucrose-non-fermenting-related protein kinase 1 (SnRK1), which results in up-regulation of biosynthetic reactions supporting photosynthesis and starch synthesis among others.[1,6] Tre6P, a central sugar signal in plants, regulates gene expressions and metabolic

pathways involved in plant growth and development *via* SnRK1, and early flowering associated with sucrose, nutrients, and starch supply and allocation, underpinning improvement of crop performance and tolerance to abiotic stresses.[5,6,8–11] Under proper symbiotic relationship, Tre has the higher capacity to confer drought tolerance by regulating hormone homeostasis, essentially ABA and jasmonic acid (JA), transcription factors targeting gene expression, and biosynthesis of osmoprotectants such as proline.[12] Under salinity and drought stresses, intracellular calcium elevation stimulates phospholipase Dα1 (PLDα1)-mediated phosphatidic acid (PA) production which subsequently interacts with SnRKs. PA/SnRKs regulates drought stress responses such as stomata movement by regulating the activity of vacuolar H^+-ATPases. These proton pumps help to maintain the proton gradient that drives Na^+/H^+ antiporter activity.[13,14] Thus, the interaction between PLDα1-induced PA with SnRKs may affect Tre/SnRK1 and thus remains to be investigated. Regulation of SnRK1 and its signaling pathways are highly dependent on post-translational modifications, such as, myristoylation,[15] SUMOylation,[16] phosphorylation and ubiquitination.[17] SnRK1 signaling is under the influence of its interactions with other proteins, such as TARGET OF RAPAMYCIN (TOR),[18,19] MAPKs, DUF 581,[18] CDPK,[20] and phytohormones such as ABA.[21] Therefore, understanding the molecular pathways underlying the relationships between Tre/SnRK1 with these regulatory components provide more information in order to improve drought stress responses in plants.

Conclusions and future remarks

Optimization of Tre/SnRK1 regulation and its signaling pathways to obtain drought tolerance with improved yield depend on Tre precursors, carbohydrate metabolism, post-translational modifications of SnRK1, symbiotic relationships, and Tre/SnRK1 interaction with molecular signals such ABA, TOR, MAPKs, DUF581, and CDPK. Carbohydrate metabolism, which can be improved by Tre/SnRK1 signals, is one of the most important parameters to establish efficient symbiotic performance and thereby stress responses. In this context, breeding programs not only for improved plant Tre/SnRK1 signaling pathways, but also for their symbionts have the potential to improve responses to multiple stresses and symbiotic performance with optimized yield, which require further studies.

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Conflict of interest

The author declares there is no conflict of interest.

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